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(REVIEW ARTICLE)

A Systematic Review on Lumpy Skin Disease

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Abstract

Lumpy skin disease (LSD) results in significant financial setbacks for the livestock sector. This condition is instigated by the Lumpy skin disease virus (LSDV), a member of the Poxviridae family, and the Neethling strain serves as its reference or prototype. LSDV is a member of the Capripoxvirus genus, which also includes the sheep pox and goat pox viruses. LSD is an enzootic, contagious, eruptive, and seldom lethal illness of cattle defined by skin nodules. The only impacted animal species are cattle and water buffalo, which have significant morbidity rates but minimal death. But calf mortality rates are greater. LSD impairs the production of milk and beef, results in female miscarriages, and makes males sterile. The initial outbreaks of Lumpy skin disease (LSD) were first documented in Zambia in the year 1929. LSD's origins date back to 1929 in Zambia. In Africa, LSD is seen as an endemic illness. In 1984, the illness was spread outside of Africa. It has been documented in Madagascar and a few Middle Eastern and Arab Gulf countries. Recent reports of the sickness in LSD-free nations (Jordan, Syria, Lebanon, Turkey, Iran and Iraq) with possible financial harm to the cattle sector. Both the World Organization for Animal Health (OIE) and the Food and Agriculture Organization (FAO) have issued warnings regarding the potential for the transmission of diseases to result in substantial economic repercussions. This disease diminishes the milk production of cows due to the presence of oral ulcers, which weaken the animals and cause a loss of appetite. The objective of this review article is to examine Lumpy skin disease (LSD) in the context of the current situation, particularly in relation to the growing concern about the disease spreading to countries that have thus far remained LSD-free.

Keywords: Lumpy Skin Disease Virus (LSDV); Nodules; Infection; Poxvirus

1. Introduction

Lumpy skin disease is an infectious disease. It is caused by Lumpy Skin Disease Virus (LSDV) which belongs to family Poxviridae, genus Capripoxvirus. Antigenically, it is very similar to the viruses that cause sheep and goat pox. Routine serological tests, however, are unable to distinguish between these viruses [1]. Cattle and water buffalo are affected by LSD. It is a vector-borne disease spread by various biting and biting arthropods that feed on blood. Emaciation, harm to hides, infertility, mastitis, a reduction in milk output, and mortality of up to 20% all contribute to significant economic losses caused by LSD. Depending on the host cattle breed and capripoxvirus strain, the severity of the clinical indications of LSD varies [2]. Until 1989, only the African Continents were affected by lumpy skin condition. However, the illness spreads from Africa to Madagascar and the Middle East, where it severely damages the livestock business. Lesions start to show up at the inoculation site in 4 to 20 days following the estimated two to five week incubation period in the field. The first symptom is a fever, which is quickly followed by the growth of nodules on the skin and mucous membranes within two days [3]. Based on the normal clinical patterns (morbidity and mortality), an LSD diagnosis is constructed. Transmission electron microscopy (TEM), immunoperoxidase (IMP) staining, an ELISA test for antigen-tracking, and a PCR test are all used to confirm a diagnosis. Treatment for LSD is not specific. However, supportive care should be provided to infected animals to alleviate clinical symptoms and manage any secondary problems. In South Africa,

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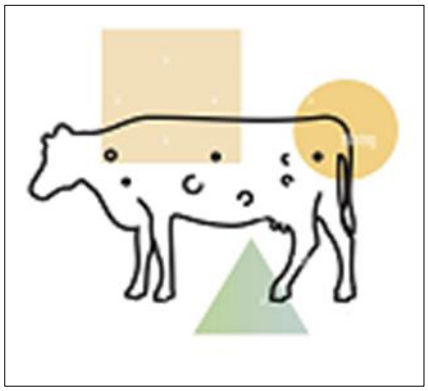
immunisation of susceptible animals is the most efficient means of disease prevention, and the Neethling strain virus is used to make the vaccines [4].



2. The Causative Organism

Lumpy skin disease is brought on by the genus Capripoxvirus, which belongs to the Poxviridae family. According to Woods (1988), the sheep and goat poxviruses and lumpy skin disease virus (LSDV) share a close antigenic relationship. Routine serological testing are unable to distinguish between these three viruses, despite their substantial differences (Figure 1). LSDV is vulnerable to temperatures of 65°C/30 minutes and 55°C/2 hours. It can be extracted from skin nodules and stored for ten years at -80 °C. At 4°C, the contaminated tissue culture fluid can be kept for six months. The virus can survive in extremely acidic or alkaline pH conditions. When maintained at pH 6.6-8.6 for 5 days at 37°C, however, there is no appreciable decrease in titre. Ether (20%), chloroform (1%), formalin (1%), and various detergents, such as sodium dodecyl sulphate, are all vulnerable to LSDV. Furthermore, it is vulnerable to phenol (2%/15 min), sodium hypochlorite (2-4%), iodine compounds (1:33 dilution), Virkon® (2%), and quarternary ammonium compounds (0.5%). The LSDV is highly stable, enduring for extended durations at room temperature, particularly in dried scabs. To be inactivated, LSDV is extremely resistant. It can last up to 33 days or more in necrotic skin nodules, 35 days or month. It has the potential to persist in the environment for an extended duration. It has a very long environmental shelf life. The virus can endure for several months in gloomy settings like contaminated animal shelters, while being susceptible to sunlight and detergents with lipid solvents or in desiccated crusts, and at least 18 days in air-dried hides. It is discovered the LSDV's genomic sequence [5]. The 156 putative genes found in the LSDV genome (151 kbp) are located in the core coding region, which is surrounded by identical 2.4 kbp-inverted terminal repeats. The chordopoxviruses of other genera, however, show 146 conserved genes, encoding proteins involved in virion structure and assembly, nucleotide metabolism, DNA replication, protein processing, transcription and mRNA synthesis, viral pathogenicity, and host range. In particular, suipoxvirus, yatapoxvirus, and leporipoxvirus genes have a significant degree of colinearity and amino acid identity (average of 65%) with genes of other known mammalian poxviruses. Poxvirus homologues are either absent or share a reduced level of amino acid identity (on average 43%) in the terminal sections, where colinearity is disturbed. Although the gene structure and content of LSDV are similar to leporipoxviruses, it also contains homologues of other poxvirus genera' interleukin-10 (IL-10), IL-1 binding proteins, G protein-coupled CC chemokine receptor, and epidermal growth factor-like protein. Although the LSDV is closely linked to other Chordopoxvirinae members, it has a distinct set of genes that determine the viral host range and pathogenicity. Several capripoxviruses, including LSDV [5], sheep poxvirus, and goat poxvirus [6], have had their whole genome sequences published.

