

REVIEW ARTICLE

Lumpy Skin Disease- A Review

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ABSTRACT

Lumpy skin disease (LSD), a pox virus disease of domestic cattle, water buffaloes, and some wild ruminants is caused by Lumpy Skin Disease Virus (LSDV) which is a member of the genus Capripoxvirus and family Poxviridae. From an antigenic standpoint, it is very similar to the goat and sheep pox viruses. Blood-feeding arthropod vectors (mosquitoes, biting flies and ticks) are the primary means of infection in animals. The LSD incubation period is 28 days, however the experimentally infected calves may show clinical indications as soon as 6 to 9 days after infection. The illness is marked by fever, numerous firm, confined skin nodules, necrotic plaques in the mucous membranes (mostly of the upper respiratory tract and oral cavity), and enlargement of the peripheral lymph nodes. Chronic illness is not a result of LSD. It does not develop latent symptoms, and the condition does not recur. Also, humans cannot contract this disease since it is not zoonotic. The World Organization for Animal Health (OIE) classifies transboundary LSD as a notifiable disease, and it has a significant economic impact on the cattle industry due to decreased milk and meat production, abortions, issues with male and female fertility, damaged cattle skins, and, ultimately, the death of severely affected animals. Indirect losses are created by national and international cattle movement and trade limitations. The aim of this review is to provide an overview of the lumpy skin disease highlighting various strategies and actions needed to control outbreaks of this emerging disease.

Keywords: Lumpy skin disease, OIE, LSDV

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INTRODUCTION

The Lumpy Skin Disease Virus (LSDV), which belongs to the family Poxviridae, subfamily Chordopoxvirinae, of the Capripoxvirus genus, is the cause of lumpy skin disease. Exanthema nodularisbovis, pseudo-urticaria, Neethling virus sickness, and knopvelsiekte are a few of the names given to the disease [3, 31]. Initially believed to be an allergic reaction to insect bites, lumpy skin disease (LSD) was first discovered in Northern Rhodesia (Zambia) in 1929 [18]. Given the significant economic effects on cattle, LSDV is one of the most significant animal poxviruses. LSD is a notifiable disease according to the World Organisation for Animal Health [20]. LSD is a transboundary, vector-borne, non-zoonotic illness that currently only affects ruminants, such as cattle and water buffaloes. Biting flies, mosquitoes, and ticks are the arthropod vectors that transfer disease [33, 16]. Fever, decreased milk output, and skin nodules are its hallmarks. Other common conditions include mastitis, swollen peripheral lymph nodes, anorexia, increased nasal discharge, and watery eyes. Infected bulls and cows might become infertile either temporarily or permanently. The condition may result in both high morbidity and low mortality [34, 33]. Despite being in close proximity to diseased cattle and buffaloes, sheep and goats have not been recorded to naturally contract the disease. However, skin lesions have been observed in sheep, goats, giraffes, Giant gazelles, and impalas following experimental infection [7]. Up until 1943, the condition there recurred sporadically before spreading to Botswana (Bechuanaland) [36], Zimbabwe (Southern Rhodesia) [11], and South Africa [28]. The OIE reports that this disease is currently widespread in a number of African, European, and Asian nations [34]. Since its introduction into India, LSD incidents have been primarily seen in the east of the nation without any notable mortality for a period of roughly 2-3 years. The current LSD wave, which began in the states of Gujarat and Rajasthan in Western India in June or July 2022, is extremely deadly [14]. Sporadic cases emerged during the following two years, especially in Gujarat and Maharashtra. However, the sickness was documented in the prior year (2022) in western and northern states as well as on the island of Andaman Nicobar. The objective of this review is

to provide present understanding on several aspects of the illness, including its transmission, clinical signs, diagnostics and preventative and control strategies

Aetiology

LSDV is a brick-shaped encapsulated virus with double-stranded DNA that has complex symmetry and replicates in the cytoplasm of the host cell. The 151 kbp long LSDV genome has 156 putative genes and is made up of identical 2.4 kbp-long inverted terminal repeats that surround the central coding region. LSDV has 30 structural and non-structural genes that are 97% nucleotide identical to sheeppox and goatpox viruses [29,30].

