# Lumpy Skin Disease - Epidemiology, Diagnosis, Prevention and Control

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Lumpy Skin Disease (LSD) is a viral disease affecting bovines caused by the Lumpy Skin Disease Virus (LSDV), a member of the Capripoxvirus genus. Initially endemic to Africa, LSD has expanded to the Middle East, Asia, and parts of Europe, primarily due to the spread of blood-feeding insect vectors, such as mosquitoes, flies, and ticks. The disease is characterized by fever, nodular skin lesions, and swollen lymph nodes, often resulting in reduced milk production, weight loss, and trade restrictions. Mortality rates are generally low, yet LSD causes significant economic losses due to decreased productivity and export limitations. The epidemiology of LSD is closely linked to environmental factors that support vector populations, with outbreaks peaking in warm, humid areas. Although all cattle breeds are susceptible, exotic high-yielding breeds often suffer more severe impacts than native cattle. Diagnosing LSD involves clinical examination and laboratory tests, primarily polymerase chain reaction (PCR) and serological assays, which help differentiate LSD from other similar diseases. Accurate and early diagnosis is crucial to controlling outbreaks. Prevention relies heavily on vaccination with live attenuated vaccines derived from Capripoxvirus, which offer effective immunity in endemic regions. Control measures include movement restrictions, quarantine, and vector control, such as insecticides and repellents, particularly in disease-free areas, to contain LSD spread. Ongoing surveillance and international collaboration are essential to manage LSD effectively, as the disease poses a substantial threat to global cattle industries.

***Keywords:*** *Transboundary disease, Vector-borne transmission, Vaccination, Livestock biosecurity, Global livestock trade*

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## Introduction

India's economy relies mainly on agriculture, and the allied livestock sector significantly impacts the country's Gross Domestic Product (GDP). In 2020-21, the contribution of livestock was 6.17 percent and 30.87 percent to national and agricultural Gross Value Added (GVA), respectively (Basic Animal Husbandry Statistics, 2022). However, the prevalence of various livestock diseases is detrimental to cattle productivity and leads to monetary losses. One such disease that has come to light in recent years is lumpy skin disease, which caused distress among the farmers. The LSD virus (Lumpy Skin Disease Virus - LSDV) (genus: *Capripoxvirus*; family *Poxviridae*) causes lumpy skin disease (LSD), a transboundary, vector-borne, non-zoonotic infection. The Capripoxvirus genus consists of the Goat pox virus (GTPV), Lumpy skin disease virus (LSDV), and Sheep pox virus (SPPV). During its expansion throughout African and Asian nations, lumpy skin disease was known by several other names, including "Pseudo-urticaria," "Neethling virus disease," "exanthema nodularis bovis," and "knopvelsiekte" (Abutarbush, 2017). The World Organization for Animal Health (WOAH Terrestrial Manual, 2024), which was formerly known as the Office International des Epizooties (OIE), has classified LSD as a "notifiable transboundary disease" due to its global spread. LSD is an infectious disease of economic importance. It affects cows, buffaloes, and some wild animals. Recently, LSD has been reported in Mithun (Reddy et al., 2024) and Yak (Reddy et al., 2023). Cattle of all age groups are affected, although the most susceptible are young animals and those nursing at their prime (Gupta et al., 2020). It reduces milk production, infertility, anorexia, emaciation, fever, and hide damage, all of which are of commercial value for dairy farmers.

## Genome

LSD has a double-stranded linear DNA genome approximately 150 kbp long and is believed to contain 156 genes. The LSDV genome consists of the primary coding region and two identical inverted terminal repeats (ITR), each having at least 2,418 base pairs (bp) at both ends. With 156 available reading frames, the LSDV has a 95% coding density. Proteins generated by these genes range from 53 to 2,025 amino acids long. It is thought that the six LSDV proteins that are known to be released contribute to the host's immune system suppression or modification. The Yaba virus, Dengue Virus (DV), Rift Valley Fever (RFV), Myxoma virus (MYX), and Swine pox (SPV) were among the recognized species whose genes and functions shared similarities with the LSDV Open Reading Frames (ORFs) (Gari et al., 2010).