2.1. Virology

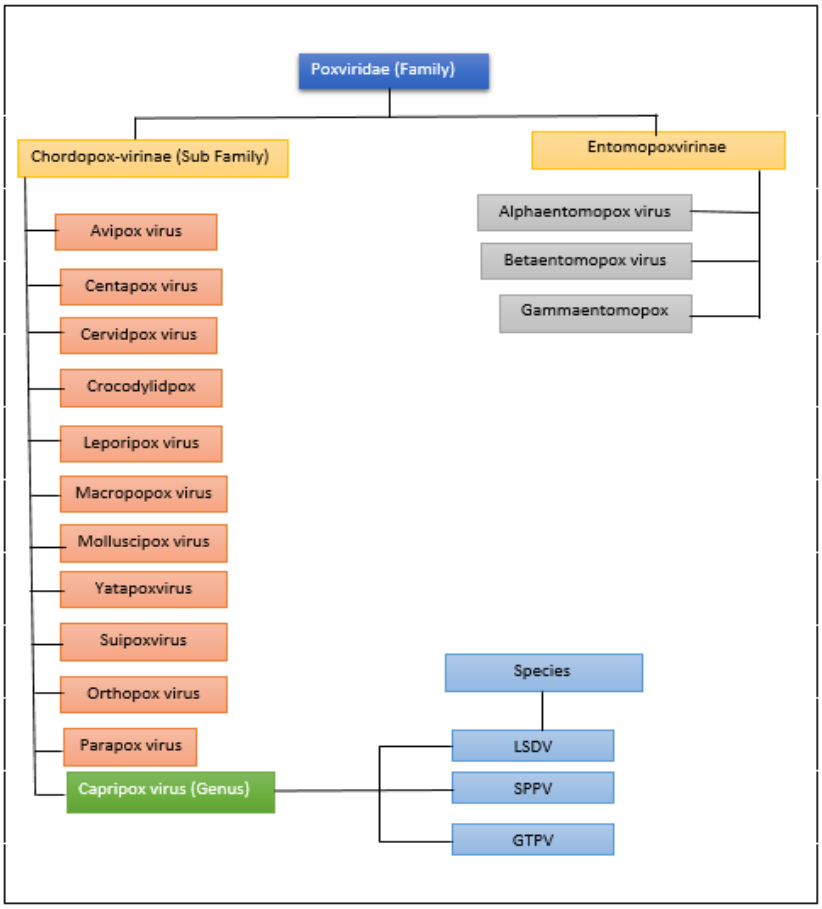


Figure 1 Taxonomy of LSDV

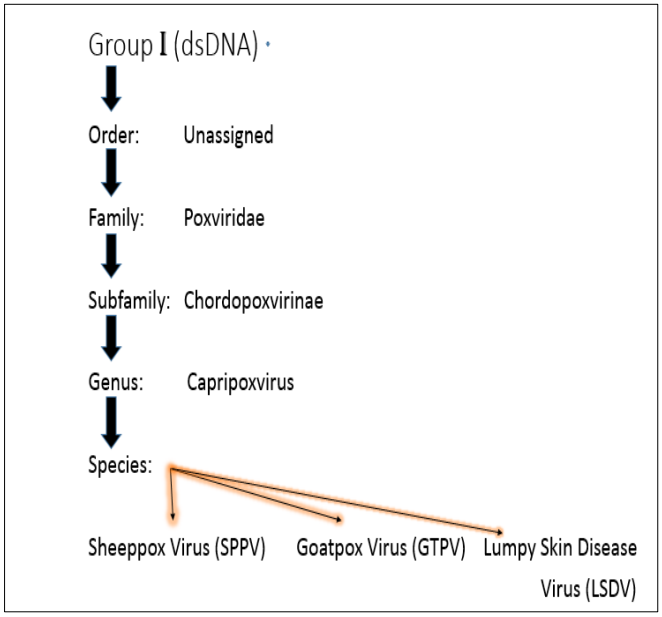


Figure 2 Classification of Lumpy skin disease virus

3. Epidemiology and historical outbreaks

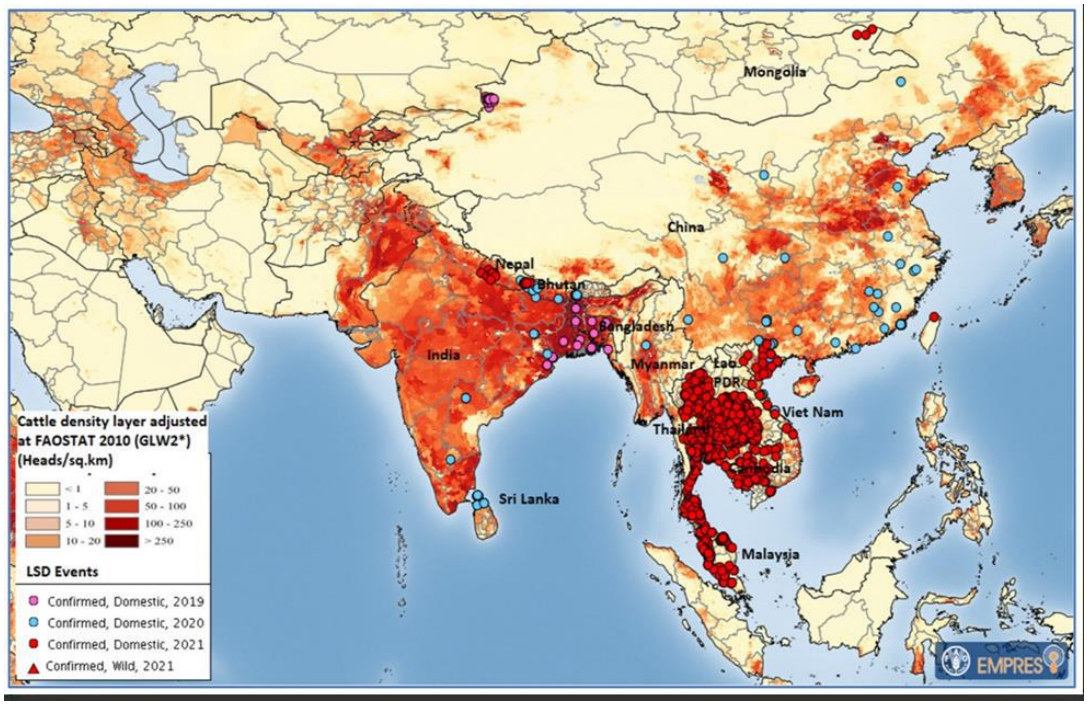


Figure 3 LSD outbreaks in Asia from 2019 to 2021 (map source: FAO Emergency Prevention System Global Animal Disease Information System data).