The virus that causes lumpy skin disease (LSDV), which affects all domestic animals except dogs, is a member of the Poxviridae family of viruses. According to Quinn et al. [22], the family is divided into two subfamilies: Entomopoxvirinae, which infects invertebrate hosts, and Chordopoxvirinae, which infects vertebrate hosts. Buffalo (*Bubalus bubalis*) and cattle (*Bos indicus* and *Bos taurus*) are susceptible hosts. Compared to local breeds of cattle, the *Bos taurus* is more vulnerable. All animals are sensitive, however calves are particularly vulnerable and develop lesions in 24 to 48 hours (Al-Salihi 2014) [2]. Under natural conditions, wild animals are resistant to infection; however, experimental infection resulted in clinical lesions in Giraffe (*Giraffe camelopardalis*), impala (*Aepyceros melampus*), Arabian oryx (*Oryx leucoryx*), springbok (*Antidorcas marsupialis*) and Thomson's gazelle [7, 21].

Mode of Transmission

LSD is a vector-borne illness that is spread by ticks, mosquitoes, and other blood-feeding insects. The main method of disease transmission is by mechanical transfer by vectors. The arrival of the seasonal rains and the summer season, which coincide with the peak activity of the vectors, dramatically increases disease incidences in the majority of the endemic nations, including sub-Saharan Africa, Egypt, and Ethiopia (Mulatu and Feyisa 2018) [19]. As mechanical vectors and reservoirs of virus, the tick *Amblyomma* spp., *Rhipicephalus decoloratus*, *Rhipicephalus appendiculatus*, and *Amblyomma hebraeum* have been identified [16,32]. Additionally, mosquitoes like *Culex mirificens* and *Aedes natrionius* as well as biting flies like *Stomoxys calcitrans* and *Biomyia fasciata* are implicated in the mechanical transmission of disease. The disease results in lumps on the animal's body, and when flies and mosquitoes sit on them, they spread the infection to other healthy animals.

Calves can contract the disease through direct contact after nursing from sick cows [4]. After infection, the virus can stay in the semen for up to 42 days [12]. Additionally, iatrogenic spread of virus through infected needle is also seen [19].

Clinical Signs

In natural conditions, the incubation period for a disease lasts between two and five weeks, whereas in lab settings, it lasts between seven and fourteen days. There are three different types of LSD: acute, subacute, and chronic. Clinical signs of the illness include fever, weakness, nasal discharge, salivation, and lachrymation, as well as swollen lymph nodes, a significant decrease in milk production, weight loss, or even death (Abutarbush et al., 2013, Tasioudi et al., 2016) [1,27]. Biphase fever is the first sign of the sickness. Clinical signs of a minor infection include one or two lumps of nodules that occur within two to three days of the commencement of fever, emaciation, ocular discharge, and agalactia. Later, painful and hyperemic nodular lesions may appear on the animal's body, particularly on the skin of the muzzle, nares, back, legs, scrotum, and perineum, as well as the eyelids, lower ear, nasal and oral mucosa, and tail [24].

The lesions then develop into papules, vesicles, pustules with exudate, and finally eventually develop into scabs. The lesions heal quite slowly. On the mucous membranes of the mouth, vulva, respiratory system, and nose, lesions form over time. The skin sores harden and becoming necrotic after two to three weeks, making the animals uncomfortable and making them reluctant to move. According to Tupurainen and Oura [33], lumpy skin disease lesions exhibit eosinophilic intracytoplasmic inclusion bodies as well as ballooning epithelial cell degeneration. In some cases, inflammatory cells such as macrophages, lymphocytes, and eosinophils are seen infiltrating the superficial dermal tissue of affected areas, as well as widespread vasculitis and severe coagulative necrosis in subcutaneous muscles [5, 25].

Tissues like the muzzle, nasal cavity, larynx, trachea, inside of the lips, dental pad, gingiva, abomasum, udder, teats, uterus, vagina and testes might be affected. The complications like keratitis, dysentery, lameness, pneumonia, mastitis and myiasis were reported [3,35].