## Economic impact

Asia produces 39% of the world's cattle and buffalo, with 300 million animals living in India alone. China and Pakistan follow with 90 million and 85 million animals, respectively. However, between 2022 and 2023, the Indian government estimated a loss of about 200 thousand heads of cattle due to LSD (Sreedevi B et al.2021). Nonetheless, the high morbidity of the disease has a higher economic impact than fatality. In bovines, it was calculated that the morbidity was 27.04% due to LSD. Severe underweight, damaged hides, mastitis, infertility in both sexes, reduced milk production, and miscarriages are the leading causes of the significant losses. According to a study, a loss of INR 18337.76 crores (USD 2217.26 million) in India was estimated due to the LSD outbreak in bovines totally from July 2022 to October 2022 alone (Gari et al., 2010).

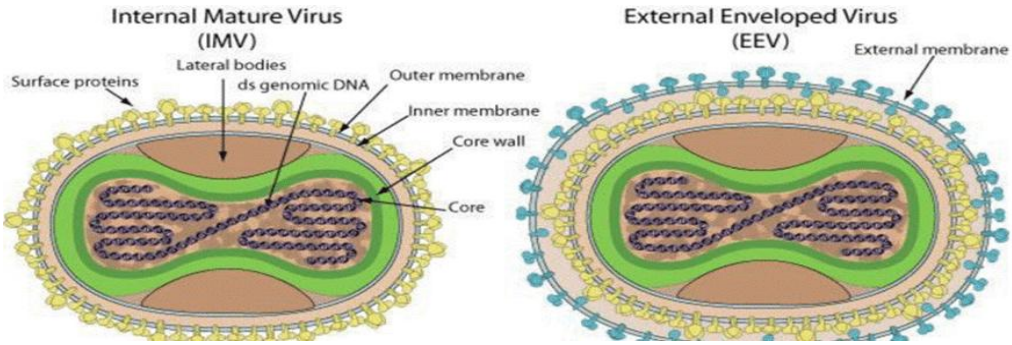


**Figure 1. Morphological structure of LSDV (Haftu et al., 2012)**

## Geographical distribution

The first reports of LSD occurred in Zambia in 1929. Cattle populations in other southern African nations were quickly affected by the disease in the 1940s. LSDV was restricted to sub-Saharan Africa until 1984, when its pathogenicity grew over time and resulted in devastating pandemics (Islam et al., 2016). Egypt and Israel saw the first LSD outbreak outside of sub-Saharan Africa in 1988 and 1989, and more occurrences in Kuwait, Bahrain, Yemen, the United Arab Emirates, and Sudan were reported. In 2006, cattle imported from the African Horn countries introduced LSD back into Egypt (Tuppurainen et al., 2011; Gari et al., 2010). Africa and Asia were among the nations with the highest incidence of LSD globally in 2016–2020, and the epidemiology fluctuated significantly over time. The disease is primarily found in Asia, Africa, the Middle East, and some parts of Europe. Still, it has also been reported from numerous other regions of the world, including Iraq (Tulman et al., 2001), Uganda, Nigeria, Saudi Arabia (Kasem et al., 2016), Ethiopia, India (Sudhakar et al., 2020), China (Lu et al. 2019), Egypt (Tassew 2018), Kazakhstan (Orynbayev et al., 2021), Bangladesh (Hasib et al., 2021), Nepal (Koirala et al., 2022), Myanmar (Maw et al., 2022), Thailand (Arjkumpa et al., 2021).

## Transmission

The regions where epidemics are prevalent include the Indian subcontinent, the Middle East, and Southwest Asia. Aerosols are the usual way viruses spread when susceptible animals and infected animals come into close or direct contact. Viruses typically gain entry to a host organism through the respiratory system (Babiuk et al., 2008). The disease can manifest several hundred kilometers from the initial outbreak locations in a brief timeframe. Although the movement of infected animals appears to be the primary mechanism for long-distance LSDV dispersal, clear seasonal patterns suggest that arthropod-borne transmission is more likely to cause the disease's rapid and aggressive short-distance proliferation. The role of different arthropod vectors in the transmission of LSDV is anticipated to vary depending on the local climate, season, humidity, and vegetation (Sprygin et al., 2018). Although any arthropod that feeds on proteins could be involved, interrupted feeders, which are common in the wild and regularly parasitize cattle, are the possible route of LSDV transmission, which includes flies - *Stomoxys calcitrans*, *Musca domestica*, mosquito - *Aedes aegypti*, and ticks – Rhipicephalus and Amblyomm (Tuppurainen et al., 2017). Other modes of transmission are also noted, namely:

**Transmission by direct contact**: The rate of transmission of LSD by direct contact is low and less efficient (Magori-Cohen et al.2012). Furthermore, no correlation was found between cattle population density and infection rate. (Reddy et al., 2022).

