सत्यमेव जयते

पशुपालन एवं डेयरी विभाग, भारत सरकार

Department of Animal Husbandry and Dairying
Government of India

STANDARD VETERINARY TREATMENT GUIDELINES FOR LIVESTOCK AND POULTRY

Food and Agriculture Organization of the United Nations

USAID

पशुपालन एवं डेयरी विभाग, भारत सरकार

Department of Animal Husbandry and Dairying
Government of India

STANDARD VETERINARY TREATMENT GUIDELINES FOR LIVESTOCK AND POULTRY

Disclaimer

The document on 'Standard Veterinary Treatment Guidelines for Livestock and Poultry' has been developed by the Department of Animal Husbandry and Dairying (DAHD), Government of India with support from the Food and Agriculture Organization of the United Nations (FAO) and the United States Agency for International Development (USAID).

It is based on extant literature on the subject and inputs from senior veterinary professionals. These are merely advocacy guidelines for use of veterinary professionals, paraprofessionals, and animal health workers in India, and should not be interpreted as 'legally binding' or mandatory 'regulation' to be followed by veterinary professionals. The final decision on the management of disease and sick animal including diagnosis, choice of drugs and their dose, duration of treatment, and preventive measures rest on the professional experience and judgment of the attending veterinarians and the clinical status of the sick animals.

While implementing the Standard Veterinary Treatment Guidelines (SVTG), all provisions of the Indian Veterinary Council Act, 1984 (especially Section 30) must be strictly adhered to. These guidelines should be applied only under the supervision of a registered veterinary practitioner, as per the Indian Veterinary Council Act, 1984, to prevent any misuse or misapplication of the guidelines.

While all reasonable precautions have been taken to verify the information contained in this publication, the material is distributed without warranty of any kind, either expressed or implied. The responsibility for the interpretation and use of the material lies with the reader. In no event shall DAHD, Ministry of Fisheries, Animal Husbandry and Dairying Government of India be liable for damages arising from its use.

The present document on "Standard Veterinary Treatment Guidelines" is a living document. It is a first step in an iterative process towards developing a set of guidelines for animal treatment which are user-friendly, sustainable, and provide a framework for economic and consistent veterinary treatment prescriptions. Henceforth, these guidelines will be put to day-to-day use in veterinary practice by a large community of veterinarians which will be a part of ground truthing. Development of this document is a part of a collaborative venture with practicing veterinarians wherein the document will be reviewed updated regularly incorporating the valuable suggestions and feedback - collated over a period of 2-3 years - received from Veterinarians Practicing in field and the newer developments in contemporary veterinary medicine practice globally.

राजीव रंजन सिंह उर्फ ललन सिंह
RAJIV RANJAN SINGH ALIAS LALAN SINGH

सत्यमेव जयते

पंचायती राज मंत्री
एवं मत्स्यपालन, पशुपालन एवं डेयरी मंत्री
भारत सरकार
Minister of Panchayati Raj and
Minister of Fisheries, Animal Husbandry and Dairying
Government of India

DO. No.3109.MIN PR&FAHD/20.24

MESSAGE

I am delighted to know that the Department of Animal Husbandry and Dairying, Ministry of Fisheries, Animal Husbandry and Dairying, Government of India, in collaboration with the Food and Agriculture Organization of the United Nations, has developed a comprehensive standard treatment guideline for major livestock and poultry diseases in India.

India is home to vast livestock resources that provide livelihood opportunities for millions of farmers and contribute to foreign reserves through the export of meat, milk products, eggs, wool, and other animal by-products. A key aspect of raising healthy and productive livestock and poultry is implementing effective strategies to mitigate losses arising from diseases. Animal diseases have multiple impacts, affecting farm-level operations as well as broader trade, welfare, and public health concerns. Endemic diseases primarily impact farms, while epidemic diseases can lead to significant economic repercussions, restricting trade in livestock and livestock products. The occurrence of such diseases marginalizes both poor and affluent livestock producers by limiting their access to higher-priced markets and constraining their capacity for value-added trade.

The development of standard veterinary treatment guidelines will assist animal health professionals in making informed decisions regarding appropriate and recommended treatments for specific clinical circumstances. Additionally, these guidelines will be valuable for policymakers, drug manufacturers, and marketing agencies in promoting better drug stewardship.

I would also like to congratulate the Department of Animal Husbandry and Dairying for leading this initiative, as well as the experts and academia from ICAR, various Veterinary Colleges and Universities, and Industries who have united on a single platform to collectively develop these standard veterinary treatment guidelines for field professionals. Their dedication and efforts will undoubtedly contribute to the betterment of India's livestock sector, ensuring healthier animals, more productive farms, and a more resilient economy.

(Rajiv Ranjan Singh)

Room No. 234, B- Wing, Krishi Bhawan, Dr. Rajendra Prasad Road, New Delhi-110 001 (India),
Ph. +91 11-23380780/81/82 Fax: +91 11-23380783 Resi. 011-23782781/82

प्रो. एस. पी. सिंह बघेल
राज्य मंत्री
मत्स्यपालन, पशुपालन एवं डेयरी
एवं
पंचायती राज मंत्रालय
भारत सरकार

सत्यमेव जयते

Prof. S. P. Singh Baghel
Minister of State
Fisheries Animal Husbandry & Dairying
and
Ministry of Panchayati Raj
Government of india

MESSAGE

I am pleased to learn that the Department of Animal Husbandry and Dairying, in collaboration with the Food and Agriculture Organization of the United Nations, has successfully developed a comprehensive Standard Treatment Guideline for major livestock and poultry diseases in India.

These guidelines will be an essential reference for veterinary professionals and paraprofessionals nationwide, empowering them to make informed and precise decisions regarding the treatment of various livestock and poultry diseases. It is set to greatly improve livestock productivity, reduce the unnecessary use of antimicrobials, encourage the responsible use of medications, and ensure compliance with food safety standards.

These standardized guidelines will not only aid animal health professionals in selecting appropriate treatments for specific clinical situations but will also serve as a crucial tool for policymakers, pharmaceutical manufacturers, and marketers in promoting responsible drug usage.

I express my sincere appreciation to all the stakeholders who contributed to this significant achievement. I am confident that this document will mark a milestone in advancing rational and consistent treatment practices across the country, fostering better health outcomes for our livestock sector.

(Prof. S. P. Singh Baghel)

Office: Room No. 199-J, 1st Floor, Krishi Bhawan, New Delhi-110001
Residence: 7, K, Kamraj Lane, New Delhi-110011
Phone: 011-23380378, 23380379, E-mail: mos-fahd@dahd.nic.in

अलका उपाध्याय, भा.प्र.से.
ALKA UPADHYAYA, IAS
सचिव
SECRETARY

सत्यमेव जयते

भारत सरकार
मत्स्यपालन, पशुपालन एवं डेयरी मंत्रालय
पशुपालन एवं डेयरी विभाग
Government of India
Ministry of Fisheries, Animal Husbandry & Dairying
Department of Animal Husbandry & Dairying
218, A-Wing, Krishi Bhawan
New Delhi-110001

October 11, 2024

MESSAGE

I am pleased to announce that the Department of Animal Husbandry and Dairying (DAHD), Government of India, has taken the initiative to develop Standard Veterinary Treatment Guidelines for the major livestock and poultry diseases prevalent across the country. Spearheaded by DAHD, this effort underscores the government's dedication to enhancing animal health systems and ensuring the welfare of rural communities that depend on livestock and poultry for income and nutrition. The guidelines aim to support rural livelihoods, strengthen food security, and safeguard public health, given the essential role of livestock and poultry in the Indian agricultural economy.

With over 536.82 million livestock and 851.12 million poultry (Livestock Census, 2019), India's varied geography and climatic conditions present significant challenges for disease management. The Standard Veterinary Treatment Guidelines will play a pivotal role in ensuring consistent, quality treatment across the country. They are designed to minimize antimicrobial misuse and enhance the effective control of diseases. The inclusion of ethno-veterinary practices in these guidelines provides additional, cost-effective treatment options for marginal and small-scale farmers, ensuring broader access to quality care.

In this initiative, the Food and Agriculture Organization of the United Nations (FAO) has provided critical technical guidance, ensuring that these treatment guidelines are in line with international standards and reflect best practices in veterinary medicine. The collaborative efforts of other key stakeholders, including ICAR, Veterinary colleges, Universities, NCDC, private sector entities, and NGOs, are also noteworthy, reflecting a shared commitment to improving animal health, safeguarding public health, and ensuring food safety in India.

This milestone marks a significant step forward in our collective effort to promote animal health and welfare while enhancing disease control, public health, and the security of the national food supply chain.

Alka

(Alka Upadhyaya)

e-mail : secyahd@nic.in, Tel.: +91-11-23382608, Fax : +91-1123388006
Website : https://dahd.nic.in

डॉ. अभिजित मित्र
Dr. Abhijit Mitra
पशुपालन आयुक्त
Animal Husbandry Commissioner

भारत सरकार
मत्स्यपालन, पशुपालन एवं डेयरी मंत्रालय
पशुपालन एवं डेयरी विभाग
नई दिल्ली–110001
Government of India
Ministry of Fisheries Animal Husbandry & Dairying
Department of Animal Husbandry and Dairying
Krishi Bhawan, New Delhi-110001

MESSAGE

The publication of the "Standard Veterinary Treatment Guidelines (SVTGs)" marks a significant step forward in ensuring the health and well-being of India's livestock and poultry sectors. These guidelines focus on critical aspects of therapeutic processes like careful identification of signs and symptoms, through and correct diagnosis, and proper use of vaccines (therapeutic), drugs or non-drug treatments options. In addition, these guidelines provide a holistic approach to disease prevention through vaccines and biosecurity measures. The goal is to ensure science-backed treatment protocols, supporting sustainable livestock management.

What sets the SVTG document apart is its synthesis as a comprehensive "Reference Document" that offers expert advocacy tools to veterinarians, paraprofessionals, and community animal health workers (CAHWs). By adopting the latest treatment approaches for a wide range of livestock and poultry diseases, the guidelines prioritize the use of high-quality, cost-effective drugs with proven efficacy, ultimately improving treatment outcomes while ensuring affordability for farmers. A shorter version of the main SVTG document providing tips on diagnosis and treatment is also available as a "Ready Reference" for day-to-day use.

Through this initiative, we aim to curb irrational treatment practices, promote veterinary drug stewardship, and foster compliance with rational antimicrobial use. This is a critical step toward addressing the growing global concern of antimicrobial resistance (AMR), which poses risks to both animal and human health. The consistent application of SVTGs will ensure that antibiotics are used prudently, minimizing the risks of overuse as well as misuse and contributing to food safety in animal-source foods (ASFs).

The creation of this monumental document would not have been possible without the contributions of over 80 experts—veterinarians from diverse backgrounds including academia, research, and field practice—who lent their expertise to make this resource as comprehensive and practical as possible. Comprising nine detailed chapters, the document covers diseases across a range of species, from cattle and poultry to camels, equines, and wildlife, offering a rich repository of knowledge for veterinary professionals.

While this document is comprehensive, it is also designed to be a "living document," open to periodic updates as new insights and practices emerge. It is important to note that the SVTGs serve as advocacy tools and are not legally binding regulations. The final decision on treatment rests with the attending veterinarian, based on their professional judgment and the specific clinical situation, as per the provisions of the Indian Veterinary Council Act, 1984.

We extend our sincere gratitude to the Food and Agriculture Organization of the United Nations (FAO) and the dedicated team of experts for their commendable work in bringing this valuable resource to fruition.

Let us work together to ensure that the "Standard Veterinary Treatment Guidelines" lead to improved animal health management, economic benefits for farmers, and enhanced public health outcomes.

(Abhijit Mitra)

2nd Floor, Chanderlok Building, 15 Janpath, New Delhi-110001
Mob: +91-9412445671, E-mail: ahc-dadf@nic.in

Message from FAO Representative, India

The Food and Agriculture Organization of the United Nations (FAO) recognizes the interdependence of the human health and animal health sectors and highlights the critical importance of strengthening animal health system to improve livestock productivity, safeguard public health, and enhance food security.

This is especially relevant in a country like India, where the diversity of landscapes, animal species, and farming practices presents unique challenges in delivering consistent and effective veterinary care. Additionally, the presence of a heterogeneous animal health service system—comprising professionally trained veterinarians, semi-trained para veterinarians, and untrained community animal health workers – has led to an uneven approach across the country.

In response to these challenges, FAO played a crucial role in developing the Standard Veterinary Treatment Guidelines (SVTGs) for Livestock and Poultry, aimed at standardizing and improving veterinary practices – through rationalization of animal treatment using SVTGs – across the country. These guidelines ensure that veterinary prescriptions are both effective and evidence-based, addressing key areas such as the misuse of antimicrobials and improving the safety of animal-source foods by reducing residues in the food chain.

The SVTGs are envisioned as a "living document," evolving through regular updates based on feedback from veterinarians and ongoing developments in veterinary medicine. These guidelines are part of a long-term, collaborative process with the veterinary community in India, promoting best practices in the animal health sector.

FAO remains committed to strengthening the animal health sector by providing a reliable framework for treatment of livestock and poultry through the SVTGs. General treatment guidelines based on symptoms and clinical signs will assist veterinarians in providing timely care, while disease-specific guidelines will ensure precise and targeted interventions once a diagnosis is confirmed.

The Standard Veterinary Treatment Guidelines for Livestock and Poultry are set to become a cornerstone of veterinary service delivery in India, contributing to healthier animals, safer food products, and a more robust animal health system.

Takayuki Hagiwara
FAO Representative in India

Acknow

Acknowledgment

The Department of Animal Husbandry and Dairying (DAHD), under the Ministry of Fisheries, Animal Husbandry, and Dairying, Government of India, is pleased to announce the successful completion of the Standard Veterinary Treatment Guidelines (SVTGs) for livestock and poultry in India. This significant achievement was made possible through close collaboration with the Food and Agriculture Organization (FAO) and the unwavering support of the United States Agency for International Development (USAID) India.

The SVTGs will empower veterinarians and animal health professionals to deliver standardized, rational treatment, ensuring improved animal health across.

We extend our deepest appreciation to the collective efforts of eminent independent consultants, esteemed reviewers, veterinary experts from various Departments and institutions namely Department of Animal Husbandry and Dairying, Government of India; Indian Council of Agricultural Research (ICAR) Animal Health Institutes, viz., Indian Veterinary Research Institute (ICAR-IVRI), National Research Centre on Equines (ICAR-NRCE), National Research Centre on Camel (ICAR-NRCC), National Research Centre on Pig (ICAR – NRCP) , National Research Centre on Mithun (ICAR – NRC Mithun), the ICAR Research Complex for North Eastern Hill Region (ICAR RC NEH), Central Institute for Research on Buffalo (ICAR-CIRB), Central Institute for Research on Goats (ICAR-CIRG), ICAR- National Institute of Veterinary Epidemiology and Disease Informatics (ICAR- NIVEDI), ICAR-Directorate of Poultry Research, Veterinary Universities and Colleges [Pandit Deen Dayal Upadhyaya Veterinary Science University (DUVASU), Guru Angad Dev Veterinary And Animal Sciences University (GADVASU), Karnataka Veterinary, Animal and Fisheries Sciences University (KVAFSU), Central Agricultural University (CAU), Assam Agricultural University (AAU), Lala Lajpat Rai University of Veterinary and Animal Sciences (LUVAS), G. B. Pant University of Agriculture and Technology (GBPUAT), Tamil Nadu University of Veterinary and Animal Sciences (TAANUVAS), Nanaji Deshmukh Veterinary Science University (NDVSU)], Veterinary Council of India (VCI) and experts from private sector and international agencies.

We sincerely thank our colleagues at FAO for their relentless support and guidance in the development of this document. In particular, we express our sincere gratitude to Mr. Takayuki Hagiwara, FAO Representative in India, Dr. Konda Chavva, Assistant FAO Representative in India (Programme), Ms. Sonia Bhalla, Assistant FAO Representative in India (Admin), Mr. Rajesh Dubey, Dr. Raj Kumar Singh, Dr. Jyoti Misri, Dr. Mohammad Hasib, Dr. Robin J Paul, Dr. Acty George, Dr. Vikram Vashisht, Ms. Ananya Manchanda, and Ms. Bushra Owaisy from the FAO India Office.

This publication is based on the consolidated opinions of experts and does not necessarily reflect the individual views of the Government of India (GoI) or FAO.

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STANDARD VETERINARY TREATMENT GUIDELINES FOR LIVESTOCK AND POULTRY

INTRODUCTION

Background

The Standard Veterinary Treatment Guidelines (SVTG) for livestock and poultry is a document comprising of a chapter on each disease with different sub-heads, *viz*., Definition and Causative agent, Transmission, Clinical signs, Lesions, Diagnosis (including differential diagnosis), Treatment and Control, and Biosecurity Measures. Information provided under various sub-heads enables the veterinary practitioners, paraprofessionals, community animal health workers (CAHWs), and other animal health care workers in making informed treatment decision making in any given animal health condition/situation. The SVTG is advantageous to all the stakeholders who would be either using the SVTG (veterinary practitioners, paraprofessionals, CAHWs, and other animal health care workers) or will be beneficiary (Farmers, animal owners, medicine suppliers, policy makers) of the use of SVTG.

The therapeutic interventions mentioned in SVTG have been designed to offer efficacious and economic treatment options for animal diseases conditions which either contribute in terms of morbidity and mortality or are of economic and/or public health significance. While writing prescription for treatment, considerations have been on devising a prescription for cost-effective treatments based on fewest drugs/medicines necessary for treating the diseases under local conditions. The farmers' perspective has always been high on the radar while writing the SVTG.

General and specific objectives

The SVTG covers treatment for almost all the diseases of livestock and poultry in various sub-heads like non-infectious/systemic/metabolic diseases of ruminants (large and small), infectious diseases of large ruminants; infectious diseases of small ruminants; and standalone chapters each on infectious/systemic/metabolic diseases of pig, poultry, yak, mithun, camel, and equine (horses, donkeys, and mules). There could be similarities amongst a few diseases which are multispecies, but they have written under different species because of differences in disease biology and management. These chapters describe treatment and biosecurity perspective – besides other information – for each disease while supporting information like drug adverse reaction reporting, vaccine adverse reaction reporting, Physiological-Haematological-Biochemical values of all animal species included in SVTG, SOP for Carcass disposal including disposal of carcasses of anthrax-infected dead animals, SOP for disposal of dead foetus, placental membranes, and uterine fluids, etc. are given as annexure.

The SVTG focuses the thinking on critical aspects of the therapeutic process like making correct diagnosis by careful identification of signs and symptoms and effective and proper use of drugs or non-drug treatments that will truly benefit the animal and the farmer.

The SVTG document will help in providing prioritized uniform treatment options for livestock and poultry in each situation. These guidelines will be used by all the authorized animal healthcare workers at all levels within public and private animal health system. The regular use of these guidelines

will lead to uniformity, effectiveness, and economy of scale in animal health prescribing.

Key features of the Guidelines

- The SVTG is developed by a group of about 80 Expert Veterinarians/Clinicians having enormous field experience.
- Standard treatment guidelines have been synthesized for almost all diseases of livestock and poultry common in the country.
- Important clinical diagnostic criteria including differential diagnosis – for all the selected diseases listed in SVTG – are given.
- Drugs/medicine along with dose, route, and duration of treatment, Withdrawal period and potential adverse reactions for a particular drug are written clearly and concisely.
- Decision on the first choice of treatment of a disease depends on the diagnosis of the disease and disease condition in the affected animals.
- Same standard line of treatment will be available for use by all animal healthcare providers which will lead to homogeneity in animal healthcare service delivery.
- The SVTG is also published as digital small, durable pocket size manual – as Ready Reckoner – which makes it convenient to be accessed through mobile phones.
- The SVTG Document should ideally be conjointly with other documents with standard case definitions of animal diseases.
- The SVTG is a dynamic "Live" document and will be updated frequently based on the field experience and globally evolving scenarios and developments in veterinary treatment domain.

Standard Veterinary Treatment guidelines benefit all the stakeholder

Every Stakeholder in animal health chain is a beneficiary of the standard veterinary treatment guidelines. The benefits to category-wise stakeholders are as under:

- **Animal patients:** The livestock and poultry get effective treatment on regular basis, consistency in prescription, treatment with decreased ambiguity, and better compliance.
- **Animal Owners:** The animal owners get quality treatment with standard set of medicine prescriptions, treatment is less-expensive, rational use of medicines makes food safer fetching good price, SVTGs build the confidence and motivates the animal owners for permitting treatment and vaccination of their livestock and poultry.
- **Animal Health Providers:** SVTGs enable correct diagnosis based on pathognomonic symptoms and lesions, decide standard set of treatment which could be the best in that situation, rationalize the treatment leading to a standard set of treatment as and when necessary, in the similar situation.
- **Medicine Supply Chain Stakeholders:** Advantages to medicine supply chain stakeholders include: quality of medicines assessed in field situation, predicted risk or no risk of routinely used medicines as per SVTG, predicted requirement of medicines in that area (local, state, or national level) in short- and long-term, assessment of use of medicines in an area may help in calculation of usage of the drugs/antimicrobials, and forewarning a disease situation, *etc.*
- **Livestock and Poultry Health Policymakers:** SVTG provides basis and methods for livestock and poultry disease control, assessment of quality of medicines and hence quality of animal health services, making budgetary provisions for regular supply of medicines in a particular area based on past supply and use, homogeneity in treatment helps in integrating the procurement supplies as well as integrating the facilities.

Process of "Development of Standard Treatment Guidelines for Livestock and Poultry"

1. Context

India is a vast country with diverse agro-climatic zones, rich animal diversity, varied farming practices, rich and not-so-rich farmers, vast array of traditions of ethnoveterinary treatment practice, inadequate animal health system (hospital infrastructure, laboratory network, workforce, funding, national surveillance system), and a massively heterogeneous prescription and treatment regimens. This heterogeneity in veterinary treatment in India is due to complex web of professionally trained (Veterinarians), professionally semi-trained (Para veterinarians), and untrained (community animal health workers – CAHWs) animal health service providers dealing with farmers majority of whom are not-so-rich with the rest being rich and resourceful. Delivering veterinary services under these situations becomes an onerous task. Veterinary service delivery, therefore, can be facilitated by making available the "Standard Veterinary Treatment Guidelines (SVTG)

for Livestock and Poultry" leading to harmonized prescription.

The wide use of the SVTG – in combination with Ready-Reckoner – will harmonize the animal treatment leading to healthy animals and safe animal-source foods (ASFs); prevent the avoidable usage of antimicrobials, drugs, hormones, and many other medicines which may not be needed in a particular scenario; reduce the antimicrobial, drug, hormone residues in ASFs; and reduce antimicrobial resistance (AMR). The SVTG is envisaged to become the component of animal health services provisioning to ensure evidence-based veterinary treatment and quality of animal care. At animal health system level, the SVTG will help in planning and costing of the services and will become important tool for monitoring and authorizing procedure(s) in a public-funded animal health insurance schemes and thus become indispensable tool – due to built-in quality control, regulatory and planning functions – both for public and private service providers.

2. Objectives for development of SVTG

The objectives of the SVTG development include the following:

i) collate and review the existing standards treatment guidelines available globally,
ii) identify the procedures/conditions for development of Standard Veterinary Treatment Guidelines,
iii) suggest principles/protocols by which the global guidelines are reviewed and incorporated in SVTG document being developed,
iv) develop the SVTG Document - keeping the country (India)-context in view – for adoption in the country.

3. Expected outputs

To develop documents, *viz*.,

i) Standard Veterinary Treatment Guidelines as a "Reference Guide",
ii) SVTG Standalone Chapters on diseases of various animal species – listed as Individual chapters in main SVTG document.
iii) "SVTG Ready-Reckoner" for day-to-day use by Veterinary Workforce in India

4. Expected Outcomes

i) routine practice using SVTGs will facilitate prescription consistency along with effective and economic treatment,
ii) improved food safety due to reduction of antimicrobial/drug residues in ASFs, and
iii) improved 'Veterinary Drug Stewardship' and rational use of drugs in veterinary practice at the field level.

5. Process development of Standard Veterinary Treatment Guidelines for Livestock and Poultry

The infographic below describes the roadmap of the development of SVTGs.

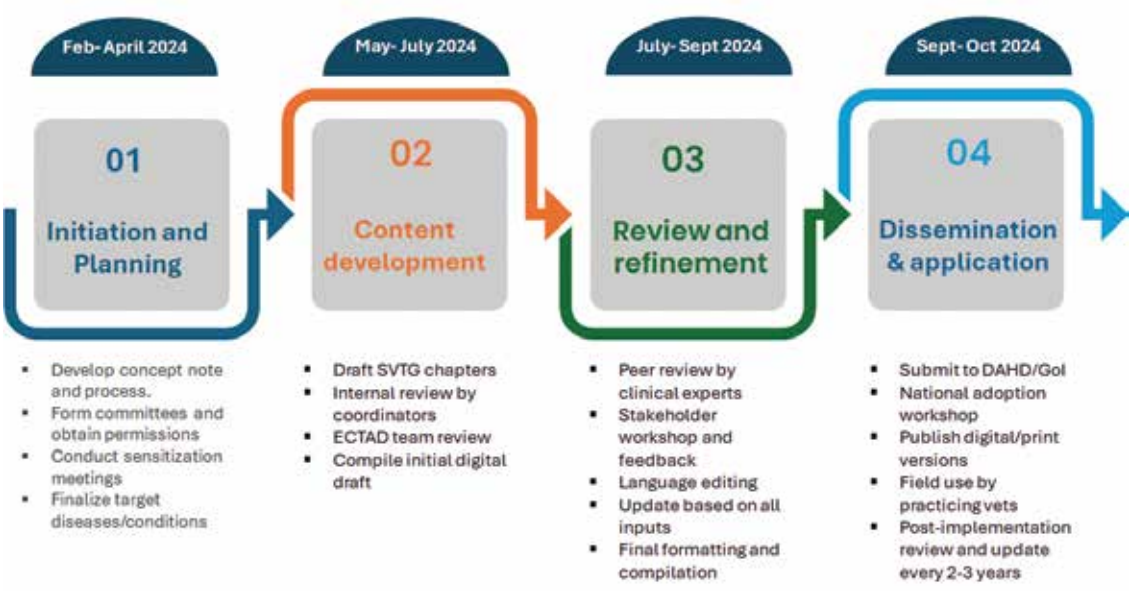


Fig. 1: SVTG ROADMAP DEVELOPMENT

5.1 Defining the scope of the SVTG for Livestock and Poultry: Scope of the proposed document being developed on "SVTG" is based on the ethos of the "prioritization of system-wise disease/ syndromes based on symptoms and clinical signs versus species-wise/Individual diseases. The species-wise individual diseases might initially be seen as a disease/syndrome based on symptoms and clinical signs until laboratory-confirmed diagnoses are available. Till such a time the laboratory confirmation of a disease is obtained, only symptomatic treatment with "General Treatment Guidelines" can be provided on the clinical signs-based presumptive diagnosis. Once the disease/ syndrome is laboratory-confirmed, the treatment would be based on the standard treatment guidelines available for that disease in the Standard Veterinary Treatment Guidelines being developed. This has also been kept in mind - while developing the SVTG document - that antimicrobials for animal treatment are judiciously prescribed. This basic principle has been followed while writing the treatment part in the proposed "Standard Veterinary Treatment Guidelines" document.

5.2 Constitution of committees: The different committees were constituted to facilitate the process of SVTG development.

"Terms of Reference – TOR" of various committees: The following committees are envisaged to facilitate the whole process of development of Standard Veterinary Treatment Guidelines for Livestock and Poultry.

5.2.1 Advisory Committee: This Working Group will comprise of the officials of the Department of Animal Husbandry & Dairying (DAHD), Government of India (GoI); Indian Council of Agricultural Research (ICAR); FAO Officials, selected Eminent Veterinary Experts, and Experts in veterinary ayurveda and ethnoveterinary medicine practice, *etc.*, who will oversee the whole process of the SVTG development and guide the various Teams/Drafting Committees wherever required to ensure timely completion of the quality document.

5.2.2 Core Planning Committee: The Core Planning Committee will comprise of the eminent senior clinical experts, practicing clinical experts, Officials of the DAHD (GoI), Indian Council of Agricultural Research (ICAR), and animal health experts from Academia, research, industry, FAOIN ECTAD Team, *etc.*

The TOR of the Core Panning Committee is to devise the concept, scope, and process flow of the development of SVTG for Livestock and Poultry.

5.2.3 Clinical Expert Committee for identifying, shortlisting, and prioritizing the potential livestock and poultry diseases for which SVTGs are to be developed: The Clinical Expert Committee will comprise of the Professionals

Fig. 2: Infographic depicting different committees constituted to develop the "Standard Veterinary Treatment Guidelines for Livestock and Poultry".

with domain expertise of Veterinary Medicine, Animal Reproduction/Veterinary Gynaecology and Obstetrics, Veterinary Bacteriology and Mycology, Veterinary Virology, Veterinary Pathology, Veterinary Parasitology, Veterinary Surgery and Radiology; Private Veterinary Practitioners; and representatives from Veterinary Council of India; Veterinary/Poultry Associations; Animal Welfare Board of India, *etc*.

The ToR of the Clinical Expert Committee Members include: (i) act as member in Sectional Drafting Committees, (ii) review the existing status of animal diseases prevalent in India, economic losses, implications in human and environmental health, (iii) shortlist the important diseases, prioritize them, and prepare a list of potential diseases and syndromes (System-wise, species-wise) for which treatment guidelines are to be developed, (iv) internal reviewing of the draft standard treatment guidelines - drafted by Drafting Committee – by Coordinators and Co-Coordinators, (v) write down the treatment part in the draft document where drafting committee has not written the treatment, *etc*. The research and academia members from this group will be members of Expert Sectional Drafting Committees wherein the Practicing Veterinary Clinical Experts will help in adding the prescription part in the sectional draft.

5.2.4 Clinical Expert Review Committee for review of Draft Standard Treatment Guidelines developed by Drafting Committees: The Clinical Expert Review Committee will comprise of 10-15 Eminent Veterinary Clinical Experts with rich experience in livestock and poultry treatment. This committee will comprise of domain experts in the areas of Veterinary Medicine, Animal Reproduction/ Veterinary Gynaecology & Obstetrics, Veterinary Surgery and radiology, Veterinary Microbiology (Virology, Bacteriology, Parasitology, Mycology), Veterinary Parasitology, Private Practitioners, *etc*.

The TOR of the Committee includes: (i) review of the list of the potential livestock and poultry diseases as short-listed and prioritized by drafting committee/Clinical Expert Committee, (ii) advise the drafting committee on writing of the draft standard treatment guidelines, (iii) Guide the process of development of SVTG, (iv) review the draft standard treatment guidelines developed by Sectional Drafting Committees/Clinical Experts, *etc*.

Expert Sectional Drafting Committees–Section-wise: Each chapter is designated as Section. The Sectional Drafting Committees will comprise of the scientists and faculty with academia/Research, *viz*., ICAR Animal Science institutes (ICAR-IVRI, ICAR-NRCE/VTCC, ICAR-NIFMD, ICAR-NIVEDI, ICAR-NRC on Camel, ICAR-NRC on Pig, IVAR-NRC on Mithun, ICAR-NRC on Yak, ICAR-CIRB), ICAR-NIVEDI, State Veterinary Universities (SVUs), Veterinary Colleges under State Agriculture Universities (SAUs) and Central Agriculture Universities, and Practitioners from State Animal Husbandry Departments, Officials from DAHD (GoI), *etc*.

Terms of Reference (TOR) of Sectional Drafting Committees include: (i) draft various chapters including Foreword, Acknowledgments, Table of Contents of diseases included in the exercise, draft Section-wise SVTG, (ii) review the existing literature on various sub-heads in a chapter and prepare the draft chapter for each disease – System/syndrome-wise or Individual Disease-wise – giving country-context-specific information which is apt for quick decision making by the Veterinary Practitioners in the field, (iii) review the chapter sub-heads (each chapter will contain: Definition and Causative agent, Case Definition, Transmission and spread, Clinical signs, Lesions, Diagnosis, Differential Diagnosis, Treatment and control, Public Health Risk, Biosecurity measures) and finalize the chapter-wise sub-heads.

Three Drafting Committees for chapters, *viz*., (i) SVTG for systemic/metabolic disease of Small and large ruminants, (ii) SVTG for infectious diseases of large ruminants, and (iii) SVTG of infectious diseases of small ruminants will have one senior person each as Coordinator and Co-coordinator to facilitate smooth conduct of the process of drafting the respective chapters/sections.

Other Drafting Committees - for chapters, *viz*., parasitic diseases of animals, Infectious and systemic disease of poultry, pig, equine, camel, yak and Mithun, along with one chapter on various annexures - will have one senior person as Coordinator to facilitate smooth conduct of the process of drafting the respective chapters/sections.

The Sectional Drafting Committees will be formed, one for each Section/Chapter or a combination of sections as per the details below:

5.2.5.1 SECTION 1: Expert Sectional Drafting Committee for Foreword, Acknowledgement, and Table of Contents: The TOR of this committee will include drafting of Foreword, Acknowledgement, and Table of Contents, any other items as the need arises.

Expert Sectional Drafting Committee for Metabolic/Non-infectious diseases and Systemic diseases of for large ruminants (cattle, buffalo, Yak, Mithun), Small ruminants (Sheep and Goat), poultry, swine, equine, cameline, *etc.*

- **Coordinator:** One Senior member of the Team is designated as coordinator.
- **Co-Coordinator:** Another Senior member of the Team is designated as Co-Coordinator.
- **Members:** A Group of Medicine Experts will be drawn by the coordinator, and they will write SVTG for the sections below. These members will work either in one group only or else sub-committees can be formed. Chairman may decide.

Expert Sectional Drafting Sub-Committees for:

1. Metabolic/Non-infectious Diseases
2. Digestive System Diseases
3. Urinary and Reproductive System Diseases
4. Cardio-pulmonary System Diseases
5. Nervous System Diseases
6. Integumentary System Diseases
7. Musculo-skeletal System Diseases

The above main committee or Sub-committees will draft the standard veterinary treatment guidelines for large ruminants and small ruminants and will also be helping the other Sectional Committees whenever any assistance is required by other sectional groups, like poultry, swine, equine, camel, yak and mithun.

The scope of the systemic disease document is to develop the "General Treatment Guidelines" aimed at providing symptomatic treatment till the disease is laboratory-confirmed. In situations where no standard treatment is available and symptomatic treatment is provided, due care has been exercised for minimal to no use of antibiotics. After the laboratory confirmation, the treatment would be based on the standard veterinary treatment guidelines for that individual disease, as described in the Standard Veterinary Treatment Guidelines.

The ToR for the above committees would be to (i) review the existing frameworks available nationally and globally, (ii) identify the best format to take it up as a starting framework, (iii) modify this framework to country-context, (iv) review the list of diseases under this category to incorporate for development of guidelines, (v) develop the draft chapter framework (syndrome-wise, disease-wise) on treatment guidelines with/without proposed standard veterinary treatment guidelines, (vi) suggest innovative ways of further improving these chapters to make them explicit.

5.2.5.2 SECTION 2: Expert Sectional Drafting Committees for Species-wise individual infectious (Bacteria, Viral, Fungal, Parasitic) diseases for

- Infectious Diseases of Large Ruminants
- Infectious Diseases of Small Ruminants
- Parasitic diseases of animals
- Infectious and non-infectious Diseases of Swine (Pig) diseases
- Infectious and non-infectious Diseases of Poultry diseases
- Infectious and non-infectious Diseases of Equine diseases
- Infectious and non-infectious Diseases of Camel diseases
- Infectious and non-infectious Diseases of Yak and Mithun disease

Details of sections and sub-sections are given in Annexure-1.

5.2.5.3 Section 3: Expert Sectional Drafting Committee for Quick Reference Guide – Ready Reckoner: After drafting the SVTG, the concise form of the same will also be published as a "Ready Reckoner" for Veterinary Practitioners.

5.2.5.4 Section 4: Expert Sectional Drafting Committee for Annexures: the following Annexures will also be placed in the file.

- Drug Adverse Reaction Reporting
- Biosecurity
- Vaccination schedule
- Physiological values in different species
- Biochemical values in different species
- Haematological values in different species
- Ethnoveterinary treatments

- Additional readings
- Table on Methods of carcass disposal
- List of Veterinary Essential Medicines
- List of antibiotics – Medically important Antimicrobials (from AMR Sameeksha)
- List of antibiotics for use in animals (WOAH)

Final List of Chapters in SVTG Document

Chapter 1: Systemic/Non-infectious Diseases of Ruminants

Chapter 2: Infectious Diseases of Large Ruminants diseases

Chapter 3: Infectious Diseases of Small Ruminants diseases

Chapter 4: Parasitic diseases of animals

Chapter 5: Infectious and non-infectious Diseases of Poultry diseases

Chapter 6: Infectious and non-infectious Diseases of Swine (Pig) diseases

Chapter 7: Infectious and non-infectious Diseases of Camel diseases

Chapter 8: Infectious and non-infectious Diseases of Equine diseases

Chapter 9: Infectious and non-infectious Diseases of Yak and Mithun disease

Chapter 10: Annexures

5.2.5.5 Section 5: *List of Contributors* – *available at the end of the Book.*

6. Develop concept note and process for the development of SVTGs

The FAOIN ECTAD Team, in consultation with Officials of the DAHD, Government of India, will synthesize the Concept note and rationalize the whole process involved in SVTG Development Value Chain which starts with conceptualizing the context of SVTG, review of global Standard Treatment Guidelines (STGs) for animals, finalization of the module of the SVTG document to be synthesized for India keeping the country-context in view, constitution of Committees with specific Terms of Reference (TOR) for drafting/ review/advisory/finalization of the document, synthesis of SVTG, internal review of the Draft by Coordinators/Co-Coordinators, technical Review of the draft SVTG, English language editing of the draft SVTG chapters, final technical edit by FAOIN ECTAD Team, collation of chapters in the form of the SVTG Digital document, seeking of comments from Stakeholders, updating of the draft SVTG document by addressing the comments, synthesis of "Ready Reckoner" for day-to-day use by practicing veterinarians, final printing of the SVTG Book and Ready Reckoner.

The **Graphical representation of the** "SVTG Development Value Chain**" is as below:**

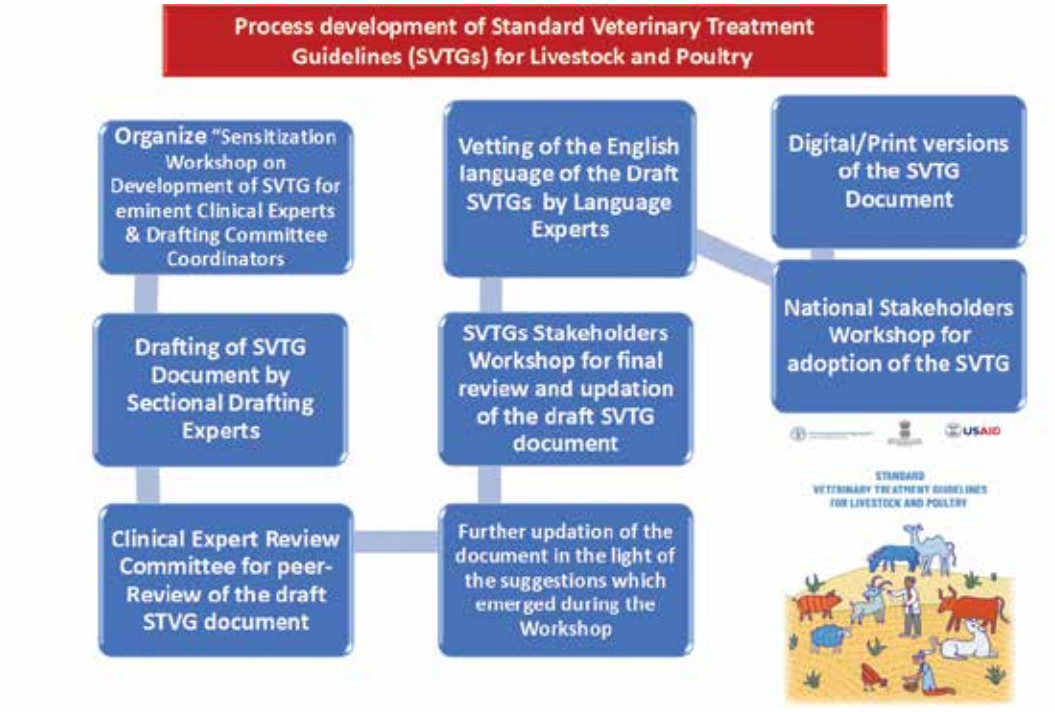


Fig. 3: Infographic on SVTG Development Value Chain

7. Desktop Review activities before organizing the Sensitization Workshop

Collate the information on species-wise diseases, prepare the draft of the SVTG development process, list of various Committees along with Terms of Reference (ToR), identify a format to be followed for writing SVTG, *etc.*

8. Online sensitization of eminent Clinical Experts & Drafting Committee Coordinators on development of SVTG

This series of online/in-person/teleconferencing conversations with Experts and Members/ Coordinators/Co-Coordinators of drafting committees is aimed at sensitizing Expert delegates involved in SVTG development and meeting expectations. After this workshop, delegates will be fully aware about the whole process and be able to guide their colleagues involved in drafting the chapters.

9. Drafting of SVTGs & Ready Reckoner

The respective committees will draft the SVTGs and Ready Reckoner and check both the drafts for plagiarism using standard plagiarism software and provide a certificate with the drafts.

10. Clinical Expert Review Committee for peer-Review of the draft Standard Treatment Guidelines prepared by Expert Sectional Drafting Committee/Clinical Experts Committee.

The ToR of the Clinical Expert Review Committee will comprise of (i) review of the draft SVTGs, (ii) update the treatment profile where needed, (iii) Guide the Drafting Committees for improvement of the technical content, and (iii) Ensure the technical quality of the Pre-final SVTG document.

11. Addressing the comments of Peer Reviewers

The reviewed Draft SVTGs will be reverted to the Clinical Experts Committee/respective Expert Sectional Drafting Committees to address the comments given by the Peer Reviewers.

12. Vetting of the English language of the Draft Manuscript guidelines by Language Expert

The draft manuscript guidelines – after technical review – need to be sent to the English Language Editors for vetting the language to make this a document of international standard.

13. SVTGs Stakeholders (all Experts involved in development of SVTGs) in-person Workshop for final review and updating the completed draft SVTGs

In this workshop, all the experts involved in the complete process of the planning/conceptualizing/ drafting/writing/ reviewing of the SVTGs, other Experts, Officials of the Various Government departments, Representatives from associated stakeholder departments/CSO/NGOs/ others, etc., will attend this workshop for face-to-face interaction and thorough discussion for finalization of this important document.

14. Collating the chapters and sending the SVTG document to Stakeholders for their review and get feedback from Field Veterinarians, higher officials, after collating the chapters, send the chapters to stakeholders, *viz.*, State Animal Husbandry Departments, Higher Officials of the various stakeholder Departments including Health Department, policy makers, ICAR, and all other stakeholders) for review and sending their feedback. Feedback of the Veterinarians in day-to-day practice is most important and valued. Once the comments are received, the document will be updated incorporating the comments.

15. Digital/Print versions of the SVTG Document

Once document is finally technically ready, initiate the Process for making the Digital/Print versions of the SVTG Documents – Complete Reference Document, Standalone Chapters of SVTG of Animal Species-wise Diseases, and Ready Reckoner.

16. National Stakeholders Consultation Workshop for validation and adoption of standard treatment guidelines

A national stakeholders consultation workshop for adoption of the standard veterinary treatment guidelines will be organized which would be attended by the Directors and staff of the State Animal Husbandry Departments; representatives from Veterinary Council of India, Animal Welfare Board of India (AWBI), Indian Veterinary Association (IVA), Animal Welfare Associations, Indian Society of Veterinary Medicine (ISVM), Indian Association of Veterinary Parasitologists (IAVP), Indian Association of Veterinary Microbiologists, Immunologists, and Specialists of Infectious Diseases (IAVMI), Indian Virological

Society (IVS); The Consumer Associations; Patients Association/Union; Indian Federation of Animal Health companies (INFAH); Veterinary Drug Manufacturers, Veterinary Drug Manufacturers Association; Para veterinarians; Community Animal Health Workers (CAHWs); Brooke Hospital for Animals in India; RAIKAS/Camel Association; CSOs working with animals including Camel, Mithun, Yak, and others; selected Members of the SVTG Development Working Committee, *etc*.

The delegates of the above Workshop will get an overall view of the SVTG document and will be able to give (i) way forward for swift implementation of the Standard Veterinary Treatment Guidelines in field and (ii) values suggestions for futuristic improvement.

17. Periodic Updating of the SVTG Document

The SVTG document will remain as a "Live Document" and will be updated periodically (every 2-3 years) incorporating practical suggestions from field veterinary Practitioners and Practicing Experts and incorporating contemporary developments in veterinary practice.

ABBREVIATIONS

AAU – Assam Agricultural University
AEV – Avian encephalomyelitis virus
AGID – Agar gel immunodiffusion
AnHV-1 – Anatid alphaherpesvirus1
AHC – Animal Husbandry Commissioner
AHS – African horse sickness
AlHV-1 – Alcelaphine herpesvirus-1
ALP – Alkaline phosphatase
ALOA – Agar Listeria according to Ottaviani and Agosti
APMV-1 – Avian paramyxovirus type-1
APPs – Acute phase proteins
ARV – Avian reovirus
ASF – African swine fever
ASFV – African swine fever virus
AST – Antimicrobial/Antibiotic sensitivity testing
BD – Border disease
BEF – Bovine ephemeral fever
BEFV – Bovine ephemeral fever virus
BEH – Bovine enzootic haematuria
BEFV – Bovine ephemeral fever virus
bid– Twice a day
BLV – Bovine leukaemia virus
BoHV-1/BHV-1 – Bovine herpesvirus 1
BPI – Bovine parainfluenza
BPV – Bovine papilloma virus
BRD complex– Bovine respiratory disease complex
BRSV – Bovine respiratory syncytial virus
BSE – Bovine spongiform encephalopathy
BTV – Bluetongue virus
BVD – Bovine viral diarrhoea
BVDV – Bovine viral diarrhoea virus
BW – Body weight
CAE – Caprine arthritis and encephalitis
CAEV – Caprine arthritis and encephalitis virus
CAGR – Compound annual growth rate
CAHWs – Community animal health workers
CAM – Chorioallantoic membrane
CAMP – Christie–Atkins–Munch–Peterson
CAU – Central Agriculture University
CAV – Chicken anaemia virus
CBC – Complete blood count
CCE – Camel contagious ecthyma
c-ELISA – Competitive enzyme-linked immunosorbent assay
CCPP – Contagious caprine pleuropneumonia
CCT – Comparative cervical test
CEM – Contagious equine metritis
CFT - Complement fixation test
CHF – Chronic (congestive) heart failure
CIAV – Chicken infectious anaemia virus
CIE – Counter-immunoelectrophoresis
CIRB – Central Institute for Research on Buffalo
CIRG – Central Institute for Research on Goats
CIT – Cervical intradermal test
CLA – Caseous lymphadenitis
CMLV – Camelpox virus
CMT – California mastitis test
CNS – Central nervous system
COD – Cystic ovarian disease
CSF – Cerebrospinal fluid
CSF – Classical swine fever
CSFV – Classical swine fever virus
CT – Computed tomography
DAHD – Department of Animal Husbandry and Dairying
DFA – Direct fluorescent antibody
DH – Diaphragmatic hernia
dl - Decilitre
DRIT – Direct rapid immunohistochemistry test
DUVASU – UP Pt DDU Pashuchikitsavigyan Vishwavidyalaya evam Go Anusandhan Sansthan
EAE – Enzootic abortion of ewes
EAV – Equine arteritis virus
EBH – Enzootic bovine haematuria

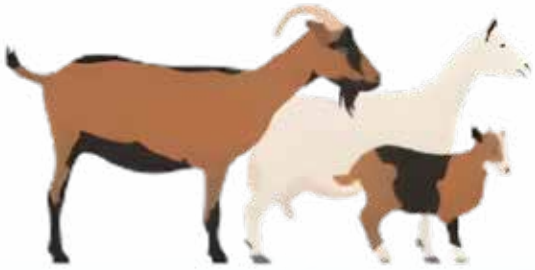
EBL - Enzootic bovine leukaemia
ECG (or EKG) – Electrocardiogram
EER – Equine Exertional Rhabdomyolysis
EHD – Epizootic Haemorrhagic Disease
EHV-1 – Equine herpesvirus-1
EHV-4 – Equine herpesvirus-4
EIA – Equine infectious Anaemia
EIAV – Equine infectious Anaemia virus
EITB – Enzyme-linked immunoelectron transfer blot assay
ELISA - Enzyme-linked Immunosorbent assay
EPG – Eggs per gram
EPM – Equine protozoal myeloencephalitis
EHEC – Enterohemorrhagic *E. coli*
EID_{50} – Egg infectious $Dose_{50}$
EPEC – Enteropathogenic *E. coli*
ETEC – Enterotoxigenic *E. coli*
EVA – Equine Viral Arteritis
EVM – Ethnoveterinary Medicine
FAO – Food and Agriculture Organization of the United Nations
FAT - Fluorescent antibody test
FAdV – Fowl Adenovirus
FAVN – Fluorescent antibody virus neutralization test
FEC – Faecal egg count
FMD – Foot and mouth disease
FMDV – Foot and mouth disease virus
FPV – Fowl pox virus
FTA cards – FTA (Flinders Technology Associates) cards
GADVASU – Guru Angad Dev Veterinary and Animal Sciences University
GANV – Ganjam virus
GGT – Gamma-glutamyl Transferase
GoI – Government of India
GAHV-1 – Gallid Herpesvirus-1
HA - Haemagglutination
HAD - Haemadsorption tests
HCN – Hydrocyanic Acid (HCN) Poisoning
HI – Hemagglutination Inhibition
HPAI – Highly pathogenic avian influenza
HRSV – Human Respiratory Syncytial Virus
HS – Haemorrhagic Septicaemia
IBDV – Infectious Bursal Disease Virus
IBH-HPS – Inclusion Body Hepatitis-Hydropericardium Syndrome
IBR – Infectious bovine rhinotracheitis
IBR/IPV – Infectious bovine rhinotracheitis/ infectious pustular vulvovaginitis
IBT - Immunoblotting test
IBV – Infectious Bronchitis virus
ICTV – International Committee on Taxonomy of Viruses
IDV – Influenza D virus
IFA - Indirect fluorescent antibody
IFAT - Indirect fluorescent antibody test
IFN-γ – Gamma-interferon
IGRAs – Interferon-gamma release assays
IHC – Immunohistochemistry
ILT – Infectious laryngotracheitis
ILTV – Infectious laryngotracheitis virus
IM – Intramuscular
INIBs – Intra Nuclear Inclusion Bodies
IM – Intramuscular
Inj – Injection
IPB – Infectious pustular balanoposthitis
IPT - Immunoperoxidase test
IPV – Infectious pustular vulvovaginitis
IU – International units
IV – Intravenous
IVRI – Indian Veterinary Research Institute
JE – Japanese encephalitis
JEV – Japanese encephalitis virus
kg - Kilogram
KV – killed virus
KVAFSU – Karnataka Veterinary, Animal, and Fishery Sciences University
KVK – Krishi Vigyan Kendra
LA – Large Animal
LAMP – Loop-mediated isothermal amplification
LDA – Left Displaced Abomasum
LFA – Lateral flow assay
LH – Luteinizing Hormone
LPAI – Low Pathogenic Avian Influenza
LPAIV – Low pathogenic avian influenza virus
LSD – Lumpy Skin Disease
LSDV – Lumpy Skin Disease Virus

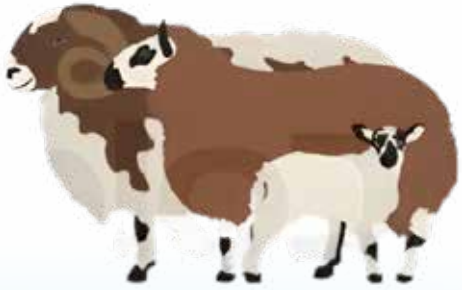
MALT - Mucosa associated lymphoid tissue
MAT – Microscopic agglutination test
MBTC – *Mycobacterium tuberculosis* complex
MCF – Malignant Catarrhal Fever
MCFV – Malignant Catarrhal Fever Virus
MDV – Marek's Disease Virus
mg – Milligram
MHD – Mulberry heart disease
MIT – Mouse inoculation test
ml – Millilitre
mm - Millimetre
MLV - Modified live virus
MLST – Multi locus sequence typing
MoFAHD – Ministry of Fisheries, Animal Husbandry and Dairying
MoAb – Monoclonal antibody
MPD – Medial patellar desmotomy
MRI- Magnetic Resonance Imaging
MVV – Maedi-Visna virus
NADCP – National Animal Disease Control Programme
NDV – Newcastle disease virus
NDDB – National Dairy Development Board
NEB – Negative Energy Balance
NiV – Nipah Virus
NRCE – National Research Centre on Equine
NRCC – National Research Centre on Camel
NSAIDS – Nonsteroidal anti-inflammatory drugs
NSDV – Nairobi Sheep Disease Virus
NTEC – Necrotoxigenic *E. coli*
OEA – Ovine Enzootic Abortion
OvHV-2 – Ovine herpesvirus-2
PCR - Polymerase chain reaction
PCR-RFLP – PCR-restriction fragment length polymorphism
PCV – Packed Cell Volume
PCPV – Pseudocowpox virus
PCV-2 – Porcine circovirus-2
PCV2-ED – PCV2-enteric disease
PCV-2-LD – PCV-2-lung disease
PCV2-RD – PCV2-reproductive disease
PCV2-SD – PCV2-systemic disease
PCV2-SI – PCV-2-subclinical infection
PCVDs – Porcine circovirus diseases
PCVAD – Porcine circovirus-associated diseases
PDNS – Porcine dermatitis and nephropathy syndrome
PDP – Project Directorate on Poultry
PED – Porcine epidemic diarrhoea
PEDV – Porcine epidemic diarrhoea virus
PEV – Post-exposure vaccination
PFGE – Pulsed-field gel electrophoresis
PI-PLC – Phosphatidylinositol-Specific Phospholipase C
PLSD – Pseudo Lumpy Skin Disease
PMN cells – Polymorphonuclear cells
PMWS – postweaning multisystemic wasting syndrome
PO – Oral administration
PPD – Purified protein derivative
PPE – Personal Protective Equipment
ppm – Parts per million
PPRV – *peste des petits ruminants*' virus
PPV - Porcine parvovirus
PRDC - Porcine respiratory disease complex
PRRS – Porcine reproductive and respiratory syndrome
PRRSV – Porcine reproductive and respiratory syndrome virus
PRNT – Plaque Reduction Neutralization Test
Pv1 – Papillomavirus type 1
Pv2 – Papillomavirus type 2
qid (6 h) – Four times a day
qod – Every other day
qRT-PCR – Quantitative RT-PCR
RAPD - Random amplified polymorphic DNA
RBC – Red Blood Cell
RBPT – Rose Bengal Plate Test
RDA – Right Displaced Abomasum
RFFIT – Rapid Fluorescent Focus Inhibition Test
RFLP - Restriction Fragment Length Polymorphism
RFM - Retention of foetal membranes
RT-PCR – Reverse-transcription PCR
RV - Rotavirus
RVF – Rift Valley Fever
RVFV –Rift Valley Fever virus
SA- Small Animal

SAT – Serum Agglutination Test
SC – Subcutaneous
SCC – Somatic cell count
SD – Single dose
SDH – Sorbitol dehydrogenase
sid (24 h) – Every day
SIV – Swine Influenza virus
SNT – Serum Neutralization Test
SPF - Specific–Pathogen–Free
SOP – Standard Operating Procedures
SRMV – Small ruminant morbillivirus
ssRNA – Single-stranded RNA
STAT – Standard Tube Agglutination Test
SwPV – Swinepox virus
SVD – Swine vesicular disease
SVDV – Swine vesicular disease virus
SVTG – Standard Veterinary Treatment Guidelines
SVV – Seneca valley virus
TD – Total dose
TGE – Transmissible Gastroenteritis
TGEV – Transmissible Gastroenteritis Virus
TIBC – Total Iron Binding Capacity
tid (8 h) – Three times a day
TRP – Traumatic Reticuloperitonitis
USAID – United States Agency for International Development
VCI – Veterinary Council of India
µm - Micrometre
VNT – Virus neutralization test
VS – Vesicular stomatitis
VE – Vesicular exanthema
WNF – West Nile Fever
WNV – West Nile virus
WOAH – World Organization for Animal Health

CHAPTER- 1

GUIDELINES FOR NON-INFECTIOUS/ SYSTEMIC DISEASES OF RUMINANTS
(SMALL AND LARGE)





1.1 Preamble

1.2 Production/Metabolic Diseases

1.2.1 Milk Fever
1.2.2 Ketosis
1.2.3 Hypomagnesemic Tetany
1.2.4 Downer Cow Syndrome
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1.1 Preamble

The Indian livestock sector is an important agrarian sector which has emerged as a vital pillar of national economic landscape. Small-scale farmers, rural communities, and marginalized sections of the society rely heavily on livestock rearing as a means of subsistence and livelihood enhancement. India has world's highest livestock comprising 535.78 million with 192.49 million cattle, 109.85 million buffaloes, 148.88 million goats and 74.26 million sheep. In spite of adopting good management practices over a period of time, livestock are susceptible to several types of infectious and/or non-infectious diseases, which not only adversely impact the health of the animals but also the productivity to a great extent. Prophylactic vaccination and treatment constitute important components of livestock disease prevention and control programmes. India being a huge country with rich animal biodiversity and varied climatic and environmental conditions needs an effective animal health system through Standard Veterinary Treatment Guidelines (SVTGs) for optimising productivity. The Guidelines are aimed to rationalize veterinary practice and protect the respective animal population from irrational therapy and hazardous consequences. Rationalizing animal health care reduces costs on animal health system and thus making the livestock enterprise more profitable. In this chapter, the Guidelines are framed based on the body system involved and diseases associated with these systems, *viz*., production/metabolic diseases, digestive system, nutritional deficiency diseases, respiratory system, urinary system, musculo-skeletal system, nervous system, hematopoietic system, skin diseases, cardiac system, reproductive system, and congenital diseases. Further, concise information on drugs and hormone usage in addition to ethnoveterinary medicines (EVMs) and formulations are made available for ready reference in the Annexures and Additional Readings.

1.2 Production/Metabolic Diseases

1.2.1Milk Fever

Definition and Etiology

Milk fever – also known as parturient paresis, hypocalcaemia, paresis puerperalis, parturient apoplexy – is a condition of adult dairy animals wherein severe deficiency of calcium (acute hypocalcaemia) causes acute to per acute, afebrile, flaccid paralysis most commonly at or just after parturition. Dairy animals are at considerable risk for hypocalcaemia at the onset of lactation caused by an imbalance where calcium output in colostrum exceeds calcium influx from the intestine and bone into the extracellular pool.

Predisposing Factors

Dietary deficiency of calcium.

Clinical Signs

Three stages of milk fever include: Stage-1 - a brief stage of excitement and tetany with hypersensitivity and muscle tremor of the head and limbs, the animal does not eat and is reluctant to move, there may be a slight shaking of the head, protrusion of the tongue, and grinding of the teeth, and the rectal temperature is usually normal to slightly above normal; Stage-2 – a prolonged sternal recumbency with a lateral kink in the neck or the head turned into the flank; the muzzle is dry, the skin and extremities are cool, rectal temperature is subnormal, there is a marked decrease in the absolute intensity of the heart sounds and an increase in rate (about 80 bpm), and the ruminal stasis, secondary bloat and constipation are common; Stage-3 - lateral recumbency, animal is almost comatose, the limbs may be stuck out with complete flaccidity on passive movement and cannot assume sternal recumbency on its own.

Diagnosis

Diagnosis is based on history and clinical signs. Confirmation of hypocalcaemia is through the response to treatment with calcium borogluconate. Total serum calcium levels are reduced to below 5 mg/dl; and in severe cases as low as 2 mg/dl.

Differential Diagnosis

Hypophosphatemia, hypomagnesaemia, downer cow syndrome, fat cow syndrome, carbohydrate engorgement, per acute coliform mastitis, aspiration pneumonia, acute diffuse peritonitis, maternal obstetrical paralysis, dislocation of coxofemoral joint.

Treatment

Treat with calcium borogluconate (23 percent solution, 400-800 mL, slow IV in large animals and 50-100 ml in small animals) or calcium magnesium

borogluconate (1.86 percent calcium borogluconate, 5 percent magnesium hypophosphite and 20 percent dextrose anhydrous, 450-800 ml, slow IV in large animals and 50-100 ml in small animals), depending upon the severity of the condition. Repeat the treatment after 10-12 hours, if needed. The heart should be auscultated throughout the administration for the evidence of gross arrhythmia, bradycardia, and tachycardia. If any of this condition occurs, the intravenous administration should be interrupted and continued only after the heart sounds return to normal and use a course of antihistaminic drug (pheniramine maleate @ 1-2 mg/kg, IM).

Prevention and control

Dietary management during the transition period (before and after calving). Administer calcium gels orally at the time of parturition and vitamin D immediately before parturition to enhance the mobilization of calcium. Feeding a prepartum diet with a DCAD (dietary cation-anion difference) between 50 and 150 meq/kg of diet dry matter is generally optimal for the prevention of parturient paresis. Supplementing anionic salts such as calcium chloride, magnesium chloride, calcium sulphate, and magnesium sulphate before calving twice weekly during prepartum period.

Sample collection for Diagnosis

Blood.

1.2.2 Ketosis

Definition and Etiology

Ketosis is a multifactorial disorder of energy metabolism in high yielding dairy animals after calving. The negative energy balance (NEB) results in hypoglycaemia, ketonemia (accumulation of acetoacetate, β-hydroxybutyrate and acetone in blood) and ketonuria. The causal factors of ketosis are (i) dietary factors (starvation/loss of appetite or feeding of low carbohydrate diet/excess feeding of protein-rich diet and silage), (ii) animal factors (heavy drain of lactose (45 g/kg milk) in high yielding animals through milk leads to NEB along with hepatic insufficiency), and (iii) hormonal factors (absence of glucocorticoids/gluconeogenesis in stress conditions such as pregnancy and parturition).

Predisposing Factors

It is disease of lactating crossbred (high yielding) stall-fed cows and buffaloes. Incidence is higher during third to fifth lactation between two weeks to two months after calving.

Clinical Signs

The two clinical forms of ketosis are (i) Wasting form - common form, refusal to eat grain, decrease in milk yield, loss of body weight (woody appearance), depression, abdominal pain, hangdog appearance and odour of ketones in breath and milk, and (ii) Nervous form - encircling straddling, head pushing, blindness and vigorous licking of skin or inanimate objects and hyperesthesia.

Diagnosis

Diagnosis is based on history, clinical signs, milk and urine cow-side tests (Rothera's test), and milk fat to protein ratio. Milk and urine ketone concentrations are detected by the reaction of acetoacetate with sodium nitroprusside. Milk fat concentration tends to increase and milk protein concentration tends to decrease during post-partum NEB.

Differential Diagnosis

Wasting form of ketosis should be differentiated from diseases characterized by wasting such as traumatic reticulitis/pericarditis/diaphragmatic hernia, vagus indigestion, pneumonia, metritis/cystitis/pyelonephritis and abomasal displacement. Nervous form of ketosis should be differentiated from tetanus, rabies, encephalitis, lead poisoning and lactation tetany/ hypomagnesemia.

Treatment

Treatment includes: Replacement therapy - Dextrose (25 percent, 500-1,000 mL, IV for 2-3 days), Propylene glycol (@ 225 g PO every 24 hours for 3-5 days) and sodium propionate (@ 110-225 g PO for 3-6 days); hormonal therapy - glucocorticoids such as dexamethasone sodium phosphate (@ 40 mg, IV, 4 to 6 days) along with IV glucose therapy; long-acting insulin (@ 200-300 U, SC) along with IV glucose and supportive therapy (vitamin B_1/ B-complex @ 10 ml IM on alternate day; feeding of mineral mixture containing phosphorus and cobalt).

Prevention and Control

Avoid starvation or overfeeding at calving, give extra concentrate ration (1.5 kg/day) during advanced pregnancy. In late lactation cows, increase the energy supply from digestible fibres and reduce the energy

supply from starch. Early lactation rations should be relatively high in non-fibre carbohydrates (38-41 percent). Monensin sodium (@ 300 mg/head/day) throughout the transition period may be used in preventing subclinical ketosis.

Sample Collection for Diagnosis

Blood, urine, milk.

1.2.3 Hypomagnesemic Tetany

Definition and Etiology

Hypomagnesemic tetany in cow (lactation tetany/ grass tetany/grass staggers) is a complex metabolic disorder characterized by hypomagnesemia (<1.5 mg/dl in serum), hyperexcitability, muscular spasm, seizures, respiratory distress, collapse, and death. It occurs in calves aged between 2 and 4 months which are fed exclusively on milk because of the fall in the magnesium absorption level from 87 percent at 2-3 weeks to 32 percent at 7-8 weeks of age.

Predisposing Factors

Hypomagnesemia arises during inclement weather due to starvation, transport, grazing on immature grass pasture containing <0.2% Mg on a DM basis. Absorption of magnesium increases with an increasing Na:K ratio (5:1), and it is impaired if ratio is <3:1.

Clinical Signs

Acute lactation tetany – twitching of the muscles and ear, severe hyperesthesia, continuous bellowing, frenzied galloping, staggering gait. During convulsive episodes, there are nystagmus, champing of jaws, frothing at the mouth, pricking of ears, and retraction of eyelids. Hyperthermia (40° to 40.5°C), and increased pulse and respiratory rates. Subacute lactation tetany - gradual onset of clinical signs which usually takes 3 to 4 days inappetence, wildness of facial expression and exaggerated limb movements, spasmodic urination and frequent defecation, decreased ruminal contractions, muscle tremor, mild tetany of hind legs and tail with an unsteady, straddling gait, and retraction of head and trismus. Chronic hypomagnesemia – sudden death with no premonitory clinical signs. Few animals show vague syndrome like dullness, unthriftiness and reduced milk yield. Clinically, in calves, the condition is characterized by hyperesthesia, shaking of the head, opisthotonos posture, retraction of the eyelids, muscle tremors of limbs and frothing at the mouth. Clinical signs occur when serum magnesium concentration falls below 0.3-0.7 mg/dl.

Diagnosis

Diagnosis is based on clinical signs and is confirmed by response to treatment. Laboratory test for measurement of blood magnesium levels (normal range in cattle: 1.8-2.4 mg/dl and sheep: 2.2-2.8 mg/dl) and low CSF (<1 mg/dl) level. Herd diagnosis can be accomplished by estimating the urinary magnesium concentration (normal level: 1-20 mg/dl). Low urine Mg is good presumptive evidence of hypomagnesemia. Urinary magnesium concentration below 1 mg/dl indicates danger of tetany. Some affected animals may have concurrent hypocalcaemia and hypophosphatemia.

Differential Diagnosis

Rabies (History of dog bite, abnormal vocalization, aggressive behaviour), lead poisoning (anaemia, blindness, hyperesthesia), nervous ketosis (sudden drop in the milk yield, anorexia), hypovitaminosis A (blindness, absence of ocular reflexes) and nitrate poisoning (chocolate brown colour mucous membrane).

Treatment

Treat with calcium magnesium borogluconate (1.86 percent calcium borogluconate, 5 percent magnesium hypophosphite and 20 percent dextrose anhydrous, 400-500 mL, slow IV in large animals and 50-100 ml in small animals), depending upon the severity of the condition. Alternatively, administer magnesium sulphate (@ 200 ml of 50 percent solution, SC). Provide magnesium sulphate (100-200 g) daily in the diet at least for 3 to 5 days after initial intravenous infusions. Administer xylazine (@0.05 mg/kg IM) to reduce convulsions. In calves, treat with magnesium sulphate injection (100 ml of 10 percent solution, slow IV) followed by oral supplementation of MgO (10 g/day).

Prevention and Control

Animal at high risk should be moved to low-risk pastures during the grass tetany season. Daily feeding of at least 60 g of magnesium oxide (MgO) or magnesium chloride per day to lactating dairy cows.

Sample Collection for Diagnosis

Blood, urine, CSF.

1.2.4 Downer Cow Syndrome

Definition and Etiology

Downer cow syndrome is a group of syndromes noticed as a complication to milk fever in high yielding dairy animals and is characterized by prolonged recumbency despite treatment with intravenous calcium preparation. Traumatic injury to limb muscles and nerves prior to parturition or at the time of parturition may also lead to downer.

Predisposing Factors

Delayed treatment of milk fever, traumatic injury to sciatic nerve and pelvic muscle, serum electrolyte abnormality (Na, K, Mg, Ca) and slippery floor in the farm premises.

Clinical Signs

Alert downers – the animal is in sternal recumbency with normal feed and water intake. Pulse and heart rates are initially normal but increase later. Creepers – alert downer which makes repeated attempts to get up. Non-creeper – alert downer cow, which makes no attempt to raise and prefers to remain in recumbency state. Non alert downer – inappetence to anorexia, lateral recumbency.

Diagnosis

History of recent parturition, dystocia, and prolonged recumbency. Elevated level of muscle specific enzymes, *viz.*, CPK (normal level - 40-280 IU/litre) and AST. Decreased serum level of calcium, phosphorus and magnesium. Mild to moderate proteinuria and ketonuria. Postmortem lesions include haemorrhage and degeneration in thigh muscles.

Differential Diagnosis

Coliform mastitis (presence of systemic signs and discolouration of milk with enlarged udder) and fracture in limb (radiographic evidence of hip bone and or femur fracture).

Treatment

Provide fluids and electrolyte therapy, which includes isotonic Ringer's lactate, calcium and magnesium infusions. Analgesics and NSAID (phenylbutazone) or steroid (prednisolone) may be preferred. Providing comfortable bedding and to roll the animals from side to side several times daily to avoid decubital ulcers. Assisted lifting using mechanical or electrical hip lifters or body sling may be helpful. Use broad spectrum antibiotic such as streptopenicillin to prevent secondary bacterial infection, vitamin E and selenium and anti-histaminic for effective management.

Prevention and Control

Prognosis depends upon the cause of recumbency. Syndrome can be prevented by prompt treatment of animals for milk fever and providing proper bedding material.

Sample Collection for Diagnosis

Blood.

1.2.5 Post-parturient Haemoglobinuria

Definition and Etiology

Post-parturient haemoglobinuria, a sporadic condition, most commonly affects high-yielding dairy animals at the onset of lactation. It is characterized by development of acute intravascular haemolysis often associated with haemoglobinuria leading to potentially life-threatening anaemia.

Predisposing Factors

High yielders.

Clinical Signs

Haemoglobinuria, loss of appetite, weakness, decrease in milk production, pale mucous membranes and increased cardiac and jugular pulse. Dung typically gets dry, brownish and firm. Dyspnea and tachycardia are common. Jaundice may be seen in the advanced stages.

Diagnosis

Diagnosis is based on clinical signs, particularly dark urine and anaemia during the characteristic stage of lactation. Lactating cows in an affected herd may have moderately low levels of phosphorus (2-3 mg/dl), however, affected animals have extremely low levels of phosphorus (0.4 to 1.5 mg/dl). Total erythrocyte count and haemoglobin levels are greatly reduced.

Differential Diagnosis

Leptospirosis, Bacillary haemoglobinuria, anaplasmosis, babesiosis and chronic copper poisoning.

Treatment

Whole blood transfusion (~5 litre of blood initially

sufficient up to 48 hours) in severe cases when PCV is <15 percent. Following successful blood transfusions, fluid therapy is essential both for support and to reduce the risk of haemoglobinuria nephrosis. Administer phosphorus in acute cases (60 g sodium acid phosphate in 300 ml of distilled water IV initially, followed by SC injections at 12-hour intervals for three occasions). Sodium acid phosphate (40.3 percent, @ 50 ml, IV once in 12 hours for 3 times) may also be given. Provide dicalcium phosphate supplementation (100-120 g twice daily for 5 days). Additionally, haematinics (iron dextran) are recommended during the recovery period.

Prevention and Control

Ensure adequate intake of phosphorus according to the requirements for maintenance and milk production, particularly in early lactation. A decrease in the incidence of the disease is reported after copper supplementation of cattle in a copper-deficient area.

Sample Collection for Diagnosis

Urine, Blood

1.2.6 Pregnancy toxaemia

Definition and Etiology

Pregnancy toxaemia is one of the most important metabolic diseases of pregnant ewes. Decrease of glucose concentration and concomitant increase of ketone bodies are the primary causes of pregnancy toxaemia. The clinical disease occurs when there is a marked decline in the plane of nutrition during the last 4-6 weeks of gestation. The ewes carrying twins or triplets are more vulnerable due to inability to meet the demand of energy on account of rapid foetal growth.

Predisposing factors

Deprivation of food, management practices such as crutching, shearing, change of environment, or drenching during the latter half of pregnancy, obesity, heavy worm infestation with *Haemonchus contortus* and triplet or twin lambs are the key predisposing factors.

Clinical Signs

Separation from the flock, lack of interest towards feed intake, head pressing against obstacle, star gazing posture, convulsions, incoordination in walking, constipation, and grinding of the teeth. In later stages, severe nervous signs with the tremors of the muscles of the head cause twitching of the lips, lateral deviation of the head, circling movement followed by recumbence. Affected ewes commonly have difficulty in lambing.

Diagnosis

Diagnosis is based on the history of pregnancy and clinical signs. A smell of ketones on the breath of the ewe is considered as early diagnostic indicator. Further, diagnosis is done by examining the glucose level in blood. Low blood glucose level (<30 mg/dL) is an indicative of the early stages of the disease. Elevation of ketone bodies in serum (ketonemia) and urine (ketonuria) are constantly found in pregnancy toxaemia. The serum β-hydroxybutyrate concentrations (>3,000 µmol/L) and plasma cortisol (>10 ng/mL) are indicative of pregnancy toxaemia. Sheep develop liver dysfunction, a severe metabolic acidosis, renal failure with a terminal uraemia, and become dehydrated in advanced stage.

Differential Diagnosis

Hypercalcaemia, listeriosis, cerebral abscess, acidosis, uterine torsion, abortion, rabies.

Treatment

Treatment includes fluid and electrolytes (oral drenching @ 3–4 litre by stomach tube, every 6 hours) and glucose (dextrose) for a prolonged period of time. Provide glucose (5-7 g, IV 6-8 times in a day) in conjunction with zinc protamine insulin (20-40 U, SC on alternate days for 3 days). Oral administration of propylene glycol (60 mL, PO, every 12 hours for 3 days, or 100 mL/day) or glycerine (110 g/d) useful to support parenteral glucose therapy. Adding oral supplementation with calcium (12.5 g calcium lactate) and potassium (7.5 g KCl), administration of protamine zinc insulin (0.4 U/kg, SC, every 24 hours) and isoflupredone (0.125-0.25 mg/kg/day, IM) increases survival rates. In severe case, caesarean section is recommended to save the dam.

Prevention and Control

Affected animals should be immediately treated with propylene glycol or glycerol or oral glucose and electrolyte solutions. Avoid excessive grain feeding. Provide adequate nutrition with a concentrate containing 10% protein during last two months of

gestation. Monitoring of glucose (40-60 mg/dL) and β-hydroxybutyrate concentrations (<1,000 µmol/L) in pooled serum samples to identify the latent pregnancy toxaemia during the last 6 weeks of pregnancy.

Sample Collection for Diagnosis

Blood for analysis of glucose, ketone body (β-hydroxybutyrate) and cortisol level.

1.2.7 Neonatal Hypoglycaemia

Definition and Etiology

Neonatal hypoglycaemia is characterized by a lower blood glucose level than normal in newborn calves, which leads to hypoglycaemia and subsequently irreversible brain damage and death. Severe hypoglycaemia is defined when plasma glucose concentration goes <79 mg/dL or 2 mmol/L. Hypoglycaemia in newborn calves occurs due to acute severe diarrhoea, deprivation of milk, endotoxemia and asphyxia.

Predisposing Factors

Malnutrition of dam.

Clinical Signs: Loss of suckling reflex, weakness, incoordination, lethargy, severe depression, recumbency, hypothermia, miosis, seizures, opisthotonus, dyspnea, coma and death.

Diagnosis

Diagnosis is based on the history of malnutrition and clinical signs such as diarrhoea, hypothermia, septicaemia, *etc*. Blood glucose examination in calves with acute severe diarrhoea may fall to below 40 mg/dL (2.2 mmol/litre) in 30-50 percent of cases. Haemato-biochemical examination – leucocytosis or leukopaenia, increase in plasma L-lactate level, hypoproteinaemia, higher serum urea and creatinine concentration. Necropsy findings – no visible lesions, absence of curd in the stomach negligible hepatic glycogen, evidence of septicaemia and generalized peritonitis.

Differential Diagnosis

Septic shock and meningitis.

Treatment

Glucose or dextrose (20 percent solution, @ 2g/kg body weight, IV). Provide warm housing to the neonates. Colostrum feeding (@ 100-200 ml) and supplementation of multivitamin and multimineral preparations.

Prevention and Control

Sustained input of exogenous glucose after first few hours of life of a newborn calf. Colostrum feeding results in two-fold increase in plasma glucose within 1-3 hours, however, the milk replacers may cause marked hyperglycaemia.

Sample Collection for Diagnosis

Blood

1.3 Digestive System Diseases

1.3.1 Simple Indigestion

Definition and Etiology

Simple indigestion is commonly observed rumen disorder of dairy animals and occurs due to the variability in quality and quantity of feed consumed. Generally, dietary abnormalities including poor quality roughage, low protein intake, mouldy feed, and excesses of grain and concentrate intake are the common causes that inhibit the activity of normal rumen microflora.

Predisposing Factors

Accidental access to large quantities of grain, sudden introduction of a new source of grain, non-availability of *ad-libitum* drinking water during dry season, prolonged or high oral dosing with antimicrobials are the key predisposing factors.

Clinical Signs

Reduced appetite with drop in milk production is the first clinical sign of affected animals. Low ruminal movements with moderate tympany are often felt on palpation. The faeces are usually dry and scanty initially followed by softness in consistency. Most cases are generally resolved spontaneously with simple treatments within 2 days.

Diagnosis

History and clinical signs help to diagnose the simple indigestion. However, examining the pH of rumen fluid and activity of the ruminal microflora are additional diagnostic aids. The pH of rumen fluid is examined by using an indicator paper, while the activity of the ruminal microflora by sediment activity and cellulose digestion tests on aspirated ruminal fluid.

Differential Diagnosis

Ketosis, secondary ruminal atony, acidosis and abomasal displacement.

Treatment

Change in diet with good quality roughage. Supplement rumenotorics like ginger, antimony tartarate, ferrous sulphate and cobalt chloride. If the pH is <5.0, treat with 400 g of magnesium carbonate orally. Administration of strained fresh ruminal juice orally by stomach tube to the animals.

Prevention and Control

Sudden change of grain should be avoided. Provide *ad-libitum* drinking water especially during dry period and good-quality green fodder and concentrate.

Sample Collection for Diagnosis

Rumen fluid by stomach tube for rumen flora analysis.

1.3.2 Rumen Impaction

Definition and Etiology

Ruminal impaction is a condition commonly observed in all species of ruminants, such as cattle, buffalo, sheep, and goats; where the contents of the rumen become impacted, or stuck together, and cannot move through the digestive system. It can also occur due to the ingestion of foreign substances or the accumulation of ingested materials such as hair or plastic, causing the stomach to expand and absence of faeces in the rectum.

Predisposing Factors

Inadequate water intake, mineral deficiency, negative energy balance, diets too low in fibres or too high in grains, changes in feed or grazing patterns, and stress are the common predisposing factors. Female animals are more susceptible than males.

Clinical Signs

Poor appetite, decreased rumen contractions, abdominal distension, reduced faecal output, and weight loss. Ruminal impaction can lead to more serious health problems such as rumen acidosis, dehydration, and even death in various species.

Diagnosis

Diagnosis is based on the history of diet. The blood biochemical changes (hypoproteinaemia, hypoalbuminaemia, hypocalcaemia, hypoglycaemia, and hypophosphataemia); haemoconcentration along with clinical signs (tympany, ruminal atony, foamy salivation, absence of faeces in rectum), and imaging techniques (radiography, ultrasonography) help in diagnosing ruminal impaction. The serum analysis reveals hyponatremia (low Na^+), hypokalemia (low K^+) and metabolic alkalosis.

Differential diagnosis

Traumatic reticuloperitonitis, secondary ruminal atony, carbohydrate engorgement, tympany and abomasal displacement.

Treatment

Treatment involves a combination of balanced fluid and electrolyte (@ 80–120 mL/kg daily, IV up to 72 hours) therapy. Supplement rumenotorics like ginger, antimony tartarate, ferrous sulphate and cobalt chloride. Give laxative (mineral oil – 0.5-3 litre) through stomach tube or magnesium sulphate (200-300 g) orally. In severe cases, surgery may be necessary to remove the impacted material.

Prevention and Control

Provide balanced diet containing high fibres and adequate water intake and avoid sudden changes in feeding or grazing patterns. Proper disposal of plastic waste.

Sample Collection for Diagnosis

Rumen fluid collected by stomach tube for rumen flora analysis.

1.3.3 Ruminal Tympany

Definition and Etiology

Ruminal tympany/bloat is abnormal distension of the rumen and reticulum caused by excessive retention of the gases on account of fermentation, either in the form of a persistent foam mixed with the rumen contents or as free gas separated from the ingesta. Primary ruminal tympany (frothy bloat) occurs when leaf material is digested rapidly by microorganisms in the rumen, causing chloroplast particles to be released into the liquid part of the rumen contents. Secondary ruminal tympany (free-gas bloat) causes physical obstruction to eructation as occurs in oesophageal obstruction caused by a

foreign body, stenosis of the oesophagus, pressure from enlargements outside the oesophagus, such as tuberculous lymphadenitis or bovine viral leukosis involvement of bronchial lymph nodes, or obstruction of the cardia. Interference with oesophageal groove function in vagus indigestion and diaphragmatic hernia may cause chronic ruminal tympany. Tympany in cattle is usually accompanied by secondary free gas bloat due to spasm of the oesophagus and inability to eruct normally.

Predisposing Factors

Excessive feeding of leaf and grains, ingestion of foreign body, sudden change in feeding patterns and conditions causing pressure on the oesophagus.

Clinical Signs

Distended abdomen, respiratory distress, increased heart rate, kicking of abdomen and open mouth breathing.

Diagnosis

Based on history, clinical signs and examination of rumen fluid.

Differential Diagnosis

Primary bloat (dietary in origin), Tetanus (limb and tail rigidity, free-gas bloat, prolapse of the third eyelid and hyperesthesia).

Treatment

Passage of stomach tube to relieve the free gas. Use trocar and cannula to relive the gas in severe cases. Treat with simethicone (@100 to 200 ml orally or intra-ruminally) and a course of antihistaminic drug (pheniramine maleate, @ 1-2 mg/kg, IM). Tie a stick in the mouth to promote the production of excessive saliva, which is alkaline and may assist in neutralization of the stable foam.

Prevention and Control

Avoid grazing on pastures with high legume content, especially when they are lush and rapidly growing. Adopt pasture rotation to prevent continuous grazing on high-risk pastures.

1.3.4 Ruminal Acidosis

Definition and Etiology

Ruminal acidosis (lactic acidosis, grain overload, acute carbohydrate engorgement) is a metabolic condition in ruminants marked by decreased blood pH and bicarbonate level caused by overproduction of ruminal D-lactate. Sudden ingestion of toxic doses of carbohydrate-rich feed, such as grains (wheat, barley, and corn). Ingestion of toxic amounts of highly fermentable carbohydrates increases gram-positive bacteria like *Streptococcus bovis* in the rumen, producing large quantities of lactic acid. This lowers rumen pH to ≤5, destroying protozoa and cellulolytic organisms, impairing rumen motility. The low pH allows lactobacilli to produce more lactic acid, increasing osmotic pressure and causing fluid influx into the rumen, leading to fluid ruminal contents and dehydration. Low ruminal pH causes chemical rumenitis, and lactate absorption, especially d-lactate, results in lactic acidosis and acidemia. Consequences include metabolic acidosis, dehydration, haemoconcentration, cardiovascular collapse, renal failure, muscular weakness, shock, and death.

Predisposing Factors

Sudden ingestion of higher amount carbohydrate-rich feed.

Clinical Signs

Clinical signs range from simple indigestion to rapidly fatal acidemia and strong ion (metabolic) acidosis. Typical symptoms of the illness include depression, dehydration, inactivity, weakness, abdominal distension, diarrhoea, and anorexia. Body temperature is usually below normal (36.5° to 38.5°C) but can rise up to 41°C if animals are exposed to the sun. Respiration is shallow and increased, with mucopurulent nasal discharge. Diarrhoea is mostly present and often profuse, with light-colour faeces with a sweet–sour odour. Severe dehydration progresses, and anuria is common in acute cases. Severely affected animals show a staggery, drunken gait and impaired eyesight, with a sluggish or absent palpebral reflex. Acute laminitis can occur, leading to lameness in all four feet, which may resolve with recovery. Recumbency typically follows after about 48 hours, with a rapid onset indicating a poor prognosis and the need for urgent treatment.

Diagnosis

Diagnosis is based on the history especially effects on multiple animals and clinical findings (low ruminal pH - <5.5, and examination of the microflora of the rumen for presence of live protozoa). Rumen

examination reveals firm and doughy feeling in animal that consumed a large amount of grain, while those with smaller amounts may have a resilient rumen due to excessive fluid. The ruminal fluid is milky green to olive brown with a pungent acidic smell and pH below 5.

Differential Diagnosis

Other ruminal disorders – tympany, simple indigestion, rumen impaction.

Treatment

The primary goal of treatment is to arrest the further production of D lactic acid and neutralize the pre-formed lactate. Restrict supply of carbohydrate rich feeding for 24 to 48 hours. Treat severely affected animals with hypertonic sodium bicarbonate (5 percent, @ 10 mL/kg body weight, IV) followed by isotonic sodium bicarbonate (1.3 percent, @ 150 ml/kg body weight, IV, daily for 2 days). Provide a course of antihistaminic drug (pheniramine maleate, @ 1-2 mg/kg, IM), rumenotoric bolus orally and injection of vitamin B_1, B_6 and B_{12} (@10 ml, IM) along with normal saline daily for 3 days. Antibiotics like procaine penicillin G or oxytetracycline is preferred to minimize the development of bacterial rumenitis and liver abscesses. During emergency, rumenotomy is done with the lavage using 5 percent sodium bicarbonate solution. Fresh ruminal cud from slaughterhouse can be transplanted into rumen.

Prevention and Control

Prevent accidental access to grain. Introduce dietary changes gradually over 7–14 days. Feed two times a day if stall fed. Use ionophores in feed to alter rumen metabolism.

1.3.4 Diarrhoea

Definition and Etiology

Diarrhoea is one of the most important clinical conditions of enteritis of farm animals and observed in all age groups. It is manifested with increased frequency of defaecation due to higher peristaltic activity of small intestine. The consistency of the faeces may be soft or liquid due to high water content and low dry matter. The frequency of defaecation depends on the severity of enteritis. Diarrhoea often leads to dehydration due to lack of absorption of fluid and loss of fluid through the faeces. Sometimes, hypovolemic shock is also seen in acute and persistent diarrhoea. Common causes of diarrhoea are enteritis, malabsorption, nutritional deficiency, grain engorgement, infections with microbes and parasites, toxicosis by physical and chemical agents, prolonged use of antimicrobials, pathological damage of abomasum or stomach, intestinal tumour, *etc.*

Predisposing Factors

Inadequate colostrum intake by newborns, inflammation or necrosis of the intestinal mucosa, structural change of intestinal mucosa, abnormal intestinal motility due to increase peristalsis and pre-existing endoparasite infection are the major predisposing factors.

Clinical Signs

Dehydration, abdominal pain, soft or fluid faeces with or without unpleasant odour, dysentery (bloody diarrhoea) or melena (faeces containing blood). The systemic effects of diarrhoea are manifested with fever, septicaemia and toxaemia in the infectious enteritis. The hypovolemic shock often occurs due to severe dehydration in acute and per acute enteritis. Weight loss is seen in chronic diarrhoea.

Diagnosis

Diagnosis is based on history and clinical findings. The faecal examination is useful to determine the presence of causative bacteria, helminths, protozoa and chemical agents. Haematology of blood samples assists in determining the presence or absence of infection. Serum biochemistry and electrolyte analysis have additional advantages for effective management of diarrhoea.

Differential Diagnosis

Dietary diarrhoea, toxicities, parasitism, *etc.*

Treatment

Treat the root cause of diarrhoea as per the etiology. The principles of treatment of diarrhoea include balanced isotonic fluids and Ringer's lactate/lactated Ringer's solution (80-120 mL/Kg), intestinal protectants and adsorbents (kaolin and pectin mixtures @ 50 g for adult cattle and 30 g for calf, sheep and goat, twice daily orally for 3 days). Use probiotic and prebiotic supplements to reduce the severity of infection. Treat pyrexia with NSAID and severe form of diarrhoea having subnormal body temperature with fluid therapy.

Prevention and Control

Reduce the parasitic load through deworming at regular intervals. Ensure adequate colostrum intake by neonates. Vaccination against important enteric diseases (*E. coli*, rotavirus, coronavirus, *Cryptosporidium* spp.) is necessary. Adopt good farm management practices to minimize environmental stressors.

Sample Collection for Diagnosis

Blood sample for haematology and serum biochemistry and faecal samples for examination of parasitic infestation.

1.3.5 Oesophageal Obstruction

Definition and Etiology

Oesophageal obstruction, either acute or chronic, is characterized clinically by inability or difficulty in swallowing feed and water and continuous drooling of saliva accompanied by bloat in ruminants. Obstruction can be intra- or extraluminal. Intraluminal obstructions are usually caused by ingestion of inappropriate sized food materials which get lodged in the oesophagus. Extraluminal obstructions are caused by enlarged mediastinal lymph nodes, cervical or mediastinal abscess, and persistent right aortic arch, and thymoma causing pressure on the oesophagus leading to partial obstruction.

Predisposing Factors

Nutritional deficiencies and high yielders tend to ingest inanimate foreign bodies owing to their craving appetite that may cause obstruction mostly in the distal cervical oesophagus.

Clinical Signs

The animal suddenly stops eating and shows restlessness, forceful attempts to swallow, regurgitation, salivation, coughing and continuous chewing movements. If obstruction is complete, free gas bloating occurs rapidly. Ruminal movements are continuous and forceful, and a systolic murmur is audible. However, chronic cases will exhibit inability to swallow, salivation and continued bloat. Persistent obstruction may cause pressure necrosis of the mucosa resulting in perforation.

Diagnosis

Obstruction at the cervical oesophagus can easily be palpated. When thoracic oesophagus is involved, a probang or stomach tube is gently passed to locate the site of obstruction. Contrast radiographic examination is helpful to outline the site of stenosis, diverticulum or dilatation. Fibreoptics endoscope can be used for confirmatory diagnosis.

Differential Diagnosis

Oesophagitis, ruminal atony, persistent right aortic arch, mediastinal lymph node enlargement, diaphragmatic hernia, and vagus indigestion.

Treatment

Stabilize the animal with proper fluid and electrolyte therapy and primary management of bloat (rumenotomy). Relieve cervical obstruction by placing thumb and fingers distal to obstruction and gradually forcing upwards towards the pharynx and then removed by inserting hand into the pharynx. Use a probang or stomach tube to gently push the round obstruction into the rumen. Surgical removal by oesophagotomy is indicated if conservative therapy fails. Pre- and post-operative management includes withholding feed for 5-7 days, and keep the animal on intravenous fluid therapy, parenteral antibiotics and analgesics.

Prevention and Control

Provide adequate nutrition to the animals especially high producing dairy animals to avoid craved appetite and ingestion of foreign bodies.

Intestinal Obstruction

Definition and Etiology: Intestinal obstruction in ruminants is characterized by decrease or absence of passage of faeces, abdominal pain with variable distension, dehydration and metabolic alkalosis. It may be either mechanical (intraluminal - ingestion of foreign bodies, impacted ingesta, faecoliths, worm infestation, phytobezoar or extraluminal - compression of lumen due to peritoneal adhesions, fibrous bands, herniation, abscess, neoplasms, volvulus, mesenteric torsion, intussusception, etc.) or functional (paralytic ileus - due to peritonitis, enteritis, prolonged distension of intestine, stasis, hypocalcaemia, heavy concentrate feeding) or sometime congenital (hypoplasia jejuni, atresia ilei, atresia coli, Meckel's diverticulum).

Predisposing Factors: Large ruminants are mostly affected. Functional obstructions are more common particularly intussusception, peritonitis and hypocalcaemia in cattle and faecoliths or impacted ingesta in buffaloes.

Clinical Signs

Decrease or absence of passage of faeces, anorexia, abdominal pain (colic) and abdominal distension with elevated heart rate if vascular supply is occluded. Fluid-splashing sounds on ballottement and simultaneous auscultation over the right abdomen in most cases.

Diagnosis

Auscultation and percussion over right flank area show tympanic resonance extending cranially towards intercostal space. It is recommended that any case with complete cessation of defaecation and presence of thick mucus in rectum for >24 hours should be subjected immediately to right flank laparotomy for correction. Laboratory examination shows increased PCV, inverted neutrophil to lymphocyte ratio, azotaemia, hypocalcaemia, hypokalaemia, hypochloraemia, hyperglycaemia and metabolic alkalosis. Rumen fluid chloride concentration is high due to abomasal reflex. Paradoxic aciduria may be present. Strangulating obstructions are characterized by increase in the total protein concentration and nucleated cell counts of peritoneal fluid which includes degenerative neutrophils, intracellular Gram-positive and Gram-negative bacteria. Plant material in the peritoneal fluid is indicative of bowel rupture. Ultrasonographic examination of the abdomen via right paralumbar fossa or per rectum may help in diagnosis of small-intestinal distension, ileus, hypomotility or atony and increased peritoneal fluid volume. Intussusception in small intestine appears as Bull's eye lesion or multiple layered, onion ring-type mass with varying echogenicity. Ultrasonographic imaging of large intestine is difficult due to its marked gas content.

Differential Diagnosis

Abomasal displacement, caecal dilatation and torsion, diaphragmatic hernia.

Treatment

Treatment is symptomatic and supportive (fluid therapy - oral or intravenous) after eliminating the inciting cause (*e.g.*, hypocalcaemia, hypokalaemia, excessive grain intake) and allowing time for normal intestinal motility to return. Restore intestinal motility with calcium and potassium solutions, neostigmine in smaller doses (2 mg SC every 4-5 hours), saline purgatives and rumen cud transplants, provided no mechanical obstruction is present. Multiple electrolyte solution or 0.9% sodium chloride solution is effective. Hypertonic saline (2.7% sodium chloride with 1.1% potassium chloride @ 12-15 ml/kg IV over 12-15 min) followed by access to drinking water is used for rapid resuscitation of hypovolemic condition. For mechanical intestinal obstruction, perform right flank laparotomy for removal of obstruction with standard pre- and post-operative care.

Prevention and Control

Avoid abrupt changes in feeding - coarse feeds, fermentable feedstuffs and management, provide adequate water intake; control parasitic infection; dental abnormalities; and access to foreign material.

Sample Collection for Diagnosis

Blood for haematology, serum biochemistry and electrolyte changes.

1.3.7 Abomasal Displacement

Definition and Etiology

Abomasal displacement refers to the abnormal placement of the abomasum in the abdominal cavity. It may be left or right displacement. Left displaced abomasum (LDA) often occurs around the calving and is associated with changes in rumen function, high-energy diets, and hormonal fluctuations. Right displaced abomasum (RDA) is less common in females than males and is typically linked to metabolic disorders like hypocalcaemia. LDA is more frequently observed as compared to RDA.

Predisposing Factors

Excess grain feeding and anatomical and physiological factors, including advanced stages of pregnancy and previous abdominal surgeries, contribute to the likelihood of displacement.

Clinical Signs

Decreased milk production, loss of appetite and affected animals may exhibit a distended left flank and a characteristic ping sound upon percussion of the flank. Rumen stasis and dehydration are also common.

Diagnosis

Diagnosis is based on the animal's history and clinical signs. Physical examination involves palpation of the distended flank and detecting a ping sound on percussion. Diagnostic imaging techniques, such

as ultrasonography, provide detailed imaging of the abdominal organs. Confirmation can be done by abdominal paracentesis (Liptek test).

Differential Diagnosis

Differential diagnosis from rumen tympany (bloat), traumatic reticuloperitonitis (TRP), peritonitis, and acidosis.

Treatment

Do surgical correction by flank abomasopexy for repositioning of the abomasum after stabilization of the condition through fluid therapy, pain management, and correction of metabolic disorders followed by standard post-operative care.

Prevention and Control

Avoid sudden change in diet and excess feeding of grains especially in last half of pregnancy.

Sample Collection for Diagnosis

Fluid obtained on paracentesis may be subjected to pH examination apart from routine pre-surgical blood examination.

1.3.8 Traumatic Reticuloperitonitis in Cattle and Buffalo

Definition and Etiology

Traumatic reticuloperitonitis (TRP), commonly referred as hardware disease, is a prevalent condition in cattle and buffalo resulting from the ingestion of foreign metallic objects. These objects – typically nails or pieces of wire – lodge in the reticulum, and their penetration can lead to severe inflammation, infection, and subsequent complications. The primary cause of TRP in cattle is the ingestion of sharp metallic objects, which occurs due to indiscriminate feeding habits (often consume feed without discerning between edible materials and foreign objects), incomplete mastication (tend to swallow food items without thorough chewing, increasing the risk of ingesting sharp objects) and increased intra-abdominal pressure (factors such as pregnancy and parturition can exacerbate the risk of foreign body penetration through the reticulum).

Predisposing Factors

Ingestion of foreign body, grazing in metallic scrap areas, feeding habits of cattle and buffalo, advanced stage of pregnancy.

Clinical Signs

A range of clinical signs which vary in severity include fever (40°C to 41°C), anorexia and weight loss, ventral oedema (swelling, especially in the brisket area), reluctance to walk (cattle may walk with short steps, exhibit a stiff gait, and grunt while moving, indicating pain), arching of the back as a typical pain response, especially when the animal is standing, tachycardia and dyspnea, venous stasis (enlargement of the jugular vein) and brisket oedema and abduction.

Diagnosis

Accurate diagnosis of TRP involves a combination of clinical observation and specialized tests findings of auscultation (listening for pericardial friction sounds), which result from fibrinous exudate deposition, and muffled heart sounds in advanced stages. The washing machine murmur is indicative of gas presence in the pericardial sac, echocardiography (ultrasound imaging) to detect fluid accumulation and thickening of the pericardium, pericardiocentesis (extracting pericardial fluid), typically using an 18-gauge spinal needle inserted at the fifth intercostal space just dorsal to the elbow. The fluid often contains fibrin and has a fetid odour, radiography (X-rays to identify the presence of the metallic foreign body within the reticulum) and pain response tests (wither pinch and poll tests to elicit a pain response confirm the presence of TRP. Laboratory findings may include leucocytosis, low RBC and haemoglobin levels, reduced packed cell volume and increased globulin and decreased albumin levels.

Differential Diagnosis

Differential diagnosis is crucial to distinguish TRP from other conditions with similar presentations, such as haemorrhagic septicaemia, pleurisy, congenital heart failure and lymphosarcoma.

Treatment

Managing TRP often requires a combination of medical and surgical approaches. Medical treatment includes broad-spectrum antibiotics, diuretics to reduce oedema, analgesics and transfaunation (reintroducing ruminal flora post-surgery to aid digestion and recovery). Surgical techniques include laparotomy and rumenotomy wherein an exploratory laparotomy is performed to access the reticulum, followed by rumenotomy to remove the

foreign body with standard post-operative care.

Prevention and Control

Feeding of magnetic pellets helps to attract and immobilize metallic objects in the rumen, preventing migration.

Sample Collection for Diagnosis

Blood for complete hemogram.

1.3.9 Diaphragmatic Hernia

Definition and Etiology

Diaphragmatic hernia (DH) is a common surgical condition in bovines especially buffaloes and characterized by protrusion of abdominal viscera into the thoracic cavity through a tear in the diaphragm. Most commonly, the reticulum herniates; however, liver, spleen, abomasum and loops of intestines may also be involved. Most cases occur because of weakening of the diaphragm due to injury from penetrating foreign body or traumatic reticuloperitonitis but can occur spontaneously without foreign body also. Congenital DH in cattle calves at an early age is occasionally reported.

Predisposing Factors

Increased intra-abdominal pressure during advanced stage of pregnancy or straining at parturition and presence of sharp metallic foreign bodies in reticulum. Right ventromedial musculo-tendinous junction of diaphragm is more prone for rupture in buffaloes.

Clinical Signs

Capricious appetite, loss of condition for several weeks before abdominal distension, persistent moderate ruminal tympany due to frothy bloat, grinding of teeth and pasty scanty faeces. In advanced cases, regurgitation of ruminal contents may occur leading to aspiratory pneumonia. The temperature usually remains normal, but bradycardia may be present (40–60 beats/min). Affected animals may die, if untreated.

Diagnosis

On auscultation, systolic murmur is present, and the intensity of the heart sounds may suggest displacement of the heart, usually anteriorly or to the left. Reticular sounds may be heard cranial to 6th rib on right side. Thoracic radiography is recommended in right lateral recumbency. Absence of clear diaphragmatic line, in the thoracic radiograph is indicative of DH. Ultrasonography can be used to detect characteristic biphasic reticular motility at fourth and fifth intercostal spaces on the right side between heart and right thoracic wall. Haemato-biochemical findings include leucocytosis with neutrophilia, hyperproteinaemia with elevated SGOT, creatine kinase and creatinine levels.

Differential Diagnosis

Chronic bloat, vagus indigestion and oesophageal obstruction.

Treatment

Two-stage surgical correction is the treatment of choice for DH. In first stage, the rumen is emptied partially through left flank rumenotomy in standing position and the foreign bodies are removed from the reticulum while in the second stage, suture the hernial ring in dorsal recumbency under general anaesthesia through post-xiphoid approach after removal of adhesions between the abdominal organs and diaphragm by blunt dissection and retraction of the organs. Suture the hernial ring with non-absorbable suture in lockstitch pattern and surgical incision in routine manner. Standard post-operative care includes adequate intravenous fluid therapy, parenteral antibiotics and analgesics for a week and antiseptic dressing of skin wound twice a day till the suture removal.

Prevention and Control

Preventing traumatic reticuloperitonitis through management of the environment and the administration of reticular magnets. Feed and fodder should be checked for any metal debris to prevent its ingestion by animals. Buffaloes in their advanced stage of pregnancy should not be allowed for wallowing in pond and should be fed highly palatable, balanced nutritious feed to avoid straining due to impaction etc.

Sample Collection for Diagnosis

Blood samples for haemato-biochemistry and rumen fluid for physical, chemical and microscopic examination may be collected.

1.4 Nutritional Deficiency Diseases

1.4.1 Goitre in Small Ruminants

Definition and Etiology

Goitre or hypothyroidism is one of the most

prevalent abnormalities in goats and defined as a non-inflammatory, non-neoplastic enlargement of the thyroid gland in the foetus. It may be congenital or simply induced. Goitre in young lambs/kids is the result of pregnant ewes/does consuming goitrogens or consuming insufficient amounts of iodine. The syndrome is characterised by dystocia, myxoedema, longer gestation, and an increase in the size of the foetus.

Predisposing Factors

Deficient intake of iodine or excessive calcium intake, diets high in *Brassica* spp., or severe bacterial contamination of feedstuffs or drinking water can all contribute to iodine deficiency. The glycoside linamarin presence in linseed meal, or prolonged diet of the grass *Cynodon aethiopicus* having high cyanogenetic glucoside and a low iodine level or rapeseed and rapeseed meal, if fed to pregnant ewes/ does, may cause goitre in lambs.

Clinical Signs

Goitre is a clear sign of iodine deficiency. Partial or complete alopecia is the most prevalent symptom of iodine deficiency, along with a high rate of stillbirths and weak newborn animals. Weakness, widespread baldness, and palpable thyroid gland enlargement are noticed in newborn lambs. There may be an increase in ewes' gestational period duration and perinatal mortality. Goats exhibit a similar clinical picture, with the exception that all anomalies are more severe than that in lambs. Goat kids are goitrous and alopecic. The glands may pulsate with the normal arterial pulse and may extend down a greater part of the neck and cause some local edema. Auscultation and palpation of the jugular furrow may reveal the presence of a murmur and the thyroid thrill, due to increased arterial blood supply of the glands.

Diagnosis

Laboratory diagnosis of iodine deficiency is done by the estimation of T3, T4 and associated hormones. This consists of serum T4 (serum thyroxine) concentration comparisons between the dam and lamb, thyroid weight, and the ratio of the lamb's thyroid to body weight. Other tests are concentrations of iodine in plasma, milk, and urine to predict current iodine status. There may be breed differences in blood iodine levels but levels of 2.4-14 µg of protein-bound iodine/ 100 mL of plasma appear to be in the normal range. In ewes, an iodine concentration in milk below 8 µg/L indicates iodine deficiency. Normal lambs at birth have twice the serum thyroxine levels of their dams, but goitrous lambs have levels lesser than that of dams. Thyroid-weight: birth-weight ratios higher than 0.8 g/kg are suggestive of an iodine deficiency in newborn lambs.

Differential Diagnosis

Abortions due to infectious agents and congenital diseases.

Treatment

Administer potassium iodide (20 mg, single dose, orally) to lambs suffering from goitre. Alternatively, sodium iodide can also be given.

Prevention and Control

Iodine can be provided in salt or a mineral mixture. Provide potassium iodate enriched salt blocks (@ 200 mg/kg salt). Potassium iodide (280 mg) or potassium iodate (370 mg) given to pregnant ewes individually twice during the fourth and fifth months of pregnancy effectively prevents goitre in lambs. Avoid feeding of goitrogenic feeds/ fodder.

Sample Collection for Diagnosis

Blood for estimation of T3, T4 and associated hormones.

1.4.2 Hypovitaminosis A

Definition and Etiology

The deficiency of vitamin A in young animals is manifested as the compression of brain and spinal cord whereas in adult animals the syndrome is characterized by night blindness, corneal keratinization, pityriasis, defects in the hooves, loss of weight, and infertility. Congenital defects are common in the offspring of deficient dams. There are two types of vitamin A deficiencies, (i) primary diseases, which result from an absolute lack of the vitamin A or its precursor carotene in the diet; (ii) secondary diseases, which occur when there is an adequate dietary supply of the vitamin A or its precursor but problems with digestion, absorption, or metabolism cause its deficiency at tissue level. A maternal deficiency of vitamin A can result in herd outbreaks of congenital hypovitaminosis A in calves.

Predisposing Factors

Young animals raised on range pastures during dry

spells experience primary vitamin A insufficiency. Secondary vitamin A insufficiency may arise in situations of chronic illness of the liver or intestines. High ambient temperatures and a high feed nitrate content decrease the conversion of carotene to vitamin A. Mineral oil administration as in bloat over time may result in a decline in vitamin A esters, plasma carotene, and buffer fat carotene levels.

Clinical Signs

Inability to see in dim light (night blindness or moonlit night) is the earliest sign in all species. True xerophthalmia, with thickening and clouding of the cornea, occurs only in the calf. A range of ocular deformities, including cataract formation, lens luxation, microphthalmia. Excessive keratinization and heavy deposits of bran-like scales on the skin are seen in affected cattle. Dry, scaly hooves with multiple, vertical cracks are other signs. In the male, libido is retained but degeneration of the germinative epithelium of the seminiferous tubules causes reduction in the number of motile, normal spermatozoa. In the female, conception is usually not interfered, but placental degeneration leads to abortion and the birth of dead or weak young ones. Placental retention is common. Signs related to damage of the nervous system include paralysis of skeletal muscles caused by damage of peripheral nerve roots, encephalopathy caused by increased intracranial pressure, and blindness caused by constriction of the optic nerve canal. Congenital defects include complete absence of the eyes (anophthalmos) or small eyes (microphthalmos), incomplete closure of the foetal optic fissure, degenerative changes in the lens and retina, corneal dermoid and aphakia (absence of lens). Edema of the limbs and brisket (anasarca) is associated with vitamin A deficiency in cattle.

Diagnosis

Plasma levels of vitamin A used widely (< 20 µg/dL) for diagnosis. Additionally, CSF pressure is used as a marker of low vitamin A status.

Differential Diagnosis

Polioencephalomalacia, Hypomagnesemic tetany, Lead poisoning, Rabies, Central blindness and Peripheral blindness.

Treatment

Administer vitamin A (@440 IU/kg BW, injection parenterally). Aqueous solution is preferred over oily one.

Prevention and Control

Provide minimum daily requirement of vitamin A in all species (@40 IU/kg BW). Alternatively, dietary supplementation of vitamin A (@3,000 to 6,000 IU/kg BW, IM) at intervals of 50 to 60 days should be given.

Sample Collection for Diagnosis

Blood

1.5 Respiratory System Diseases

1.5.1 Pneumonia in Ruminants

Definition and Etiology

Pneumonia is the inflammation of lung tissue, often involving the bronchioles and pleura. Causes could be infectious (viruses, mycoplasmas, bacteria, fungi, parasites), physical agents and chemical agents. In animals, pneumonia is usually bronchogenic but can also spread through the bloodstream with septicaemia. Mycoplasmal pneumonia significantly affects cattle and goats.

Predisposing Factors

Weaning calves in cold climates, long-distance transport, housing in poorly ventilated, overcrowded barns and significant weather changes. Factors that weaken either innate or adaptive immunity increase susceptibility to the condition.

Clinical Signs

Symptoms include increased respiratory rate, altered breathing patterns, coughing, nasal discharge (purulent or serous), fever, abnormal breath sounds, and toxaemia in bacterial cases. Rapid shallow breathing (polypnea) is an early sign, but dyspnoea occurs when significant lung tissue becomes non-functional. Coughing - a key symptom - varies with the type of pneumonia. It is moist and painful in bacterial bronchopneumonia, and frequent, dry, and hacking in viral interstitial pneumonia. Cyanosis appears only when large lung areas are affected. Nasal discharge in the presence of upper respiratory inflammation and foul breath odour in anaerobic bacterial pleuropneumonia. Lungs reveals abnormal sounds (crackles, wheezes) on auscultation.

Diagnosis

Diagnosing pneumonia in ruminant species is based on clinical signs and findings of physical examination and diagnostic tests. Examination includes auscultation of the lungs for abnormal sounds (crackles, wheezes), tapping the chest to detect abnormal fluid or air accumulation, radiographic imaging the chest to visualize lung consolidation, interstitial infiltrates, or pleural effusion, collecting fluid from the trachea or deep airways to analyse cellular content and presence of pathogens, complete blood count (CBC) and serum biochemistry to assess inflammation and overall health status.

Differential Diagnosis

Other conditions causing polypnea and dyspnea include congestive heart failure, severe anaemia, hydrocyanic acid poisoning, hyperthermia, and acidosis, typically without abnormal lung sounds. Viral interstitial pneumonia often mistaken for conditions like pulmonary oedema, congestion, pulmonary artery embolism, and emphysema, which lack fever and toxaemia.

Treatment

Administer long-acting antimicrobials in bacterial pneumonia with short-acting ones in severe cases. Selection of antimicrobials involve effectiveness against the causative agent, ability to reach in therapeutic concentrations to the lungs, ease of administration, affordability, availability and approval for use in food animals. The choice of antibiotic, dosage, routes and duration can vary based on factors like severity of the disease, the specific pathogens involved, veterinary guidelines and clinician judgement. Effective antimicrobials for lung diseases are ceftiofur, amoxycillin, marbofloxacin, potentiated penicillin, fluoroquinolones (danofloxacin, enrofloxacin), and florfenicol, *etc*. Administration routes include oral, parenteral, or inhalation. Use anthelmintics such as ivermectin, moxidectin, and benzimidazoles for treatment of parasitic lung diseases. Viral pneumonia lacks specific treatment, and mycoplasma-associated pneumonia does not respond well to antimicrobials due to intracellular location. For the treatment of fungal pneumonia, use a combination of sulphonamide and trimethoprim (specifically sulfamethoxazole-trimethoprim). Supportive treatment includes NSAIDs, antihistaminic and corticosteroids for reducing inflammation and improving clinical signs. Treat with Bronchodilators like beta-2 adrenergic agonists (restricted in food animals) to enhance mucociliary clearance, nebulization in critical stages. Provide warm, well-ventilated house, with draft-free environments, ample freshwater and nourishing food.

Prevention and Control

Vaccinate against common pathogens. Provide well-ventilated housing to minimize drafts and maintains optimal temperature and humidity. Avoid overcrowding to reduce stress and transmission of pathogens. Implement strict protocols for introducing new animals and isolate sick individuals promptly. Avoid premature return to work or exposure to bad weather.

Sample Collection for Diagnosis

Tracheal aspirates or and washings retrieve through endoscopy or transtracheal wash to detect bacterial pathogens like *Mannheimia haemolytica* and *Pasteurella multocida* and assess inflammation. Blood for testing systemic markers of infection and inflammation, such as white blood cell count and serum fibrinogen levels. Culture the collected samples to identify specific bacteria and fungi involved in the infection and drug sensitivity.

1.5.2 Aspiration Pneumonia

Definition and Etiology

Aspiration pneumonia is a lung condition caused by inflammation and tissue death resulting from inhaling/aspiration of foreign substances in farm animals like food, fats, edible oil, fluids, medications, meconium, or excessive dust through sloppy drenching, oral medication administration, dipping, feeding or accidental placement of a tube into the trachea instead of the stomach during treatment. The severity of the condition depends on type of the material inhaled/aspirated and its spread in the lungs.

Predisposing Factors

Weakness, regurgitation process during poor health conditions, fluid feeding, dipping, anaesthesia with unprotected airways, cows with hypocalcaemia induced casting.

Clinical Signs

Common symptoms include cough, difficulty in breathing, rapid breathing, exercise intolerance,

isolation from other animals, loss of appetite with poor ruminal contractions and sometimes fever. In acute cases, animal stands with an arched back, extended neck, lowering of head, dark red mucous membranes suggesting endotoxemia and walks slowly. The rectal temperature is typically elevated up to 41.0°C with higher heart rate (often exceeding 100 beats/minute), and respiratory rate (>40–60 breaths/minute). The animals show pain with bilateral mucoid or purulent nasal discharge, occasionally reddish brown or green with a foul odour. Lactating animals may stop producing milk. Lung's auscultation usually reveals widespread crackles over the affected lung with reduced sounds and increased sounds over normal lung tissue, often with pleural fluid accumulation.

Diagnosis

Diagnostic ultrasound imaging (5-MHz linear probe) shows consolidated or hepatized lung tissue and hyperechoic pleural fluid in the cranioventral lung fields. Thoracic X-rays reveal consolidated lung tissue and bronchoalveolar patterns. Ultrasound examination can easily identify superficial consolidated lung tissue and fibrous pleurisy lesions. In mild to moderate cases, these lesions may not be immediately visible, but treatment should be aggressive in suspected cases. Hospitalized animals should undergo arterial blood gas analysis to monitor cardiopulmonary function.

Differential Diagnosis

Phlebitis or bacteraemia due to faulty or contaminated calcium injection, chronic suppurative respiratory disease, pleurisy, hepatocaval thrombosis, endocarditis or pericarditis, left-sided pyothorax associated with traumatic reticulitis and peritonitis.

Treatment

Treat with broad spectrum antibiotic with need based supportive care. Use culture and sensitivity test for antibiotic selection from tracheal/bronchial/nasal samples. Maintain hydration and provide nutritional support through oral or intravenous fluids. Manage pain and combat toxaemia with appropriate medications. Use humidified nasal oxygen in severe cases or neonates, as guided by arterial blood gas analysis indicating hypoxemia.

Prevention and Control

Use appropriate techniques by trained personnel for naso-/oro-gastric administration to avoid accidental inhalation of fluids or medications. Focus on preventing underlying diseases that lead to prolonged weakness and lying down. Ensure adequate ventilation and comfortable temperature during recovery.

Sample Collection for Diagnosis

Tracheal aspirates or washings helps to identify the presence of bacteria, fungi, or other pathogens directly from the respiratory tract.

1.5.3 Epistaxis

Definition and Etiology

Epistaxis (nosebleed) is the bleeding from the nostrils, nasal cavity, or nasopharynx and indicative of various underlying health issues. It can occur in one or both nostrils and may range from a mild trickle to a significant haemorrhage. External injuries (kicks, collisions), nasal foreign bodies, Iatrogenic causes (nasogastric intubation), bacterial infections (*e.g.*, rhinitis, sinusitis), certain parasites, like nasal bots (*Oestrus ovis*) in sheep and nasal schistosomiasis (*Hirudinaria granulosa*; leech) in buffaloes, tumour, blood clotting disorders, anticoagulant toxins, or inherited conditions, aneurysms or arteriovenous malformations, snake bite particularly viperid bite can result in nasal bleeding.

Predisposing Factors

Deficiencies in certain vitamins (vitamin C) and minerals (copper) can weaken blood vessels and increase the risk of bleeding. High blood pressure (uncommon in cattle) can induce spontaneous bleeding from the nose. Dry air, dust, and extreme temperatures dry out and irritate the nasal mucosa, making more susceptible to bleeding. Congenital or acquired abnormalities in the nasal passages can predispose cattle to epistaxis.

Clinical Signs

Blood comes out from one or both nostrils, ranging from a trickle to a heavy flow. Cattle may sneeze more often due to irritation or blood in the nasal passages. Swelling around the nose due to trauma or infection, attempts to relieve discomfort or irritation in the nasal area, difficulty in breathing, noisy breathing, increased respiratory rate and salivation, weakness, reduced feed intake, blood loss and anaemia if the bleeding is significant and prolonged. General signs of weakness due to blood loss. Blood may be present in the saliva if the epistaxis is severe.

Diagnosis

Inspect the nasal passages and surrounding areas for trauma or foreign bodies. Radiography to identify fractures, tumours, or sinusitis. Blood tests for coagulation profiles, infectious disease panels, and liver function tests.

Differential Diagnosis

Nasal or facial trauma, foreign bodies (grass awns, sticks, or other objects lodged in the nasal passages), bacterial infections (abscesses), fungal infections (aspergillosis), parasitic infections (nasal bots), nasal tumours, disorders affecting blood clotting (thrombocytopenia or haemophilia), ingestion of anticoagulant rodenticides or toxic plant, liver disease, *etc.*

Treatment

Control the bleeding with topical vasoconstrictors, cold compress, or packing the nasal passages. Treat epistaxis according to the cause. Treating coagulopathies with appropriate medications or blood products, *viz.*, epinephrine (adrenaline, 1:1,000 solution, soak gauze and apply to the bleeding site), vitamin K_1 (phytonadione @0.5 to 1.5 mg/kg body weight, SC or IM, one daily for 3-5 days) to manage anticoagulant poisoning (warfarin) and specific coagulopathies. Administer tranexamic acid (@ 10-15 mg/kg of body weight, IV every 8-12 hours for duration of 3-5 days or until bleeding controlled). Treat with flunixin meglumine (@1.1 to 2.2 mg/kg body weight, IV or IM, one daily and up to 5 days) or meloxicam (@ 0.5 mg/kg body weight, IV or SC, once daily for a maximum of 5 days). Use saturated solution of salt into the nostrils for treatment of leeches. Sodium iodide (10% solution, @66 mg/kg body weight every 7 to 10 days via IV infusion) for fungal infections. Give supportive therapy, *viz.*, fluid therapy to maintain blood volume and prevent shock, normal saline or lactated Ringer's solution, haemostatic drug (ethamsylate @ 5-10 mg/ kg bodyweight/ day for 1-3 days), calcium injection, and blood transfusion (@10-20 ml/kg of body weight) as per the need and severity of blood loss. Surgical intervention for tumours or severe trauma cases.

Prevention and Control

Accurate diagnosis and appropriate treatment are crucial for managing epistaxis. Ensure proper restraint and handling to minimize stress and further injury to the animal.

Sample Collection for Diagnosis

Blood for complete blood count (CBC) to evaluate overall health and detect anaemia, infections, or clotting disorders. Coagulation profile to assess blood clotting function and detect any clotting disorders. If a tumour or granuloma is suspected, a biopsy of the nasal passage or any visible mass may be necessary.

1.5.4 Pneumothorax

Definition and Etiology

Pneumothorax is characterized by the presence of air (or other gases) in the pleural cavity and can affect one or both the sides of the chest. When a significant amount of air enters pleural cavity, it can compromise the respiratory gas exchange resulting into respiratory distress and lung collapse. In cattle, the anatomy of mediastinum often results in unilateral pneumothorax, where air leaks into the pleural space on one side only. Lung rupture, often due to trauma or disease like bronchopneumonia, is a common cause in cattle. Thoracic wall trauma due to the accidents involving farm machinery around cattle or impalement on objects, can also lead to pneumothorax. In newborns, lung injury can occur if a rib fractures during birth. Additionally, bullet or arrow wounds, injuries to the upper respiratory tract or subcutaneous emphysema, surgical procedures like thoracotomy or lung biopsies can cause pneumothorax.

Predisposing Factors

Trauma, lungs diseases, thoracic surgery, bovine respiratory disease complex, actinomycosis (lumpy jaw), lungworms *(Dictyocaulus viviparus)*, and improper use of medical instruments etc predisposes to pneumothorax.

Clinical Signs

Acute onset of inspiratory dyspnea can be fatal within minutes if pneumothorax is bilateral and severe. If only one pleural sac collapses, the affected side's ribcage collapses and shows decreased movement with concomitant compensatory increased movement and bulging of the chest wall towards the unaffected side. The mediastinum may bulge towards the unaffected side, displacing the heart and apex beat, with accentuated heart sounds and a metallic note on the affected side. The apex beat may

be absent on the affected side. Auscultation reveals markedly decreased or absence of breath sounds. Percussion of the affected thorax detects hyper-resonance over the dorsal aspects. Affected animals are anxious, tachypnoeic, and in varying degrees of respiratory distress. Pneumothorax in cattle is often secondary to lung disease, typically infectious with signs like fever, toxaemia, purulent nasal discharge, and cough. Pneumothorax from chest wall trauma is usually obvious, though lung lacerations by fractured ribs can be missed, especially in newborns.

Diagnosis

Diagnosis is confirmed by radiographic or ultrasonographic examination. Radiography detects both bilateral and unilateral pneumothorax, as well as other air leakage syndromes (pneumomediastinum, pneumoperitoneum, pneumopericardium). Many cattle with pneumonia and pneumothorax show radiographic evidence of emphysematous bullae. Ultrasonography helps to determine pneumothorax extent and presence of consolidated lung and pleural fluid.

Differential Diagnosis

The potential conditions like pleuropneumonia, pulmonary abscess, lungworms, and bloat including several respiratory conditions, some systemic and traumatic causes need to be differentiated. These conditions can present with signs that overlap with pneumothorax, such as respiratory distress, abnormal lung sounds, and dyspnea.

Treatment

Treatment depends on the cause and severity of pneumothorax, respiratory distress, and hypoxemia. No specific treatment for closed pneumothorax without respiratory distress or hypoxia, but such animals should be confined and restrict exercise until signs resolve. Surgical intervention is required for closing an open pneumothorax caused by thoracic wound. Use broad spectrum antimicrobials to prevent septic pleuritis and analgesics to relieve pain.

Prevention and Control

Implementing measures to prevent traumatic injuries that could lead to pneumothorax.

Sample Collection for Diagnosis

Blood sample to assess oxygenation and ventilation status. It can show hypoxemia and respiratory acidosis in severe cases. If pleural fluid is present, aspiration can be performed to evaluate for characteristics such as colour, clarity, and cellular content to rule out other conditions causing respiratory distress.

1.6 Urinary System Diseases

1.6.1 Urolithiasis and Urethral Obstruction

Definition and Etiology

Urolithiasis is defined as the formation of uroliths due to multiple congenital and/or acquired pathophysiologic processes that result in increased concentration of less soluble crystalloids in the urine. Urethral obstruction may result from identifiable or specific causes like urethral plugs and uroliths and non-identifiable or non-specific causes like inflammatory swelling of urethra, urethral muscular spasm, reflex dyssynergia, intraluminal accumulation of sloughed tissue, red blood cells. Urinary pH plays an important role in development of calculi in the presence of urolith components. Struvite crystallization occurs only at a pH range of 7.2 to 8.4, whereas apatite stones develop at a urine pH of 6.5 to 7.5. Infection is another important factor that affects the composition of urinary calculi.

Predisposing Factors

Calcium deficiency in feed, imbalanced diet, non-availability of water, and urinary tract infection are some of the important predisposing factors.

Clinical Signs

Dullness, anorexia, restlessness, constipation, tympany, uraemic odour from mouth, rough hair coat and erect hair. Rumen motility decreases in most of the cases. Pressure develops in the bladder, producing mild colicky symptoms such as stretching, treading with the rear limbs and occasional kicking at the abdomen. Rectal examination reveals distended bladder, and calculi may be palpated in the distal flexure. Encrustation on the preputial hair may also be noted. In case of rupture of urinary bladder, there will be bilateral abdominal distension and water belly condition, and fluid thrills on abdominal ballottement.

Diagnosis

Diagnosis is based on the clinical signs, rectal

examination, abdominal palpation to feel the distended bladder and presence of abdominal fluid thrill in case of rupture of the bladder. Ultrasonography, contrast cystography or urethrography may be used to detect the condition of the urinary bladder and location of calculi.

Differential Diagnosis

In small animals, the condition should be differentiated from distended abdomen due to tympany and ascites.

Treatment

Do surgical management of urolithiasis in case of complete obstruction, by performing one or the combination of the urethrotomy, urethrostomy, tube cystostomy and bladder marsupialization. Provide urinary acidifier by feeding ammonium chloride (@100-200 mg/day orally for a week).

Prevention and Control

Adequate supply of water, balanced diet rich in calcium and intermittent use of urinary acidifiers like ammonium chloride in feed @ 100-200 mg/kg depending upon the size of the animal. Feeding of common salt also reduces the incidence of urolithiasis in ruminants.

Sample Collection for Diagnosis

Abdominal fluid by paracentesis for the presence of urea and creatinine and blood for haemato-biochemical parameters estimation.

1.6.2 Enzootic Bovine Haematuria

Definition and Etiology

Enzootic bovine haematuria (EBH) or bovine enzootic haematuria (BEH), a chronic disease of cattle and buffaloes, is characterised by the intermittent presence of blood in urine due to the occurrence of tumours in the urinary bladder. EBH is frequently seen in animals reared on pasturelands, where bracken fern (*Pteridium* spp.) grows abundantly. The urinary bladder of these herbivores is a target for bracken genotoxins, such as ptaquiloside (PT). A synergism between chronic ingestion of bracken fern and bovine papillomavirus (BPV) infection can result in chronic inflammation and urinary bladder tumour formation, responsible for EBH.

Predisposing Factors

The disease mainly occurs in hilly areas, which are wooded and situated in both high and low mountains with bracken fern grass.

Clinical Signs

In clinical stage, the colour of urine becomes red (macro-haematuria) and it contains a large number of intact erythrocytes. As the disease progresses, haematuria may disappear altogether and reappear after a few weeks or months. In the terminal stages, animals pass large clots in their urine and become emaciated, urinary flow may be interrupted especially in male animals resulting in secondary complications like cystitis, pyelonephritis, hydronephrosis and ascending bacterial infection.

Diagnosis

Clinical signs may be indicative of EBH that can be confirmed by ultrasonographic examination. In doubtful cases, diagnosis can be confirmed by cystoscopy of the bladder. The endoscopic examinations of the animals affected with EBH detects lesions such as congestion, haemorrhage, oedema, hypertrophy, haemangioma, ulcer and polyploid proliferation. Microscopic examination of urine sediment for the presence of significant numbers of RBCs is critical.

Differential Diagnosis

EBH should be differentiated from haematuria caused by other reasons and haemoglobinuria. Distinguishing haemoglobinuria (babesiosis, post-parturient haemoglobinuria, *etc.*) from haematuria is an important diagnostic consideration. Systemic disorders leading to intravascular haemolysis is associated with significant haemoglobinuria in the presence of a normal urinary system.

Treatment

No rational therapy to cure EBH and only symptomatic treatments, which include repeated douches of the bladder with solution of 1.3 percent acridine dyes, 2 percent silver nitrate or 1 percent alum. Use haematinics (iron dextran) and blood transfusion (~ 5 litre of blood) in severe cases.

Prevention and Control

Restrict the access of animals to bracken fern. Avoid grazing in fern-infested pastures especially during scarcity periods and allow stall feeding.

Sample Collection for Diagnosis

Urine for urinalysis, blood for haematology, fern samples for identification and analysis of fern toxin.

1.6.3 Cystitis

Definition and Etiology

Cystitis is the inflammation of the urinary bladder characterized by signs of pollakiuria, haematuria, and dysuria. Haematuria may be more noticeable at the end of the urine stream. Urinary tract infection with bacteria may occur which may be associated with catheterization or parturition in females and urolithiasis in males.

Predisposing Factors

Cystoliths, faulty catheterization, ascending and descending infections. Chronic glucocorticoid administration, hyperadrenocorticism, chronic kidney disease, and diabetes mellitus may predispose the animal to cystitis.

Clinical Signs

Haematuria, pollakiuria, and dysuria are the common clinical signs of cystitis. Colic may be observed in severe cases.

Diagnosis

Clinical signs can give indications of cystitis, but it can be confirmed with ultrasonography and urinalysis. Urinalysis may reveal increased protein and haemoglobin. The urine pH may be more alkaline if the infective bacteria are urease positive. A urine culture and antimicrobial sensitivity test should be done.

Differential Diagnosis

Cases of cystitis should be differentiated from urethritis, pyelonephritis, and abdominal colic.

Treatment

Treat with antimicrobials based on urine culture and antibiotic sensitivity test report for a minimum period of 2 weeks. Repeated urine culture during treatment is warranted. Supportive therapy includes antispasmodic, anti-inflammatory drugs and pH modifiers as per the severity of the condition.

Prevention and Control

Supply adequate water. If catheterization is needed only sterile catheters should be used and then removed as early as possible. Intermittent salt feeding may prevent cystitis.

Sample Collection for Diagnosis

Urine sample for bacterial culture and sensitivity.

1.7 Musculo-Skeletal System Diseases

1.7.1 Luxation of Patella

Definition and Etiology

Patellar luxation exists in three forms in cattle and buffaloes. Dorsal patellar luxation or upward fixation of patella occurs in adult bovine and more commonly in buffaloes. Lateral patellar luxation is a congenital problem and usually not common. Similarly, medial patellar luxation is also a congenital problem and rarely reported. In upward fixation of patella, the patella may be fixed temporarily or permanently on the upper part of medial femoral trochlear ridge. Etiology of upward fixation of patella is not clearly known but imbalanced diet, excessive workload and traumatic injury to the patella or its ligaments may lead to this condition.

Predisposing Factors

Trauma to the joint, excessive workload and imbalanced diet are the common predisposing factors.

Clinical Signs

Stiffness of the limb, excessive extension of the hind limb caudally for longer than normal, followed by forward jerky movement in temporary fixation. In many cases, there may be intermittent extension and dragging of the limb. Position of patella may be evident on patellar palpation.

Diagnosis

Clinical signs and palpation of the patella and its ligaments that may appear stiff and tense.

Differential Diagnosis

Displacement of biceps femoris muscle, spastic paresis and acute gonitis.

Treatment

Complete recovery follows with medial patellar desmotomy (MPD), performed in recumbency or in standing position using close or open method.

Prevention and Control

Selective breeding, proper nutrition, work and exercise management. Avoid excessive strain on

young and growing animals. Prompt and early treatment of any joint injury is important to prevent chronic issues.

1.7.2 Fracture

Definition and Etiology

Fracture, a break in the continuity of a bone or cartilage, is a common clinical condition occurring in all animals at any location of the bone. It can be incomplete or complete, open or closed, and simple transverse or slight oblique to severely comminuted. The causes of fracture can be either extrinsic or intrinsic. Extrinsic causes are either direct trauma like automobile accidents, infighting, a direct hit or trauma during dystocia or handling, or indirect trauma due to running, falling or jumping/galloping of an animal. Intrinsic causes can be violent muscular contractions or local/systemic diseases (pathological) such as bone tumours/cysts, bone infection and metabolic causes that may lead to bone weakness (rickets, osteomalacia, osteoporosis, etc.).

Predisposing Factors

Superficial nature with little soft tissue covering make distal limb bones more susceptible to fractures. Smooth roads, slippery floors of sheds and stables predispose animals for slipping/falling leading to fractures. Pathological conditions such as osteomalacia and osteoporosis can predispose the animal for fractures. Allowing the animals to roam around can predispose for accidents causing fractures.

Clinical Signs

Acute non-weight bearing lameness, shortening of the affected limb with bending, rotation, adduction or abduction of the affected limb, soft tissue swelling at the site of injury are classical signs of fracture. Palpation of the injured area may reveal pain and crepitation. In open fractures, broken bone fragments may be visible piercing through the skin. Some special fractures may show specific signs like, fracture at femoral head/neck along with dislocation of hip joint may result in hyperextension of the stifle joint, whereas in diaphyseal fractures of humerus may show knuckling of paw/digits due to involvement of radial nerve.

Diagnosis

Diagnosis is based on the classical clinical signs and radiographic examination. It also helps to find the type and location of fracture and ultimately helps the surgeon to plan for surgical fixation.

Differential Diagnosis

Fracture has to be differentiated from joint luxation, tendon/ligament and nerve injuries, and bone pathologies, which can cause lameness or/and soft tissue swelling.

Treatment

Technique of fracture management depends on factors like age and species of the animal, the fracture type and location, the extent of soft tissue injury, and presence or absence of open wound, *etc.* Provide first aid and emergency treatment to ensure clear airway and haemostasis in fracture cases. Clean the open wound thoroughly with antiseptic solution and apply splints/ casts and bandage. The joints proximal and distal to the injured bone should be included in the co-optation. Once the animal is haemodynamically stable, undertake definitive fracture fixation as early as possible. In general, treat fractures below the stifle or elbow joint, especially forelimb fractures, using external fixation techniques like splint and cast, however, fracture in proximal limb bones such as femur and humerus need internal fixation like IM pin fixation, bone plate or interlocking nail. Treat open fractures by any of the external skeletal fixation technique like multiplanar epoxy-pin fixation in light weight animals and metallic fixators in heavy animals. Post-operatively, restrict the animal movement for 2-3 weeks and ensure regular antiseptic dressing of surgical wound till complete healing. Maintain fixation till the radiological healing of fracture and can be removed after bone union.

Prevention and Control

As fracture generally occurs due to trauma, it is difficult to prevent the occurrence of fracture. However, better management practices including providing non-slippery floor, restricting the animal from free roaming, careful handling of foetus during dystocia, and maintaining good bone health (sufficient intake of mineral calcium, vitamin D_3 and protein) can reduce the incidence of fractures.

Sample Collection for Diagnosis

Blood biochemical tests (serum calcium, phosphorus, alkaline phosphatase etc.) to get the

status of metabolic condition of the animal and bone healing.

1.7.3 Radial Nerve Paralysis

Definition and Etiology

Radial nerve paralysis is characterized by dropping of elbow due to relaxation of triceps muscle and flexion of the limb due to paralysis of triceps brachii, extensor carpi radialis and digital extensors. Contusion or trauma to the radial nerve may be due to hyperextension of shoulder, physical trauma, bite wounds, injury from fracture fragments of humerus or brachial plexus involvement because of vertebral injury and fracture of radius and ulna. Nerve may also get entrapped in the callus during fracture healing.

Predisposing Factors

Casting on hard surface and inappropriate restraint of the forelimbs predispose for radial paralysis.

Clinical Signs

Dropped elbow and flexion of the limb at carpus are prominent clinical signs. It causes supporting limb lameness and dragging of limb in case of complete paralysis.

Diagnosis

Diagnosis is made based on history, clinical signs and physical examination.

Differential Diagnosis

Radial paralysis is a non-painful condition and needs to be differentiated from rupture of the medial collateral ligament of the elbow, elbow arthritis and myopathy of the biceps brachii, triceps brachii, anconeus, and extensor carpi radialis muscles.

Treatment

Treatment with anti-inflammatory drugs, vitamin B_{12}/vitamin B complex and massage with complete rest ensures reversal of the condition. Apply bandage and splint on the lower limb to prevent friction injury on anterior aspect of the limb.

Prevention and Control

Avoid casting on hard surface and be careful while reducing and fixing the fractures.

1.7.4 Laminitis

Definition and Etiology

Laminitis is the inflammation of sensitive laminae of the hoof, affecting the feet of ungulates including cattle, sheep, and goats. It affects the walking surface of the claws (sole and white line region), which is the papillae region of the claw and not the lamellae region. The onset of laminitis may be acute, sub-acute or chronic. It is caused by excessive intake of dietary energy and grain, grazing on green pasture high in sugars (fructan), compensatory weight bearing due to injury to the opposite limb and ingestion of toxic plants. Excessive work/load on hard surfaces and inadequate rest (prolonged weight bearing) and systemic diseases such as sepsis, acidosis, septicaemia, mastitis and metritis, *etc*., are other the causative factors for laminitis. In dairy animals, it occurs mostly around the calving period and is influenced by managemental and nutritional factors.

Predisposing Factors

Sudden changes in diet, grain overload, hard concrete flooring, systemic diseases-sepsis/ endotoxaemia, and compensatory weight bearing due to injury (like fracture) of the opposite limb can predispose to development of laminitis. Sudden variations in the diet changes rumen microbial populations, resulting in acidosis and endotoxaemia, affecting vascular endothelium to great extent and predispose to laminitis.

Clinical Signs

Clinical signs vary depending on the extent of damage to the laminae. Variable degree of lameness, foot tenderness and warmth in one or more legs is common. There may be increased digital pulses and temperature in the hooves. Venous distension of superficial veins and reddening of the skin above the coronet are also seen. Often signs of primary systemic illness such as bloat, diarrhoea, toxaemia, and increased respiratory and heart rates. In severe cases, foot deformities are evident.

Diagnosis

Detailed history including information on diet, exercise regimes, recent managemental changes, calving status, previous medical issues, along with complete clinical findings help to diagnose laminitis. Radiograph is used in confirmatory diagnosis of

laminitis. It allows the visualization and evaluation of the hoof capsule and detects the presence of a lamellar wedge or seroma. Venography (contrast radiography) of the palmar digital vein also helps to delineate the vasculature of the foot.

Differential Diagnosis

Acute laminitis should be differentiated from meningitis, encephalitis and tetanus. The local changes should be differentiated from interdigital necrobacillosis (foot rot) and tenosynovitis, *etc*. Differential diagnoses should also include nutritional and metabolic diseases that produce lameness, stiff gait and recumbency.

Treatment

Treatment involves management of pain, supportive treatment, and prevention or minimizing the effects of the inciting causes of laminitis. First, keep the animal under stall rest with deep soft bedding (straw/sand). Provide cold therapy (ice baths/packs) to minimize inflammation of laminae in early stages and address concurrently the underlying causes such as sepsis or endotoxaemia. Use a course of antihistaminic drug (pheniramine maleate, @ 1-2 mg/kg, IM). A low volar nerve block (injection of 2 percent lignocaine over the nerves located posterior to the artery on the posterolateral aspect of the fetlock) can be used to relieve pain and inhibit vascular constriction within the foot. Regular trimming and shoeing are essential. Provide good-quality hay with low non-structural carbohydrate content and a low-calorie ration.

Prevention and Control

Limiting free access to lush green pasture, minimizing carbohydrates and sugars in the diet, preventing exposure to large amounts of concentrate, ensuring adequate forage intake, adding rumen buffers to the diet (in ruminants), giving adequate exercise around the calving time, correcting housing errors, and providing free access to salt lick to increase production of saliva. Regular farrier care can ensure properly balanced hooves. Prevention of infectious diseases and interdigital dermatitis causing overgrowth of claws can reduce the incidence of laminitis.

Sample Collection for Diagnosis

Blood tests to detect suspected underlying endocrine disease.

1.7.5 Arthritis

Definition and Etiology

Arthritis is defined as inflammation of articular surface and synovium of the joint often manifesting structural damage and pain. Trauma to the structures of the joint, uneven wear and tear of the joint cartilage (also in senility), haematogenous spread of infection (septic arthritis in calves due to naval ill) may be the inciting causes of arthritis.

Predisposing Factors

Conformational defect, excess body weight, older age and presence of chronic septic foci in the body may be the predisposing factors.

Clinical Signs

Signs of septic arthritis in calves are depression, fever, pain and anorexia with or without lameness. Joint swelling, stiffness and inability or partial ability to bear weight may occur in adults. Involvement of more than one joint and joint deformities and crepitus are also observed.

Diagnosis

Diagnosis is based on clinical signs and physical examination followed by radiography and synovial fluid examination, haematology, and serum biochemistry.

Differential Diagnosis

Spasticity of muscles and lameness of musculo-skeletal or nervous origin need to be differentiated.

Treatment

Provide rest and treat first with the NSAIDs and cold compress. Administer intra-articular antibiotic like gentamicin and corticosteroids to control the inflammation and infection and preserve the integrity of joint structures. In septic arthritis, copious joint lavage with sterile saline followed by autologous synovial transplant in addition to systemic antibiotic therapy.

Prevention and Control

In newly born calves, ligate the umbilical stump and apply iodine containing antiseptic. Avoid housing on hard flooring for prolonged periods.

Sample Collection for Diagnosis

Synovial fluid and blood for haematology and serum biochemistry.

1.7.6 Ruptured Achilles Tendon

Definition and Etiology

Partial or complete rupture of Achilles tendon may occur due to trauma in small and large ruminants and is clinically manifested by dropping to the hock and flattening of the limb distal to hock in a way to support weight on plantar surface of the metacarpal bone.

Predisposing Factors

Older animals are more susceptible due to decreased elasticity and strength of tendons. Highly active or working animals (*e.g.*, racing horses, herding dogs) have a higher risk.

Powerful contraction during forced hyperextension of the muscle-tendon unit and prior tendon injuries can weaken the structure and increase the likelihood of rupture.

Clinical Signs

Peculiar clinical sign is dropped hock and presence of external wound just above the hock or a depression if subcutaneous rupture has occurred due to intrinsic forces. Tarsal hyperflexion is always noted in animals with Achilles tendon rupture. If the injury results from chronic Achilles tendon stretching, the patient will bear weight but walk plantigrade due to tarsal hyperflexion.

Diagnosis

Diagnosis is made by physical examination by assessing the range of motion, palpation for defects, and observing gait abnormality. Ultrasonography helps in diagnosis of extent of subcutaneous rupture and for evaluating healing after surgery.

Differential Diagnosis

Traumatic Achilles rupture needs to be differentiated from muscle strains or tears, fracture of calcaneus process, joint dislocation, inflammatory conditions such as tendinitis or bursitis, and obturator and/or sciatic nerve injury (parturitional trauma).

Treatment

The first step is rest and immobilization by limiting movement and applying splints, casts, or bandages. Administer anti-inflammatory drugs and pain relievers to reduce pain and swelling. Surgical repair as early as possible is indicated in severe cases, which involves primary anastomosis of the tendon (tenorrhaphy) with post-operative immobilization and support. Physical rehabilitation is vital to reverse the effects of immobilization on the joints.

Prevention and Control

Avoiding overexertion by monitoring and limiting strenuous activities, especially in at-risk animals. The animal should be gradually returned to normal activity as the premature weight bearing will result in failure of the tendon to heal.

1.8 Nervous System Diseases

1.8.1 Epilepsy

Definition and Etiology

Epilepsy in animals is characterized by paroxysmal, self-limited cerebral disturbances marked by abnormal brain electrical activity, resulting in seizures, fits, or convulsions. These seizures often involve a sudden loss of consciousness and can vary widely in severity and frequency depending on the underlying cause. Etiologically, epilepsy can stem from various factors, including genetic predisposition in certain breeds, traumatic head injuries, infections such as encephalitis or meningitis, exposure to toxins, metabolic disorders like hypocalcaemia or hypomagnesemia, and in many cases the cause remains idiopathic.

Predisposing Factors

Certain breeds or familial lines may have a higher genetic predisposition, making them more susceptible to epileptic seizures. Traumatic head injuries, whether from accidents or physical trauma, can also trigger or exacerbate seizures. Metabolic conditions such as imbalances in calcium, magnesium, or other electrolytes, and nutritional deficiencies can lower seizure thresholds in animals in addition to the developmental disorders, chronic systemic diseases, stress, previous history of seizures, and exposure to specific toxins.

Clinical Signs

Animals manifest various types of seizures. Generalized seizures affect the entire body, often characterized by convulsions, vocalization, loss of consciousness, excessive salivation, and involuntary

urination or defecation. In contrast, focal seizures impact specific parts of the body, resulting in localized twitching, anxiety, repetitive behaviours such as head shaking, and abnormal posturing of the head and limbs. Animals may exhibit pre-ictal signs, including restlessness, a vacant stare, or twitching, signalling an impending seizure. Post-ictal signs may involve depression, fatigue, temporary blindness, and coordination difficulties as the animal recovers from the seizure episode.

Diagnosis

Diagnosis is based on the history of frequency, duration, and nature of seizures. A thorough clinical examination, particularly neurological assessment, is important to evaluate motor functions, reflexes, and sensory responses. The laboratory tests of blood to assess for infections and metabolic status, along with cerebrospinal fluid (CSF) analysis to detect abnormalities in the central nervous system aid the diagnosis. Advanced imaging techniques like magnetic resonance imaging (MRI) and computed tomography (CT) scans play a pivotal role in identifying structural lesions or abnormalities within the brain that may be causing or contributing to seizures.

Differential Diagnosis

Metabolic disorders such as hypocalcaemia or hypomagnesemia, infectious diseases like tetanus, rabies, or listeriosis, neurological conditions such as Polio encephalomalacia or toxicosis from various toxins, including lead poisoning or nervous ketosis.

Treatment

Treatment strategies aim to manage and reduce the frequency and severity of seizures. Use antiepileptic drugs such as diazepam (0.25 to 0.5 mg/kg body weight, IV), mannitol (20% solution, 0.5–1 g/kg body weight, IV), dexamethasone (0.1–1mg/kg body weight, IV) and hepatoprotectants. Correct metabolic imbalances and nutritional deficiencies through dietary management. Treating underlying systemic diseases that may be triggering seizures is crucial in managing epilepsy in animals. Tailoring treatment plans based on the underlying cause and response to therapy is key to successful management of epilepsy.

Prevention and Control

Providing a stress-free environment and minimizing potential triggers play a vital role in preventing seizure episodes. Providing safe housing and handling practices to avoid head injuries. Regular vaccinations and parasite control to prevent brain infections. Avoid breeding animals with history of epilepsy. Provide balanced diets to prevent metabolic disorders.

Sample Collection for Diagnosis

Whole blood for complete blood count examination and cerebrospinal fluid analysis for haemato-biochemical examination.

1.8.2 Meningitis

Definition and Etiology

Meningitis, inflammation of the meninges, is usually caused by bacterial or viral invasion of the central nervous system. It is clinically manifested by fever, cutaneous hyperaesthesia, and muscle rigidity. Common bacterial agents include *Streptococcus*, Coliforms, *Salmonella, Listeria, Haemophilus, Pasteurella, Leptospira, Mycoplasma mycoides*, and others, often spreading via the bloodstream. Viral encephalitides frequently lead to meningitis as well. Infections can also arise from direct extension due to skull fractures, osteomyelitis, sinusitis, otitis, or spinal injuries, and from metastatic or embolic events related to left-sided endocarditis.

Predisposing Factors

Animals with complication of a pre-existing disease as well as the young animals and those with weakened immune systems are at higher risk. Close contact with infected animals or environments contaminated with infectious agents can facilitate the spread of the disease. Animals with head trauma or infections in nearby structures, such as the ears or sinuses, are more susceptible to meningitis. Stress and poor nutritional status can also compromise ability of animals to fight off infections, increasing the likelihood of developing meningitis.

Clinical Signs

The clinical signs vary widely depending on the severity and underlying cause of the inflammation. Common signs include fever, lethargy, anorexia, and a stiff neck. Neurological symptoms are seizures, disorientation, sensitivity to touch, generalized hyperaesthesia, trismus, opisthotonus, and rigidity of the neck and back, leading to refusal to lower the head to eat. Behavioural changes like increased

aggression or depression, disturbed consciousness, and nervous signs varying from excitement or mania to drowsiness and coma. Vomiting is common in the early stages, particularly in pigs, and blindness can occur in cases of cerebral meningitis. Additional symptoms include ophthalmitis with hypopyon in young animals, slower pupillary light reflex, slow and deep respiration, and muscle rigidity or paralysis caudal to the affected area.

Diagnosis

History and clinical signs are important in detecting the disease. There is neutrophilic leucocytosis of peripheral blood. The CSF analysis reveals elevated white blood cell counts, increased protein levels, and the presence of infectious agents. CSF is turbid with a tendency to clot. It contains high protein (20-270 mg/dl), increased cell count (>100 neutrophils/µl) and normal (80% of blood level) or decreased (<50 percent of blood level) glucose level. Bacteria may be seen on Gram's staining.

Differential Diagnosis

Meningitis should be differentiated from encephalitis, acute cerebral oedema, spinal cord compression, cervical intervertebral disc protrusion, hypoglycaemia and hypomagnesemia, brain tumours, abscesses, or vascular accidents. Infections localized to the ears, sinuses, or other regions near the central nervous system should also be considered.

Treatment

Treat bacterial meningitis with broad-spectrum antibiotics which cross blood-brain barrier, such as sulpha, cefotaxime, *etc.*, and often given intravenously. Viral meningitis is managed with supportive care, including fluids, anti-inflammatory medications, vitamin B complex and anticonvulsants if seizures are present. Fungal and parasitic infections require specific antifungal or antiparasitic treatments. Corticosteroids are used to combat the harmful effects of inflammation. Supportive therapy, including intravenous fluids, pain management, and anticonvulsants, is essential in all cases.

