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Popular Article

Significances of Lumpy Skin Disease in Cattle

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Abstract

Lumpy skin disease (LSD) is a contagious viral disease of cattle, buffaloes and wild ruminants which is caused by Lumpy skin disease virus. The disease is characterized by high fever, lymphadenitis, severe emaciation, bilateral epiphora, drop in milk production, sterility, skin erythema and development of nodules. A major route of transmission is an ulcerated skin lesion, while the virus can also be shed through bodily excretions and secretions. Hematophagic arthropods, such as biting flies, mosquitoes, and ticks, mechanically spread infection. Decreased milk production, abortions, temporary or permanent sterility, damage to the hide, and deaths will ensue, all of which causes significant economic loss to the country. Therefore, the most efficient method of reducing the spread and financial burden of lumpy skin disease is mass vaccination along with other suitable control measures.

Introduction

Lumpy skin disease (LSD) is a contagious and re-emerging transboundary viral disease of cattle, buffaloes and wild ruminants which is caused by Lumpy skin disease virus (LSDV), which belongs to genus Capripoxvirus, subfamily Chordopoxvirniae and family Poxviridae. The World Organization for Animal Health considers LSD as a notifiable disease since virus can spread globally and result in significant economic loss to the farmers. LSD are also be called as Neethling virus disease, exanthema nodularis bovis, Pseudo-urticaria and knopvelsiekte. The disease is characterized by high fever, lymphadenitis, severe emaciation, bilateral epiphora, drop in milk production, sterility, skin erythema and development of nodules (10–50 mm in size). The economic loss to the farmer is

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attributed by poor reproductive efficiency, loss in terms of hide quality, reduced milk yield and consistent degenerative emaciation (Amenu *et al.,* 2018). Usually the mortality rate is low (1–5%), the morbidity rate can be as high as 100% (Casal *et al.,* 2018). The morbidity rate varies according to the immune status of animals and frequency of mechanical vectors.

Host Range

The most vulnerable host for LSD is cattle and buffalo, both significantly gets affected irrespective of species, age, gender and breed but few reports suggest that *Bos taurus* is fairly more predisposed to the development of LSD than *Bos indicus*; younger animals and immunocompetent animals are also predisposed to LSD. Under normal circumstances, wild animals are invulnerable to infection yet a clinical infection has been successfully produced by experimental introduction of contagion in certain animals like Thomson's gazelle, Giraffe, oryx, springbok, impala and Arabian oryx. Wildlife and feral animal population is believed to have an insignificant role in the propagation of this disease (Khan *et al.,* 2021).

Epidemiology

In 1929, LSD was first reported in Zambia (Gumbe, 2018). In 1988, First outbreak of this disease in Egypt was reported due to unrestricted movement of cattle from African countries (Salib and Osman, 2011). In 1989, the first confirmed transcontinental spread of LSD from the African to Middle-East Asian countries occurred when the disease was reported in Israel which is believed to be spread by *Stomoxys calcitrans* vector that migrated from Egypt (Yeruham *et al.,* 1995). In July 2019, the virus made its way into the South East Asian country among which Bangladesh first reported an epidemic of LSD where sixty-six cases were reported (Das *et al.,* 2021). On the 3rd of August 2019, China became the second country in Southeast Asia to have an epidemic where 65 animals were infected in the Ili Kazak region, near the border of Kazakhstan. In India, first outbreak of the disease was reported in Odisha state in the month of August, 2019, in monsoon season with high humidity and vector density.

