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Lumpy Virus: A Devastating Disease for Indian Farmers in Agriculture

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Abstract
Lumpy Skin Disease has been associated with high morbidity in domesticated animals resulting in adverse effects on the affected economy. Even though the disease is by no means a novel phenomenon, the lack of an effective treatment regimen makes it detrimental to the infected animal. It was endemic in African continent where it was reported in first half of 20th century and was reported in India in 2019; however its worst wave hit the country in 2022 claiming lives of as many as 97,000 cattle in just 3 months. This article discusses the aetiology, transmission and economic impact of the disease as well as possible prevention measures.

Introduction
Lumpy skin disease (LSD) is classified as one of the notifiable diseases of terrestrial animals due to its major impact on the economy and financial importance making it a disease of international concern. It is caused by the Lumpy Skin Disease Virus (LSDV), a member if the Poxviridae family noted for their infection causing ability in domesticated animals. The disease is known by various names such as "LSD", "Pseudo-urticaria", "Neethling virus disease", "exanthema nodularis bovis", and "knopvelsiekte". The first clinical manifestations of this disease were observed in Zambia in the year 1929 in the form of skin nodules however it was not until another incidence of epidemic stuck in Botswana in 1943 that evidences of involvement of an infectious agent was suspected. During this time, the disease spread rampantly throughout the continent of Africa as a panzootic including in the Republic of South Africa where the term "Lumpy Skin Disease" was coined. As of today, LSD is prevalent in most African countries and has extended to South-East Europe, Russia and Kazakhstan. The first report of a LSDV outbreak in India was reported in August 2019, in the state of Odisha and then days later in Patliputra. The cases in India were attributed to transboundary spread from China or Bangladesh which already had cases for the infections. A first published report of the strain of LSDV in India (which was genetically closer to South African strains) revealed 7.1% morbidity.

One of the defining features of the causal agent *i.e.*, LSVD is that it is highly host specific and affects only domestic cattle *(Bos indicus* and *Bos taurus*) and buffaloes (*Bubalus bubalis*) and even though the genome of LSVD is strikingly similar to Sheeppox virus and

Goatpox virus, no infection of either sheep or goat have been reported however experimental infection in giraffe (*Giraffe camelopardalis*), impalas((*Aepyceros melampus*), sheep, goats and Giant gazelles have yielded skin lesions cultivating belief that wild animals are naturally resistant to the infection under natural conditions. Humans have also found to be resistant to the virus.

The disease is associated with high morbidity but low mortality which affects the economic value of an animal in incidences of reduced quality of produce, meat, leather and live animal trade incurring heavy economic losses. The recent LSDV outbreak in India in July 2022 which started from the sates of Gujarat and Rajasthan and slowly spread to over 15 states has resulted in death of over 97,000 cattle in a span of three months with Rajasthan recording the maximum (about 65%) of all reported cases. The rampant spread of the disease and associated vectors calls for immediate measures to be employed to control the extent of damage.

Aetiology

The Lumpy Skin Disease is caused by Lumpy skin disease virus (LSVD), a member of the *Capripoxvirus* genus and the *Chordopoxvirinae* subfamily (consisting of 10 genera), family *Poxviridae.* There are two additional viral species under this genus namely Sheepox virus (SPPV) and Goatpox virus (GTPV) which too are highly host-specific. Except for dogs, the *Poxviridae* family comprises the largest viruses capable of causing disease in most domestic animals. The LSDV is a double stranded DNA virus with dimensions of 300×270×200 nm and a brick-like shape with complicated symmetry and an envelope. LSDV has a genome that is 151 kbp large and having 146 conserved sequences for synthesis of proteins involved in nucleotide metabolism, replication, transcription and structure formation and stability. The replication occurs in the cytoplasm of the host cell.

Transmission

The transmission of LSDV is mainly attributed to arthropod vectors in a process called mechanical transmission perhaps best demonstrated by the occurrence of disease in seasonal rains when the population of certain arthropods exponentially increases and relative decrease in incidences with the onset of winters. LSDV can be transmitted by a large selection of hematophagous arthropod vectors like the bitting flies (*Stomoxy calictrans* and *Biomyia fasciata*), mosquitoes (e.g., *Aedes natrionus*). The tick *Amblyomma* sp., *Rhipicephalus decoloratus*, *Rhipicephalus appendiculatus* and *Amblyomma hebraeum* etc. have been reported as a mechanical vectors and reservoirs of virus.

