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Lumpy skin disease: An emerging transboundary viral disease

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Abstract
Lumpy skin disease is a severe viral disease of cattle caused by the lumpy skin disease virus, a member of the *Capripoxvirus* genus of the *Poxviridae* family. The disease is characterized by fever, enlarged lymph nodes, firm, circumscribed nodules in the skin, and nodules that are particularly noticeable in the hairless areas. Blood-feeding insects, such as specific types of flies, mosquitoes, and ticks, are thought to be the primary vectors of lumpy skin disease virus transmission. Epidemic of disease occurs mainly during rainy season. Disease causes serious economic loss to the animal owners. Diagnosis is mainly based on using various advanced techniques like virus isolation and culture, serological tests, polymerase chain reaction, dot blot hybridization, agar-gel immunodiffusion tests, indirect enzyme-linked immunosorbent assays, direct fluorescent antibody tests, west blotting, routine histopathology, and immune histopathological staining. Disease should be differentiated from other diseases including dermatophilosis, cutaneous tuberculosis, dermatophytosis and others. Effective control measures for this disease are achieved through mass vaccination, import restrictions on livestock and their products, control of vectors, and quarantine stations. Therefore, this communication reviews existing information on the various aspects of the disease, such as its etiology, transmission, epidemiology, diagnosis, prevention, and control measures.

Keywords: Cattle, control, diagnosis, lumpy skin disease, lumpy skin disease virus, prevention

Introduction
Since the lumpy skin disease virus (LSDV) has spread to new geographic areas and has had negative economic effects on a global scale, there has been an unprecedented surge in scientific reports and publications on the virus over the past ten years. This has increased interest in both the disease and its causative agent (Bianchini *et al*., 2023) [6]. LSDV is a type of double-stranded DNA (dsDNA) poxvirus belonging to the genus *Capripoxvirus*. It spreads similarly to other *Capripoxviruses*, such as the goatpox (GTPV) and sheeppox (SPPV) viruses, through indirect contact and arthropod bites (Nesterov *et al*., 2022) [20]. While direct and indirect contact is the most effective way for viruses to spread, LSDV is primarily spread mechanically through blood-feeding arthropod vectors (Hamdi *et al*., 2021) [14]. Viruses can be spread by touch, water, and food, but they are primarily spread by insects such as flies, mosquitoes, and ticks. This is because the disease is also vector-borne (EFSA Panel, 2015) [10].

Nodules that appear on the skin, lips, and upper respiratory tract are the hallmarks of lumpy skin disease, an infectious acute or sub-acute viral illness in cattle. According to Davies (1991) [9], there is a breed difference in the severity of the clinical indications in affected cattle, although they experience significant emaciation and productivity loss for long periods. In addition to draft, skins, and dung fuel, diseases are a major factor in decreased milk and meat yield. One of the most economically significant viral emerging disorders is lumpy skin disease (LSD), which is characterized by hardness, enlarged lymph nodes, fever, and confined nodules (OIE, 2010) [23]. It is a disease that affects cattle of all ages and breeds, with a high morbidity and low fatality rate. Reduced milk production, lost meat, draft power, abortion, infertility, condition loss, and hide damage all result in large economic losses (CFSPH, 2008) [7]. Therefore, this paper delineates the importance of lumpy skin disease as an emerging transboundary viral disease of cattle.

Etiology
The *Poxviridae* family includes Lumpy skin disease virus that causes devastating infection in cattle. There are two subfamilies within the family *Poxviridae*:

Chordopoxvirinae (poxviruses of vertebrates) and *Entomopoxvirinae* (poxviruses of insects). Within the subfamily *Chordopoxvirinae* and genus *Capripoxvirus*, lumpy skin disease virus is found. As for the prototype strain of LSDV, there is just one serotype. Routine virus neutralization or other serological tests can distinguish it from the sheep and goat poxvirus, which is closely linked antigenically to the Neethling virus. Although it has been shown to impact sheep and goats in tests, the LSDV mostly affects cattle. Lamb testis cells give the highest yield when the virus-causing lumpy skin disease is grown in tissue culture of cow, ovine, or caprine origin (Gelaye *et al*., 2015) [13]. Among all viruses, those belonging to this family are the biggest. It has the shape of an envelope and contains linear or ovoid molecules that are virions that are 140-266 nm by 220-450 nm in size. It is mentioned by Stram co-workers (2008) [27], that the DNA genome of LSDV is roughly 151 kb.

Transmission

LSDV is transmitted through arthropods, particularly blood-sucking insects (MacLachlan & Dubovi, 2010) [19], contaminated feed and water, and direct transmission in the later stages of the disease via saliva, nasal secretions, and semen (Tuppurainen *et al*., 2017) [28]. As most LSD outbreaks have occurred in the summer when arthropods are most active, it may indicate the involvement of various vector species, especially blood-feeding insects, in virus spread (Sprygin *et al*., 2018) [26].

Numerous investigations have hypothesized that hard ticks may play a part in the spread of viruses (Lubinga *et al*., 2015) [18]. There have been reports linking three kinds of bloodsucking hard ticks *Aedes aegypti* mosquitoes and *Stomoxys calcitrans* flies to the spread of LSDV in sub-Saharan Africa. *Rhipicephalus (Boophilus) decoloratus* (blue tick), *Rhipicephalus appendiculatus* (brown ear tick), and *Amblyomma hebraeum* are the three tick species that have been discovered as both disease vectors and viral reservoirs (Lubinga *et al*., 2014) [17].

