University of Mosul
Lecture No.:
College of Veterinary Medicine
Date:
Unit of Scientific Affairs
Website:

Lecture title:

Diseases of Bovine

Part -2

Lecturer Affiliation:
Ahmed M.A. Alsaidya *, PhD*
Asst. Prof. , Department of Pathology and Poultry Diseases College of Veterinary Medicine, University of Mosul, Mosul, Iraq

Summary:

Bovine Viral Diarrhea
(mucosal Disease)

It is an acute infectious disease of cattle, which caused by pestivirus and characterized by diarrhea and erosions on the digestive tract.

affects cattle of all ages but is most common in animals 8 months to 2 years of age.

Animals infected early in life with non cytopathic BVD virus develop a persistent infection. Later in life, if exposed to cytopathic virus, they may have disease develop.

Cause

It is caused by RNA pestivirus.. Susceptible hosts Cattle and buffalo are the only susceptible hosts. Young animals without maternal immunity and less than 2 years of age are most susceptible to the disease.

Mode of transmission

The disease is transmitted by direct and indirect contact, particularly through ingestion and inhalation of infected saliva, oculonasal discharge, urine and feces.

The virus is present in two forms, cytopathogenic BVDV and noncytopathogenic BVDV according to its effect on tissue culture.

The cattle postnatally infected with cpBVDV between 6 months to 2 years develop bovine viral diarrhea with high morbidity ((Morbidity in a herd varies from 2%to 50%)) and low mortality. Meanwhile, mucosal

disease with low morbidity and high mortality developed in cattle infected intra-uterine with noncpBVDV .

Pathogenesis

The pathogenesis of BVDV infection is complex, with infection pre- and post-gestation leading to different outcomes. Infection of the dam during gestation results in fetal infection, which may lead to embryonic death, teratogenic effects or the birth of persistently infected (PI) calves. PI animals shed BVDV in their excretions and secretions throughout life and are the primary route of transmission of the virusw.

Replication of BVDV occurs within the cytoplasm of the host cell and viruses exit via exocytosis. The noncpBVDV replicates in leukocytes, lymphoid tissue, the proximal colon and the respiratory tract , while cpBVDV was found to replicate in the gastrointestinal tract. In persistently infected males BVDV replicates within the seminal vesicles and the prostate gland.

Acute infection with BVDV results in transient viraemia prior to seroconversion and can lead to reproductive dysfunction and immunosuppression leading to an increased incidence of secondary disease.

Clinical Signs

The diseased cattle show fever, anorexia, depression and diarrhea. Moreover, excessive salivation with stringy mucus hanging from the muzzle to ground is characteristic. Erosion and ulcers are seen on muzzle and nose. The conjunctiva may be congested and the animal is emaciated and dehydrated.

Abortion , still birth and congenital defects are recorded in pregnant animals. Congenital cerebral hypoplasia, cataract, micorphthalmia and others malformation are seen in calves born to infected dam. Calves born alive may be persistently infected(PI) and later develop mucosal disease.

Gross appearance

the diseased animals are dehydrated and emaciated. The lesion usually seen in the gastrointestinal tract.

Erosions or well circumscribe ulcers of irregular shape are seen on inside the lips, gums, check, dental pad, posterior part of the hard palate, and on the lateral surface of tongue . Ulcers are seen also on muzzle and external nares. 13

The pharynx shows ulcer, which may be extended, to larynx. The esophagus shows ulcer and erosion.

Rumen and omasum show ulcer and erosion beside erosions in the folds of the fundus and submucosal hemorrhage and edema of the abomasum wall.

In the intestine, the characteristic lesion is sharply demarcated foci of necrosis in the mucosa over the GALT. Congestion and erosions are noticed in small intestine, cecum and colon particularly the areas above Payer‟s patches.

Necrosis is seen in lymph node and spleen.

In cattle with chronic mucosal disease, chronic ulcer are seen on the oral cavity and on skin particularly perineum, skin horn junction and around hooves

Microscopic appearance

Erosions are represented by necrosis of the cells of stratum spinosum and granulosum with intact basal cell layer followed by necrosis of the upper layers their removal leading to ulcer are seen on buccal cavity and esophagus No vesicles are seen. The abomasum shows atrophy and cystic changes of the gastric glands, besides edema and erosion and hemorrhage are common in the lamina propria and submucosa. The intestine showed catarrhal and necrotic inflammation focal areas of necrosis and erosions of the epithelium particularly over Payer's patches are seen in small and large intestine. The lymphoid follicles and lymph nodes show necrosis of lymphocytic elements.