Prevention and Control

Preventing meningitis in animals involves vaccination, hygiene practices, and environmental management. Vaccines are available for certain pathogens, like the distemper virus in dogs. Maintaining a clean environment, minimizing stress, and promptly treating localized infections in the ears or sinuses reduce the risk. Biosecurity measures, monitoring and surveillance, proper nutrition, and management practices are essential for prevention and control.

Sample Collection for Diagnosis

Cerebrospinal fluid (CSF) through a lumbar puncture, analysis for cell counts, protein levels, and infectious agents. Peripheral blood samples to assess general health and identify systemic infections.

1.9 Hematopoietic System Diseases

1.9.1 Anaemia

Definition and Etiology

Anaemia is defined as an absolute decrease in the red blood cell mass as measured by red blood cell (RBC) count, haemoglobin concentration, and/or packed cell volume (PCV). Anaemia can be classified as haemorrhagic anaemia, haemolytic anaemia or anaemia due to reduced production of erythrocytes from the bone marrow. It can also be classified as either regenerative or non-regenerative anaemia based on the production of reticulocytes from bone marrow and its release in the circulation. The causes of anaemia are multifactorial in nature. Causes of haemorrhagic anaemia include endo-parasitism like amphistomiasis, fasciolosis, haematophagus lice (*Linognathus vituli*), tick infestation and abomasal ulcer due to theileriosis. Causes of haemolytic anaemia include *Babesia* spp., *Anaplasma* spp., *Mycoplasma* spp., *Trypanosoma* spp., *Leptospira* spp. (*L. interrogans* serovar pomona), *Clostridium haemolyticum*, bovine viral diarrhoea, post-parturient haemoglobinuria, water intoxication and drinking cold water in calves induces haemolytic anaemia. Snake bite especially haemotoxic snakes like vipers besides nutritional deficiency of iron and copper also lead to anaemia.

Predisposing Factors

Nutritional deficiency of iron and copper, ectoparasites, endoparasites, certain bacterial and viral infections, ulcers, snake bites, water intoxication.

Clinical Signs

Paleness of all visible mucous membranes, muscular weakness, dullness and depression, inappetence to

anorexia, tachycardia, jaundice, haemoglobinuria, haematuria and oedema.

Diagnosis

Diagnosis is based on history and clinical signs and complete blood test (reduced haemoglobin, RBC and PCV levels). If haemolytic disease is suspected, blood can be evaluated for autoagglutination, or a direct Coombs test. The normal concentration of iron and total iron binding capacity (TIBC) in the blood is 10-29 pmol/litre (0.445-1.2905 µg/litre) and 20-63 pmol/litre (0.89-2.8035 µg/litre).

Differential Diagnosis

Differentiate among the specific causes and other conditions causing anaemia like haemorrhage due to trauma, accidental blood loss, *etc*.

Treatment

First treat the underlying causes leading to anaemia. Administer haematinic preparations containing Fe, Cu and Co either by oral or parenteral route depending on the condition. Iron dextran (@ 5 mg/kg BW IM on alternate days basis) along with cyanocobalamin (vitamin B_{12}) and folic acid. Animals with autoimmune haemolytic anaemia respond well to administration of corticosteroids which include prednisolone or dexamethasone. Whole blood transfusion is indicated in severe cases and volume calculated as per the following formula.

$$\text{Transfusion amount in litres} = \frac{\text{Desired PCV-Current PCV x (Weight of recipient x 0.08)}}{\text{PCV of Donor}}$$

Prevention and Control

Exclude the specific causes and provide supplement rich in iron, copper and cobalt.

Sample Collection for Diagnosis

Blood for the estimation of RBC count, haemoglobin concentration, and/or PCV, examination of the parasites.

1.10 Skin Diseases

1.10.1 Photosensitization

Definition and Etiology

It is the inflammatory condition caused by either ingestion of pre-formed photodynamic agents (primary photosensitization - Type I) or production of photodynamic agent in the body (aberrant pigment synthesis - Type II) due to abnormal metabolism or faulty excretion owing to hepatopathy (hepatogenous photosensitization - Type III) or idiopathic (Type - IV) leading to sensitization of mucosa, cornea, lightly pigmented areas, and dorsal skin surface of the animal body on exposure to sunlight. The hepatogenous photosensitization is most common in animals. The primary photosensitization caused by excess ingestion of exogenous photodynamic agent present in lush green and rapidly growing plants such as *Hypericum perforatum* (hypericin), *Polygonum fagopyrum* (fagopyrin), *Cymopterum* spp. (furocoumarins), *Lolium perenne* (perloline), *Brassica* spp., *Trifolium* spp. or chemicals (phenothiazine, Rose Bengal and Acridine dyes, corticosteroids). The photosensitization due to aberrant pigment synthesis is caused by inherited congenital porphyria in domestic animals. In hepatogenous photosensitization, the phylloerythrin, a normal product of chlorophyll metabolism is retained due to its inability to be excreted in bile owing to hepatitis or bile duct obstruction. Several hepatotoxic plants (*Lantana camara*, *Lippia rehmanni*, *Tetradymia* spp., *Senecio jacobea*, *etc*.), fungus (*Pithomyces chartarum*), blue green algae (*Microcystis flosaquae*), plants rich in steroidal saponins leading to cholangio-hepatopathy (*Agave* spp., *Tribulus terrestris*, *Panicum* spp.) or inherited congenital defective hepatic function (Corridale and Southdown lambs) contribute to the development of hepatogenous photosensitization. The idiopathic photosensitization is caused by exposure to water-damaged or mouldy alfalfa hay *(Medicago sativa) and Erodium* spp., mouldy straw, orchard grass hay containing foxtails, winter wheat, *Brassica rapa*, and phenanthridium used in treatment of trypanosomiasis.

Predisposing Factors

Light skin animals, hepatopathy, damaged or mouldy feed stuffs, excess feeding of lush pasture, and exposure to the poisonous plants.

Clinical Signs

Erythema, intense irritation with constant rubbing and edema of affected part of skin followed by weeping and shedding of hair and finally necrosis and dry gangrene leading to sloughing of large area of skin. The lesions are typically more pronounced in the unpigmented areas of facial region (tongue,

ears, conjunctiva, cornea, eyelids, muzzle), and dorsum (vulva and perineum), less intense lesion of sides and usually ventral body except teats not affected, serous discharge with blepharospasm and swollen eyelids, and resentment to suckling. Systemic manifestations such as shock due to extensive tissue damage, pyrexia, nervous signs such as ataxia, posterior paresis, blindness, depression or excitement and signs of hepatic insufficiency (jaundice, ascites, *etc.*) may become apparent in some cases.

Diagnosis

Diagnosis is based on history of exposure to photodynamic agents or hepatotoxins, grazing in area dominated by hepatotoxic plants and characteristic lesions (erythema and edema of hairless, non-pigmented areas of skin). There is no suitable field test available but the measurement of porphyrin concentration in blood, urine, faeces help in diagnosis. The serum biochemistry (elevated concentration of gamma-glutamyl transferase - GGT, sorbitol dehydrogenase – SDH, alkaline phosphatase - ALP, and direct bilirubin level) aids in diagnosis.

Differential Diagnosis

Mycotic dermatitis (occur in all parts of body), *Clostridium novyi* infection, pink eye disease (only eye affections not associated with dermatitis).

Treatment

Immediate withdrawal of toxic feed material. Housing of affected animal in cool and shaded places. Use toxin binder or laxative to remove the ingested toxin from body, hepatoprotectant, NSAIDs, antihistaminics, vitamin E and Se drugs. The parenteral antibiotics in animals showing systemic signs of septicaemia or shock.

Prevention and Control

Do not breed animals with photosensitization due to a genetic defect. Identify and remove the plant source and drugs (CCl_4, phenothiazine, phenanthridium) causing photosensitization.

Sample Collection for Diagnosis

Blood, urine, faeces for the measurement of porphyrin concentration and serum biochemistry.

1.10.2 Degnala Disease

Definition and Etiology

Disease caused by toxin (aflatoxins) produced by mould *Aspergillus* spp. (*A. flavus* and *A. parasiticus*) grown on the spoiled feedstuffs (maize, green chopped, sorghum, ground nut cake, cottonseed cake etc.) stored under damp environment. The aflatoxins (AF) include AFB1, B2, G1, G and second-generation metabolites (M1 and M2) excreted in the milk. Among them, AFB1 is most toxic. AF is believed to be derived from dicoumarin compounds. All animal species are susceptible; however, outbreaks are commonly reported in cattle, sheep and pig. Aflatoxicosis has public health importance due to excretion in milk (M1 and M2). Aflatoxins primarily affect the liver causing hepatosis and hepatic insufficiency. Further, they are also mutagenic, carcinogenic, teratogenic and immunosuppressive.

Predisposing Factors

The storage of feedstuffs having high moisture (grain moisture >15 percent) and environment having relative humidity (>75 percent) and warm temperatures are conducive for the mould growth.

Clinical Signs

In cattle and sheep, the symptoms are icterus, haemorrhage, clotting defects, blindness, circling, ear twitching, bruxism, photosensitive dermatitis, abortion, diarrhoea, anal prolapse, and terminally convulsions. Calves (3-6 months age) are most susceptible and affected animals die within 48 hours.

Diagnosis

Diagnosis is based on clinical signs, serum chemistry [for estimation of marked elevation of Alkaline phosphatase (ALP), Alanine transaminase (ALT), Aspartate aminotransferase (AST), bilirubin, and serum bile acids], milk testing (aflatoxin M1 and M2; > 0.5 ppb), complete blood count, coagulation test (prolonged prothrombin time), and postmortem examination of carcass (enlarged and firm liver with marked fatty accumulations, massive centrilobular necrosis and haemorrhage in liver, icterus, and ascites).

Differential Diagnosis

Fascioliasis, Lantadene poisoning, Pyrrolizidine alkaloid poisoning, and Sporidesmin poisoning.

Treatment

No specific antidote for aflatoxins is available. The treatment is based on the clinical signs. Essentially, the hepatoprotective agents (silymarin), multivitamins, diet containing balanced amount of protein, amino-acid supplements, trace minerals and antioxidants should be considered for treatment of affected animals.

Prevention and Control

Avoid aflatoxin contaminated feed.

Sample Collection for Diagnosis

Whole blood, serum, liver, urine, and feed.

1.10.3 Horn Cancer in Bovine

Definition and Etiology

Bovine horn cancer, mostly squamous cell carcinomas, arises from the mucosa of the frontal sinus and invades the horn core. Chronic irritation of horns at their base due to any reason like painting of horns for aesthetic purposes, tying of ropes at the horn base for restraint and prolonged exposure to direct, harsh sunlight. It mainly occurs in castrated bullocks, but cases are also seen in cows as well. The condition is mostly unilateral, but both the horns can also be affected.

Predisposing Factors

Horn cancer is a widespread cancer reported in long-horned Indian zebu cattle (*Bos indicus*). Horn cancer is more common in male animals, especially bullocks, with higher frequency – due to genetic predisposition – in Kankrej and Gir breeds than other zebu cattle, nondescript cattle or crossbred cattle.

Clinical Signs

Striking the head against hard objects, shaking of head, asymmetry of horn, slimy blood-tinged discharge from the nostrils of affected side, softness of the horn base which feels hot on touch and pain on palpation. There may be suppurative sanguinous discharge from the affected horn. Spontaneous separation of horn may reveal the presence of cauliflower like growth at the base.

Diagnosis

It is based on clinical signs, radiography and histopathological examination of the tissue biopsy from the horn base.

Differential Diagnosis

Horn fracture, horn avulsion and frontal sinusitis.

Treatment

Horn amputation under local analgesia with removal of all neoplastic tissue (cryosurgery also useful). Ensure providing autoimmune therapy (administration of saline phenol extract of the horn core tissue, SC) and standard post-operative care.

Prevention and Control

Avoid prolonged direct exposure to sunlight and application of irritants to the horn. Adopt debudding practice.

Sample Collection for Diagnosis

Aspirate from the horn core for cytology and tissue sections for histopathology.

1.10.4 Burn injuries

Definition and Etiology

Burn is a thermal injury to the skin or other tissues when it comes in contact with hot objects/flame and hot scalding vapours or liquids. It may also be caused by radiations, electricity, friction, or corrosive chemicals. Depending upon the depth of tissue involved, burns can be first degree (superficial burn, involving only epidermis), second degree (partial thickness, involving epidermis and mid of dermis), and third degree (full thickness, involving entire epidermis and dermis and underlying structures).

Predisposing Factors

Exposure to very high temperatures/fire, corrosive chemicals/ionizing radiations/electricity. Unattended heating devices, poor electrical connections/appliances in and around animal housing, inappropriate storage of inflammable material/irritant chemicals, and lack of proper fire safety/fire-fighting equipment.

Clinical Signs

Erythema, oedema, and desquamation of the superficial layers of the skin in the first-degree burns. Erythema and oedema at the epidermal-dermal

junction, necrosis of the epidermis, blister and eschar formation in the second-degree burns. White to black colour wound having loss of epidermal and dermal components, eschar formation and loss of pain sensation in the third-degree burns, burn shock (hypovolemic shock with reduced cardiac output), and dehydration.

Diagnosis

History and clinical signs of burn injury. Total body surface area involved in the burn should be assessed for its prognostic value.

Differential Diagnosis

Lacerations, pyoderma and viral dermatoses wherein blisters or bullae are formed.

Treatment

Management of burn involves intravenous fluid administration in all the cases of extensive burn injury with colloidal solutions (@ 4ml/kg body weight, hydroxyethyl starch/plasma), followed by additional crystalloids. Administer broad-spectrum antibiotics, anti-inflammatory/analgesic drugs, H2 blockers and corticosteroids. Protect the wound from further mechanical trauma/self-mutilation and keep dry. Blisters should be left intact for first 24-36 hours followed by incision/broken aseptically, with the application of antiseptic ointments, like silver sulfadiazine ointment and dressing of wound.

Prevention and Control

Use of fire resistant/retardant materials for construction of animal housing, proper upkeep of electric circuitry and equipment in the animal houses and following the fire safety norms.

1.11 Cardiac System Diseases

1.11.1 Chronic (Congestive) Heart Failure

Definition and Etiology

Chronic (congestive) heart failure (CHF) is characterized by congestion either in the pulmonary venous circulation or systemic venous circulation or both. It may be left-sided (pulmonary congestion) or right-sided (systemic venous congestion). Causes of chronic (congestive) heart failure include valvular disease, myocardial disease, congenital anatomical defect producing shunts and hypertension.

Predisposing Factors

Congenital anatomical defect, excess salt intake.

Clinical Signs

In right-sided congestive heart failure, the heart rate is increased and there is venous distension and subcutaneous oedema. In ruminants, there is subcutaneous oedema occurring in the brisket region, under the jaw and along the ventral midline, and ascites. In left-sided congestive heart failure, the heart rate is increased and there is an increase in the rate and depth of respiration at rest with cough, the presence of crackles (discontinuous sounds) at the base of the lungs and severe dyspnea and cyanosis terminally.

Diagnosis

Based on clinical signs, electrocardiogram (ECG), echocardiography, radiography, phonocardiogram and phonocardiography.

Differential Diagnosis

Related cardiac diseases having similar symptoms.

Treatment

Use diuretics (furosemide @1-2 mg/kg BW) for cattle and cardiac glycosides (digoxin, an initial intravenous loading dose @ 2.2 mg/100 kg BW followed by 0.34 mg/100 kg BW every 4 hours). Stall rest in a thermoneutral environment is also an important treatment. Salt restriction in diet.

Prevention and Control

Salt restriction in diet.

1.11.2 Traumatic Pericarditis

Definition and Etiology

It is inflammation of the pericardium due to perforation of the pericardial sac by an ingested foreign body that often results in accumulation of fluid or exudate between the visceral and parietal pericardium. Ingestion of potentially penetrating foreign bodies like pieces of wire, needles or nails, sharp metal fragments, broom bristles, and sometimes external penetrating traumatic injury.

Predisposing Factors

Traumatic injury, ingestion of foreign body, grazing in metallic scrap areas.

Clinical Signs

Fever, anorexia, depression, weight loss, drop in milk yield, stiff gait with slightly hunched back, abducted elbows with tucked up belly, and mild ruminal tympany. Slight expiratory grunt that becomes more obvious when the animal tries to lie down. Hard, scanty faeces, peripheral oedema with distention and pulsation of the jugular vein. Tachycardia, muffling of heart sounds, fluid splashing sound (washing machine murmurs) on cardiac auscultation. Tachypnoea, absence of lung sounds in the ventral thorax (dorsally, the lung sounds are louder than normal). Congested mucous membranes with prolonged capillary refill time is also important sign.

Diagnosis

Diagnosis is based on clinical signs and findings of the pole test (putting pressure on the ventral thorax though a bamboo stick will elicit pain, as evidenced by grunting), wither pinch test (pinching the withers will elicit pain, and the animal will be reluctant to bend its spine ventrally), haematology (leucocytosis, absolute neutrophilia with a shift to left; lymphopenia, hypoalbuminemia, hyperfibrinogenaemia, hyperglobulinemia, and elevated cardiac troponin I, lactate dehydrogenase-I and creatinine kinase - MB isoenzyme), cytology and bacterial culture of pericardial fluid, radiography (presence of metallic foreign body in cranial reticulum or caudal thorax; fluid and gas accumulation in the pericardium; enlarged, globose cardiac silhouette), electrocardiography (low voltage QRS complexes (<1.5 mV in the base-apex lead), electrical alternans, and ST-segment elevation or slurring. A right-axis deviation in the standard limb leads may be apparent), echocardiography (anechoic fluid filled space between the parietal and visceral pericardium; presence of echogenic fibrinous strands in the pericardial sac; hyperechoic regions with sharp shadows in the pericardial sac indicating the gas produced by infection of gas producing bacteria; right ventricular/right atrial collapse), metal detector test (can detect only ferromagnetic foreign bodies) and necropsy findings (hyperaemia of the pericardial lining with fibrin deposits; accumulation of turbid, putrid, fluid, thickening of epicardium and pericardium; adhesion of pericardium to the epicardium).

Differential Diagnosis

Pleuritis, cardiac valvular disease, mediastinal abscess, diaphragmatic hernia, and aspiration pneumonia.

Treatment

Treat with antibiotics only as per the findings of the pericardial fluid culture and antibiotic sensitivity test. In the absence of culture report, broad-spectrum antibiotics can be attempted. Pericardiocentesis with pericardial lavage using sterile, warmed normal saline solution, pericardial marsupialization to the thoracic wall, and pericardiotomy or pericardiectomy (if constrictive pericarditis). Keep the animal standing with forequarters elevated. Perform rumenotomy for the removal of the potential foreign body along with supportive therapy (fluid therapy, anti-inflammatory/analgesic drugs).

Prevention and Control

Provision of clean fodder, prevention of grazing in the metallic scrap infested areas, regular administration of bar magnets in the feed, feeding of magnet.

Sample Collection for Diagnosis

Blood, pericardial fluid.

1.11.3 Bovine Myocarditis

Definition and Etiology

Inflammation of the myocardium could be due to bacterial, viral or parasitic organisms, thromboembolism caused by these organisms, nutritional deficiencies and some toxic agents. Various categories include: (i) bacteria (*Staphylococcus aureus*, *Streptococcus* spp., *Clostridium chauvoei*, *Histophilus somni*, *Mycobacterium* spp., or any other bacterial infection causing bacteraemia and septicaemia); (ii) spirochaete (*Borrelia burgdorferi*), viral (foot-and-mouth disease virus, especially in calves); (iii) parasitic (*Sarcocystis* spp., cysticercosis, toxoplasmosis, *Neospora caninum*); (iv) nutritional deficiency (vitamin E/selenium deficiency, chronic copper deficiency - falling disease); (v) toxic agents (inorganic poisons -selenium, arsenic, mercury, phosphorus, thallium, fluoroacetate); (vi) toxic plants (*Pavetta* spp., *Asclepias* spp., *Lantana* spp., *Bryophillum* spp., *Colchicum spp*., *Lolium rigidum*, *etc*.); and (vii) drugs (catecholamines, xylazine, succinylcholine, bracken fern).

Predisposing Factors

Any systemic bacterial infection, ensuing pericarditis, epicarditis, or endocarditis, deficiency of vitamin E, selenium or copper.

Clinical Signs

Fever, exercise intolerance, arrhythmia, gallop rhythm, cardiac murmurs, dyspnoea, generalized oedema and syncope.

Diagnosis

Diagnosis is based on findings of the haematology (neutrophilic leucocytosis), electrocardiography (sinus tachycardia or other cardiac arrhythmias), ECG (decreased ejection fraction and fractional shortening with myocardial dyskinesis), bacterial culture from blood or pericardial and pleural fluid sample, estimation of serum enzymes (cardiac Troponin I, lactate dehydrogenase-I and creatinine kinase - MB isoenzyme), examination for trace element deficiency, toxicological examination of the feed and fodder and necropsy findings (abscesses or inflammation, and pallor/petechiae of the myocardium; flabby heart having thin walls and patches of shrunken, tough fibrous tissue; myocardial vacuolation, calcification and degeneration with necrosis and fibrosis).

Differential Diagnosis

Dilation cardiomyopathy, congenital heart defects, cor pulmonale, nutritional myodegeneration, bacterial endocarditis, cardiac neoplasia, thoracic abscess, pericarditis, pleuritis and diaphragmatic hernia.

Treatment

Corticosteroids (dexamethasone @ 0.5 mg/kg BW, IM/IV) in severe toxaemia, lignocaine (@ 0.25-0.5 mg/ kg BW, slow IV, can be repeated after 15 minutes) for the treatment of ventricular tachyarrythmias. Symptomatic and supportive treatment including vitamin E and Selenium administration (@ 1 ml/25-50 kg BWIM).

Prevention and Control

Adequate supplementation of vitamin E, selenium, and copper in the feed with provision of good quality feed and fodder without any toxin or toxic plant.

Sample Collection for Diagnosis

Blood for bacterial culture, estimation of serum enzymes and trace minerals.

1.11.4 Bovine Endocarditis

Definition and Etiology

Inflammation of the endocardium, due to bacterial infection, mostly leads to valvular lesions and systolic insufficiency. The bacteria causing bovine endocarditis are alpha-haemolytic *Streptococci*, *Trueperella* (*Actinomyces* or *Corynebacterium*) *pyogenes*, *Micrococcus* and *Staphylococcus* spp., *Pseudomonas spp*., *Clostridium chauvoei* (blackleg), *Mycoplasma mycoides*, *Erysipelothrix rhusiopathiae*.

Predisposing Factors

Chronic bacteraemia, Iatrogenic (cardiac/ intravenous catheterization), ongoing septic process such as mastitis, metritis, foot abscess or traumatic reticular peritonitis, and low body condition score.

Clinical Signs

Cardiac murmurs or thrills, persistent tachycardia, constant and moderate fluctuating fever, loss of condition and mucosal pallor, sinus tachycardia and low voltage QRS complex, signs associated with embolism of different organs (embolic nephritis, arthritis, tenosynovitis or myocarditis).

Diagnosis

Diagnosis is based on findings of haematology (nonregenerative anaemia, leucocytosis, neutrophilia, hyperfibrinogenaemia, hyperglobulinemia, hypergammaglobulinemia), bacterial culture (from blood sample at febrile stage), electrocardiography (sinus tachycardia and low voltage QRS complex), echocardiography (valvular lesions characterized by hypoechoic or echogenic masses, and irregular thickening; rupture of the chordae tendinea), radiography (evidence of cardiac enlargement, increased pulmonary vascular pattern, or pulmonary oedema), urine examination (proteinuria and bacteriuria) and necropsy findings (large, cauliflower-like vegetative or small, wartlike verrucose lesions on the atrioventricular or aortic valves; shrunken, distorted and thickened valve leaflets; embolic lesions in other organs).

Differential Diagnosis

Congenital valvular dysplasia, pericarditis, brisket disease and cardiac lymphosarcoma.

Treatment

Long-term use of antibiotics as per the findings of the blood culture and antibiotic sensitivity test.

Administer fluroquinolone group of antibiotics (@ 5-10 mg/kg BW IM/IV for five days), Aspirin (100 mg/kg/day orally or SC); low dose of sodium heparin (30-40 U/kg SC / IV twice daily) to prevent platelet adhesion at the endocardial surface. Furosemide (@ 1-2 mg/kg, IM/ IV) for managing volume overload caused due to valvular lesions and digoxin (at a loading dose of 22 µg/kg BW IV, followed by a maintenance dose of 11 µg/kg BW thrice daily) to counter congestive heart failure.

Prevention and Control

Proper asepsis with intravenous administration and other invasive procedures.

Sample Collection for Diagnosis

Blood, serum, endocardial tissue at the time of necropsy.

1.12 Reproductive System Diseases

1.12.1 Anoestrus/ Sub-oestrus

Definition and Etiology

Anoestrus, one of the most common reproductive disorders in large and small ruminant species, can be defined as absence of periodic manifestation of oestrus, with no palpable follicular and luteal structures (smooth, inactive and quiescent ovaries) called true anoestrus, or absence of normal physiological signs of oestrus associated with a corpus luteum called sub-oestrus/silent oestrus. Major causes are malnutrition with deficiency of micronutrients, loss of body conditions during transition period, suckling, and hormonal disturbances.

Predisposing Factors

Malnutrition with deficiency of macro- and micro-nutrients, non-availability of green fodder, heat stress, loss of body conditions during transition period, suckling by calves, high yielding dam, hormonal disturbances, heavy parasitic infestations and chronic wasting diseases (brucellosis, FMD, tuberculosis, paratuberculosis), debility and senility.

Clinical Signs

Absence of periodic manifestation of overt signs of oestrus after attaining puberty for long (pubertal anoestrus) and calving (after 90 days post-calving, post-partum anoestrus) and sometimes very poor expression of behavioural oestrus (sub-oestrus).

Diagnosis

On the basis of history and no sign of heat/oestrus for the long time after attaining puberty or >90 days post-partum. Ultrasonographic examination helps to detect different types of anoestrus based on the presence and growth of follicular and luteal structures. Ovaries of affected dairy animals appear small, quiescent, flat and smooth on per-rectal examination with low levels of blood calcium, phosphorus, glucose, total proteins and progesterone concentration (<1ng/ml) in true anoestrus cases. However, milk or blood progesterone concentration >1 ng/ml with the presence of corpus luteum indicates sub-oestrus.

Differential Diagnosis

Pregnancy, uterine pathology, cystic ovarian disease, *etc*., must be differentiated carefully.

Treatment

Treat anoestrus according to the causative factor being a multi-factorial functional ovarian disorder of dairy animals. Prescribe high energy ration, concentrates, minerals and trace elements including vitamin A to address nutritional deficiency. Simple ovarian massage and Lugol's iodine painting on posterior part of the cervix. Hormonal (GnRH, progesterone oral/ injection/skin implant/vaginal pessaries alone or in combination with PGF2α) and herbal preparations to induce cyclicity in true anoestrus cases. Administer PGF2α Injection for the treatment of sub-oestrus/silent oestrus after ruling out the pregnancy.

Prevention and Control

Provision of sheds, shade, shower, and wallowing to the buffaloes during summer improves anoestrus management. Provide high energy ration during transition period after parturition and growing heifers along with proper deworming. Awareness about proper oestrus signs, record keeping, and detection of oestrus twice or thrice daily help to manage anoestrus condition.

Sample Collection for Diagnosis

Blood samples for the estimation of progesterone, calcium, phosphorus, glucose, and total proteins levels.

1.12.2 Repeat Breeding

Definition and Etiology

Repeat breeding (cyclic non-breeder) animals are those who are coming into heat regularly but fail to conceive with three or more than three inseminations from an apparently normal healthy bull or semen. The clinical examination does not reveal any definite conditions to explain the failure of conception. Fertilization failure and early embryonic death are two most important causative factors.

Predisposing Factors

Infection of the genital tract, poor quality semen/low fertile bulls, poor reproductive health management, anatomical problems like a cervical abnormality, uterine adhesions, ovaro-bursal adhesions, large sized herd, early and late reproductive stages.

Clinical Signs

The animal does not conceive after service and returns to heat repeatedly after about 20-22 days of breeding/oestrus.

Diagnosis

Diagnosis is based on the history of breeding, reproductive health management practices, and per-rectal examination of the genital organs at 10-11 days interval for the presence of corpus luteum. Tupal patency test - phenolsulphonphthalein (PSP) dye test - helps to detect normal oviducts.

Differential Diagnosis

Uterine pathology, oviductal blockage.

Treatment

Treat Repeat breeding animals with the following interventions – intrauterine broad-spectrum antibiotic therapy at oestrus followed by insemination at the subsequent oestrus; injection of hCG or GnRH to combat anovulation or delayed ovulation; and injection of progesterone, GnRH, hCG post-AI during luteal phase of the cycle to support the CL functions and establishment of pregnancy.

Prevention and Control

Proper management of calving and post-partum period to avoid uterine infection. Ensure proper oestrus detection and right-time insemination with properly thawed quality semen.

Sample Collection for Diagnosis

Cervical mucus for detection of pathogens and drug sensitivity in the unsuccessful treatment cases.

1.12.3 Cystic Ovarian Disease

Definition and Etiology

Cystic ovarian disease (COD) also called ovarian cysts/cystic ovaries are the persistence of large follicular structures in either of the ovary on account of ovulation failure with aberrant reproductive function. Normally, size of a cyst is >25 mm in diameter if single but may be less when multiple cysts are present. Hormonal deficiency especially luteinizing hormone (LH) is the major cause. Ovarian cysts can be classified as either follicular or luteal. Follicular cysts are thin walled, fluctuating structures that can be single or multiple. Luteal cysts are thick-walled and are usually single and differentiation by ultrasonography is generally more accurate than by manual palpation.

Predisposing Factors

Hereditary predisposition, stress, higher milk yield, age, and plane of nutrition.

Clinical Signs

The main clinical signs are persistent oestrus, anoestrus, or masculinization. In follicular cyst, the characteristic symptoms are nymphomania in which cows display excessive, prolonged signs of oestrus, with oedematous swelling of the vulva, frequent and copious discharge of clear mucus, and a shortened interval between successive heats. Such cows are sexually aggressive as a bull, often called buller; attempt to ride other cows and will also stand to be mounted by other cows. In long-standing cases of nymphomania, the relaxation of pelvic ligaments causes tipping of the pelvis and elevation of the tail-head called sterility hump. Luteal cyst is characterized by the long anoestrus. Prolonged cases of COD can result in mucometra, in which there is distension of the uterus with mucoid fluid, thinning of the uterine wall, and cessation of oestrus. The owner believes that the cow is pregnant, and the clinician should cautiously examine such cows.

Diagnosis

The diagnosis is based on history, symptoms, clinical examination either per rectal or by ultrasound examination, and progesterone concentration in milk/ blood. Follicular cyst is thin walled, fluid-filled structure ≥ 2.5 cm in diameter, either single or frequently multiple in one or both ovaries. Affected cows are either anoestrous or nymphomaniac with

low peripheral progesterone concentrations (< 1 ng/ml). Luteal cyst is thick walled, fluid-filled structure ≥ 2.5 cm diameter and usually single associated with high peripheral progesterone concentrations (>1 ng/ml) and affected cows are anoestrous.

Differential Diagnosis

Follicular cyst with luteal cyst and vice-versa and other cystic conditions of ovary.

Treatment

Treat follicular cyst with the use of hormones, *viz*., hCG and GnRH and luteal cyst with the PGF2α. Manual rupture of cyst by applying digital pressure per rectum is one of the oldest methods but not advocated as it leads to ovarian trauma and ovaro-bursal adhesion.

Prevention and Control

Provide good managemental practices to reduce stress and high plane of nutrition.

Sample Collection for Diagnosis

Blood/milk for progesterone estimation.

1.12.4 Uterine infections – Endometritis, Metritis, Pyometra

Definition and Etiology

Metritis, endometritis (sub-clinical and clinical), and pyometra are most commonly reported uterine abnormalities. Among all, endometritis is most commonly encountered under field or farm conditions in bovines. Metritis is commonly seen during the puerperal period with systemic illness, fever, and an enlarged uterus with a watery or purulent discharge. Clinical endometritis is defined as a purulent or mucopurulent discharge after calving with no signs of systemic illness. Sub-clinical endometritis is defined as the presence of inflammatory cells within the uterine lumen but without signs of clinical endometritis. Endometritis, clinical endometritis, and subclinical endometritis are often used interchangeably. Pyometra is characterized by a collection of purulent exudates of variable amounts within the uterine lumen. It may be closed or open. A wide range of bacterial species contaminate the uterus, but clinical disease is only associated with particular uterine pathogens, including *E. coli*, *Arcanobacterium pyogenes*, *Prevotella* spp., *Fusobacterium* spp. and *P. melaninogenicus*. Apart from bacterial agents, several fungi, *viz*., *Candida albicans*, *C. tropicalis*, *C. pseudotropicalis*, *C. guilliermondii*, Mucor, and *Aspergillus* spp. were associated with endometritis and endo-cervicitis. Uterine abnormalities delay recovery of ovarian function, uterine involution, increase days open and thus extend the calving interval.

Predisposing Factors

Bacterial contamination of the uterus especially after calving, dystocia, premature birth, retained placenta, induction of parturition, abortion, foetal maceration, laceration of the genital tract, uterine inertia, lack of exercise, injury during insemination, unhygienic conditions at the time of calving, lactational stress, *etc*., predispose to develop uterine infections. Some metabolic diseases like ketosis, hypocalcaemia, and overfeeding during the dry period also cause endometritis.

Clinical Signs

In metritis, there will be systemic illness, fever, and an enlarged uterus with a watery or purulent discharge. Purulent or mucopurulent discharge with no signs of systemic illness is observed in clinical endometritis, however, sub-clinical endometritis goes without any observable symptoms except the presence of inflammatory cells in the uterine lumen. In pyometra, there is collection of purulent exudates of variable amounts within the uterine lumen.

Diagnosis

Diagnosis of metritis, endometritis, or pyometra is usually done by rectal palpation and fortuitous observation of the patterns of discharge. Diagnosis of endometritis and particularly sub-clinical endometritis by rectal palpation is often challenging because uterine size and palpable quality of content may vary between individuals and strongly based on subjective assessment. In addition, vaginoscopic examination, endometritis clinical score, white side test, ultrasonography, and uterine biopsy are used for efficient detection of endometritis. Ultrasonographically, pyometra is characterized by the presence of a corpus luteum on an ovary, an accumulation of fluid of mixed echo density in the uterine lumen, and distention of the uterus.

Differential Diagnosis

Among the different uterine pathology conditions as well as mucometra, uterine abscesses, and tumours

standout .

Treatment

Ideal therapy for uterine infection should involve eliminating bacteria from the uterus without inhibiting the normal uterine defense mechanisms and reducing the chances of adulteration of milk or meat for human consumption. Treatment in principle is based on the severity of the condition and clinical observations, include use of antibiotics, NSAIDs, hormones, uterotonics. The most commonly used antibiotics include tetracycline, amoxicillin, ampicillin, and sulphonamides, trimethoprim, cephapirin, ceftiofur, and benzyl penicillin. The antibiotic selection should be based on legal restrictions, effectiveness against Gram negative and anaerobic microbes, and the form and severity of disease. The most frequently used NSAIDs are flunixin meglumine, ketoprofen, meloxicam, and carprofen. Hormones and uterotonic drugs include oxytocin initially in the first few hours after calving and synthetic or natural prostaglandin F2α later. Supportive therapy includes fluid therapy, calcium, and energy supplementation. The immunomodulators used in the treatment of endometritis include endotoxins (lipopolysaccharides of *E. coli*), serum, plasma or hyperimmune serum, colostrum whey, polymorphonuclear (PMN) cells extracts and its components, bacteria-free filtrate, oyster glycogen, leukotriene B4, granulocyte-macrophage colony-stimulating factor (GMCSF), herbal extracts like *Tinospora cordifolia*, neem oil and others.

Prevention and Control

Provide balanced ration with supplements, housing with a spacious calving pen and a clean environment to avoid infection during and after parturition and good management practices. With the global spread of antibiotic resistance, there is a growing demand for developing alternative treatment options and focusing more on prevention in farm animal medicine. Therefore, problems of uterine infection can be minimized by using certain immunomodulators in the early stages of postpartum, adopting a standard set of diagnostic protocols along with package of practices in managing herd hygiene.

Sample Collection for Diagnosis

Biological samples collected are blood, uterine swabs, or cervicovaginal mucus for bacterial culture, drug sensitivity and endometrial cytology using cytobrush for cellular morphology and PMNs cell infiltration percentage.

1.12.5 Cervico-Vaginal Prolapse

Definition and Etiology

Protrusion of both the cervix and almost the entire vagina through the vulvar opening is called cervico-vaginal prolapse, while the whole or part of the vagina protrusion called vaginal prolapse. It occurs mostly in late gestation (last 2 to 3 months) and incidence is usually higher in buffaloes as compared to cows. If the cervical seal is disturbed, there is a danger of septic abortion. Increased intra-abdominal pressure is due to increased size and weight of the uterus during pregnancy. Sometimes, it has hereditary predisposition.

Predisposing Factors

Increased intra-abdominal pressure due to increased size and weight of the uterus during pregnancy, rumen distention, intra-abdominal fat, softening of the pelvic girdle, *etc*., may lead to cervico-vaginal prolapse.

Clinical Signs

In the beginning, the mucous membrane, *i.e.*, floor of the part of the vagina lying cranial to the urethral opening protrudes out followed by whole of the vagina and the cervix may protrude out in severe cases. In mild cases, prolapse usually appears when the animal is recumbent and recedes/attains the normal position in standing position. Thrombosis, ulceration, necrosis of the prolapsed mass with toxaemic signs and even rectal prolapse are seen in severe cases due to injury, infection and irritation that lead to enhanced straining.

Diagnosis

Appearance of cervico-vaginal mass from the vulva.

Differential Diagnosis

Rectal and uterine prolapse.

Treatment

Successful management involves reduction, replacement and retention of prolapsed mass under epidural anaesthesia (2 percent lignocaine HCl, 4-8 ml). Clean the prolapsed mass with non-irritant

antiseptic solution and replace with the palm taking utmost care avoiding trauma. Retain the prolapsed mass with either tape or stout nylon suture tying the vulva and perineal skin in mild cases. In severe cases, retention is achieved by a horizontal mattress across the vulval lips involving some portion of perineal skin. Provide parenteral administration of calcium and phosphorus preparation along with fluid therapy and broad-spectrum antibiotic.

Prevention and Control

Provide regular exercise during pregnancy. Ensure supplementation of calcium during last trimester of gestation.

1.12.6 Uterine Prolapse

Definition and Etiology

In this condition, the uterus gets everted out of the birth canal and is also called casting of the calf-bed. It mostly occurs within 24 hours of calving and the main causes are dystocia, straining, retained foetal membranes (RFM), and mineral (calcium and phosphorus) deficiency. Strong tenesmus after the second stage of labour, excessive relaxation of the perineal and pelvic region, and also forced extraction of the foetus may lead to uterine prolapse.

Predisposing Factors

Advance stage of pregnancy, lack of exercise, confinement of the animals, slopy sheds, mineral deficiency (Ca, P, Mg), faulty correction of dystocia, winter months, and prolonged recumbency.

Clinical Signs

The uterus is everted out of the vulva with visible placentomes on the exposed endometrium, and the cervix is present at the level of the vulva. The prolapsed organ is often highly contaminated with bedding material, faeces, and dirt. Bleeding may occur due to injury to the endometrium or placentomes and dam may go under shock due to blood loss. Animals show restlessness, pain, anxiety, straining, weakness, depression, increased respiration rate, subnormal temperature, struggling, and prostration are observed in most of the cases.

Diagnosis

Visually by seeing the prolapsed mass hanging through the vulva.

Differential Diagnosis

Cervico-vaginal prolapse, vaginal prolapse, rectal prolapse.

Treatment

Successful management involves reduction, replacement and retention of prolapsed mass under epidural anaesthesia (2 percent lignocaine HCl, 4-8 ml). Isolate the animal with restricted movement to prevent injury and keep the prolapsed mass moist with sterile (boiled in water) clean cotton cloth. Do correction in sternal recumbency with both hind legs pulled out towards the backside. Clean the prolapsed mass with non-irritant antiseptic solution (1 percent solution of potassium permanganate) and it should rest on elevated surface to prevent stretching of the organ and constriction of blood vessels. If the bladder is present inside the prolapsed uterus, it should be evacuated first by elevation of the prolapsed mass. Push gently the prolapsed mass with cupped palms taking utmost care avoiding trauma/rupture by using finger pressure. Push the organ first at the cervical end near the vulva then the lower portion and then all. After repositioning, the organ should be gently rocked and shaken to ensure complete retention and to reduce the chance of re-prolapse. Use broad-spectrum antibiotic inside the uterus to prevent infection. Apply horizontal mattress suture to the vulva to prevent re-prolapse. Parenteral administration of calcium magnesium borogluconate increases the tone of uterine muscles and ligaments and reduces the chance of re-occurrence along with fluid therapy, broad-spectrum antibiotic.

Prevention and Control

Provide balanced ration and mineral mixture with extra calcium, as a regular feed supplement, to pregnant animals. Ensure regular exercise to the stall-fed animals.

1.12.7 Uterine Torsion

Definition and Etiology

Uterine torsion can be defined as the rotation/revolution/twisting of the pregnant uterus on its longitudinal axis leading to the obstruction of birth canal and causing dystocia. Torsion is most common in buffaloes than in cattle. Exotic breeds

(Bos taurus) of cattle are more susceptible to torsion than indigenous breed *(Bos indicus)*. Torsion is usually seen in the last trimester of the pregnancy and in stall-fed animals. The important cause is the anatomy of the broad ligament and its attachment to uterus in bovine (broad ligament is attached towards lesser/ventral curvature of uterus and the greater/dorsal curvature of uterus remains free making gravid uterus vulnerable for torsion; when dam lies down usually front legs first, the uterus gets temporarily suspended and a sudden jerk or twist at that time causes the uterus to turn on its axis) besides increased foetal weight, reduced amount of amniotic fluid and low ratio of foetal fluid to foetal size, transportation during advanced stage of pregnancy, and poor exercise.

Predisposing Factors

Increased abdominal capacity, hilly terrain, slipping, sudden movement of dam, repeated lying down and raising, unsteady walk, stall fed animals, less exercise, and transportation are the factors leading to torsion.

Clinical Signs

Whenever a late gestation pregnancy experiences signs of dystocia, uterine torsion is suspected. Careful anamnesis reveals that owner complaints that dam was about to calve as exhibited by letdown of milk, relaxation of pelvic ligaments, but adequate time has passed and still, there is neither the rupture of foetal water bags nor the appearance of the foetus from vulvar lips. Shrinkage of udder over a period of time was also noticed. In some cases, torsion may be so severe that an external examination of vulva reveals the dorsal commissure being pulled forward and to one side either right or left. In advance cases, foetus may die leading to uterus or vaginal rupture, rupture of major blood vessels and broad ligament attached to the uterus, adhesions to tissue surrounding uterus and necrosis of cervix, constipation, toxaemia, tucked up appearance of dam, finally leading to death of animal.

Diagnosis

History of dystocia, prolonged labour pain, restlessness, uneasiness, kicking at abdomen, *etc.*, with low body temperature, weak and rapid pulse, anaemia, and weakness on external examination. In post-cervical torsion, direction of the spiral folds of vaginal wall will be palpated in the direction of torsion on per-vaginal examination. Per-rectal examination is done to diagnose pre-cervical uterine torsion. In the right uterine torsion – the left-side broad ligament is pulled tightly across over uterine body and cervix. In contrast, in left uterine torsion – the right-side broad ligament is pulled strongly under the uterine body and cervix. Post-cervical, right-side uterine torsion is the most common type of uterine torsion.

Differential Diagnosis

Colic pain, indigestion, and other causes of foetal and maternal dystocia.

Treatment

Schaffer's method is used widely for the detorsion of the uterus in cows and buffaloes with the principle of rolling the dam to the same degree and direction to which the uterus has rotated, keeping the foetus fixed using a wooden plank (9-12 foot long, 8-12 inches wide, and 2-inch thickness). Restrain the dam in lateral recumbency and place a plank of wood on the flank region (upper paralumbar fossa) over the top of the uterus at the side of torsion. The lower end of the plank should be resting on the ground. An assistant stands on the lower end while the dam is being rolled. This anchors the uterus in place thus increasing the chance of success. Caesarean section may be performed as the last resort.

Prevention and Control

Advance pregnant animals should be housed separately on a non-slippery floor and avoid transportation of such animals.

1.12.8 Retention of Foetal Membranes

Definition and Etiology

Retention of foetal membranes (RFM) - earlier called retention of placenta (ROP) - is the most common complication of bovine parturition which denotes failure to expel foetal membranes within 24 hours after parturition. It occurs when the calf's side of the placenta (the foetal membranes) fails to separate from the mother's side. Occurrence of placental retention is associated with the failure of the normal processes of dehiscence and expulsion. Hence, the factors that cause retention of placenta are those that interfere with the detachment of foetal villi from maternal crypts. It is most commonly associated with dystocia, milk fever (metabolic diseases) and twin births.

Predisposing Factors

Abortion (associated with placentitis), abnormal gestation length (prolonged or shortened), dystocia, primary uterine inertia, delivery by caesarean section, fatty liver, deficiency of selenium/vitamin E/vitamin A, failure of placental maturation (twin birth, induced parturition, heat stress) and secondary uterine inertia due to hypocalcaemia.

Clinical Signs

Main clinical sign is hanging of foetal membranes from vulva. In mild/early cases, normal pulse, temperature, appetite and milk yield with no fetid smell is recorded, However, in severe complicated cases, there is high fever, accelerated pulse, straining, reduced appetite and drop in milk yield. Foetal membrane is discoloured and dry with foul smelling reddish discharge. Further, due to ascending infection with dung and filth, the foetal membrane gets inflamed, oedematous and emphysematous.

Diagnosis

Diagnosis is based on history, clinical signs and per-vaginal examination.

Differential Diagnosis

No need being a characteristics symptom of RFM.

Treatment

Manual removal of foetal membranes is the most preferred method and the best time for this is 24-hour post-calving. To avoid microbial contamination, provide broad-spectrum antibiotics intrauterine or parenteral depending upon the severity of the condition. The effective prophylactic measures include use of ecbolic agents (oxytocin, PGF2α, Ergot derivatives), calcium preparation, selenium supplementation alone or in combination with vitamin E.

Prevention and Control

The proper nutrition supplemented with vitamins and mineral mixture during prepartum period should be given to avoid RFM and other metabolic diseases. RFM associated with brucellosis can be prevented by proper immunization.

Sample Collection for Diagnosis

The aseptic sample of retained foetal membrane for *Brucella* diagnosis.

1.12.9 Pseudopregnancy

Definition and Etiology

Pseudopregnancy/cloud burst in goats is defined as an accumulation of aseptic/sterile fluid in the uterine lumen (hydrometra) which is devoid of a viable conceptus and presence of persistent corpus luteum. Pseudopregnancy can occur in all ruminants; however, it is most common in goats of all ages and during the non-breeding season. The incidence is significantly higher in goats mated outside the normal breeding season and those subjected to oestrus synchronization treatment. The etiology is the persistence of functional corpus luteum subsequent to early embryonic death and reabsorption; however, the exact etiology is not known. The other factors include decreased production or release of PGF2α from the endometrium, exposure to phytoestrogens.

Predisposing Factors

Out-of-season breeding, excessive hormonal manipulation of the reproductive cycle, exposure to phytoestrogens, and congenital malformations of the cervix and uterus.

Clinical Signs

There is copious watery or mucoid discharge in the absence of foetuses. Pseudopregnancy in goats is categorized into two types. First, when fertilization occurs after mating followed by early embryonic death, the functional CL persists and does pretend as if she is pregnant. There is abdominal enlargement and udder development, and lactogenesis also occurs. The "does" that are lactating may have a fall in milk yield. This type of pseudopregnancy persists for the duration of the gestation period and even longer till the CL regresses spontaneously. However, when the pseudopregnancy is terminated, the abdominal distension disappears, and the "does" may search for their missing kids. Secondly, when the "does" in oestrus are not mated, the cyclical ovarian activity ceases, but there is no marked hydrometra. The "does" may expel a bloody vaginal discharge when the acyclicity ends.

Diagnosis

Diagnosis is based on history and breeding records, ultrasonography and serum oestrogen sulphate test. Serum estrone sulphate levels can be helpful in differentiating pregnancy and pseudopregnancy

after 50 days of anoestrus. B-mode transabdominal ultrasonography helps in the differential diagnosis of hydrometra by the presence of fluid-filled uterus with the absence of foetus and cotyledons from a normal pregnancy.

Differential Diagnosis

Differential diagnosis from normal pregnancy in goats.

Treatment

Termination of pseudopregnancy by injecting PGF2α results in the expulsion of a large amount of fluid (hydrometra) and oestrus ensues in approximately 4 days. A second PGF2α injection after 12 days further improves fertility and conception rate. Dopamine agonists like bromocriptine (1 mg, SC for 6–10 days) can also be used.

Prevention and Control

Limit 'out of season' breeding, exposure to phytoestrogen, and hormonal manipulation of the reproductive cycle. Ensure balanced diet with selenium supplement.

Sample Collection for Diagnosis

Blood samples for estimation of estrone sulphate to rule out pregnancy.

1.12.10 Dystocia

Definition and Etiology

Dystocia is defined as difficulty in birth process due to inability of the dam to deliver its young one on its own efforts. The major causes of dystocia are classified into maternal (inadequate expulsive forces due to uterine inertia or weak abdominal contractions, inadequate size of birth canal due to inadequate pelvis, incomplete dilation or constriction of birth canal) and foetal causes (foetal oversize, foetal monsters, foetal pathologies like anasarca, ascites and emphysema, and faulty disposition in the presentation, position and posture). Dystocia may lead to the death of a foetus, death of the dam, reduced fertility, sterility, and severe production losses, *etc*.

Predisposing Factors

Breed, parity of dam, sex of foetus, birth weight of foetus, pelvic size of dam, gestation length, nutrition, year and season of calving in addition to the genetic, environmental, and periparturient managemental factors. Risk factors affecting dystocia are broadly grouped into four, *viz*., direct factors (malpresentation and uterine torsion), phenotypic factors (foetus birth weight, pelvimetry, body weight and body condition of dam, gestation length), non-genetic factors (parity of dam, year and season of calving, place of calving, sex of the foetus, nutrition, level of hormones) and genetic factors (genotype of the dam, inbreeding, muscular hypertrophy, selection and quantitative trait loci).

Clinical Signs

Restlessness and abnormal posture of dam due to prolonged first stage labour with no progression towards delivery of calf even 2 hours after rupture of amniotic bag, vigorous straining, and obvious malpresentation, malposture or malposition of foetus. Appearance of detached chorio-allantois, foetal meconium or blood-stained amniotic fluid at the vulva.

Diagnosis

Diagnosis is based on history, clinical signs and gynaeco-clinical examination of the dam for prompt treatment/management.

Differential Diagnosis

Important to distinguish between dystocia and a normal parturition.

Treatment

Dystocia management involves different obstetrical methods – mutation (repulsion, rotation and version), forced traction after correction, caesarean section and foetotomy. Preferred methods of correcting dystocia when foetus is live include mutation, forced traction and caesarean section while mutation, forced traction, caesarean section or foetotomy with standard pre- and post-operative cares when foetus is dead. Resolving dystocia has three goals which include survival of the dam, survival of the foetus, and maintenance of dam fertility. A prompt decision to perform caesarean section is important for optimum success.

Prevention and Control

Adopt standard reproductive and husbandry practices. Early intervention minimizes the losses. Provide balanced ration and timely assistance.

1.12.11 Foetal Mummification and Maceration

Definition and Etiology

When foetus dies usually between 3-8 months of gestation and foetal membranes becomes shrivelled and dried; fluids of allantois, amnion and foetus are resorbed and uterus contracts on foetus and moulds it into a dry contorted mass – this condition called as foetal mummification. The two forms of mummification in domestic animals include haematic and papyraceous types. In bovine, haematic type of mummification is common which is characterized by accumulation of semifluid viscous and adhesive substance of haematogenous origin between uterus and chorion which imparts a reddish-brown colour to the foetus and foetal membrane. If the condition is undiagnosed, mummified foetus will remain in the uterus beyond the normal gestation period which is associated with a persistent corpus luteum interrupting the process of parturition/abortion mechanism to occur. The major causes include torsion or compression of umbilical cord, genetic factor (inherited endocrine defects or autosomal recessive genes), and torsion of uterus. All mummified foetuses and uteri - when examined and samples thereof cultured - are sterile. However, when aborting foetus fails to be expelled due to uterine inertia in a dilated cervix and undergoes a gradual bacterial digestion in the uterus - it is called foetal maceration. Foetal maceration may occur at any stage of gestation. The causes of foetal maceration include uterine inertia, improper dilation of cervix, uterine torsion, and disease conditions like Trichomoniasis and Vibriosis.

Predisposing Factors

Any complication during pregnancy leading to death of the foetus, infection, uterine inertia, uterine torsion.

Clinical Signs

In foetal mummification, pregnant uterus is relatively small and lacks fluid. The pregnant uterus in such situation contains irregular inert foetal mass, no cotyledon, and small uterine artery with no fremitus. In the ovary, palpable corpus luteum is present. In foetal maceration, intermittent straining with a foul, reddish grey vulval discharge is common. On per-rectal examination, one experiences a distended swollen foetus with foetal bones floating in pus or crepitating against each other in the uterus, as well as thick and heavy uterine wall with a large and hard cervix.

Diagnosis: Both the conditions are diagnosed by history, clinical signs and gynaeco-clinical examination. In foetal mummification, clinical examination reveals that the foetus is dead, although the dam is pregnant. Transrectal palpation reveals an irregularly shaped, contracted uterus with a foetal mass but no foetal fluid within it. There is no fremitus in the uterine artery. Ultrasonographic examination of accessible parts of the uterus per rectum confirms the diagnosis. In sheep, foetal mummification can be diagnosed by abdominal palpation supported by transabdominal ultrasonography. Prognosis is grave with and questionable future breeding life if condition existed for longer, causing the greater damage to endometrium.

Differential Diagnosis

Pregnancy complication occurring during mid to late gestation, ovarian and uterine disorders.

Treatment

Rational approach to treat foetal mummification with good success is to induce parturition using $PGF_{2\alpha}$ (@ 25 mg natural/500 µg synthetic analogue, IM) that dilates cervix within 24- 48 hours and remove mummified foetus manually. However, foetal maceration can be best manged by careful removal of bones per vaginum, broad spectrum antibiotic and fluid therapy at the earliest possible.

Prevention and Control

Affected animals are normally culled due to economic considerations.

1.13 Congenital Diseases

1.13.1 Atresia Ani

Definition and Etiology

Atresia ani (or *et recti* or *imperforate anus*) is a congenital condition in which the hindgut fails to fully communicate with the perineum manifested as absence of anus/anal perforation sometimes associated atresia recti, recto-vaginal fistula, recto-cystic fistula, vagino-urethral agensis, tail lessness, hypospadias and cleft scrota.

Predisposing Factors

Atresia ani is associated with chromosomal abnormality, and developmental defects.

Clinical Signs

Absence of anal opening or canalization of rectum, tenesmus, bulging of anal area and abdominal distension. In atresia recti, the blind end does not bulge out on applying abdominal pressure and the defect is usually located near the pelvic brim. Cranial location of malformation causes more serious condition. In females, it is commonly associated with recto-vaginal fistula.

Diagnosis

Based on the history of not passing faeces and straining for defecation since birth and clinical signs.

Differential Diagnosis

Since the condition is identified on the basis of imperforate anal opening, differential diagnosis is not needful.

Treatment

In atresia ani or imperforate anus, a circular incision of 2 to 3 cm diameter is made at the location of anal opening. In atresia recti, blind end of the rectum is freed from the adhesions if present and the mucosa is sutured with the skin using non-absorbable suture. Proper post-operative care should be done to ensure patency of the anal orifice.

Prevention and Control

Animal with a history of this condition should not be bred further/propagated since it is a genetic disease.

1.13.2 Ocular Dermoid

Definition and Etiology

Dermoid cysts are usually congenital and possibly hereditary condition occurring consequent to *in utero* epithelial dislocations.

Predisposing Factors

Genetic factors and embryological developmental anomalies may predispose for formation of ocular dermoids.

Clinical Signs

Visible growth of a hair patch at an aberrant location on the conjunctiva or cornea or both leading to epiphora, blepharospasm, and corneal ulceration or scarring are the prominent clinical signs.

Diagnosis

Diagnosis is based on clinical signs and a thorough ocular examination.

Differential Diagnosis

Clinical signs are so peculiar and obvious so no need of any differentiation from other ocular conditions.

Treatment

Surgical excision of dermoid (with or without superficial keratotomy) under retrobulbar eye block using local analgesics.

Prevention and Control

Exclusion of the affected animals from breeding programs.

1.13.3 Pervious Urachus

Definition and Etiology

It refers to a congenital anomaly where the urachus, a tube-like structure connecting the foetal bladder to the umbilicus during development, fails to close properly after birth.

Predisposing Factors

Hereditary components and intra-uterine factors that affect foetal development or intra-uterine environment may predispose for this condition. In some cases, trauma or mechanical pull at umbilicus immediately after birth also interferes with the normal closure process of the urachus.

Clinical Signs

The most common sign is urinary leakage from the umbilicus or a persistent dampness around the umbilical area.

Diagnosis

Based on clinical signs, physical examination and if needed by contrast radiography and ultrasonography.

Differential Diagnosis

The condition is identified in immediate post-natal life and has peculiar signs hence no need of any differentiation from other ocular conditions.

Treatment

Standard surgical intervention by double ligation of urachus and umbilical vessels proximal to the infection site if any. The infected mass is dissected out and the standard surgical closure of the wound

is done.

Prevention and Control

Exercise caution at the time of umbilical separation and ligation and consider exclusion of such animals from future breeding program.

1.13.4 Contracted Tendon

Definition and Etiology

It is defined as Contracture or shortening of the flexor tendons which results in knuckling of the fetlock joint frequently and of carpal joint rarely. It is one of the most prevalent congenital musculoskeletal abnormalities in neonatal calves. It can result from inherited factors, *in utero* mal-positioning and overcrowding due to foetal size relative to the dam. It may occur with other congenital abnormalities, such as cleft palate, dwarfism, and arthrogryposis.

Predisposing Factors

Maternal nutritional deficiencies during gestation, poor foetal disposition and genetic factors. Acquired tendon contracture may be due to fracture or radial nerve paralysis, physitis or contracture following tendon trauma.

Clinical Signs

Contracted tendon is classified as mild, moderate, and severe. In mild cases, animals exhibit slight metacarpophalangeal flexion, and they step with the tips of their hooves when moving. In moderate cases, calves intermittently bear their weight on the over-flexed joint and in severe cases, calves bear their entire weight on the dorsal surface of the joint and may fall when attempting to stand. Untreated severe cases in calves can lead to the development of severe skin and phalangeal lesions, increasing the risk of suppurating arthritis and extensor tendon rupture.

Diagnosis

Most cases of contracted tendons in calves are detected within the initial days after birth based on clinical signs. In cases where joint or tendinous lesions are suspected, radiographic examination can be useful.

Differential Diagnosis

Arthrogryposis, crooked calf and neurogenic flexion of the limb.

Treatment

Commonly used treatments for mild cases include applying splints and bandages fibreglass/POP cast to keep straight and in severely contracted cases, superficial and/or deep flexor tenotomy is recommended.

Prevention and Control

Breeding of animal with the congenital anomalies should be avoided to prevent the occurrence of congenital contracted tendon in newborn.

1.14 General Systemic Conditions

Pyrexia

Definition and causes

Pyrexia, also known as fever, is rise in body temperature above the normal range due to elevation in body temperature set point. True fever involves body temperatures ranging from 39.5° C to 41.1°C (103° F to 106°F). It is a physiological reaction to infections, inflammation, or other disease processes. The hypothalamic set point can be elevated by exogenous pyrogens, which include drugs, toxins, and products from viruses or bacteria (such as endotoxins). These pyrogens stimulate inflammatory cells to release cytokines known as endogenous pyrogens. Eventually, prostaglandin E_2, produced locally in the hypothalamus, raises the set point, causing fever.

Clinical signs

In animals, clinical signs of pyrexia include an elevated body temperature above 102.5°F, lethargy, appetite loss, and depression. Additional signs can include shivering, dehydration, rapid breathing or panting, increased heart rate, and warm ears and paws.

Diagnosis

Diagnosis of pyrexia in animals can be done thorough physical examination to check elevated body temperature and other signs of infection or inflammation. A detailed medical history, including vaccination status and recent exposures, is essential. Laboratory tests such as a complete blood count, serum biochemistry, and urinalysis help identify underlying causes of pyrexia.

Treatment of pyrexia

NSAIDs (meloxicam in small animals and

phenylbutazone in large animals) and IV fluids (lactate ringer's solution or 0.9% normal saline) therapy may be helpful. Before initiating antimicrobial therapy, obtain samples for culture for antibiotic sensitivity test (ABST). Consider using antimicrobials (antibiotics, antifungals, anthelmintics) (Amoxicillin-clavulanate or Ceftiofur or Enrofloxacin) and immunosuppressive or anti-inflammatory drugs, such as corticosteroids (prednisone and dexamethasone), if a precise diagnosis cannot be made.

Septicaemia or Sepsis

Definition

Septicaemia in animals, also known as sepsis, is a severe systemic infection where pathogens (usually bacteria) enter the bloodstream, causing a widespread inflammatory response. This pathological state is life-threatening and requires immediate veterinary care.

Causes

Septicaemia in animals is primarily caused by bacterial infections originating from wounds of gastrointestinal, respiratory, urinary, and reproductive systems. Viral infections can predispose animals to secondary bacterial infections leading to septicaemia, while fungal and parasitic infections, though less common, can occur in immunocompromised animals. Additional causes include traumatic injuries, surgical complications, and chronic diseases that compromise the immunity.

Clinical Signs

Clinically, the symptoms of septicaemia in animals vary widely but often include high temperature or hypothermia, weakness, and anorexia. Rapid heart rate and breathing, dehydration, and changes in mucous membrane colour (pale, red, or blue gums) are also common. Gastrointestinal disturbances such as diarrhoea, vomiting, and signs of septic shock including collapse, weak pulse, and cold extremities might be observed.

Diagnosis

Septicaemia in animals is diagnosed thorough clinical examination and various diagnostic tests. Blood tests, including blood cultures, complete blood count, liver and kidney function test, electrolyte and lactate levels, are necessary to diagnose the causative organism and assess infection severity. Imaging techniques like radiography, ultrasound, or advanced imaging techniques help to locate the infection source. Additional tests, such as urine analysis, urine cultures, faecal examination, and thoracic or abdominal fluid analyses are conducted, if specific infections are suspected.

Treatment

The status of septicaemia should be carefully assessed. In case of sepsis, due care should be taken to maintain a living subject. For treatment, broad-spectrum antibiotics (combination of a beta-lactam antibiotics ampicillin-sulbactam) and an aminoglycoside (amikacin) or a fluoroquinolone (enrofloxacin) are started immediately to cover a wide range of pathogens, with adjustments made based on culture and sensitivity results. Supportive care consists of oxygen support for respiratory distress, IV fluids (Isotonic crystalloids, *e.g.*, Lactated Ringer's Solution) to regulate blood pressure and hydration, and nutritional support, if the animal is not eating. Specific treatments may involve vasopressors (Dobutamine or Norepinephrine) for regulation of blood pressure (BP), analgesics (Opioids like morphine or fentanyl) for pain management, and surgical intervention for drainage of abscesses and removal of necrotic tissue.

Toxaemia

Definition

Toxaemia is a generalized disease condition brought on by toxins from bacteria or tissue injury. Endotoxaemia is the most prevalent type of toxaemia in large animals, resulting from Gram-negative bacterial lipopolysaccharides in the blood. Endotoxaemia causes various abnormalities, including cardiopulmonary dysfunction, leukopenia, thrombocytopenia, coagulopathies, increased vascular permeability, and decreased organ blood flow, potentially leading to heart and renal failure.

Causative agents

Toxaemia in animals is caused by antigenic and metabolic toxins. Antigenic toxins – include exotoxins and endotoxins – produced by bacteria and helminths stimulate antibody development. Exotoxins (those released from *Clostridium spp.*), and enterotoxins (from *E. coli*) cause specific effects like diarrhoea. Endotoxins released from

Gram-negative bacteria lead to endotoxaemia when released during bacterial growth or cell wall breakdown. Metabolic toxins accumulate due to impaired elimination or abnormal metabolism, especially in liver dysfunction or gastrointestinal obstruction, causing toxaemia.

Clinical Signs

Clinical findings of acute toxaemia in animals include depression, anorexia, muscular weakness, increased heart rate, weak and rapid pulse, and fever, which may later normalize or drop. Severe endotoxaemia can cause cardiovascular collapse, leading to 'toxic' shock resulting in severe vasodilation, pallor, hypothermia, tachycardia, weak pulse, and muscle weakness. Chronic toxaemia manifests as lethargy, inappetence, poor growth, and emaciation. Endotoxaemia can produce bleeding, diarrhoea, and disseminated intravascular coagulopathy (DIC).

Diagnosis of Toxaemia

Diagnosis of Toxaemia includes ***Haematology***: Identify changes in leukocyte counts; mild endotoxaemia shows leucocytosis and neutrophilia, while severe cases exhibit leukopenia, neutropenia, and lymphopenia; ***Serum Biochemistry:*** Observe for low plasma glucose, high serum urea, and low albumin and total protein; ***Electrolytes analysis:*** Monitor for mild hypocalcaemia, hypomagnesemia, hypokalaemia, and hypophosphatemia in adult herbivores, indicating inappetence and reduced gastrointestinal motility; and ***Hemagglutination inhibition assays*** can identify endotoxins in horse blood, although their applicability for routine diagnostics is restricted

Lesions

At necropsy, visible abnormalities are confined to lesions caused by the toxin. Under the microscope, degeneration is observed in the parenchyma of liver, kidney glomeruli and tubules, myocardium and adrenal glands.

Treatment of Toxaemia

Treatment principles for endotoxaemia involves removal of infectious sources and using antibiotics effective against Gram-negative bacteria (Co-administration of aminoglycosides with β-lactams). Providing fluids (lactate ringer's solution or 0.9% normal saline) and electrolyte therapy (Balanced Electrolyte Solutions) to address fluid deficits, hypoglycaemia, and electrolyte imbalances. Considering NSAIDs (meloxicam) or glucocorticoids (Dexamethasone) to inhibit cyclooxygenase pathway products. Additional treatments may include inotropic or vasopressors agents (Dobutamine or Norepinephrine), polymyxin B via IV or intra-mammary routes, and hyperimmune plasma with antibodies against core lipopolysaccharide antigens.

Heat Stroke

Definition

Elevated body temperature - mainly owing to physical reasons – those results from either insufficient heat dissipation or excessive heat production is called hyperthermia or heat stroke. Heat stroke or heat exhaustion is frequently observed in clinical condition.

Aetiology/Etiology

Hyperthermia primarily results from high environmental temperature and intense physical exertion, exacerbated by factors like humidity, obesity, dense hair coats, or poor ventilation. Animal management under heat conditions is crucial due to differences in their tolerance levels, influenced by breed, sunlight exposure, and exercise. The additional aetiology of hyperthermia include dehydration, neurogenic factors, poisonings (*e.g.*, strychnine, dinitrophenols), and specific conditions like malignant hyperthermia in pigs and hyperkalemic periodic paresis in horses.

Clinical Signs

In most animals, hyperthermia is defined by a rise in body temperature above 39.5°C, frequently surpassing 42°C. Sweating, restlessness, and elevated respiration and heart rate are the initial symptoms. These are followed by a decrease in sweating and fatigue. Animals seek cool environments but may exhibit laboured breathing and distress as temperatures rise to 41°C, leading to shallow breathing, rapid weak pulse, collapse, convulsions, and coma at higher temperatures. Prolonged hyperthermia can impact reproductive efficiency in ruminants and cause summer infertility in swine. Horses with hyperthermia experience fatigue, electrolyte imbalances, and impaired sweating, leading to decreased performance and potentially fatal complications.

Lesions

Nonspecific gross observations at necropsy include peripheral vasodilation, delayed blood clotting, early rigor mortis onset, and fast putrefaction. Usually, no particular or persistent histological alterations are seen.

Treatment

Adequate drinking water, shade, and air movement are crucial for animals in high temperatures. In severe hyperthermia, a cold-water hose and fans should be provided to cool an animal shed. Intravenous fluids (such as 0.9% NaCl) are necessary for weak or dehydrated animals.

Control

To mitigate heat stress in livestock, provide adequate shade using trees, roofs, or cloth structures. For dairy cattle and buffalo, offer cool, clean water with ample trough space, use shade and intermittent sprinkler systems, enhance airflow with fans or mounds, adjust feeding times to cooler periods, minimize handling during heat peaks, and select housing based on breed susceptibility.

Dehydration

Definition

Numerous diseases in farm animals involve disruptions in body fluids, electrolytes, and acid-base imbalance. When more fluid is lost from the body than is absorbed, it may result into decrease in circulating blood volume and tissue dehydration.

Etiology

The main causes of dehydration in livestock are decreased water intake and diarrhoea. The primary issue in most cases of dehydration is electrolyte loss.

Clinical signs

In dehydration, dry wrinkled skin that appears shrunken and a sluggish recovery of skin elasticity are the main clinical indicators in animals. Dehydration is more noticeable if electrolyte and fluid loss has occurred over several days. The degree of eye recession into the orbit, which can be determined by rolling the lower eyelid and measuring the recession distance, is the important sign of hydration in calves. Skin elasticity on the lateral thorax and neck is the second-best indicator. Dehydrated animals exhibit rapid weight loss, reduced appetite, and weakness of muscle.

Diagnosis

Dehydration in animals is diagnosed through physical examination and clinical signs like delayed skin turgor, eye recession (especially in calves), dry mucous membranes, delayed capillary refill time, and rapid weight loss. Common signs include dry wrinkled skin, sunken eyes, lethargy, and reduced urine. Severity of dehydration is evaluated by laboratory tests, *i.e.*, total protein, serum electrolytes, packed cells volume(PCV), and specific gravity of urine.

Treatment

Treatment involves fluid replacement (balanced electrolyte solution and 0.45% normal saline solution for hypertonic dehydration) and addressing underlying causes. For mild cases of dehydration, oral rehydration solutions including glucose and electrolytes are utilized; for moderate to severe cases, intravenous fluids like normal saline or ringer's lactate are required.

Acidemia

Definition

Acidemia develops when excessive amounts of acid accumulate in the blood or in tissue. Acidemia refers to the state of abnormally high acidity (pH <7.35) in the body while acidosis refers to the physiologic processes that lead to acidemia. A condition known as acidemia, or non-respiratory acidosis, can be brought on by an excess loss of bicarbonate, and deposition of exogenous or endogenous acid.

Etiology

Acidosis brought on by variations in ion concentrations, such as hyponatremia or hyperchloremia. The other specific causes of acidemia include acute diarrhoea, enteritis, and engorgement of carbohydrate, difficult parturition, shock, kidney damage, or excessive acidifying treatments.

Clinical Signs

In kids of goats and newborn calves, metabolic acidosis manifests as mental depression, muscle weakness, and reluctance to suckle. Severe cases could result into coma and shallow breathing due to respiratory compensation failure. Tachycardia, low BP, and hyperkalaemia may bring about severe complications, including sudden death. Some calves

show minimal dehydration but exhibit significant ataxia and weakness.

Diagnosis

Diagnosis often reveals low blood pH, PCO_2 and bicarbonate levels, along with elevated D-lactate and blood urea nitrogen (BUN).

Treatment

Depending on the extent of the disease condition, intravenous isotonic 1.3-1.4 % solution of sodium bicarbonate solution is given as treatment.

Alkalosis

Elevated blood pH is the hallmark of alkalemia, also known as metabolic alkalosis, which is often driven on by excessive acid loss, increased absorption of alkali, or a carbon dioxide deficiency.

Etiology

Metabolic alkalosis commonly arises from condition like abomasal atony, which may occur due to abomasal impaction. Under these events, potassium and hydrochloric acid are continuously secreted into the abomasum, resulting in hyperchloremic and hypokalaemia alkalosis. Furthermore, metabolic alkalosis can cause a shift in potassium from extracellular to intracellular locations, which may exacerbate hypokalaemia even in cases where the body potassium levels are acceptable.

Clinical signs

Animal alkalosis may be challenging to diagnose due to a lack of specific clinical symptoms. It frequently causes convulsions, tetany, tremors in the muscles, and shallow respirations. In more severe phases, dyspnoea and hyperpnea can appear.

Treatment

Treating alkalosis in animals involves fluid therapy with 0.9% isotonic saline along with electrolyte management, including giving potassium chloride (KCl) at 20-40 mEq/L IV, are crucial.

Electrolyte imbalances

Definition and causes

Net losses from gastrointestinal diseases account for most animal electrolyte abnormalities. The etiologies of electrolyte imbalance include vomiting, burn exudation, sweating, and excessive salivation. Nevertheless, these conditions are less prevalent in livestock, except ruminant dysphagia and horse sweating.

Clinical signs

Animals with electrolyte imbalances exhibit tremors, seizures, cardiac arrhythmias, weakening in their muscles, and lethargy.

Diagnosis

Diagnosis of electrolyte imbalances in animals involves clinical assessment for symptoms like weakness, tremors, seizures, or abnormal heart rhythms. This is followed by specific blood tests to quantify levels of calcium, sodium, chloride, potassium, and phosphorus. Employing tests to analyse renal function and acid-base imbalance is helpful in arriving at a diagnosis.

Treatment

Intravenous fluids and electrolyte supplements, such as calcium gluconate for hypocalcaemia, potassium chloride for hypokalemia, 10% sodium phosphate (monohydrate) solution for hypophosphatemia, and 25% Epsom salt solution for hypomagnesaemia, are employed to correct the ionic imbalance. Regular monitoring of blood electrolytes and clinical status is crucial.

Burn

Definition

An injury to the skin or other tissues brought on by exposure to heat, chemicals, electricity, or radiation is called a burn.

Causes

Burns can occur by thermal sources such as flames, hot liquids, or steam; chemical exposure to acidic or alkaline substances; electrical currents passing through the body; and prolonged exposure to UV rays or other radiation sources.

Clinical signs

According to extent of severity and depth, burns are categorized into three degrees, First Degree, Second Degree, and Third Degree. Burns of the first-degree cause discomfort, redness, and some swelling without blistering. Skin in second degree burns turns in red blistered skin with swelling and severe pain. The skin in third degree burns is charred, white or black, and may be numb as a result of nerve loss.

Diagnosis

Assessing burn severity in an animal requires a complete history along with physical examination. The burn areas can be inspected visually for signs like redness, blistering, charring, pain and tenderness. The severity of the burn is determined using tools like the Rule of Nines adapted for animals. Additional assessments can be done to identify potential complications including blood tests and monitoring for shock.

Treatment

For first degree burns, rinse with cold water, apply ointments, use pain relievers, and cover with a sterile bandage. Second-degree burns need 10-15 minutes of cold-water rinsing, sterile bandaging without popping blisters. Third-degree burns require emergency veterinary attention, cool, moist sterile bandages, shock monitoring, and advanced treatments like fluids (5% glucose saline), antibiotics (Topical antibiotics like silver sulfadiazine cream on burn wounds and systemically used common antibiotics include cephalosporins, amoxicillin-clavulanate, or fluoroquinolones, depending on the suspected bacteria), and potential surgery.

Anorexia

Definition

Anorexia in animals refers where an animal experience decreased appetite and reduced food intake, leading to significant weight loss and potential health complications.

Causes

Anorexia in animals is the first sign of any disease or discomfort and can result from abrupt dietary changes or ingestion of indigestible substances, leading to digestive disturbances. Inadequate digestive secretions, dental problems and liver disease can impair nutrient processing and diminish appetite. Prolonged antibiotic use may disrupt gut flora and impacting digestion. Additionally, bacterial, viruses, and parasitic infections can cause inflammation of digestive tract, causing discomfort and reducing the desire to eat.

Clinical signs

Decreased feed intake and loss of appetite in animals with digestive disturbances often coincide with slow or weak ruminal movements, indicating potential digestive problems in ruminants. Signs like decreased milk production, weight loss, weakness, and dehydration like sunken eyes or dry gums are usually noticed in anorexic animals.

Diagnosis

Diagnosis is made through comprehensive assessment of blood, urine, and feed analysis.

Treatment

To aid with digestion, rumenotonics (magnesium hydroxide) or stomachics (nux vomica powder, thyme, asafoetida, ginger and capsicum) might be given. Multivitamins (Vitamin B complex) and hepatoprotective agents (Liv 52) may be administered to improve overall health condition. Antimicrobial drugs (penicillin, oxytetracycline or chloramphenicol) may be recommended in infections. Prevention and control strategies focus on maintaining good nutrition and regular veterinary supervision to optimize animal health and prevent recurrence of digestive issues.

Poisoning

Definition

When a hazardous material is swallowed, breathed, injected, or absorbed through the skin, it can cause poisoning in animals, which can have a negative impact on their health. The nature and amount of toxin exposure determines the degree and type of poisoning.

Causes

Ingested poisons in animals include certain feed and toxic plants. Household chemicals such as antifreeze, cleaning products, rodenticides, and pesticides, also pose significant risk and cause poisoning. Inhaled poisons include gases and fumes from sources like carbon monoxide, smoke, and chemicals such as bleach and ammonia. Injected poisons primarily come from venomous snake bites, insect stings, or spider bites, while absorbed poisons are typically from topical insecticides or herbicides contacting the skin.

Clinical signs

In animals, common signs of poisoning include vomiting, diarrhoea, drooling, and loss of appetite. Respiratory problems like coughing and difficulty in breathing, as well as neurological symptoms including tremors, convulsions, and tiredness

are also observed. Animals can also demonstrate symptoms like jaundice, altered behaviour, excessive salivation, irregular pulse, and a shift in urination.

Diagnosis

Diagnosing poisoning in animals involves a detailed history of potential toxin exposure, physical examination, and conduction of some specific diagnostic tests such as blood test and urine analysis.

Treatment

Animal poisoning is treated by quickly eliminating the toxin, giving activated charcoal to absorb ingested toxins, and giving intravenous fluids containing electrolytes (balanced electrolyte solution) to maintain renal and hydration functions. The first and foremost aim should be to maintain the vital parameters followed by tentative treatment. Specific antidotes, such as atropine for organophosphate poisoning or Vitamin K for anticoagulant rodenticide exposure, can be given. Symptomatic treatment, including seizure control and respiratory support, is important for stabilizing the condition of the animals.

Sudden death

Definition

Sudden death in animals refers to the rapid and unexpected demise of an animal without exhibiting signs of illness or distress. Usually, this kind of death happens minutes to hours after the symptoms start.

Causes

Sudden deaths in animals occur due to spontaneous internal haemorrhage, which can result from cardiac tamponade in cows, ruptured aorta or atrium, inherited aortic aneurysm or verminous mesenteric arterial aneurysm in horses, and esophagogastric ulcer or intestinal haemorrhagic syndrome in pigs. Rapid death can result from digestive disorders such as per acute enteritis, volvulus, and stomach rupture. Per acute endogenous toxaemia can arise from the rupture of the stomach, abomasum, or colon, while exogenous toxaemia could result from snakebites. Iatrogenic deaths may occur due to overdose or improper administration of medications. In horses, cardiovascular accidents and massive haemorrhage are common causes, with gastrointestinal and respiratory tract lesions also significant contributors. Apart from these factors, allergies, dietary inadequacies, poisoning, viral infections, and lightning strikes may also result in deaths in a group of animals. Anaphylaxis from injections can also lead to sudden deaths, particularly in piglets with low selenium-vitamin E status.

Clinical signs

Sudden death in animals is often an unexpected and distressing event. Clinical signs can include general weakness or lethargy, respiratory distress and cardiovascular collapse or fainting. Neurological symptoms, including seizures or sudden loss of consciousness, and gastrointestinal signs such as vomiting or diarrhoea, may also occur.

Diagnosis

Diagnosing the cause of sudden death in animals requires a comprehensive approach. The process begins with a detailed history and physical examination to gather clues about the possible cause. A necropsy, or animal autopsy, is often the most definitive method for determining the cause, involving a detailed examination of the animal's body by a veterinary pathologist. Toxicology tests are crucial if poisoning is suspected, requiring analysis of blood, stomach contents, and tissue.

Treatment

When sudden death occurs, immediate treatment is not possible, but prompt action can save an animal showing severe symptoms. Emergency measures like Cardio-pulmonary Resuscitation (CPR), oxygen therapy, and fluid resuscitation are crucial in critical situations. Administering medications, such as anti-seizure drugs or antidotes for poisoning, may stabilize the animal. Surgical intervention might be necessary for trauma or acute conditions. Veterinary hospitals provide vital support through intensive monitoring and care during these emergencies.

1.15 Annexure-I Drugs usage in ruminants

Drugs	**Dose (mg/kg BW); Route; Frequency/Day**
Abbreviations: sid (24 h) - Every day; bid (12 h) - Twice a day; qid (6 h) - Four times a day; qod - Every other day; tid (8 h) - Three times a day; Inj - Injection; IM – Intramuscular; IV - Intravenous; PO - Orally; SC - Subcutaneous; SD - Single Dose; TD - Total Dose; LA - Large Animal; SA - Small Animal	
Antimicrobial drugs: Use broad-spectrum antibiotics for 3-5 days. However, clinician should choose appropriate antimicrobial drug and its usage on the basis of system affected, clinical condition of the animals and antibiotic sensitivity test.	
Amikacin sulphate	LA/SA: 7; IM/IV; tid
Amoxicillin sodium	LA/SA: 22; IM/SC; bid
Amoxicillin trihydrate	LA/SA: 11-22; IM/SC; sid/bid
Amoxicillin sodium + Sulbactam sodium	LA/SA: 7-10; IM/IV; bid
Ampicillin sodium	LA/SA: 22; SC/ IV/IM; bid
Ampicillin trihydrate	LA/SA: 4-22; IM/SC; sid/bid
Cefotaxime	LA/SA: 10-11; IM/SC; bid
Ceftiofur sodium	LA/SA: 1.1-2.2; IM/IV; sid
Ceftriaxone	LA/SA: 5-10; IM/IV; bid
Enrofloxacin	LA/SA: 2.5-5; IM/SC; sid
Florfenicol	LA/SA: 20; IM; Repeat after 48h/40; IM; SD
Gentamicin sulphate	LA/SA: 2.2-6.6; IM/IV; bid/tid
Marbofloxacin	LA/SA: 2; IM/IV/SC; sid
Oxytetracycline	LA/SA: 5-20; IV/IM; sid/bid
Penicillin G, benzathine (Long Acting)	LA/SA: 44000 – 66000 IU; IM/SC; 48-72 h
Penicillin G, procaine	LA/SA: 10,000-60,000 IU; IM/SC; sid/bid
Streptomycin	LA/SA:11; IM/SC; bid
Sulphadoxine/Trimethoprim	LA/SA: 15; IM/SC/IV; sid/bid
Sulphadimethoxine	LA/SA: 55-110, PO; sid
Sulphonamide/Trimethoprim	LA/SA: 15-30; IM/IV/PO; sid/bid
Tylosin	LA/SA:18; IM; sid
Anthelmintic drugs: repeated/ extended based on parasitic/ faecal eggs count load.	
Albendazole	LA/SA: 7.5-10; PO; SD
Closantel	Sheep: 10; SC/PO; SD
Fenbendazole	LA/SA: 5-10; PO; SD
Ivermectin	LA/SA: 0.2; SC; SD
Levamisole	LA/SA: 5.5-11; PO; SD LA/SA: 3.3-8.0; SC; SD
Morantel tartrate	LA/SA: 8-10; PO; SD
Moxidectin	LA: 0.2; PO/SC; SD SA: 0.2-0.5; PO/SC; SD
Pyrantel pamoate	LA/SA: 25; PO; SD
Triclabendazole	LA/SA: 10-12; PO; SD
Antifungal drugs	
Griseofulvin	LA/SA: 10-20; PO; sid
Whitfield's ointment (Benzoic acid 6% and Salicylic Acid 3% w/w) mixed with excipient (Emulsifying Wax 21.84 w/v)	LA/SA: Topical; bid

Drugs	Dose (mg/kg BW); Route; Frequency/Day
Anti-Inflammatory agents: NSAID'S indicated to control inflammation, fever and pain.	
Carprofen	LA: 1.4; SC/IV; SD
Flunixin meglumine	LA/SA; IM/IV; sid/bid
Meloxicam	LA/SA: 0.5, IV/SC/IM; SD LA/SA: 0.5-1.0; PO; 24-48 h
Ketoprofen	LA: 2-4; IM/IV; sid
Tolfenamic acid	LA: 2.0; IM; sid
Corticosteroids	
Prednisolone	LA/SA: 1-4; IV; SD/sid
Dexamethasone	LA/SA: 0.02-2.0 (Anti-inflammatory)/ 5-20 (ketosis)/ TD -20-30 (Induction of parturition), TD; IV/IM; SD/sid
Anti-histaminic Drugs	
Diphenhydramine hydrochloride	LA/SA: 0.5-1.0, IV/IM; tid/qid/bid
Pheniramine Maleate	
Chlorpheniramine Maleate	
Multivitamins: To improve the efficacy of standard therapy. Dose should be as per the availability of preparations and manufacturer instructions.	
Vitamin A	LA: 440 IU; IM; sid
Vitamin D3 (Cholecalciferol)	LA: 10 million IU TD; IM (2-8 days before calving)
Vitamin E (Tocopherol acetate) and Se	LA: 2 ml/45 kg BW; IM
Thiamine hydrochloride (vitamin B1)	LA/SA: 5-50; IM/IV; bid
Ascorbic acid (Vitamin C)	LA (Calves): 3 g TD; SC; SD
Haematinics	
Iron dextran	LA/SA: 2.0; IM; SD
Immunomodulators: To improve the efficacy of standard therapy.	
Levamisole	LA/SA: 2.5; SC; SD
Cardiac glycoside: For Congestive Heart Failure	
Digoxin	0.022 loading dose then 0.0034; IV, 4 h interval
Diuretic	
Furosemide	LA/SA: 0.5 -1.0; IV/IM (Adult Cattle); sid/bid
Mannitol	LA/SA:1-3g; IV; SD
Common drugs	
Ammonium Chloride	LA/SA: 50-200; PO; sid/bid
Potassium Iodide	LA/SA: 1.5; IV; sid
Kaolin pectate	LA/SA: 0.25-1mL; PO; qid
Ferrous Sulphate	LA/SA: 10-30; PO; sid
Magnesium Oxide	LA/SA: 1000-2000; PO; SD
Magnesium Hydroxide	LA: TD - 400-500g; SA: 10-30g; PO; sid/bid/tid
Magnesium Sulphate	LA: TD – 200-300 g; PO; SA: 30-50 g; PO

Drugs	Dose (mg/kg BW); Route; Frequency/Day
Fluid and electrolyte: Crystalloids are balanced and similar to plasma when contain electrolytes (K, Mg, Ca) in addition to Na and Cl. Lactated Ringer's is a balanced solution than the normal saline. Dose should be as per the clinical condition of the animal till hydration/ normalization. However, clinician can assess the degree of dehydration based on symptoms like skin tenting, sunken eyeball, capillary refilling time and body temperature. Mild dehydration (6-8%): Slight eyeball recession, skin tent slightly prolonged (2-4 seconds), mucous membranes moist; Moderate dehydration (8-10%): Eyes obviously sunken, skin tent obviously prolonged (4-8 seconds), mucous membranes tacky; Severe dehydration (10-12%): eyes severely sunken into orbits, skin remains tented indefinitely, mucous membranes dry.	
Maintenance dose	LA/SA: 50-80ml/kg/day; IV; Flow rate: 7ml/kg/hr
Based on degree of Dehydration	
4-6%	LA/SA: 20-25 ml/Kg BW; IV
6-8%	LA/SA: 30-50 ml/Kg BW; IV
8-10%	LA/SA: 50-80 ml/Kg BW; IV
10-12 percent	LA/SA: 80-120 ml/Kg BW; IV
Oral electrolyte solutions - (NaCl -7 g/litre, KCl -1.25 g/ litre, $CaCl_2$ - 0.5 g/litre in water)	LA: 5-10 liter/ day; PO (Through stomach tube); Repeat as necessary
Isotonic crystalloids - Normal Saline (0.9% NaCl)/ Lactated Ringer's Solution (LRS)/ 5% Dextrose saline solution (DNS)	LA:10-20L; SA: 2-4 litre; IV continuous
Colloids - Hydroxyethyl Starch (HES) and Dextrans	5-10 mL/kg (Shock); IV (short-term)

1.16 Annexure-II Reproductive hormones/ drugs

Name of Hormone/ Drug	Indications	Administration (Dose, Routes and Duration)
Hormones for the management of reproductive disorders should be used judiciously depending upon the clinical conditions and clinician judgment. Avoid overuse / misuse of the hormones		
Abbreviations - LR: Large Ruminant; SR: Small Ruminant, PO: Oral; IV: Intravenous; IM: Intramuscular; SC: Subcutaneous; TD: Total dose, * Avoid frequent use		
GnRH analogue (0.0042mg/ml)	Anovulation/ Delayed Ovulation	LR: TD 2.5 ml, IM/ IV at estrus
	True anoestrus	LR: TD 5 ml, IM/ IV
		SR: TD 1 ml, IM/ IV
	Follicular cyst	LR: TD 5.0 ml, IM/ IV
	Enhancing Conception Rate	LR: TD 2.5 ml, IM at estrus/ luteal phases (early/mid/late)
PMSG* (Pregnant Mare Serum Gonado tropin, FSH like activity)	True anoestrus	LR: TD 1500 -3000 IU, IM/ IV
		SR: TD 1000 IU, IM/ IV
hCG* (Human Chorionic Gonadotropin, LH like activity)	Repeat Breeding	LR: TD 1500-3000 IU, IM at breeding
		SR: TD 200-500 IU, IM, IM at breeding
	Cystic Ovarian Disease and Delayed Ovulation	LR: TD 1500-3000 IU, IM
	Early abortion	LR:1500-3000 IU, IM, Every week for 4 weeks
	Enhancing Conception Rate	LR: TD 1000-2000IU, IM at luteal phases (early/mid/late)

Name of Hormone/ Drug	Indications	Administration (Dose, Routes and Duration)
Hydroxy progesterone caproate (250mg/ml)	Cystic Ovarian Disease	LR: TD 500 mg, IM
	Habitual abortion (Late)	LR: TD 500 mg/ IM, for 3 days and then every week (As per the clinician observation)
	Habitual abortion (Early)	LR: TD 500 mg/ IM, after 1.5 months of pregnancy
	Post-Partum Anestrus	LR: TD 500 mg/ IM (As per the clinician observation)
Prostaglandin $F_{2\alpha}$ (Natural 5mg/ ml or Synthetic 250 mcg/ml) (Contraindications in pregnancy)	Sub-oestrus, Luteal cyst, Mummified foetus, Chronic endometritis, Pyometra, Induction of parturition	LR: TD Natural - 25mg/ Synthetic - 500mcg
		SR: LR: TD Natural – 10 mg/ Synthetic - 250mcg
Oxytocin (5 units/ml)	Uterine inertia, Dystocia, RFM, Uterine prolapse	LR:50-70 IU; SR: 20-30IU; IM/ IV

CHAPTER- 2

GUIDELINES FOR INFECTIOUS DISEASES OF LARGE RUMINANTS

2.1 Preamble

India has a vast animal population, and their diseases have significant adverse impact on the health of large ruminants leading to huge economic losses every year in the form of morbidity and mortality. The control and management of infectious diseases have been improved substantially with the advent and access of new diagnostics, vaccines, and therapeutics. Early and accurate diagnosis is the central component for effectively combating any infectious disease. In today's scenario of globalization, the diseases have no geographical barriers, hence continuous update of the knowledge of the pathogen is consequential to steer the pathway for its efficient restriction. The losses due to diseases can be drastically reduced by adopting appropriate and timely measures for their prevention, control and treatment. The Standard Veterinary Treatment Guidelines for Large Ruminants – developed based on thorough review and comprehensive discussion amongst the experts – will help the practicing veterinarians to protect the animal population from irrational therapy and hazardous consequences of excessive use of antibiotics and other drugs ensuring safety of the animal source foods (ASFs). Rational use of drugs, antibiotics, hormones, and other medicines will reduce the cost of animal health care system and make the system more effective.

2.2 Bovine Parainfluenza (BPI)

Definition and Causative Agent

This infectious viral disease of cattle, characterized by purulent nasal discharge, cough, fever, anorexia and rapid respiration, is caused by Bovine parainfluenza-3 virus (BPI-3V) belonging to genus *Respirovirus*, family Paramyxoviridae and order Mononegavirales. BPI-3V is an important respiratory pathogen in cattle associated with bovine respiratory disease complex or shipping fever. The virus is pleomorphic and enveloped in nature with herringbone-shaped helically symmetrical nucleocapsid of 150-300 nm diameter. The genome of the virus is 15 to 16 kb, consisting of negative-sense, single-stranded RNA.

Transmission

BPI-3 virus infection occurs worldwide and is usually inapparent. Following natural infection, BPI-3 virus persists for several weeks in infected animals, which may act as source for transmission of disease. Aerosol and direct contact are the major modes for the transmission of this virus, and both are accentuated in crowded and inadequately ventilated conditions. The infection is common in calves raised under intensified farming system. Additionally, this disease is commonly seen in 2- to 8-month-old calves due to the wanes of maternal immunity. This virus is also recovered from intestinal contents, milk, and aborted foetuses but the importance of these sources of contagion is unknown.

Clinical Signs

In cattle, BPI-3 virus infection is marked by febrile reaction (104°-105°F) at about day 5 followed by rhinitis and pneumonia. Generally, the infection remains sub-clinical but sometimes it manifests as lacrimation, dyspnea and harsh hacking cough and depression. Furthermore, some animals may develop broncho-interstitial pneumonia. An uncomplicated infection runs a brief clinical course of 3-4 days, followed by complete recovery.

Lesions

BPI-3 virus infection results in necrosis and inflammation in small airways in the lungs – specifically bronchiolitis and bronchitis. Usually, epithelial cells of the respiratory tract are the primary target for virus but it can also infect type II pneumocytes and alter the surfactant production via accumulation of exudate. Constant irritation may cause the reddening of nasal mucosa. Syncytium formation and both intranuclear and intracytoplasmic inclusion bodies are encountered in bronchiolar and alveolar epithelial cells. Inclusions are less common in calves that die of respiratory disease because they are absent or obscured late in the infection. Apart from that, infiltrates of lymphocytes and plasma cells accumulate in the peribronchial and perivascular interstitium.

Diagnosis

Definitive diagnosis of BPI-3 virus requires recovery of the virus from respiratory secretions and tissues. Because of the haemadsorbing nature of this virus, it can be diagnosed by isolation in cell culture followed by haemadsorption with guineapig erythrocytes. This virus can be isolated in bovine turbinate and bovine kidney cell cultures, which need to be incubated at 35°C. BPI-3 virus may

also be detected in nasal discharges or respiratory tissues by immunofluorescence staining, RT-PCR, and/or immunohistochemistry. Interpretation of results requires an assessment of the overall clinical condition in the individual animal and the herd.

Differential Diagnosis

BPI-3 virus shows the similarities in clinical signs with many other microbial pathogens. Therefore, laboratory diagnosis is an essential tool for its differentiation from bovine adenovirus, bovine coronavirus, bovine viral diarrhoea virus, infectious bovine rhinotracheitis virus, bovine respiratory syncytial virus, *Mannheimia haemolytica* and *Mycoplasma bovis.*

Treatment

Being a viral disease, there is no specific treatment for BPI-3 virus infection. However, to treat secondary bacterial infection, antibiotics (enrofloxacin @ 3-5 mg/kg BW IM/ ceftiofur @ 1.1-2.2 mg/kg BW IM) may be indicated for bacterial pneumonia. Antihistaminics (pheniramine maleate/chlorpheniramine @ 0.5 mg/kg BW IM), antitussives (diphenhydramine @ 0.5-1 mg/kg BW IM/IV), and nebulization (ceftiofur @ 1mg/kg BW) can be recommended as per the clinical condition of the sick animal. NSAIDs (meloxicam @ 0.2-0.5 mg/kg BW, flunixine meglumine @ 1.1-2.2 mg/kg BW) can restore the virus damaged host defense mechanisms.

Prevention and Control

Vaccination is the most efficacious preventive measure to control BPI-3 virus infection. Several attenuated virus vaccines for intranasal and parenteral use are available internationally that are able to induce protective mucosal IgA antibodies against BPI-3V. Typically, multivalent vaccines are formulated containing various combinations of BPI-3V, bovine herpesvirus 1 (infectious bovine rhinotracheitis virus), bovine respiratory syncytial virus, bovine viral diarrhoea virus, and *Mannheimia haemolytica*. These vaccines are usually able to control disease problems associated with BPI-3 virus infection in dairy cattle in different management situations.

Biosecurity Measures

The main aim of the biosecurity measure is to reduce pathogen exposure. Basic cleaning and hygienic procedures play pivotal role to prevent or at least reduce the infection pressure. The major biosecurity measures include good animal husbandry practices (GAHPs), strategic vaccination, strict movement control, scientific calf care, proper ventilation, minimal mixing of animals of different types, and avoidance of contact of the animals with outside animals.

Sample Collection for Diagnosis

Nasal swabs or tracheal wash fluids are the samples of choice for virus detection, and the virus can be isolated from the nasal discharges collected even during 7-9 days after infection.

2.3 Bovine Influenza

Definition and Causative Agent

Bovine influenza, an important contagious viral disease of bovine caused by influenza D virus (IDV), is characterized by nasal discharge, fever and lower respiratory pneumonia. IDV play a role in bovine respiratory disease complex (BRD), with a proven ability to cause respiratory disease. The virus belongs to genus *Deltainfluenza virus* under family Orthomyxoviridae. This virus is enveloped in nature with segmented genome of 80-120 nm diameter. The genome of the virus is 10 kb in size and consists of seven negative-sense, single-stranded RNA segments.

Transmission

Cattle are the natural reservoir of the virus. The transmission of IDV can be through – direct contact with infected animals, via aerosol at short distances, and contaminated fomites.

Clinical Signs

IDV infected animals show mild to moderate respiratory signs like spontaneous to repeated dry cough and serous/mucoid discharge. In severe disease condition, tachypnoea, dyspnea and abnormal lung sounds (wheezing) can be observed.

Lesions

The infection results in inflammation of turbinate and nasal mucosa with increased neutrophils and infiltration of lamina propria by mononuclear cells in nasal epithelium. Sometimes, superficial nasal epithelium of mucosa shows mild multifocal loss of cilia, necrosis, and erosions. Moderate inflammation of trachea can be observed with neutrophilia in

mucosa and submucosa. In severe condition, bronchitis and bronchointerstitial pneumonia can be seen.

Diagnosis

IDV can be detected in both the upper and lower respiratory tracts. RT-PCR is used for molecular diagnosis; HI and ELISA assays for serological diagnosis (detection of antibodies); and immunohistochemistry for microscopic lesion and viral presence.

Differential Diagnosis

Laboratory diagnosis is essential for its differentiation from bovine adenovirus, bovine coronavirus, bovine viral diarrhoea, infectious bovine rhinotracheitis, bovine respiratory syncytial disease, *Mannheimia haemolytica* and *Mycoplasma bovis.*

Treatment

There is no specific treatment for IDV, only symptomatic treatment with NSAIDs (meloxicam @ 0.2-0.5 mg/kg BW, flunixine meglumine @ 1.1-2.2 mg/kg BW), antibiotics (enrofloxacin @ 3-5 mg/kg BW IM; ceftiofur @ 1.1-2.2 mg/kg BW IM) and supplements (vitamin C @ 3g SC and B-complex @10 ml IM) for the control of secondary infections.

Prevention and Control

Currently there are no vaccines available for bovine influenza due to diverse lineages and strains. It can be controlled by isolation of infected animals and quarantine of the newly purchased animals.

Biosecurity Measures

The main aim of the biosecurity measure is to reduce pathogen exposure. Basic cleaning and hygienic procedures play pivotal role to prevent or at least reduce the infection pressure. The important biosecurity measures include good animal husbandry practices (GAHPs), strategic vaccination, strict animal movement control, scientific calf care, proper ventilation, minimal mixing of animals of different types, and minimization of overcrowding and avoidance of contact of the animals with outside animals.

Sample Collection for Diagnosis

Nasal swabs, bronchoalveolar, transtracheal lavage, blood and serum samples can be useful for molecular and microbiological diagnosis. Additionally, lung tissue, trachea, and nasal turbinates may be collected during postmortem examination

2.4 Foot and Mouth Disease (FMD)

Definition and Causative Agent

Foot and mouth disease is a highly contagious viral disease affecting cloven-hoofed animals, including cattle, pig, sheep, goat, and various wildlife species. It is characterized by fever and blister-like sores on the tongue, lips, mouth, teats, and between the hooves. The disease is caused by the foot and mouth disease virus (FMDV), belonging to the genus Aphthovirus under family Picornaviridae. FMDV is a non-enveloped, single-stranded positive sense RNA virus. There are seven distinct serotypes of FMDV, viz., O, A, C, Asia1, SAT1, SAT2, and SAT3 – each serotype having numerous subtypes. These serotypes are immunologically distinct, so infection or vaccination with one serotype does not confer immunity against others. Currently, serotype O, A and Asia 1 are circulating in India with serotype O being the most prevalent.

Transmission

Direct contact with infected animals or their secretions such as saliva, milk, urine, and faeces. Indirect contact via contaminated objects (fomites) like equipment, vehicles, clothing, and feed. The virus can become air-borne and spread over several kilometres under favourable conditions, particularly in cool and humid weather. Recovered animals may become carriers and serve as the source of infection for a longer period of time.

Clinical Signs

The common clinical signs in bovine include initial high fever (40°-40.6°C) which often precedes other symptoms by 1-2 days. Appearance of fluid-filled blisters (vesicles) on the tongue, gums, inner cheeks, teats and between the hooves. These vesicles rupture, leaving painful erosions. Animals also exhibit lameness due to lesions in feet and drooling of saliva due to oral lesions. Reduced appetite, lowered milk production, significant weight loss and subsequent reduction in growth are important economic consequences. Morbidity rate in FMD is close to 100 percent, whereas mortality is generally low in adults but is high in young ones due to myocarditis.

Lesions

Characteristic lesions include appearance of vesicles on nose, lips, tongue, oral mucosa, between the hooves and on teats. These vesicles rupture, leaving raw, red erosions, which can become secondarily infected. Lesions around the hooves (coronary band) may cause the hoof wall to separate. FMDV in young animals can cause myocarditis leading to sudden death.

Diagnosis

FMD can be initially suspected on the basis of clinical signs. Disease can be confirmed by RT-PCR and antigen capture ELISA. Serotyping can also be carried out by qPCR and sandwich ELISA. Virus isolation from suspected samples can be performed in primary bovine thyroid cell cultures and cell lines such as BHK-21, IBRS-2. It is difficult to identify carrier samples but oropharyngeal fluid - as the sample of choice - increases the rate of detection of the FMD virus.

Differential Diagnosis

FMD should be differentiated from injury in mouth and other vesicular diseases such as vesicular stomatitis, swine vesicular disease, vesicular exanthema of swine and bluetongue.

Treatment

There is no specific treatment for FMD. However, the external application of antiseptics contributes to the healing of wounds and guards from attacks by flies. Rational treatment with dihydrostreptomycin @ 10 mg/kg BW IM or oxytetracyline @10 mg/kg BW IV for 3-5 days is recommended to treat secondary infection. For localized treatment, it is recommended to rinse the vesicle erosions using normal saline or citric acid 1 percent or potassium permanganate 0.01 percent. After cleaning the teats and limbs, apply antiseptic ointment and bandage. Infected and in-contact animals should be isolated to prevent the spread. Soft food and *ad lib.* water should be provided.

The validated ethnoveterinary medicine practices for treatment and better health management of FMD cases are in use to reduce the irrational use of antibiotics, thus reducing the cost of treatment.

Prevention and Control

Control of the FMD depends on the epidemiological scenario of the affected country. Mass vaccination remains the most important measure of control. Presently, inactivated trivalent (FMDV O, A, Asia-1) vaccine with 6 monthly booster is administered across the nation in large ruminants under National Animal Disease Control Programme (NADCP). For effective control, systematic vaccination with the quality vaccine is of utmost significance. Routine and systematic sero-surveillance to monitor the vaccine induced immune response. Control measures are – strict restriction of the movement of animals, people, vehicles in and out of the affected areas; and cleaning and disinfection of premises, equipment, and vehicles of affected farms.

Biosecurity Measures

Immediate and proactive reporting of the outbreak to restrict further spread of infection. Limiting of farm access only to essential personnel. Use of protective clothing and disinfection protocols for visitors and farm workers. Strict regulations on import of animal and animal products to prevent entry of the new virus.

Sample Collection for Diagnosis

Samples include fluid from unruptured vesicles, vesicular swabs, and oropharyngeal fluid. Blood shall be collected to obtain serum for detection of antibodies against FMDV. Proper labelling, storage, and transportation of samples to the diagnostic laboratory is of utmost significance to ensure accurate diagnostic results.

2.5 Lumpy Skin Disease (LSD)

Definition and Causative Agent

Lumpy skin disease (LSD), an economically important and highly contagious viral disease of cattle, is characterized by fever, nodules on the skin, mucous membrane and other parts of the body. The severity of LSD is highly variable and depends on several factors, including the virus strain, the age of the host, immunological status and breed. The causative agent is the lumpy skin disease virus (LSDV) which – along with sheep pox virus (SPPV) and goat pox virus (GTPV) – is a member of the genus *Capripoxvirus* in the subfamily Chordopoxvirinae and family Poxviridae. LSDV is closely related to the SPPV and GTPV.

Transmission

LSDV primarily spreads through insect vectors

including biting flies (*Stomoxys* spp.), mosquitoes (*Aedes* spp., *Culex* spp.), and ticks (*Rhipicephalus* spp., *Amblyomma* spp.). Direct contact between infected and susceptible animals as well as contaminated feed, water, and equipment can also contribute to the transmission. The virus can survive in scabs and lesions for extended periods, increasing the risk of indirect transmission.

Clinical Signs

The incubation period for LSDV ranges from 4 to 14 days. Sudden onset of high fever (up to 105°F) which may exist for 1 week or more, decrease in feed intake, marked enlargement of superficial lymph nodes and reduction in milk yield of lactating cattle. Firm, round nodules appear on the skin, typically 2-5 cm in diameter. These nodules can cover the entire body, including the muzzle, nostrils, eyes, and genitalia. Nodules can develop on mucous membranes of the mouth, respiratory tract, and gastrointestinal tract. The ocular and nasal discharge becomes mucopurulent, and keratitis may develop. The dermal lesions include vasculitis with fibrinoid necrosis, oedema, thrombosis, lymphangitis, dermal-epidermal separation, and mixed inflammatory infiltrate. Limbs become oedematous and painful leading to the lameness. Bulls may become permanently or temporarily infertile and may shed virus in the semen.

Lesions

Lesions caused by LSDV progress through several stages. Initially multiple, firm circumscribed nodules involving dermis and epidermis are formed. These nodules may extend up to the subcutis and even muscles in occasional cases. With time, these nodules may exude serum and become necrotic. Histopathological studies reveal dermal vasculitis and epidermal vacuolar changes with intracytoplasmic inclusion bodies. In chronic cases, nodules may ulcerate and form thick scabs circumscribed by granulation tissue.

Diagnosis

Laboratory confirmation is done by virus isolation in cell cultures or embryonated eggs; detection of virus by PCR (polymerase chain reaction) and visualization of virus particles by electron microscopy; and ELISA and virus neutralization tests for detection of LSDV antibodies.

Differential Diagnosis

LSD must be primarily differentiated from Pseudo-LSD, a milder skin disease caused by bovine herpesvirus-2 (BoHV-2); bovine papular stomatitis, bluetongue, foot and mouth disease, infectious bovine rhinotracheitis, malignant catarrhal fever and mucosal disease. The skin lesions of LSD also resemble the dermatophilosis-a bacterial infection causing scabby skin lesions. Other differential conditions may include – dermatophytosis, bovine farcy, actinomycosis, actinobacillosis, urticaria, and pseudocowpox.

Treatment

There is no specific treatment for LSD. Management should focus on supportive care and preventing secondary infection using antibiotics (oxytetracyline @ 10 mg/kg BW IM or dihydrostreptomycin @ 10 mg/kg BW IM). NSAIDs (meloxicam @ 0.2-0.5 mg/kg BW IM, flunixine meglumine @ 1.1-2.2 mg/kg BW IM) may be used to reduce pain and inflammation. Cleaning of lesions with antiseptic solutions (povidone iodine, potassium permanganate/chlorhexidine), and supplementation of vitamin C @ 3g SC and B-complex @ 10 ml IM, can be recommended. Adequate nutrition and hydration are required to support recovery.

The validated ethnoveterinary medicine practices for treatment and better health management of LSD cases are in use to reduce the irrational use of antibiotics and thereby reducing the cost of treatment.

Prevention and Control

Control of LSD relies on vaccination, vector control, farm management practices, prophylactic mass vaccination of cattle against LSD using Live attenuated LSD, sheep pox and goat pox vaccines have been carried out internationally for control of LSD in cattle. Careful selection of LSD vaccine for use in prophylactic mass vaccination of cattle is most important – any vaccine as approved by the Government of India should be used.

Other preventive measures include isolation of the affected animals to prevent further spread of the virus; regular disinfection of animal sheds, equipment, and feeding areas; implementation of measures to control biting flies, mosquitoes, and ticks through insecticides, repellents; and environmental management.

Biosecurity Measures

Strict biosecurity measures are essential to prevent the introduction and spread of LSDV. Various important biosecurity measures to control LSD include GAHPs, good farm management practices (GFMPs), quarantine – and monitoring for any signs of disease – of animals before being introduced to the herd, ensure farm workers maintain and observe good personal hygiene including handwashing and changing clothing after handling animals, regular cleaning and disinfection of animal sheds/ feeding areas/equipment and vehicles that come into contact with animals, and restricted access to the farm visitors and ensuring visitors follow biosecurity protocols, *etc.*

Sample Collection for Diagnosis

Collect biopsies from active nodules or lesions. Ensure samples are placed in viral transport media for virology and in 10 percent formalin for histopathology. Collect scabs from lesions for virus isolation and PCR testing. Collect blood samples for serological testing to detect antibodies against LSDV. Collect swabs from nasal, ocular, or oral secretions for PCR testing. Samples should be collected aseptically, packed, labelled, stored appropriately, and transported to the laboratory under cold chain to maintain viral viability

2.6 Corona/Rotavirus Diarrhoea

Definition and Causative Agent

Neonatal calf diarrhoea (calf scours) is caused by rotavirus during first seven days of life with signs of yellowish, watery faeces, dehydration. Viral diarrhoea in bovines is caused by *corona/rota and other enteric viruses along with E. coli infection in young calves. Corona* viruses possess a positive-sense single-stranded RNA genome and are characterized by their crown-like appearance under electron microscopy due to spike proteins on their surface. Rotaviruses possess double-stranded segmented RNA genome.

Transmission

Coronavirus and rotavirus infections are highly contagious and can spread rapidly among animals through multiple routes. Most important route is faecal-oral route where infected animals excrete the virus in their faeces, contaminating the environment, feed, and water sources. The virus can persist in the environment, especially in faecal matter and calves can become infected through contact with contaminated bedding, feed, water, clothing and hands of farm workers.

Clinical Signs

In coronavirus infections, calves typically show signs of enteritis, including diarrhoea, dehydration, and weight loss. The diarrhoea is often watery, yellow, and may contain blood. In rotaviral diarrhoea, incubation period is 1-3 days. Clinical signs are most severe in 1- to 3- week-old calves and include watery, yellow-to-greenish diarrhoea often containing mucous and undigested milk. Rapid loss of fluids due to diarrhoea leads to dehydration and loss of body weight.

Lesions

Lesions associated with corona/rotavirus infections are primarily found in the gastrointestinal tracts. The mucosa may show congestion, haemorrhages, and necrosis. In rotavirus infection, lesions are confined to the small intestine, particularly the jejunum and ileum. Rotavirus replicates in intestinal epithelial cells near the tips of villi. Coronavirus has the affinity for the epithelial cells of the villi of the small intestine and also the surface epithelial cells of colon. Histopathologically, there is blunting and fusion of the intestinal villi, leading to malabsorption and osmotic diarrhoea. Other symptoms include loss of surface epithelial cells, cystic dilatation and accumulation of cellular debris in underlying crypts. The corona/rota viruses infect and destroy enterocytes, resulting in impaired digestive and absorptive functions.

Diagnosis

Clinical diagnosis is based on the characteristic signs of diarrhoea and dehydration in young calves. Virus detection in faeces, nasal swabs, and tissues is done by immunofluorescent staining, ELISA, and RT-PCR. Histopathological examination of intestinal tissues is done for assessing extent of villous atrophy and enterocyte damage. Virus isolation in cell culture is less commonly used method for diagnosis.

Differential Diagnosis

Differential diagnosis is essential to distinguish corona/rotavirus infections from *Cryptosporidium parvum* (a protozoan parasite causing diarrhoea in young ruminants), enterotoxigenic strains of

Escherichia coli and *Salmonella* spp. (enteritis and septicaemia with diarrhoea), and *Clostridium perfringens* type C (necrotizing enteritis with acute and often bloody diarrhoea).

Treatment

Treatment of corona/rotavirus infections is primarily supportive, as there are no specific drugs available. Fluid therapy by oral rehydration solutions (ORS) or intravenous fluids (Ringer's lactate/Normal saline/ dextrose normal saline) to correct dehydration and electrolyte imbalances. Continued feeding, to maintain energy intake, using easily digestible and nutrient-rich feeds is beneficial. Rumenotoric, probiotics, and liver tonics can be supplemented. Antibiotics (co-trimoxazole @ 15-30 mg/kg BW IM; amoxicillin-sulbactum @ 5-10 mg/kg BW IM) may be used to treat secondary bacterial infections. NSAIDs (meloxicam) @ 0.2-0.5 mg/kg BW IM may also be given. Use antidiarrheal agents (kaolin-pectate @ 1mg/kg), and nutritional support by continued feeding of milk or milk replacers in small frequent amounts to maintain energy intake.

The validated ethnoveterinary medicine practices for treatment and better health management of bovine diarrhoea cases are in use to reduce the use of antibiotics and thereby reducing the cost of treatment.

Preventions and Control

Vaccines for bovine coronavirus and rotavirus can be used in pregnant cows to boost colostral antibodies, providing passive immunity to newborn calves. Ensure that newborns receive adequate, high-quality colostrum within the first few hours of life to confer passive immunity. Regular cleaning and disinfection of calf pens, feeding equipment, and water troughs to reduce environmental contamination and to prevent the spread of the virus to healthy calves is important. Provide balanced nutrition to calves to support their immune system. Segregating infected animals to prevent the spread of the virus to healthy animals is essential.

Biosecurity Measures

Quarantine new animals for a minimum of two weeks and monitor for signs of illness. Minimize animal movement during outbreaks to prevent the spread of the virus. Ensure that farm personnel follow strict hygiene protocols, including the use of personal protective equipment (PPE) and disinfection of hands and clothing. Implement measures to control insects/pests that may act as mechanical vectors for the virus. Use effective disinfectants on equipment, clothing, and boots that come into contact with calves. Place disinfectant footbaths at the entrance of calf housing areas to reduce the risk of contamination.

Sample Collection for Diagnosis

Fresh faecal samples should be collected using clean gloves and sterile containers. Rectal swabs can be taken if faecal samples are not readily available. Samples should be stored at 4°C and transported to the laboratory within 24-48 hours to ensure the integrity of the virus for accurate diagnosis. During postmortem examination, samples from the small intestine (particularly the jejunum and ileum) should be collected and preserved in 10 percent formalin for histopathological examination or frozen for diagnosis by RT-PCR and virus isolation. Samples should be kept cool and transported to the laboratory as soon as possible.

2.7 Bovine Respiratory Syncytial Virus (BRSV)

Definition and Causative Agent

Bovine respiratory syncytial virus (BRSV) is a highly contagious viral pathogen that primarily affects cattle, particularly young calves, but can also infect sheep and goats. BRSV is a significant cause of respiratory disease in cattle worldwide and is part of the bovine respiratory disease complex, which also includes other viruses and bacteria. The causative agent is a single-stranded RNA virus belonging to the genus *Orthopneumovirus* of family Pneumoviridae. The virus is closely related to human respiratory syncytial virus (HRSV), which causes similar respiratory infection in humans, especially infants.

Transmission

BRSV is primarily transmitted via direct contact with infected animals or indirect contact through contaminated surfaces, equipment, and handlers. The virus is shed in nasal and ocular secretions, making close contact between animals a significant risk factor. Aerosol transmission is also possible, particularly in crowded or poorly ventilated environments. The virus can spread rapidly within a herd, especially in settings where animals are stressed due to factors like transportation, mixing of

animals from different sources, or abrupt changes in weather.

Clinical Signs

The clinical signs of BRSV infection vary depending on the age, immune status of the animal, and the presence of secondary infections. Common clinical signs include, fever, nasal discharge (initially serous, then mucopurulent), coughing, increased respiratory rate and effort, dyspnoea (difficulty breathing), open-mouth breathing, wheezing and crackles upon auscultation, depression and reduced feed intake. In severe cases, especially in young calves, BRSV can lead to pneumonia and acute respiratory distress, which can be fatal if not managed promptly.

Lesions

Gross lesions are observed in the respiratory system. Key findings during necropsy may include consolidation of lung lobes, especially the cranial and middle lobes, emphysema (air-filled spaces) in the lungs, interstitial pneumonia, bronchiolitis and bronchiolar epithelial necrosis, and presence of syncytial cells (multinucleated giant cells) in the lung tissue. Histopathological examination reveals inflammation, necrosis of bronchiolar and alveolar epithelium, and formation of characteristic syncytial cells.

Diagnosis

Key diagnostic approaches include detection of viral genome by RT-PCR, virus isolation in cell culture, immunofluorescence assay (IFA) and serological detection by ELISA. Histopathological examination of lung tissue for characteristic lesions and syncytial cells supports the diagnosis.

Differential Diagnosis

Infectious bovine rhinotracheitis (BHV-1), bovine parainfluenza virus type 3 (BPI-3V), bovine viral diarrhoea virus (BVDV), *Mannheimia haemolytica*, *Pasteurella multocida*, *Histophilus somni*, and *Mycoplasma bovis*.

Treatment

Treatment of BRSV is primarily supportive, as there is no specific treatment. The supportive treatment includes use of NSAIDs (meloxicam @ 0.2-0.5 mg/kg BW IM or flunixin meglumine @ 1.1-2.2 mg/kg BW IM) that can help reduce fever and inflammation. Supportive care with fluids (Ringer's lactate/Normal saline/dextrose normal saline) and electrolytes may be necessary for severely affected animals. Use of antibiotics (enrofloxacin @ 3-5 mg/kg BW IM; ceftiofur @ 1.1-2.2 mg/kg BW IM) to treat secondary bacterial infections, antihistaminics (pheniramine maleate/ chlorpheniramine @ 0.5 mg/kg BW IM) and antitussives (diphenhydramine @ 0.5-1 mg/kg BW IM/IV) can also be used. Bronchodilators (aminophylline @ 6 mg/kg BW BD IM) can be used to alleviate respiratory distress. Ensuring that affected animals are in a comfortable environment with adequate ventilation and minimal stress is crucial.

Prevention and Control

Control of BRSV involves a combination of vaccination, good management practices, and biosecurity measures. Modified live virus vaccines can reduce the severity of disease and restrict the spread of the virus within a herd.

Biosecurity Measures

Implementing strict biosecurity measures can help prevent the introduction and spread of BRSV in cattle. Variouis biosecurity measures include isolating new or sick animals to prevent transmission to healthy stock, regular cleaning and disinfection of facilities, equipment, and vehicles, implementing quarantine protocols for new arrivals or animals returning from shows or markets, reducing stress through proper handling, adequate nutrition, and minimizing abrupt changes in the environment.

Sample Collection for Diagnosis

Samples should be collected as early as possible in the early course of the disease. Use a sterile swab to collect nasal secretions from both nostrils, ideally from animals showing early signs of respiratory disease. At necropsy, collect samples of affected lung tissue for virus isolation, RT-PCR, and histopathology. Bronchoalveolar lavage fluid can be collected from live animals using a bronchoscope or catheter inserted into the lower respiratory tract. Collect blood samples to test for antibodies against BRSV, which can indicate exposure or infection.

2.8 Bovine Leukemia

Definition and Causative Agent

Bovine leukaemia, also known as bovine leukosis or

enzootic bovine leukaemia (EBL), is a contagious disease in cattle caused by the bovine leukaemia virus (BLV). It belongs to the genus *Deltaretrovirus* in family Retroviridae. Retrovirus integrates into the host's DNA and can lead to the development of lymphosarcoma, a type of cancer affecting the lymphatic system.

Transmission

Bovine leukaemia virus (BLV) primarily transmits through infected blood or body fluids. Transmission can occur during procedures like dehorning, castration, and vaccination if hygiene is inadequate. Biting insects and flies can also facilitate virus transmission between cattle. Infected cows may transmit BLV to their calves during pregnancy and calves can acquire the virus through colostrum or milk consumption. The virus can spread via contaminated needles, surgical tools, and other equipment used for injections and blood sampling, as well as through contaminated gloves during rectal examinations. Close contact among cattle, particularly in intensive farming system, further facilitates the transmission of BLV.

Clinical Signs

The disease often shows no clinical signs in many infected cattle. However, when clinical signs do appear, they can be categorized based on the progression of the disease:

In asymptomatic phase - most infected cattle do not show any obvious symptoms and can remain asymptomatic for long periods.

In persistent lymphocytosis - some cattle may develop persistent lymphocytosis, characterized by an elevated number of lymphocytes in the blood.

In lymphosarcoma - a small percentage of cattle infected with BLV may develop lymphosarcoma, presenting various clinical signs, *viz*., enlargement of superficial lymph nodes near the jaw, shoulder, or hindquarters, progressive weight loss, and a noticeable decline in milk production in dairy cows. Gastrointestinal tumors may cause diarrhoea, constipation, bloating, or abdominal distention. In respiratory system involvement, affected cattle exhibit coughing or labored breathing. Tumors in the central nervous system may lead to weakness, lameness, or paralysis. Other symptoms include fever, lethargy, and reduced appetite.

Lesions

Animals with BLV-associated lymphosarcoma often exhibit lesions in central or peripheral lymph nodes, leading to lymphadenopathy. Abomasal lesions can cause cranial abdominal pain, melena, or abomasal outflow obstruction, while extradural spinal lesions may result in pelvic limb paresis progressing to paralysis. In right atrium lesions, arrhythmias, murmurs or heart failure may occur. Uterine lesions may result in reproductive failure or abortion, and lesions of internal organs typically involve the spleen, liver, or kidneys and ureters. Liver lymphosarcoma - usually asymptomatic - can lead to jaundice and liver failure, while kidney and ureter disease may cause abdominal pain, hydroureter, hydronephrosis, and renal failure. Lymphosarcoma appears as yellow-tan nodular masses or a diffuse tissue infiltrate, with histological examination revealing densely packed, monomorphic lymphocytic cells.

Diagnosis

Diagnosis of BLV is done by clinical examination. The initial signs may include lymphadenopathy, weight loss, decreased milk production, and other symptoms related to specific organ involvement, such as abdominal pain, paralysis, or cardiac issues. In serological testing-detection of antibodies against BLV in the blood by ELISA, and by agar gel immunodiffusion (AGID). In molecular diagnosis, PCR detects BLV DNA in blood samples and is used to confirm the presence of the virus, especially when serological tests are inconclusive. Histopathologically, tissue samples showed the presence of neoplastic lymphocytes typical of lymphosarcoma. On haematology, blood smear may reveal atypical lymphocytes.

Differential Diagnosis

It should differentially be diagnosed from tuberculosis, Johne's disease, lymphadenitis, bovine viral diarrhoea, actinobacillosis, actinomycosis, brucellosis, lymphoma etc.

Treatment

There is no treatment for lymphosarcoma in cattle, although parenteral corticosteroids (prednisolone @ 1-4 mg/kg BW IM) can transiently decrease the severity of clinical signs.

Prevention and Control

There are several strategies to prevent and control

the disease internationally. ***Testing and segregation:*** Regular testing of the herd to identify infected animals and subsequently segregating or culling infected animals to prevent the spread of the virus. ***Avoidance of use of shared instruments and equipment:*** Avoid using needles, dehorners, and other equipment that could transmit the virus. ***Breeding strategies:*** Using BLV-free bulls for natural service and semen from BLV-negative donors for use in artificial insemination to reduce the risk of transmission. ***Colostrum management:*** Ensuring that newborn calves receive colostrum from BLV-negative cows to prevent early infection. ***Vector control:*** Controlling insects and other vectors that can transmit BLV between animals. ***Vaccination:*** Use vaccines, if available. However, currently, commercial vaccines are not available.

Biosecurity Measures

Biosecurity measures include implementing strict biosecurity protocols to prevent the introduction and spread of BLV. avoiding the use of shared needles, dehorners, and other equipment that could transmit the virus, regular testing of the herd to identify BLV-infected animals, segregating or culling of infected animals to prevent the spread of the virus, and controlling the insects and other vectors that can transmit BLV between animals.

Sample Collection for Diagnosis

Blood samples are the most common samples collected. However, lymph node biopsies and milk samples can also be used.

2.9 Bovine Viral Diarrhoea (BVD)

Definition and Causative Agent

It is caused by the bovine viral diarrhoea virus (BVDV) belonging to the genus *pestivirus* of family flaviviridae. The virus has two genotypes, type 1 and type 2, each classified as separate species within the genus pestivirus. Both cytopathic and non-cytopathic biotypes of BVDV type 1 and type 2 exist, but non-cytopathic strains are more commonly found in field infections. Cattle across all ages are susceptible to this infection.

Transmission

BVDV can be transmitted both congenitally and after birth. Congenital infections may lead to abortion, resorption, or stillbirth. Foetuses that survive congenital BVDV infection are born as BVDV-infected calves and will persistently carry and shed the virus throughout their lives, contaminating the farm environment. The virus can also spread via fomites, semen, biting insects, biological products, and other animals, including sheep, goat, swine, camelids, and wild ruminants.

Clinical Signs

The clinical signs in adults can vary widely. Incubation period lasts for 6 to12 days and clinical signs for 1 to 3 days. Signs in acute infection include pyrexia, reduced appetite, lethargy, discharge from the eyes and nose, diarrhoea and reduced milk yield. Chronic infection can result in symptoms resembling mucosal disease. Cerebellar hypoplasia is the most commonly observed congenital abnormality in calves. Signs include ataxia, tremors, stumbling and failure to nurse. The disease may lead to death in severe cases of infected calves. Transient infections include calf pneumonia, diarrhoea, reproductive disorders, decreased milk production, increased incidence of other illness, and death.

Lesions

Focal ulcerations are the predominant gross findings in the mucosa of the caecum, proximal colon, or rectum. In the small intestine, there is sunken appearance over the mucosa of Peyer's patches.

Diagnosis

PCR is the most sensitive method for early detection of persistently infected calves, allowing farmers to promptly remove these animals from the herd, which shed the virus extensively. Isolation of viral antigen or virus in clinical samples and tissues, and assays to detect anti-BVDV antibody in milk or serum are done for diagnosis. Serological tests include virus neutralization and ELISA.

Differential Diagnosis

BVD needs to be differentiated from malignant catarrhal fever, bluetongue and bovine herpes virus.

Treatment

Treatment of BVD remains limited primarily to supportive therapy, as there are no specific drugs available. Fluid therapy by oral rehydration solutions (ORS) or intravenous fluids (Ringer's lactate/ normal saline/dextrose normal saline) to correct dehydration and electrolyte imbalances should be

given. Antibiotics (co-trimoxazole @ 15-30 mg/kg BW IM; amoxicillin-sulbactum @ 5-10 mg/kg BW IM) may be used to treat secondary bacterial infections. NSAIDs (meloxicam) @ 0.2-0.5 mg/kg BW IM and antidiarrheal agents (kaolin-pectate @ 1mg/kg BW) may also be given.

Prevention and Control

Modified live virus (MLV) and killed virus (KV) vaccines are available. MLV vaccines typically require only a single dose for initial immunization. Vaccination in pregnant cows is contraindicated. Colostral antibodies protect calves for 3–6 months, thus requiring vaccination at 5–9 months of age.

Biosecurity Measures

Implement quarantine period or physically separating replacement cattle from the existing herd for 2 to 4 weeks. Detect and remove persistent infectors.

Sample Collection for Diagnosis

Swabs from the nostrils and conjunctiva of animals suffering from respiratory disease or from faeces or rectum, if the gastrointestinal signs present. From dead animals, lungs and spleen are collected.

2.10 Infectious Bovine Rhinotracheitis (IBR)

Definition and Causative Agent

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis (IBR/IPV), caused by bovine herpesvirus 1 (BoHV-1or BHV-1), is a disease of domestic and wild cattle. Disease is characterized by acute inflammation of the upper respiratory tract. The virus can infect the genital tract causing pustular vulvovaginitis in females and balanoposthitis in males, and also causes abortions. BoHV-1 is a member of the genus *Varicellovirus* in the subfamily Alphaherpesvirinae, which belongs to the Herpesviridae family. Bovine herpesvirus-1 subtypes: BHV-1.1 (respiratory); BHV-1.2a and 1.2b (genital); BHV-1.3 (renamed BHV-5; encephalitic). The BoHV-1.2 subtypes may be less virulent than that of subtype 1.1.

Transmission

The main sources of infection are the nasal exudate and coughed-up droplets, genital secretions, semen, and foetal fluids and tissues. Aerosol infection spreads respiratory disease, and venereal transmission spreads genital diseases.

Clinical Signs

Respiratory form – the BHV-1 virus infects the nasal cavities and upper respiratory tract, resulting in rhinitis, laryngitis, and tracheitis, severe hyperaemia of the nasal mucosa with numerous greyish necrotic foci, discharge from the eyes and nose, and increased salivation. Nasal discharges are initially watery then turn to mucopurulent, hyperaemia of the muzzle (red nose disease), and conjunctivitis in which one or both eyes may be affected, and the conjunctiva is reddened and oedematous. Drop in milk production is also observed. Abortion may occur at 4-7 months gestation, and the virus is also reported to cause mastitis. Gastroenteritis form may occur in adult cattle and is a prominent finding in the generalized disease of neonatal calves. Infectious pustular vulvovaginitis (IPV) – the vulva is swollen, and small papules, then erosions and ulcers are present on the mucosal surface. Mucosal ulcers may coalesce and sloughing of brown necrotic tissue may occur. Infectious pustular balanoposthitis (IPB) – swelling of the prepuce and mucopurulent discharge may also be seen.

Lesions

Lesions primarily occur in the upper respiratory tract and trachea, with petechial to ecchymotic haemorrhages in the nasal cavity and paranasal sinuses, and necrotic areas in the nose, pharynx, larynx, and trachea. The sinuses often contain serous or serofibrinous exudate, and as the disease progresses, the pharynx and trachea may be coated with a serofibrinous exudate and blood-tinged fluid. Pharyngeal and pulmonary lymph nodes are enlarged and haemorrhage, with tracheitis sometimes extending into the bronchi and bronchioles, leading to epithelial sloughing. In young animals with generalized BHV-1 infection, erosions and ulcers may occur in the nose, oesophagus, and fore-stomachs, with necrotic foci in the liver, kidneys, spleen, and lymph nodes, and aborted foetuses may exhibit pale, focal, necrotic lesions, particularly in the liver.

Diagnosis

IBR can be diagnosed by identifying the characteristic clinical signs and lesions, such as rhinitis, tracheitis, and conjunctivitis. For

confirmation, viral isolation from nasal swabs is done especially when samples are collected early in the disease. A rise in serum antibody titre can also support the diagnosis, although this method is not applicable in case of abortion. BHV-1 abortion diagnosis involves identifying characteristic lesions and detecting the virus in foetal tissues using PCR assay, viral isolation, immunoperoxidase, or fluorescent antibody staining.

Differential Diagnosis

IBR is to be differentially diagnosed from bovine viral diarrhoea (BVD) as both BVD and IBR produce respiratory signs in cattle and also from bovine respiratory syncytial virus, Mycoplasma bovis and other viruses such as parainfluenza 3 virus, bovine adenovirus and bovine respiratory coronavirus using specific diagnostic tests targeting each pathogen.

Treatment

Broad-spectrum long-acting antibiotics such as oxytetracyline 10 mg/kg BW IM or streptopenicillin 10 mg/kg BW IM are used to treat secondary bacterial pneumonia. Additionally, the use of NSAIDs (*e.g.*, meloxicam 0.2-0.5 mg/kg BW IM) relieves the respiratory symptoms and pyrexia. Antihistaminics (pheniramine maleate/ chlorpheniramine 0.5 mg/kg BW IM) can also be given.

Prevention and Control

Current strategies for controlling IBR/IPV involve natural exposure, biosecurity measures, vaccination, or potentially eradicating the virus from a herd or entire cattle population within a country. Natural exposure entails relying on immunity gained from recovering cattle, although this approach carries risks as not all animals may become infected and develop immunity, leading to potential abortion storms. Vaccination is recommended in high-prevalence areas where eradicating the virus is impractical due to extensive cattle populations and animal movements between regions. Effective disinfectants such as 1 percent quaternary ammonium bases, 1 percent phenolic derivatives, and 10 percent Lugol's iodine are sensitive to the virus, aiding in biosecurity efforts.

Biosecurity Measures

Biosecurity measures involve implementing strict protocols to prevent transmission. These measures include isolating infected animals, maintaining hygiene through regular cleaning and disinfection of facilities, restricting movement of animals, and implementing vaccination programs where effective vaccines are available. Additionally, monitoring and controlling vectors like insects and rodents, and educating staff on biosecurity protocols, are crucial to minimize the spread of these diseases within livestock populations.

Sample Collection for Diagnosis

Samples used for diagnosing IBR and IPV in cattle include – nasal swabs, vaginal/vulvar swabs to detect virus; blood samples to check the antibodies; and scrapings from lesions and tissue samples from affected organs to confirm the diagnosis. In outbreaks, swabs of the environment can also help to find the source of the infection.

2.11 Malignant Catarrhal Fever (MCF)

Definition and Causative Agent

Malignant catarrhal fever is an acute, infectious, usually lethal disease that impacts cattle, buffalo, deer, but also giraffes, domestic pigs, and antelopes. Malignant catarrhal fever (MCF) is caused by several viruses classified within the family Herpesviridae, subfamily Gammaherpesvirinae, genus *Macavirus*. This subgroup of Macaviruses, known as MCFV, comprises at least 10 members, with five currently identified as causing the disease.

Transmission

Inhalation is considered the main route of transmission for all MCF viruses, although ingestion may also be feasible.

Clinical Signs

The clinical signs of MCF vary widely, spanning from peracute to chronic forms. In the peracute phase, animals may exhibit no noticeable clinical signs, or they may experience depression followed by diarrhoea and dysentery shortly before death. Generally, initial signs include fever, increased serous lachrymation, and mucopurulent nasal discharge. Animals may also show reduced appetite and and milk yield. Neurological signs include incoordination, trembling, head pressing, involuntary eye movements, heightened sensitivity to touch, aggression, and seizures.

Lesions

The main lesions include inflammation and necrosis of the mucosal epithelium in the respiratory, urinary or alimentary tracts; subepithelial lymphoid infiltration; widespread vasculitis and generalized lymphoid proliferation and necrosis. Mucosal erosions and haemorrhages are common. A classic, though not pathognomonic, histologic lesion is fibrinoid necrosis of small muscular arteries.

Diagnosis

PCR test is the preferred diagnostic method, detecting all MCF viruses. Other tests include virus neutralization, ELISA, immunoblotting, immunocytochemistry or immunofluorescence.

Differential Diagnosis

It includes bovine viral diarrhoea, bluetongue, infectious bovine rhinotracheitis, epizootic haemorrhagic disease, foot and mouth disease, rinderpest, vesicular stomatitis, ingestion of some toxic plants or caustic materials.

Treatment

No treatment provides any consistent benefit. Stress reduction of subclinical or mildly affected animals using antioxidants (vitamin @ C 3g SC, vitamin E @ 1,000-4,000 IU/day, selenium @ 0.1 mg/kg DM) is indicated.

Prevention and Control

Currently, no vaccine is available. The only effective control strategy is separating persistent infectors from susceptible species.

Biosecurity Measures

Avoid contact between carriers and vulnerable species. Keep susceptible animals separated from sheep, goat, wildebeest, or other suspected reservoir hosts. Prevent access to contaminated objects, particularly for highly susceptible species. During outbreaks, promptly separate susceptible animals from the suspected source.

Sample Collection for Diagnosis

In cattle, samples for PCR include nasal swabs, scrapings from lesions and tissues from affected organs. Blood samples are collected to check the antibodies. During postmortem, tissue samples from different organs in 10 percent formalin are taken for histopathological and over ice for microbiological diagnosis.

2.12 Bovine Ephemeral Fever

Definition and Causative Agent

This acute and febrile illness affecting cattle and water buffalo is also known as Three-Day Sickness. It is caused by the bovine ephemeral fever virus (BEFV), belongs to the genus *Ephemerovirus* within the family *Rhabdoviridae*. BEFV is an RNA virus with a characteristic bullet-shaped morphology. The disease is named for its typically short duration, with most animals recovering within three days, although the condition can occasionally lead to prolonged convalescence or severe complications.

Transmission

BEFV is primarily transmitted by arthropod vectors, particularly mosquitoes and culicoides midges. The virus can also be spread mechanically by biting insects, though this is less common. There is no evidence of direct animal-to-animal transmission without vector involvement.

Clinical signs

There is sudden onset of high fever (up to 105°F). Animals may exhibit loss of appetite, stiffness, lameness, and reluctance to move due to muscle pain and joint inflammation. Excessive salivation and drooling serous nasal and ocular discharge are common. Swollen joint particularly in the limbs may be observed. In severe cases, animals may show signs of ataxia, tremors, and rarely, recumbency. Most animals recover within three days, hence the name Three-Day Sickness, but some may suffer from prolonged weakness and reduced productivity.

Lesions

The pathological lesions observed in BEF are primarily associated with inflammation and oedema. Oedema and haemorrhages in muscles and joints, leading to stiffness and lameness. Other lesions include congestion and oedema in the lungs with occasional pleuritis, enlarged and oedematous lymph nodes, mild meningoencephalitis in severe cases, and congestion and oedema in the gastrointestinal tract.

Diagnosis

The sudden onset of fever, stiffness, and lameness provides initial diagnostic clues. Laboratory confirmation requires detection of viral genome by PCR. Virus isolation is performed in cell culture but virus isolation is less commonly used for diagnosis. Detection of specific antibodies using ELISA or virus neutralization test is useful for retrospective diagnosis or surveillance. Hemagglutination Inhibition (HI) test can also be used to detect antibodies against BEFV.

Differential Diagnosis

Differential diagnosis is to be made from bovine respiratory syncytial virus (BRSV), bovine viral diarrhoea (BVD), foot-and-mouth disease (FMD), bluetongue, epizootic haemorrhagic disease (EHD).

Treatment

Treatment for BEF is primarily supportive, as there are no specific drugs for BEFV. NSAIDs like meloxicam or flunixin meglumine may be given for 2-3 days to reduce fever, pain, and inflammation. To maintain hydration and support recovery in severely affected animals, fluid therapy should be used. Ensure affected animals continue to receive adequate nutrition. Antibiotics like streptopenicilin @ 10 mg/kg BW IM; oxytetracycline @ 10 mg/kg BW IV may be used to treat secondary bacterial infections.

Prevention and Control

Control measures for BEF focus on vector management, vaccination, and good husbandry practices. Reducing vector populations through the use of insecticides, repellents, and environmental management to eliminate breeding sites. Implementing good management practices to reduce stress and improve overall herd health to help mitigate the impact of BEF. Minimize stress and isolating affected animals to prevent further spread of the disease.

Biosecurity Measures

Effective biosecurity measures are crucial to prevent the introduction and spread of BEF. Isolate new arrivals for a period to monitor for signs of disease and prevent introduction of the virus. Limit animal movement during vector season to reduce the risk of disease spread. Ensure that personnel follow strict hygiene protocols, including the use of personal protective equipment (PPE) and disinfection of hands and clothing. Eliminate standing water and other potential mosquito breeding sites around farm premises.

Sample Collection for Diagnosis

Whole blood samples in anticoagulant (heparin) are used for PCR and virus isolation while serum samples are used for serological tests. During postmortem examination, collect tissue samples from the lymph nodes, spleen, and lungs for virus isolation and in 10 percent formalin for histopathology. Nasal or ocular swabs are collected for PCR testing. Samples should be stored at 4°C and transported to the laboratory as soon as possible, ideally within 24-48 hours, to preserve the integrity of the virus for accurate diagnosis.

2.13 Bovine Papillomatosis

Definition and causative agent

Bovine papilloma, also known as bovine papillomatosis, is a viral disease of cattle characterized by the development of benign skin tumors, commonly referred to as warts. These tumors are caused by various types of bovine papilloma viruses (BPVs). BPVs belong to the Papillomaviridae family and have double-stranded DNA genome. There are multiple types of BPVs, with BPV-1 through BPV-10 are the most commonly identified in cattle. Each type has a predilection for specific tissues, leading to distinct forms of papillomatosis.

Transmission

Bovine papilloma viruses are transmitted primarily through direct contact between infected and susceptible animals. The virus can also spread indirectly through fomites such as grooming equipment, halters, and other objects that come into contact with infected skin. The virus enters the body through abrasions or micro-injuries in the skin or mucous membranes. Young cattle are more susceptible due to their developing immune systems and higher likelihood of engaging in behaviours that lead to skin abrasions.

Clinical signs

The clinical presentation of bovine papillomatosis can vary depending on the type of BPV involved and the location of the tumours. Common signs include appearance of cutaneous papillomas (warts) which is

firm, cauliflower-like growths on the skin. Common sites include the head, neck, shoulders, and teats. Lesions may vary in size from a few millimetres to several centimetres. Warts on the teats can interfere with milking and may lead to mastitis. Also occur in the oral mucosa, oesophagus, and rumen.

Lesions

The primary lesion of bovine papillomatosis is the papilloma, which is a benign epithelial tumor. Histologically, papillomas are characterized by hyperplasia of the epidermis, thickening of the outer layer of the skin and formation of finger-like projections, fibrosis and presence of inclusion bodies within the cells. In some cases, papillomas can undergo malignant transformation, particularly those caused by BPV-4, leading to squamous cell carcinoma.

Diagnosis

Diagnosis of bovine papillomatosis is primarily based on clinical examination and the characteristic appearance of the lesions. Additional diagnostic methods include microscopic examination of biopsy samples to confirm papilloma by histopathology, detection of BPV DNA in tissue samples by PCR.

Differential Diagnosis

It is important to differentiate it from other conditions that cause skin lesions in cattle, such as bovine viral diarrhoea (BVD) which produces erosive and ulcerative lesions on the mucosa; foot and mouth disease (FMD) with vesicular lesions in the mouth, feet, and teats; bovine herpesvirus (BHV-1) with ulcerative lesions on the mucosa and skin; and dermatophytosis (ringworm), a fungal infection causing circular, crusty lesions.

Treatment

Treatment of bovine papillomatosis often involves a combination of management practices and therapeutic interventions. Many papillomas regress spontaneously over time due to the animal's immune response. Cryotherapy by freezing the lesions with liquid nitrogen is recommended sometimes. Anthiomaline @ 10 -15 ml IM for 4 doses at the interval of 48 hours. Ivermectin @ 0.2 mg/kg BW SC once in 15 days. Teat dip using iodine-based preparation is recommended for herd outbreaks. Surgical removal of large or problematic warts may be required.

The validated ethnoveterinary medicine practices for treatment and better health management of bovine papilloma cases are in use to reduce the irrational use of antibiotics and thereby reducing the cost of treatment.

Prevention and Control

Control of bovine papillomatosis involves reducing the risk of viral transmission and enhancing herd immunity by quarantine of infected animals to prevent spread. Other measures include regular cleaning and disinfection of equipment and facilities, avoiding sharing of equipment, and use of the commercially available or autogenous vaccines in endemic areas. Autogenous vaccine made from the affected animal's papillomas can be used.

Biosecurity Measures

Effective biosecurity measures are essential to prevent the introduction and spread of BPV within a herd. Animal should be purchased only from reputable sources with no history of papillomatosis. Screen and quarantine new animals before introducing them to the herd. Reduce stress factors that can compromise the immune system, such as overcrowding and poor nutrition. Maintain clean and dry housing conditions.

Sample Collection for Diagnosis

Collect tissue samples from the lesion in 10 percent formalin for histopathological examination; swabs from the lesion surface for PCR testing; and blood for serological tests if necessary. Proper handling and transportation of samples to the laboratory are essential to preserve the integrity of the specimens and ensure accurate diagnostic results.

2.14 Buffalopox

Definition and Causative Agent

Buffalopox, an infectious viral disease, affects domestic buffaloes and cows. The disease manifests in cutaneous and mucosal forms, causing significant economic losses due to decreased milk production, poor growth, and secondary infections. Buffalopox is caused by the buffalopoxvirus BPXV), a member of the genus *Orthopoxvirus* within the family Poxviridae. The virus is closely related to the vaccinia virus, which causes cowpox, and it has a similar morphology and replication cycle.

Transmission

The virus is primarily transmitted through direct contact with infected animals or contaminated materials. Mechanical vectors like flies and other insects also play a role in transmission. Infected animals can shed the virus in lesions, and the virus can survive in the environment for extended periods, facilitating indirect transmission. Virus is zoonotic in nature and infected cows and buffaloes can infect human beings.

Clinical Signs

The incubation period of buffalopox is usually 3-7 days. Clinical signs vary depending on the form of the disease. In cutaneous form, initial appearance of papules which develop into vesicles, pustules, and eventually scabs. Lesions are commonly found on the udder, teats, inner thighs, perineum, and muzzle. Lesions can be painful and may lead to secondary bacterial infections. In severe cases, animals may exhibit fever, lethargy and reduced appetite. In mucosal form, lesions occur on the mucous membranes of the mouth, nostrils, and eyes. These can cause excessive salivation, nasal discharge, and conjunctivitis.

Lesions

Lesions caused by buffalopox progress through several stages. Initially, small, raised, red papules appear, followed by the development of fluid-filled blisters, which later form the pustules filled with pus. Pustules rupture and dry up, forming thick crusts. Histopathologically, lesions show ballooning degeneration of epithelial cells, eosinophilic intracytoplasmic inclusions, and infiltration of inflammatory cells.

Diagnosis

Clinical diagnosis includes characteristic lesions and history of exposure. Laboratory confirmation is performed by detection of virus genome by PCR, virus isolation in embryonated eggs or cell cultures; ELISA and other serological tests to detect antibodies.

Differential Diagnosis

Buffalopox must be differentiated from cowpox; bovine herpes mammillitis; pseudocowpox; foot and mouth disease.

Treatment

No specific treatment is advised, but palliative treatment including antibiotics (penicillin G @ 10,000-60,000 IU/kg BW IM; amoxicillin-sulbactum @ 5-10 mg/kg BW IM; enrofloxacin @ 3-5 mg/kg BW IM) to treat secondary bacterial infections, NSAIDS (meloxicam @ 0.2-0.5 mg/kg BW IM) for management of fever and pain, supportive therapy with antioxidants (vitamin C @ 3 g SC; and B-complex @10-15 ml IM) and wound dressing with potassium permanganate solution (0.01percent) may be necessary in severely affected animals.

The validated ethnoveterinary medicine practices for treatment of skin lesions due to buffalopox are in use to reduce the irrational use of antibiotics/medicines and thereby reducing the cost of treatment.

Prevention and Control

Control of buffalopox relies on a combination of vaccination, hygiene, and management practices. Vaccination with live attenuated vaccines provides protection. Isolate affected animals to prevent the spread of the virus. Regular disinfection of housing, equipment, and milking areas to reduce environmental contamination. Control of mechanical vectors like flies through the use of insecticides and proper waste management.

Biosecurity Measures

New animals should be quarantined and monitored for signs of disease before being introduced to the herd. Farm workers should practice good personal hygiene, including handwashing and changing clothing after handling animals. Regular cleaning and disinfection of equipment and vehicles that come into contact with animals. Restricting access to the farm and ensuring visitors follow biosecurity protocols.

Sample Collection for Diagnosis

Collect swabs from active lesions, particularly vesicles and pustules. Ensure the swabs are placed in viral transport media. Collect blood samples for serological testing to detect antibodies against the virus. Samples should be collected aseptically, stored appropriately, and transported to the laboratory under cold chain to maintain viral viability.

2.15 Pseudocowpox

Definition and Causative Agent

Pseudocowpox, also known as milker's nodule, is a contagious viral disease that primarily affects cattle, particularly dairy cows. It is caused by the pseudocowpox virus (PCPV), a member of the genus *Parapoxvirus* in the family Poxviridae. The virus causes characteristic ring or horseshoe-shaped scabs on the teats and udder of cows, and can also infect humans, resulting in localized skin lesions.

Transmission

Pseudocowpox virus is transmitted through direct contact with infected animals or contaminated materials. Common routes of transmission include direct contact by handling infected animals or encountering infected lesions. Contaminated milking machines, towels, and other equipment can spread the virus between cows. Farm workers can inadvertently transmit the virus through their hands, clothing, or milking practices. Flies and other insects act as mechanical vectors, spreading the virus from infected to susceptible animals. The virus can persist in the environment, particularly in scabs and lesions, which can serve as sources of infection for extended periods.