**Indirect transmission:** An indirect source of infection is the sharing of feeding and watering. Twelve to eighteen days following infection, traces of the virus were detected in nasal and oral secretions in a study (Li et al., 2023). A significant point in this transmission mode is that the manifestation of nodules in affected animals covers just about 25% of their skin. However, viral load in the nasal and oral mucous membranes is comparable to those found in skin lesions.

**Epidemiology**: Either legal or illicit migration of bovines is typically the cause of the initial outbreak in an unaffected area. Usually occurring during damp and humid conditions, outbreaks coincide with an abundance of vector populations. The infected animal recovers from the infection as a sequela, and there is no scientific proof that the recovered animals have a carrier status. Even though sheep and goats live alongside cattle and buffalo in rural households, it is yet unknown if small ruminants serve as LSDV reservoirs (Reddy et al., 2022).

**Risk factors:** The feeding behavior of cattle is significantly impacted by temperature. Cows do not require protection from the cold or warmth in tropical and subtropical climates. They can continue to graze outside all year long freely, which significantly raises the risk of coming into touch with bloodsucking insects. Because of their diverse range of activity, bloodsucking insects carry the virus and disseminate it further after biting sick cattle. Arthropods are also better equipped to thrive and survive in warmer temperatures. Arthropods are far less common and mostly found indoors in temperate zones, where winter temperatures are low. Cattle are also kept indoors to keep them warm and shield them from the cold. Therefore, the limited host and vector activity spectrum is not conducive to disease emergence and dissemination in temperate locations (Gari et al., 2010).

**Histopathological identification:** Lower lip and muzzle revealed healing necrotic scab infections. The tongue's dorsal surface displayed enhanced necrotic scab sores. Severe congestion, haemorrhages, oedema, and necrosis were observed in the skin and muscle subcutaneous tissue. Internal organs showed septicaemic lesions. Multiple areas of consolidation with pneumonic lesions were seen in the lungs. The trachea displayed necrotic, confined, raised nodular lesions, congested mucosa, and frothy exudate in the lumen. Diffuse, many circumscribed, high hyperaemic, and necro-haemorrhagic ulcerative lesions were observed on the serosal surface of the rumen, reticulum, and abomasum (Reddy et al., 2024).

**Diagnosis:** Diagnosis is crucial for early control, prevention, and treatment of a disease. Early detection of LSDV is challenging because the symptoms are not readily apparent. Clinical symptoms, such as the development of skin nodules, particularly in the neck, back, legs, and other body regions, do not show until later in the disease. The illness may worsen and eventually kill the animal in its later stages. As a result, it becomes imperative to identify the illness as soon as feasible. A range of laboratory methods, including gross pathology, histology, immunohistochemistry, PCR, virus isolation, SNT, IFAT, indirect ELISA, and clinical symptoms, are employed in the diagnosis of LSD (Seerintra et al., 2022; Odonchimeg et al., 2022; Tuppurainen et al., 2017). A veterinarian can identify the disease by observing the clinical indications and laboratory diagnosis.

