

Popular Article

Lympy skin disease of cattle

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Introduction

As the name implies, lumpy skin disease is an infectious virus that affects cattle and buffalo. It is characterized by fever; multiple firm circumscribed raised nodules on the skin, ulcerative lesions in the mucosa, pneumonia, generalized lymphadenitis and edema of the ventral body parts and limbs. Especially in animals not previously exposed to the virus, it can cause death. The condition is often aggravated by secondary bacterial infections. LSD can cause considerable variation in mortality and morbidity depending on the breed of cattle, the population's immune status, insect vectors involved in transmission, and virus

Etiology-

Lumpy skin disease is caused by the lumpy skin disease virus (LSDV), a member of the genus Capripoxvirus (CaPV) within the family Poxviridae. Frequently, new foci of infection appear in areas far removed from the initial outbreak. Its incidence is highest in wet summer weather, but it may occur in winter. The morbidity rate in LSD is 10-20%, while the mortality rate is up to 5%.

Epidemiology

There has been no reported case of LSD in sheep and goats even when kept in close contact with infected animals. LSD has limited host range and does not complete its replication cycle in non-ruminant hosts. The disease is most commonly found in cattle breeds such as Holstein-Friesian and Jersey, but it has been experimentally produced in sheep, goats, impalas, and Grant's gazelles without systemic disease. There are more severe clinical symptoms in young calves and cows at peak lactation, but the disease affects all age groups. Water buffalo (*Bubalis bubalis*) were observed to have lumpy skin disease during an outbreak in Egypt in 1988, but the morbidity rate was much lower than that of cattle (1.6%). In cattle, *Bos taurus* is more susceptible to clinical disease than *Bos indicus*, probably due to a decrease in ectoparasite susceptibility. LSDV affects both sexes and all ages, but some evidence suggests that young animals are more likely to contract the disease in its severe form.

1. Transmission

LSDV is transmitted via cattle movement. Infected cows excrete infectious LSDV in saliva, nasal discharges, and ocular discharges, which can contaminate shared feeding and drinking areas.

The virus can be transmitted to pregnant cows through the semen of infected bulls, natural mating or artificial insemination. Infected bulls deliver skin lesions to their calves. Infected milk or skin lesions in teats may transmit the virus to suckling calves.

There are also local blood-feeding insects that can transmit the virus. Staple fly (*Stomoxys calcitrans*), mosquito (*Aedes aegypti*), and some tick species (*Rhipicephalus* and *Amblyomma* spp.) are known to spread the LSDV.

2. Clinical findings

As a result, lachrymation, nasal discharge, and high fever may occur for a week before characteristic nodular eruptions of the skin and body appear. There is a firm mass of creamy gray or yellow tissue within the skin nodules. A mild case may have a few skin lesions, whereas a severe infection may have multiple lesions. Preditis is most prevalent on the head, neck, perineum, genitalia and udder of animals. A painful ulcerative lesion may develop on the cornea of one or both eyes, resulting in blindness in the worst-case scenario. Regional lymph nodes (prefemoral and subscapular lymph nodes are swollen, and edema develops in the udder, brisket, and legs. There may be secondary bacterial infection persist that causes extensive suppuration and sloughing, so the animal may become extremely emaciated. Mastitis are common complication of disease. With the time, nodules either regress, or necrosis of the skin results in hard, raised areas (“sit-fasts”) clearly separated from the surrounding skin. These areas slough to leave ulcers, which heal and scar.



Figure 1. skin nodule lesions in infected cattle and buffalo

Diagnosis

Viral isolation

Lumpy skin disease virus (LSDV) may be recovered from skin biopsies and blood samples collected from clinically infected cows.

Histopathological findings

In addition to providing a basis for diagnosis, histopathological findings provide typical findings. As well as ballooning and degeneration of cell layers, the presence of eosinophilic intracytoplasmic inclusion bodies is a pathognomic finding detected microscopically in keratinocytes, macrophages and endothelial cells from skin nodules. The affected area is infiltrated by macrophages, lymphocytes, and eosinophils. Furthermore, diffuse vasculitis reflecting the viral tropism for endothelial cells is seen histopathologic ally. Muscular damage during the course of LSD can cause severe coagulative necrosis in subcutaneous muscles.