Clinical Signs

The incubation period for pseudocowpox is typically 5-7 days. Clinical signs include appearance of small, red papules on the teats and udder which progress to vesicles, pustules, and then to scabs. The scabs are typically ring- or horseshoe-shaped, which are pathognomonic for pseudocowpox. Affected cows may show signs of discomfort during milking due to the painful lesions. The open lesions can become infected with bacteria, leading to mastitis or other complications. Pain and secondary infections can result in decreased milk production. In humans, pseudocowpox manifests as a localized skin lesion, often on the hands or fingers, characterized by a raised red nodule that may ulcerate.

Lesions

Lesions caused by pseudocowpox progress through several stages starting from formation of small, red, raised areas on the skin. Fluid-filled blisters convert into pustules filled with pus. Pustules later rupture and form thick, crusty scabs, typically ring or horseshoe shaped. Histopathologically, the lesions show hyperplasia of the epidermis, ballooning degeneration of epithelial cells, and the presence of eosinophilic intracytoplasmic inclusion bodies.

Diagnosis

Clinical diagnosis is based on the characteristic ring or horseshoe-shaped lesions on the teats and udder. Laboratory confirmation is done by detection of virus by PCR and/or virus isolation in the cell cultures. ELISA and other serological tests are used to detect antibodies.

Differential Diagnosis

Pseudocowpox must be differentiated from bovine herpes mammillitis, vesicular stomatitis, foot and mouth disease , and udder impetigo.

Treatment

There is no specific treatment for pseudocowpox. Management focuses on supportive care and controlling secondary infections. Clean and disinfect lesions with antiseptic solutions. Administer antibiotics to treat secondary bacterial infections. NSAIDs like meloxicam or flunixin meglumine may be used to reduce pain and inflammation. Application of soothing topical ointments or creams to affected areas to reduce discomfort.

The validated ethnoveterinary medicine practices for treatment and better health management of pseudo-cowpox cases are in use to reduce the irrational use of antibiotics/medicines and thereby reducing the cost of treatment.

Prevention and Control

Control of pseudocowpox relies on good milking hygiene, management practices, and biosecurity measures. Various measures include ensuring proper cleaning and disinfection of milking equipment and teats before and after milking, isolating affected animals to prevent the spread of the virus, regular disinfection of housing, equipment and feeding areas to reduce environmental contamination. Implement measures to control flies and other insects through insecticides, repellents, and environmental management.

Biosecurity Measures

Implementing strict biosecurity measures is crucial to prevent the introduction and spread of

pseudocowpox. Measures include quarantining new animals and monitoring for signs of disease before being introduced to the herd. Farm workers should practice good personal hygiene, including handwashing and changing clothing after handling animals. Regular cleaning and disinfection of equipment and vehicles that come into contact with animals. Restricting access to the farm and ensuring visitors follow biosecurity protocols.

Sample Collection for Diagnosis

Collect swabs from active lesions, particularly vesicles and pustules, and place them in viral transport media or sterile PBS for virus isolation and in 10% formalin . Collect scabs from lesions for virus isolation and PCR testing. Collect blood samples for serological testing to detect antibodies against the virus. Collect biopsies from active lesions for histopathological examination. Samples should be collected aseptically, stored appropriately, and transported to the laboratory under cold chain to maintain viral viability.

2.16 Rabies

Definition and Causative Agent

Rabies is an infectious invariably fatal disease in man and other warm-blooded animals caused by rabies virus belonging to genus *Lyssavirus* of family Rhabdoviridae. Lyssavirus is an enveloped single-stranded, negative-sense RNA virus having bullet shaped structure.

Transmission

Dogs are the main source (up to 99 percent) of rabies transmission to domestic animals including cattle. The rabies virus is mainly transmitted through saliva, however, less often virus from saliva, salivary glands, or neural tissues can cause infection by entering the body through intact mucous membranes or fresh wounds. Mongoose, foxes, hyena and jackals are the major reservoirs of the virus in the wild in Indian sub-continent. In sylvatic cycle of rabies, wild animals including bats, raccoons, and foxes also serve as the maintenance host for the virus.

Clinical Signs in Cattle

The paralytic form of rabies is the main form in cattle; however, some animals also show depression and excitation. In furious form - abnormal bellowing due to laryngeal paralysis, violent behavior, hitting and biting any object, excessive salivation, hyperexcitability. In paralytic form - knuckling of hind fetlock, sagging and swaying of hind quarter, and deviation of tail.

Lesions

There are no characteristic gross lesions. The typical histological signs, found in the central nervous system, involve multifocal, mild, polio-encephalomyelitis and craniospinal ganglionitis with mononuclear perivascular infiltrates, diffuse glial proliferation, regressive changes in neuronal cells, and glial nodules. Intracytoplasmic inclusions (Negri bodies) can be seen in some but not all cases.

Diagnosis

Definitive signs and symptoms in furious form and direct fluorescent antibody (DFA) test are "Gold Standard" assays for rabies diagnosis. Other tests used for rabies diagnosis include direct rapid immunohistochemistry test (dRIT), ELISA, virus isolation using cell culture or mouse inoculation test (MIT), PCR assays (RT-PCR, real-time PCR), fluorescent antibody virus neutralization test (FAVN), and rapid fluorescent focus inhibition test (RFFIT) which is "Gold Standard" for assessing the viral neutralising antibodies.

Differential Diagnosis

Rabies should be differentially diagnosed for cattle disease with abnormal behaviour, inability to swallow, neurological abnormalities, and lameness, *e.g.*, neuronal ketosis, bovine spongiform encephalopathy (BSE), listeriosis, hypomagnesemia tetany and botulism.

Treatment

There is no specific treatment for rabies. Symptomatic treatment may be attempted to relieve the discomfort and pain.

Prevention and Control

Control of stray dog population, post-exposure vaccination (PEV) with ARV (anti-rabies vaccine) on 0, 3rd, 7th, 14th, and 28th day as per standard protocol. Providing prompt first aid for bite wounds is crucially important.

Biosecurity Measures

Immediately report any suspected rabies case to veterinary and public health authorities for

confirmation and response. Quarantine the exposed animal. Use gloves, masks, and protective clothing when handling cattle suspected of having rabies. Implement measures to prevent contact between cattle and potential rabies vectors such as bats, raccoons, mongoose, foxes, and stray dogs.

Sample Collection for Diagnosis

Antemortem samples: Saliva, serum and cerebrospinal fluid.

Postmortem samples: Fresh brain (tissue samples of cerebellum, cerebral cortex, brainstem, medulla and hippocampus) is the preferred specimen for confirmatory testing of rabies and should be submitted in glycerol-phosphate buffered saline. Additionally, cerebrospinal fluid, neck skin biopsy and serum samples may also be collected.

Public Health Risk

Rabies poses a potential public health threat associated with human exposures that result during handling of sick animals and through bites or saliva from rabid cattle, leading to fatal encephalitis, if not promptly treated.

Do's

- Wash the wound immediately with plenty of water and soap for a minimum of 15 minutes.
- Consult the physician immediately and seek advice regarding post-bite immunization.
- Complete the course of anti-rabies vaccination as per Doctor's advice following standard protocol.

Don'ts

- Do not suture or bandage the wound.
- Do not apply turmeric powder, mud, *etc.*, to the bite wound.
- Do not drink raw milk of cow bitten by rabid dog.

2.17 Hemorrhagic Septicemia (HS)

Definition and causative agent

It is a severe septicaemic disease caused by *Pasteurella multocida*, mostly affecting buffaloes and cattle. The causative agent is a Gram-negative, coccobacillary organism inhabiting as commensal in the upper respiratory tract of the animals and is shed during stress periods, leading to outbreaks in the herd. The serotypes B:2 and E:2 are mostly responsible for the outbreak of classical HS. It is an economically important bacterial disease of livestock accounting for high morbidity and mortality in Asia.

Transmission

Various stress factors are commonly associated with the transmission and outbreak of HS. High humidity temperature, malnutrition, and co-infection with other parasites or vial pathogens act as precipitating factors. Most outbreaks of HS occur in the monsoon season. Direct contact with oral /nasal secretions from the clinically infected/healthy carrier animals, as well as consumption of contaminated water and feed, may lead to the transmission of the infection to the susceptible animals.

Clinical signs

Most cases are per-acute in nature with sudden death occurring within 8-24 hours. High temperature (104-l06°F), nasal discharge, profuse salivation, and dyspnoea may be evident but may go unnoticed due to the shorter duration of the disease episode. The acute form of the disease may last longer for 3-5 days, with high, restlessness, profuse salivation, ocular discharge, mucopurulent nasal discharge, severe dyspnoea, cyanosis, and recumbency. A severe form of subcutaneous edema in the pharynx, neck, brisket and sometimes extending to the foreleg, is a characteristic finding in HS. The clinical signs are more prominent and severe in water buffaloes. Mortality in endemic regions is mostly restricted to young adults and older calves.

Lesions

Characteristic subcutaneous edema with blood-stained fluid at the neck, pharynx, and brisket are characteristic postmortem findings. Profuse petechial and ecchymotic hemorrhages on the serosal surface of internal organs and prominent hemorrhages in cervical and pharyngeal lymph nodes. Sero-sanguinous or sero-fibrinous fluid in pericardial, thoracic, and abdominal cavities. Pulmonary congestion with edema and frothy exudates in the bronchi, trachea, and nasal cavity. Haemorrhages with mucosal erosions are also encounterd in small intestine.

Diagnosis

Tentative diagnosis may be done from the clinical history of the animal. The confirmatory diagnosis requires the isolation of *P. multocida* (serotype B:2 or E:2) from the clinical samples viz., blood, tissues, or

bone marrow by *in vitro* cultivation (casein/sucrose/yeast (CSY) agar containing 5% blood as the suitable medium) and biological methods (inoculation of small amount of suspected sample subcutaneously or intramuscularly in mice). Death occurs in 24-36 hrs and pure culture of *P. multocida* can be seen in a blood smear. Biochemical, serological (Rapid slide agglutination test, Bipolar coccobacilli are also demonstrated in blood smears prepared from clinical case and stained by Giemsa/Methylene blue stain.

Differential diagnosis

HS should be differentiated from other conditions with similar clinical pictures such as acute salmonellosis, anthrax, blackleg, lightning strike, snakebite, and non-infectious toxicities.

Treatment

Animals may be treated with Sulphadimidine 33.3% (100 mg/kg BW IV) or Sulphatrimethoprim @ 30 mg/Kg BW IM/IV SID or Ceftiofur @ 1.1-2.2 mg/Kg BW SID IM for 3-5 days. Flunixin meglumine @ 1.1 – 2.2 mg/Kg BW IM SID and NSAID to be used. Animals with jowl edema may be treated with Furosemide @ 1-2 mg/Kg BW SID IM/SC. Bronchodialator such as Aminophyline @ 6 mg/Kg BW BID IM.

Prevention and Control

Three different types of inactivated vaccine formulations are commonly used for the prevention of HS, *viz*., Bacterins, Alum-Precipitated Vaccine, and Oil-Adjuvanted Vaccine. In general, the age of primary vaccination is 4-6 months and repeated yearly at least 15-20 days before the monsoon.

Biosecurity measures

Disease outbreak should be notified to the concerned authorities and the movement of the animals should be strictly controlled in the affected area to check the spread of infection. Early detection of the disease and effective surveillance are necessary for the proper control of the infection during an outbreak. Regular monitoring of the animals, proper cleaning and disinfection of the animal sheds, equipment and vehicles following all the biosecurity measures to be adopted in an outbreak of HS.

Sample collection for diagnosis

Blood and nasal swabs are collected aseptically from sick moribund animals for the diagnosis as septicemia peaks at the terminal stage of the disease. Blood samples collected from animals undergoing antimicrobial therapy are not suitable for the diagnosis. Blood sample or swab collected from the heart immediately after the death is suitable for the isolation and identification of *P. multocida*. Bone marrow from long bones is a sample of choice for isolation if the animal has been dead for a long period. All samples should be kept in any transport medium, tightly packed, and transported on ice.

2.18 Brucellosis

Definition and Causative Agent

Brucellosis is one of the most prevalent zoonoses affecting animals and humans, causing significant socio-economic and trade losses. In farm animals, it causes abortion and leads to huge economic losses. The *Brucella* is Gram-negative, aerobic, facultative intracellular, non-motile, non-spore- forming, partially acid-fast microorganisms. Currently, there are 12 species of *Brucella*, of which the four commonly occurring species with zoonotic potential include *B. melitensis, B. abortus, B. suis* and *B. canis*.

Transmission

Transmission of brucellosis in cattle occurs mainly through ingestion of contaminated food and water, infected semen and tail splashing of urine leading to aerosolization of bacteria and subsequent transmission through conjunctival route.

Clinical Signs

The reproductive system is most commonly affected in cattle leading to abortion in the third trimester along with retention of placenta. Birth of stillborn or weak calves can also occur. *Brucella* organisms localise in the supramammary lymph nodes and mammary glands, and the cattle secrete pathogens in milk throughout their life. The disease decreases average milk yield in infected animals.

Lesions

Granulomatous inflammatory lesions occur in the reproductive tract, udder, supramammary lymph nodes, and joints and synovial membranes. Other lesions include mild to severe endometritis after an abortion, thickened and oedematous placenta, and leathery intercotyledonary region with a wet appearance and focal thickening.

Diagnosis

Isolation and identification of the *Brucella:* Staining of bacterial smears, culture and identification by biochemical tests; PCR assays for species identification; Rose Bengal Plate Test (RBPT) for screening of the animals/herd; Indirect-ELISA and competitive ELISA; Standard Tube Agglutination Test (STAT); and Milk Ring Test/Abortus Bang Ring Test which is a herd screening test.

Differential Diagnosis

Trichomoniasis, vibriosis, leptospirosis, listeriosis and infectious bovine rhinotracheitis.

Treatment

Detection of positive animals and subsequent elimination from the herd is the best option because no practical treatment option is available. Treatment with doxycycline can be used for precious animals but not economical.

Prevention and Control

For immunization have calf-hood vaccination in 4-8 months old female calves with B. abortus strain19 (S19) as per the National Animal Disease Control Programme (NADCP) guidelines. Good management practices, prompt reporting of abortion cases to the nearest dispensary, cleaning of animal sheds with disinfectants like phenol, segregation of infected animals, proper disposal of aborted foetus, personal protection using gloves and masks during handling of infected animals and aborted material; education and awareness of public.

Biosecurity Measures

Proper biosecurity measures should be followed at the farms along with regular herd screening. Test- positive animals must be removed from the herd following the test and segregate policy. Proper screening of breeding bulls meant for natural service or for production – and use of – use of disease-free semen for artificial insemination to avoid sexual transmission. Important biosecurity considerations include a surveillance programme for monitoring of Brucella status in cattle herd, scientific disposal of contaminated materials including aborted foetal contents, and thorough disinfection of the infected premises till the lochial discharge ceases.

Sample Collection for Diagnosis

Aborted foetus (stomach contents, spleen, and lungs), foetal membranes, lochial discharge, colostrum, milk and fluid collected from arthritic areas/hygroma, and blood/serum sample/FTA Card for routine screening.

Public Health Risk

Humans get infected through direct contact with infected cattle, tissues, or fluids, particularly during calving, handling, or slaughter. Ingesting unpasteurized dairy products (milk, cheese, cream) or undercooked meat from infected cattle can lead to human brucellosis. Farmers, veterinarians, abattoir workers and dairy workers are at higher risk.

Do's

- Segregate the infected animal(s).
- Maintain cleanliness in animal shed.
- Drink pasteurized/boiled milk only.
- Calf-hood vaccination of cow/buffalo with nationally recommended Brucella vaccine.

Don'ts

- Do not handle infected animals and aborted material without gloves.
- Do not drink raw milk and do not eat uncooked meat.

2.19 Tuberculosis

Definition and Causative Agent

Tuberculosis (TB) is an infectious granulomatous, chronic debilitating disease of animals and humans caused by *Mycobacterium tuberculosis* complex. Zoonotic tuberculosis, a long- neglected zoonosis, is primarily caused by *Mycobacterium bovis*. It is capable of infecting a broad range of hosts, including ruminants (predominantly domestic cattle) and humans. It is an acid-fast, Gram-positive rods belonging to the family Mycobacteriaceae.

Transmission

In cattle herds, common route of infection is inhalation of infected aerosol during close contact. Ingestion of *M. bovis* directly from infected animals or indirectly from contaminated pastures, and water. Calves become infected by ingesting colostrum or milk from infected cows.

The zoonotic form is primarily transmitted indirectly to humans through the consumption of contaminated milk, dairy products, or meat containing infected material.

Clinical Signs

Infection is often subclinical and clinical signs are not specifically distinctive and include progressive emaciation, weight loss, lethargy, weakness, anorexia, diarrhoea, and a low-grade fluctuating fever, enlargement of superficial lymph nodes that can sometimes rupture and drain, and respiratory involvement with a moist intermittent cough that is worse in the morning or during cold weather and/or exercise.

Lesions

Tuberculous granuloma is yellowish in appearance and could be caseous, caseo-calcareous, or calcified in consistency enclosed in capsule of varying thickness. The lesions occur commonly in the lungs and the retropharyngeal, bronchial and mediastinal lymph nodes. Lesions can also be found in the mesenteric lymph nodes, liver, spleen, on serous membranes, and in other organs.

Diagnosis

Diagnosis is based on isolation and identification of the bacteria. Culture the sample for Brucella growth and identify by Ziehl–Neelsen staining. PCR assays are the best assays for species identification. Other methods include tuberculin skin test (delayed hypersensitivity test) - caudal fold test, the single cervical intradermal test (CIT); comparative cervical test (CCT); gamma-interferon (IFN- γ) release assays; ELISA; and molecular typing of isolates for strain identification.

Differential Diagnosis

Contagious bovine pleuropneumonia, aspiration pneumonia, Caseous lymphadenitis, traumatic pericarditis, and chronic aberrant liver fluke infestation.

Treatment

Treatment with isoniazid, ethambutol and rifampicin has limited efficacy in animals and also uneconomical. Test and segregation are the best practical approach to control the infection.

Prevention and Control

Important considerations include adopting "Test and Segregation" strategies for brucella-infected animals, disease (animal and human) reporting, farm sanitation and disinfection, open air housing rather than confinement, and avoidance of crowding. If an infected herd is found, the reactors are removed, and the herd is quarantined until all animals test negative. Isolation of the sick and weak animals showing marked symptoms. Tuberculin testing-segregation of tuberculin positive animals, abattoir surveillance.

Biosecurity Measures

Practical and implementable biosecurity measures include general/outline sanitary and hygienic measures implemented on day-to-day at the livestock farms in general, heightened biosecurity program implementation at the farm premises during the disease aimed at maintaining farm premises neat and clean, scientific disposal of disinfecting contaminated premises are very important. Proper and hygienic disposal of waste. Wildlife barriers around feed storage areas. Biosecurity measures on farms to decrease mixing of wildlife and domesticated animals.

Sample Collection for Diagnosis

Whole blood, serum, sputum, nasopharyngeal swabs, lymph node aspirates, tissue samples from lungs, liver, spleen, or during necropsy are suitable samples for any work related to TB testing.

Public Health Risk

Bovine TB in humans occurs through direct contact with infected cattle, infected aerosol inhalation or consumption of unpasteurized milk and milk products. Immunocompromised individuals are particularly at risk. Controlling bovine TB is crucial to protect human health, particularly in rural and agricultural communities. Public health measures, including pasteurization of milk, animal testing, and culling of infected cattle, are essential to mitigate this risk.

Do's

- Always use pasteurised/boiled milk
- Segregate sick animals from healthy ones
- Maintain hygiene in animal shed
- Avoid overcrowding of animals
- Visit doctor if cough persists for a longer period

Don'ts

- Do not make curd, paneer and cream from raw milk
- × Do not skip medications if diagnosed with TB

2.20 Colibacillosis

Definition and Causative Agent

Colibacillosis is the common bacterial infections causing serious economic losses to the livestock industry. The disease mostly affects less than 2-week-old calves. Virulent pathogenic serotypes of *Escherichia coli* such as enterotoxigenic *E. coli* (ETEC), enteropathogenic *E. coli* (EPEC), enterohemorrhagic *E. coli* (EHEC), and necrotoxigenic *E. coli* (NTEC) are the main agents for the clinical outcome of colibacillosis. Low birth weight and deficiency of immunoglobulins due to inadequate colostrum feeding are the predetermining factors for the disease in newborns. Sometimes the invasion of the organism occurs into the bloodstream which leads to the condition called as colisepticemia.

Transmission

The organism is shed in the nasal discharge, saliva, urine and faeces of bacteraemic calves and lambs. Transmission occurs via direct contact with the infective materials or the contaminated environment. Invasion of the organism occurs through nasal route, oro-pharyngeal route or through intestinal mucosa and sometime infection may occur through umbilicus.

Clinical Signs

Two clinical forms of the disease are enteric colibacillosis and coliform septicaemia. Enteric colibacillosis is the most common form of colibacillosis in newborn calves with diarrhoea being the major manifestation. Mucoid, pasty or fluid, foul smelling, grey or yellowish-white colour excreta containing partially digested milk is voided by the animal. Severe diarrhoea with dehydration and weight loss is seen in complicated cases. Coliform septicaemia occurs as peracute and acute form with signs of septic shock such as listlessness, inappetence, depression, collapse, loss of sucking reflex, poor response to external stimuli, recumbency, subnormal rectal temperature, cold extremities, stupor and coma. In prolonged course of bacteraemia, localization of infection results in omphalitis, polyarthritis, and meningitis. Peracute septicaemic cases characterized by sudden death without evident clinical signs are mostly encountered in lambs and kids.

Lesions

In enteric colibacillosis, carcass is dehydrated with fluid-filled flaccid intestines and abomasum containing undigested milk clots. Petechial haemorrhages in abomasal mucosa, hyperaemic intestinal mucosa, atrophy of ileal and jejunal villi and oedema of the mesenteric lymph nodes on postmortem examination. In coliform septicaemia, gross lesions are not much evident. Submucosal haemorrhages, subserosal hemorrhages, enteritis and gastritis may be present in mild infections. Fibrinous exudates in the serous cavities and joints, pneumonia, omphalophlebitis and meningitis may be present infrequently.

Diagnosis

Preliminary diagnosis is based on the history and clinical signs. Isolation and identification of *E. coli* from the intestines and the faeces confirms enteric colibacillosis. Detection of enterotoxin by ligated intestinal loop test or infant mouse test, and the histopathology of the intestine can complement the diagnosis. Enzyme immunoassay (EIA) and latex agglutination test (LAT) can also be performed for the presence of toxins. Direct FAT and ELISA can be used to detect K99+ ETEC.

Differential Diagnosis

Colibacillosis in calves should be differentiated from neonatal calf diarrhoea caused by rotavirus, coronavirus, *Cryptosporidium* spp., *Salmonella* spp., and *Eimeria* spp. as well as septicaemia caused by *Salmonella* spp., *Listeria monocytogenes*, *Mannheimia haemolytica*, *Streptococcus* spp. and *Pneumococcus* spp., and *Clostridium perfringens* type C.

Treatment

Fluids and electrolyte therapy according to the level of dehydration. Sulphatrimethoprim @ 30 mg/kg BW IM/IV BID for 3-5 days or ampicillin @ 5-10 mg/kg BW IM/IV BID for 3-5 days.

The validated ethnoveterinary medicine practices for treatment and better health management of colibacillosis cases are in use to reduce the use of antibiotics and thereby reducing the cost of treatment.

Prevention and Control

Adequate and timely feeding colostrum to neonates,

use of probiotics, improved managemental conditions at the farms, avoid exposing the umbilical cord to the environment, and vaccination of the newborn or the pregnant dam can be practiced to improve the specific immunity of neonates to infection.

Biosecurity Measures

Ensure proper hygienic measures and managemental facilities where neonates are housed. Personnel handling the neonates should take utmost care to avoid infection to the animal facility. Parturition should be facilitated in a hygienic environment and the practices like cutting of umbilical cord should be done following aseptic precautions. Foot dips should be installed in front of the calf shed.

Sample Collection for Diagnosis

In coliform septicaemia: Blood, liver, spleen, lungs, umbilicus and meninges may be collected and transported for isolation of E. coli.

In enteric colibacillosis: Piece of ileum and colon along with the contents, duodenum, jejunum and mesenteric lymph nodes may be collected aseptically.

2.21 Anthrax

Definition and Causative Agent

Anthrax is a rapidly progressing, acute septicaemic disease caused by *Bacillus anthracis.* Anthrax bacilli is a Gram positive, aerobic or facultative anaerobic, non-motile, non-haemolytic, spore- forming, rod-shaped bacteria and develops capsule inside the body of the host.

Transmission

Ingestion of contaminated fodder, water and animal products (bone meal, fertilizers); spore inhalation during wallowing in contaminated water sources; mechanical transmission by biting flies (*e.g.*, *Hippobosca* spp., *Tabanus* spp.) and the use of contaminated surgical instruments for dehorning and docking

Clinical Signs

In cattle, the disease occurs in peracute from and is characterized by sudden and rapid onset. Signs like high fever, muscle tremors and dyspnoea occur shortly before the animal dies. Exudation of tarry unclotted blood occurs from natural orifices. Rigor mortis is absent, and carcass appears bloaty.

Lesions

Upon necropsy, spleen is found enlarged having a dark semi-fluid pulp of 'blackberry jam' like consistency and poorly clotted blood is seen. Haemorrhage from the nose, mouth, vagina and/or anus may be found at death.

Diagnosis

Isolation and identification of the bacteria; capsule visualization by MacFadyen's reaction; blue rods in a background of purple/pink-stained capsule; immunofluorescence and PCR for detection of protective antigen (PA).

Differential Diagnosis

Clostridium infection, bloat, and lightning strike, acute leptospirosis, anaplasmosis, and acute poisonings by sweet clover and bracken fern.

Treatment

Animals should be treated with long-acting penicillin such as benzathine penicillin @ 22,000-44,000 IU/kg BW IM or alternatively with long-acting oxytetracycline @ 5-10 mg/kg BW IM.

Prevention and Control

Globally, anthrax Sterne strain 34-F2, a live attenuated non-capsulating vaccine is used in animals; during an outbreak, no postmortem of suspected dead animals; plug orifices of dead animals with cotton soaked in carbolic acid/Lysol; safely dispose the carcass as per the guidelines; disinfect the site of the dead animal with lysol or 3–5 percent formaldehyde and disinfect slaughter sites, processing factories and retail outlets as per the guidelines.

Biosecurity Measures

The disease should be notified to the appropriate regulatory officials when outbreak occurs; rigid enforcement of quarantine for imported animals; prompt disposal of dead animals, faeces, bedding, or other contaminated material by cremation or deep burial should be done; isolation of sick animals and removal of healthy animals from the contaminated areas, and cleaning and disinfection of sheds, pens, milking barns, and equipment used on production animals.

Sample Collection for Diagnosis

Peripheral blood and tissue samples may be collected carefully to avoid contamination of the

environment and to prevent human exposure to anthrax organisms.

Public Health Risk

In humans, anthrax manifests in three distinct patterns (cutaneous, gastrointestinal and pulmonary). More than 95 percent of human anthrax cases are of the cutaneous form and result from handling infected carcasses or the hides, hair, meat, or bones from such carcasses.

Do's

- Isolate the sick animals
- Prompt treatment of sick animals
- Proper carcass disposal
- Maintain hygiene in animal barn

Don'ts

- Do not open suspected carcass
- Do not handle animals without protective clothing including full personal protective equipment (PPE)
- Do not eat raw or improperly cooked meat

2.22 Actinomycosis

Definition and Causative Agent

Actinomycosis is primarily caused by *Actinomyces bovis (A. bovis)*, a Gram-positive, anaerobic bacterium. *A. bovis* causes lumpy jaw in cattle which is manifested as a localized, chronic, progressive, granulomatous abscess, often affecting the mandibles, maxillae, or other bony tissues in the head. Additionally, *A. bovis* also infects sheep, pig, dog, and other mammals including chronic fistulous withers and poll evil in horses. Other species of this genus including *A. israelii* and *A. naeslundii* are reportedly found in suppurative or pyogranulomatous conditions in various mammalian species including humans.

Transmission

Animal-to-animal transmission of actinomycosis is rare. The oral and gastrointestinal tracts of normal cattle harbour *A. bovis* and the bacteria are usually introduced to underlying soft tissue through penetrating wounds of the oral mucosa (*e.g.*, from wire, coarse hay, or sticks). Involvement of adjacent bone can lead to facial distortion, loose teeth, and swelling into the nasal cavity.

Clinical Signs

Lumpy jaw is manifested as localized, chronic, granulomatous abscess affecting the mandible, maxillae, or other bony tissues in the head. The primary lesions appear as a slow-growing, firm mass that is attached to, or is part of the mandible. In some cases, abscess may extend and produce sinus to the skin surface leading to drainage of purulent discharges. Examination of oral cavity may exhibit loose teeth or missing teeth and foul smell from the mouth (halitosis).

Lesions

The lesions within tissue give characteristic sulphur granule appearance due to the aggregation of bacteria and debris. Histologically, the lesions exhibit granulomatous inflammation, characterized by the presence of central necrosis surrounded by macrophages, multinucleated giant cells and a peripheral zone of fibrosis. The sulphur granules can be identified within the necrotic centres.

Diagnosis

Tentative diagnosis is usually based on clinical signs, radiography (X-rays) for evaluating lumpy jaw cases and demonstration of Gram-positive bacteria in purulent material obtained from the lesions. Culture and molecular techniques PCR/real-time PCR are recommended for definitive identification of the causative organism.

Differential Diagnosis

The disease must be differentiated from conditions that cause abscesses or granulomatous lesions, such as abscesses caused by other bacterial infections (*e.g.*, *Staphylococcus* spp., *Streptococcus* spp.), foreign body reactions, neoplasia, bacterial infections like nocardiosis and tuberculosis, fungal infections such as mycetoma caused by fungi like *Madurella* spp. or *Pythium* spp., and osteomyelitis.

Treatment

Animal may be treated with penicillin @ 22,000-44,000 IU/kg BW and sodium iodide (10 percent) @ 1g/14 kg BW IV for 5 doses at the interval of 7 days along with NSAIDs like meloxicam or flunixin meglumine.

Prevention and Control

There is no vaccine against actinomycosis in animals. Regular veterinary inspection and prompt treatment

of any injuries or wounds must be followed to treat secondary infection with actinomycetes. Ensure clean and hygienic living conditions for animals. Regularly clean and disinfect housing areas, feeding troughs, and water sources. Proper waste management should be followed to prevent buildup of organic material where actinomycetes can thrive. Practice strict biosecurity protocols to minimize transmission between animals and farms.

Biosecurity Measures

Implement strict quarantine measures for newly acquired animals to prevent introducing organisms to the herd or flock. Isolate sick animals promptly to minimize spread within the group. Maintain good hygiene within the animal housing areas.

Sample Collection for Diagnosis

Pus material, tissue samples or fluids and swabs from the affected bony tissues are the ideal sample for isolation of organism and molecular diagnosis.

2.23 Actinobacillosis

Definition and Causative Agent

Actinobacillosis is caused by *Actinobacillus lignieresii,* a Gram-negative cocco-bacilli which leads to tumorous abscesses of the tongue, usually referred to as wooden tongue. It is primarily seen in cattle but also in sheep, horses, pigs and dogs.

Transmission

Actinobacillus lignieresii is normally found in the oral cavity of healthy animals. When wounds occur in the mouth, these bacteria can infect the soft tissues, leading to localized infections. The bacteria can spread from one animal to other through infected saliva or contaminated feed. The bacteria can survive 4 to 5 days in feed.

Clinical Signs

The tongue, along with nearby soft tissue, is swollen and firm giving characteristic wooden tongue appearance. Other visible signs include inability to eat or drink, drooling of saliva, protrusion of tongue between the lips, swollen and painful soft swelling around the lower jawbones (bottle jaw), nodules and ulcers on the tongue and rapid loss of condition. In a few cases, enlargement of regional mandibular lymph nodes may occur due to spread of the infection.

Lesions

The lesions in actinobacillosis include fibrous texture of tongue leading to wooden tongue appearance and presence of nodules, ulcers or abscesses in oral cavity. Histologically, the primarily lesion is a granulomatous abscess. Chronic cases of wooden tongue may lead to the formation of fibrous adhesions between the affected tongue and adjacent structures within the oral cavity.

Diagnosis

The diagnosis of actinobacillosis can be made based on clinical signs, histopathology of affected tissue, bacterial culture and 16S rRNA based PCR. The combination of culture and molecular methods make the confirmatory diagnosis.

Differential Diagnosis

The disease must be differentiated from foreign body ingestion leading to inflammation and drooling of saliva, dental abscesses, tumors, lymphadenitis, parasitic bottle jaw, and actinomycosis.

Treatment

Animal may be treated with penicillin @ 22,000-44,000 IU/kg BW and sodium iodide (10 percent) @ 1g/14 kg BW IV for 5 doses at the interval of 7 days. Oral potassium iodide (KI) @ 5 g daily for 10 days.

Prevention and Control

There is no vaccine against this disease. A combination of good hygiene, strict biosecurity and prompt veterinary care is required for controlling the disease. The associated environment must be cleaned properly, and infected material must be disposed after decontamination to prevent spread of disease to other animals.

Biosecurity Measures

Implement strict quarantine measures for newly acquired animals to prevent introducing organisms to the herd or flock. Isolate sick animals promptly to minimize spread within the group. Maintain good hygiene within the animal housing areas.

Sample Collection for Diagnosis

Pus material, tissue sections and swabs from the affected tissues/abscess are the ideal samples for isolation of organism and molecular diagnosis.

2.24 Leptospirosis

Definition and Causative Agent

Leptospirosis, one of the most common zoonotic bacterial diseases affecting multiple species of animals, is caused by several pathogenic serovars *of Leptospira interrogans* and *Leptospira borgpetersenii.* Clinical infections in cattle are mostly associated with serovar Hardjo or Pomona. Leptospirosis has a worldwide distribution, occurs most commonly during warm and wet climates, and is endemic in tropical areas. It affects most ruminants including cattle, sheep, and goats with a broad range of clinical manifestations. Carrier state is reported in chronically infected animals which act as a reservoir of infection to other animals and human.

Transmission

The prevalence of a particular serovar in a region depends on the abundance of the maintenance host which act as the reservoir of infection. The transmission occurs through direct contact with the urine, milk, placental fluid, aborted foetus, uterine discharge, or body fluids of infected animals or the contaminated environment. It can also be transmitted through venereal or transplacental route depending on the host species and the infecting serovar. The organism gains entry to the body by penetrating the skin of the legs and feet as well as the mucosa of the eye, mouth, and nose.

Clinical Signs

In acute leptospirosis - high fever, inappetence, tachycardia, dyspnoea, depression, anaemia, haemoglobinuria, jaundice, and pale mucous membranes are observed in young ones. Rapid onset of agalactia (milk drop syndrome) occurs in adult milking animals and abortions are manifested in pregnant animals. Subacute leptospirosis is characterized by most of the clinical signs of acute infections but in milder forms. In chronic leptospirosis - abortion, premature birth, stillbirth, birth of weak offspring, and infertility may occur.

Lesions

Major lesions are seen in the kidney of affected bovines wherein petechiae and small white foci are observed on the surface of the kidneys in acute cases. The liver appears congested with yellowish discoloration. The lungs may be congested with pneumonic changes. Icterus and petechiae are seen in mucous membranes.

Diagnosis

Preliminary diagnosis depends upon clinical signs and vaccination history. Isolation of the causative agents from various organs and body fluids (liver, kidneys, lungs, brain, urine, blood, milk, peritoneal fluid, *etc.*) and subsequent identification by immunohistochemistry and molecular detection methods like PCR specific to pathogenic *Leptospira provide confirmatory diagnosis.* Serological detection methods include the microscopic agglutination test (MAT) and ELISA. MAT is the most used and the gold standard reference serological test for the diagnosis of leptospirosis. It is always recommended to combine serological tests with the detection of leptospires in organs or body fluids for the confirmation of active infection.

Differential Diagnosis

Acute leptospirosis should be differentiated from babesiosis, anaplasmosis, (post-parturient hemoglobinuria) and bacillary hemoglobinuria. Chronic leptospirosis causing abortion should be differentiated from all other common causes of abortions, *viz.*, IBR, BVD, brucellosis, mycotic abortions, *etc.* Milk drop syndrome should be differentiated from other mastitis cases and drop in milk yield due to managemental issues.

Treatment

Penicillin G @ 10,000-20,000 IU/kg BW BID IM for 3-5 days followed by dihydrostreptomycin @ 12 mg/kg BW IM for 3-5 days in large animals.

Prevention and Control

Restrict the contact of the animals with maintenance hosts for *Leptospira* like rodents. Commercial whole-cell inactivated vaccines are available for the prevention of disease in cattle. Serovar-specific immunity is noticed in leptospirosis necessitating the use of polyvalent vaccines incorporating possible serovars endemic to an area. Several mono- and poly-valent vaccines are commercially available. Regular epidemiological surveillance to investigate the incidence of different serovars in an area is required for a successful vaccination program.

Biosecurity Measures

A regular surveillance program in the endemic areas is necessary to trace and prevent the new serovars.

Ensure proper rodent control methods in the animal sheds. Movement control and quarantine of infected and in-contact animals are necessary to check the spread in the herd. Proper cleaning and disinfection of all the equipment, animal sheds, and vehicles used for the transport of animals. Farm workers handling the diseased animals should avoid the spread of infection to uninfected herds by adopting the hygienic measures.

Sample Collection for Diagnosis

Liver, kidneys, brain, lungs, adrenal glands and urine, blood, milk, CSF, peritoneal and thoracic fluid from clinically infected and dead animals may be collected aseptically for the isolation of leptospires. Paired serum samples are preferable for the serodiagnosis. In case of abortions, aborted foetuses, placental fluid, and uterine discharges may be preferred as samples for possible detection. All the samples should be refrigerated and immediately transported to the laboratory.

2.25 Aspergillosis

Definition and Causative Agent

Aspergillosis is an opportunistic fungal disease caused by the members of genus *Aspergillus*. It is a ubiquitous mold which mainly causes infection by spores. The disease mainly affects the respiratory and reproductive system of the cattle. The major species involved are *Aspergillus. fumigatus*, *A. flavus*, *A. niger*, *A. terreus* and *A. nidulans*. However, *A. fumigatus* is the most important and pathogenic causes in majority of the infections. *Aspergillus* spp. also produces many toxins such as aflatoxins, citrinins, gliotoxins, ribotoxins, ochratoxins, *etc*. Sporadic cases of aspergillosis reported in cattle, however, mycotoxicosis associated with *Aspergillus* spp. is quite common in India.

Transmission

Aspergillus can survive in the soil, decaying organic matter as well as in and around the farms producing enormous quantities of the spores. The disease is mainly acquired by inhalation of conidiospore present in the environment. Damp feeding and bedding materials may also lead to enormous production of spores. Inhalation is the main route of infection followed by ingestion. The use of contaminated intramammary medications may also be a source of infection to the cattle.

Clinical Signs

Bronchopulmonary pneumonia is accompanied by nasal discharge, fever, labored breathing, *etc*. Intramammary inoculation of the spores may lead to mastitis. The udders appear firm and there is drop in milk production. However, unlike bacterial mastitis, milk appears normal. Haematogenous spread of the infection may lead to abortion in cattle usually around 6 to 9 months of gestation. Retention of the foetal membrane is a common sequela. Rarely, aspergillus infection can cause gastritis in calves.

Lesions

Acute pneumonic lesions. Chronic form may result in formation of granulomas in the lungs. Abortion may result in necrotizing placentitis. The inter-cotyledonary area of the foetal membrane and the inter-caruncular area of the uterus may appear thick and leathery. The cotyledons may be thick and yellow to greyish. The aborted foetus may show circumscribed ring worm-like lesions in the skin.

Diagnosis

Pneumonia may be detected based on the postmortem findings. The organism can be visualised in the histopathology of the lung tissue. Fungus may be isolated from the suspected samples using Sabouraud's dextrose agar (SDA) media. Milk samples from the suspected animals may be useful in identifying mastitis cases. The lesions in the aborted foetus and placenta may help categorize the abortion cases. The foetal abomasal contents may also be cultured for the diagnosis.

Differential Diagnosis

Pneumonia and mastitis caused by the *Aspergillus* should be differentiated from other similar mycotic infections. Abortion cases should be differentiated from the bacterial abortion mainly caused by *Brucella* and *Leptospira*.

Treatment

Treatment of systemic mycoses largely by amphotericin and nystatin but the newer compounds such as enilconazole, fluconazole, itraconazole, ketaconazole administered orally appear to be highly effective.

Prevention and Control

Reducing the burden of fungal spores in the farm and surrounding is the key to prevent the aspergillosis in

bovines. The storage conditions of feed and fodder must be improved. Regular removal of the dung from the shed premises may be helpful in minimizing the spore production and dissemination. Proper hygiene and ventilation in and around the shed.

Biosecurity Measures

Proper disposal of the infective material like aborted foetus, foetal membranes, contaminated feed and bedding materials by autoclaving followed by incineration.

Sample Collection for Diagnosis

Lung tissue, sterile milk samples and foetal abomasal contents are the ideal samples for the diagnosis. Samples should be collected at the time of postmortem without any delay.

2.26 Dermatophytosis

Definition and Causative Agent

Dermatophytosis, a superficial mycotic disease, affects the host's keratinized tissue. The disease is caused by a group of keratinophilic molds called dermatophytes which can thrive on the keratinous structures by producing an array of protease enzymes. This keratinolytic fungi group includes *Microsporum*, *Trichophyton*, *Nannizzia*, *Arthroderma*, *Epidermophyton*, *Lophophyton* and *Paraphyton*. The most common cause of dermatophytosis in cattle and goats is *Trichophyton verrucosum*, a zoophilic dermatophyte. Dermatophytes secrete endoproteases, exoproteases, and sulphite for the breakdown of keratin to amino acids.

Transmission

The hyphal structures of dermatophytes breakdown to form smaller spores called arthrospores, which is the major infective propagules. The spores once enter to the host body surface will germinate in the stratum corneum.

Clinical Signs

The disease is more common in calves than the adult animals. The affected calves exhibit non-pruritic lesions.

Lesions

Loss of hair in patches and crust formation in the neck and face are the most commonly observed lesions. Sometimes suppurative lesions can also be observed. In cows, lesions may be observed in the limbs and chest region.

Diagnosis

Direct microscopic examination of samples after treatment with 10 percent KOH reveals the arthrospores in the clinical samples. The isolation of dermatophytes from the samples can be attempted by the inoculation in Sabouraud's dextrose agar supplemented with chloramphenicol and cycloheximide. The dermatophyte isolates can be characterized by microscopic examination, colony morphology, and molecular techniques such as PCR. Production of the chain of chlamydospores in the Corn Meal Agar is characteristic of *T. verrucosum*.

Differential Diagnosis

Ringworm should be differentiated from other common skin ailments such as bacterial infections, insect bites, interdigital dermatitis, *etc.*

Treatment

Crusty lesion can be topically treated with Whitfield's ointment or with 4 percent lime sulphur. Povidone iodine (1 percent) or 0.5 percent sodium hypochlorite have also shown similar success rate. Systemically animals may be treated with 10 percent sodium iodide @ 1g/14 kg. BW IV.

Prevention and control

A live vaccine containing freeze-dried fungal structures of *T. verrucosum* is used in some countries as a prophylactic measure against *T. verrucosum* infections. However, vaccination is not commonly practiced in India.

Biosecurity Measures

The disease is highly contagious and zoonotic. The infected animal should be isolated and treated till clinical and mycological cure. Contaminated bedding material should be discarded. The farm equipment and other types of machinery should be properly disinfected with sodium hypochlorite.

Sample Collection for Diagnosis

Skin scrapings from the periphery of the lesion are mainly collected for the isolation of dermatophytes. Infected hairs along with follicles can also be plucked using sterile forceps.

2.27 Black leg (Black Quarter)

Definition and Causative Agent

Black quarter (BQ) – an acute infectious fatal disease of cattle and occasionally of other species, such as sheep, goat, swine, mink and rarely horses – is caused by *Clostridium chauvoei* and characterized by toxaemia, inflammation and necrosis of skeletal and cardiac muscles with high mortality. *Cl. Chauvoei* is a Gram positive, motile, sporulating rod, with subterminal spores, saccharolytic produces four toxins, *viz*., alpha toxin (necrotizing and hemolytic); beta toxin (DNase); gamma toxin (hyaluronidase), and delta toxin (hemolysis). *Cl. chauvoei* is also (along with other clostridia) a common secondary invader of traumatic or surgical wounds resulting in gas gangrene or clostridial sepsis.

Transmission

Black leg is a soil-borne infection, and the portal of entry is through the alimentary mucosa after ingestion of contaminated feed/fodder. It is postulated that disturbing the soil in some way exposes the bacteria or causes them to become pathogenic. In sheep, the disease is invariably a wound infection. The skin wound during shearing, docking, fighting and of the navel at birth infected with the organisms may cause the development of disease. The infections of the genitalia of the ewe at lambing may also cause serious outbreaks. Spores ingested multiply in intestine and cross the intestine mucosa which are then taken away by macrophages to general circulation after which they are deposited in various organs and tissues including skeletal muscles. Spores remain dormant until damage to the muscle. Anaerobic environment is required for germination and proliferation of spores. Organisms when multiply release the toxins locally leading to myonecrosis and systemic spread of toxins via the bloodstream causing death.

Clinical Signs

Disease is acute, usually fatal and affected animals are often found dead before appearance of the clinical signs. In some cases, there may be lameness or visible crepitating swelling of muscle groups. Any striated muscles may be affected including the tongue, diaphragm and myocardium, but the shoulder and pectoral muscles are most often involved. The disease is most common in young animals (6 months to 2 years of age) on a good plane of nutrition. The infected animals show anorexia, ruminal stasis and high temperature (up to 106°F).

Lesions

Crepitating swelling in the musculature, affected muscles on incision at necropsy are dark brown or dark red, streaked with black, central part is dry and surrounding is moist and on pressing yield dark gas filled exudates. A peculiar sweetish odour (rancid butter) may be noticed. Subcutaneous tissue overlying affected muscles are usually yellowish, gelatinous, blood tinged and contain gas bubbles. Similarly, lesions are often found in heart but rarely in tongue. Microscopically, there are irregular areas of necrosis and collection of neutrophils and lymphocytes, with presence of Gram-positive bacilli appearing singly or in small irregular clumps.

Diagnosis

Gross lesions, demonstration of organism with subterminal spores, spores are in greater diameters than bacilli.

Differential Diagnosis

The differential diagnosis is to be made from other acute clostridial infections, lightning strike and with anthrax. Differentiation from *Cl. septicum* by cultural characteristics and immunofluorescence.

Treatment

Penicillin G @dose of 22,000-44,000 IU / kg BW IM may be administered for 4-5 days.

Prevention and Control

In enzootic area, annual vaccination of all cattle between 6 months and 2 years of age may be carried out during spring and summer. The ewes may be vaccinated 3 weeks before lambing to protect lambs against umbilical infections at birth and subsequent docking. The animal dying of black leg may be disposed off by burning or deep burial to check the soil contamination.

Biosecurity Measures

Proper cleaning and disinfection of all the equipment and animal sheds. Parturition should be facilitated in a hygienic environment and the practices like cutting of umbilical cord should be done following aseptic precautions. Proper disposal of carcasses is required to prevent the contamination of pasture and also to check the access of decomposed carcass

to the grazing and free-range animals.

Sample Collection for Diagnosis

The biopsy samples from affected muscles and heart blood may be collected for isolation and characterization of the organisms. During postmortem, samples from affected muscles and heart blood may be collected for microbiological and histopathological diagnosis of the disease.

2.28 Bacillary Hemoglobinuria

Definition and Causative Agent

This is an acute highly fatal toxaemia of cattle and sheep, caused by *Cl. haemolyticum*, which is very similar in some respect to *Cl. novyi* type B. Characterized by high fever, haemoglobinuria and jaundice with presence of necrotic infarcts in the liver. Bovine bacillary haemoglobinuria is also known as red water disease and infectious ictero-haemoglobinuria.

Transmission

The infection is associated with irrigated or poorly drained pasture contaminated with *Cl. hemolyticum* along with snails harbouring the intermediate stage of liver fluke. Heavy mortality is reported when cattle from the uninfected area are brought into the infected farm. Animals in good body conditions are more susceptible. The disease spreads from infected to non-infected area by flooding, natural drainage, and contaminated fodder from infected area by carrier animals. The disease mostly occurs in cattle and occasionally in sheep and rarely in pigs. Organisms in the soil are ingested which then multiply in the intestinal tract and enter the systemic circulation. The organisms localize in the liver and remain latent until anaerobic condition develops (migration of liver fluke cause hepatic damage) which initiate activation of latent organisms releasing exotoxins which further contribute to hepatic damage and produce characteristic infarcts, haemolysis and death. The principal toxin is beta toxin (identical to that of *Cl. novyi*) – a phospholipase C – that hydrolyzes lecithin and sphingomyelin and hemolyzes red blood cells.

Clinical Signs

The illness is of short duration and cattle may be found dead without showing any signs of disease. The animals show anorexia with abdominal pain, high fever (103°–106°F), arched back, and disinclination to move. Disease is characterized by sudden onset of haemoglobinuria, fever, anaemia, leukocytosis, collapse, and death within a day or two. Oedema of the brisket is commonly seen in most of the cases. Animals exhibit diarhoea with mucus and blood-tinged faeces and hence the faeces are dark brown in colour. The urine is dark red with sign of jaundice.

Lesions

Infected animals show generalized subcutaneous haemorrhages including haemorrhagic abomasitis and enteritis with presence of blood mixed contents. The typical solitary lesion is a large area of necrosis – resembling an infarct in the liver – in contrast to black disease where multiple areas of hepatic necrosis can occur. Microscopically, the necrotic tissue contains the Gram-positive bacilli, surrounded by polymorphonuclear infiltrate. The kidneys are mottled as a result of haemoglobinuria and bladder contains red urine.

Diagnosis

Cl. hemolyticum can be isolated from heart blood, liver and other organs from a fresh carcass. The organisms can be demonstrated in liver impression smears by immunofluorescence technique. Gross and microscopic lesions along with isolation and characterization of the organism confirm the diagnosis.

Differential Diagnosis

The disease is to be differentiated from other diseases showing haemoglobinuria, myoglobinuria and haematuria like post-parturient haemoglobinuria, babesiosis, anaplasmosis, leptospirosis, enzootic haematuria, chronic copper poisoning, anthrax, black leg and infectious necrotic hepatitis.

Treatment

The cases may be treated with penicillin, tetracycline with supportive fluid and electrolyte therapy. Convalescent animals should be protected from nutritional and climatic stress until they are fully recovered. Mineral supplement containing iron, copper and cobalt may be provided to the recovered animals. Haemostatic like ethamsylate or tranexamic acid may be used.

Penicillin G @22,000-44,000 IU/kg BW IM may be administered for 4-5 days.

Prevention and Control

In enzootic area, vaccination is to be carried out over 6 months of age. Proper sanitation and hygienic measures are to be adopted at the farm. Proper disposal of the carcasses dying of the disease is essentially required.

Biosecurity Measures

Biosecurity measures include proper cleaning and disinfection of all the equipment and animal sheds, proper disposal of carcasses to prevent the contamination of the pasture, check the access of decomposed carcass to the grazing and free-range animals, and control of parasitic load by timely anthelmintic treatment.

Sample Collection for Diagnosis

The biopsy samples from affected liver and heart blood may be collected for isolation and characterization of the organisms. During postmortem, samples from affected liver and heart blood may be collected for microbiological diagnosis of the disease. Further, the morbid samples in 10 percent formalin are required for histopathological diagnosis of the disease.

2.29 Malignant Oedema (Gas gangrene)

Definition and Causative Agent

Malignant oedema is an acute wound infection caused by *Clostridium* organisms with acute inflammation at the site of infection and severe systemic toxaemia. *Cl. septicum*, *Cl. chauvoei*, *Cl. perfringens*, *Cl. sordellii* and *Cl. novyi* are involved in the causation of malignant oedema. The organisms are found in soil and in the gastrointestinal tract of animals. Malignant oedema is seen as a sequel to wounds, such as those occurred in shearing or docking or to parturition. It is most common in horses, sheep and cattle, and rare in dogs and cats.

Transmission

Mostly a wound is the main portal of entry of the *Clostridium* organisms. Deep puncture wounds accompanied by severe trauma result into anaerobic condition for the growth of organisms. Infection may also occur through surgical or accidental wounds following vaccination, venipuncture or through umbilical cord of the newborn.

Clinical Signs

Clinical signs appear mostly within 12-48 hours of infection. At the site of infection, there is a soft doughy swelling which is hot with marked erythema and pain on palpation. The swollen areas later become even more oedematous, tense but less painful and cooler. Animals show high fever (106°F), depression, weakness, muscle tremors and lameness. Post-parturient infection reveals swelling of the vulva with reddish brown discharge. In fighting injury, rams showed local oedema in the head region.

Lesions

It is mainly a form of gas gangrene although in malignant oedema, the gas production is minimum. Haemolytic and necrotizing alpha toxin is responsible for the lesions. Grossly, tissues are oedematous and often haemorrhagic and contain some gas bubbles. Septicaemia often occurs, with widespread haemorrhages throughout the body. The lungs are congested and oedematous. Serous blood-tinged effusions in peritoneum are also observed. *Cl. septicum* is readily demonstrable in the affected tissues. A foul, putrid odour is generally present in infection with *Cl. perfringens* and *Cl. sordellii*. Microscopically, there is extensive necrosis of muscle with extensive oedema and cellulitis. The lesion is the result of potent toxins, released from the invading bacteria which may enter the circulation and lead to haemolytic anaemia and necrotizing lesions at other sites.

Diagnosis

Clinical diagnosis is made based on toxaemia, local inflammation and emphysema. Laboratory diagnosis is made on isolation and characterization of *Clostridial* organisms.

Differential Diagnosis

The differential diagnosis is to be made from black leg and anthrax.

Treatment

Penicillin G @ 22,000-44,000 IU/kg BW IM may be administered for 4-5 days. Supportive therapy includes NSAIDs and antihistamines.

Prevention and Control

Sanitary and hygienic measures are required at lambing, shearing, castration and docking to control the infection in sheep. In enzootic area, the vaccination with specific or combined vaccine is required for prevention and control.

Biosecurity Measures

Biosecurity measures include proper cleaning and disinfection of all the equipment and animal sheds, facilitate parturition in a hygienic environment, follow aseptic procedures while cutting the umbilical cord, and proper disposal of carcasses to prevent the contamination of pasture.

Sample Collection for Diagnosis

The swab or tissue samples from local lesions is required for microbiological investigation. Further, the morbid samples in 10 percent formalin are required for histopathological diagnosis of the disease.

2.30 Tetanus

Definition and Causative Agent

Tetanus is a fatal infectious disease of all species of domestic animals caused by the exotoxins of *Cl. tetani*. Tetanus or lock jaw occurs in humans and animals. The causative agent – *Cl. tetani* – is a normal inhabitant of the intestinal tract of herbivorous animals and is found in humas rich soil. *Cl. tetani* is Gram-positive, sporulating, anaerobic, rod-shaped bacillus. Tetanus is usually sequel of wounds, such as nail pricks, castration, docking, shearing or those received during parturition, and accidental injuries on roads as well as injuries due to grazing of rough fibrous feeds.

Transmission

The *Cl. tetani* organisms enter through deep puncture wounds but the spores may remain dormant in the tissues for sometimes till they get the anaerobic environment in the tissues. In some cases, the toxin is produced in the gut or ingested preformed in the feed. The grazing of rough fibrous feeds before the outbreaks is a common finding and suggests that the entry of infection may occur via wounds in the mouth.

Clinical Signs

The incubation period varies between 1-3 weeks with rare cases occurring even after 7 months of the infection. In sheep and lambs, the clinical signs appear in 3-10 days of shearing or docking. The clinical picture is similar in all the species. In general, increased muscle stiffness is observed first and is accompanied by muscle tremors. There are restricted jaw movements, prolapse of third eye lid, stiffness of the hind limbs causing straddling gate with stiff tail. The animal looks anxious and alert with erect ears and dilation of nostrils and exaggerated response through normal stimuli, drooling saliva, constipation with retention of urine. As the disease progress the muscular tetani increases and the animal shows the sawhorse posture. Animals show inability to walk and inclined to fall. The entire musculature is eventually involved, and death follows. The duration of foetal illness in horse and cattle is usually 5 to 10 days but sheep usually die within 3 to 4 days.

Lesions

There are no appreciable gross or microscopic lesions which can help in the diagnosis of the disease. However, search should be made for the site of infection for demonstration and isolation of the organisms in the laboratory.

Diagnosis

Based on clinical sings and a history of trauma. However, a wound is not often demonstrable and also the bacilli are difficult to demonstrate. The clinical case of tetanus is so distinctive and rarely confused with other diseases.

Differential Diagnosis

In the early stages, tetanus may be confused with other diseases like strychnine poisoning which is uncommon in farm animals. Hypocalcaemic tetani (Eclampsia) of mares also simulate the tetanus but it is seen in lactating mares and respond to calcium therapy. The lactation tetani of cattle accompanied by tetani and convulsions which are more severe than those seen in tetanus. Enterotoxaemia of lamb is accompanied by severe nervous signs.

Treatment

Parenteral administration of large doses of penicillin eliminates the organism. The tetanus antitoxins are also administered in high doses of 300,000 units twelve hourly for 3 days in horses. In large ruminants (Cattle, Buffalo), the dosage is 10,000-50,000 units which is given twelve hourly for 3 days.

Local administration of antitoxin around the wound is also effective. Combination of chlorpromazine or diazepam may be administered to reduce the hyperesthesia. Urinary catheterization is done to relieve the urine. Supportive therapy includes isotonic balanced electrolyte solution.

Prevention and Control

Tetanus can be controlled to a great extent by proper disinfection of instrument used for castration, docking and shearing. Any surgical intervention on the animals may be carried out in clean surroundings and proper use of disinfectant. As prophylaxis, antitoxin @1,500–3.000 IU may be injected subcutaneously in horses for passive immunity. In enzootic area, all susceptible animals should be given anti-tetanus toxoid for active immunization. The tetanus can be controlled in neo-natal lambs by proper vaccination of the ewe in the late pregnancy.

Biosecurity Measures

Biosecurity measures include proper cleaning and disinfection of all the equipment and animal sheds, proper disposal of carcasses to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Sample Collection for Diagnosis

The swab from the wound may be collected for isolation and characterization of Cl. tetani. The serum sample may be collected for toxin-antitoxin neutralization test.

2.31 Botulism

Definition and Causative Agent

Botulism is a fatal and motor nerve paralysis caused by the ingestion of preformed toxin of *Cl. botulinum*. The organisms proliferate in dead decomposing animals and plant materials. The disease also occurs due to infection of wound with the organisms and the formation of potent exotoxins. *Cl. botulinum* is responsible for an extremely serious food intoxication-botulism. It is divided into seven different toxigenic strains based on antigenically distinguishable toxins produced by the organisms.

Type	Principal victim	Source/vehicle
A	Human and mink	Canned vegetable, fruits, meat and fish
B	Human, horses, cattle, sheep	Meat, usually pork, silage and forage
C	Cattle, sheep, horses, dogs, mink, birds	Fly larvae, rotting vegetation, silage, carrion (forage poisoning, limber neck)
D	Cattle, horses, birds	Carrion (Lamsiekte)
E	Humans, mink, fish	Fish and marine animal foods
F	Humans	Liver paste
G	Humans	Soil

Transmission

Transmission is generally by ingestion of the feed and fodder contaminated with preformed botulinum toxins. The spores of *Cl. botulinum* are very resistant and survive for long period in the environment. The lethal toxins also remain active for long time in the bones of the dead animals. The organisms are common inhabitant of the alimentary tract of herbivores. It is also found in the soil. The soil and water contamination occurs from faeces and decomposed carcasses. The source of infection for animals is almost always dead decaying carcasses. The cattle with phosphorus deficiency chew the carcasses and suffer from botulism. The spoiled silage and hay also facilitate the growth of botulinum organism and may be the source of botulism in the animals.

Clinical Signs

The disease's incubation period varies with the amount of ingested toxin, with individual susceptibility, and may last from days to weeks and characterized by a descending and symmetrical paralysis. After absorption into blood stream, the toxin enters the peripheral nerves at their synaptic junctions, binds to the nerve membrane, and prevents release of acetylcholine by synaptic vesicles. Only peripheral cholinergic nerves are affected, as the toxin does not enter the CNS. Cranial nerves are first affected, but ultimately all muscles of the body may be affected. Almost all animal species, including birds and fishes

are susceptible. Progressive muscular paralysis affects the hind limb muscles and muscles of the jaw and throat. The muscle paralysis occurs in the hind quarter and progresses to the fore quarters, head and the neck, marked by muscle tremors. Animal showed restlessness incoordination, stumbling and ataxia followed by inability to rise and lift the head. In some cases, the tongue becomes paralyzed and hangs out from the mouth.

In cattle, mostly the botulism results from ingestion of animal carcasses and their contaminated food stuffs. Many small animals and birds carry *Cl. botulinum* type D organisms as part of their normal intestinal flora, and after death, the carcass can become extremely toxic. The phosphorus deficient cattle chew the bones and bites the decaying meat.

Forage poisoning is a form of botulism in cattle, which results from the ingestion of silage or hay that has been contaminated with a dead animal. *Cl. botulinum* type C is most often implicated in this disease. Botulism in sheep is comparable to that in cattle and is associated with phosphorus deficiency and bone chewing. In horses, botulism is seen sporadically world-wide. It is usually caused by ingestion of hay contaminated with dead animals. Wound botulism resulting from *Cl. botulinum* type B toxin is reported in horses and foals. Swine are relatively resistant to botulism because the toxin is poorly absorbed. Botulism is seen in most species of birds except vultures, which are resistant. A peculiar form of torticollis - called limber neck - is seen that mostly results from *Cl. botulinum* type C toxin, but *Cl. botulinum* type D and E toxicoses have also been reported. There are no specific histopathological lesions in the dead animals.

Lesions

There are no specific gross or microscopic lesions. Presence of the suspicious foreign materials in stomach may be suggestive. Nonspecific sub-endocardial and sub-epicardial haemorrhages and congestion of intestinal mucosa and serosa may be present. Microscopically, perivascular haemorrhages may be seen in the cerebellum and cerebrum with destruction of Purkinje cells.

Diagnosis

Presumptive diagnosis can be made on history and clinical signs. Confirmatory diagnosis can be made by demonstration of toxin in the stomach and intestinal content of the animal and also in the suspected feed/fodder/carcass samples.

Differential Diagnosis

Differential diagnosis is to be made from parturient paresis in cattle and hypocalcaemia in sheep. Paralytic rabies in cattle, encephalomyelitis in horses, and louping ill and scrapie in sheep require differential diagnosis.

Treatment

Specific or polyvalent antitoxin serum may be useful in early stage of the disease. Purgatives such as magnesium sulphate/liquid paraffin are useful for removal of the toxin from the alimentary tract.

Prevention and Control

Mineral (Ca, P) and protein supplement need to be added to overcome the dietary deficiencies in the animals. Proper disposal of carcasses is necessary to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Biosecurity Measures

Follow standard biosecurity measures. Dispose of the dead carcass safely following all biosecurity norms recommended for disposal of carcasses of infected animals.

Sample Collection for Diagnosis

Samples from gastrointestinal tract of dead animals and from decomposed carcasses may be collected for demonstration of botulinum toxin, and also to carry out the toxin-antitoxin neutralization test.

2.32 Bovine Genital Campylobacteriosis

Definition and Causative Agent

Bovine genital campylobacteriosis (BGC), also known as bovine venereal campylobacteriosis, is a sexually transmitted disease of cattle characterized by early embryonic death, infertility, and abortions. Although *Campylobacter fetus* subsp. *venerealis* is the major etiological agent, *Campylobacter fetus* subsp. *fetus* is also sporadically reported from infertility and abortion cases in ruminants.

Transmission

C. fetus is transmitted through sexual contact as

well as via contaminated semen or instruments at the time of artificial insemination. Infected bulls may become carrier and amplify the disease spread. The primary factor associated with this variability is attributed to age-related depth of the preputial and penile epithelial crypts. In young bulls (<3-4 year old), the crypts are less developed and, hence, the clearance is faster whereas in older bulls (>3-4 year old), the deeper crypts provide the proper microaerophilic environment required for chronic infections to establish. Infected cows may carry infection for ≥2 years.

Clinical Signs

Cows infected with bovine genital campylobacteriosis are apparently healthy but may exhibit varying degrees of mucopurulent endometritis. Other symptoms include early embryonic death, extended luteal phases, irregular estrous cycles, and repeat breeding resulting in prolonged calving intervals. Abortions are relatively uncommon. In herds that are not managed intensively, the disease may become evident only through pregnancy examinations that reveal low or borderline pregnancy rates and significant variations in gestation lengths, particularly if the disease has occurred recently. In subsequent years, infertility is typically limited to replacement heifers and a few susceptible cows. Bulls remain asymptomatic and produce normal semen.

Lesions

The lesions of bovine genital campylobacteriosis include mucopurulent endometritis, erosion and ulceration of uterine wall, presence of fibrinous deposits and thickening of the uterine mucosa. The cervix may show signs of inflammation or fibrosis. The presence of mucopurulent discharge from the vagina is indicative of underlying endometrial inflammation.

Diagnosis

Culturing and identification of the organism is the only suitable method for declaring the animal negative for campylobacteriosis. Monoclonal antibody (MAb)-based capture ELISA can specifically detect *C. fetus* subsp. from incubated Clarke's transport enrichment medium. Only positive ELISA results samples are to be cultured for confirmation of the specific subspecies using standard isolation and identification methods. MALDI-TOF mass spectrometry can be used to identify *C. fetus* at species level but is not able to differentiate between subsp. *venerealis* and *fetus*. Bacterial culture followed by PCR is the recommended method for diagnosis of *C. fetus* up to subspecies level. Checking for presence of IgA Abs in vaginal mucous can help in detecting the disease prevalence in cattle herd.

Differential Diagnosis

Bovine genital campylobacteriosis must be differentiated from bovine viral diarrhoea, leptospirosis, brucellosis, neosporosis, chlamydiosis, mycoplasmosis, uterine pathologies like pyometra, metritis, or other forms of endometritis due to non-infectious causes, nutritional deficiencies or toxicities and hormonal disorders such as cystic ovarian disease or luteal cysts.

Treatment

The infection in bulls can be treated with streptomycin @ 20 mg/kg BW IM along with application of 5 g of streptomycin in an oil-based suspension to the male genitalia for 3 consecutive days. Cows are not usually treated for genital campylobacteriosis.

Prevention and Control

No proven vaccines are available against the disease. Culling of affected animals from the herd is suggested to prevent the disease spread, as *C. fetus* is shed subsequent to pregnancy in cattle. Artificial insemination is an excellent way to prevent or control genital campylobacteriosis. As *C. fetus* may be isolated from cows for >6 months even after the end of pregnancy. Artificial insemination should be continued until all the cows in a herd are through at least two pregnancies. Routine testing of animals for campylobacter must be practiced monitoring the health status of the herd and identify the carriers.

Biosecurity Measures

Implement mandatory testing for newly acquired animals to prevent introducing organisms to the herd. Testing of semen stock to be used for AI in the farm and the breeding bulls must be carried out at regular intervals to check the spread of disease. Implement biosecurity measures in routine at the farm premises.

Sample Collection for Diagnosis

Preputial wash samples are the sample of choice for culture and identification of pathogen in breeding bulls. The stomach contents of aborted foetus, placental membranes, and vaginal mucous are recommended samples to be collected from the affected cattle.

2.33 Mastitis

Definition and Causative Agent

Mastitis is the inflammation of the mammary glands or udder in dairy animals. The term mastitis is taken from the Greek word *Mastos* for breast and *itis* for inflammation. The condition is manifested by hardening of the udder glandular tissue, physical and chemical changes in milk quality, decreased production, and increased somatic cell count of milk. Although bacteria, virus, mycoplasma, and fungus have been implicated as causes of mastitis, bacteria are the most commonly isolated agents. Most commonly encountered bacteria in mastitis cases include various species of streptococci, staphylococci, and Gram-negative lactose-fermenting organisms of enteric origin, commonly termed coliforms. Based on severity, mastitis can be categorized as subclinical and clinical mastitis.

Transmission

Mastitis occurs when microbes enter the teat through the teat canal and proliferate in the glandular tissue. While *Mycoplasma* species can be transmitted between cows via aerosols and subsequently invade the udder following bacteraemia, most contagious pathogens are spread during milking through the milkers' hands or the liners of the milking machine. The primary environmental source of pathogens is the bedding used for housing cattle, however, contaminated teat dips, water used for udder washing, intramammary infusions, water ponds, skin lesions, teat trauma, and flies are also implicated as sources of infection.

Clinical Signs

Based on severity, the mastitis can be categorised into clinical and subclinical mastitis. Cows with clinical mastitis exhibit redness, swelling, heat, and pain in the udder. The milk contains flakes and there is change in colour or presence of fibrin clots. The cow may develop fever as a systemic response to the infection. Other symptoms may include loss of appetite, oedema around the udder and decreased milk flow due to changes in milk consistency or udder tissue. Cases with only local clinical signs are termed mild or moderate while those with systemic response are termed as severe. In subclinical mastitis, there are no obvious signs of inflammation or visible changes in the milk although transient episodes of abnormal milk may appear. Unlike clinical mastitis, subclinical mastitis does not present with noticeable symptoms such as swelling, redness, or pain in the udder. One of the main indicators of subclinical mastitis is an elevated somatic cell count (SCC)in the milk, which can be measured by California mastitis test (CMT). There may be changes in milk quality, such as reduced milk yield, altered milk components (*e.g.*, lower butterfat or protein content), though these changes are often subtle. Once established, it is termed as subclinical chronic mastitis after two months.

Lesions

There are no obvious lesions in the udder in subclinical mastitis. The clinical form is manifested by oedematous swelling of udder tissue, hyperaemia and hardening of the affected quarter. If such cases are not treated and infection lasts for a longer duration, this may lead to cracks on the udder tissue, ulceration, abscess formation, and/or fibrosis in long standing cases.

Diagnosis

Preliminary diagnosis of mastitis is based on the presence of clinical signs and changes in the milk quality parameters along with somatic cell count of the milk. The milk quality can be tested by California mastitis test, strip cup test or direct somatic cell counting. A SCC of ≥ 200,000 cells/ml in a cow indicates a high likelihood of infection. Specific diagnosis can be made by bacterial culture of the milk and identification of causative organism by standard biochemical tests or PCR.

Differential Diagnosis

Mastitis must be differentiated from udder oedema in early lactation or due to nutritional deficiency, traumatic injury associated inflammation, chemical burns, and teat cysts.

Treatment

Parenteral antimicrobial therapy based on antimicrobial sensitivity testing is the most appropriate. However broad-spectrum antimicrobial treatment may be started with beta lactam antibiotics or enrofloxacin. The affected teat should be properly cleaned and washed with antiseptic solutions, and the teat should be emptied, then the broad-spectrum intramammary infusions containing suitable drugs can be administered. To reduce the inflammation of the affected quarter, the NSAIDs such as flunixin meglumine or meloxicam or tolfenamic acid may be given. Supportive drugs such as vitamin E and selenium or vitamin C should be given for reducing oxidant injury.

The ethnoveterinary medicine (EVM) and herbal drugs have been tried at large scale by National Dairy Development Board (NDDB) for treatment of mastitis and found efficacious. The validated EVM practices for treatment and better health management of bovine mastitis cases are in use to reduce the irrational use of antibiotics and thereby reducing the cost of treatment.

Prevention and Control

Maintaining the hygienic conditions in the animal shed is the first step to prevent or control the incidence of mastitis in the animals. Maintaining hygiene of milking equipment, use of an effective pre- and post-milking germicidal teat dip, individual towels to dry teats, gloves for milkers' hands, cleaning milking units after an infected cow has been milked, and segregation of infected cows are the basic steps to prevent mastitis. Cows should be provided dry and clean bedding. Inorganic bedding supports less bacterial growth than cellulose-based material; thus, sand is preferred over sawdust, straw, recycled paper, or manure solids. Regular cleaning or changing of bedding, decreasing heat stress, maintaining healthy teat condition, decreasing udder oedema in periparturient cows, maintenance of stalls for proper lying behaviour, preventing frostbite or fly exposure are the measure to prevent mastitis of environmental origin.

Biosecurity Measures

Biosecurity measures include maintaining higher standards of hygiene and cleanliness in the farm premises, isolation of diseased animals to avoid contamination of shed or the farm premises and quarantining newly purchased animals before they are introduced in the herd to prevent spread od pathogens causing mastitis. As such, strict quarantine and testing must be followed before introducing the animal into the herd to prevent the introduction of new pathogens into the farm premises.

Sample Collection for Diagnosis

Milk from the affected quarter is the ideal sample for diagnosis of mastitis and identification of causative agent. The brief protocol is given here:

1. Label the tubes prior to sampling including animal ID and respective quarter of udder.
2. Brush loose dirt and bedding from the udder and teats. Thoroughly wash and dry grossly dirty teats and udders before proceeding with sample collection.
3. Clean the teat ends with cotton swabs dipped in 70 percent alcohol.
4. Remove the cap from the tube and maintain at approximately a 45-degree angle while taking the sample. Do not allow the sample tube to touch the teat end.
5. The initial stream of milk should be discarded, and subsequent streams are collected. Collect 3 to 5 ml of milk and immediately replace and tightly secure the cap.
6. To collect a composite sample (milk from all quarters in the same tube), 1 to 2 ml of milk should be collected from each quarter of the udder.

Store samples immediately on ice and transport to the laboratory as early as possible.

2.34 Collection, preservation and dispatch of samples to laboratory for disease diagnosis

Necropsy examination of animal helps in diagnosis of diseases and ultimately their control. It is said "Necropsy is the message of wisdom from dead to living". It includes systemic examination of dead animal, recording of gross pathological lesions and their correlation with history to make diagnosis of diseases. Sometimes, it is difficult to make any conclusion merely on the basis of postmortem. In that situation, samples are to be collected for further laboratory analyses such as histopathology, microbiology and toxicology for confirmation of the etiological agents. Samples from all vital organs' tissues showing lesions and lymph nodes

are necessarily required. Tissues from putrefied carcasses should also be collected.

Purpose

- Diagnosis of disease and identification of new disease.
- Confirmation of tentative diagnosis.
- To observe the effect of treatment and give direction for future therapy.

Precautions

- The tissue sample should be fully representative of the organ and the lesions.
- Collect the tissues as early as possible after the death of an animal.
- Representative tissue/sample should be collected.
- Sharp knife should be used for cutting.
- Collect the tissues directly in fixative.
- Size of tissue should not be more than 1 cm for histopathology.
- Hollow organs should be taken on paper to avoid shrinkage.
- Hard organs like liver and kidneys should be collected along with capsule.
- Wide mouth glass or plastic bottle of varying capacity should always be used.
- 10 percent formalin solution is the best fixative for routine histopathological diagnosis.
- For virological examination, small pieces of spleen, lymph node and tissues from lesion sites may be sent either in 50 percent buffered glycerine or on ice.
- For bacteriological isolation, heart blood, tissue pieces and swab may be sent on ice.
- Proper labeling of the bottle/specimen samples is very essential.
- A piece of absorbent cotton should be placed on the surface of the sample in formalin to keep the tissue moist in case the bottle is broken during transit.
- The mouth of the specimen bottle or bags may be sealed carefully by paraffin wax or adhesive tape to avoid the leakage during transit and make it watertight.

Samples for bacteriological examination

- Collect the tissues under sterile condition.
- Sterilize knife/scalpel/spatula on flame or in boiling water.
- Sterilized the surface by hot spatula.
- Cut with knife and collect sample from inner tissue.
- Body fluids/blood should be collected in sterilized syringe or in Pasteur pipette.
- Specimens should be collected directly in media (liquid media-nutrient broth, peptone water, tetrathionate broth or even in normal saline solution/phosphate buffer saline).
- Seal, pack and transport the collected material to laboratory on ice/under cold chain conditions.

Samples for virological examinations

- Collect tissue under sterilized condition
- Body fluids/blood in sterilized syringe or in Pasteur pipette.
- Tissues in buffered glycerin
  - PBS pH 7.2 –50 percent
  - Glycerin – 50 percent
- Avoid samples in glycerine for sensitive viruses especially enveloped viruses, *e.g.*, PPR, and canine distemper
- Seal and mark the specimen bottle and transport to laboratory immediately.

Samples for toxicological examination

- Stomach/intestinal contents (about 100 g) in clean glass or plastic bottles over ice.
- Liver, kidneys, muscles (about 500 g), heart blood (about 50 ml) over ice.
- Urine in clean glass container on ice.
- Leftover feed/fodder in manger (about 1 kg) in airtight container over ice.
- Seal, label, transport to laboratory.
- In veterolegal cases, all specimens must be collected in presence of police/witness.
- Type of poison suspected along with detailed history, signs, lesions/treatment etc. should be written on letter with specimens.

Dispatch of material

Following points must be kept in mind while dispatching the material to laboratory for diagnosis:

- Describe the clinical signs, lesions, tentative diagnosis and treatment given to animal in the letter. Also mention the type of test required as per the tentative diagnosis.
- Write correct address on letter as well as on the parcel preferably with pin code, if the material is sent through post.
- Mark the parcel 'Biological Material', 'Handle with care', 'Glass material', 'Fragile', *etc.*, to avoid

damage in parcel. Also mark the side to be kept on upper side with arrows.

- Seal the container so that it should not leak in transit.
- Try to send the material as soon as possible after its collection from animal.
- Keep one copy of covering letter inside the parcel and sent another copy by hand or post in a separate cover.
- Keep adequate padding material like cotton in the parcel, which will save the material from outside pressures/jerks.
- Use dry ice, if available otherwise use ice pack in sealed containers.

CHAPTER- 3

GUIDELINES FOR INFECTIOUS DISEASES OF SMALL RUMINANTS

3.1 Preamble

Small ruminants - often called poor man's cow - play a pivotal role in national economy through meat, milk, and skin production. The major health problems encountered in small ruminants are mainly due to improper healthcare including vaccination. Therefore, timely diagnosis, treatment and control measures are essential to contain the illness. In this context, Standard Veterinary Treatment Guideline (SVTG) for small ruminants will help in rationalizing veterinary practice. This guideline will also give adequate information regarding dosage, frequency and route of administration, duration of therapy, prevention, control and withdrawal time of pharmaceutics in animals before use of products for human consumption. Further, information on proper biosecurity measures is aimed to reduce the pathogenic agents in the animals as well as in the environment. Thus, appropriate management together with strategic use of vaccines and drugs will decrease animal losses. These guidelines describe the different categories of the pathogen which include viruses, bacteria, mycoplasma, fungi, rickettsia, and chlamydia.

3.2 *Peste des Petits Ruminants* (PPR)

Definition and Causative Agent

PPR (goat plague), a viral disease of sheep, goats, camels and wild ruminants, is characterized by oculo-nasal discharge, severe oral necrosis, enteritis, and pneumonia. The disease is caused by PPR virus (PPRV) - also known as small ruminant morbillivirus (SRMV) or *morbillivirus caprinae* - classified within the genus *Morbillivirus*, family Paramyxoviridae.

Transmission

Transmission is mainly by aerosols or direct contact with infected animals. All the secretions and excretions retain infectivity for at least seven days after onset of disease. Fomites may also spread the infection.

Clinical signs

Clinical manifestations include high temperature, oculo-nasal discharges, ulcers, gastroenteritis, and bronchopneumonia, followed by death or recovery from the disease. The mortality usually ranges from 50 to 90 percent, and morbidity varies from 10 to 100 percent. Most of the animals show mortality within 10–12 days following onset of disease. Abortions are often recorded in field outbreaks.

Lesions

Numerous necrotic lesions in the oral cavity, bronchopneumonia, and zebra marking in large intestine.

Diagnosis

Presumptive diagnosis is based on clinical observations and postmortem lesions. Laboratory confirmation of the disease is usually done through virus isolation, virus neutralization assay, ELISA, reverse-transcription PCR (RT-PCR), loop-mediated isothermal amplification (LAMP), lateral flow assay (LAF), PCR-ELISA, real-time PCR, *etc.*

Differential diagnosis

The disease should be differentiated with pasteurellosis, contagious caprine pleuropneumonia (CCPP), bluetongue, heartwater, contagious ecthyma, foot-and-mouth disease, coccidiosis and plant or mineral poisoning.

Treatment

There is no specific treatment since PPR is a viral disease. Supportive treatment includes fluid therapy (based on clinical assessment of dehydration) and broad-spectrum antibiotics to treat secondary bacterial infections, wherever needed, and NSAIDs (*e.g.*, meloxicam @ 0.2-0.5 mg/kg BW IM), antioxidants (*e.g.*, vitamin C @ 3 g) and B-complex (3-5 ml on alternate day) can also be given. Lesions around the eyes, nostrils and mouth should be cleaned with potassium permanganate solution (0.01 percent) and boro-glycerine paste should be applied, along with good nursing care. Besides theses, herbal immunomodulators can also be given (refer EVM practices).

Prevention and Control

The only way to prevent PPR is by vaccination using live attenuated PPR vaccine (Sungri/96). Vaccination at 3 months of age provides lifelong immunity. The animals suspected to be exposed within the infected zone should not be vaccinated. The vaccine is safe for use in pregnant animals also.

Biosecurity Measures

In an outbreak, rapid identification and isolation of sick animals and their contacts (since manifestation

of first symptoms) and elimination and disposal of dead animals by incineration or deep burial of carcasses is important. Other measures include strict quarantine and control of animal movements as well as thorough cleaning and disinfection every 24 hours - with disinfectants like phenol or sodium hydroxide (2 percent) - of contaminated areas of all premises including physical perimeters, equipment and clothing.

Sample Collection for Diagnosis

i) Antemortem materials

Nasal, ocular and oral swabs (best material for antigen detection and virus isolation) in viral transport media* or phosphate buffer saline*; unclotted blood (use EDTA or Heparin) for virus isolation and immunohistochemistry; paired serum 21 days apart for serology.

ii) *Post-mortem materials (tissue samples)*#

Piece of lymph node, spleen, lungs, intestine (caecum/rectum), ileo-caecal junction for antigen detection and histopathology.

* Concerned laboratory may be contacted for sample collection and transportation; # One set of the tissue samples is chilled but not frozen. This set of samples can be used for making impression smears for immunofluorescence assay; #One set of the tissue samples is collected aseptically and stored frozen at -20°C. Such tissues are used for virus isolation in natural host or in cell culture; #One set of tissues is preserved in 10% neutral buffered formalin solution. Such tissue samples are generally used for histopathological examination.

3.3 Sheep pox and Goat pox

Definition and causative agent

Sheep pox and goat pox are viral diseases of sheep and goat is caused by sheep pox virus (SPPV) and goat pox virus (GTPV), respectively. It is characterized by fever, generalized papules or nodules, internal lesions (particularly in the lungs), and death. The SPPV and GTPV are classified under the genus *Capripoxvirus*.

Transmission

Disease is commonly transmitted by aerosol infection associated with close contact with infected animals. Virus is present in nasal and oral secretions for several weeks after infection and can live in scabs that have fallen off the animal for several months. Transmission can also take place by indirect transmission from contaminated fomites.

Clinical Signs

Rise in rectal temperature to >40°C. Macules develop in 2 to 5 days followed by papules which may cover the body or be restricted to the groin, axilla and perineum. Within 24 hours of the appearance of generalized papules; affected animals develop rhinitis, conjunctivitis and enlargement of all superficial lymph nodes, especially prescapular lymph nodes. Skin papules undergo necrosis and scab formation, which persist for up to 6 weeks, leaving small scars.

Lesions

Various lesions in sheep pox and goatpox affected animals include congestion, cutaneous haemorrhage, oedema, vasculitis and necrosis involving all skin layers, enlargement of lymph node, lymphoid proliferation, oedema, pock lesions distributed widely on mucous membranes of various organs like eyes, mouth, nose, pharynx, trachea, ruminal, abomasal, muzzle, nares, vulva, as well as lesions on prepuce, testicles, udder, and teats. Severe and extensive pock lesions include focal and/or uniformly distributed pock lesions throughout the lungs particularly in the diaphragmatic lobes along with congestion, oedema, focal areas of proliferation with necrosis and lobular atelectasis. The mediastinal lymph nodes are enlarged, congested, oedematous and show haemorrhages.

Diagnosis

A tentative diagnosis can be made based on clinical signs consisting of skin lesions, persistent fever, lymphadenitis, and often pneumonia. Laboratory diagnosis is based on polymerase chain reaction (PCR), real-time PCR, sequencing, electron microscopy, and serological tests like counter-immunoelectrophoresis (CIE), virus neutralization, ELISA kits available commercially or be obtained from ICAR-IVRI.

Differential Diagnosis

The clinical signs of severe sheep pox and goat pox are highly characteristic. However, in their mild form, differential diagnosis includes contagious ecthyma (orf), insect bites, bluetongue, *peste des petits*

ruminants, photosensitisation, dermatophilosis, parasitic pneumonia, caseous lymphadenitis, and mange.

Treatment

No specific treatment is available. supportive treatment includes antibiotics to treat secondary bacterial infections, NSAIDS (*e.g.*, meloxicam @ 0.2-0.5 mg/kg BW IM) for management of fever and pain, antioxidants and B-complex. Rumenotoric, probiotics, and liver tonics for management of anorexia. Topical treatment with potassium permanganate solution (0.01 percent) and wound dressing may be necessary in severely affected animals.

Prevention and Control

Live vaccines are used to control goat pox and sheep pox using Goat pox vaccine (Uttarkashi strain) and Sheep pox vaccine (Srinagar strain), respectively, as per Manufacturer's recommendations. These are used at age of 4 months and above and give immunity for more than 4 years as tested so far.

Biosecurity Measures

Various biosecurity measures include isolation of infected herds and sick animals for at least 45 days after recovery, slaughtering of infected herd, if possible, proper disposal of cadavers and products by burning or burial (often used), stringent cleaning and disinfection of farms and equipment, quarantine of new animals before introduction into herds, and movement control of animal and vehicles within the farm premises including infected areas. Mass-scale vaccination should be done when the disease has spread more widely.

Sample Collection for Diagnosis

From live animals, collect full skin thickness biopsies, scabs, skin scrapings, lymph node aspirates, whole blood into heparin or EDTA, and at 28 days interval collect paired serum samples.

At necropsy, collect pieces of skin containing lesions, lymph nodes, lung lesions, full set of tissues (especially those with lesions) for histopathology.

Samples for virus isolation should be collected within the first week of the occurrence of clinical signs and/or before the development of neutralizing antibodies.

Samples for viral genome detection by PCR may be collected before or after the development of neutralizing antibody responses.

Samples for histopathology should include tissue from the surrounding area and should be placed immediately following collection into ten times the sample volume of 10 percent formalin or neutral buffered 10 percent formal saline.

3.4 Contagious Ecthyma (orf)

Definition and Causative Agent

Contagious ecthyma - also known as contagious pustular dermatitis, sore mouth, or orf - is an acute proliferative dermatitis of sheep and goat characterized by the presence of papules, vesicles or pustules and subsequently scabs around lips, and coronary bands of the feet. The disease is caused by orf virus of the genus *Parapoxvirus*.

Transmission

Orf virus enters the skin through cuts and abrasions. Loss of epithelial integrity is the important predisposing factor for initiation and the damage caused by eruption of incisors in young animals is enough to predispose to infection.

Incidence is higher in young lambs or kids. Stressors such as transportation, immunosuppression or primary infections due to other agents are the predisposing factors. Seasonal occurrences immediately after lambing and after entry into a feedlot are common. The virus is extremely resistant to environmental conditions and can contaminate small ruminant facilities for many years.

Clinical Signs

Clinically, the proliferative lesions appear on the lips, muzzle, mucocutaneous junctions, nostrils, gums and sometimes spread to internal organs including the tongue. Dermal lesions are sometimes observed on other body parts such as the ears, eyelids, forehead, and the coronets. In lactating animals, lesions may appear on the udder/teats. The lesions are also often self-limiting. During the disease (1–4 weeks), the scabs drop off and the tissues heal without scarring. Younger lambs and kids will have difficulty in nursing and become weak.

Lesions

In addition to skin and mucosal lesions, gross lesions may be found rarely in the oesophagus, rumen, omasum, lower intestinal tract, heart and respiratory

tract. Skin and mucosal lesions may sometimes be accompanied by regional lymphadenopathy. Lesions are highly vascular in nature leading to profuse bleeding when injured or biopsied.

Diagnosis

Laboratory diagnosis of orf is based on following assays:

i) *Identification of the agent*: Counter-immunoelectrophoresis (CIE), viral genome detection by PCR and/or real-time PCR, viral genome sequencing, electron microscopy to rapidly identify typical virions of orfvirus, virus isolation in cell culture (primary lamb testis or lamb kidney).
ii) *Serological tests*: Virus neutralization, counter-immunoelectrophoresis (CIE), ELISA.
iii) *Differential diagnosis*: Capripox, dermatophilosis, ulcerative dermatosis, bluetongue, staphylococcal dermatitis, FMD.

Treatment

No specific treatment is available. Removal of the scabs and the application of ointments (boric acid-glycerine/zinc oxide), antiseptics (povidone iodine/potassium permanganate/ chlorhexidine) or astringent lotions on wound helps but might also delay healing in most cases. Provide soft and palatable food to sick animals. The combined use of diathermy debridement and cryosurgery is claimed to be effective for the proliferative intraoral lesions in young lambs. EVM or herbal preparations can be used for applying on the wound for enhanced wound healing (Refer EVM Section and Annexures).

Prevention and Control

The removal of harsh vegetation from pastures or feed might help in reducing abrasions around the mouth that facilitate virus entry. Individuals handling infected animals should be advised of taking precautionary steps like workers wearing gloves, avoiding mixing of clothes worn while working and PPE, regular cleaning and disinfection of – clippers, ear tagging devices, and other similar equipment after each use, *etc.*

Vaccinating lambs and kids with commercial vaccine is the best practice to prevent disease. The primary objective of the vaccination is to reduce the severity and duration of clinical course as solid immunity is unlikely. All the newcomer animals, on their arrival, must be vaccinated before introducing them to a new environment. Precautions must be taken when vaccinating the animals because the vaccine may induce orf in animal handlers. It is not recommended to vaccinate animals in flocks already free of the disease.

Biosecurity Measures

Various biosecurity measures include segregation of the infected animals to avoid spread of infection, isolate the freshly introduced animals avoiding contact with the existing stock on the farm, thorough cleaning and disinfection of farm premises and equipment, and strict movement control of animals/personnel/vehicles within the farm premises. Vaccination with live vaccine is also not recommended on a farm with no previous history of the outbreak.

Sample Collection for Diagnosis

Collect skin scabs/scrapings in a sterile sample container containing PBS (pH, 7.2–7.4), place on ice and transport to the laboratory.

3.5 Nairobi Sheep Disease Virus or Ganjam Virus

Definition and Causative Agent

Ganjam is a highly infectious tick-borne viral disease of small ruminants (sheep and goat) caused by Nairobi sheep disease virus (NSDV) or Ganjam virus (GANV), which is characterized by biphasic fever, acute haemorrhagic gastroenteritis, profuse diarrhoea and abortion. The GANV is recognized as an Asian variant of Nairobi sheep disease virus and has been named after the location of the first-time virus isolation, Ganjam city in Odisha, India. The virus is classified under genus Nairovirus, and family Bunyaviridae. This virus is enveloped in nature and its genome is consisting of single-stranded, three segmented, negative sense RNA.

Transmission

GANV is tick-borne and mostly transmitted through the bite of any stage of the infected tick. The virus is primarily transmitted through the tick *Haemaphysalis intermedia*, *H. wellingtoni*, *R. haemaphysaloides*, and the mosquito *Culex vishnui*. All tick hosts are able to maintain the virus though both transovarian and transstadial transmission. The virus is present in urine and faeces of infected

animals; however, direct contact does not result in infection.

Clinical signs

The clinical signs begin with a steep rise in body temperature (40.55°- 41.11°C) that persists for 1 to 3 days and follows an incubation period of 4 to 5 days. Sometimes leucopoenia and viraemia usually coincide with the febrile phase of the disease. At the onset of the disease, there is profuse and watery diarrhoea which at later stage may convert into bright to dark-green faeces. Death may occur in the early febrile viraemic phase, or it may follow two days after remission of the fever without appearance of any clinical signs. Goats show similar clinical signs although with low severity compared to the sheep.

Lesions

On external examination of the carcass, presence of ticks attached on the body surface especially in the ear and head is the most striking feature. Petechial haemorrhages are observed in the coronary band above the hoofs. Additionally, petechial haemorrhages are observed on the nasal mucosa also and there is excessive froth in the trachea. The prescapular lymph nodes on one side are notably enlarged and oedematous with subcapsular haemorrhages. Haemorrhages and oedema are observed in the submucosa and muscular layer of gastrointestinal tract. Ulceration may be seen in the mucosa of ileum, caecum and colon. Glomerular-tubular nephritis in the kidney is a constant feature.

Diagnosis

Diagnosis can be made on the basis of clinical signs. However, these clinical signs are common to other viral infection and, therefore, laboratory diagnosis is necessary. Virus isolation can be considered as gold standard for the diagnosis of GANV. BHK_{21}-C13 is the most suitable cell line for isolation of GANV. Agar gel immunodiffusion test can be used as valuable diagnostic tool for detection of viral antigen in the infected animal tissues like spleen and mesenteric lymph nodes. Serological test like ELISA can be used for the detection of GANV-specific antibodies. The RT-PCR is the most sensitive molecular diagnostic tool, which can be used for the confirmation of virus as a causative agent of the disease.

Differential diagnosis

Laboratory diagnosis is an essential tool for its differentiation from *peste des petits ruminants* (PPR), coccidiosis, salmonellosis, Rift valley fever, cowdriosis and sometimes toxicity like arsenic poisoning in sheep and goat.

Treatment

No specific treatment is available. Supportive treatment including NSAIDs, broad-spectrum antibiotics to treat secondary bacterial infections, provision of good shelter and quality feed, may improve survival. Unaffected animals in the flock may be treated with acaricides (cypermethrin pour-on products, or various dip preparations).

Prevention and Control

Disease reporting and quick response is necessary for the control of the outbreaks in disease-free area. Veterinarians who suspect the infection should follow the national and local guidelines for disease reporting. Currently, vaccines are not commercially available.

Biosecurity Measures

Being a zoonotic emerging infection, strict biosecurity measures need to be taken at all levels. Rapid detection of pathogen, vector control, and handling of suspected samples in biocontainment is required.

Sample Collection for Diagnosis

Infected animal tissues like spleen and mesenteric lymph nodes can be used for the antigen detection, while paired serum (collected 21 days apart) can be collected for the antibody detection.

3.6 Rift Valley Fever

Definition and Causative Agent

Rift valley fever (RVF) is caused by a segmented single-stranded negative-sense RNA virus known as Rift valley fever virus (RVFV), The RVFV is a member of the *Phlebovirus* genus in the Bunyaviridae family. The RVF disease is an acute viral zoonotic disease primarily affecting domestic animals like sheep, goat, cattle, as well as humans. The disease is marked by high mortality rates in young animals and abortions in pregnant females.

Transmission

RVFV is predominantly transmitted by mosquito vectors, specifically *Aedes* and *Culex* species. Between outbreaks, the virus spreads between

mosquito vectors and susceptible domestic or wild ruminants, as well as vertically through specific *Aedes* spp. mosquitoes. RVFV can also be transmitted and arise or re-appear through the migration of viraemic animals. Sporadic, sometimes massive, outbreaks of illness in ruminants are typically connected with excessive rains and localized flooding.

Clinical Signs

Clinical signs of RVF are often nonspecific, making individual cases challenging to identify. In lambs, biphasic fever may develop, and they become listless, reluctant to move, and may exhibit signs of abdominal pain. Mortality in young lambs is high, resulting in death within 2–3 days. Adult sheep show generalized febrile responses, hematemesis, haematochezia, and nasal discharge. Abortion can, sometimes, be the only indication of infection, with the aborted foetus usually autolyzed.

Lesions

In aborted foetuses and newborn lambs, the liver is notably enlarged, soft, and friable, exhibiting irregular congested patches. Although numerous greyish-white necrotic foci are typically present, they may not always be clearly visible. Common findings include haemorrhage and oedema in the gallbladder wall and the abomasum mucosa. The spleen and peripheral lymph nodes are often enlarged, oedematous, and may show petechial haemorrhages. Severe and extensive liver lesions, with hepatic necrosis is the characteristic histological feature of RVF.

Diagnosis

RVF should be suspected in sheep experiencing abortions and neonatal mortality, especially following heavy rains and flooding. The presence of specific histopathological lesions, particularly in the liver of lambs, is indicative of the disease and confirmed by immunohistochemical detection of viral antigens in fixed tissues. RT-PCR in serum, tissues, and mosquitoes is used for viral RNA detection. ELISA for IgM antibodies, or seroconversion observed in paired serum samples through virus neutralization, ELISA, or haemagglutination inhibition, can confirm the disease. Rift Valley fever is zoonotic; hence, all isolation and identification procedures should be conducted under biosafety level 3 conditions.

Differential Diagnosis

RVF must be differentiated from bluetongue, *peste des petits ruminants* (PPR), Nairobi sheep disease, Liver fluke infection, and enterotoxaemia.

Treatment

No specific treatment is available. NSAIDs are recommended in fever and pain. Supportive therapy with antioxidants (*e.g.,* vitamin C or vitamin E and selenium) and vitamin B-complex can be used.

Prevention and Control

Immunization is the primary and most effective method for protection against RVF. In endemic areas, inactivated or live-attenuated vaccines are used. Controlling mosquito populations through insecticides, environmental management, and eliminating breeding sites reduces the risk of RVF. PPE must be worn when handling potentially infected animals or their tissues to prevent the spread of the disease.

Biosecurity Measures

Implementing effective pest control programs to minimize contact between animals and mosquitoes.

Sample Collection for Diagnosis

For laboratory diagnosis, blood from viraemic animals and foetal organs as well as postmortem specimens of the liver, spleen, and brain are the preferred samples. However, tissues and fluids from infected animals pose a high risk of infection to human handlers.

3.7 Caprine Arthritis and Encephalitis

Definition and Causative Agent

Caprine arthritis and encephalitis (CAE), a chronic viral disease affecting goats and occasionally sheep, is caused by the caprine arthritis encephalitis virus (CAEV). It is an enveloped, single-stranded RNA lentivirus belonging to the Retroviridae family. The CAE manifests as polysynovitis-arthritis in adult goats. In kids, it is less commonly seen as leukoencephalomyelitis, with symptoms such as progressive weakness, ataxia, and proprioceptive deficits. Additionally, the virus can cause subclinical or clinical interstitial pneumonia, indurative mastitis, and chronic wasting.

Transmission

The virus is primarily transmitted through the ingestion of colostrum and milk from infected animals, but it can also spread via direct contact with infected fluids, including saliva, nasal secretions, and faeces. A significant route of infection is vertical transmission from the dam to the kid through the placenta or milk.

Clinical Signs

Polysynovitis-arthritis is the most prevalent manifestation of infection, with symptoms including joint swelling and varied degrees of lameness. Affected goats typically lose weight and have weak hair coats. Depression, head tilt, circling, opisthotonos, torticollis, and leg pedalling are observed. Chronic interstitial pneumonia in adult goats with CAEV serology might evolve to dyspnea over time. CAEV infection causes the hard udder condition with a firm, enlarged mammary gland and agalactia during parturition. Milk quality is generally unaffected.

Lesions

During physical examination, infected goat's lungs may be viewed as hard and greyish pink, with many tiny white foci that do not collapse. In chronic cases, soft-tissue calcification of joint capsules, tendon sheaths, and bursae is common. Gross lesions, associated with the neurologic form include asymmetric reddish pink swelling regions in the cervical and lumbosacral spinal cords. Histopathology reveals multifocal mononuclear cell inflammatory infiltrates and different degrees of demyelination.

Diagnosis

AGID and ELISA are used for laboratory validation of virus-specific antibodies. Virus isolation and/or PCR assays can be utilized to confirm the presence of viral antigen in tissues.

Differential Diagnosis

The disease should be differentiated from enzootic ataxia, spinal cord abscess, cerebrospinal nematodiasis, spinal cord trauma, and congenital anomalies of the spinal cord and vertebral column. If a neurological examination suggests brain involvement, polio encephalomalacia, listeriosis, and rabies should also be considered.

Treatment

No specific treatment is available. However, goats with polyarthritis may be treated with NSAIDs like meloxicam or phenylbutazone. Antibiotics having good tissue penetrations can be used to treat secondary bacterial infection.

Prevention and Control

Avoid pooled colostrum for controlling disease transmission in kids. Implementing semi-annual serologic testing of the herd with concomitant identification and segregation of seronegative from seropositive goats will check the spread of infection and eventual culling of the seropositive goats.

Biosecurity Measures

Conduct regular serological testing to monitor the health status.

Sample Collection for Diagnosis

Serum, milk and tissue samples are collected for serological tests to detect antibodies and for PCR.

3.8 Coronavirus Infection in Sheep and Goats

Definition and Causative Agent

Coronavirus infections in sheep and goats are caused by viruses belonging to the family Coronaviridae and the genus *Betacoronavirus*. These viruses possess a positive-sense single-stranded RNA genome and are characterized by their crown-like appearance under electron microscopy due to spike proteins on their surface. Coronaviruses infect sheep and goats, causing respiratory and gastrointestinal illnesses.

Transmission

Coronavirus infections are highly contagious, and the most important route is faecal-oral route. The infected animals excrete the virus in their faeces, contaminating the environment, feed, and water sources. The virus can be transmitted through aerosols and droplets generated by coughing, sneezing, or close contact with infected animals. Direct contact with infected animals or their secretions, including nasal discharge and saliva, facilitates transmission. Indirect transmission can occur through contaminated equipment, bedding, clothing, and hands of personnel.

Clinical Signs

Lambs and kids often exhibit gastrointestinal signs, including diarrhoea, dehydration, and reduced feed intake. Respiratory symptoms such as coughing, nasal discharge, and fever may also occur. In adult sheep and goats, the respiratory symptoms predominate, including coughing, nasal discharge, dyspnea, and fever. Gastrointestinal signs are less common but can occur in outbreaks.

Lesions

Lesions are primarily found in the gastrointestinal and respiratory tracts. In gastrointestinal tract, infected animals exhibit villous atrophy and blunting in the small intestine, leading to malabsorption and diarrhoea. The mucosa may show congestion, haemorrhages, and necrosis. In respiratory tract, the lesions include congestion, oedema, and inflammation of the nasal passages, trachea, and bronchi. In severe cases, interstitial pneumonia and bronchopneumonia can develop.

Diagnosis

Observing clinical signs such as diarrhoea and respiratory symptoms provides initial clues. PCR detects viral RNA in faeces, nasal swabs, or tissues. ELISA is also commonly used for detection of viral antigens and antibodies. Virus isolation in cell culture is less commonly used.

Differential Diagnosis

Differential diagnosis is essential to distinguish coronavirus infections from rotavirus, cryptosporidium, and ETEC infections.

Treatment

Treatment of coronavirus infections is primarily supportive, as there are no specific antiviral drugs available. Fluid therapy by oral rehydration solutions (ORS) or intravenous fluids (Ringer's lactate/Normal saline/dextrose normal saline) to correct dehydration and electrolyte imbalances should be given. Continued feeding to maintain energy intake, using easily digestible and nutrient-rich feeds is beneficial. Rumenotorics, probiotics, and liver tonics can be supplemented. Antibiotics may be used to treat secondary bacterial infections along with NSAIDS.

Prevention and Control

Vaccines are available for bovine coronavirus and can be used in sheep and goat to boost colostrum antibodies, providing passive immunity to newborn kids. The pregnant dam is vaccinated 6 and 2 weeks before parturition to stimulate antibody production. This ensures that newborns receive adequate, high-quality colostrum within the first few hours of life to confer passive immunity. Regular cleaning and disinfection of housing, equipment, and feeding areas to reduce environmental contamination and segregating infected animals to prevent the spread of the virus to healthy individuals are important preventive measures.

Biosecurity Measures

Implementing strict biosecurity measures is crucial to prevent the introduction and spread of coronavirus infections. Quarantine new animals for a minimum of two weeks and monitor for signs of illness. Minimize animal movement during outbreaks to prevent the virus spread. Ensure that personnel follow strict hygiene protocols, including the use of PPE and disinfection of hands and clothing. Implement measures to control pests that may act as mechanical vectors for the virus.

Sample Collection for Diagnosis

Fresh faecal samples should be collected using clean gloves and sterile containers. These samples are particularly useful for detecting enteric coronaviruses. Collect nasal swabs using sterile swabs and transport media for detecting respiratory coronaviruses. In postmortem examination, collect tissue samples from the intestines, lungs, and lymph nodes for histopathology and PCR testing. Collect blood samples in sterile tubes for serological testing to detect antibodies against coronaviruses. Store samples at 4°C and transport them to the laboratory as soon as possible to preserve viral integrity for accurate testing.

3-9 Border Disease (BD)

Definition and Causative Agent

Border disease (BD), also known as hairy shaker disease, is a congenital condition in lambs, often leading to outbreaks of infertility and abortion in ewes. It is caused by *Pestivirus* of Flaviviridae family and is closely related to bovine viral diarrhoea virus and classical swine fever virus.

Transmission

The infection spreads vertically and horizontally,

and both directly and indirectly. Persistently infected rams often carry substantial viral loads in their semen.

Clinical Signs

Late abortions can occur, with kids able to lift only their head and neck. Lambs may be undersize with excessively hairy or pigmented fleece. Skeletal abnormalities include shortened limbs and cranium. Some lambs show muscle tremors, especially in the trunk and hindlegs, which worsen with movement. Affected lambs have poor survival rates, but nervous signs can disappear within 3-4 months in survivors. Border disease virus may cause low fertility in ewes and poor viability in lambs.

Lesions

In severe cases, necropsy may reveal abnormal development of the cerebrum, such as hydrocephalus, hydranencephaly, microcephaly or porencephaly. Cerebellar hypoplasia or dysplasia can also occur. Typically, the characteristic lesions are microscopic and affect the white matter of the central nervous system (CNS).

Diagnosis

Clinical signs often lead to a diagnosis of border disease. Confirmation is by histological examination showing characteristic lesions in the CNS and immunocytochemical staining for the virus. In lambs with typical hairy-shaker symptoms, the virus or viral antigen can be easily detected in blood and tissues using methods such as virus isolation, fluorescent antibody tests (FAT), immunohistochemistry, or polymerase chain reaction (PCR).

Virus isolation from serum or buffy coat cell samples in cell cultures is possible, and a viral antigen detection ELISA using heparinized or EDTA blood is available. Reverse transcriptase-PCR can also detect viral RNA in clinical samples and distinguish between different ruminant pestiviruses.

Differential Diagnosis

Differential diagnoses in live-borne lambs include bacterial meningoencephalitis, swayback (enzootic ataxia), daft lamb disease and focal symmetric encephalomalacia.

Treatment

No specific treatment is available. Affected lambs should be provided with colostrum to maintain adequate energy intake. Supplementation with vitamin B complex can be done.

Prevention and Control

Promoting flock immunity and prevent early pregnancy exposure to infection. Persistently infected sheep continuously spread the disease, and those that survive to breeding age can perpetuate it, necessitating identification and culling.

Biosecurity Measures

In infection-free flocks, new ewes and rams should undergo screening before purchase or be quarantined upon farm arrival. Newly introduced sheep should be kept apart from the main flock until after lambing. Ideally, pregnant sheep should not share pasture or housing with cattle.

Sample Collection for Diagnosis

Formalin-fixed samples of skin, spinal cord, half of the mid-sagittally sectioned brain, thyroid, distal ileum, colon, caecum, thymus, spleen, liver, heart, and kidney, for histological examination using light microscopy and immunohistochemistry. Serology can be conducted using heart blood serum or thoracic fluid for virus neutralization testing. Samples from placenta/caruncle, thymus, lymph node, spleen, thyroid, brain, and ileum are used for virology testing using immunostaining, ELISA, FAT and PCR.

3.10 Bluetongue

Definition and Causative Agent

Bluetongue is an infectious, non-contagious viral disease affecting domestic and wild ruminants, particularly sheep and goats. It is caused by the bluetongue virus (BTV) of the genus *Orbivirus* of the family Reoviridae. BTV has a segmented, double-stranded RNA genome enclosed in a double-layered protein capsid. The virus is highly variable, with 29 known serotypes.

Transmission

Bluetongue is transmitted primarily by biological vectors like certain species of Culicoides midges as these insects become infected by feeding on the blood of an infected animal and can then transmit the virus to other susceptible hosts during subsequent feedings. Transmission is influenced by temperature, humidity, and the presence of suitable habitats for the midges. Mechanical

transmission via contaminated needles and instruments can occur, although it is less common. There is no evidence of direct animal-to-animal transmission.

Clinical Signs

High fever followed by swelling of the face, lips, muzzle, and ear, as well as ulcerations and erosions in the mouth lead to excessive salivation and difficulty in swallowing. The tongue may become swollen, cyanotic (blue), and protrude from the mouth, giving the disease its name. Inflammation and swelling of the coronary band and laminitis can cause lameness. Difficulty in breathing due to pulmonary oedema and serous to mucopurulent nasal discharge is observed. Loss of wool due to dermatitis and itching. Clinical signs are more severe in sheep than other ruminants.

Lesions

The lesions observed in bluetongue-infected animals are primarily due to vascular damage and inflammation. Common lesions include generalized oedema of the face, lips, and submandibular area. Petechial and ecchymotic haemorrhages in the oral and nasal mucosa, heart, lungs, and gastrointestinal tract. Necrotic ulcers on the tongue, dental pad, cheeks, and hard palate. Reddening and ulceration of the coronary band, leading to lameness and congestion and oedema in the lungs.

Diagnosis

Laboratory diagnostic methods include detection of viral genome by PCR, viral antigen detection by sandwich ELISA and/or virus isolation in cell lines or embryonated chicken eggs. Serological detection includes detection of antibodies against BTV by ELISA and AGID test.

Differential Diagnosis

The disease should be differentiated from foot and mouth disease (FMD), PPR, orf (contagious ecthyma), sheep pox and goat pox.

Treatment

No specific treatment. Supportive care includes treatment of secondary bacterial infections, and use of NSAIDs, antihistamine, fluid and electrolyte therapy.

Prevention and Control

Controlling bluetongue involves vector control, vaccination, and management practices. Available vaccines include inactivated pentavalent vaccine in India. Implementing quarantine and movement controls during outbreaks to prevent the spread of the virus is important.

Biosecurity Measures

Effective biosecurity measures are crucial to prevent the introduction and spread of bluetongue. Quarantine new animals or returning animals for a period to monitor for signs of disease. Minimize animal movement during peak midge activity periods. Regular cleaning and disinfection of equipment and facilities. Use insect-proof housing and insecticides to reduce midge populations. Regular monitoring of animals for clinical signs and implementing early diagnostic testing during outbreaks.

Sample Collection for Diagnosis

Collect whole blood samples in anticoagulant (heparin) tubes for PCR and virus isolation. Serum samples can be used for serological tests. Collect tissue samples from necropsied animals, including lymph nodes, spleen, lungs, and heart, for virus isolation and histopathology. Nasal or oral swabs can be collected for PCR testing. Samples should be collected in sterile VTM/PBS for PCR and virus isolation, whereas samples should be collected in 10 percent formalin for histopathology. Capture and submit Culicoides midges for virus detection to identify vector involvement in transmission. Samples should be kept cool and transported to the laboratory as soon as possible, ideally within 24-48 hours, to preserve the integrity of the virus for accurate testing.

3.11 Rabies

Definition and Causative Agent

Rabies, an acute, invariably fatal disease in man and other warm-blooded animals, is caused by rabies virus belonging to genus *Lyssavirus* of family Rhabdoviridae. Lyssavirus is an enveloped single-stranded, negative- sense RNA virus having bullet shaped structure.

Transmission

Dogs are the main source (up to 99 percent) of rabies transmission to domestic animals including cattle, buffalo, sheep and goat. The rabies virus is mainly transmitted through saliva. However, less

often; virus from saliva, salivary glands, or neural tissues can cause infection by entering the body through intact mucous membranes or fresh wounds. Mongoose and jackals are its major reservoirs in the wild in Indian sub-continent. In sylvatic cycle of rabies, wild animals including bats, raccoons, and foxes also serve as the maintenance host for the virus.

Clinical Signs

Profuse salivation and restlessness (furious form) are the only observed clinical signs of rabies in goats. Behavioural change, or mania, aggression or hyperesthesia can also be seen in affected goats. Trembling, lateral recumbency, convulsion, opisthotonos, and fever are the symptoms in sheep. Other symptoms like, arched back, tremors, and a swimming movement of all four limbs, followed by paralysis and death are other symptoms in this disease.

Lesions

There are no characteristic gross lesions. The typical histological signs, found in the central nervous system, involves multifocal, mild, polio encephalomyelitis and craniospinal ganglionitis with mononuclear perivascular infiltrates, diffuse glial proliferation, regressive changes in neuronal cells, and glial nodules. Negri bodies can be seen in some but not all cases.

Diagnosis

Signs and symptoms are definitive symptoms in furious form which aid in diagnosis. Direct fluorescent antibody (DFA) test is the gold standard assay recommended by OIE for rabies diagnosis. Other tests for rabies diagnosis are direct rapid immunohistochemistry test (dRIT), ELISA, virus isolation using cell culture or mouse inoculation test (MIT), and PCR assays – RT-PCR, fluorescent antibody virus neutralization test (FAVN); and rapid fluorescent focus inhibition test (RFFIT) as gold standard for assessing the viral neutralising antibodies.

Differential Diagnosis

Rabies should be differential diagnosed with nervous ketosis, scrapie, listeriosis, hypomagnesemia tetany, botulism.

Treatment

There is no treatment for rabies. Post-exposure vaccination (PEV) with ARV (anti-rabies vaccine) on 0, 3rd, 7th, 14th, and 28th day is recommended.

Prevention and Control

Control of stray dog population is important for prevention of rabies in India. Irrigation of the wound with soap solution for 15 minutes or a solution of benzalkonium chloride for at least 5 minutes is crucial.

Biosecurity Measures

Immediately report any suspected rabies cases to veterinary and public health authorities for confirmation and response. Quarantine the exposed animal; use gloves, masks, and protective clothing when handling affected animals suspected of having rabies; implement measures to prevent contact between affected animals and potential rabies vectors such as bats, raccoons, foxes, and stray dogs.

Sample Collection for Diagnosis

Samples of saliva, serum and cerebrospinal fluid are the preferred samples for antemortem examination while for postmortem while fresh brain (including sections of cerebellum, cerebral cortex, brainstem, medulla and hippocampus) is the preferred specimen for confirmatory testing of rabies and should be submitted in glycerol-phosphate buffered saline. Additionally, cerebrospinal fluid, neck skin biopsy, and serum samples may also be collected.

Public Health Risk

Rabies poses a potential public health threat associated with human exposures that result during handling sick animals and through bites or saliva or milk from rabid cattle, leads to fatal encephalitis if not promptly treated.

Do's

- Wash the wound immediately with plenty of water and soap for a minimum of 15 minutes
- Consult the Physician immediately and seek advice regarding post-bite immunization
- Complete the course of anti-rabies vaccination, as per your Doctor's advice.

Don'ts

- Do not suture or bandage the wound
- Do not apply turmeric powder, mud, *etc.*, to the bite wound
- Do not drink milk of sheep, goat and cow bitten by rabid dog

3.12 Foot and Mouth Disease (FMD)

Definition and Causative Agent

Foot and mouth disease (FMD) is a viral infection that affects cattle, sheep, goat, swine and other cloven-hooved domestic animals caused by foot and mouth disease virus (FMDV) of genus *Aphthovirus* and family Picornaviridae. FMDV is a non-enveloped, single-stranded RNA virus of genome size ~8.2 kb. It exists in seven serotypes (O, A, C, Asia-1, SAT-1, SAT-2, SAT-3) antigenically and, of these only three serotypes (O, A, Asia-1) are prevalent in India. However, none of the serotypes confer immunity against others. FMD is rarely fatal for adults but leave the animals weakened and debilitated leading to severe production losses. FMD is generally characterised by fever and blister-like sores on the tongue and lips, in the mouth, on the teats and between the hooves but it is often inapparent in sheep and goat. Nevertheless, sheep and goat can become carriers spreading the infection. Thus, it is imperative to study the epidemiology of FMD in small ruminants and take measures to restrict its spread in them.

Transmission

FMD is a highly contagious disease and is found in all excretions and secretions of infected animals. It can be transmitted via aerosols through oral and nasal route. FMD can be easily spread through direct contact with infected animals or their secretions such as saliva, milk, urine, semen and faeces. Indirect contact with contaminated pens, vehicles, equipment, clothing, and feed can also transmit FMD. The virus can become air-borne and spread over several kilometres under favourable conditions, particularly in cool and humid weather. Recovered animals may become carriers for a long time and serve as the source of new outbreaks.

Clinical Signs

The severity of clinical signs in FMD depends on age and immune status of the host, species affected and strain of the virus. Clinical signs in sheep and goats are generally inapparent or mild in comparison to cattle. Nevertheless, sheep and goat have ability to become carriers (6-9 months) and act as reservoirs of infection which poses major risk of spread of disease especially in FMD-free countries through trade in these animals. The silent nature of FMD in small ruminants has led to several outbreaks in the past. In sheep and goats, the clinical signs mainly include fever, lameness due to the vesicles along the coronary band or interdigital spaces, vesicles on dental pad and mild oral lesions. Vesicles may appear on teats and may also lead to agalactia in milking sheep and goats. FMD has high morbidity, but mortality is usually low in adults. However, mortality is generally higher in young ones and may cause death of young stock without clinical signs.

Lesions

The characteristic lesions for FMD include appearance of vesicles along the coronary band and interdigital cleft. Lesions on the dental pad may also be present but usually remain unnoticed. Lesions are also seen on tongue, oral mucosa, and between the toes and teats. These vesicles rupture leading to secondary infection with bacteria or other pathogens.

Diagnosis

FMD can be initially suspected based on clinical signs. The gold standard for FMD diagnosis is virus isolation in primary cells or cell lines like BHK-21, IBRS-2 and LFBK. FMD and its serotype can be confirmed by Sandwich-ELISA. Reverse transcriptase - polymerase chain reaction (RT-PCR) assay can be used which is also serotype-specific. Reverse transcriptase loop-mediated isothermal amplification (RT-LAMP) can also be used as it is easy, quick and does not require thermal cycler. It is difficult to identify carrier samples though but oropharyngeal fluid as the sample of choice increases the rate of detection. Further, real-time RT-PCR assay can be used as the main test for the detection of carrier animals.

Differential Diagnosis

FMD should be differentiated from vesicular stomatitis, bluetongue, and *peste des petits ruminants*. For lameness, FMD needs to be differentiated from laminitis, hoof abscess, and footrot.

Treatment

No specific treatment is available. However, the external application of antiseptics contributes to the healing of the ulcers and guards off attacks by flies. Rational treatment with broad spectrum antibiotics (β-lactam/streptomycin) to treat secondary bacterial infection is recommended. For management of foot lesions, use antiseptic

solution (potassium permanganate 0.01 percent) or ethnoveterinary formulations. For oral lesion, 1 percent boroglycerine is recommended.

Prevention and Control

FMD can be prevented and controlled with good veterinary services, rapid diagnosis and implementation of control measures that mainly includes mass vaccination. Vaccination programmes should target at least 80 percent of the population to achieve herd immunity. In India, sheep and goat are vaccinated using inactivated trivalent (O, A, Asia 1) vaccine at six-monthly intervals under the flagship scheme National Animal Disease Control Programme (NADCP) funded by Government of India. The protective immune response shall be monitored by routine sero-surveillance.

Biosecurity Measures

Sound biosecurity practices are a must on farms to prevent the introduction and spread of the FMDV into facilities, and further restrict its spread. Various measures include strict movement control of animals, people, and vehicles in and out of affected areas; proper cleaning and disinfection of premises, equipment, and vehicles of affected farms, immediate reporting of the outbreak, if any, to restrain further spread of infection; use of protective clothing and disinfection protocols for visitors and farm workers; and strict regulations on import of animal and animal products.

Sample Collection for Diagnosis

The samples for the FMDV diagnosis include vesicular epithelium or fluid from unruptured vesicles on tongue, teat, and vesicular swabs. The epithelium should be placed in a transport medium of phosphate-buffered saline (PBS) or equal parts glycerol and phosphate buffer and kept refrigerated or transported on ice to diagnostic laboratories. Oropharyngeal fluid can be collected using probang cup or pharyngeal swabbing. Serum samples shall be collected for serological assay. All the samples shall be properly labelled, stored and transported to the laboratories for accurate results.

3.13 Maedi-Visna

Definition and Causative Agent

Maedi-Visna - also known as ovine progressive pneumonia, is a chronic disease in sheep. It is caused by the Maedi-Visna virus (MVV) belonging to the *Lentivirus* genus, which is an enveloped, single-stranded RNA virus within the Retroviridae family. The term 'Maedi' refers to the progressive pneumonia caused by the virus, while Visna denotes the neurologic form of the disease.

Transmission

The virus is primarily transmitted through direct contact with infected animals. It can also spread through the ingestion of colostrum or milk containing viral particles, or by inhaling aerosol droplets from infected animals. Additionally, the virus can persist in the environment for extended periods, spreading it further.

Clinical Signs

The respiratory form of the disease is characterized by a persistent cough, laboured breathing, nasal discharge, and weight loss. The disease progresses slowly, with wasting and increasing respiratory distress being the primary symptoms. In adult sheep, the neurologic form typically manifests as encephalitis, with clinical signs of head tilt and circling. The spinal form presents with unilateral or bilateral deficits in pelvic limb proprioception, which can progress to paresis and eventually complete paralysis.

Lesions

Macroscopic lesions of progressive pneumonia are confined to the lungs and the associated lymph nodes. Histological examination reveals interstitial pneumonia, with perivascular and peribronchial lymphoid hyperplasia and hypertrophy of smooth muscle throughout the lung. Additionally, chronic inflammation and demyelination are observed in the central nervous system, particularly affecting the brain and spinal cord. Lesions may also include encephalitis and various degenerative changes.

Diagnosis

Clinical diagnosis can be primarily based on symptoms and confirmed serologically. Serological testing is useful for detecting infected animals, if the disease is confirmed in the flock through histopathology or virus isolation. Commonly used serological assays are AGID, ELISA, and western blotting. PCR assays and virus isolation are sensitive and specific methods for detecting the virus.

Differential Diagnosis

The disease must be differentiated from pulmonary adenocarcinoma, pleural abscesses, and pulmonary caseous lymphadenitis. Listeriosis, scrapie and cerebrospinal nematodiasis should be considered in the neurologic form of the disease.

Treatment

No specific treatment is available. Supportive treatments include broad-spectrum antibiotics to treat secondary bacterial pneumonia. Antihistaminics, antitussives, and NSAIDs can also be used. Methylcobalamine and glucocorticoids can be added in neurological symptoms.

Prevention and Control

Control and prevention rely solely on serologic testing and the culling of seropositive animals. Newborn lambs should be separated from infected dams at birth and reared in isolation. Owing to the extended incubation period and delayed seroconversion, it may be necessary to retest animals annually or even semi-annually.

Biosecurity Measures

Various biosecurity measures include isolation of infected animals, regular monitoring of flock and controlled access to the farm.

Sample Collection for Diagnosis

Blood samples are collected for serological analysis, while tissue samples from the lungs, brain, spinal cord, and udder (if affected) are obtained for postmortem examination or for PCR analysis. Milk samples are collected to detect viral presence in lactating animals.

3.14 Foot Rot

Definition and Causative Agent

Foot rot, a contagious bacterial disease affecting the epidermis of the interdigital skin and hoof matrix, is caused primarily by *Dichelobacter nodosus*, a gram-negative anaerobe and obligate pathogen. *Fusobacterium necrophorum*, another gram-negative anaerobic bacterium, may play a synergistic role in the disease's pathogenesis. The disease progresses from the interdigital horn to the heel, then to the sole, and finally to the lateral side of the hoof. There are at least 20 strains of *D. nodosus*, each varying in pathogenicity.

Transmission

Foot rot primarily spreads through direct contact with infected animals or contaminated environments. Wet, muddy, or dirty conditions facilitate the proliferation and transmission of the bacteria. Transmission occurs most rapidly in warm-moist conditions.

Clinical Signs

The most obvious clinical sign of foot rot in sheep is lameness, though affected limbs are rarely carried or non-weight-bearing. In sheep with chronic infections, the hoof becomes enlarged and distorted besides swelling and redness of the interdigital skin and a characteristic foul-smelling discharge from the infected hoof.

Lesions

Interdigital dermatitis is the earliest lesion, characterized by mild inflammation and redness of the skin between the toes. Advanced cases show slight detachment of the hoof wall at the junction of the interdigital skin and the hoof. This leads to necrosis of hoof tissue, resulting in separation of hoof horn from underlying structures.

Diagnosis

Diagnosis of foot rot is primarily based on identifying clinical signs. For confirmatory diagnosis, the isolation and identification of *D. nodosus* from interdigital swabs or tissue samples are performed using bacteriological culture, PCR, or ELISA tests. *D. nodosus* is a fastidious organism that requires anaerobic conditions for growth and can be challenging to isolate. Virulent strains can be identified via thermostability testing of the proteases or via real-time PCR assay for virulence genes.

Differential Diagnosis

The disease must be differentiated from foot scald and contagious ovine digital dermatitis, which are lacking the characteristic foul odour of foot rot.

Treatment

Foot rot should be treated with systemic streptopenicillin or tetracycline antibiotics. NSAIDs are recommended for managing lameness, and 5 percent copper sulphate or 10 percent formalin for foot bath.

Prevention and Control

Regular use of footbaths with antiseptic solutions such as zinc sulphate or copper sulphate and routine hoof trimming.

Biosecurity measures

Monitoring and quarantining animals, along with restricting movement, can prevent the introduction and spread of foot rot.

Sample Collection for Diagnosis

Dry or moistened sterile swab samples taken from the interdigital space of all four extremities and tissue samples from the lesions can be used for bacterial culture or pooled for PCR assay.

3.15 Haemorrhagic Septicaemia (HS)

Definition and Causative Agent

Pasteurellosis in sheep and goat is mainly caused by *Pasteurella multocida* and *Mannheimia haemolytica*. *Pasteurella multocida* is mostly isolated from polyarthritis in young lambs whereas *Mannheimia haemolytica* from cases of mastitis, especially in sheep. Both the bacteria cause severe fibrinonecrotic pneumonia in sheep and goats. The disease is characterized by acute onset of illness, high fever, dyspnoea, anorexia, and often death. Death losses are high in severely affected animals. It is an economically important bacterial disease of livestock accounting for high morbidity and mortality in Asia.

Transmission

Various stress factors commonly associated with the transmission and outbreak of HS include high humidity, temperature, malnutrition, and co-infection with other parasites or vial pathogens which act as precipitating factors. Most outbreaks of HS occur in the monsoon. Direct contact with oral/nasal secretions from the clinically infected/healthy carrier animals, and consumption of contaminated water and feed may lead to the transmission of the infection to the susceptible animals.

Clinical signs

Affected animals have fever (40°- 41.1l°C), listlessness, poor appetite, coughing, oronasal discharge and sudden death especially in young ones. Tonsilitis, pneumonia and polyarthritis in young lambs can also be seen. The organism is thought to move from the tonsils to the lungs and pass into the blood. This results in septicaemia and localization of the infection in one or more tissues, such as the joints, udder, meninges, or lungs.

Lesions

Characteristic subcutaneous oedema with blood-stained fluid at the neck, pharynx, and brisket are characteristic postmortem findings. Other lesions include profuse petechial and ecchymotic haemorrhages on the serosal surface of internal organs; prominent haemorrhages in cervical and pharyngeal lymph nodes; sero-sanguinous or sero-fibrinous fluid in pericardial, thoracic, and abdominal cavities; pulmonary congestion with oedema and frothy exudates in the bronchi, trachea, and nasal cavity; and polyarthritis in young lambs.

Diagnosis

Tentative diagnosis may be done from the clinical history of the animal. The confirmatory diagnosis requires the isolation of *Pasteurella multocida* and *Mannheimia haemolytica* from the clinical samples, *viz*., blood, tissues, or bone marrow by *in vitro* cultivation (on casein/sucrose/yeast (CSY) agar containing 5 percent blood as the suitable medium) and biological methods (inoculation of small amount of suspected sample subcutaneously or intramuscularly in mice - death occurs in 24-36 hours and pure culture of *P. multocida* can be seen in a blood smear). Biochemical, serological (rapid slide agglutination test, indirect haemagglutination test, agar gel immunodiffusion test, counter immunoelectrophoresis, agglutination test) and molecular techniques (PCR, real-time PCR, and loop-mediated isothermal amplification (LAMP) assays) can also be used for the identification of the causative agent.

Differential Diagnosis

HS should be differentiated from acute salmonellosis, anthrax, blackleg, lightning strike, snakebite, and non-infectious toxicities.

Treatment

Animals may be treated with sulphadimidine or sulphatrimethoprim @15-30 mg/kg BW IM/IV sid or ceftiofur @ 1-2 mg/kg BW OD IM for 3-5 days. Flunixin meglumine @ 1.1 – 2.2 mg/kg BW IM may be preferred for its anti-endotoxaemic property. Animals with jowl oedema may be treated with

furosemide @ 1-2 mg/kg BW tid IM/SC.

Prevention and Control

Three different types of inactivated vaccine formulations are commonly used for the prevention of HS, *viz*., bacterins, alum-precipitated vaccine, and oil-adjuvanted vaccine. In general, the age of primary vaccination is 4-6 months and repeated yearly at least 15-20 days before the monsoon.

Biosecurity Measures

Outbreak of the disease should be notified to the concerned authorities, and animal movement should be strictly controlled in the affected area to check disease spread. Early detection of the disease and effective surveillance are necessary for its proper control during an outbreak. Regular monitoring of the animals, proper cleaning and disinfection of the animal sheds, equipment, and vehicles, and following all other biosecurity measures are the best practices to be adopted in an HS outbreak situation.

Sample Collection for Diagnosis

Samples from live animals: Blood samples collected aseptically from moribund animals are preferable for the diagnosis of HS, as septicaemia peaks at the terminal stage of the disease. Blood samples collected from animals undergoing antimicrobial therapy are not suitable for the diagnosis.

Samples from dead animals: A blood sample or swab collected from the heart immediately after the death is suitable for the isolation and identification of both *P. multocida* and *M. haemolytica.* Bone marrow from long bones is a sample of choice for bacterial isolation if the animal has been dead for a long period. In case of non-availability of a postmortem facility, blood may be aspirated or collected by incision from the jugular vein aseptically. All samples should be kept in suitable transport medium, tightly packed, and transported on ice. Samples may be frozen (stored in a refrigerator), if there is no facility to transport to the laboratory within a few hours.

3.16 Brucellosis

Definition and Causative Agent

Brucellosis, one of the most prevalent zoonoses affecting animals and humans, is causing significant socio-economic and trade losses. The causative agents in sheep (*B. ovis*) and goat (*B. melitensis)* are Gram- negative, aerobic, facultative intracellular, non-motile, non-spore-forming, partially acid-fast microorganisms. These infections cause abortion and lead to huge economic losses.

Transmission

Transmission of brucellosis in sheep and goats occurs mainly through ingestion of contaminated food and water, infected semen and tail splashing of urine leading to aerosolization of bacteria and subsequent transmission through conjunctival route.

Clinical signs

The clinical signs of brucellosis in sheep and goat are similar to those in cattle. The reproductive system is most commonly affected leading to abortion in the third trimester along with retention of placenta. Birth of stillborn or weak calves can also occur. Brucella organisms localise in the supramammary lymph nodes and mammary glands, and the affected animals secrete pathogens in milk throughout their life. The disease is associated with decrease in average milk yield in infected animals. Arthritis, epididymitis, orchitis and impaired fertility are also some other key observations in this disease.

Lesions

Granulomatous inflammatory lesions in the reproductive tract, udder, supramammary lymph nodes, and joints and synovial membranes; mild to severe endometritis may be seen after an abortion with thickened and oedematous placenta; the intercotyledonary region is leathery, with a wet appearance and focal thickening.

Diagnosis

Isolation and identification of the agent (staining of bacterial smears, culture and identification by biochemical tests); PCR assays for species identification; Rose Bengal plate test (RBPT) as screening test; serological assays like ELISAs (indirect-ELISA and competitive ELISA) amenable for mass-scale serosurveys; and molecular typing methods like multilocus sequence typing (MLST) and multilocus variable number of tandem repeats analysis (MLVA).

Differential Diagnosis

The disease should be differentiated from chlamydiosis (enzootic abortion), coxiellosis, toxoplasmosis, and listeriosis.

Treatment

Detection of positive animals and subsequent elimination from the herd is the best option because no practical treatment option is available.

Prevention and Control

Vaccination of young (3- to 8-month-old) sheep and goats with the Rev. 1 strain SC or intra-conjunctival routes is common. Good management practices like prompt reporting of abortion cases to the nearest dispensary; cleaning of barn with good disinfectants; segregation of infected animal; proper disposal of aborted foetus. Personal protection using gloves and masks during handling of infected animals and aborted material. Education and awareness of public is crucial in Brucella prevention and control.

Biosecurity Measures

Proper biosecurity measures should be well-aligned with good farm management practices. Regular herd screening for brucellosis is very important. Positive-tested animals must be removed from the herd as per test-and-segregate policy. Careful selection of replacement animals with inspection of health certificate and observing proper quarantine procedures are important. Properly screen the breeding stock including breeding males for Brucella-negativity for use in natural service and/or use tested Brucella-free semen for artificial insemination to avoid sexual transmission. Surveillance programmes to control the disease in the small ruminants should be carried out. Proper disposal of infected/contaminated materials [like aborted foetus (stomach content, spleen, and lung), foetal membranes, lochial discharge, colostrum, milk] and disinfection of the infected premises till the lochial discharge ceases may effectively control the disease.

Sample Collection for Diagnosis

Samples from aborted foetus (stomach content, spleen, and lung), foetal membranes, lochial discharge, colostrum, milk as well as fluid collected from arthritis or hygroma are good for diagnostic works. Blood/serum sample/FTA Card is good for routine screening.

Public Health Risk

Humans contract brucellosis through direct contact with infected sheep and goat, tissues, or fluids, particularly during kidding or lambing, handling, or slaughter. Ingesting unpasteurized dairy products (milk, cheese) or undercooked meat from infected animals can lead to human brucellosis. Farmers, veterinarians, abattoir workers, and dairy handlers are at higher risk.

Do's

- Segregate the infected animal
- Maintain cleanliness in animal barn
- Drink pasteurized/boiled milk only
- Vaccination of sheep and goats with appropriate vaccine

Don'ts

- Do not handle infected animals and aborted material without gloves
- Do not drink raw milk and do not eat uncooked meat

3.17 Tuberculosis

Definition and Causative Agent

Tuberculosis (TB) is an infectious granulomatous, chronic debilitating disease of animals and humans caused by *Mycobacterium tuberculosis* complex. Sheep are susceptible to infection with *Mycobacterium bovis* and *Mycobacterium caprae*, but they rarely show signs of disease. Lesions are usually restricted to the respiratory tract; however, generalization is possible. Goats are highly susceptible to infection with *Mycobacterium tuberculosis* complex, particularly *M. caprae* but also *M. bovis* and *M. microti*. *M. avium* complex also infects goats. Infection is widespread and herd prevalence can be high. *M. bovis* and *M. caprae* are the main pathogens associated with sheep and goat. Bacteria are acid-fast, gram-positive rods belonging to the family Mycobacteriaceae. Sheep are susceptible to infection, but they rarely show signs of disease, goats are highly susceptible to infection leading to high herd prevalence.

Transmission

The infection spreads through inhalation of infected aerosol during close contact, ingestion of bacteria directly from infected animals, or indirectly from contaminated pastures and water. Newborns become infected by ingesting colostrum or milk from infected animals.

Clinical Signs

TB should be considered in cases of chronic loss

of condition and appetite, reduced milk yield and debilitating disease, with or without respiratory signs. A chronic cough can be a sign of TB in goats, and should in particular be considered when a goat has failed to respond to antibiotic treatment for a respiratory infection.

Lesions

Cases of TB in goats are usually identified at post-slaughter inspection or at post-mortem examination in a veterinary laboratory. The lungs and respiratory lymph nodes are most frequently affected. Lung lesions are usually white or creamy and contain white or creamy semi-liquid pus. TB lesions in sheep are primarily reported in the lungs and thoracic lymph nodes. Lesions are creamy, yellow or green and may be calcified (gritty). Lesions are usually restricted to the respiratory tract; however, generalization is possible.

Diagnosis

Diagnostic assays used in cattle can be applied to sheep and goat. The diagnostic tests used for isolation and identification of the bacteria are Ziehl Neelsen staining, PCR assays for species identification, tuberculin skin test (delayed hypersensitivity test) - caudal fold test (CFT), the single cervical intradermal test (CIT) and the comparative cervical test (CCT); gamma-interferon (IFN- γ) release assays; ELISA; and molecular typing of isolates for strain identification by REA, RFLP, VNTR, *etc*. Goats must be 12-month-old or older for an official TB test, a caudal fold test. As a reminder, the TB test for goats requires 0.1 ml PPD Bovis intradermally in the caudal fold. Inspect visually and palpate at 72 hours.

Differential Diagnosis

The disease should be differentiated with contagious caprine pleuropneumonia, aspiration pneumonia, caseous lymphadenitis, traumatic pericarditis, and chronic aberrant liver fluke infestation.

Treatment

Test and segregation are the best practical approach to control the infection. Treatment with isoniazid, ethambutol and rifampicin has limited efficacy in animals but uneconomical.

Prevention and Control

Test-and-segregation strategies; disease reporting; farm sanitation and disinfection, open air housing rather than confinement, and avoidance of crowding; if an infected herd is found, the reactors are removed, and the herd is isolated and quarantined until all animals test negative; isolation of sick and weak animals showing marked symptoms; tuberculin testing: segregation of tuberculin positive animals; abattoir surveillance.

Biosecurity Measures

Routine hygienic measures aimed at cleaning and disinfecting contaminated premises, as well as strict biosecurity, are useful. Proper and hygienic disposal of waste; wildlife barriers around feed storage areas; biosecurity measures on farms to decrease interactions between wildlife and domesticated animals.

Sample Collection for Diagnosis

Whole blood, serum, nasopharyngeal swabs, lymph node aspirates, tissue samples from lungs, liver, spleen, or during necropsy.

Public Health Risk

Controlling TB in small ruminants is crucial to protect human health, particularly in rural and agricultural communities. Public health measures, including pasteurization of milk, animal testing, and culling of infected animals, are essential to mitigate this risk.

Do's

- Always use pasteurised/boiled milk
- Segregate sick animals from healthy ones
- Maintain hygiene in animal barn
- Avoid overcrowding of animals
- Visit Doctor if cough persist for a longer period

Don'ts

- Do not make curd, paneer and cream from raw milk
- Do not skip medications, if diagnosed with TB

3.18 Paratuberculosis

Definition and Causative Agent

Paratuberculosis (Johne's disease) is a chronic, infectious, granulomatous enteritis of ruminants clinically characterized by chronic diarrhoea and progressive emaciation. The disease is caused by *Mycobacterium avium* subsp. *paratuberculosis* (MAP), a fastidious, non-spore forming, acid-

fast, gram-positive rod belonging to the genus *Mycobacterium*.

Transmission

Infected animals shed the bacterium in manure, colostrum, and milk. Primary source of infection is faeces from infected sheep and goat or milk contaminated with the faeces of diseased animals. It can also be transmitted from an infected pregnant animal to its foetus.

Clinical Signs

It has a long incubation period; hence infected animals may excrete organisms in the faeces for 15-18 months before clinical signs appear. Emaciation is the most noticeable sign, accompanied by shedding of wool in advance stage. Though in cattle, diarrhoea may be constant or intermittent; in sheep, goats, and other ruminants, diarrhoea may not be seen. This leads to low concentrations of total protein and albumin in plasma, although gamma globulin levels are normal.

Lesions

Carcasses may be emaciated, with loss of pericardial and perirenal fat in more advanced cachectic cases. Sheep and goats, sometimes, develop foci of caseation with calcification in the intestinal wall and lymph nodes. The terminal part of the ileum is the most affected and shows specific lesions like thickening, sometimes oedematous, along with presence of corrugations on mucosal surface, which does not disappear when the intestinal wall is stretched.

Diagnosis

Ziehl Neelsen staining of faecal smears or intestinal mucosa; faecal culture- gold standard for the diagnosis of paratuberculosis in live animals; serological tests - ELISA (widely used), complement fixation test (CFT), agar gel immunodiffusion test (AGID); intradermal Johnin test (DTH).,

Differential Diagnosis

Intestinal TB, caseous lymphadenitis, parasitic gastroenteritis.

Treatment

Treatment with streptomycin, isoniazid, clofazimine and monensin has limited efficacy in animals. Test and segregation or slaughter is the best practical approach to control the infection in sheep and goats.

Prevention and Control

Keep young ones separated from older animals to reduce the risk of MAP transmission. Use screening tests for newly introduced animals to identify and segregate infected animals. Ensure kids or lambs are housed in clean well-bedded areas. Kidding/lambing pens to be thoroughly cleaned and disinfected. The dam's teats must be as clean as possible to prevent ingestion of faecal matter.

Biosecurity Measures

Biosecurity measures include implementation of good sanitation and management practices, reduction of faecal contamination in animal housing areas, regular cleaning and disinfection of feeding and watering equipment, using colostrum from sheep and goats that test negative for Johne's disease, and avoiding pooling of colostrum from multiple animals.

Sample Collection for Diagnosis

Antemortem samples: Faecal samples, blood, rectal mucosal scraping.

Postmortem samples: Intestinal tissue mainly from ileo-caecal junction, ileo-caecal lymph nodes, faecal sample.

Public Health Risk

Paratuberculosis is not a major zoonosis. MAP organism is occasionally isolated from patients with Crohn's disease, which is a chronic, painful, diarrhoeal inflammatory disease of the intestinal tract in humans resembling Johne's disease.

3.19 Colibacillosis

Definition and Causative Agent

Colibacillosis is considered as one of the most common bacterial infections causing serious economic losses to the small ruminant sector. It is commonly manifested in intensive farming systems, mostly affecting kids and lambs of less than 2-week-old. Certain virulent pathogenic serotypes of *Escherichia coli* [enterotoxigenic *E. coli* (ETEC), enteropathogenic *E. coli* (EPEC), enterohemorrhagic *E. coli* (EHEC) and necrotoxigenic *E. coli* (NTEC)] are responsible for the clinical outcome of colibacillosis. Age, low birth weight and deficiency of immunoglobulins are the predetermining

factors for the disease in newborns. Invasion into the blood stream leads to the condition called as colisepticaemia.

Transmission

The organism is shed in the nasal discharge, saliva, urine and faeces of bacteraemic lambs. Transmission occurs via direct contact with the infective materials or the contaminated environment. Invasion of the organism occurs through nasal, oro-pharyngeal or intestinal mucosa and sometime through umbilicus.

Clinical signs

There are two clinical forms of the disease:

Enteric colibacillosis: Most common form of colibacillosis in newborn animals. Diarrhoea is the most striking manifestation of enteric colibacillosis. Frequent and effortless defecation, faeces appear mucoid or haemorrhagic, pasty or fluidy, foul smelling, containing partially digested milk and is grey or yellowish white in colour. Severe diarrhoea with dehydration and weight loss occurs in complicated cases.

Coliform septicaemia: Kids and lambs of 1 to 2 day and 3 to 8 weeks old are mostly susceptible to septicaemia. In the per acute and acute cases of colisepticaemia, septic shock may occur. Other signs may be listlessness, inappetence, depression, collapse, loss of sucking reflex, poor response to external stimuli, recumbency, subnormal rectal temperature, cold extremities, stupor and coma. In the prolonged course of bacteraemia, localized infection is seen resulting in omphalitis, polyarthritis, and meningitis. Per acute septicaemic cases are characterized by sudden death without evident clinical signs. Stiff gait, hyperaesthesia, recumbency, tetanic convulsions and collapse are seen in acute cases while chronic cases exhibit arthritis.

Lesions

Enteric colibacillosis: Lesions include gross dehydration of the carcass with fluid-filled flaccid intestines and abomasum containing undigested milk clots, petechial haemorrhages in abomasal mucosa, hyperaemic intestinal mucosa, atrophy of ileal and jejunal villi, and oedema of the mesenteric lymph nodes.

Histopathology reveals the presence of adherent bacteria on the brush border epithelia of enterocytes, erosion of enterocytes and atrophic ileal villi. Pseudomembranous ileitis and muco-haemorrhagic lesions in the large intestine may be present in EPEC infections.

Coliform septicaemia: Gross lesions are not much evident. Submucosal haemorrhages, subserosal haemorrhages, enteritis and gastritis may be present in mild infections. Fibrinous exudates in the serous cavities and joints, pneumonia, omphalophlebitis and meningitis may be present infrequently. The histopathological findings are the same as of septicaemia and toxaemia.

Diagnosis

Primary diagnosis is based on the history and clinical signs. Isolation and identification of *E. coli* from the intestines and the faeces confirms enteric colibacillosis. Detection of enterotoxin by ligated intestinal loop test or infant mouse test and the histopathology of the intestinal mucosa can complement the diagnosis. Other tests used include EIA and LAT for the presence of toxins, direct FAT and ELISA to detect K99+ ETEC, and PCRs using primers specific for pathotypes, enterotoxin and adhesins genes to characterize the isolates of *E. coli.*

Differential diagnosis

Colibacillosis in lambs and kids should be differentiated from neonatal diarrhoea caused by coronavirus, rotavirus, *Cryptosporidium* spp., and *Clostridium perfringens* type C; as well as septicaemia caused by *Salmonella* spp., *Listeria monocytogenes* and *Erysipelothrix insidiosa*.

Treatment

Fluids and electrolyte therapy according to the level of dehydration. Sulphatrimethoprim @15-30 mg/kg BW IM/IV sid for 3-5 days or ampicillin @ 5-10 mg/kg BW IM/IV bid for 3-5 days. Supportive therapy includes B-complex with antihistamine.

Prevention and Control

Various interventions include timely feeding of adequate quantity of colostrum to neonates, use of probiotics, good farm management practices, avoid exposing the umbilical cord to the environment to prevent infection, vaccination of the newborn, and vaccination of the pregnant dam to improve the specific immunity of neonates to infection.

Biosecurity Measures

Biosecurity measures include ensuring proper

hygienic measures and managemental facilities where neonates are housed, personnel handling the neonates taking utmost care to avoid infection to the animal facility, facilitating parturition in a hygienic environment, and following aseptic precautions while cutting of umbilical cord.

Sample Collection for Diagnosis

In coliform septicaemia: blood, liver, spleen, lung, umbilicus and meninges may be collected and transported for isolation of *E. coli*. Samples to be collected aseptically for enteric colibacillosis include segments of colon and ileum along with the contents, duodenum, jejunum and mesenteric lymph nodes.

3.20 Anthrax

Definition and Causative Agent

Anthrax is a rapidly progressing, per acute and acute septicaemic disease caused by *Bacillus anthracis*. *Anthrax bacilli* is a gram-positive, aerobic, or facultative anaerobic, non-motile, non-haemolytic, spore-forming, rod-shaped bacteria and develops capsule inside the body of the host. The per acute form (common in cattle and sheep) is characterized by sudden onset and a rapidly fatal course.

Transmission

Ingestion of contaminated fodder, water, and animal products (bone meal, fertilizers); spore inhalation during wallowing in contaminated water sources; mechanical transmission by biting flies (*e.g.*, *Hippobosca* spp., *Tabanus* spp.);

Clinical signs

In sheep and goats; staggering, dyspnea, trembling, collapse, a few convulsive movements, and death after only a brief period of illness may occur. Exudation of tarry unclotted blood occurs from natural orifices. Rigor mortis is absent, and carcass appears bloaty.

Lesions

Upon necropsy, spleen is enlarged having a dark semi-fluid pulp blackberry jam-like consistency and poorly clotted blood is seen. Haemorrhage from the nose, mouth, vagina and/or anus may be found at death.

Diagnosis

Isolation and identification of the bacteria is considered as gold standard. *B. anthracis* may be identified in a stained smear from the live animals by Gram-stain or polychrome methylene blue through capsule visualization by MacFadyen's reaction wherein blue rods in a background of purple/pink-stained capsule are seen, immunofluorescence, and PCR for detection of protective antigen (PA).

Differential diagnosis

Clostridium infection, bloat, and lightning strike need to be differentiated for anthrax.

Treatment

Animals should be treated with long-acting penicillin such as benzathine penicillin @ 22,000IU/kg BW IM or alternatively with long acting oxytetracycline @ 5-10 mg/kg BW IV.

Prevention and Control

Globally, Anthrax Sterne strain 34-F2 vaccine - a live attenuated non-capsulating vaccine - is used for vaccination of animals. During an outbreak, do not conduct postmortem of suspected dead animals, plug orifices of dead animals with cotton soaked in lysol, safely dispose the carcass as per the guidelines, disinfect the site of the dead animal with lysol or 3–5% formaldehyde, disinfect slaughter sites, processing factories and retail outlets as per the guidelines.

Biosecurity Measures

The disease should be notified to the appropriate regulatory officials when outbreak occurs. Other measures include rigid enforcement of quarantine for imported animals; prompt disposal of dead animals, faeces, bedding, or other contaminated material by cremation or deep burial; isolation of sick animals and removal of healthy animals from the contaminated areas; as well as cleaning and disinfection of animal sheds, pens, milking barns, and equipment used on production animals.