3.1. Mortality and Morbidity rates

The death and morbidity rates associated with LSD outbreaks vary enormously. Geographical location, climate, management practises, animal nutrition and overall health, afflicted breed of cattle, immune state, population sizes, and distribution of potential insect vectors in different habitats, as well as virus virulence, are some of the variables that may affect the outcome. LSD has a morbidity rate that can range from 5 to 45%. However, 1 to 5 percent morbidity rates are thought to be more typical. Epizootics in Southern, West, and East Africa as well as the Sudan have seen higher rates, yet it's possible that the same epizootic might also have far lower rates. Additionally, in 2009, Oman reported high rates of illness and mortality in a farm population of Holstein cattle of 30-45% and 12%, respectively [7].

3.2. Susceptible animals

Narrow vertebrate host range for LSD. The species that naturally contract the disease in the course of field outbreaks are cattle and buffalo. Asian water buffalo Bubalus bubalis has been the subject of five clinical cases of LSD [8]. During field epidemics, no other domestic ruminant species gets sick naturally. The disease seems to affect all types of cattle equally. As opposed to native breeds with thicker skins, such as the Afrikaner and Afrikaner cross-breeds, imported varieties with thin skins, like the Bos taurus, Friesland cattle, and Channel Island breeds, were far more susceptible [9].

3.3. Transmission

According to (Weiss 1968, Kitching and Mellor 1986, Carn and Kitching 1995) [10,11,12], the method of lumpy skin disease virus transmission is not entirely known. The mechanical spread of the LSD virus has primarily been linked to flying insects, and every indication supports field findings that outbreaks of LSD happen when biting insects are most active. The majority of cases are thought to have been caused by arthropod vectors. However, extremely high morbidity rates that follow rain are prevalent in areas with large mosquito breeding grounds. In a recent investigation, even though the virus was detected in various arthropods, including mosquitoes (Anopheles stephensi, Culex quinquefascuatus), the stable fly, and a biting midge (Culicoides nebeculosis) after they had fed on cattle with lumpy skin disease, it was observed that the infection did not transfer to susceptible cattle when these arthropods were allowed to re-feed on infected cattle [10,13]. Infection has frequently been introduced through the transportation of animals from infected herds, frequently months after recovery. It is believed that ancient skin sores are where the virus originated. The disease has been noted to manifest itself across the majority of Sub-Saharan Africa after the seasonal rains. Different species of arthropods are constantly expanding in number [14].