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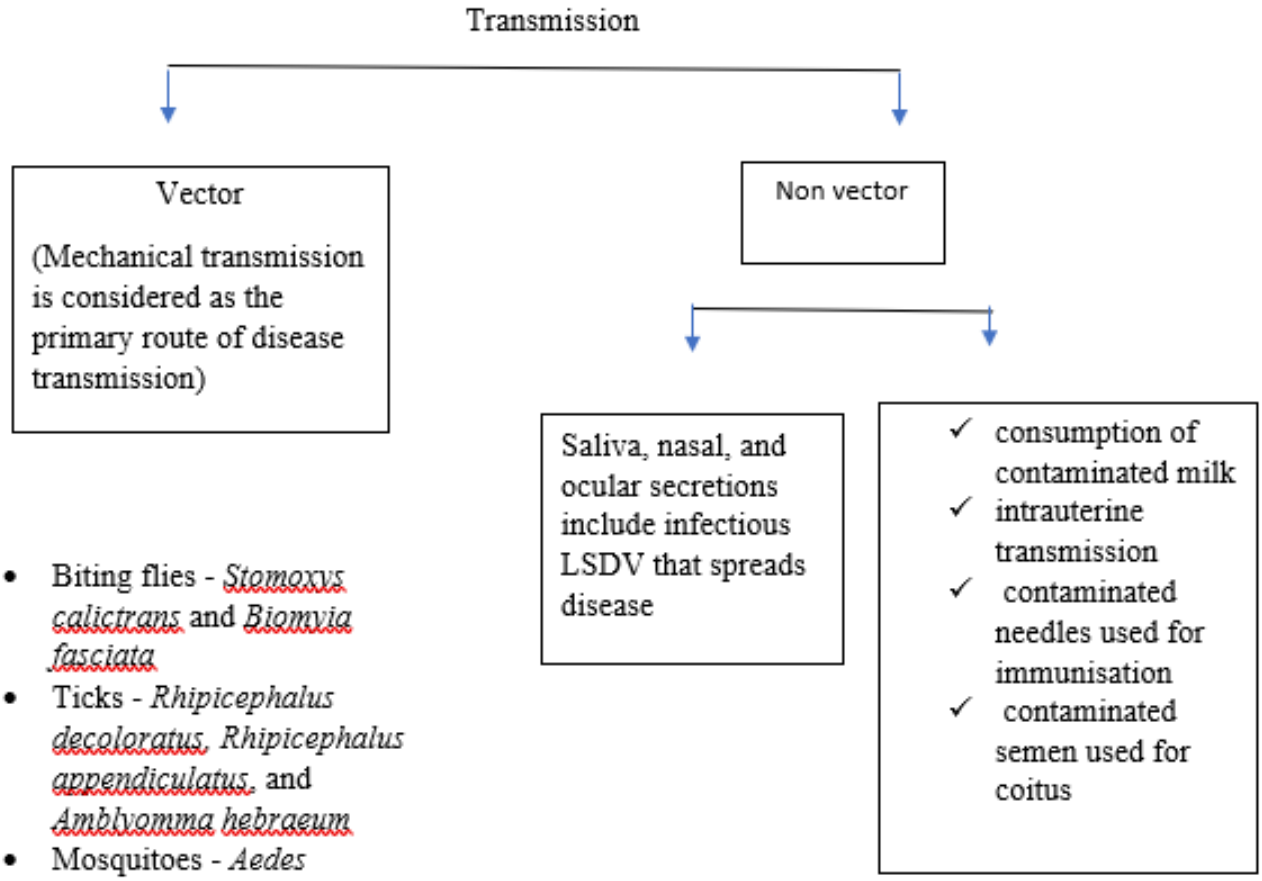
Transmission



(Gumbe, 2018; Tuppurainen *et al.*, 2017)