Sample Collection for Diagnosis

Peripheral blood and tissue samples are collected for diagnosis. Samples should be collected carefully to avoid contamination of the environment and to prevent human exposure to bacteria.

Public Health Risk

In humans, anthrax manifests in three distinct patterns (cutaneous, gastrointestinal and inhalational). More than 95 percent of human

anthrax cases are of the cutaneous form and result from handling infected carcasses or the hides, hair, meat, or bones from such carcasses.

Do's

- Prompt treatment of sick animals
- Proper carcass disposal
- Maintain hygiene in animal barn

Don'ts

- Do not open suspected carcass
- Do not handle animals without protective clothing including full personal protective equipment (PPE)
- Do not eat raw or improperly cooked meat.

3.21 Listeriosis

Definition and Causative Agent

Listeriosis, a food-borne illness, is caused by member of the genus *Listeria*. It is a gram-positive bacterium belonging to family Listeriaceae. *L. monocytogenes* is the primary pathogen associated with human and animal listeriosis. It is an intracellular, non-sporulating, rod-shaped facultative anaerobic microorganism. The disease course in sheep and goats is rapid, and death may occur between 24 and 48 hours after onset of clinical signs.

Transmission

Listeriosis mainly occurs from the ingestion of contaminated feed and water particularly by eating contaminated silage having pH >5.0-5.5.

Clinical Signs

Rhombencephalitis - also referred to as circling disease - is the most common manifestation of disease in sheep and goats in which circular movement of the diseased animals occurs with head turned or twisted to one side. Other symptoms include dullness, unilateral facial nerve paralysis causing dropping of the eyelid and ear, and the drooling of saliva because of partial pharyngeal paralysis. In goats, recumbency and death occur within 2 or 3 days. Septicaemic or visceral listeriosis is found in young ruminants before the rumen is functional. Listeria abortion usually occurs in the last trimester without prior clinical signs. Foetuses usually die *in utero*, but stillbirths and neonatal deaths also occur.

Lesions

In the encephalitic form, the cerebrospinal fluid may be cloudy and the meningeal vessels congested. Gross lesions are characterised by vascular congestion. In the septicaemic form, multiple foci of necrosis in the liver are evident. Aborted foetuses of ruminants show very few gross lesions, but autolysis may be present if the foetus was retained before being expelled.

Diagnosis

Isolation and identification of agent (cold enrichment method for isolation from brain tissue); ALOA (Agar Listeria according to Ottaviani and Agosti), a selective chromogenic medium for detecting PI-PLC (phosphatidylinositol-specific phospholipase C); Christie–Atkins–Munch–Peterson (CAMP) test for identifying *Listeria* spp.; PCR assays; subtyping using pulsed-field gel electrophoresis (PFGE); nucleic acid sequence-based typing using multi locus sequence typing (MLST); and loop-mediated isothermal amplification (LAMP).

Differential Diagnosis

The disease should be differentiated from rabies, chlamydiosis (enzootic abortion), coxiellosis, and toxoplasmosis.

Treatment

Penicillin G @ 22,000-44,000 IU/kg BW IM for 1-2 weeks is the drug of choice for listeriosis. Alternatively, ceftiofur may be given @ 1.1-2.2 mg/kg BW bid IM for 5-7 days. Mannitol @ 1-2 mg /kg BW IV in encephalitis

Prevention and Control

Feed only good quality silage to prevent listeriosis in sheep and goats. Other measures include discarding any spoiled or mouldy silage, avoiding feeding of the top few inches of silage that are exposed to air, keeping the animals away from rotting vegetation to minimize exposure to harmful organisms and reduce the risk of faecal contamination of feed, removing any feed source known to be or suspected for listeriosis outbreak, investigating the cases promptly, isolating sick animals, and removing and destroying the sources of contamination like placenta and foetus from infected animals.

Biosecurity Measures

Standard operating procedures (SOPs) should be framed to target minimizing the pathogen(s) load in the environment and manage the pathogen risks within the environment at the farms, and food processing plants/facilities, and ensure a cleaner farm environment. Other measures include cleaning and disinfecting the animal sheds regularly to prevent disease transmission, storing animal feeds in conditions that inhibit microbial growth to prevent contamination by *L. monocytogenes*, restricting the entry of wild and stray animals to the farm and feed storage areas, and operationalizing the plans for proper implementation and monitoring of the personal hygiene of workers of the food processing units.

Sample Collection for Diagnosis

Brain tissues, cerebrospinal fluid (CSF), aborted foetus, placenta, blood, joint fluid, amniotic fluid, uterine swabs, and milk.

Public Health Risk

Humans develops fatal meningitis, sepsis, and papular exanthema on the arms after handling aborted material. In pregnant women, infection may result in abortion, stillbirth or premature birth. Therefore, all material from suspected clinical cases of listeriosis should be handled carefully.

Do's

- Proper disposal of aborted materials
- Cleaning and disinfection of farm equipment
- Maintain cleanliness in the housing areas for cattle

Don'ts

- Do not consume unpasteurized milk and raw milk products
- Do not handle aborted foetuses without PPEs.

Sample collection for diagnosis

Placenta, vaginal discharges and tissues of aborted foetuses (spleen, liver, lung or stomach content). For investigation of bacterial shedding, samples can be taken from vagina, milk and colostrum.

3.22 Q-fever

Definition and Causative Agent

Coxiellosis, also known as Q-fever, is a zoonotic bacterial infection associated primarily with parturient ruminants. Coxiellosis is one of the 13 global priority zoonoses and is classified as a potential bioterror weapon under Category 'B' of the CDC. Q-fever is a bacterial disease caused by *Coxiella burnetii* which is a Gram-negative obligate intracellular bacterium. The organism displays different morphological forms in its developmental cycle and some forms can survive extracellularly and even accumulate in the environment.

Transmission

Contaminated aerosols from drying of infected placentas and body fluids and/or dust from contaminated manure act as significant sources of infection. Reactivation of the bacterium during pregnancy results in the shedding of a high number of infectious agents into the environment during abortion or via birth fluids, placenta and foetal membranes. Ticks can also be involved in Q-fever transmission.

Clinical signs

Infected animals show no symptoms of the disease until abortion in late pregnancy. The disease may occasionally be a cause of stillbirths, and birth of weak offspring. The placenta, foetuses, and uterine fluids from infected animals contain high numbers of infective bacteria. Some animals show depression and lack of appetite one to two days prior to abortion.

Lesions

Necrotizing placentitis, intercotyledonary fibrous thickening, and endometritis are some of the notable changes observed in this disease.

Diagnosis

Isolation and identification of the bacteria; staining techniques using Stamp, Gimenez, Macchiavello, Giemsa and modified Koster methods; serological testing using the ELISA; immunofluorescence assay – a Gold standard; and PCR assays.

Differential Diagnosis

Infectious and non-infectious agents that cause abortion should be differentiated from this disease.

Treatment

Ruminants may be treated with oxytetracycline @ 5-10 mg/kg BW bid IM/IV. At the known infected farm premises, pregnant animals may be treated

with oxytetracycline @ 5-10 mg/kg BW bid IM/IV.

Prevention and Control

Measures include control of tick vector to prevent outbreaks, testing of abortion cases, reducing the exposure to raw milk, consumption of only pasteurised milk and milk products, and educating public on sources of infection.

Biosecurity Measures

Biosecurity measures include destruction and disposal of the placenta and dead foetus to reduce environment contamination, preventing the access to contaminated biorisk materials to domestic or wild carnivores to prevent dissemination of the pathogen, manure management to prevent aerosolization, separating the identified infected herds from healthy animals, and quarantine the newly introduced animals before introducing them to the main herd.

Sample Collection for Diagnosis

Placenta, vaginal discharges, and tissues of aborted foetuses (spleen, liver, lung or stomach content). For investigation of bacterial shedding, samples can be taken from vagina, milk and colostrum.

Public Health Risk

Q-fever is an important zoonosis which impacts veterinarians, laboratory workers, farmers and abattoir workers. Farmers and veterinarians are at risk while assisting parturition whereas slaughterhouse workers are at risk from contact with infected carcasses. Human transmission occurs by inhalation of infectious aerosols.

Do's

- Handle animal tissue with care
- Wear protective clothing to prevent tick bites
- Wear disposable gloves and sleeves while handling animal parturition

Don'ts

- Do not consume unpasteurized milk and milk products
- Do not touch newborn animal or birthing products (placenta, birth fluids) from an infected animal
- Do not get exposed to urine, milk, or blood from an infected animal.

3.23 Enzootic Abortion of Ewes

Definition and Causative Agent

Enzootic abortion of ewes (EAE), also referred to as ovine chlamydiosis or ovine enzootic abortion (OEA), is caused by the bacterium *Chlamydophila abortus,* which is a gram-negative, obligate intracellular bacteria. The condition typically occurs in the last 2–3 weeks of pregnancy, leading to stillborn lambs and inflamed placentas. Infection is typically introduced through infected replacements. Initially, there are only a few abortions in the first year. However, in the second year, an abortion storm can affect up to approximately 30 percent of ewes.

Transmission

Ewes become infected via the oro-nasal route, often by sniffing around areas where other ewes have lambed. Lambs fostered onto an aborted ewe can become infected by coming into contact with vaginal discharge on the udder and wool. The bacteria can survive on pasture for several days during spring, contributing to transmission.

Clinical Signs

Abortion, stillbirth and vulvar discharge are the most common signs of EAE. Infected ewes typically show fever, depression, and lack of appetite for about 24 hours before abortion. Lambs born from infected ewes may be weak and unable to suckle. Ewes infected with EAE may exhibit a red/brown vulval discharge that stains the wool around the tail and perineum, which is highly characteristic of EAE. Infected ewes remain latent carriers of the infection, until the microorganism reactivates in the next pregnancy.

Lesions

EAE leads to inflamed placentas in affected ewes. Gross lesions may include necrotic, reddish-brown cotyledons. The intercotyledonary areas become thickened and brown, often covered by exudate. Gross lesions in the foetus are rare but may include ascites (fluid accumulation in the abdomen), lymphadenopathy (enlarged lymph nodes), and liver congestion.

Diagnosis

Aborted placental contents and vaginal discharge are the sample of choice for diagnosis of EAE. Examination of placental/vaginal smears stained

with modified Ziehl Neelsen stains combined with PCR/real-time PCR is the suggested method for confirmatory diagnosis of EAE. *Chlamydophila abortus* can be isolated in embryonated chicken eggs or in cell culture, the latter being the method of choice for isolation of new strains. The *Chlamydophila abortus* is zoonotic and thus isolation and identification procedures should be carried out under biosafety level-2 conditions.

Differential Diagnosis

The disease must be differentiated from other causes of abortion in small ruminants that include toxoplasmosis, leptospirosis, brucellosis, campylobacteriosis, listeriosis and Q fever.

Treatment

Ewes can be treated with long acting oxytetracycline @ 5- 10 mg /kg BW IM.

Prevention and control

It is suggested to immediately isolate the affected ewes, dispose-of infected materials, clean and disinfect housing and avoid fostering. Subsequently, serology may be used to confirm the EAE in the herd and specific control measures must be implemented. Currently, inactivated and attenuated live vaccines are available for EAE in sheep.

Biosecurity Measures

Maintain a closed flock and restrict the access to the farm.

Sample collection for diagnosis

Freshly aborted foetus and placenta are the sample of choice for laboratory diagnosis. Serum samples from aborted ewes collected between 3 weeks to 3 months after abortion can be tested for *Chlamydophila abortus* specific antibodies.

3.24 Contagious Agalactia

Definition and Causative Agent

Contagious agalactia is a disease syndrome of sheep and goats that is characterised by mastitis, arthritis, keratoconjunctivitis and occasionally abortion. *Mycoplasma agalactiae* is the main cause of the disease in sheep and goats, but *M. capricolum* subsp. *capricolum*, *M. mycoides* subsp. *capri* and *M. putrefaciens* produce a clinically similar disease, more often in goats, which may be accompanied by pneumonia. *M. agalactiae* and *M. capricolum* subsp. *capricolum* have also been isolated from wild small ruminants such as ibex and mountain goats.

Transmission

The bacteria are transmitted through direct contact with the infected animal and indirect contact with fomites and contaminated feed, water, surface or equipment. The main sources of infection include ocular and nasal secretions, faeces, milk, urine, and excretions from joint lesions. Venereal transmission through sexual contact and transmission to young ones through contaminated colostrum or milk is also reported. Some animals may become asymptomatic carriers of the bacteria, shedding it intermittently and serving as a reservoir of infection within a herd or flock.

Clinical Signs

The disease caused by *M. agalactiae* is recognised by elevated temperature, inappetence and alteration in the consistency of the milk in lactating ewes with decline and subsequent failure of milk production, often within 2– 3 days, because of interstitial mastitis. After several days, the affected udder shrinks because of damage to the secretory tissue. Abscesses within the udder and enlargement of the retromammary lymph nodes may also be seen. Lameness due to unilateral or bilateral arthritis and keratoconjunctivitis affects about 5-10 percent of infected animals. Fever is common in acute cases and may be accompanied by nervous signs, but these are rare in the more frequently observed subacute and chronic infections where the disease is endemic. Pregnant animals may abort due to bloodstream infection. *M. agalactiae* may occasionally be found in lung lesions, but pneumonia is not a consistent finding. Other three species (*M. capricolum* subsp. *capricolum*, *M. mycoides* subsp. *capri* and *M. putrefaciens*) also produce similar clinical signs in affected animals, which include agalactia, abortions, arthritis and keratoconjunctivitis.

Lesions

The infected udder is grossly atrophic in one or both the sides. Microscopically, the chronic inflammatory reaction in the stroma shows increased fibrosis and a reduced number of glandular acini. Infected joints exhibit swelling of the joint capsules, with the synovial lining containing clusters of fibrins. Joint surfaces may show erosion and occasionally become

ankylosed. In early stages of keratitis, the cornea becomes swollen and infiltrated with white blood cells; later, there is a substantial buildup of pus in both the cornea and the ciliary body. Colonization of nervous tissue is well documented, although clinical signs are rarely reported.

Diagnosis

Definitive diagnosis requires the isolation of the causative mycoplasmas from the affected animals, which are identified by biochemical, serological and, molecular tests such as the 16S rRNA-based PCR. Though detection of antibodies in serum by ELISA provides rapid diagnosis of disease, confirmation of infection by isolation and identification or detection by PCR is usually necessary.

Differential Diagnosis

The disease syndrome must be differentiated from bacterial and fungal mastitis; septic arthritis caused by *Staphylococcus aureus*, *Streptococcus* spp., and *Escherichia coli*; traumatic or immune-mediated arthritis; infectious conjunctivitis associated with *Chlamydia* spp., *Moraxella* spp., and *Staphylococcus* spp.; contagious caprine pleuropneumonia (CCPP), and the abortions caused by other agents like *Chlamydia abortus, Toxoplasma gondii*, and *Brucella melitensis*, *etc.*

Treatment

Fluroquinolones may be preferred over penicillin and oxytetracycline. Treatment with erythromycin and tylosin may destroy the mammary parenchyma in small ruminants.

Prevention and Control

Infection can be prevented by adopting good managemental practices and following continuous surveillance/monitoring for the pathogen. Strict quarantine and herd screening should be followed to detect the carrier animals. Proper disposal of litter and other materials, including discharges and aborted foetus, and proper sterilization of contaminated utensils are recommended. Use of disinfectants as hypochloric acid, formalin, cresols, and phenolic substances along with commonly used quaternary ammonium compounds is effective against the organism. Proper screening of the semen for artificial insemination and bucks to be used should be conducted on regular basis. In endemic areas, vaccination with nationally approved vaccine(s) is effectively applied throughout world.

Biosecurity measures

Implement strict biosecurity protocols to prevent introduction and spread of the disease. This includes quarantine and testing of new animals before introduction to the herd or flock. Control access of people, vehicles, and equipment to farm areas to minimize the risk of introducing pathogens.

Sample Collection for Diagnosis

Preferred samples for diagnosis include milk, nasal swabs and secretions, joint fluid from arthritic cases, conjunctival swabs from cases of ocular disease, blood and serum for antibody detection. The sampling of bulk milk tank provides a convenient way of monitoring flocks and herds for causative mycoplasmas. From dead animals, samples should include udder and associated lymph nodes, joint fluid, lung tissue (at the interface between diseased and healthy tissue) and pleural/pericardial fluid.

3.25 Caseous Lymphadenitis

Definition and Causative Agent

Caseous lymphadenitis (CL) is a chronic infectious disease caused by the gram-positive bacterium *Corynebacterium pseudotuberculosis*. It is characterized by the formation of abscesses either in or near major peripheral lymph nodes (external form) or within internal organs and lymph nodes (internal form). Although, both the forms of CL occur in both sheep and goats, the external form is more common in goats, and the internal form is more common in sheep (thin ewe syndrome).

Transmission

CL spreads through direct contact of healthy animals with the infected ones having draining lesions or through indirect contact with contaminated feed, water, premises, beddings, *etc.* Once established on a farm, *C. pseudotuberculosis* can survive up to 2 months on fomites such as beddings and 8 months in soil. As it is a zoonotic pathogen, care should be taken when handling infected animals or purulent exudates from active draining lesions.

Clinical Signs

The clinical signs of CL vary with the external or internal form of the disease. Infected animals may have an elevated body temperature and decreased milk production. The typical clinical signs associated

with external form include painless firm nodules or swellings near submandibular (under the jaw), parotid (near the ear), prescapular (in front of the shoulder), and prefemoral (in the thigh) lymph nodes. Abscesses may progress to open sores that discharge purulent (pus-filled) material. The pus is typically odourless and can vary in consistency, with softer and pasty pus is more common in goats and thicker or caseous (cheese-like) pus is more common in sheep. In internal CL, abscesses can form within internal organs such as the lungs, liver, spleen, and kidneys. In general, animals may show signs of weight loss, decreased appetite, lethargy, and respiratory distress if the lungs are affected.

Lesions

Firm nodules or swellings near peripheral lymph nodes and finding of abscesses in internal organs are the major lesions associated with external and internal form of CL, respectively. Initially, the abscesses are firm and may be painless. As the disease progresses, they eventually rupture, forming open sores that discharge purulent material. The abscesses contain caseous material that results from necrosis and liquefaction of affected tissues. Surrounding tissues may show inflammation and fibrosis as a response to the infection.

Diagnosis

Though the presence of external abscess near to peripheral lymph nodes is suggestive of CL, confirmatory diagnosis can be made by bacterial culture and PCR-based identification of *C. pseudotuberculosis* from the affected lymph nodes. In suspected internal CL, imaging techniques such as ultrasound or radiography may be used to visualize abscesses within internal organs, which must be confirmed by culture of tracheal washes or aspirated needle contents. Testing of paired serum samples in indirect ELISA is effective in control and eradication programs.

Differential Diagnosis

The infection with *Corynebacterium pseudotuberculosis* must be differentiated from abscesses caused by *Staphylococcus* spp., *Trueperella pyogenes* and *Actinobacillus ligneresii*, diseases like tuberculosis, brucellosis, and *Mycoplasma* infection, neoplastic conditions like lymphoma, parasitic abscesses in lymphatic tracts, and trauma or foreign body induced abscesses.

Treatment

Treatment is not recommended due to recurrence of the disease and most of the antibiotics do not penetrate abscess well.

Control

The most practical management approach for commercial animals infected with CL is to cull them from the herd. When culling is not an option, an effective strategy could be dividing the herd into clean and infected groups, and gradually removing older or less valuable animals. Additionally, lambs and kids from CL-infected dams can be raised on pasteurized colostrum away from infected animals. However, since the internal form of CL and asymptomatic carriers can still pose a risk, this approach may have limited success in completely eradicating the infection. The animals with draining abscesses should not be sent through sale barns until draining has ceased and the wound has healed. Following strict biosecurity measures, culling of diseased animals from the herd or flock, disinfection of equipment, serologic screening, and quarantine before introduction of new animals are the basic needs to prevent infection in the herd.

Commercial CL vaccines are currently licensed for use in sheep and goat. It is advisable to vaccinate young replacement animals, while older infected animals should be gradually culled as financial resources permit. When CL prevalence in the herd decreases to a low level, vaccination should be discontinued, and all seropositive, unvaccinated animals should be culled. In herds or flocks that have no history of CL (clean herds), vaccination is not recommended.

Biosecurity Measures

Enforce rigorous biosecurity measures to prevent the introduction and spread of the disease. This involves quarantine and testing of new animals before they are introduced into the herd. Additionally, regulate access for people, vehicles, and equipment to farm areas to reduce the risk of introducing pathogens.

Sample Collection for Diagnosis

Pus or exudate from draining abscesses and tissue samples from affected lymph nodes are the sample of choice for confirmatory diagnosis of CL. Serum samples may be collected for serological tests

such as ELISA to detect antibodies against *C. pseudotuberculosis*.

3.26 Dermatophytosis

Definition and Causative Agent

Dermatophytosis, a superficial mycotic disease, affects the host's keratinized tissue. It is caused by a group of keratinophilic moulds called dermatophytes which can thrive on the keratinous structures by producing an array of protease enzymes. This keratinolytic fungi group includes *Microsporum, Trichophyton, Nannizzia, Arthroderma, Epidermophyton, Lophophyton* and *Paraphyton*. The most common cause of dermatophytosis in cattle and goats is *Trichophyton verrucosum*, a zoophilic dermatophyte. Dermatophytes secrete endoproteases, exoproteases, and sulphite (Na_2SO_3) for the breakdown of keratin to amino acids.

Transmission

The hyphal structures of dermatophytes breakdown to form smaller spores called arthrospores, which are the major infective propagules. The spores once enter to the host body surface will germinate in the stratum corneum.

Clinical signs

The disease is more common in kids and lamb than in the adult animals. The affected animals exhibit non-pruritic lesions.

Lesions

Loss of hair in patches and crust formation in the neck and face are the most commonly observed lesions. Sometimes suppurative lesions can also be observed. Lesions may be observed in the limbs and chest region.

Diagnosis

Direct microscopic examination of samples after treatment with 10 percent KOH reveals the arthrospores in the clinical samples. The isolation of dermatophytes from the samples can be attempted by the inoculation in Sabouraud's dextrose agar supplemented with chloramphenicol and cycloheximide. The dermatophyte isolates can be characterized by microscopic examination, colony morphology, and molecular techniques such as PCR. Production of the chain of chlamydospores in the corn meal agar is characteristic of *T. verrucosum*. Trichophyton agar can also be used to assess the vitamin and amino acid requirements of *T. verrucosum* isolates.

Differential Diagnosis

Ringworm should be differentiated from other common skin ailments such as bacterial infections, insect bites, interdigital dermatitis, etc.

Treatment

Crusty lesion can be topically treated with Whitfield's ointment or with 4 percent lime sulphur. Povidone iodine (1 percent) or 0.5 percent sodium hypochlorite have similar success rate. Systemically, animals may be treated with 10 percent sodium iodide @ 1g/14 kg BW IV.

Prevention and Control

A live vaccine containing freeze-dried fungal structures of *T. verrucosum* is used in some countries as a prophylactic measure against *T. verrucosum* infections. However, vaccination is not commonly practiced in India.

Biosecurity Measures

The disease is highly contagious and zoonotic. The infected animal should be isolated and treated till clinical and mycological cure. Contaminated bedding material should be discarded. The farm equipment and other types of machinery should be properly disinfected with sodium hypochlorite.

Sample Collection for Diagnosis

Skin scrapings from the periphery of the lesion are mainly collected for the isolation of dermatophytes. Infected hairs along with follicles can also be plucked using sterile forceps.

3.27 Clostridial Diseases

Organisms of the genus *Clostridium* are sporulating, anaerobic large bacteria about 3-8µ in length and 0.8µ in width with terminal or sub-terminal spores, occur singly, in pairs or in chains. Organisms are commonly found in soil as well as in the intestinal tract of humans and animals. Members of the group are responsible for several diseases of humans and animals. Some organisms produce disease through tissue invasion and others through production of toxins. Though all clostridia produce toxins that contribute to their pathogenicity.

Cl. botulinum, the cause of botulism, is completely

non-invasive disease and results from ingestion of toxins formed outside the body. The *Cl. Tetani*, the cause of tetanus, is not a particularly invasive disease and results from circulating toxin produced by organisms, which after entry into the body, and remain localized within the sites of tissue damage with low oxygen tension. The invasive clostridia such as *Cl. chauvoei*, *Cl. novyi* and *Cl. haemolyticum* cause diseases characterized by extensive tissue invasion and necrosis. Tissue invasion by these organisms is by means of tissue damage initiated through other mechanisms to produce an anaerobic environment. The pathogenesis of diseases caused by *Cl. perfringens* may involve invasive and non-invasive mechanisms.

3.28 Braxy (Bradsot)

Definition and Causative Agent

Braxy (bradshot), an acute infection of sheep, is caused by *Cl. septicum* and characterized by haemorrhagic abomasitis, toxaemia and high mortality.

Transmission

The disease occurrence is associated with cold weather and the ingestion of frost covered feed and fodder. *Cl. septicum* is a soil-borne organism and considered as a normal inhabitant of the ovine intestinal tract. The infection mainly affects young sheep and usually occurs during the winter months.

Clinical Signs

Death is sudden, with few or no clinical signs. Animals showed anorexia, depression, and segregation with high fever (41.11°C). Abdomen may be distended with pain. The sheep become recumbent, comatose and die within a few hours.

Lesions

The wall of the abomasum showed thickening, oedema, congestion, haemorrhages, necrosis and ulceration. Small intestine may also show congestion.

Diagnosis

Cl. septicum can be isolated from the cut surface of the abdominal wall or by culture from heart blood and other fresh morbid samples. The causative bacilli can be seen in tissue section and are readily isolated from the lesions.

Differential Diagnosis

Disease may be differentiated from overeating of grains which cause focal ruminitis and reticulitis but no lesion in the abomasum. It is also to be differentiated from the black disease (infectious necrotic hepatitis).

Treatment

No treatment is found to be effective. Procaine penicillin can be opted to treat the clinical cases.

Prevention and Control

Management of the flock is important during winter; the sheep may be given hay before allowing them to frosted pasture in the morning. The vaccination using killed whole culture vaccine of *Cl. septicum* is also effective.

Biosecurity Measures

Proper cleaning and disinfection of all the equipment and animal sheds. Proper disposal of carcasses is required to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Sample Collection

Morbid samples from abomasum and heart blood may be collected within an hour of death for culture isolation and confirmation.

3.29 Black Disease (Infectious Necrotic Hepatitis)

Definition and Causative Agent

Black disease, an acute fatal infection of sheep and rarely cattle and pigs, is caused by the toxin of *Cl. novyi* (*Cl. odematiens*) excreted in damaged liver tissue. Under field conditions, it is usually associated with fascioliasis. Three strains of the organisms, *viz.*, A, B and C are widely distributed in soil and are common inhabitant of intestinal tract of sheep. Strains are differentiated on the basis of toxin production.

Type A produces alpha toxin – Necrotizing and lethal

Type B produces both alpha and beta toxin – necrotizing, haemolytic and lethal

Type C is non-toxigenic and non-pathogenic

Cl. novyi type B, nearly identical to *Cl. haemolyticum*,

is the strain that causes black disease. Pathogenesis and lesions are similar to bovine bacillary haemoglobinuria.

Transmission

Grazing on the contaminated pasture is the important source of infection. The carcass of the sheep died of the disease may also cause contamination of the pasture. Spores of the *Cl. novyi* pass through the intestinal wall and lodge in the liver and remain as a latent infection. Anaerobic environment - produced by the migration of liver flukes - activates the proliferation of the bacteria leading to release of exotoxin which further contributes to hepatic necrosis and produces fatal toxaemia.

Clinical Signs

Few animals may be found dead without showing any signs of illness. Animals may show high temperature (40.55°-41.66°C), hyperaesthesia, sternal recumbency and death within a few hours. In few cases, death may occur without any premonitory signs.

Lesions

Blood stain froth may exude from the nostrils. Pathologic changes include characteristic multiple foci of necrosis in the liver and petechiae on the epicardium, endocardium, and hydropericardium. Subcutaneous venous congestion causes a dark discoloration of the pelt, which gives the name of the disease "Black disease". The migratory tracts of fluke with haemorrhages are usually evident on the cut surface of the liver.

Diagnosis

Diagnosis may be made on postmortem lesions particularly in the liver and isolation of *Cl. novyi* from morbid samples. FAT may also be used on smears and/or sections of liver or confirmatory diagnosis.

Differential Diagnosis

Black disease is to be differentiated from acute fasciolosis, enterotoxaemia, black leg, malignant oedema and anthrax. Laboratory confirmation is necessary for definite diagnosis.

Treatment

No effective treatment is available. However, penicillin and other broad-spectrum antibiotics along with NSAIDs may be useful.

Prevention and Control

The control of liver fluke and other parasitic infestation is effective in the control of the disease. Proper disposal of carcasses is helpful to prevent the pasture contamination by the organisms. Vaccination also effective for the control of disease.

Biosecurity Measures

Biosecurity measures include proper cleaning and disinfection of all the equipment and animal sheds, scientific disposal of carcasses to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals and controlling parasitic load by timely anthelmintic treatment.

Sample Collection

Morbid samples from liver and heart blood may be collected within an hour of death for culture, isolation of bacteria and confirmation.

3.30 Enterotoxaemia (ET)

Definition and Causative Agent

Five strains of *Cl. perfringens* (Type A, B, C, D and E) are responsible for causation of enterotoxaemia by elaboration of toxins in the GI tract. Some of these are true enterotoxaemia (caused by type D), but others are characterized by necrotizing enterocolitis.

Type	Alpha	Beta	Epsilon	Iota	Disease
A	+	-	-	-	Gas gangrene, food poisoning, enterotoxaemia in lambs, cattle, goat, horses, dog
B	+	+	+	-	Lamb dysentery, enterotoxaemia in calves, foals
C	+	+	-	-	Enterotoxaemia (necrotic enteritis) in lambs, goats, cattle, pigs, struck in adult sheep
D	+	-	+	-	Enterotoxaemia (over eating disease, pulpy kidney disease) in sheep, goat, cattle
E	+	-	-	+	Enterotoxaemia in calves, lambs

3.30.1 Enterotoxaemia caused by Clostridium perfringens Type A

The organisms are the normal part of the bacterial flora of the alimentary tract of many animal species. The illness is characterized by an acute profuse watery diarrhoea and high mortality with presence of large number of *Cl. perfringens* Type A organisms in intestine during postmortem examination. Haemorrhagic gastroenteritis is the common manifestation of the disease with development of colic syndrome. The organism is also associated with food poisoning and diarrhoea in pigs and human. In the haemolytic condition, animal shows severe depression, pale mucus membrane, jaundice, haemoglobinuria, dyspnea, and severe abdominal pain. Temperature may rise up to 41.11°C.

Transmission

The organisms are commonly found in the soil and in the alimentary tract of normal animals. The spores of the bacteria survive for long periods in the soil. The toxins produce by type A is alpha toxin, which is necrotizing and haemolytic.

Clinical Signs

Type A enterotoxaemia is often termed as yellow lamb disease. It has short course and high rate of mortality characterized by intense icterus, haemolytic anaemia and haemoglobinuria. Type A is also associated with colitis 'x' in horses, characterized by foul smelling profuse diarrhoea and dehydration.

Lesions

Lesions at postmortem include pale anaemic mucus membranes, jaundice and haemoglobinuria, swollen kidneys dark brown in colour which may contain infarcts. The liver is pale, swollen, enlarged and friable. There may be hydropericardium and pulmonary oedema. The intestine shows extensive necrosis with presence of large number of clostridium organisms.

Diagnosis

Diagnosis can be made on the basis of clinical findings. Confirmation may be made by the presence of large number of colonies of *Clostridium perfringens* on culture of faecal sample, and also in the contents of small intestine with demonstration of alpha toxin in the intestinal contents.

Differential Diagnosis

Differential diagnosis is to be made from chronic copper poisoning and leptospirosis in calves.

Treatment

Use of hyper immune antiserum is the only effective treatment. Oral administration of penicillin may prevent further proliferation of the organisms and production of the toxins. The use of chelating agents is also effective.

Prevention and Control

Cl. perfringens Type A antiserum is effective in prevention of the disease in calves. The use of killed vaccine provides the immunity in sheep.

Biosafety Measures

Biosecurity measures include proper cleaning and disinfection of all the equipment and animal sheds, scientific disposal of carcasses to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Sample Collection

Faecal samples and intestinal contents from small intestine may be collected during postmortem for isolation of the organisms, detection of alpha toxin, and also for toxin-antitoxin neutralization test in mice.

3.30.2 Enterotoxaemia caused by Clostridium perfringens Type B

Cl. perfringens type B and C cause severe enteritis with diarrhoea and dysentery in young lambs, calves, pigs, and foals. *Cl. perfringens* type B causes lamb dysentery, *Cl. perfringens* type C causes 'struck' and haemorrhagic enterotoxaemia. Necrotic haemorrhagic enteritis in calves is caused by *Cl. perfringens* type E.

Transmission

The organisms are commonly found in the soil and in the alimentary tract of normal animals. The well-nourished growing animals are more susceptible. The spores of the bacteria survive for long periods in the soil. The toxins produce by type B are alpha, beta and epsilon and by type C, alpha and beta. The occurrence of the disease is common in very young lambs and kids due to immature alimentary tract.

Clinical Signs

Lamb dysentery occurs in young lambs <2 weeks of age manifested by per acute death without any clinical signs. In acute form of the disease, there is loss of suckling, severe abdominal pain manifested by bleating, stretching and looking toward abdomen. Faeces may be blood tinged with painful straining during defecation. Lambs go in recumbency, coma and death within 24 hours. Struck in adult sheep is also manifested by sudden death with abdominal pain and convulsions.

Lesions

The intestinal contents are blood stained with excessive serous fluid in peritoneal cavity. The characteristic lesion is haemorrhagic enteritis often with ulceration and occasionally with perforation and peritonitis. Lesions are usually restricted to the small intestine but may also affect the colon. Petechiae and ecchymoses are common on serous membranes. Microscopically, lesions are characterized by focal areas of necrosis involving the entire thickness of the mucosa which extends into the muscularis.

Diagnosis

Diagnosis can be made on the basis of rapid course and typical necropsy findings. The specific toxins may be detected in the intestinal/faecal contents with toxin-antitoxin neutralization tests. Severe hypoglycaemia is observed in young pigs dying of this disease.

Differential Diagnosis

Differential diagnosis is to be made with lamb dysentery and haemorrhagic enterotoxaemia and septicaemia caused by *E. coli* and *Salmonella* spp.

Treatment

Use of hyper immune antiserum is the only effective treatment. Oral administration of penicillin may prevent further proliferation of the organisms and production of the toxins. The use of chelating agents is also effective.

Control

Cl. perfringens Type B antiserum is effective in prevention of the disease in lambs and calves, and the use of killed vaccine provides the immunity in sheep.

Biosafety Measures

Proper cleaning and disinfection of all the equipment and animal sheds. Proper disposal of carcasses is required to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Sample Collection

Faecal samples and intestinal contents from small intestine may be collected during postmortem for isolation of the organisms and detection of toxins and also for toxin-antitoxin neutralization test in mice.

3.30.3 Enterotoxaemia caused by Clostridium perfringens Type C

Cl. perfringens type C induced enterotoxaemia in adult sheep is called struck which occurs mostly during winter and early spring months, clinical signs are usually not noted.

Clinical Signs

Clinically there is diarrhoea. Type C enterotoxaemia also occurs in suckling piglets, usually during first week of life. Most affected piglets die within 12 to 48 hours. Haemorrhagic, necrotizing enteritis principally affects the jejunum. There is haemorrhagic lymphadenitis of draining lymph nodes, sero-sanguineous fluid in the peritoneal, pleural and pericardial cavities and haemorrhages in the epicardium, endocardium, and kidneys.

Lesions

The lesions include haemorrhagic enteritis, with ulceration of the mucosa of duodenum and jejunum and peritonitis with large amount of fluid in peritoneal cavity. Another form of type C enterotoxaemia - known as enterotoxic haemorrhagic enteritis - occurs in calves, lambs and foals in the first few days of life.

Diagnosis

Diagnosis can be made on the basis of rapid course and typical necropsy findings. The specific toxins may be detected in the intestinal/faecal contents with toxin antitoxin-neutralization tests.

Differential Diagnosis

Differential diagnosis is to be made with lamb dysentery as well as haemorrhagic enterotoxaemia

and septicaemia caused by *E. coli* and *Salmonella* spp.

Treatment

Use of hyper immune antiserum is the only effective treatment. Oral administration of penicillin may prevent further proliferation of the organisms and production of the toxins. The use of chelating agents is also effective.

Prevention and Control

Cl. perfringens Type C antiserum is effective in prevention of the disease in lambs and calves and also the use of killed vaccine provide the immunity in sheep.

Biosafety Measures

Proper cleaning and disinfection of all the equipment and animal sheds. Proper disposal of carcasses is required to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Sample Collection

Faecal samples and intestinal contents from small intestine may be collected during postmortem for isolation of the organisms and detection of toxins and also for toxin-antitoxin neutralization test in mice.

3.30.4 Enterotoxaemia caused by Clostridium perfringens Type D

This is an acute toxaemia of ruminants caused by proliferation of *Cl. perfringens* type D in the intestine and thereafter liberation of toxins. The disease is characterized by the onset of diarrhoea, convulsions, paralysis, and rapid death. The organisms produce many toxins but epsilon toxin is most important and results in vascular damage and lesions in the nervous system.

Transmission

The organisms are natural inhabitant of the intestine and present in the soil contaminated by the faeces. The disease primarily affects the lambs and is worldwide in distribution. The animals in good nutrition and in good body condition are affected. In suckling calves, enterotoxaemia is most common between 1 and 4 months of age.

Clinical signs

There is profuse mucoid diarrhoea and stimulation followed by depression of the central nervous system. There is hyperglycaemia due to mobilization of hepatic glycogen and effusion of protein-rich fluid in the heart, brain and lungs. In lambs, the course of the disease is very short, and many lambs may be found dead without showing any clinical signs. The signs may be dullness, off feed, depression, yawning, and facial movement. Animals show green pasty diarrhoea, staggering, recumbency, opisthotonus and severe colonic convulsions, muscle tremor, grinding of teeth, and salivation. Adult sheep shows staggering, clamping of jaw, salivation, and rapid shallow irregular respiration. There is blot in the later stages and hyperglycaemia with glycosuria.

Lesions

The epsilon toxins of *Cl. perfringens* type D increase the permeability of the intestinal mucosa thereby enhancing the absorption of the toxins; increased amount of straw-coloured fluid in pericardial cavity, petechiae on the serosal surface and pulmonary oedema, congestion of the abomasum and intestinal mucosa. The kidneys are soft and pulpy within a few hours of death. There is symmetrical area of haemorrhages, oedema and liquefaction in the brain.

Diagnosis

Demonstration of large number of Gram-positive bacilli in the smears prepared from the small intestine. The intestinal content should be tested for detection of toxin and toxin-antitoxin neutralization test in the mice. An ELISA test may be used for detection of epsilon toxin in the intestinal filtrate.

Differential Diagnosis

In lambs, differential diagnosis is to be made with acute pasteurellosis and hypocalcaemia with hypomagnesaemia. In adult sheep and calves, differential diagnosis is to be made with rabies, acute lead poisoning, and pregnancy toxaemia.

Treatment

The course of the disease is so acute for effective treatment. Hyper immune serum is effective as short-term therapy. Antitoxin in combination with oral sulphadimidine is effective in goats. Chelating agents are very effective in neutralizing the toxins.

Prevention and Control

Twin control measures including reduced food intake and vaccination are effective to control the disease. Reduced feed intake may affect the growth of the lambs. Alum-precipitated formalin-killed vaccine is available for vaccination.

Biosecurity Measures

Proper cleaning and disinfection of all the equipment and animal sheds. Proper disposal of carcasses is required to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Sample Collection

Intestinal content is required for demonstration of epsilon toxin of *Cl. perfringens*. The organisms may also be isolated from intestinal contents. The toxin-antitoxin neutralization test may be carried out in mice.

3.31 Tetanus

Definition and Causative Agent

Tetanus is a fatal infectious disease of all species of domestic animals caused by the exotoxins of *Cl. tetani*. Tetanus or lock jaw occurs in humans and animals. The causative agent *Cl. tetani*, is a normal inhabitant of the intestinal tract of herbivorous animals and is found in humas-rich soil. It is Gram-positive, sporulating, anaerobic, rod-shaped bacillus. Tetanus is usually a sequel of wounds, such as nail pricks, castration, docking, shearing or those received during parturition.

Transmission

The *Cl. tetani* organisms enter through deep puncture wounds but the spores may remain dormant in the tissues for some time till the environment gets anaerobic in the tissues. In some cases, the toxin is produced in the gut or ingested preformed in the feed. The grazing of rough fibrous feeds before the outbreaks is a common finding and suggests that the entry of infection may occur via wounds in the mouth.

Clinical Signs

The incubation period varies between 1 and 3 weeks with rare cases occurring after 7 months of the infection. In sheep and lambs, the clinical signs appear in 3-10 days of shearing or docking. The clinical picture is similar in all the species. In general, increased muscle stiffness is observed which is first accompanied by muscle tremors. There are restricted jaw movements, prolapse of third eye lid, and stiffness of the hind limbs causing straddling gait with stiff tail. The animal looks anxious and alert with erect ears and dilation of nostrils and exaggerated response through normal stimuli, drooling saliva, and constipation with retention of urine. As the disease progresses, the muscular tetani increases, the animal shows the sawhorse posture and inability to walk and is inclined to fall. The entire musculature is eventually involved, and death follows. The sheep usually dies within 3 to 4 days.

Lesions

There are no appreciable gross or microscopic lesions, which can help in the diagnosis of the disease. However, search should be made for the site of infection for demonstration and isolation of the organisms in the laboratory.

Diagnosis

Diagnosis is based on clinical signs and a history of trauma. However, a wound is often not visible apparently, and the bacilli are difficult to demonstrate. The clinical case of tetanus is so distinctive that it is rarely confused with other diseases.

Differential Diagnosis

The disease must be differentiated with strychnine poisoning, meningitis, hypomagnesemia, white muscle disease, Polioencephalomalacia, and enterotoxaemia.

Treatment

Parenteral administration of large doses of penicillin eliminates the organism. The tetanus antitoxins are also administered @ 3,000-15,000 IU subcutaneously twelve hourly for 3 days. Local administration of antitoxin around the wound is also effective. Combination of chlorpromazine or diazepam may be administered to reduce the hyperesthesia.

Prevention and Control

Tetanus can be controlled largely by proper disinfection of instruments used for castration, docking, and shearing. Any surgical intervention on the animals may be carried out in clean surroundings and with proper use of disinfectant. As prophylaxis, antitoxin may be used for passive immunity by injecting a dose of 200 IU of antitoxin

subcutaneously in sheep and goats. In enzootic area, all susceptible animals should be given formalin-inactivated and adjuvanted toxoids for active immunization. Primary vaccination requires two doses 3-6 week apart followed by annual booster dose. The tetanus can be controlled in newborn lambs by proper vaccination of the ewe in the late pregnancy.

Biosecurity Measures

Proper cleaning and disinfection of all the equipment and animal sheds. Proper disposal of carcasses is required to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Sample Collection

The swab from the wound may be collected for isolation and characterization of Cl. tetani. The serum sample may be collected for toxin antitoxin neutralization test.

3.32 Botulism

Definition and Causative Agent

Botulism is a fatal and motor nerve paralysis caused by the ingestion of preformed toxin of *Cl. botulinum.* The organisms proliferate in dead decomposing animals and plant materials. The disease also occurs because of infection of wound with the organisms and the formation of potent exotoxins. *Cl. botulinum* is responsible for an extremely serious food intoxication-botulism. It is divided into seven different toxigenic strains based on antigenically distinguishable toxins produced by the organisms.

Type	Principal victim	Source/vehicle
A	Human and mink	Canned vegetable, fruits, meat and fish
B	Human, Horses, Cattle, Sheep	Meat, usually pork, silage and forage
C	Cattle, sheep, horses, dogs, mink, birds	Fly larvae, rotting vegetation, silage, carrion (Forage poisoning, limber neck)
D	Cattle, horses, birds	Carrion (Lamsiekte)
E	Humans, mink, fish	Fish and marine animal foods
F	Humans	Liver paste
G	Humans	Soil

Transmission

Transmission is generally by ingestion of the feed, fodder, contaminated with preformed botulinum toxins. The spores of *Cl. botulinum* are very resistant and survive for a long period in the environment. The lethal toxins have also remained active for long time in the bones of the dead animals. The organisms are common inhabitant of the alimentary tract of herbivores. Organisms are also found in the soil and the soil and water contamination occurs from faeces and decomposed carcasses. Sheep - suffering from pica associated with protein deficiency - also chew the carcass and suffer with botulism. The spoiled silage and hay also facilitate the growth of botulinum organism and may be the source of botulism in the animals.

Clinical Signs

In sheep; stiffness, incoordination, and some excitability can be noticed in the early stages. The affected animals will show holding of head on one side or bobbed up and down while walking (limber neck). Lateral switching of tail, salivation and nasal discharge may be noticed. In the terminal stage of the disease, the animal shows abdominal respiration, paralysis of limbs and death. Risk of botulism in goats is comparatively lower than the sheep due to their feeding habits. The symptoms in exposed animals are more or less similar to sheep but with less intensity.

Lesions

There are no specific gross or microscopic lesion seen in sheep and goats. However, presence of suspicious feedstuff in stomach may be suggestive. Like other species, nonspecific sub-endocardial and sub-epicardial haemorrhages and congestion of intestinal mucosa and serosa may also be present in small ruminants. Microscopically, perivascular haemorrhages may be seen in the cerebellum and cerebrum with destruction of Purkinje cells.

Diagnosis

Presumptive diagnosis can be made on history and clinical signs. Confirmatory diagnosis can be made by demonstration of toxin in the stomach and intestinal content of the animal and also in the suspected feed/fodder/carcass samples.

Differential diagnosis

Differential diagnosis is to be made from parturient

paresis in cattle and hypocalcaemia in sheep and many other diseases of nervous system including paralytic rabies in cattle, encephalomyelitis in horses and louping ill, and scrapie in sheep.

Treatment

Specific or polyvalent anti-toxin serum may be useful in early stage of the disease. Purgatives such as magnesium sulphate and liquid paraffin are useful for removal of the toxin from the alimentary tract.

Prevention and Control

Mineral (Ca, P) and protein supplements need to be added to overcome the dietary deficiencies in the animals. Proper disposal of carcasses is required to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Biosecurity Measures

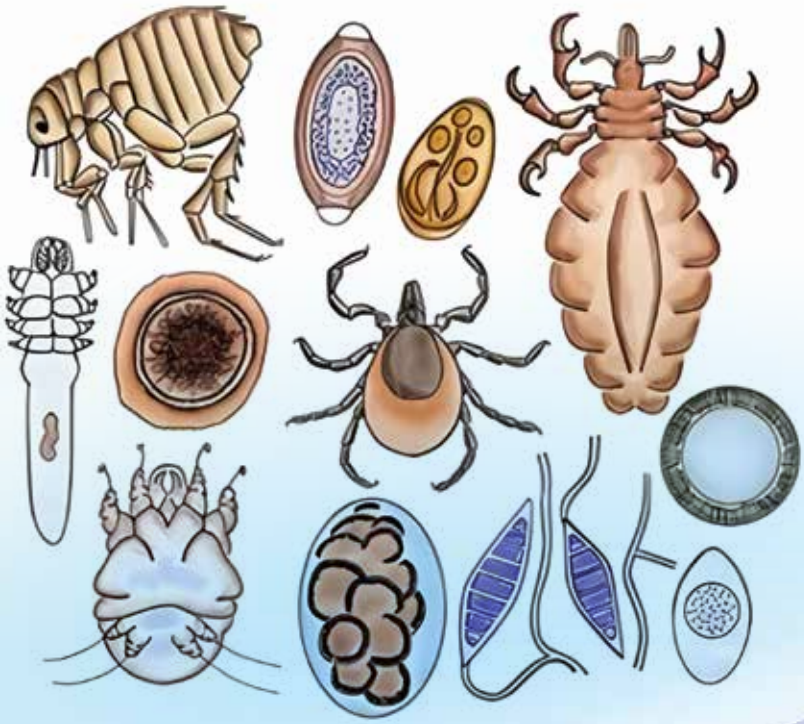
Proper disposal of carcasses is required to prevent the contamination of pasture and also to check the access of decomposed carcass to the grazing and free-range animals.

Sample Collection

Samples from gastrointestinal tract of dead animals and from decomposed carcasses may be collected for demonstration of botulinum toxin, and also to carry out the toxin-antitoxin neutralization test.

CHAPTER- 4

GUIDELINES FOR PARASITIC DISEASES OF ANIMALS



4.1 Preamble

Livestock are generally exposed to many parasitic infections (flukes, tapeworms, roundworms and protozoan parasites) and infestations (tick, mite, lice and flies, *etc.*) depending upon the husbandry practices adopted for their rearing. Impact of these parasitic infections/infestations are more in tropical countries like India, because of prevailing climatic conditions (suitable temperature, relative humidity and rainfall) which facilitate rapid regeneration of almost all the internal and external parasites. A number of parasitic diseases, *viz.*, fasciolosis, paramphistomosis, echinococcosis/hydatidosis, gastrointestinal nematodosis, trypanosomosis, babesiosis, theileriosis, bovine trichomonosis and external parasite infestations have been found to affect the health and production potentials of animals significantly, resulting in huge economic losses to the farmers. Further, the costs of medication of animals and manpower involved in animal care also reduce the net profit from the animal husbandry sector. The present veterinary services available in the country need to be updated in terms of technical skills, knowledge and also the minimum essential facility for disease diagnosis. Sincere initiatives taken by Food and Agriculture Organization of the United Nations and Government of India to formulate the Standard Veterinary Treatment Guideline (SVTG) for the country will prove beneficial to all the Veterinary Professionals in selecting suitable therapeutic remedies against the major parasitic diseases of livestock.

4.2 Bovine Anaplasmosis

Definition and Causative Agent

Bovine anaplasmosis, also called yellow bag or gall sickness, is an arthropod-borne rickettsial disease caused by an obligate intra-erythrocytic pathogen belonging to the genus *Anaplasma*. *Anaplasma marginale* is the most common aetiological agent for bovine anaplasmosis, eminently pathogenic affecting the health and productivity of bovines causing huge economic losses to animal husbandry in both temperate and tropical regions of the world, including India. The organism is located as dot-like intra-erythrocytic stages (known as marginal bodies) in the margin of RBCs. Purebred, pregnant, undernourished, lactating, high yielding cattle with concurrent infections and more than one year of age are worst sufferers.

Transmission

Anaplasma marginale is transmitted biologically by ixodid ticks belonging to the genera *Rhipicephalus*, *Hyalomma*, *Dermacentor* and *Ixodes*. Even the soft ticks like *Argas* and *Ornithodorus* can transmit this infection. The infection can also be mechanically transmitted by blood-sucking flies (*Stomoxys*, Tabanids, Deer fly, Mosquitoes) and unsterilized surgical instruments/needles. Transfusion of *A.marginale* infected blood to an uninfected host can also transmit this infection. Trans-placental transmission is also reported in cattle. Wild ruminants and deer act as carriers/reservoirs of *A. marginale* and play a decisive role in transmission of anaplasmosis.

Clinical Signs

In cattle, the disease is generally chronic in nature. Fever is noticed in the early stages of the disease, but as the disease progresses, sub-normal temperature is recorded along with severe anaemia (Hb: 2-3 g percent, PCV: 5-10 percent, TEC: 1 million/mm^3), anorexia, depression, cessation of rumination, increased cardiac and respiratory rates, prominent jugular pulse, pallor of conjunctiva, icterus, panting, exhaustion and severe depression in milk yield. Few animals may even succumb to the infection.

Lesions

Postmortem examination reveals presence of watery blood, enlarged spleen (splenomegaly), yellowish fluid accumulation in the body cavity, yellow body fat and pale mucus membranes.

Diagnosis

A presumptive diagnosis is based on the clinical signs and haematological profile indicating severe anaemia. For confirmatory diagnosis, microscopic examination of Giemsa-stained peripheral blood smears reveal presence of *A. marginale* dots (0.2-0.5µm in size) with a light hallow around them at the periphery of RBCs. Currently two serological tests, *viz.*, cELISA and CAT are preferred for diagnosing chronic infections in infected animals. Suspected blood samples may also be screened by PCR-based assays targeting conserved regions of the 16S rRNA gene or major surface protein.

Differential Diagnosis

Bovine anaplasmosis should be differentially

diagnosed from leptospirosis, babesiosis, bacillary haemoglobinuria, hepatotoxic plant poisonings, and other causes of anaemia or icterus.

Treatment

For treatment of infected cattle, intramuscular injection of oxytetracycline@ 6.0-10.0 mg/kg BW by I/M or I/V route should be given for 3-5 days. Imidocarb@ 1.2-2.4 mg/kg BW by I/M or S/C route may also be used in place of oxytetracycline. Supportive therapy with haematinics and, if required, blood transfusion is also recommended.

Prevention and control: For prevention and control of anaplasmosis in a herd, it is necessary to test all the animals for presence of infection. Subsequently, all the animals detected positive on testing should be immediately treated to eliminate the infection and prevent its further spread. Control of ticks and flies in the areas is beneficial for preventing the spread of the disease. Iatrogenic transmission can be prevented by avoiding the re-use of needles and sanitizing equipment in the dairy farms. Vaccinating the cattle with live or killed Anaplasma vaccines have been attempted in many countries.

4.3 Bovine Babesiosis

Definition and Causative Agent

Bovine babesiosis, also known as red-water fever, is caused by intra-erythrocytic apicomplexan parasites of the genus *Babesia*. In India, *Babesia bigemina* (large form) is mainly responsible for producing clinical disease in bovines but very occasionally *B. bovis* – like infection has been reported. Bovine babesiosis is more common in adult animals, as inverse age resistance protected the calves.

Transmission

Rhipicephalus (*Boophilus*) *microplus* is the main vector of *B. bigemina* in India. Vectors (ticks) get infection of *B. bigemina* (gametocytes) while feeding on infected animals. The infected female ticks transmits the infection to next generation through transovarian transmission. The larva emerging from eggs laid by infected ticks can transmit the disease to other animals while sucking blood. The sporozoites invade host erythrocytes and cause erythrolysis.

Clinical Signs

High rise of temperature, anorexia, weakness, severe anaemia, haemoglobinuria, icterus, depression and gastrointestinal stasis are the most commonly observed signs in infected animals. Diarrhoea or constipation, increase heart rate and difficult breathing may also be evidenced in severely infected animals. Infection in pregnant animals may cause abortion.

Lesions

Pale mucous membranes, watery blood, petechial haemorrhages on kidneys, presence of dark brown urine in the bladder, oedema of lungs and ecchymosis on the surface of heart and brain are the principal lesions in infected animals.

Diagnosis

Diagnosis of babesiosis can be made on the basis of clinical symptoms and detection of piroplasms in the Giemsa-stained blood smear. Further, immunodiagnosis using ELISA or IFAT may also be helpful in herd level diagnosis.

Differential Diagnosis

Bovine babesiosis must be differentiated from other haemoparasitic diseases like trypanosomosis and anaplasmosis as well as other conditions like bacillary haemoglobinuria, leptospirosis, post-parturient haemoglobinuria, toxic hepatopathies, and chronic copper poisoning.

Treatment

Diminazene aceturate: dosage: 3.5-7.0 mg/kg BW, I/M as a single dose is effective. Imidocarb dipropionate @ 2.2-4.0 mg/kg BW, I/M or S/C every 72 hours for two to four times is also very effective. It has prophylactic activity up to 8 weeks. Imidocarb is nephrotoxic, cholinesterase inhibitor and slowly metabolized.

Caution: Imidocarb can have side effects such as cholinergic reactions (salivation, lacrimation, urination, defecation) and hepatotoxicity

Supportive therapy: Fluids and electrolytes– I/V fluids to maintain hydration and support kidney function. Non-steroidal anti-inflammatory drugs (NSAIDs) like flunixin meglumine to reduce fever and inflammation. Blood transfusions may be necessary in severe anaemia.

Control

Bovine babesiosis can be controlled by integration of several approaches including segregation and

treatment of sick animals, protection of healthy animals and control of tick vectors. Besides, mapping/surveillance of disease in tropical and sub-tropical regions of the country is crucial and based on that information, foolproof control strategy is to be executed.

Biosecurity Measures

Strict biosecurity measures must be in place to prevent the introduction of new diseases in cattle farms. Basic measures include quarantine of newly purchased animals, their screening and suitable treatment, if found positive. Bovine babesiosis is spread through tick vectors, hence measures should be taken to control tick infestation in animals and surrounding environment.

4.4 Bovine Tropical Theileriosis

Definition and Causative Agent

Theileriosis, also known as bovine tropical theileriosis, is caused by a haemoprotozoan - *Theileria annulata*. *Theileria* spp. infect the blood cells (leukocytes and RBCs) of bovines and is transmitted by hard tick of the genus *Hyalomma*. The disease is more common in crossbred animals and is characterized by high fever, enlarged lymph nodes, loss of appetite, lacrymation, nasal discharge, coughing, weakness, weight loss, conjunctival petechiae and anaemia. Lateral recumbency, diarrhoea, and dysentery are also associated with later stages of infection. The parasite has very wide geographical distribution and has been considered endemic in Mediterranean basin, Middle East and Southern Asia. In India, around 10 million cattle are at risk for theileriosis with an annual economic loss of US $800 million.

Transmission

T. annulata infection occurs in bovines following inoculation of the sporozoite stage of parasite by infected ticks (*Hyalomma anatolicum*) while feeding. The tick vectors transmit the infection through stage-to-stage transmission (trans-stadial transmission), *i.e.,* infection picked up by the larvae and transmitted by nymphs or the infection is picked up by nymphal stage and transmitted by adult ticks.

Pathogenesis

Parasite may produce acute, sub-acute or chronic form of infection in animals. In acute form of disease, symptoms last for only 3-4 days otherwise it may go for about 20 days. Both the schizont and piroplasm stages of the parasite are responsible for the development of leukopenia and anaemia, which constitute the main pathogenic features of tropical theileriosis. Pathology of the disease is associated with the presence of intra-leucocytic stage of the parasite (schizont) as well as intraerythrocytic piroplasm stage.

Clinical Signs

The main clinical signs associated with *T. annulata* infection include swelling of superficial lymph nodes, fever (up to 42°C), lacrymation, nasal discharge, coughing, inappetence, icterus, tachycardia, dyspnoea and weakness. The most common haematological alteration in acute infection is anaemia, leukopenia and lymphocytopenia. Severity of infection depends upon parasite virulence, strain variation, quantum of infection as well as immune status and age of the host. Mortality may occur in heavily infected crossbred cattle during the clinical course of disease, however, the recovered animals become carriers after the treatment and parasites hide in macrophages and lymph nodes. In cerebral form of theileriosis, nervous symptoms are evident.

Lesions

Postmortem findings may include emaciated carcass, pale mucous membranes, enlarged superficial lymph nodes, spleen and liver. There is oedema in lung and punched out ulcerative lesions (button-shaped) in the abomasum (pathognomonic lesions of theileriosis) of infected cattle.

Diagnosis

Diagnosis of clinical theileriosis cases in cattle can be made on the basis of clinical signs coupled with detection of parasitic stages in the blood or lymph node aspirate smears following microscopic examination. However, detection of sub-clinical and/or chronic infection may not always be possible through this method. Therefore, molecular tests like PCR, real-time PCR and reverse line blot (RLB) and serological assays like IFAT and ELISA are used. Amongst all, IFAT is the prescribed test for theileriosis, which is less user-friendly than the other tests. Nervous form of disease may be diagnosed by impression smear examination made from the brain.

Differential Diagnosis

The clinical sings of theileriosis present in animals are generalized in nature and mostly seen in haemoparasitic infections like babesiosis, anaplasmosis and trypanosomosis. However, very close observations on the febrile phase and haemogram of the infected animals may rule out involvement of other haemoparasitic organisms.

Treatment

Buparvaquone (2.5 mg/kg BW by I/M route) is the drug of choice for treating clinical cases of theileriosis in animals. If required, this drug can be repeated after 48hours. ***Suggested milk withdrawal period of the drug is 2 days***. Other drugs like Rolitetracycline (4mg/kg BW I/M for 4 days) or single injection of long acting Oxytetracycline formulation (20.0 mg/kg BW I/M or I/V) is also effective in treating early-stage infection of *Theileria* spp. Vitamin B complex supplements can be provided as a supportive therapy.

Control

Theileria annulata infection in bovines can be controlled through integration of different strategies including vaccination (tissue culture vaccine; Rakshavac-T), timely treatment, vector control and regular monitoring of the disease. Since, hard ticks of the genus *Hyalomma* are incriminated as major vector, scientific use of acaricides and repellents is advised to maintain the tick population under control. Asymptomatic and carrier animals play important role in maintaining organism in a given geographical area, hence regular monitoring of the pathogen is of outmost importance. Early diagnosis is the key point for effective management of Theileriosis, hence reach of farmers to the disease diagnostic facilities must be facilitated.

Biosecurity Measures

Adoption of quarantine measures, screening of newly purchased animals before introducing them in the main herd and treatment of infected animals is essentially required. Further, a clean and hygienic housing facility free of disease transmitting vectors must be ensured.

4.5 Echinococcosis/Hydatidosis

Definition and Causative Agent

Echinococcosis/hydatidosis is a zoonotic parasitic infection caused by the taenid tapeworms of the genus *Echinococcus* with important parasites being *Echinococcus granulosus* and *E.multilocularis*. The adult tapeworms are found in the small intestine of canids (Echinococcosis) whereas the metacestode stage of the parasite, hydatid cyst, is found in the visceral organs including liver and lungs of intermediate hosts (hydatidosis) like human, cattle, buffaloes, sheep, goat, pig and other animals as well as wildlife.

Transmission

In the complex life cycle of the parasite, canids like dogs act as definitive hosts in which the adult tapeworms develop. The dogs get infected by consuming the offal containing the hydatid cysts from the infected intermediate hosts. Infected dogs excrete the eggs of the parasite through faeces which can contaminate feed, water and pasture. The animals and humans get infection by ingesting these eggs through contaminated food or water leading to the development of hydatid cysts in their visceral organs. Among different animals, sheep is considered as the most important and successful intermediate hosts, as they harbour fertile hydatid cysts for disease transmission.

Clinical Signs

In affected dogs, a large number of adult parasites in the intestine may cause enteritis. In intermediate hosts, as the cyst grows slowly over months, a higher infection rate is observed in older animals. The clinical signs of hydatidosis will not be evident until the disease is in the advanced stage. In heavily infected animals, symptoms include reduced productivity, weight loss, respiratory distress and even death in severe cases. Rupture of hydatid cyst may lead to anaphylactic shock and death.

Lesions

Postmortem examination reveals presence of fluid-filled hydatid cysts in different visceral organs, especially in the liver and lungs. The cysts may be unilocular or multilocular depending on the species of the parasite involved. The cysts may vary in size and may be encapsulated with clear or turbid fluid.

Diagnosis

Diagnosis can be achieved by detecting *Echinococcus* spp. eggs in the faecal sample of dogs. However, it is difficult to differentiate it from the eggs of other

Taenia spp. Diagnosis of hydatidosis is entirely made on the basis of imaging techniques like ultrasound scanning or X-ray imaging. In addition, serological tests like ELISA are available for the detection of specific antibodies of hydatidosis in animals.

Differential Diagnosis

Hydatidosis should be differentially diagnosed from other cystic conditions like liver abscess, tuberculosis, cysticercosis, and neoplastic conditions.

Treatment

Treatment of hydatidosis is generally impractical due to the high cost of the treatment and the difficulty in accessing the cysts. In humans, surgical intervention is the primary choice of treatment along with the administration of anthelmintics like albendazole to prevent the recurrence of infection. The echinococcosis affected dogs are treated with praziquantel @ 5.0 mg/kg body weight orally to break the life cycle of the parasite.

Control

Infection can be controlled by regular deworming of dogs, proper disposal of offal to prevent its access to stray dogs, implementing good slaughterhouse practices to prevent feeding of dogs with offal, and educating farmers about the disease transmission and its zoonotic importance.

Biosecurity Measures

The biosecurity measures required to effectively control the infection are – ensuring clean and hygienic food and water at the livestock farms, controlling stray dog population in the vicinity of the farms, and proper disposal of offal, carcasses, and other biowastes generated at the farm.

4.6 Paramphistomosis

Definition and Causative Agent

Paramphistomosis is caused by a group of trematode parasites (rumen flukes) which are commonly known as amphistomes. These flukes have global distribution, especially in tropical and subtropical part of the world. The disease is manifested by juvenile or immature stage of the parasites while the adults are almost non-pathogenic. In India, the most common paramphistomes reported are *Paramphistomum epiclitum* and *Gastrothylax crumenifer* in the rumen and *Gigantocotyle explanatum* in the bile duct of domestic ruminants. *Calicophoron, Cotylophoron, Fischoederius* and *Orthocoelium* are other genera found in the rumen of animals.

Transmission

Adult rumen flukes lay eggs which are passed out in the faeces of the hosts and develop in the environment, ultimately giving rise to miracidia, which infects snails of the families Lymnaeidae and Planorbidae. After passing through the different larval stages, cercariae comes out from the snails and encyst as metacercaria on the aquatic plants/ vegetation, which are picked up by the hosts during grazing.

Clinical Signs

Immature amphistomiasis is characterized by sporadic epizootics of acute gastro-enteritis with high morbidity and mortality, particularly in young animals. Infected animals show persistent foul smelling watery diarrhoea, accompanied by weakness, depression, dehydration, anorexia, sub-mandibular oedema and death leading to severe economic losses. Faeces may contain streaks of blood with large number of immature flukes and mucous.

Diagnosis

Diagnosis of immature paramphistomosis can be made by finding the pink-coloured immature flukes in the diarrhoeic faeces with the help of a hand lens (faecal examination for eggs may turn negative). Paratuberculosis, liver fluke and roundworm infection, and copper deficiency are the conditions with which paramphistomosis is most likely to be confused. Poisoning due to arsenic, lead and poisonous plants can be differentiated only by their detection either in the environment or tissues at necropsy. Coprological examination does not give any clue, since the clinical disease chiefly occurs during the prepatent period. In an outbreak situation, diagnosis is made by postmortem examination in which immature flukes are recovered from small intestine.

Treatment

Once diagnosed, condition can be treated using flukicidal drugs. Oxyclosanide (@ 20.0 mg/kg BW, oral) is the drug of choice for treating immature paramphistomosis. This drug has very good efficacy against mature flukes as well. Niclosamide is also

effective against immature flukes (@ 160.0 mg/kg BW orally as a single or in 2 divided doses 3 days apart in cattle). Niclofolan (@ 6.0 mg/kg, oral) and bithionol sulphoxide (@ 40.0 mg/kg, oral) are effective against immature flukes.

Control

Control of immature paramphistomosis relies on the holistic approach for treating animals with effective drugs (use of chemical) as well as to limit the contact between infected pasture and animals. Draining of low-lying swampy areas, if possible, to reduce snail habitat can be an effective alternative to chemical control. Use of anthelmintics at the appropriate time will remove flukes from infected animals. To reduce snail population in endemic areas, precocious use of copper sulphate or N-tri-tylomorpholin may be useful. Besides, introduction of ducks in waterbodies may prove beneficial.

4.7 Bovine Fasciolosis

Definition and Causative Agent

Fasciolosis is a snail-borne parasitic disease of cattle caused by the liver flukes *Fasciola hepatica* and *F.gigantica*. The parasite resides in the liver of cattle resulting in profound damage to animal health and productivity.

Transmission

The freshwater snails such as *Lymnaea truncatula* for *F.hepatica* and *L. auricularia* or *L. natalensis* for *F.gigantica* serve as the intermediate host of the parasite in which different larval stages like miracidium, sporocyst, redia and cercariae will develop. The cercariae will get encysted as metacercariae on the vegetation. The susceptible cattle will acquire the infection by ingestion of metacercariae, the infective larval stage of the liver fluke, while grazing. Transmission is highest in areas with poor drainage where the cattle can get access to water bodies, and also during the rainy season when water logging favours the breeding of freshwater snails. The preference of cattle to graze in wet marshy areas increases the risk of transmission of the disease.

Clinical Signs

Clinical signs and severity of fasciolosis in cattle depend on the number of metacercariae ingested and the stage of development of the parasite in the liver. Acute fasciolosis, often manifested after heavy infection, is characterized by sudden death due to massive liver damage (traumatic hepatitis). The more common form of fasciolosis in cattle is chronic wherein the clinical manifestations include weight loss, reduced appetite, lethargy, diarrhoea, submandibular oedema (bottle jaw), anaemia, jaundice and decreased milk production. The morbidity rate in cattle in endemic areas may be 20-90 percent whereas the mortality rate ranges from 1-10 percent, especially in acute cases with severe liver damage accompanied by secondary bacterial infection.

Lesions

On necropsy, show typical enlarged friable liver with necrotic tracts and fibrosis and thickening of bile ducts with presence of adult flukes in the bile ducts. The calcification of the walls of the bile ducts resembles the stem of a clay pipe (pipe stem liver). Frequently, the parasite can be found in lungs in hazel nut-sized cysts. Black disease is generally associated with the liver damage caused by the migrating young flukes and the secondary bacterial infection of the liver with *Clostridium novyi* type B bacteria.

Diagnosis

Diagnosis of acute fasciolosis can be made on the basis of clinical symptoms and the laboratory test results. In the field, the diagnosis of fasciolosis is made following the microscopic detection of eggs by faecal examination. During microscopic examination, *Fasciola* ova should be differentially distinguished from the eggs of *Paramphistomum* spp. The eggs of *Fasciola* spp. have yellowish brown shell with indistinct operculum and embryonic cells whereas amphistome eggs have transparent shell, distinct operculum and embryonic cells and eggs of some amphistomes possess a small knob at the posterior end. The long pre-patent period of infection often limits early detection of infection by this method, thus delaying the treatment. Coproantigens can be detected as early as 5 weeks of infection by ELISA, which is also used in endemic areas. The serum enzyme assay can be conducted to detect the elevated level of liver enzymes.

Differential Diagnosis

The clinical symptoms of other diseases that can mimic fasciolosis in cattle include Johne's disease

and bovine tuberculosis.

Treatment

Triclabendazole @ 12.0 mg/kg BW orally is effective against both immature and mature flukes. This drug has limited efficacy in infected buffaloes. Wherever, the triclabendazole resistance is suspected Closantel can be administered @10.0 mg/kg BW orally. Oxyclozanide (15.0-20.0 mg/kg BW, PO) and rafoxanide (7.5 mg/kg, PO) can also be used to treat fasciolosis in cattle. The secondary bacterial infection may be treated with antibiotics. Vitamin supplements may be provided as a supportive therapy.

Control

Fasciolosis in cattle can be controlled with pasture management, preventing cattle from grazing in low-lying areas near the water bodies, proper disposal of excreta, treatment of forage before giving to animals, employing snail control measures, and the specific integrated therapeutic management of the infected animals.

Biosecurity Measures

Implementing biosecurity measures to reduce the risk of fasciolosis in cattle include ensuring provision of clean drinking water, fencing of water-logged areas to prevent access to high-risk areas, as well as by creating awareness among the farmers about the transmission of the disease.

4.8 Fasciolosis: Sheep and Goat

Definition and Causative Agent

Fasciolosis is a parasitic disease of sheep and goats caused by the liver flukes *Fasciola hepatica* and *F.gigantica*. Fasciolosis is a snail-borne disease which can result in massive destruction of the liver in the infected animals due to migration of a large number of immature flukes in the liver parenchyma. The prevalence of fasciolosis in goat is low as compared to sheep due to their feeding habits.

Transmission

The susceptible sheep and goats can acquire the infection by the ingestion of infective larval stage of the liver fluke, metacercariae, while grazing in marshy areas. The freshwater snails of the family Lymnaeidae serves as the obligatory intermediate hosts of the parasite without which the parasite cannot complete its life cycle. The larval stages miracidium, sporocyst, redia and cercariae will develop inside the snail host, and the cercariae that emerged out from the snails will get encysted as metacercariae on the vegetation. Transmission is highest in the areas where the susceptible animals are allowed to graze near the infected water bodies.

Clinical Signs

Acute fasciolosis commonly occurring disease in sheep wherein the animal dies suddenly without manifesting any clinical symptoms. There may be blood-stained froth at the nostrils as well as discharge of blood from the anus. In chronic cases, the clinical signs include anorexia, anaemia, submandibular oedema (bottle jaw), weight loss, lethargy, brittle wool and diarrhoea.

Lesions

Necropsy will reveal enlarged, pale, and friable liver with numerous haemorrhagic tracts and fibrinous clots. There will be marked destruction of the liver parenchyma with hyperplastic cholangitis and fibrosis of bile duct.

Diagnosis

Diagnosis is based on the clinical symptoms and the laboratory test results. In the field, the diagnosis of fasciolosis is based on the microscopic detection of eggs by faecal sample examination. The eggs of *Fasciola* spp. have yellowish brown shell with indistinct operculum and embryonic cells. Coproantigens in faeces can be detected as early as 5 weeks after infection by ELISA and can be used in endemic areas. The serum enzyme assays can be conducted to detect the elevated level of liver enzymes.

Differential Diagnosis

The clinical symptoms of acute fasciolosis in sheep may mimic anthrax, haemonchosis, clostridial infections, and pasteurellosis. Chronic fasciolosis should be differentially diagnosed from haemonchosis, cobalt deficiency, and Johne's disease.

Treatment

Triclabendazole @12.0 mg/kg BW, PO is effective against both immature and mature flukes. Closantel@7.5 mg/kg by I/M route is effective for adult flukes. Oxyclozanide and rafoxanide are also used for the effective treatment of fasciolosis in sheep.

Control

Fasciolosis can be controlled with pasture management to minimize the exposure of susceptible flocks to infested vegetation and water. Strategic deworming, rotational grazing, drainage of water-logged areas and adopting snail control measures will reduce the incidence of fasciolosis. Regular screening and treatment of infected animals is highly advised to prevent the transmission of infection.

Biosecurity Measures

To reduce the risk of fasciolosis in sheep and goats, the biosecurity measures that need to be considered are avoiding overstocking, maintaining clean feeding and watering areas and regular monitoring of the health status of the herd.

4.9 Gastrointestinal Nematodosis (GIN)

Definition and Causative Agent

GIN, a ubiquitous, economically devastating disease of ruminants, is a front-line disease affecting the health and production of small ruminants. Its control is arguably hampered due to the widespread prevalence of drug-resistant strains. The infection is predominantly caused by the members of genus *Haemonchus, Trichostrongylus, Bunostomum, Cooperia,* and *Oesophagostomum* in the country; however, other nematodes like *Chabertia ovina, Oestrertagia/Teladorsagia* (both in temperate climates), *Gaigeriapachyscelis* (central India), and *Nematodirus* (Himalayan states) also cause sporadic potential damage to the ruminants.

Transmission

These are soil-transmitted nematodes. Environmental factors such as temperature, moisture, and humidity play important roles in development and transmission. The third larval stage is infective to the host and development from eggs to the infective stage occurs in the environment. The infective larva invades the host by ingestion (per os), however, *Bunostomum* spp. and *G.pachyscelis* larvae may also enter the host by skin penetration.

Clinicalsigns

Anaemia is a predominant sign in blood-feeding species. *Haemonchus* spp., *Mecistocirrus digitatus*, *Bunostomum* spp., and *G. pachyscelis* are voracious blood suckers. Besides, frequent changes in their site of attachment in the abomasal/intestinal mucosa lead to biting injuries with continuous bleeding from the biting wound, further facilitating the loss of blood. Initially, the anaemia is normocytic and normochromic; later, it turns out to be microcytic and hypochromic. In chronic cases, hypo-proteinaemia and jaw oedema are common. The generalised symptoms in GIN include anorexia, enteritis (black diarrhoea in trichostrongyliasis, greenish diarrhoea in oesophagostomiasis, tarry diarrhoea in bunostomiasis), loss of body weight, dehydration, depressed growth, production losses and sometimes death. Morbidity varies between 50-70 percent; sometimes, mortality occurs between 8-10percent in young animals.

Lesions

Pin-point bite marks in the abomasum/small intestine may be observed in haemonchosis and bunostomosis. Nodules may be present on the serosal layer of the intestines in oesophagostomiasis. The lesions include pale mucous membranes, watery blood, fluid in cavities, fat depletion (jelly-like fat), slight to moderate pale colour of internal organs, and fatty changes in the liver. Ringworm-like lesions are usually noticed in the pyloric end on the abomasum in *T.axei* infection. In heavy teladorsagiasis, hyperplastic abomasal mucosa appears like Morocco leather.

Diagnosis

Diagnosis is mainly based on clinical signs (using FAMACHA, Dag score and intermandibular oedema), epidemiology of GIN and coprological examination for both qualitative and quantitative faecal egg count (McMaster technique). Copro-culture of faecal samples to obtain larvae and subsequent examination of the infective larva is a standard method for species identification. Nucleic acid-based techniques are accurate methods for the identification of various nematode species.

Differential diagnosis

GIN is often misdiagnosed with chronic Type D enterotoxaemia in goats, salmonellosis, Johne's disease, coccidiosis, cryptosporidiosis, immature amphistomosis and subacute rumen acidosis.

Treatment and Control

Broad-spectrum anthelmintics such as benzimidazoles, imidazothiazoles/ tetrahydropyrimidines, and avermectins are effective against GIN. Benzimidazole derivatives: fenbendazole 5.0-7.5mg/kg BW PO, albendazole 5.0-10.0 mg/kg BW PO; imidazothiazoles: levamisole 7.5 mg/kg BW PO or S/C; tetrahydropyrimidines: pyrantel 7.5 mg/kg BW PO, morantel 15.0 mg/kg BW PO and ivermectin 200µg/kg by S/C route should be administered whenever needed. Closantel (5.0 mg/kg S/C or 10.0 mg/kg PO) is effective against anthelmintic resistant strains of *Haemonchus*.

Control

Control of GIN in small ruminant is largely based on strategic use of anthelmintics. Selective treatment to maintain refugia (keeping a portion of parasites in a population unexposed to treatment), reduces the spread of drug-resistant GIN). The selective treatment indicators are anaemia, diarrhoea, body score, sub-mandibular oedema. Resting/burning of pasture can kill free living stages of nematodes. Advising farmers to avoid grazing of animals at early morning and late evening.

4.10 Trichinellosis

Definition and Causative Agent

Trichinellosis, caused by a nematode parasite of the genus *Trichinella*, is one of the most important meat-borne zoonotic diseases. However, this parasite has little impact on the animal health. Presently, there are 10 valid species (*Trichinella spiralis*, *T. nativa*, *T. britovi*, *T. murrelli*, *T. nelsoni*, *T. patagoniensis*, *T. chanchalensis*, *T. pseudospiralis*, *T. papuae*, and *T. zimbabwensis*) and 3 genotypes (T6, T8 and T9) under this genus. However, *T. spiralis* is the most pathogenic and widespread species.

Clinical Signs

The infected animals do not show any clinical signs and act as reservoir host. However, the clinical signs of trichinellosis in human include generalised fever, vomiting, abdominal pain, diarrhoea, periorbital oedema, eosinophilia, myalgia and prostration. In severe disease, there may be pulmonary, cardiovascular, and neurology related complications which can sometimes be fatal.

Lesions

In muscles, the *Trichinella* spp. larvae are coiled and enclosed in a collagen capsule (encapsulated species) or may exist freely in the muscle fibres (non-encapsulated species). Calcification of collagen capsule and nurse cell commences after the considerable time period which results in death of larvae.

Diagnosis

Conventional antemortem diagnosis (trichinoscopy) involving biopsied muscles is very difficult and thus serological tests utilizing suspected serum (ELISA and Western blot) are now being used for diagnosing the infection in humans. Confirmation of infection in animals can be made following examination of preferred muscles (masseter, diaphragm, tongue and intercostal) by compressorium technique. Further, artificial muscle digestion technique (acid pepsin digestion) can be used to increase the sensitivity of larvae detection.

Differential Diagnosis

Differential diagnosis must be carried out with food poisoning, typhoid fever, influenza, intolerance to pork, muscle rheumatism, cerebrospinal meningitis, dermatomyositis, periarteritis nodosa, and eosinophilic leukaemia.

Treatment

Benzimidazole compounds (albendazole or mebendazole) are recommended for treating human infection. If treatment is started in the early phase (less than 3 days), further migration of larva can be prevented in muscles. Albendazole is recommended for humans @ 400mg twice a day by oral route for 8-14 days. Mebendazole may be used @ 200 to 400 mg thrice a day for 3 days by oral route followed by 400 to 500 mg thrice a day for 10 days by oral route. Pyrantel may be used in pregnant women and children below 2 years of age @ 10.0 to 20.0 mg/kg BW by oral route. This drug needs to be repeated for 2 to 3 days. Steroids may also be administered to reduce the severity of symptoms.

Control

Trichinellosis is less known disease in India and there is an urgent need to create public awareness regarding the safe meat consumption. Postmortem examination of meat producing pigs and other reservoir animals needs to be made mandatory.

People must be educated regarding the life cycle, distribution pattern (globally as well as in Indian scenario) and preferred host of different species along with risk factors associated with clinical disease in humans. Apart from that, reach of domestic pigs to wild animal carcasses in the forest premises may also initiate infection in domestic cycle. Therefore, timely and safe disposal of wild animal carcasses is essential.

Biosecurity Measures

Trichinella larvae have tendency to survive even in putrefying carcasses/carrion and hence proper carcass disposal procedure must be in place. Movement of rodents and carnivorous birds in pig farms must be restricted. Free-ranging pigs are always at risk so, scientific pig rearing must be popularized. Pig feed formulations must be free from the garbage and/or meat scraps.

4.11 Trypanosomosis

Definition and Causative Agent

Trypanosomosis, commonly known as surra, is caused by the flagellated haemoprotozoan parasite, *Trypanosoma evansi*. The parasite causes debilitating wasting type of disease in cattle.

Transmission

The transmission of *T. evansi* from the infected to the susceptible cattle occurs through mechanical means mainly through biting flies like *Tabanus*, *Stomoxys* or *Lyperosia*. In addition, iatrogenic transmission through contaminated needles as well as surgical instruments can also contribute to the spread of the disease within a herd. In endemic areas, the presence of reservoir animals including wild animals contributes to the maintenance of infection.

Clinical Signs

In most of the cases, the affected cattle will be asymptomatic carriers. The disease is chronic and is characterized by fluctuating fever, corneal opacity, lachrymation, inappetence, progressive weakness with staggering gait or recumbency, emaciation, laboured breathing, salivation, poor weight gain, hypoglycaemia, reduced milk yield and draught ability, nervous symptoms, convulsions and abortion in pregnant animals. In affected animals, progressive anaemia will be evident due to haemolysis of red blood cells and erythrophagocytosis.

Lesions

On postmortem examination, enlargement of liver and spleen may be evident. Other symptoms include congestion of the lungs and petechial haemorrhages on the serous surface and parenchyma of the liver and kidneys.

Diagnosis

Diagnosis is based on the clinical symptoms as well as the laboratory test results. Wet film examination of peripheral blood samples reveals the moving parasites under the microscope. The microscopical examination of Giemsa stained thick/thin blood smears will identify the parasite based on the morphological characteristics like the presence of free flagellum, well developed undulating membrane and subterminal kinetoplast. Mouse inoculation test can be used for the diagnosis of infection in cattle. PCR tests can also be used for the sensitive detection of infection from the blood sample. Card agglutination test for *T.evansi* (CATT/*T.evansi*) is available for the detection of IgM antibodies in the serum samples. Further, whole cell lysate-based indirect ELISA is being used for the detection of IgG antibodies in the serum samples against *T.evansi* in infected cattle.

Differential Diagnosis

Differential diagnosis of trypanosomosis in cattle is often challenging due to the presence of non-specific clinical manifestations that overlap with other diseases of cattle. The condition may be differentially diagnosed from haemoprotozoan parasitic diseases like babesiosis, anaplasmosis and theileriosis. Considering the clinical history, geographic features of that area and the presence of vectors arecrucial while arriving at a diagnosis along with laboratory confirmation.

Treatment

Diminazene aceturate @3.5 to 7.0 mg/kg BW by deep I/M route is the most extensively used trypanocide. Surramin@0.5g/45kg BW by I/V route is also effective against *T.evansi* infection in cattle. Isometamidium chloride can be used for both prophylactic and curative purposes wherein the dosage of Isometamidium chloride is @0.5 mg/kg BW by I/V or S/C route for curative purpose whereas @ 1.0mg/kg BW for prophylactic purpose. Quinapyramine salts (sulphate plus chloride) can be used @3.0-5.0 mg/kg BW by S/C route for

prophylaxis and treatment purposes. The vitamin supplements can be provided as a supportive therapy.

Control

Effective control of *T.evansi* infection in cattle can be achieved by chemoprophylaxis, vector control, regular monitoring and adopting required management practices. Since haematophagous flies can transmit the disease, vector control through insecticides and repellents is advised to effectively check the infection. The recovered animals can act as carriers of infection and also as a source of infection to the susceptible animals. As such, the asymptomatic carrier animals should be identified and treated to effectively control the disease. The animal at risk can be treated with chemoprophylactic drugs. Further, good husbandry practices like good nutrition and stress management strategies must be adopted to enhance the overall health of the herd.

Biosecurity Measures

Implementing biosecurity measures helps prevent the introduction and spread of disease in cattle farms. The measures include quarantining and screening of newly introduced animals followed by treatment of infected animals, maintaining clean and hygienic housing conditions to avoid any potential fly breeding sites, adopting fly control measures at the farm, and minimizing contact of animals with potential vectors.

4.12 Bovine Trichomonosis

Definition and Causative Agent

Bovine trichomonosis, also called as bovine trichomonad abortion or bovine genital trichomonosis, is a venereal disease of cattle caused by a protozoan parasite, *Tritrichomonas foetus*(*T. foetus*). The protozoon infects the genital tract of cattle, where it multiplies asexually by longitudinal binary fission, and is responsible for early abortions in cows, usually between 1-16 weeks after conception. The disease is more commonly seen in dairy herds.

Transmission

Under natural conditions, *T. foetus* is transmitted during coitus. The parasite may also be transmitted by artificial insemination (if the semen used is infected or the equipment used is contaminated), infected teaser bulls or during gynaecological examination if proper precautions are not taken. The infected bulls become a permanent source of infection, while in cows, the infection is self-limiting, and the parasites disappear after sexual rest for at least three oestrus cycles.

Clinical Signs

In bulls, the principal site of infection is preputial cavity. However, epididymis, testes and seminal vesicles may also be involved. The clinical signs include pain on micturition, disinclination to serve the cows and mucopurulent preputial discharge along with red nodules on mucus membrane of prepuce.

In cows, initially there is mucopurulent discharge from vagina with a slight swelling and reddening of the vulva and vagina. The organism invades the uterus and produces inflammation and catarrh. The cow may conceive, but aborts early (1-16 weeks post-conception) with subsequent uterine discharges and irregular oestrus cycles. In some cases, foetus and membranes are not expelled out completely, as a result maceration occurs which may even lead to sterility. In cases where cervix is closed, pyometra occurs and a thin greyish-white and odourless fluid containing trichomonads accumulates. There is associated anoestrus and cows appear apparently pregnant during this period.

Lesions

Chronically infected bulls show no gross lesions. At the most, there may be orchitis, which subsides as the case progresses. In the infected cow, the initial lesion is a vaginitis, with swelling and reddening of vulva and vagina. The organism invades the uterus and produces inflammation and catarrh. In pregnant animals, there may be placentitis leading to early abortion.

Diagnosis

A presumptive diagnosis is based on herd history, which will reveal an increased incidence of cows returning to service, failure of cow to conceive after repeated artificial insemination, early abortions in cows, increased incidence of vaginal discharges and pyometra in cows following introduction of a new bull in the herd. Confirmatory diagnosis is achieved by demonstration of trichomonads in vaginal or uterine discharges or in amniotic and allantoic fluids or in stomach contents of

aborted foetus or in preputial washings from the suspected bull.

Differential Diagnosis

Bovine trichomonosis should be differentially diagnosed from diseases having clinical signs like infertility, vaginitis, pyometra, abortions and vaginal discharge like campylobacteriosis, leptospirosis, brucellosis, neosporosis, chlamydiosis, bovine viral diarrhoea, and infectious bovine rhinotracheitis, *etc*.

Treatment

In bulls, the treatment is difficult. However, boroflavin ointment may be applied into the preputial cavity and massaged for 15-20 minutes giving pudendal anaesthesia to the bulls. Less than 1 percent solution of Acriflavin is injected in preputial cavity and retained for 15 min. One or two courses with five successive treatments per day may eliminate the infection. Dimetridazole @ 50.0 mg/kg BWby I/V route may also be given daily for five days.

Important: Since the infection is self-limiting in cows, a sexual rest for 3 consecutive oestrus cycles in aborted cows is recommended

Prevention and Control

Early diagnosis is the key for prevention and control of bovine trichomonosis. Since the treatment is difficult, all the infected bulls must be either castrated or slaughtered. Only the thoroughly tested and authorized bulls should be used for breeding purpose. Use of artificial insemination with proper care and hygiene can reduce the incidence of the disease in the herd. The aborted cows must be given sexual rest for three consecutive oestrus cycles.

4.13 Tick infestations

Definition and Causative Agent

Ticks are the most important blood-sucking ectoparasites of livestock in humid tropical and subtropical areas. Theyare responsible for severe economic losses through their haematophagous activity and transmission of deadly pathogens. The infestation is mainly due to the genus *Rhipicephalus* and *Hyalomma* in the country; however, another genus, *Haemophysalis* is also prevalent. Several tick species have been recognised as vectors for many deadly haemoparasitic diseases, *viz.*, babesiosis, theileriosis and anaplasmosis in animals and Crimean Congo haemorrhagic fever, Kyasanur forest disease, *etc*., in humans.

Transmission

The animals get infestation from the environment. Ticks infesting bovines are divided into one- host, two-host, and three-host life cycles based on life cycle patterns. In one-host life cycle (*Rhipicephalus microplus*), the tick remains on the host throughout life; only engorged females leave the animals to lay eggs. The eggs hatch to larva stage, which further seek a new host for attachment and feeding. In two-host ticks, the newly emerged larva attaches to the host and, after blood meal, moult to nymph, and drop out from the host for moulting to adult in the environment. Further, newly moulted males and females seek a host for a blood meal, and engorged females drop out for egg laying. In three-host ticks, every stage seeks a new host for a blood meal, *viz*., larvae, nymphs, and adults. A hot and humid condition is a favourite environment for the development and transmission of ticks.

Clinical Signs and Diagnosis

The infected animals show attachment of ticks on the body. Tick infestation is manifested as anaemia, severe debilitation, reduced production, delayed growth rate and reproduction. The blood may ooze out due to bite injury from sensitive skin areas such as the udder and external genitalia region. Ticks are associated with tick toxicosis and tick paralysis, which is described as ascending paralysis. Tick bites may damage skin at the biting site and lead to alopecia, pruritis, and crust formation.

Treatment and Control

Broad-spectrum acaricides such as pyrethroids, formamidines, avermectins, and phenylpyrazole are effective against ticks. Pyrethroids derivatives like cypermethrin@ 10 percent w/v(topical application over the skin), deltamethrin @ 2.5 percent w/v topical application, Flumethrin pour on (@1ml/10 kg BW drop by drop on the skin over vertebral column) are very effective. Formamidine-amitraz @12.5 percent w/v topically; avermectins: ivermectin @ 200 µg/kg BW by S/C route; and phenylpyrazole: fipronil@ 0.25 percent topical applications are regularly used for the management of tick infestation.

Control

Chemical-based acaricides are used to control, strictly based on epidemiology to maintain the efficacy of currently available drugs, however, the development and spreading of acaricide resistance is a global problem. Frequent monitoring of acaricide resistance and rotational use of different broad spectrum acaricides/combination of acaricides could be an option to control acaricide resistance. Resting/burning of pasture can kill larvae of ticks. Phytochemical based acaricides may be applied alternatively to chemical drugs to integrated tick control strategy. Cattle can be immunized against ticks, by vaccines like TICKGAURD/GAVAC, but the genetic diversity in the vaccine candidates limited their efficacy as a global vaccine.

4.14 Mange in Animals

Definition and Causative Agent

Mange (acariasis) is a highly contagious disease, mainly caused by mites belonging to three different families, *viz*., Sarcoptidae (Genus-*Sarcoptes*, *Notoedres*), Psoroptidae (Genus-*Psoroptes*, *Chorioptes*, *Otodectes*) and Demodecidae (Genus-*Demodex*). Based on their habits, these mites are classified in two categories, *i.e.*, burrowing (*Sarcoptes*, *Notoedres, Demodex*) and non-burrowing mites (*Psoroptes*, *Chorioptes*). These mites can be differentiated easily to the generic level based on morphological features. Mite infestation (*i.e.*, mange) is more common during winter, whereas spontaneous recovery occurs during summer. In fact, infestation becomes latent during summer and at the advent of winter they again migrate to general body surface. The major mange mites are listed below:

1. *Sarcoptes scabiei*: Cattle, buffalo, goats, equines, camels and pigs
2. *Psoroptes ovis*: Sheep and other ungulates
3. *Chorioptes* spp.: Cattle, goats, sheep, camel, equines, rabbits

. *Demodex bovis*: Bovines

Transmission

Female mites are larger than the males and lay eggs in the skin lesions. These eggs further develop into larva (3 pairs of legs), which further develop to the nymphal stage (4 pairs of legs). Finally, nymphs develop to the adult stage (male and female individual). These stages may spread to new healthy areas or even the new hosts. Since all the life cycle stages of these mites are present on host and thus infection to new host occurs through close contact. Entire life cycle of the mites can be completed within 2-3 weeks.

Pathogenesis

Mites pierce the host skin to suck the lymph and may also feed on young epidermal cells. Their activities produce a marked irritation on the skin, which causes intense itching and scratching of the body parts. Resulting inflammation of the skin causes exudation, which coagulates and forms a thick crust. The excessive keratinisation and proliferation of connective tissue leads to the thickened and wrinkled skin. There is concomitant loss of hair, which may be spreading to the wider areas.

Clinical Signs

General clinical signs observed in mange infected animals are scratching, alopecia, redness and discolouration of the skin. Further, the infected animal emits offensive odour, its skin pH becomes acidic, and skin thickness increases due to inflammatory reaction followed by exudation and scab formation. Secondary bacterial infection may aggravate the condition. In general, clinical signs of disease may appear any time between day 10 and 8 weeks after contact from the source of infection or animals.

Sarcoptes spp. infestation in bovines starts from head, neck and shoulder region but it may spread to the whole body.

Likewise, infestation of *Psoroptes* spp. may begin on the back and sides of animals but may become generalized as well.

Chorioptes spp. is able to infest the skin surface or epidermal debris and usually infest the lower body parts like legs, udder, scrotum, tail head and perineum. Choriopic mange is the most common infestation in cattle and equine. The disease produced (*i.e.*, barn itch) by these mites is less pathogenic in comparison to psoroptic and sarcoptic mange infestation.

Otodectic mange has similar morphology as *Psoroptes ovis* and they are mainly infesting the ear canal but sometimes may infest ear pinnae as well. Clinical signs may include rubbing and scratching of the ear, head shaking, depression, excessive exudation, and haematoma of the ear.

Demodectic mange infestation is observed mostly in immunocompromised hosts. The clinical disease - also known as red mange - is very often seen in dogs but it may also be seen in other hosts species as well. Clinical signs in animals may include papules on the skin to large size nodules and excessive loss of hair. Secondary bacterial and yeast infections may aggravate the condition, and severely infested animals may die.

Lesions

Piercing of mites may results in oozing of serum from the skin. The most common lesions in mange infestation include thickening of skin, loss of hair, papules, pustules and crust formation on the skin. Ear canker and bronze discoloration of skin are produced by the specific group of mites.

Diagnosis

Microscopic examination of skin scraping is helpful in the diagnosis of mange. Collection of scrapings from infected part is important to reach at confirmatory diagnosis. Samples are collected carefully till the oozing of blood from blood capillaries and examined after digestion (boiling) in 10 percent potassium hydroxide (KOH) or sodium hydroxide (NaOH) solution. After digestion, the sediment is examined for the presence of eggs, larvae, nymphs or adult mites. Morphological differentiation of different genera of mites infecting livestock is possible. Serological (ELISA) and molecular tests (PCR assay and genome sequencing) are also helpful in the diagnosis of mange infestation.

Differential Diagnosis

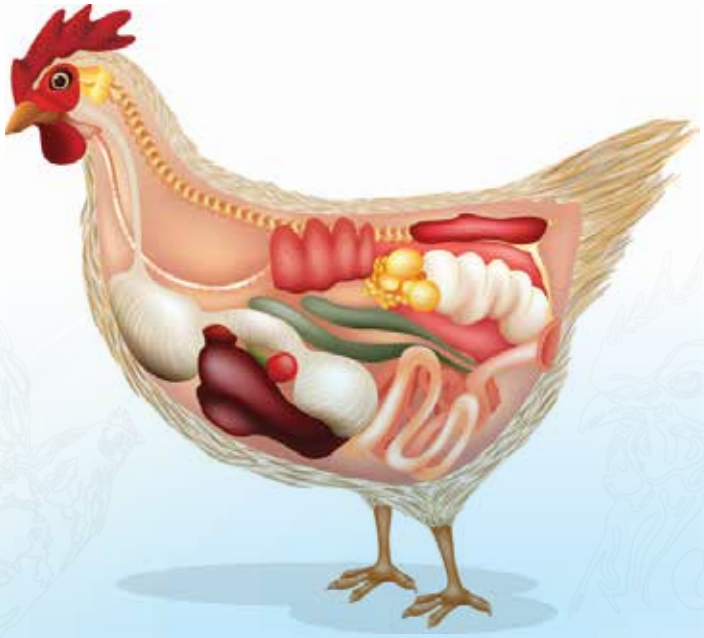
Flea bite allergy, bacterial pyoderma, atopic dermatitis, autoimmune diseases, dermatophytosis/ ringworm and other fungal infection may be differentiated with mange infestation for using an effective treatment.

Treatment and Control

After confirmatory diagnosis, systematic treatment of animals using effective acaricides at the appropriate interval is necessary. The most preferred treatment is the use of macrocyclic lactones (ivermectin @ 200-300 µg/kg BW by S/C route; or moxidectin @2.5mg/ kg BW by S/C route, 4 week apart for 2 occasions). Treatment needs to be repeated after 14-day interval for 2-4 occasions. Other effective drug in common use is Amitraz (topical application of 0.0025 percent solution) at 1- to 2- week interval for 2-6 occasions. Synthetic pyrethroids or organophosphate compounds are also in use. Pour-on preparations of different drugs are also available in the market. Lime sulphur application/dips at weekly interval is also effective in young animals. Before topical application of drugs, clipping of hairs and removal of crust from the skin must be undertaken. To prevent secondary bacterial infection, use of antimicrobial medicines is required.

CHAPTER- 5

GUIDELINES FOR POULTRY DISEASES



5.1 Preamble (Current status of poultry industry in India)

Poultry production in India has taken a quantum leap in the last few decades, emerging from conventional farming practices to commercial production systems with state-of-the-art technological interventions. With a production of 851.81 million birds, India continues to be in second position in egg production and fifth position in broiler meat production in the world. The Indian poultry market was valued at USD 28.18 billion in 2022. Aided by the increasing popularity of online services and growing online food delivery channels, the market is expected to witness a compound annual growth rate (CAGR) of 8.1% during 2023-2028 and projected to reach USD 44.97 billion by 2028 and contributing 1.2% of India's gross domestic product (GDP).

Egg production in the country has increased from 78.48 billion Nos. in 2014-15 to 129.60 billion Nos. in 2021-22 with a CAGR of 7.4% per annum. The per capita availability of egg is at 101 eggs per annum as per Basic Animal Husbandry Statistics-2023 (DAHD, Government of India). In India, exotic commercial layer strains, *viz*., Babcock, Bovans, Hyline and Lohmann are contributing nearly 85 percent total egg production. The birds are totally in cage system of rearing for up to 100 weeks. White shelled eggs are predominating in Indian egg market.

Indian broiler meat production is estimated at around 5 million metric tonnes (MMT) annually, and currently witnessing an annual growth of 6–7 percent according to trade estimates. The share of commercial broiler birds in total meat production is around 80-85 percent, and of backyard poultry 15-20 percent. The north-eastern states contribute majorly towards meat production through backyard poultry. The poultry market is primarily a wet market system in India and only 5 percent is processed market (10 major players) with 20 to 30 percent price premium. Total broiler placement in India ranges between 700 and 800 million birds per week depending upon the season. Vertical integration system of broiler rearing is highly predominant with an average flock size of 5,000 birds/flock. India's per capita consumption of broiler meat is just 3.35 kg/person per year. However, the consumption is low when compared to ICMR recommendations of 180 eggs/person a year and 11 kg meat/person a year. Therefore, the poultry industry has tremendous potential to grow rapidly in near future. In India, more than 50 percent of total egg and chicken meat are produced in South Indian states, *viz*., Tamil Nadu, Andhra Pradesh, Telangana and Karnataka. To meet out the increasing demands of chicken meat and eggs, expansion of poultry industry is taking place in a very rapid manner, but this expansion takes place only in already existing poultry producing regions which further increases the poultry density in such thickly poultry populated regions of the country. This further increases the risks of infectious diseases which lead to morbidity, mortality, loss of production and welfare issues. Hence it is of paramount importance to develop standard veterinary treatment guidelines for the following economically important diseases of poultry which cause significant economic losses in India:

5.2 Poultry Viral Diseases

5.2.1 Newcastle Disease (Ranikhet Disease)

Definition and Causative Agents

Newcastle disease (ND) – caused by Newcastle disease virus (NDV) - is one of the most devastating diseases of domestic poultry and wild birds causing a fatal respiratory, enteric and neurological disease leading to 100 percent morbidity and mortality. The etiological agent is avian paramyxovirus type-1 (APMV -1), a member of the genus *Orthoavulavirus* in the Paramyxoviridae family under the order Mononegavirales. The APMV-1 has one serotype and four pathotypes, *viz*., velogenic, mesogenic, lentogenic (mostly vaccine strains) and apathogenic enteric. Based on the genomic analysis, NDV is further classified in two main distinct genetic clades – class I (low virulent strains), contains single genotype with three sub genotypes; and class II (mostly high virulent) comprises at least 18 (I–XVIII) genotypes. Class II genotypes VII/ XIII viruses are the most prevailing viruses in India.

Transmission

Infected birds may shed the virus in exhaled air, respiratory discharges and faeces, contaminating the environment. Birds in the pigeon family can shed the virus intermittently for a year or more. Transmission can occur by direct contact with faeces and respiratory discharges/by inhalation of small infective particles produced from dried faeces or by contaminated feed, water, footwear, clothing,

tools, equipment, and the environment. The disease is very contagious. When the virus is introduced into a susceptible flock, virtually all the birds will be infected within two to six days. Viruses transmitted by the respiratory route may spread extremely rapidly, whereas viruses excreted in the faeces and transmitted by the faeco-oral route may spread extremely slowly.

Clinical signs

The clinical signs vary considerably with the pathotype of the infecting virus. As such, no signs can be regarded as pathognomonic of the disease. Infection with velogenic virus may result in per acute deaths. Typical cases exhibit marked depression, prostration, nerves symptoms such as torticollis, head twitching, oedema of the head, inappetence, increased respiration, progressive weakness and greenish diarrhoea. The appearance of shell-less or soft-shelled eggs is followed by complete cessation of egg laying. Mortality in susceptible flocks is >90 percent or higher. In mesogenic form, respiratory distress and coughing, followed by nervous symptoms are noticed. Mortality may occasionally reach 50 percent. The lentogenic virus causes mild respiratory symptoms, a sudden drop in egg production and reduction in feed intake. The mortality is negligible and complete recovery occurs within 1 to 8 weeks.

Lesions

Viruses causing respiratory disease may induce haemorrhagic lesions and marked congestion of trachea. Air sacculitis and thickening of the air sacs with catarrhal or caseous exudates are often observed. The intestinal lesions include pinpoint haemorrhages in the tips of the proventricular glands, dark red haemorrhagic ulceration with necrosis in the intestinal wall, enlarged and necrotic caecal tonsils, necrosis and haemorrhage in intestinal lymphoid aggregates, and splenic necrosis on the capsular surface. Laying hens infected with velogenic viruses usually reveal egg yolk in the abdominal cavity. The ovarian follicles are often flaccid and degenerate and may even show haemorrhagic stigmata.

Diagnosis

Diagnosis of ND comprises of isolation of virus in 9- to11- day-old embryonated chicken eggs and virus antigen identification by the haemagglutination inhibition test. Alternative methods based on molecular biological techniques like RT-PCR and real-time PCR are used for the detection and identification of NDV. In addition to RT-PCR, the nucleotide sequencing is used for the determination of the virulence of NDV

Differential Diagnosis

Avian Influenza, Infectious Bronchitis, Fowl Cholera.

Treatment

No specific treatment is available, but supportive treatment like spraying of antiviral disinfectant and giving disinfectant through drinking water also as per recommended dosages may be useful. Ethnoveterinary preparations and immunomodulators like Beta Glucans, vitamin E, and Se can also help.

Control

Effective Newcastle disease prevention needs a multifaceted strategy which includes stringent biosecurity, appropriate and timely vaccination, and management. In India, the commercial and backyard chickens are vaccinated with live lentogenic vaccines such as LaSota, F Clone, and recombinant vaccines, while mesogenic vaccines like R_2B, K, and inactivated vaccines at various stage of production cycle. New generation Vector-ND vaccines are also used in day-old broilers and layers either *in ovo* or subcutaneous. Usually, the conventional live vaccines are used by eye drop or mass application either in drinking water, or spray; mesogenic vaccines are applied by intramuscular route; and inactivated vaccines are administered by sub-cutaneous route. Further ND vaccination must ensure that >85 percent of the flock is sero-converted with sufficient titres, needed for herd immunity. The right application method includes withdrawal of water sanitizers one day before and after administration of drinking water vaccination. Disinfectant sprays should not be used 2 days after live ND vaccination. The storage conditions and consistency of inactivated vaccines are needed to be checked before administration. Regular sero-monitoring is necessary to assess the NDV antibody titres. To maintain the desired protective titres, revaccination (with LaSota in drinking water) is advised if the titre falls below 128(2^7).

Biosecurity

The common biosecurity methods include foot and vehicle dip/bathing along with disinfection facilities for the visitors, farm workers, and service providers, which should be provided at the farm entrances. Adequate floor space and ventilation should be provided to minimize the stress. Feral pigeons and other wild birds can asymptomatically transmit NDV to poultry. Therefore, it is necessary to make birdproof feed mills, water tanks, and chicken sheds. The ND virus is usually transmitted by contaminated shoes, clothes, and equipment, including trucks, crates, egg fillers, containers, and tools for vaccination and beak trimming. Therefore, it is important to follow the right decontamination protocols before introducing such items into the farm.

5.2.2 Infectious Bronchitis (IB)

Definition and Causative Agent

Avian infectious bronchitis (IB) – caused by infectious bronchitis virus (IBV) - is a highly contagious, acute, and economically important viral disease of chickens that causes respiratory, renal and reproductive disease characterized by moderate mortality rates, and severe losses on the productive performance of poultry flocks. The IBV is placed in group-3 of genus *Coronavirus* of the order Nidovirales. Many different IBV serotypes/variants are recognized on the basis of antigenic variation, out of which IBVs of economic importance reported in India include Massachusetts, 793B (4/91), Indian nephropathogenic variants and QX variant.

Transmission

IBV is considered the most contagious and infectious as only a few virus particles may initiate an infection. Naturally infected chickens and those vaccinated with live IBV may shed virus intermittently for up to 20 weeks after infection. Virus is shed via both the respiratory tract and the faeces. Direct air-borne transmission of virus from the respiratory tract is probably the most common method. However, transmission through infected faeces is also important, and virus spread might happen by mechanical means such as on clothing, poultry crates and equipment.

Clinical Signs

Mortality may be as high as 25 percent or more in chickens less than 6-week-old, and usually negligible in chickens older than 6 weeks. The characteristic respiratory signs in young chickens are gasping, coughing, sneezing, tracheal rales and nasal discharge. Wet eyes and dyspnoea may be seen, and facial swelling may also occur with concurrent bacterial infection of the sinuses. In broiler chickens, IBV infection is a major cause of poor feed conversion, reduced growth rate, and condemnation of meat at processing. Apart from respiratory symptoms, broiler chickens infected with one of the nephropathogenic viruses show signs of depression, ruffled feathers, huddling, wet droppings, and increased water intake. In layers, IBV damages reproductive organs permanently causing reduced fertility and hatchability, delayed sexual maturity and increased rate of non-layers. The egg production may drop by as much as 70 percent, and deterioration in egg qualities like watery albumen, mishappened/discoloured eggs and soft-shelled eggs are the other manifestation due to permanent damage in oviducts.

Lesions

Infected chickens have serous, catarrhal, or caseous exudates in the nasal passages, sinuses and trachea. The air sacs may appear cloudy or contain a yellow caseous exudate. A caseous plug may be found in the lower trachea or bronchi of dead chicks. Small areas of pneumonia may be observed around the large bronchi. Nephropathogenic infections produce swollen and pale kidneys with the tubules and ureters often distended with urates. The relative kidney weight and kidney asymmetry are increased. Despite of lack of kidney gross lesions, microscopic changes of nephritis may still be present. Associations with myopathy and proventriculitis are also reported. Fluid yolk material may be found in the abdominal cavity of chickens that are in production. Gross changes in the oviduct caused by early infection may vary from the presence of a continuous patent but underdeveloped structure to a blind sac projecting forward from the cloaca. Fibrinous pericarditis, perihepatitis and caseous air sacculitis will be observed in case of *E. coli* complications.

Differential Diagnosis

Newcastle Disease, avian metapneumovirus, infectious laryngotracheitis, mycoplasmas, *Avibacterium paragallinarum*, and *Ornithobacterium rhinotracheale.*

Diagnosis

Diagnosis of IB is based on the clinical history, lesions, finding sero-conversion or rising antibodies titres using ELISA; determining the serotype of the virus include virus isolation and neutralization tests, which are now being replaced with rapid, specific and confirmatory test like RT-PCR and RFLP.

Treatment

There is no treatment for this disease. Secondary bacterial infections are common and treatment with antibiotics may reduce losses from these infections. Reducing the protein concentrations in feed and providing electrolytes and essential oils in drinking water may assist in fighting the outbreaks caused by nephropathogenic strains.

Control by Vaccination

The only practical means of controlling IB is vaccination, the multiplicity of serotypes identified in the field presents a challenge in designing an effective vaccination program. In India, the common IBVs used in most vaccination programs are the Massachusetts (H120 and Ma5 live), M41 (Inactivated), 4/91 (live) and nephropathogenic IBV(Inactivated). Vaccination programmes and procedures may differ in different regions of India depending on local conditions. Usually in egg laying birds, live vaccines are used in first week onwards at different intervals either by eye drop or in drinking water. Inactivated vaccines are used at the point of lay. In broilers, live vaccines are used in day-old. Usually one Mass type vaccine (H120 or Ma5) followed by boosting with one variant IB vaccine (4/91) are recommended for broader protection of birds against variant IBVs in India.

Biosecurity

Basic biosecurity management practices such as limited controlled site access, separate footwear and equipment for each site/house, and footbaths at the entrance to sites/houses all minimize the risk of introducing the IBV. Prevention is best achieved through good management by creating dust-free environments, reduced ammonia levels, good ventilation and optimal temperature. The virus is easily destroyed by heat and ordinary disinfectants. In young chickens, it is helpful to increase the brooder temperature and to optimise environmental conditions. The downtime between successive chicken flocks must be maximized (a minimum of 10 days is recommended).

5.2.3 Avian Influenza

5.2.3.1 Highly pathogenic avian influenza

Definition and Causative Agents

Highly pathogenic avian influenza (HPAI) - also known as fowl plague - is an acute, generalized, highly infectious and dynamically evolving disease of domestic poultry and wild birds causing huge morbidity and mortality. The recent wave of HPAI in Asia, Africa, Europe and America has made the global impact of this transboundary animal disease. The latest outbreaks are beyond the scope and resources of a single region to control. HPAI is caused by infection with antigenically, genetically and biologically diverse influenza A viruses of the family Orthomyxoviridae. They are differentiated into type A, type B, type C and type D influenza viruses on the basis of the identity of the major internal protein antigens, the nucleoprotein (NP) and matrix (M) proteins. All avian influenza viruses are classified as type A, which is typically spherical to pleomorphic but can be filamentous. The RNA is single-stranded and negative sense and further subtyping of this is based on the antigenicity of two surface glycoproteins, hemagglutinin (HA) and neuraminidase (NA). Currently, 16 HA and 9 NA subtypes have been identified among influenza A viruses. Each virus has one H and one N antigen, apparently in any combination; all subtypes and the majority of possible combinations have been isolated from avian species, but only H5 and H7 subtypes were found to cause HPAI in domestic poultry, wild birds and mammals. The avian influenza virus is classified into HPAI and LPAI based on their amino acid sequences in their HA cleavage site. The primary difference between LPAI and HPAI virus is local versus systemic replication, respectively. The definition adopted by World organization for Animal Health (WOAH) for HPAI is an infection of poultry caused by any influenza A virus of the H5 or H7 subtypes or by any AI virus with an intravenous pathogenicity index (IVPI) greater than 1.2 (or as an alternative at least 75 percent mortality) or presence of multiple basic amino acids at the cleavage site of the precursor haemagglutinin molecule (HA0).

Transmission

The wild birds of order *anseriformes* (duck family)

and *charadriiformes* (gull family) are acting as the natural reservoirs of the AIVs. The virus is usually introduced in one country through the wild migratory birds to the domestic poultry flocks. Infected birds excrete virus from the respiratory tract, conjunctiva, and faeces. Therefore, likely modes of transmission include both direct contact between infected and susceptible birds, and also by an indirect contact through contaminated fomites such as boots, wheels, straw and contaminated water.

Clinical Signs

The clinical symptoms showed by HPAI were characterized by sudden onset of severe illness, rapid death, high morbidity and mortality reaching up to 100 percent in 3-10 days. In per acute cases, the birds were found dead without any clinical symptoms. However, the birds surviving for 3-7 days exhibit depression, prostration, ruffled feathers, conjunctivitis, cyanosis of head, combs, wattles and shanks, facial oedema/ swelling, and decrease in feed and water consumption. Respiratory signs observed include severe respiratory distress, rales, coughing, snicking, sneezing and dyspnoea. Nervous disorders such as tremors of head and neck, torticollis, opisthotonus, nystagmus, paresis and paralysis were also noticed. Layer birds experienced precipitous drop in egg production within six days usually accompanied by an increase in numbers of poor-quality egg.

Gross Lesions

In highly pathogenic viruses, there may be no prominent lesions in the birds that die very acutely, before development of gross lesions, however, the parenchyma of the visceral organs and skin might show variety of oedematous, haemorrhagic and necrotic lesions in the affected birds. Haemorrhagic lesions were commonly observed in the coronary fat and on the epicardium, serosa of the proventriculus and gizzard, and within the pectoral muscles. Initial changes include oedema of the head with swollen sinuses and cyanotic combs and wattles. As the disease progresses, subcutaneous oedema of head, face, upper neck and feet, generalized congestion of the carcass, petechial/ecchymotic haemorrhages of the shank, conjunctiva, skeletal muscles over breast and thigh, surface of the heart, abdominal fat, and mucosa of the proventriculus, mucous/muco-haemorrhagic tracheitis, pale and necrotic pancreas, as well as atrophy of the thymus and bursa of Fabricius.

Differential Diagnosis

The HPAI should be differentiated from other diseases which cause acute mortality and haemorrhages such as Newcastle disease and fowl cholera.

Diagnosis

The diagnosis of HPAI includes clinical inspection, postmortem examination for the identification of lesions in tissues and organs and laboratory tests on serum, tracheal swabs and tissue samples to detect the virus or specific antibodies against it. The clinical signs vary constantly and are mostly nonspecific. So, the isolation of virus from clinical specimens can often aid significantly in the accurate diagnosis of this disease in poultry and other avian species. RT-PCR can detect the HA gene, even in specimens that were negative for virus by standard testing in eggs. This can be combined with sequencing of the HA cleavage site, which offers a sensitive way to access the virulence potential of avian influenza virus.

Disease Management

A quick response is vital for controlling HPAI outbreaks. In addition to national notification requirements, HPAI viruses and LPAI viruses that contain H5 or H7 must be reported to the WOAH by member nations. Veterinarians and state governments who encounter or suspect a HPAI should follow the DAH&D's latest Avian Influenza action plan on guidelines for proper containment operations.

5.2.3.2 Low Pathogenic Avian Influenza

Definition and Causative Agents

Low pathogenic avian influenza virus (LPAIV) generally causes asymptomatic infection or a mild disease, but without involvement of co-infections. The H9N2 subtype is the widely circulated LPAI type in the world since its first detection from turkeys in Wisconsin in 1966.

Transmission

The virus is usually introduced in one country through the wild fauna, and to the domestic flocks either by a direct contact between the wild birds and the domestic ones or by an indirect contact through contaminated fomites such as boots, wheels, straw and by contaminated water. The virus could spread by wind-borne route in very closely situated farms

and also by flying insects that get contaminated with infected faeces. Secondary spread was principally due to the movement of personnel and equipment between farms.

Clinical Signs

The disease produced by LPAI may be inapparent or slight disease at one extreme. Most frequently subclinical disease associated with mild clinical manifestations including respiratory diseases, decrease in egg production, diarrhoea and renal syndrome. At the other extreme, infections of LPAI viruses may be associated with severe disease and high mortality when they are associated with secondary bacterial/ viral infections.

Gross Lesions

In LPAI infections, mild lesions were observed in the sinuses, respiratory, digestive, reproductive tracts, and kidneys. The frequently observed lesions were swelling of the periorbital tissues and sinuses, typical respiratory discharge, extensive hyperaemia of the respiratory system followed by exudation and cast formation at the tracheal bifurcation extending into the secondary bronchi, thickened air sacs, fibrinous yolk peritonitis, salpingitis, oophoritis, nephritis, oedema of the mesentery of the oviduct, and pale swollen kidneys, *etc.*

Differential Diagnosis

Newcastle Disease, Infectious Bronchitis, Fowl Cholera.

Laboratory Diagnosis

Biosafety Level 2 facilities, practices and procedures are recommended for diagnostic, research and production activities utilizing contemporary, circulating LPAI strains. The diagnosis of AIV includes clinical inspection, postmortem examination for the identification of lesions in tissues and organs, and laboratory tests on tracheal swabs, blood and tissue samples to detect the virus or specific antibodies against it. The isolation of virus from clinical specimens can often aid significantly in the accurate diagnosis of infectious disease in poultry and other avian species. The virus isolation remains the gold standard for diagnosis and is indispensable for virus characterization, yet rapid laboratory confirmation of suspected avian influenza in routine diagnostic laboratories is usually performed by RT-PCR detection of viral nucleic acids in specimens. RT-PCR methods allow for sensitive and specific detection of viral nucleic acids and increase the diagnostic sensitivity for many viral pathogens compared to culture or antigen detection methods.

Disease Management

Biosecurity

Effective biosecurity programs prevent contact of poultry with wild/migratory/aquatic birds, their excretions, and other materials that could contain viral particles. Routine biosecurity measures must be effective enough to prevent an outbreak. Every poultry operation is different and should develop a biosecurity plan that identifies its vulnerabilities for virus introduction and puts into place programs that mitigate these risks. Movement of birds, people, equipment, feed and materials coming onto a poultry facility must be strictly controlled, because if any of these is contaminated can infect the commercial poultry. Restrict access to only those people essential to the farm's operation with a change into farm dedicated footwear, clothing and hairnets. The vehicles used on farms should be dedicated for use only on the farm. The movement and marketing of old hens must be strictly controlled. On-farm sales of eggs and end-of-lay hens should not be arranged. The egg trays and bird crates used for product sales to traders should not be returned to the farm or should be fully cleaned and disinfected prior to return to the facility. Use caution and strict control plans when utilizing third-party contractors shared by commercial egg layer companies for vaccinations, moving old hens, pullets, and manure, as these services played a critical role in the spread of LPAI. Live bird markets were found involved in several past influenza outbreaks. Live bird markets are often unhygienic and not regulated. It is common that birds from multiple farms are in close proximity, increasing the possibility of genetic shift and spread of the virus. Movement of manure and dead birds pose a significant risk for spreading the virus. Flocks infected with LPAI shed high levels of infectious virus in tissues and manure. When workers and manure handling equipment move between farms, a complete cleaning and disinfection is required. Composting manure and dead birds for 10 days at 60°C is an effective way to inactivate influenza virus. Regular disinfection in and outside farms with products like Virkon-S and other broad-spectrum

disinfectants available commercially with dosages as per their label claims, must be conducted.

Vaccination

Inactivated vaccines are available in different countries for control of LPAI. Government of India has also allowed production of inactivated vaccines by Indian manufacturers only and may be available commercially by the end of 2024. It may require initial vaccination during first week of age in layers and breeders with subsequent boosters after every 8-10 weeks along with control of secondary bacterial infections due to *Mycoplasma* and *Escherichia coli* besides following strict biosecurity precautions mentioned above. While vaccination may not prevent infection, properly vaccinated birds are protected from the mortality, respiratory disease, and egg production losses associated with LPAI infection as observed in countries following vaccination. Vaccinated birds are more resistant to infection after a field challenge; and shed less virus into poultry farms and environment.

5.2.4 Infectious Laryngotracheitis

Definition and Causative Agent

Infectious laryngotracheitis (ILT) is a highly contagious, acute, upper respiratory disease of chickens, having worldwide distribution affecting growth and egg production leading to significant economic losses. The etiologic agent is ILT virus (ILTV), also designated as Gallid herpesvirus-1 (GaHV-1, ILT-like viruses) which belongs to the subfamily Alphaherpesvirinae of the family Herpesviridae.

Transmission

The virus gets introduced into a flock by direct contact with respiratory exudates or indirect/ mechanical transmission of contaminated equipment, litter, feed bags, feathers, vehicles, dust, footwear, clothes, and movement of people. Darkling beetles and mealworms also act as a source of infection to the birds. Dogs and cats retrieving dead bird carcasses from affected poultry houses also spread the infection. Wind-borne transmission of ILTV is also found between commercial poultry operations.

Clinical Signs

The incubation period, following the introduction of ILTV into a flock, varies from 6 to 12 days. The morbidity due to ILTV is 50-100 percent and mortality usually 10-20 percent but sometimes may be up to 70 percent. Typically, there is a sudden increase in daily mortality in a single house on a farm. Often, birds in one area of the house show clinical signs and die, and the disease spreads relatively slowly through the other birds in the house. Some birds even in good body condition may die due to difficulty in breathing with extension of the neck and gasping in an attempt to inhale. Affected birds also exhibit sinusitis, nasal discharge, swollen orbital sinuses, and purulent conjunctivitis with frothy exudate at the canthus of the eye. There is also gurgling, rattling, and coughing when birds try to expel obstructions in the trachea. Clots of blood may be coughed up and found on the floor and walls of the house.

Postmortem Lesions

The postmortem lesions are limited to the upper respiratory tract and consist of haemorrhagic tracheitis with stains/blood clots/casts/diphtheritic membrane throughout or part of its length or is filled with bloodstained mucus. The primary bronchi may also be affected and sometimes yellow caseous exudate (cheesy plug) is also noticed. Apart from tracheal haemorrhages, mucoid rhinitis may also be present.

Differential Diagnosis

Newcastle disease, avian influenza, infectious bronchitis, infectious coryza, Fowl pox (wet pox), chronic respiratory disease, and vitamin A deficiency.

Diagnosis

Infectious laryngotracheitis in chicken can be tentatively diagnosed based on the clinical signs and necropsy findings. The suspected cases are subjected to laboratory diagnosis by conventional and molecular diagnostic tests. The conventional methods include histopathology as well as virus isolation in embryonated chicken eggs and cell culture. Detection of pathognomonic syncytial cells and intranuclear inclusion bodies in the trachea, eyelid, and lung tissues using histopathology is routinely practiced. Molecular techniques like Reverse transcription-PCR (RT-PCR) and quantitative real-time PCR (qRT-PCR) are widely used due to their higher diagnostic sensitivity and accuracy.

Treatment

No effective treatment options are available for ILT, however, various Ethnoveterinary products like essential oils along with spraying of antiviral disinfectants may help.

ILT is usually prevented by a combination of biosecurity measures and vaccination.

Vaccination

- For layers and breeders, apply one dose of CEO vaccine at 6 to 8 weeks by eye drop followed by a booster vaccination of CEO vaccine at 12 to 15 weeks by eye drop. Do not introduce ILT live vaccine (CEO) in clean areas as well as where multi-age birds are kept at one location.
- The eye drop method is considered comparatively safer and gives more protection than mass application methods like drinking water and spray administration.
- A superior ILTV vaccine must contain a titre of $>10^2$ plaque-forming units/ml to induce adequate immunity.
- The highest protective immunity is attained from 15 to 20 weeks post-vaccination, which may last over a year.
- However, the CEO vaccines have undesirable properties of reversal to the virulent form following bird-to-bird passages leading to vaccinal laryngotracheitis.
- To overcome the disadvantages of CEO vaccines, recombinant ILT vaccines may be used in the field. These vaccines can be administered *in ovo* at day 18^{th} of embryonating period or subcutaneous route during one-day of age.

Biosecurity

- Following biosecurity measures may be adopted for safety.
- Isolate affected birds.
- Use good biosecurity measures to reduce exposure, especially during movement of birds out of pullet houses and into or out of layer houses.
- Sanitation of people, equipment, and vehicles should be practiced to minimise the risk of carrying infected material into contact with the flock.
- Mixing of birds of different ages should be avoided.
- The ILT virus can be mechanically transmitted or carried from one site to another through flies and rodents, therefore, ongoing efforts should be made to control them.
- Wild birds and pets should be prevented from entering poultry facilities.
- Backyard birds should not be maintained near commercial poultry operations.

Control

- Revaccination of birds during an outbreak will prevent the transmission of field ILTV stains.
- Thorough cleaning and disinfection of facilities, and extended downtime before repopulation is a more effective method.
- Proper carcass disposal (incineration, deep burial and composting) is an important central measure in controlling the natural spread of ILTV.

5.2.5 Infectious Bursal Disease

Definition and Causative Agent

Infectious bursal disease (IBD) is an acute, highly contagious immunosuppressive viral disease of young chickens, seen worldwide. It can present as a clinical or subclinical disease, but immune suppression and related secondary infections are typically seen. Immunosuppression can lead to vaccination failures, *Escherichia coli* infections, gangrenous dermatitis and inclusion body hepatitis-anaemia syndrome. The virus responsible for infectious bursal disease (IBD) is a member of the *Avibirnavirus* genus, within the family Birnaviridae.

Transmission

IBD is highly contagious. The affected birds excrete the virus in faeces as early as 2 days post-infection (thus shortly before the first clinical signs) and for at least 10–14 days. Transmission in a flock occurs mainly via the faecal–oral route. Mechanical vectors are likely to play a part in the spread of the virus. Meal worms and litter mites are found infective for up to 8 weeks.

Clinical signs

The clinical signs include depression, white watery diarrhoea, soiled vents, vent picking, inflammation of the cloaca, anorexia, reluctance to move, closed eyes, and death. Mortality commences on the third day of infection, reaches a peak four days after start of mortality, then drops rapidly, and the surviving chickens recover a state of apparent health after five to seven days.

Postmortem lesions

The gross lesions consist of haemorrhages in the thigh and pectoral muscles, proventriculus and gizzard junction, hypertrophic, hyperaemic and oedematous bursas.

Treatment

No effective treatment is available for IBD, however, homeopathic preparations, ethnoveterinary preparations and immuno-stimulants such as vitamin E, selenium, and electrolytes can be given to reduce the mortality. Virucidal disinfectants can be sprayed as per dose to reduce the spread.

Control

Regular biosecurity and personal hygiene of farm workers must be adhered to, along with vaccination.

Vaccination

It is always advisable to vaccinate against IBD after checking maternal antibody levels in day-old chicks (20 chicks) from each hatchery. Vaccination practices differ according to the type of birds and local husbandry conditions in India. Usually, immune complex vaccines are administered *in ovo* at hatchery on 18th day of incubation. Vectored vaccines are given at day-old by subcutaneous route. Conventional live IBD vaccines (intermediate and intermediate plus) are administered either in first or second week of age and followed by booster during third week of age in egg laying birds. Similarly, the inactivated vaccines are also administered during first or second weeks of age in egg laying birds. Apart from that, inactivated vaccines are administered at regular interval during laying period to ensure sufficient maternal antibody titre in breeder birds.

Biosecurity

The best method of IBD prevention is all-in/all-out farming methods, along with an extended down time. Very high resistance of IBDV to physical and chemical agents accounts for persistence of the virus. Hence, rigorous disinfection protocols should be implemented by using very effective disinfectants like Virkon-S @ 5 g/litre (oxidizing type) for aerial disinfection in the presence of birds. Prior to cleaning, all insects and pests (*e.g.*, rats and mice) must be eliminated as soon as the farm premises are empty. Old bedding and faeces must be discarded and composted. The farm buildings, immediate surroundings and farm equipment must be dry-cleaned first to eliminate all dust, and then washed using hot water (60°C) with a detergent, at a pressure of 80 bar to 150 bar. A second disinfection of the full premises must be performed before the introduction of the chicks. Feed silos must be emptied completely and cleaned inside and outside. The infective materials/litter and carcasses should be disposed by incineration, composting, or deep burial.

5.2.6 Marek's Disease

Definition and Causative Agent

Marek's Disease (MD) is a highly contagious viral infection that predominantly affects chickens. The disease causes morbidity of 10-50 percent and mortality up to 100 percent. In late Marek's, the mortality can extend up to 50 weeks of age. Marek's disease virus (MDV) is a member of the genus *Mardivirus* within the subfamily Alphaherpesvirinae. Within the genus *Mardivirus*, there are three closely related species, *viz.*, Gallid herpesvirus-2 (MDV-1) that represents all virulent Marek's disease virus strains, and is further divided into pathotypes, designated as mild (m), virulent (v), very virulent (vv), and very virulent plus (vv+); Gallid herpesvirus-3 (MDV-2) and Meleagrid herpesvirus-1 (turkey herpesvirus, MDV-3) represent avirulent virus strains isolated from chickens and turkeys, respectively, and are commonly used as vaccines against Marek's disease.

Transmission

The route of infection is usually respiratory, and the disease is highly contagious being spread by infective feather-follicle dander, fomites, *etc.* Infected birds remain viraemic for life. Vertical transmission is not considered to be important.

Clinical Signs

Typically, affected birds show paralysis of legs/wings/neck, loss of weight, grey iris or irregular pupil, vision impairment, and follicular tumours. Affected birds are immunosuppressed and, as a consequence, are more susceptible to other infectious diseases.

Postmortem Lesions

The postmortem lesions associated with MD include diffuse or nodular lymphoid tumours in various organs, particularly the liver, spleen, gonads, heart, lung, kidney, muscle, and proventriculus. Enlarged feather follicles (commonly termed skin

leukosis) noted in broilers after defeathering during processing and are a cause for condemnation. There is enlargement of vagus, brachial, and sciatic nerves with loss of cross-striations.

Treatment

There is no specific treatment available for MDV. Hence, regular grading and culling of the infected chicken can minimize the disease transmission. Immuno-stimulation strategies by supplementation of vitamin E, selenium, and mannan oligosaccharides (MOS) in the feed can help to minimize the loss.

Control

Vaccination–Vaccination is the central strategy for the prevention and control of MD after disinfection. In commercial flocks, it is virtually impossible to eliminate all MD virus exposure, so vaccination is used to help minimize losses. Vaccines are typically applied once in the hatchery to day-old chicks. In India, bivalent vaccines consisting of HVT (with 1,500 PFU) and either the SB-1 (with 2,500 PFU) or 301B/1 strains (cell associated) of MDV are used to provide additional protection against challenge with virulent Marek's disease virus isolates. Proper handling of vaccine during thawing and reconstitution is crucial to ensure that adequate doses are administered.

Immunosuppressive diseases such as infectious bursal disease, chicken infectious anaemia (chicken anaemia virus), and reovirus infections can prevent adequate immunological response to MD vaccination, or favour development and maintenance of latent MD field virus in the chicken. Hence, appropriate vaccination and control strategies for immunosuppressive diseases should be practiced to minimize MD susceptibility.

Biosecurity

Proper disposal of litter, feathers and dead carcasses by incineration, composting and deep burial along with proper cleaning, disinfection, biosecurity and all-in all-out system is always advisable.

Shedding of the MDV in dead skin and feather follicle epithelial cells contributes virus to the dust found in chicken houses wherein virus can remain infectious for many months. Young chicks become infected as they breathe the virus-containing dust, therefore, the first intervention in MD prevention should be to limit exposure of young chicks to infectious dust. This can be achieved by controlling the dust generation in the farm by regular burning of feathers/dandruff by using flame gun and thorough cleaning and disinfection of brooder houses and equipment before placement of new chicks.

5.2.7 Fowlpox

Definition and Causative Agent

Fowlpox is a slow spreading disease accompanied by the development of discrete, nodular, proliferative skin lesions on the non-feathered parts of the body (cutaneous form), or fibrino-necrotic and proliferative lesion in the mucous membrane of the upper respiratory tract, mouth, and esophagus (diphtheritic form). The etiologic agent of fowlpox is the fowlpox virus (FPV)under the genus *Avipoxvirus;* family *Poxviridae*.

Transmission

Fowlpox virus is transmitted by direct contact, inhalation or ingestion of dust/aerosols, or mechanically by biting insects.

Clinical Signs and Lesions

The disease may occur in one of the two forms: cutaneous or diphtheritic, or both. Its course in individual birds varies between 10 and 14 days and on a flock basis, it generally lasts in 6 to 10 weeks. The chickens infected with the fowlpox virus will show poor growth, poor feed conversion and a precipitous fall in egg production. Mortality will seldom be marked if the lesions are limited to the skin. However, death may occur if the oral cavity or air passages become involved.

Cutaneous lesions appear on the un-feathered skin of the head, comb, wattle, earlobes, neck, legs, and feet. First, there is a papule, which rapidly progresses through the vesicle to pustule, and finally to the crust or scab stage. In most outbreaks, the terminal scab stage is present on some of the birds presented for diagnosis. After about 2 weeks, the scab drops off, and a healed lesion is left, which may or may not leave a scar. Multiple lesions usually develop and often coalesce. Lesions in various stages of development may be found on the same bird. Cutaneous lesions on the eyelids may cause complete closure of one or both the eyes. Lesions are prominent in some birds and cause irritation and may significantly decrease flock performance, but these flocks generally return to normal production after recovery.

In the diphtheritic form (wet pox), slightly elevated white opaque nodules are observed in the upper respiratory and digestive tracts. These nodules coalesce to form raised yellow plaques on the mucous membranes. Most lesions are found in the mouth, but others are present in the larynx, trachea, and oesophagus. These lesions can give rise to inappetence, depressed droopy attitude dyspnoea, with sneezing and cough. Lesions in nares can cause nasal discharge. Those on the conjunctiva cause ocular discharge, and in rare cases, result in blindness. These symptoms are similar to those of other respiratory diseases and may cause confusion in making a diagnosis.

Differential Diagnosis

ILT, Newcastle disease, avian influenza, *Mycoplasma gallisepticum* (MG), and vitamin A deficiency may complicate an initial diagnosis of wet pox because of similar respiratory lesions.

Diagnosis

Diagnosis can be made on the basis of the clinical signs. The presence of cutaneous lesions is suggestive of fowlpox. Microscopic examination of lesions reveals intracytoplasmic eosinophilic inclusion bodies. Material can be scraped from the lesions, and smears made on glass slides. Using the appropriate stain, the virions (Borrel bodies) can be seen under the light microscope. The diphtheritic form is more difficult to diagnose on the basis of clinical signs alone. The lesions are adherent, and if removed leave ulcers. This fact helps in differentiating it from infectious laryngotracheitis and avitaminosis A. However, in both forms, confirmation can be made by detection of the virus from the lesion. Ground-up scabs inoculated onto the chorioallantoic membrane of 9-12 day-old embryonated chicken eggs produce characteristic pock lesions. Microscopically, inclusion bodies can be identified in pock lesions. PCR is used to differentiate field and vaccine strains of the virus. The genomic profiles of field isolates and vaccine strains of fowlpox virus can be compared by restriction fragment length polymorphism.

Treatment

- No effective treatment options are available for fowlpox on commercial farms, however, the alternative medicines like homoeopathic preparations and ethnoveterinary preparations may help.

Control

- Prevention is by vaccination of the birds using live fowlpox vaccine.
- Fowlpox is usually prevented by a combination of vaccination and biosecurity measures. Ensure that infected birds have access to clean water and nutritious feed to maintain their strength and immunity. Tripple salt antiviral preparations in spray is also helpful.

Vaccination

- The fowlpox vaccine is usually administered as a live vaccine, either through wing-web or intramuscular route.
- A suitable potency for an attenuated live fowlpox vaccine is in the region of 10^5 EID_{50} (50 percent embryo infective dose) per ml or $\geq 10^2$ $TCID_{50}$ of virus per dose.
- Vaccination can be done in layer and breeder chickens two to three times depending upon the disease intensity in the farm and may use live pigeonpox vaccine, if outbreaks occur in early age.
- The first vaccine is recommended at about 4-6 weeks old and booster can be given at 5 to 7 weeks intervals, depending on the specific vaccine used and the recommendations of veterinarians or poultry health specialists.

Biosecurity

- Immediately isolate infected chickens from rest of the flock to prevent further spread.
- Clean and disinfect all equipment, including feeders, waterers, and tools, regularly.
- Ensure proper disposal of bedding and manure to minimize viral contamination.
- Fowlpox can spread through mosquitoes and other biting insects. Implement measures to control insect populations around the chicken coop and utilize insecticides as needed.
- Ensure that chicken housing areas are well-maintained and free from standing water areas where insects can breed.
- Maintain good hygiene in the poultry premises.

5.2.8 Chicken infectious anaemia

Definition and Causative Agent

Chicken infectious anaemia - caused by chicken anaemia virus (CAV) - is highly contagious disease of young chickens, characterized by severe anaemia, generalized lymphoid atrophy and increased mortality, with concomitant severe immunosuppression enhancing susceptibility to

other infectious agents and diminished vaccines response leading to severe economic losses. Subclinical immunosuppression by CAV in chickens older than 2 week of age is now recognized as economically important in production. The CAV belongs to the family Anneloviridae and the genus *Gyrovirus*. The CAV is classified into four distinct genotypes (I, II, III, IV and V) based on variations in the VP1 gene.

Transmission

The virus spreads both horizontally and vertically. Horizontal transmission of CAV is by the faecal-oral route, perhaps by the respiratory route, and through infected feather follicle epithelium. In vertical transmission, seronegative hens become infected and continue to remain infective until neutralizing antibodies develop. Chicks hatched from these eggs are viraemic, and CAV can rapidly spread horizontally from these chicks to susceptible, maternal antibody-negative hatch-mates. The virus is present in high concentrations in the faeces of chickens 5- 7 weeks after infection. Roosters shedding CAV in semen is another source of vertical transmission.

Clinical Signs

Chicken infectious anaemia infections are manifested in either clinical or sub-clinical forms and cause up to 60 percent mortality. In clinical CAV, the early signs start at the end of the second or third week of age. The affected birds show stunted growth, weakness, ruffled feathers, depression, anorexia, paleness, anaemia with low haematocrit values and watery blood. In its acute form, the mortality reaches peak within a week. The affected birds exhibit focal skin lesions characterised by oedematous, reddish blue gangrenous areas releasing a sero-sanguinous exudate in the head, wings, sides of the thorax, abdomen, thigh region and feet. These clinical conditions are defined as haemorrhagic anaemia syndrome, anaemia-dermatitis syndrome and blue wing disease. In few flocks, a second but smaller peak of mortality is also observed after 2 weeks of first incidence due to horizontal spread of the disease in seronegative birds. This kind of mortality is due to secondary or mixed infections of CAV and other agents, such as fowl adenovirus, Marek's disease virus and infectious bursal disease virus, or from secondary bacterial infection of the skin lesions like *Clostridium perfringens, Staphylococcus* and E. *coli.*

Postmortem lesions

The pathological lesions are characterised by pale and anaemic carcass including visceral organs, subcutaneous haemorrhages with accumulation of gelatinous and oedematous fluid in the subcutis of the affected skin, atrophy of the thymus, spleen and bone marrow; petechial to ecchymotic haemorrhages in the thigh and breast muscles; and haemorrhages between the proventriculus and gizzard junction.

Diagnosis

A tentative diagnosis of CAV can be made based on the history with the clinical signs and pathological findings indicative of anaemia dermatitis syndrome. Laboratory diagnosis by using ELISA test, and isolation of CAV by using yolk sac route in embryonated chicken eggs, and in MDCC-MSB1 cells or in SPF chicks. However, virus isolation is not followed mostly as it is time-consuming, expensive, and also difficult in growing CAV in common chicken cell cultures. Therefore, polymerase chain reaction (PCR) is used to demonstrate viral DNA in tissues of the affected flocks.

Treatment

No specific treatment is available for CAV, however, haematinics, immunomodulatory agents such as vitamin C, E, selenium, zinc, copper, *etc*., could be given in feed for 2 weeks. Administration of probiotics in the feed as per recommended dose can restore the gut microflora.

Control

Vaccination

The breeders should be vaccinated between 8 and 16 weeks of age with a live vaccine; The clinical infection of CAV can be controlled by ensuring transfer of sufficient level of maternal antibodies to the progeny by vaccination of breeders and use of immunomodulatory agents, herbal extracts and protein supplements. The level of protection against clinical CAV infection improves with a higher level of maternal antibody. In CAV endemic regions, layer chickens can be vaccinated with live CAV vaccine subcutaneously before 4 weeks of age.

The CIAV vaccination program may be initiated keeping in view the nature and immunopathogenesis of CIAV infection in relation to other agents such

as IBDV and MDV. Since co-infection with IBDV, MDV enhances the pathogenicity of CIAV, stringent IBD and MDV control is an important pre-requisite for CIAV control.

Biosecurity

Limited controlled site access, separate footwear and equipment for each site/house, and footbaths at the entrance to sites/houses minimize the risk of introducing the virus apart from through terminal disinfection programmes in the shed. Applying suitable disinfectants at the correct concentration with a specific contact time is critical to reduce infectivity of virus particles. Products containing formaldehyde, chlorine releasing agents, or quaternary ammonium compounds are used for reducing the infective virus load in the environment. The downtime between successive flocks must be a minimum of 10 days.

5.2.9 Inclusion Body Hepatitis-Hydropericardium Syndrome (IBH-HPS)

Definition and Causative Agent

IBH-HPS caused by fowl adenovirus (FAdV) is an important immunosuppressive disease of poultry, affecting 3- to 6-week-old broiler chicks. The HPS is called as Litchi (or Leechi) disease in India as the heart of the affected birds surrounded by hydropericardium resembled a peeled Indian Litchi fruit. FAdV belongs to family Adenoviridae and the genus *Aviadenovirus*. Based on serum neutralization test (SNT) and restriction endonuclease analysis, FAdVs are divided into five major species (A to E) with 12 serotypes (1 to 8a and 8b to 11). In India, there is circulation of FAdV2, FAdV3, FAdV4, FAdV5, FAdV6, FAdV8, FAdV11 and FAdV12 denoting that FAV is one of the major immunosuppressive diseases and contains majority of the different serotypes reported in the world.

Transmission

The disease is highly contagious and can spread quickly from one flock to another by horizontal and mechanical routes of transmission and with contaminated litter. The FAdV is prolifically excreted through the faeces of infected birds and serves as a major source of infection to young broilers. It's horizontal transmission in a flock is via oro–faecal route followed by its mechanical spread along with faecal contamination.

Clinical Signs

The disease usually begins with observance of stunting and unevenness in the flock followed by a sudden and sharp increase in mortality reaching highest after 3-4 days, which usually returns to normal on day 5 but occasionally continues for 2- 3 weeks. Sick birds adopt a crouching position with ruffled feathers, yellow mucoid droppings and die within 48 hours, or recover. Overall feed conversion and weight gain decrease.

Postmortem Lesions

The predominant and most consistent gross lesion is hydropericardium, characterized by the accumulation of clear or amber green colour, watery or jelly-like fluid in the pericardial sac. Flabby heart with its apex floating in pericardial sac and yellowish discoloration and petechial haemorrhage of pericardial fat are also commonly observed. The changes observed are – liver becomes pale, swollen, friable and mottled with large areas of focal necrotic patches and petechial and ecchymotic haemorrhages; lungs oedematous and congested; and kidneys pale yellow, swollen and friable containing deposits of urates in the tubules and ureters. Atrophy of bursa and thymus, pancreatic necrosis, and gizzard erosions are also reported.

Differential Diagnosis

It may be confused with mycotoxicosis, ascites, and sudden death syndrome based on gross lesions.

Diagnosis

Specimens of choice are faeces, kidney, and liver. Diagnosis can be done on the basis of typical gross lesions, histopathological lesions, particularly observation of intra-nuclear inclusion bodies (INIBs) in hepatocytes; demonstration of adenovirus particles in the nucleus of infected liver cells by transmission electron microscopy; or isolation of virus either in chick embryo liver cells, or chick embryo kidney cells. Cytopathic changes like cell rounding and degeneration are found within 24 hours to 4 days. When the tissue extracts are inoculated into 7- to 8- day-old specific–pathogen–free (SPF) eggs via the yolk sac and/or chorio-allantoic membrane routes; mortality, haemorrhages, intestinal sloughing, enlarged green livers with intranuclear inclusion bodies, poor feathering and stunting are observed. ELISA is used to detect the antibodies, which is sensitive

but expensive. Polymerase chain reaction (PCR) tests are considered specific and reliable diagnostic methods.

Treatment

There is no specific antiviral treatment available for FAdV. Supportive care including maintaining optimal environmental conditions (temperature, humidity) and providing appropriate nutrition and hydration, is crucial. Quality of feed plays significant role because high mycotoxin levels can act as a predisposing factor. Inclusion of double dose of liver tonics, kidney flushers, immunity stimulants like vitamin E and ethnoveterinary products in diet could be useful in recovery.

Vaccination

Inactivated vaccines containing FAV-4, FAV-8 and FAV-11 are available and are administered in breeders during 10th to 15th day of age (Half dose) followed by full dose boosters during 11th to 12th week and 19th to 21st week. Commercial day-old broilers can be vaccinated particularly during rainy season when incidence increases.

Biosecurity

Preventing Aviadenovirus infection is mainly based on biosecurity practices. Strict managemental practices, cleaning and disinfecting of premises and equipment; and restriction of entry and/or disinfection of personnel/visitors and vaccination crews into poultry house, all play an important role in IBH prevention.

Prevention

The resistance of Aviadenoviruses to inactivation by heat (up to 70°C) as well as high resistance to common disinfectants (particularly lipid solvents such as ether, chloroform, and phenol) poses a significant challenge, particularly in poultry houses with impervious floors and walls. Application of glutaraldehyde and calcium hydroxide liquid combination during the downtime inside and outside the house has been found successful. Effective FAdV control begins at the primary breeder level with optimum disinfection and vaccination as two parallel lines that could prevent infection thus protecting against vertical transmission. The horizontal spread should also not be overlooked, and it takes some effort to keep a commercial flock free of FAdV infection. Controlling and/or eliminating IBD virus and CIAV is also critical in reducing FAdV disease because they increase the pathogenicity of FAdV.

5.2.10 Reovirus Infection

Definition and Causative Agent

Reovirus infections of poultry are widespread and all commercial poultry flocks probably become infected at some time during the life of the flock. The disease primarily affects meat-type birds but may be seen occasionally in light egg-laying breeds. Pathogenic strains of reovirus are associated with viral arthritis/tenosynovitis, malabsorption syndrome, stunting/runting syndromes, enteric disease, immunosuppression, and respiratory disease. This infection can result in poor growth, lameness, and significant economic losses in poultry production due to reduced feed efficiency, increased morbidity, and mortality. The causative agent of reovirus infection in chickens is the avian reovirus (ARV). It belongs to the family Reoviridae and the genus *Orthoreovirus*. They are non-enveloped, double-stranded RNA viruses with a segmented genome.

Transmission

These viruses are highly resilient in the environment, capable of surviving under various conditions, which facilitates their spread among poultry populations. Reoviruses can be transmitted both vertically (from hen to egg) and horizontally (through faecal-oral routes, contaminated equipment, or direct contact).

Clinical Signs

Reoviruses are also isolated from birds with mal-absorption/mal-digestion/pale bird syndrome. Affected birds are stunted, unthrifty, show abnormal feather development, proventriculitis, have poor feed conversions and generally look sick. Orange-tinged diarrhoea may be present as can be various degrees of diarrhoea and mal-digestion. Some birds may lose colour in the legs and beak while others may have a distended abdomen. Some individuals may display helicopter-like feathers in their wings and other feather abnormalities.

Runting-Stunting Syndrome- The first sign of a broiler flock being affected is usually the appearance of uneven growth. With experience, this can be detected as early as 4-day- old, and it becomes obvious between 1 and 2 weeks old. Typically,

between 5 to 20 percent of the flock may be stunted. Infected birds often show poor growth and reduced weight gain. There is a decrease in feed efficiency, leading to poorer production performance.

Arthritis or tenosynovitis - One of the most common signs is lameness or reluctance to walk, often due to viral arthritis or tenosynovitis (inflammation of the tendons and tendon sheaths). Swelling of the hock joints and tendon sheaths is frequently observed. Some birds may exhibit mild respiratory signs, such as coughing and sneezing, although these are less common.