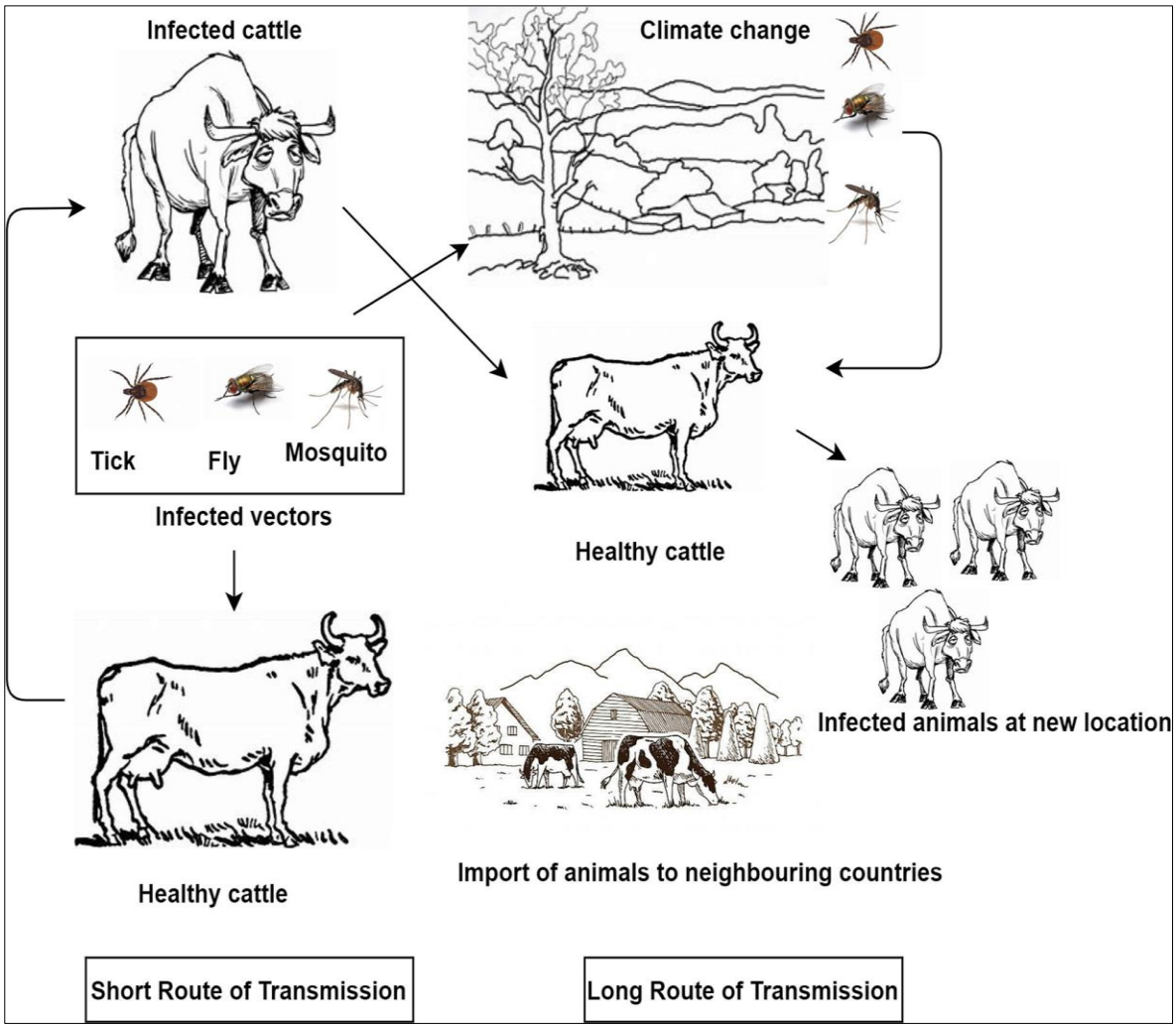


Figure 4 Summary of transmission of LSD virus [15]

4. Pathogenesis

Research on the pathogenesis of LSD in cattle is limited [16]. In the typical progression, it starts with viremia and fever, followed by the localization of the virus in the skin and the formation of inflammatory nodules [17]. After administering LSDV through subcutaneous or intradermal inoculation in cattle, localized swelling typically emerges between 4 to 7 days post-inoculation (DPI). This swelling can vary in size, ranging from 1 to 3 cm, and may cover approximately 25% of the skin surface. Enlargement of the nearby lymph nodes and a widespread eruption of skin nodules typically occur between 7 to 19 days post-inoculation (DPI) in cattle following LSDV exposure. Viremia and low levels of viral shedding in oral and nasal secretions can be detected between 6 and 15 days post-inoculation (DPI) and 12 and 18 DPI, respectively, following the febrile reaction in cattle. LSDV has also been observed in saliva, semen, and skin nodules for a minimum of 11, 42, and 39 days, respectively, after the onset of fever in cattle[18,19]. Viral replication in various cell types such as macrophages, fibroblasts, pericytes, endothelial cells, and potentially other cells within the walls of blood vessels and lymph vessels can lead to vasculitis and lymphangitis in specific vessels in affected areas. In severe cases, this can result in thrombosis (blood clot formation) and infarction (tissue death due to a lack of blood supply) [18]. In cases of natural infection, it appears that calves in their early stages, cows that are currently producing milk, and animals suffering from malnutrition tend to experience more severe illness, potentially because their humoral immunity may be compromised. Antibodies were observable at 21 days post-infection (DPI) through serum neutralization testing [20]. The immunity obtained after recovering from a natural infection is lifelong. Calves born to immune cows acquire maternal antibodies and remain resistant to clinical disease for approximately six months [19, 21]. In due course, animals that have been affected by the disease clear the infection, and there is no recognized carrier state for LSDV (Lumpy Skin Disease Virus) [22].

5. Clinical Manifestations and Pathology

5.1. Clinical manifestations

The period between inoculation and the initial observation of widespread clinical symptoms varies from 7 to 14 days in cattle subjected to experimental infection, regardless of the infection route, and from 2 to 5 weeks in cases of natural infection [21,23]. Lumpy Skin Disease (LSD) can be categorized into mild and severe forms depending on factors such as the quantity of lumps (nodules), the presence of complications, the dosage of the inoculum, the susceptibility of the host, and the density of the insect population. The clinical manifestations of mildly affected cattle include the emergence of one or two lumps (Figure 5.A) or nodules, typically within 2 days after the onset of fever. These lumps or nodules are usually 1 to 5 cm in diameter. Additional signs may encompass feelings of sadness, diminished appetite (referred to as anorexia), heightened salivation, secretions from the eyes and nasal passages, insufficient milk production (known as agalactia), and an overall state of emaciation. Additionally, painful and red nodular lesions may be noticeable on various parts of the animal's body, particularly on the skin of the muzzle, nostrils, back, legs, scrotum, perineum, eyelids, lower ears, nasal and oral mucosa, as well as the tail [24]. In severe cases, which can endure for 7-12 days, the animal may exhibit continuous high fever (40-41.5°C), severe depression, loss of appetite (anorexia), and a distinct feature: numerous nodules, typically numbering more than hundreds, which are usually fairly uniform in size and distributed all over the animal's body (Figure 5.B) [10].

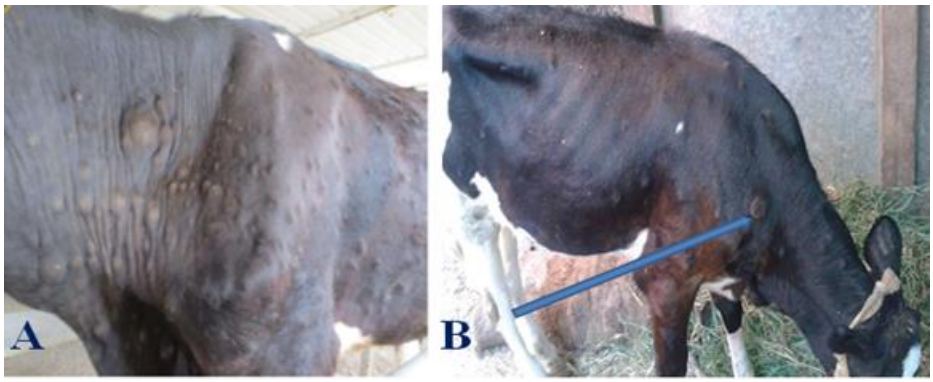


Figure 5 Distinctive LSD nodular lesions that signal the degree of severity: In severe cases, the lesions can spread across the entire body (A), while in milder cases of LSD, there may only be a few skin nodules (B), adapted from [25,26].

These nodules are firm in texture and slightly raised above the surrounding healthy skin. They are often separated from the normal skin by a narrow ring of hemorrhage. These nodules affect not only the epidermis and dermis but also extend into the adjacent subcutaneous tissue and even the underlying musculature. These nodules have the potential to resolve, but in some cases, they may persist as firm lumps or undergo changes, becoming moist, necrotic, and eventually sloughing off or developing into ulcers. Lesions where skin is lost may leave lasting marks and remain visible for extended periods. When these lesions merge or come together, they can create sizable areas of exposed raw tissue, which are vulnerable to infestation by screwworm fly larvae [17]. Once a lesion undergoes sloughing, it can give rise to a void that penetrates the entire skin thickness, displaying a distinctive attribute known as the "inverted conical zone" of necrosis, which is commonly recognized as a "sit fast" lesion [15].