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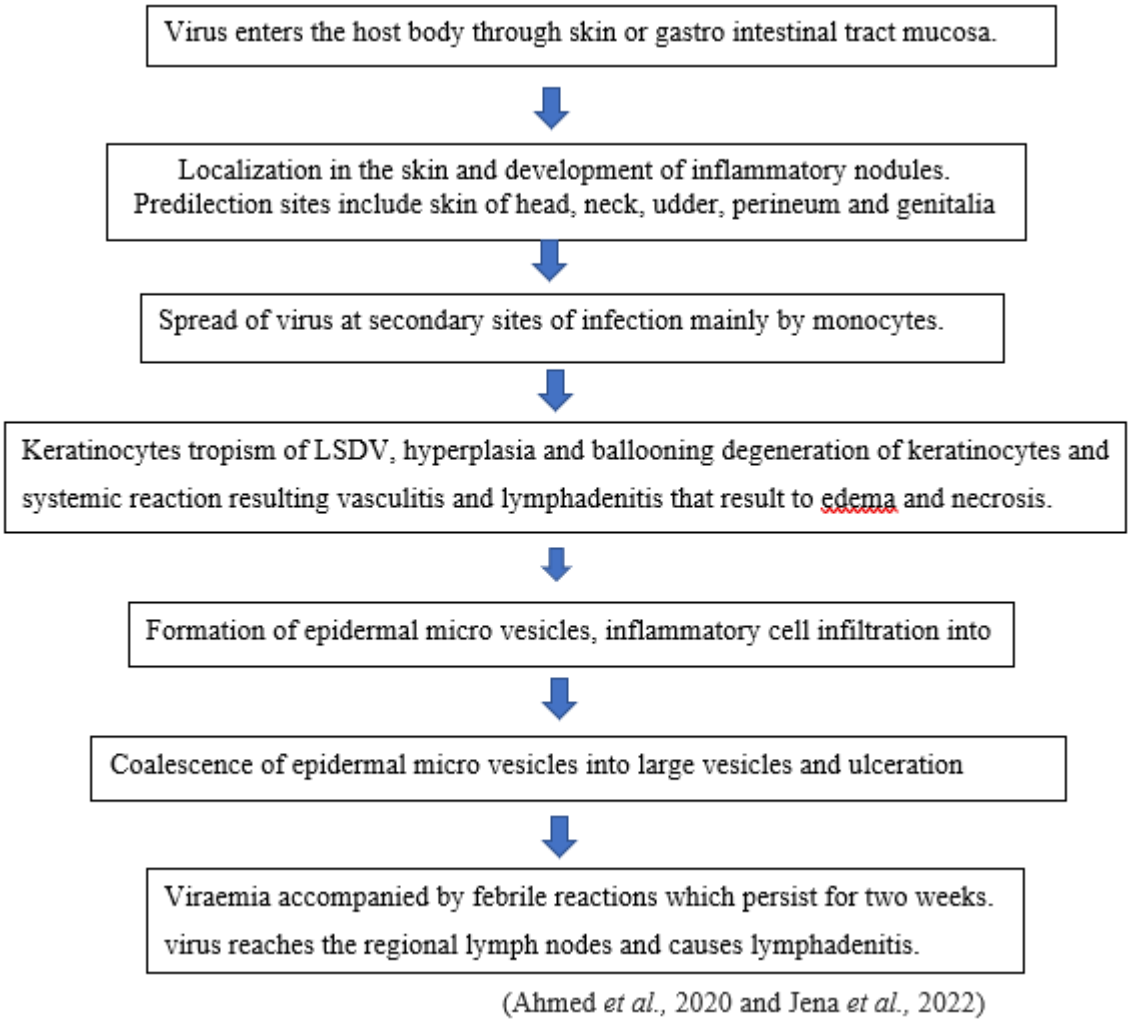
Pathogenesis



(Ahmed *et al.*, 2020 and Jena *et al.*, 2022)

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Large nodules may become necrotic and eventually fibrotic and persist for several months (sitfasts); the scars may remain indefinitely. Small nodules may resolve spontaneously without consequences (Gumbe, 2018; Lubinga *et al.*, 2014).

Clinical Signs

- The incubation period of disease in natural condition is between 2 and 5 weeks but in experimental condition, the duration ranges from 7 to 14 days (Gupta *et al*., 2020).
- Lumpy skin disease is clinically presented in three forms; acute, subacute and chronic form.
- In mild form of the disease, initially there is intermittent fever followed by development of 1–2 nodules on skin. Emaciation, agalactia, and epiphora are the most challenging symptoms to

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treat clinically. In later stages, erythema, pruritis, and nodular lesions are apparent on the skin. Painful lesions appear on the pubic, perineal, and oro-nasal mucosae.