Hematological and Biochemical analysis

As a result of hemochemical and serum biochemical analysis, red blood cells, hemoglobin, packed cell volume, and mean corpuscular hemoglobin concentrations were significantly decreased in experimentally infected animals with a significant increase in mean corpuscular volume, which is interpreted as macrocytic hypochromic anemia. In contrast, the leucogram showed leucopenia, lymphopenia, and granulocytic leukocytosis, which may have resulted from secondary acute bacterial infections, including pyogenic bacterial infections. In addition to inflammatory thrombocytopenia, hyperfibrinogenemia, decreased creatinine concentrations, hyperchloremias, and hyperkalemias in naturally infected cattle, LSD is also associated with hyperfibrinogenemia. Studies by Neamat-Allah and Abutarbush revealed that serum protein and albumin levels were significantly decreased [46,52], but globulin levels, especially gamma globulins, increased significantly in LSD infected cows. A serum biochemical analysis of cattle infected with LSD by Sevik et al. revealed an increase in aspartate aminotransferase and alkaline phosphatase, along with higher levels of globulin protein and creatinine. LSDV infection was found to cause changes in serum biochemical analysis due to liver and kidney failures, severe inflammation, anorexia, and reduced muscle mass.

Molecular and serological diagnosis

Polymerase chain reaction (PCR), dot blot hybridization (DBH), and indirect enzyme-linked immunosorbent assay (iELISA) were used for the diagnosis of lumpy skin disease in clinically infected animals.

Differential diagnosis

In the case of pseudo lumpy skin disease, also known as Allerton virus, which is caused by bovine herpesvirus-2, skin lesions may resemble those of LSD, and must be confirmed in the laboratory. A pseudo lumpy skin disease is characterized by circular superficial lesions that can cover the entire body and reach a diameter of 2 cm. Bovine herpes mammillitis is the disease in which lesions appear only on cows' teats and udders. An intact central area and raised edges are present, along with hair loss. Pseudo-lumpy skin disease is a milder disease than true lumpy skin disease, but differentiation depends essentially on isolation and identification of the causal virus. Among the differential diagnoses of LSD are urticaria, streptotrichosis (infection by *Dermatophilus congolensis*), ringworm, hypoderma bovis infection, photosensitization, bovine papular stomatitis, foot and mouth disease, bovine viral diarrhea, and malignant catarrhal fever. The pox virus of lumpy skin disease can be demonstrated by electron microscopy in the early skin lesions. The two diseases can be distinguished by PCR.

Treatment

There are no specific antiviral drugs available for the treatment of lumpy skin disease, only supportive therapy is available. This can include treatment of skin lesions using wound care sprays and the use of antibiotics to prevent secondary skin infections and pneumonia. Anti-inflammatory painkillers can be used to keep up the appetite of affected animals. Intravenous fluid administration may be of benefit; however this may not be practical in the field. The lack of treatment options for lumpy skin disease virus emphasizes the need of using effective vaccination for preventing disease.

Prevention and control

- A careful surveillance of the disease onset and spread of the disease.
- Introduction of recent new animals into herds should be limited. New purchased animals should be examined and declared free from clinical signs before to movement and on arrival, and should be kept quarantined from the herd for a minimum of 28 days.
- Animal should be treated with insect repellents regularly to reduce the chance of vector transmission of the disease. This measure cannot fully prevent transmission but may reduce the danger.
- Destroyed vector breeding sites such as standing water sources, slurry and manure and improving drainage system are sustainable, affordable ways of limiting the number of vectors on and around animals.
- Attenuated virus vaccines may help control spread. Vaccination with attenuated virus offers the most promising method of control and was effective in halting the spread of the disease.