Animals affected by LSD also display symptoms such as excessive salivation, increased tear production (lacrimation), nasal discharge, and emaciation. These symptoms are often associated with necrotic plaques and the characteristic LSD lesions present in the oral cavity, conjunctiva (eye membranes), and nasal cavity, respectively. Enlargement of superficial lymph nodes and lymphadenopathy are additional features commonly observed in cases of LSD (Lumpy Skin Disease). Furthermore, lactating cows may experience a reduction in milk production, develop mastitis, and in some cases, pregnant cows might face the risk of abortion. Additionally, calves with extensive skin lesions, presumably acquired through intrauterine infection, may be born. Swelling of the testicles and orchitis, which is inflammation of the testicles, can also occur in infected bulls. As a consequence of lesions in the reproductive organs, both bulls and cows may experience temporary or permanent sterility [17]. In infected cows, you may also observe edematous (swollen due to fluid retention) and inflammatory swellings in the brisket, face, and one or more limbs. These swellings can severely limit the animal's movement. Additionally, deep ulcerative skin lesions and cases of keratitis (inflammation of the cornea), which can be unilateral (affecting one eye) or bilateral (affecting both eyes), can be seen in some infected cows [24,27,28]. Pox lesions may also manifest in the pharynx, larynx, trachea, lungs, and throughout the alimentary

(digestive) tract. These lesions in the respiratory tract are frequently followed by the development of pneumonia [20]. Severe cases of LSD (Lumpy Skin Disease) have distinctive and easily recognizable symptoms. However, in the early stages of infection and in milder cases, the symptoms may resemble those of other diseases affecting the skin, which can lead to confusion in diagnosis. An example of a disease that can be confused with LSD is Pseudo Lumpy Skin Disease, also known as Allerton virus, which is caused by bovine herpesvirus-2 (BHV). It presents with skin lesions similar to those of LSD and often requires laboratory confirmation to differentiate between the two diseases. Pseudo lumpy skin disease is characterized by circular superficial lesions that can encompass the entire body and measure up to 2 cm in diameter. These lesions have a distinct intact central area and raised edges, often accompanied by hair loss. Urticaria, Streptotrichosis (caused by Dermatophilus congolensis infection), ringworm, Hypoderma bovis infection, photosensitization, bovine papular stomatitis, foot and mouth disease, bovine viral diarrhea, and malignant catarrhal fever are all potential differential diagnoses when evaluating cases that may be mistaken for LSD (Lumpy Skin Disease). Accurate identification and differentiation of these diseases require careful examination and laboratory tests [17,22,26].

6. Pathology

6.1. Gross pathological lesions

Skin nodules typically exhibit a consistent size, with a firm, round, and raised appearance. However, some of these nodules may amalgamate to form larger, irregularly shaped, and well-defined plaques. When incised, the nodule's surface appears reddish-gray and displays edematous characteristics in the subcutaneous layer. Upon incision, the nodule's surface appears reddish-gray and exhibits edematous characteristics within the subcutaneous layer.Circular necrotic lesions can be observed in various parts of the alimentary, respiratory, and urogenital tract. For example, these circular necrotic lesions may be found in areas such as the muzzle, nasal cavity, larynx, trachea, bronchi, inside of the lips, gingiva, dental pad, abomasum, uterus, vagina, teats, udder, and testes [22,27]. The regional lymph nodes can become significantly enlarged, swelling to sizes up to 10 times their normal dimensions. They also exhibit edema (fluid retention), congestion, and the presence of pyemic foci. In addition to these lymph node changes, local cellulitis can occur in the affected areas [24]. In severe cases, pleuritis (inflammation of the pleura, the lining of the chest cavity) and enlargement of mediastinal lymph nodes are also part of the clinical picture. The characteristic LSD nodular lesions extend into the musculature and the fascia over the limbs, appearing as grey-white nodules surrounded by red inflammatory tissue. Additionally, these lesions tend to be distinctly separated from the necrotic epithelium and extend a considerable distance from healthy tissue. They leave behind ulcers that heal gradually through the process of granulation. Severely infected animals may experience complications such as secondary bacterial pneumonia, tracheal stenosis (narrowing of the windpipe), acute and chronic orchitis (inflammation of the testicles), mastitis (inflammation of the mammary glands) with secondary bacterial infection, and similar lesions in the female reproductive tract [29].

6.2. Histopathological findings

The histopathological findings in LSD are distinctive and serve as a foundation for diagnosis. Microscopic examination can reveal the pathognomonic LSD lesion, which consists of eosinophilic intracytoplasmic inclusion bodies. These inclusion bodies may be found in keratinocytes, macrophages, endothelial cells, and pericytes derived from skin nodules. Additionally, the affected cells may display ballooning and degeneration of their cell layers. Inflammatory cells, such as macrophages, lymphocytes, and eosinophils, infiltrate the affected area. Moreover, there is a histological observation of widespread vasculitis, indicating the viral preference for endothelial cells [17] [30]. If muscular damage occurs during the course of LSD, histopathologically, severe coagulative necrosis in the subcutaneous muscle may be observed [31].