- Severe form is characterized by emergence of hundreds of nodules all over the skin, lasting for about a week until they become firm and narrow haemorrhagic ring on the skin start to encapsulate them. The lesions continue to scab and may spread to other mucosae.
- The skin lesions harden and becoming necrotic after two to three weeks, making the animals uncomfortable and making them reluctant to move. The characteristic lesion "sitfast" may slough, leaving holes that could invite bacterial invasion and screwworm fly invasion, both of which could result in septicaemia (Abutarbush *et al.*, 2015; Constable *et al*., 2016).

Fig. 1. (left) Multiple skin nodules on the neck ; Open wound near coronary band (right)

Post Mortem Changes

- Extensive greyish-pink skin nodules with caseous necrotic centres
- Similar nodules may also be found in the nasopharynx, trachea, bronchi, lungs, rumen, abomasum, renal cortex, testicles and uterus
- Swollen, congested lymph nodes with petechial haemorrhage.

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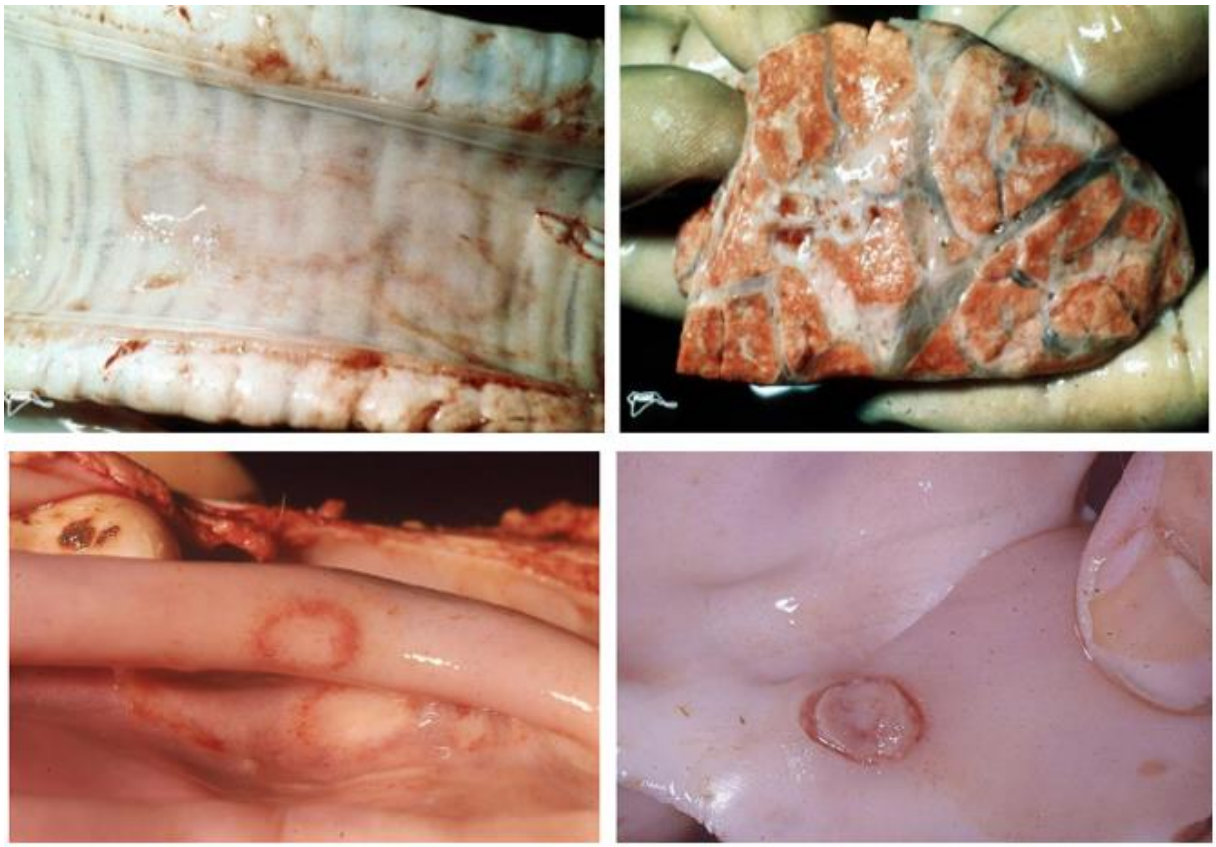