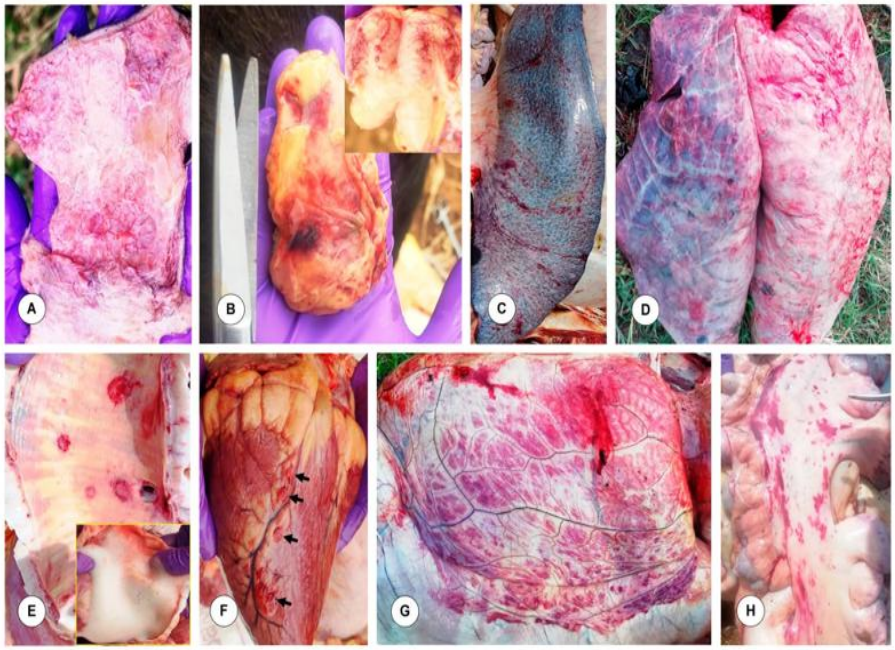


**Figure 2. Post-mortem lesions in an early stage of LSD. (A) Subcutaneous tissue showed congestion, haemorrhages, oedema, necrosis, and serous atrophy of fat with a yellow gelatinous appearance. (B)Enlarged, congested, and haemorrhagic prescapular lymph node. (C) Enlarged spleen with haemorrhages in the serosa. (D) Lungs showed congestion, hepatisation, and prominent interlobular septa. (E) Fresh haemorrhagic ulcers on tracheal mucosa with frothy exudate (Inset). (F) Necrohaemorrhagic epi-carditis. (G) The serosal surface of the rumen showed haemorrhages. (H)Mesentery showed haemorrhages (Reddy et al., 2024).**

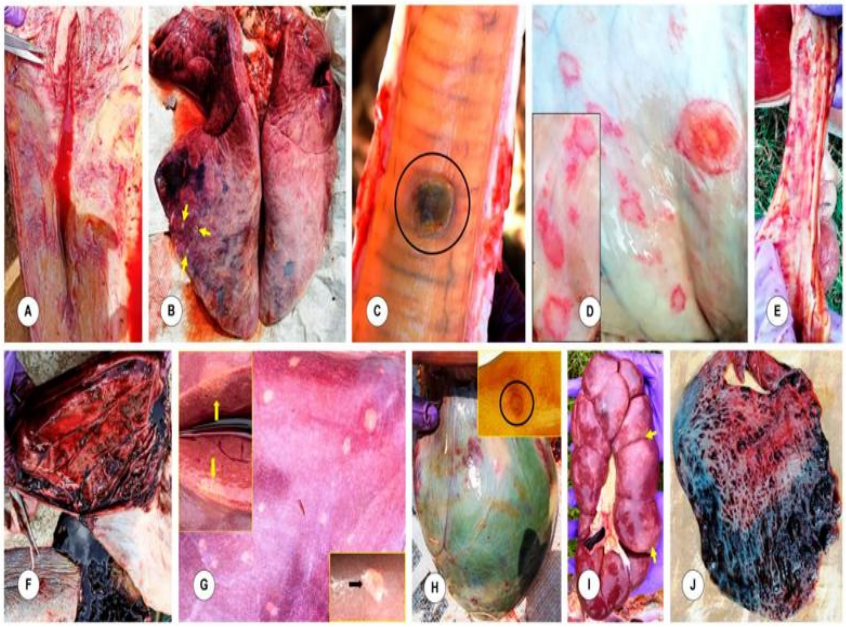


**Figure 3. Post-mortem lesions in mid-stage of LSD. (A) The skin's subcutaneous tissue showed congestion, haemorrhages, and necro- sis with sero-sanguinous edematous fluid. (B) Lungs showed congestion, haemorrhages, focal pneumonic lesions, and multiple circumscribed nodular lesions(arrow). (C) The tracheal mucosa showed necrotic circumscribed nodular lesions. (D) Multiple**

**circum-scribed elevated hyperaemic necro-haemorrhagic lesions on the serosal surface of the rumen (Inset). (E) The small intestine showed congestion and diffuse haemorrhagic ulcerative lesions in the mucosa. (F) Congestion and haemorrhages on the mucosal surface of the abomasum. (G) The liver showed necrotic foci (yellow arrow) and circumscribed nodular lesions (black arrow). (H) Markedly dis-tended gallbladder with serosal haemorrhages and ulcerative lesions in the mucosa (Inset). (I) Multifocal necrotic lesions on the surface of the kidney (arrow). (J) The mucosa of the urinary bladder showed severe haemorrhages. (Reddy et al., 2024).**