6.3. Hematological and serum biochemical changes

Hematological and serum biochemical analyses of animals have been naturally and experimentally infected with LSDV have been conducted and described in recent studies [25] [31,32]. The findings from Neamat-Allah's research indicate a noteworthy decrease in red blood cells, hemoglobin levels, packed cell volume, and mean corpuscular hemoglobin concentration in experimentally infected animals. Simultaneously, there is a substantial increase in mean corpuscular volume, which is typically interpreted as a macrocytic hypochromic anemia [25]. Conversely, the leucogram results demonstrated leucopenia (a decreased white blood cell count) and lymphopenia (a decreased lymphocyte count), which may be attributed to the viral infection. Additionally, granulocytic leukocytosis (an increased number of granulocytes) was observed, which could be a response to secondary acute bacterial infections, particularly those caused by pyogenic bacteria. Lumpy Skin Disease (LSD) has also been reported to be linked with various hematological and biochemical abnormalities in naturally infected cattle. These abnormalities include inflammatory thrombocytopenia (a decrease in platelet count due to inflammation), hyperfibrinogenemia (an elevated level of fibrinogen in the blood), decreased creatinine concentration (lower levels of creatinine in the blood), hyperchloremia (elevated blood chloride levels), and

hyperkalemia (higher-than-normal blood potassium levels) [32]. The studies conducted by Neamat-Allah and Abutarbush have revealed a significant decrease in the levels of total protein and albumin in the serum of LSD-infected cows. In contrast, there was a significant increase in globulin levels, particularly in the gamma globulins, in these infected cows [25,32]. Furthermore, study on the serum biochemical analysis of LSD-infected cattle indicated an increase in levels of aspartate aminotransferase (AST) and alkaline phosphatase (ALP). Additionally, there were elevated concentrations of globulin protein and creatinine in the serum of these infected cattle [31]. In conclusion, the studies have suggested that the changes observed in the serum biochemical analysis may be attributed to liver and kidney dysfunction, a severe inflammatory response, and disease-related complications, such as anorexia and a reduction in muscle mass, that occur during LSDV infection.

7. Diagnosis

The diagnosis of LSD relies on the recognition of characteristic clinical symptoms, supplemented by laboratory verification of the virus or antigen's presence.

A preliminary field diagnosis of LSD can be established by considering the following:

- ✓ **Morbidity, mortality, and clinical indicators that mirror LSD, including:**
  - A contagious illness characterised by widespread skin nodules
  - Low mortality, emaciation, and ongoing fever
  - Enlargement of the lymph nodes draining the affected areas, along with the typical inverted conical necrosis of skin nodules (sitfast).
  - Mouth, pharynx, tongue, epiglottis, digestive tract, nasal cavity, trachea, and lung mucous membranes are among the mucous membranes affected by shingles.
  - In the lungs, there is edoema and spots of localised lobular atelectasis.
  - Fibrin in the synovial fluid is present in synovitis and tendosynovitis.
- ✓ **A definitive diagnosis of LSD can be established by:**
  - **Laboratory investigations and identification of the agent** [33,34]
- ✓ **Isolation of the virus**

The isolation and identification of the virus is necessary for the confirmation of lumpy skin disease in a new area. Before the establishment of neutralising antibodies, samples for viral isolation should be collected within the first week of the emergence of clinical symptoms [35, 36]. Early lesions (those without necrosis) on the skin can be biopsied to obtain samples for viral isolation and electron microscopy. Additionally, during the viraemic stage of LSD, blood samples taken into EDTA or heparin can be used to isolate the LSD virus from buffy coat. For viral isolation, samples aspirated from swollen lymph nodes can also be employed. For viral isolation, samples aspirated from swollen lymph nodes can also be employed. In tissue cultures of bovine, ovine, or caprine origin, the LSD virus multiplies. The most vulnerable cells are thought to be lamb testis (LT) cells or ovine dermis cells (primary or secondary culture). LSD capripoxvirus has also been modified to grow on the chorioallantoic membrane of embryonized chicken eggs and African green monkey kidney (Vero) cells, which is not advised for primary isolation[33].

- ✓ **Electron microscopy**

Within a few hours after receiving the samples, a transmission electron microscopy (TEM) diagnosis of LSD can be verified. In preparations of biopsy specimens taken from afflicted skin or mucous membranes that were negatively stained, TEM demonstrated the virus.

Orthopox virions have a much rounder profile and smaller lateral bodies than mature capripox virions, which have an average dimension of 320 × 260 nm.[33]

- ✓ **Fluorescent antibody tests**

Using fluorescent antibody testing, capripoxvirus antigen can also be found on infected tissue culture slides or cover slips.

- ✓ **Enzyme-linked immunosorbent assay**

It is manufactured by producing monoclonal antibodies (MAbs) using expressed recombinant antigen and P32 monospecific polyclonal antiserum [12]

- ✓ **Polymerase chain reaction (PCR)**

Capripoxviruses have been more sensitively detected using the loop-mediated isothermal amplification (LAMP) assay [37,38].

7.1. Differential diagnosis

Similar LSD symptoms are caused by a variety of illnesses. In order to implement the most effective preventive and control measures for susceptible herds, it is crucial to establish a definitive diagnosis. These conditions can be mistaken for LSD:

- Bovine virus diarrhoea/mucosal disease
- Demodicosis (Demodex)
- Bovine malignant catarrhal fever (Snotsiekte)
- Rinderpest
- Besnoitiosis
- Insect bite allergies
- Oncocercariasis

