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Lumpy Skin Disease in Cattle

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Introduction:

Lumpy skin disease (LSD) is a viral disease caused by lumpy skin disease virus (LSDV), a member of *Capripoxvirus* genus, subfamily *Chordopoxvirniae* and *Poxviridae family*. Lumpy skin disease virus (LSDV) is a double-stranded DNA containing around 150 kilobase pairs (kbp) with relatively large sizes (230–260 nm), enclosed in a lipid envelope The disease is known by various names such as "LSD", "Pseudo-urticaria", "Neethling virus disease", "exanthema nodularis bovis", and "knopvelsiekte". The world organization for animal health (OIE) categorises the LSD as notifiable diseasedue to its significant economic losses and the potential for rapid spread. The first case of LSD was reported from Zambia in 1929. According to OIE, at present this disease is prevalent in countries including various African, European and Asian countries. The reasons of the disease spread to India are unknown but it may be due to livestock movement across international borders or may be due to vectors movement from the neighbouring countries. In recent years, LSD has been reported from countries neighbouring India like China and Bangladesh. Therefore, understanding the epidemiology of exotic diseases becomes necessary for timely planning the effective disease management.

Species affected:

LSD is a non-zoonotic, vector borne and transboundary disease with limited host range and currently restricted to ruminants viz. cattle and water buffaloes. Animals of all ages are susceptible but calves are more susceptible and develop lesions within 24 to 48 hours to natural infections, probably due to impairment of humoral immunity. Normally the role of wildlife in the transmission and maintenance of LSDV has been found almost negligible. Humans are also resistant to the virus. Animals that have recovered from natural infection by the virus have shown lifelong immunity. Calves from their infected dams are resistant to clinical disease for approximately 6 months because of the acquired maternal antibodies. Affected animals clear the infection and no carrier state has known for LSDV yet.

Transmission:

The arthropod vectors responsible for the disease spread include biting flies, mosquitoes and ticks. The main sources of infection are considered to be skin lesions as the virus persists in the lesions or scabs for long periods. The virus is also excreted via the blood, nasal and lachrymal secretions, saliva, semen and milk. *Aedes aegypti* is the sole dipteran to be able to fully transmit the virus to susceptible cattle. Mosquitoes such as *Culicoides nubeculosus*, *Culex quinquefasciatus* Say and *Anopheles stephensi* Liston were not able to transmit the virus. Natural infection of sheep and goat has not been reported even in close contact with infected cattle and buffaloes but skin lesions have been seen after experimental infection in sheep, goat, giraffe, Giant gazalles, impalas.

Pathogenesis:

Following LSDV infection, virus replication, viremia, fever, cutaneous localization of the virus and development of nodules occur

1. 4 to 7 days post-infection (DPI): localized swelling as 1–3 cm nodules or plaques at the site of inoculation.
2. On 6 to 18 DPI: viremia and shedding of the virus via oral and nasal discharge.
3. On 7 to 19 DPI: regional lymphadenopathy and development of generalized skin nodules.
4. On 42 days after fever: presence of virus in semen can be detected.

Clinical Signs:

The incubation period of disease in natural condition is between 2 and 5 weeks. The illness begins with biphasic fever. The clinical features of the disease include fever, inappetence, nasal discharge, salivation and lacrymation, enlarged lymph nodes, a considerable reduction in milk production, loss of body weight and sometimes death. LSDV can lead to abortion, mastitis and orchitis. The disease is characterized by firm, slightly raised, circumscribed skin nodules that are 2–7 cm in diameter and typically appear on the neck, legs, tail and back, shortly after the beginning of fever. The number of the lesions may vary from a few in mild cases, to multiple lesions, covering the entire body in severely infected individuals. In addition, necrotic plaques may appear in the mucous membranes of the oral and nasal cavities, causing purulent or mucopurulent nasal discharge and excessive salivation. Moreover, ulcerative lesions may appear in the cornea of one or both eyes, leading to restricted vision, and even to blindness. Severe cases may show characteristic lesions throughout the entire digestive and respiratory tracts and on the surface of almost any internal organ.

Diagnosis:

Despite a primary clinical diagnosis of LSD which can be confused with other diseases like foot and mouth disease (FMD), insect bite, demodicosis and hypersensitivity. Tentative diagnosis can be made on the basis of skin nodules observed on face, eyelid, neck, muzzle, nostrils, udder, and limbs. Skin biopsy sample can be collected for further confirmation of disease. The diagnosis is confirmed by using conventional PCR or real-time PCR techniques.

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Molecular methods are more precise, reliable and rapid compared with other methods. Skin samples can be checked by electron microscopy to identify virus. Virus isolation can be used for the confirmatory diagnosis in new niches. The bovine testes and pre-pubertal lamb, primary and secondary culture is most sensitive for isolation of virus. The virus persists in the skin and could be isolated 38 days post-infection, whilst viral DNA was detected using PCR in skin lesions for more than 90 days.

Prevention and Control:

The treatment of LSD is only symptomatic and targeted at preventing secondary bacterial complications using a combination of antimicrobials, anti-inflammatory, supportive therapy and anti-septic solutions. In order to halt the spread of LSD by vectors in particular, culling of all susceptible animals that have been exposed to the infection, or at least those showing clinical signs, is a generally recommended control measure for LSD. However, regarding the role of arthropod vectors, elimination of the disease is likely to be difficult and any delays in the removal of infected animals increase the risk of LSD transmission. Educating veterinarians and livestock workers would enable them to perform timely diagnoses of clinical cases, helping to slow the spread of disease. Vectors movement due to prevailing winds may cause disease transmission. Vector control methods like use of vector traps, use of insecticides can also be used for preventing the disease. Members of the capripoxvirus are known to provide cross-protection. Hence, homologous (Neethling LSDV strain) and heterologous (sheeppox or goatpox virus) live attenuated vaccines can all be used to protect cattle against LSD infection. The reasons behind the entry of LSD in India need to be investigated along with epidemiological random screening in different regions to access the actual disease prevalence. Besides, effective quarantine methods, vector control methods, vaccination is the only method to prevent the disease.

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