Postmortem Lesions

Joints, particularly the hock (tibiotarsus) joints, are often swollen due to the accumulation of inflammatory exudate. Thickening and inflammation of the tendon sheaths, commonly affecting the gastrocnemius tendon. Cartilage erosion within the joints and sometimes ulcerations are seen. Inflammation of the tendon sheaths, especially in the legs. In severe cases, tendons may rupture due to ongoing inflammation and weakening. Other lesions include myocarditis, hepatomegaly, splenomegaly, proventriculitis, intestinal atrophy, *etc*.

Differential Diagnosis

Reovirus infection can resemble other poultry diseases such as Avian encephalomyelitis, BCO lameness, *Mycoplasma synoviae* and mycotoxicosis.

Diagnosis

Clinical signs are usually nonspecific and vary depending upon the type of strain involved hence laboratory diagnosis is the reliable method. Virus isolation involves collecting samples (cloacal or respiratory swabs, internal organs, or faeces) from affected birds and isolating the virus in cell culture. PCR tests can detect reovirus genetic material (RNA) in samples, providing a rapid and specific diagnosis. Blood samples can be tested for the presence of antibodies against reovirus, indicating exposure or previous infection. Histopathological demonstration of characteristic lesions in various organs.

Treatment

No effective treatment is available for reovirus infection. Virucidal disinfectants such as triple salt can be sprayed @5g/litre to reduce the spread. Providing high-quality feed and clean water to support the immune system, overall health and support recovery of the birds along with supplementing the water with electrolytes and vitamins, particularly during times of stress or outbreak.

Control

Regular biosecurity and personal hygiene of farm workers must be adhered along with vaccination.

Vaccination

Since avian reovirus is vertically transmitted, vaccinate breeders with live vaccine containing 1133 strain during first week followed by killed vaccines that contain all the three pathogenic strains during 11th and 18th to 20th week of age. Inactivated Immunogenic strains of Reo virus (VH/ARV/TS/5, VH/ARV/MAP/4, S1133, 1733 and S2408 strains) used for SC or IM injection. Combination vaccines (four-way) along with vaccines for other viral diseases are available.

Prevention

Good broiler farm hygiene, flock nutrition, sanitation and avoidance of intercurrent disease will reduce the burden of challenge caused by multiple infectious organisms. Feeds should be analyzed for dietary toxins, and feeds with high levels of toxins should not be knowingly fed to commercial poultry.

Biosecurity

Limit access to the poultry houses. Require all personnel to wear clean protective clothing, including boots and gloves, which should be disinfected before entering and after exiting poultry houses. Regularly clean and disinfect poultry houses, equipment, and vehicles. Use effective disinfectants against reoviruses. Regularly replace and properly dispose of litter to minimize the risk of infection, besides regular disinfection of water and cleaning of pipe lines.

5.2.11 Avian Encephalomyelitis (epidemic tremor)

Definition and Causative Agent

Avian encephalomyelitis (AE) is an infectious viral disease of young chickens, turkeys, pheasants, and quails. It is characterized by ataxia, rapid tremors, especially of the head and neck in young chicks, reduced egg production and an accompanying lowered hatchability of fertile eggs in laying breeder

hens. Avian encephalomyelitis virus (AEV) belongs to the genus *Tremovirus* of the Picornaviridae family. All strains seem to be antigenically uniform but there are variations in neurotropism and virulence. Field strains are mainly enterotropic while fowl-embryo-adapted strains such as the Van Roekel (VR) strain are mainly neurotropic and are much more likely to kill embryos.

Transmission

Infection occurs via vertical and horizontal routes. If a breeder flock becomes infected during egg production, the virus is vertically transmitted to the offspring resulting in a major outbreak. The disease often appears in a series of flocks hatched from the infected breeder flock. Egg transmission occurs during the period from the infection of susceptible laying hens to the development of immunity, normally a period of 3-4 weeks. Field strains of the virus are enterotropic and multiply in the intestine. Infected birds shed the virus in their faeces for a few days to a few weeks, which serves to spread the infection to hatch mates.

Clinical Signs

The incubation period varies from 5 to 14 days depending on the route of infection. Morbidity is mostly 15 percent but may be as high as 60 percent of the flock. The mortality in affected birds is high. Vertically infected chicks commonly show clinical signs during the first week after hatching. Clinical signs appear later in hatch-mates that are horizontally infected by the faecal-oral route. Vertical infection followed by horizontal infection causes a characteristic biphasic mortality pattern. The main clinical signs are ataxia and leg weakness that varies from sitting on hocks to paresis that progresses to paralysis and recumbency. Fine tremors of the head and neck are evident in some birds and are characteristics of the disease. Tremors vary in frequency and severity and are mostly seen in disturbed or excited birds. Ataxia usually progresses until the chick is incapable of moving about, followed by inanition (loss of vitality from lack of food and water), prostration (lying down), and finally death due to inability to eat or drink, or through being trampled. Survivors may later develop blindness from an opacity giving a bluish discoloration to the lens. Mature birds may experience a temporary drop in egg production, but do not develop neurological signs. In layers, the fall in egg production is about 5–10 percent and lasts for 5–14 days, with return to full potential production at the end of this time. The fall in hatchability accompanying the depression in production is about 5 percent of fertile eggs.

Post-mortem Lesions

There are no gross lesions in the young or older birds apart from rare pale areas in the gizzard muscle of chicks and opacity and fixation of the lens in a small proportion of survivors.

Differential Diagnosis

Nutritional encephalomalacia (vitamin E deficiency/ vitamin A deficiency/riboflavin deficiency/rickets), Newcastle disease, Marek's disease, peripheral neuropathy.

Diagnosis

The clinical signs in young birds like the absence of gross lesions and the histological lesions in the brain, spinal cord and viscera, together with the absence of other virus infections and nutritional deficiencies affecting the nervous system are strongly suggestive of avian encephalomyelitis and frequently used for routine presumptive diagnosis. For virus isolation, a suspension of brain, pancreas or duodenum from affected chicks is inoculated into the yolk sac of 5- to 6-day-old susceptible chick embryos. AEV positive embryos are characterised by inertia, muscular dystrophy and occasional mortality. The remaining embryos are hatched and during the first 10 days of life, the chicks are observed for typical clinical signs of avian encephalomyelitis. However, the PCR test is routinely used in the diagnosis of AEV infection.

Treatment

There is no specific treatment for AE once a bird is infected. Supportive care includes providing a stress-free environment, good and balanced nutrition, clean water and administration of vitamin B complex and vitamin E in water.

Control

Vaccination is able to control the spread of the disease.

Vaccination

Live AE vaccine is typically recommended in breeders orally or through drinking water before 14 weeks of age. This method ensures that chicks

receive the vaccine effectively and start building immunity early.

Biosecurity

Regularly clean and disinfect all equipment, vehicles, and tools that come into contact with birds or their environment. Disinfectant triple salts are effective against the AE virus. Basic poultry farm management standards which must be followed include limiting access to poultry areas to essential personnel only; keeping foot dips and hand washing stations at entry points; controlling rodents, wild birds, and insects that may carry and transmit the AE virus; securing feed storage areas; disposing of dead birds promptly and safely; and quarantining sick birds or birds showing symptoms of AE immediately to prevent further spread.

5.2.12 Leukosis/Sarcoma Group

Definition and Causative Agent

Avian leukosis is caused by retroviruses associated with several neoplastic diseases in poultry. These viruses are grouped into different envelope subgroups and induce diseases such as lymphoid leukosis (LL) and myeloid leukosis (ML) that are widespread in many countries causing major economic losses and animal welfare issues. Avian leukosis group viruses are placed in the Alpha *Retrovirus* genus of the family Retroviridae. Members of this family are RNA viruses characterized by the possession of the enzyme reverse transcriptase, which is necessary for the formation of a DNA provirus that is integrated in the host genome during virus replication.

Transmission

Exogenous ALVs are transmitted vertically from hen to progeny through the egg and horizontally from bird-to-bird by direct or indirect contact. Although usually only a small percentage of chicks are infected vertically, this route of transmission is important epizootiologically because it affords a means of maintaining the infection from one generation to the next. Most chickens become infected by close contact with congenitally infected birds. Although vertical transmission is important in the maintenance of the infection, horizontal infection may also be necessary to maintain a rate of vertical transmission sufficient to prevent the infection from dying out. The infection does not spread readily from infected birds-to-birds in indirect contact (in separate pens or cages), probably because of the relatively short life of the virus outside the birds. However, contact exposure at hatch is an effective method of spread of ALV-J among broiler breeder chickens and can be prevented by small group rearing.

Clinical Signs

Leukotic diseases in birds cause inappetence, weakness, diarrhoea, dehydration, and emaciation. In LL, abdominal enlargement may occur. The course is usually rapid, with birds dying within weeks. Myelocytomatosis can cause protuberances, eye orbital haemorrhage, skin haemorrhage, renal tumours, and connective tissue tumours. Osteopetrosis affects long bones, with irregular thickening and unusually warm areas. Advanced lesions have boot-like shanks, stunted, pale birds, and a stilted gait. Avian leukosis virus is associated with fowl glioma.

Postmortem Lesions

Tumours can be found in various organs and tissues, including the liver, spleen, kidneys, gonads, heart, and mesentery. These are typically soft and can vary in size and number. Liver enlargement (hepatomegaly) with nodular or diffuse tumours is common. It may appear pale or mottled with white to yellow nodules. Spleen is often enlarged (splenomegaly) and may contain nodules similar to those in the liver. Kidney is enlarged and has nodules. Tumours may be present on the surface or within the myocardium in heart. Involvement of Bursa of Fabricius is common in younger birds, and the bursa may be enlarged and have nodular masses. Bone Marrow can be affected, showing infiltration by neoplastic cells, leading to changes in the colour and consistency of the bone marrow.

Diagnosis

The clinical signs and lesions in chickens affected by leukosis are nonspecific. Diagnosis involves pathological, virological and molecular examinations to determine the type of virus involved in the neoplasm responsible for mortality. Histopathological examination can reveal the presence of neoplastic cells and help differentiate between different types of tumours. PCR can be used to detect viral DNA or RNA in blood or tissue samples. Specific PCR tests can differentiate between different subgroups of avian leukosis viruses (ALV-A, ALV-B, ALV-C, etc.)

Treatment

As no treatment is available for LLV, only regular grading and culling of the infected chicken can minimize the disease transmission. Immuno-stimulation strategies by supplementation of vitamin E, selenium, and MOS in the feed can minimize the loss.

Control

Identifying and propagating lines that show natural resistance to the ALV. Eliminating infected breeders from the flock to reduce vertical transmission and repopulation with certified ALV-free stock. Conducting regular serological testing of flocks to detect the presence of ALV antibodies or antigens. Utilize polymerase chain reaction (PCR) and other molecular techniques for early detection of the virus in breeding and commercial flocks. Implement an all-in/all-out management system to reduce the risk of disease carryover between batches of birds.

Vaccination

Although no commercial vaccines are available for ALV, it is important to take advantage of prophylactic measures such as use of vaccines against other diseases to maintain overall flock health and reduce the susceptibility to secondary infections.

Biosecurity

Maintain strict isolation of breeding stock from commercial flocks to prevent horizontal transmission. Implement rigorous cleaning and disinfection protocols for housing, equipment, and farm vehicles to minimize the risk of virus spread. Restrict access to poultry houses to essential personnel only and enforce the use of protective clothing and footbaths.

5.3 Bacterial Diseases

5.3.1 Avian Mycoplasmosis

Definition and Causative Agent

Avian mycoplasmosis - due to *Mycoplasma gallisepticum* and *Mycoplasma synoviae* - causes significant economic losses on poultry farms because of chronic respiratory disease, reduced feed efficiency, decreased growth and decreased egg production in chickens, as well as conjunctivitis and sinusitis in turkeys and game birds. *Mycoplasma gallisepticum* and *Mycoplasma synoviae* are bacteria belonging to the class Mollicutes and the family Mycoplasmataceae.

Transmission

The disease can be transmitted vertically through eggs from infected breeders to progeny as well as via infectious aerosols, contamination of feed, water, and environment as well as human activity on fomites which can come from equipment and shoes. Backyard flocks, multiple-age layer flocks, and some free-ranging songbird species also act as potential reservoirs.

Clinical Signs

In chickens, infection may be inapparent or result in varying degrees of respiratory distress, with slight to marked rales, difficult breathing, coughing, and/or sneezing. Morbidity is high and mortality low in uncomplicated cases. Nasal discharge and conjunctivitis with frothiness around the eyes may be present. The disease is generally more severe in turkeys than that in chickens and swelling of the infraorbital sinuses is common. In laying flocks, birds may fail to reach peak egg production, and the overall production rate is lower than normal.

Postmortem Lesions

The affected birds exhibit catarrhal sinusitis, tracheitis, and airsacculitis. *E. coli* infections are often concurrent and result in severe air sac thickening and turbidity, with exudative accumulations, adhesive pericarditis, and fibrinous perihepatitis.

Treatment

Mycoplasma infection is treated by combination of anti-Mycoplasma drugs in recommended dosages and duration. The antibiotic therapy should be strictly administered as per antibiotic sensitivity test (ABST) under the supervision of a poultry veterinarian and the withdrawal period should be strictly followed.

Vaccination

Before going for vaccination in breeders administer antimycoplasmal drugs during the first and fourth weeks of age as a clean-up programme as well as screen the flocks either by PCR or ELISA for mycoplasma. If the flocks are found free of mycoplasma, MG live vaccines either 1/85 or Ts-11 and MS live (MSH) may be administered by eye drop separately. No antimycoplasmal drugs should be administered at least for 6 weeks. Booster with

MG and MS killed vaccine can be administered at 10th and 15th weeks of age. In mycoplasma endemic farms, second MG live vaccine is recommended at 12th week of age followed by MG killed vaccine at 15th week of age.

Prevention

In general, purchase fertile hatching eggs or day-old chicks from the breeding flock free of Mycoplasma infection. In India, the commercial poultry are reared in open air poultry houses with multi age system of rearing. Poultry density is very high in many poultry producing regions of the country, sometimes mixed types of commercial poultry are kept in proximity thus making infection-free flocks more than difficult, if not impossible. Therefore, suitable antibiotic medication may be used to alleviate clinical signs and reduce production losses or egg transmission of Mycoplasma in commercial flocks.

Biosecurity

A high level of biosecurity of breeder flocks, with

a) production in single-aged, all-in/all-out farms,
b) routine monitoring by serological testing backed up by rapid and specific confirmatory tests. and
c) immediate slaughter of infected breeding flocks and vaccination to prevent transmission of *Mycoplasma* to the progeny.

5.3.2 Avian Pathogenic E. coli

Definition and Causative Agent

Avian colibacillosis is an infectious disease of birds caused by *Escherichia coli* (*E. coli*), which is considered as one of the principal causes of morbidity and mortality, associated with heavy economic losses to the poultry industry by its association with various disease conditions, either as primary pathogen or as a secondary pathogen. *E. coli* is a gram-negative, non-acid-fast, uniform staining, non-spore-forming bacillus that grows both aerobically and anaerobically and may vary in size and shape. Many strains are motile and have peritrichous flagella. Although *E. coli* is present in the normal microbiota of the intestinal tract, other host mucosal surfaces, and in the bird's environment; only a certain number of these strains possessing specific virulence attributes, designated as Avian Pathogenic *E. coli* (APEC), are able to cause disease. These strains are mainly associated with extra-intestinal infections, majority of them come under "O" serogroup.

Transmission

The *E. coli* persists for long periods outside the bird's body in dry and dusty conditions. Faecal contamination of the eggs may result in the penetration of *E. coli* through the shell and is considered to be the most important source of infection. Other sources may be ovarian infection or salpingitis. The organism is present between 0.5 and 6 percent of eggs from normal hens. *E. coli* may spread to other chickens during hatch, and is often associated with high mortality rates, or it may cause yolk sac infections. Feed is often contaminated. Rodent droppings usually contain pathogenic coliforms. Contaminated well water can also leave pathogenic serotypes into the poultry flocks.

Clinical Signs

The clinical symptoms in poultry are yolk sac infection, omphalitis, respiratory tract infection, swollen head syndrome, septicaemia, polyserositis, coli granuloma, enteritis, cellulitis and salpingitis. Generally, the clinical disease is characterized by drop in feed consumption, dejected appearance of the affected birds with ruffled feathers, respiratory distress, laboured breathing and gasping. Morbidity and mortality is variable, and losses are usually lesser than 5 percent of the group, but morbidity can be over 50 percent.

Postmortem Lesions

The characteristic gross lesions include air sacculitis, peritonitis, perihepatitis, and pericarditis. Besides these lesions, pneumonia and pleuropneumonia are also usually present. Less commonly, salpingitis may occur following sepsis. When the left abdominal air sac is infected by *E. coli,* females may develop chronic salpingitis characterized by a large caseous mass in a dilated, thin-walled oviduct. Size of the caseous mass may increase with time. Infection of the peritoneal cavity (peritonitis) is characterized by acute mortality, fibrin, and free yolk. Infection occurs when bacteria ascending through the oviduct grow rapidly in yolk material that has been deposited in the peritoneal cavity. Salpingitis can also occur from an ascending infection from the cloaca. The carcass is septicaemic. Liver, spleen, lungs and kidneys are dark and congested. The air sacs are thickened, opaque and white with caseous deposits.

Diagnosis

Diagnosis is based on isolation and identification of *E. coli* from lesions typical of colibacillosis. Care must be taken to avoid faecal contamination of samples. Material should be streaked on eosin-methylene blue (EMB) or MacConkey agar, as well as non-inhibitory media. A presumptive diagnosis of *E. coli* infection can be made if most of the colonies are characteristically dark with a metallic sheen on EMB agar and bright pink with a precipitate surrounding the colonies on MacConkey agar. Strains of *E. coli* can be slow or non-lactose fermenters and appear as non-lactose fermenting colonies. Multiplex PCR for 6 virulence genes of *E. coli* help to distinguish between commensal and pathogenic isolates.

Treatment

Antimicrobial therapy - as per the antimicrobial sensitivity test - under the supervision of the poultry veterinarian along with supportive therapy will reduce the losses in clinical infection. Strict withdrawal period should be followed after administration of antibiotics. Water sanitation and acidification of feed and water helps in prevention.

Control

Proper breeder farm and hatchery hygiene and sanitization. Feed hygiene and gut health management.

Prevention/Biosecurity

Feed contamination with *E. coli* can be a source of infection for poultry. Hence, it is advisable to use acidifiers (organic acids, pre- and pro-biotics (competitive exclusion)) and essential oils as per recommended doses in the feed to maintain the gut integrity and reduce the *E. coli* contamination.

Water hygiene includes following steps:

- Regular administration of water sanitizers such as Chlorine dioxide@0.2 ml/L
- Use of water acidifiers to reduce the pH.
- Regular removal of biofilms in the pipelines by using 10% H_2O_2.
- Ensure regular care of clean drinking water pipes.
- Rodent, insect, and pest control.
- Personal hygiene of farm workers.
- Avoiding open defecation in farm premises.
- Regular cleaning and disinfection of poultry sheds can help to reduce the environmental buildup of *E. coli.*

Waste disposal in a poultry farm is important to check further infection. Various steps include used poultry litter, carcasses, and other potentially contaminated farm waste should be transported and disposed of in a safe manner (deep burial, incineration or composting) to prevent the direct or indirect exposure of humans, livestock and wildlife to *E. coli*.

5.3.3 Salmonella Infections in Poultry

Definition and Causative Agent

Infections with bacteria of the genus *Salmonella* are responsible for a variety of acute and chronic diseases in poultry. Isolations of salmonella are reported more often from poultry and poultry products than from any other animal species. The genus *Salmonella* consists of more than 2,300 serologically distinguishable variants (serotypes/serovars). These serotypes are usually named after the place of initial isolation. Infections of poultry with salmonellae can be grouped into three categories.

The first group includes infections with two non-motile serotypes, *S. pullorum* and *S. gallinarum,* which are generally host-specific for avian species. Pullorum disease, caused by *S. pullorum*, is an acute systemic disease of chicks and poults (young turkeys). Fowl typhoid, caused by *S. gallinarum*, is an acute or chronic septicaemic disease usually affecting mature birds. Both of these diseases cause serious economic losses to poultry farmers.

The second group includes infections with a group of motile Salmonella serotypes, referred collectively as paratyphoid salmonellae. The disease produced by them is called salmonellosis or paratyphoid infections. Paratyphoid infections are important mainly as a cause of food-borne disease in humans. Paratyphoid infections of poultry are very common, but they rarely cause acute systemic disease, except in highly susceptible young birds subjected to stressful conditions.

The third group includes infections with the various motile serotypes of the sub-genus Arizona, the most important species being *S. arizona.* It was formerly designated as *Arizona hinshawii* and causes arizonosis, which is of economic significance in turkeys.

Transmission

Salmonella is passed from hen to chick by vertical transmission and then there is rapid lateral spread from chick-to-chick in hatcheries and rearing units. The organism can survive outside the body for many months. Red mites, rodents, farm pets may be involved in the transmission of disease and persistence in poultry houses.

Clinical signs

S. pullorum causes pullorum disease, which is seen mainly in under 3-week-old chicks. First indication is usually excessive numbers of dead-in-shell chicks, and deaths soon after hatching. Signs include depression with a tendency to huddle, respiratory distress, lack of appetite, and white viscous (thick and sticky) droppings, which adhere to the feathers around the vent. The mortality varies considerably, and in extreme cases can be 100 percent. A subacute form with lameness and swollen hock joints may be seen in growing birds, and result in poor growth rates.

S. gallinarum causes fowl typhoid which is a more acute septicaemic condition that mainly affects mature birds and may be particularly severe in commercial laying flocks. It spreads rapidly with high morbidity and acute or subacute mortality. Clinical signs are anorexia, diarrhoea, dehydration, weakness and death.

Postmortem Lesions

In pullorum disease, the chicks which die soon after hatching have peritonitis with an inflamed, unabsorbed yolk sac. Lungs may be congested and liver dark and swollen with haemorrhages visible on the surface. Sometimes, chicks which die in the acute phase do not show specific lesions, however, they may show lesions only those of a septicaemia with the liver congested and the subcutaneous blood vessels dilated and prominent. In chicks which die after showing signs of disease for 1 or 2 days may have typhlitis (inflammation of the caecum). The caeca are enlarged and distended with casts of hard, dry necrotic material. Discrete (separate), small, white, necrotic foci are also usually found in the liver, lungs, myocardium, and gizzard wall. In growers affected with arthritis, the hock joints are usually enlarged due to the presence of excess orange-colour gelatinous material around the joints. In general, the lesions in chicks are neither characteristic nor constant. In adult birds, the characteristic lesions of fowl typhoid are swollen, friable, and bronze colour liver, with or without necrotic foci; an abnormal ovary with the ova being irregular, cystic, misshapen, discolored and pedunculated with prominent thickened stalks.

Diagnosis

Avian salmonellosis should be diagnosed and confirmed by isolation, identification, and serotyping of *Salmonella* strains. Serologic testing can be used to detect infections in mature birds. Necropsy examination and microbiologic culture and typing can then be used to confirm further. Serological ELISA test is used to diagnose *S. typhimurium* or *S. enteritidis*. Real-time PCR system is used for quick and accurate detection of *Salmonella* at genus- and serovar-specific (*S. enteritidis* and *S. typhimurium*) levels for monitoring the chicken food chain.

Control

Time and frequency of testing are as follows:

Breeders and hatcheries		
Breeder flocks before lay	Breeder flocks in lay	Hatcheries
• Before the end of the first week of life • Within the four weeks before being moved to another house, or before going into production if the birds will remain in the same house • One or more times during the growing period if there is a culling policy in place. The frequency would be determined on commercial considerations	• At least at monthly intervals during the laying period • Additional testing should be determined by the veterinary services	• Testing at hatcheries should complement on farm testing • The minimal frequency should be determined by the veterinary services

Layer flocks	Broiler flocks
• Flocks grown to be layers • Before the end of the first week of life when the status of the breeder flock or the hatchery is not known • Within the four weeks before being moved to another house, or before going into production if the birds will remain in the same house for the production period • Layer flocks • At expected peak of lay for each production cycle (the period of time in the laying cycle when the production of the flock is highest) • One or more times during the laying period	• Flocks should be sampled at least once as late as possible before the first birds are transported to the slaughterhouse/ abattoir

Control measures to reduce the risk of transmission of Salmonella to humans

i) In breeders, control measures may be implemented to reduce the transmission of *Salmonella* to the next generation, especially for trans-ovarian transmitted serotypes such as *S. enteritidis*.

ii) In layer flocks, control measures will reduce and may eliminate contamination of eggs with *Salmonella*.

iii) In poultry for meat production, control measures may be implemented at slaughter or further down the food chain.

Sourcing of commercial chicks: Day-old birds used to stock a poultry house should be obtained from breeder flocks and hatcheries free from *S. enteritidis* and *S. typhimurium*.

Feed hygiene and gut health management: Feed contamination with *Salmonella* is a source of infection for poultry. Monitor the *Salmonella* status of poultry feed and take corrective measures, if feed is found positive for *Salmonella*. Use of pelleted feed can reduce the *Salmonella* risk. It is advisable to use acidifiers (organic acids, pre- and pro-biotics (competitive exclusion)) and essential oils as per recommended doses in the feed to maintain the gut integrity and reduce the *Salmonella* contamination.

Other control measures include rodent control, insect control, wild bird proofing, dog and cat proofing, waste disposal, and hatching egg hygiene, *etc*.

Vaccination

Killed *Salmonella enteritidis* and *S. typhimurium* vaccines are licensed to use in breeders to control salmonellosis and to prevent infection transmission through poultry products for human consumption. These vaccines can be used at 10-12th week of age followed by vaccination at the point of lay. For prevention of fowl typhoid, use live vaccine SG9R during 7-8th week of broiler breeders/layers.

Biosecurity

Houses and buildings should be cleaned and disinfected thoroughly; adequate downtime must be given. Restocking can be done only after swabs taken to check for the persistence of *Salmonella*.

5.3.4 Infectious Coryza

Definition and Causative Agent

Infectious coryza, a highly contagious and acute respiratory disease of chickens, is characterized by foamy conjunctivitis, sinusitis, nasal discharges, depression, and lethargy. The disease occurs worldwide and causes economic losses due to an increased number of culls and a marked (10 percent to more than 40 percent) drop in egg production, particularly on multi-age farms. The disease is caused by *Avibacterium paragallinarum* - a Gram-negative, non-motile, non-spore-forming, capsulated and microaerophilic rod. It was classified based on Kume serovar typing. Currently, nine recognized Kume serovars include A-1, A-2, A-3, A-4, B-1, C-1, C-2, C-3 and C-4.

Transmission

The main source of infection is clinically affected and carrier birds, especially from replacement stock. The organism can be transmitted by drinking water contaminated by nasal discharge as well as by air-borne means over a short distance. Lateral transmission occurs readily by direct contact. Spread between batteries - with nipple drinkers - occurs more slowly. Birds can be more susceptible if already infected with other respiratory viral or bacterial infections.

Clinical Signs

The disease in flocks on floor management is characterized by rapid spread, high morbidity, and low mortality. The affected birds exhibit symptoms within 24-72 hours PI. The first typical signs include sero-mucoid nasal and ocular discharge (lacrimation) with offensive odour, facial oedema and swelling. In severe cases, marked conjunctivitis with closed eyes, purulent discharge from eyes, swollen wattles (wattle disease), difficulty in breathing, sneezing, loss of appetite, and weight loss can be seen. The birds may have diarrhoea. The swelling usually subsides in 10–14 days; however, if secondary infection occurs, swelling can persist for months. There may be varying degrees of rales depending on the extent of infection. Feed and water consumption is usually decreased resulting in a drop in egg production (10–40 percent) or an increase in the rate of culls.

Postmortem Lesions

In acute cases, lesions are found in infraorbital sinuses. There is a copious, tenacious, greyish, and semifluid exudate. As the disease becomes chronic or other pathogens become involved, the sinus exudates may become consolidated and turn yellowish. Other lesions may include subcutaneous oedema, conjunctivitis, tracheitis, bronchitis, and airsacculitis, particularly if other pathogens are involved.

Differential Diagnosis

Differentiate from Mycoplasmosis, respiratory viruses, chronic or localised pasteurellosis, and vitamin A deficiency.

Diagnosis

Culture should be attempted by swabbing from the infraorbital sinus of two to three acutely diseased chickens on to blood agar plates cross-streaked with a feeder organism such as *Staphylococcus epidermidis*. The isolated organism can be identified by phenotypic tests or by confirmatory PCR. The PCR can also be applied directly to nasal exudate.

Treatment

Antibiotics as per antibiotic sensitivity test such as sulpha group of drugs or quinolones can be recommended for 3-5 days under the supervision of poultry veterinarian. Strict withdrawal periods should be followed after antibiotic therapy.

Vaccination

Vaccination of susceptible chicken flocks is the most efficacious preventive practice against infectious coryza. One to three killed vaccines are commonly administered in commercial layers and breeders according to the endemicity of the disease at 4th week, 9th week and 14th weeks.

5.3.5 Fowl Cholera

Definition and Causative Agent

Fowl cholera (FC), a contagious disease affecting domestic and wild birds, typically occurs as a fulminating disease with massive bacteremia. It is caused by *Pasteurella multocida and* can range from acute septicaemia to chronic and localised infections. The morbidity and mortality may be up to 100 percent. High environmental temperature and humidity may act as predisposing factor during rainy season.

Transmission

Transmission is via contaminated nasal exudates, faeces, contaminated soil, equipment, and people. Incubation period usually ranges from 5-8 days. The bacterium is easily destroyed by environmental factors and disinfectants but may persist for prolonged periods in soil. Reservoirs of infection may be present in other species such as rodents and cats. Predisposing factors include high density and concurrent infections such as respiratory viruses.

Clinical signs

Fowl cholera occurs in several forms, *viz.*, per acute, acute, and chronic and localized disease.

In the per acute form, there are no warning signs and large numbers of birds are found dead, but in good bodily condition (web-footed birds in

particular). Before death, the birds may exhibit convulsions, uncoordinated fluttering, stiffness, and rapid breathing.

In the acute form, marked depression, anorexia, listlessness, shivering, huddling, mucus discharge from the orifices, ruffled feathers, and cyanosis may be seen. In acute cases, a green diarrhoea can be an early symptom. Pneumonia is particularly common in turkeys. Respiratory sound, sneezing and sticky nasal discharges are sometimes observed. The feathers surrounding the vent, eyes and beak may become matted with secretion. The droppings which start out as pasty and yellow, fetid (foul-smelling), become bloodstained due to intestinal ulceration.

The chronic form occurs in birds which survive the more acute disease, or it may result from infection with an organism of relatively low virulence. The signs are mainly due to localized infections of joints, tendon sheaths, footpads, and abscesses of the head (cranial bones, infraorbital sinuses, sternal bursae, wattles, subcutaneous tissue, comb and wattles), oviduct and the respiratory tract (dyspnea and rales). There may be exudative conjunctivitis and pharyngitis. The clinical signs include depression, weight loss, abdominal distention, conjunctivitis, dyspnea, and - in a few cases - lameness, torticollis, and swelling of the wattles. Torticollis may be associated with infections of the cranial bones, middle ear, and meninges. Dermal necrosis in turkeys may also be observed.

Postmortem Lesions

Lesions observed in per acute and acute disease are dominated by general septicaemic lesions, including vascular disturbances in the form of congestion throughout the carcass and enlargement of liver and spleen. Often there are petechial and ecchymotic haemorrhages at subepicardial fat of the heart, in mucous membranes, gizzard and in abdominal fat. Increased amounts of peritoneal and pericardial fluids are frequently seen. Hemorrhagic consolidation of the lungs is often seen. With respiratory involvement, there is evidence of a catarrhal exudate which is most prominent in the upper trachea. The intestinal tract is empty or contains a thin greenish fluid and haemorrhages may be present on the serosal surface. In addition, acute oophoritis with hyperaemic follicles may be seen. The lungs are often involved, especially in turkeys, where the lesions may be very characteristic. In the most acute infection, haemorrhages dominate the lung lesions, soon followed by necrosis and fibrinous pleuropneumonia where affected areas are clearly marked from unaffected tissue.

Differential Diagnosis

Avian Influenza, Newcastle Disease, and toxicities causing acute mortality.

Diagnosis

Although the history, signs, and lesions may aid diagnosis; *P. multocida* should be isolated, characterized, and identified for confirmation. Primary isolation can be accomplished using blood agar or dextrose starch agar, or trypticase soy agar that yield colonies up to 3 mm in 24 hours (no growth on McConkey agar). The addition of 5% heat-inactivated serum may improve isolation. *P. multocida* can be readily isolated from viscera of birds dying from per acute/acute fowl cholera, whereas isolation from suppurative lesions of chronic cholera is more difficult. At necropsy, bipolar microorganisms are demonstrated by using Wright's or Giemsa stain on impression smears obtained from the liver in acute cholera.

Treatment

Several antibiotics are used to treat fowl cholera, however, if the source of infection is not eliminated, the mortality may resume after discontinuation of antibiotic therapy. Pecking and cannibalism also contribute to spread of disease within a flock; therefore, prompt removal and disposal of carcasses is critical to decrease further losses. An early treatment with antimicrobials and in adequate dosages is important, which should be administered under the supervision of a veterinarian. The antibiotics should be selected always based on antibiotic sensitivity tests, and withdrawal period should be followed after antibiotic treatment. Sulphamethazine @ 134–195 mg/kg, PO or sulpha-di-methoxine @30-50mg/kg in feed or water usually controls mortality rates. Sulpha drugs should be used with caution in breeders because of potential toxicity and cannot be used in hens laying eggs for human consumption.

Prevention/Biosecurity

Thorough sanitation, rodent control, and a rigorous biosecurity plan are essential to preventing infection. Vaccines are available to aid in the control of an outbreak within a flock taking precautions as per

advice of local poultry veterinarian.

5.4 Poultry Parasitic Diseases

5.4.1 Coccidiosis

Definition and Causative Agents

Coccidiosis is caused by protozoa of the phylum Apicomplexan. Most species affecting poultry belong to the genus *Eimeria* and infect various intestinal sites. The disease course is rapid (4–7 days) and is characterized by parasite replication in host cells with extensive damage to intestinal mucosa. Coccidia in poultry are generally host-specific and the different species infect specific portions of the intestine.

In chickens, depending on the source, seven to nine species of *Eimeria* that can cause coccidiosis are described. The main identified species of *Eimeria* are *E. acervulina, E. maxima, E. tenella, E. necatrix, E. brunetti, E. preacox, E. mitis,* and E. *mivati.*

Transmission

Coccidia are almost universally present in poultry-raising operations, but clinical disease occurs only after ingestion of relatively large numbers of sporulated oocysts by susceptible birds (those that are immunosuppressed and/or with concurrent disease). Both clinically infected and recovered birds shed oocysts in faeces which contaminate feed, dust, water, litter, and soil. Oocysts may be transmitted via equipment and personnel, (shoes) as well as the presence of insects (flies) and rodents. Fresh oocysts are not infective until they sporulate under optimal conditions (21°–32°C) with adequate moisture and oxygen which requires 1–2 days. The prepatent period is 4–7 days. Sporulated oocysts may survive for long periods, depending on environmental factors.

Clinical Signs and Lesions

Signs of coccidiosis range from decreased growth rate in many sick birds, severe bloody diarrhoea, and high mortality. During an outbreak, the birds also exhibit decreased feed and water consumption, weight loss, and decreased egg production. Mild infections - which would otherwise be classed as subclinical - may potentially lead to secondary infection, particularly due to *Clostridium* spp.

E. tenella lesions are easier to recognize due to their type and location and increased mortality rates. *E. tenella* is considered one of the most pathogenic *Eimeria* spp. The parasite is primarily found in the caeca, and in severe disease cases, there is an associated increase in morbidity, blood in the faeces, weight loss, dehydration, loss of appetite, anaemia, and diminished skin pigmentation. Bloody caeca cores may also be seen in the caecum.

E. acervulina infections can result in mild to severe coccidiosis, and more severe infections can reduce body weight. Watery/mucoid droppings can also be observed. Pigmentation in skin is lost due to carotenoid and reduced xanthophyl absorption in the small intestine. Gross lesions are usually in the duodenum but can extend into the rest of the small intestine if the infection is severe. These lesions are characterized by white plaques.

E. maxima is considered moderately to severely pathogenic. Symptoms can vary from mild to severe, with mortality rates of up to 30 percent. It can cause poor weight gain, diarrhoea, ruffled feathers, loss of appetite and pale skin. *E. maxima* affects the mid-gut anywhere after the duodenum and past the Meckel's diverticulum; however, heavy infections can be seen throughout the small intestine. An intestine infected with *E. maxima* may be oedematous, flaccid and thickened and can reveal increased mucus - typically, yellow or orange - and blood. In more severe infections, the mucosa can slough off.

E. necatrix lesions are found in the small intestine as with *E. maxima* lesions and are typically seen in the pullet stage and in older birds. *E. necatrix* is considered one of the most pathogenic *Eimeria* spp. Infections of *E. necatrix* can cause high morbidity and mortality rates of more than 25 percent. Additionally, decreased body weight, decreased egg production, emaciation and secondary infections can also be observed. Gross lesions in the gut are typically ballooning and involve thickened mucosa and blood. Usually, *E. necatrix* lesions are described as salt-and-pepper in appearance due to the white and black plaques seen in the mucosa.

E. brunetti is found primarily in the lower intestine after the Meckel's diverticulum; however, in severe infections, it can be in the upper and lower intestines, caeca and cloaca. Symptoms can vary from mild to severe in heavy infections. Moderate mortality rates and reduced feed conversion may be observed. Gross lesions present in the gastrointestinal tract

are characterized by petechiae, watery contents, thickened mucosa, pallor and, in severe cases, erosion of the mucosal layer. Digested or coagulated blood may be observed in the faeces.

Differential Diagnosis

Salmonellosis, Necrotic Enteritis, Cannibalism.

Diagnosis

Diagnosis is based on the location of lesions in the host and their appearance; the size of the oocysts is used to determine the species. The coccidial infections are readily confirmed by demonstration of oocysts in faeces or intestinal scrapings; however, the number of oocysts present has little relationship to the extent of clinical disease. Severity of lesions as well as knowledge of flock appearance, morbidity, daily mortality, feed intake, growth rate, and rate of lay are important for diagnosis. Necropsy of several fresh specimens is advisable. Classic lesions of *E. tenella* and *E. necatrix* are pathognomonic, but infections with other species are more difficult to diagnose. In laboratories, may use faecal flotation as well as histopathology of intestinal tissue samples.

Treatment

Anti-coccidial compounds in feed or water, vaccination, or a combination of both is used to prevent clinical signs. Once clinical signs appear, use of antibiotics and supportive care is advisable to minimize dehydration and secondary bacterial infection.

Control

Poultry maintained at all times on wire floors to separate birds from faeces have fewer infections; clinical coccidiosis is seen only rarely under such circumstances. Other methods of control are vaccination or prevention with anticoccidial drugs.

Anticoccidials like ionophores: Monensin, lasalocid, narasin and maduramicin are given in the feed prophylactically because most of the damage occurs before signs become apparent and because drugs cannot completely stop an outbreak. Therapeutic treatments with Amprolium, toltrazuril, sulphonamides, *etc.*, are usually given by water because of the logistical constraints on feed administration and by following withdrawal periods as per recommendation for the product. Antibiotics may be used in the ration to improve rate of recovery and prevent secondary infections.

Poultry producers may use one anticoccidial continuously through successive flocks, change to alternative anticoccidials in different phases of growing chickens, or change anticoccidials during a single grow out (*i.e.*, a shuttle program). Anticoccidials are commonly withdrawn from broilers 3–7 days before slaughter to meet regulatory requirements and to reduce production costs.

Vaccination

Commercial vaccines consist of live, sporulated oocysts of the various coccidia species administered at low doses. Modern anticoccidial vaccines should be given to day-old chicks, either at the hatchery or farm. Many commercial vaccines contain live oocysts of coccidia that are not attenuated. However, the risk of disease outbreak due to these vaccines led to attenuated vaccines. The virulence of attenuated anticoccidial vaccine is usually reduced. This is done by screening for more precocious *Eimeria* isolate. More effective administration techniques and choice of coccidia strains in the product are improving the feasibility of vaccination in poultry. Attenuated vaccines are proving helpful in maintaining uniformity in breeder flocks as well as resulting in better egg production.

Biosecurity

Litter management particularly during high humidity and rainy season plays important role in prevention. Sporulated oocysts of coccidia are very resistant to normal farm conditions and may survive even up to a year if proper cleaning and disinfection was not used after an outbreak of coccidiosis at the farm. Dispose of the infected litter and faeces either by composting or incineration. Farm labourers and visitors may spread it to different sheds/farms through their footwear.

5.4.2 Red Mite infestation (Ectoparasite)

Definition and Causative Agent

The poultry red mite (PRM) or chicken mite – known as *Dermanyssus gallinae* - infests chickens, turkeys, pigeons, canaries, and various wild birds worldwide. These bloodsucking mites will also bite people but cannot survive on or infest them. Economic losses from poultry mite infestations severely affect the productivity in the egg-laying/breeding hens. The poultry red mite is an ectoparasitic mite in laying

hen farms worldwide and is a major pest in the poultry industry. Poultry red mites come out to feed on poultry at night and during the daytime they normally hide in cracks and crevices under manure, and on roosts of the chicken house, where they deposit eggs. Populations develop rapidly during the warmer months and more slowly in cold weather; the life cycle may be completed in only 1 week. A house may remain infested for up to 9 months after birds are removed.

Transmission

PRM transmission is by dispersion of mite or by contact with infested birds, animals, or inanimate objects. In the integrated poultry industry, mites are dispersed most frequently on inanimate objects such as egg trays, crates, or by personnel going from house to house or farm to farm. The mite - besides sucking blood - may act as a vector for several pathogenic poultry infections that spread diseases caused by bacteria and viruses The mite can transmit several viral diseases (Newcastle disease paramyxovirus, the avian influenza A virus, fowlpox virus, *etc.*) as well as bacterial infections (*Escherichia coli*, *Erysipelothrix rhusiopathiae*, *Pasteurella multocida*, *Salmonella gallinarum* and *S. enteritidis*).

Clinical Signs

The mite feeds on the blood of hens in only 30 to 60 min. The mite induces severe stress on hen health and welfare by causing anaemia. Mite infestations are a problem for hens, inducing reductions in egg production, feed conversion efficiency, body weight gains, and egg size, and also causing significant animal health problems such as increased mortality, stress, weight loss, anaemia, feather pecking or cannibalism and compromised immunity. Heavy infestations of poultry red mites decrease reproductive potential in males also.

Diagnosis

An adult female PRM is approximately 1 mm in length and 0.4 mm in width, and adult colours range from grey to red depending on blood engorgement. The mite has 5 life stages, *viz.*, egg, larva, protonymph, deutonymph, and adult. They can very quickly go from a low-level infestation to having mite visibly hanging down from feeder tracks and drinker lines.

Treatment and Control

PRM is treated by using synthetic neurotoxic acaricides such as organophosphates and pyrethroids. The oral administration of systemic ectoparasiticides, such as fluralaner administered through drinker lines in two treatments, seven days apart. It is totally harmless to hens, but if present in their blood will kill the mites that feed on them.

Use of chemical pesticides and synthetic acaricides for mite treatment in veterinary medicine presents challenges such as ineffectiveness of active ingredients, mite resistance, undesirable residues in the environment, and unacceptable risks to nontarget organisms.

Among the alternative treatments available, botanicals/ethnoveterinary products (also known as natural pesticides or phytochemicals, essential oils) represent an alternative control strategy that is less harmful to the environment and human health. The substances derived from these natural products can be grouped into two categories based on their uses: acaricides are toxic to the pest, and the repellents deter the pest.

Azadirachta indica oil, the neem oil, has a good acaricide effect associated with a low repellent effect at a concentration of 15 to 20 percent under laboratory conditions. Trials with various inorganic chemicals/materials are also going on PRM.

These mites can remain dormant for up to 18 months, hidden in cracks between concrete on commercial farms, where no amount of cleaning or disinfection will reach them. Once birds are placed on the farm and the shed environment heats up, the mite will reactivate and, if untreated, will multiply. Therefore, it requires a comprehensive programme for control of menace due to PRM.

5.5 Duck Diseases

5.5.1 Duck plague

Definition and Causative Agent

Duck plague or duck viral enteritis is caused by Duck enteritis virus – also known as *Anatid alphaherpesvirus1* (AnHV-1), Duck plague is an acute disease affecting duck, geese and swan throughout the globe. All age groups from 1 week of age are susceptible to duck plague. Duck plague outbreaks occur from March to September of the year, although 86 percent of the disease is seen during March to June.

Transmission

The Duck enteritis virus is transmitted by both vertical, horizontal way (principal mode) through contaminated feed, water and bedding material, and by direct contact. The migratory waterfowl are responsible to spread this disease in distance places, as they often act as carrier of this disease. The incubation period of duck enteritis virus is 3-7 days with high mortality rate. The morbidity and mortality of birds range from 5 to 100 percent.

Clinical Signs

The clinical signs depend on age, sex, immune status, species and virulence of the virus strain, *etc*. Severity in clinical symptoms is observed with the progression of infection in the flock. In acute cases, there is sudden and persistent death in a flock due to duck plague. The clinical signs may include depression, loss of appetite, sudden decrease in egg production, *i.e.*, up to 20-40 percent, nasal discharge, increased thirst, ataxia, dropped wing appearance, photophobia, greenish and watery diarrhoea, soiled vent, tremors, and prolapse of penis in male. Some birds often refuse to drink water due to ocular sign and end up having dehydration. The mortality is seen within 5 days of onset of symptoms.

Post-mortem Lesions

The Commonly observed lesions are vascular damage, necrotic changes, eruptions on the mucosal surface and disseminated intravascular coagulopathy of the digestive tract. The degenerative lesions in parenchymatous and lymphoid organs are evident. Severe enteritis, haemorrhage in intestine, body cavities, heart, pericardium, liver and spleen, plaques in oesophagus and intestine are observed. Petechial or larger extravasations of blood could be seen on myocardium and epicardium giving a red paintbrush appearance. This characteristic lesion is often observed in adult ducks rather than in ducklings. In parenchymatous organs like liver, the surface may have pale copper colour with pin-point haemorrhages and white foci giving a speckled appearance which changes to dark bronze colour with bile stains in later stage of infection. Surfaces of organs like pancreas, lungs, and kidney may also show petechial haemorrhages. The pathognomonic lesions of duck plague are petechial haemorrhage in the conjunctivae, mucous membrane of intestine, trachea, and syrinx.

Diagnosis

The clinicals signs and the gross pathological lesions conclude the primary diagnosis. PCR, FAT, immune-chromatographic strip tests and agar gel immunodiffusion tests are used for confirmatory diagnosis. Diagnosis can be strengthened by demonstration of intra-nuclear inclusion bodies in epithelial cells of the digestive, respiratory and reproductive tracts, liver, and spleen.

Differential Diagnosis

Differential diagnosis of duck plague should be made between duck virus hepatitis, fowl cholera, necrotic enteritis, coccidiosis and specific intoxications.

Treatment

Duck plague is a viral disease, as such there is no specific treatment available. However, symptomatic treatment is given as required.

Vaccination

Live attenuated vaccine is administered @1ml/ duck at 8-12 weeks of age through subcutaneous route. Booster dose is given after 1 month interval of the first dose and annual immunization is recommended.

Biosecurity Measures

Contact with wild, free-flying waterfowl and direct or indirect contact with contaminated birds or material (*e.g.*, free-flowing water) should be avoided. Control of DVE is affected by depopulation, removal of birds from the infected environment, sanitation, and disinfection. The disease is prevented through immunization or by maintaining susceptible birds in a disease-free environment.

5.5.2 Duck Cholera

Definition and Causative Agent

It is an important disease of domestic ducks though mostly prevailing in Asia yet is of global concern. It affects both domestic and wild birds with high morbidity and mortality rates. Duck cholera - also known as fowl cholera - is a contagious bacterial disease caused by *Pasteurella multocida*.

Transmission

Transmission of *P. multocida* occurs within a flock primarily by contaminated exudates from the mouth, nose, and conjunctiva of affected birds that act as a source of transmission to their

surrounding environment, feed, and water. Pecking and cannibalism of infected carcasses contributes to disseminate the organism through affected flocks to others.

Clinical Signs

In acute duck cholera outbreak, large number of dead birds without any clinical finding, is usually noticeable. In protracted or chronic cases, symptoms like fever, loss of appetite, ruffled feather, mucous discharge from the mouth, greenish diarrhoea, laboured breathing, *etc*., are seen. Lameness, exudative conjunctivitis, and pharyngitis are often noticed. Infected meninges, middle ear, or cranial bones cause torticollis. Mortality rate often increases rapidly, if untreated. Birds may keep on dying for months.

Postmortem Lesions

Lesions in dead birds include haemorrhages on heart muscle, mesentery and abdominal fat. The liver is enlarged with white narcotic foci, copper coloured and friable (easily crumbled) in nature. Hyperaemia of the abdominal viscera is also seen.

Differential Diagnosis

Differential diagnosis with other infection like *Escherichia coli, Salmonella enterica, Ornithobacterium rhinotracheale*, gram-positive cocci, and *Erysipelothrix rhusiopathiae* (erysipelas) may be carried out.

Diagnosis

Although, clinical sings and lesions can aid the tentative diagnosis in field level, however, confirmatory diagnosis can be done by bacterial culture as well as PCR.

Treatment and Control

Since, duck cholera is a bacterial disease, the use of antibiotics as per antibiotic sensitivity test can be treated. Oxytetracycline, chlortetracycline and third generation cephalosporins may be used. Alternate treatment can be done by use of sulpha drug in water. Suppurative treatment is essential along with antibiotics.

Vaccination

Duck cholera vaccine is available. By vaccinating the flock with killed duck Pasteurella alum-precipitated vaccine, the disease can be prevented. Vaccination is done by injecting 2 ml SC in each duck followed by booster dose after 10 days of the primary dose. Since the ducks are poor natural immune responder, it is always advisable to use immunomodulators like interferon, levamisole and Tulsi leaves (in water).

Biosecurity Measures

All classical biosecurity norms - besides following – need to be implemented: separation of diseased birds by clinical signs from healthy flock; regular cleaning of all the debris, utensils from duck shed and disinfecting it; use of lime wash and disinfectant for floor and walls of the shed, and disposal of carcasses should by either burning or burying at a sufficient depth using disinfectant, lime or salt, *etc*.

5.6 Biosecurity Practices in Poultry Farming

Definition

Biosecurity means a set of farm protocols designed to prevent the exposure of birds to the pathogenic microorganisms. It helps to reduce introduction and transmission of pathogens into and between the farms. In a broader sense, biosecurity encompasses measures like isolation, traffic control, water, feed and environmental quality, worker hygiene, pest control, cleanliness, vaccination, and monitoring that help keep pathogens out of the farm and prevent their entry. An efficient biosecurity programme is vital for profitable poultry farming. Basically, the biosecurity operations in poultry farm are divided into the following categories:

Conceptual Biosecurity

It deals with all operations related to the physical location of the farm and it is primary level of biosecurity. It always accounts for the proximity of the farm to main road, wild bird activity sites such as water bodies, neighbouring farms, abattoirs, *etc*. It should be designed and executed during planning and construction phase of the poultry farm to make sure that sufficient distances should be maintained between different facilities like poultry houses, feed mills, neighbouring farms, main roads, water bodies, *etc*., to prevent the introduction of pathogens.

Structural Biosecurity

Considers the factors pertaining to farm design and layout of farm buildings and other structures. This

is the second level of biosecurity which includes – fencing around the farm with single entry gate to restrict entry of unwanted visitors as well as other domestic and wild animals, poultry housing design and orientation, drainage systems, feed storage, dead bird disposal, change rooms, and washroom facilities for workers. In the intermediate term, structural biosecurity can be improved with the appropriate financial investment. Otherwise, when a new disease or epidemic of a deadly disease, like highly pathogenic avian influenza, occurs; the remedial action taken is too late to prevent further damage.

Operational Biosecurity

It deals with the regular protocols and procedures that are followed in regular day-to-day operations. It is the final line of protection against the incursion of pathogenic micro-organisms. The key components of operational biosecurity include:

1. How the visitors are permitted to access the farms
2. How the routine cleaning and disinfection protocols including rodent and pest control within and outside the farm are practiced
3. How the poultry health monitoring plan including disease control measures are adopted
4. How the depopulation measures during routine culling as well as during disease outbreaks are used

The operational procedures include record maintenance, disinfection procedures at entry gate including vehicle hygiene, all routine sanitization and disinfection protocols such as disinfection of the equipment, poultry houses, worker hygiene, feed, water, environmental sanitation, rodent and pest control measures, dead bird disposal, overall cleanliness within the farm facilities.

In general, the biosecurity operations of commercial poultry farms in Indian field conditions should be as practical as possible and these start from:

i. Selection of farm site
ii. Fencing with single entry
iii. Foot and vehicle bath at farm gate
iv. Restriction of visitors
v. Vehicle hygiene
vi. Worker's hygiene
vii. Safe disposal of farm waste and dead birds - composting and incineration
viii. Rodent and pest control
ix. Regular water sanitization with chlorine di-oxide/hydrogen peroxide or any other Government-approved commercial water sanitizers
x. Routine testing of water and removal of biofilm inside the pipelines, if any contamination is found in water
xi. Feed hygiene practices
xii. Spraying the disinfectants once or twice a week
xiii. Routine sero-monitoring and vaccination and
xiv. Proper terminal disinfection practices including reasonable downtime between the batches.

In contrast to commercial poultry, the backyard poultry are free-roaming in the villages therefore strict biosecurity measures cannot be adopted. Hence,

i. Proper vaccination
ii. Deworming
iii. Daily cleaning of watering utensils and use of chlorine tabs/ potassium permanganate for disinfection of water
iv. Use of suitable litter, its frequent removal and disposal
v. Routine spraying of disinfectants in the perching areas.

Such practices could help to reduce the disease burden in backyard poultry.

ANNEXURE 5.1

Table 5.1: Suggested Vaccination and debeaking schedule for commercial layers

Sl #	Age	Vaccine	Route
	1st Day	MD (HVT+SB1)	SC
		IB Ma-5	IO or beak dip
	5th Day	IB (H120) + LaSota/NDVH Clone	IO
	10th Day	Debeaking	
	13th Day	IBD plus/IBD	IO
		ND killed	SC
	24th Day	IBD+	IO
	30th Day	IB (Ma 5) + LaSota	IO
	35th Day	Infectious Coryza killed (optional)	SC
	42nd Day	Fowlpox	IM/wing web
	48-49th Day	ILT	IO
	55th Day	Fowl Cholera (optional)	SC
	70th Day	R2B or RDVK	IM
	77th Day	Fowlpox (optional)	Wing web/IM
	84th Day	Infectious Coryza /Fowl Cholera (optional)	SC
	86-90 Day	Debeaking	
	100-105 Day ILT	IO	
	110-115 Day	IB+ La Sota	IO
		ND Killed	SC
	150-160 Days	ND Killed	SC

After 22nd week, Mid-lay ND killed can be given during 40th and 60th week depending up on the farm productivity and ND titers.

Alternatively, ND LaSota along with IB-H120/Ma5/ 4/91 can be given once in 6-8 weeks depending upon the farm productivity and ND/IB titers.

Deworming may be carried out at 8-10 weeks.

Depending upon endemicity of infections in different areas, consult local poultry veterinarian for addition/ deletion in schedule.

Table 5.2: Suggested Vaccination Schedule for Broiler Breeders

Sl #	Age	Vaccination	Route
	Day 1	MD	SC
		(IB) H-120	Eye drop
	Day 3	IB Killed	SC
	Day 5	ND Killed +	SC
		ND Clone	Eye drop
	Day 7or 8	IBH Killed	SC
	Day 12	IBD intermediate	Eye drop
		IBD Killed	SC

Sl #	Age	Vaccination	Route
	Day 18	IB 4/91/Mass	Eye drop
		IB Killed	SC
	Day 23	IBD intermediate	SC
	5 Week	ND Killed	SC
	5.3 Week	IB Killed	SC
	6 Week	Fowl Pox Live	Wingweb/IM
	8 Week	SG 9R (*Salmonella*)	SC or IM
	9.1Week	ND+ IB Multi Killed	SC
	10 Week	Tri- Reo Killed	SC
	11 Week	FC Killed	SC
	11.3 Week	AE+Pox	Wing web
	12 Week	IBH Killed	SC
	12.3 Week	Salmonella Killed	SC
	13 Week	EDS -76 Killed	SC
	14 Week	AE Killed	SC
	15 Week	MG- Bac Killed	SC
	15.3 Week	FC +IC Killed	SC
	16 Week	Tri-Reo Killed	SC
	17 Week	ND Killed (Genotype XIII)	SC
	18 Week	AE Killed	SC
	20 Week	IB Killed	SC
	22 Week	4 way (ND+IBD+IB+ Reo)	SC
	23 Week	IBH Killed	SC
	31-32 Weeks	ND + IB Multi Killed	SC
	39 Week	IB (Nephro) Killed	SC
	40 Week	4 way (ND+IBD+IB+ Reo)	SC
	42 Week	IBH Killed (Rainy season)	SC
	43 Week	IB New Killed	SC
	48 Week	ND+IB Multi Killed	SC
	58 Week	ND+ IB Multi Killed	SC
	68 Week	ND+ IB Multi Killed	SC

Table 5.3: Vaccination schedule for commercial broilers

Sl #	Age	Vaccine	Route
1.	1st Day	ND Killed	SC
		ND+IB	Spray or IO
2.	14	IBD+	IO
3.	19-21	ND live	IO

*Alternative to ND is HVT Vector-based ND or ND+IBD. Such vaccines can be given at the time of hatching on 18th day or on day-old chicks.

***Alternative to IBD - Immune complex vaccines can be given in ovo or on day-old.**

Table 5.4: Vaccination schedule for backyard poultry

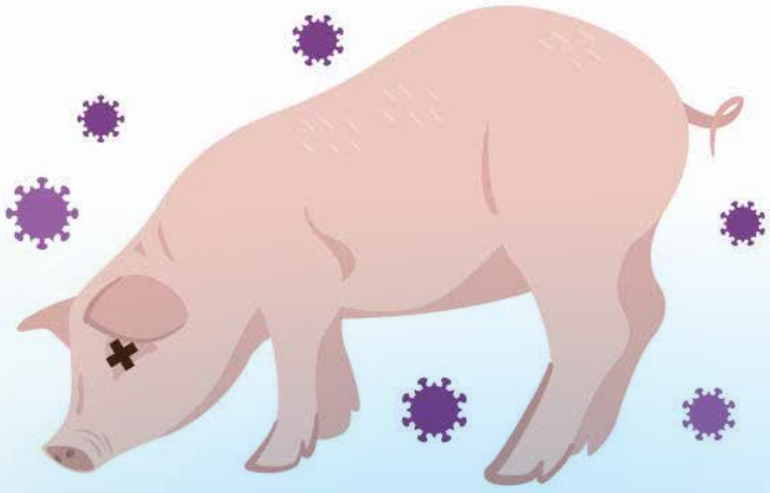
Sl #	Age	Vaccine	Route
1.	7th Day	ND-B1	IO
2.	14th Day	IBD	IO
3.	28th Day	LaSota	IO
4.	45th Day	Fowlpox (Optional)	IM
5.	55th Day	Infectious Coryza (Optional)	SC
4.	70th Day	LaSota	IO
5.	120th Day	LaSota	IO

Note:

*Ensure before vaccination that flock is healthy and there is no concurrent infection

CHAPTER- 6

GUIDELINES FOR PIG DISEASES



Contents

Abbreviations

AGID	agar gel immunodiffusion test
ASF	African swine fever
ASFV	African swine fever virus
BW	body weight
CFT	complement fixation test
c-ELISA	competitive ELISA
CSF	classical swine fever
CSFV	classical swine fever virus
dL	decilitre
ELISA	enzyme-linked immunosorbent assay
ETEC	Escherichia coli
FAT	fluorescence antibody test
FMD	Foot and mouth disease
FMDV	Foot and mouth disease virus
HAD	haemadsorption tests
HA	haemagglutination
HI	haemagglutination inhibition
IBT	immunoblotting test
IFA	indirect fluorescent antibody
IFAT	immunofluorescence antibody test
IHC	immunohistochemistry
IM	intra-muscular
IPT	immunoperoxidase test
IU	international units
IV	intravenous
JE	Japanese encephalitis
JEV	Japanese encephalitis virus
kg	kilogram
MALT	mucosa associated lymphoid tissue
MAT	Microscopic Agglutination Test
MIC	minimum inhibitory concentration
MBTC	Mycobacterium tuberculosis complex
mg	milligram
MHD	mulberry heart disease
ml	millilitre
mm	millimetre
NSAIDs	non-steroidal anti-inflammatory drugs
PCR	polymerase chain reaction
PCR-RFLP	PCR-restriction fragment length polymorphism
PCV-2	Porcine circovirus-2
PCV2-ED	PCV2-enteric disease

PCV2-LD	PCV2-lung disease
PCV2-RD	PCV2-reproductive disease
PCV2-SD	PCV2-systemic disease
PCVDs	porcine circovirus diseases
PCVAD	porcine circovirus-associated diseases
PDNS	porcine dermatitis and nephropathy syndrome
PED	porcine epidemic diarrhoea
PEDV	porcine epidemic diarrhoea virus
PMWS	post-weaning multisystemic wasting syndrome
PO	oral administration
ppm	parts per million
PPV	porcine parvovirus
PRDC	porcine respiratory disease complex
PRNT	Plaque Reduction Neutralization Test
PRRS	porcine reproductive and respiratory syndrome
qRT-PCR	quantitative RT-PCR
RAPD	random amplified polymorphic DNA
RBPT	Rose Bengal Plate Test
RFLP	Restriction Fragment Length Polymorphism (RFLP
RT-PCR	Reverse-transcription polymerase chain reaction
RV	Rotavirus
SAT	Serum Agglutination Test
SIV	swine influenza virus
SC	sub-cutaneous
SD	single dose
SwPV	swinepox virus
SVD	swine vesicular disease
SVDV	swine vesicular disease virus
SVV	Seneca Valley virus
TD	total dose
TGE	transmissible gastroenteritis
TGEV	Transmissible gastroenteritis virus
VNT	virus neutralization test
WOAH	World Organization for Animal Health

6.1. Preamble

Pig farming provides substantial livelihood and income generation opportunities to the economically backward sections of society. With the consumption of pork increasing in India, pig rearing is also expanding, with a significant number of livestock farmers taking it up, as rural and peri-urban communities find it a cost-effective source of income. However, despite the huge economic potential of the piggery sector, the occurrence of various diseases, particularly the infectious diseases that result in huge mortality, is hampering pig production and productivity. The problem is exacerbated by the limited availability of information on the frequency of occurrence of the different diseases and their detrimental effects on pig production. The non-availability of proper treatment guidelines and control measures for common diseases of pigs creates further obstacles to the development of the piggery sector in India. These Standard Treatment Guidelines for swine diseases have been prepared with the objective of providing a ready reckoner for the treatment of prevailing common diseases of pigs in India. These guidelines will be very useful for the field veterinarians and will enable efficient and cost-effective treatment of porcine diseases in India. The current document is developed based on available literature on porcine diseases in India and Expert consultation. The 36 porcine diseases covered in this document have been classified into two main categories – infectious diseases and non-infectious diseases. Infectious diseases include viral, bacterial and parasitic diseases whereas non-infectious diseases include metabolic diseases, deficiency diseases and toxicity-related diseases.

6.2. Infectious diseases of pig

6.2.1 Viral diseases

6.2.1.1 Classical swine fever

Definition and causative agent

Classical swine fever (CSF), or hog cholera, is a highly infectious viral disease of domestic, feral and wild suids. It is caused by a small single-stranded positive sense RNA virus of genus *Pestivirus* under family Flaviviridae. The virus is closely related to the border disease virus and bovine viral diarrhoea virus.

CSF is a notifiable and economically important disease of swine. The disease was first reported from the United States of America in 1810 and spread worldwide after 1960. It is endemic to many countries, though there are some which have been declared CSF-free territories. In India, the State of Uttar Pradesh reported the first case in 1944, and the disease is now prevalent in all the pig rearing states. The north-eastern states have the highest pig population in India and, hence, CSF is endemic and among the most economically important diseases of pigs in this region. The common genotypes found in India are 1.1, 2.1 and 2.2.

Transmission

The CSF virus (CSFV) can be transmitted directly or indirectly. Direct contact between healthy and infected pigs is the most common method of transmission. The virus is excreted in nasal secretions, saliva, urine and faeces. The most common route of transmission is oronasal through direct or indirect contact with infected pigs and wild boars (which are carriers), consumption of virus-contaminated feed or swill, infected raw pork and pork products, and artificial insemination. There can be vertical transmission from infected sows to foetuses throughout the period of gestation.

Clinical signs

The incubation period of the disease ranges from 3–10 days. The acute form is characterized by fever, lethargy, general weakness, anorexia, conjunctivitis, respiratory distress, diarrhoea, neurological signs such paresis, incoordination, paralysis and convulsions. Skin haemorrhages or cyanosis can be seen at the ears, limbs, tails, extremities and ventral abdomen. In the chronic form, intermittent fever, chronic enteritis, anorexia, depression, cough, diarrhoea, staggering gait, purple discolouration of the skin and wasting is commonly encountered. Abortion, stillbirth, foetal mummification, malformations and congenital tremors are observed in prenatal infection.

Lesions

The common lesions observed in the acute form of CSF are swollen and haemorrhagic lymph nodes, and petechial haemorrhages in the inner organs particularly in the kidneys, spleen, gastro-intestinal tract and tonsils. Spleen infarction is considered as pathognomonic for CSF. In the case of chronic infection, the haemorrhagic lesions are not

common, but there are alterations in certain organs which include atrophy of the thymus, ulcerative and necrotic lesions in the intestine (button ulcers) and necrotic ulcers in larynx and epiglottis.

Diagnosis

Isolation of CSFV in a suitable cell line (PK-15, SK6, STE) is a classical method of diagnosis. The CSFV antigen can be detected in organs of affected pigs by methods like sandwich enzyme-linked immunosorbent assay (ELISA) and fluorescence antibody test (FAT). The genomic RNA of CSFV can be detected in infected pigs by sensitive molecular assays like reverse transcriptase polymerase chain reaction (RT-PCR) or real-time based quantitative RT-PCR (qRT-PCR). Antibodies against CSFV can be detected by indirect ELISA or indirect immunoperoxidase test (IPT) but cross-reacting antibodies against bovine viral diarrhoea virus (BVDV) and bovine viral diarrhoea (BVD) may interfere with CSF diagnosis.

Differential diagnosis

Differential diagnosis is to be made with African swine fever, porcine circovirus 2 infection, porcine reproductive and respiratory syndrome (PRRS), as well as with bacterial septicaemia which include *erysipelas*, *pasteurellosis*, *salmonellosis*, *actinobacillosis* and infections with *Haemophilus parasuis*.

Treatment and control

Treatment

There is no specific treatment for CSF. In a majority of cases, the affected animals are treated with supportive medication – non-steroidal anti-inflammatory drugs (NSAIDs) like meloxicam @ 0.5 milligram (mg)/kilogram (kg) body weight (BW) single dose or repeated after 24 hours, antihistaminics like Chlorpheniramine maleate @ 0.5mg/kg BW IM every 24 hours for three to five days, multivitamin injection @ 1–2 millilitres (ml) IM for three to five days.

If secondary bacterial infection is suspected, antibiotics like Enrofloxacin @ 5mg/kg BW IM every 24 hours for three to five days or Ceftriaxone @ 3–5 mg/kg, IM, every 24 hours for three consecutive days should be given.

Control

Live attenuated vaccines are used globally as they have the capability to prevent CSF. Different strains of CSFV are used to prepare the live attenuated vaccines. In endemic areas, the primary dose is given just after weaning and a booster is given 30 days after the primary vaccination. Revaccination is done at six-month intervals.

Biosecurity measures

The pig farms should have fences, good hygiene practices should be followed in the farm premises and there should be dedicated decontamination tanks for footwear. Newly purchased animals should be quarantined for at least 15 days. Farm equipment, utensils, clothes and shoes of workers should be disinfected, and the entry of visitors should be restricted. Virucidal agents containing active ingredient potassium monopersulfate can be used for the disinfection of pig sheds, including equipment.

6.2.1.2 African swine fever

Definition and causative agent

African swine fever (ASF) is a highly contagious, fatal haemorrhagic viral disease of domestic and wild pigs, which can cause mortality approaching 100 percent. ASF is a transboundary disease and a major threat to the global swine industry. It is caused by the African swine fever virus (ASFV) which is a large enveloped, double-stranded DNA virus belonging to *Asfivirus* genus under the family Asfarviridae. Till date, 24 genotypes (I-XXIV) and eight serotypes of ASFV has been identified.

ASF was first reported from Kenya in 1921 and remained confined to the African countries for a long period. In due course, it has spread to Europe and Asia. In India, the first report of ASF was from two north-eastern states of Assam and Arunachal Pradesh in early 2020. However, within two years it spread to other north-eastern states as well as to several states of southern and northern India. The genomic sequence analysis revealed that the most circulating genotype of ASFV in India belongs to genotype-II.

Transmission

The disease can be transmitted to healthy pigs by direct contact with the oral and nasal fluids, blood, faeces and urine of infected pigs through various routes. Soft ticks of the genus *Ornithodorus* are the

natural reservoir of the virus and help in spreading the disease in certain geographical locations. Wild pigs of Africa, including bush pigs (*Potamochoerus porcus*), wart hogs (*Phacochoerus aethiopicus*), and giant forest hogs (*Hylochoerus meinertzhageni*), act as reservoir hosts and these wild species are resistant to the disease. Indirectly, the disease can be transmitted through contaminated feed, pork and pork products, and fomites (clothes, shoes, vehicles, farm equipment and the like).

Clinical signs

The incubation period of the disease usually ranges from 4–19 days. Depending on the virulence of the virus, a wide range of clinical manifestations are encountered which includes per-acute, acute, sub-acute, chronic and sub-clinical. In the per-acute or acute form of the disease, there is high fever, respiratory distress, oozing of blood, nasal and conjunctival discharge, anorexia, lethargy, diarrhoea, vomiting, abdominal pain, abortion, haemorrhages in the skin and internal organs and death (mortality ɑ100 percent). In per-acute cases, there may be sudden death without any clinical signs being exhibited. In the sub-acute form, while the clinical symptoms are similar to the acute cases - the severity is low and mortality ranges from 30–70 percent. The chronic cases may be marked by intermittent or low fever, respiratory distress, emaciation, growth retardation, arthritis, multifocal necrosis in the skin, abortion and stillbirth.

Lesions

The common lesions that are encountered in acute cases are haemorrhagic splenomegaly, multifocal haemorrhages lymphadenitis and petechial haemorrhages in the surface of kidneys Sometimes haemorrhages are observed in other organs such as heart, walls of the urinary bladder, stomach and intestines and the gallbladder may also be distended. Similar lesions in the spleen and lymph nodes are also observed in the sub-acute form. Other changes include hydropericardium, ascites and multifocal oedema in the perirenal fat and in the wall of the gall bladder. Vascular changes are rarely observed in the chronic form.

Diagnosis

Suspected diagnosis is done based on disease epidemiology, clinical signs and lesions. Laboratory confirmation is done by virus isolation and identification by haemadsorption tests (HAD). PCR or real-time PCR will help in reliable detection of the genomic DNA of ASFV. Antigens may be detected by FAT or sandwich ELISA whereas the antibodies can be detected by ELISA, indirect immunofluorescence assay, IPT and immunoblotting test (IBT). In per acute cases, no antibodies can be detected.

Differential diagnosis

ASF should be differentiated from CSF. As it is difficult to differentiate on the basis of clinical signs and lesions, laboratory confirmation is essential. It should also be differentiated from Aujeszky's disease or pseudorabies, PRRS, porcine dermatitis-nephropathy syndrome and other bacterial septicaemia such as erysipelas, salmonellosis and pasteurellosis.

Treatment and control

Treatment

There is no specific treatment for ASF.

Control

Presently, no vaccine for ASF is available in India. Enforcement of strict biosecurity measures is the main method of control.

Biosecurity measures

Adoption of biosecurity measures is essential for preventing the spread of ASF to nearby farms or healthy pigs. Zoning and compartmentalization are very helpful approaches and should be followed as per national guidelines for control of the disease. Besides biosecurity, bio-exclusion and bio-containment measures are also necessary. Swill feeding should be avoided to the extent possible. There should be restrictions on the movement of pigs, pork and pork products across intra-national and international boundaries. Vehicles, clothes, shoes and equipment should be disinfected. Quarantine is essential before introduction of new pigs or piglets to a farm. Infected carcasses and culled animals should be disposed of properly.

6.2.1.3 Foot and mouth disease

Definition and causative agent

Foot and mouth disease (FMD) is an acute and highly contagious viral disease affecting cloven-hoofed animal species. It is characterized by the formation of vesicles and erosions in the buccal cavity

and on the feet, teats and nose. It has a significant impact on the economy, as the trade of livestock and their products is disrupted both regionally and internationally. Mortality is rare in adult animals but can be high in young animals due to myocarditis or lack of milk when the dam is infected. FMD is endemic to India and regular episodes of outbreaks have been reported from different species of animals, including pigs, every year.

FMD is caused by the foot and mouth disease virus (FMDV) that belongs to the genus *Aphthovirus*, under the family Picornaviridae. At present, there are seven viral serotypes, *viz.*, O, A, C, Asia 1, South African Territories (SAT) 1, SAT 2 and SAT 3. These serotypes do not provide immunity against one another. FMDV serotypes O, A, and Asia-1 are present in India.

Transmission

Animals get infection when they come in direct contact with another infected animal, by feeding on products contaminated with FMDV, or when placed in an environment that is highly contaminated with the virus. Transmission of FMDV may also occur via fomites. Pigs are resistant to natural aerosol infection of FMDV, but pigs infected with FMDV are considered robust emitters of the airborne virus and are also referred to as amplifier hosts of the FMDV. Primary infection occurs in the epithelium overlaying the MALT (mucosa associated lymphoid tissue). Infected pigs produce more virus in aerosol form than ruminants do. Ruminants act as carriers after recovery, but pigs do not show any carrier stage. Virus shedding can be seen in all excretions and secretions, and the virus can be found in milk and semen four days before the onset of clinical signs in a diseased animal.

Clinical signs

The incubation period of the disease varies from 1 to 14 days based on the dose and strain of virus, route of infection, environment around the animal and individual susceptibility. When the exposed viral dose is low, then infected animals may only show sub-clinical or mild disease which may be characterized by no detectable clinical signs or lesions and very low level or undetectable viraemia and antibody response.

Clinical signs in the initial stages may be characterized by fever, inappetence and the infected animal may show reluctance to move. Severe lesions may occur on the feet, and lameness along with blanching of coronary bands may be observed initially. Chronic lameness may occur in adult pigs due to ruptured blisters, and disease recovery depends on the severity of lesions. Blisters heal within seven days – they can take longer sometimes – but complications can occur due to secondary bacterial complications. Weight gain in fattening pigs may be slower. Sudden deaths may occur in pigs below 14 weeks age. Piglets are highly susceptible, and mortality may be considered as the first sign of disease in a herd. Disease morbidity can be as high as 100 percent in non-vaccinated herds. Mortality in adult animals is low (1–5 percent) but can be very high in piglets (20 percent or more) under weaning age.

Lesions

Vesicles are generally seen on the snout, tongue and interdigital clefts. Commonly, lesions are found on the feet. Sometimes lesions can also be found on the teats of lactating sows, or the scrotum of boars. Hoof may also slough. Vesicles generally rupture and haemorrhages are seen on underlying tissue because of sloughing of the superficial epidermis. In piglets, necrosis of the myocardium is often observed.

Diagnosis

Diagnosis of the disease can be made based on the clinical signs and lesions. FMD should be tentatively considered in farms showing sudden high mortality in piglets, lameness, fever and vesicular lesions in a considerable number of pigs. FMD is a notifiable disease and should be reported immediately whenever a vesicular disease is seen in domestic animals.

Disease can be confirmed by laboratory diagnosis by employing different techniques. Detection and identification of the causative agent can be done by virus isolation and immunological methods like ELISA and complement fixation test (CFT). According to the World Organization for Animal Health (WOAH), ELISA is preferred over the CFT as it is more sensitive and not affected by pro- or anti-complementary factors. Nucleic acid recognition methods like agarose gel-based RT-PCR and real-time RT-PCR can be performed for the presence of the virus. Serological tests can be performed for FMD in order to confirm the suspected cases of disease, to import or export disease-free animals

and for demonstration of vaccine efficacy. Virus neutralization test (VNT), solid-phase competitive ELISA (c-ELISA) and liquid-phase blocking ELISA can be performed under serological tests. Presence of antibodies against non-structural proteins of FMDV can be detected by indirect ELISA and enzyme-linked immuno-electro-transfer blot assay (EITB).

Differential diagnosis

Diseases like vesicular stomatitis (VS), swine vesicular disease (SVD) and vesicular exanthema (VE) show clinical signs which may be indistinguishable from FMD in pigs, so laboratory confirmation of FMD is important to rule out these diseases.

Treatment and control

Treatment

There is no specific treatment for FMD, but supportive treatment can be provided for good recovery and to get rid of complications. Antibiotics are given to avoid or reduce secondary bacterial infections of blisters. Foot and mouth wash solution containing ingredients like 1 percent potassium permanganate ($KMnO_4$) or 2 percent sodium bicarbonate solution, 2 percent copper sulphate or 2–4 percent sodium carbonate can be used. Boro-glycerine (one part of boric acid and nine parts of glycerine) can be used on the mouth lesions for quick recovery.

Control

Disease can be controlled by identifying and eliminating the source of infection, which can be achieved by epidemiological surveillance programmes. Contact between infected and healthy, but susceptible, animals should be avoided. The susceptible animal population can be reduced by vaccination. Ring vaccination around the affected area can contain the disease to some extent. FMD-free countries or areas prevent the entry of disease by imparting strict import rules on animals and their products. The most convenient strategy to control and prevent the disease can be chosen locally on the basis of the epidemiological status, recovery of animals and economic resources.

Biosecurity measures

WOAH recommends the following four steps to protect animals from the disease:

Clean: All surfaces should be scraped, scrubbed and swept regularly to remove organic waste.

Wash: All surfaces should be soaked with water and detergent and later washed by spraying, wiping or scrubbing. Other good alternatives are steam and high-pressure washers.

Disinfect: Thorough application of disinfectants as per their recommended concentrations and contact time should be ensured.

Track: Record keeping of vehicle, equipment and holding yard cleaning should be up-to-date and followed diligently.

6.2.1.4 Transmissible gastroenteritis

Definition and causative agent

Porcine transmissible gastroenteritis (TGE) is an extremely contagious enteric disease of pigs. The disease is characterized by severe diarrhoea and vomiting in neonatal pigs that can cause mortality up to 100 percent in piglets below two weeks of age. The disease is caused by the transmissible gastroenteritis virus (TGEV) belonging to the genus *Alphacoronavirus* of the family Coronaviridae. TGEV is a single-stranded, positive sense RNA virus and the virion exhibits "corona-like" structure due to the presence of spike protein at its outer surface. The TGEV was first reported in 1946 in the United States. Although TGEV is reported from many swine-populated countries, there is limited documentation from India pertaining to detection of the antigen or nucleic acid of the virus. However, detection of antibodies against TGEV in pig serum samples collected from the State of Assam has been reported.

Transmission

TGEV is transmitted directly via oral and nasal routes, as the virus is readily excreted in the faeces and nasal secretions of infected pigs. Although faeces are the main source of infection, infected sows can transmit the virus to piglets via milk. Other mechanical carriers of the virus are fomites, insects and other animals like fox, cat and dogs.

Clinical signs

Watery diarrhoea is the common clinical manifestation of the disease. Other major clinical signs are vomiting, dehydration and weight loss. Neonatal piglets generally die within two to three

days after the onset of clinical signs. However, piglets above four weeks of age often survive. In adult pigs, besides diarrhoea and vomiting, there is loss of appetite with low mortality, and they usually recover spontaneously within 5–10 days.

Lesions

In piglets, the intestine may be filled with undigested milk curd and yellow, foamy, odoriferous fluid. Due to malabsorption of fat in the small intestinal mucosa, the mesenteric lymphatics may be devoid of chyle.

Diagnosis

Diagnosis can be done based on clinical signs. Confirmatory diagnosis can be done by isolation of the virus from faeces and small intestine. Virus antigen is detected by the fluorescent antibody test and sandwich ELISA. Nucleic acid of TGEV is detected by molecular tests such as RT-PCR and qRT-PCR. Serological tests to detect TGEV specific antibodies are virus neutralization test (VNT) and ELISAs.

Differential diagnosis

The disease should be differentiated from rotavirus infection, porcine circovirus 2 infection, colibacillosis, *Strongyloides* infection and porcine epidemic diarrhoea.

Treatment and control

Treatment

There is no specific treatment. Supportive treatment can be provided to correct dehydration. Provision should be made for oral electrolytes and free access to water. Oral fluid intake will help in correcting severe dehydration, especially in infected pigs that are older than three to four days of age. Zinc oxide within permissible limits (150 parts per million or ppm) can be given to the affected animals.

Control

Currently, no vaccine is available in India. Control relies on the adoption of strict biosecurity measures.

Biosecurity measures

TGE outbreaks can be prevented by following strict biosecurity measures, especially in winter. Practice of strict "All-in-all-out" system helps to prevent the disease. Thorough cleaning and disinfection of the farm is essential. Farm building structures should have the potential to exclude animal carriers (rodents, cats, dogs and birds among others). There should be minimum human traffic and fomites should be disinfected regularly.

6.2.1.5 Porcine reproductive and respiratory syndrome

Definition and causative agent

Porcine reproductive and respiratory syndrome (PRRS) is one of the most economically important diseases of pigs throughout the world. It is mainly characterized by reproductive failure in breeding animals and respiratory disease in any age group of pigs. The causative agent is PRRS virus which is an enveloped single-stranded RNA virus in the genus *Porartevirus* under family Arteriviridae. At present, PRRSV exists as two distinct virus species, – PRRSV-1 (known as the European genotype, type strain Lelystad) and PRRSV-2 (known as North American genotype, strain VR-2332).

Transmission

The most common method of PRRSV transmission is close contact between pigs, or exposure to contaminated body fluids. One of the important aspects of PRRSV infection is the ability of infected pigs to transmit the virus for period longer than 200 days. Infected pigs with few or no symptoms shed the virus, and this is probably the most common method of introduction of PRRSV to a herd. The virus is highly infectious, with the infectious dose being as little as 10 virus particles. Nasal secretions, urine, semen, mammary secretions and faeces of affected animals contain the virus. Infected pregnant sows may deliver persistently infected piglets as a congenital PRRS virus infection. Virus can transmit from infected piglets or dams to other piglets. The virus is excreted in semen for up to 92 days after infection and can serve as source of infection for dams. Mechanical transmission by mosquitoes and house flies is also possible.

Clinical signs

Gilts, sows and boars

Clinical signs such as high temperature, loss of appetite, lethargy, depression, respiratory distress and nausea may be observed in affected animals. Bluish discolouration of the ears, abdomen and vulva may also be seen in some cases. The most common reproductive problems often observed are increased

premature farrowings, late abortions, stillbirth or birth of weak piglets and mummification. Nursing pigs may show signs of dyspnoea ("thumping") and pre-weaning mortality is high.

Young, growing and finishing pigs

The main clinical signs in young pigs include increase in temperature, melancholy, dullness and pneumonia. Sneezing, high temperature and dullness are generally followed by dyspnoea and stunting. Respiratory disease reaches a peak in pigs in the age group of 4–10 weeks. Older pigs also exhibit similar respiratory signs.

Lesions

Swelling of the lymph nodes is generally observed. In uncomplicated cases of PRRS, the majority of the foetuses and stillborn pigs will not have distinguished lesions, however, in some cases - enlarged lymph nodes, swollen eyelids, haemorrhagic skin and dehydration may be observed.

Diagnosis

In acute outbreak cases, clinical signs and history may suggest involvement of PRRSV. Diagnosis can be made based on either isolation of virus, antigen detection by FAT or immunohistochemistry (IHC), or detection of PRRSV by PCR. Serological tests will be helpful in assessing the presence and stage of PRRS infection in the herd.

Differential diagnosis

PRRS should be differentiated from respiratory diseases of pigs caused by pathogens such as influenza A virus, porcine circovirus 2 (PCV2), porcine respiratory coronavirus, pseudorabies virus, *Mycoplasma hyopneumoniae*, *Actinobacillus pleuropneumoniae*, *Streptococcus suis*, *Salmonella choleraesuis*, *Haemophilus parasuis* and *Pasteurella multocida*. PRRS should also be differentiated from reproductive failure in pigs caused by agents such as porcine parvovirus (PPV), influenza A virus, porcine teschovirus/enterovirus, pseudorabies virus, CSFV and leptospirosis.

Treatment and control

Treatment

There is no specific treatment. Antibiotics may be used to control secondary bacterial infections. In case of acute disease, anti-inflammatory products can be administered. Vitamin supplementation and provision of electrolytes to affected animals are also important.

Control

There is no vaccine for PRRS in most countries. Accurate diagnosis of the disease is of paramount importance to know the disease status in the farm. Clear understanding on the PRRS status of replacement gilts and boars is very essential to prevent the introduction of PRRS to a herd. In addition, proper isolation and acclimatization of the incoming stock are also critical in order to prevent PRRS getting introduced into a herd. After successful elimination of PRRS from a herd, adoption of biosecurity measures like strict adoption of quarantine procedure, regular testing, the purchase of animals/semen only from PRRS-negative herd, proper sanitation of animal carriers, regulation of personnel movement including fomite in the farms as well as their premises must be ensured to prevent re-entry of the virus into the herd.

Biosecurity measures

A quarantine facility is an essential requirement of a PRRSV biosecurity programme. It should be more than 120 meters away from the breeding unit. It is important to keep the incoming animals in the quarantine shed for at least 30 days. Farm personnel should monitor these newly brought animals daily for any clinical signs. Blood samples of the newly introduced animals should be tested within 24–48 hours after arrival in the isolation facility as well as five to seven days ahead of their entry to the breeding unit. It is advisable to test the samples by PCR to enhance the detection of per-acute infections. Samples collected late in the isolation period can also be tested by ELISA for PRRSV antibodies. All routine biosecurity measures should be strictly adopted so that the entry and spread of PRRSV to the herd can be prevented. If infection is detected in the farm, affected animals should be isolated at the earliest and treated individually. In case an animal dies, the carcass must be buried deeply and covered with adequate lime. No new pigs should be introduced to the farm during an outbreak.

6.2.1.6 Porcine circovirus 2 infection

Definition and causative agent

Porcine circovirus 2 (PCV-2) is the smallest known virus that affects pigs of all ages and is associated with a wide range of clinical syndromes collectively

called porcine circovirus-associated diseases (PCVAD). These diseases include post-weaning multisystemic wasting syndrome (PMWS), PCV-2-lung disease (PCV-2-LD), porcine dermatitis and nephropathy syndrome (PDNS) and reproductive disorders. It is a highly infectious virus that can lead to huge economic losses in the swine industry due to elevated mortality rates, sub-optimal growth performance and reproductive failures. It belongs to the *Circovirus* genus of the Circoviridae family. PCV-2 has been classified into eight distinct genotypes (PCV-2a to PCV-2h) based on a recent genotyping method. In addition, a new genotype – PCV-2i – has also been identified recently in the United States. In India, six genotypes – PCV-2a, PCV2b, PCV-2d, PCV-2f, PCV-2g and PCV-2h – and a few recombinants are circulating. There are reports of PCV-2 from almost all regions of the country.

Transmission

Like most viral infections, transmission of PCV-2 occurs by direct contact with infected pigs. It has been detected in most natural secretions and excretions of affected pigs such as nasal, ocular, saliva, urine and faeces. PCV-2 has also been detected in the semen of affected boars, but it is practically significant only when present in high loads. It is also assumed that contaminated feeds, fomites and insects may play a role in disease transmission.

Clinical signs

Diseases caused by PCV2 are collectively called PCVADs or porcine circovirus diseases (PCVDs). PCV2-systemic disease (PCV2-SD), which was previously called PMWS, affects pigs of the 8–16 weeks age group and is characterized by wasting, diarrhoea, skin pallor, jaundice, dyspnoea and lymph node enlargement. PCV2-reproductive disease (PCV2-RD) is clinically characterized by abortions in late terms, still-births and mummifications. PCV2-lung disease (PCV2-LD) causes symptoms of fever, dyspnoea, cough and anorexia in affected pigs, which are mostly of the 14–20 weeks age group. PCV2, along with other pathogens, contribute to the development of PRDC (porcine respiratory disease complex) in pigs. PCV2-enteric disease (PCV2-ED) is clinically manifested as diarrhoea, colitis and enteritis. Previously, PCV2-LD and PCV-2 ED were considered as separate disease conditions, but recent studies have revealed that both the conditions may be a part of the systemic disease. PDNS is one of the most important disease conditions caused by PCV2. While it affects mostly weaners, growers and finisher pigs, breeding pigs are also affected sporadically. This condition in pigs is characterized by the occurrence of macules and papules on the perineal area of the hindquarters, limbs, thorax and ears. Sometimes, the skin lesions merge to form large irregular patches. General clinical signs such as anorexia, depression, stiff-gait and prostration are also observed. The most common form in which PCV-2 is manifested in pigs is the PCV-2-subclinical infection (PCV2-SI). In this, there is an increase in the average daily loss in the pigs without showing any discernible clinical signs.

Lesions

The lymph node is substantially enlarged and pale on the cut surface in PCV2-SD affected pigs. Splenic infarcts, atrophied thymus, and thinner than normal tonsils are some other gross lesions. When the lung is affected, the severity of the lesions depends upon the disease duration and presence of concurrent infections. Firm lungs which fail to collapse, presence of diffuse pulmonary oedema, mottling and consolidation are common lesions. In PCV2-RD, stillborn and non-viable neonatal piglets exhibit chronic passive congestion of the liver and cardiac hypertrophy, along with multifocal areas of myocardial discolouration. The lesions in PDNS are easily recognized as red or dark red macules and papules on the skin. Pigs that die of PDNS have firm and bilaterally enlarged kidneys.

Diagnosis

PCVAD is diagnosed on the basis of three criteria – presence of specific clinical signs, presence of specific gross and corresponding microscopic lesions in affected organs, and presence of PCV2 antigen or DNA in lesions. Herd diagnosis is very important for the prevention and control of PCVAD. This is done on the basis of two criteria – significant increase in post-weaning mortality and clinical signs compatible with PCVADs, and fulfilment of individual criteria of diagnosis in at least one out of three to five affected pigs. Currently, the most commonly used technique for the detection of PCV2 antigen or nucleic acid in lesions include IHC, *in situ* hybridization and PCR, including the different variants of PCR. Virus isolation can be done on several cells of porcine origin, the most common being PK-15 cells. Serological techniques including

indirect fluorescent antibody test (IFA) and ELISA are used for the detection of PCV2 antibodies, but they cannot be used for clinical diagnosis of PCVADs as PCV2 antibodies may also be present in healthy pigs.

Differential diagnosis

Differential diagnosis of PCVADs depends upon the type of syndrome exhibited. For example, differential diagnosis of PCV-2-SD will include other conditions causing decreased growth rates and increased mortality in piglets such as PRRS, Glasser disease, and Salmonellosis, to name a few. The differential diagnosis of PCV-2-RD include PRRS, Leptospirosis, pseudorabies, porcine parvovirus infection, among others. Cutaneous signs of PDNS need to be differentiated from ASF, CSF, swine erysipelas, porcine stress syndrome, septicaemic salmonellosis, transient erythema caused by chemical burns, contact with urine-soaked floors, among others. Kidney lesions in PDNS should be differentially diagnosed from ASF, CSF, swine erysipelas, and septicaemic salmonellosis.

Treatment and control

Treatment

There is no specific treatment. Anti-inflammatory drugs and antimicrobials may be used. Antibiotics given in feed or water may suppress secondary infections. Drugs like ivermectin has potent antiviral activity towards PCV2 both *in vitro* and *in vivo*. Vitamin D3 can be used parenterally as this vitamin plays a central role in the immune response of swine. It may be injected at the anticipated time of onset of infection in piglets.

Control

Vaccination is considered the most effective way to control PCV2 infections. Vaccine (Ingelvac Circo FLEX®) is administered @ 1 millilitre (mL) dose by the I/M route to pigs at three weeks of age. Other control measures include enforcement of strict biosecurity measures, control of concomitant viral infections, especially PRRS, and vaccination against commonly prevalent bacterial pathogens.

Biosecurity measures

Enforcing strict biosecurity and sanitary measures is pivotal to prevent or control PCV2 infections. "All-in-all-out" practice, limited contact between animals, isolation of sick pigs and prevention of cross-fostering and mixing of batches is crucial. Proper disinfection of transport vehicles and buildings should be done. Addition of antioxidant feed additives, spraying of dried plasma and conjugated linoleic acid in piglet feed, coupled with the use of anti-parasitic drugs such as ivermectin, have also been seen to have controlled PCVADs.

6.2.1.7 Porcine rotavirus infection

Definition and causative agent

Porcine rotavirus infection is a viral infection that primarily affects young pigs, causing acute gastroenteritis characterized by diarrhoea, dehydration and weight loss. Although pigs of all ages are susceptible, the incidence among nursing and weaned piglets is higher. Rotavirus infection in pigs leads to productivity losses, so it is an economically significant swine pathogen. The causative agent – Rotavirus (RV) - belongs to the genus *Rotavirus* within the Sedoreoviridae family. As per the International Committee on Taxonomy of Viruses (ICTV), nine antigenically distinct rotavirus species – RVA, RVB, RVC, RVD, RVF, RVG, RVH, RVI, and RVJ – are classified. Of these, only RVA, RVB, RVC and RVH have been reported from swine. RVE, which was also reported to occur in swine, was removed from the list of species in 2019 due to lack of more isolates and sequence data. RVA is most common in weaned piglets, and RVC in nursing piglets that are 1–10 days old. These two species are also the most common species affecting pigs. RVs are common agents of piglet diarrhoea in India and have been detected from several states.

Transmission

RVs are easily transmitted by direct contact. Transmission occurs through the faeco-oral route. Healthy carrier sows can shed rotavirus in their faeces during the periparturient period, potentially infecting their litters. Newly weaned piglets are often exposed to RVs that linger in nursery facilities and in other carrier pigs during mingling.

Clinical signs

Rotaviral diarrhoea can manifest in suckling pigs soon after birth, leading to a clinical condition known as milk scours, white scours or pre-wean scours. The clinical manifestation of the disease occurs when neonatal pigs fail to receive protective levels of maternal antibody. The clinical symptoms

vary depending upon the age of the affected animal. Sows may show short-term diarrhoea, though many may not show any sign of the disease. In piglets, profuse watery diarrhoea with dehydration and wasting is common. The diarrhoea may persist for up to 72–96 hours. The severity of the disease may increase if the condition is complicated by other pathogens such as pathogenic *E. coli* strains.

Lesions

Pigs that die of RV infection show staining of the perineal area with faeces and are extremely dehydrated. The small intestines appear more transparent and have thinner walls. The presence of watery stool and fluid and debris may be found in the intestines, most commonly in the caecum and colon.

Diagnosis

History, clinical manifestations and lesions are indications of RV infection but are not confirmatory diagnosis. PCR to detect the presence of RVs in faecal samples is the most commonly used test. Electron microscopy aided direct examination of the faeces or affected epithelial cells to demonstrate typical "cart-wheel" appearance of rotaviral particle may be helpful. To detect RVs in epithelial cells, FAT and IHC can also be used. ELISA can also be used for the detection of RV antigen in faecal samples or intestinal fluid. Postmortem samples may be subjected to histopathology to see villous atrophy in the intestinal segments.

Differential diagnosis

RV infection in pigs should be differentiated from other causes of diarrhoea such as viruses (porcine epidemic diarrhoea virus, transmissible gastroenteritis virus, porcine deltacoronavirus), bacteria (*Salmonella* spp., *Escherichia coli*, *Clostridium* spp., to name a few), and parasites (*Isospora suis*, *Cryptosporidium* spp.).

Treatment and control

Treatment

There is no specific treatment for rotaviral enteritis in piglets. Symptomatic treatment should be given to address diarrhoea and manage secondary infections. Animals should have free access to water. Fluid and electrolytes should be provided immediately for rehydration irrespective of the degree of dehydration. Rehydration solution like Ringer's lactate (sodium lactate solution) can be used at 4–10 ml per piglet and may be repeated if required. Affected weaned pigs should be provided a warm, dry, draft-free environment and receive frequent, small feedings which will help to prevent starvation, secondary diseases and long-term stunting.

Control

Currently, no commercial vaccine is available in India and control depends upon the implementation of strict biosecurity measures and hygiene management practices.

Biosecurity measures

Mingling of pigs from different sources must be avoided. "All-in-all-out" system of management should be practised. Floors, surfaces and equipment should be thoroughly cleaned and disinfected to reduce viral load. Phenol-based disinfectants are shown to be effective against RVs. When an outbreak occurs, affected animals must be segregated from healthy animals.

6.2.1.8 Swine influenza

Definition and causative agent

The swine influenza virus (SIV) is a highly contagious upper respiratory viral infection of swine. Although not all affected pigs demonstrate clinical signs of infection, the disease spreads within pig units at a quick pace, which is followed by rapid recovery of the animals. The causative agent of this infection is the influenza A viruses, that are again divided into various subtypes. The common subtypes responsible for causing infection in pigs are the H1N1, H1N2 and H3N2. Mortality due to SIV in pigs is usually low, although the morbidity may reach up to 100 percent. It can be considered an economically significant disease as it slows the weight gain in pigs, thereby increasing the number of days required to reach market weight. SIV is of public health importance since it affects humans as well. SIV has also been detected in swine population in India.

Transmission

Pigs infected with SIV shed the virus from as early as day one to nearly 10 days of infection. It is mostly found in pigs, but humans and birds such as turkey and ducks are also affected. As such, chances of production of novel subtypes and spread of SIV are more in settings where pigs and birds are kept

in close proximity to each other. The virus spreads within the pig population mainly through the aerosol route, direct contact with infected pigs and through fomites. In the case of outbreaks, initially one or two pigs are affected and then the disease spreads rapidly to the entire herd.

Clinical signs

An outbreak of SIV in pigs is characterized by sudden onset and rapid spread throughout the herd. Clinical signs of SIV appear within one to three days, and a majority of the animals recover within three to seven days unless there is a secondary infection or other complications. As it affects the upper respiratory tract of pigs, the infection manifests as fever, anorexia, lethargy, laboured breathing and weight loss. Respiratory signs like sneezing, coughing and nasal discharge are commonly observed. Conjunctivitis and abortions may also occur.

Lesions

Lesions are usually limited to the thoracic cavity unless there are further complications. Upon examination, the lungs of affected pigs appear purplish red and collapsed. Patchy pneumonia may be distributed throughout the lungs and unaffected areas appear pale and emphysematous. Severe pulmonary oedema, oedema of the bronchial and mediastinal lymph nodes, serous or sero-fibrinous pleuritis may also be observed. The airways may be filled with copious mucopurulent exudate.

Diagnosis

History and clinical signs are indicative of SIV infection. For laboratory diagnosis, deep nasal swabs at the earliest after the development of clinical signs or lung tissue at postmortem examination may be collected, and these may be subjected to RT-PCR for diagnosis and also for virus sub-typing. Virus isolation on continuous cell lines such as Madin-Darby canine kidney or embryonated eggs may be attempted. For subtyping of the virus, RT-PCR or haemagglutination inhibition (HI) and the neuraminidase inhibition tests are the tests of choice. IHC and FAT tests are used for detection of SIV antigen in tissues. For serology, HI test in paired serum samples is usually done, where a four-fold or higher rise in antibody titre reflects positivity. VNT, ELISA, IFAT, and agar gel immunodiffusion test (AGID) may also be deployed to check for SIV antibodies. Both conventional RT-PCR and real-time RT-PCR tests are the most frequently used tests for detection of SIV in swab and tissue samples.

Differential diagnosis

SIV in pigs should be differentiated from other causes of respiratory disease, the most common of which are viruses (PRRS, PCV2 infection, pseudorabies), bacteria (Pasteurellosis, Bordetellosis, Actinobacillosis) and *Mycoplasma hyopneumoniae* infection.

Treatment and control

Treatment

There is no specific and effective treatment for SIV. Antipyretics should be given in case of fever. Antimicrobials are to be given to treat secondary bacterial infections. Adequate water should be provided to prevent dehydration.

Control

Currently, no commercial vaccine is available in India. As such, control of this disease relies on early detection and good biosecurity measures.

Biosecurity measures

Measures such as "All-in-all-out" management system, combined with good hygiene practices, must be strictly followed. Once the entire herd is affected with SIV, it is difficult to eliminate the virus from the farm without complete depopulation. Segregation of early weaned piglets and partial depopulation may be adopted to minimize the economic impact in a farm. Stressors, such as crowding and dusting, should be avoided to minimize transmission and associated losses. Since SIV is a zoonotic infection, exposure of pigs to humans with influenza-like illness or related symptoms must be avoided.

6.2.1.9. Japanese encephalitis

Definition and causative agent

Japanese encephalitis (JE) is a vector-borne zoonotic viral disease which can cause reproductive failure in pigs. Mostly, subclinical infection is observed but it may be presented with abortions and stillbirths among pigs. The causative agent is the Japanese encephalitis virus (JEV), an enveloped RNA virus belonging to the genus *Orthoflavivirus* under the Flaviviridae family. Although five genotypes of JEV have been described till date, there is only a single

serotype. Japanese encephalitis has been reported from pigs in India.

Transmission

Pigs and water wading birds belonging to the Ardeidae family act as amplifying hosts and play a crucial role in the biological transmission cycle of JEV. *Culex tritaeniorhynchus* mosquitoes feed on the blood of these viraemic animals and cause its zoonotic spillover. In pigs, JEV persists in the tonsils and direct transmission has been documented experimentally. Since JEV is shed in the semen of affected boars, transmission through semen is also possible.

Clinical signs

Mummified (desiccated, dead foetus), stillbirth and weak piglets with or without neurological ailments are major clinical signs of JE infection. Piglets that are infected with the virus after birth may have inflammation of the brain in the initial six months of life. In other cases, wasting, hind limb paralysis and depression is observed among suckling piglets. Adult sows do not exhibit clinical symptoms. Infertility is seen among boars, along with swollen and congested testicles.

Lesions

Macroscopic lesions like subcutaneous oedema (fluid accumulation in subcutaneous tissue), ascites, haemorrhages, hydrocephalus (abnormal CSF accumulation inside brain cavities), congested lymph node and necrotic foci in the spleen and liver are seen among stillborn and infected piglets. Other lesions include spinal hypomyelinogenesis, congestion in meninges and hypoplasia of cerebellum.

Diagnosis

Virus isolation and identification is done for definitive diagnosis of JEV infection. The virus can be isolated from brain extracts and inoculated into cell culture or suckling mice and identified by neutralization test. Plaque reduction neutralization test (PRNT) is considered the gold standard test as it shows minimal cross reactivity with other flaviviruses. In cell culture, cell lines such as Vero, BHK-21, C6/36 mosquito cell line, and primary cell cultures of chicken embryo may be used for virus isolation. Molecular assays like quantitative RT-PCR and Restriction Fragment Length Polymorphism (RFLP) are highly sensitive tests that can be used for JEV diagnosis. Alternatively, FAT can be done to detect viral antigen in tissues extracted from stillborn piglets or infected foetuses. Compliment fixation test, IgM capture ELISA and haemagglutination inhibition assay are other tests used for JEV serodiagnosis. These serological tests should be interpreted carefully as maternal antibodies might be present in piglets for up to eight weeks and there are chances of cross-reactivity with other flaviviruses.

Differential diagnosis

JE in pigs should be differentiated from other diseases with similar presentation, the most common ones being PRRS, CSF, porcine parvovirus infection, pseudorabies, coronavirus infection and porcine brucellosis. Among the non-infectious causes, water deprivation or excess salt is a condition which needs to be differentially diagnosed from JEV.

Treatment and control

Treatment

There is no specific treatment for JEV. Treatment should be provided based on symptoms. Special attention has to be paid to nutrition and fluid therapy. Supplemental therapeutic doses of vitamin D3 and multivitamin and other micronutrients (iron, zinc, copper, chromium) at recommended dose for pigs must be given. Antibiotics can be used as and when necessary to protect against secondary bacterial infection.

Control

Currently, there is no vaccine available in India for JE. Controlling the abundance of mosquitoes near pig farms is a crucial step in breaking the transmission cycle of this virus. Other measures like using insecticides and screening of barns can be useful.

Biosecurity measures

Efficient mosquito control methods that target all the life stages of mosquitoes and prevents their breeding near pig farms is important. Regular spraying of approved insecticides on the outside of sheds and addition of larvicides to water bodies located near sheds are important steps in this regard. Monitoring and surveillance on a regular basis are important for keeping the mosquito population under check near pig-farms. Before semen collection from boars, quarantine protocols should be followed, and boar

should be checked for clinical signs of JEV infection before collection. JEV can be inactivated by common disinfectants such as commonly available detergents, phenolic compounds and 1 percent sodium hypochlorite.

6.2.1.10. Aujeszky's diseases

Definition and causative agent

Aujeszky's disease - more commonly known as pseudorabies - is an acute, contagious viral disease primary affecting pigs and seen in other domestic and wild animal as well. It is often fatal. It is rarely seen in equines and humans. The disease is prevalent across the world.

Aujeszky's disease is caused by neurotrophic α-herpesvirus. It is an enveloped DNA virus and pig is the only reservoir host. Although a stain difference is identified but a single recognized serotype is only recorded.

Transmission

The Aujeszky's disease virus is transmitted through direct and indirect contact. Direct contact includes nose-to-nose contact, oral or faecal contact. Indirect contact is through aerosol, fomite, infected carcasses and sometime sexual transmission through boars to sows to piglets. The virus can survive for four days in bedding material, three days in nasal washing, plastic material and feed, and up to seven hours in well water. In certain weather conditions, the virus may travel up to 2 km via aerosol. Since the virus is an enveloped one, it can be destroyed by temperature higher than 37°C. Other species of animals can be infected through direct contact with infected pigs or by eating infected pork.

Clinical signs

The clinical signs of Aujeszky's disease are age dependent. Young piglets - less than seven days of age, which are highly susceptible - show tremors, seizures, ataxia, circling and paddling and then perish. The mortality rate is 100 percent in piglets of less than one month of age.

In weaning piglets, respiratory symptoms like sneezing and dyspnoea are the dominant clinical signs. If secondary bacterial infection occurs, then the disease becomes complicated. In grown pregnant pigs; abortion, stillbirth, absorption of litter or birth of mummified foetus are common, with general symptoms like high fever (41-42ɑ), anorexia, weight loss being seen. With age, the mortality rate declines – from 50 percent in month-old piglets to 10 percent in piglets that are six months of age and 1–2 percent in adult pigs.

In all other non-porcine species, intense pruritis (itching), CNS (jaw and pharyngeal paralysis) and respiratory signs along with high fever is recorded. Any secondary host lives for only one or two days after symptoms are seen.

Lesions

No gross lesion appears in Aujeszky's disease. Severe rhinitis and necrotic tonsillitis, along with haemorrhagic pulmonary lymph nodes, are found. Animals in which respiratory symptoms are predominant are found with pulmonary oedema. In young piglets, multiple necrotic foci of 2–3 mm in diameter can be found scattered throughout the liver.

When examined microscopically, meninges are found to be thickened, and mononuclear perivascular cuffing and neuronal necrosis may be seen. Nonsuppurative meningoencephalitis is present in both white and grey matter. Necrotic tonsilitis, bronchitis, bronchiolitis as well as alveolitis are often seen. In macerated foetus, the liver, spleen, lymph nodes and adrenal glands are frequently found with focal necrosis.

Diagnosis

The diagnosis can be done by clinical findings, identifying gross and microscopic lesions. Additionally, diagnosis can also be made by virus isolation, PCR and ELISA. The false positive result is typically reassessed by using the serum neutralizing test or latex agglutination test. ELISAs can be used to differentiate between an immune response due to natural infection or due to vaccination.

Differential diagnosis

Differential diagnosis of Aujeszky's disease has to be made with classical swine fever, African swine fever, teschovirus, *streptococcus suis*, influenza, PRRS, PCV2, parvovirus and salt toxicity.

Treatment and control

Treatment

There is no specific treatment. Symptomatic and supportive line of treatment protocol should be followed. Antibiotics should be administered to

control any secondary infection.

Control

Vaccine for Aujeszky's disease is not available in India and strict biosecurity measures should be implemented to control the disease.

Biosecurity measures

Standard biosecurity measures like restricted entry into farms, cleanliness and sanitization of farm premises and implements, provision of clean water and feed, proper vaccination and deworming, isolation and quarantine as needed should be strictly followed.

6.2.1.11. Swinepox

Definition and causative agent

Swinepox is an acute infectious disease of pigs, characterized by eruptions on the skin. All age groups of pigs are affected, but the incidence is higher in young, growing pigs. Occasional infections are also seen as congenital cases and in neonates. It is caused by the swinepox virus (SwPV), the only member of the genus *Suipoxvirus* in the family Poxviridae. SwPV is a large DNA virus which is enveloped. The mortality rate due to this virus is generally very low (<5 percent), but the morbidity rates can be very high in pigs up to the age of four months. Many outbreaks of poxviruses in pigs in the past were attributed to vaccinia virus, which is a zoonotic poxvirus. However, SwPV has now emerged as the main cause of poxvirus infection in pigs. The disease is present in a majority of pig rearing countries. Outbreaks of SwPV have also been reported from India.

Transmission

Transmission of SwPV in pigs occurs either through direct contact or through mechanical vectors. *Haematopinus suis* (pig lice) and *Musca domestica* have been identified as mechanical vectors. Occasional infection of neonates occurs when SwPV passes through the placenta in viraemic sows. Contaminated items such as clothes and equipment can also transmit the virus from one farm to the other.

Clinical signs

SwPV infection in adult pigs is generally self-limiting and the disease is usually seen in pigs up to four months of age. The incubation period of SwPV infection in pigs is usually up to seven days but can go up to 14 days in some cases. Small red spots often appear on the face, ears, inner legs and abdomen. These spots develop into papules and, within a few days, pustules or small vesicles may form. The centre of the pustules dry out and scab over, and are surrounded by a raised, inflamed zone, creating a characteristic umbilicated appearance. Eventually, dark scabs of 1–2 cm in diameter form, giving the affected piglets a spotted look. These scabs eventually fall off or are rubbed off, leaving no scars. Severe cases of SwPV are characterized by lesions in the upper respiratory and digestive tracts. Piglets that are affected congenitally may be born with lesions covering the entire body. The infection in pigs typically does not produce a pruritic response. Systemic signs of infections are rare, but sometimes mild pyrexia, inappetence and dullness might be observed.

Lesions

Skin lesions, typical of poxvirus infections, are observed. Lesions at various stage of development as described earlier may be seen. A report on SwPV infection in a wild boar has described the lesion as "cherry-sized" papules.

Diagnosis

SwPV infections are easily diagnosed by identifying the lesions. Histologically, identification of large, intracytoplasmic inclusion bodies in the lesions may be used for diagnosis. Electron microscopy-aided visualization also helps in identifying the agent. The virus can be cultured in a wide range of cells in the laboratory. IHC and immunofluorescence assays are used for the detection of SwPV antigens in infected cells. PCR-based assays can be used for detection of SwPV and differentiation from vaccinia virus that produces similar symptoms in pigs. Serological assays to detect antibody against SwPV are the VNT, AGID test and immunoelectrophoresis.

Differential diagnosis

SwPV infection in pigs should be differentiated from other agents producing similar lesions such as FMD, vaccinia virus, greasy pig disease, ringworm, streptococcal dermatitis, calicivirus infection, Pityriasis rosea, Dermatosis vegetans, swine vesicular disease and vesicular stomatitis.

Treatment and control

Treatment

There is no specific treatment for swinepox. Supportive treatment should be provided to the affected animals for faster recovery. Antiseptic or antibiotic ointments or lotions may be applied to control secondary bacterial infection and augment wound healing.

Control

Currently, there are no vaccines available for SwPV infection in pigs in India. Control of the disease is dependent on herd immunity and prevention of transmission. Pigs can be treated with ectoparasiticides, or insecticides may be used to control the lice and fly population which are responsible for disease transmission.

Biosecurity measures

It should be ensured that the animals are housed in closed premises or with screens to keep out flies. Manure should be removed regularly to prevent laying of eggs by flies. The premises should have adequate ventilation and fans so that the flies do not sit on the animals frequently. Cleaning and thorough disinfection of the pens are needed as SwPV may persist in the environment for long periods. SwPV is susceptible to commonly used disinfectants such as quaternary ammonium compounds, acids, alcohols, aldehydes, phenolic compounds and oxidizing agents.

6.2.1.12. Nipah virus encephalitis

Definition and causative agent

A highly contagious zoonotic disease characterized by a sudden onset of fever in pigs, the Nipah virus encephalitis causes a gamut of illness from asymptomatic mild infection to respiratory and neurological ailments, and fatal encephalitis. The causative agent is the Nipah virus, an enveloped RNA virus belonging to the genus *Henipavirus* of the Paramyxoviridae family. The disease has been reported in humans and bats from India but not from pigs. However, the Nipah virus has been detected in pigs in neighbouring Bangladesh as well as Malaysia and Singapore.

Transmission

The Nipah virus is present in the saliva, urine and faeces of the Pteropus bat, which is the reservoir host of this virus. It gets transmitted to pigs through the consumption of food and water contaminated with bat waste. Within pig farms, it gets transmitted by direct contact and through fomites. Inadequate biosecurity practices and transport of infected animals also lead to virus transmission.

Clinical signs

Nipah virus disease in pigs is also known as porcine respiratory and neurologic syndrome (PRNS), porcine respiratory and encephalitic syndrome (PRES) or barking pig syndrome. The clinical signs in pigs differ with the age of the infected animal and the specific response towards the virus. The majority of the infected pigs develop fever, with severe coughing and dyspnoea, because of which this disease is also called "barking pig syndrome". Some pigs, especially sows and boars, can develop encephalitis with signs like motor incoordination, tremors, twitching, muscle fasciculations, spasms and paraplegia. Morbidity is high among all the age groups, but mortality is minimal, except in suckling pigs. Abortion in the first trimester has also been reported in sows with Nipah virus infection.

Lesions

In this infection, the most notable damage occurs in the lungs which become firm with large areas of consolidation and a reddish colour. Lung lobes swell due to fluid build-up in the septal walls, partial blockage of the airways being caused sometimes by foamy fluids. Lymph nodes in the respiratory areas become enlarged and may bleed. Bleeding is also seen in the lungs and kidneys. Blood-tinged or clear frothy exudate may fill up the trachea and bronchi. Oedema of the meninges - accompanied by congestion of the cerebral blood vessels - may be evident in the brain.

Diagnosis

The methods that can be employed for diagnosis of the Nipah include virological, immunohistochemical, serological and molecular methods. For virus isolation lung, spleen, kidney or brain samples are used. After three days of infecting Vero cell monolayer with the virus, cytopathic effects (CPE) (plaques or syncytia) can be observed. Further, RT-PCR is performed using culture supernatant for virus identification. RT-PCR is considered as the gold standard for Nipah virus detection from different biological samples. Indirect ELISA and sandwich

ELISA have also been developed which can detect anti-NiV IgM and IgG antibodies from porcine serum samples. PRNT and VNT in microtitre plates is also commonly used for detection of Nipah virus antibodies. Immunohistochemical staining is another method in which anti-NiV antibodies are utilized for staining formalin-fixed tissues of organs like lymph nodes, heart, CNS, spleen and lung for detecting viral antigens. These antibodies allow for the identification of NiV-associated lesions such as necrosis, vasculitis in tissue sections.

Differential diagnosis

Identifying Nipah virus infection in pigs is challenging because there are no specific clinical signs. This makes diagnosing the disease difficult, especially when considering other potential illnesses like African swine fever, Aujeszky's disease, pasteurellosis, CSF, PRRS, enzootic pneumonia and pleuropneumonia.

Treatment and control

Treatment

There is no specific treatment. Supportive care should be given for treating the symptoms as they occur. Supportive treatment includes rehydration therapy and medicines to relieve pain.

Control

Currently, there are no vaccines developed for Nipah virus infection in India. It is important to maintain appropriate biosecurity measures and follow quarantine protocols within facilities to prevent the spread of infection. For people working in the livestock and agricultural fields, following good practices in pig farming is crucial and crops should be protected from contamination by bats.

Biosecurity measures

Stringent biosecurity measures are important for preventing spread of infection among domestic animals. The potential for contact between the bat reservoir and susceptible animals in virus affected areas must be minimized. In regions with confirmed cases, pigs must be kept away from fruit tree plantations, and they must not be fed any fruits that may have been contaminated by bats. In case of a suspected (or confirmed) outbreak, it is advisable for personnel to use personal protective equipment (PPE) including masks, gloves, protective goggles, gowns and boots. These PPE should be thoroughly washed and disinfected after use. All the animals testing positive must be culled immediately to curtail the spread of the disease to other animals or humans. The site where the infected animals are buried must be disinfected with chlorinated lime.

6.2.1.13. Swine vesicular disease and Seneca Valley virus disease

Definition and causative agent

Swine vesicular disease (SVD) and Seneca Valley virus disease (also called porcine idiopathic vesicular disease) are viral vesicular diseases of swine caused by picornaviruses. The causative agent of SVD is swine vesicular disease virus (SVDV), which belongs to the genus *Enterovirus*, whereas Seneca Valley virus disease is caused by senecavirus A1 or Seneca Valley virus (SVV), which belongs to the genus *Senecavirus*, both under the Picornaviridae family. Swine are the only natural host of SVDV and SVV, even though the viruses have been detected in another host. Antigenically, SVDV and human coxsackie virus B5 of human are similar. SVD has not been reported in India but has been from other Asian countries and is thought to be endemic there. SVV, however, has been reported from India.

Transmission

The route of transmission of SVV is yet to be understood. Since it belongs to the same family as FMDV, it is presumed that the spread is by direct contact with pigs suffering from Seneca Valley virus disease, through aerosols and by fomites. SVDV is spread in pigs through damaged skin or ulcerated mucosa, ingestion and inhalation routes. Transmission of SVDV takes place by direct contact with SVD-affected animals or their excretions. Transmission through contaminated faeces, often aided by vehicles, is also a major source of SVDV. Pigs affected with SVDV excrete the virus 48 hours before clinical signs appear. SVDV may be continued to be excreted in the faeces for up to three months.

Clinical signs

It is very difficult to distinguish SVD and Seneca Valley virus disease clinically. Cutaneous vesicular lesions may develop on the snout, lips, tongue, teat, coronary band and inter-digital space. In SVD, coronary band and inter-digital space are more common sites for the development of vesicular lesions than the snout and other places. Signs

accompanying these vesicular lesions include sudden onset of lameness, fever, anorexia and lethargy. Seneca Valley virus disease in neonatal pigs may result in neurological symptoms and diarrhoea. Neurological symptoms have also been reported in SVDV infection, but it is considered a rare event.

Lesions

Vesicular lesions are seen in both the disease as described above. Petechial haemorrhages on the kidney have also been reported in Seneca Valley virus disease. In addition, piglets that have diarrhoea when infected with Seneca Valley virus may show subcutaneous and mesenteric oedema on necropsy.

Diagnosis

Samples of choice in SVD include lesion materials in case of clinical disease, and faecal samples in case of sub-clinical disease. Serum samples need to be collected for antibody detection. Detection of virus antigen is done most commonly by RT-PCR and virus isolation in cell culture of porcine origin. Sandwich ELISA for antigen detection is less sensitive than RT-PCR and virus isolation, and so it can be used to test lesion material homogenate. ELISA and VNTs are used for detection of SVD antibodies. For Seneca Valley virus disease, tissue homogenate from neonatal pigs and other samples as those mentioned for SVD need to be collected. Conventional and quantitative RT-PCR is the gold standard for diagnosis. Isolation of SVV can be done in various cell line of porcine and human origin. Human retinoblast and human lung cancer cell lines are examples of cell line where SVV can be grown. Other methods for virus detection include *in situ* hybridization and IHC tests. ELISA and VNTs can be employed for detection of antibodies against SVV.

Differential diagnosis

Both the diseases need to be differentially diagnosed from each other and other infectious vesicular diseases of swine like FMD, vesicular exanthema, and vesicular stomatitis. Differential diagnosis also includes non-infectious causes like blisters due to mechanical causes and chemical or thermal burns.

Treatment and control

Treatment

There is no specific treatment for both the diseases. Antiseptics may be applied on ulcerated lesions to make them heal faster and fend off fly attacks. Broad-spectrum antibiotics may be given to prevent secondary bacterial infection. One percent potassium permanganate may be used to rinse ulcerated vesicle. After cleaning and application of antiseptics, the ulcers may be bandaged.

Control

Currently, there is no commercial vaccine available for both SVD and SVV disease in India. Prevention and control of both the disease relies on enforcement of strict biosecurity measures.

Biosecurity measures

India is non-endemic for both the diseases. Strict biosecurity practices need to be enforced. All the pigs brought from outside should be screened thoroughly before introduction to the main farm. Garbage feeding should be avoided or, if it is used, it must be cooked properly. In case of any suspected outbreak, sick animals should be isolated and any movement to and from the farm should be restricted till a definite diagnosis is done. In case of an outbreak, culling all the animals, whether infected or in contact, should be done. The farm premises, vehicles and equipment should be disinfected thoroughly. One percent sodium hydroxide, hydrogen peroxide, bleach, and iodophors may be used with detergents for disinfection.

6.2.1.14. Porcine epidemic diarrhoea

Definition and causative agent

Porcine epidemic diarrhoea (PED) is a viral disease of pigs caused by the PED virus (PEDV). It affects pigs of all age groups and is characterized by watery diarrhoea and weight loss. The PEDV is highly contagious, and an outbreak of the disease can have serious economic implications. Neonatal pigs are worse affected by the disease as the morbidity and mortality percentage can reach up to 100 percent in them. PEDV belongs to the genus *Alphacoronavirus* in the family Coronaviridae and has two main genotypes – G1 and G2. Each genotype has two sub-genotypes, *viz.*, classical G1a and recombinant G1b under the G1 genotype, and local epidemic G2a and global epidemic or pandemic G2b under the G2 genotype. No cases of the PED disease in pigs have been reported in India till now even though the antibodies to PEDV in pigs have been detected in Assam. The PEDV has been circulating in other Asian countries such as China, Japan, South Korea,

Vietnam and Taiwan.

Transmission

The faeco-oral route via any contact with infected pigs or diarrhoeal faeces/vomitus has been identified as a prime mode of transmission of PEDV. Although infectious PEDV was shown to be shed in the faeces for the initial two weeks post-infection diarrhoea (PID), viral RNA has been shown to be present up to 49 PID. Indirect transmission via contaminated fomites such as contaminated vehicles, equipment, feed, and feed ingredients and additives are also possible. Additionally, PEDV transmission through the use of contaminated semen and vertical transmission through milk are reported.

Clinical signs

PED infection cannot be distinguished from other diarrhoeal causes and varies in severity based on the age, past exposures, and immunological state of the pigs. Severe disease is seen in nursing piglets under the age of two weeks. The main clinical signs include flocculent and fetid diarrhoea, and there may be occurrence of vomiting. Secondary clinical signs may include dehydration and metabolic acidosis.

Lesions

Necropsy findings in affected pigs may include coating of skin and hair with yellow faeces, distension of the intestine with watery contents, thinning of the intestines (particularly small intestines), occurrence of undigested milk in the stomach and necrosis of the back muscles in fatteners.

Diagnosis

Faecal samples, small intestine and oral fluids can be used for virus detection and serum to detect antibodies. For virus detection, tests such as RT-PCR, sandwich ELISA, and IHC are commonly employed. Virus isolation may also be attempted, although it is difficult. For antibody detection, virus neutralization, ELISA, IHC and immunofluorescence tests are used.

Differential diagnosis

Diarrhoea caused by PEDV should be differentiated from diarrhoea due to other agents such as viruses (transmissible gastroenteritis, rotavirus), bacteria (*Escherichia coli*, *Salmonella* spp., *Clostridium* spp., to name a few), and parasites (*Cryptosporidium* spp. and *Isospora suis*, among others).

Treatment and control

Treatment

There is no specific treatment for PED. Symptomatic treatment should be given for diarrhoea and to control secondary infections. Affected animals should be kept in a warm and dry state. They should be provided with free access to water and electrolyte solutions for managing dehydration.

Control

Vaccines for PED are currently not available in India as the disease is not present. Therefore, control of the disease in case of any future outbreak will depend on early detection and prompt biosecurity measures.

Biosecurity measures

Implementation of stringent biosecurity practices is the single most effective measure to control and prevent PED. Pigs should be introduced in farms from reliable sources, where the health status of animals is known. Pigs brought from different sources should not be mixed. Animals brought from outside have to be quarantined, ideally for 28 days but at least for 14 days in case of any exigency. Proper sanitation and disinfection practices of equipment, vehicles and the barn as a whole must be adopted. PEDV is susceptible to commonly available disinfectants such as formalin, sodium carbonate, accelerated hydrogen peroxide and glutaraldehyde/ quaternary ammonium. Commercially available disinfectants such as 0.5 percent Virkon S and 2.06 percent Clorox have been shown to be very effective for inactivation of PEDV in faeces. In case of an outbreak, suspected animals must be segregated immediately from healthy pigs. Equipment, dead pigs and slurry must be disposed of appropriately. The "All-in-all-out" practice is an effective method to break the transmission cycle in a farm.

6.2.2. Bacterial diseases

6.2.2.1. Porcine brucellosis

Definition and causative agent

Porcine brucellosis is a zoonotic disease caused primarily by *Brucella suis*, although *B. abortus* and *B. melitensis* may occasionally be involved. The disease is characterized by infertility, abortion, orchitis and bone and joint lesions. There are five biovars of *B. suis*, of which biovars 1, 2 and 3 are responsible for porcine brucellosis worldwide. *Brucella suis*

biovars 1 and 3 are distributed worldwide. Biovar 1 is present in the Americas and Asia, while biovar 3 has been reported in China, the United States and the Europe Union.

Transmission

Transmission usually takes place through direct contact with infected pigs. The organism gains entry through oral, nasopharyngeal, conjunctival and vaginal mucosa. Pigs may get infected if they ingest feed or water contaminated with aborted foetuses, placenta, foetal membranes, foetal fluids or vaginal discharges from an infected sow. Transmission can also take place via infected semen during natural breeding and artificial insemination. Milk or urine of infected animals may also be a source of infection.

Clinical signs

The major clinical signs of the disease include abortions, stillbirth, increased neonatal mortality, infertility and decreased litter size. Abortion can occur at any stage of gestation but most commonly in the mid to late gestation periods. Metritis can be seen in some sows. In boars, signs like reluctance to mate and abnormalities in the semen may be observed and there may be abscess formation and swelling of the testes in some cases. Boars can excrete the organism asymptomatically in the semen, and sterility may be the only sign of infection. Signs like swollen joints, lameness, incoordination and posterior paralysis may be observed. Usually, most infected pigs do not exhibit clinical illness on visual examination.

Lesions

Abscesses and other purulent or inflammatory lesions may be observed in the testes as well as in the joints.

Diagnosis

Isolation of bacteria is the gold standard for diagnosis of porcine brucellosis. But isolation of *Brucella* poses some difficulties such as slow growth of the organism, low rate of recovery of isolates from chronically infected swine and biosafety issues associated with working with *B. suis*. As a result, serologic testing has become the standard for diagnosis of porcine brucellosis. Serological tests like Rose Bengal Plate Test (RBPT), Serum Agglutination Test (SAT), complement fixation test (CFT) and c-ELISA can be used for diagnosis of porcine brucellosis. Of these tests, the most commonly used serological tests are RBPT and SAT. The organism can also be detected through molecular techniques such as the PCR.

Differential diagnosis

Porcine brucellosis should be differentiated from other porcine diseases such as Aujeszky's disease, leptospirosis, erysipelas, salmonellosis, CSF and porcine parvovirus infection.

Treatment and control

Treatment

Antibiotics might reduce the clinical signs, but the organism may persist in treated animals and there is possibility of resurgence. Antibiotics like chlortetracycline, streptomycin, oxytetracycline, singly or in combination with dihydrostreptomycin or gentamicin, can be used. As treatment of brucellosis is not very cost-effective, the infected animal should preferably be culled and disposed of properly.

Control

Currently, there is no vaccine for porcine brucellosis in India. In a herd, *Brucella suis* is generally introduced by an infected animal. Therefore, strict biosecurity measures must be adopted so that *B. suis*-free herds do not come in contact with positive herds, including feral pigs or contaminated environments. Introduction of new animals to a herd should always be made from *Brucella*-free herds only. In addition, semen for artificial insemination should be collected from *Brucella*-negative animals.

Biosecurity measures

Herd biosecurity is critical for the prevention of brucellosis. Any new additions to the farm should be made only from Brucella-negative herds and they should be kept in an isolation shed for 30 days and have their blood tested prior to introduction. Entry of visitors to the farm should be restricted. Exposure of domestic pigs to feral pigs should be avoided.

6.2.2.2. Leptospirosis

Definition and causative agent

Leptospirosis is a contagious disease of pigs caused by several serovars of *Leptospira*. The disease also occurs in other animals as well as human beings. There are eight pathogenic species of *Leptospira,*

of which three are important for swine: *Leptospira interrogans* (serovars *pomona, icterohaemorrhagiae, canicola* and *bratislava*); Leptospira *borgpetersenii* (serovars *sejroe* and *tarassovi*); and *Leptospira* kirschneri (serovar grippotyphosa). Serovars *pomona* and *bratislava* are uniquely adapted to swine whereas the others are maintained in other species but can infect swine sometimes.

Transmission

Infection is usually introduced in a herd through infected pigs (commonly through replacement gilts or boars). Infection can also occur by direct or indirect contact with incidental hosts (rats, mice, foxes, among others) or through water, soil or effluent contaminated with the organism. Carrier animals used as replacement stock for breeding can introduce the infection into recipient herds. Infection can also take place during coitus.

Clinical signs

In pigs, the majority of the infections are subclinical in nature and clinical infection mostly occurs in piglets and pregnant sows.

Acute leptospirosis: In the acute phase of the disease, the signs may not be evident in mature, non-pregnant or growing pigs. Upon careful observation, mild fever and inappetence for a few days may be noticed. Signs like fever, anorexia, haemolytic anaemia, haemoglobinuria, icterus, convulsions and a failure to grow and gain weight may be observed in very young pigs.

Chronic leptospirosis: Clinical signs such as abortions, stillbirths, decreased litter size and birth of weak piglets may be observed when sows are infected with *L. pomona*. Pregnant animals often abort in the late gestation period. In a majority of the cases, foetuses are carried almost full-term but may be mummified, dead or weak at birth. Generally, most of the infected neonatal pigs die within a few days, although some survive. The dams usually recover promptly. The recovered animals often conceive again and carry their litters to term.

Lesions

As most infected adults recover, lesions are hardly evident in sows. Petechial haemorrhages are common, and organs are often discoloured due to the degree of jaundice. White-spotted kidneys may be observed at slaughter. Placental lesions include oedema and scattered haemorrhages.

Diagnosis

Diagnosis of porcine leptospirosis poses a challenge. Diagnostic tests such as serology, FAT, histopathology and culture are commonly used for diagnosis of the disease. Serovars such as *pomona*, *grippotyphosa* or *icterohaemorrhagiae* can be readily diagnosed using serology and FAT. Because of the poor serologic response of the infected pigs, diagnosis of serovar *bratislava* infection is more difficult. Definitive diagnosis of *L. interrogans* can be made by isolating the organism from tissues. The culture of Leptospira is a bit difficult, time consuming and requires specialized culture medium and technical expertise. Microscopic Agglutination Test (MAT) is commonly used for the diagnosis of porcine leptospirosis. The presence of Leptospira in tissues or body fluids can be detected by FAT. Their presence in fresh or frozen tissues can also be confirmed by FAT. Histopathology can be used to detect the organism in porcine tissues. PCR may also be used for the diagnosis of swine leptospirosis.

Differential diagnosis

Porcine leptospirosis should be mainly differentiated from PRRS. Other infectious causes of pregnancy losses in pigs include brucellosis, PCV and PPV.

Treatment and control

Treatment

Cases of porcine leptospirosis should be treated with dihydrostreptomycin G @ 25 mg/kg IM for three to five days as early as possible after signs appear. Although the efficacy of the treatment may vary depending on the serovar involved, other antibiotics such as oxytetracycline @ 40 mg/kg IM daily for three or five days, tylosin @ 44 mg/kg IM daily for five days and erythromycin @25 mg/kg IM daily for five days can also be used.

Control

Because of the involvement of several species of animals, control of porcine leptospirosis is generally difficult. However, it will be possible to control the disease in pig populations when the animals are managed under the confinement system, coupled with a combination of medication, rodent control and the provision of contamination-free drinking water.

Biosecurity measures

Biosecurity measures such as avoidance of introduction of infected animals into the herd, implementation of strict isolation and quarantine of new animals until proven negative, and restricting access of livestock to outside sources of infection with provisions such as double fencing of perimeters as well as restricting access to open waterways may be adopted.

6.2.2.3 Listeriosis

Definition and causative agent

Listeriosis is a sporadic bacterial infection that affects several species of animals, including pigs. The disease has also been reported in pigs from India. It is caused by a bacterium, *Listeria monocytogenes*. *L. monocytogenes* is a Gram-positive, non-spore forming, non-capsulated, pleomorphic, facultative anaerobic, rod-shaped coccobacillus with an ability to switch from an environmental saprophyte to a potentially fatal pathogen.

Transmission

The natural reservoirs of *L. monocytogenes* is soil and mammalian gastrointestinal tracts, both of which contaminate vegetation. Animal-to-animal transmission occurs via the faecal-oral route. Feed is considered as the main source of contamination with *Listeria*, but an environmental source is also possible due to the soil origin of *Listeria* spp. *L. monocytogenes* is generally present in animal products such as processed meat, fish and dairy products. The organism is also found in vegetable products. Many outbreaks of listeriosis were found to be associated with consumption of listeria-contaminated pork.

Clinical signs

Monogastric animals, including pigs, show signs of septicaemia (infection in the blood and several organs). Abortion, stillbirths and neonatal death occur in all species. Although the septicaemic form is the most common form in pigs, a few reports of encephalitic form have also been reported. Sudden death, high fever, septicaemia, nervous symptoms (possible meningitis), head tilting and ear infections have also been recorded in piglets.

Lesions

The most frequent gross lesions observed is congestion of meninges. Necrotic foci are generally found in most of the organs (mostly in the liver) in septicaemic form. Focal hepatic necrosis is invariably observed in listeriosis of neonatal pigs. High-grade alveolar pulmonary oedema and severe diffuse fibrinonecrotic typhlocolitis with large amounts of intralesional bacteria have also been reported.

Diagnosis

Diagnosis is usually based on history, clinical signs, pathological lesions and detection of the pathogen. Confirmatory diagnosis is made by isolation of the organism. Sero-diagnostic tests such as SAT, CFT, haemagglutination (HA), HI, antibody precipitation, growth inhibition test and ELISA can also be used. Molecular tools such as PCR, multiplex PCR and real-time PCR are also used for rapid, specific, and reliable detection of the organism.

Differential diagnosis

Listeriosis in pigs should be differentiated from all diseases of pigs associated with septicaemia, reproductive problems and diseases associated with nervous signs.

Treatment and control

Treatment

Penicillin is the drug of choice for treating listeriosis. Procaine penicillin G @ 40 000 international units (IU) per kg BW IM for five days should be given. Other antibiotics such as ceftiofur @ 3 mg per kg BW for three days can also be used. Supportive therapy, including providing fluids and electrolytes, is recommended to correct dehydration.

Control

Proper disposal of contaminated materials, beddings, litters and infected carcasses by incineration or burning should be done. Pigs should not be fed rotten vegetables and proper hygiene, and sanitation should be maintained at the farms. Protective clothes should be worn when infected animals and aborted material are being handled or while removing retained placenta.

Biosecurity measures

Biosecurity plays a crucial role in the control of listeriosis, as the organism is transmitted to animals through direct (animal-to-animal/animal-to-workers) or indirect contact. Indirectly the organism may be carried by farm workers. The organism may

also be present in contaminated objects.

6.2.2.4. Oedema disease

Definition and causative agent

Oedema disease mainly affects recently weaned pigs. This disease occurs commonly in countries where pigs are reared under intensive system of management. The disease has also been reported in pigs from India. It is caused by toxins produced by pathogenic serogroups of *E. coli*. Most have pilus type F18 or F4 (K88) and elaborate Shiga-like toxin.

Transmission

Pathogenic *E. coli* that causes oedema disease generally persists in the environment of the pig shed/ farrowing rooms and may have been introduced by carrier dams. The presence of antibodies from the mother in colostrum and milk may also be a factor. Oedema disease occurrence can also be influenced by type of rations (especially high protein diets) and frequency of feeding.

Clinical signs

The disease generally occurs after weaning and it affects the healthiest piglets of the litter. Clinical signs include loss of appetite, ataxia and recumbency, which is often accompanied by paddling and running movements. Lateral recumbency with paddling movements leading to coma and death, normally follow within 4–36 hours or the onset of clinical signs. Swelling of the face and eyelids may be present in some cases.

Lesions

The only significant gross changes that can be seen in dead pigs is the congestion of the viscera. In some cases, facial oedema and oedema of the eyelids may be present. Increased fluid may be present in the peritoneal cavity, chest or pericardial sac. Occasionally haemorrhages are found under the epicardium or endocardium. The brain may have unique haemorrhagic and malacia lesions in the brain stem, or basal ganglia in less acutely affected pigs. These lesions are very suggestive of oedema disease.

Diagnosis

Tentative diagnosis can be made based on the history, clinical signs and lesions. Bacterial culture of the small intestine from an untreated diseased pig will yield high populations of haemolytic colonies of *E. coli*. Genotyping of the organism will help in confirming the virulence factors. Typical microscopic vascular lesions help in confirming the diagnosis.

Differential diagnosis

Oedema disease must be differentiated from other causes of neurologic signs (water deprivation or organic arsenical poisoning) or septicaemia and meningoencephalitis caused by *Streptococcus suis*, *Salmonella* serotype *choleraesuis*, erysipelas, or *Haemophilus parasuis*

Treatment and control

Treatment

Antimicrobials and acidifiers can be given in water (acidifiers can be used at 2–8 kg per tonne of feed). Antimicrobials can also be used parenterally, particularly in less severe cases. The choice of antibiotic depends on sensitivity test on the isolate from affected pigs.

Withdrawal of feed along with the provision of zinc oxide (with legal limit of 150 ppm of total zinc in complete feed) via drinking water is recommended. Along with antibiotics, supportive therapy for correction of acidosis and dehydration is essential.

During an outbreak, efforts should be made to reduce the incidence of infection in the remaining animals at risk. High-fibre and low-protein diet is recommended in severe outbreaks.

Control

Environmental stresses such as temperature variation and dampness should be minimized. Provision should be made for creep feeding, restricted feeding, multiple feedings (small quantities three to six times a day) after weaning and the rations should contain high fibre foods. Antimicrobial intervention (antibiotics in rations or water) should be done.

Biosecurity measures

General biosecurity measures for the control of infectious diseases such as routine hygienic measures including cleaning, disinfection and restrictions on entry of visitors need to be followed judiciously.

6.2.2.5. Enteric colibacillosis

Definition and causative agent

Enteric colibacillosis is a common disease of nursing and weaned piglets caused by the colonization of

enterotoxigenic strains of *Escherichia coli* (ETEC) in the small intestine. The ETEC are gram-negative bacilli with flagella. Pathogenic strains produce smooth or mucoid colonies. It produces several virulence factors that are responsible for disease production like fimbriae, capsules, enterotoxins, and endotoxins. ETEC make use of specialized fimbria for adherence to the absorptive surface of the intestine, thereby facilitating colonization. Then, through the actions of toxins, localized or systemic effects are seen in affected pigs. Five common fimbriae, which are antigenically distinct from one another, are found in pigs. They are F4 (K88), F5 (K99), F6 (987P), F41 (associated with neonatal colibacillosis) and F18 (associated with post-weaning colibacillosis). F4 fimbriae are also implicated in post-weaning colibacilloses. It is one of the most commonly prevalent bacterial infections in swine population worldwide, including India.

Transmission

The primary route for ETEC transmission in pigs is faeco-oral but aerosols and contaminated environment are also a source of infection for naïve piglets. If the sanitation on the farm is poor, then the organism builds up in the environment. Nursing piglets often ingest the organisms from the skin and mammary glands of sows that are contaminated with *E. coli*.

Clinical signs

Colibacillosis in pigs is usually manifested by the appearance of diarrhoea, which varies in severity. It is accompanied by dehydration, acidosis and, sometimes, death. Hypersecretory diarrhoea typically has an alkaline pH but can vary in colour. It may be clear and watery, particularly in neonates, or it can appear white or yellow, depending on the type of ingesta and the duration of the illness. Occasionally, affected pigs may also vomit. Body temperature is invariably subnormal, and it is accompanied by shivering.

Lesions

Dehydration-related changes are the most common sign. In the small intestine and colon, excess fluid or gas may be present resulting in their distention. Mild congestion of the stomach may also be seen.

Diagnosis

Rectal swabs, faecal samples or intestinal tissues are the specimens of choice. The bacteria should be isolated and cultured from theses specimens. Smooth, mucoid or haemolytic colonies are suggestive of *E. coli*. Histologically, shortening of the villi with the presence of small coccobacilli on the absorptive surface are also suggestive of colibacillosis. To confirm the serogroup, a slide agglutination test can be used. PCR-based assays are used to identify the nucleic acid of the pathogen. Multiplex PCR is used to detect specific fimbriae and toxin genes.

Differential diagnosis

Colibacillosis in pigs must be differentiated from other diarrhoeal diseases such as rotavirus infection, coronavirus infection, porcine epidemic diarrhoea, salmonellosis, clostridium infection, transmissible gastroenteritis (TGE), coccidiosis and Strongyloides infestation.

Treatment and control

Treatment

Aminoglycosides are the choice of antibiotic for treatment of colibacillosis and should be selected based on results of minimum inhibitory concentration (MIC) testing. It should be used only on a case-to-case basis and not over a long-term. Restoration of fluids with electrolytes is crucial to prevent dehydration and electrolyte balance are a good choice.

Precaution: Aminoglycosides are the choice of antibiotic for treatment of colibacillosis and should be selected based on results of minimum inhibitory concentration (MIC) testing. Drug should be used only on a case-to-case basis and not over a long-term.

Control

Currently, vaccines for colibacillosis are not available for pigs in India. The disease is controlled mainly by good housing and nutritional interventions. Sudden feed changes at weaning should be avoided. Rice-based diets are highly digestible and reduce the *E. coli* colonization in the small intestine. Prebiotics, probiotics and organic acids can be used instead of antimicrobials to control the infection. Zinc oxide at permissible limits can be added in the feed. It should be ensured that all piglets consume the colostrum to build up resistance. Feed intake of sows and weaned piglets can be reduced by providing high protein

and high fibre diets. Other than these points, good biosecurity measures are imperative for the control of colibacillosis.

Biosecurity measures

The "All-in-all-out" system of management should be followed. The farrowing area or facility should be thoroughly cleaned, disinfected and dried between farrowings. A good sanitation programme prevents build-up of pathogenic bacteria. The farrowing area can be kept at 70°C and the piglet area even warmer. Used bedding from previous farrowing should be removed or burned.

6.2.2.6. Porcine pasteurellosis

Definition and causative agent

Swine pasteurellosis is a condition in pigs caused by the bacteria *Pasteurella multocida*. This pathogen is associated with atrophic rhinitis, enzootic pneumonia and pleuropneumonia in pigs. Pneumonic pasteurellosis or septicaemic pasteurellosis are forms of the disease when the bacterium acts as primary pathogen. The causative agent, *P. multocida*, is a gram-negative coccobacillus that does not form spores and show bipolar staining characteristics. The bacteria has five capsular sub-groups: A, B, D, E, and F. Pneumonic pasteurellosis is caused by types A and D, acute septicaemia caused by type B, and atrophic rhinitis by type D. Non-toxigenic type A *P. multocida* is the predominant form in pneumonia cases. Most cases of pasteurellosis in pigs are as secondary pathogens, in association with other agents. Atrophic rhinitis is a condition in pigs where there is atrophy of the turbinate bones, distortion of the nasal septum and twisting or shortening of the jaw. Atrophic rhinitis is categorized into two types: non-progressive atrophic rhinitis and progressive atrophic rhinitis. Non-progressive atrophic rhinitis – caused by *B. bronchiseptica* – is mild, temporary and typically has minimal impact on growth and performance. In contrast, progressive atrophic rhinitis – caused by toxigenic *P. multocida* – is severe, permanent and often results in poor growth. *P. multocida* is a worldwide pathogen reported from almost all countries of the world, including India.

Transmission

The modes of transmission of pasteurellosis in pigs are direct contact, through aerosol or sometimes through ingestion. Usually, it occurs in poor husbandry conditions like overcrowded and dusty conditions. It occurs in breeding or finishing herds where enzootic pneumonia is present.

Clinical signs

In cases of acute pasteurellosis, affected animals may exhibit depression, difficulty in breathing with laboured abdominal respiration, coughing, slight nasal discharge and a fever ranging from 40-41ɑ. Mouth breathing and cyanosis of the extremities can also occur. Lung sounds are often pronounced. These clinical signs typically last for 5–10 days and may result in either recovery or death, but symptoms can persist for three to five weeks. Animals that recover often remain thin.

In chronic infection, there are symptoms of fever, chronic coughing and reduced daily weight gain, suggestive of complicated enzootic pneumonia cases. In atrophic rhinitis cases, the disease is characterized by sneezing, dacryoadenitis and coughing. Nasal haemorrhage may also be seen in severe cases. Occlusion of the lacrimal ducts and presence of tear stains below the medial canthi of eyes is also seen. In severely affected pigs, distortion of the nasal septum and twisting or shortening of the jaw is seen. In less severe cases, some degree of turbinate atrophy will be present without any apparent outward distortion.

Lesions

Petechial and ecchymotic haemorrhages in the serous and mucous membranes of various organs and systems are seen in the septicaemic form. The lungs, mesenteric and mediastinal lymph nodes are congested and oedematous. Atrophy of the turbinate bones, with or without distortion, is evident in atrophic rhinitis cases. If the inflammation is active, the mucosa appears blanched, and there might be presence of purulent substances.

Diagnosis

Diagnosis of swine pasteurellosis is based on history, clinical signs, lesions and laboratory-based identification of the organism. Routine monitoring of herds to check turbinate atrophy should be undertaken. For isolation of the agent, mucous and blood are the preferred specimens. For atrophic rhinitis cases, nasal swabs should be used for isolation. Lesion materials may also be used for isolation at necropsy. Mouse pathogenicity test

may be used to verify the culture of *P. multocida*. In cases of suspected pasteurellosis caused by serogroup B, hyaluronidase production test can be utilized. The use of PCR and its variants is crucial to detect and differentiate toxin-producing and non-toxin producing *P. multocida* isolates, thereby differentiating pneumonic pasteurellosis and atrophic rhinitis.

Differential diagnosis

Septicaemic pasteurellosis should be differentiated from Salmonellosis and CSF. For atrophic rhinitis, a differential diagnosis is necessary to exclude diseases or irritants that can cause rhinitis like PPS, pseudorabies, inclusion body rhinitis and excessive dust or ammonia.

Treatment and control

Treatment

Severely infected animals may be treated with commonly available antibiotics such as ceftiofur, ampicillin, amoxicillin, tetracycline, penicillin and streptomycin for three to five days to be administered parenterally. Along with parenteral administration, the drinking water of affected animals may be medicated with doxycycline or any of the above antimicrobials in its soluble form. Long acting oxytetracyclines can also be used. Supportive therapy such as use of NSAIDs and antihistamines is recommended along with the antimicrobials.

Control

No commercial vaccine for pasteurellosis in swine is available in India at present. In such a situation, control of the disease relies on biosecurity and management practices.

Biosecurity measures

All measures that prevent the entry of *P. multocida* are included in the biosecurity measures. The "All-in-all-out" system of rearing should always be practised. Mingling of pigs from different sources should be avoided. Newly purchased pigs should be from reliable source and quarantined before addition to the herd. In case of atrophic rhinitis, if a decision is made to stamp out the disease from the herd rather than controlling it, the herd should first be depopulated and then restocking of the stock should be done from a trusted source. Management and housing of the pigs should be improved, where stressors such as overcrowding, excessive dust, ammonia, and other respiratory diseases should be avoided. The animal house should be thoroughly disinfected as *P. multocida* are susceptible to commonly used disinfectants and dies in dry conditions.

6.2.2.7. Anthrax

Definition and causative agent

Anthrax is generally not common in pigs unless the causative organisms are ingested by them in large numbers. It is caused by the bacterium *Bacillus anthracis*. It is an aerobic, or facultatively anaerobic, capsulated bacterium which produces spores upon exposure to the environment. The vegetative cells do not last long outside the host body without sporulation. The dwarf pigs are resistant to anthrax, where anthrax spores remain ungerminated in tissues.

Transmission

Infection can occur through ingestion (from any wound of the digestive tract), skin abrasions or skin lesions and spore inhalation.

Clinical signs

One of the important signs of anthrax in pigs is sudden death. Anthrax may occur in pigs in three forms, *viz*., pharyngeal anthrax, intestinal anthrax and septicaemic anthrax.

The pharyngeal form is the most common form and is characterized by massive swelling of cervical region and dyspnoea. Piglets develop the pulmonary form of this disease which is characterized by lobar pneumonia and exudative pleurisy.

