MJF COLLEGE OF VETERINARY AND ANIMAL SCIENCE, CHOMU, JAIPUR

DEPARTMENT OF VETERINARY PATHOLOGY

Blue Tongue

- It is a viral disease is caused by *orbi* virus and characterized by catarrhal stomatitis, rhinitis, enteritis, and lameness.
- Bluetongue virus is an arthropod-borne *orbi* virus in the Family *Reoviridae.*

Synonyms:-

- Sore Muzzle
- catarrhal fever of sheep
- Pseudo Foot-and-Mouth Disease
- Muzzle Disease

Etiology

- Blue tongue results from infection by the bluetongue virus, a member of the genus *Orbi* virus and family *Reoviridae*.

- 24 serotypes have been identified worldwide; six serotypes (1, 2, 10, 11, 13, and 17) have been found in domesticated or wild ruminants in the United States.

- Bluetongue viruses are closely related to the viruses in the epizootic hemorrhagic disease (EHD) serogroup.

Species Affected

- Bluetongue virus infects many domesticated and wild ruminants including sheep, goats, cattle, buffalo, deer, antelope and bighorn sheep.
- Clinical disease is seen often in sheep, occasionally in goats, and rarely in cattle.
- The virus is present in some regions without associated clinical disease.

Transmission

- Bluetongue is transmitted by biting insects of the genus Culicoides.
- Infection has been transmitted by insemination from an infected bull, since the virus is present in the semen.

Incubation Period

- In sheep, the incubation period is usually 5 to 10 days.
- Cattle can become viraemic starting at four days post-infection, but rarely develop symptoms.
- Animals are usually infectious to the insect vector for several weeks.

Pathogenesis

- The pathogenesis of bluetongue, epizootic hemorrhagic disease, and Ibaraki disease is fundamentally similar in all species in which disease is seen.
- After the insect bite, Primary viral replication occurs in regional lymph nodes and spleen.
- Viremia about 4-6 days after inoculation results in secondary infection of endothelium in arterioles, capillaries, and venules throughout the body, but especially in lung microvascular endothelium.
- Microscopic lesions, fever, and lymphopenia begin a day or so later, about a week after inoculation.

- BTV in the blood appears to be closely associated with, or in, both leukocytes and erythrocytes.

- Endothelial damage caused by viral infection initiates local *microvascular thrombosis and increased permeability.*

- *This is reflected microscopically* by the presence of swollen endothelium, and fibrin and platelet thrombi in small vessels, with edema and hemorrhage in surrounding tissue.

- These lesions in turn mediate the full spectrum of gross findings. These are fundamentally *ischemic necrosis of many tissues; edema* due to vascular permeability; and *hemorrhage resulting from vascular* damage.

- In severe cases, coagulopathy due to thrombocytopenia and depletion of soluble clotting factors.

Clinical Signs

- The vast majority of infections with bluetongue are clinically inapparent.
- In sheep, the clinical signs may include fever, excessive salivation, depression, dyspnoea and panting.
- Initially, animals have a clear nasal discharge; later, the discharge becomes mucopurulent and dries to a crust around the nostrils.
- The head and ears may also be edematous.

- Erosions and ulcerations are often found in the mouth; these lesions may become extensive and the mucous membranes may become necrotic and slough.

- Oedematous swellings appear in the lips, tongue, ears, face, and inter-mandibular space.
- Oedema and cyanosis of the tongue (bluish) are so striking that they have given the disease its name.
- The coronary bands on the hooves are often hyperemic and the hooves painful- Stiffness and lameness.

Clinical Signs-contd

- Pregnant ewes may abort their fetuses, or give birth to "dummy" lambs.
- The disease may terminate in severe emaciation, prostration (lying down), and muscular weakness (sometimes with torticollis - twisted neck drawing the head to one side), which may last 3 weeks or more.
- This is followed by pulmonary oedema and death from pneumonia.

Sheep, Bilateral nasal discharge, Excessive salivation, erosion of nasal planum

Sheep, mouth. - linear erosion and reddening of the right buccal mucosa.

multiple erosions and crusts on the muzzle and lips.

Post mortem Lesions

- In sheep, the face and ears are often edematous.
- A dry, crusty exudates may be seen on the nostrils. The coronary bands of the hooves are often hyperemic; petechial or ecchymotic hemorrhages may be present and extend down the horn.
- Petechiae, ulcers and erosions are common in the oral cavity, particularly on the tongue and dental pad, and the oral mucous membranes may be necrotic or cyanotic.
- The nasal mucosa and pharynx may be edematous or cyanotic, and the trachea hyperemic and congested. Froth is sometimes seen in the trachea, and fluid may be found in the thoracic cavity.

Post mortem Lesions-contd

- Hyperemia and occasional erosions may be seen in the reticulum and omasum. Petechiae, ecchymoses and necrotic foci may be found in the heart.
- In some cases, hyperemia, hemorrhages and edema are found throughout the internal organs. Hemorrhage at the base of the pulmonary artery is particularly characteristic of this disease.
- In addition, the skeletal muscles may have focal hemorrhages or necrosis, and the intermuscular fascial planes may be expanded by edema fluid.

Post mortem Lesions-contd

- In deer, the most prominent lesions are widespread petechial to ecchymotic hemorrhages.
- Chronic case: deer may have ulcers and necrotic debris in the oral cavity.
- They may also have lesions on the hooves, including severe fissures or sloughing.