Poxviruses are extremely resistant and that accounts for their prolonged survival in infected tissues for over 120 days and even longer. The virus has been identified in semen, saliva, nasal discharge, lachrymal secretion and blood which are presumed to be primary routes of indirect LSD transmission to animals that may share feed, shelter and water with infected animals. Some iatrogenic routes of infections have also been suggested especially in mass vaccination drives that do not use a separate needle for every cattle. The transmission in these cases occurs from skin scabs or crusts. The infection can also be transmitted intrauterine *i.e.,* from mother to foetus along with incidences of transmission to calves via milk secretions and skin abrasions.

Clinical features and pathogenesis

The incubation period for LSDV is 2-4 weeks in the field, in experiment however it has been reported to be 7-14 days and spreads widely after cutaneous infection to include other organ systems. The onset of symptoms starts with a biphasic fever, and increase in respiratory rate. The characteristic nodules start to appear on the skin within 2-3 days of the onset of fever and start few in number before covering most of the epidermis ranging 2-7 cm in diameter which

appear well defined regions of erect hair, round, firm, and slightly raised from the surrounding skin, surrounded by a haemorrhagic ring. The fever is accompanied by lachrymation (ocular discharge), cessation of milk production (agalactia), emaciation, swelling of lymph nodes and nasal discharge. The nodules later become extremely painful, increasing in number and involve both dermis and epidermis. These raised nodular lesions later ulcerate and become extremely painful and spread over the entire body and are concentrated around anterior nares, eyelids, oral, nasal and lower ear mucosa, tail, regions of the groin (scrotum and vulva), perineum and legs (figure 1). The disease takes three forms-acute, sub-acute and chronic. The acute stage is characterised by a number of histo-pathological changes like enlargement, swelling and inflammation of lymph nodes and vessels (lymphangitis) and inflammation of blood vessels (vasculitis), infarction resulting from development of thrombosis and subsequent oedema and necrosis. Histo-pathological reveals the presence of eosinophilic intracytoplasmic inclusions bodies and ballooning degeneration of epithelial cells. Acute phase of LAS also accounts for the infertility in both males and females (anoestrus) and abortions.

In severe cases, the numbers of nodules increase to over a hundred and persist for over 7 to 12 days. This is followed by development of lesions over the respiratory tract with exudation turning necrotic after three weeks and cause discomfort in movement. The presence of haemorrhages and oedema in surrounding/adjacent tissue also occurs and the slow recovery leaves the cattle open to high incidences of secondary bacterial infections like mastitis and pneumonia.

Diagnosis

Clinical diagnosis of LSD is based off of the appearance of animal symptoms. The occurrence of nodules, pyrexia and enlargement of superficial lymph nodes are considered distinctive to this disease.