**Clinical signs:** Initially, it was believed that LSD was caused by an allergic reaction (urticaria) in cattle because of the nodule-like appearance of the clinical manifestations. As a result, the illness was given the moniker "Pseudo-urticaria." The first and simplest clinical sign to recognize is the development of nodules in various body areas. These hard, round skin nodules, measuring approximately 2.5–5 cm in diameter, appear on multiple body areas, including the head, neck, limbs, and udder. Large, necrotic nodules may develop from these, and eventually, they will transform into fibrotic tissue. It raises the possibility of developing myiasis (Tuppurainen et al., 2017).

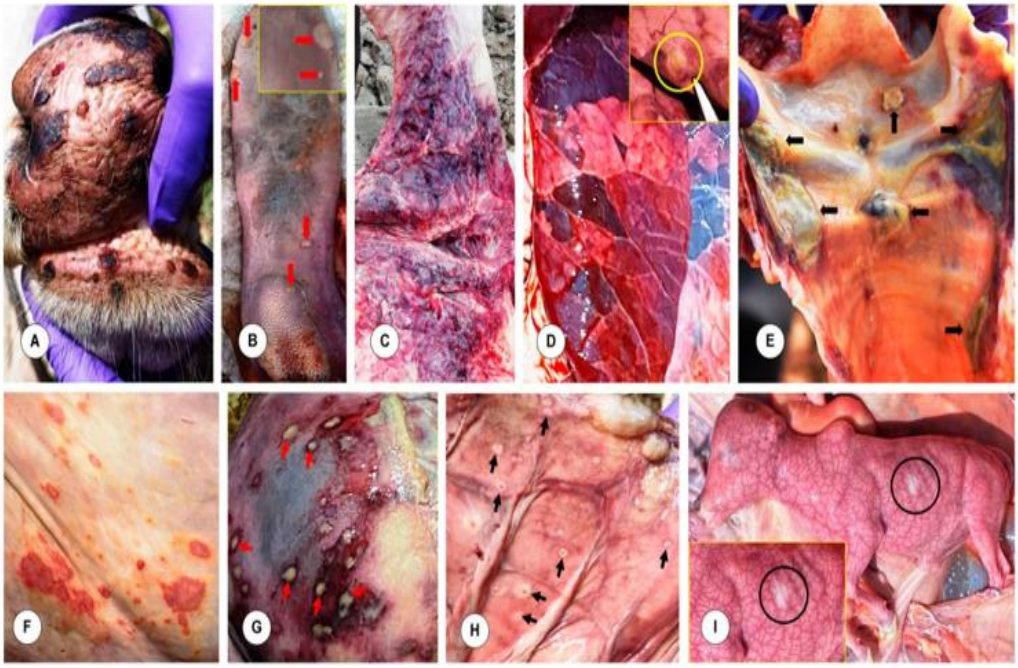


**Figure 4. Post-mortem lesions in late-stage of LSD. (A) The muzzle and lower lip showed necrotic scab lesions with healing. (B) The dorsal surface of the tongue showed elevated necrotic scab lesions (arrow). (C) Subcutaneous tissue and muscle showed severe congestion, haemorrhages, oedema, and necrosis. (D) Lungs showed circumscribed nodular scab lesions (Inset) and a marbling appearance due to prominent interlobular septa, severe congestion, haemorrhages, and pneumonic lesions. (E) Upper tracheal mucosa showed necrotic circumscribed elevated nodular lesions (arrow). (F) Multiple circumscribed elevated hyperaemic necro-haemorrhagic ulcerative lesions on the serosal surface of the rumen. (G) Diffuse haemorrhages with necrotic ulcerative lesions in the serosal reticulum surface (arrow). (H) Circumscribed ulcerative lesions in the mucosal surface of the abomasum (arrow). (I) An aborted foetus showed severe congestion of cutaneous blood vessels and necrotic lesions in the skin (Inset) (Reddy et al., 2024).**

**Sample processing:** The initial and crucial stage in laboratory diagnosis is isolating and identifying viruses through gathering clinical samples. The collected samples must be handled carefully and preserved appropriately for future use. After being gently mixed and processed for virus isolation, the blood samples containing anticoagulant with a buffy coat should be immediately placed on ice to maintain the virus's specificity. If processing takes 48 hours, clinical materials like tissue and serum should be stored at -20 ºC. Clinical samples of adequate size should be transported across long distances without refrigeration if the transport medium contains 10% glycerol. It prevents transport media from penetrating the biopsy's core region (OIE Terrestrial Manual, 2018).

