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Lumpy Skin Disease

Rahul Singh, Rakesh Kumar*, R. K Asrani

Department of Veterinary Pathology, Dr. G.C. Negi College of Veterinary and Animal Sciences, CSKHPKV, Palampur, Himachal Pradesh, India-176062

*Corresponding Author
Rakesh Kumar*
E-mail: rkvetpath@gmail.com

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INTRODUCTION

Lumpy skin disease (LSD) is a highly infectious disease affecting cattle, buffaloes and occasionally other wild species of hoofstock which is caused by Lumpy skin disease virus (LSDV)of the genus Capri poxvirus (Neethling virus). This disease is characterised by the eruption of multiple well circumscribed skin nodules all over the skin and mucous membrane, as well as fever, ventral oedema, and generalised lymphadenopathy.

Etiology

LSDV is double-stranded DNA virus. It is a member of the *Capripoxvirus* genus of family *Poxviridae*. Capripoxviruses belongs to the *Chordopoxvirus* subfamily. The capripoxvirus genus consists of LSDV, as well as sheeppox virus, and goatpox virus. The infections of capripoxvirus are generally host-specific in certain geographical ranges, but they are serologically undifferentiated.

Epidemiology

LSD is present throughout the African continent and Madagascar, with sporadic reports from the Middle East and Israel. LSDV mostly affects cattle and zebus, although it has also been observed in giraffes, water buffalo, and impalas. Cattle of all ages, genders, and breeds are affected, however Channel Island breeds are more severely impacted. Both Bos indicus and Bos taurus cattle are vulnerable; however, the illness in Zebu strains can be less severe. The illness is particularly contagious in fine-skinned *Bos taurus* cow breeds like Holstein-Friesian and Jersey. Thick-skinned *Bos indicus* breeds, such as the Afrikaner and Afrikaner cross-breeds, exhibit less severe illness symptoms. This is most likely owing to Bos indicus breeds lower sensitivity to ectoparasites compared to Bos taurus strains. Young calves and cows in peak lactation have more severe clinical signs.

The morbidity is highly varied, and it is usual to have an infection that is not visible. Mortality is typically modest, about 1%, but can reach 50%. Economic losses arise from debilitation, loss of milk and meat production, hide damage, and reproductive wastage caused by fever-related abortions and temporary sterility in bulls.

Transmission

Blood-feeding arthropods are thought to be the primary vectors of transmission. The natural incubation period for lumpy skin disease is 2-4 weeks. Epidemics typically related with high temperatures and humidity and periods of extended rainfall, which favours population growth in vector species. It is most common during the wet summer and fall months, especially in low-lying places or near bodies of water; but outbreaks can occur during the dry season as well. Mechanical vectors of the illness include blood-feeding insects such as mosquitos and flies. Outbreaks of lumpy skin disease are sporadic because they are affected by animal migrations, immunological state, and wind and rainfall patterns, all of which impact vector populations. The virus can be spread by blood, nasal discharge, lacrimal secretions, sperm, and saliva. The illness can also be passed on to suckling calves through contaminated milk. There is no one species of vector identified. Instead, the virus was recovered from the species *Stomoxys, Biomyia fasciata, Tabanidae, Glossina,* and *Culicoides*. LSDV, like other pox viruses that are known to be extremely resistant, can survive in infected tissue for more than 120 days. During the interepidemic phase, an infectious reservoir is considered to be a forest maintenance cycle, most likely including Cape buffalo. Aside from cattle, no reservoir hosts have been discovered.

Clinical Signs

- Fever, copious drooling, oculonasal discharge, swollen superficial lymph nodes, and numerous nodules (measuring 2–5 centimetres in diameter) on the skin and mucous membranes (including those of the respiratory and gastrointestinal tracts) are all symptoms of LSD.
- Infected cattle may also develop oedematous swelling in their limbs and exhibit lameness.
- Afflicted animals skin suffers irreversible damage, reducing the market worth of their hide.
- The illness frequently causes chronic debility, ventral oedema, widespread lymphadenopathy, decreased milk supply, poor development, infertility, miscarriage, and, in rare cases, death.
- Secondary bacterial infection may lead to suppuration of nodules.
- In mild illness there may be a few solitary nodules and no prodromal fever.