Differential diagnosis:

1. Rinder pest
2. foot and mouth disease
3. parasitic and bacterial enteritis. 14

Lumpy Skin Disease
(bovine nodular exanthema)

It is a highly infectious skin disease of cattle and less common in buffalo characterized by generalized cutaneous nodules.

Cause

Lumpy skin disease is caused by a virus in the genus Capripoxvirus of the family Poxviridae.

Lumpy skin disease virus (LSDV) is closely related antigenically to sheep and goat poxviruses. Although these three viruses are distinct, they cannot be differentiated with routine serological test

Transmission

LSDV is thought to be transmitted primarily by biting insects. This virus has been found in mosquitoes in the genera Aedes and Culex during some outbreaks. Experimentally infected Aedes aegypti are infectious for 6 days and can transmit LSDV mechanically during this time. Flies (e.g. Stomoxys calcitrans) and other insects might also be involved in transmission, but this remains unproven.

Direct contact could be a minor source of infection. LSDV occurs in cutaneous lesions, saliva, respiratory secretions, milk and semen. Shedding in semen may be prolonged; viral DNA has been found in the semen of some bulls for at least 5 months after infection. Animals can be infected experimentally by inoculation with material from cutaneous nodules or blood, or by ingestion of feed and water contaminated with saliva. LSDV is very resistant to inactivation, surviving in desiccated crusts for up to 35 days, and can remain viable for long periods in the environment.

Susceptible hosts

Lumpy skin disease is primary a disease of cattle and less common in buffaloes. 15

Signs

The disease usually occurs is hot season. The incubation period is about two to five weeks. Persistent fever, anorexia, reduce milk production, emaciation besides generalized suddenly skin nodules is observed. The skin nodules are most numerous on the neck, brisket, back, thighs, legs, perineum, udder, scrotum, and around the muzzle and eyes. Morbidity is high but mortality is generally low, but may be approach 10%.

Post Mortem Lesions

The post mortem lesions can be extensive. Characteristic grayish-pink deep nodules with necrotic centers are found in the skin; these nodules often extend into the subcutis and underlying skeletal muscle, and the adjacent tissue exhibits congestion, hemorrhages and edema. The regional lymph nodes are typically enlarged. Flat or ulcerative lesions may also be found in the mucous membranes of the oral and nasal cavities, pharynx, epiglottis and trachea. Nodules or other lesions can occur in the gastrointestinal tract (particularly the abomasum), udder, urinary bladder, lungs, kidneys, uterus and testes. In the lungs, the lesions are difficult to see and appear as focal areas of atelectasis and edema. The mediastinal lymph nodes are enlarged in severe cases, and pleuritis may be seen. Some animals may have synovitis and tendosynovitis with fibrin in the synovial fluid. Aborted fetuses do not always have the characteristic external lesions, but some may be covered in nodules.

Microscopically

The dermis shows edema, perivascular infiltration by lymphocytes, macrophages and neutrophils. Acanthosis, parakeratosis and hyperkeratosis are seen in the epidermis. Moreover, eosinophilic intracytoplasmic inclusion bodies are seen in keratinocytes, macrophages and fibroblasts . Later on the hyperplastic cells undergo vesiculation and necrosis, which requires three to 5 weeks to heal. The lungs show coagulative 16

necrosis, surrounded by mononuclear cells mainly lymphocytes and plasma cells and finally followed by fibrous tissue. Other organs show necrosis infiltrate by round cells.

Diagnosis

Clinical Lumpy skin disease should be suspected when the characteristic skin nodules, fever and enlarged superficial lymph nodes are seen. The mortality rate is usually low.

Differential diagnosis

Differentials include

- Bovine herpes mammillitis* pseudo-lumpy skin disease), Dermatophilosis
- Ringworm
- Insect or tick bites,
- Hypoderma bovis infestation
- Photosensitization
- Bovine papular stomatitis
- Cutaneous tuberculosis.

Most of these diseases can be distinguished from lumpy skin disease by the clinical signs, including the duration of the disease, as well as histopathology and other laboratory tests.