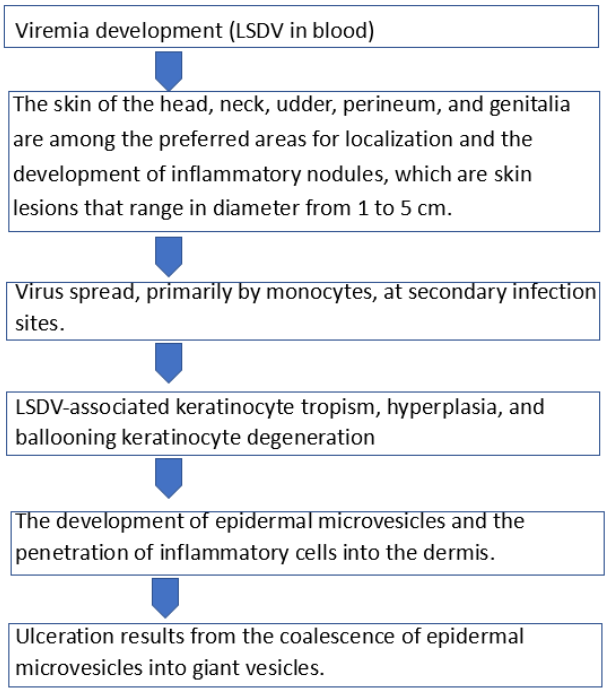


**Figure 5. Flow chart of pathogenesis**

**Virus isolation:** Typically, cell cultures of bovine (cattle), ovine (sheep), or caprine (goat) origin are employed for LSDV propagation. The virus is isolated using primary lamb testis (PLT) and Madin-Darby bovine kidney (MDBK). Mediums such as Dulbecco's Modified Eagle Medium (DMEM) or Glasgow's Minimum Essential Medium (GMEM) supplemented with 10% fetal calf serum and antibiotics are employed for cell growth. As the virus multiplies, its cytopathic effect (CPE) and intracellular inclusion bodies are examined. The CPE develops for 9 days during primary isolation (Sanz-Bernardo et al., 2020).

**Method of microscopic detection:** Capripox virions can be identified with a transmission electron microscope because they resemble bricks and are roughly 270 - 290 nm in size. However, the method is unreliable since it is hard to distinguish the orthopoxvirus that causes buffalo pox from the Capripoxvirus (Parvin et al., 2022; Roche et al., 2021). Immunohistochemistry using F80G5 monoclonal antibody specific for Capripoxvirus ORF 057 has been reported to detect LSDV antigen in the skin of experimentally infected cattle (Chihota et al., 2001).

**Prevention and control**

One of the most important aspects of LSD prevention is vector control. Repellants or pesticides are sprayed in cow sheds to control vector population, and following good managemental practices to reduce the number of vector breeding places is among other measures taken to manage their population. To clean and disinfect the harmed regions and fomites, use the appropriate chemicals (Gentamicin (@5mg/kg B.Wt. intramuscularly), Meloxicum (@0.2mg/kg B.Wt.). The carcasses of the afflicted animals should be disposed of carefully (Milovanović et al., 2019).

**Molecular detection:** The most simple, fast, and affordable technique for LSDV detection is the conventional polymerase chain reaction. The P32 envelope protein gene and fusion protein gene are the two primers most frequently used for PCR amplification. About 192 bp comprise the P32 envelope protein gene