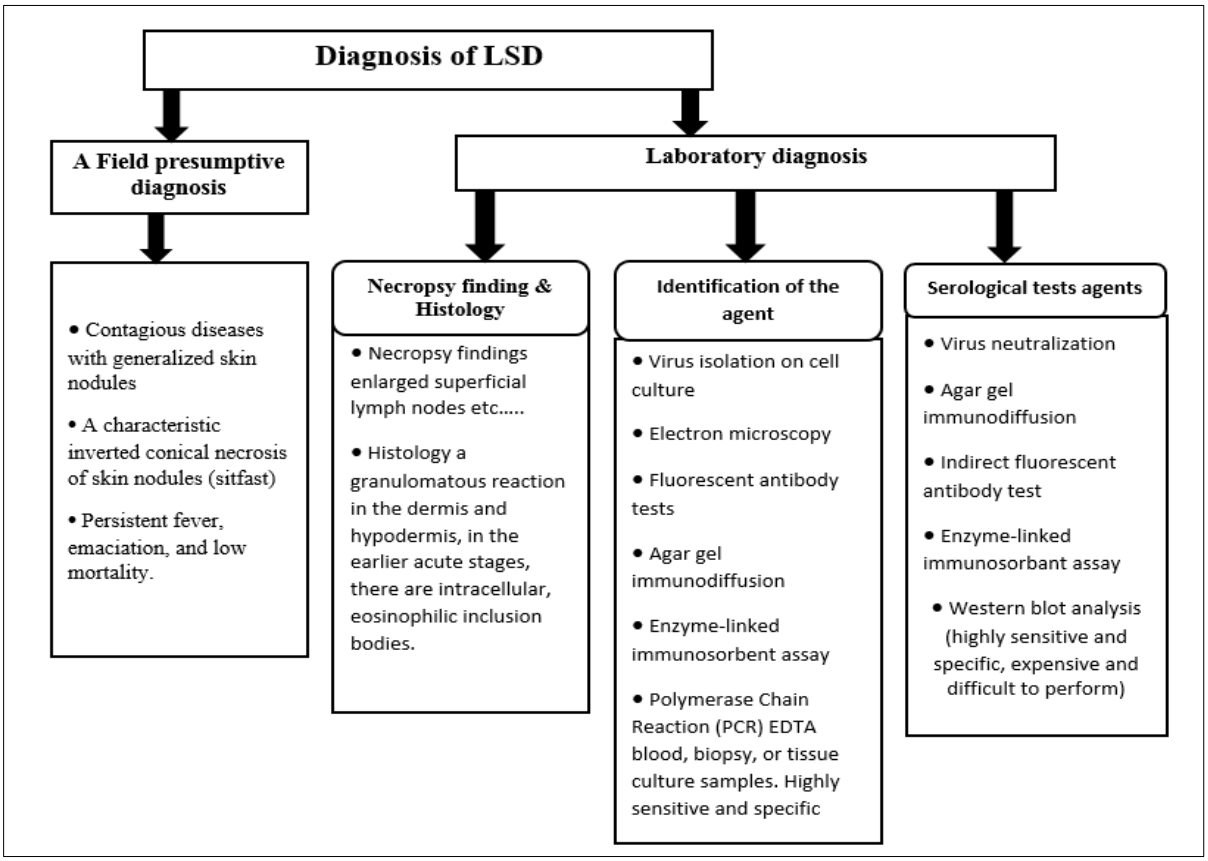


Figure 6 The diagnostic procedures of the LSD

8. Treatment

Lumpy skin disease is the result of a viral infection and, as a consequence, there is currently no identified treatment available. Nevertheless, antibiotics, anti-inflammatory medications, or vitamin injections are occasionally administered in certain instances to address secondary bacterial infections, alleviate fever or inflammation, and enhance the animal's appetite [39].

9. Control Measures

Due to LSD being a viral infection, there is presently no targeted cure available. The treatment for LSD primarily focuses on alleviating symptoms, with the use of antibiotics to prevent potential bacterial complications (Table and Figure). Antibiotics like penicillins, cephalosporins, tetracyclines, and fluoroquinolones are typically prescribed for a duration of 5 to 7 days, which may vary based on the seriousness of the illness. In 2011, Salib and Osman initiated treatment studies aimed at mitigating the consequences of LSD and preserving lives. Their approach proved successful, employing a combination of medications designed to combat bacterial infections, reduce inflammation, offer relief, and treat various infections [24,40]. They also recommended the use of antihistamines and non-steroidal anti-inflammatory drugs (NSAIDs). In addition, an antipyretic medication like paracetamol was administered to lower fever. To aid in the recovery of appetite loss, it was advised to take regular doses of multivitamins and liver-supporting medications[41]. Nonetheless, managing LSD and its associated consequences can be expensive, and it does not always guarantee a complete recovery. Therefore, prevention proves to be a more effective strategy for minimizing significant financial losses arising from hide damage, milk loss due to mastitis, and losses of food products caused by death, miscarriage, fever, and myiasis.

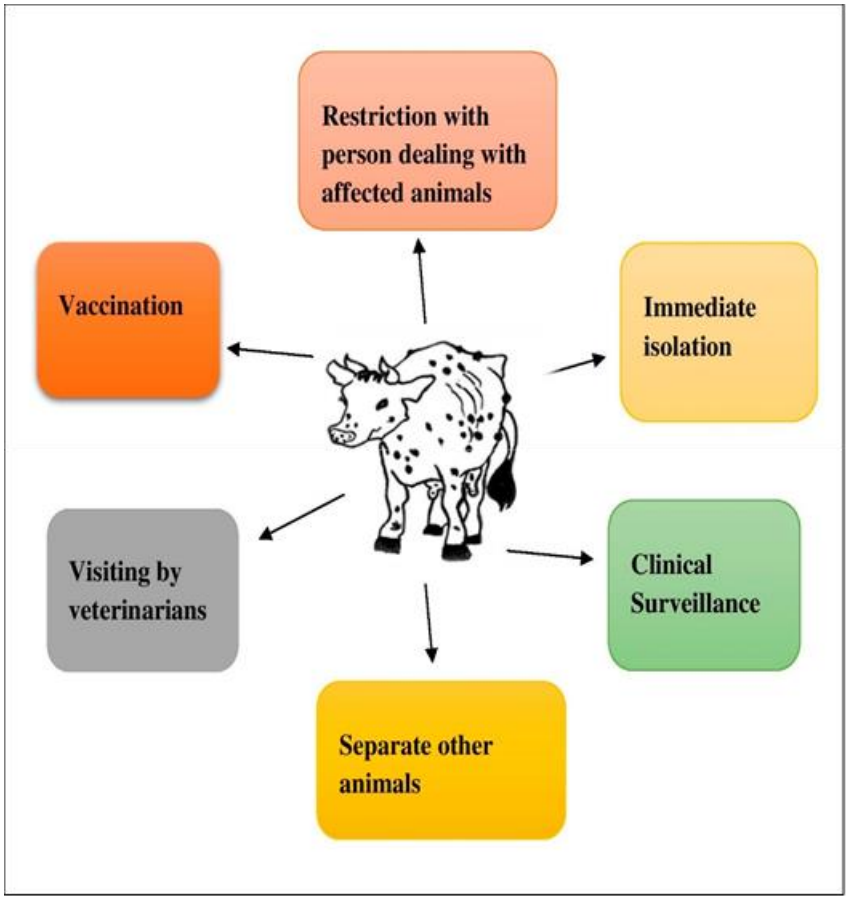


Figure 7 Different control measures to prevent LSD

9.1. Control of Cattle Movement

When a disease is initially identified in a country or region, the immediate and critical steps to be taken include implementing a standstill and quarantine measures. It is essential to establish zones with minimal movement restrictions while focusing on clinical surveillance in areas at high risk [42]. Additionally, for the successful execution of eradication measures, such as quarantine, the culling of infected and exposed animals, appropriate carcass disposal, premises cleaning and disinfection, and pest control, it is crucial to promptly identify clinical cases as soon as they occur during the outbreak [43]. Nevertheless, in endemic areas, disease control primarily relies on vaccination, imposing restrictions on animal movement, and the euthanasia of animals showing signs of illness [44]. In India, where cattle are considered sacred, euthanasia is generally not permitted as a control measure for diseases, which can pose a unique challenge in disease management and control strategies.