In the intestinal form, the signs are similar, but apparent cervical oedema and dyspnoea may be absent. In chronic cases, the clinical sign may be vomiting, jaundice, enteritis, diarrhoea and constipation. Both forms can occur in the same animal.

In the septicaemic form, sudden death is common, and the course of the disease is short.

Lesions

In anthrax affected pigs, there may be lack of rigor mortis and the blood may not clot. In a majority of cases, visible external lesions are not found except pharyngeal swelling. In septicaemic anthrax, paleness of the carcass and dehydrated is generally observed.

Diagnosis

The carcass of a pig suspected to have died of anthrax should not be opened because of the risk of human infection, bacterial sporulation and environmental contamination.

The organism can be identified in a stained smear from live animals by Gram-stain or polychrome methylene blue (McFadyen stain) stain. Although time-consuming, the blood culture is considered as the gold standard method.

Other tests such as FAT, Ascoli's thermos-precipitation test and PCR can also be used.

Precaution: The carcass of a pig suspected to have died of anthrax should not be opened because of the risk of human infection, bacterial sporulation and environmental contamination.

Differential diagnosis

Anthrax in pigs should be differentiated from diseases like pharyngeal malignant oedema, acute cases of swine erysipelas, acute CSF and ASF.

Treatment and control

Treatment

Treatment is effective if cases are treated at the initial stage (post-exposure sub-clinical and prophylaxis) of the disease. The most effective antibiotics for the treatment of anthrax are penicillin and oxytetracycline. Penicillin @ 10 000 units per kg BW given twice daily through parenteral route was found to be effective against anthrax. Oxytetracycline given @ 10 mg/kg daily IM or intravenous (IV) is also effective. The daily dose should be divided and given for 12 hours in the initial period of therapy.

Control

Anthrax can be controlled by adopting measures such as breaking the source of infection, proper disease monitoring, proper disposal of anthrax-affected pig carcass and contaminated material, and proper disinfection of the infected area. Attenuated live vaccine, which is used in some species for prevention, is not used commonly in pigs because of their natural resistance to the disease.

Biosecurity measures

The carcass of an anthrax-affected animal or suspected for anthrax should never be opened. If there is an anthrax outbreak, the relevant regulatory officials should be informed immediately. Strict quarantine measures should be adopted in the anthrax-prone areas. The carcass should be buried properly and covered by quick lime. Persons dealing with carcass disposal must adopt adequate hygiene measures.

6.2.2.8. Swine erysipelas

Definition and causative agent

Swine erysipelas is mostly a disease of grower or older pigs. The disease might be clinically unapparent, cause acute illness or might cause signs like joints enlargement, lameness and endocarditis which is observed in chronic cases. It is caused by the bacterium *Erysipelothrix rhusiopathiae*. Twenty-eight different serotypes of *Erysipelothrix* spp. have been reported and at least 15 are found in pigs. The disease has also been reported in pigs from India.

Transmission

Domestic pig is the major reservoir of *E. rhusiopathiae*. Young pigs are naturally exposed to *E. rhusiopathiae,* if the disease is endemic in the farm where they are located. The organism is excreted through faeces and oronasal secretions of infected pigs and, thereby, contaminate the environment. The pigs which recover from the disease and those infected chronically become carriers. Healthy pigs may also act as asymptomatic carriers. The infection generally occurs through ingestion. Contaminated water, feed or faeces are sources of infection. Infection can also occur through skin abrasions. Contaminated pork is also a source of infection.

Clinical signs

There are four clinical forms of the disease in pigs: acute septicaemic form, subacute urticarial form, joint or arthritic form and chronic cardiac.

In the acute septicaemic form, there may be sudden death without the pig showing any prior clinical signs of having contracted the disease. This is mostly observed in grower and finisher pigs. There is an increase in temperature (40-42ɑ) and animals may be reluctant to move. Affected pigs shift their weight from limb to limb when standing. Abortion may occur in pregnant sows.

In the subacute urticarial form, some pigs may show classic cutaneous rhomboid urticaria (diamond skin) after the acute febrile stage. These lesions are mostly seen over the back, rump or shoulders.

If animals are not treated, they develop the chronic form of the disease with signs like arthritis, vegetative valvular endocarditis or both. Because of the chronic arthritis (most common in chronic infection), the animals exhibit mild to severe lameness.

Lesions

Diamond skin lesions are observed in the urticarial form of the disease. Arthritis may be observed in one or more legs. There may be thickening and enlargement of affected joints with presence of inflammatory exudates.

Diagnosis

Clinical signs, typical lesions and favourable response to the high doses of penicillin will help in making tentative diagnosis of the disease. Confirmatory diagnosis should be based on isolation and identification bacterium or molecular confirmation (PCR) using tissues from affected animals. The presence of diamond skin lesions is almost diagnostic, but should be differentiated from diseases of pigs showing similar lesions.

Differential diagnosis

The disease should be differentiated from septicaemia caused by *Salmonella choleraesuis*, hog cholera and infections caused by *Streptococcus suis*. It should also be differentiated from Glasser's disease and infection caused by *Mycoplasma hyosynoviae* in pigs.

Treatment and control

Treatment

The drug of choice for treatment of swine erysipelas is penicillin. High doses of penicillin (50 000 IU/kg BW) may be required for effective response. The diseased pigs should be treated for at least three days at 12-hour intervals.

Tetracycline @ 500 mg/gallon may be added in drinking water on a herd basis. In severe cases, treatment needs to be continued for a longer period. Drugs like lincomycin and tylosin can also be used. To manage fever in acute cases, NSAIDs such as flunixin meglumine parentally or aspirin in drinking water may be used. Treatment of chronic cases is not cost-effective.

Control

Acute cases can be controlled through the use of parenteral penicillin.

Biosecurity measures

Standard farm biosecurity measures, including routine sanitary practices, should be in place for the effective prevention and control of the disease.

6.2.2.9. Greasy pig disease

Definition and causative agent

Greasy pig disease mostly affects young pigs less than eight weeks of age and is found in almost all pig producing countries. It is characterized by variable to high morbidity and variable mortality. It is caused by *Staphylococcus haikus*, a Gram-positive coccus. At least six serotypes have been identified. The disease has also been reported from India.

Transmission

Transmission generally occurs through carrier pigs, but it can also occur from contaminated utensils or clothing. The disease normally occurs when only non-immune pigs are infected, or a new strain is introduced into the farm. Other risk factors such as trauma and environmental irritation are also likely to play a role in the occurrence of the disease. Outbreaks have also been reported in farms after introduction of pigs from other herds.

Clinical signs

Early signs of the disease include rise of temperature, loss of appetite, listlessness or depression, and it may occur in all or some piglets of a litter. The skin may become reddened, but there is no itching. Initial lesions are found in the axilla or groin but often go unnoticed. Brownish spots, which are covered by exudate, are generally found on the skin of the face or head. In later stages of the disease, the lesions turned into brown to black crusts. In young pigs, death may occur within three to five days. In older pigs, there are only a few lesions which are largely confined to the head.

Lesions

Carcass of the affected pigs are dehydrated and have bad smell. There is presence of fissure in the affected areas containing dirt and filth from the environment. Inflammation of the external ears may be observed in chronic cases. Lesions are mostly found on hairy areas, including the feet, but it may affect the mouth and tongue.

Diagnosis

Tentative diagnosis can be made based on clinical signs and lesions. Confirmatory diagnosis is made by PCR, isolation of *S. hyicus and*/or by histopathology.

Differential diagnosis

When only one or a few localized lesions are present in older pigs, the disease must be differentiated from sarcoptic mange infestation and other infectious diseases affecting skin.

Treatment and control

Treatment

Successful treatment can be achieved with daily spraying of antiseptics (0.05 percent chlorhexidine solution, 1:50 dilution of 10 percent povidone iodine) on the entire body surface. In severe cases, treatment with antibiotic is required. The antibiotics need to be given in high dosages in the early stages of the disease and for 7–10 days.

Antimicrobials such as ampicillin, penicillin, oxytetracycline, gentamicin, enrofloxacin, norfloxacin and ciprofloxacin are effective against greasy pig disease. For quick recovery, supportive treatment with skin ointment should be provided.

Control

Control is possible only through the adoption of comprehensive biosecurity measures including, but not limited to, strict hygiene measures such as regular disinfection and cleaning of the pig shed.

Biosecurity measures

Standard biosecurity measures for the control of infectious diseases, such as adoption of strict sanitary measures in the farm, restriction on the entry of visitors, quarantine procedures for introduction new animals to the farm and the like need to be followed meticulously.

6.2.3. Parasitic Diseases

6.2.3.1. Porcine cysticercosis

Definition and causative agent

Porcine cysticercosis is a systemic parasitic disease caused by the metacestodes (cysticercoid) – the larval stage of the human cestode called *Taenia solium*. It primarily affects the muscles and central nervous system in pigs. *Taenia solium* is one of the important zoonotic parasites of pigs and, hence, is of serious public health concern. It can occur in two forms, *viz.*, taeniasis and cysticercosis. Cysticercosis occurring in humans as well as pigs is caused by the ingestion of excreted tapeworm eggs. The ingested eggs develop into larvae and then migrate through the body. Porcine cysticercosis has been reported from many Indian states and is considered to be endemic.

Transmission

Porcine cysticercosis occurs when pigs ingest the eggs of *Taenia solium* from either faeces or any environmental source that was contaminated with faeces from humans that were affected by the adult stages of the parasite.

Clinical signs

Porcine cysticercosis is normally considered to be asymptomatic in pigs. However, some studies in India and abroad have reported clinical signs like excessive salivation, tearing, eye blinking, dullness, apathy, sluggishness, drowsiness and loss of consciousness in affected pigs. Neurocysticercosis, a serious neurological form of cysticercosis that occurs in humans, has also been reported in pigs and is characterized by seizures.

Lesions

Cystic lesions are found mainly in the skeletal muscles (like tongue, masseter and pterygoids) and parts of the central nervous system.

Diagnosis

Diagnosis of cysticercosis mainly relies on visual examination of the muscles of affected pigs at the time of slaughter. Histopathological examination of the affected tissues and grading of the inflammatory changes also helps in diagnosis. Tongue palpitation is a method which can detect cysticercosis in live animals, albeit with very low sensitivity. ELISA for detection of both antigen and antibodies against *T. solium* antigens are available. In the molecular tests, PCR and PCR-restriction fragment length polymorphism (PCR-RFLP) are commonly deployed. Medical imaging techniques like computed tomography (CT) scan and magnetic resonance imaging (MRI) used for the diagnosis of neurocysticercosis in humans may also be used in pigs, but it may not economically be feasible for resource-poor countries.

Differential diagnosis

Porcine cysticercosis should be differentiated from other conditions in pigs causing similar lesions like cysticerci of *Taenia hydatigena* (*Cysticercus tenuicollis*), *Taenia asiatica*, *Echinococcus granulosus*, and non-infectious causes like congenital serous cyst.

Treatment and control

Treatment

The administration of Oxfenbendazole as a single dose of 30 mg/kg BW is highly effective. Drugs like albendazole and praziquantel have had varying levels of success.

Control

Immunization of pigs with Cysvax® vaccine (Indian Immunologicals Ltd) at 1 ml per pig may be given by deep intramuscular route behind the ear area to control the disease.

Mass awareness can be an effective strategy to break the life cycle *of T. solium*. The focus of this should be on the risk factors that contribute to the occurrence of the disease and measures to address public hygiene, especially the construction of adequate sanitary toilets. In addition, strict enforcement of biosecurity measures is essential.

Biosecurity measures

Special emphasis should be given to hygiene and sanitation in order to break the oral-faecal route, and to educating people about the disease, cause, transmission and probable consequences. Application of temperature of 60°C or more for five minutes inactivates the eggs of the parasite. Although the parasite is resistant to many chemical disinfectants, higher concentrations and a longer period of contact can inactivate the eggs. The number of viable eggs can be reduced by Formalin, freshly prepared iodine solutions and chlorine gas. Hypochlorites like 1 percent sodium hypochlorite may be used for disinfection.

Pigs should not be exposed to environments that are contaminated by human faeces. Sewage used to fertilize land used for food crop or pig forage should be treated appropriately to ensure that the *Taenia solium* eggs are inactivated and no untreated sewage effluent should be used for this. Deliberate use of human faeces as pig feeds must be avoided. Adequate number of sanitary facilities like toilets should be made available for humans at the swine farm premises so that exposure to human faeces is avoided.

6.2.3.2. Porcine trichinellosis

Definition and causative agent

Trichinellosis or trichinosis is a serious parasitic zoonosis of mammals caused by nematodes (roundworms). It has a broad host range including humans, animals, birds and reptiles. The causative agents are nematodes under the genus *Trichinella*. This genus consists of ten species and three genotypes as per geographic and host preferences. Among the different species, *Trichinella spiralis* commonly affects pigs, and is also the most common species affecting humans. It also affects rats, horses and many carnivores. Humans get infected by eating raw or undercooked animal meat, including pork. Trichinellosis infection has been reported in India from both pigs and humans.

Transmission

The parasite in pigs is transmitted when they scavenge through the infected carcass of rats, swine and other mammals. It is also transmitted by feeding the pigs with uncooked garbage that contains meat scraps or uncooked meat products that harbour the parasite. Cannibalism, resulting in tail biting, is a possible mechanism of transmission among pig population.

Clinical signs

Natural infection of *Trichinella* spp. usually does not produce any clinical signs in affected pigs. However, death of the affected animal may occur in case of heavy infection.

Lesions

No gross lesions in affected swine are observed, except for white streaking in some cases; that happens if the affected muscle fibres are calcified.

Diagnosis

Direct methods for detection of trichinellosis in pigs involve trichinoscopy and muscle digestion. Trichinoscopy is a rapid test but lacks sensitivity, whereas the muscle digestion test is more sensitive. In trichinoscopy, suspected muscle tissue which is squeezed between plates of glass is examined microscopically. In the muscle digestion test, the

suspected muscle tissue is digested with enzymes like HCl-pepsin, following which the digested residue is concentrated and then examined in microscope. Apart from these two techniques, molecular techniques such as PCR and random amplified polymorphic DNA analysis (RAPD) methods can also be used. Detection of antibodies against the larva of *Trichinella* spp. is another approach. For this, tests such as ELISA, indirect immunofluorescence (IFAT) and western blot are used. Tests such as ELISA have the advantage of the ability to be done in a large number of animals before their slaughter and thus can be used for surveillance purposes.

Treatment and control

Treatment

Thiabendazole at the rate of 50 mg/kg BW may be given orally.

Control

Currently, no vaccine is available for the control of trichinellosis in pigs in India. Control relies on good hygiene management and following biosecurity practices.

Biosecurity measures

Since rats are a source of infection to pigs, rodent control becomes an integral component of biosecurity practices in the control of trichinellosis. Garbage that is used as pig feed must be boiled at 100° C for a minimum of 30 minutes before feeding. Raw meat in feed and any access to the carcass of wild animals should be avoided. Additionally, cannibalism, *i.e.*, tail biting, must be prevented.

6.2.3.3. Porcine ascariasis

Definition and causative agent

Ascariasis is the infestation of pigs by the roundworm, *Ascaris suum*, which is the most prevalent and significant internal parasite of pigs. Ascaris infestation negatively impacts pig health and performance, leading to decreased feed efficiency and significant economic losses. The condition is reported in pigs worldwide, including in India.

Transmission

The disease is spread by the ingestion of the ascarid eggs from the environment. Pigs of all ages are affected. The adult stages of this parasite are found in the small intestine of pigs. Adult parasites are round, thick-bodied worms measuring 15–40 cm in length. An adult worm can produce up to 2 million eggs daily. These adults typically live in a pig for about six months before they start being expelled naturally, but they can survive for an year or more. In cases of heavy infection, a single animal can host hundreds of ascarids in the intestine.

Clinical signs

The symptoms and lesions observed in ascariasis depend upon the level of infestation and the site of predilection. Young and growing piglets show signs of unthriftiness, coughing and have a pendulous abdomen and rough hair coat. They fail to gain weight at the expected rate. Sometimes, abdominal expiratory dyspnoea is seen in affected pigs, which is commonly called "thumping". Severe respiratory disease, which sometimes turns out to be fatal, may be seen in previously unexposed pigs that are placed in heavily contaminated places.

Lesions

Small haemorrhagic lesions may be seen throughout the lungs. Small airways may be seen to be obstructed with larvae and inflammatory exudate. Secondary suppurative bronchopneumonia might be present in few cases. Emphysema often accompanies the pneumonia. Migration of the larvae on the liver produces grey to white lesions, commonly referred to as "milk spots". Heavy infestations lead to diffuse fibrosis of the entire liver.

Diagnosis

Postmortem confirmation of ascariasis depends upon typical "milk spots" lesions or detection of adult worms in the small intestine. Identification of ascarid eggs (thick, golden brown) in faeces by floatation method is a commonly used technique for diagnosis. PCR tests and serological assays have been developed, but not used on a wide scale.

Differential diagnosis

Migrating larvae of other parasites such as *Toxocara canis* and *Stephanurus dentatus* may produce similar lesions and thus ascariasis should be differentially diagnosed from these conditions.

Treatment and control

Treatment

For treatment of ascarids in pigs, imidazole and benzimidazole anthelmintics are the drugs of choice.

Tetramisole @ 15 mg/kg BW, benzimidazoles such as Parbendazole @ 30 mg/kg BW or Fenbendazole @ 5 mg/kg BW orally are suitable in the intestinal stages of the disease. Injectable ivermectin and levamisole are more useful in pneumonic cases. Levamisole is given subcutaneously @ 7.5 mg/kg BW or orally as a drench @ 8 mg/kg BW or mixed into the feed @ 0.72 g/kg feed for herd treatment. Supportive treatment includes antibiotic therapy to prevent secondary bacterial infections, in cases with respiratory involvement.

Control

Control of ascariasis may be achieved by proper deworming of animals. Sows should be dewormed around seven days before farrowing and piglets at six to eight weeks of age with the aforementioned anthelmintic. Early weaning, before ascarid eggs become active, may be done. Good hygiene management and sanitation practices are crucial to control the disease in pigs.

Biosecurity measures

Good management practices are more important than treatment to control ascariasis in pigs. "All-in-all-out" production system should be followed with proper cleaning and sanitation between the groups. Sows should be washed to remove any ascarid egg attached to the skin before they are put in sanitized farrowing crates. A solution of hot caustic soda or hot steam can be used for washing of contaminated pig pens to render them safe. Pigs without ascarids are susceptible to severe pneumonia if they are mixed with ascaris-affected grower and finisher pigs. In such cases, providing a continuous dewormer in their feed for the first 30 days is advisable. To avoid further contamination of the facility, these pigs should be dewormed every eight weeks or less. Sows should be dewormed before moving them to a clean pasture if an open housing system is to be followed. Pasture rotation can be practised as it greatly decreases the exposure of pigs to worm eggs.

6.3 Non-infectious diseases of pig

6.3.1 Piglet anaemia

Definition and etiology

Piglet anaemia, also called iron deficiency anaemia, is a hypochromic-microcytic anaemia mostly found in rapidly growing young piglets which are deprived of dietary iron or are denied access to sources of environmental iron. At birth, piglets have anormal blood haemoglobin level of 12–13 g/dL, which quickly drops to 6-7 g/dL by the tenth to fourteenth day after birth. This shortage of iron results in reduced haemoglobin levels in the red blood cells, which in turn causes decreased oxygen supply to the body and an increased susceptibility to disease.

Piglet anaemia occurs mainly due to three factors: piglets are denied access to soil; they grow very fast and, hence, their iron requirements are high; and sows' milk is deficient in iron.

Clinical signs

Clinical signs observed in piglet anaemia are general weakness along with rough hair coat, wrinkling of skin, and pale mucus membrane. Affected piglets are listless and this is evidenced by drooping of the head and ears, coupled with lack of appetite, diarrhoea and reduced growth rate. As the blood viscosity is reduced, there is presence of systolic murmurs on auscultation. There may be sudden death due to anoxia and there is presence of subcutaneous oedema in the neck, shoulder, and limb areas.

Diagnosis

Diagnosis is based on nutritional history, clinical signs and clinic-pathological findings such as decreased haemoglobin level (8 gm/dL) and total erythrocyte counts (3-4 x 10^{12}/L).

Treatment and control

Treatment

Iron dextran (@150–200 mg) injection by IM route is the simplest method for treatment of piglet anaemia. Piglets should be treated on the fourth and fourteenth day of their life.

Iron mixtures can be given orally and are generally given on the back of the tongue. For better results, these formulations are usually given within 36 hours of birth. It can also be provided through drinking water using a dispenser placed in the creep area. Oral administration of 1.8 percent Ferrous sulphate @ 4 ml/day for seven days starting from birth will also help in preventing piglet anaemia.

Iron sulphate paste can be painted/smeared on the teats of the sow every two to three days.

Control

Piglets must have access to soil. If piglets are raised

on a concrete floor some soil must be evenly spread on the floor. Iron blocks or licks can be installed inside the pig shed/farrowing unit. Iron oral pastes are also commercially available and are used for the control of piglet anaemia but the uptake within any litter is varied.

6.3.2. Rickets

Definition and etiology

Rickets (bowed legs) is usually the result of an inadequate intake of calcium or phosphorus or both, or to a faulty proportion of these minerals in the diet, together with a deficiency of vitamin D. Pigs fed indoors on a ration consisting largely of cereal grains - without access to palatable sun-cured roughage - are, therefore, very likely to develop this deficiency disease. Lack of access to sunshine due to modern confinement production systems and improper orientation of the farm sheds that limit natural access to sunrays predisposes the pigs more to hypovitaminosis D. Sow's milk contains less vitamin D and placental transport of vitamin D is poor and these may be other causes of congenital rickets.

Clinical signs

Common clinical signs of rickets in pig are swollen and painful joint and stiff gait. Swelling is very common in the area of the metaphysis of long bones and some long bones may appear improper, irregular or widened. Posterior paralysis and weakness, along with difficulty in rising and lying down, are also observed. Bowed limbs may be seen in some piglets along with pathologic fracture without any history of trauma. When standing, the front and hind feet are often brought very close together. The back is usually abnormally arched.

Diagnosis

Diagnosis can be made based on clinical signs supported by radiography and serum biochemistry for calcium, phosphorus and Vitamin D concentration. There is hypocalcaemia along with increase in plasma alkaline phosphatase (ALP) activity. In advanced cases, blood-serum calcium is usually lowered from a normal value of 10 to 12 mg/dL to 6 mg or less.

Treatment and control

Treatment

Therapeutic approaches include pain management with the use of analgesics or NSAIDs. It is necessary to administer vitamin D3 supplements orally or via injection and calcium supplements such as calcium carbonate or calcium gluconate to the affected pigs through feed or water.

Control

Adequate outdoor space and appropriate housing to facilitate access to sunlight to enable natural synthesis of vitamin D through the skin must be provided. It must be ensured that the diet contains adequate amounts of the minerals (calcium and phosphorus) in the correct ratio (usually 1.2:1 to 2:1). Parenteral vitamin D3 injection before parturition by intramuscular injection of vitamin D3 to sows at 20 days pre-partum is an effective method for enhancing the vitamin D status of piglets. Proper dietary management so that pregnant sows receive a well-balanced diet is necessary to prevent congenital rickets in piglets.

6.3.3. Zinc deficiency (Parakeratosis)

Definition and etiology

Zinc is an important component of many enzymes and affects the metabolism of carbohydrates, proteins and lipids. Grains and oilseeds contain zinc at low levels which are mostly associated with phytate that makes zinc unavailable to the pig. Usually, the maximum tolerable dietary zinc level for pig is 1000 ppm, with the exception of zinc oxide that may be incorporated at higher levels for a short duration immediately after weaning. Deficiency of zinc is manifested by parakeratosis, reduced growth rate and reproductive problems.

Clinical signs

The clinical signs observed are anorexia, reduced weight gain and feed efficiency, extended parturition time, increased still birth rate, reduced litter size and pig birth weight, alopecia and poor wound healing. Affected pigs show few signs of illness other than skin lesions and reduced growth rate.

Lesions

Initially, reddened papules can be seen on the abdomen and the surface of the thighs. Subsequently, these lesions are covered by crusts. Later, more prominent lesions are seen on the lower legs and on the dorsum. Lesions are also found around the eyes, ears, snout and tail. The affected skin becomes hyperkeratotic and epidermal fissures may be seen

along with secondary infection. Focal or diffuse hyperkeratosis on the tongue is most common feature.

Diagnosis

Diagnosis of zinc deficiency cannot always be made based only on response to nutritional therapy, particularly if it is a long-term deficiency. A nutritional deficiency needs to be diagnosed based on several factors such as clinical signs, dietary review, presence of diseases and management history.

Differential diagnosis

Differential diagnosis of zinc deficiency in pigs should be made with diseases like sarcoptic mange and greasy pig disease. Itching is not observed in parakeratosis, whereas itching is present in sarcoptic mange infestation. Itching is also generally not associated with greasy pig disease. In the case of parakeratosis, the affected animal will recover faster if excessive calcium is removed from the ration and effectively supplemented with zinc.

Treatment and control

Treatment

Inclusion of dietary zinc between 2000 and 3000 ppm is a common recommendation for nursery diets to reduce post-weaning diarrhoea and improve growth performance. Good outcomes can be obtained by adjusting the dietary intake of calcium or zinc or both.

Control

Nursery pig diets should contain 0.8 percent–0.85 percent calcium (with standardized total tract digestible phosphorus of 0.4 percent–0.45 percent) and 100 mg/kg of zinc, assuming daily feed intake of 280–500 g. Grower diets should contain 0.6 percent–0.65 percent calcium and 60 ppm zinc, whereas finisher diets should contain 0.45 percent–0.5 percent calcium and 50 ppm zinc. Sow and boar diets should contain 0.9 percent calcium and 150 ppm zinc. Correction of the deficiency results in rapid recovery.

6.3.4. Iodine deficiency (Goitre)

Definition and etiology

Iodine is a chemical element which is primarily known for its role in the synthesis of thyroid hormones, reproductive performance as well as overall growth and development of animals. Though iodine deficiency is rarely seen in commercial farms due to the incorporation of vitamin and mineral premix in the daily rations, sporadic cases may cause losses to pig farmers due to mortality of the animals. Iodine should be provided to pigs @ 0.14 mg/kg of diet. Iodine content of stabilized iodized salt is 0.007 percent iodine and, when fed sufficiently, it will also meet the iodine requirements of pigs.

Iodine deficiency in pregnant sow occurs mainly due to insufficient feeding of iodized salt. Genetic defect in the sow for the biosynthesis of thyroid hormones is another possible factor. Iodine absorption in the gastrointestinal tract is also affected by fluorine and sulphur. Other factors include ingestion of goitrogenic substances like certain plants, drugs or chemicals by the gestating sow.

Clinical signs

Common clinical signs include birth of weak or dead piglets that are largely devoid of hair. These piglets suffer from myxoedema, especially of the skin of the neck. The skin in the affected area becomes thick and doughy. The tongue may protrude from the oral cavity which is often found to be oedematous. Reproductive problems like stillbirth, decreased fertility or prolonged gestation period may be seen in a few cases. Hairless piglets have greater susceptibility to sun-burning and general loss of stamina and vitality when compared to unaffected litters.

Diagnosis

Diagnosis of deficiency always cannot be made based only on response to nutritional therapy, particularly if it is a long-term deficiency. A nutritional deficiency needs to be diagnosed based on several factors such as clinical signs, dietary review, presence of diseases and management history.

Treatment and control

Treatment

The diet should be supplemented with adequate iodine (for example, iodized salt and mineral mixtures).

Control

The control of iodine deficiency in farm animals, including pigs, can be achieved by the use of iodized

salt containing 0.02 percent of potassium iodide. The iodized salt can be purchased from feed dealers, or salt containing approximately the same iodine content can be prepared by thorough mixing of powdered potassium iodide and granulated stock salt. Iodine levels should be regularly monitored and goitrogenic substances in feed like soybeans and soybean meal should be avoided.

6.3.5 Hypovitaminosis A

Definition and etiology

Unlike other grazing animals such as cattle and sheep, pigs are generally enclosed in pens without access to green fodders, which are the natural source of vitamin A, and hence they are usually deficient in this vitamin. Their main food, whether it be grain, boiled garbage or skim milk, is very low in vitamin A and the levels are often insufficient to supply the pigs' requirements. Vitamin A is stored in the liver where it is released slowly as and when required by the animal. The main tissues for which Vitamin A are essential are lining tissues, especially those of the eyes, respiratory system and reproductive tract, bone and eyes.

Clinical signs

Adult pigs: Adult pigs are rarely visibly affected by a vitamin A deficiency. Night blindness, which is a common symptom, is very seldom noticed in them. In severe cases, the sows either fail to come into heat or infertility is common. Boars deficient in vitamin A are also infertile.

Weaners to growers: The most frequent sign observed in weaners to growers is paralysis. The paralysis usually begins with incoordination of the gait and swaying of the hindquarters. This gradually progresses until the hindquarters are completely paralyzed. The pig eats normally, and growth rate is not greatly affected. In some cases, the affected pig will live indefinitely, while in other cases increasing pressure on the nervous system leads to nervous fits and death. The pigs suffering from advanced vitamin A deficiency would easily become excited and fall over on one side in a spasm or convulsion, roll their eyes, struggling a little or lying with legs extended, squeal as if in pain, and give evidence of laboured breathing.

Newborn piglets: Newborn piglets from sows deficient in vitamin A may be born dead and those born alive are very weak. They may lay on their side, squeal a lot and show no interest in suckling. The stronger ones show a tendency to burrow and hide under the bedding. The eyes of these piglets present the most obvious symptoms – the eyelids are gummed together with a brown wax -like material and the eyeballs are either bulging, cyst-like or practically absent. There is often a lot of fluid in the tissues and the abdominal cavity.

Some piglets may be born normally but with insufficient vitamin A. These piglets suckle normally and appear to make quick initial growth but are very susceptible to intestinal and respiratory infections. Enteritis with scouring, and pneumonia with coughing are commonly seen. In some of the piglets in these litters, bone growth around the brain is abnormal and the resulting pressure on the brain causes various nervous symptoms. Some of the pigs become paralyzed before they are very old and throw nervous fits. The nervous damage usually becomes more severe until death occurs.

Diagnosis

Vitamin A deficiency should be suspected when any litters are born dead or weak, as also when abnormalities of the eyes are present in newborn piglets. If the litters of young pigs are very susceptible to enteritis and pneumonia, or if paralysis of the hindquarters is often seen in pigs in the piggery, then it is necessary to check if there is enough green feed/fodder or vitamin A in the diet.

Enteritis, pneumonia and paralysis are not necessarily caused by vitamin A deficiency. A common disease causing a similar birth of dead piglets is leptospirosis. In this disease, the infected sow does not show any obvious symptom until she aborts, which may occur two to four weeks before farrowing takes place. If the sow farrows at full time, the piglets are born dead or weak. For diagnosis of hypovitaminosis A, testing of sow's blood serum is essential.

Treatment and control

When there are obvious symptoms of vitamin A deficiency, a dose of 10 000 IU of the vitamin A supplement should be given as a drench. Pigs suffering from vitamin A deficiency will usually respond very quickly when the vitamin is supplied in adequate amounts, but animals with advanced symptoms such as marked muscular incoordination or blindness seldom recover completely.

Control

To prevent the occurrence of vitamin A deficiency, good quality fresh green feed should be provided daily, or a vitamin A supplement added to the feed. About 60 g of green feed should be provided to each pig daily. Whole milk has fairly large amounts of vitamin A, but skim milk is a very poor source as are most milk products, meat meal and grain (except maize which is very high in carotene). Cod liver oil and fish oils contain large amounts of vitamin A and can be used as supplements, but it is easier and cheaper to use one of the commercial synthetic stabilized vitamin A supplements. These can be obtained in powder or liquid emulsion form for mixing with the feed; details about the quantities to be fed are supplied with the various preparations. Many commercial preparations contain vitamin A together with vitamin D.

6.3.6 Goose stepping (Pantothenic acid deficiency)

Definition and etiology

Pantothenic acid, also known as vitamin B5, is essential for normal physiological functions in pigs. It plays an important role in the synthesis and metabolism of carbohydrates, proteins and fats. Deficiency of this vitamin can lead to a variety of clinical symptoms, particularly affecting growing pigs and pregnant sows.

Inadequate dietary intake of pantothenic acid, particularly in growing pigs and pregnant sows, lead to the development of deficiency symptoms. Low-quality or improperly formulated feeds may lack sufficient levels of essential vitamins and minerals, including pantothenic acid. Any concurrent gastrointestinal disease/problems that affect the absorption of nutrients from the gastrointestinal tract can also contribute to deficiencies.

Clinical signs

Clinical signs include loss of appetite, leading to significant weight loss and poor growth rates. Goose-stepping gait is a distinctive, exaggerated high-stepping style of walking, primarily affecting the hind limbs, that is commonly observed in pigs with this deficiency. It results from nerve damage and muscle weakness, resulting in a lack of muscle coordination leading to an unsteady and staggering walk. Non-infectious bloody diarrhoea in few cases is also reported. In severe deficiency, death may occur as a result of complete anorexia.

Diagnosis

Diagnosis of deficiency cannot always be made based only on response to nutritional therapy, particularly if it is a long-term deficiency. A nutritional deficiency needs to be diagnosed based on several factors such as clinical signs, dietary review, presence of diseases and management history.

Treatment and control

Treatment

Treatment involves dietary supplementation to correct the deficiency. As per the National Research Council, growing-finishing pigs have a pantothenic acid requirement of 6.0 to 10.5 ppm. D-calcium pantothenate has an availability of 92 percent and can be used to meet the dietary requirement of pantothenic acid and to restore normal levels. Affected pigs should be provided supportive care, including fluid therapy for dehydration caused by diarrhoea. They should be monitored, and secondary infections or complications managed.

Control

It is necessary to ensure that the diet of pigs, particularly growing pigs and pregnant sows, is balanced and contains adequate levels of all essential nutrients, including pantothenic acid. This can be achieved through high-quality commercial feeds or carefully formulated home-mixed rations. Regular assessment and planning of diets to ensure that they meet the nutritional requirements of pigs at different stages of growth and production will help prevent the disease. High-quality commercial feeds that are fortified with essential vitamins and minerals, including pantothenic acid, should be used.

6.3.7. Mulberry heart disease

Definition and etiology

Mulberry heart disease (MHD) occurs due to deficiency of vitamin E (alpha-tocopherol) and selenium, which causes cardiovascular problems and rapid death in young pigs that could be a few weeks to four months of age and are in good health. Typically, there is presence of necrosis and haemorrhages throughout the myocardium, and presence of excessive fluid and fibrin strands in the pericardial sac. Often, there is presence of straw-coloured fluid in the pleural cavity and the lungs are found to be oedematous.

Apart from the main cause, additional factors may contribute to the disease by hindering either the intake or absorption of vitamin E and selenium. Ration fed to piglets which have low methionine and cysteine levels and high in fat and vitamin A deficiency may also predispose the animal to vitamin E and selenium deficiency. Faster growth rate in piglets can also be a risk factor. Storage of grains at high temperatures along with increased moisture content and presence of fungus may significantly interfere with vitamin E levels.

Clinical signs

The main clinical signs of MHD are muscle weakness, lowered body temperature and cyanosis before death. The cells of the liver, heart and skeletal muscle are severely affected. The condition mainly develops due to congestive heart failure along with hydropericardium. Sudden mortality occurs as a result of arrhythmia caused by myocardial lesions. Clinical signs like jaundice, cyanosis and tachycardia may also be observed.

Lesions

At postmortem, increased volume of clear fluid is generally seen in the pericardium. Multiple areas of haemorrhages are observed over the heart surface, which gives a mulberry like appearance. Pulmonary oedema may also be present.

Diagnosis

Diagnosis is generally made based on the gross lesions at postmortem. However, the liver can be submitted for additional diagnostic testing.

Differential diagnosis

MHD should be differentiated from other causes of rapid deaths in piglets. The most common causes like *Streptococcus suis* infection and chronic gastritis resulting ulcers should also be taken into consideration.

Treatment and control

Treatment

Parenteral products of vitamin E and selenium are available and are found to be suitable in preventing mulberry heart disease in fast-growing pigs. If there is sufficient evidence to suspect this disease, or sporadic outbreak is reported, all pigs must receive parenteral vitamin E and selenium. It can also be given through water. The recommended levels for vitamin E are 100 IU/kg in the ration of growing pigs and 60 IU/kg in the ration for pigs in the finishing stage. Generally, MHD responds better to vitamin E than selenium, so the focus should be on correcting the vitamin deficiency for better response. Piglets born from treated sows (injected in late gestation) were found to have increased levels of both the compounds.

Control

As faulty feed storage, higher copper and fat levels and inferior quality feed constituents easily destroy vitamin E in feed, due care should be given during formulation of feeds. Animals must have access to pastures and soil.

6.3.8. Salt poisoning (water deprivation)

Definition and etiology

Salt poisoning (water deprivation; salt toxicosis or sodium ion toxicosis) is seen in pigs either as a consequence of water deprivation or from sudden excessive salt ingestion.

Management factors such as mechanical failure of waterers, overcrowding, unpalatable medicated water, new surroundings or frozen water sources may predispose the animal to salt poisoning. Swine on normal diets can be severely affected when water intake is completely restricted or when consuming high-salt diets with moderate water restriction. The use of whey as a feed or as a component of wet mash can also result indirectly in increase in sodium intake. High-saline ground water, brine or seawater also contains excess sodium.

Clinical signs

Clinical signs of sodium ion toxicosis are acute cerebral oedema that occurs as a result of multiple central nervous system (CNS) lesions. In pigs, early signs may include increased thirst, pruritus and constipation. Signs like blindness and deafness may be exhibited by the affected pig and they may not eat, drink and respond to external stimuli. They may wander aimlessly, head pressing or may bump into objects, circle or pivot around a single limb. Affected animals may show intermittent seizures, and backward and upward jerking of the head, and signs of opisthotonus may also be observed after one to five days of limited water intake. The affected pigs may show signs of paddling, and, in the terminal stage, the animals lie on their sides and may die

within a few to 48 hours. Dog-sitting posture is also observed in some cases. Sometimes, the animals may then rise and continue their wandering.

Diagnosis

Diagnosis associated with water deprivation may be suggested by history, signs and elevated sodium levels in the serum or cerebrospinal fluid. Gross lesions may be absent or limited to gastroenteritis. Gastroenteritis is more likely in pigs consuming salty brine and may be accompanied by diarrhoea.

Differential diagnosis

Differential diagnosis of salt poisoning in pigs can be made by taking into consideration all other encephalitic diseases of pigs. In an affected pen, a clue to the occurrence of water deprivation will be the absence of any urine or wet faeces on the pen floor.

Treatment and control

Treatment

Water-deprived or affected pigs should be reintroduced to water slowly, given only small amounts of water at frequent intervals. This may suppress mortality. Re-hydration of the pig is important, and this can be achieved by dripping water through the rectum or allowing water to drip onto the tongue slowly. An alternate technique is to inject sterile water at body temperature into the abdominal cavity. The sterile water is gradually absorbed into the surrounding blood vessels, which distribute the fluid throughout the body, contributing to overall hydration. If brain oedema is suspected, mannitol 20 percent @ 1.5–2.0 g/kg BW and dexamethasone @ 0.1–0.2 mg/kg BW is advisable.

Control

Supply and access to clean and fresh water is of paramount importance for prevention and control. Proper monitoring of the herd for the presence of signs like the absence of or reduced urine or wet faeces on the floor of the pig shed is very important, as the condition mostly occurs secondary to water deprivation (rather than a primary toxic intake of salt) and salt poisoning is frequently apparent at a pen or herd level.

6.3.9. Neonatal hypothermia in piglets

Definition and etiology

Neonatal hypothermia is a common problem in newborn piglets, especially within the first few days of life. It occurs when piglets are unable to maintain their body temperature within the normal range, leading to significant health risks and increased mortality rates.

The thermoregulation system in newborn piglets have limited ability to regulate their body temperature. Moreover, they have a high surface area-to-body mass ratio, which also increases the heat loss. Other factors include exposure to cold environment and cold air. Wet or damp bedding and poor insulation of the housing or shed can increase heat loss through the skin surface. Piglets that do not suckle enough or properly may not receive the energy needed to maintain body temperature. Also, piglets with low birth weight often have less body fat reserves and are more susceptible to hypothermia.

Clinical signs

Extremities such as ears and limbs are found cold when touched. Shivering and huddling together is commonly observed. Erect hairs, difficulty in standing, reduced activity and convulsion may also be observed. Rectal temperature may drop down below 37.8ɑ.

Diagnosis

Diagnosis can be made based on clinical signs. Shivering, neonatal piglets huddling together or burying themselves in bedding and changes in appearance (bluish extremities, hair erection) may suggest presence of hypothermia.

Treatment and control

Treatment

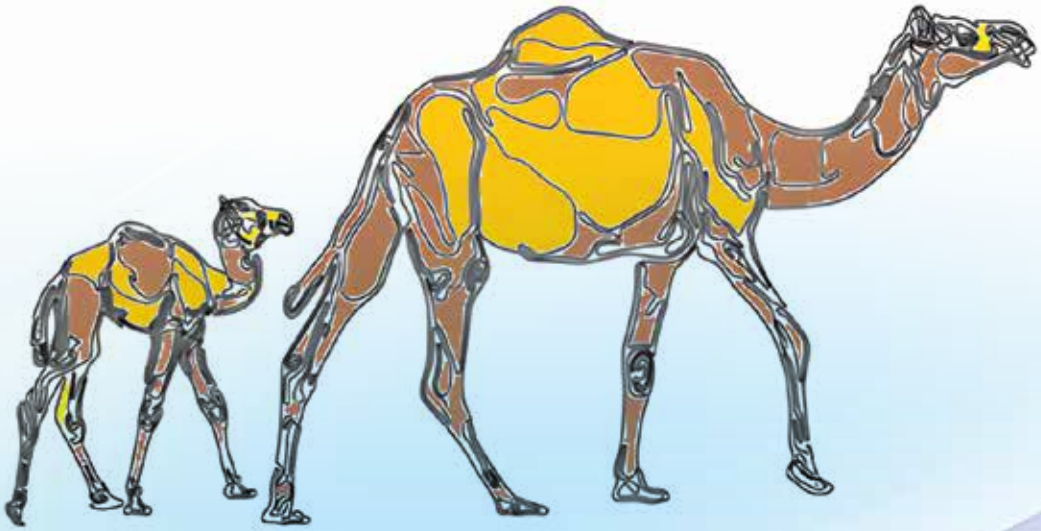
Gradual re-warming by shifting the piglets to a warm, dry environment in order to gradually raise their body temperature is essential. Heat lamps and heat pads can be used to create a warm area for the piglets. Additionally, supplemental feeding with milk replacers must be given to support energy requirements, especially in cold seasons.

Control

Adequate nesting material for sows and piglets should be ensured. Uniform suckling should be encouraged to avoid energy deficiency in piglets. Proper insulated housing should be provided for sows and piglets.

CHAPTER- 7

GUIDELINES FOR CAMEL DISEASES



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Abbreviations

7.1 Preamble

These Standard Treatment Guidelines for camels have been developed based on the comprehensive category so as to help in rationalizing veterinary practices. The objective of the Guidelines is to protect the camel population from irrational therapy and the hazardous consequences following from this, as well as to educate farmers and animal health professionals about the hazards of irrational curative care. Rationalizing clinical camel health care will (a) reduce costs for the animal health system and (b) make the system more effective for the same level of expenditure.

The Guidelines are divided into three sections:

- Infectious diseases: This section comprises disease causative agents -which include viruses, bacteria, fungi and parasites.
- Non-infectious diseases: This deals with metabolic and deficiency diseases.
- Systemic diseases: This covers diseases associated with the respiratory and digestive systems.

7.2 Infectious diseases of camel

Viral diseases

7.2.1.1 Camelpox

Definition and causative agent

Camelpox is a contagious viral disease which is characterized by the formation of skin lesions. Camelpox is caused by the camelpox virus (CMLV) which is a member of a genus *Orthopoxvirus* belonging to Poxviridae family. This virus is closely related to other poxviruses such as cowpox virus in cattle.

Transmission

The CMLV is primarily transmitted through direct contact with infected animals or through contact with contaminated materials such as feed, water, bedding or equipment. It can also be transmitted through arthropod vectors such as mosquitoes, ticks or flies, although this mode of transmission is less common. Infected camels can shed the virus into the environment through scab materials and salivary secretions.

Clinical signs

Incubation period is usually 4–15 days until the onset of fever. Development of skin lesions is followed by pruritis and vesicular rupture, leading to secondary bacterial infections. The disease can also affect the eye, with symptoms of conjunctivitis and lacrimation. In generalized conditions, the camels have fever, anorexia, lymph node enlargement, lethargy and dehydration. Abortion in pregnant camels and drop in milk production of lactating camels has also been observed. In severe infections, respiratory involvement showing nasal discharges and cough are seen, particularly in young camels. Morbidity and mortality (25–100 percent) are high in young camels.

Lesions

Development of characteristic skin lesions include erythematous macules which progress further to papules, vesicles, pustules and later brown crust or scabs.

Diagnosis

Diagnosis of camelpox is based on clinical signs, history of exposure to infected animals and laboratory tests. Detection of viral antigen in pock lesion material can be done by immunohistochemistry, quantitative real-time reverse-transcription polymerase chain reaction (qRT-PCR), antigen-capture enzyme-linked immunosorbent assay (AC-ELISA). Polymerase chain reaction (PCR) with primers for the C18L gene is specific for the detection of camelpox virus. Virus isolation is done by inoculation onto the chorioallantoic membrane of embryonated chicken eggs. Seroprevalence can be studied by ELISA and viral neutralization tests (VNT).

Differential diagnosis

Differential diagnosis for camelpox includes other vesicular diseases such as camel contagious ecthyma and papillomatosis.

Treatment and control

Treatment

There is no specific treatment for poxviral infections. Broad-spectrum antibiotics like oxytetracycline @ 10 milligram (mg)/kilogram (kg) body weight (BW) is administered intramuscularly (IM) as a single dose once daily for three to five days. Non-steroidal anti-inflammatory drugs (NSAIDs) such as meloxicam @ 0.2 mg/kg BW can be given IM daily for five to seven days, depending on the severity of the infection. Topical antiseptic sprays and ethno-herbal formulations to lessen pruritic

lesions are also beneficial. Management primarily involves supportive care to alleviate symptoms, prevent secondary bacterial infections, therapeutic wound management as well as providing adequate nutrition and hydration therapy.

Control

Recovered animals are immune and colostrum can afford some degree of immunity for the first five to six months. Live attenuated viral vaccines can be given in endemic areas. Inactivated vaccines need to be administered yearly. To prevent the introduction and spread of camelpox, quarantine of new animals for 28 days is beneficial. Isolation of infected from non-infected camels and controlling the vector reduce the risk of transmission.

Biosecurity measures

Quarantine of newly introduced animals, isolation of infected camels from healthy ones, proper disposal of carcass and decontamination of the area should be done. The virus is susceptible to various disinfectants including 1 percent sodium hypochlorite, 1 percent sodium hydroxide, 1 percent per acetic acid, 0.5-1.0 percent formalin and 0.5 percent quaternary ammonium compounds. The virus is zoonotic and poses a potential risk for spread through camel handlers.

Sample collection

For diagnosis, samples like fluid from blisters (papules/vesicles/pustules), tissues of scabs or crusts from skin lesions and biopsies from the edge of fresh lesions are collected. In respiratory signs, nasal and oral swabs are taken. If gastrointestinal symptoms are present, then a rectal swab should be collected. Blood samples are required for immunological and molecular diagnostic tests.

7.2.1.2. Camel contagious ecthyma

Definition and causative agent

The camel contagious ecthyma (CCE) – also known as contagious pustular dermatitis (CPD), sore mouth, or orf – is a highly contagious viral disease, mostly of young camels, that contributes to calf debility and, occasionally, calf loss. CCE is characterized by pustular lesions around the mouth, lips, buccal cavity and swelling of the head. The disease is caused by the virus belonging to the genus *Parapoxvirus* of the Poxviridae family.

Transmission

Transmission usually occurs through direct contact with infected animals or contaminated environments. The virus can enter the body through abrasions, cuts or mucous membranes. It can also be spread indirectly through contaminated equipment or fomites.

Clinical signs

CCE is marked by the development of characteristic skin lesions and facial swelling. The lesions include papules, vesicles, pustules and crust formation. There is marked facial swelling in the initial stages of the disease, resulting in vision impairment. Lesions in the oral cavity can lead to excessive salivation or drooling and reduced appetite. Systemic symptoms such as fever, lethargy, weakness and weight loss may occur, especially if secondary bacterial infection develops.

Lesions

Lesions are commonly found on the lips, nostrils, eyelids and areas with less hair coverage, such as the abdomen and inner thighs and, occasionally, on the udder or teats in lactating females. In severe cases, lesions can also develop in the oral cavity, including the tongue, gums and palate. Histopathology of scab lesions reveal hydropic degeneration of keratinocytes with intracytoplasmic eosinophilic inclusion bodies.

Diagnosis

Diagnosis is usually based on clinical signs and history of exposure to infected animals or environments. Laboratory tests such as PCR or virus isolation confirm the presence of the CCE virus.

Differential diagnosis

Differential diagnosis may include other skin conditions affecting camels, such as camelpox, camel papillomatosis, dermatophilosis, bacterial skin infections and insect bite reactions.

Treatment and control

Treatment

There is no specific antiviral treatment for CCE. The disease is usually self-limiting with high morbidity, but a low mortality rate. Symptomatic treatment with antibiotics, NSAIDs and multi-vitamins helps in quick recovery in acute cases. Administration of NSAIDs like Flunixin meglumine @ 1.0 mg/

kg BW IM once daily for three to five days is recommended to reduce facial swelling. Secondary bacterial infections can be treated with systemic broad-spectrum antibiotics, *viz.*, oxytetracycline @ 5.0-10.0 mg/kg BW IM for three to five days or long acting oxytetracycline @ 10–20 mg/kg BW IM which is repeated after 72 hours. Topical application of skin antiseptics for the lesions is also useful.

Control

Control measures include prophylactic vaccination of camels. Vaccination using CCE virus-containing material appears promising. However, immunization with vaccinia virus and a vaccine against infectious ecthyma in sheep and goats did not protect camels from infection. Providing good nutrition and comfortable housing de-stresses camels making them less susceptible to CCE infection. Maintaining good hygiene practices, such as regular cleaning and disinfection of facilities, minimizing contact between infected and susceptible animals, and observance of farm biosecurity measures help prevent the spread of the virus. Additionally, early detection and isolation of infected animals prevents further transmission within herds or populations.

Biosecurity measures

Biosecurity measures include quarantining new batch of camels as well as camels returning from animal fairs, isolating the infected animals, disinfecting equipment and facilities, restricting movement of animals between infected and non-infected areas, and allowing only designated personnel to enter the farm. Handlers and caretakers should follow proper hygiene practices, such as hand washing and wearing protective clothing. Proper disposal of farm waste is an important step in preventing further spread of disease.

Sample collection

Samples to be collected include fluid from vesicles/ papules/pustules, scabs or crusts from skin lesions and biopsies from the edge of fresh lesions. Nasal and oral swabs from camels with respiratory signs and rectal swab from camels with gastrointestinal symptoms should be taken. Blood samples are required for immunological and molecular diagnostic tests.

7.2.1.3 Bluetongue

Definition and causative agent

Bluetongue is an insect-borne infectious viral disease, characterized by mild fever, erosions in the oral cavity, oedema of the cheeks and lips, conjunctivitis and necrotic gingivitis in camels. The disease is caused by the *Bluetongue virus* (BTV) which is a double-stranded RNA virus belonging to genus *Orbivirus* and family Reoviridae.

Transmission

The disease spreads upon exposure to insect vectors, especially the biting midges of *Culicoides* spp. Camels can act as a reservoir for the BTV and play a key role in its epidemiology and transmission.

Clinical signs

The disease is mostly subclinical, but mild to moderate symptoms can be seen in camels. Mild fever, salivation, erosions around nostrils and oral mucosa, facial oedema and conjunctivitis are seen. BTV affected camels may also show symptoms of muscular necrosis, stiffness in limbs, lameness, sterility or abortion. Lactating camels show reduction in milk production.

Lesions

Lesions include haemorrhages and ulcers in the oral cavity, necrosis of skeletal and cardiac muscles, pulmonary oedema, hydrothorax and pericardial effusion.

Diagnosis

The diagnosis of BTV infection is based on either pathogen identification or immune response detection. RT-PCR, real-time RT-PCR, and classical virus isolation are the methods for pathogen identification while competitive ELISA (C-ELISA, serogroup specific), VNT (serotype-specific), and agar gel immunodiffusion test (AGID) are the methods for the detection of the immune response in the host.

Treatment and control

Treatment

No specific treatment is available for BTV disease. If clinical signs are noticed, symptomatic treatment can be given by local dressing of the lesions with mild disinfectants.

Control

There are no vaccines available for BTV in camel. Insect repellents can be used to control vector

populations.

Biosecurity measures

In the absence of vaccines and therapeutics, strict implementation of farm biosecurity measures is the best way to prevent bluetongue. It is necessary to restrict the movement of camels, prevent mixing of camels from various sheds at the same premises as well as from other farms and implement vector control programmes at the farm premises to control the midge population.

Sample collection

Blood samples, nasal, oral and ocular swabs can be collected for virus isolation and PCR assay. During postmortem, spleen, liver, lung and lymph nodes can be sampled for virus isolation and PCR. Heart and skeletal muscles can be sampled if haemorrhages or necrosis is observed on them. Serum samples are required in order to detect antibodies against BTV.

7.2.1.4. Camel papillomatosis

Definition and causative agent

Camel papillomatosis**,** also known as camelid viral papillomatosis or camelid warts, is caused by various strains of Papillomavirus, primarily Papillomavirus type 1 and Papillomavirus type 2. These viruses belong to the Papillomaviridae family.

Transmission

Camel papillomatosis spreads through direct contact with infected animals or contaminated objects such as grooming tools, feeders and water troughs. Additionally, it can be transmitted through biting insects or mechanical vectors. Mange caused by *Demodex* spp. And camelpox can act as a predisposing factor for the development of papillomas.

Clinical signs

The disease primarily manifests as the development of warts or papillomas on the skin and mucous membranes. These growths are typically painless but can become irritable or inflamed, leading to discomfort for the animal. Papillomas can be found as wart-like growth anywhere on the body. Most cases are seen in young animals and occur in the late rainy season, coinciding with outbreaks of CCE and camelpox.

Lesions

The lesions associated with camel papillomatosis are typically raised, firm and cauliflower-like in appearance. They can vary in size and number, ranging from small, isolated papillomas to larger clusters covering significant areas of the skin or mucous membranes.

Diagnosis

Diagnosis of camel papillomatosis is usually based on clinical signs and history of exposure to infected animals or environments. Viral identification can be done by electron microscopy and immunohistochemistry. PCR is to be done for amplification of partial sequences of L1 ORF of *camel papillomavirus* DNA to confirm the diagnosis.

Differential diagnosis

Differential diagnosis may include other skin conditions affecting camels, such as camelpox, CCE, dermatophilosis, ringworm and bacterial or fungal infections. However, the characteristic appearance of the papillomas and the confirmation of papillomavirus infection through laboratory testing help differentiate camel papillomatosis from other diseases.

Treatment and control

Treatment

There is no specific antiviral treatment for camel papillomatosis. In most cases, the disease is self-limiting, and the lesions usually regress spontaneously over time, as the animal's immune system mounts a response against the virus. However, in severe cases, where papillomas cause significant discomfort or obstruction, surgical removal is necessary.

Control

Vaccine against *camel papillomavirus* is available in some countries; it helps to reduce the incidence and severity of the disease. Additionally, maintaining good hygiene practices, such as regular cleaning and disinfection of housing and feeding areas, can help minimize the risk of transmission. Furthermore, it is important to avoid overcrowding and stressors that may weaken the immune system. Control of the fly population also helps in reducing incidence of the disease.

Biosecurity measures

Implementing strict biosecurity measures is crucial

to prevent the spread of camel papillomatosis within and between herds. These measures include quarantine of new arrivals at the farm as well as camels returning from animal fairs, isolation of affected animals, minimizing contact between healthy and infected individuals, disinfection of equipment and facilities, and limiting access to the farm to only authorized persons.

Sample collection

Samples should be taken from the edge of fresh lesions or warts using sterile swabs. Saliva swab or oral secretions should be examined, if lesions are present in or around the mouth. Serum samples are required to detect antibodies against BTV. During postmortem, lymph nodes and any other affected organ can be sampled for virus isolation and PCR assays.

7.2.1.5 Rabies

Definition and causative agent

Rabies is a viral zoonotic disease that affects the central nervous system. It is caused by the rabies virus, which belongs to the *Lyssavirus* genus within the Rhabdoviridae family.

Transmission

Rabies is typically transmitted through the saliva of infected animals, usually via a bite or scratch. The virus can also spread through contact with mucous membranes or open wounds. In camels, transmission can occur through interactions with infected animals, such as dogs or wildlife.

Clinical signs

Rabies in camels occurs with a wide range of clinical signs, typically progressing through different stages. Once clinical signs appear, rabies is almost always fatal.

Prodromal phase (one to three days): Initially, the camel may display subtle behavioural changes such as restlessness, nervousness or increased irritability. There could also be loss of appetite or changes in feeding habits.

Excitative or furious phase (one to seven days): The camel may exhibit uncharacteristic aggression, attacking humans, other animals or objects without provocation. The camel may also exhibit other signs like aimless wandering, agitation, excessive vocalization, growling, snarling, hypersensitivity to touch, light or sound, profuse salivation and difficulty in swallowing. Pupils may become dilated (mydriasis), and the camel may display a fixed, glassy stare. Ataxia or incoordination may be evident, leading to stumbling, falling or difficulty in walking. Seizures, including convulsions or spasms, may occur.

Paralytic or dumb phase (one to four days): The affected camels show decreased muscular activity and signs of paralysis in the limbs or neck. They may appear lethargic. Paralysis of facial muscles with excessive salivation and dropped jaw, paralysis of throat muscles showing difficulty in swallowing, and paralysis of the respiratory system leading to respiratory distress can be noticed in this phase.

Rabies symptoms can vary between individual camels. However, once clinical signs appear, the disease progresses rapidly, typically leading to death within a few days to a few weeks.

Lesions

There is no specific lesion associated with the disease. However, histopathological examination of brain tissue may reveal characteristic changes such as neuronal degeneration, inflammation and the presence of viral inclusions known as Negri bodies. Negri bodies are most common in the hippocampal pyramidal cells, cerebellar Purkinje cells and brainstem nuclei.

Diagnosis

Diagnosis is based on clinical signs and history of exposure to potentially rabid animals. The most reliable method of confirming rabies is through postmortem examination of brain tissue using fluorescent antibody testing (FAT), PCR and RT-PCR to detect viral antigens or nucleic acids.

Treatment

There is no specific treatment for rabies. Prompt and thorough wound care following suspected exposure to rabid animals is very important to reduce the risk of infection. For bite injuries, immediate wound cleaning with 20 percent soft soap solution for at least 15 minutes can prevent the establishment of infection. Benzalkonium chloride and iodine solutions can be used as first aid measures. Post-exposure rabies vaccination is required to prevent occurrence of the disease. In case of bites closer to the head, neck and foreleg; infiltration of rabies

immunoglobulin or monoclonal antibodies into the wound is required.

Biosecurity measures

Biosecurity measures for preventing rabies in camels include controlling access to potentially rabid animals, such as stray dogs or wildlife, and avoiding contact with animals exhibiting abnormal behaviour or signs of illness. Additionally, maintaining secure enclosures and implementing vaccination programmes can help reduce the risk of rabies transmission within camel populations.

Sample collection

Key samples to be collected to confirm the presence of the rabies virus are saliva and cerebrospinal fluid. On postmortem, brainstem and cerebellum samples should be collected for direct FAT, which is the gold standard for rabies diagnosis. Hippocampus area is also to be sampled for detecting the rabies virus. Given the zoonotic nature and serious health implications of rabies, it is crucial to follow proper sample collection, handling and safety protocols.

7.2.1.6 Bovine viral diarrhoea

Definition and causative agent

Bovine viral diarrhoea (BVD) in camels is characterized by gastrointestinal and systemic manifestations. The causative agent of BVD is the bovine viral diarrhoea virus (BVDV), a member of the *Pestivirus* genus in the Flaviviridae family.

Transmission

Transmission can occur through direct contact with infected animals, ingestion of contaminated feed or water, or exposure to contaminated fomites such as equipment or clothing. Additionally, vertical transmission from an infected dam to her offspring can occur, leading to the birth of persistently infected calves that serve as a reservoir of the virus.

Clinical signs

The infected camels exhibit fever, loss of appetite and are lethargic. Respiratory signs include clear or mucopurulent nasal discharges, dyspnoea and cough. Watery diarrhoea or dysentery may be seen, and prolonged diarrhoea may lead to signs of dehydration. BVD infection in pregnant camels can lead to abortion, stillbirths or the birth of weak calves. Symptoms vary in camels from asymptomatic to varying severity. Camels may act as carriers of infection without showing any clinical signs.

Lesions

Lesions include gastrointestinal inflammation and ulceration, Hepatic necrosis, immunopathological lesions in lymphoid tissues, oedema in various organs and tissues and respiratory lesions in cases with concurrent respiratory involvement.

Diagnosis

Diagnosing BVD in camels typically involves a combination of clinical signs, histopathological examination for characteristic lesions and laboratory tests like viral detection assays, namely PCR to detect viral genetic material, and serological tests such as ELISA to detect antibodies against BVDV.

Differential diagnosis

Differential diagnosis for BVD in camels may include other causes of viral or bacterial gastroenteritis, respiratory infections or reproductive disorders. Some of the diseases that may need to be ruled out include *rotavirus* or *coronavirus* infections, salmonellosis, enterotoxaemia, infectious bovine rhinotracheitis and leptospirosis.

Treatment and control

Treatment

There is no specific treatment for BVD in camels. Supportive care may include administration of fluids, electrolytes and nutritional support to manage dehydration and maintain body conditions.

Control

Control measures include isolation of infected animals to prevent further transmission. Hygiene measures such as cleaning and disinfection of the premises and equipment is of utmost importance. No BVDV vaccine is currently approved for use in camelids, although several vaccines are available for use in cattle. Biosecurity protocols must be implemented to prevent the introduction of the virus into susceptible herds. It is also necessary to protect camels from exposure to wildlife reservoirs (or vectors) that may contribute to BVDV transmission.

Biosecurity measures

Implementation of strict hygiene practices at the farm to minimize the risk of fomite transmission is very important. Other effective biosecurity measures include quarantining new batches of

camels as well as camels returning from animal fairs, isolating infected animals, disinfecting equipment and facilities, restricting the movement of animals between infected and non-infected areas, and allowing only designated personnel to enter the farm. Monitoring and surveillance for early detection of BVDV infection should be done.

Sample collection

For the diagnosis of BVD in camels, serum samples are required for detecting antibodies against the virus. Blood samples, nasal and oral swabs, and faecal samples if gastrointestinal symptoms are present, are to be collected for molecular tests. On postmortem, affected lymph nodes, spleen, liver, lungs and intestinal mucosa from areas showing lesions or inflammation are analysed for virus isolation and PCR assay.

7.2.2 Bacterial diseases

7.2.2.1 Brucellosis

Definition and causative agent

Brucellosis is a bacterial infection primarily affecting the reproductive system in camels. It is caused by *Brucella melitensis* and *Brucella abortus*.

Transmission

Transmission typically occurs through direct contact with infected bodily fluids such as placental membranes, aborted foetuses or vaginal discharge. Camels can contract brucellosis by consuming contaminated feed, water or through licking contaminated surfaces. Vertical transmission from an infected dam to its offspring can occur *in utero* or during parturition.

Clinical signs

Infected camels show persistent fever with temperatures range from 40°C to 42°C. They appear lethargic and often exhibit weakness and fatigue. Brucellosis causes abortions in pregnant camels, typically occurring in the last trimester. It can also lead to retained placenta, infertility and decreased milk production in lactating camels. Swollen joints and lymphadenopathy, especially in the neck and under the jaw, are noticed. Few camels can show respiratory signs of cough and nasal discharges. Severe infections manifest in neurological signs of disorientation, torticollis, head pressing and seizures.

Lesions

Aborted foetuses show cutaneous and sub-cutaneous lesions. Nodular lesions or cystic lesions with yellowish fluid are seen in the liver, spleen, kidney, lungs and mammary gland. Epididymitis and testicular lesions are noticed in male camels. Inflammatory lesions and scarring of reproductive tract in camels lead to reduced fertility or infertility. Osteitis and arthritic lesions are noticed in the bones and joints.

Diagnosis

Clinical signs and lesions constitute "suspected diagnosis". Serological tests like Rose Bengal Plate Test (RBPT), Serum Agglutination Test (SAT) and ELISA are employed to detect antibodies against *Brucella melitensis* in the camel's blood or serum, while PCR is commonly used for detection of *Brucella* DNA in blood, tissue or other samples. Isolation of *Brucella* bacteria from blood samples and culturing them on specific media is the gold standard for confirmation of the disease.

Treatment

Treatment with long-term antibiotic regimens such as combination of long acting oxytetracycline at a dose of 25 mg/kg BW IM every two days for 30 days, and streptomycin @ 25 mg/kg BW given IM every two days for 16 days can help in clinical recovery. Clinical symptoms disappear after treatment, but permanent cure is never achieved.

Control

Vaccination is an essential component of brucellosis control in camels. There are two Brucella vaccines in use, namely, *Brucella abortus* (strain 19) which is commonly used in camels and provides immunity against *Brucella abortus and Brucella melitensis* (strain M5-90), and *Brucella melitensis* Rev 1 which provides immunity against *Brucella melitensis*. Vaccination should be performed according to the recommended schedule, typically starting at the weaning age (around six to eight months). Herd management includes culling of infected animals, proper disposal of aborted foetuses and cleaning of the parturition area. Due to the zoonotic nature of the disease, camel owners are to be educated about the disease risk.

Biosecurity measures

Quarantine of newly introduced animals and

animals returning from animal fairs, isolation of sick and recovering animals, preventing contact between infected and healthy animals, thorough cleaning and disinfection of calving pens, proper disposal of placenta and vaginal fluids/discharges after calving of infected animals are crucial biosecurity measures. Strict biosecurity protocols must be implemented to prevent contact with infected camels. Hygiene measures at the farm include regular cleaning and disinfection of the premises, equipment and vehicles in order to minimize disease transmission. Preventing contact between domestic camels and wild animals that may serve as reservoirs for *Brucella* bacteria is also crucial.

Sample collection

For the diagnosis of brucellosis; blood sample, swabs from the vagina in females with reproductive disorders and from aborted foetus and placenta, as well as nasal swabs are required for PCR test and culture. Milk samples can be used for the Milk Ring Test and culture. Serum is used for serological tests such as ELISA and RBPT.

7.2.2.2 Tuberculosis

Definition and causative agent

Tuberculosis is a bacterial infection primarily affecting the lungs, but can potentially affect any part of the body, including the lymph nodes, bones, and other soft tissues. It is caused by *Mycobacterium tuberculosis* complex (MBTC) bacteria, which include *Mycobacterium tuberculosis*, *Mycobacterium bovis* and other related species.

Transmission

Transmission typically occurs through the inhalation of respiratory droplets containing the bacteria, often from close contact with infected animals during coughing or sneezing. Ingestion of contaminated feed or water may also lead to infection, especially if the bacteria are present in the environment in high concentration. Transmission can also occur through direct contact with infected bodily fluids or tissues.

Clinical signs

Clinical signs vary depending on the stage of the disease and the organs affected. Chronic cough, difficulty in breathing and nasal discharge are common respiratory manifestations. Fever, weight loss, lethargy and decreased appetite may occur as the infection progresses. In systemic cases of tuberculosis, emaciation and muscle wasting are major symptoms. In female camels, reproductive problems such as infertility or abortions are noticed. Camels may exhibit changes in behaviour, such as increased restlessness or isolation from the herd.

Lesions

Characteristic granulomas, consisting of aggregates of immune cells, may be present in affected tissues, particularly in the lungs and lymph nodes. Areas of caseous necrosis, where the tissue becomes necrotic and cheese-like, are typical tuberculosis lesions.

Diagnosis

Tuberculin skin test can be done with purified protein derivative (PPD) to detect delayed-type hypersensitivity reactions which are indicative of exposure to MBTC. In the single intradermal test, 0.1 ml of bovine PPD is injected intradermally and changes in skin thickness are noted after 72 hours. An increase in thickness >0.6 mm is considered a highly suspected case. However, this test gives variable results, and in advanced stages of the disease the test may give a negative result. Isolation and cultural identification of the causative organism from clinical samples such as sputum, lymph nodes or other tissues is a gold standard diagnostic test. Molecular diagnosis can be done by PCR assays which can detect *Mycobacterium tuberculosis* DNA in clinical samples with high sensitivity and specificity.

Differential diagnosis

The disease is to be differentiated from other respiratory infections in camels, such as pneumonia caused by bacteria, viruses, fungi and chronic granulomatous conditions caused by fungal infections or reactions to invasion by foreign bodies.

Treatment and control

Treatment

The treatment of tuberculosis in camels involves a comprehensive approach that includes antibiotic therapy, supportive care, monitoring and biosecurity measures. Treatment typically involves long-term administration of antibiotics such as rifampicin @ 5 mg/kg BW IM ---route-----, and streptomycin sulphate 5 mg/kg BW IM once daily for 10 weeks. Use of isoniazid in camels is not recommended as it may cause toxicity. Treatment efficacy may vary from one camel to another, and some strains of MBTC may

be resistant to antibiotics. Providing a well-balanced diet rich in essential nutrients is crucial to support the camel's immune system and overall health during the treatment. Regular veterinary check-ups are necessary to monitor the camel's response to treatment, assess any side effects of antibiotics and make adjustments to the treatment plan as needed. Complete cure is seldom achieved.

Precaution: Use of isoniazid in camels is not recommended as it may cause toxicity.

Control

Infected animals should be isolated from susceptible ones to prevent further transmission. In cases of severe outbreaks or when treatment is not feasible, culling of infected animals is necessary to prevent the spread of the disease. Herd management practices such as regular screening for tuberculosis, prompt treatment of infected animals, and proper disposal of carcasses can help prevent the spread of the disease. Educating camel owners and workers about the risks of tuberculosis transmission to humans and promoting good hygiene practices can reduce the risk of zoonotic transmission

Biosecurity measures

Strict biosecurity protocols must be implemented to prevent contact with infected animals. Quarantining of newly introduced animals as well as animals returning from animal fairs is one of the very effective biosecurity measures. Other important biosecurity measures to be implemented regularly include regular cleaning and disinfection of premises, equipment and vehicles as well as preventing contact between domestic camels and wildlife that may serve as reservoirs for the MBTC.

Sample collection

Clinical samples such as sputum, lymph nodes or other tissues are collected for bacterial culture and PCR tests. Serum samples are collected for serological tests.

7.2.2.3. Anthrax

Definition and causative agent

Anthrax is caused by the bacterium *Bacillus anthracis*, which forms spores that can survive for long periods in the environment. These spores are highly resistant to heat, drying and disinfectants.

Transmission

Bacillus anthracis spores can persist in soil for many years, creating an environmental reservoir of bacteria. Camels can become infected with anthrax by ingesting spores present in contaminated soil, water or vegetation while grazing. Direct contact with contaminated materials, such as soil or carcasses of infected animals, can also lead to transmission. In rare cases, camels can inhale anthrax spores, leading to respiratory anthrax.

Clinical signs

Sudden death without preceding signs is common in the per acute form of anthrax. In acute cases with a more prolonged course, clinical signs may include fever, depression, swelling in the neck, difficulty in breathing and rapid pulse. Ingestion of anthrax spores can lead to gastrointestinal symptoms such as severe abdominal pain and bloody diarrhoea. Cutaneous anthrax in camels can manifest in the form of skin lesions like red bumps on the skin, vesicles or blisters, ulceration with a black centre (eschar or scab), as well as swelling and inflammation around the lesion. Some animals may exhibit signs of internal or external haemorrhage, such as bloody discharge from the orifices and neurological signs like convulsions, disorientation and agitation.

Lesions

Enlargement and congestion of the spleen are characteristic lesions of anthrax. Oedematous and haemorrhagic lesions may be present in various organs and tissues, including the lymph nodes, liver and lungs. Blood may appear dark and fail to clot properly due to the action of anthrax toxins. Cutaneous anthrax in camels is characterized by skin lesions like papule, vesicle or blister filled with fluid; the lesion then ulcerates, forming a black, necrotic centre known as an eschar (scab). Surrounding oedema (swelling) and inflammation may be present. Anthrax can cause ulceration and haemorrhage in the gastrointestinal tract and haemorrhages and congestion of lung tissue in the respiratory tract.

Diagnosis

Clinical signs suggestive of anthrax in combination with sudden death in otherwise healthy animals should raise suspicion. Characteristic lesions observed during necropsy, including spleen enlargement and haemorrhagic oedema can support the diagnosis. If the animal is suspected of dying

of anthrax, then postmortem examination is not recommended.

Precaution: Postmortem of camels suspected of dying due to anthrax is not recommended.

Differential diagnosis

The disease is to be differentiated from other diseases showing sudden death, acute febrile illnesses, septicaemia in camels, acute viral infections and deaths due to poisoning. The disease is also differentiated from malignant oedema or blackleg, which show similar clinical and pathological findings.

Treatment and control

Treatment is not recommended.

Precaution: Treatment of camels suspected of anthrax is not recommended.

Control

Susceptible camel population may be vaccinated with a spore vaccine containing the avirulent Sterne strain of *B. anthracis* (Anthrax spore vaccine) 1 millilitre (ml) annually by subcutaneous injection. Prompt disposal of carcasses and contaminated materials, followed by thorough disinfection of the farm premises helps prevent the spread of anthrax. Avoiding grazing or watering animals in areas known to be contaminated with anthrax spores can reduce the risk of exposure. Educating camel owners and workers about the signs of anthrax, zoonotic risk of anthrax, the importance of vaccination, and proper carcass disposal can help prevent outbreaks.

Biosecurity measures

Isolation of suspected cases to prevent further transmission to healthy animals is necessary. Access to areas where anthrax cases have occurred should be limited and strict biosecurity protocols for personnel and vehicles should be implemented. Regular monitoring for signs of anthrax and prompt reporting of suspected cases to veterinary authorities is necessary.

Sample collection

For the diagnosis of anthrax, blood samples, nasal swabs and swabs from cutaneous lesions are required for bacteriological analysis and microscopic detection of spores. If contamination of the environment is suspected, samples should be collected from soil or feed.

7.2.2.4 Botulism

Definition and causative agent

Botulism - characterized by nervous manifestations - is a toxin-mediated disease caused by the ingestion of botulinum neurotoxins produced by the bacterium *Clostridium botulinum*. This bacterium is commonly found in soil and can produce potent neurotoxins under anaerobic conditions, such as in decaying organic matter or improperly preserved feed.

Transmission

Camels can contract botulism by consuming food or water contaminated with the botulinum toxin or the bacteria itself. This contamination can happen if the feed or water contains decaying organic matter where the bacterium can thrive and produce toxin. Improper storage of feed or silage can sometimes lead to the growth of *Clostridium botulinum* bacteria and the production of its toxin.

Clinical signs

The clinical signs can vary depending on the severity of the infection and age of the animal. One of the most distinctive signs of botulism in camels is drooping or sagging of the eyelids, which can be bilateral or unilateral. Weakness or paralysis of the face, neck and limbs can occur, leading to difficulty in walking, standing or moving. Affected camels may have difficulty in eating or swallowing due to weakness of the jaw and tongue muscles. Botulism can cause facial asymmetry, head tilt, abeyance of pupillary light reflex, corneal reflex and withdrawal reflex, decrease in appetite or refusal to eat. In severe cases, respiratory failure, leading to hypoxia may occur. Affected camels may produce abnormal sounds, adopt an abnormal posture and show urinary and faecal incontinence due to muscle weakness or nerve damage.

Diagnosis

Clinical presentation with progressive muscle weakness and paralysis are suggestive of botulinum toxicity. For diagnosis, serum samples are used for toxin detection assays such as ELISA or mouse bioassay. Faecal samples are examined to detect the presence of *C. botulinum* spores or toxin. During postmortem, gastrointestinal content may be examined to identify toxins or spores. Suspected feed or water samples can be tested for the presence

of the botulinum toxin.

Differential diagnosis

The disease is to be differentiated from other causes of muscle weakness, paralysis, spinal cord injuries, tick paralysis, tetanus as well as other toxicoses affecting camels.

Treatment and control

Treatment

Prompt administration of specific botulinum antitoxin can help neutralize the circulating toxin and prevent further progression of clinical signs. A monovalent (type B) or multivalent antitoxin (total 30 000 international units (IU) administered intravenous (IV) can help in recovery before the animal is recumbent. Muscle relaxants may be administered to reduce muscle spasms and improve muscle tone. Methocarbamol @ 40 to 60 mg/kg BW orally once daily can be tried. A high-calorie diet may be provided to help camels maintain energy levels and support muscle function. Supportive measures such as fluid therapy, assisted feeding and respiratory support may be helpful.

Control

Vaccines against botulism are available in some countries and these provide protection against specific strains of *Clostridium botulinum*. Good sanitation and hygiene practices, including proper carcass disposal and cleaning of feeding equipment, help prevent contamination with botulinum toxin. The consumption of spoiled or improperly preserved feed that may harbour *Clostridium botulinum* spores must be prevented.

Biosecurity measures

Proper storage and handling of feed is required to prevent contamination with *Clostridium botulinum* spores. Regular cleaning and maintenance of water sources is essential to prevent bacterial colonization. Rodent control measures must be implemented to minimize the risk of contamination of feed and water sources.

Sample collection

Identification of botulinum toxin in serum, faeces or feed samples using specialized tests such as ELISA or mouse bioassay as well as isolation and identification of *Clostridium botulinum* from environmental samples or faeces provides confirmatory diagnosis.

7.2.2.5 Enterotoxaemia

Enterotoxaemia, also known as "pulpy kidney disease", is a bacterial disease affecting various animal species, including camel calves.

Definition and causative agent

Enterotoxaemia is a toxaemic condition caused by the toxins produced by the bacterium *Clostridium perfringens*, primarily types C and D. *Clostridium perfringens* is a ubiquitous bacterium commonly found in the environment and the gastrointestinal tract of animals.

Transmission

Animals typically acquire enterotoxaemia by ingesting feed or water contaminated with *Clostridium perfringens* spores. Factors such as sudden changes in diet, overeating or stress can predispose animals to the development of enterotoxaemia.

Clinical signs

Clinical signs can vary depending on the severity of the infection and the age and health status of the camel.

In mild cases, signs start as a watery or loose stool and progress to a more severe, watery diarrhoea. Some camels may vomit in the early stages of the infection. Animals may become lethargic, depressed and show signs of abdominal pain that may vary from mild discomfort to severe colic.

In severe cases, diarrhoea can become very watery and voluminous, leading to dehydration and electrolyte imbalances. Frequent vomiting and severe abdominal pain can lead to colic or even bloody stools. Dehydration and electrolyte imbalances lead to extreme lethargy or recumbency.

Acute onset of enterotoxaemia often manifests suddenly, with affected animals appearing healthy at one moment and severely ill the next moment. Camels may exhibit neurological signs such as ataxia, convulsions and recumbency. Colic-like abdominal pain, abdominal distension and diarrhoea may also be observed. The disease can progress rapidly, leading to death within hours of the onset of clinical signs.

Lesions

The typical lesion of enterotoxaemia is pulpy necrosis of the kidney, characterized by softening and disintegration of the renal tissue. Haemorrhagic lesions may be present in the gastrointestinal tract, particularly in the small intestine. Gas accumulation in the intestinal tract may be observed due to fermentation of feed by *Clostridium perfringens*.

Diagnosis

Sudden onset of neurological abnormalities and abdominal pain are suggestive of the disease. For the diagnosis, faecal samples are examined to detect the presence of *C. perfringens* and its toxins. Intestinal contents, intestinal mucosa and other affected tissue samples are collected during postmortem for detection of clostridial toxins and bacterial identification. Samples of feed or water that the camel may have consumed can be tested for contamination with *C. perfringens*.

Differential diagnosis

Differential diagnosis should be done with diseases showing sudden death, acute neurological signs as in botulism, lead poisoning, viral encephalitis, bacterial meningitis and rabies in camels.

Treatment and control

Treatment

Administration of specific antitoxin against *Clostridium perfringens* toxins is required. Five to 25 ml of a multivalent antitoxin can be administered intravenously, depending upon the body weight of the animal in order to neutralize circulating toxins and mitigate clinical signs. Antibiotic therapy with Penicillin G sodium @ 22 000 to 44 000 IU/kg BW can be given IV or IM, every 6 to 12 hours. Supportive measures such as fluid therapy, electrolyte supplementation and nutritional support are essential for treating affected animals.

Control

Implementing routine vaccination programmes for susceptible camel populations can provide long-term protection against enterotoxaemia. Sudden dietary changes should be avoided, and adequate roughage should be provided in the diet to prevent overeating and gastrointestinal disturbances. Minimizing stressors such as overcrowding, transportation or abrupt environmental changes help reduce the risk of enterotoxaemia outbreaks.

Biosecurity measures

It is important to ensure proper storage and handling of feed to prevent contamination with *Clostridium perfringens* spores. Biosecurity regime of regular cleaning and disinfection of water troughs, feeders and housing facilities should be implemented to minimize the risk of bacterial contamination. Vaccination against *Clostridium perfringens* types C and D provides protective immunity against enterotoxaemia.

Sample collection

Intestinal contents, intestinal mucosa and other affected tissues are collected for toxin identification and bacterial culture. Feed or water samples are collected when contamination with *C. perfringens* is suspected.

7.2.2.6 Tetanus

Definition and causative agent

Tetanus, also known as lockjaw, is a serious and potentially fatal neurological disease caused by the neurotoxin produced by the bacterium *Clostridium tetani* and is characterized by muscle stiffness and spasms. *Clostridium tetani* is a ubiquitous bacterium commonly found in soil, dust and animal faeces. It enters the body through wounds or puncture injuries.

Transmission

Clostridium tetani spores enter the body through wounds, especially deep puncture wounds, lacerations or surgical incisions like castration. The bacterium thrives in anaerobic conditions, such as deep tissue wounds, where it produces toxin.

Clinical signs

Tetanus can cause depression, lethargy and abnormal behaviour, such as restlessness, agitation or aggression. Camels may exhibit stiffness in their muscles, particularly in the neck, back and legs. As the infection progresses, camels may exhibit abnormal movements, such as difficulty in standing or walking, stiff-legged walking, trembling, staggering and lock jaw. Muscle spasms may lead to abnormal postures, such as arching of the back or stiffening of the legs. Other unusual symptoms include increase in salivation and frothing, increased or difficult respiration, hunched posture, decreased appetite, seizures, coma and death.

Lesions

Tetanus typically does not cause significant macroscopic lesions. Histopathological examination may reveal neuronal degeneration and necrosis in affected areas of the brain and spinal cord.

Diagnosis

Disease can be diagnosed based on the history of wounds or puncture injuries during castration or shearing, especially those in areas prone to contamination with *Clostridium tetani*. Characteristic clinical signs of muscle stiffness, spasms and difficulty in opening the mouth aid in the diagnosis.

Differential diagnosis

Differential diagnosis should rule out other neurological disorders causing similar signs, such as rabies and botulism, spinal cord injuries and infectious diseases such as viral encephalitis or bacterial meningitis.

Treatment and control

Treatment

A calm, stress-free environment must be provided to the animal. Administration of specific antitoxin against *Clostridium tetani* toxin (10 000 to 50 000 IU) by IV route once can neutralize the circulating toxin and prevent further progression of clinical signs. Antibiotic therapy with penicillin G Sodium @ 22 000 to 44 000 IU/kg BW IM every 6 to 12 hours for four to five days or metronidazole (5–10 mg/kg BW administered intravenously every 12 hours), may be given for four to five days. Proper wound care and debridement must be ensured to remove contaminated tissue and reduce bacterial load. Supportive measures such as fluid therapy, muscle relaxants and sedatives may be used to manage muscle spasms and pain.

Control

The camels should be vaccinated against tetanus prior to castration, shearing and long-distance transportation. Prompt and thorough cleaning and disinfection of sheds should be a routine when animals are sick. Minimizing stressors such as overcrowding, or transportation reduce the risk of injuries and wounds.

Biosecurity measures

Measures to prevent wounds should be implemented, such as providing safe housing and minimizing exposure to sharp objects or rough surfaces. Prompt cleaning and disinfection of wounds should be done to prevent contamination with *Clostridium tetani* spores.

Sample collection

For the diagnosis of tetanus in camels, wound exudate or swabs collected from the site of injury or suspected entry point of the infection must be examined. Muscle tissue or tissue from the wound site can also be collected. Serum samples are required for toxin detection assays or serological tests.

7.2.2.7 Caseous lymphadenitis

Definition and causative agent

Caseous lymphadenitis (CLA) is a chronic bacterial infection characterized by the formation of abscesses in the lymph nodes and other tissues. The disease is caused by the bacterium *Corynebacterium pseudotuberculosis*, which is commonly found in the environment and can survive for long periods in soil and on contaminated surfaces.

Transmission

Transmission typically occurs through direct contact with contaminated materials, such as feed, water or equipment, or through contact with infected animals. Entry of the bacterium through wounds or abrasions on the skin can also lead to infection. Contaminated fomites such as shared needles or surgical instruments can serve as vehicles for transmission.

Clinical signs

Some infected camels may not show any clinical signs and remain carriers of the bacterium. Camels may develop firm, painless swellings (abscesses) in the lymph nodes, typically in the neck, shoulder or groin regions. Abscesses may gradually enlarge over time and may rupture spontaneously, leading to the discharge of purulent material. In severe cases, camels may exhibit systemic signs such as fever, lethargy and weight loss.

Lesions

The typical lesions of CLA are encapsulated abscesses containing caseous material, which may vary in size and location. Abscesses are commonly found in the lymph nodes, particularly those draining the head and neck regions. In advanced cases, abscesses may

develop in internal organs such as the liver or lungs.

Diagnosis

The presence of characteristic abscesses in the lymph nodes, combined with systemic signs of infection, are suggestive of CLA. Pus or exudate from abscesses or enlarged lymph nodes, swabs from the surface of abscesses or draining tracts can be collected for bacterial examination. Isolation and identification of the *Corynebacterium pseudotuberculosis* from clinical samples or abscess contents aids in the confirmatory diagnosis.

Differential diagnosis

The disease should be differentiated from other diseases causing lymphadenopathy and abscess formation in camels, such as tuberculosis, brucellosis, fungal infections and certain non-infectious conditions causing swelling, masses such as neoplasia or foreign body reactions.

Treatment and control

Treatment

Surgical drainage of abscesses combined with debridement and irrigation of affected tissues is necessary. Corynebacteria are very sensitive to penicillin, tetracyclines and cephalosporines, but the fibrous capsule and the pus in the abscess prevents the medication from reaching the bacteria. Since erythromycin is better able to penetrate tissues, a combination of Procaine penicillin G @ 10 000 to 20 000 IU/kg BW IM once daily and erythromycin @ 5 to 10 mg/kg BW administered intramuscularly once daily may be used to treat CLA. Intravenous injection of 20 percent sodium iodine in 500 ml sodium chloride as a single treatment, which can be repeated two to three weeks later, can also be tried.

Control

Vaccination against CLA may be available in some regions and can help reduce the incidence and severity of the disease. Implementing strict biosecurity measures, such as screening new additions to the herd, practising good hygiene and minimizing exposure to contaminated environments can help prevent CLA transmission. Regular monitoring for clinical signs of CLA and prompt intervention in suspected cases can help prevent outbreaks and limit the spread of the disease within camel populations.

Biosecurity measures

All infected animals must be removed from sheds and the sheds should be disinfected. Dung, bedding and topsoil from sheds must be removed, and the herd must be moved immediately to a newly erected area. Animal housing should be free from wire and other causes of skin trauma. External parasites must be controlled. Purchase of animals should only be allowed from herds with no history of abscessation. The entire herd must be subjected to serological screening and reactors removed; no serological positive animal should be detected after the second screening. Affected animals must be quarantined and isolated immediately to prevent further spread of the disease within the herd. Thorough cleaning and disinfection of facilities, equipment and vehicles must be ensured. Measures to minimize direct and indirect contact between infected and susceptible animals, such as separating clinically affected animals from the rest of the herd, must be implemented.

Sample collection

For the diagnosis of caseous lymphadenitis, pus or exudate from abscesses or enlarged lymph nodes, swabs from the surface of abscesses or draining tracts can be collected for bacterial examination. Tissue samples can be collected from affected lymph nodes or abscesses during postmortem examinations.

7.2.2.8. Pasteurellosis

Definition and causative agent

Pasteurellosis/haemorrhagic septicaemia is an acute fatal disease of camels characterized by fever, oedema of the throat region, dyspnoea and sudden death. The disease is caused by *Pasteurella multocida* type A or several serotypes of *Mannheimia haemolytica*. Both organisms reside mostly as a part of the normal respiratory flora of camels and other animals. It may become pathogenic when the vitality of the camel is lowered by malnutrition and parasitism or due to inclement weather such as high humidity or rainfall.

Transmission

The worst epidemics occur during the rainy season and particularly affect animals in poor physical condition. The disease largely occurs when the resistance of the body is lowered by transportation over long distances, deficiencies of dietary vitamins and minerals, heavy parasitic infestation,

trypanosomosis and sudden changes in weather. *P. multocida* is transmitted by direct contact with infected animals and through fomites. Camels become infected when they ingest or inhale the causative organism, which may originate in the nasopharynx of infected animals. Mortality is nearly 100 percent unless the animal is treated very early in the disease.

Clinical signs

Affected animals show a wide range of pulmonary and septicaemic manifestations. This is an acute febrile respiratory disease with fulminating fibrinopurulent bronchopneumonia and fibrinous pleurisy. The disease develops within 10 to 14 days with signs of cough, dyspnoea, and mucopurulent nasal and ocular discharges. Newborn camels of two to three days of age can die due to toxaemia before the development of pulmonary lesions.

There are three clinical forms of Pasteurella infection in camels: per acute, acute and abdominal forms. The acute form is identical to haemorrhagic septicaemia. Clinical signs of pasteurellosis include increased rectal temperature (40°C), pulse and respiration rate, dyspnoea, dullness, depression and abdominal pain associated with haemorrhagic enteritis. There is subcutaneous swelling of the neck and between the mandibles. Mandibular and cervical lymph nodes also become enlarged and painful. Affected camels also show signs of dilated nostrils and open-mouthed breathing. In some cases, there is tar-coloured faeces (melena), abdominal pain and coffee-coloured urine. Prognosis is guarded in these cases. Both recovered and sick animals will discharge the organism through excretions and bodily secretions.

Lesions

Lesions of pneumonia, including consolidation and necrosis of lung tissue, may be observed on postmortem examination. Haemorrhages may be present in various organs and tissues, such as the lungs, liver, spleen and lymph nodes. Oedema and congestion of mucous membranes, particularly in the respiratory tract, may also be evident.

Diagnosis

Characteristic clinical signs of acute respiratory disease, combined with rapid progression to septicaemia and shock, provide clues to diagnosis. On postmortem examination, characteristic lesions such as pneumonia, haemorrhages and oedema are evident. Isolation and identification of *Pasteurella multocida* can be done from clinical samples, such as nasal swabs, lung tissue or blood. In freshly dead animals, a heparinised blood sample or swab should be collected from the heart within a few hours of death, as well as a nasal swab. Spleen and bone marrow provide excellent samples for the laboratory isolation of the organism by cultural and biological methods. Serotyping methods include the rapid slide agglutination test, indirect haemagglutination test, somatic antigen agglutination tests, AGID and counter immunoelectrophoresis. PCR technology is routinely used for rapid, sensitive and specific diagnosis.

Differential diagnosis

The disease can be differentiated from acute respiratory illness in camels like bacterial pneumonia caused by other pathogens, and septicaemia and haemorrhagic syndromes associated with anthrax or leptospirosis.

Treatment and control

Treatment

Antibiotics such as penicillin, amoxicillin, cephalothin, ceftiofur, cefquinome, streptomycin, gentamicin, spectinomycin, tetracycline, sulfonamides, trimethoprim/sulfamethoxazole, erythromycin, enrofloxacin, amikacin and norfloxacin can be used for treatment. Antibiotic for treatment can be selected based on antibiotic sensitivity test. However, ceftiofur, gentamicin, oxytetracyline and sulphonamides appear to give better clinical response. For clinical management of respiratory distress and septic shock; fluid therapy, NSAIDs and oxygen therapy can be beneficial.

Control

Vaccination against *P. multocida* can help reduce the severity of the clinical disease. Implementing routine vaccination programmes for susceptible camel populations can provide long-term protection against pasteurellosis. Routine implementation of good hygiene practices and minimizing stressors such as transportation or overcrowding are important to reduce the risk of infection.

Biosecurity measures

Important biosecurity measures include quarantining of new arrivals and animals returning

from animal fairs, isolation of infected animals to prevent further spread of the disease within the herd, regular cleaning and disinfection of the farm premises, utensils, equipment and vehicles, proper disposal of animal waste, limiting contact between infected and uninfected animals and preventing contact between personnel handling infected and healthy herds.

Sample collection

For the diagnosis of pasteurellosis in camels, nasopharyngeal swabs collected from the nasal passages or throat are examined to detect the presence of bacteria. Blood samples are used for bacteriological culture and serological testing. Exudate from any external lesions or abscesses, lung tissue collected during post-mortem are examined to identify the bacteria.

7.2.2.9 Salmonellosis

Definition and causative agent

Salmonellosis is an infectious disease caused by different serotypes of bacterium Salmonella including *Salmonella enterica* serotypes such as *S. typhimurium*, *S. enteritidis*, and *S. enterica Serovar Newport*. These bacteria are Gram-negative, facultative anaerobes that commonly reside in the intestines of many animals, including camels.

Transmission

The faecal-oral route is the most common mode of transmission. Infection may occur by ingesting food or water contaminated with Salmonella bacteria, and through contaminated environment such as surfaces or objects like feeding troughs, water sources or bedding materials. Camels that are stressed or have weakened immune systems are more susceptible to this infection. Environmental conditions, such as temperature and humidity, can influence the survival and proliferation of Salmonella bacteria in the camel's surroundings. Some camels may act as carriers without showing clinical signs of illness and shed the bacteria in their faeces, potentially contaminating the environment and infecting other animals.

Clinical signs

A variety of clinical signs, ranging from mild gastrointestinal upset to severe systemic illness may be observed. Diarrhoea may range from mild to severe, with watery, mucoid and foul-smelling faeces. Camels may show signs of abdominal discomfort, including restlessness, lying down frequently, or standing in a stretched-out posture. Systemic signs include fever that commonly ranges between 39°C and 41°C. Infected camels may appear weak, depressed, anorectic and reluctant to move. In rare cases, Salmonella infection can lead to pneumonia, which may manifest as coughing or respiratory distress. Abortion or stillbirths may occur in pregnant camels.

Lesions

Postmortem examination may reveal characteristic lesions of enteritis, including congestion, haemorrhage and ulceration of the intestinal mucosa. In severe cases, especially with certain serotypes like *Salmonella typhimurium*, camels may develop pseudomembranes on the intestinal mucosa. Systemic lesions may include splenomegaly, hepatomegaly and hepatitis in severe cases. Lymphadenopathy, particularly enlargement and congestion of mesenteric lymph nodes, may be observed. Multifocal necrosis, pneumonic change in lungs and placentitis are other common lesions.

Diagnosis

Diagnosis is based on the presence of characteristic clinical signs (such as diarrhoea, fever and abdominal pain), isolation and identification of Salmonella bacteria from faecal samples/rectal swabs/affected tissues, and identification of Salmonella by detection of Salmonella DNA in clinical samples by PCR assays with high sensitivity and specificity. Blood serum can be tested for antibodies against Salmonella; however, serological tests may not differentiate between active infection and previous exposure. In cases of severe illness or mortality, a postmortem examination of the camel may reveal characteristic lesions.

Differential diagnosis

The disease should be differentiated from other gastrointestinal disease in camels, such as coccidiosis, rotaviral infections, dietary indiscretion, clostridial enteritis and colibacillosis.

Treatment and control

Treatment

Fluid therapy is essential for managing dehydration and electrolyte imbalances associated with diarrhoea. Administration of antibiotics, such as

fluoroquinolones (Enrofloxacin @ 5–10 mg/kg BW IM/IV once daily for five to seven days); third-generation cephalosporins (Ceftiofur @ 2.2–4.4 mg/kg BW IM/IV once daily for five to seven days); Aminoglycosides (Gentamicin @ 4-– 6 mg or Amikacin @ 10–15 mg /kg BW IM/IV once daily for five to seven days); Tetracyclines (Doxycycline @ 5–10 mg/kg BW orally or IV once daily for 10 to 14 days);. Sometimes, Trimethoprim-Sulfonamides combinations are used (Trimethoprim @ 15 mg and sulfamethoxazole @ 75 mg/kg BW orally or IV twice daily for five to seven days). Supportive treatment includes anti-diarrhoeal medications and rehydration fluid support. NSAIDs may be prescribed to alleviate fever, reduce inflammation and alleviate pain associated with Salmonellosis.

Control

Practising good hygiene, including regular cleaning and disinfection of feeding and watering equipment, can minimize the risk of Salmonella contamination. Clean and uncontaminated feed and water sources should be provided to reduce the risk of exposure to Salmonella. Vaccination against specific serotypes of Salmonella may be available in some regions and can help reduce the incidence and severity of salmonellosis outbreaks.

Biosecurity measures

Affected animals should be isolated to prevent further spread of the disease within the herd. Quarantine measures should be put in place for new additions to the herd to prevent the introduction of Salmonella in the herd. Thorough cleaning and disinfection of facilities, equipment and vehicles can help eliminate Salmonella contamination.

Sample collection

For the diagnosis of salmonellosis, fresh faecal samples/blood samples/rectal swabs are examined for bacteriological culture and serological testing. During postmortem, samples are collected from affected organs such as the intestines, liver, spleen and lymph nodes for bacterial examination

7.2.2.10 Mastitis

Definition and causative agent

Mastitis in camels is an inflammatory condition of the udder, typically caused by bacterial infection. It can affect one or more quarters of the udder and is characterized by swelling, heat, pain and changes in the chemical and microbial quality of milk. Mastitis in camels can lead to decreased milk production, changes in milk composition and, in severe cases, systemic illness. Multiple bacterial agents can cause mastitis in camels, including *Staphylococcus aureus, Streptococcus* spp., and other environmental pathogens including *Klebsiella* spp., *Pseudomonas* spp., and *Enterobacter* spp.

Transmission

Transmission of mastitis pathogens occurs through various routes, primarily involving the introduction of pathogenic bacteria into the udder through the teat canal. Common modes of transmission include contaminated milking equipment, poor hygiene practices during milking (improper udder sanitation), environmental contamination (contaminated bedding, soil, faecal contamination and the like) and trauma to the udder and teat.

Clinical signs

Clinical signs of camel mastitis can vary depending on the severity and duration of the infection, as well as the specific bacterial agents involved. Common clinical signs include swelling in the udder and teat that appears warm and painful to touch, changes in milk appearance, decrease in milk yield and presence of blood or pus in milk. Systemic signs of illness like fever, lethargy, loss of appetite may be noted in severe cases. Abscess may develop in the udder and chronic cases are characterized by udder and teat fibrosis.

Diagnosis

Diagnosing camel mastitis typically involves a combination of clinical examination, milk sampling and culture and, sometimes, imaging techniques. A thorough examination of the camel's udder and milk can reveal clinical signs including swelling, heat, pain, changes in milk appearance and systemic signs of illness. The California Mastitis Test (CMT) is recommended for routine screening of lactating camels. Somatic Cell Counting (SCC) is a more sensitive test, particularly for diagnosis of subclinical mastitis. Other animal side tests used for cattle and buffalo may also be used. Milk samples are collected aseptically from affected udder quarters for bacterial culture and sensitivity testing to identify the causative bacterial agents and their antibiotic sensitivity profile for selection of suitable antimicrobials and determine appropriate antibiotic

therapy.

Differential diagnosis

Differential diagnosis includes traumatic injury to the udder like bruising, lacerations or other injuries to the udder. Neoplastic growths or abscesses within the mammary gland can present with swelling, pain and changes in milk production. Conditions such as mycotic mastitis, contagious agalactia or viral infections may manifest with similar clinical signs but require different treatment approaches.

Treatment and control

Treatment

Treatment and control of camel mastitis involve several key strategies aimed at eliminating the causative bacteria, reducing inflammation and restoring udder health. Prompt treatment increases chances of complete recovery.

Antimicrobials should be selected based on antibiotic sensitivity testing. Systemic administration of antimicrobials is required. Intramammary infusion of antibiotics directly into the affected udder quarters can also be considered, but care should be taken that the septum dividing the teat canal should not be damaged. NSAIDs may be used to reduce inflammation, alleviate pain and improve the comfort and welfare of the animals. Feeding mineral mixture regularly to camels has been found to lower intramammary infections. Measures such as frequent milking or massage of affected udder quarters can help improve milk flow and drainage, reducing swelling and discomfort.

Control

Mastitis control involves a multifaceted approach including change in management practices, such as regular udder health checks, proper nutrition and appropriate milking techniques. Post-milking teat dipping also helps in reducing the disease incidence. Housing facilities should be maintained in a clean and dry condition with adequate ventilation to reduce the risk of environmental contamination. It is a good practice to keep the camels standing for 30 minutes post-milch by luring the animals with green fodder and feed concentrate.

Biosecurity measures

New animals should be quarantined before being introduced into the herd, and thorough health screenings, including testing for mastitis and other infectious diseases, should be conducted. Camels showing signs of mastitis or other infectious diseases should be isolated to prevent the spread of the infection to healthy animals. Contact between camels and potential sources of environmental contamination, such as contaminated bedding materials, soil or standing water, should be minimized. Animal handlers should be trained in proper milking techniques, udder hygiene and disease recognition.

Sample collection

Milk samples are collected taking aseptic precautions.

7.2.3 Fungal diseases

7.2.3.1 Camel dermatomycoses

Definition and causative agent

Camel dermatomycoses, also known as dermatophytosis or ringworm, is a fungal infection of the skin and hair follicles in camels. It is caused primarily by fungi belonging to the genera *Trichophyton*, *Microsporum* and *Epidermophyton*.

Transmission

These fungi thrive in warm, humid environments and can infect camels through direct contact with contaminated objects, infected animals or environmental sources like soil or bedding. Minor skin injuries, which can occur through grazing, fighting or during handling, provide an entry point for the fungi, facilitating infection.

Clinical signs

Clinical signs can vary depending on the specific fungal species involved, the extent of the infection and individual factors such as the camel's immune status. Alopecia is a characteristic sign of dermatomycoses in camels. Alopecic lesions may be localized or diffused. Erythema and inflammation of the skin are common features of dermatomycoses. Scaling and crusting may occur in conjunction with hair loss and erythema, contributing to the overall appearance of lesions. Pruritus (itching) can range from mild to severe and may lead to self-trauma by the infected animals, including rubbing, scratching and biting the affected areas. Ulcerated lesions are prone to secondary bacterial infections, which can exacerbate clinical signs and delay healing.

Lesions

Lesions are distinctive, circular or ring-shaped. Ring-shaped lesions have raised inflamed borders, which are reddened compared to the centre. Lesions can vary in size from small spots to large patches, depending on the severity and duration of the infection. The infection weakens the hair shafts, causing them to fall, leaving bald patches. In some cases, hairs may break off near the surface of the skin, resulting in a stubby appearance. As the infection progresses, the lesions can develop crusts, which are typically yellowish and hard. These crusts form dried exudate, dead skin cells and fungal elements. Common sites include the head, neck, limbs and, occasionally, the body. Prolonged scratching and irritation can lead to secondary skin changes such as lichenification (thickening of the skin) and hyperpigmentation.

Diagnosis

Clinical examination of the skin and hair coat should be conducted for characteristic signs of fungal infection, such as alopecia (hair loss), erythema (redness), scaling, crusting and pruritus (itching). Lesions may be localized or widespread, and their distribution and severity can provide valuable diagnostic clues. Microscopic examination of skin scrapings should be done to check for the presence of fungal elements such as hyphae, spores or arthroconidia. Fungal culture is considered the gold standard for diagnosing dermatomycoses in camels. Samples collected from affected skin areas or hair follicles are cultured on specific fungal growth media under controlled conditions. Histopathological examination involves the microscopic evaluation of skin biopsy samples obtained from affected areas. PCR-based assays can be used to detect and identify fungal DNA in clinical samples with high sensitivity and specificity.

Differential diagnosis

Conditions that may mimic camel dermatomycosis include other causes of alopecia and skin lesions, such as bacterial infections, mange, and autoimmune skin diseases.

Treatment and control

Treatment

Topical applications include any of the following: Miconazole (2 percent) cream to the affected areas twice daily for two to four weeks; Clotrimazole (1 percent) cream to the lesions twice daily for two to four weeks; Terbinafine (1 percent) cream to the affected areas once or twice daily for one to two weeks; Ketoconazole (2 percent) cream to the lesions once or twice daily for two to four weeks.

Medicated shampoos can also be used. Camels can be bathed with either Chlorhexidine or Miconazole shampoo once or twice a week, with the shampoo being left on the skin for 10–15 minutes before rinsing thoroughly or with Ketoconazole shampoo twice or thrice a week, leaving the shampoo on the skin for 10–15 minutes before rinsing.

Systemic antifungal drugs like Griseofulvin @ 10–15 mg/kg BW can be administered orally once daily for four to eight weeks. This should be administered along with fatty food to enhance absorption and monitored for potential side effects such as bone marrow suppression and gastrointestinal upset. Itraconazole @ 5–10 mg/kg BW can be administered orally once daily for three to six weeks. Fluconazole @ 2.5–5.0 mg/kg BW can be administered orally once daily for two to four weeks. It is well-absorbed and has fewer drug interactions. Camels should be monitored for any signs of hepatotoxicity.

Control

The camels' living environment, including bedding, feeding equipment and any surfaces they are frequently in contact with should be regularly and thoroughly cleaned and disinfected. Effective antifungal disinfectants such as diluted bleach (1:10 dilution) or commercial antifungal disinfectants should be used. It is necessary to ensure that the camel receives a balanced diet rich in vitamins and minerals to support its immune system. Supplements such as vitamin A, vitamin E and zinc to promote skin health and recovery can be considered. Regular grooming and inspection of animals can help detect early signs of infection, allowing for prompt treatment and control measures.

Biosecurity measures

Implementing strict biosecurity measures is crucial for preventing the spread of camel dermatomycosis within a herd or facility. Infected animals should be isolated from healthy ones to prevent direct contact and minimize transmission. Contaminated objects, equipment and living areas should be regularly cleaned and disinfected to remove fungal

spores. New animals introduced to the herd should be quarantined and tested for dermatomycosis to prevent the introduction of infection. Handlers and caretakers should use appropriate personal protective equipment (PPE), such as gloves and protective clothing, when handling affected animals or cleaning contaminated areas.

Sample collection

For the diagnosis of camel dermatomycosis, skin scrapings, and hair plucks can be collected by scraping the edge of the lesion. If lesions are moist, swabs can be used to collect the exudate by rubbing over the lesion.

7.2.3.2 Camel candidiasis

Definition and causative agent

Candidiasis in camels is an opportunistic fungal infection caused by various species of the *Candida* genus, most commonly *Candida albicans*. This infection can affect the skin, mucous membranes and internal organs, and is more likely to occur in immunocompromised animals or those with underlying health conditions. Young calves are more susceptible to the infection.

Transmission

Transmission of camel candidiasis can occur through direct contact with infected animals, ingestion of contaminated feed or water, or exposure to contaminated environments. Conditions like malnutrition, concurrent infections or high stress levels can weaken the camel's immune system and can act as predisposing factors. Warm and humid environments favour the growth of *Candida*. Unsanitary living conditions can promote infection. Skin cuts, abrasions or other injuries can serve as entry points. Damage to the mucous membranes can facilitate infection.

Clinical signs

In prolonged cases, the disease causes itching and uneasiness, and may lead to bleeding and ulceration of skin, leading to weakness and debility of calves. Oral lesions of camel candidiasis often manifest as red, inflamed mucosa covered by white or yellowish plaques on the mucous membranes of the mouth, tongue and gums. These lesions may be painful and lead to difficulty eating and swallowing. In female camels, the lesions may involve the vaginal mucosa and vulva and may cause a white, cheesy discharge from the vulva, along with redness and inflammation of the genital area. Scraping the lesion with a scalpel reveals foul-smelling blackish-brown dry crusts bunched with hairs along with roots.

Lesions

Skin lesions of the disease are initially observed on the back near the hump; later the lesions extend towards the abdomen and may cover the whole body. Lesions are initially round in shape and measure less than 1 cm in size which may enlarge to more than 10 cm and may coalesce. The lesions are hard and fibrous crusts with papules accompanying alopecia.

Diagnosis

Diagnosing camel candidiasis involves a combination of clinical examination, cytological evaluation and fungal culture. Scrapings or swabs of affected areas can be examined microscopically for the presence of yeast cells and hyphae characteristic of Candida infection.

Differential diagnosis

Conditions that may mimic camel candidiasis include other causes of oral lesions (such as bacterial infections, viral infections and traumatic lesions) and skin infections (such as dermatophytosis or bacterial folliculitis and sarcoptic mange). Differential diagnosis may require further diagnostic tests, such as bacterial culture, viral PCR and skin biopsies.

Treatment and control

Treatment

Topical antifungal treatments like Clotrimazole (1 percent) or Miconazole (2 percent) cream may be applied to the lesions twice daily. Systemic antifungal treatments include Fluconazole @ 2.5–5 mg/kg BW administered orally once daily for two to four weeks or Itraconazole @ 5–10 mg/kg BW administered orally once daily for three to six weeks. For oral candidiasis, Nystatin at 100 000–400 000 IU per dose, may be administered orally for two to four times daily. In addition, a balanced diet rich in essential vitamins and minerals should be ensured and be supplemented with probiotics to restore normal gut flora, especially after antibiotic therapy. Management of underlying predisposing factors, such as immune suppression or poor hygiene, is also essential for successful treatment and prevention of recurrence.

Control

Preventing camel candidiasis involves maintaining optimal hygiene practices, minimizing stressors that may compromise the immune system and monitoring camel health regularly. Providing a balanced diet, adequate housing and veterinary care can help support overall health and reduce the risk of Candida overgrowth and infection. Additionally, avoiding unnecessary use of antibiotics and immunosuppressive medications can help preserve the natural microbial balance and prevent dysbiosis.

Biosecurity measures

Infected animals should be isolated from healthy ones to prevent direct contact and minimize transmission. Maintaining good hygiene practices, such as regular cleaning and disinfection of housing facilities and equipment, can help reduce the spread of Candida spores. Minimizing exposure to contaminated feed, water and environments can help prevent the introduction and spread of the infection. Handlers and caretakers should use appropriate PPE, such as gloves and protective clothing, when handling affected animals or cleaning contaminated areas.

Sample collection

Skin swabs or scrapings from the edge of the lesions, milk samples if the udder is affected and genital swabs for genital infections may be examined.

7.2.4 Parasitic diseases

7.2.4.1 Trypanosomosis

Definition and causative agent

Trypanosomosis is an important haemoprotozoan disease of camel caused by *Trypanosoma evansi*, which also infects cattle, buffalo, goat, sheep, pig, horse, donkey, camel and dog. It is commonly known as Surra, which means rotten as the animal becomes highly emaciated during the chronic course of infection. It is characterized by intermittent rise of temperature, anaemia, wasting and cutaneous eruptions.

Transmission

Transmission is essentially mechanical, in which the trypanosomes are transferred from one mammalian host to another by the interrupted feeding habit of biting insects, notably, tabanids and *Stomoxys*.

Clinical signs

The course of trypanosomosis infection in camels may be acute, sub-acute or chronic. Acute infection is characterized by high fever, anaemia, weakness and sudden death. In sub-acute cases, infection leads to case fatality within a few months. Signs of illness appear with intermittent fever (41°C) for approximately a week. The animals appear dull, lustreless and become progressively weaker. Loss of appetite, weight loss, anaemia, pale mucous membranes, petechial or ecchymotic haemorrhages, and oedema on the udder, scrotum and sheath are noticed in camels. Pregnant camels show abortions. All age groups can be infected, but the disease generally starts occurring shortly after weaning. Nervous signs are sometimes observed, such as periodic convulsions. A specific odour of the urine is detected by camel owners, which is sufficient for diagnosing the disease.

Lesions

Necropsy findings and clinical signs are non-specific. In acute fatal cases, extensive petechial haemorrhages of the serosal membranes, especially in the peritoneal cavity, may occur. Lymph adenomegaly and splenomegaly are frequently observed. In chronic cases, swollen lymph nodes, serous atrophy of fat and anaemia are generally seen.

Diagnosis

Diagnosis is based upon clinical evaluation, serological and molecular tests. Clinical evaluation is based on anaemia (pale mucous membrane), intermittent fever and sudden decline in weight bearing capacity. Conventional parasitological examinations are not sensitive but can be used in the field with very little investment in equipment. To increase the sensitivity of the method, a haematocrit centrifuge technique or a buffy coat method can be used. Various serologic tests measure antibody to trypanosomes, but their use is more suitable for herd and area screening rather than for individual diagnosis. The serologic tests include ELISA, immunofluorescence antibody test (IFAT), complement fixation test (CFT) and agglutination tests. Molecular techniques, *viz*., PCR, real-time PCR, loop-mediated isothermal amplification (LAMP) can also be used for detection of low level of infection and species differentiation.

Differential diagnosis

The symptoms may overlap with chronic form of

other haemoprotozoan diseases like theileriosis and babesiosis.

Treatment and control

Treatment

Quinapyramine methyl sulphate (Antrycide) is the most used drug with 5 mg/kg subcutaneously administered for treatment; it confers a short period of prophylaxis. For more and prolonged protection, a modified quinupramine known as 'Antrycide pro-salt' containing slow releasing quinupramine chloride in 3:2 is also available. A new arsenical drug, melarsomine dihydrochloride (cymelarsan) has been developed and is considered as drug of choice for camels. It has the ability to cross the blood-brain barrier and has been recommended for acute and chronic trypanosomosis in camels @ 0.25 mg/kg deep IM.

Control

Disease control is principally based on the use of trypanocides and preventive management methods to protect animals from infection. Flies can be partially controlled by frequent spraying of animals, aerial and ground spraying of insecticides on fly-breeding areas, use of insecticide-impregnated screens and targets, bush clearing, and other habitat removal methods. Drug resistance must be carefully monitored by frequent blood examinations for trypanosomes in treated animals.

Biosecurity measures

The measures include screening of newly introduced camels in the herd. Infected camels should be isolated from healthy ones to prevent direct contact and minimize transmission and treated. Good hygiene practices should be followed, and fly control measures adopted in the farm to minimize contact of camels with potential vectors.

Sample collection

Blood and serum are required for parasitological, serological and molecular tests.

7.2.4.2 Coccidiosis

Definition and causative agent

Gut-dwelling coccidia, mainly *Eimeria* spp., are distributed widely among camel populations. The prevalence rates are high, involving several coccidian species. The main coccidian species affecting camels are *E. dromedarii*, *E. cameli*, *E. pellerdyi*, *E. rajasthani*, *Sarcocystis spp.* and *Isospora spp.* A higher prevalence of coccidiosis is seen in the rainy season and in camel calves below one year of age.

Transmission

Transmission occurs through contaminated feed, fodder and water with sporulated oocysts. Unsporulated oocysts are excreted in faeces. Sporulation occurs rapidly.

Clinical signs

Severe enteritis and diarrhoea in heavy infections.

Lesions

There are marked inflammatory changes in the intestinal mucosa, and disruption of villous architecture with presence of schizonts.

Diagnosis

Diagnosis is based on history, clinical signs, faecal examination and histopathology. During microscopic faecal examination, unsporulated oocysts (25–35 µm long) and sporocysts (20 × 15 µm) containing elongated or ovoid sporozoites are seen.

Differential diagnosis

Coccidiosis in camels is to be differentiated with other gastrointestinal helminthic infections *viz.*, *Haemonchus longistipes, Nematodirrela* spp. and *Nematodirus* spp.

Treatment and control

Treatment

Sulfadimidine may be given orally @ 30 mg/kg BW daily for ten days.

Control

Affected camels must be treated. Good hygiene practices must be adopted to minimize contamination of feed, fodder and water sources.

Biosecurity measures

Avoid overcrowding and follow good hygienic management practices.

Sample collection

Faecal samples are required for examination of parasitic eggs and intestinal tissues are required for mucosal examination by histopathology.

7.2.4.3 Haemonchosis

Definition and causative agent

The disease is caused by nematode *Haemonchus longistipes.*

Transmission

Camels get infected by ingestion of infective L3 larvae of *Haemonchus longistipes* through contaminated feed and water. The life cycle of the parasite involves parasitic eggs which hatch into L1, develop to L2 then L3 in a period of five days, but development may be delayed for weeks or months under cool conditions. The larvae moult twice near the gastric glands and, just before the final moult, they develop the piercing lancet which enables them to obtain blood from the mucosal vessels. As adults, they move freely on the surface of the mucosa.

Clinical signs

H. longistipes worms are voracious blood suckers producing symptoms similar to *H. contortus* in domestic ruminants. Acute haemonchosis is characterized by anaemia, lethargy, dark coloured faeces and variable degrees of oedema, of which the submandibular form and ascites are the most easily recognized. Diarrhoea is generally not a regular feature of *Haemonchus* infection. Chronic haemonchosis is associated with progressive weight loss and weakness. However, severe anaemia and gross oedema are not regularly observed.

Lesions

In cases of acute haemonchosis, numerous small haemorrhagic lesions are observed at the abomasal mucosa during postmortem examination. The abomasal contents are fluid and dark brown due to the presence of altered blood. The carcass is pale and oedematous, and the red marrow is found to have expanded from the epiphyses into the medullary cavity.

Diagnosis

The history and clinical signs are often sufficient for the diagnosis of acute haemonchosis, which can be further validated with faecal worm egg counts. Necropsy examination of abomasum is also useful for diagnosis of haemonchosis. The diagnosis of chronic haemonchosis is more difficult to differentiate from poor plane of nutrition, and confirmation may depend upon the gradual disappearance of the syndrome after anthelmintic treatment.

Differential diagnosis

The disease is to be differentiated with coccidiosis and other gastrointestinal helminthic infections, *viz*., *Nematodirrela* spp. and *Nematodirus* spp.

Treatment and control

Treatment

In cases of acute haemonchosis outbreak, infected camels should be treated with benzimidazoles/ levamisole/ivermectin at recommended doses (see annexure) and immediately moved to a pasture not recently grazed by any other ruminant. When the original pasture is grazed again, prophylactic measures should be undertaken, as enough larvae may have survived to institute a fresh cycle of infection. Chronic haemonchosis is also dealt with in a similar fashion. If possible, the new pasture should have good nutritional value; alternatively, some supplementary feeding may be given.

Control

In the tropics and subtropics, rainfall and temperature permit high pasture levels of *Haemonchus* larvae. It may, therefore, be necessary to use an anthelmintic at intervals of two to four weeks depending upon the eggs per gram (EPG) count of animals. Camels should also be treated at least once before the start of the rainy season to remove persisting hypobiotic larvae, whose development could pose a future threat. For this purpose, one of the benzimidazole drugs or ivermectin is recommended. Fenbendazole may be given @ 5 mg/kg BW given orally. Ivermectin, when given orally @ 0.2 mg/kg BW, is reported to be more effective than equivalent doses given subcutaneously. In some areas where *Haemonchus* is endemic; closantel, which has a residual prophylactic effect, may be used. However, bilateral blindness - as a side effect - may be closely monitored.

Biosecurity measures

Avoid overcrowding and follow good hygiene practices.

Sample collection:

Faecal samples are to be collected for examining unsporulated eggs in the faeces. Abomasal contents are to be collected on postmortem.

7.2.4.4 Other strongyles infections (except *Haemonchus*)

Definition and causative agent

Other strongyle species prevalent in camels includes *Nematodirrela, Nematodirus Trichostrongylus*, *Ostertagia* and *Strongyloides.*

Transmission

Adult strongyles produce eggs that are passed out in the faeces and disseminated into the environment. These eggs then develop into infective larvae (L3) that exist on the pasture vegetation or in stalls and infection is transmitted through contaminated feed or water.

Clinical signs

The symptoms of strongyles infection are anorexia, enteritis and diarrhoea, and anaemia.

Lesions

Lesions take the form of inflammatory changes in the intestinal mucosa causing extensive destruction and tunnelling.

Diagnosis

Diagnosis is based on history, clinical signs, faecal examination and histopathology. Laboratory examination for internal parasites includes faecal examination by direct smear, sedimentation and flotation, as well as faeces EPG counts using a modified McMaster's technique.

Differential diagnosis

The disease is to be differentiated from coccidial infections and Haemonchosis in camels.

Treatment and control

Treatment

Limited information is available on the efficacy of anthelmintics against gastrointestinal nematodes in camels. Benzimidazoles and ivermectin may be given for treatment at recommended dose rates mentioned in the Annexure.

Control

Treatment of affected camels can control the spread. Good hygiene management practices must be adopted to minimize contamination of feed, fodder and water sources.

Biosecurity measures

Overcrowding should be avoided, and good hygiene practices should be followed.

Sample collection

Faecal samples must be collected for antemortem examination. Intestine and intestinal contents are required for postmortem examination.

7.2.4.5 Mange

Definition and causative agent

Mange is one of the most common parasitic skin diseases of camel caused by the mite *Sarcoptes scabiei var. cameli*, having zoonotic importance. It is locally known as Khujli, Khaj, Paanv. The disease is highly contagious and occurs throughout the year but is more severe during the winter months. Stress due to other diseases, overcrowding, poor management and heat are the important predisposing factors. The incidence is observed to be high in adult camels as compared to young camels.

Transmission

The mites can also infect humans through close contact with infected animals while riding camels and cleaning camel sheds and surfaces. The mite can survive outside the camel's body for about one or two weeks, and they propagate in low temperature and high humidity conditions. The fertilized female mite creates a winding burrow or tunnel in the upper layers of the epidermis, feeding on liquid oozing from the damaged tissues. The eggs are laid in these tunnels, hatch in three to five days, and the six-legged larvae crawl on to the skin surface. These larvae, in turn, burrow into the superficial layers of the skin to create small 'moulting pockets' in which they moult to nymph and adult. The adult male then emerges and seeks a female either on the skin surface or in a moulting pocket. After fertilization, the females produce new tunnels, either *de* novo or by extension of the moulting pocket. The entire life cycle is completed in 17–21 days. New hosts are infected by contact, presumably from larvae, which are commonly, present on the skin surface.

Clinical signs

Host reaction starts on the head, neck, mammary glands, prepuce and flanks. The first lesions appear as erythema, papules and intense pruritus with hair loss, which becomes reddened and moist. During the chronic form of disease; the neck, head and flanks are badly affected leading to severe irritation, itching and rubbing. The animals become lethargic and weak, and this is followed by anaemia. This affects the general performance and production potential of camels.

Lesions

The lesions may become generalized with hyperkeratosis on the neck and legs, with intense pruritus leading to loss of appetite, weight loss and emaciation.

Diagnosis

Mites and eggs can be isolated by taking deep skin scrapings from different places. For the ectoparasitic infections, skin-scraping examination is done for identification of mites. For confirmatory diagnosis, skin scraping from different affected sites are to be taken, boiled in 10 percent potassium hydroxide/ water and examined under the microscope for mites, eggs and faecal pellets. The size of the mite may vary from 0.2 to 0.5 millimetre (mm).

Differential diagnosis

Conditions that may mimic camel dermatomycosis, candidiasis and other skin fungal infections include other causes of alopecia and skin lesions, such as bacterial infections and autoimmune skin diseases.

Treatment and control

Treatment

The commonly available acaricides in the market are Amitraz 12.5 percent (500 parts per million or ppm), Deltamethrin (50 ppm), Cypermethrin (700 ppm) and Fenvalerate (500 ppm). Normally the spray is to be repeated after 15 days. Additional spray is also required depending on the severity of the disease. Individual affected animals can also be treated by Ivermectin injection @ 0.2 mg/kg BW administered subcutaneously once a week for four to five weeks. One percent Flumethrin is applied on the vertebral column at 20 ml/100 kg BW.

Control

Timely care and adopting good hygiene practices and treatment will be beneficial in the control of the disease as well as in maintaining the work potential and productivity of camels. Nomadic people generally apply acaricidal drugs mixed with sulphur powder dissolved in mustard oil (*Brassica juncea*) topically once weekly or fortnightly.

Biosecurity measures

Infected animals should be isolated from healthy ones to prevent direct contact and minimize transmission. Maintaining good hygiene practices, such as regular cleaning and disinfection of housing facilities and equipment, can help reduce the spread of mite infestations.

Sample collection

Deep skin scrapings from the edge of the lesion may be examined for the presence of adult mite, larvae, and eggs.

7.2.4.6 Ticks infestation

Definition and causative agent

Hard ticks, *Hyalomma dromedarii* and *Hyalomma anatolicum anatolicum,* are commonly prevalent, and the camel-specific hyalommine species is a desert-adapted two or three host tick. Soft tick, *Ornithodorus savignyi*, also occurs on camel in India. These ticks are responsible for transmission of various haemoprotozoan, rickettsial and viral infections.

Transmission

The presence of adult *Hyalomma* tick is abundant in winter due to the intact hair coat of the camel facilitating lodgement of ticks. Overcrowding of animals in indoor night shelters result in increased contact and spread. In the months preceding winter, the nymph stage of the tick predominates. Larvae and nymphs usually feed on small wild mammals and birds, while adults feed on camels. The adult females suck blood, drop down and lay 4 000–5 000 eggs.

The adult *Ornithodoros* tick female lay batches of 100 eggs in the sand which hatch in about eight days. The larvae that emerge remain quiescent until they have moulted to nymphal stages. Several nymphal instars are passed through and attack hosts for a short period. The adult females suck blood, drop down and lay eggs.

The most frequent attachment sites for adult ticks are the groin, perineum, udder, axilla and facial region, while nymphs are found hidden in areas well-covered by the hair coat, particularly along the back, around the hump and the intermandibular region.

Clinical signs

The signs of tick disease are marked irritation at the site of the tick bite, inflammatory reaction characterized by congestion, oedema, hyperplasia, cellular infiltration around the site of insertion of mouth parts and anaemia.

Lesions

Inflammatory reaction on the skin is noticed at tick bite sites, often resulting in wound formation which are complicated further due to secondary bacterial infection.

Diagnosis

Infestation of ticks can be visually observed at different sites of the camel's body. The ticks should be removed and examined for identification of the tick species based on body morphological features.

Differential diagnosis

Tick bite wounds should be differentiated from injuries or wounds on skin caused by other ectoparasites.

Treatment and control

Treatment

Acaricides, *viz*., deltamethrin @ 25 ppm and Amitraz @ 250 ppm, can be used in two sprays 10 days apart.

Control

Following good hygiene practices and spraying of insecticide in the infected premises, cracks and crevices, and breeding sites of ticks can help control the spread of ticks.

Biosecurity measures

Maintaining good hygiene practices, such as regular cleaning of housing facilities, can help in reducing the tick infestations. Overcrowding of animals should be avoided.

Sample collection

Ticks should be collected from the camel body and the premises they are housed in.

7.2.4.7 Myiasis

Definition and causative agent

Cephalopina titillator (Camel nasal bot fly) is an oestrid fly whose larvae are obligate parasites specifically of the camel. It is widely distributed in camel raising areas, causing nasopharyngeal myiasis.

Transmission

The flies deposit young larvae around the nostrils of the camel, from where they crawl upward and enter the nasal cavity, frontal sinus and pharynx of camel. Different larval stages develop there, and finally full-grown larvae crawl out and pupate on the ground. After three to six weeks the fly emerges. The flies hide in warm corners or crevices in the camel premises.

Clinical signs

The flies cause great annoyance when they attack the camel to deposit larvae around the nostril. The animal become restless and is reluctant to take feed. The larvae irritate the nasal mucosa with their oral hooks and spines, causing secretion of viscid mucous exudate. The symptoms are exhibited based on presence of larvae in the nasal cavity, frontal sinus, pharynx and other parts of the camel.

Lesions

There is inflammatory reaction and damage of tissues in the nasal cavity, frontal sinus and pharynx.

Diagnosis

Diagnosis is based on clinical signs and presence of larvae in the nasal tract.

Differential diagnosis

The disease is to be differentiated from chronic bronchial and pulmonary disease due to bacterial/ viral infections. The larva of *C. titillator* resembles the larva of *Hypoderma* spp.

Treatment and control

Treatment and control

Successful treatment through the use of ivermectin @ 0.2 mg/kg BW administered subcutaneously in clinical cases have been reported.

Control

Following good hygiene practices, spraying of insecticide in the premises and the use of fly repellents can check the spread of the disease.

Biosecurity measures

It is important to maintain good hygiene practices, such as regular cleaning of housing facilities.

Sample collection

Larvae should be collected from the nasal tract of the camel and flies should be collected from the housing premises.

7.3 Non-infectious diseases of camel

7.3.1 Metabolic disease

7.31.1. Pica

Definition and etiology

Pica in camels refers to a behavioural disorder characterized by the consumption of non-food substances, such as soil, sand, stones or other foreign objects. Etiology is diverse and include nutritional deficiencies of certain minerals or nutrients, such as salt or minerals like phosphorus or calcium. Confinement of camels can lead to boredom or stress, prompting camels to engage in abnormal behaviours like pica. Some gastrointestinal conditions, such as gastric ulcers or parasitic infections, may cause discomfort or irritation, leading camels to seek relief by ingesting non-food items. Pica may also be a coping mechanism for camels to alleviate stress or anxiety.

Clinical signs

The most obvious sign of pica in camels is the consumption of non-food items. This behaviour may be observed regularly or intermittently and can vary in severity. Ingestion of indigestible materials like sand or dirt can lead to ruminal distension or impaction. This is manifested as abdominal discomfort, bloating, decreased appetite and changes in defecation patterns. Pica can result in reduced nutrient absorption or displacement of essential feed, leading to weight loss or poor body condition despite adequate food intake.

Prolonged chewing or ingestion of abrasive substances like sand or gravel can cause oral and dental abnormalities such as worn teeth, dental fractures or oral ulcers. Ingestion of large or sharp objects can lead to gastrointestinal obstruction or perforation, resulting in severe abdominal pain, colic, anorexia and potentially life-threatening complications. Pica can lead to secondary to gastrointestinal disorders, parasitic infections, metabolic disorders or neurological abnormalities. Ingestion of foreign objects or abrasive materials like sand can irritate the gastrointestinal tract leading to diarrhoea, colic or other digestive disturbances.

Diagnosis

Diagnosis is based on unusual ingestion patterns by the affected camel, examining oral cavity and gastrointestinal tract for signs of abrasions, inflammation or foreign material. Blood tests may be performed to assess for nutritional deficiencies or underlying health conditions contributing to the pica behaviour.

Faecal samples need to be checked for parasitic infections and presence of undigested material. Feed samples are analysed to assess the nutrient content and to identify any deficiencies or imbalances. Soil samples need to be checked for contaminants or substances that might attract the camel to consume soil. Blood samples should be collected for haematological and biochemical parameters studies.

Differential diagnosis

This includes gastrointestinal disorders causing discomfort or pain, nutritional deficiencies and behavioural issues related to stress or boredom.

Treatment and control

Treatment

Underlying health conditions, such as gastrointestinal disorders or nutritional deficiencies, should be identified and addressed. Camels should be provided opportunities for mental and physical stimulation to reduce boredom and stress. Camels must have access to a balanced diet, including adequate minerals and nutrients, to minimize the likelihood of nutritional deficiencies. Positive reinforcement training techniques must be employed to discourage pica behaviour and encourage more appropriate behaviours. Measures such as fencing off areas with potentially harmful objects should be taken to prevent camels getting access to non-nutritive substances. This is necessary to monitor any complications arising from pica behaviour.

Control

Pica can be controlled through proper nutrition, environmental management and regular veterinary care.

7.3.2 Nutritional deficiency disease

7.3.2.1 Vitamin A deficiency

Definition and etiology

Vitamin A deficiency is caused by inadequate intake or absorption of vitamin A, an essential fat-soluble vitamin necessary for various physiological processes in the body. Etiological factors include insufficient dietary intake of preformed vitamin A (retinol) or provitamin A carotenoids, which are converted into vitamin A in the body. Conditions affecting the absorption of dietary fat or bile acids, such as liver disorders or intestinal malabsorption syndromes, can impair the absorption of vitamin A. Liver damage or dysfunction can impair the storage

and conversion of vitamin A into its active forms, leading to deficiency. Feeding camels low-quality or degraded forage with reduced vitamin A content can contribute to deficiency. Seasonal changes in forage availability or quality can affect the vitamin A intake of camels, particularly in arid regions with limited vegetation.

Clinical signs

Affected camels may exhibit signs of xerophthalmia (dryness), inflammation and ulceration of the cornea and conjunctiva, night blindness (nyctalopia) or impaired vision in low-light conditions and, later, complete blindness. Vitamin A deficiency can show up as rough, dry or scaly skin, Hyperkeratosis particularly on the elbows, knees and muzzle, and hair loss or thinning of the coat. The deficiency can result in infertility or reproductive failure in females, and reduced libido and sperm quality in males. Affected camels have heightened susceptibility to infections due to impaired immune function. Vitamin A deficiency slows growth and development, particularly in young camels.

Diagnosis

Observation of characteristic signs such as ocular abnormalities, skin changes and reproductive disturbances can raise suspicion of vitamin A deficiency. Measurement of serum retinol levels can help confirm vitamin A deficiency. Low serum retinol concentrations indicate deficiency. Evaluation of ocular health and visual function, including assessment for xerophthalmia, can aid in diagnosis. Feed and fodder may be analysed to assess the vitamin A content.

Differential diagnosis

Differential diagnoses include other nutritional deficiencies, such as vitamin E or selenium deficiency, infectious diseases affecting the eyes or skin and environmental factors contributing to ocular or skin abnormalities.

Treatment

Camels should be provided with vitamin A supplements orally or by injection to correct the deficiency. Camels must have access to a balanced diet containing adequate sources of vitamin A, such as green forage, fresh grass and vitamin A-rich supplements. Response to the treatment should be monitored and supportive care provided as needed, especially for ocular or reproductive complications.

7.4 Systemic diseases of camel

7.4.1 Diseases of respiratory system

7.4.1.1 Camel pneumonia

Definition and etiology

Camel pneumonia is characterized by inflammation of the lung tissue. It can be caused by the following infectious agents:

Bacteria: Common bacterial pathogens associated with camel pneumonia include *Mannheimia haemolytica*, *Pasteurella multocida*, *Streptococcus* spp. and *Escherichia coli*.

Viruses: Viral infections such as camel respiratory coronavirus and camel influenza virus can also lead to pneumonia.

Fungi: Fungal pneumonia can occur, though it is less common, and may include *Aspergillus* spp. and *Cryptococcus* spp.

In addition, camel pneumonia can be caused by non-infectious factors such as environmental irritants – direct contact with infected animals, as well as through respiratory droplets and aerosols. It can also be transmitted indirectly through contaminated feed, water or fomites.

Clinical signs

Persistent or intermittent coughing is one of the characteristic signs of pneumonia. Camels with pneumonia often exhibit nasal discharge, which can vary from clear and watery to thick and purulent (containing pus). Tachypnoea (rapid breathing) is commonly observed, and the animal may exhibit shallow or laboured breathing, especially during exertion or stress. Dyspnoea (difficulty in breathing) may be present, and is manifested as open-mouth breathing, flared nostrils or increased use of accessory respiratory muscles. Abnormal lung sounds such as crackles or wheezes may be auscultated upon listening to the chest with a stethoscope. These sounds indicate the presence of fluid or mucus in the airways.

Systemic signs like elevated body temperature are common signs of pneumonia. However, in some chronic and severe cases, the temperature may be normal or even subnormal. Camels with pneumonia may appear lethargic, depressed or reluctant to move. They may isolate themselves from the herd

and exhibit reduced appetite or interest in their surroundings. Chronic or severe pneumonia can lead to weight loss and dehydration, especially if the animal has an accompanying fever or is reluctant to drink water. In addition to difficulty in breathing, camels with pneumonia may exhibit nasal flaring, which is an attempt to increase airflow through the nostrils to compensate for respiratory distress.

Physical examination may reveal cyanosis (bluish discoloration of the mucous membranes) in severe cases of pneumonia, indicating hypoxemia (low oxygen levels in the blood).

Lesions

Lung congestion occurs when blood vessels within the lungs become dilated and engorged with blood, leading to reddening of the affected tissue. Accumulation of exudative fluid within the air spaces and interstitial tissue of the lungs is also evident. This can result in the formation of fibrinous adhesions between the lung and the chest wall. Severe or chronic pneumonia may result in necrosis (tissue death) of lung parenchyma, leading to the formation of cavities or cystic lesions within the lung tissue.

Diagnosis

Abnormal lung sounds become evident during auscultation of the chest. Radiography can reveal areas of consolidation or inflammation in the lungs. Swabs from the nasal passages and tracheal washes from the lower respiratory tract are to be examined. Blood samples can be collected for complete blood count (CBC), blood cultures and serological tests to detect the specific pathogens. If there is pleural effusion, a thoracocentesis can be performed to collect pleural fluid. CBC and serum biochemistry may show signs of infection or inflammation. Differential diagnoses for camel pneumonia may include other respiratory conditions such as bronchitis, pleuropneumonia, lung abscess and inhalation of irritants.

Treatment and control

Treatment

Ceftiofur @ 2.2 to 4.4 mg/kg can be given IV/ IM at 12 to 24 hours intervals for five to six days. Other broad-spectrum antibiotics like cefquinome can also be given initially until specific pathogens are identified. Supportive care like rest, adequate nutrition and hydration are also crucial for early recovery. NSAIDs may be used to reduce inflammation and alleviate discomfort.

Control

Strict quarantine protocols must be implemented for new animals. Good hygiene and sanitation measures must be practised to prevent the spread of infectious agents. Contact between healthy and sick animals must be limited. Vectors such as insects and rodents must be controlled. Vaccines against common respiratory pathogens such as Pasteurellosis may be available and can help reduce the risk of pneumonia. Proper ventilation, clean housing, and regular monitoring can help prevent the spread of infectious agents and reduce the likelihood of pneumonia outbreaks.

7.4.2 Diseases of the digestive system

7.4.2.1. Indigestion

Definition and etiology

Abrupt changes in diet, especially transitioning from a high-fibre diet to the one high in grains or vice versa, can upset a camel's digestive system. Camels may ingest foreign objects, which can cause blockages or irritation in the digestive tract. Infections by bacteria, viruses or parasites can lead to indigestion, causing symptoms like diarrhoea or constipation. Camels, especially when food is abundant, may overeat which might lead to indigestion. Environmental stressors like transportation and changes in climate can also affect a camel's digestive health.

Clinical signs

Common clinical signs are decreased appetite, signs of abdominal discomfort like pawing at the ground, rolling or stretching. Diarrhoea, constipation or unusual consistency or colour in faeces are other signs. Colic is manifested by signs of abdominal pain, restlessness, and frequent lying down and getting up. Indigestion can lead to poor nutrient absorption and subsequent weight loss and lethargy.

Diagnosis

Fresh faecal samples taken directly from the ground or rectally using a sterile glove should be examined for parasites, bacteria and undigested feed particles. Rumen fluid analysis can reveal pH, microbial populations and fermentation products. Blood tests can help assess overall health, liver function and

metabolic status. Urine samples analysis can provide information on kidney function and hydration status. Feed samples may be examined to assess nutrient content and quality.

Treatment

Dietary management includes adjusting the camel's diet to include more fibre and gradual introduction of new foods. In cases of dehydration, the animal must be rehydrated through intravenous or oral fluids. Depending on the cause, medications such as antacids, probiotics, antibiotics, anthelmintics, anti-inflammatory drugs, prokinetic agents and analgesics can be given. Stressors in the camel's environment must be minimized and the animal must be provided a calm atmosphere. Light exercise should be encouraged to help stimulate digestion and alleviate discomfort. Prompt recognition and management of indigestion in camels is essential to prevent complications and ensure the animal's well-being. Regular monitoring of feeding, behaviour and overall health aids in early detection and intervention.

7.4.2.2 Bloat/tympany

Definition and etiology

Tympany is a condition characterized by the accumulation of gas in the digestive system, leading to distension of the abdomen. It can be caused by several factors and may be presented as either frothy bloat or free gas bloat.

Dietary factors like rapid consumption of lush, high-moisture forage or feeds rich in fermentable carbohydrates can contribute to bloat. Certain legumes, such as alfalfa, can also predispose camels to bloat due to their high protein and soluble carbohydrate content. Microbial fermentation in the forestomaches (rumen and reticulum) produces gasses such as methane and carbon dioxide. When fermentation processes are disrupted or imbalanced, excessive gas production takes place which will lead to bloat. Physical obstruction or blockages in the digestive tract, such as foreign objects or impacted ingesta, can interfere with normal gas expulsion, resulting in bloat. Ingestion of toxic plants can disrupt normal digestive processes and contribute to bloat. Temperature and climatic factors, including temperature extremes and changes in weather conditions, can influence microbial activity in the digestive tract and predispose camels to bloat.

There are two types of bloats:

Frothy bloat: Also known as primary or dietary bloat, this occurs when stable foam forms in the rumen, preventing the escape of gas. This type of bloat is often associated with the consumption of rapidly fermentable feeds.

Free gas bloat: Also called secondary or obstructive bloat, this occurs when gas accumulates in the rumen and cannot be expelled due to physical obstruction or other factors inhibiting eructation (belching).

Clinical signs

Signs of abdominal distension and discomfort such as restlessness, pawing at the ground, or frequent lying down and getting up may be recorded. In severe cases, bloat may compress the diaphragm and interfere with normal breathing, leading to respiratory distress. Camels may show a decreased interest in feed due to abdominal discomfort. Weakness and lethargy is observed, particularly if it is accompanied by dehydration or other systemic effects. Excessive drooling or frothing at the mouth may occur, especially in cases of frothy bloat.

Diagnosis

Diagnosis is done by examination of rumen fluid for motility, pH and microbial counts. Blood samples may be examined for CBC and to check for signs of infection or inflammation. Serum samples can be analysed for kidney and liver function, electrolyte balance and overall metabolic status. Faecal sampling can be done for parasitological examination and microbial culture. Urinalysis should be conducted to assess hydration status, presence of ketones, glucose or any signs of urinary tract infection.

Treatment and control

Treatment

Passage of a stomach tube or use of a trocar and cannula to release trapped gas from the rumen can provide immediate relief in severe cases of bloat. Dietary management, including removal of offending feed or forage and providing access to dry, low-moisture forage, can help alleviate bloat and prevent recurrence. Fluid therapy may be necessary, especially if dehydration has occurred. Gentle exercise can help stimulate rumen motility and aid in the expulsion of gas. Medication, including anti-

foaming agents, or oral bloat treatments containing surfactants or vegetable oils may be administered to help break down frothy foam in the rumen. Surgical intervention in cases of severe or recurrent bloat and surgical procedures such as rumenotomy may be necessary to remove obstructions or address underlying issues contributing to bloat.

Control

Close monitoring of the camel's condition, along with supportive care such as warmth and comfort, is essential during recovery from bloat. Prompt recognition and treatment of bloat are crucial to prevent complications such as respiratory compromise, circulatory shock or rumen acidosis. Additionally, preventive measures such as gradual dietary transitions, proper forage management and avoidance of toxic plants can help reduce the risk of bloat occurrence.

7.4.2.3. Impaction

Definition and etiology

Impaction in camels refers to a condition where the normal movement of ingesta through the digestive tract is obstructed, leading to the accumulation of dry or hardened faecal material. This can occur in various parts of the digestive system, including the stomach, intestines or colon. Impaction can be caused by several factors and may manifest with a range of symptoms.

Consumption of low-fibre diets or inadequate water intake can contribute to the formation of dry, compacted faecal material, predisposing camels to impaction. Limited physical activity or confinement to small enclosures can reduce gastrointestinal motility and contribute to the development of impaction. Ingestion of foreign objects, such as stones, sand or fibrous material, can obstruct the digestive tract and lead to impaction. Dental issues that interfere with chewing or grinding food properly can result in inadequate breakdown of feed material, increasing the risk of impaction.

Clinical signs

Camels may exhibit signs of constipation or pass small, dry faecal pellets infrequently. Signs of abdominal pain or discomfort, such as restlessness, pawing at the ground or lying down and getting up repeatedly also indicate impaction. Impacted camels may show a reduced interest in food or refuse to eat altogether. Swelling or bloating of the abdomen may be observed, particularly in cases where impaction affects a large portion of the digestive tract. Impacted camels may appear weak, lethargic or depressed. Straining during attempts to defecate, with little or no faecal output, may indicate obstruction in the digestive tract. Signs of colic, such as rolling, stretching or kicking at the abdomen, may be present in severe cases of impaction.

Diagnosis

Faecal sampling may be done for parasitological examination, microbial culture and to assess the composition and type of fibre to determine if it is contributing to the impaction. Blood sampling may be done for CBC, biochemical profile to assess kidney and liver function, electrolyte balance and overall metabolic status. Elevated levels of packed cell volume (PCV) and total protein may indicate dehydration. Rumen fluid may be examined for pH measurement and to assess the motility and population of protozoa and bacteria. Urinalysis must be carried out to assess hydration status, presence of ketones, glucose or any signs of urinary tract infection.

Treatment and control

Treatment

Rehydration with intravenous or oral fluids is essential to address dehydration and soften impacted faecal material, making it easier to pass. Providing access to fresh water and high-fibre forage can help promote normal bowel movements and prevent further impaction. Administration of laxative medications or enemas may be necessary to help soften and evacuate impacted faecal material. Mustard oil – 1.5 to 2.0 litres – may be given once or may be repeated after 48 hours. Analgesic medications may be used to alleviate abdominal discomfort and improve the camel's comfort during recovery. In cases of severe or persistent impaction, surgical procedures such as enterotomy (intestinal incision) may be required to remove obstructive material and restore normal gastrointestinal function. Rectal enema using soap water may be helpful, particularly in calves. Chances of peritonitis should be ruled out in cases of impaction. Blood examination may be helpful in deciding the need for antibiotics. Providing supportive care, including warmth and comfort, and monitoring of vital signs

is important for the overall well-being of the camel during treatment.

Control

Early recognition and intervention are crucial for successful management of impaction in camels. Veterinarian consultation and diagnostic evaluation may be necessary to determine the extent of impaction and develop an appropriate treatment plan tailored to the individual animal's needs.

7.4.2.4 Enteritis

Definition and etiology

Enteritis in camels refers to inflammation of the intestines, particularly the small intestine, which can have various causes ranging from infectious agents to dietary indiscretion. Enteritis, if not promptly diagnosed and treated, can lead to severe gastrointestinal symptoms and potentially life-threatening complications. Different etiological factors include infectious agents like bacteria, viruses and parasites. Common pathogens include *Escherichia coli*, *Salmonella* spp., Rotavirus, Coronavirus and various species of intestinal parasites. Dietary factors include abrupt changes in diet, ingestion of spoiled or contaminated food or water, consumption of toxic plants or exposure to mycotoxins – these can irritate the intestinal lining and trigger enteritis. Environmental stressors such as transportation, overcrowding, extreme temperatures or social disturbances within a herd can weaken the camel's immune system and predispose it to enteric infections. Underlying conditions such as inflammatory bowel disease, gastrointestinal neoplasia or metabolic disorders can contribute to chronic or recurrent enteritis in camels. Ingestion of chemicals, medications or toxic substances can cause irritation and inflammation of the intestinal mucosa, leading to enteritis.

Clinical signs

Watery or mucoid stools, possibly containing blood or mucus, are common symptoms of enteritis. Diarrhoea may be accompanied by an increased frequency of defecation. Signs of abdominal discomfort, such as pawing at the ground, stretching or lying down and getting up repeatedly, are also observed among affected camels. Fluid loss through diarrhoea can lead to dehydration, which may manifest as sunken eyes, dry mucous membranes, lethargy and reduced skin elasticity. Loss of appetite or refusal to eat is a common clinical sign in camels with enteritis. Elevated body temperature is often observed in affected camels, reflecting the body's inflammatory response to infection or inflammation. Enteritis can cause generalized weakness, lethargy and depression in affected camels, leading to decreased activity levels. Chronic or severe enteritis can result in weight loss due to impaired nutrient absorption.

Diagnosis

Faecal sampling is to be done for parasitological examination and microbiological culture to identify viral agents like rotavirus or coronavirus, and to detect protozoa like coccidia. Blood sampling for CBC, biochemical profile and to detect antibodies against specific pathogens may be done. Rumen fluid may be collected for pH measurement and to assess the motility and population of protozoa and bacteria. Abdominal ultrasound or radiography may be performed to evaluate intestinal motility, detect abnormalities or assess for signs of intestinal obstruction or perforation. Endoscopic examination of the intestinal mucosa allows direct visualization of the gastrointestinal tract and collection of biopsy samples for histopathological evaluation.