A typical and frequently fatal LSD side effect is pneumonia. In addition to greater areas of bronchopneumonia and focal or widespread areas of grey consolidation in the lungs, there may be lesions throughout the upper respiratory tract. When a necrotic slough develops from an old tracheal lesion, inhalation is a complication of the respiratory tract's necrotic lesions and it can be lethal even months

after the initial infection [8]. Cattle recovering from disease may remain in extremely poor conditions for about 6 months.

Diagnosis

A tentative diagnosis of LSD can be made based on the presence of an epizootic disease in cattle that causes the distinctive skin nodules and systemic sickness. It spreads to random in cattle of all ages, and no chemotherapeutic drug or antibiotic has any effect on the disease's progression. Herd movement restrictions do not prevent the spread of disease to surrounding herds.

It is preferable to have a laboratory diagnosis; this can be done by inoculating animals, histopathologically examining the lesions, isolating the virus, or determining the virus' identity using electron microscopy or serology [6].

Typical histological changes in skin samples include vasculitis and perivascular infiltration with white cells that results in a thrombosis of the vessel in the dermis and subcutis. Epithelial "cellesclavelaues" cells, which are also described in sheep pox, are invading the lesion. The agar gel precipitation test is not specific for LSD because other capripoxviruses and parapoxviruses share the LSDV antigen [10]. Molecular diagnosis using conventional PCR and real time PCR has been used for differential diagnosis [15].

Prevention and Control

Bovine movement restrictions with widespread immunisation of cattle is the most efficient method of controlling an outbreak (EFSA et al., 2020)[9]. Culling of all vulnerable animals that have been exposed to the infection, or at least those showing clinical indications, is widely advised as a control measure for LSD in order to stop the spread of the disease through vectors in particular [35]. However, this measure might not be affordable or practical in many nations with limited resources. Additionally, slaughtering cattle or other endangered animals could not be allowed by the law or for religious or customary reasons, making eradication impossible [23]. The need of quick mass immunizations of cattle and water buffaloes using a high-quality vaccine with established effectiveness against the virus is highlighted by all of the above facts. Successful control and eradication of LSD relies on early detection of the case index, followed by a rapid and widespread vaccination campaign.

Vaccination

Vaccination plays an important role in controlling the spread of disease. Vaccines currently used to protect cattle from LSD are primarily live attenuated vaccines based on attenuated strains of wild isolates passaged by cell culture. There are three licensed vaccines for bovine dermatosis (LSD): lumpy skin disease virus (LSDV) Neethling vaccine, Kenyan sheep and goat pox (KSGP), O-180 strain vaccines and Gorgan goat pox (GTP) vaccine. Live-attenuated vaccines (LSD and sheep/goat pox vaccines) are commercially available. Although the OIE Manual does not currently include information on inactivated LSD vaccines, some manufacturers have developed inactivated vaccines against LSD that countries may be willing to consider. Cattle are well protected against virulent field strains by homologous vaccinations. The KSGP O-240 and O-180 strains and the well-known South African Neethling strain are both included in LSDV vaccinations. The vaccine strain was attenuated from the virulent strain by 61 serial passages in lamb kidney (LK) cells, followed by 20 passages in the chorioallantoic membrane of embryonated chicken eggs, and three passages in LK cells. The Neethling strain was isolated from the first LSD outbreaks in South Africa [13,37]. Compared to live attenuated vaccinations, inactivated vaccines have both benefits and drawbacks. The security of inactivated vaccines is the main advantage. Their lack of replication prevents the vaccine virus from spreading to animals, reverting to virulence, and recombining with virulent viral strains. Recently, Lumpi-ProVacInd, the vaccine, has been developed by two institutes under the Indian Council of Agricultural Research (ICAR) – National Equine Research Center, Hisar (Haryana) in collaboration with the Indian Veterinary Research Institute, Izzatnagar (Bareilly).

CONCLUSION

Large ruminants, such as cattle and domestic water buffalo, are susceptible to the lumpy skin disease. Clinical symptoms include mild to severe fever, enlarged lymph nodes, and visible nodules all over the body, which are followed by the formation of necrotic tissue and scarring. Despite the low mortality rate of LSD, lesions can develop and pose problems for which there is no specific cure. LSD can be prevented effectively with immunization in conjunction with vector management, the control of animals, especially those from endemic areas, and ongoing disease surveillance of epidemic scenarios.

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