Pathology

Macroscopically, the cutaneous lesions are firm, confined, flat-topped papules and nodules ranging in size from 0.5 to 5.0 cm and have a creamy grey to white hue on cut portion and may leak serum and cover the whole width of the cutis, reaching into the subcutis and occasionally neighbouring muscles. Sometimes, the nodules may coalesce together to make larger nodules. Nodules affecting the scrotum, perineum, udder, vulva, glans penis, eyelids, and conjunctiva are often flatter and surrounded by a zone of severe hyperaemia in nonpigmented tissue.

The nodules often undergo central necrosis and sequestration, although some may resolve quickly and completely, while others may fail to detach and instead become indurated, persisting as hard intradermal lumps for many months. As the separation process progresses into the dermis, the nodule develops a core or sequestrum of necrotic material (“sit-fast”) that is cone shaped and flat topped. Large craterous ulcers cause lymphangitis and lymphadenitis. Localized lesions can lead to blindness, tenosynovitis, arthritis, or mastitis. Nodules in the respiratory system may induce oedema severe enough to cause dyspnoea and suffocation.

Microscopically, the nodule is distinguished by an inflammatory response in the dermis. The nodule is oedematous, with perivascular aggregates of lymphocytes, macrophages, plasma cells, and neutrophils, as well as proliferating fibroblasts. Acanthosis (thickened epidermis), parakeratosis (thickened stratum corneum with pyknotic nuclei), and hyperkeratosis (thickened stratum corneum) of the epidermis are present, followed by necrosis and vesicle formation. In keratinocytes, fibroblasts, and macrophages eosinophilic cytoplasmic inclusion bodies develop. The complete nodule necrosis is followed by healing, which generally takes 3 – 5 weeks, however some nodules might last for months. The presence of eosinophilic cytoplasmic inclusion bodies in sections of skin lesions and viral tissue cultures is required for diagnosis.

Diagnosis

- Histopathology
- Virus isolation
- PCR
- Electron microscopy is utilised to show the typical pox virions in the lesions.
- Use of fluorescent antibody and serum neutralisation testing is widespread.

Differential Diagnosis

The most common differential diagnosis is pseudo–lumpy skin disease caused by a Bovine Herpesvirus 2 (BHV-2) that is identical to the Bovine Herpes Mammillitis virus but was formerly identified as the Allerton virus. Clinically, pseudo–lumpy skin disease is a milder illness with superficial nodules that resemble only the early stages of lumpy skin disease. Moreover, BHV-2 has a shorter course than LSD. Intranuclear inclusion bodies characterise BHV-2, as opposed to intracytoplasmic inclusions that characterise LSD. The presence of poxvirus particles in fresh or formalin-fixed tissue is the best way to confirm the latter. Electron microscopy can also help distinguish between the two illnesses.

Treatment

Antibiotics are administered to control secondary bacterial infection and supportive treatment along with good nursing care is recommended.

Immunity

Natural Immunity

After recovering from a normal illness, most cattle gain lifetime immunity. Furthermore, calves of immunological cows develop maternal antibodies and are resistant to clinical illness until they are around 6 months old. Calves under 6 months of age whose mothers were naturally infected or vaccinated should not be vaccinated to prevent interfering with maternal antibodies. Calves born from vulnerable cows, on the other hand, are likewise susceptible and should be vaccinated.

Artificial Immunity

Immunization against LSDV has been approached in two ways. The virus's Neethling strain was initially attenuated in South Africa by 20 passes through the chorio-allantoic membranes of hen eggs. The vaccine virus is now being grown in cell culture. A vaccine made from sheep or goatpox viruses has been proven to confer protection in cattle in Kenya. Adult cattle that are vulnerable to LSDV should be vaccinated yearly to guarantee optimal protection. Swelling (10–20 millimetres (12–34 in) in diameter) develops in around 50% of cattle at the site of inoculation. This swelling goes down in a few weeks. Dairy cows may have a transient reduction in milk output after being inoculated.

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Circumscribed, raised, firm, confined, flat-topped papules and nodules ranging in size from 0.5 to 5.0 cm all over the skin of buffalo