Treatment and control

Treatment

Rehydration with intravenous or oral fluids is crucial to correct dehydration and electrolyte imbalances resulting from diarrhoea. Providing easily digestible, highly palatable feed or specialized liquid diets can help maintain nutritional support and energy intake during recovery. Antibiotics or antiparasitic medications may be prescribed to target specific infectious agents identified in faecal samples or, if suspected, based on clinical signs where confirmatory diagnostic services are not available. NSAIDs or corticosteroids may be used to reduce intestinal inflammation and alleviate pain associated with enteritis. Flunixin meglumine can be given @ 1.1 mg/kg BW IM once a day for three to four days. Symptomatic treatment to control diarrhoea, such as anti-diarrhoeal agents or adsorbents, may be administered to manage clinical signs and improve faecal consistency. Supportive care to provide a quiet, stress-free environment and monitoring the camel's hydration status, appetite, and faecal output are essential components of supportive care

during treatment. Surgical intervention is needed in severe cases of enteritis complicated by intestinal obstruction, perforation or unresponsive medical therapy.

Control

Good hygiene practices for maintaining clean feeding and watering areas, proper sanitation and regular removal of faeces can help minimize the risk of enteric infections. Dietary management by providing a balanced diet, avoiding abrupt changes in feed and ensuring access to clean, fresh water can help support optimal gastrointestinal health in camels. Vaccination against common enteric pathogens, such as Salmonella or Rotavirus, may be recommended in regions where these diseases are prevalent. Minimizing stressors in the camel's environment, such as overcrowding, transportation or sudden changes in routine, can help bolster the camel's immune system and reduce susceptibility to enteritis. Implementing regular deworming protocols and pasture management practices can help control gastrointestinal parasites and reduce the risk of parasitic enteritis.

Annexures

Dose rate of common drugs used in camel

Pharmacokinetics of different drugs in dromedary camel has not been widely investigated so far. Fixing standard dose rates for a given drug in camel is a difficult task due to several limitations. The volume of distribution for a given drug varies largely with the hydration status of the camel. In literature, one may find different studies where the volume of distribution at steady state differed by up to 100 percent. Hence, the dose and frequency of drug administration, particularly those eliminated largely through urine – like penicillins and aminoglycosides – should be adjusted according to the hydration status of the animal. Whenever toxicity is suspected, fluid therapy should be preferred. Use of drugs with high margin of safety should be preferred to avoid chances of drug toxicity. Nevertheless, higher doses are generally recommended to avoid sub-therapeutic drug concentrations. In general, antibiotics have longer elimination in camels than in other domestic ruminants, perhaps due to lower rate of urine production. Thus, the therapeutic effect of a drug is longer than that in other domestic ruminants. Calculation of total dose to be given under field conditions is often empirical as facilities for determination of accurate body weight are seldom available.

Table 1: Drugs used for treating camel trypanosomosis

Drug	Dose rate	Remarks
Quinapyramine methylsulphate	@3 to 5 mg/kg BW SC	Weak and dehydrated animals may show transient salivation, muscle tremors, stiffness and depression. Has nephrotoxic effects. Curative. Resistance to this drug is a common problem.
Quinapyramine chloride/ methyl sulphate	@5 to 8.3 mg/kg BW SC	Use with caution in weak/dehydrated animals Curative as well as prophylactic.
Cymelarsen	@0.25 mg/kg deep IM	Curative. Effective in cases resistant to Quinapyramine
Suramin	@12 mg/kg BW IV	Leakage of drug into soft tissues while injecting may cause severe irritation and abscess formation. Curative as well as prophylactic effects (for three weeks)
Isometamidium chloride	@0.5 mg/kg BW IV as 2 percent solution	Leakage of drug into soft tissues can cause severe irritation and abscess formation.

Table 2: Drugs used for treatment of fungal diseases in camel

Drug	Dose rate	Remarks
Whitfield's ointment	Topical, twice weekly for two to three weeks	More suitable for localized small lesions.
Lugol's iodine solution	Topical, twice weekly	
Agricultural Bordeaux mixture	Topical, once daily for five days, thereafter weekly	
0.5 percent lime sulphur	Topical, twice weekly	
1:300 Captan (or two tablespoonfuls in 2 litres water)	Topical, twice weekly for two to three weeks	Captan powder is an irritant when applied as solution.
0.5 percent Sodium hypochlorite/0.5 percent Chlorhexidine	Topical, twice weekly	
10 percent sodium iodide at 1 g/14 kg BW	IV, twice weekly at one week interval	Use with care in weak animals.

Table 3. Antiparasitic and acaricidal drugs used in camel

Drug	Dosage	Remarks
Oxfendazole	@4.5 mg/kg BW orally once	Used for gastrointestinal nematodes.
Fenbendazole	@5 mg/ kg BW orally once	Safe in pregnant animals.
Albendazole	@5–7.5 mg/kg BW orally once	
Morantel	@7.5 mg/kg BW orally once	
Thiabendazole	@40–100 mg/kg BW orally once	
Triclabendazole	@10 mg/kg BW orally once	
Ivermectin	@0.2 mg/kg BW SC once	Effective against GI nematodes and mange mite. Adverse reaction: salivation, drooping of the lower lip.
Levamisole	@7.5 mg/kg BW orally @6 mg/kg SC	Oral: as anthelmintic SC: as immunomodulator.

Table 4. Sedatives and analgesics used in camel

Drug	Dose rate	Comments
Propionylpromazine	@0.5 to 1.0 mg/kg BW IM	Used as premedication in epidural anaesthesia with Lidocaine
Xylazine	@0.25 mg/kg BW IM, IV	Used for sedation and muscle relaxation. Effect starts after 10 to 20 minutes and last for 90 min to 8 hours.
Chlorpromazine	@3 mg/kg BW IM	Used for sedation.
Doxapram	@0.05 to 0.13 mg/kg BW IM	Used as antidote of Xylazine.
Ketamine HCL	@2.5 to 5.5 mg/kg BW IM or IV	Used as sedative. Can be used as anaesthetic in combination with Xylazine.
Triflupromazine	@2 mg/kg BW IM	Used as sedative. Effect is evident after half an hour and may last for up to two hours.

Table 5. Antimicrobials used in camel

Drug	Dose rate	Remarks
Penicillin G Na	@22 000-44 000 IU/ kg BW every 6–12 hours IV/ IM/ SC	
Procaine Penicillin G	@10 000–20 000 IU/ kg BW every 6–12 hours IV/ IM/ SC	
Streptomycin	@10 to 25 mg/kg BW IM every 24 hours	Effective against Actinobacillosis and Actinomycosis.
Ampicillin	@10 to 20 mg/kg BW IV/IM/SC every 8–12 hours	
Ceftiofur	@2.2 to 4.4 mg/kg BW IV/IM/SC every 12–24 hours	In neonates up to 8 mg/kg.
Amikacin	@20 mg/kg BW IV/IM/SC every 24 hours	
Gentamicin	@4.4 to 6.6 mg/kg BW IV/IM/SC every 24 hours	
Tobramycin	@1.3 to 2.5 mg/kg BW IM every 12 hours	
Kanamycin	@6.0 to 8.5 mg/kg BW IM every 12 hours	
Enrofloxacin	@2.5 to 5.0 mg/kg BW IV/IM/SC every 12–24 hours @7.5 mg/kg BW SC every 72 hours (long-acting preparation) @10 mg/kg BW orally every 24 hours	Useful in mastitis.
Norfloxacin	10 mg/kg BW IM	Rapid passage in milk, can be used in mastitis.
Marbofloxacin	@8 mg/kg BW every 24 hours	Broad spectrum. Effective against *Pasteurella* spp. and Mycoplasma.
Oxytetracycline	@5 to 10 mg/kg BW IV every 12–24 hours	High dose (up to 20 mg/kg): every 24 hours. Low dose: every 12 hours. Very useful antibiotic.
Oxytetracycline long acting	@10 to 20 mg/kg BW IM/SC every 24–72 hours	
Tylosin	@4 to 10 mg/kg BW IV/ IM	
Cefquinome	@1 mg/kg BW IM/IV/SC	

Drug	Dose rate	Remarks
Florfenicol	@10 mg/kg BW IM every 48 hours @20 mg/kg BW SC every fourth day	
Lincomycin	@22 mg/kg BW IM every 12–24 hours	Effective in purulent abscess.
Marbofloxacin	@2 mg/kg BW IM/SC every 24 hours	
Neomycin	@15 mg/kg BW PO once daily	Treatment of diarrhoea in calf.
Toltrazuril	@20 mg/kg BW once	Effective in coccidiosis.
Trimethoprim-sulfadoxine	@30 mg/kg BW once daily for three days	Used in coccidiosis.
Amprolium hydrochloride	@10–15 mg/kg BW orally once daily for three to five days	
Metronidazole	@20 mg/kg BW every 24 hours IV	Used in clostridial enteritis.
Sulfadimidine	@30 mg/kg BW orally once daily for 10 days	Used in coccidial infections.

Table 6. Non-steroidal anti-inflammatory drugs (NSAIDs) used in camel

Drug	Dose rate	Remarks
Meloxicam	@0.5 mg/kg BW orally every 72 hours @0.5 mg/kg BW SC/IV every 24 hours	Effective in lameness and musculoskeletal pain.
Flunixin meglumine	@1.1 mg/kg BW IV every six hours @1.1–2.2 mg/kg BW IM every 24 hours	
Phenylbutazone	@4.4 mg/kg BW IV/IM every 24 hours maximum up to five days	
Ketoprofen	@2.2 mg/kg BW IM/ IV every 24 hours	
Butorphanol	@0.5 mg/kg BW SC every 24 hours 0.1 mg/kg BW IV	
Tolfenamic acid	@4 mg/kg BW IV every 24 hours	

Table 7. Hormones and vitamins used in camel

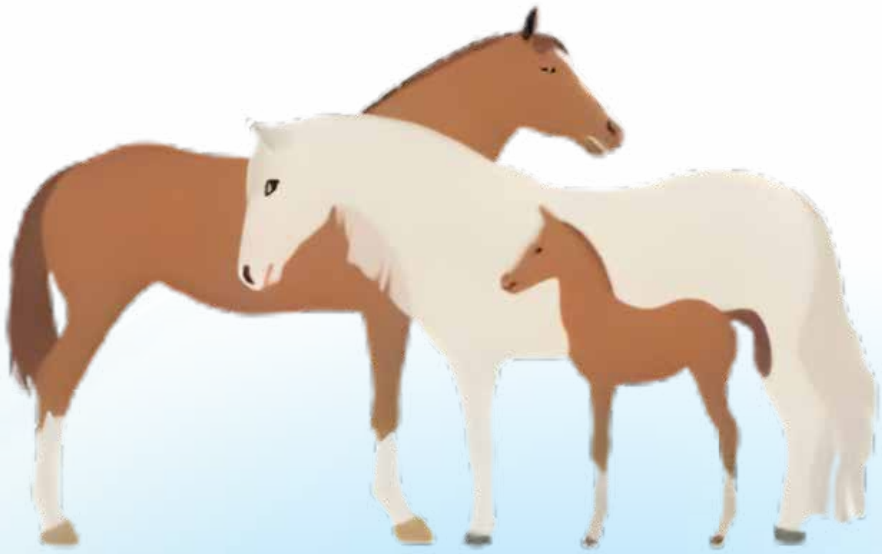
Drug	Dose rate	Remarks
Oxytocin	@10–20 IU IM/SC (up to 50 IU SC total)	To induce parturition. As ecbolic.
Vitamin D	@1000 IU IM once	In calf or pregnant animals for prevention of rickets
Vitamin A	@30 000 to 50 000 IU IM	Particularly indicated for pregnant camels.
PMSG (FSH like action) (To increase conception rate)	@1 000 to 2 000 IU IM once daily for two to three days	Go for mating three to five days after treatment.
PGF2 alpha	@30 mg total IM	Indications: dystocia, retained placenta etc.
Cloprostenol	@500 µg IM total once	As ecbolic

Table 8. Drugs contraindicated in camel

Drug	Comments
Diaminazine aceturate	Toxic. Toxicity symptoms include hyperaesthesia, salivation, convulsions, frequent urination and defecation, itching and sweating. Hepatic and renal damage may occur particularly at higher doses.
Salinomycin/Lasalocid/Monensin/Narasin	Highly toxic to camel.
Isoniazid	Not effective and even toxic.
Sulphonamides and aminoglycoside antibiotics	Use in lower diseases in dehydrated camels.

CHAPTER- 8

GUIDELINES FOR EQUINE DISEASES



8.1 Preamble

Diseases in equines represent a significant concern in the management of horse health, as they can lead to widespread morbidity, mortality, and substantial economic losses. As equines are often involved in racing, showing, transportation and activities that require close interaction, hence, risk of infectious disease outbreaks is heightened, necessitating vigilant management practices. Research and advances in veterinary medicine are to play a vital role in combating these threats by offering new vaccines, diagnostic tools, and treatments. However, ongoing challenges come from the global movement of horses and varying prevalence of different viral diseases across regions. Thus, awareness and education about the specific diseases that can affect equines are essential for horse owners, veterinarians, and all those involved in equine care.

Viral and bacterial diseases are caused by a variety of viruses and bacterial pathogens, respectively, that can affect the respiratory, neurological, reproductive systems and other systems in equines. Some of these infections can spread rapidly through populations of horses, often with severe consequences, while others may persist in a chronic form, causing long-term health issues and decreased performance. Protozoan diseases caused by protozoa, often target the blood, muscles, and nervous system of horses. Among the most significant protozoan diseases in equines are equine piroplasmosis and equine trypanosomosis. Parasitic infestations in horses are caused by a variety of internal and external parasites. Parasitic infections can lead to mild discomfort and poor coat condition to severe colic, anaemia, weight loss, and even death. Non-infectious diseases in horses are not caused by pathogens but due to genetics, environment, nutrition, and mechanical injuries. These diseases can affect various systems in the horse's body, including metabolic disorders, musculoskeletal issues, and respiratory conditions.

Effective control and prevention of diseases in equines require a multifaceted approach, including vaccination, biosecurity measures, regular monitoring, and prompt intervention when outbreaks occur. Moreover, understanding the transmission routes - whether through direct contact, vectors, or contaminated equipment - is crucial for implementing appropriate preventive strategies. The management of bacterial diseases in equines involves a combination of good hygiene practices, proper management, appropriate use of antibiotics, and preventive measures such as vaccination. The emergence of antibiotic resistance also underscores the need for a judicious use of antimicrobials and adherence to veterinary guidance in treating bacterial infections. Control and prevention of protozoan diseases in equines are challenging due to the complex life cycles of the causative agents and their modes of transmission, which often involves vectors or intermediate hosts. Effective management requires a combination of measures, including vector control, regular health monitoring, prompt diagnosis, and appropriate treatment with anti-protozoal medications.

Effective management of parasitic infestations involves regular deworming, pasture management, and vigilant monitoring for signs of infection. Maintaining proper hygiene, implementing strategic deworming programs, and ensuring regular veterinary care are essential in preventing and controlling parasitic infestations in horses, thereby, safeguarding their health and ensuring optimal performance. Managing non-infectious diseases often involves preventive care, such as proper nutrition, regular exercise, and minimizing stress, as well as early detection and appropriate medical intervention to maintain the horse's health and performance.

Correct sample collection and handling are essential for identifying the specific pathogen affecting the horse and for guiding appropriate treatment strategies. Use appropriate sterile techniques to avoid contamination. Samples should be handled and stored correctly depending on the type of analysis required.

Understanding and managing equine diseases is crucial for maintaining the health and performance of horses, as well as ensuring the safety and well-being of all involved in their care and use. Ongoing research into equine diseases aims to improve understanding, develop better diagnostic tools, and create more effective treatments and vaccines.

8.2 Equine Viral Diseases

8.2.1 Equine influenza

Definition and Causative Agent

Equine influenza, a highly infectious respiratory disease, is caused by influenza A viruses, primarily

subtypes H7N7 and H3N8. The currently circulating strain of equine influenza in the country is H3N8 (clade 2 Florida lineage).

Transmission

Equine influenza spreads directly through inhalation of infective respiratory secretions and indirectly via fomites such as clothing, hands, or shared water. In naïve horses, shedding typically lasts for around 7-10 days post-infection. Epidemics often occur when acutely infected horses are introduced to susceptible groups. Vaccinated horses can shed the virus sub-clinically.

Clinical Signs

Equine influenza typically presents with high fever (41.1°C), accompanied by depression, anorexia, and weakness. Nasal discharge starts as serous and may become mucopurulent, if a secondary bacterial infection develops. Lymphadenopathy may be evident as slight swelling in the submandibular or retropharyngeal areas. Horses commonly exhibit a dry, harsh, and nonproductive cough. The incubation period ranges from 1-3 days, with clinical signs appearing 3-5 days after exposure. In uncomplicated cases, symptoms last less than 3 days; recovery can take 2-3 weeks for mildly affected horses and up to 6 months for severely affected ones. Secondary bacterial infections can cause pneumonia, pleuropneumonia, and chronic bronchitis.

Diagnosis

Diagnosis of equine influenza virus infection includes RT-PCR on nasopharyngeal swabs collected approximately 1-2 days after the onset of illness. Antigen-capture ELISA and serologic testing on paired serum samples are the other additional diagnostic methods, which should be taken shortly after appearance of clinical signs and again after about 2 weeks. Virus isolation on SPF embryonated chicken eggs can also be used.

Treatment

There is no specific treatment for equine influenza virus infection. A minimum of three weeks of rest, along with supportive care, is recommended for recovery. Supportive care may include use of anti-bacterial drugs for secondary bacterial pneumonia (*refer* Annexure - I) and non-steroidal anti-inflammatory drugs (NSAIDs).

Control

Currently, vaccination is not practiced in India. The updated vaccines using indigenous strains were developed but are not commercially available, and vaccination provides short-lived immunity with the potential need for multiple boosters.

Biosecurity

Controlling equine influenza virus infection involves enforcing biosecurity measures like isolating newly introduced horses for 2 weeks - during outbreaks; isolating sick horses following biosecurity guidelines for 21 days after resolution of signs in the last newly infected horse; controlling environmental contamination as the virus can survive ~2-3 days on fomites and in water and a few hours in aerosolized droplets.

Sample Collection for Diagnosis

Nasopharyngeal swabs collected approximately 1-2 days after illness onset

Paired (acute and convalescent) serum samples collected 2-3 weeks apart

8.2.2 Equine herpesvirus infection (Equine viral rhinopneumonitis)

Definition and Causative Agent

Equine herpesvirus-1 (EHV-1) and equine herpesvirus-4 (EHV-4) are two distinct viruses from the Herpesviridae family, primarily causing respiratory disease (rhinopneumonitis) in horses. In addition to respiratory issues, these viruses can also lead to abortion and neurological disorders in horses. EHV-1 can impact multiple organs leading to serious complications, whereas EHV-4 mainly affects the respiratory system.

Transmission

EHV-1 and EHV-4 are transmitted directly through the inhalation of infectious respiratory secretions from infected horses and indirectly via fomites such as contaminated clothing, equipment, and hands, with an incubation period of 2-10 days.

Clinical Signs

Clinical signs of equine herpesvirus infections include general symptoms such as fever (38.9°–41.7°C), serous nasal discharge, malaise, pharyngitis, cough, inappetence, and submandibular or retropharyngeal lymphadenopathy, along with leukopenia/neutropenia. EHV-1 specifically presents with a biphasic fever pattern, abortions

(2–12 weeks post-infection), neonatal foal death, and equine herpesvirus myeloencephalopathy (EHM), which causes neurological signs such as incoordination, caudal paresis, and paralysis. EHV-4 primarily causes respiratory disease and sometimes leads to abortion.

Diagnosis

A combination of clinical examination, clinical signs, and laboratory tests is used to diagnose the equine herpesvirus infection. Laboratory methods include PCR assay to detect EHV DNA in samples from nasal swabs, blood, or tissues, which is highly sensitive and can differentiate between EHV-1 and EHV-4. Virus Isolation, although less commonly used, involves culturing samples to identify the virus. The ELISA, a serological test to detect antibodies against EHV-1 or EHV-4, indicates past infections or immune status.

Treatment

There is no specific treatment for equine herpesvirus infection (EHV). A minimum of three weeks of rest along with supportive care is recommended for recovery. Supportive care may include use of anti-bacterial drugs for secondary bacterial pneumonia (*refer* Annexure-I) and non-steroidal anti-inflammatory drugs (NSAIDs).

Control

During an outbreak, the control measures include immediately isolating the affected horses, monitoring exposed the horses for signs of infection, and restricting the movement completely in and out of the affected premises. Imported vaccines are available in the Indian market to help control diseases caused by EHV-1/4. Vaccination is effective in reducing the incidence of abortions, paralysis, perinatal foal mortality, and respiratory diseases associated with these viruses.

Biosecurity

Biosecurity measures include quarantining new animals brought from outside for 28 days, quarantining animals – brought back from fairs/ melas, shows, and competitions – for 28 days, isolating new arrivals and horses showing clinical signs, implementing quarantine during outbreaks, and disinfecting equipment, stables, and transport vehicles.

Sample Collection for Diagnosis

Nasopharyngeal swabs, blood, aborted tissue materials and brain (in case of EHM).

Paired (acute and convalescent) serum samples collected 2-3 weeks apart.

8.2.3 Japanese encephalitis (JE)

Definition and Causative Agent

Japanese encephalitis, an arthropod-borne viral infection, is caused by the Japanese encephalitis virus (JEV) belonging to the genus *Flavivirus* in the family Flaviviridae. It is widely distributed in South-East Asia including India, Western Pacific regions, and northern Australia.

Transmission

Japanese encephalitis virus is transmitted through the bite of infected mosquitoes, with no evidence of human-to-human or animal-to-human transmission. The primary vectors are *Culex* mosquitoes, including species like *Culex tritaeniorhynchus* in Asia and *Culex annulirostris* in Australia. The natural life cycle of the virus involves water birds, such as herons and egrets, and mosquitoes. Water birds and pigs serve as reservoirs, while pigs being important amplifying hosts. Horses and humans are incidental hosts; and they are considered dead-end hosts because they do not develop sufficient viraemia to infect mosquitoes.

Clinical Signs

In horses, JEV infection is mostly subclinical, which shows no noticeable symptoms. Some horses may develop a transitory syndrome with mild fever, loss of appetite, and impaired locomotion, typically recovering within 2 to 3 days. The lethargic form of the infection is marked by high fever (41°C), stupor, bruxism, difficulty in swallowing, neck rigidity, impaired vision, paresis, and paralysis, with recovery generally occurring in about a week. The hyperexcitable form is more severe with high fever (41°C or higher), profuse sweating, muscle tremors, aimless wandering, behavioural changes, loss of vision, neurologic sequelae, collapse, and coma, leading to death.

Diagnosis

Initial diagnosis is based on observed clinical signs and symptoms. Molecular tests, such as RT-PCR and TaqMan-based real-time PCR are used to detect the virus in blood (collected during the early stage

of infection), brain, spinal cord, and cerebrospinal fluid (CSF). Serological tests are also used to detect virus-specific IgM antibodies in serum or CSF, using methods like ELISA, virus neutralization (VN), hemagglutination inhibition (HI), and complement fixation (CF) tests. In endemic areas, paired serum samples may be required due to the possibility of inapparent infection. Additionally, virus isolation in cell culture, using BHK-21, Vero, or C6/36 cell lines, can confirm the presence of JEV.

Differential Diagnosis

Equine herpes myeloencephalopathy, rabies, West Nile encephalitis, equine viral encephalitis, Borna disease, tetanus, acute piroplasmosis, equine protozoal myeloencephalitis and leucoencephalomalacia.

Treatment

There is no specific treatment for JE in horses and hence supportive care according to the signs observed is advisable. Supportive care includes I/V fluids - if the horse is unable to drink, use of appropriate anti-inflammatory agents (phenylbutazone and flunixin meglumine) and anticonvulsants, if necessary, and sling support for unstable or paralyzed animals.

Control

Vaccination is not practiced in India, currently, though vaccines are available in other countries. It is crucial to follow manufacturer guidelines for vaccine schedules based on age, disease risk, and previous vaccination history. Surveillance and reporting should be strengthened with timely diagnosis and outbreak forecasting, supported by effective diagnostic facilities and reporting mechanisms.

Biosecurity Measures

Ensuring environmental sanitation through effective water drainage and clean piggeries, using insect repellents, spraying larvicides, and housing animals indoors in screened stabling to protect them from mosquitoes.

Sample Collection for Diagnosis

Blood (collected during the early stage of infection), brain, spinal cord, and cerebrospinal fluid (CSF).

Paired (acute and convalescent) serum samples collected 2-3 weeks apart.

8.2.4 Rabies

Definition and Causative Agent

Rabies is a severe, rapidly progressing neurological disease caused by the rabies virus. It is a neurotropic, negative-sense, non-segmented, single-stranded RNA virus belonging to the *Lyssavirus* genus of the Rhabdoviridae family. It is almost always fatal once symptoms manifest.

Transmission

In horses, rabies is primarily transmitted through bites from rabid animals such as dogs, foxes, mongooses, and jackals, with dog bites accounting for 95-97 percent of global rabies cases. Additionally, rabies can also be transmitted through exposure to infected saliva or brain tissue via open wounds, abrasions, or mucous membranes.

Clinical Signs

The incubation period for rabies in horses typically ranges from 3 to 12 weeks, though it can vary from as short as 5 days to several years. Rabid horses may display symptoms such as hyperesthesia, fever, and neurological signs like ataxia or paralysis. While most horses appear depressed, some may exhibit aggression. Other symptoms can include bruxism (teeth grinding), anorexia, and refusal to drink, although some horses may continue eating and drinking until shortly before death. As the disease progresses, horses may become recumbent, experience convulsions, enter a comatose state, and show violent thrashing before death.

Diagnosis

For ante-mortem diagnosis, viral nucleic acid detection is conducted using real-time TaqMan PCR on pooled saliva samples collected at 3–6-hour intervals. Although cerebrospinal fluid (CSF) can be used, it is less sensitive. A key limitation of ante-mortem diagnosis is that a negative result does not rule out rabies. Post-mortem diagnosis is carried out through brain tissue examination, using direct fluorescent antibody tests (DFAT) and RT-qPCR on brain samples, while CSF, salivary gland, skin (particularly tactile facial hair follicles), and corneal impression smears are less effective. Serological tests such as the rapid fluorescent focus inhibition test (RFFIT), fluorescent antibody virus neutralization test (FAVN), and ELISA have limited diagnostic value due to the rapid progression of rabies and the short survival time of rabid animals.

Differential Diagnosis

Equine herpes myelencephalopathy, Japanese encephalitis, West Nile encephalitis, equine viral encephalitis, Borna disease, Tetanus, Acute babesiosis, Equine protozoal myeloencephalitis and Leucoencephalomalacia.

Treatment

There is no specific treatment once rabies symptoms develop. Every effort should be made to prevent the development of the disease.

Treatment of horses prior to the development of clinical signs (after exposure to a rabid animal): For horses bitten by a confirmed or suspected rabid animal, treatment (before the development of clinical signs) includes thorough wound cleaning by flushing with running tap water for 15 minutes and washing with soap, applying povidone-iodine or 70 percent ethanol, and covering large wounds with a simple dressing. Post-exposure prophylaxis using five-vaccine doses should be administered on days 0, 3, 7, 14, and 28, with an optional dose on day 90. Tetanus toxoid may be administered to prevent tetanus, while antibiotic prophylaxis can be used to prevent secondary bacterial infections, depending on the specific case. Although, the rabies immunoglobulin (RIG) is rarely used in animals due to cost and availability, it can provide additional protection if administered (as demonstrated in dogs and sheep). An exposed horse should be quarantined and monitored for signs of rabies. Previously vaccinated horses need to be observed for 45 days following post-exposure prophylaxis (PEP), while unvaccinated horses may require a quarantine period of six months or longer.

Treatment of horses after development of clinical signs: Once clinical signs of rabies appear, the disease is fatal. While supportive care may alleviate suffering, the horse will either die naturally or may be euthanized, if local regulations allow.

Control

Effective control also relies on a multidisciplinary approach involving government agencies, local bodies, medical and veterinary professionals, animal owners, public health workers, ecologists, and vaccine producers. Vaccinating at least 70 percent of dogs can reduce canine rabies and thereby break the transmission cycle. Pre-exposure vaccination may be administered to horses in rabies-endemic country like India. The recommended age for the initial vaccination and booster doses in horses can vary by manufacturer. Typically, horses should receive their first vaccination starting at 3-6 months of age. A booster may be needed 4 weeks later, followed by annual boosters.

Biosecurity Measures

When handling animals suspected of having rabies, the handler must wear protective face masks, gloves, eye protection, clothing, and shoes. Disinfect any surfaces, tools, instruments, floors, and walls that may have been contaminated with animal fluids. Use cutting tools like scissors and scalpels carefully during postmortem procedures to avoid injury to self and potential viral contamination of the wound.

Sample Collection for Diagnosis

Antemortem samples: Saliva (three pooled samples taken at 3–6-hour intervals). The virus might also be found in CSF, salivary glands, skin (tactile facial hair follicles), and corneal impression smears, but detection is less efficient.

Post-mortem samples: The brain is the preferred tissue for postmortem diagnosis of rabies. For direct fluorescent antibody test (DFAT) on animal brains, impression smears should be taken from a composite brain tissue sample including the brain stem and cerebellum. If the cerebellum is unavailable, a cross-section of the Ammon's horns may suffice.

Serum: Serum have limited diagnostic value due to the rapid progression of rabies and short survival of the animals. However, paired serum (taken 2-3 weeks apart) can be useful in cases where animal survives beyond a week. A significant antibody rise in paired serum samples collected confirms the diagnosis in such cases.

8.2.5 Equine viral arteritis (EVA)

Definition and Causative Agent

Equine Viral Arteritis (EVA) is a contagious viral disease affecting the respiratory and reproductive systems of equines, including horses, donkeys, mules, and zebras. EVA is caused by the Equine arteritis virus (EAV) - from the Arteriviridae family – which is an enveloped, positive-sense, single-stranded RNA virus with two distinct phylogenetic lineages., *viz*., North American (NA) and European (EU).

Transmission

The EAV can be transmitted through respiratory, venereal, congenital routes, or indirectly. Carrier stallions shed the virus in their semen which contributes to genetic and phenotypic divergence of the virus and can lead to new outbreaks. Horizontally, EAV spreads via respiratory routes during acute infection, posing risks in close-contact environments such as racetracks, veterinary hospitals, and breeding farms. It can also be transmitted through contact with placental materials and indirectly via contaminated fomites or handlers. Vertically, EAV can be transmitted during natural breeding or artificial insemination with semen from infected stallions, which may shed the virus persistently, from weeks to a lifetime.

Clinical Signs

The incubation period for EAV varies, with respiratory transmission, typically taking 2 to 3 days and venereal transmission 6 to 8 days. Clinical signs range from mild to severe, influenced by the viral strain, host immunity, and environmental conditions. Acute symptoms include high fever (up to 41°C), depression, loss of appetite, leukopenia, conjunctivitis, respiratory issues, clear to mucus-like nasal discharge, and severe pneumonitis in young foals, along with vascular lesions, swelling in dependent areas, pinpoint haemorrhages, and urticaria. EAV can cause abortions in pregnant mares (10-70 percent), and 10-70 percent of stallions may become persistently infected, shedding the virus in their semen.

Diagnosis

Diagnosis involves reviewing the animal's history, including purchase and semen use for artificial insemination, and observing clinical signs. Confirmation is achieved through postmortem lesions such as oedema, congestion, haemorrhages, and excess fluid in the peritoneal, pleural, and pericardial cavities. Microscopy may reveal vasculitis in small arterioles and venules, and immunohistochemistry (IHC) can detect viral antigens. Virus isolation and nucleic acid detection are performed using RT-PCR and RT-qPCR on nasopharyngeal swabs, washings, and unclotted blood samples. Serological tests, including virus neutralization tests (VNT) and ELISA, are used to detect the humoral antibody response.

Differential Diagnosis

Differential diagnosis includes equine herpesvirus 1 and 4 (EHV-1, EHV-4), equine influenza virus (EIV), equine rhinitis A and B viruses, equine adenovirus, Getah virus infection, Dourine, African horse sickness (AHS), equine infectious anaemia (EIA), purpura haemorrhagica, and allergy-induced urticaria.

Treatment

There is no specific antiviral treatment for EVA. Supportive care during the acute phase includes antipyretics, anti-inflammatory drugs, diuretics, rest, and good nursing care. There is no effective treatment for EVA pneumonia or pneumoenteritis in foals, and early euthanasia (if regulations permit) is recommended to minimize the risk of further spread of the virus.

Control

Preventing and controlling EAV focuses on limiting its spread in breeding populations and reducing outbreak risks. Although no vaccine is available in India, attenuated or inactivated vaccines used abroad provide immunity for 2-3 years.

Biosecurity Measures

Effective management practices include isolating new arrivals for 3-4 weeks, keeping pregnant mares in isolated groups, managing carrier stallions separately, and taking precautions during semen collection. Additionally, testing fresh-cooled or frozen semen for EAV, halting breeding activities during outbreaks, and sanitizing stalls and equipment are crucial measures.

Sample Collection for Diagnosis

Nasopharyngeal swabs or washings.

Unclotted (citrated or EDTA) blood samples.

Paired (acute and convalescent) serum samples collected 3–4 weeks apart.

8.2.6 African Horse Sickness

Definition and Causative agents: African horse sickness (AHS) - a highly infectious and often fatal viral disease affecting horses, mules, and donkeys - is caused by the African horse sickness virus (AHSV) which belongs to the genus *Orbivirus* within the Reoviridae family. India is free from this disease.

Transmission

African Horse Sickness is transmitted primarily through the bite of infected biting midges (*Culicoides* species), which are the main vectors of the virus.

Clinical Signs

African horse sickness can present in various forms with different severities:

Pulmonary (Per acute) Form: Characterized by high fever (40-41°C), severe respiratory distress with rapid breathing and frothy nasal discharge, and infected animals often standing with forelimbs spread and nostrils fully dilated. Coughing starts dry and progresses to moist, with death often occurring within a few hours to a day after the onset of respiratory signs, and a mortality rate exceeding 95%.

Cardiac (Sub acute) Form: Features fever (39-40°C), followed by oedema starting in the eyelids and extending to other areas such as the cheeks, lips, and sometimes the neck and chest. Congestive heart failure signs include jugular vein distension and petechial haemorrhages, with a mortality rate up to 50% and death occurring 4-8 days after onset.

Mixed Form: Exhibits a combination of pulmonary and cardiac symptoms, with a high mortality rate ranging from 70-80%.

Horse Sickness Fever (Mild Form): Presents with mild fever (37.8-39°C) and mild swelling of the supraorbital fossa (area above the eyes), with most horses recovering.

Diagnosis

Diagnosing AHS involves several steps owing to its resemblance to other equine respiratory diseases. Diagnosis involves observing clinical signs such as high fever, respiratory distress, and oedema. For antemortem diagnosis, blood samples (in EDTA) are taken, while spleen, lung, and lymph nodes are collected after necropsy. Postmortem examination of lesions, including pulmonary oedema and cardiac haemorrhages, further aids in confirming the diagnosis. Laboratory tests include PCR to detect viral RNA; serological tests like ELISA and virus neutralization to identify antibodies; and virus isolation from blood or tissues.

Differential Diagnosis

Differential diagnosis includes equine encephalosis virus, equine infectious anaemia, equine viral arteritis, anaplasmosis, or theileriosis.

Treatment

There is no specific treatment for AHS; management emphasizes supportive care, which includes fluid therapy, anti-inflammatory drugs, and pain relief to address symptoms and complications.

Control

Different approaches exist for endemic versus AHS-free countries or zones. India is presently free of AHS, and vaccination is not currently practiced. Controlling potential infections relies on ongoing surveillance and prompt reporting to detect outbreaks early and a swift response. Vector control and implementation of biosecurity measures help maintain disease-free status.

Biosecurity Measures

Stabling horses during peak vector activity (dawn to dusk) reduces transmission risk. Stables can be protected by covering openings with insecticide-treated shade cloth, using double-door systems and automatic insecticide dispensers. Additionally, applying topical insect repellents and insecticides, such as permethrin and DEET, to horses and stable buildings effectively prevents insect bites.

Sample Collection for Diagnosis

Antemortem: Blood samples (in EDTA).

Postmortem: Spleen, lung, and lymph nodes.

Paired (acute and convalescent) serum samples collected 3-4 weeks apart.

8.2.7 Equine infectious anaemia

Definition and Causative agents

Equine infectious anaemia (EIA) - a viral disease affecting horses, mules, and donkeys – is caused by the Equine infectious anaemia virus (EIAV). It is a *Lentivirus* in the Retroviridae family. EIA is also known as swamp fever due to its association with wet, swampy areas where vectors such as horseflies and deerflies are prevalent.

Transmission

EIA is primarily transmitted through blood-feeding insects like horseflies and deerflies. The virus can also spread through the use of contaminated needles, surgical instruments, or blood transfusions. It can

be passed from mare to foal through the placenta.

Clinical Signs

EIA can manifest in acute, subacute, or chronic forms, with varying symptoms. In the acute form, affected horses may experience high fever up to 40.5°C, severe anaemia, and oedema of the lower abdomen and legs. They often show weakness, depression, rapid weight loss, and can die within 2-3 weeks. The subacute form is characterized by moderate fever, anaemia, weight loss, and a prolonged illness with less severe symptoms. In the chronic form, horses have recurrent episodes of fever and anaemia, persistent weight loss, lethargy, and ongoing edema and muscle wasting.

Diagnosis

Diagnosis of EIA is primarily conducted through serological tests. The Coggins test, also known as the agar gel immunodiffusion assay, is the gold standard for diagnosing EIA. Additionally, the ELISA is a quicker and more sensitive method that can detect early stages of infection. For molecular diagnosis, PBMCs separated from whole blood are utilized to confirm the presence of the virus.

Differential Diagnosis

Equine viral arteritis, purpura haemorrhagica, piroplasmosis, severe strongylosis or fasciolosis and autoimmune haemolytic anaemia.

Treatment

There is no antiviral treatment or cure for EIA. Infected horses, being lifelong carriers, are euthanized as per the rules of the land.

Control

Control and prevention of EIA involves several measures. Regular testing of horses - particularly before transport or entry into new stables - is essential, along with quarantine and euthanasia of infected animals to prevent spread. No safe and effective vaccine is available for EIA and hence vaccination is not yet a standard practice for EIA. All horses participating in shows or performance events must have proof of EIA testing within a specified timeframe.

Biosecurity Measures

Strict adherence to proper hygiene and disinfection practices is crucial to prevent iatrogenic transmission of EIA. Reducing exposure to blood-feeding insects with repellents and fly control measures, and stabling horses during peak insect activity. Sterile practices are crucial, such as using sterilized needles and surgical equipment and avoiding the sharing of equipment that could be contaminated with blood. Movement restrictions and event bans are imposed in affected areas, and surveillance systems with regular examinations and sample analysis help detect and track outbreaks.

Sample Collection for Diagnosis

Unclotted (citrated or EDTA) blood samples and serum samples.

8.3 Equine Bacterial Diseases

8.3.1 Strangles (Equine Distemper)

Definition and Causative Agent

Strangles or equine distemper - a highly contagious disease of the upper respiratory tract in horses and other equines - is caused by the Gram-positive bacterium *Streptococcus equi*. The bacterial infection leads to lymphadenopathy, causing obstruction of the pharynx, larynx, and trachea, which can result in sudden death, hence the name strangles. The abscesses are typically restricted to the head and neck, in chronic cases they may extend to other body parts such as the abdomen and brain, a condition known as bastard strangles. The disease is commonly reported more in younger animals compared to that in older ones.

Transmission

Transmission occurs through direct or indirect contact with infected horses. Discharge from abscesses and nasal cavities can contaminate pastures, feed, water troughs, stalls, and the hands and clothes of grooms and veterinarians, and thereby spreading the infection to other susceptible animals in the herd. About 10-30% of clinically affected horses have persistent infections and can act as carriers. Guttural pouch empyema is common in these carrier horses. Proper quarantine of recovered horses and repeated culture or PCR of nasopharyngeal swabs are required to confirm the carrier state.

Clinical Signs

In strangles, the upper respiratory tract and lymph nodes in the head and neck of the affected animals is primarily affected. One of the earliest and most

common signs of strangles is fever, with temperature rising significantly, often between 39.4°C and 41.1°C. As the disease progresses, horses develop a nasal discharge that starts as clear and watery discharge but quickly becomes thick, yellow, and purulent, typically draining from both the nostrils.

Swelling of the submandibular and retropharyngeal lymph nodes is the cardinal sign of strangles. These swollen lymph nodes are painful and may eventually rupture, releasing thick pus (with thick, cream-yellow pus). This swelling can sometimes become so severe that it obstructs the airway, making it difficult for the horse to breathe, which is why the disease is called strangles. Additionally, horses may show difficulty swallowing, known as dysphagia, due to the pressure from swollen lymph nodes or abscesses in the throat area.

Fatal complications such as suppurative necrotic bronchopneumonia may occur due to aspiration of pus from the upper airways.

Lesions

Suppurative lesions can be seen on visceral organs such as the liver, spleen, lungs, pleura, and peritoneum.

Diagnosis

Diagnosis is based on history and clinical signs, but confirmation requires detection of *S. equi* by PCR or culture from nasopharyngeal swabs, abscess discharges, or guttural pouch lavage. Owing to the possibility of false-negative results, a combination of PCR and culture is used for more accurate diagnosis.

Differential Diagnosis

Strangles can be differentiated from other upper respiratory tract conditions such as - equine viral arteritis; equine adenovirus; equine viral rhinopneumonitis.

Treatment

Antibiotics should be administered before abscess formation or in severe infection. Penicillin is the drug of choice. Procaine penicillin G @ 22,000 IU/kg I/M every 12 hours or sodium penicillin G @ 22,000 IU/kg I/V every 6 hours. If penicillin administration is not possible due to anaphylaxis or unavailability, use alternatives like animopenicillins, cephalosporins. Other antibiotics can be used are - oxytetracycline: 6.6 mg/kg IV every 12-24 hours; sulphonamide-trimethoprim combinations: 15-30 mg/kg orally or IV every 12 hours.

NSAIDs (*e.g.*, phenylbutazone, flunixin meglumine) are included in the treatment to manage pain and reduce fever. Ensure proper hydration and nutrition.

Abscess should be managed by performing its proper drainage and flushing with antiseptic solutions to promote healing. Hot compresses may help to promote drainage.

Prevention and Control

The vaccine is available which contains typically killed or inactivated bacteria (bacterin) or a purified protein extract from *Streptococcus equi*. These can be given through I/N or I/M route. It enhances mucosal immunity and provides strong local immunity at the site of infection.

Biosecurity Measures

Avoid importing infected animals into existing clean groups of horses. Proper biosecurity protocols should be followed to prevent the spread of the disease. Quarantine new arrivals for at least 2-3 weeks and monitor for symptoms. Isolate infected horses to prevent the spread of the disease.

Sample Collection for Diagnosis

It involves obtaining specimens from the affected horse to diagnose the presence of *Streptococcus equi* subsp. *equi*, the bacterium that causes the disease. The key types of samples typically collected are – nasal swabs, nasopharyngeal swabs/washes, abscess aspirates, guttural pouch lavage, blood, and serum samples. Ensure that all sample collection is done using sterile techniques to avoid contamination. Samples should be immediately transported to the laboratory under appropriate conditions, often cooled but not frozen, to preserve the bacteria for accurate diagnosis.

8.3.2 Glanders

Definition and Causative Agent

Glanders is a highly contagious zoonotic disease affecting equids such as horses, mules, and donkeys. It is clinically characterized by nodular lesions on the skin, respiratory tract, and lymphatic system. While most countries are free from glanders, focal outbreaks are still reported in some parts of the world. The disease is caused by *Burkholderia mallei*, a non-motile, non-sporulating, facultative

intracellular, gram-negative bacillus. *B. mallei* is an obligate mammalian pathogen and was first isolated by Loeffler and Schutz in 1882. The pathogen can remain viable at room temperature for about a month but is inactivated by heat, direct sunlight, and common disinfectants, including hypochlorites. Historically, *B. mallei* has been used as a bioterrorism agent.

Transmission

Recovered animals that act as carriers can be a potential source of infection for healthy animals. Transmission can occur through ingestion or inhalation of infected materials, such as contaminated feed, watering troughs, and utensils. Direct contact with infected animals having cutaneous lesions is also a common mode of transmission.

Clinical Signs

Glanders can manifest in acute or chronic forms:

Acute Form: Characterized by fever, cough, nasal discharge, ulcers on the nasal mucosa, and nodules on the skin of the lower limbs or abdomen. Death may occur within a few days due to toxaemia.

Chronic Form: Characterized by inappetence, cough, dyspnea, nasal discharge, weight loss, enlargement of submandibular lymph nodes, and exercise intolerance. Ulcers may appear on the nasal septum or as nodules and ulcers on the limbs.

There are three major forms of glanders:

Pulmonary Form: Characterized by cough, frequent epistaxis, and laboured respiration.

Nasal Form: Characterized by 1 cm diameter nodules on the nasal septum and turbinate, which later ulcerate. Initial serous nasal discharge becomes purulent and blood-stained. Healing ulcers are replaced by stellate scars.

Skin Form: Characterized by 1-2 cm subcutaneous nodules that ulcerate and discharge honey-like pus.

Lesions

The disease occurs in two forms, *viz*., acute and chronic form described as below:

Acute Form - Multiple petechial haemorrhages throughout the body, catarrhal bronchopneumonia, and enlargement of the bronchial lymph nodes. ***Chronic Form*** - Miliary nodules in the lungs and ulcers on the mucosa of the nasal cavity, larynx, trachea, and bronchi.

Diagnosis

Diagnosis is based on clinical signs and serological tests, preferably the complement fixation test. The Mallein test, involving an I/D injection of Mallein is also used. Positive reaction is indicated by a significant skin thickness around the injection area after 48 hours of its application.

Differential Diagnosis

Glanders should be differentiated from epizootic lymphangitis, ulcerative lymphangitis, sporotrichosis, melioidosis, strangles, *Rhodococcus equi* infection, and equine pleuropneumonia.

Treatment

No treatment is undertaken. Infected animals are euthanised to prevent the spread of the disease as per Prevention and Control of Infectious and Contagious Diseases in Animals Act, 2009.

Control

Control measures include early identification, isolation, and euthanasia of infected animals. In endemic areas, regular screening of animals is essential.

Biosecurity

Precautionary measures such as wearing surgical masks, face shields, gloves, and gowns are advised while handling animals in endemic areas. Strict biosecurity and quarantine measures are crucial for prevention and control.

Sample Collection for Diagnosis

It involves obtaining specimens to detect *Burkholderia mallei*, the bacterium responsible for the disease. The following types of samples are typically collected - nasal swabs, lymph node aspirates, blood samples, skin lesion samples, bronchoalveolar lavage (BAL). Use sterile techniques to avoid contamination. Glanders is highly contagious, and appropriate biosafety measures should be taken during sample collection and handling. Samples should be promptly transported to the laboratory under appropriate conditions, typically refrigerated, for bacterial culture or PCR testing.

8.3.3 *Rhodococcus equi* Infection

Definition and Causative Agent

Rhodococcus equi infection – a zoonotic disease primarily affecting horses and foals worldwide – is a major cause of foal mortality, characterized by pyogranulomatous lesions in pulmonary and extrapulmonary tissues. While most *Rhodococcus* species are non-pathogenic, but *Rhodococcus equi* (*R. equi*) is pathogenic. The disease is common in foals between 3-week to 5-month-old, though it can also occur in older foals. Immunocompromised individuals involved in handling and care of stud farms are also at risk.

Transmission

R. equi is transmitted through aerosolization, entering the respiratory or digestive tract. The primary route of transmission in both animal and human populations is exposure to contaminated soil. Infection may also occur through traumatic inoculation or superinfection of wounds. Disease progression is facilitated by continuous shedding of the organism in faeces on stud farms. Humans can contract the infection through contact with infected animals' waste, farm utensils, or direct oropharyngeal transmission.

Clinical Findings

Clinical signs are often not present until the advanced stages of the disease thus making early confirmation challenging. Symptoms can be both pulmonary and extrapulmonary. The *pulmonary symptoms* include reduced exercise capacity, nasal discharge, bronchopneumonia, pyogranulomatous pneumonia, and lung abscesses while *extrapulmonary symptoms comprise of* diarrhoea, fever, polysynovitis, uveitis, enterocolitis, abdominal abscesses, and osteomyelitis.

Diagnosis

Diagnosis is based on the history of *R. equi* infection on the farm and distinct clinical signs. Neutrophilic leucocytosis is highly suggestive clinical findings. Bacteriological culture, PCR on tracheal aspirate samples, ultrasonography, and radiology are used for definitive diagnosis.

Differential Diagnosis

The disease should be differentiated from other lower respiratory tract infections. Musculoskeletal abnormalities caused by *R. equi* should be distinguished from septic arthritis caused by *Streptococcus zooepidemicus* and *Salmonella* spp.

Treatment

Combination antibiotic therapy is recommended at least for 30-40 days**.** The recommended antibiotics are *Macrolides:* Erythromycin, azithromycin, clarithromycin, or gamithromycin are commonly used due to their effectiveness against *R. equi*; and *Rifampin:* Often used in combination with macrolides to enhance efficacy.

Dosage and Administration

Erythromycin: 25 mg/kg orally every 8-12 hours. *Azithromycin:* 10 mg/kg orally once daily for 5 days, then every 48 hours. *Clarithromycin:* 7.5 mg/kg orally every 12 hours. *Rifampin:* 5 mg/kg orally every 12 hours. Macrolides are not to be used in hot weather as they may cause hyperthermia in foals.

Supportive Care

NSAIDs: Non-steroidal anti-inflammatory drugs like flunixin meglumine can be used to reduce inflammation and pain. *Hydration and Nutrition:* Ensure that the foal remains hydrated and receives proper nutrition. *Oxygen Therapy:* In severe cases with significant respiratory distress, oxygen therapy may be necessary.

Surgical Intervention

In cases of large abscesses or where antibiotic therapy is insufficient, surgical drainage of abscesses may be required.

Prevention and Control

Passive Immunization - Administration of hyperimmune plasma from mares vaccinated with autogenous vaccines can provide passive immunity to foals. The foals may receive 1 litre of hyperimmune plasma intravenously within the first 24 - 48 hours of life. This early administration is crucial as it provides immediate passive immunity to the foal during the critical early period when they are most vulnerable to infection. A second booster dose of 1 litre of hyperimmune plasma is often administered at 2 to 4 weeks of age. This boosts the foal's immunity during the period when maternal antibodies from the colostrum may begin to wane.

Repeat Imaging and Testing: Follow up imaging and laboratory tests to assess the effectiveness of treatment and adjust therapy as needed.

Biosecurity

It is crucial for controlling and preventing the spread

of *Rhodococcus equi* on equine farms, particularly those involved in breeding and raising foals. Reduce dust in the environment to minimize inhalation of *R. equi*. Regular removal and composting of manure to reduce environmental contamination. Designate specific, clean areas for foaling that are well-drained and free from accumulated manure. Regularly clean and disinfect these areas to reduce the bacterial load. Implement quarantine procedures for any new animals arriving on the farm, or even animals returning back to farm from fairs/events/markets.

Sample Collection for Diagnosis

This is crucial for diagnosing of *Rhodococcus equi* infection particularly in foals. Samples specifically collected are - tracheal wash/bronchoalveolar lavage (BAL) fluid, blood samples, nasal swabs, and lung biopsy (in severe cases). Ensure all sampling is done using sterile methods to prevent contamination. Samples should be transported to the laboratory promptly, usually chilled, to maintain sample integrity for accurate diagnosis. Proper sample collection and handling are essential for diagnosing *Rhodococcus equi* infections and determining the appropriate treatment and management strategies.

8.3.4 Anthrax

Definition and Causative Agent

Anthrax is a fatal infectious bacterial disease affecting all warm-blooded animals, including horses and humans. Horses are considered less prone to anthrax compared to cattle and sheep owing to differences in their digestive systems. The disease is caused by *Bacillus anthracis*, a gram-positive, spore-forming, facultative anaerobic bacterium. Upon the death of an infected animal, the vegetative form of the bacterium converts into spores when exposed to the environment. These spores can survive in soil for many years due to their low nutritional demands and high tolerance to environmental conditions. Neutral or alkaline soil rich in calcium or lime is more favourable for the pathogen's multiplication.

Transmission

Transmission can occur via ingestion, inhalation, or skin penetration by biting flies, insects, or trauma. Horses grazing in areas where anthrax is common may consume the spores and become infected. Sometimes, contaminated forage can also lead to infection. The incubation period ranges from 3 to 7 days. After infecting the animal, the bacteria multiply and spread throughout the body, producing lethal toxins that cause cell death, organ damage, and failure. The bacteria spread through the blood and lymphatic system.

Clinical Findings

Death is often the only clinical sign observed. Prior to death, signs may include high fever, agitation, chills, severe colic, anorexia, depression, staggering, respiratory distress, exercise intolerance, muscle weakness, seizures, and in rare cases, bloody diarrhoea. Oedematous swelling around the neck may cause suffocation. Swelling of the chest, lower abdomen, and external genitals can also occur. If spores infect open cuts or abrasions, a localized skin infection occurs. Death usually occurs within 2 to 3 days of the first signs. Unclotted blood may ooze from natural body openings like nostrils, mouth, anus, and vulva. Abortions can occur in pregnant mares, and blood-stained milk may be seen in lactating mares with anthrax.

Diagnosis

Diagnosis is based on clinical signs. The sudden death of one or more animals, particularly in a herd, along with the presence of dark, unclotted blood from orifices, should raise immediate suspicion of anthrax.

Laboratory analysis of blood samples (bacterial culture identification) is required to confirm the presence of the bacteria. Smears prepared from oedematous tissue should be stained with polychrome methylene blue and examined.

Differential Diagnosis

Anthrax should be differentiated from other causes of sudden death, such as lightning strikes, per acute blackleg, malignant edema, bacillary haemoglobinuria, and hypomagnesemia tetany.

Postmortem Lesions

Post-mortem findings indicative of anthrax include dark-colored, tarry blood from natural orifices, failure of the blood to clot, widespread ecchymoses. Avoid movement of carcass and do not proceed for autopsy.

Treatment

Anthrax is a toxigenic zoonosis and treatment is not undertaken as per as per Prevention and Control of Infectious and Contagious Diseases in Animals Act,

2009.

Control

The primary aim of anthrax control is preventing human transmission. During outbreaks, key measures include quarantining farms, destroying carcasses, and vaccinating survivors. Avoiding consumption of milk and meat from infected farms is crucial. Infected carcasses should not be opened and must be immediately burned *in situ* along with contaminated manure, bedding, and soil. Quicklime for deep burial is common but not recommended due to groundwater contamination risks. If burial is delayed, 5 percent formaldehyde should be applied to the carcass and surroundings to prevent scavenging. Disinfecting premises, hides, bone meal, fertilizer, wool, and hair by heat (60°C for a few minutes) or other disinfectants like 5 percent Lysol, formalin, or sodium hydroxide (5-10 percent) is essential. Infected clothing should be sterilized by soaking in 10% formaldehyde. Hides and wool can be sterilized commercially by gamma-irradiation.

Biosecurity

Disposal of carcasses from animals that have died of anthrax is a critical process that must be handled with extreme caution to prevent the spread of *Bacillus anthracis* spores. Improper handling can lead to environmental contamination, which can pose serious risks to public health and other animals. Burning/cremation of carcass should be performed. Anyone involved in the disposal process should wear appropriate PPE, including protective clothing, gloves, masks, and eye protection. PPE helps prevent human exposure to the spores.

Sample Collection for Diagnosis

The sample collection for anthrax in horses requires extreme caution due to the zoonotic nature and potential environmental contamination risk posed by *Bacillus anthracis*. Samples can be collected are - blood samples, aspirates from exudates. Carcasses should not be opened to avoid releasing spores into the environment. Instead, samples should be collected from intact orifices (such as nasal or rectal swabs). Full PPE kit, including gloves, masks, and protective clothing, is essential. Samples must be securely contained and transported under strict biosafety conditions to prevent contamination and exposure. Given the risk associated with anthrax, sample collection should only be performed by trained professionals with appropriate biosecurity measures in place.

8.3.5 Contagious Equine Metritis

Definition and Causative Agent

Contagious equine metritis (CEM) is a non-systemic, venereal bacterial disease of mares affecting both the proximal and distal reproductive tract, causing temporary infertility and, in rare cases, abortion. It is caused by *Taylorella equigenitalis* which is a fastidious, microaerophilic, Gram-negative coccobacillus.

Transmission

CEM can be transmitted directly or indirectly. Stallion-to-stallion transmission can occur through contaminated fomites at semen collection centres or veterinary clinics. It can be transmitted by natural cover and artificial insemination with infected raw, chilled, or possibly frozen semen. Occasionally, transmission occurs from mare to foal either *in utero* or post-parturition. Inappropriate biosecurity measures during breeding can also contribute to transmission.

Clinical Findings

Clinical signs of CEM include endometritis, cervicitis, and vaginitis of variable severity, along with a slight to copious mucopurulent vaginal discharge. *T. equigenitalis* remains a commensal organism in asymptomatic stallions. In mares, the infection is confined to the reproductive tract. Mares bred to a carrier stallion by natural cover or AI with contaminated semen may develop a mucopurulent vaginal discharge lasting up to 15-21 days. Affected mares may face difficulties in conceiving on the first or second oestrus but often achieve normal pregnancies in subsequent breeding. Most mares eliminate the infection, but some may become persistently infected carriers for an extended period.

Diagnosis

Diagnosis is based on clinical signs and isolation of the organism by swabbing specific urogenital sites in the mare. Cultural isolation of the bacteria (the gold standard) and the complement fixation (CF) test are the official diagnostic tests. Since the organism is slow-growing, multiple cultures over 1 week are necessary for a positive outcome.

Differential Diagnosis

CEM should be differentiated from other reproductive tract infections, such as coital exanthema, metritis due to other causes, and pyometra.

Treatment

Cleaning and Disinfection – Mares: Thoroughly clean the clitoral fossa and clitoral sinuses with a 2 percent chlorhexidine solution. Repeat this treatment daily for 5-7 days. Stallions: Thoroughly clean the penis, prepuce, and urethral fossa with a 2 percent chlorhexidine solution. Repeat this treatment daily for 5-7 days.

Antibiotics – Administer systemic antibiotics such as trimethoprim-sulphonamide (30 mg/kg) for 10-14 days. Follow-Up Testing - Retest the mare 10 days after completing treatment to ensure that the infection has been cleared.

Control

Isolate infected animals to prevent the spread of CEM. Quarantine new arrivals and test them before introducing them to the herd. Regular testing of breeding animals to ensure that they are free from *T. equigenitalis*.

Biosecurity

Implement strict biosecurity measures at breeding facilities. Use disposable or sterilized equipment for breeding and semen collection. Ensure proper hygiene and sanitation practices. Report suspected cases of CEM to appropriate authorities as it is a notifiable disease.

Sample Collection for Diagnosis

It involves obtaining specimens from specific sites to detect the presence of *Taylorella equigenitalis*, the causative bacterium. Proper sample collection is crucial for accurate diagnosis and prevention of disease spread.

Mares - *Clitoral Swabs*: Collect swabs from the clitoral fossa and sinuses; *Endometrial Swabs*: May be collected during oestrus for more accurate detection.

Stallions - *Penile Swabs*: Collect swabs from the urethral fossa, urethra, and prepuce. *Pre-ejaculatory Fluid*: Obtain, if possible, as it can contain the bacterium. Collect samples during the breeding season when the bacteria are most likely to be present. Use proper personal protective equipment (PPE) to prevent contamination and ensure safe handling of samples.

Samples must be transported to a certified laboratory under appropriate conditions for culture or PCR testing to confirm the presence of *Taylorella equigenitalis*. Proper sample collection and handling are vital for the diagnosis and control of CEM.

8.3.6 Tetanus

Definition and Causative Agent

Tetanus is a life-threatening disease caused by the toxin produced by the bacterium *Clostridium tetani*. This bacterium is commonly found in soil, manure, and the gastrointestinal tract of horses. The toxin affects the nervous system, leading to muscle stiffness and spasms.

Transmission

Clostridium tetani spores enter the body through wounds, surgical incisions, or punctures. The spores germinate in anaerobic (low oxygen) conditions and produce the potent neurotoxin tetanospasmin.

Clinical Signs

The clinical signs of tetanus typically develop within days to weeks after the bacteria enter the body through a wound, puncture, or other break in the skin. The first signs often include stiffness and muscle spasms, particularly in the muscles of the neck, back, and jaw, leading to the characteristic lockjaw where the horse has difficulty opening its mouth. As the disease progresses, the horse may exhibit a sawhorse stance, with rigid, extended legs, and a stiff, uncoordinated gait. The ears are often erect, and the tail may be held stiffly outwards. The horse's sensitivity to stimuli increases, with loud noises or sudden movements triggering exaggerated muscle spasms. In severe cases, the spasms can be so intense that the horse has difficulty in breathing causing respiratory failure. The third eyelid may protrude across the eye, and the horse may have difficulty in swallowing. Without prompt and aggressive treatment, tetanus can rapidly progress to complete muscle rigidity, convulsions, and death due to respiratory failure or exhaustion.

Diagnosis

The clinical signs and history of recent wounds or surgeries may help in diagnosis. There are no specific laboratory tests for tetanus; and diagnosis is

primarily based on clinical presentation.

Treatment

Neutralize the toxin by administering tetanus antitoxin to neutralize circulating toxin. Dosage: 10,000 - 50,000 IU intravenously or intramuscularly.

Antibiotics: Penicillin is the drug of choice to eliminate *C. tetani* infection. Penicillin G, 22,000 IU/kg body weight IM every 6-12 hours. *Alternative:* Metronidazole, 15 mg/kg body weight orally or IV every 8-12 hours.

Supportive Care:

Sedation and Muscle Relaxation - Administer sedatives and muscle relaxants to control spasms and provide relief. Acepromazine: 0.03-0.1 mg/kg BW IM or IV; Diazepam: 0.05-0.2 mg/kg BW IV; Methocarbamol: 15-30 mg/kg BW IV. Keep the horse in a dark, quiet, and padded stall to minimize external stimuli that may trigger spasms.

Wound Care: Thoroughly clean and debride any wounds to remove necrotic tissue and promote aerobic conditions. Ensure the wound is open to air to prevent anaerobic conditions that favour *C. tetani* growth.

Nutrition and Hydration: Provide supportive nutrition and hydration, often through IV fluids if the horse is unable to eat or drink.

Monitoring: Continuous monitoring of vital signs, muscle tone, and response to treatment. Intensive care may be required for several weeks.

Prevention

Vaccination: The typical dosage for a tetanus vaccine in horses is 1 ml of the tetanus toxoid administered by I/M route. This dose is the same for foals, adult horses, and pregnant mares.

Foals: The first dose of the tetanus toxoid is usually given at 4-6 months of age. A second dose is administered 4-6 weeks after the first dose.

Adult Horses (Unvaccinated or Unknown Vaccination History): Administer the first dose of the tetanus toxoid, followed by a second dose 4-6 weeks later.

Annual Booster: Administer a tetanus toxoid booster annually.

Biosecurity

It is aimed at preventing the introduction and spread of *Clostridium tetani* bacteria, which cause tetanus in horses. While vaccination is the primary means of protecting horses from tetanus, biosecurity measures can significantly reduce the risk of exposure to the bacteria in the environment.

Sample Collection for Diagnosis

It is not typically used for diagnosis, as tetanus is diagnosed based on clinical signs rather than by isolating the causative organism, *Clostridium tetani*. If required, wound exudate or tissue samples from the suspected site of infection could be collected under sterile conditions for culture or PCR, although this is rarely done due to the fastidious nature of the bacterium and the urgency of treatment.

8.4 Protozoan Diseases of Horses

8.4.1 Equine Piroplasmosis

Definition and Causative Agent

Equine piroplasmosis is a clinical or sub-clinical disease caused by protozoan parasites *Theileria equi* and/or *Babesia caballi*. These haemoprotozoa are transmitted by ixodid ticks, primarily from the genera *Hyalomma* and *Rhipicephalus*. The disease can be fatal and is characterized by fever, intravascular haemolysis, leading to progressive anaemia, haemoglobinuria, and jaundice.

Clinical Findings

Acute Signs: Fever, lethargy, weakness, depression, anaemia, jaundice, hemoglobinemia, and haemoglobinuria. Pregnant mares may abort, and temporary infertility may occur.

Chronic Signs: Many animals recover but may remain infected for years with *T. equi* or for a few months with *B. caballi* without showing clinical signs during the carrier state.

Diagnosis

It involves microscopic examination and certain tests.

Microscopic Examination: Demonstration of intra-erythrocytic parasites in blood smears stained with Giemsa's stain remains the best, most reliable, and economical method for diagnosing equine babesiosis, though it is not suitable for detecting carrier animals.

Diagnostic Tests: Indirect fluorescent antibody (IFA)

test and enzyme-linked immunosorbent assay (ELISA) are preferred and recommended by the OIE. Various forms of ELISA have been standardized and validated on field samples. PCR tests have been developed for detecting the DNA of the parasites.

Treatment

Imidocarb Dipropionate: Effective against both *T. equi* and *B. caballi*. Dosage: 2.2 to 4 mg/kg body weight I/M or S/C every 72 hours for two to four treatments.

Caution: Imidocarb can have side effects such as cholinergic reactions (salivation, lacrimation, urination, defecation) and hepatotoxicity.

Diminazene Aceturate: Dosage: 3.5-7 mg/kg BW I/M as a single dose.

Supportive Therapy: Fluids and Electrolytes: Intravenous fluids to maintain hydration and support kidney function. NSAIDs: Non-steroidal anti-inflammatory drugs like flunixin meglumine to reduce fever and inflammation. Blood Transfusions: May be necessary in cases of severe anaemia.

Control and Biosecurity

Tick Control - Regular Application of Tick Repellents: Use of acaricides and insect repellents on horses can reduce tick attachment and feeding. Topical treatments, such as permethrin-based sprays are commonly used.

Monitoring and Surveillance: Regular Blood Testing - Implement regular blood testing, especially in areas where piroplasmosis is endemic. Testing can identify carriers and help in monitoring the disease status of the herd.

Screening New Arrivals: Test all new horses entering a facility or region for equine piroplasmosis to prevent introducing infected animals into the population.

Clinical Monitoring: Regularly monitor horses for signs of piroplasmosis, including fever, lethargy, anaemia, jaundice, and swelling. Early detection is key to effective treatment and control.

Sample Collection for Diagnosis

Good quality samples are crucial for diagnosing the infection caused by *Theileria equi* and *Babesia caballi*. The primary types of samples collected include whole blood samples collected in EDTA and serum samples**.** Ensure that sample collections are performed using sterile techniques to avoid contamination. Samples should be transported to the laboratory under refrigeration to maintain their integrity for accurate diagnostic testing. These samples are typically analysed using microscopy, PCR, or serological tests to confirm the presence of *Theileria equi* or *Babesia caballi*. Proper sample collection and handling are essential for accurate diagnosis and effective management of equine piroplasmosis.

8.4.2 Equine Trypanosomosis

Definition and Causative Agent

Trypanosomosis is an infectious disease caused by several species of the genus *Trypanosoma*, a blood and tissue parasite found in mammals, including humans. The disease is transmitted by biting insects where biological transformation of the parasite occurs. In equines, the most common causative agents are *Trypanosoma evansi* (causing *Surra*) and *Trypanosoma equiperdum* (causing Dourine).

Transmission

Trypanosomosis is a vector-borne disease transmitted mechanically by various species of biting flies. *Surra* is transmitted by *Tabanus* and *Stomoxys* species, while *T. equiperdum* is transmitted sexually. *T. evansi* is transmitted mechanically by blood-sucking flies, vampire bats, and possibly sexually.

Clinical Signs

Equine trypanosomiasis manifests in different forms:

***Surra*:** Affects horses and camels, known as *Surra* in Asia and Africa, meaning rotten. Incubation period: 1-4 weeks, can extend up to 8 weeks. Symptoms: Fever, anaemia, emaciation, urticarial plaques, ulcerative lesions at mucocutaneous junctions, dullness, weakness, anorexia, weight loss, petechial haemorrhages, oedema (ventral parts, udder or scrotum and sheath), low milk and meat yield, poor traction power, increased abortion, and death.

Dourine: Meaning unclean. Incubation period: 8-12 weeks. Three stages:

Stage of Oedema: Urethral or vaginal discharge, oedema of the vagina and prepuce extending to the belly.

Dollar Stage: Circular urticarial plaques (≥3 cm diameter) beneath the skin, mainly on the flank, known as Dollar spots.

Stage of Paralysis: Incoordination and unilateral paralysis of hind legs.

Diagnosis

Diagnosis is based on the prevalence of infection history, presence of biting flies, clinical signs, and laboratory examination of blood and body fluids. Methods include:

Direct Examination: Confirmatory diagnosis by demonstrating the organism in fresh blood smears. In acute infections, organisms are readily detectable; in chronic cases, thick and thin blood smears from lymphoid fluid are studied.

Serological Tests: Include complement fixation test and enzyme-linked immunosorbent assay (ELISA), among others.

Treatment

Three major groups of drugs are used for treating equine trypanosomiasis, divided into curative and preventive drugs:

Quinapyramine: Quinapyramine methyl-sulphate: 8 mg/kg BW S/C. Quinapyramine sulphate and chloride combination for curative/preventive use in horses.

Isometamidium Chloride: Curative and preventive drug from the phenanthridine family, given @ 0.5 mg/kg BW (curative) and 1 mg/kg BW (preventive) I/M or S/C.

Diaminazine Aceturate: Curative drug, mainly for *T. evansi*, is given @ 7.0 mg/kg BW I/M. In horses and dogs, it has limited use due to poor efficacy and tolerance.

Control

Control measures include reducing vector exposure, managing environmental factors conducive to vector breeding, and using preventive chemoprophylactic drugs. Proper sanitation and hygiene, quarantine measures, and monitoring and treating infected animals promptly are also crucial.

Biosecurity

Regular application of insecticides and repellents on horses can reduce the risk of fly bites that transmit *Trypanosoma* species. Topical treatments, such as permethrin or pyrethroid-based sprays, can be effective. Deploy fly traps and insect screens around stabling areas to reduce the number of flies. Screens can be particularly useful in high-risk areas to prevent flies from coming into contact with horses. Reducing the breeding grounds for flies by managing vegetation, removing manure, and improving drainage can help decrease the local fly population. Keep stables clean and dry, removing manure and leftover feed regularly to avoid attracting flies.

Sample collection for Diagnosis

It is essential for diagnosing the disease caused by *Trypanosoma* species, particularly *Trypanosoma evansi* in horses. The samples typically collected include whole blood samples collected in EDTA and serum samples.

Use sterile methods to prevent contamination. Transport samples to the laboratory under refrigeration for accurate testing. Samples are analysed using microscopy, PCR, or serological tests to confirm the presence of *Trypanosoma* species, aiding in the diagnosis and management of equine trypanosomosis.

8.4.3 Toxoplasmosis

Definition and Causative Agent

Toxoplasmosis is a parasitic disease caused by the protozoan *Toxoplasma gondii*. The significance of toxoplasmosis in donkeys lies in its potential zoonotic impact, as humans can acquire infection of *T. gondii* through contact with infected donkeys.

Transmission

Ingestion of Oocysts: Donkeys can ingest oocysts shed by infected cats in contaminated feed, water, or soil. Congenital Transmission: Rarely, pregnant mares can transmit the infection to their foals trans-placentally. Tissue Cysts: Donkeys can become infected by ingesting tissue cysts present in the tissues of other intermediate hosts, although this is not very common.

Clinical Signs

Clinical toxoplasmosis in donkeys is rare and usually subclinical. When clinical signs do occur, they may include - Neurological Symptoms: Ataxia, weakness, circling, and seizures; Respiratory Symptoms: Coughing, nasal discharge, and respiratory distress; Ocular Lesions: Uveitis, retinitis, and other eye infections; Systemic Signs: Fever, lethargy, anorexia, and weight loss.

Diagnosis

Serological Tests: Detection of antibodies against *T. gondii* in the blood using tests such as ELISA or IFAT. *PCR (Polymerase Chain Reaction):* Detection of *T. gondii* DNA in blood, cerebrospinal fluid, or tissue samples. *Clinical Signs and History:* Observing clinical signs consistent with toxoplasmosis in conjunction with a history of exposure to cats or contaminated environments.

Treatment

Antiparasitic Drugs: *Pyrimethamine and Sulfadiazine:* Combination therapy can be used to inhibit the replication of *T. gondii*. Pyrimethamine is usually administered @ 1 mg/kg BW daily, while sulfadiazine is given @ 20 mg/kg BW twice daily. *Trimethoprim-Sulfamethoxazole (TMS):* TMS is an alternative combination that is commonly used due to its availability and effectiveness. *Clindamycin:* Administered @5-10 mg/kg BW twice daily, it is effective against *T. gondii*.

Supportive Care: NSAIDs can reduce inflammation and manage pain. Ensure the horse remains hydrated and receives adequate nutrition during treatment.

Prevention and control

Control Cat Population: Limit access of cats to horse feeding and watering areas to reduce contamination with *T. gondii* oocysts. *Clean Feed and Water:* Ensure that feed and water sources are clean and not contaminated with cat faeces. *Hygiene Practices:* Clean stables and feeding areas regularly to minimize the risk of contamination. *Proper Feed Storage:* Store feed in sealed containers to prevent contamination by rodents or cats. *Routine Serological Testing:* Regular testing of horses, especially those in contact with cats, to monitor for *T. gondii* infection. *Prompt Veterinary Attention:* Seek veterinary care, if clinical signs suggesting toxoplasmosis are observed.

8.5 Parasitic Infestations

8.5.1 Strongylosis

Definition

Strongyles are a group of parasitic nematodes (roundworms) that infect the gastrointestinal tract of horses. They are divided into two main groups: large strongyles (*Strongylus* spp.) and small strongyles (Cyathostomins).

Large Strongyles: *Strongylus vulgaris, Strongylus edentatus, Strongylus equinus*

Small Strongyles: *Cyathostomins* - Over 40 species of small strongyles can infect horses.

Lifecycle: Eggs are passed in the horse's faeces and develop into infective larvae in the environment. Infective larvae (L3) are ingested by grazing horses. *Large Strongyles*: Larvae migrate through various tissues and organs before returning to the intestines. *Small Strongyles*: Larvae penetrate the gut wall and may encyst in the mucosa, later emerging to complete their lifecycle.

Clinical Signs

Grazing - horses usually carry a mixed burden of large and small strongyles and the major clinical signs associated with heavy infections in animals up to 2-3 years of age are unthriftiness, anaemia and sometimes diarrhoea. Marked clinical signs are not very common in older animals, although general performance may be impaired.

Strongylus vulgaris is responsible for causing anaemia, poor condition and performance, varying degrees of colic, temporary lameness, intestinal stasis, rarely intestinal rupture and death. Larval forms of *S. vulgaris* cause arteritis in the mesenteric circulation, resulting in colic and thromboembolic infarction of the large bowel, while the adults cause anaemia and ill-thrift. The pathogenesis of infection with adult worms is associated with damage to the large intestinal mucosa due to the feeding habits of the worms and, to some extent, to the disruption caused by emergence of young adults into the intestine following completion of their parasitic larval development. The gross damage and subsequent loss of blood and tissue fluids is responsible for the unthriftiness, and anaemia associated with intestinal helminthosis in the horse.

Diagnosis

Faecal Egg Count (FEC) - Identifies the number of eggs per gram (EPG) of faeces. *Larval Culture*: Differentiates between large and small strongyle larvae. *Rectal Examination*: May detect arterial damage in case of *Strongylus vulgaris*. *Blood Tests*: Can indicate anaemia or protein loss. *Clinical Signs and History*: Assessing for symptoms and previous parasite control measures.

Treatment

There are a number of broad-spectrum anthelmintics including the benzimidazoles, pyrantel and the

avermectins, milbemycins, which are effective in removing lumen-dwelling adult and larval strongyles, and these are usually marketed as in-feed or oral preparations.

Benzimidazoles: Fenbendazole (10 mg/kg orally for 5 days for encysted small strongyles). Oxibendazole.

Tetrahydropyrimidines: Pyrantel pamoate (6.6 mg/kg orally).

Macrocyclic lactones: Ivermectin (0.2 mg/kg orally); Moxidectin (0.4 mg/kg orally; effective against encysted small strongyles).

Prevention

Regular Deworming Schedule: Follow a strategic deworming program tailored to the specific needs of the horse and the farm's parasite load.

Pasture Hygiene: Implement pasture management practices to reduce parasite contamination.

Faecal Egg Counts: Regularly perform FECs to identify and treat high shedders, and adjust deworming protocols accordingly.

Biosecurity: Quarantine new arrivals and perform FECs before introducing them to the herd.

8.5.2 *Oxyuris equi* Infestation

Definition and Causative Agent

Oxyuris equi, commonly known as the equine pinworm, is a nematode parasite that primarily affects the large intestine and rectum of horses. Infection with *Oxyuris equi,* is extremely common. Although of limited pathogenic significance in the intestine yet the female parasites may cause an intense anal pruritis during egg laying.

Transmission

Horses become infected by ingesting *O. equi* eggs from contaminated feed, water, or surfaces. Eggs are laid around the perianal region and then fall off into the environment, contaminating bedding, stalls, and pasture.

Clinical Signs

The presence of parasites in the intestine rarely causes any clinical signs. However, intense pruritis around the anus (Pruritus Ani) causes the animal to rub, resulting in broken hairs, bare patches and inflammation of the skin over the rump and tail head. Yellowish-white egg masses can be seen around the anus. An infected animal is restless due to the irritation and discomfort caused by the itching. Skin abrasions and secondary bacterial infections may occur due to constant rubbing and scratching.

Diagnosis

This is based on signs of anal pruritis and the finding of greyish yellow egg masses on the perineal skin. The large white long-tailed female worms are often seen in the faeces, having been dislodged while laying their eggs. *O. equi* eggs are rarely found on faecal examination of samples taken from the rectum. Therefore, Tape Test, which involves applying clear tape to the perianal region to collect eggs and examining them under a microscope, is conducted in the diagnosis of pinworm infections. Occasionally, eggs or adult worms can be found in faecal samples, though this is less common than finding eggs on the perianal skin.

Treatment

Anthelmintic Therapy

Ivermectin: Administer @ 0.2 mg/kg BW orally. Ivermectin is effective against both adult and larval stages of *Oxyuris equi*.

Moxidectin: Administer @ 0.4 mg/kg BW orally. Moxidectin is also effective against pinworms.

Pyrantel Pamoate: Administer @ 6.6 mg/kg BW orally. Pyrantel pamoate is effective and often used in rotation with other anthelmintics.

Fenbendazole: Administer @ 5 mg/kg BW orally for five consecutive days. Fenbendazole is effective but may require repeated dosing for complete eradication.

Topical Treatments

Anti-inflammatory Creams: To reduce itching and inflammation around the perianal region.

Antiseptic Solutions: To clean and soothe irritated skin, preventing secondary infections.

Environmental Management

Regular Cleaning: Thoroughly clean and disinfect stables, stalls, and paddocks to remove eggs.

Frequent Bedding Changes: Replace bedding frequently to reduce the risk of reinfection.

Tail Washing: Regularly wash the perianal area and tail to remove eggs and soothe irritation.

Preventive Measures

Routine Deworming Program: Implement a strategic deworming schedule, rotating between different classes of anthelmintics to prevent resistance.

Environmental Sanitation: Maintain a clean and sanitary environment to reduce the risk of infection.

Quarantine New Arrivals: Isolate and treat new horses before introducing them to the herd.

Regular Health Checks: Conduct regular health checks and faecal exams to monitor for pinworm infestations.

By following these treatment and preventive measures, horse owners and veterinarians can effectively manage and control *Oxyuris equi* infestations, ensuring the health and well-being of their horses.

8.5.3 Botfly Infestation (*Gasterophilus* spp.)

Definition and Causative Agent

Botflies (*Gasterophilus* spp.) are parasitic flies that affect horses, causing botfly infestation. The larvae of these flies are internal parasites of the gastrointestinal tract of horses. The most common species include *Gasterophilus intestinalis, Gasterophilus nasalis*, and *Gasterophilus haemorrhoidalis*.

Lifecycle

Adult botflies lay eggs on the horse's coat, especially on the legs, shoulders, and around the mouth. The larvae hatch and migrate to the mouth, where penetrate the mucous membranes and then move to the stomach and intestines. After several months, the larvae pass out with the faeces and pupate in the soil. Adults emerge from the pupae, and the cycle begins again.

Transmission

Botfly eggs are laid on the horse's body, primarily on the hairs of legs and around the mouth. Transmission is through ingestion when the horse ingests the larvae by licking or biting at the areas where the eggs are attached.

Clinical Signs

Behavioural Signs

Licking or biting at legs and other infested areas.

Restlessness and irritation.

Gastrointestinal Signs

Mild to severe gastritis or ulcers due to larvae attaching to the stomach lining.

Colic symptoms and digestive disturbances.

Oral Signs

Lesions in the mouth and throat due to migrating larvae.

Excessive salivation and mouth irritation.

Weight Loss

Poor condition and weight loss due to gastrointestinal distress and nutrient absorption issues.

Diagnosis

Visual Inspection: Checking for botfly eggs on the horse's coat, especially on the legs, shoulders, and around the mouth. Oral Examination: Observing lesions or larvae in the mouth and throat. Faecal Examination: Occasionally, botfly larvae can be found in the faeces.

Treatment

Anthelmintics: Ivermectin - Administer @ 0.2 mg/kg BW orally. Ivermectin is highly effective against botfly larvae in the stomach and intestines. Moxidectin - Administer @ 0.4 mg/kg BW orally. Moxidectin is another effective treatment for botfly larvae.

Topical Treatments: Insecticides - Use topical insecticides on areas where botfly eggs are attached to the coat of a horse. Products containing permethrin or pyrethrin can be effective in killing eggs before they hatch.

Manual Removal: Bot Knife or Grooming Tool - Manually remove botfly eggs from the horse's coat using a bot knife or similar grooming tool. Focus on the legs, shoulders, and areas around the mouth where eggs are commonly found.

Supportive Care: Hydration and Nutrition - Ensure the horse has access to clean water and a balanced diet to support overall health and recovery. Monitoring for Complications - Monitor for signs of gastric irritation or ulcers, and consult a veterinarian, if severe symptoms arise.

Environmental Management:

Stable Hygiene: Regular Cleaning - Clean and disinfect stables, stalls, and paddocks regularly to reduce the presence of botfly eggs and larvae.

Bedding Management - Replace bedding frequently to minimize contamination.

Pasture Management - Rotate Pastures: Rotate grazing areas to reduce the buildup of botfly larvae in the environment.

Manure Management: Properly dispose of manure to interrupt the botfly lifecycle.

Preventive Measures

Fly Control: Apply fly repellents to horses during botfly season to reduce the likelihood of egg laying. Use fly traps and other control methods to reduce the population of adult botflies.

Regular Deworming: Implement a regular deworming schedule, particularly during and after botfly season, using effective anthelmintics like ivermectin or moxidectin. Inspect horses daily for botfly eggs and remove them promptly to prevent larvae from hatching and being ingested.

8.5.4 Tapeworms Infestation

Definition and Causative Agent

Equine tapeworms, primarily *Anoplocephala perfoliata*, are parasitic flatworms that inhabit the intestines of horses. These tapeworms can cause various gastrointestinal issues, including colic, weight loss, and diarrhoea.

Transmission

Tapeworms require an intermediate host, the oribatid mite, which is found in soil and on pasture. Horses become infected by ingesting mites that contain the tapeworm larvae while grazing.

Clinical Signs

In most infections, there are no clinical signs. However, when there are significant pathological changes in the intestine there may be unthriftiness, enteritis and colic, particularly spasmodic or ileocecal colic. There is a gradual weight loss despite adequate nutrition, intermittent or chronic diarrhoea, dull coat due to nutrient absorption issues, anaemia in severe cases, due to intestinal damage and nutrient loss. Perforation of the intestine will prove rapidly fatal.

Diagnosis

Faecal Examination: Detection of tapeworm eggs using faecal flotation methods; however, this method has limited sensitivity.

Treatment

Anthelmintics:

Praziquantel: Administer @ 1 mg/kg BW. Praziquantel is highly effective against tapeworms and is often combined with other deworming agents like ivermectin or moxidectin for broad-spectrum control.

Pyrantel Pamoate: Administer at a double dose of 13.2 mg/kg BW. Effective but less commonly used compared to praziquantel.

Combination Products:

Ivermectin/Praziquantel: Combines the broad-spectrum efficacy of ivermectin with the tapeworm-specific action of praziquantel.

Moxidectin/Praziquantel: Another combination product providing comprehensive parasite control.

Preventive Measures:

Regular Deworming Program: Implement a strategic deworming schedule based on the local prevalence of tapeworms and other parasites. Use combination anthelmintics (e.g., ivermectin/praziquantel) at least once or twice a year, typically in the spring and fall.

Pasture Management: Rotate grazing areas to reduce the accumulation of oribatid mites. Avoid overgrazing and maintain clean pastures to minimize exposure to mites.

Manure Management: Regularly remove and properly dispose of manure from pastures to reduce the risk of tapeworm egg contamination. Compost manure thoroughly before spreading it on fields.

Environmental Control: Reduce mite populations by maintaining dry, well-drained pastures and avoiding excessive moisture.

8.5.5 *Parascaris equorum* infestations

Definition and Causative Agent

Parascaris equorum, commonly known as the equine roundworm, is a parasitic nematode that primarily affects foals and young horses. Adult roundworms are large, white, and can measure up to 40 cm in length.

Lifecycle:

Egg Stage: Eggs are passed in the faeces and become infective after 10-14 days in the environment.

Larval Stage: Foals ingest infective eggs from contaminated feed, water, or surfaces. The larvae hatch in the intestines, migrate through the liver and lungs, and are coughed up and swallowed back into the intestines, where they mature into adults.

Adult Stage: Adult worms reside in the small intestine, where they lay eggs that are excreted in the faeces, continuing the cycle.

Transmission:

Transmission is through faecal-oral route. Horses ingest infective eggs from contaminated feed, water, or surfaces.

Clinical Signs

During the migratory phase of experimental infections, up to four weeks following infection, the major signs are frequent coughing accompanied in some cases by a greyish nasal discharge although the foals remain bright and alert. Light intestinal infections are well tolerated, but moderate to heavy infections will cause unthriftiness in young animals with poor growth rates, dull coats and lassitude. Other clinical signs are poor growth and weight loss due to nutrient competition between the host and the parasite, pot-bellied appearance which is common in heavily infected foals, diarrhoea and intestinal obstruction or impaction which in severe cases cause colic and potential life-threatening situations.

Diagnosis

Faecal Examination: Detection of characteristic eggs through faecal flotation methods.

Clinical Signs and History: Observing the typical signs in young horses, especially those with a history of inadequate deworming programs.

Treatment

Fenbendazole: Administer @ 5 mg/kg BW orally for 5 consecutive days. Effective and safe for young foals. Ivermectin: Administer @ 0.2 mg/kg BW orally. Effective against both larval and adult stages but may cause complications in heavy infestations due to rapid worm kill-off. Pyrantel Pamoate: Administer @ 6.6 mg/kg BW orally. Effective and safe for young foals. Oxibendazole: Administer @ 10 mg/kg BW orally. Effective against adult worms.

Management of Heavy Infestations

Gradual Deworming: In heavy infestations, consider using a low-dose anthelmintic initially to reduce the risk of intestinal blockage from dead worms. Follow up with a full-dose treatment after a few weeks. Supportive Care: Provide supportive care, including fluid therapy and anti-inflammatory medications, as needed for foals with severe infestations and signs of colic.

Preventive Measures

Regular Deworming Program: Implement a strategic deworming schedule for foals starting at 2-3 months of age and continuing every 6-8 weeks until they are 1-year-old. Rotate between different classes of anthelmintics to prevent resistance.

Pasture Management: Rotate pastures to reduce the buildup of infective eggs in the environment. Avoid overgrazing and maintain clean, dry pastures to minimize exposure.

Manure Management: Regularly remove and properly dispose of manure from paddocks and stables to reduce environmental contamination. Compost manure thoroughly before spreading it on fields.

8.5.6 Microfilariasis

Definition and Causative Agent

Microfilariae in horses are the larval stages of filarial nematodes, which can cause various parasitic infections. The most commonly known filarial nematode affecting horses is *Setaria equina*, although others like *Onchocerca* species can also infect horses. These parasites are transmitted by biting insects such as mosquitoes and midges.

Lifecycle:

Adult Worms: Live in the body cavities or tissues of the host (*e.g.*, abdominal cavity for *Setaria equina*).

Microfilariae: Released into the bloodstream or tissue fluids by the adult worms.

Intermediate Host: Biting insects ingest microfilariae during a blood meal.

Larval Development: Microfilariae develop into infective larvae within the insect vector.

Transmission: Infective larvae are transmitted to a new host when the insect bites again, continuing the cycle.

Transmission

Vector-Borne: Transmitted through the bites of infected mosquitoes, midges, or other blood-sucking insects.

Clinical Signs

Microfilariasis in horses is caused by filarial worms, leading to the presence of microfilariae in the blood or tissues. The clinical signs can vary depending on the species of filarial worm involved and the tissues affected. Common clinical signs include *Skin Lesions* – Pruritus (itchiness), dermatitis, and non-healing sores, often on the ventral midline or limbs. *Ocular Involvement*: Conjunctivitis, uveitis, and in severe cases, blindness, if the microfilariae invade the eye. *Neurological Symptoms*: In rare cases, microfilariae can migrate to the brain or spinal cord, causing neurological symptoms. Ataxia, head tilt, and other signs, if the central nervous system is affected. *Systemic Signs*: Fever, lethargy, and general malaise.

Diagnosis

Blood Smear: Microscopic examination of blood smears can reveal the presence of microfilariae.

Skin Biopsy: A skin biopsy or skin scraping can be examined for microfilariae, especially in cases of dermatitis.

Treatment

Anthelmintics: Ivermectin: Administered @ 0.2 mg/kg BW orally; effective against microfilariae and some adult worms. Moxidectin: Administered @ 0.4 mg/kg BW orally; effective against microfilariae.

Anti-Inflammatory Medications: NSAIDs (Non-Steroidal Anti-Inflammatory Drugs) - to manage inflammation and discomfort associated with the infection.

Supportive Care: Topical Treatments: For skin lesions and dermatitis, topical anti-inflammatory and antiparasitic treatments may be used. Ocular Care: Specific treatments for eye infections, including anti-inflammatory eye drops or ointments.

Preventive Measures

Vector Control:

Insect Repellents: Regular application of insect repellents to horses, especially during peak insect activity periods.

Stable Management: Use of screens, fans, and other measures to reduce the presence of biting insects in stables.

Environmental Control: Eliminate standing water and other breeding sites for mosquitoes and midges.

Strategic Deworming Program: Implement a regular deworming schedule using anthelmintics effective against filarial parasites.

8.5.7 Cutaneous Habronemiasis (Skin sores)

Definition and causative agent

Cutaneous habronemiasis is caused by *Habronema* and *Draschia* larvae, which are deposited by infected flies in existing wounds.

Transmission

Cutaneous habronemiasis occurs when intermediate hosts, *i.e.*, *Musca* spp. flies deposit infective larvae on skin, open wounds, or chronically wet areas.

Life cycle: Eggs or L1, are passed in the faeces and the L1 are ingested by the larval stages of various muscid flies including *Musca, Stomoxys* and *Haematobia* which are often present in faeces. Development to L3, occurs synchronously with the development to maturity of the fly - the intermediate host. When the fly feeds around the mouth of the horse, the larvae pass from its mouthparts on to the skin and are swallowed. Alternatively, infected flies may be swallowed whole. Development to adult takes place in the glandular area of the stomach in approximately two months. When *Habronema* larvae are deposited on a skin wound or around the eyes, they invade the tissues but do not complete their development.

Clinical Signs

Typical skin wounds (ocular and cutaneous habronemiasis) which are not healing and containing larvae. During the early stages, there is intense itching of the infected wound. These wounds tend to heal spontaneously during the period when flies are inactive (winter, dry season) but often recur in subsequent seasons when fly activity starts again (summer, wet season).

Diagnosis

Non-healing, reddish brown, greasy skin granuloma that contain rice-grain-size, calcified material is indicative for cutaneous habronemiasis. The larvae, recognized by spiny knobs on their tails, may be found in material from these lesions. Gastric infection is not easily diagnosed since *Habronema* eggs and larvae are not readily demonstrable in the

faeces by routine techniques.

Treatment

Cutaneous lesions are best treated with ivermectin. Ivermectin (200 µg/kg, IM) and moxidectin are the treatments of choice for adult *Habronema* and *Draschia* species. Ivermectin is approved for the treatment of summer sores caused by larvae of *Habronema* and *Draschia* species. An insecticide (Lindane) ointment should afterwards be applied to the wound to prevent reinfection. Trichlorfon (25 mg/kg, IV) administered in 1 litre isotonic saline solution cured summer sores within 30 days. Premedication with atropine was necessary. Organophosphates (coumaphos, lindane) applied topically to the abraded surface may kill the larvae.

Insect repellents showed benefit but radiation therapy and cryosurgery are to be used in more chronic cases. Any measures taken to prevent injuries and to control fly populations will be beneficial.

Control

Any measures taken to prevent injuries and to control fly populations will be beneficial. Fly prevention by stacking manure and using insecticides. Skin wounds should be treated with fly repellents (citronella oil, diethyltoluamide and diethyl phthalate). Cypermethrin (0.1 percent solution, using 150-250 ml per animal) and pyrethrins (0.01 percent) are highly effective to control flies on horses.

8.5.8 Equine Onchocerciasis

Definition and Causative Agent

Equine onchocerciasis is a parasitic disease caused by the nematode *Onchocerca cervicalis*, which affects horses worldwide. This condition primarily targets the ligamentum nuchae and, less frequently, the suspensory ligaments and flexor tendons of the lower limbs.

Transmission

Vectors: The disease is transmitted by midges (*Culicoides* species).

Hosts: Adults of *Onchocerca cervicalis* reside in the nuchal ligament, while their microfilariae are distributed in the skin and other connective tissues, including the ocular conjunctiva.

Lifecycle: The lifecycle is indirect, involving the ingestion of microfilariae by midges from infected horses. The larvae develop to the infective third-stage in the midge, which then transmits the larvae to another horse during feeding. These larvae migrate to the nuchal ligament to mature.

Clinical Signs

Many horses with *O. cervicalis* show no clinical signs. However, in some cases, the microfilariae cause pruritic skin lesions like inflammation, scaling, and depigmentation, often worse in the summer.

Sweet itch or Queensland itch – hypersensitivity to midge saliva, causing pruritic lesions along the caudal back and tail head is distinct from cutaneous onchocerciasis.

Ligamentum nuchae lesions – painless, diffuse swellings that may become palpable lumps and then regress, leaving a calcified focus.

Open purulent lesions, known as fistulous withers, can also occur. Lower Limb Lesions include soft painless swellings that develop into small fibrous nodules.

Diagnosis

Diagnosis of onchocerciasis depends on the finding of microfilariae in skin biopsy samples. In most species the microfilariae are concentrated in the preferred feeding sites of the vectors, which for *Simulium* spp. and *Culicoides* spp. are usually the shaded lower parts of the trunk. It is usually recommended that sample should be taken from the region of linea alba. The piece of skin is placed in warm saline, teased to allow emergence of the microfilariae and is incubated for six hours or more. The microfilariae are readily recognized by their sinuous movements in a centrifuged sample of the saline.

Treatment

Earlier treatment consisted of daily administration of diethylcarbamazine over a period as a microfilaricide, but now a single dose of ivermectin is found highly efficient in this respect, although the dying microfilariae may provoke local tissue reactions. In equine ventral midline dermatitis, local treatment with synthetic pyrethroids controls hornflies and aids resolution of the lesions.

Ivermectin: A single dose (0.2 mg/kg BW orally) is highly effective against microfilariae and some adult worms, though it may cause local tissue reactions as

the microfilariae die.

Moxidectin: Administered @ 0.4 mg/kg BW orally, effective against microfilariae.

Anti-inflammatory Medications: NSAIDs to manage inflammation and discomfort.

Topical Treatments: Anti-inflammatory and antiparasitic treatments for skin lesions.

Control and Prevention

Vector Control:

Insect Repellents – regular application of insect repellents to horses, especially during peak insect activity periods.

Stable Management – use of screens, fans, and other measures to reduce the presence of biting insects in stables.

Environmental Control – Eliminate standing water and other breeding sites for mosquitoes and midges.

Strategic Deworming Program: Implement a regular deworming schedule using anthelmintics effective against filarial parasites.

8.5.9 *Thelazia lacrymalis*

Thelazia lacrymalis is a parasitic nematode that affects the eyes of horses. This worm is transmitted by face flies (*Musca autumnalis*), which deposit the larvae into the conjunctival sac of the horse while feeding on tears. The adult worms reside in the tear ducts, conjunctival sac, and under the eyelids, causing irritation and inflammation.

Clinical Signs

Conjunctivitis: Inflammation of the eye's conjunctiva.

Excessive Tearing (Epiphora): Due to irritation.

Swelling and Redness: Around the eyes.

Discomfort and Sensitivity to Light: Photophobia. *Corneal Ulcers*: In severe cases.

Diagnosis

Visual Inspection – The worms can often be seen directly in the eye.

Microscopic Examination: Examination of eye discharge for larvae.

Treatment

Mechanical Removal: The worms can be mechanically removed carefully using forceps.

Antiparasitic Medication: Systemic administration of ivermectin or moxidectin is effective against the adult worms.

Topical Antibiotics: Apply topical antibiotics to prevent or treat secondary bacterial infections resulting from the damage caused by the worms.

Prevention

Fly Control: Use of fly repellents, fly masks, and maintaining clean environments to reduce the horse's exposure to flies.

Regular Eye Examinations: Especially in endemic areas, regular check-ups can help catch infestations early before they cause significant damage.

8.5.10 Mite Infestation (Mange)

Mange in horses is caused by various species of parasitic mites that infest the skin, leading to severe itching, hair loss, and skin lesions. Mange mites can be highly contagious and spread quickly among horses, causing significant discomfort and health issues.

Types of Mange Mites in Horses:

Sarcoptic Mange (*Sarcoptes scabiei* var. *equi*): Burrowing mites that create tunnels in the skin, causing intense itching and irritation.

Psoroptic Mange (*Psoroptes equi*): Surface-dwelling mites that cause itching, crusting, and scabbing on the skin.

Chorioptic Mange (*Chorioptes bovis*): Mites that primarily affect the lower legs, causing dermatitis and itching.

Demodectic Mange (*Demodex equi*): Less common in horses; mites live in hair follicles and sebaceous glands, causing localized or generalized mange.

Clinical Signs

Intense itching and scratching, hair loss, redness and inflammation of the skin, crusty scabs and lesions, thickened skin, weight loss and poor condition (in severe cases).

Diagnosis

Clinical Examination: Observation of clinical signs and affected areas.

Skin Scrapings: Microscopic examination of deep skin scrapings to identify mites and their eggs.

Treatment

1. Topical:

Lime Sulphur Dips: Effective for various types of mange mites. Usually applied once a week for several weeks.

Permethrin or Pyrethrin Sprays: Commonly used insecticides. Applied according to the product instructions.

2. Systemic:

Ivermectin: Effective against Sarcoptes and Psoroptes mites. Dosage: Typically administered at 0.2 mg/kg BW. Given orally or via injection, repeated after 14 days if needed.

Moxidectin: An alternative to ivermectin, effective against similar mites. Dosage and administration as per veterinary prescription.

Doramectin: Another systemic option. Dosage and administration as per veterinary prescription.

3. **Environmental Management:**

Cleaning and Disinfecting: Thoroughly clean stables, grooming tools, and equipment. Use appropriate disinfectants to kill mites in the environment. Isolation: Isolate infected horses to prevent the spread of mites. Treat all horses that have been in contact with the infected animal.

4. **Supportive Care:**

Soothing Shampoos and Conditioners: Use products that alleviate itching and promote skin healing. Avoid harsh chemicals that may irritate the skin further. Nutrition and Overall Health: Ensure a balanced diet to support the horse's immune system. Provide vitamins and minerals as needed.

Treatment Schedule Example

Week 1:

Day 1: Administer ivermectin orally or via injection.

Day 2-7: Apply lime sulphur dip as per instructions.

Week 2:

Day 8: Reapply lime sulphur dip.

Day 9-14: Monitor the horse and provide supportive care.

Week 3:

Day 15: Administer a second dose of ivermectin if needed.

Day 16-21: Apply lime sulphur dip if necessary.

Prevention

Regular Grooming – early detection and management of skin issues.

Maintain Hygiene – clean living conditions and equipment.

Quarantine New Arrivals – prevent introducing mites to the herd.

Regular Veterinary Check-Ups – monitor for early signs of infestations.

8.6 Non-Infectious Diseases

8.6.1 Laminitis

Definition

Laminitis is the inflammation of the digital laminae of the hoof, which are structures that provide suspension to the digital bones within the hooves and absorb shock during locomotion.

Causes

Common causes of laminitis include high carbohydrate diet, obesity, pyometra or metritis, retained placenta, loss of sleep during transportation, water deprivation and dehydration, excessive concussion of the hoof (road founder), large water intake after overheating (water founder), excess weight bearing due to injury in the opposite foot, and enteritis, colitis, or endotoxins absorption from the intestine

Pathogenesis

Excess carbohydrates that are not digested in the foregut move to the hindgut, where they ferment leading to lactic acid proliferation and acidosis.

Acidosis kills beneficial bacteria and increases toxin-producing harmful bacteria.

Toxins absorbed due to increased gut permeability cause endotoxemia and inflammation in the lamina of the feet.

Fresh spring grasses high in fructan can lead to laminitis by causing bowel microflora imbalance and endotoxin production.

Systemic bacterial infections, retained placental membranes, and metritis in mares can also release endotoxins.

Long toes working on hard ground, certain drugs

(especially corticosteroids), and exposure to certain agrichemicals can also cause laminitis.

Clinical Signs

The clinical signs include repeated shifting of affected feet and lameness, preference for a sitting position. hind legs positioned under the body and forelegs stretched out if the front hooves are affected, bilateral systemic laminitis affecting any feet, more common in the front feet, and possible rotation or sinking of the third digit, leading to abscesses within the hoof capsule

Types of Laminitis

Rotation: Affected toe area due to less severe laminae damage. The deep digital flexor tendon and the horse's weight pull the coffin bone away from the hoof wall, leading to rotation. Severe cases may see the bone tip penetrate the sole.

Sinking: Complete failure of inter-digitation between sensitive and insensitive laminae around the entire hoof wall. Complete separation of hoof wall from the rest of the hoof, with possible pus discharge at the white line or coronary band. The coffin bone may penetrate the sole.

Diagnosis

Diagnosis is based on history and clinical signs as well as radiography to assess bone rotation and sinking

Treatment

Immediate Management

Rest: Provide complete rest to the horse to reduce stress on the hooves.

Cryotherapy: Apply cold water or ice to the hooves to reduce inflammation and pain, especially in the initial stages.

Supportive Hoof Care: Use foam or soft bedding to cushion the hooves. Hoof pads or supportive shoes can help distribute weight more evenly.

Medications

NSAIDs: Phenylbutazone (4.4 mg/kg twice daily initially, then 2.2 mg/kg twice daily) or flunixin meglumine (1.1 mg/kg IM once daily) to reduce pain and inflammation.

Hyaluronic acid: Sodium hyaluronan can be administered intra-articularly (20 mg) or intravenously (40 mg) to improve joint fluid elasticity and lubrication.

Polysulphated Glycosaminoglycans: Oral supplements of glucosamine chondroitin sulphate for 2-6 weeks can improve cartilage health and inhibit degrading enzymes.

Pentoxifylline and Isoxsuprine: These drugs improve peripheral circulation and have anti-inflammatory effects.

Dietary Management

Caloric Restriction: Reduce caloric intake to manage weight.

Control Dietary Non-Structural Carbohydrates (NSC): Essential for insulin-resistant animals.

Levothyroxine or Metformin: May be necessary for animals with obesity or insulin resistance that do not respond to conventional treatment.

Surgical Interventions

Deep Digital Flexor Tenotomy: May be performed in severe cases to relieve tension on the laminae.

Hoof Trimming and Corrective Shoeing: Regular trimming and appropriate shoeing to support the hoof structure and reduce pain.

Long-Term Management

Weight Management: Maintain a healthy weight to reduce stress on the hooves.

Regular Hoof Care: Ensure proper and regular hoof trimming.

Avoiding Trigger Factors: Monitor and manage diet, avoid sudden changes, and provide appropriate exercise.

8.6.2 Osteoarthritis

Definition

Osteoarthritis in horses, also known as mechanically induced arthritis, is a slow-progressing degenerative disease affecting synovial joints. It results in joint pain and reduced functionality, characterized by the degeneration of cartilage, sclerosis of subchondral bone, osteophyte formation, and inflammation of synovial membranes.

Pathogenesis

Chronic trauma to the joint is a common etiology in osteoarthritis.

Repetitive injuries damage the cartilage, which does not recover once damaged.

Overuse and conformational deformities (e.g., hoof deformities due to laminitis) lead to inappropriate pressure on joints like the fetlock, resulting in osteoarthritis and fibrosis.

Clinical Signs

Lameness and stiffness. Joint swelling and heat. Reduced range of motion. Pain on palpation of affected joints. Decreased performance and reluctance to move.

Diagnosis

Clinical signs and history. Radiographs to assess joint changes. Ultrasound for soft tissue evaluation. Synovial fluid analysis. CT scans for detailed imaging.

Treatment

Removal of Etiological Factors:

Reduce undue pressure on joints

Address trauma or overuse

Provide rest from work or racing

Medications:

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs):

Phenylbutazone: Loading dose of 4.4 mg/kg BW twice a day for the initial two days, then 2.2 mg/kg BW or less, IM, twice a day for the remaining treatment period. Excessive dosing can cause gastric ulceration, renal damage, and vascular thrombosis.

Flunixin Meglumine: 1.1 mg/kg BW IM/IV once daily.

Corticosteroids: Intra-articular corticosteroids can be used for reducing inflammation within the joint. Examples include methylprednisolone, triamcinolone, and betamethasone.

Hyaluronic Acid: Administer intra-articularly (20 mg) or intravenously (40 mg) to replace depleted hyaluronate, restore elasticity of synovial fluid, and provide lubrication to the articular tissues.

Polysulphated Glycosaminoglycans (PSGAGs): Oral supplements of glucosamine chondroitin sulphate for 2-6 weeks. They form stable complexes with fibronectin and collagen fibres in the cartilage and inhibit degrading enzymes.

Chondroprotective Agents: Pentosan polysulphate and other chondroprotective agents to support cartilage health.

Bisphosphonates: These can help manage bone changes and pain, particularly in cases involving subchondral bone pain.

Physical Therapy:

Rest and Controlled Exercise: Provide periods of rest followed by controlled, gradual reintroduction of exercise to strengthen muscles and support joints.

Hydrotherapy: Swimming or water treadmills can help reduce joint stress while maintaining fitness.

Cryotherapy: Applying cold packs to the affected joints to reduce inflammation and pain.

Nutraceuticals and Supplements

Omega-3 Fatty Acids: Anti-inflammatory properties can help manage arthritis.

Antioxidants: Vitamin E and other antioxidants to reduce oxidative stress in the joints.

Joint Supplements: Glucosamine, chondroitin sulphate, and MSM (methylsulphonyl methane) to support joint health.

Surgical Interventions:

Arthroscopic Surgery: To remove loose cartilage or bone fragments and debride damaged joint surfaces.

Joint Fusion (Arthrodesis): In severe cases, surgical fusion of the joint may be considered to alleviate pain.

Management and Preventive Measures

Weight Management: Maintain an optimal weight to reduce stress on joints.

Proper Hoof Care: Regular trimming and corrective shoeing ensure proper alignment and reduced joint stress.

Avoid Overexertion: Avoid overworking the horse, particularly on hard surfaces or with high-impact activities.

8.6.3 Equine Exertional Rhabdomyolysis (Monday Morning Disease)

Definition

Equine exertional rhabdomyolysis (er), also known as azoturia, tying up, or Monday morning disease, is a condition characterized by severe muscle cramps

in horses, primarily affecting the muscles of the hindquarters and back. It typically occurs after a period of rest followed by exercise.

Pathogenesis

During rest, the horse's energy requirements decrease, leading to glycogen storage in muscles. Upon resuming exercise, the high energy demand cannot be met by aerobic respiration, resulting in glycogen conversion to lactic acid. This lactic acid buildup damages muscle tissues, releasing myoglobin, which can cause myoglobinuria and potentially lead to kidney failure.

Clinical Signs

Severe muscle cramps. Stiffness and pain. Sweating and labored breathing in severe cases. Reluctance to move forward, especially in riding horses. Hardness and pain in the muscles upon palpation. Recumbency in severe cases.

Diagnosis

Laboratory tests: Elevated levels of creatine kinase (CK), aspartate transaminase (AST), and serum creatinine.

Treatment

Immediate Rest:

Provide rest until the horse recovers.

Use slings for recumbent horses to avoid decubital ulcers.

Pain and Inflammation Management:

NSAIDs such as phenylbutazone (1-2 mg/kg BW twice daily) for pain relief and inflammation reduction.

Thiamine Supplementation:

Administer 4 mg/kg BW intramuscularly for 5 to 7 days to enhance lactic acid utilization.

Fluid Therapy:

Administer normal saline (5 to 10 litre IV daily) to maintain kidney function.

Antibiotic Coverage:

To prevent secondary bacterial infections.

Supportive Care:

Hot towelling of affected muscles to provide relief.

Prevention

Proper conditioning and gradual increase in exercise intensity.

Adequate warm-up and cool-down periods.

Maintaining a balanced diet with appropriate carbohydrate levels.

Avoiding sudden changes in exercise routines.

8.6.4 Diarrhoea in Adult Horses

Definition

Diarrhoea in adult horses can result from various causes and may require monitoring even if it is often self-limiting.

Bacterial Diarrhoea: Common bacteria include *Salmonella* and *E. coli*. Treat with antibiotics like sulphadimidine and biotrim, and supportive fluid therapy. *Clostridium difficile* can cause acute, fatal colitis after antibiotic treatment.

Sand Ingestion: In horses grazing on sandy pastures, sand ingestion causes colic and diarrhoea. Preventive use of psyllium husk is beneficial.

Intestinal Parasites: Strongyles cause diarrhoea, controlled with broad-spectrum anthelmintics.

Viral Diarrhoea: Commonly caused by rotavirus in foals.

Feed/Fodder: Sudden feed changes or excess lush green fodder can induce diarrhoea.

Miscellaneous: Mycotoxin-contaminated feed, irritant chemicals, and poisons like organophosphorus.

Treatment

Supportive Care: Fluids and Electrolytes: Intravenous Fluids– Normal saline or Ringer's lactate solution @ 2-4 liter per 100 kg BW per day to maintain hydration and electrolyte balance. Oral Rehydration Solutions– If the horse is not severely dehydrated and can drink.

Medications: NSAIDs: Flunixin meglumine (1.1 mg/kg IV or IM once daily) or meloxicam for pain and inflammation. Antibiotics– Only if a bacterial infection is confirmed or highly suspected. Sulphonamides: Potentiated sulphonamides (trimethoprim-sulphonamide) are commonly used. Enrofloxacin– Effective against Gram-negative bacteria. Metronidazole– For *Clostridium difficile* infections.

Adsorbents: Activated Charcoal: 1 g/kg BW orally to absorb toxins. Bismuth Subsalicylate: 10-15 ml per 100 kg BW orally every 4-6 hours.

Anti-Diarrheal Agents: Kaolin-Pectin: To coat and soothe the gastrointestinal tract.

Probiotics and Prebiotics: *Lactobacillus* spp. and *Saccharomyces boulardii*– To restore and maintain gut flora balance.

Dietary Management: Gradual Reintroduction of Feed: Start with easily digestible feeds, such as grass hay. Avoid high-concentrate feeds initially. Gradually reintroduce normal diet over several days. Avoid Sudden Dietary Changes– Maintain a consistent feeding schedule. Ensure high-quality feed free of Mold and toxins.

Environmental Management: Reduce Stress– Minimize changes in environment and routines. Good Hygiene– Regular cleaning of feeding and watering equipment. Proper Deworming– Regular deworming program to control parasitic infections.

Specific Treatments for Underlying Causes: Sand Colic– Psyllium husk (50 g daily) to help clear sand from the gut. Toxin Exposure– Identify and remove the source of the toxin. Use adsorbents like activated charcoal.

Prevention

Regular Deworming: Follow a deworming schedule to control parasitic infections.

Stable balanced Diet: Avoid sudden changes in feed and ensure a balanced diet.

Clean Water: Provide clean and fresh water at all times.

Hygiene: Maintain clean feeding areas and equipment.

Stress Reduction: Minimize stressors, especially during travel and environmental changes.

8.6.6 Colic

Definition

Colic refers to abdominal pain in horses, often related to disturbances in the gastrointestinal tract, particularly the colon. It is a clinical sign rather than a diagnosis.

Etiology

Dietary changes, especially in hay or straw

Lack of regular deworming programs

Horses at pasture have a lower risk than stall-fed horses

History of previous colic, recent changes in stabling conditions, diet, or activity level

Large amounts of concentrated high-energy feeds, low forage rations, low feeding frequency, and spoiled feed

Low plasma electrolyte levels in exercising horses

Environmental factors, such as sudden weather changes or stress from travel

Clinical Signs

Pawing, scraping, stretching, frequent attempts to urinate; Flank watching, biting the stomach, pacing; Repeated lying down and rising, rolling, groaning, teeth grinding.

Indicators – Pulse rate, pulse character, jugular vein filling, skin temperature, mucous membrane colour, capillary refill time, sweating, depression, skin turgor. Indicators – Pulse rate, pulse character, jugular vein filling, skin temperature, mucous membrane colour, capillary refill time, sweating, depression, skin turgor;

Diagnostic tools – Rectal palpation, passing a stomach tube, abdominal distension, auscultation, faecal examination.

Diagnosis - Clinical and Laboratory Examination

History: Record clinical signs, progression, elapsed time, treatments, diet changes, activities, deworming history.

Cardiovascular Parameters: Monitor heart rate, mucous membrane color, and packed-cell volume (PCV) to assess hydration and circulatory status.

Rectal Examination: Diagnose conditions of the large intestine and detect dilated small intestinal loops.

Naso-Gastric Intubation: Relieves pressure and fluid buildup in the stomach.

Abdominocentesis: Assess peritoneal fluid for indications of infarction or leucocyte presence.

Abdominal Distension: Indicates conditions affecting the large intestines.

Auscultation: Increased gut sounds suggest spasmodic colic; decreased sounds suggest

impaction, ileus, or strangulating obstruction.

Faecal Examination: Check for sand content, color, smell, and consistency.

Blood Tests: Assess hydration, electrolyte balance, and organ function.

Treatment

Immediate Management: Pain Relief: NSAIDs– Flunixin meglumine (1.1 mg/kg IV or IM once daily), phenylbutazone (2.2-4.4 mg/kg orally or IV). Sedation and Analgesia – Xylazine (0.3-0.5 mg/kg IV or IM), detomidine (0.01-0.02 mg/kg IV), butorphanol (0.01-0.02 mg/kg IV).

Fluid Therapy: IV Fluids– Ringer's lactate or normal saline (5-10 litre initially, adjust based on hydration status). Oral Fluids– Administer via naso-gastric tube if no reflux is present.

Gastrointestinal Support:

Mineral Oil or Liquid Paraffin: 2-4 litre via naso-gastric tube to help with impactions.

Laxatives: Psyllium husk (50 g daily) for sand impactions.

Antibiotics: If bacterial infection is suspected or confirmed. Broad-Spectrum Antibiotics like Trimethoprim-sulpha (30 mg/kg orally), penicillin (22,000 IU/kg IM), gentamicin (6.6 mg/kg IV).

Antispasmodics: Buscopan (N-butylscopolammonium bromide) – 0.3 mg/kg IV to relieve spasmodic colic.

Surgery: Emergency surgery is required for severe cases such as torsion, strangulating obstruction, or unresponsive impactions.

Monitoring and Supportive Care

Regular Monitoring: Check heart rate, respiratory rate, mucous membrane colour, capillary refill time, and hydration status.

Repeat Examinations: Re-evaluate rectal findings and naso-gastric reflux periodically.

Preventive Measures

Diet Management: Maintain a consistent feeding schedule with high-quality forage and minimal concentrates.

Deworming: Follow a regular deworming program to control parasites.

Hydration: Ensure access to clean water at all times.

Exercise: Provide regular, moderate exercise and avoid sudden increases in workload.

Specific Treatments for Different Types of Colic:

Spasmodic Colic:

Treatment: Flunixin meglumine, Buscopan, and adequate hydration.

Prognosis: Usually responds well to treatment.

Impaction Colic:

Treatment: Mineral oil, fluid therapy, and analgesics.

Prognosis: Good - if treated early; severe cases may require surgery.

Gas Colic:

Treatment: NSAIDs, walking the horse, and naso-gastric tubing if necessary.

Prognosis: Generally, responds well to treatment.

Sand Colic:

Treatment: Psyllium husk, mineral oil, and fluid therapy.

Prognosis: Good if treated early; preventive measures are crucial.

Strangulating Obstruction:

Treatment: Emergency surgery, aggressive fluid therapy, and supportive care.

Prognosis: Guarded to poor without timely surgical intervention.

Displacement or Torsion:

Treatment: Emergency surgery.

Prognosis: Dependent on the severity and duration before surgery.

Prevention

Regular Feeding Schedule: Avoid sudden changes in diet.

Deworming Program: Follow a strict deworming schedule.

Hydration: Ensure constant access to clean water.

Pasture Management: Minimize sand ingestion and ensure high-quality forage.

Stress Reduction: Minimize stressors, especially during travel and changes in environment.

Regular Dental Care: Ensure proper dental care to prevent chewing problems that could lead to impactions.

8.6.7 Chronic Obstructive Pulmonary Disease (COPD)

Chronic Obstructive Pulmonary Disease (COPD) in horses - also known as Recurrent Airway Obstruction (RAO) or heaves - is a chronic, non-infectious respiratory disease. It is characterized by inflammation and narrowing of the small airways in the lungs, leading to breathing difficulties.

Causes

Environmental Allergens – dust, mold, and spores in hay and bedding are the primary triggers.

Seasonality – symptoms often worsen in winter when horses are stabled and exposed to poor ventilation.

Genetics – some horses may be more genetically predisposed to developing COPD.

Clinical Signs

Chronic Cough – persistent coughing, especially when exposed to dust.

Nasal Discharge – often clear but may become more pronounced during flare-ups.

Laboured Breathing – horses with COPD exhibit increased respiratory effort, particularly during exercise.

Heave Line – a noticeable line along the horse's abdomen due to the extra effort needed to breathe. Exercise Intolerance – reduced ability to perform physical activities.

Diagnosis

Clinical Examination – a veterinarian will observe symptoms such as coughing, nasal discharge, and respiratory effort.

Auscultation – listening to the lungs with a stethoscope to detect abnormal lung sounds.

Endoscopy – an endoscope may be used to visualize the airways.

Bronchoalveolar Lavage (BAL) – collecting and analyzing fluid from the lungs to check for inflammation.

Treatment

Bronchodilators**:** To open the airways and make breathing easier. Clenbuterol: A common bronchodilator given orally to relax the airway muscles. Albuterol: Administered via inhalation for more direct effect.

Corticosteroids: To reduce inflammation in the airways. Dexamethasone or Prednisolone: Administered orally or via injection to reduce airway inflammation. Inhaled Steroids: Such as fluticasone, to reduce systemic side effects.

Mucolytics: To help clear mucus from the respiratory tract. Acetylcysteine: To break down mucus, making it easier for the horse to expel.

Antibiotics *(if secondary infections are present):* Used to treat bacterial infections that may complicate COPD.

Management

Environmental Control: Reducing exposure to allergens by improving stable ventilation, soaking hay, using low-dust bedding, and keeping horses outdoors as much as possible.

Nutritional Management: Ensuring the horse's diet supports overall health and reduces the risk of flare-ups.

Prognosis: COPD is a chronic condition that can be managed but not cured. With proper care, horses can live comfortable, productive lives. However, flare-ups can occur, particularly if environmental controls are not strictly maintained.

Control and Prevention

Good Ventilation: Ensuring stables are well-ventilated to reduce the buildup of dust and allergens.

Feed Management: Feeding dust-free hay or soaking hay before feeding to reduce dust exposure.

Bedding: Using low dust bedding materials and maintaining cleanliness in the stable.

8.6.8 Neonatal Isoerythrolysis

Neonatal Isoerythrolysis (NI) is a life-threatening condition in foals, where the foal's red blood cells (RBCs) are destroyed by antibodies in the mare's colostrum. This occurs when the mare has been sensitized to the foal's blood type during a previous pregnancy or transfusion, and she produces antibodies against the foal's RBCs.

Causes

Blood Type Incompatibility: The foal inherits a blood type from the sire that the mare lacks but has been sensitized to.

Pathophysiology

Antibody Transfer: When the foal ingests colostrum, it also ingests antibodies that attack its own RBCs. Haemolysis: The antibodies cause haemolysis (destruction of red blood cells), leading to anaemia and jaundice.

Clinical Signs

Clinical signs usually appear 24-48 hours after birth. The foals may become weak, lethargic, and have a reduced suckle reflex. Yellowing of the mucous membranes and skin due to the breakdown of RBCs is observed. Haemoglobinuria (dark urine) due to haemolysis and increased heart and respiratory rates due to anaemia are also observed.

Diagnosis

Coombs' Test: Detects antibodies attached to the foal's red blood cells. Blood Typing and Cross-

Matching: Helps identify the blood type incompatibility between the mare and foal. Clinical

Signs: Diagnosis is often based on the clinical signs and history of blood type incompatibility.

Treatment

Immediate Withdrawal of Colostrum: Prevent further ingestion of antibodies by bottle-feeding or using a colostrum substitute.

Supportive Care: Provide fluids and nutritional support.

Blood Transfusion: A blood transfusion may be necessary using washed RBCs from the dam (to remove antibodies) or a donor with a compatible blood type.

Prevention

Blood Typing and Antibody Screening: Perform blood typing of mares and sires before breeding. If incompatibility is detected, prevent the foal from nursing the mare's colostrum and provide an alternative colostrum source.

Monitoring: In high-risk pregnancies, monitor the foal closely after birth for signs of NI.

8.7 Annexure – I

Common Antibiotics Used in Horses and their Dose Rates

The following are the common antibiotics used in horses for treatment, along with their recommended dose rates and administration methods.

Precaution: It is recommended that broad-spectrum antibiotics in horses should be used precautionary.

Penicillin

Types: Penicillin G, Procaine Penicillin G

Dosage:

Penicillin G: 22,000 IU/kg IV every 6 hours

Procaine Penicillin G: 22,000 IU/kg IM every 12-24 hours

Administration: Intravenous (IV) or Intramuscular (IM)

Indications: Respiratory infections, skin infections, and soft tissue infections.

Trimethoprim-Sulphamethoxazole (TMS)

Dosage: 30 mg/kg orally every 12 hours

Administration: Oral (tablets, powder, or paste)

Indications: Respiratory infections, urinary tract infections, skin infections, and wounds.

Gentamicin

Dosage: 6.6 mg/kg IV or IM every 24 hours

Administration: Intravenous (IV) or Intramuscular (IM)

Indications: Severe systemic infections, uterine infections, and joint infections.

Enrofloxacin

Dosage: 5 mg/kg orally or IV every 24 hours

Administration: Oral or Intravenous (IV)

Indications: Respiratory infections, urinary tract infections, and joint infections. Not to be used in young animals (>4 years age) and pregnant animals.

Doxycycline

Dosage: 10 mg/kg orally every 12 hours

Administration: Oral (tablets or powder)

Indications: Respiratory infections, ehrlichiosis, and Lyme disease.

Metronidazole

Dosage: 15 mg/kg orally every 6-8 hours

Administration: Oral (tablets or powder)

Indications: Anaerobic infections, gastrointestinal infections, and abscesses.

Oxytetracycline

Dosage: 6.6-11 mg/kg IV every 12-24 hours

Administration: Intravenous (IV)

Indications: Respiratory infections, ehrlichiosis, and Potomac horse fever.

Ceftiofur

Types: Ceftiofur Sodium, Ceftiofur Crystalline Free Acid

Dosage:

Ceftiofur Sodium: 2.2 mg/kg IM every 24 hours

Ceftiofur Crystalline Free Acid: 6.6 mg/kg IM once every 4 days

Administration: Intramuscular (IM)

Indications: Respiratory infections, uterine infections, and skin infections.

Amikacin

Dosage: 10 mg/kg IV every 24 hours

Administration: Intravenous (IV)

Indications: Severe systemic infections, joint infections, and uterine infections. Not indicated in growing horses.

Chloramphenicol

Dosage: 50 mg/kg orally every 6 hours

Administration: Oral (tablets or powder)

Indications: Respiratory infections, gastrointestinal infections, and joint infections.

8.7 Annexure - II

Dosage of Common Dewormers for Horses

Deworming is an essential part of horse health management to control internal parasites. The choice of dewormer and dosage depends on the type of parasites present and the horse's weight. Here are some commonly used dewormers and their recommended dosages:

1. Ivermectin

Dosage: 0.2 mg/kg body weight

Administration: Oral paste or liquid

Target Parasites: Large and small strongyles, ascarids, bots, lungworms, pinworms, and threadworms.

2. Moxidectin

Dosage: 0.4 mg/kg body weight

Administration: Oral gel

Target Parasites: Large and small strongyles (including encysted small strongyles), ascarids, bots, pinworms.

3. Fenbendazole

Dosage: 5 mg/kg body weight for 5 days or 10 mg/kg body weight as a single dose

Administration: Oral paste, granules, or liquid

Target Parasites: Large and small strongyles, ascarids, pinworms.

4. Pyrantel Pamoate

Dosage: 6.6 mg/kg body weight

Administration: Oral paste or liquid

Target Parasites: Large and small strongyles, ascarids, pinworms, tapeworms (at double the dose).

5. Praziquantel

Dosage: 1 mg/kg body weight (when combined with ivermectin or moxidectin)

Administration: Oral paste

Target Parasites: Tapeworms.

6. Oxibendazole

Dosage: 10 mg/kg body weight

Administration: Oral paste

Target Parasites: Large and small strongyles, ascarids, pinworms.

7. Pyrantel Tartrate

Dosage: 2.64 mg/kg body weight daily

Administration: Daily feed additive

Target Parasites: Large and small strongyles, ascarids, pinworms.

8.7 Annexure - III

General Guidelines and Schedule for Deworming of Horses

A proper deworming schedule is crucial for

maintaining the health and well-being of horses. The schedule can vary based on the age, health status, and living conditions of the horses. Here is a general guideline for deworming horses, which should be customized to individual needs with the advice of a veterinarian.

General Guidelines:

Foals (0-6 months):

2-3 months: First deworming with a benzimidazole (e.g., fenbendazole) to target ascarids (*Parascaris equorum*).

4-6 months: Second deworming, rotate to a different class of anthelmintic (e.g., pyrantel pamoate or ivermectin).

Before weaning (approximately 6 months): Deworm again, possibly with ivermectin and praziquantel if tapeworms are a concern.

Weanlings and Yearlings (6-18 months):

Every 2-3 months: Continue deworming, rotating between different classes of anthelmintics to prevent resistance. Target strongyles, ascarids, and tapeworms.

Adults (18 months and older):

Spring (April-May): Deworm with ivermectin or moxidectin to target strongyles and bots. Consider adding praziquantel to target tapeworms.

Summer (July-August): Depending on the parasite load and pasture management, deworm with pyrantel pamoate or fenbendazole.

Fall (October-November): Deworm with moxidectin or ivermectin and praziquantel to target strongyles, bots, and tapeworms.

Winter (January-February): Deworm with a benzimidazole or pyrantel pamoate if necessary, based on faecal egg count (FEC) results.

Pregnant Mares:

Deworm before breeding, 1 month before foaling, and after foaling to reduce the transmission of parasites to the foal. Avoid moxidectin in pregnant mares.

Faecal Egg Count (FEC) Testing:

Twice a Year: Perform FEC tests in spring and fall to determine the parasite burden and adjust the deworming schedule accordingly.

Low Shedders: Horses with low FEC may only need deworming once or twice a year.

Moderate to High Shedders: Horses with higher FEC may need more frequent deworming.

Strategic Deworming:

Targeting Specific Parasites:

Strongyles: Use ivermectin, moxidectin, or fenbendazole.

Ascarids (Foals and Young Horses): Use benzimidazoles (e.g., fenbendazole), pyrantel pamoate.

Tapeworms: Use praziquantel or a combination product containing praziquantel.

Bots: Use ivermectin or moxidectin.

Pasture Management:

Manure Removal: Regularly remove manure from pastures to reduce parasite load.

Pasture Rotation: Rotate pastures to disrupt the lifecycle of parasites.

Co-Grazing: Graze horses with other species (e.g., cattle) to reduce parasite burden.

8.7 Annexure - IV

Vaccination Schedule in Horses

Vaccination - designed to prevent various infectious diseases - is a crucial aspect of equine health management. The schedule for vaccinations may vary based on geographic location, the age of the horse, its use (*e.g.*, racing, breeding), exposure risk, and local regulations. Here's a general overview of common vaccinations recommended for horses:

Core Vaccinations

These vaccines are recommended for all horses owing to the widespread risk and potential severity of the diseases.

Tetanus (Lockjaw)

Pathogen: *Clostridium tetani*

Vaccine Schedule: Foal – 2-3 ml, adult 3-5 ml. Initial series of two doses, 4-6 weeks apart, followed by annual boosters. More frequent boosters may be recommended based on wound management and exposure risk.

Rabies

Pathogen: Rabies virus.

Vaccine Schedule

Unvaccinated adult horses: Single dose, then annual revaccination.

Foals of unvaccinated mares: One dose at 4-6 months (as per manufacturer recommendations), annual revaccination.

Foals of vaccinated mares: Administer a 2-dose series—first dose at 4-6 months, second dose 4-6 weeks later (to address the potential for maternal antibody interference), with annual revaccination recommended

Mares: Vaccinate before breeding or 4-6 weeks before foaling.

Risk-Based Vaccinations

These vaccines are administered based on the horse's risk of exposure to these diseases.

Equine Influenza

Pathogen: Influenza virus.

Vaccine Schedule: Foals start with an initial series of three doses. Adults receive booster shots every 6-12 months depending on their exposure risk and the type of vaccine used.

Equine Herpesvirus (Rhinopneumonitis)

Types: EHV-1 and EHV-4.

Vaccine Schedule: Foals receive an initial series starting at 4-6 months with boosters, while adults get boosters every 6 months. Broodmares, should be vaccinated late in pregnancy to prevent abortion caused by EHV-1.

Strangles *(Streptococcus equi)*

Pathogen: *Streptococcus equi* bacterium.

Vaccine Schedule: Initial series of two or three doses for foals, with annual boosters depending on exposure risk. Intranasal vaccines provide localized immunity and are preferred for initial vaccinations.

Equine Viral Arteritis (EVA)

Pathogen: Equine arteritis virus.

Vaccine Schedule: Recommended for stallions and breeding mares based on risk. Stallions intended for breeding and non-pregnant mares may be vaccinated, with annual boosters and pre-breeding checks.

Vaccine Management Tips:

Consultation: Always consult a veterinarian to tailor a vaccination program to your horse's specific needs.

Documentation: Keep accurate records of all vaccinations, including the date, type, and any reactions noted.

Adverse Reactions: Monitor horses for any adverse reactions post-vaccination, such as swelling at the injection site, fever, or lethargy.

Best Practices:

Store vaccines according to the manufacturer's recommendations.

Use sterile needles for each injection to prevent infections.

Follow biosecurity measures to minimize the risk of disease spread.

Vaccinations are an essential part of preventive health care in horses, protecting them from serious diseases, enhancing their quality of life, and ensuring their performance and longevity in various equestrian disciplines.

Vaccination Schedule: A typical vaccination schedule for a horse is discussed here.

Foals (from vaccinated mares):

Tetanus: 4-6 months, booster at 10-12 months.

EEE/WEE: 4-6 months, booster at 10-12 months.

WNV: 4-6 months, booster at 10-12 months.

Rabies: 4-6 months, booster 4-6 weeks later, Annual booster.

Foals (from unvaccinated mares):

Tetanus: 3-4 months, booster at 4-6 months.

EEE/WEE: 3-4 months, booster at 4-6 months.

WNV: 3-4 months, booster at 4-6 months.

Rabies: 4-6 months, Annual booster

Adults:

Core vaccines: Single dose, then annual revaccination.

Risk-based vaccines according to exposure risk.

Considerations:

Pregnant Mares: Vaccinate with EHV-1 during the

5th, 7th, and 9th month of pregnancy. Ensure tetanus and EEE/WEE boosters 4-6 weeks before foaling.

High-Risk Horses: Horses that travel frequently, are in contact with other horses, or participate in events may require more frequent vaccination, especially for influenza and herpesvirus.

Geographical Factors: Some vaccines, like Potomac Horse Fever, may be more critical in certain areas.

8.7 Annexure-V

Normal Clinical Values in Equines

S No	Parameter	Normal Values
	Physiological Parameters	
1	Rectal Temperature	37.2° to 38.6°C
2	Heart Rate	28 to 44 beats per minute (bpm)
3	Respiratory Rate	8 to 16 breaths per minute
4	Oestrous Cycle	21 days (range 18 to 24 days)
5	Gestation Period	340 days (range 320 to 360 days)
	Haematological Indicators	
1	Haemoglobin	11 to 17 g/dL
2	Packed Cell Volume (PCV)	32 to 48 %
3	Red Blood Cell Count (RBC)	6.5 to 12.5 million/µL
4	White Blood Cell Count (WBC)	5500 to 12500 cells/µL
5	Platelet Count	100000 to 350000/µL
	Liver Functions Biomarkers	
1	Aspartate Aminotransferase (AST)	180 to 380 U/L
2	Alanine Aminotransferase (ALT)	2 to 50 U/L
3	Alkaline Phosphatase (ALP)	70 to 300 U/L
4	Gamma-Glutamyl Transferase (GGT)	10 to 35 U/L
5	Lactate Dehydrogenase (LDH)	240 to 600 U/L
6	Creatine Kinase (CK)	100 to 350 U/L
	Kidney Functions Biomarkers	
1	Blood Urea Nitrogen (BUN)	10 to 25 mg/dL
2	Creatinine	< 2.0 mg/dL
	Other Biochemicals Parameters	
1	Total Protein	5.5 to 7.9 g/dL
2	Albumin	2.7 to 3.9 g/dL
3	Globulin	2.4 to 4.0 g/dL
4	Triglycerides	14-65 mg/dL
5	Cholesterol	163-397 mg/dL
6	Glucose	60 to 130 mg/dL
	Electrolytes	
1	Calcium	10.2 to 13.6 mg/dL
2	Phosphorus	2.5 to 4.5 mg/dL
3	Magnesium	1.5 to 2.5 mg/dL
4	Sodium	132 to 146 mEq/L
5	Potassium	2.4 to 4.7 mEq/L
6	Chloride	95 to 110 mEq/L

CHAPTER- 9

GUIDELINES FOR MITHUN AND YAK DISEASES

9.1 Preamble

The yak (*Poephagus grunniens* or *Bos grunniens*) is a unique multipurpose bovid, reared mostly by the poor and marginal, tribal farmers of rural and remote India in the Himalayan belts. Yak are found at an altitude of 3,000-4,500 m above mean sea level (amsl)- and even at 6,000 m amsl- in the People Republic of China, Mongolia, Bhutan, Nepal, Russia and India. This multi-utility bovid is the lonely source of livelihood for the highlanders. In India, yaks are found in West Kameng and Tawang districts of Arunachal Pradesh; North and East districts of Sikkim; Lahaul, Spiti and Kinnaur districts of Himachal Pradesh; Ladakh and Kargil areas in Jammu and Kashmir; and Garhwal Himalayas of Uttaranchal. The dwindling domestic yak population for last few decades is a matter of serious concern for the environmentalists and scientific community from wildlife ecology viewpoint. Yaks are usually reared in cold high altitude alpine pastures. Therefore, the incidence of diseases in these animals is low unlike other livestock. However, during summer, these animals are brought to comparatively lower altitude (<3000 m amsl) where they came in close contact of other animals and are exposed to the various pathogens responsible for dreadful diseases like foot and mouth disease (FMD), infectious bovine rhinotracheitis (IBR), bovine viral diarrhoea (BVD), tuberculosis, brucellosis, haemorrhagic septicaemia (HS), ascariasis, *etc*. Moreover, many endo-parasitic and ecto-parasitic infestations are also observed in yaks.

The Mithun (*Bos frontalis*), a semi-domesticated and unique bovine species, is mostly concentrated in the North Eastern (NE) region of India. Arunachal Pradesh has the highest population of mithuns. Besides, Nagaland, Mizoram and Manipur also have a reasonable population. Mithuns are distributed at 300-3000m amsl. Owing to their preference of a cold climate, they avoid bright sunlight and like to stay in the deepest part of the forest during daytime. Like yaks, mithuns are also susceptible to various infectious and non-infectious diseases and they mostly get the infection because of their contact with cattle and other wild animals during migration through the hilly terrains.

Diseases always remain a major obstacle for any livestock industry to grow and flourish. Lack of appropriate health strategy, suitable therapeutic and prophylactic measure and paucity of veterinary dispensaries in the remote hilly areas are serious drawbacks for the yak and mithun farming. All these diseases add to hurdles faced by these two unique ruminants. Both mithuns and wild yaks are considered vulnerable according to International Union for Conservation of Nature (IUCN).

These standard veterinary treatment guidelines are designed to give a brief overview to plan suitable therapeutic and prophylactic strategies against the prevalent diseases of yaks and mithuns.

9.2 Bacterial diseases

9.2.1 Anthrax

Definition and Causative Agent

Anthrax is a highly infectious, fatal and zoonotic disease of all the warm-blooded animals including yaks and mithuns. It is also called Splenic fever due to extensive engorgement and enlargement of spleen. The disease is caused by *Bacillus anthracis*, a gram positive, large rod- shaped non-motile, capsulated, spore-forming organism.

Transmission

The susceptible animals may get the infection through contact, inhalation and ingestion. As anthrax spores remain viable for long deeply buried in the soil, occasional outbreaks with fatal outcome are very common in the endemic/enzootic areas. The sporulation occurs with the change in the soil pH, coinciding with the onset of the monsoon.

Clinical Signs

Sudden death is noticed in per-actute cases. Besides fever, bloat, dyspnoea, convulsion and prostration may accompany. Unclotted blood usually comes out of the natural orifices after death. Cutaneous form of anthrax has also been reported in yaks.

Lesions

Rigor mortis is usually absent and dark red or black color-stained blood can be noticed coming out of the nostrils, mouth and anus. Splenomegaly, congestion of liver and kidney, and accumulation of serosanguineous fluids in the body cavity may be observed.

Diagnosis

The clinical symptoms are very characteristic and blood swabs from the suspected animals may be

processed for cultural examination. Thick blood films collected from the suspected animal may be stained with polychrome methylene blue (PMB) for detection of blue rod-shaped organisms in short chains with pink capsules (Mc'Fadyean Reaction). Ascoli's test may be performed for detection of pathogens in the suspected hide samples. Fluorescent antibody staining and PCR are used for specific diagnosis. Contaminated soil samples may be subjected to bacterial culture for isolation of the causative agent and subsequent identification.

Differential Diagnosis

The disease should be differentiated from acute tympany, snake bite, hemorrhagic septicaemia, and black quarter where such acute or per-acute onset with sudden death is common.

Treatment

In cutaneous form, crystalline penicillin G @ 10,000-20,000 IU/kg BW OD IM or oxytetracycline @5-10 mg/kg BW IM/IV parenterally can be used for at least 5 days. Antipyretics such as flunixin meglumine @ 0.5-2.2 mg/kg BW, IM/IV, OD, SOS; antihistaminics such as chlorpheniramine maleate @ 0.5-1.0 mg/kg BW, IM, OD, for 3 days can offer relief to the animal.

Control

In the endemic zones, the animals aged four months or above should be vaccinated annually with live anthrax spore vaccine prepared from *B. anthracis* Sterne strain in the pre-monsoon season.

Biosecurity Measures

As it is OIE List A notifiable disease, the State Veterinary department should intimate such occurrence to the competent authority at the earliest. It is advised to avoid the opening of a suspected carcass to avoid accidental exposure to spores. The carcass should be deeply buried with application of lime with a thickness of 3 cm above the burial ground. The adjacent areas should be sprayed with disinfectants like 3 percent acetic acid or 10 percent caustic soda or 10 percent formaldehyde or 3 percent hydrogen peroxide.

Precaution: It is advised to avoid the opening of a suspected carcass to avoid accidental exposure to spores.

Standard Operation procedure (SOP) for disposal of anthrax infected carcasses by burial procedure

Select an appropriate site for carcass burial such that it is not mutilated by wild dogs, foxes, jackals, *etc*., to prevent the spread of the spores.

Due consideration should be given to avoid contamination of water sources, residential areas, livestock facilities, pastures and other establishments in the vicinity. Preferably, it should be away from any footpaths or roads leading to the site.

Prepare a pit with sufficient width to accommodate the carcass with a minimum depth of 2 m considering the size of the carcasses.

Wear apron, face masks, eye shields and gloves before handling the carcass.

Drop the carcass into the pit.

Cover the carcass with soil (400 mm) and add an unbroken layer of lime (calcium carbonate) or calcium hypochlorite (bleaching powder).

Do not put lime directly on to the carcass (it will slow decomposition process).

Close the pit with sufficient soil and make a heap over the site.

Then put a layer of lime over the soil.

Secure the disposal site by fencing (if possible) and place a notification mark.

Standard Operating Procedure (SOP) for disinfection of contaminated premises

Prepare 1 percent hypochlorite solution in a bucket.

Spray and wash barn utensils, tools and equipment with the above solution thoroughly and dry them for reusing.

Bury the beddings with carcasses, if it is in small quantities. Burn it in a pit, if the materials are in larger quantities.

Contaminated premises should be disinfected thoroughly with 1 percent hypochlorite spray @ 1.0-1.5 litre/sq m. Allow contact time of 2-3 hours.

Contaminated laboratory materials can be disinfected by immersing them in 1 percent hypochlorite solution for at least 30 minutes.

Disposable items, including used PPEs, must be incinerated/burnt in a pit.

Samples to be collected

Blood, hide, and hair.

9.2.2 Tuberculosis

Definition and Causative Agent

Tuberculosis (TB) - a highly contagious, chronic granulomatous and debilitating disease - is caused by members of the *Mycobacterium tuberculosis* complex, mainly by *M. bovis* in animals. Infections may also be caused by *M. caprae* and to a lesser extent by *M. tuberculosis*. The organisms are non-motile and non-sporulating bacteria detected by Ziehl-Neelsen staining. The disease is characterized by development of tubercles in lungs, pleura, liver, kidney and lymph nodes.

Transmission

The disease may be transmitted by contact of infected animals, inhalation of infected aerosol and consumption of infected materials including the milk from infected animals.

Clinical Signs

Infected animals may remain asymptomatic for a long period. Generally, they display progressive emaciation, dyspnea, intermittent hacking paroxysmal cough, lethargy, weakness, anorexia, a low-grade fluctuating fever, and enlargement of lymph nodes particularly retropharyngeal and supra-mammary lymph nodes.

Lesions

Appearance of nodular or miliary tubercles in lungs, liver, peritoneum, kidney, lymph nodes and udder during postmortem examination. Bunch of grapes-like lesions may be noted in pleura and peritoneum.

Diagnosis

The intradermal tuberculin skin test is generally recommended to detect the positive reactors in a herd. Animals which are doubtful should be re-tested. Further, gamma interferon release assay, bacterial culture, and pathogen isolation are recommended for disease confirmation.

Differential Diagnosis

Tuberculosis should be differentiated from aspiration pneumonia, nasal schistosomiasis, rhinosporidiosis, actinobacillosis, theileriosis and bovine leucosis.

Treatment

Treatment is not recommended due to high treatment cost, low cure rate and chance of spread of infection among other healthy animals of the herd. However, animals may be treated with Isoniazid @ 20 mg/kg BW orally daily for 3 months or Clofazimine @ 600 mg TD orally for 3 months or streptomycin @50 mg/kg BW, IM for minimum 12 months along with vitamin C and E and hepatoprotectants.

Control

If the incidence rate is very low (<5 percent), disease can be eradicated by test and elimination policy. In India, as no test and elimination policy are in practice due to socio-economic reason, confirmed cases should be kept in isolation.

Biosecurity Measures

The infected animals should immediately be isolated and separated from herd with proper biosecurity measures like quarantine of new animals for 30 days, restrictions of visitors and vehicles in the farm, and periodic disinfection of footwear, farm equipment, utensils, and clothes. Animals should be bi-annually tested with intradermal test and all the positive reactors should be segregated and culled.

Samples to be collected

Lung, spleen, liver, kidneys and regional lymph nodes along with broncho-alveolar lavage, oro-nasal discharges.

9.2.3 Black Quarter

Definition and Causative Agent

Black quarter or black leg is an acute, febrile, infectious disease of ruminants caused by Gram-positive rod-shaped, spore forming toxin-producing anaerobe, *Clostridium chauvoei* characterized by emphysematous swelling, usually in the heavy limb musculature (clostridial myositis).

Transmission

The disease is soil-borne and can spread from soils contaminated with infected carcass. The organisms gain entry through ingestion of contaminated feed or through open wound. Healthy animals may harbour bacteria in the spleen, liver and alimentary tract and thus the animals may excrete the organisms through their faeces.

Clinical Signs

Most of the clinical signs in yaks and mithuns are akin to cattle and buffaloes. Pyrexia (up to 108°F),

anorexia, suspended rumination, limb stiffness or lameness, and oedematous swelling in the hip, shoulder, chest, back, neck and perianal regions are evident. On palpation, swollen areas emit crackling or crepitation sound due to emphysema. Animal may succumb to infection following a rapid course due to severe toxaemia.

Lesions

Lesions are limited to affected heavy muscles, tongue, diaphragm and myocardium. The affected tissues turn black and emanate a rancid odour. Thoracic cavity and pericardium show large quantity of blood-stained fluid.

Diagnosis

Bacterial culture and isolation with the samples collected from affected muscles. Microscopic examination of the stained impression smears to detect Gram-positive bacteria. The fluorescent antibody test and PCR to detect the pathogen.

Differential Diagnosis

Diseases causing sudden death such as anthrax, haemorrhagic septicaemia and other acute diseases like malignant oedema and bacillary hemoglobinuria.

Treatment

Penicillin G @ 40,000 units/kg BW IM TID interval for at least 7 days along with surgical debridement of the affected tissue. Metronidazole infusion can be instilled locally into the affected muscles. Antipyretics such as Flunixin meglumine @ 1.1- 2.2 mg/kg BW, IM/IV, OD may be given.

Control

The dead animals should not be de-skinned and burnt or buried. The young animals should not be allowed to graze in the endemic pasture. All the animals aged 6 months or above from the endemic zones should be vaccinated annually before onset of monsoon with formaldehyde inactivated Black Quarter vaccine or combined vaccine of *Pasteurella multocida* and *Clostridium chauvoei* can be used.

Biosecurity Measures

The dead bodies should be buried or burnt with lime application. Opening of carcasses should be avoided to prevent contamination of soil and environment. Burning of the upper layer of the soil with straw may be done to minimize the dissemination of the spores.

Precaution: Opening of carcasses should be avoided to prevent contamination of soil and environment.

Samples to be Collected

Heart blood, peritoneal fluids and affected muscles.

9.2.4 Haemorrhagic Septicaemia

Definition and Causative Agent

Haemorrhagic septicaemia – an acute, subacute and highly fatal infectious disease of septicemic nature – is caused by Gram-negative coccoid, short-rod or filamentous bipolar organism, *Pasteurella multocida*. It is characterized by acute gastroenteritis, subacute oedema, serofibrinous pleuropneumonia and oedema of intra-alveolar tissues. The organism remains in respiratory tract without any clinical symptom and may turn pathogenic only in the presence of predisposing stress factors like malnutrition, fatigue, starvation, transportation, overexertion, and inclement weather.

Transmission

The pneumonic form occurs through inhalation of contaminated droplets or ingestion of contaminated feed or water. Salivary transmission is also possible as it contains abundant *Pasteurella* organisms.

Clinical Signs

High fever (up to 110 ^{0}F) with concurrent shivering followed by profuse salivation, lacrimation and nasal discharge, congested mucus membrane, and labored breathing are characteristic symptoms. The affected animals may show abdominal pain, and severe diarrhoea or dysentery. Auscultation of lungs reveals increased vesicular and moist rales. Hot and painful oedematous swellings may be observed in the subcutaneous pocket of the head, neck, dewlap and brisket region.

Lesions

The pneumonic changes are manifested by marked hepatization of lungs with deposition of serofibrinous exudates in the interlobular space. Oedema and thickening of the interlobular septa and petechial haemorrhages can be observed on the thoracic membrane and pericardial sac.

Diagnosis

PCR, examination of heart blood smears, bacterial cultures from oral or nasal secretions and sometimes

from faeces are indicative.

Differential Diagnosis

Verminous pneumonia, *Ascaris* pneumonia, allergic rhinitis, pulmonary abscess, aspiration pneumonia and enzootic nasal granuloma.

Treatment

Ceftiofur sodium @ 1.1-2.2 mg/kg BW, IM OD for 3-5 days or enrofloxacin @ 5 mg/kg BW IM/IV OD for 5 days are effective.

Alternatively, animals may also be treated with sulphathiazole or sodium sulphadimidine @ 150 mg/kg BW IM/IV OD for 5 days or oxytetracycline @ 10 mg/kg BW IM for 5 days. Symptomatic treatment includes diuretics, NSAIDs (pregnant animals) and steroids like prednisolone or dexamethasone (preferred for non-pregnant animals).

Control

Annual vaccination with formaldehyde inactivated *Pasteurella multocida* or combined vaccine of *Pasteurella multocida* and *Clostridium chauvoei* can be used before onset of monsoon.