Histopathology (Skin and Lymph nodes)

- Eosinophilic intracytoplasmic inclusion bodies.
- Necrotic vasculitis in dermal arteriole with infiltration of neutrophils.
- Zenker's necrosis in the dermal muscles and mononuclear cells aggregation.
- Severe edema and infiltration of neutrophils (Jena *et al.*, 2022).

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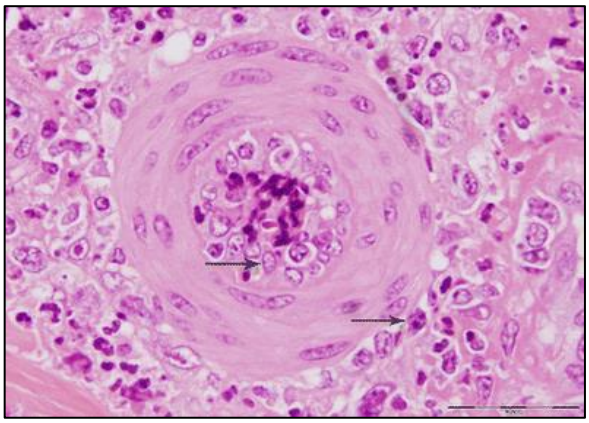

Fig. 3. Skin nodule: vasculitis indicated by the presence of inflammatory cells inside and around the blood vessels (*arrows*). H&E x 400

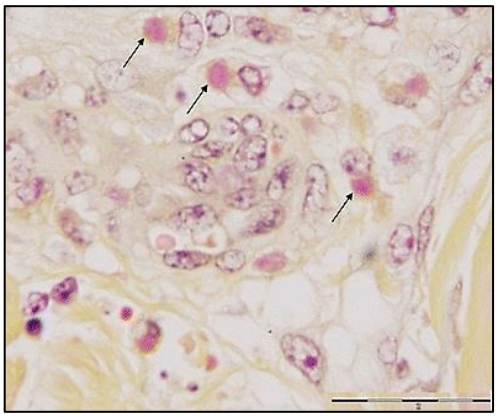

Fig. 4. Mononuclear cells displaying intracytoplasmic inclusions (*arrows*) Phloxine–tartrazine staining x 1000

Diagnosis

Lumpy skin disease (LSD) can be diagnosed based on peculiar clinical signs of the disease and the incidence of disease outbreak in nearby areas. However confirmatory diagnosis is furnished by laboratory tests with the presence of viral antigen or antibody (Amenu *et al*., 2018).

- Detection of viral DNA using the polymerase chain reaction.
- Electron microscopy
- Virus neutralization test
- Indirect fluorescence test
- Agar gel immunodiffusion test
- Western blot and
- Enzyme-linked immunosorbent assay. (OIE, 2016)

Differential Diagnosis

- Bovine herpes mammillitis (pseudo-lumpy skin disease)
- Bovine papular stomatitis (Parapoxvirus), Pseudocowpox (Parapoxvirus)
- Demodicosis
- Photosensitisation Urticaria
- Cutaneous tuberculosis
- Onchocercosis. (Tuppurainen *et al*., 2017)

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Economic Importance

The Office International des Epizooties (OIE) has designated Lumpy skin disease as a notifiable outbreak due to potential spread internationally and threat from agroterrorism (Abutarbush *et al.,* 2015). The economic effects of lumpy skin disease are much more indirect because it does not have a significant death rate. Considering its severe impacts on the health of the diseased animals, the overall economic effects are reflected in the trade of animals and their by-products due to reduced quality and quantity. Hence, huge economic losses have to be faced by the industries associated with livestock and its by-products e.g., meat, milk and leather industries. Beside other industries, the livestock farmers also have to face the economic crises incorporated by the LSD (Khan *et al.*, 2021).

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