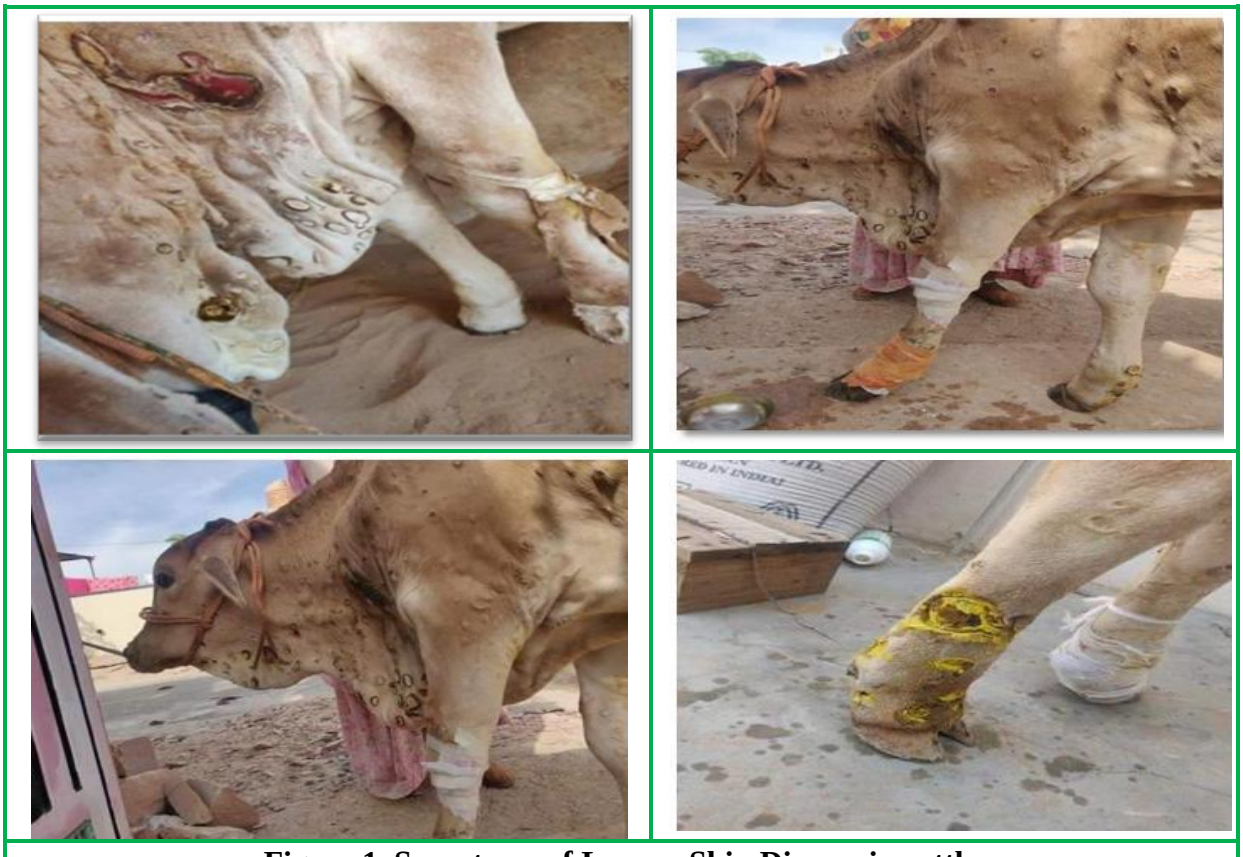

Figure 1. Symptoms of Lumpy Skin Disease in cattle

Viral isolation is critical for confirmation of clinical disease and determination of isolate. The viruses grows slowly on cell cultures and usually are grown on chorioallantoic membrane of embryonated chicken eggs and African green monkey kidney and grows typically after 5 to 7 days of inoculation. The distinct cytopathic effect of LSD and intra-cytoplasmic inclusion bodies differentiate it from those of Bovine herpesvirus 2 which causes pseudo-lumpy skin disease. The differential diagnosis in the case of LSD is mainly pseudo-LSD induced by bovine herpesvirus 2 which is a milder disease compared to the former and also causes cutaneous lesions however those are limited to only the epidermis and produce a scab after sloughing and systemic symptoms do not occur. Other differential diagnoses include, but not limited to dermatophytosis, Bovine farcy, Cowpox and demodicosis.

Serological tests for the detection of LSD have also been utilised including Indirect Fluorescent Antibody Test (IFAT), Enzyme Linked Immunosorbent Assay (ELISA) Western blotting and viral neutralisation. Viral naturalisation is by far the most specific and effective test, more so that agar gel diffusion or IFAT along with western blotting which is specific but rather expensive and difficult. Neutralising antibodies occur about 3 to 4 days after the onset of pyrexia and maximum titer levels are reached in about 2 to 3 weeks. Molecular detection methods are also employed for monitoring the spread and controlling of disease outbreaks and are another most often utilized way of diagnosis is detection of viral DNA using conventional or real-time polymerase chain reaction (PCR) specifically a *Capripoxvirus* specific PCR approach using blood, tissues and sperm material. Another possibility is via the demonstration of characteristic Capripoxvirions in biopsy material or dried crusts using transmission electron microscopy.

Control measures

In the absence of any treatment for LSD, the clinical approach rests on reducing inflammation and prevention of subsequent secondary bacterial infections. Use of antibiotics and anti-inflammatory drugs only helps with symptomatic treatment and effective control measures should be put in place to stop the transmission of disease to unaffected hosts.

- The transmission of LSDV us contributed to its vectors therefore it is imperative to restrict vector movements. Frequent and diligent use of insecticides and vector traps can be effective for restricting the transmission and breaking the disease cycle.
- Isolation of infected animals in the form of quarantine so their exudates do not contribute to the spread of the virus and restricting their movements to prevent trans-boundary spread is also important.
- Timely long-term immunisation of cattle with 100% coverage against prevalent strains of LSDV should be undertaken. These vaccines are widely available and are the most effective prevention measure for this disease.

Impact on Economy

The financial impact of LSD is incurred by farmers and herd owners who rely on animal by-products for revenue. The major economic setbacks of LSD are attributed to its high morbidity rates that translate to decreased milk production, abortion and infertility in both, males and females which may be either temporary or permanent, damaged hides, mastitis, emaciations etcetera. The industries most severely affected include the meat, milk and leather industries and because the quality of animal is also significantly affected in LSD, the overall trade of live animals is also affected and for its significant economic impact it has been declared as a notifiable disease by the WHO. The outbreak in India resulted in major economic losses where the state Rajasthan recorded a fall of 20% in total milk production and Gujarat reported decrease in daily milk collection amounting to approximately 1,00,000 litres per day.