Biosecurity Measures

In enzootic areas, prophylactic vaccination is carried out one or two months before the onset of monsoon. The animal-shed should be well ventilated, and periodic disinfection of sheds is recommended with exposure to heat, sunlight and application of 0.5 percent phenol.

Samples to be Collected

Smear/swabs from heart blood, liver, spleen, lung, broncho-alveolar and intestinal contents.

9.2.5 Brucellosis

Definition and Causative Agent

It is an acute or chronic, zoonotic and contagious disease of animals caused by small Gram-negative, non-motile, non-sporing facultative intracellular bacterium *Brucella abortus*. It is characterized by placentitis, birth of stillborn or weak calves, retained placenta and abortion at third trimester of pregnancy.

Transmission

The infection spreads through ingestion of food and water contaminated with discharges of aborted fetus or fetal membranes, abraded skin or conjunctiva, grazing on infected pasture, artificial insemination with semen from infected bull, congenitally, and milk from infected dams.

Clinical Signs

It is the most common cause of abortion, which usually takes place in the last trimester of pregnancy along with retention of placenta, persistent uterine infection and muco-purulent discharges from vagina. Animals exhibit low conception rate, reduced milk production, hygroma, bursitis of limbs. In male, epididymitis and orchitis may be seen. Infertility in both male and female is common.

Lesions

Important lesions include swelling of the foetal membranes, adhered cotyledons with yellow-brownish necrotic changes, blood-stained fluid in the aborted fetal serous cavities. In males, necrotic orchitis with involvement of seminal vesicle is the common lesion.

Diagnosis

Infection can be confirmed by isolation and identification of organism from aborted foetal tissues and excretions. Serological tests like serum agglutination tests, Rose Bengal Plate Test (RBPT), Milk Ring Test, CFT, ELISA may be useful to detect sero-positive animals.

Differential Diagnosis

Trichomonosis, campylobacteriosis, leptospirosis, listeriosis, vibriosis and mycotic abortion.

Treatment

There is no proven treatment which can cure brucellosis in yaks and mithuns. Test and removal of the reactor is recommended. Regular cleaning and disinfection of the farms and proper disposal of uterine discharges, placenta, foetus and foetal membranes should be conducted to eliminate the major source of infection to susceptible animals.

Control

Vaccination of female calves with the live attenuated *Brucella abortus* strain 19 vaccine, between 4 and 8 months of age, can provide adequate immunity. Where artificial insemination is practiced, prepucial washing of mithun/yak bulls should be tested for *Brucella* infection. Bulls should be tested before being used for semen collection for AI.

Biosecurity Measures

All the newly purchased animals are to be quarantined for 30 days. Regular screening of the herds with a history of abortion should be conducted. RBPT is a reliable and practical test for herd screening and animals should be tested 21 days following abortion to identify and eliminate the positive reactors.

Aborted fetus, placenta and fluid should be isolated and disinfected with phenolic/quaternary ammonium compound (at 5-10 percent) solution for disposal.

Farm attendants, visitors and veterinarians in contact with infected material, should use ~4 percent chlorohexidine gluconate as the disinfectant.

Samples to be Collected

Fetal fluid, aborted fetus, placenta and uterine discharges.

9.2.6 Leptospirosis

Definition and Causative Agent: Leptospirosis is an acute or chronic and sometimes clinically inapparent zoonotic disease characterized by fever, anemia, hemoglobinuria, icterus and abortion. Leptospirosis is caused by an obligate aerobic, Gram-negative, corkscrew-shaped spirochaete *Leptospira interrogans*.

Transmission

Infected urine, placental fluids, and milk are the major source of transmission. Venereal or transplacental routes are sporadically reported. Direct contact with urine of infected animals, particularly of rodents and ingestion of feed or water contaminated with urine may play an important role.

Clinical Signs

Fever, dyspnea, icterus, haemoglobinuria and abortion at the third trimester of pregnancy with retention of placenta. Subclinical infection is more common.

Lesions

Major changes are observed in kidney and liver. Affected kidneys are characterized by interstitial nephritis and petechial hemorrhages. Liver is enlarged and discolored due to cholestatic hepatitis.

Diagnosis

Dark field microscopic examination of urine and blood from suspected animals and IGM antibody detection by ELISA can be useful. Microscopic agglutination test with prevalent serovars in the area is the gold standard. Immunohistochemistry of the formalin-fixed tissue and PCR can effectively detect pathogens in blood, urine, or tissue samples.

Differential Diagnosis

Bacillary haemoglobinuria, post-parturient haemoglobinuria, babesiosis, anaplasmosis and poisoning/toxicity.

Treatment

Prolonged antibiotic and supportive therapy should be provided to speed up the recovery of liver and complete elimination of the bacteria from the kidneys.

Penicillin G @ 20,000 IU/kg BW, IM, 5 days or streptomycin/dihydrostreptomycin @ 10 mg/kg BW, IM, 5 days will eliminate the carrier state. Alternatively, a high dose of enrofloxacin @ 15 mg/kg BW, OD, IM administered over 5 days. It should not be used in lactating animals. In pregnancy, amoxicillin @ 10-20 mg/kg BW BID IM/IV for 7 days may be given.

However, clinicians must evaluate status of liver and kidney function before deciding about the antibiotic treatment. Commercial indigenous hepatoprotective formulation may be used to support the liver function.

Control

Urine is the major source of infection, hence, strict sanitary measures should be adopted in the farm to avoid contamination of drinking water through urine. Segregation of infected animals, use of disinfectants like diluted bleach solution (one-part household bleach and 10 parts water) for washing the floor and premises, and proper disposal of aborted foetus, foetal membranes and fluid must be taken into consideration for controlling the disease.

Biosecurity Measures: Precautions must be taken to prevent any contamination by excreta of infected domestic and wild animals. Convalescent animals should be kept isolated for at least two months before being re-introduced in the herd. Serological screening of all the animals in the herd is a necessary step. Farms should be kept free from rodents, cats and wild/stray dogs. Disinfection of farm utensils with disinfectants like sodium hypochlorite and

sodium hydroxide is helpful.

Samples to be Collected

Blood, urine, milk, foetus and infected tissues.

9.2.7 Colibacillosis

Definition and Causative Agent

Colibacillosis, a bacterial disease of neonatal calves caused by *Escherichia coli*, is characterized by profuse diarrhoea and septicaemia. *E. coli* is a Gram-negative motile bacteria present in the gut as commensal. However, there are several variants of diarrhoeagenic *E. coli* and the enterotoxigenic *E. coli*, which produce heat labile and heat-stable enterotoxins that are mostly responsible for causing colibacillosis. However, Shiga-toxin producing and enteropathogenic *E. coli* may also have a role in causing diarrhoea in yaks and mithuns.

Transmission

Oro-fecal route is the main source, along with nasopharyngeal or naval routes. Discharges from uterus, vagina, umbilicus or aborted foetus and infected milk may act as a source of infection to the newborns.

Clinical Signs

Two forms are common - enteric and septicaemic forms. Enteric form can occur during the first 3 weeks of life. Affected animals pass watery or pasty faeces, usually chalky white with offensive rancid odour leading to dehydration and death. Septicaemic forms are often seen with an initial rise of temperature. But the animals may become hypothermic with profuse diarrhoea and dehydration. Sub-acute forms usually accompany joint ill, meningitis and panophthalmitis.

Lesions

Congestion, haemorrhages and sloughing of mucosa are noticed in the jejunum and colon. Occasionally, haemorrhages may be detected in endocardium and epicardium. Chronic forms have changes in joints, liver, spleen and kidney.

Diagnosis

Bacteriological isolation and identification and molecular tests such as PCR may be used to identify diarrhoeagenic strains.

Differential Diagnosis

Salmonellosis, listeriosis, pasteurellosis, coccidiosis, rotavirus infection, cryptosporidiosis and *Clostridial perfringens* type C infections.

Treatment

Fluid and electrolyte therapy, diet changes and antimicrobial therapy are the cornerstone for management. In yak or mithun calves, oral rehydration solution may be given @ 50-100 ml/kg BW, for 4-6 hours followed by 100-140 ml/kg BW for next 20 hours depending upon the degree of dehydration. Alternatively, in severe dehydration, parenteral fluids such as Ringer's lactate may be advocated up to 90 ml/kg BW; IV followed by a maintenance dose of 5-10 ml/kg BW IV BID/TID until recovery. Use of commercial probiotic and prebiotic or synbiotics formulation may be beneficial.

Tannic acid (15 g), kaolin (30g) and pulverized ginger (15 g) may be added in gruel and fed to the affected animals BID until recovery.

Control

If the animals do not improve on fluid and supportive therapy, antimicrobial therapy may be initiated. Ampicillin @ 10 mg/kg BW TID for 7 days or oxytetracycline @ 5 mg/kg BW BID for 5 days are effective. Sulphamethoxazole and trimethoprim @ 30 mg/kg BW OD for 5 days may also be given. The calf should be allowed to suck colostrum for first 3-4 days. The mother should get adequate protein in diet and vitamin A @ 50,000 units may be given 10-15 days prior to parturition. Calves may be fed with purified gamma globulin.

Biosecurity Measures

The disease may be prevented by feeding of colostrum to calves @ $1/10^{th}$ of their body weight within 6 hours of birth. The farm may be made free of calf scour, if good animal husbandry practices and proper hygienic measures are in place. The teats of the mother must be kept clean and disinfected. Overcrowding is to be avoided in the animal shed and regular disinfection of the farm utensils should be done. In newborns, the naval should be swabbed with 2 percent iodine and also the pens should be well-bedded, clean and disinfected. Sick animals should immediately be segregated with proper disposal of excreta and washing of floors.

9.2.8 Infectious Bovine Keratoconjunctivitis

Definition and Causative Agent

It is a highly contagious ocular disease of yak and mithun, caused by Gram-negative bacteria, *Moraxella bovis*. The infection is characterized by conjunctivitis, lacrimation, corneal opacity and blepharospasms. There may be involvement of other pathogens like BHV-1 and *Neisseria*. *Moraxella bovis* is a Gram-negative, aerobic, coccobacillus which usually appear in short chains. This is the most common ocular disease in yaks and mithuns.

Transmission

The disease transmits through direct contact with nasal or ocular discharges or mechanically through vectors like *Musca domestica*.

Clinical Signs

The disease is characterized by profuse lacrimation from the affected eye followed by conjunctivitis, blepharospasms, and drooping of eyelids resulting in photophobia, matting of eye lashes and eventually corneal opacity. Besides keratitis, corneal ulceration also develops. Corneal scarring may be noticed even after recovery, and it takes months to resolve. In severe cases, scarring may lead to complete blindness.

Lesions

Scleral congestion, viscid lachrymal discharges along with hypertrophy of conjunctiva and keratitis are frequently observed. In later stage, animals usually develop diffuse corneal opacity and corneal ulceration.

Diagnosis

Diagnosis is based on isolation and identification of organism from affected eyelids and ocular discharges. Molecular techniques and matrix-assisted laser desorption/ionization time-of-flight mass spectrometry can be used for pathogen detection.

Differential Diagnosis

Traumatic conjunctivitis, infectious bovine rhinotracheitis, eye worms such as *Thelazia* spp., photosensitization, rickettsial conjunctivitis and mycoplasmal keratoconjunctivitis.

Treatment

Ophthalmic ointment containing, penicillin, oxytetracycline and streptomycin and steroidal anti-inflammatory drugs is suggested. Boro-zinc solution (1-2 percent) may be instilled in the affected eyes. In corneal opacity, sub-conjunctival injection of dexamethasone @ 0.25 ml for 5-7 days is recommended. Irrigation of affected eyes with lukewarm normal saline solution is beneficial. Vitamin A @ 440 IU/kg BW IM once in a week may be given for better response. Non-responsive cases may be treated systemically with enrofloxacin @ 5 mg/kg BW OD for 5 days.

Control

Keeping the affected animals away from direct sunlight is essential.

Biosecurity Measures

In endemic area, animals are to be maintained under proper surveillance and that may reduce the transmission. Isolation of diseased animals from the rest of the herd, fly control and administering adequate minerals like selenium, copper and zinc, as their deficiencies lead to increased risk of such condition. Insecticide-impregnated fly traps in the vicinity of the animals helps in disease prevention.

Samples to be Collected

Swabs from affected eyes.

9.2.9 Chlamydiosis

Definition and Causative Agent

A zoonotic disease, also known as Enzootic abortion, is caused by *Chlamydophila abortus*, a Gram-negative, obligate intracellular pathogen. It is characterized by abortion or birth of weak newborns. Placenta is often the site of predilection for this pathogen and thus placentitis ensues with extensive tissue damage and abortion.

Transmission

Animals may contract the disease by direct contact with the infected animal, inhalation of aerosolized dried faeces, ingestion of contaminated feed or water and also through infected semen.

Clinical Signs

In acute cases, there may be fever, respiratory disorders, vaginitis, enteritis, pericarditis, encephalitis, arthritis and hepatitis. At a later stage; drop in milk yield, premature calving and early-stage abortion is the most evident clinical sign. Bulls suffer from vesiculitis and can shed *Chlamydia* via

semen.

Lesions

Lesions depend on the site of infection and forms. In respiratory form, acute pulmonary lesions develop often with bronchiolitis and pulmonary oedema. Hepatic lesions include hepatocyte necrosis, lympho-histiocytic infiltrates and sinusoidal histiocytes. Reproductive lesions include endometritis, vaginitis and vesiculitis.

Diagnosis

Isolation of organism from vagina, rectum, tracheal washing or broncho-alveolar lavage or swab from nose. Detection of nucleic acid by direct hybridization or nucleic acid amplification (PCR) are some of the newer diagnostic methods. Besides PCR, ligase chain reaction and strand displacement amplification are also used. ELISA can be used for serological screening.

Differential Diagnosis

Toxoplasmosis, brucellosis, leptospirosis, Q-fever and other causes of abortion.

Treatment

Oxytetracycline @ 10 mg/kg BW IM or enrofloxacin @ 2.5–5.0 mg/kg BW OD IM can be used. Treatment should be started at the earliest and to be continued for at least seven days.

Control

Isolating sick animals from rest of the herd and improvement of ventilation to reduce aerosol concentrations are required.

Biosecurity Measures

This is a zoonotic pathogen, therefore, the veterinarians and animal handlers particularly, pregnant mothers should take proper precaution like wearing of personal protective equipment (PPE) whenever such suspicion arises at the farm. Separation of the affected animals, regular screening, hygienic disposal of the aborted materials, disinfection of materials used in handling such animals are essential footsteps for optimum biosecurity. Disinfectants like quaternary ammonium compounds (1:1,000), 1 percent lysol and bleach (1:100) are effective to reduce such pathogens in fomites and farm premises.

Samples to be Collected

Serum samples for serology, vaginal, ocular and nasal swabs, tracheal washing or bronchoalveolar lavage fluid, aborted tissues and fluids for pathogen isolation and molecular diagnosis.

9.3 Viral Diseases

9.3.1 Lumpy skin disease

Definition and Causative Agent

Lumpy Skin Disease (LSD) - caused by the lumpy skin disease virus (LSDV), a member of the genus *Capripoxvirus* within the family Poxviridae - is a disease of bovines characterized by fever, nodules on the skin, mucous membranes and internal organs, emaciation, enlarged lymph nodes, oedema of the skin, and sometimes death. The disease primarily affects cattle and water buffaloes. This disease has also been documented in mithun from the Northeastern hilly regions of India. All age groups are susceptible.

Transmission

LSDV primarily spreads through insect vectors such as mosquitoes, ticks, and flies. It can also be transmitted through direct contact with infected animals, contaminated feed, water, equipment, and infected semen.

Clinical Signs

The incubation period ranges from 4 to 14 days. The disease is characterized by high fever, lymphadenopathy, anorexia, fall in milk yield, gradual weight loss, firm, raised, and painful nodules with a necrotic centre on the skin and mucous membranes, oedematous swelling, particularly in the limbs. Later, the nodules rupture eventually leading to magot infested open wounds that act as a source of infection. In severe cases, respiratory tract may be involved resulting in severe dyspnoea.

Lesions

Leukocytosis or leukopenia, lymphopenia, and neutrophilia in secondary infections are evident. Bullet-shot wounds in the trachea, respiratory tract and nodular lesions in the gastrointestinal tract are found.

Diagnosis

Diagnosis is based on the appearance of characteristic skin nodules and clinical signs such as fever, lymphadenopathy, and oedema. Polymerase

Chain Reaction (PCR) is a highly sensitive and specific method for detecting LSDV DNA in clinical samples. Besides, the virus can be isolated from the clinical samples. Serological tests like VNT and ELISA may detect antibody in the serum.

Differential Diagnosis

Severe cases of LSD are highly distinctive; however, milder forms can be mistaken for other diseases such as bovine herpes mammillitis (also known as pseudo lumpy skin disease), bovine papular stomatitis (parapoxvirus), pseudocowpox (parapoxvirus), dermatophilosis, demodicosis, and photosensitization.

Treatment

There is no treatment for lumpy skin disease. Non-specific treatments using anti-inflammatory drugs like Flunixin meglumine @ 1.1-2.2 mg/kg BW IM, OD, 3 days and vitamin A and C are usually advocated to relieve inflammation and fever, and to improve the appetite of the animals. Local wounds are to be dressed with antiseptic solution like Lugol's iodine.

For better response and prevention of spread of LSD, ivermectin may be given @ 200-400 mcg /kg BW SC. Secondary infection may be treated with enrofloxacin @ 2.5-5 mg/kg BW, IM or SC, OD for 5 days.

For alternative treatment, refer EVM section.

Control

Vaccination is the most effective method for controlling LSD. Live attenuated Goatpox vaccine is recommended for prophylactic vaccination of cattle and buffalo against LSD and the same can be used for prophylactic vaccination of yak and mithun. Fly-impregnated traps in the farms may be effective to control vectors.

Biosecurity Measures

Important biosecurity measures include quarantining of newly coming animals for at least 14 days for disease monitoring before introducing into the herd, immediate isolating the LSDV-infected or suspected animals to prevent disease transmission and restricting the access to only essential personnel for their entry into livestock areas. It is effective to carry out proper disinfection procedures using one of the disinfectants like 3 percent sodium hypochlorite, 4-5 percent acetic acid, 4 percent sodium carbonate, and 2 percent sodium hydroxide and phenolic compounds for footwear and equipment, and farm-specific clothing to avoid cross-contamination. Important steps to minimize the vector population at farm premises include application of insecticides (malathion, propoxur, plant oils like neem, garlic, or citronella) to animal housing and surrounding areas to manage mosquito, fly, and tick populations; installing fine mesh screens on windows and doors; removal of standing water, and proper disposal of wastes, *etc.*

9.3.2 Foot and Mouth Disease

Definition and Causative Agent

The foot and mouth disease (FMDV) is a highly contagious viral disease that can affect the health and production of all cloven-footed animals, including mithuns and yaks, over an extended period of time. Epithelium of buccal cavity, tongue, nares, muzzle, teats and feet and hooves are affected with vesicular eruptions. FMD virus is an *Aphthovirus*, which is a member of the Picornaviridae family. There are seven major serotypes of FMDV, *viz.*, O, A, C, Asia 1, Southern African Territories (SAT) 1, SAT-2, and SAT-3, along with several variants. Only four serotypes of FMDV, *viz.*, O, A, C, and Asia-1 had been present in India. Currently, only three serotypes of FMDV like O, A, and Asia-1 are responsible for FMD in India.

Transmission

FMDV is primarily transmitted by direct contact with secretions and excretions from infected animals. It can be transmitted via fomites, such as contaminated feed, water, bedding, equipment, clothing, and vehicles. People can inadvertently spread FMDV by carrying the virus on their clothing, footwear, and hands. FMDV, when air-borne, can spread virus/disease over long distances. The virus can be present in meat, milk, and other products derived from infected animals. Wildlife-livestock interfaces are critical points for potential virus spillover.

Clinical Signs

Like other ruminants, yak and mithun are highly susceptible to FMD. Its symptoms are sudden onset of high fever typically lasting for 2-3 days before the formation of vesicles (blisters) on the tongue, gums, inside the cheeks, and lips. Vesicles and subsequent

ulcers may appear on the coronary band, interdigital space, and sometimes on the heels. Vesicles may rupture, leading to erosions and ulcers, causing excessive salivation and drooling. Lameness due to painful lesions leads to reluctance to walk or stand. Serous nasal discharge can be observed, which may become mucopurulent (containing mucus and pus), if secondary bacterial infection occurs.

Lesions

In young animals, myocardial necrosis and inflammation is often associated with sudden death. Besides, petechial haemorrhages in the kidney are commonly observed in mithun during postmortem. Blood profile often reflects leukopenia (lymphopenia and neutropenia particularly). Increased levels of acute phase proteins (APPs) such as fibrinogen and serum amyloid A (SAA), muscle enzymes like creatine kinase (CK) and aspartate aminotransferase (AST) due to myocardial damage, especially in severe cases affecting the myocardium may be observed.

Diagnosis

Clinical signs are often indicative of FMD. PCR is a highly sensitive and specific method for detecting viral RNA in various clinical samples. Further, FMDV can be isolated from vesicular fluid, tissue samples, or secretions in cell cultures or laboratory animals. ELISA or VNT can be used to detect FMDV-specific antibodies in serum samples. Electron microscopy allows direct visualization of FMDV particles in clinical samples.

Differential Diagnosis

Other vesicular diseases with similar clinical signs, particularly vesicular stomatitis, swine vesicular disease, and vesicular exanthema.

Treatment

The lesions should be washed with 1 percent potassium permanganate or other antiseptic solution 3-4 times a day. Washing the foot lesions with 2 percent copper sulphate solution is also effective. Fly repellents are useful to avoid maggot formation. In secondary bacterial infection, enrofloxacin @ 2.5-5.0 mg/kg IM OD may be given for 7 days along with NSAIDs (flunixin meglumine @ 0.5-2.2 mg/kg BW, IM/IV, OD, SOS) and antihistaminics (chlorpheniramine maleate @ 0.5-1.0 mg/kg BW, IM, OD, for 3 days). Vitamin A @ 440 IU/kg BW, IM may be given once in a week for better results. Alternatively, vitamin C @ 10 mg/kg, BW, IM may also be given.

Control

Vaccination of farm animals on a regular basis using inactivated trivalent FMD vaccine as recommended under National Animal Disease Control Programme (NADCP) is the only way to control FMD. The first vaccination may be carried out at three months of age followed by a booster after a month. Subsequently, vaccines should be given once in every six months. In yaks, the protective antibody titre after FMD vaccine was reported to fall after 3 months; hence, the antibody titre in yaks may be monitored periodically and vaccination strategy can be made accordingly so as to maintain herd immunity.

Sodium hydroxide (2 percent), sodium carbonate (4 percent), and citric acid (0.2 percent) are effective to disinfect floor, farm premises, the surrounding area, and all other contaminated goods. The lime powder should be sprinkled in the surroundings of the animal premises. The farm's entry and exit ways should be equipped with a foot bath and wheel dip.

For herbal alternative treatment, refer EVM section.

Biosecurity Measures

Infected animals must be immediately isolated, and confined, and their movements must be restricted. Infected animals should must not be allowed to graze in common grazing pastures and drink water from common drinking facilities. The movements of animals and animal handlers or visitors should be restricted and regulated as per the farm design for effective biosecurity. Farm personnel and visitors should use 4 percent sodium carbonate solution for disinfection of farm equipment, and utensils and clean themselves. Calves should not be fed milk from infected animals. Introduction of newly purchased animal in the herd is to be suspended during outbreaks.

Samples to be Collected

Tissue samples from the affected areas like epithelial tissues or membrane, vesicular fluid, saliva, milk, and blood, myocardial tissues are collected for virus isolation and molecular diagnosis while serum samples are collected for sero-diagnosis and sero-monitoring.

9.3.3 Infectious Bovine Rhinotracheitis

Definition and Causative Agent

Infectious bovine rhinotracheitis (IBR) is a highly contagious viral disease primarily affecting cattle, but can also infect goat, sheep, buffalo, mithun, and yak. Because of the endemicity of the disease in India, the dairy industry has suffered significant financial losses as a result of decreased milk production, repeated breeding, infertility and miscarriages. IBR is caused by bovine herpesvirus-1 (BoHV-1) classified under the genus *Varicellovirus* of the subfamily Alphaherpesvirinae under the family Herpesviridae.

Transmission

Direct contact with infected animals or with their respiratory and genital secretions are important transmission routes. Infection may occur from contaminated equipment, feed, water, human handlers and other fomites. This virus can establish latency and reactivate during periods of stress in the animals causing intermittent shedding of the viral particles. Yaks and mithuns are reared in the high mountainous terrains where animals are exposed to extreme stress and nutritional deprivation during prolonged and harsh winter. During such conditions, the virus gets a convenient opportunity to reactivate and start secreting through nasal and genital secretions.

Clinical Signs

Bovine herpesvirus causes diverse ailments, *viz*., infectious bovine rhinotracheitis (IBR), infectious pustular vulvovaginitis (IPV), infectious balanoposthitis, keratoconjunctivitis and neurological disorders. The different forms of clinical manifestations of IBR range from mild to severe and infection may affect the respiratory tract, genital tract or ocular tissues. In yak, BHV-1 was recorded to precipitate mild respiratory, abortive and ocular form. In general; fever, nasal secretion, keratoconjunctivitis, vaginal discharges and abortion usually in the second half of gestation are the most commonly seen symptoms.

Lesions

Upper respiratory tract and genital tract may show petechial to ecchymotic haemorrhages and significant necrotic and inflammatory changes. Pharyngeal and pulmonary lymph nodes are usually swollen and haemorrhagic. Aborted foetus may show lesions in all the major organs, particularly liver.

Diagnosis

Serological tests such as ELISA for detection of antibodies, and virus neutralization tests and molecular techniques like PCR can be used for antigen or genomic detection. Virus isolation from nasal swabs, conjunctival swabs, or genital secretions will provide a confirmatory diagnosis. The virus can be demonstrated in foetal tissues by PCR, immunoperoxidase, or fluorescent antibody staining.

Differential Diagnosis

Malignant catarrhal fever, mucosal disease (bovine viral diarrhoea), foot and mouth disease and other respiratory illness like bovine parainfluenza, bovine respiratory syncytial syndrome, bovine coronavirus infection, bacterial pneumonia and lungworm infection.

Treatment

There are no specific antiviral drugs for treatment of IBR in animals. Management primarily focuses on supportive care and prevention of secondary bacterial infection. Ceftiofur @ 2.2 mg/kg BW IM for 7 days may provide relief from respiratory infection and enrofloxacin @ 5 mg/kg BW OD IM may be given for 5 days in urogenital infections. Supportive care includes access to good quality feed and drinking water *ad libitum*. Anti-inflammatory drugs (flunixin meglumine @ 1.1-2.2 mg/kg IM/IV OD for 3 days) can help reduce fever and inflammation along with chlorpheniramine maleate @ 0.4 to 0.5 mg/kg BW/day for 3 to 5 days. Commercial multivitamin preparation and mineral mixture formulation may help in boosting immunity.

Control

In regions where IBR is prevalent, screening of animals and culling of carrier animals are important. Good management practices such as minimizing stress through proper handling, adequate feeding and optimal housing conditions are essential. Providing adequate nutrition to support immune function and ensuring proper ventilation in animal sheds may reduce the risk of respiratory infections, and help in controlling of outbreaks.

Biosecurity Measures

Implementing effective biosecurity measures is

crucial in preventing the introduction and spread of BHV-1 in a herd. Quarantine of all newly purchased animals for at least 2-4 weeks before introducing them to the main herd is crucial. Movement of yak and mithun should be restricted to avoid their closeness with cattle and wildlife. Regular disinfection (0.5 percent NaOH, 0.01 percent $HgCl_2$, 1 percent chlorinated lime, 1 percent phenolic derivatives, 1 percent quaternary ammonium bases or 10 percent Lugol's iodine) is helpful in preventing the disease incursions.

Samples to be Collected

Serum for serological assays and foetal tissue for virus isolation and molecular diagnosis.

9.3.4 Bovine Viral Diarrhoea

Definition and Causative Agent

Bovine viral diarrhoea (BVD) – caused by bovine viral diarrhoea virus (BVDV) – is a significant viral disease of ruminants which can cause respiratory, gastrointestinal, and reproductive disorders. The BVDV is a member of the genus *Pestivirus* within the family Flaviviridae. BVDV exists in two biotypes – cytopathic (CP) and non-cytopathic (NCP), with NCP being more common and associated with persistent infections (PI).

Transmission

The disease is primarily transmitted through direct contact with infected animals, particularly through bodily secretions like nasal discharge, saliva, urine, faeces, and semen. Persistently infected (PI) animals, which harbour the virus for life, are the main source of transmission, shedding large amounts of the virus into the environment. Vertical transmission from an infected dam to her foetus can occur, leading to the birth of PI calves.

Indirect transmission can also occur through contaminated equipment, feed, water, and fomites. Additionally, the virus can spread through blood-sucking insects, such as mosquitoes and flies. Insemination with contaminated semen is another potential route of transmission.

Clinical Signs

The disease can be manifested as an acute infection with fever, diarrhoea, nasal discharge, oral erosions, and immunosuppression leading to secondary infections. Infertility, abortions, congenital defects, and stillbirths are also found. The mucosal form of the disease (MD) is the predominant form with high fever, severe diarrhoea, and erosive lesions in the gastrointestinal tract.

Lesions

Erosions and ulcers can be seen on the tongue, gums, palate, oesophagus, rumen, intestines, and abomasum along with depletion and necrosis of Peyer's patches in the intestines.

Diagnosis

Confirmatory diagnosis is achieved by laboratory tests such as virus isolation, PCR for viral genomic detection, ELISA for viral antigen detection, and serology for antibody detection; and histopathological examination of tissue samples for observing characteristic lesions specific to BVD.

Differential Diagnosis

Foot-and-mouth disease, malignant catarrhal fever, and infectious bovine rhinotracheitis and other diarrheal diseases based on other characteristic lesions and laboratory examination.

Treatment

There is no specific treatment for BVD. Supportive care needs adequate fluid and electrolyte therapy to prevent dehydration. Further, NSAIDs like flunixin meglumine @ 1.1-2.2 mg/kg BW (slow IV/IM) may be given to control inflammation and fever. Antidiarrheal preparation containing kaolin and pectin may be given. Commercial multivitamin and mineral mixture may be given to revitalize the infected animals.

Control

Testing herds for BVDV and culling of the persistently infected animals is the cornerstone for control of BVDV/MD in a herd. Avoiding a high stocking density and community grazing can greatly help in the control and prevention of BVD. Effective control strategies focus on identification and removal of PI animals, and stringent biosecurity practices to minimize the risk of introducing and spreading the virus in the herd.

Biosecurity Measures

Quarantine of the newly purchased animals for at least 28 days and testing the animals for BVDV before introduction to the herd, avoidance of contact with potentially infected animals, regular

cleansing and disinfection of equipment, premises and vehicles and use of protective clothing by the visitors and staff, help in control of the disease.

Samples to be Collected

Serum, milk, nasal swab, tissue (spleen, lymph node, and ulcerated segments of the GI tract).

9.3.5 Malignant Catarrhal Fever

Definition and Causative Agent

Malignant catarrhal fever (MCF) is a viral disease characterized by systemic illness and mucosal lesions causing significant economic impact due to mortality and reduced productivity. It is caused by members of the Herpesviridae family, particularly the alcelaphine herpesvirus-1 (AlHV-1) and the ovine herpesvirus-2 (OvHV-2). While the AHV-1 primarily affects wildebeests and other wild ruminants, OvHV-2 usually affects domestic sheep and can be transmitted to in-contact cattle, mithun and yak. These viruses are closely related to ruminant gamma-herpesviruses of the Macavirus genus.

Transmission

Transmission is possible directly by contact with infected animals or their secretions, such as saliva, nasal discharge, or ocular secretions, and indirectly via contaminated equipment or the environment.

Clinical Signs

Sudden onset of high fever, depression, anorexia, profuse discharge from the eyes and nose, ulcerative stomatitis, and mucosal erosions in the mouth, dyspnoea due to nasal and tracheal involvement, corneal opacity, head pressing, circling, incoordination and progressive weight loss are the characteristic findings.

Lesions

Ulcerative lesions in the mucosa of the oral cavity and upper respiratory tract are distinct. Necrotizing tracheitis, lymphadenopathy, multifocal hepatic necrosis, and other hepatic lesions may be observed in some cases.

Diagnosis

Characteristic clinical findings, history of exposure to sheep or wild ruminants, PCR to detect viral DNA, serological tests like VNT, immunoperoxidase technique, immunofluorescence, and ELISA can be indicative.

Differential Diagnosis

Bovine viral diarrhoea (BVD), infectious bovine rhinotracheitis (IBR), foot and mouth disease (FMD), and bluetongue.

Treatment

There is no specific treatment for MCF. Supportive therapy includes anti-inflammatory and antihistaminic drugs depending upon the severity of infection. Adequate nutrition and hydration need to be ensured. Visible mucosal lesions may be washed with local antiseptic lotion (Lugol's iodine/ 1 percent potassium permanganate).

Control

Avoidance of the contact with cattle and sheep or wild ruminants, isolation of new animals in the herd and close monitoring for specific clinical finding are helpful.

Biosecurity Measures

Physical separation between yak/mithun and potential reservoir hosts is to be maintained along with regular disinfection of equipment and premises. Animal should be closely monitored for any sign and promptly isolated, if suspected.

Samples to be Collected

Anticoagulated blood, kidney, intestinal wall, lymph node, and brain.

9.4 Parasitic Diseases in Yaks

9.4.1 Babesiosis

Definition and Causative Agent

Babesiosis is an acute tick-borne disease of adult mithun, yak and yak-cattle hybrids. The disease is characterised by sudden dullness, inappetence, very high fever (≥105°F), haemoglobinuria, and mortality. It is caused by the blood protozoa *Babesia bigemina*.

Transmission

The disease is transmitted by the *Boophilus microplus* tick. Cattle act as a reservoir for yaks and yak-cattle hybrids. Outbreaks usually occur during autumn when yaks and yak-cattle hybrids migrate from alpine pastures to mid-altitude pastures recently grazed by cattle.

Clinical Signs

The disease usually starts with sudden onset of

dullness, anorexia, and mortality of a few lactating animals in the herd. High fever and dark-colour urine may accompany.

Lesions

Lesions include pale and icteric mucous membrane, thin/watery blood, an enlarged icteric liver with a distended gallbladder, and an enlarged spleen with a friable consistency. The kidneys are black in colour, and the urinary bladder contains brown-colour urine.

Diagnosis

The disease can be confirmed by microscopic detection of the parasite in a Giemsa-stained blood smear and PCR-based identification of *B. bigemina.*

Differential Diagnosis

The disease should be differentiated from other conditions causing dark-colour urine prevalent at high altitudes.

Treatment

Diminazene aceturate @ 3.5 mg/kg BW IM is usually effective to control the infestation. NSAIDs like flunixin meglumine @ 1.1-2.2 mg/kg BW slow IV/IM SOS should be used as antipyretic. Glucose therapy (DNS 5 percent @ 5 ml/kg BW, I/V) in afebrile animal may help promote restoration of hepatic function. Commercial hematinic preparation may be used in anaemia.

Control

Tick control in combination with the early diagnosis and treatment of susceptible yak populations is the only feasible measure, especially in endemic areas.

Biosecurity Measures

The biosecurity measure of this tick-borne disease should aim at control of tick population in the endemic areas. Periodic spraying of acaricides may help control the tick population in a herd. The yaks living in close contact with the cattle should be checked regularly.

Samples to be Collected

Blood, ticks.

9.4.2 Coccidiosis

Definition and Causative Agent

Coccidiosis, caused by an apicomplexa protozoa under the genus *Eimeria*, is an intestinal disease affecting multiple species of animals including yak and mithun calves. Although mostly asymptomatic, it may lead to diarrhoea (with or without mucus), hematochezia, lethargy, weight loss, vomiting, signs of abdominal pain, pallor, anorexia and chronic emaciation.

Transmission

Young calves get infected through accidental ingestion of oocyst passed through the infected faeces.

Clinical Signs

Most infected calves are asymptomatic, even if oocysts are detected in faecal examination. Clinical forms may accompany lethargy, inappetence, and diarrhoea (sometimes blood-stained). Clinical exhibits are mostly observed in calves housed in poor flooring with water stagnation or in calves under stress.

Lesions

Diffuse inflammation may be observed in various areas of small or large intestine with pinpoint haemorrhages on mucosal surface. Loss of epithelial surface and villous atrophy are evident in histopathology.

Diagnosis

Modified McMaster technique with flotation method is an effective tool in detection and quantifying the load of *Eimerian* oocysts per unit of faecal matter.

Differential Diagnosis

Other diarrheal conditions caused by bacteria, viruses, or parasites.

Treatment

Coccidiosis is a self-limiting disease; therefore, in subclinical cases, treatment is not advised. Under severe infection, oral medication with amprolium @10 mg/kg BW or sulphaquinoxaline @ 13 mg/kg BW for 5 days is effective.

Control

Good floor hygiene in the calf-pen, prevention of moisture stagnation and faecal contamination of water reduces the incidence of the clinical disease.

Biosecurity Measures

The farm should be well ventilated and overcrowding of the animals should be avoided. The feeding and

watering utensils, pans, floor and farm environment should be regularly disinfected using 10 percent bleach solution.

9.4.3 Toxocariasis

Definition and Causative Agent

Toxocariasis, caused by the ascarid *Toxocara vitulorum,* is the leading cause of mortality in young yak and mithun calves particularly under 6 months of age.

Transmission

Prenatal and trans-mammary infections constitute the major modes of transmission for young calves.

Clinical Signs

Inappetence, weakness, and intermittent diarrhoea accompanied by colic, mud-colour foul- smelling faeces are the major signs. In many cases, adult worms are passed in faeces. The calves with heavy infection die mostly due to intestinal obstruction, within a few days from the onset of clinical signs.

Diagnosis

Detection of ova in the faecal examination and an adult worm passed out in faeces or ova/adult worm detected on necropsy are indicative of the disease. Eggs appear in the faeces of calves as young as 3-week-old, and are easily recognised by their thick, pitted shells and dark brown centres.

Treatment

Piperazine citrate @ of 200-300 mg/kg BW PO once or fenbendazole @ 5 mg/kg BW PO once, or levamisole @ 7.5 mg/kg BW PO or ivermectin @ 200 mcg/kg BW SC is effective.

Control

In endemic areas, preventive deworming of calves at 10th day of birth can minimize the infestation. Prevention of milk feeding from infected dams is advocated. Regular faecal examination of calves, prompt deworming in positive cases, and pasture rotation should be practised to prevent reinfection. Deworming of pregnant yak and mithun dams with fenbendazole 2-4 weeks before the expected date of calving could be beneficial.

9.4.4 Helminth Parasites

Definition and Causative Agent

Yaks and mithuns, like other grazing ruminants, are commonly infected by several helminth parasites. These parasites impact their growth and production and sometimes causes diarrhoea, anorexia or inappetence and reduced milk yield. Helminth parasites recorded in yaks include *Haemonchus contortus*, *Strongyloides papillosus*, *Mecistocirrus digitatus*, *Ostertagia* spp., *Bunostomun* spp., *Trichostrongylus* spp., *Trichuris lobulosa*, *Capillaria* spp., *Moniezia* spp., *Fasciola gigantica*, *Dicrocoelium dentriticum,* Amphistomes, *etc.* Gastrointestinal nematodes and *Moniezia* spp. are commonly encountered in routine faecal examinations. *Fasciola* and *Dicrocoelium* have been reported from isolated pockets, mostly at necropsy. Mithuns are also affected by these gastrointestinal helminths; however, clinical cases are relatively less.

Transmission

Oral ingestion of infective larvae from pasture is the main source of infection. Helminthic diseases are seasonal in occurrence, with a higher level of infection during the warmer months. Infected yaks/mithuns pass parasitic eggs in the faeces on the pasture, where the eggs develop into infective larvae. The seasonality of parasitism is different for free-range and farmed animals. In yaks or mithuns maintained on organised farms, parasitic load is higher during the rainy season. In contrast, free ranging yaks/mithuns, infestation is at the peak during spring, when they come in contact of other domestic ruminants in lower altitude. Infection slowly decreases, as they start up migrating to the alpine pastures.

Clinical Signs

Clinical diseases are rare, since the parasite load in free-ranging animals is generally low. In a few cases, general symptoms of parasite infestation are retarded growth, weight loss, a dull body coat, and occasional diarrhoea, especially among young calves.

Diagnosis

Detection of eggs in the faecal examination or detection of adult parasites at necropsy is indicative.

Treatment

A broad-spectrum anthelmintic is effective in treating GI nematode infection. Commonly used anthelmintics such as albendazole, mebendazole and fenbendazole at an oral dose of 5 mg/kg BW or levamisole @ 7.5 mg/kg BW PO or ivermectin

@ 200 mcg/kg BW SC are effective. In a mixed infestation of GI nematode with cestode or fluke, a combination of fenbendazole @ 5-10 mg/kg BW PO and praziquantel @ 5 mg/kg BW PO is effective.

Control

Half-yearly deworming is usually effective in yaks and mithuns. The first deworming is done before the commencement of migration in late May to early June, and the second in October, when the herd usually migrates to lower altitude. In addition to deworming, the pasture rotation helps keeping parasitic infestation under control. However, decision on deworming should be taken judiciously to avoid anthelmintic resistance.

9.5 Systemic diseases

9.5.1 Dietary Diarrhoea

Definition and Etiology

It is probably the most common systemic illness encountered in mithun and yak calves. Ingestion of poor-quality milk replacer, excess milk and excess starchy food may cause such illness. Undigested substances often lead to bacterial proliferation in the gut complicating the condition.

Pathophysiology

Animals suffer from dehydration and circulatory failure due to progressive fluid loss. Loss of sodium, potassium and bicarbonate is most common. Hypokalemia causes muscle weakness while bicarbonate loss may lead to metabolic acidosis. Profuse diarrhoea causing severe dehydration may result in renal hypoperfusion, cortical necrosis and acute renal failure, which is a life-threating condition.

Clinical Findings

Profuse offensive diarrhoea, tympany, increased pulse rate, colic, prostration, and sunken eye ball are evident. Auscultation of intestinal region may reveal gurgling sound. In severe dehydration, animal may become hypothermic.

Laboratory Findings

Biochemical profile indicating values of serum creatinine, blood urea nitrogen (BUN), sodium, potassium, bicarbonate and chloride and PCV is a useful indicator.

Treatment and Management

Correction of the fluid and electrolyte balance is the mainstay of therapy. In severe dehydration, Ringer's lactate @ 100 ml/kg BW IV for first 4-6 hours followed by 140 ml/kg BW IV for next 20-24 hour is to be given. Use of adsorbents or protectants like kaolin (calves 20 g; adult 200 g), bismuth subcarbonate (calves 5 g; adult 20 g) BID, PO is helpful and can be continued, until the condition improves.

If there is evidence of hypokalemia, 10 ml solution of 15 percent potassium chloride may be given with 500 ml of 5 percent dextrose with extreme caution. If there is any indication of cardiac dysrhythmia, the therapy should be discontinued. Daily dose should not exceed 6 g/50 kg BW.

Precaution: The therapy for hypokalemia - with 10 ml solution of 15 percent potassium chloride given with 500 ml of 5 percent dextrose – should be given with extreme caution. If there is any indication of cardiac dysrhythmia while treating hypokalemia with 15 percent potassium chloride, the therapy should be discontinued.

Various indigenous antidiarrhoeal/anti-scour preparation can be given following the instruction of the manufacturer. Unnecessary use of antibiotics and anthelmintic should be avoided, unless there is evidence of infection. The calves should be fed at least 2 kg of colostrum within first 6 hours of birth. Maintaining proper hygiene and cleanliness as well as adequate nutritional supplement helps to control such conditions.

9.5.2 Bloat / Tympany

Definition and Etiology

In tympany, rumen and reticulum get filled with excessive gas resulting from excessive intake of easily fermentable feeds. Excessive feeding of leguminous fodders, soluble carbohydrate, finely powdered grains – all can contribute to gas production. Further, the slime produced by certain bacteria prevents coalescence of gas particles resulting in frothy bloat or primary tympany.

Mechanical or physical obstruction of esophagus due to foreign objects or space occupying lesions like enlarged retropharyngeal lymph node may lead to secondary tympany or free-gas bloat.

Pathophysiology

Grazing animals may develop tympany with

excessive intake of green or leguminous plants, which is more commonly known as pasture bloat. The saponin, hemicellulose and pectin in plants and leaf cytoplasmic protein all may help in the formation of foams, which are stable in acidic pH. Further, excessive feeding of finely ground grains and less roughage in feed helps in formation of excessive gas. Alteration of diet may also change rumen flora leading to formation of bloat. Excessive ruminal gas may press lung and diaphragm causing severe dyspnea and asphyxia. Displacement of heart may result in sudden cardiac failure and death.

Clinical Findings

Abdominal engorgement is mostly evident at left paralumbar fossa. The animal is dyspneic and extend its head and neck and may show tachycardia, increased respiration rate, grinding of teeth, and colic. Auscultation of cardiac region may reveal cardiac murmur. Sudden death due to cardiac failure may be observed.

Laboratory Findings

Ruminal pH falls below 6.3 with increased time for methylene blue reduction. Sedimentation activity time of ruminal fluid is increased.

Treatment and Management

Turpentine oil (20-30 ml) added with linseed oil (400-450 ml) or 400 ml of mineral oils (soyabean oil) may be given to an adult yak or mithun through stomach tube. Mineral oil helps in breaking down of the foams. Commercial preparations containing simethicone or dimethicone may be used as indicated. A wooden stick may be tied to the mouth to help salivation and reduced fermentation. As this may cause sudden death, excessive and uncontrolled tympany may be treated with trocarisation with a long needle and cannula. Passage of stomach tube or probang may help get the gas out of rumen. Emergency rumenotomy can save those animals that are unresponsive to other treatments.

Ration should be refined with addition of ~15 percent of chopped roughage, and it is always advisable to avoid grounding or dusting the grains. Periodic feeding of antifoaming agent like linseed/ soybean oil is helpful. Antifoaming agent may be sprayed over the tympanogenic pasture.

9.5.3 Epistaxis

Definition and Etiology

Epistaxis is the bleeding from the nasal cavity and it is fairly common in yaks. It is usually due to leakage or damage of the capillaries of the nasal mucosa.

Often it is difficult to ascertain the exact cause of such bleeding. While external injuries and leech infestation may be detected in several cases, other systemic diseases like poisoning, tumour, nasal granuloma or blood protozoa infection cannot be ruled out. In many yaks, it may become a recurrent problem. Exercise-induced pulmonary haemorrhage may be seen in yaks when they are migrating to the higher terrains.

Pathophysiology

It is mostly due to the damage of the surface capillaries of nasal mucosa which causes the bleeding. Animal may become weak and anemic, if not treated on time.

Clinical Findings

Unilateral or bilateral nasal bleeding, hyperirritability, sneezing and shaking of heads and sometimes fever may be noticed. The color of the blood may be fairly indicative of its origin. If it is of nasal origin, it is bright red.

Laboratory Findings

The animal should be checked for any parasite, foreign object, lesion or external injury in the nasal cavity. Blood smear should be checked for complete blood count, specially, thrombocyte count and haemoprotozoan infection. Radiography of the head, neck and thorax is helpful for detection of local injury or lesion.

Treatment and Management

Cold water or ice pack compression on the frontal and nasal region after proper restraining of the animal, instillation of adrenaline (1:1000) in the affected nose and application of cotton swab with lidocaine and adrenaline is helpful. Alternatively, 1 ml of 1 percent lidocaine with 1:100,000 epinephrine may be instilled into the greater palatine foramen. The underlying cause should be treated.

9.5.4 Anaemia

Definition and Etiology

Anaemia is a condition caused by either fall in the number of RBCs or quantity of haemoglobin or both in per unit volume of blood. The disease is characterized by pale mucus membrane, increased

heart rate, dyspnea, weakness, loss of body condition and multiple organ failure in severe cases.

Multiple etiological factors may be involved in anaemia. In yaks, macrocytic normochromic anaemia and normocytic hypochromic anaemia are seen mainly because of deficiency in essential vitamins and trace minerals like copper, cobalt, iron and zinc, particularly during long period of nutritional deprivation in harsh winter. Persistent protozoan infection like *Babesia* may cause macrocytic hypochromic anaemia while pyrrolizidine (*Senecio spp.*) alkaloid poisoning may lead to normocytic, normochromic anaemia.

Pathophysiology

Reduction in oxygen supply to the vital organ leads to compensatory tachycardia and may increase respiration. In prolonged anaemia, animals become weak and vital organs may fail to function because of long standing tissue anoxia. Increased force of cardiac pumps and increased heart rate may cause cardiomegaly and animal may die of cardiac failure.

Clinical Findings

Pale or blanched mucus membranes, tachycardia, dyspnoea, extreme fatigue and muscular weakness are the prominent findings. Cardiac murmur may be heard on auscultation.

Laboratory Findings

Either the total RBC count or haemoglobin level or both may fall distinctly. The liver enzymes (aspartate amino-transferase, alanine amino-transferase, ornithine carbamyl transferase, gamma-glutamyl transferase, alkaline phosphatase) and kidney parameters (BUN and creatine) should be checked to rule out the possible involvement of any poisoning. Hepatomegaly and splenomegaly may be observed depending upon the associated etiology.

Treatment and Management

Treatment should be decided based on the causative factor. Symptomatic treatment may be done in yaks using the following formulation – particularly when deficiency is suspected – comprising of Ferric sulphate-5g, Copper sulphate -2g, Zinc chloride-0.2g, Cobalt sulphate-0.2g, and Treacle-QS. This formulation should be given orally for a month or until the condition improves.

Commercial preparations of iron dextran injection @ 2.5-5.0 mg/kg BW IM and multivitamin injection/ supplement (copper, cobalt and iron) may be used as per instructions of the manufacturer.

9.5.5 Hepatic Disorders

Definition and Etiology

Hepatic disorders are common in all ruminants including yak and mithun. Hepatic diseases in yaks may be obstructive (in parasitic infestation), or due to pyrrolizidine alkaloid toxicosis, hepatic lipidosis, hepatic abscesses, cholangiohepatitis, endotoxaemia, or because of other unknown factors.

Precaution: Hepatitis, the inflammation of hepatic cells, may be acute or chronic in nature and should be treated with extreme caution.

Pathophysiology

The hepatic insult caused by any pathogen or poison causes hepatocytolysis or hepatic cell degeneration and subsequently infiltration by mononuclear cells. The damage is prominent in the centrilobular region. There is obvious loss of glycogen storage leading to hypoglycaemia. Loss of plasma protein may cause a fall in plasma colloidal osmotic pressure and development of oedema. Failure to detoxify the microbial or food protein may lead to accumulation of blood ammonia and neurological signs.

Clinical Findings

Clinical findings are non-specific and may be misleading except when it is clinically apparent as icterus, which may range from mild to severe. Anorexia, diarrhoea or constipation, recurrent fever, weight loss, oedema, dullness or prolonged prostration, muscle tremor or nervous signs, poor hair coat may be found.

Laboratory Findings

Blood smear should be checked for complete blood count, and haemoprotozoan infection. Fecal sample is to be analyzed for parasite eggs/larva.

Estimation of plasma proteins, clotting time, gamma-glutamyl transferase (GGT), sorbitol dehydrogenase (SDH), glutamate dehydrogenase (GLDH), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), bilirubin, and bile acid concentrations in blood are very useful to evaluate hepatic dysfunction. In yak and mithun, these enzymes should always be compared with their healthy counterparts in the same herd.

Percutaneous liver biopsy may be conducted for histopathological observation. Ultrasonographic evaluation of the hepatic echotexture is useful.

Treatment and Management

Adequate, easily digestible, bioavailable carbohydrate and low protein with good quality amino acid and fat-free diet is the mainstay of therapy. Dextrose 25 percent @ 500 ml to 2 litre/adult yak or mithun OD for 10 days or until the condition improved. Probiotic formulation containing probiotic strains like *Lactobacillus*, *Saccharomyces cerevisiae*, *Streptococcus, Enterococcus, Lactococcus*, and bifidobacteria @ 10^9 CFU/kg of feed for 1-3 months may be helpful.

Commercial preparation of vitamin B complex with liver extract may be given as suggested for 7 days.

For herbal alternative treatment, refer EVM section.

Protozoan or parasitic infestation to be treated with broad-spectrum anthelmintics such as benzimidazoles.

9.5.6 Pneumonia

Definition and Etiology

The term pneumonia – or pneumonitis – means inflammation of lung. Bronchopneumonia is pneumonia accompanied with inflammation of bronchioles, and pleuropneumonia is pneumonia accompanied with pleuritis.

The disease may be due to various etiological factors. Yak and mithun are infected by bacterial pathogens (*Staphylococcus*, *Streptococcus*, *Klebsiella*, *Mycoplasma*, *Pasteurella*, *Mycobacterium, etc.*), viral (BHV-1, Adeno or rhino virus) pathogens and other etiological triggers like aspiration pneumonia.

Pathophysiology

Infective organism or foreign elements cause acute fibrinous reaction with formation of exudates in lung. Subsequently, secondary pathogens get entry as the inflammation proceeds with bronchiolitis and alveolitis. Chronic caseous or granulomatous changes develop in long standing infections.

Clinical Findings

Fever, anorexia, dullness, moist painful cough, nasal discharge and laboured breathing are evident. Adventitious breath sounds are audible on auscultation on lung region and heart sound is usually muffled. Thoracic lymph nodes are usually enlarged.

Laboratory Findings

Complete blood count, specially, differential leucocyte count is usually indicative. Bronchial and tracheal exudate are to be collected for detection of pathogens. Radiographic examination of lung is useful.

Treatment and Management

Enrofloxacin @ 2.5-5.0 mg/kg BW/day SQ/IM for 7 days or Streptomycin sulphate 2.5-5.0 g BID I/M for 7 days or ceftiofur @1.1-2.2 mg/kg BW IM for 7 days may be given, if there is evidence of bacterial infection.

Precaution: Enrofloxacin should not be used in young animals.

Dexamethasone @ 0.4 mg/kg OD IM may be given to induce anti-inflammatory and antiallergic response.

Precaution: Dexamethasone should not be given in pregnancy and also if viral infection is suspected. Antihistaminic compound diphenhydramine hydrochloride @ 0.5-1.0 mg/kg BW IM OD for 3 days. If there is accumulation of fluid in lung, furesomide @ 0.5-1.0 mg/kg BW IM or IV may be given SOS.

9.5.7 Dermatitis and alopecia

Definition and Etiology

Dermatitis and alopecia are fairly common conditions in yaks and mithuns. The condition is usually acquired and mostly common after a prolonged nutritional deprivation. Besides, bacterial infection, dermatophytosis, parasitic infestation or secondary alopecia due to systemic disorders like hepatopathy may be the cause.

Pathophysiology

Acquired alopecia because of the infection starts due to temporary or permanent destruction of the hair follicles. Prolonged deficiency of protein or trace minerals may cause slow growth or replacement of hair follicles. Similarly, peripheral neuropathy or skin eruptions may lead to discomfort and self-trauma, which may subsequently turn into patchy alopecia. The exact pathophysiology is not understood in yak and mithun.

Clinical Findings

There is a general loss of hair throughout the body which can be diffused or localized. Papules, vesicular eruptions with crust formation may be present. The animals exhibit extreme irritation, itching and pruritus like licking, rubbing, scratching.

Laboratory Findings

Examination of hairs with follicles, skin scrapings, trichogram, cutaneous and auricular cytology are useful tools to arrive at specific diagnosis. Skin biopsy, complete blood count and serum chemistry can be useful, if the condition does not improve with conventional therapy and also to identify systemic anomalies.

Treatment and Management

Removal of the hairs, debris and tissues by thorough cleansing with medicated shampoos or by applying lukewarm water or normal saline solution followed by gentle rubbing with medium soft brushes.

Topical antibiotics (tetracycline or gentamicin) and antifungals (griseofulvin) should be applied three times a day (TID).

List of Contributors

Oral supplementation of vitamin A and mineral mixtures based on nutritional profiling of the soil should be provided.

Injection of chlorpheniramine maleate @ 0.5-1.0 mg/kg BW, IM, OD for 5 days may reduce pruritus.

***9.5.8* Parturient Paresis**

Definition and Etiology

Parturient paresis is not as common in yaks and mithuns as in dairy cattle. However, yaks and mithuns occasionally suffer from this condition following parturition. It is mainly due to fall in blood ionized calcium level. Although it is not well established in yaks or mithuns, if the blood calcium level drops to 5-6 mg/dL as in cattle, it can result in parturient paresis.

Pathophysiology

Drop in calcium may cause nervous excitability initially and it may reduce muscle contractility leading to paresis.

Clinical Findings

Muscle weakness, flaccidity, tremors, cold extremities, and inability to stand up after parturition leading to sternal recumbency are usual findings. Prolonged recumbency may lead to bloat, dyspnoea, and pressure atrophy resulting in downer cow syndrome.

Laboratory Findings

A history of parturition and specific clinical findings may indicate the condition. Estimation of blood calcium level is helpful to differentiate it from conditions like traumatic injury, calving paralysis, hypokalemia, hypophosphatemia and poisoning.

Treatment and Management

Treatment with calcium borogluconate 25 percent solution @ 500 ml, slow IV is indicated at the earliest. Alternatively, compounds containing Ca-Mg-boro-gluconate @ 200- 350 ml, IV followed by SC route may be given. The cardiac rate should be monitored during intravenous calcium injection to rule out arrythmia.

Precaution: Always monitor the cardiac rate - while administering intravenous calcium injection - to rule out arrythmia.

Prevention and Control

Provision of a high phosphorus and low calcium diet during the last month of pregnancy, and a single IM injection of vitamin D_3 @10 million units 72 hours before calving and feeding of calcium gel containing about 50 percent calcium chloride during the time of parturition are effective in preventing the condition.

Advisory Committee Experts

Dr Abhijit Mitra
Animal Husbandry Commissioner
Department of Animal Husbandry & Dairying, Government of India,
Chanderlok Building, 36, Janpath, New Delhi-110 001, INDIA

Dr Raghavendra Bhatta
Deputy Director General (Animal Science)
Animal Science Division,
Indian Council of Agricultural Research (ICAR)
Krishi Bhavan, Dr Rajendra Prasad Road,
New Delhi-110 001, INDIA

Dr KML Pathak
Fmr Deputy Director General (Animal Science), ICAR
Tower B2, Flat No. 2204, Cleo County,
Rani Avantibai Marg, Sector-121, NOIDA, UP, INDIA

Dr Devendra Swarup
Fmr Director, ICAR-CIRG
Flat# 32152, Prestige Falcon City,
Konanakunte Cross, Kanakpura Main Road,
Bengaluru-560062, INDIA

Dr S K Agarwal
Fmr Director, ICAR-CIRG
76, North City, Pilibhit Road Byepass,
Post-Izatnagar-243 122, Bareilly, UP, INDIA

Dr N Punniyamurthy
Emeritus Professor
Mill Complex, 8th Street
Rethinasamy Nagar,
Thanjavur-613006, Tamil Nadu, INDIA

Dr N K Mahajan
Retd Dean, College of Dairy Science & Technology, LUVAS
House No. 448, Sector 15A,
Govind Place Road, Hisar-125001, INDIA

Experts from Research & Academia

Dr Praveen Malik
Chief Executive officer (CEO)
AgrInnovate, NASC Complex, Dev Prakash Shastri Marg,
Krishi Kunj, Inder Puri, New Delhi, Delhi-110012, INDIA

Dr Mahesh Kumar
Fmr Director, CoVAS, Pant Nagar
C-23, Green Park,
Kashipur Road, Rudrapur-263153
Udham Singh Nagar, Uttarakhand, INDIA

Dr Ramesh Somvanshi
Fmr Joint Director (CADRAD), ICAR-IVRI
A-336, Rajendra Nagar
Bareilly-243 122, UP, INDIA

Dr M N Balakrishnan Nair
Emeritus Professor
74/2 Jarakabandekaval, Attur post, via Yelahanka,
Bengaluru -560064, Karnataka, INDIA

Dr R P Pandey
Director Clinics
UP PT DDU Pashuchikitsa Vishwavidyalaya Evam Go Anusandhan Sansthan
DUVASU, Mathura, UP, INDIA

Dr J L Singh
Professor & Head
Department of veterinary Medicine,
College of Veterinary and Animal Sciences,
GB Pant University of Agriculture and technology
Pantnagar-243145
Distt: U S Nagar, Uttarakhand, INDIA

Dr (Ms) M A Kshama
Professor, Department of Veterinary Medicine,
College of Veterinary Science & Animal Husbandry,
Karnataka Veterinary, Animal, and Fishery Sciences (KVAFSU),
Bengaluru-560024, INDIA

Dr Ravindra Sharma
Consultant Animal Welfare
R-265B GF, Greater Kailash-1,
New Delhi-110048, INDIA

Dr Thakur Uttam Singh
Head (Acting), Division of Pharmacology & Toxicology,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI), Izatnagar-243 122, Bareilly, UP, INDIA

Dr (Ms) Anu Rahal
Principal Scientist

ICAR-Central Institute for Research on Goat (ICAR-CIRG)
Makhdoom, PO-Farah, Mathura, UP, INDIA

Dr Ujjwal Kumar De
Senior Scientist
Division of Medicine
ICAR-Indian Veterinary Research Institute (ICAR-IVRI),
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Vivek Joshi
Scientist (SS)
Division of Medicine
ICAR-Indian Veterinary Research Institute (ICAR-IVRI),
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Raguvaran R
Scientist (SS)
Division of Medicine
ICAR-Indian Veterinary Research Institute (ICAR-IVRI),
Izatnagar-243 122, Bareilly, UP, INDIA

Dr K Mahendran
Senior Scientist
Division of Medicine
ICAR-Indian Veterinary Research Institute (ICAR-IVRI),
Izatnagar-243 122, Bareilly, UP, INDIA

Dr A M Pawde
Principal Scientist, Division of Surgery and Radiology
and Incharge, Centre for Wildlife
ICAR-Indian Veterinary Research Institute (ICAR-IVRI), Izatnagar-243 122, Bareilly, UP, INDIA

Dr Ricky Jhamb
Asstt Professor
Department of Veterinary Medicine, College of Veterinary Sciences,
Lala Lajpat Rai University of Veterinary & Animal Sciences (LUVAS),
Hisar, Haryana, INDIA

Dr Shanker K Singh
Associate Professor
Department of Veterinary Medicine,
College of Veterinary Science & Animal Husbandry,
UP Pt Deen Dayal Upadhyay Pashuchikitsa Vishvavidyalaya Evam Go Anusandhan Sansthan (DUVASU), Mathura-281 001, UP, INDIA

Dr Ashish Srivastava
Asstt Professor
Department of Veterinary Medicine,
College of Veterinary Science & Animal Husbandry,
UP Pt Deen Dayal Upadhyay Pashuchikitsa Vishvavidyalaya Evam Go Anusandhan Sansthan (DUVASU), Mathura-281 001, UP, INDIA

Dr Amarpal
Principal Scientist, Division of Surgery and
Incharge TVCC & RVP
ICAR-Indian Veterinary Research Institute (ICAR-IVRI),
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Rekha Pathak
Principal Scientist
Division of Surgery,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI),
Izatnagar-243 122, Bareilly, UP, INDIA

Dr A C Saxena
Scientist (Sr Scale),
Division of Surgery,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI),
Izatnagar-243 122, Bareilly, UP, INDIA

Dr H P Aithal
Principal Scientist & Station Incharge
Training & Evaluation Centre
ICAR-Indian Veterinary Research Institute (ICAR-IVRI), Pune Centre
Pune, Maharashtra, INDIA

Dr D B Mondal
Principal Scientist & Head,
Division of Medicine,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI), Izatnagar-243 122, Bareilly, UP, INDIA

Dr Kiranjit Singh
Principal Scientist & Head
Division of Surgery,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Rohit Kumar
Scientist, Division of Surgery,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr S. K. Singh
Joint Director (Research)
ICAR- Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Brijesh Kumar
Senior Scientist,
Division of Animal Reproduction
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Manas Kumar Patra
Senior Scientist,
Division of Livestock Production & Management
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Nitin Bajaj
Assistant Professor
Department of Veterinary Gynaecology and Obstetrics
Veterinary College, Mhow, MP, INDIA

Dr Abhishek
Senior Scientist,
Division of Bacteriology and Mycology,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI), Izatanagr-243 122, Bareilly, UP, INDIA

Dr Gaurav Sharma
Senior Scientist,
Centre for Animal Disease Diagnosis & Research (CADRAD),
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Akhilesh Mishra
Senior Scientist, Division of Medicine,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Sameer Shrivastava
Principal Scientist & Incharge, Veterinary Immunology Section
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr K P Singh
Joint Director (CADRAD) & Incharge CDDL
Centre for Animal Diseases Research & Diagnosis (CADRAD)
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Kaushal K Rajak
Principal Scientist, Division of Biological Products,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatanagr-243 122, Bareilly, UP, INDIA

Dr Vikramaditya Upamanyu
Senior Scientist, Division of Biological Standardization,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatanagr-243 122, Bareilly, UP, INDIA

Dr Sonalika Mahajan
Senior Scientist, Division of Biological Standardization
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Sonal
Senior Scientist, Division of Veterinary Biotechnology,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Himani Dhanze
Scientist (Senior Scale), Division of Veterinary Public Health,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Mukesh Bhatt
Scientist (Senior Scale)
Centre for Animal Diseases Research & Diagnosis (CADRAD)
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, Bareilly, UP, INDIA

Dr Samiran Bandyopadhyay
Principal Scientist, Eastern Regional Station,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI), Kolkata Centre
Belgachia, Kolkata, West Bengal, INDIA

Dr P S Banerjee
Principal Scientist, Eastern Regional Station,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI), Kolkata Centre
Belgachia, Kolkata, West Bengal, INDIA

Dr Muthu Sankar
Principal Scientist, Division of Parasitology,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI), Izatnagar-243 122, INDIA

Dr Rajat Garg
Principal Scientist, Division of Parasitology,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, INDIA

Dr Hira Ram
Principal Scientist, Division of Parasitology,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, INDIA

Dr Siju Susan Jacob
Senior Scientist, Parasitology Laboratory,
ICAR-National Institute of Veterinary Epidemiology and Disease Informatics (ICAR-NIVEDI),
Ramagondanahalli, Post Box No.: 6450, Yelahanka,
Bengaluru-560064, INDIA

Dr Rajender Kumar
Principal Scientist
ICAR-National Research Centre on Equines (ICAR-NRCE)
Sirsa Road, Hisar-125001, Haryana, INDIA

Dr D Suchitra Sena
Principal Scientist, Avian Health Section,
ICAR-Directorate of Poultry Research (ICAR-DPR),
Rajendra Nagar, Hyderabad-500030, INDIA

Dr Rakesh Ranjan
Principal Scientist,
ICAR-National Research Centre on Camel (ICAR-NRCC),
Jorbeer, Bikaner-334001, INDIA

Dr F C Tuteja
Senior Scientist,
ICAR-Central Institute for Research on Buffalo (ICAR-CIRB)
Sub Campus Bir Dosanjh, Nabha, Punjab, INDIA

Dr Sanjay Kumar
Principal Scientist,
ICAR-National Research Centre on Equines (ICAR-NRCE)
Sirsa Road, Hisar-125001, Haryana, INDIA

Dr Riyesh Thachamvally
Senior Scientist
National Centre for Veterinary Type Cultures (NCVTC),
ICAR-National Research Centre on Equines (ICAR-NRCE)
Sirsa Road, Hisar-125001, Haryana, INDIA

Dr R K Dedar
Principal Scientist,
Equine Production Campus,
ICAR-National Research Centre on Equines (ICAR-NRCE)
Jorbeer, Bikaner-334001, INDIA

Dr Kapil Kumar Gupta
Asstt Professor
College of Veterinary Science, Rampura Phul
Guru Angad Dev Veterinary and Animal Science University (GADVASU),
Bathinda, Punjab 151103, INDIA

Dr V K Gupta
Director
ICAR-National Research Centre on Pig (ICAR-NRCP),
Rani, Guwahati-781131, Assam, INDIA

Dr Swaraj Rajkhowa
Principal Scientist
ICAR-National Research Centre on Pig (ICAR-NRCP),
Rani, Guwahati-781131, Assam, INDIA

Dr Vishal Rai
Scientist,ICAR-National Research Centre on Pig (ICAR-NRCP),
Rani, Guwahati-781131, Assam, INDIA

Dr Durlav Prasad Bora
Asstt Professor (Sr Scale)
Department of Microbiology,
College of Veterinary Science,
Khanapara, Assam Agriculrural University, Guwahati-781022, Assam, INDIA

Dr Ilakshy Deka
Subject Matter Specialist (Animal Science)
Krishi Vigyan Kendra, Kamrup, Assam Agriculture University,
Kakikuchi Campus, Assam, INDIA

Dr Lukumoni Buragohain
Assistant Professor
Department of Animal Biotechnology,
College of Veterinary Science and Animal Husbandry,
Assam Agriculture University,
Khanapara, Guwahati-781022, Assam, INDIA

Dr Ms Plabita Goswami
Scientist,ICAR-NRC on Mithun (NRCM)
Jharnapani, Medziphema, Nagaland, INDIA

Dr Ms Joken Bam
Scientist,
ICAR-RC for NEH Region, Arunachal Pradesh Centre, Basar-791101,
Leparada District, Arunachal Pradesh, INDIA

Dr Gowthaman Vasudevan
Asstt Professor
Poultry Disease Diagnosis and Surveillance Laboratory
Veterinary College and Research Institute Campus TANUVAS,
Namakkal, Tamil Nadu, INDIA

Dr T R Kannaki
Principal Scientist
ICAR-Directorate of Poultry Research (ICAR-DPR)
Rajendra Nagar, Hyderabad, INDIA

Dr Asok Kumar M
Senior Scientist,Division of Pathology,
ICAR-Indian Veterinary Research Institute (ICAR-IVRI)
Izatnagar-243 122, INDIA

Experts from Department of Animal Husbandry & Dairying, Government of India

Dr Vijay Teotia
Joint Commissioner (LH)
Department of Animal Husbandry & Dairying, Government of India,
Chanderlok Building, 36, Janpath, New Delhi-110 001, INDIA

Dr R G Bambal
Joint Commissioner (AH)
Department of Animal Husbandry & Dairying, Government of India,
Chanderlok Building, 36, Janpath, New Delhi-110 001, INDIA

Dr Aruna Sharma
Deputy Commissioner (LH)
Department of Animal Husbandry & Dairying, Government of India
Chanderlok Building, 36, Janpath, New Delhi-110 001, INDIA

Dr Adhiraj Mishra
Assistant Commissioner (LH)
Department of Animal Husbandry & Dairying, Government of India,
Chanderlok Building, 36, Janpath, New Delhi-110 001, INDIA

Experts from Private Sector

Dr AV Harikumar
DGM (Animal Health)
National Dairy Development Board,
Anand-388001, INDIA

Dr Gopal Kulshrestha
General Manager
Skylark Hatcheries Pvt Ltd.
Saffidon, Jind, Haryana, INDIA

Dr Nitin Bhatia
Vice President- Technical & Vet Regulatory
Intas Pharmaceuticals, Ahmedabad, Gujarat, INDIA
Dr Anup Kalra
Joint Secretary, INFAH, Dabur Ayurvet
Unit #19, 103 First Floor, KM Trade Tower, Plot # H3,
Sector 14, Kaushambi, Ghaziabad-201010, UP, INDIA

Dr Shirish Nigam
President, INFAH
Indian Federation of Animal Health Companies (INFAH),
311, Mastermind IV, I.T. Park,
Royal Palm Estate, Aarey Colony,
Goregaon (E), Mumbai – 400064, Maharashtra, INDIA

Dr A K Rajput
Executive Director
All India Poultry Breeders' Association
A-1-201, Glaxo Apartments, Mayur Vihar Phase-1,
New Delhi-110091, INDIA

Experts from International Agencies

Dr Bhavin Vadera
U.S. Agency for International Development (USAID),
Health Office, American Embassy, Chanakyapuri,
New Delhi, 110021

Dr Dinesh Kumar Gupta
Brook Hospitals for Animals (India)
Joy Tower, C-20/1-A/1, Sector 62, Noida-201301, Uttar Pradesh

Dr Priyanka Kharkwal
JHPIEGO India Country Office
A Wing, 5th, Prius Platinum, D3, P3B,
Saket District Centre, District Centre, Sector 6,
Saket, New Delhi, Delhi 110017

FAOIN ECTAD Team

Mr Rajesh Dubey
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

Dr Raj Kumar Singh
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

Dr Jyoti Misri
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

Dr Mohd Hasib
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

Dr Vikram Singh Vashist
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

Dr Acty George
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

Dr Robin Jacob Paul
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

Ms Bushra Owaisy
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

Ms Ananya Manchanda
Food and Agriculture Organization of the United Nations
55 Lodi Estate, New Delhi - 110003 INDIA

ANNEXURES

ANNEXURE-1: DRUG INTERACTION

Drug interaction means qualitative and/or quantitative altered pharmacological outcome of one drug in the presence of an additional drug, food, herb/spices, or an environmental xenobiotic. It can be either:

Drug-drug interaction

Drug-food interaction

Drug-xenobiotic interaction

Drug-disease interaction

The drug interaction is useless and unsolicited if it causes

elevated toxicity, like

amplified muscle damage risk due to statins and azoles co-administration

Acute life-threatening hypertensive crisis due to tyramine-rich foods and monoamine oxidase inhibitor combination or drugs inducing dehydration in presence of thermal stress.

reduced clinical efficacy

Coadministration of bacteriostatic and bactericidal drugs

warfarin and rifampicin

tetracyclines /quinolones with antacids and milky foods

Some drug interactions can be beneficial (synergism or addition in response) and done intentionally, such as

Trimethoprim and sulfonamides

Penicillin with probenecid

Beta-lactams and Beta-lactamase inhibitors

Anti-inflammatory combinations- Paracetamol with NSAID

Major cause of drug interactions

Combination drug therapy

Multiple prescribers

Non-selective drug use

Altered physiological state

Characteristics of drug interaction

Incidence –more the array of drugs, food and herbs being exposed to the animal body, higher is the possibility of interaction to take place.

Many interactions may be clinically insignificant or merely hypothetical.

Hyposenstivity and hypersensitivity of the animal plays a major role

Higher incidence is seen in newborns and aged animals due to inefficient functioning of the kidneys and liver.

Both doctors and animal rearer may fail to recognize interactions.

The drug interactions are either a

formulation incompatibility (also known as pharmaceutical incompatibility or interaction),

pharmacokinetic interaction, or

pharmacodynamic interaction.

In some drug combinations, both drugs act independent of each other and the interaction visible is the sum of their clinical effects (two CNS depressants or antihypertensives). Both pharmacokinetic and pharmacodynamic interactions are also possible at the same time.

Pharmaceutical interaction

Pharmaceutical incompatibility means ***in vitro*** undesirable/unacceptable non-uniform product as a result of mixing two or more drugs simultaneously which may affect the clinical efficacy or safety of the treatment.

Occur frequently

Occur between active ingredients, excipients, and even drug delivery system being used for administration

can be either physical (color/odour change, effervescence, turbidity, and precipitation) or chemical (pH changes, or complex reactions).

Physical Incompatibility: These changes which occur as a result of physical incompatibility are usually

visible as either one of the following:

Immiscibility

Insolubility

Precipitation

Liquefaction

It can be easily corrected by using any one or more of the following methods:

Emulsification: Oils and water can be made miscible by emulsification.

Add suspending agent: Powder or mucilage of tragacanth is used as a suspending agent to make Phenacetin suspension.

Change the form of ingredients: Ephedrine sulphate is an alkaloidal salt and insoluble in liquid paraffin, but anhydrous ephedrine is soluble and makes a clear solution.

Add, substitute or omit therapeutically inactive compound: Gradual addition of undiluted resin tincture along with vigorous stirring to the diluted suspension or by simply adding a thickener avoids agglomeration.

Change the order or mixing of the prescription: When certain low melting point solids like camphor, menthol, thymol, phenol, chloral hydrate and aspirin are mixed, a liquid or soft mass known as "eutectic mixture" is produced as water of hydration is released So, first Menthol, Camphor, and Ammonium chloride are mixed together to form liquid and then light magnesium carbonate is added to make free flowing powder.

Chemical Incompatibility

Chemical incompatibility often occurs due to oxidation–reduction, acid-base hydrolysis or combination reactions, and may be either-

Intentional: Physician consciously prescribes the incompatibility drugs keeping the therapeutic target in mind. Bismuth sub nitrate when combined with sodium bicarbonate as in Triple carb antacid powder, carbon dioxide gas is liberated in the presence of water.

Unintentional: When the prescriber prescribes the drugs ignorant of their incompatibility. When phenytoin is added to dextrose infusion to control seizures, an insoluble precipitate of phenytoin salt forms and is no longer clinically effective. Amphotericin is perfused to treat aggressive fungal infections in urinary bladder but if it is administered in saline, the drug precipitates and can erode through the bladder wall.

Precipitate Yielding Interactions

The precipitate formed through the chemical incompatibility may be either diffusible or indiffusible.

When diffusible precipitates are formed in very small quantity, dissolve each chemical separately followed by mixing the two portions by slowly adding one portion to the other by rapid stirring as in Alkaloids with alkaline preparations, such as, strong solution of ammonium acetate, aromatic spirit of ammonia, solution of ammonia. Ammonium bicarbonate.

When indiffusible precipitates are formed in large quantity, dissolve one of the reacting substances separately and add powder gum acacia or tragacanth (2g per 100 ml of finished product) to second to produce a smooth mucilage. Mix the two portions by slowly adding one portion to the other with rapid stirring at the time of use.

Pharmacokinetic interaction can be

Absorption interaction

Distribution interaction

Biotransformation interaction

Elimination interaction

Absorption interactions- Drug absorption means translocation of a drug from its site of administration into the bloodstream. An altered biochemical/ physiological state, either due to co-drug, food or any xenobiotic, will alter the rate as well as extent of drug absorption. It is rather varied but a primary determinant of drug bioavailability and depends upon the characteristics of the physical barriers and physicochemical properties of the drug. It occurs mainly due to

Adsorption and/or complexation: Oral tetracyclines and fluoroquinolones chelate divalent metals. Chelation also reduces oral availability of Penicillamine, the universal chelator.

Altered gastric pH: Proton pump blocker and antacid induced increase in gastric pH cause significant reduction in the absorption of water insoluble azole compounds which get ionized only at low pH.

Altered gastrointestinal motility or emptying: Enteral absorption of orally administered drugs depends on gastric emptying parameters, intestinal surface area and motility, and hepatic first pass. most of them are absorbed in the small intestine. Anticholinergics like Propantheline reduce gut motility and improve absorption of benzodiazepines, cimetidine or ranitidine.

Altered digestive enzymes: Some drugs are primarily absorbed through gastric mucosa, while some undergo first pass effect, before reaching the systemic circulation. The susceptibility of a drug to hepatic first-pass metabolism may decide the preferential route of administration like sublingual route for nitro-glycerine. Age is further likely to influence absorption through maturation/geriatric degradation of hepatic enzymes and transporters. in addition to this, lower rectal administration of drugs susceptible to hepatic first pass is of potential therapeutic importance, especially in case of noncooperative animals, to bypass the liver.

Altered gastrointestinal microflora: Non-specific antibiotics usually eliminate a significant fraction of normal intestinal microflora and elevates digoxin toxicity

Malabsorption: Bioavailability of tetracycline gets reduced from capsules but not from syrup in presence of cimetidine due to a pH dependent dissolution of capsule. Astringents, specifically tannins precipitate the mucosal proteins and reduce the absorption of coadministered drugs

Distribution Interaction

Drugs exist in blood in two forms - free form, which is readily available for distribution, metabolism and excretion; and the plasma protein-bound form, which just replenishes the free drug in the blood. Very high protein binding usually restricts drugs to plasma as a reservoir. Within plasma, acidic drugs bind to albumin while basic drugs bind to alpha-1-acid glycoprotein.

Food and drug molecules compete with each other for binding sites of plasma protein binding, depending on their relative concentrations, affinity for plasma proteins and the total quantity of plasma proteins available for binding. Drugs with lesser affinity are displaced from plasma protein binding sites competitively and their free concentration in blood rises. Such interactions become clinically significant if coupled with elimination interaction with very few known clinically relevant examples like digoxin and anti-arrhythmic drugs, leading to digoxin toxicity.

In the case of hypoproteinemia or increased total drug concentrations along with food metabolites, significant competitive interaction occurs, shifting an otherwise safe pharmacodynamic interaction to an enhanced toxic prospective. Such conditions are commonly seen when one of the drugs has very high affinity for plasma/tissue protein or having a small volume of distribution, very low therapeutic index, inconsequential hepatic metabolism, or those that are administered intravenously in bolus dose as in coadministration of warfarin with NSAIDs, Phenylbutazone, salicylates, or sulfonamides.

The plasma protein availability is altered with physiological state, important considerations being pregnancy and lactation.

Only unbound drug undergoes renal glomerular filtration. When the concentration of unbound drug reduces, the bound drug dissociates to maintain equilibrium, thus prolonging the duration of drug action. Most assays do not distinguish free drug from bound drug, leading to incorrect estimation of drug levels in plasma.

In addition to this, p-glucoprotein transporters can also play an important role in absorption and distribution interactions in GIT as well as blood brain barrier and can be inhibited as well as induced by quite a number of drugs.

Biotransformation Interaction

Drugs are metabolized to inactive metabolites by hepatic microsomal enzymes, mainly the cytochrome P450 family of enzymes in two phases, termed Phase I and Phase II.

Phase I reactions prepare lipophilic drug molecules for Phase II enzymatic reactions by to finally forming a more polar and water-soluble metabolite that can be more easily excreted in the urine and/or bile. Phase II reaction is rarely rate-limiting and usually not involved in drug interactions. The Phase I reactions carried are more frequently rate limiting and are the target of clinically significant drug interactions.

Drugs also compete amongst themselves and nutrients for drug metabolizing enzymes. A

drug may induce/inhibit the enzyme that is responsible for its own and/or another drug metabolism. Inhibition of metabolism could result in potentially toxic concentrations of the parent compound (NSAIDs cause a rise in the antibiotic concentrations in general), while in case of prodrug (codeine, clopidogrel), it causes therapeutic failure. Induction of drug metabolizing enzymes could similarly result in a reduced drug levels below that required for efficacy. Sudden buildup of drug or its metabolites can also be toxic, such as liver damage by paracetamol metabolites or the seizures by of meperidine metabolites. Usually 2- 3 weeks of exposure is required for induction, and it can further continue up to 2- 3 weeks post withdrawal

Elimination Interaction

Major mechanism are:

Altered renal blood flow – non-steroidal anti-inflammatory drugs (NSAIDs) diminish renal blood flow.

Altered urine pH – probenecid reduces the renal clearance of anionic drugs such as methotrexate and penicillin.

Competitive active secretion like Probenecid and Penicillin.

Forced dieresis – leads to rapid removal of drug from the system.

Pharmacodynamic Interactions

It can be either direct or indirect and results in additive, synergistic or antagonistic activity. The combination of sulfonamide and trimethoprim is used as synergistic antimicrobial combination. Both verapamil and a β-blocker slow the heart rate by different mechanisms, but their combination can cause heart block and is contraindicated. Other common examples include:

competition at muscarinic site – atropine Ach

inhibition of adrenergic neuronal uptake – antidepressants and guanithidine

influence on electrolytes – thaizide & digitalis

Drug Food Interactions

Occur as a result of pharmacokinetic or pharmacodynamic mechanisms.

Physiological response to feed intake causes gastric acid secretion, which may increase or reduce the bioavailability of certain drugs.

Soluble fibres – as in guar gum, glucomannans and mucilage – stimulate satiety and slow down digestion.

Fat causes delayed gastric emptying and a subsequent delay in intestinal transit of drug, but the total amount of drug absorbed is unchanged.

Solid food stimulates all digestive juices and improves the dissolution of acidic drug and facilitates their absorption.

Feed rich in vitamin K like **Mustard greens and Turnip greens interfere with warfarin.**

Fodder rich in cruciferous plants and nitrite rich plants, especially new plant shoot, can interfere with the metabolism of iodine and synthesis of T3 and T4 thyroid hormones.

Thiazide and loop diuretics prevent magnesium reabsorption in kidney.

Tetracyclines and quinolones, and gastric antisecretory drugs, *i.e.*, PPI and H2 receptor antagonists cause reduced Iron absorption,

Diuretics and fibrates may cause vitamins B deficiency (in particular B12, B6 and B3).

Antacids as H2-antagonists and PPI may decrease vitamin B12 absorption by reducing gastric acidity.

Vitamin B12 deficiency may also occur with acetylsalicylic acid, antipsychotics (*i.e.*, trifluoroperazine), colchicine, estrogens and metformin.

Folate deficiency is seen with antibiotics (penicillins, cephalosporins, tetracyclines), fibrates, acetylsalicylic acid and antirheumatic drugs (*viz.*, Methotrexate), anticonvulsants (*i.e.*, phenytoin, phenobarbital, primidone) and neuroleptics (phenothiazines).

Drugs able to cause vitamin C deficiency include diuretics and acetylsalicylate.

Vitamin D deficiency is more common amongst fat-soluble vitamins (A, D, E and K) and is caused by statins, antacids, anticonvulsants (*i.e.*, phenytoin), cholestyramine and glucocorticoids.

Antineoplastic, immunomodulating, anti-bacterial agents and CNS drugs most frequently cause taste disorders and result in the loss of appetite and reduced food intake, potentially leading to protein–

energy wasting.

Orange, apple, kiwi and papaya juices share similar flavonoids (naringine, hesperidine, floridzin, floretin) and can inhibit the transport polypeptides of organic anions at the usual doses.

Naringin a flavanone glycoside in the pericarp of citrus fruit reduces uptake of aliskiren (antihypertensive) and inhibits CYP3A enzymes and raises concentrations of CYP 3A4 substrates like Felodipine, midazolam, cyclosporine above toxic levels.

Furanocoumarines and active bioflavonoids present in echinacea, banaba, ginkgo, and soybean inhibit organic anion transporting polypeptide and can reduce the oral bioavailability of co-administered OATP substrate, fexofenadine.

Molasses, syrup, and vitamin-mineral supplement with a significant concentration of iron and calcium, decrease oral bioavailability of fluoroquinolone by approximately 50%.

Phenytoin is less absorbed in the presence of diet protein, folic acid or pyridoxine.

Griseofulvin is better absorbed when given after a fatty meal.

Albendazole should be given along with meals for echinococcosis therapy and on an empty stomach might be more appropriate when intraluminal effects are desired, *e.g.*, for intestinal parasites.

Milk reduces bioavailability of tetracyclines and sotalol.

Licorice inhibits 11-beta-hydroxyl steroid dehydrogenase, increases cortisol, causing sodium retention and potassium depletion, hypertension and depression of the renin-angiotensin-aldosterone system.

Milk casein and calcium present in milk decrease the absorption of ciprofloxacin.

Azithromycin bioavailability is reduced by 43% when taken with food.

Tetracycline should be taken one hour before or two hours after meals, and not taken with milk because it binds calcium and iron, forming insoluble chelates, and influencing its bioavailability.

Food may slow down the oral absorption of paracetamol.

NSAIDs cause stomach irritation and thus they should be taken along with food or milk.

Drug Herb Interaction

The herbs and spices are an amalgam of bioactive phytomolecules in traces and the interactions can be easily identified at high doses, specifically with drugs having a low safety margin.

Piperine (black pepper) inhibits both P-glycoprotein and cytochrome CYP3A4, so it can modify the concentration of majority of drugs.

Capsaicin (Chili pepper) is vasodilator, analgesic, antibacterial, antioxidant, anticancer, and antidiabetic. It is contraindicated with aspirin, clopidogrel, diclofenac, naproxen, ibuprofen, oral anticoagulants (warfarin) and heparin.

Both cinnamon and turmeric inhibit cytochrome isoforms and increase the clinical effect of drugs.

Garlic potentiates diabetes medication and may cause heartburn and flatulence.

Garlic interacts with anticoagulants.

Ginger can increase NSAID induced gastric discomfort.

Hypericum modulate cytochrome isoforms as well as P-glycoprotein, and can pharmacokinetically interact with anaesthetics, anticoagulants, statins, beta-blockers, antimicrobials, antidepressants and anxiolytics (benzodiazepines and buspirone).

Hyperphorin present in H. perforatum inhibits serotonin, norepinephrine, dopamine, glutamate, GABA neurotransmitters thereby affecting the effectiveness of large number of CNS drugs.

Senna (Cassia angustifolia and acutifolia) is a natural cathartic and should be avoided with drugs that may induce hypokalemia, like thiazide or loop diuretics.

Glycyrrhizin acid (liquorice Glycyrrhina glabra) reduces hepatic and renal metabolism of corticosteroids by inhibiting 11-beta-hydroxysteroid-dehydrogenase and increases the aldosterone-like activity of cortisol inducing pseudo primary hyperaldosteronism, resulting in hypertension, hypokalemia, metabolic alkalosis and hydro-saline retention.

Drug Disease Interaction

Animal body biochemical and physiological status along with diet affects the drug biotransformation.

Stress plays a vital function in the multi-factorial regulation of a drug's pharmacokinetic as well as pharmacodynamic profile.

Hypoalbuminaemia can occur in liver diseases and malnutrition, an increase in alpha-1 glycoprotein is seen in tissue injury, cancer and infection.

Kidney dysfunction frequently leads to loss of albumin and thus, the major binding site for drugs.

Diarrhoea limits the availability of drug for absorption in general.

Hepatic damage or dysfunction usually increases the lifespan of the drug in the body while it limits the clinical usefulness of prodrugs.

Obesity decreases CYP3A4/5, CYP1A2 and CYP2C9 activity but increases the activity of CYP2E1.

Guidelines to avoid therapeutic incompatibility

Administer drug at least one hour before or 2 hrs after feeding to avoid absorption interactions.

Be familiar with compatibility references and know how to interpret the data.

Consider the effect of diet on gastric and urinary pH.

When reconstituting a drug, ensure thoroughly mixing before administering it or adding it to a solution.

If the compatibility of drugs cannot be determined from the available resources, avoid mixing them.

Aminoglycosides, tetracyclines, chloramphenicol, penicillin, and amphotericin B are frequently incompatible if mixed with other drugs or solutions.

Do not mix salts of hydrochloric acid (*e.g.*, dobutamine HCl, dopamine HCl, and epinephrine HCl) with alkaline solutions. Vitamin B1 (thiamine hydrochloride) is unstable in alkaline solutions and should not be mixed with alkalinizing solutions, carbonates, or citrates.

Consume oral antihistaminics with food if gastric irritation occurs.

Avoid antihistaminics with sedatives and antidepressants.

Take NSAIDs with food/milk to avoid gastric irritation.

Avoid adding two or more additives to a large volume solution whenever possible.

When two or more drugs, particularly antibiotics are prescribed for intermittent infusion, devise a staggered time schedule so that each can be infused individually. As a rule, more than one antibiotic should not be given at a time

When preparing a drug, follow all instructions and note all precautions given by the manufacturer.

Be alert for admixtures involving drugs with a very high or very low pH.

Calcium and magnesium salts frequently cause precipitation when added to other basic salts.

Whenever possible, prepare drugs and admixtures afresh immediately before administration.

Never mix two drugs in a syringe if they are to be administered intravenously.

Administer drugs separately; and as further precaution, the IV tubing should be flushed between drugs.

Common Clinical drug Incompatibilities

Main drug	codrug	Clinical outcome
Sympatholytics	Cholinergics	Additive effect, toxicity
	Sympatholytics	
CNS depressants (sedatives, hypnotics)	CNS depressants (sedatives, hypnotics)	
Warfarin	salicylates	Indirect potentiation of anticoagulant activity
Digoxin	Metoclopramide	Reduced digoxin absorption
	Propantheline	Increased digoxin absorption (due to changes in gut motility)
Digoxin Warfarin	Colestyramine	Reduced absorption due to complexation with colestyramine
Ciprofloxacin	Sucralfate, antacids	Reduced absorption
tetracycline		
azithromycin		
Atorvastatin	fluconazole	CYP3A inhibition, atorvastatin toxicity, muscle injury
QT interval prolonging Antiarrhythmic drugs	azole antifungal drugs	cytochrome P450 3A inhibition
	macrolide antibiotics, *except azithromycin*	
	Cimetidine	
Ketoconazole	Antacids H2-receptor antagonists Proton pump inhibitors	Reduced ketoconazole absorption due to reduced dissolution
Aminoglycosides	Beta-lactams	Covalent bonding, markedly reduced antibiotic efficacy
Tetracyclines	Antacids, mineral supplements with divalent metal ions	Complexation
Fluoroquinolones		
Penicillamine		
Sulfonamides Aspirin Ferrous sulfate	Antacid	Better dissolution and absorption
	sod bicarbonate, calcium carbonate	Reduced dissolution and absorption
Aspirin Diazepam Mexiletine	Prokinetics (Metoclopramide, cisapride)	Rapid GI motility, improved absorption
	anticholinergics	Reduced GI motility and absorption
Digoxin	antibiotics	Better bioavailability
Vitamin A, B12	Neomycin	Malabsorption
Methotrexate		
Tolbutamide	Sulfonamide	Potentiated hypoglycemia
Penicillin	Probenecid	Competitive active renal tubular secretion
Cephalosporins		
Nalidixic acid		

Main drug	codrug	Clinical outcome
Amphetamine	Antacids	Altered urine pH causes toxicity
	Thiazide diuretics	
	acetazolamide	
Lithium bicarbonate	NSAID	Reduced clearance, increased toxicity
Levodopa	Neuroleptics	Blockade of anti-parkinsonian action of levodopa
	Metoclopramide	
Penicillin	Tetracycline	pharmacodynamic antagonism-Penicillin is “cidal” while tetracycline is a “static” drug
	Heparin	increase the risk of bleeding
Heparin	anticoagulants	Potentiate heparin and increase the risk of bleeding
	NSAIDs	
Coumarins	metronidazole	increase the risk of bleeding
	phenylbutazone	
Aminoglycoside	carbenicillin, azlocillin, or mezlocillin	Inactivation of aminoglycoside
	heparin	precipitation of aminoglycoside
Piperacillin-tazobactam	acyclovir	particle formation invisible in normal room light
	amphotericin B	precipitate flocculently
	mitomycin	blue discoloration
cefepime	theophylline	Cefepime degradation up to 25% because of a severe chemical incompatibility
Bronchodilator	Corticosteroid	hypokalemia
NSAID	betablocker	Reduced antihypertensive effect
	ACE blocker	Impaired renal function
	Statins	Reduced NSAID metabolism
	H. antihistaminics	NSAID toxicity/ antagonism
	corticosteroid	NSAID antagonism
	furosemide	Reduced effect of furosemide
	omeprazole	NSAID toxicity
	anticoagulants	increase the risk of bleeding
	benzodiazepines	Delayed absorption of NSAID
	nifedipine	Reduced antihypertensive effect
Betablockers	Vitamin C	Reduced activity
	H. antihistaminics	Beta blocker toxicity, ophthalmoplegia
	corticosteroid	Reduced Beta blocker effect
	furosemide	Beta blocker toxicity,
	Thiazide diuretics	Additive effect, Hyperglycemia, arrhythmia

Main drug	codrug	Clinical outcome
Corticosteroids	furosemide	hypokalemia
	Barbiturates	Reduced effect of corticosteroid
	antiepileptics, phenytoin, carbamazepine	
	Rifampicin	
	omeprazole	
	estrogens	Potentiate corticosteroids by reduced metabolism
	anticoagulants, warfarin	Variable results on blood clotting time

ANNEXURE-2:
COLLECTION, PRESERVATION AND DISPATCH OF SAMPLES TO LABORATORY FOR DISEASE DIAGNOSIS

Necropsy examination of animal helps in diagnosis of diseases and ultimately their control. It is said "**Necropsy is the message of wisdom from dead to living".** It includes systemic examination of dead animal, recording of gross pathological lesions and their correlation with history to make diagnosis of diseases. Sometimes it is difficult to make any conclusion merely on the basis of postmortem. In that situation samples are to be collected for further laboratory analyses such as histopathology, microbiology and toxicology for confirmation of the etiological agents. Samples from all vital organs, tissues showing lesions and lymph nodes are necessarily required. Tissues from putrefied carcasses should also be collected.

Purpose

- Diagnosis of disease and for identification of new disease.
- Confirmation of tentative diagnosis.
- To observe the effect of treatment and give direction for future therapy.

Precautions

- The tissue sample should be fully representative of the organ and the lesions.
- Collect the tissues as early as possible after death of animal.
- Representative tissue/sample should be collected.
- Sharp knife should be used for cutting.
- Collect the tissues directly in fixative.
- Size of tissue should not be more than 1cm for histopathology in 10% formalin.
- Hollow organs should be taken on paper to avoid shrinkage.
- Hard organs like liver, kidneys etc. should be collected along with capsule.
- Wide mouth glass or plastic bottle of varying capacity should always be used.
- 10% formalin solution is the best fixative for routine histopathological diagnosis.
- For virological examination, small pieces of spleen, lymph node and from lesion sites may be sent either in 50% buffered glycerine or on ice.
- For bacteriological isolations heart blood, tissue pieces and swab may be sent on ice.
- Proper labeling of the bottle/specimen samples is very essential.
- A piece of absorbent cotton should be placed on the surface of the sample in formalin to keep the tissue moist in case the bottle is broken during transit.
- The mouth of the specimen bottle or bags may be sealed carefully by paraffin wax or adhesive tape to avoid the leakage during transit and make it watertight.

SAMPLES FOR BACTERIOLOGICAL EXAMINATION

- Collect the tissues under sterile condition.
- Sterilize knife/scalpel/spatula on flame or in boiling water.
- Sterilized the surface by hot spatula.
- Cut with knife and collect sample from inner tissue.
- Body fluids/blood should be collected in sterilized syringe or in Pasteur pipette.
- Specimens should be collected directly in media (liquid media-nutrient broth, peptone water, tetrathionate broth or even in normal saline solution/phosphate buffer saline).
- Seal, pack and transport the collected material to laboratory on ice/under cold chain conditions.

SAMPLES FOR VIROLOGICAL EXAMINATIONS

- Collect tissue under sterilized condition
- Body fluids/blood in sterilized syringe or in Pasteur pipette.
- Tissues in buffered glycerin
- PBS pH 7.2 –50%
- Glycerin – 50%
- Avoid samples in glycerin for sensitive viruses e.g. PPR, canine distemper
- Seal and mark the specimen bottle and transport to laboratory immediately.

SAMPLES FOR TOXICOLOGICAL EXAMIANTION

- Stomach/intestinal contents (about 100 g) in clean glass or plastic bottles over ice.

- Liver, kidneys, muscles (about 500g), heart blood (about 50ml) over ice.
- Urine in clean glass container on ice.
- Leftover feed/fodder in manger (about 1 kg) in airtight container over ice.
- Seal, label, transport to laboratory.
- In Veterolegal cases, all specimens must be collected in presence of police/witness.
- Type of poison suspected along with detailed history, signs, lesions/treatment etc. should be written on letter with specimens.

DISPATCH OF MATERIAL

- Following points must be kept in mind while dispatching the material to laboratory for diagnosis.
- Describe the clinical signs, lesions, tentative diagnosis and treatment given to animal in the letter. Also mention the type of test required as per the tentative diagnosis.
- Write correct address on letter as well as on the parcel preferably with pin code, if the material is sent through post.
- Mark the parcel 'Biological Material', 'Handle with care', 'Glass material', 'Fragile' etc. in order to avoid damage in parcel. Also mark the side to be kept on upper side with arrows.
- Seal the container so that it should not leak in transit.
- Try to send the material as soon as after its collection from animal.
- Keep one copy of covering letter inside the parcel and sent another copy by hand or post in a separate cover.
- Keep adequate padding material like cotton etc. in the parcel, which will save the material from outside pressures/jerks.
- Use dry ice, if available otherwise use ice pack in sealed containers.

ANNEXURE-3:
PACKAGE OF PRACTICES ON DISPOSAL OF ANIMAL CARCASS AND DISINFECTION

During an animal health emergency or animal disease outbreaks, there may be a need for proper animal carcass disposal measures, which is considered as a key component of a successful response and the costliest too. The proper disposal of animal carcasses can help prevent or mitigate the further spread of pathogens. If any materials are potentially contaminated with an animal disease virus or other pathogen, they must undergo treatment or disposal to inactivate or contain the virus or other pathogen (*Miller et al, 2020). Catastrophic natural disasters or large-scale disease outbreaks can result in a large number of dead animals. In these situations, the timely and safe disposal of animal carcasses and related materials will be necessary to prevent the spread of disease. This document gives an overview of potential methods of animal carcass disposal, as well as factors to consider for each disposal method.

General precautions

- Unauthorized and unrestricted access of disposal sites to humans, pets, wild animals, domestic animals, birds shall be prevented.
- Rodent and insect control measures should be considered to prevent disease transmission risk from disposal sites.
- In case of delay disposal, the carcass and related material should be disinfected,
- In case of infectious diseases, the carcass disposal should be divided into high risk disease (animals died of infectious diseases such as Highly Pathogenic Avian influenza, Foot and mouth Disease, Lumpy Skin Disease, Classical Swine Fever, New Castle Disease, Glanders etc.) and low risk disease based on zoonotic importance and transmissibility to other animals. High risk category carcasses and material should be disinfected and preferably incinerated.
- The carcass transport vehicle should be leak-proof, clean and disinfected (before loading and unloading) and carcass should not be sliced before loading.
- The vehicle should not be overloaded and driven slowly.
- Staff should carry approved disinfectant and equipment to handle spills during the journey.
- Small carcasses (if required) may be placed in a plastic trash bag (industrial strength bags with 3mm thick plastic) or water-tight barrel for transport to disposal area. In case of delay disposal, carcasses may be stored in a top-loading chest freezer.
- For mass burial, the site shall be at least two hundred fifty meters away from human habitat.

Common Waste types

Selection of disposal methods depends upon the type of material. Once the waste material is identified, characterized and quantified, proper disposal method should be determined. A list of common waste materials likely to be encountered are listed below

animal carcasses

animal products (milk, meat, eggs etc.)

bedding and manure

feed and feeding stuff

contaminated equipment, supply and materials

veterinary medical products, PPE kits, Syringes and needles

*Before disposal, the following animal disease outbreak response activities are assumed to be in progress or completed:

- disease confirmation – completed/ongoing.
- quarantine – ongoing.
- movement control (animals, delivery trucks, vehicles, and fomites) – ongoing.
- appraisal and compensation – completed/ ongoing.
- biosecurity procedures – ongoing.
- euthanasia – completed/ongoing.
- security measures and crowd control – completed/ongoing.
- surveillance – ongoing.
- monitoring, countermeasure use, and inoculation – ongoing; and
- health and safety procedures – ongoing.

Different methods for disposal of dead animals and related materials are as under

Rendering

Burial

Deep Burial

Superficial Burial (Above-ground burial)

Landfills

Incineration

Burning

Open burning

Pit burning

Composting

Bin Composting

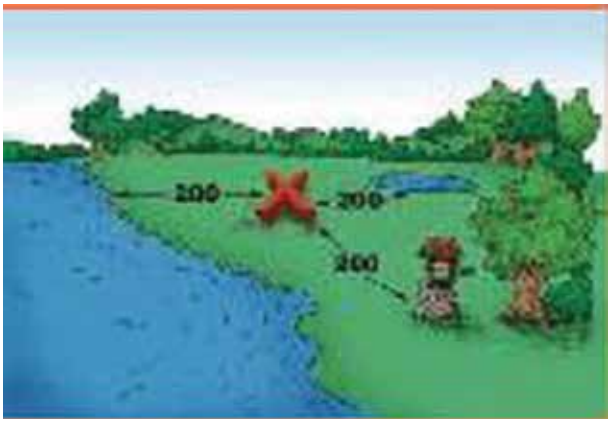
Windrow Composting

Rendering

Rendering is the one of the safest way of converting animal carcasses to pathogen-free byproducts such as feed protein. During the process of rendering animal carcasses are exposed to high temperatures (about 130 C or 265 F) under high pressure which results in destruction of most pathogens. BSE (Bovine Spongiform Encephalopathy) had its impact on the rendering industry, and the rendering process also requires huge capital investment. Burial - Burial sites and process.

The site should not be near a drinking water catchment area and near to the coast and should be away from towns, dwellings, roads and free from underground pipelines, power and telephone lines.

The site should be on soils of low permeability with significant clay content (lining pits with clay soil may be considered). The pits should not be on a slope greater than 6% and digging of 5-meter depth is possible.



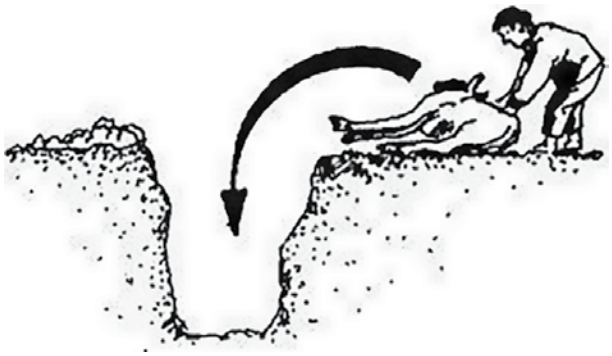
The groundwater table level should be a minimum of 6 meters below the lower level of deep burial pit.

The watercourse should be away from the burial sites such as lakes (1000 ft), rivers (400 ft), tube well (200 ft)

The pit should be **2-meter deep and half filled with waste, then covered with lime within 50 cm of the surface**, before filling the rest of pit with soil. On each occasion when waste is added to the pit, a layer of 10 cm soil shall be added to cover the waste.

Burial pit/trench should be at least **2.3 meter (not more than 3 meter) wide and 3 meters deep (7x9 ft)**. The length should be as per the number of carcasses.

A floor space of 1.3 m^2(15 ft^2): May accommodate mature bovine/equine carcass, 5 mature pigs/sheep, 100 mature chickens/40 mature turkeys. For each additional meter (3 ft) in depth, the number of animals per 1.3 m^2 of floor space may be doubled. The weight of dead animals in the pit should not exceed 2500 kg.



Land requirement

1.5 cubic meters for adult cattle carcass

0.3 cubic meter for pig/sheep carcass

1.0 cubic meter for 200 chickens

The carcasses should be covered with at least **400 mm soil with an unbroken layer of slaked lime -Ca (OH)2 (avoid lime in Anthrax carcass**). *Lime should not be placed directly on carcasses, because in wet conditions it slows and may prevent decomposition.*

The burial pit should be covered **with at least 2 m (6 ft) soil**. Soil should not be compact. During closing the pit surplus soil should be heaped over it as overfill. Lime should be added to pits, to prevent earthworms from bringing contaminated material to the surface after pit closure.

Monitor ground water quality and fence the area with visible sign of restricted entry

Landfills/Subsurface disposal

This is like burial. Carcasses are layered between compacted soil and solid waste materials. Established sites should have minimal potential risks to groundwater, surface water and other environmentally sensitive areas. Landfill design incorporates liners, leachate containment systems and gas collections systems to minimize environmental impacts. May **require 3-5 cubic yards of cover materials per 1000 carcass**. The recommended **height for a pile is 5-7 feet**.

Incineration

Incineration: Incineration is thermal destruction of carcass by using high-temperature (>850^{0}C) combustion (by using fuels like diesel, natural gas, electric energy) to convert carcasses to inert gases and sterile ash as well as deactivate pathogens. Incineration shall be practiced only on-site by agencies and institutes that have adequately trained manpower in operating the rendering plant. The site identified for incineration shall be **at least two hundred fifty meters away** from human habitat.

Burning

Burning of carcasses within a farm on pyres is also a common waste treatment practice that involves combustion of organic substances contained in waste material. **It is not suitable for large volume of material.**

Burning sites and process

Burning space: 8x3 ft. for each mature cattle/horse, 5 mature pigs/sheep, 100 mature chickens and 40 mature turkeys. Also at least 1 meter fire bed length

may be assumed for 1 adult cattle carcass/5 swine/ sheep carcass/200 chickens.

In pyre burning: Place carcasses on top of solid fuel with sufficient airflow, on their backs lower and alternating head to tail. (Approximately one cord of wood (128 cubic feet or 3.4 meter^3) is required per 500 kg of carcass)

Burn pit: The pit should be 0.5 m deep and extended 0.75 m beyond each end of pyre. The pit should be 25 cm wider than the pyre on each side. The bottom of the pit should be covered with accelerant (diesel, kerosene etc. in less quantity to avoid contamination), soaked wood, hay, straw etc. Solid

fuels should be used to maintain combustion. Pieces of heavy timber are placed across the pit to support the pyre.

Anthrax carcass can also be disposed of by burning (if incinerator is not available). All vessels and instruments should be disinfected with **3% solution of sodium carbonate**.

Composting

Composting is a natural biological process that transforms organic material in a predominantly aerobic environment into useful and biological end product. It destroys nearly all pathogenic virus, bacteria, fungi, protozoa and helminth except endospore forming bacteria (B. anthracis) and prions (including BSE).

Bin composting

Windrow Composting

Composting process

Composting should be at least 100 meter away from water sources and residence and 300 meter away from roads.

It involves layering/mixing carcass with co-compost material (sawdust, silage etc.) with at least 60 cm covering of composting material.

Material should be removed from the compost pile after the carcass/related material is completely composted with minimum odors.

Compost piles kill most pathogens in 10-14 days in case of small carcasses, longer in large carcasses.

Assume land area as 17 square meter for cattle carcass, 3.5 square meter for pig/sheep carcass and 8.7 square meter for 100 chickens. The site should be 120 cm above seasonal high-water level and at least 1 meter above bed rock. The site should not be located on flood plains.

On the base of litter, the carcass and related material along with bulking agent are added in layers so that the carbon-to-nitrogen ratio is in the range of 15:1 to 35:1 (optimal 23:1).

Necessary measures should be taken to minimize odour, flies, rodents, bird menace and fire hazard.

Leachate should be re-circulated in the compost plant for moisture maintenance.

Turning piles may increase the rate of decomposition. First stage of composting normally completed within about 3 weeks for poultry, 12 weeks for large animals. Second stage composting takes additional 3 weeks for poultry and up to about 8 months for large animals

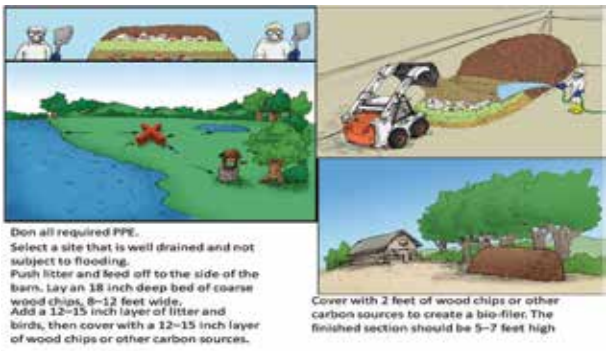
The volume of dead animal(s) in the compost pile must not exceed 25% of the total volume of the compost pile.

Break the eggs prior to composting.

Finished product can be recycled, stored or added to the land as a soil amendment subject to the fulfillment of standards prescribed by Fertilized Control Orders.

Clean and disinfect all the equipment and area.

The operation should be under expert care for proper composting.



Commonly used disposal methods and disinfectants for animal diseases

Name of the disease	Preferred Disposal Method(s)	Chemical inactivating agents for pathogens and Preferred disinfectants for farm structures, equipment, animal houses, etc.
Anthrax	Burial or Burning Burn. If incineration or cremation is not possible, burying the carcass deep (at least 6 feet) is acceptable. Carcass should be decontaminated Ensure sealing of all body openings (anus, mouth, nose etc.) of carcass with absorbent material to prevent leakage of exudates. Ensure that the head of carcass is covered with heavy duty plastic bag. There should be 1 m clay at the base of the pit and also carcass should be covered with minimum 1 m clay	10% formaldehyde, 4% glutaraldehyde, 3% hydrogen peroxide, and 1% peracetic acid. Note: Hydrogen peroxide and peracetic acid will not work in the presence of blood. Soil from areas of anthrax contamination should be removed for incineration or soaked with 5% formaldehyde. Contaminated materials should be incinerated, and non-disposable items should be soaked with 4% formaldehyde or 2% glutaraldehyde. *Avoid using lime and other calcium products on carcass or contaminated ground.*
Avian influenza / Newcastle Disease	Burial or Burning Approximately 5 quintals of wood would be required to burn 100 kg of dead birds. For burial, cover with calcium hydroxide followed by at least 40 cm layer of soil. More layers of lime and soil can be applied to level the pit. A pit of 2x2x2 meters will accommodate around 1800 birds (fowls) and about 450 turkeys. Prior to the commencement of operations, briefing must be given to all involved on the importance of the kit, its use and disposal, *etc.* PPE must be used by RRTs and all persons having direct and active exposure to infected poultry. Operations should not be started without the use of PPE and filter (N-95).	5-6% sodium hypochlorite, 5% calcium hypochlorite, 2-4% glutaraldehyde solution, 250-500 ppm Dioctyl dimethyl ammonium chloride, 4% formalin. Disinfect the walls, floors and ceilings of the sheds in the premises to remove organic material with either or a combination of the following: 3% calcium-hydroxide solution Sprinkling of bleaching powder and lime on the floors of the sheds Whitewashing of concrete areas with lime Fumigation of closed chambers and sheds with Potassium-permanganate ($KMnO_4$) and formalin Treating all the equipment with 2% sodium-hypochlorite solution for 48hrs Cages and other large metal structures may be decontaminated by heat treatment (flame gun) Feathers spread around the farm or attached to metal net, if any, should be burnt with the flame gun All units and items which are physically or functionally connected to the establishment (*e.g.*, hatchery, egg storerooms, packaging rooms, egg trolleys and egg product plants etc.) must also be properly disinfected. Vehicles used for transporting live birds, eggs and feed must also be disinfected. Water-reservoirs must also be emptied, washed and disinfected ➢ Feed tanks (silos) need to be emptied, washed with a hot water-pressure pump and subsequently fumigated After washing and disinfecting, all units must be fumigated twice with at least two weeks between the fumigations Use 2% solution of NaOH should be used at the entrance on foot mats to clean the shoes gumboots and other items
FMD / Swine vesicular disease	Burial or burning	Virkon® (2%), 2-4% Glutaraldehyde, Citric acid, Sodium carbonate, 0.5% sodium hypochlorite solution (5000 ppm available chlorine)

Name of the disease	Preferred Disposal Method(s)	Chemical inactivating agents for pathogens and Preferred disinfectants for farm structures, equipment, animal houses, etc.
Lumpy Skin Disease	Burial or burning	Ether (20%), Chloroform (20%), formalin (1%), phenol 2% in 15min, Sodium hypochlorite 2-3%, Iodine compounds (1:33) dilution, Virkon® (2%) and quaternary ammonium compounds (0.5%) can be used for disinfection of affected premises, vehicles plying through the affected animal holdings should be carried out with appropriate chemicals/disinfectants.
African Swine Fever	Burial or Burning or Rendering Carcasses shall not be allowed to move out of the area and shall be disposed of in the Infected premises itself. In case of exceptions where the carcass disposal is not possible, the transport of carcasses should be undertaken by agencies under the control of District Veterinary/ Administrative authority following strict biosecurity protocols and using leak proof vehicles. Carcasses shall be destroyed under official veterinary supervision ONLY.	Appropriate disinfectants for ASF include 2% sodium hydroxide, hypochlorite (0.5% available chlorine for 30 minutes), detergents and phenol substitutes, sodium or calcium hypochlorite (2-3% available chlorine), Ortho-phenyl phenol 3% for 30 minutes, formalin 0.3 % for 30 minutes, iodine compounds and Virkon® (2%). **Disinfection must be made in three steps –** ***Pre-disinfection:*** This is to prevent the spreading of virus in the room. Clean the surface with a broom, spray the disinfectant - keeping a distance of approximately 50 cm - on the surface and let the agent react for 30 minutes. ***Cleaning:*** This will eliminate more than 90% of the present virus in the area. Hence, after pre- disinfection, brush the surface with water and soap and let it dry. ***Disinfection:*** The remaining virus will be destroyed during the step of disinfection. Spray the disinfectant on the surface and let it react for 2 hours.
Classical Swine fever	Burial or burning	B-propiolactone (0.4%). Cresol (5%), Sodium hydroxide (2%), formalin (1%), Sodium carbonate (4%) - anhydrous or 10% crystalline), Ionic and Non-ionic detergents as well as strong Iodophors (1%) in phosphoric acid, Virkon® (2%).
Bluetongue disease	Burial or burning	0.5-1% sodium hypochlorite, 3% sodium hydroxide.
Rabies	Burial or burning	0.5-1% sodium hypochlorite solution, Phenolic compounds and 70% ethanol, Virkon® (2%)
Hemorrhagic Septicemia	Burial or burning	3% hydrogen peroxide, 5% acetic acid, Virkon® (1%)
***peste des petits ruminants* (PPR)**	Burial or burning	70% ethanol, phenol, and 5% sodium hydroxide, Virkon® (2%)
Glanders	Burial or burning A pit of minimum 8 ft. deep is to be made. The area requirement is about 3 sq. yards per carcass The dead animal is put into the pit with feet upwards. The carcass is covered with quick lime followed by filling of the pit. Personnel in close contact with the diseased animal should follow high standards of personal hygiene and strict antiseptic measures.	*B. mallei* is susceptible to sodium hypochlorite (500 ppm), 70% ethanol, 2% glutaraldehyde, iodine, Benzalkonium chloride (1/2000), mercuric chloride in alcohol and potassium permanganate. It is less susceptible to phenolic disinfectants. This organism can be destroyed by heating to 55°C (131°F) for 10 minutes, or exposure to ultraviolet irradiation. In the environment, *B. mallei* is susceptible to drying and sunlight.

Name of the disease	Preferred Disposal Method(s)	Chemical inactivating agents for pathogens and Preferred disinfectants for farm structures, equipment, animal houses, etc.
Other common bacterial and viral diseases	Burial or burning	Quaternary Ammonium compounds: 5% Sodium hypochlorite, 5% calcium hypochlorite, 5% acetic acid, 5% Sodium hydroxide, 4% Sodium carbonate, 2-4% Glutaraldehyde, 1% Formalin, and Formaldehyde gas. Some of the commercially available disinfectants such as Virkon® (1%), AlkaSept™ Active, PowerCull™ Extra, CombiSept, Bactrex Plus, Germitol, Germisol, Potassium permanganate (1- 2 grams/liter of water) and Lysol (500 ml of Lysol in 9.5 liter of water) can also be used to sanitize the premises depending on type of disease organisms and related factors.

*Miller, L.P., Miknis, R.A. and Flory, G.A. (2020). Carcass management guidelines – Effective disposal of animal carcasses and contaminated materials on small to medium-sized farms. FAO Animal production and health Guidelines no. 23. Rome, FAO [https://doi.org/10.4060/cb2464en].

ANNEXURE-4 :
CARCASS DISPOSAL METHODS IN SPECIFIC DISEASES

Disposal of Anthrax Carcasses and Contaminated Materials

Disposal by incineration is preferred; however, deep burial is also an acceptable method. There may be some jurisdictional restrictions on incineration or deep burial.

Evaluate each animal before disposal and ensure that all body openings are plugged with an absorbent material (e.g. non-perforated paper towel, cloths, etc.) before a carcass is moved.

Note: It is important to record the land location (e.g. global positioning system [GPS] coordinates) for all burn and burial sites.

Note: Research has shown that using lime or other calcium products on carcasses or contaminated ground is contraindicated. Calcium has been shown to protect, rather than destroy, anthrax spores. AVOID USING LIME or other calcium products on carcasses or contaminated ground.

A. Prevent Escape of Bloody Exudates from the Carcass

Note: AVOID performing an autopsy when anthrax is being considered:

Ensure that **all** body openings (e.g. anus, mouth, nose, etc.) are plugged with an absorbent material (e.g. non-perforated paper towel, cloths, etc.) to prevent leakage of exudates.

Ensure that the entire head is covered with a heavy duty plastic bag that is secured at the neck, behind the ears, and across the poll with duct tape, or tied with rope or twine.

Move the carcass on a conveyance that can be destroyed with the carcass or easily cleaned and disinfected (e.g. wood pallet, etc.).

AVOID USING LIME or other calcium products on carcasses or contaminated ground.

B. Protect Carcasses until their Disposal

To prevent scavenging and spreading of spores by insects, birds, or mammals, once all body openings are plugged and the head securely covered, cover the carcass with a tarp, heavy plastic, or other appropriate material. Weigh down the edges of the covering to prevent removal by wind or predators.

The natural decomposition of a carcass destroys most of the vegetative anthrax organisms within 48 to 72 hours in warm weather conditions. These carcasses pose a smaller risk of environmental anthrax contamination during subsequent handling for disposal. The carcass, however, may be friable, and thus may easily pull apart, posing other difficulties in handling.

C. Incineration/Burning C Pyre or Pit

The following guidelines provide information on evaluating disposal efforts and in confirming complete incineration.

The goal is to destroy as many spores as possible, thereby decreasing environmental anthrax contamination. A complete burn should be achieved. The carcass should be completely reduced to ash. An effective burn primarily leaves ash and bits of bone, with minimal fly attraction to the site.

General considerations:

Incinerate or burn by either pyre or pit, which is the preferred method of disposal (particularly when a carcass has been inadvertently opened for post-mortem examination or scavenging).

Be aware that burn permits may be required by municipal or provincial governing authorities.

Know that there may be jurisdictional restrictions on materials used in incineration or burning efforts. (British Columbia, for example, prohibits the burning of railroad ties.)

Be aware that ventilation and adequate airflow within a pyre or pit are essential. Prevailing winds should be considered in providing a good supply of air to the fire.

Understand that, if the fire burns too quickly, a complete burn will not be achieved, necessitating a secondary burn.

Avoid using materials that may be environmentally harmful (e.g. rubber tires).

Ensure that an adequate amount of fuel is available to completely reduce the carcass to ash.

For complete incineration, add any carcass parts or materials that fall off a pyre during the burn back onto the fire.

Burn contaminated materials with the carcass.

1. Pyre System:

a) Wood

Bottom layer: large-sized logs, fence posts, railroad ties, wood pallets spaced 8–10 inches (20–25 cm) apart in a criss-crossed fashion to allow air to enter the fire from below. Align these materials with the direction of the prevailing wind.

Middle layer: smaller pieces of wood or coal placed over top of the bottom layer.

Top layer: prop up the carcass to lay it on its back, placing any soil that is potentially contaminated by the animal/exudates on top of the pyre.

Kerosene or diesel fuel (accelerant) to soak down all the materials (approximately 5 gallons or 23 litres per carcass).

Light the fire from two opposing ends of the pyre.

Note: Approximately one cord of wood (4› x 4› x 8› or 128 cubic feet; 1.2 x 1.2 x 2.4 or 3.4m^3) is required per 1000 lbs (~ 500 kg) of carcass to be incinerated.

b) Straw

Bottom layer: large-sized logs, fence posts, railway ties, wood pallets spaced 8–10 inches (20–25 cm) apart in a criss-crossed manner to allow air to enter the fire from below. Align these materials with the direction of the prevailing wind.

Middle layer: two large round bales per carcass, approximately 1200 lbs (545 kg) each. The bales can be laid on their sides or placed on end. Place a layer of wood pallets on top of the bales to make a platform for the carcass. Pallets wedged between the bales will increase airflow into the pyre.

Top layer: prop up the carcass to lay it on its back, placing any soil that is potentially contaminated by the animal/exudates on top of the pyre.

Note: Flax bales burn at a very high temperature and are well-suited for burning carcasses; however, when used as the sole fuel, they may burn too fast for effective incineration of the carcass. Using flax bales in the centre of the pyre surrounded by other straw bales will burn hot enough for complete carcass incineration. When other types of straw bales are used as the sole fuel source, more accelerant will be required.

Note: An effective burn primarily leaves ash and bits of bone with minimal fly attraction to the site.

Soak the ashes with acceptable disinfectant. **AVOID USING LIME or other calcium products.**

Decontaminate any contaminated ground that is associated with the carcass disposal by using a torch and/or soaking with acceptable disinfectant. **AVOID USING LIME or other calcium products.**

2. Burn Pits/Trenches

The use of a pit facilitates the burial of ashes and prevents fire from spreading. Sloped sides on the pit facilitate airflow to the fire.

Burn pit/trench considerations:

For a mature animal, the pit should be 18–20 inches (0.5 m) deep, and extend approximately 2.5 feet (0.75 m) beyond each end of the pyre that will be constructed.

The pit should be approximately 10 inches (25 cm) wider than the pyre on each side; this allows airflow around the carcass.

The bottom of the pit is covered with accelerant (e.g. diesel fuel, kerosene, etc.), soaked straw, or wood, etc., placed in such a way that facilitates airflow.

Pieces of heavy timber (or other beams) are placed across the pit to support the pyre.

Note: It will be necessary to decontaminate the ground where the carcass lay, as well as the equipment, tools, etc. used in handling the carcass and any contaminated materials. Decontamination is carried out by burning the area, using a propane torch and/or soaking with an acceptable disinfectant. **AVOID USING LIME or other calcium products.**

In all cases, assess the burn site to ensure that there was adequate incineration of the carcass.

D. Burial

If incineration is not feasible or cannot take place immediately, deep burial may be a viable option.

Note: The location of burial sites, using GPS or other mapping methods, should be recorded by those involved, and kept indefinitely.

Burial considerations:

Be aware that burial permits may be required by municipal or provincial governing authorities.

Require heavy excavating equipment (i.e. backhoe) to dig a suitable hole.

Ensure that the pit is 6–8 feet (2 m) deep – the bottom of which should be well above the water table (minimum 3 feet (0.9 m).

Consider the water table level and soil composition – clay soil is preferable, whereas, sand or gravel should be avoided.

Have a minimum of 3.2 feet (1 m) of clay at the base of the pit, and cover the carcass with a minimum of 3.2 feet (1 m) of clay and topsoil to prevent access by scavengers.

Use 10% formalin or 5% lye solution (sodium hydroxide), or another acceptable disinfectant, to decontaminate the carcass and all soil put into the burial pit. **AVOID USING LIME or other calcium products.**

E. Rendering

Moving infected carcasses to rendering facilities poses too great a risk of spreading disease; therefore, rendering anthrax-contaminated carcasses is contraindicated. Rendering is not a recommended method of disposal of carcasses (in whole or part).

F. Delayed Disposal C Special Circumstances

Under specific environmental conditions – for instance, prolonged rain; carcass inaccessibility (e.g. standing water, heavy bush); or logistical problems (e.g. lack of proper equipment, manpower etc.) – the prompt disposal of infected carcasses may be impossible. In these circumstances, to prevent or minimize anthrax environmental contamination, assess the situation to decide on an appropriate course of action within a realistic timeline for disposal. Cover the carcass and the surrounding area with disinfectants, such as 10% formalin or 5% solution of lye (sodium hydroxide), and repeated as needed. Protecting the carcass from scavenging is also indicated. **AVOID USING LIME or other calcium products.**

Other points

Methods of carcass disposal – in general – include burial, landfill, incineration or combustion, rendering, composting (see Box 20-2), and alkaline hydrolysis (chemical digestion). The remains of animals suspected of having scrapie or zoonotic diseases should be burned, incinerated, buried, or chemically digested.

The carcass of animal died of anthrax should not be opened, since exposure to oxygen will allow the bacteria to form spores. Because sporulation of B. anthracis requires oxygen and therefore does not occur inside a closed carcass, regulations in most countries forbid postmortem examination of animals when anthrax is suspected.

Avoid direct contact with the dead animal's body fluids (i.e., blood, urine, feces).

The carcass should be placed in a plastic body bag and sealed as soon as possible.

Wrap it in a sealed plastic bag or a garbage bag depending on its size. Carry out your chosen disposal method, whether that's dropping the bag off at a local waste collection site or burying the animal in your yard. After removing the body, clean and sanitize the surrounding area thoroughly.

The carcass should be placed in a plastic body bag and sealed as soon as possible. Since anthrax is a zoonotic disease, it is recommended to double bag the carcass. Avoid direct contact with the dead animal's body fluids (*i.e.*, blood, urine, feces).

Disposal is specified to be by delivery to a rendering plant, burial, composting or incineration. Leaving mortalities outside for scavengers to feed on is not an acceptable method of dead animal disposal.

Composting is a relatively safe and simple method of carcass disposal that uses naturally occurring microbes (bacteria and fungi) to decompose carcasses. The process generates elevated temperatures which destroys disease-causing organisms.

The carcasses should be covered with at least 400 mm soil with unbroken layer of slaked lime -Ca (OH)2 (avoid lime in Anthrax carcass). Lime should not be placed directly on carcasses, because in wet conditions it slows and may prevent decomposition. Burial pit should be covered with at least 2 m (6 ft) soil.

Package of Practices for Carcass Disposal of Brucella-Affected Ruminants

General guidelines:

Site Selection: Choose a site far from water sources,

human dwellings, and livestock. Ensure the site is accessible and allows for secure containment of the carcass. Consider soil type and drainage; sandy soils are preferable for burial to avoid water contamination.

Personal Protective Equipment (PPE): Wear gloves, masks, coveralls, boots, and eye protection. Disinfect PPE after use and dispose of single-use items properly.

Notification and Documentation: Notify local veterinary authorities immediately upon confirmation of Brucella infection. Maintain records of carcass disposal, including the number of animals, date, and location.

Disposal of Large Ruminants (Cattle, Buffalo)

Carcass Handling: Minimize movement of the carcass to prevent contamination. Use a front-end loader or similar equipment to transport the carcass to the disposal site.

Disposal Methods: Burial: Dig a pit at least 2.5 meters deep. Place the carcass in the pit and cover it with lime to accelerate decomposition and reduce odor. Fill the pit with soil, compacting it to prevent scavenger access.

Incineration: Use a high-temperature incinerator designed for animal carcasses. Ensure complete combustion to reduce the risk of environmental contamination. Dispose of ash in an approved landfill.

Disinfection: Disinfect equipment, tools, and surfaces that have come into contact with the carcass. (Sodium hypochlorite, Betadine, and Dettol)

Disposal of Small Ruminants (Sheep, Goats)

Carcass Handling: Use appropriate tools (e.g., shovels, stretchers) to minimize direct contact with the carcass. Avoid dragging carcasses across the ground.

Disposal Methods: Burial: Dig a pit at least 2 meters deep. Place the carcass in the pit with lime, and cover with soil. Compact the soil to prevent access by scavengers. **Incineration:** Use a small-scale incinerator appropriate for small ruminants. Ensure complete combustion and proper disposal of ash.

Disinfection: Follow similar disinfection protocols as outlined for large ruminants. Ensure all contaminated surfaces and equipment are thoroughly disinfected.

ANNEXURE-5 :

PHSIOLOGICAL, HAEMATOBICHEMICAL VALUES IN DIFFERENT SPECIES

Table 1: Physiological vitals of clinical significance of different species of animals

Parameters (Range Values)	Cattle	Buffalo	Sheep	Goat	Pig	Horse	Camel	Yak	Mithun	Dog	Poultry
Rectal /Body Temperature(^{0}F)	100-103	99-102	102-105	102-105	100-104	99-102	99-102	100-101	100-101	100-102	105-107
Pulse Rate (Per minute)	40-70	15-30	60-90	60-90	60-100	28-44	60-90	69-100	65-75	60-140	20-30
Respiration Rate (Per minute)	18-28	38-35	12-20	12-20	8-18	10-24	10-30	34-65	20-22	18-34	250-300

Table 2: Haematological values of different species of animals

Parameters (Range Values)	Conventional Units	Cattle	Buffalo	Sheep	Goat	Pig	Horse	Camel	Yak	Mithun	Dog	Poultry
RBC	$\times10^6$/mcL	5-10	8-11	9-15	8-18	5-8	6-10	8-14	4-6	6-7	5-8	2-4
Hb	g/dL	8-15	14-18	9-15	8-12	10-16	10-16	11-15	9-13	10-11	12-19	7-11
PCV	%	24-46	43-45	27-45	22-38	32-50	27-43	20-32	24-38	31-45	35-57	27-42
MCV	fL	40-60	61-65	28-40	16-25	50-68	37-49	7-31	51-63	55-64	66-77	82-89
MCH	pg	11-17	20-22	8-12	5-8	17-21	14-18	10-14	16-23	15-18	21-26	27-29
MCHC	g/dL	30-36	35-37	31-34	30-36	30-34	35-39	43-49	33-39	24-33	32-36	32-33
Platelets	$\times10^3$/mcL	100-800	264-439	800-1,100	300-600	200-500	117-256	129-454	136-364	-	211-621	1-4
WBCs	$\times10^3$/mcL	4-12	12-15	4-8	4-13	11-22	6-12	7- 18	3-9	9-11	5-14	20-33
Neutrophils	%	15-33	34	10-50	30-48	28-47	52-70	50-58	26-65	24	58-85	29-37
Lymphocytes	%	45-75	54	40-55	50-70	39-62	21-42	30-42	24-58	63	8-21	49-58
Monocytes	%	0-8	5	0-6	0-4	2-10	0-6	4-6	0-3	7	2-10	8-10
Eosinophils	%	0-20	7	0-10	1-8	0.5-11	0-7	2-6	5-24	5	0-9	1-7
Basophils	%	0-2	-	0-3	0-1	0-2	0-2	0-1	0-3	1	0-1	0.2-0.5
Plasma Proteins	g/dL	7–7	5-9	6-8	6-7	8-9	6-8	5-6	7- 8	7-9	5-7	3-5

Abbreviations Used: RBC-Red Blood Cells, Hb-Haemoglobin, PCV-Packed Cell Volume, MCV-Mean Corpuscular Volume, MCH-Mean Corpuscular Haemoglobin, MCHC-Mean Corpuscular Haemoglobin Concentration, WBC-White Blood Cells, $\times10^6$/mcL million cells per microliter, g/dL grams per decilitre, Fl- femtoliters, pg-picograms %-percent and $\times10^3$/mcL-thousands cells per microliter.

NB: Data on various species compiled and adapted in part from multiple sources. (Including https://www.msdvetmanual. com/multimedia/ table/ haematology-complete-blood-count-reference-ranges, international and Indian publication). Reference ranges vary between laboratories. Values provided by the reference laboratory should be always used.

Table 3: Serum biochemistry values of different species of animals

Parameters (Range Values)	Convent-ional Units	Cattle	Buffalo	Sheep	Goat	Pig	Horse	Camel	Yak	Mithun	Dog	Poultry
Total Protein	g/dL	7-7	5-9	6-8	6-7	8-9	6-8	5-6	7- 8	7-9	5-7	3-5
Albumin	g/dL	2-4	2-4	2-3	3-4	2-4	3-4	4-4	3-4	4-5	2-3	1-3
Globulin	g/dL	3-3	3-6	3-6	3-4	5-6	3-4	2-3	3-5	3-4	3-4	1-4
ALT	U/L	11-40	7-48	26-34	6-19	31-58	-	22-44	0.6-82	36-89	10-109	21-29
AST	U/L	51-169	24-93	60-280	167-513	32-84	160-412	36	5-100	77-122	13-15	115-151
Alk Phos	U/L	41-172	44-311	68-387	93-387	118-395	-	40-595	60-264	118-225	1-114	10-17
GGT	U/L	6-17	13-55	20-52	20-56	10-60	6-17	4-17	-	-	-	103-155
Bilirubin	mg/dL	0-2	0.1-0.8	0.1-0.5	0-0.1	0-1	0-3	0- 0.03	0.31-0.43	-	0-0.3	1-1
Cholesterol	mg/dL	-	43-113	52-76	80-130	36-54	-	36-28	90-120	48	135-278	129- 297
Creatinine	mg/dL	0.5-2	1-2	1-3	1-2	1-2	0.4-2	1-1	2-2	-	0.5-2	0.5-2
Blood Urea Nitrogen	mg/dL	10-25	45-47	8-20	10-20	10-30	11-27	20-25	16-22	23-39	8-28	4-6
Sodium	mEq/L	136-144	130-160	139-152	142-155	135-150	128-142	148-155	117-123	136	142-152	134-174
Potassium	mEq/L	4-5	4-7	4-5	3-7	4-7	3-5	5-7	8-9	10	4-5	2-3
Chloride	mEq/L	99-107	73-117	95-103	99-110	94-106	98-109	125	97	-	110-124	17-19
Calcium	mg/dL	8-11	8-14	11-13	9-12	7-12	10-13	2-3	10-11	10	9-12	5-7
Phosphorus	mg/dL	6-8	6-10	5-7	4-10	5-10	1-5	1-3	-	8	3-5	9-28
Magnesium	mg/dL	1-3	2-4	2-3	3-4	3-4	1-2	1-2	2-2	2	2-2	0.71-1
Glucose	mg/dL	40-100	22-97	50-80	50-75	85-150	62-134	110-106	**61-66**	49-70	76-119	197- 299

Abbreviations Used: ALT-Alanine Aminotransferase, Alk Phos-Alkaline Phosphatase, AST-Aspartate Aminotransferase, GGT-Gamma Glutamyl transferase, g/dL- grams per deciliter, U/L- units per litre, mg/dL-milligrams per deciliter, mEq/L- milliequivalents per litre and – Information not available.

NB: Data on various species compiled and adapted in part from multiple sources. (Including https://www.msdvetmanual.com/ multimedia/ table/ serum-biochemical-analysis-reference-ranges, international and Indian publication). Reference ranges vary between laboratories. Values provided by the Reference Laboratory should be always used.

ANNEXURE-6 :
REPRODUCTIVE Hormones/ Drugs

Name of Hormone/ Drug	Indications	Administration (Suggested Dose, Routes & Duration)
Hormones for the management of reproductive disorders should be used judiciously depending upon the clinical conditions and clinician judgment. Avoid overuse / misuse of the hormones		
Abbreviations - LR: Large Ruminant; SR: Small Ruminant, PO: Oral; IV: Intravenous; IM: Intramuscular; SC: Subcutaneous; TD: Total dose, * Avoid frequent use		
GnRH analogue (0.0042mg/ml)	Anovulation/ Delayed Ovulation	LR: TD 2.5 ml, IM/ IV at estrus
	True anestrus	LR: TD 5 ml, IM/ IV
		SR: TD 1 ml, IM/ IV
	Follicular cyst	LR: TD 5.0 ml, IM/ IV
	Enhancing Conception Rate	LR: TD 2.5 ml, IM at estrus/ luteal phases (early/mid/ late)
PMSG* (Pregnant Mare Serum Gonado tropin, FSH like activity)	True anestrus	LR: TD 1500 -3000 IU, IM/ IV
		SR: TD 1000 IU, IM/ IV
hCG* (Human Chorionic Gonadotropin, LH like activity)	Repeat Breeding	LR: TD 1500-3000 IU, IM at breeding
		SR: TD 200-500 IU, IM, IM at breeding
	Cystic Ovarian Disease & Delayed Ovulation	LR: TD 1500-3000 IU, IM
	Early abortion	LR:1500-3000 IU, IM, Every week for 4 weeks
	Enhancing Conception Rate	LR: TD 1000-2000IU, IM at luteal phases (early/mid/ late)
Hydroxy progesterone caproate (250mg/ml)	Cystic Ovarian Disease	LR: TD 500 mg, IM
	Habitual abortion (Late)	LR: TD 500 mg/ IM, for 3 days and then every week (As per the clinician observation)
	Habitual abortion (Early)	LR: TD 500 mg/ IM, after 1.5 months ofpregnancy
	Post-Partum Anestrus	LR: TD 500 mg/ IM (As per the clinician observation)
Prostaglandin $F_{2\alpha}$ (Natural 5mg/ml or Synthetic 250 mcg/ml) (Contraindications in pregnancy)	Sub-estrus, Luteal cyst, Mummified fetus, Chronic endometritis, Pyometra, Induction ofparturition	LR: TD Natural - 25mg/ Synthetic - 500mcg
		SR: LR: TD Natural – 10 mg/ Synthetic - 250mcg
OXYTOCIN (5 units/ ml)	Uterine inertia, Dystocia, RFM, Uterine prolapse	LR:50-70 IU; SR: 20-30IU; IM/ IV

ANNEXURE-7 :
Drugs usage in ruminants

Drugs	Suggested Dose (mg/kg B.W.); Route; Frequency/Day
Abbreviations: sid (24 h) - Every day; bid (12 h) - Twice a day; qid (6 h) - Four times a day; qod - Every other day; tid (8 h) - Three times a day; Inj - Injection; IM – Intramuscular; IV - Intravenous; PO - Orally; SC - Subcutaneous; **SD** - Single Dose; **TD** - Total Dose; **LA** - Large Animal; **SA**-Small Animal	
Antimicrobial drugs: Use broad-spectrum antibiotics for 3-5 days. However, clinician should choose appropriate antimicrobial drug, its dosage, its usage on the basis of system(s) affected, and clinical condition of the animals. ***Note: Conduct antibiotic sensitivity test (AST) before recommending the antibiotic.***	
Amikacin sulphate	LA/SA: 7; IM/IV; tid
Amoxicillin sodium	LA/SA: 22; IM/SC; bid
Amoxicillin trihydrate	LA/SA: 11-22; IM/SC; sid/bid
Amoxicillin sodium + Sulbactam sodium	LA/SA: 7-10; IM/IV; bid
Ampicillin sodium	LA/SA: 22; SC/ IV/IM; bid
Ampicillin trihydrate	LA/SA: 4-22; IM/SC; sid/bid
Cefotaxime	LA/SA: 10-11; IM/SC; bid
Ceftiofur sodium	LA/SA: 1.1-2.2; IM/IV; sid
Ceftriaxone	LA/SA: 5-10; IM/IV; bid
Enrofloxacin	LA/SA: 2.5-5; IM/SC; sid
Florfenicol	LA/SA: 20; IM; Repeat after 48h/40; IM; SD
Gentamicin sulphate	LA/SA: 2.2-6.6; IM/IV; bid/tid
Marbofloxacin	LA/SA: 2; IM/IV/SC; sid
Oxytetracycline	LA/SA: 5-20; IV/IM; sid/bid
Penicillin G, benzathine (Long Acting)	LA/SA: 44000 – 66000 IU; IM/SC; 48-72 h
Penicillin G, procaine	LA/SA: 10,000-60,000 IU; IM/SC; sid/bid
Streptomycin	LA/SA:11; IM/SC; bid
Sulphadoxine/Trimethoprim	LA/SA: 15; IM/SC/IV; sid/bid
Sulphadimethoxine	LA/SA: 55-110, PO; sid
Sulphonamide/Trimethoprim	LA/SA: 15-30; IM/IV/PO; sid/bid
Tylosin	LA/SA:18; IM; sid
Anthelmintic drugs: repeated/ extended based on parasitic/ faecal eggs count load.	
Albendazole	LA/SA: 7.5-10; PO; SD
Closantel	Sheep: 10; SC/PO; SD
Fenbendazole	LA/SA: 5-10; PO; SD
Ivermectin	LA/SA: 0.2; SC; SD
Levamisole	LA/SA: 5.5-11; PO; SD LA/SA: 3.3-8.0; SC; SD
Morantel tartrate	LA/SA: 8-10; PO; SD
Moxidectin	LA: 0.2; PO/SC; SD SA: 0.2-0.5; PO/SC; SD
Pyrantel pamoate	LA/SA: 25; PO; SD
Triclabendazole	LA/SA: 10-12; PO; SD
Antifungal drugs	
Griseofulvin	LA/SA: 10-20; PO; sid

Drugs	Suggested Dose (mg/kg B.W.); Route; Frequency/Day
Whitfield's ointment (Benzoic acid 6% and Salicylic Acid 3% w/w) mixed with excipient (Emulsifying Wax 21.84 w/v)	LA/SA: Topical; bid
Anti-Inflammatory agents: NSAID'S indicated to control inflammation, fever and pain.	
Carprofen	LA: 1.4; SC/IV; SD
Flunixin meglumine	LA/SA; IM/IV; sid/bid
Meloxicam	LA/SA: 0.5, IV/SC/IM; SD LA/SA: 0.5-1.0; PO; 24-48 h
Ketoprofen	LA: 2-4; IM/IV; sid
Tolfenamic acid	LA: 2.0; IM; sid
Corticosteroids	
Prednisolone	LA/SA: 1-4; IV; SD/sid
Dexamethasone	LA/SA: 0.02-2.0 (Anti-inflammatory)/ 5-20 (ketosis)/TD -20-30 (Induction of parturition), TD; IV/IM; SD/sid
Anti-histaminic Drugs	
Diphenhydramine hydrochloride	LA/SA: 0.5-1.0, IV/IM; tid/qid/bid
Pheniramine Maleate	
Chlorpheniramine Maleate	
Multivitamins: To improve the efficacy of standard therapy. Dose should be as per the availability of preparations and manufacturer instructions.	
Vitamin A	LA: 440 IU; IM; sid
Vitamin D3 (Cholecalciferol)	LA: 10 million IU TD; IM (2-8 days before calving)
Vitamin E (Tocopherol acetate) & Se	LA: 2 ml/45 kg B.W.; IM
Thiamine hydrochloride (vitamin B1)	LA/SA: 5-50; IM/IV; bid
Ascorbic acid (Vitamin C)	LA (Calves): 3 g TD; SC; SD
Haematinics	
Iron dextran	LA/SA: 2.0; IM; SD
Immunomodulators: To improve the efficacy of standard therapy.	
Levamisole	LA/SA: 2.5; SC; SD
Cardiac glycoside: For Congestive Heart Failure	
Digoxin	0.022 loading dose then 0.0034; IV, 4 h interval
Diuretic	
Furosemide	LA/SA: 0.5 -1.0; IV/IM (Adult Cattle); sid/bid
Mannitol	LA/SA:1-3g; IV; SD
Common drugs	
Ammonium Chloride	LA/SA: 50-200; PO; sid/bid
Potassium Iodide	LA/SA: 1.5; IV; sid
Kaolin pectate	LA/SA: 0.25-1mL; PO; qid
Ferrous Sulphate	LA/SA: 10-30; PO; sid
Magnesium Oxide	LA/SA: 1000-2000; PO; SD
Magnesium Hydroxide	LA: TD - 400-500gm; SA: 10-30gm; PO; sid/bid/tid

Drugs	Suggested Dose (mg/kg B.W.); Route; Frequency/Day
Fluid and electrolyte: Crystalloids are balanced and similar to plasma when contain electrolytes (K, Mg, Ca) in addition to Na & Cl. Lactated Ringer's is a balanced solution than the normal saline. Dose should be as per the clinical condition of the animal till hydration/ normalization. However, clinician can assess the degree of dehydration based on symptoms like skin tenting, sunken eye ball, capillary refilling time and body temperature. Mild dehydration (6-8%): Slight eyeball recession, skin tent slightly prolonged (2-4 seconds), mucous membranes moist; Moderate dehydration (8-10%): Eyes obviously sunken, skin tent obviously prolonged (4-8 seconds), mucous membranes tacky; Severe dehydration (10-12%): eyes severely sunken into orbits, skin remains tented indefinitely, mucous membranes dry.	
Maintenance dose	LA/SA: 50-80ml/kg/day; IV; Flow rate: 7ml/kg/hr
Based on degree of Dehydration	
4-6%	LA/SA: 20-25 ml/Kg BW; IV
6-8%	LA/SA: 30-50 ml/Kg BW; IV
8-10%	LA/SA: 50-80 ml/Kg BW; IV
10-12 %	LA/SA: 80-120 ml/Kg BW; IV
Oral electrolyte solutions - (NaCl -7 g/L, KCl -1.25 g/L, $CaCl_2$ - 0.5 g/L in water)	LA: 5-10 litres/ day; PO (Through stomach tube); Repeat as necessary
Isotonic crystalloids - Normal Saline (0.9% NaCl)/ Lactated Ringer's Solution (LRS)/ 5% Dextrose saline solution (DNS)	LA:10-20L; SA: 2-4 L; IV continuous
Colloids - Hydroxyethyl Starch (HES) and Dextrans	5-10 mL/kg (Shock); IV (short-term)

ANNEXURE-8 :
Drug Adverse Reaction Reporting Proforma

Part 1: Information regarding reporting person (Animal Owner/Veterinarian/ Technical)

- Name:
- Designation/ Position/Other:
- Institute/Organization/Other:
- Address:
- Mobile:
- Email:

Part 2: Information related to animal

- Species:
- Breed:
- Age:
- Sex:
- Weight:
- Reproductive Status:
- Overall health status prior to drug administration:

Part 3: Adverse Drug Reaction Information

- Number of animals treated with the drug/ product:
- Number of animals reacted:
- Number of animals died:
- History (pre-existing disease, medications used if any):
- Description of adverse drug reaction events such as symptoms/clinical signs, severity of reaction, etiological factors, treatment:
- Adverse drug reaction event diminished after stopping using the drug/product: Yes/No
- Adverse drug reaction event reappeared after re-introducing the drug/product: Yes/No
- Outcome of the drug adverse effect:

Part 4: Information regarding suspected product/ drug

- Start date of adverse effect:
- End date of adverse effect:
- Brand name of drug/product:
- Active ingredients:
- Name of drug manufacturer:
- Identification number of drug on the label:
- Lot number of drug/product:
- Reason for using the drug /product:
- Dosage form of drug/product:
- Start date of treatment:
- End date of treatment:
- Duration of drug/product use:
- Expiry date of drug/product:
- Drug administered by: Veterinarian/ Technician/ Animal owner
- Animal(s) have been treated with this drug/ product in the past: Yes/ No
- Drug/product was used as per the label: Yes/ No

Note: Reporting of drug adverse event should be confidential

Signature:

Date:

Additional Readings

ETHNO-VETERINARY FORMULATIONS-1 (dairyknowledge.in)

OIE LIST OF ANTIMICROBIALS OF VETERINARY IMPORTANCE (woah.org)

IITKA_BOOK_TRADITIONAL-KNOWLEDGE-IN-AGRICULTURE-ENGLISH_0_0 (ICAR.ORG.IN)

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