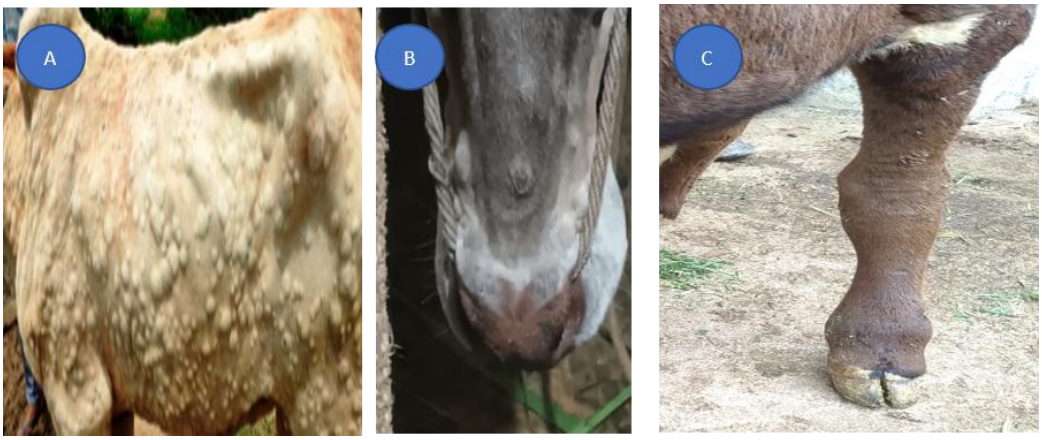


**Figure 6. Shows the affected cattle. Skin nodule/scab lesions in early stage (A); Nasal discharge from the affected cattle (B); Lymphadenopathy(C)**

fragment, LSDV074. Real-time PCR (Chihota et al., 2001) and Restriction fragment length polymorphism (RFLP), among other molecular approaches, are also employed for quick and accurate identification of LSD. Another extremely sensitive and specific detection method for on-site LSD detection has been reported. It combines CRISPR-Cas12a fluorescence (RPA-Cas12a-fluorescence) with recombinase polymerase amplification utilizing the orf068 gene (Bowden et al., 2008). To effectively restrict the spread of this transboundary disease, the CRISPR-powered technology provides a novel diagnostic tool for portable, ultra-sensitive, rapid, and adaptable LSD disease screening (Menasherow et al., 2014). A virus neutralization test (VNT) of test sera titrated against a constant dilution of the standard viral strain is used to assess the neutralization index. Less employed methods for LSDV detection are Western blotting and Agar gel immunodiffusion (AGID). AGID has a limited specificity, and western blotting is time-consuming and costly. The Immunoperoxidase Monolayer Assay (IPMA) is used to detect lumpy skin disease antibodies. It shows high sensitivity and specificity when compared with VNT and ELISA. Furthermore, LSDV-IPMA was able to detect the LSDV antibodies earlier in infected, vaccinated, and vaccinated/infected animals, in comparison to other methods. (Jiang et al., 2022). Recent studies with CRISPR/Cas12 on Gold nanoparticles were used to detect LSDV. These are the new recent techniques for the detection of LSDV. Advancements in Biosensing play a vital role in the rapid and accurate diagnosis of LSDV and Veterinary Research (Liao et al., 2022).

**Therapeutic intervention**

Lumpy skin disease has no effective treatments, similar to most viral infections. Supportive care is the only mode of treatment. Antibiotics are administered to infected animals to prevent secondary bacterial infection, while other therapeutic medications are used to alleviate clinical symptoms. Antibiotics and local dressing are used to treat ulcerated lesions on the epidermal layer to keep insects like flies away (Haegeman et al., 2020).

**Vaccination**

Vaccination is the primary strategy for both preventing and managing LSD. Current vaccines for the lumpy skin disease outbreak include the Kenyan sheep and goat pox strain (KS-1), the South African Neethling strain, the Romanian sheep pox strain, the Yugoslavian RM 65 strain, and the South African strain (Ahmed et al., 2020). It must be safe to use LSD immunizations on cattle of various ages, genders, breeds, and species. GTPV and SPPV immunizations in cattle generally have no adverse effects. However, when cattle were given a higher dosage of vaccination based on the RM65 SPPV strain, some mild adverse reactions were seen. In Israel between 2006 and 2007, the Yugoslavian RM 65 (Ramyar) sheep pox immunization proved to protect cattle only partially against LSD. Recently, GTPV vaccines against LSD have been implemented with promising results in Bangladesh and India (Tuppurainen et al., 2021).

**Conclusion**

Lumpy skin disease, caused by a member of the Poxviridae family, is a highly contagious viral disease that affects cattle and buffaloes. Even though the fatality rate is low, preventive measures should be taken to stop the disease's spread because it harms the dairy industry and causes financial losses. Reducing the incidence of LSDV may be possible with a comprehensive understanding of the various ways the virus spreads. Since there is no known treatment for LSDV, vaccination is the only way to avoid contracting the disease.

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