Bovine. The muzzle is covered by an adherent crust, and the underlying (eroded) tissue is hyperemic.

Sheep, mouth. Most of the dental pad is eroded; the remaining pale mucosa is necrotic.

Bovine, mammary gland. There is extensive coalescing ulceration of the teat skin.

Sheep, foot. There are multiple petechiae in the hoof wall, and there is marked hyperemia of the coronary band.

Sheep, tongue. The lateral mucosa contains several ulcers that are covered by exudate and surrounded by zones of hyperemia.

Sheep, eye. There are foci of bulbar and palpebral conjunctival hemorrhage.

Sheep, rumen. There are multiple mucosal hemorrhages in the rumen

Sheep, fetuses. The larger of these aborted macerated fetuses exhibits torticollis.

Morbidity and Mortality

- In sheep, the severity of disease varies with the breed of sheep, virus strain and environmental stresses.
- The morbidity rate can be as high as 100% in sheep. The mortality rate is usually 0-30%, but can be up to 70% in highly susceptible sheep.
- Similar morbidity and mortality rates are seen in bighorn sheep.
- Most of infections in cattle and goats are asymptomatic.
- In cattle, up to 5% of the animals may become ill, but deaths are rare. In some animals, lameness and poor condition can persist for some time.

Diagnosis

- Clinical:

  Clinical Signs

  lesions

- Differential Diagnosis

  foot-and-mouth disease, vesicular stomatitis, peste des petits ruminants, plant photosensitization, malignant catarrhal fever, bovine virus diarrhea, infectious bovine rhinotracheitis, contagious ecthyma (contagious pustular dermatitis), sheep pox, foot rot and *Oestrus ovis infestation.*

Diagnosis-contd

- Laboratory Tests

  Isolation via inoculation in Chicken egg embryo

  AGID

  Antigen capture ELISA

  Immunofluorescence

VESICULAR STOMATTIS

➢Vesicular stomatitis is a contagious viral disease of animals caused by vesiculovirus and characterized by the presence of vesicles in oral cavity, necrosis and intercellular oedema. It is also known as ***sore mouth of cattle .***

➢Vesicular stomatitis (VS) affects horses, cattle, and pigs, and may also affect wildlife species and some rodents. Goats and sheep are resistant.

➢The *disease is important because it causes a loss in production, especially in dairy herds, and it must be differentiated from foot and mouth disease in cattle and pigs.*

Etiology

✓*Vesiculovirus* of *Rhabdoviridae* family

✓RNA virus with 70-175 nm size, bullet shape

✓Insects are believed to be the principal vectors of transmission. Mosquitoes, black flies, sand flies, gnats, and midges have been identified as possible vectors. The disease is limited to the western hemisphere.

Pathogenesis

✓Transmitted through insects

✓Viraemia and virus settles in oral mucosa causing vesicular glossitis

Clinical signs

cattle :

- ✓mild fever, and the development o f vesicles o n the dorsum of the tongue, dental pad, lips and the buccal mucosa.
- ✓The vesicles rupture, and the resulting irritation causes profuse salivation and anorexia.

Horses:

- ✓the signs are more or less similar but usually the lesions are limited to the dorsum of the tongue or the lips.

Pigs:

- ✓vesicles develop on or behind the snout or on the
- ✓feet, and lameness is more common than in other animals.

Post Mortem Lesions

- Lesions are most common on the tongue and oral mucosa, but may also occur on the teats and the coronary band.
- Vesicles on tongue and snout of pigs

Vesicles on tongue

Microscopically

- Intercellular oedema in the stratum spinosum. This leads to cell dissociation and necrosis.

- Neutrophils and macrophages infiltrate the necrotic t issue, which sloughs, leaving erosions that are subject to secondary infection.

- The intra-epithelial oedema may become abundant enough to result in a vesicle.

- Healing usually occurs in 7-10 days.

VESICULAR EXANTHEMA

Vesicular exanthema is an acute, febrile, infectious disease of pigs caused by a *calicivirus.*

It is characterized by f ever and vesicle formation on the snout, mouth, non-haired skin, and feet.

It is indistinguishable clinically from foot-and-mouth disease in pigs, vesicular stomatitis, and swine vesicular disease.

Etiology

- *Calcivirus* of 35-40 nm diameter.

- Viruses of *Calciviridae* family were once included in *Picornaviridae*. Now they are classified as a separate group. *Caliciviruses* a re slightly larger than *picornaviruses*, and virion has 32 cup-shaped surface depressions.

Spread

- Raw garbage containing infected pork scraps is the most common medium of spread from farm to farm.

- The sources of infection are infected live pigs and infected pork.

- On infected premises, the disease spreads by direct contact.

Pathogenesis

- Direct contact
- Viraemia
- Buccal mucosa and skin

Characteristic symptoms

- Fever, anorexia
- Vesicles on mouth, snout, teats, claws
- Rupture of vesicle leads to ulcer

Macroscopic features

- Vesicles on snouts, lips, tongue, coronary band and mammary gland
- Ruptured vesicles covered by brown and dry dead tissue
- Ulcer on secondary bacterial infection

Microscopic features

- Vesicles, hydropic degeneration on stratum spinosum
- Infiltration of mononuclear cells

Diagnosis

- Symptoms and lesions
- Detection of antibody or antigen using ELISA


Thank You.