9.2. Vector Management

Consider vector control as a complementary measure rather than a preventive one since it cannot completely halt the spread of LSD or its transmission to individuals. Regularly using pour-on insect repellents and insecticides on cattle and buffalo, along with implementing additional pest control measures, can contribute to controlling vectors in farm environments. Nevertheless, completely eradicating the disease is likely to be challenging due to the involvement of arthropod vectors, and any delays in removing infected animals raise the risk of LSD transmission [38]

9.3. Vaccination

The most effective approach to preventing the transmission of LSD is through vaccinating cattle with a proven and effective vaccine, particularly when administered as a preemptive measure or before the virus reaches a susceptible region or country. A study on the epidemiological traits and economic implications of lumpy skin disease in Ethiopia. This study underscores the importance of immunization as a crucial component of LSD management in regions where the disease is endemic [45]. Live vaccines are known to trigger a robust and enduring immune response, making them

highly effective for preventing diseases [46]. Nonetheless, live vaccinations can sometimes lead to localized inflammation and result in mild illness characterized by skin lesions [46].

Members of the Capripoxvirus family have been observed to offer cross-protection. Consequently, cattle can be safeguarded from LSD (Lumpy Skin Disease) infection through the use of live-attenuated vaccines that are either homologous (such as the Neethling LSDV strain) or heterologous (derived from sheep pox or goat pox viruses). Commercially available Capripoxvirus (CaPV) vaccines comprise various strains, such as the LSDV Neethling strain, KSGPV O-240, and O-180 strains, as well as the GTP strains. Additionally, vaccines may include strains derived from Kenyan sheep and goat pox viruses, such as the Gorgan goat strain, Romanian SPP strain, and Yugoslavian RM65 sheep pox (SPP) strain. These strains are utilized to develop vaccines for the prevention of diseases like Lumpy Skin Disease (LSD) and sheep and goat pox [47]. The effectiveness of three Capripoxvirus (CaPV) strains against LSD (Lumpy Skin Disease) in Ethiopia, it was found that the Gorgan GTP vaccine can effectively provide protection to livestock against LSDV. However, the Neethling and KSGP O-180 vaccines appeared to be ineffective in this study. This highlights the need for further molecular diagnosis and evaluation of these less effective vaccines to enhance their efficacy in preventing LSD [48]. In the past, various factors contributing to vaccine ineffectiveness were recognized. These factors encompassed disparities in strains between the vaccine and the circulating field strain, inadequate vaccine potency, vaccination of animals already in the incubation phase of the disease, and mishandling of the vaccine during its transportation and storage [49]. Subsequently, the absence of cross-protection with the vaccine strain and the reduced immunogenicity of the vaccine resulting from excessive attenuation could be responsible for its limited effectiveness and the Neethling vaccine's failure in Ethiopia [48,49].

As a precaution against potential safety issues associated with the live-attenuated LSDV vaccine, it is recommended to employ the same vaccination in countries that have traditionally remained free of LSD and also offer protection against sheep pox [50]. Even though inactivated vaccines can be expensive and necessitate multiple doses, they are regarded as safe and can be combined with other antigens to create polyvalent immunizations suitable for use in regions where the infection is not prevalent. Furthermore, inactivated vaccines can serve as the concluding phase in a strategy that initially employs live vaccines to eliminate a disease [50].

Because the LSD virus remains stable and viable in the environment for an extended period, it is crucial to institute compulsory, comprehensive, and enduring immunization programs with full coverage to effectively control and prevent the disease. Additionally, it is recommended to vaccinate newly acquired animals before introducing them to farms affected by LSD to mitigate the risk of disease transmission. Calves that have been nursed by mothers who were vaccinated or naturally infected should be safeguarded when they reach 3 to 4 months of age. Additionally, it is advisable to administer vaccinations to pregnant cows and breeding bulls annually [51].

9.4. Awareness

Effective disease control in cattle cannot be successfully achieved without strong collaboration and coordination among farmers and all other stakeholders within the cattle value chain. Efforts to enhance awareness should be targeted towards a diverse audience, including public and private veterinarians, veterinary students, farmers, herders, cattle merchants, cattle truck drivers, and artificial inseminators, spanning across various settings, including the field and abattoirs. Providing education to veterinary professionals and livestock workers can empower them to promptly diagnose clinical cases, which in turn can play a crucial role in preventing the further spread of the disease [52].

10. Conclusion and Recommendations

Lumpy skin disease (LSD), a vector-borne illness caused by the genus CaPV, was previously limited to sub-Saharan Africa. Nonetheless, in recent times, it is gradually spreading to new regions, including Europe. From a clinical perspective, this disease is distinguished by unique nodular lesions primarily found on the skin and underlying tissues of afflicted animals. There are occasional instances of involvement in various body parts, including the conjunctiva, alimentary, respiratory, and urogenital tracts. As a result, these lesions lead to significant economic losses due to diminished hide quality, chronic weakness, decreased milk production, weight loss, infertility, abortion, and mortality. Additionally, these effects can have a profound impact on rural livelihoods, particularly those heavily reliant on cattle, resulting in substantial reductions in production. The consequences of this disease are also devastating on a national scale, as its presence has led to the implementation of stringent trade restrictions. To address these alarming situations, the following recommendations are put forward:

- It is essential to identify the clinico-hematological and biochemical profiles of cattle affected by LSD in addition to recognizing the typical clinical signs.

- Timely and precise diagnosis is crucial for implementing effective control measures.
- In areas where LSD is endemic, it is mandatory to implement an annual vaccination strategy using a homologous strain of the LSDV.
- It is vital to implement vector control measures and restrict the movement of animals during the active period of insect movement to mitigate the spread of the disease.
- Bulls used for breeding should undergo diagnostic testing for LSDV to ensure they are free from the disease.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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