Blood, nasal and lachrymal secretions, semen, saliva, milk, and nodules on the mucous membranes of the eyes, nose, mouth, rectum, udder, and genitalia that ulcerate, are all significant sources of transmission of infection (Babiuk *et al*., 2012) [4]. These secretions contain all of the animal viruses that are infected. Lefèvre and others (2010) [16] also reported that infected milk can spread the LSD virus to nursing calves. Thus, blood-feeding and biting arthropods, such as ticks, mosquitoes, and biting flies, are the primary vectors of transmission. Additionally, though it is uncommon, contamination of feed and water can potentially lead to transmission by direct contact (Ali *et al*., 2012) [1].

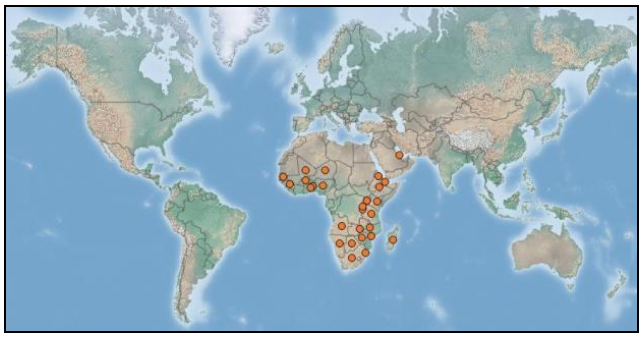
Clinical symptoms

The symptoms of the illness include fever, emaciation, swollen lymph nodes, edema of the skin, and occasionally even death. There are also nodules on the skin, mucous membranes, and internal organs. The typical skin lesions are solid, flat-topped papules that are 0.5-5 cm in diameter, well-circumscribed nodules, and numerous papules that coalesce. The nodules affect the dermis and epidermis, and on occasion, they may even spread to the nearby striated muscle and the subcutis beneath it. The regions that are predisposed include the skin of the head, neck, perineum, genitalia, udder, and limbs. At first, these nodules may exude serum and have a creamy gray-to-white appearance on the area that has been sliced. A cone-shaped central core, sequestrum of necrotic material, or necrotic plug (also known as a "sit-fast") may, however, form within the nodule within the next two weeks (WOAH, 2014) [30].

As soon as the nodules on the mucous membranes of the eyes, nose, mouth, rectum, udder, and genitalia begin to ulcerate, the virus is present in all secretions, including saliva, ocular and nasal discharge, and the nodules on the genitalia. Many cattle suffer severe emaciation and loss of production for several months. The skin lesions cause permanent damage to the hides. The disease is of economic importance as it can cause a temporary reduction in milk production, temporary or permanent sterility in bulls, damage to hides, and, occasionally, death. LSD can lead to mastitis, orchitis, and abortion. However, nodules were not observed in aborted fetuses (Tuppurainen *et al*., 2017) [28]. Intrauterine transmission of LSD is possible; pregnant cattle may abort, bulls may become permanently or temporarily infertile, and the virus can be excreted in the semen for prolonged periods (WOAH, 2014) [30].

Geographical distribution

The geographic distribution of lumpy skin conditions varies (OIE, 2010) [23]. In Zambia, the illness was initially discovered in 1929. Because it was the year with the highest number of biting insects at the time, it was initially thought to be the result of either poisoning or hypersensitivity to bug bites (Bagla, 2005) [5]. Cases were reported in Botswana, Zimbabwe (Southern Rhodesia), and the Republic of South Africa between 1943 and 1945. At this point, the disease's infectious character was acknowledged (OIE, 2008) [22]. Figure 1 below shows the distribution of LSD around the world. From 1929 until 1986, LSD was only legal in sub-Saharan Africa; it is now endemic in the majority of African nations, including Madagascar. Reports of the virus throughout the Middle East then followed. According to later reports from Oman, Yemen, Israel, Kuwait, Bahrain, Egypt, Iran, and Saudi Arabia, LSD has been spreading on an exceptionally massive scale throughout the Middle East since 2012 in the United Arab Emirate, Jordan, and Lebanon (Zeynalova *et al*., 2016) [31].



Source: https://www.cabi.org/isc/datasheet/76780#toDistributionMaps

Fig 1: Geographical distribution of Lumpy skin disease

Diagnosis

The clinical signs and symptoms that the animal’s exhibit can be used to make a diagnosis of lumpy skin disease. Depending on the animal's state, the symptoms may be abrupt, mild, or persistent. Advanced techniques such as virus isolation and culture, serological testing, polymerase chain reaction (PCR), dot blot hybridization (DBH), agar-

gel immunodiffusion tests (AGID), indirect enzyme-linked immunosorbent assays (ELISA), direct fluorescent antibody tests, west blotting, routine histopathology, and immune histopathological staining can all be used in the lab to assist in confirmatory diagnosis (Awad *et al*., 2010 [3]; OIE, 2021) [21]. The following conditions may be considered in the differential diagnosis of the disease: Cutaneous tuberculosis, ringworm, insect bites, dermatophilosis, stomatitis, Vaccinia virus, pseudo-cowpox, and bovine herpes abnormalities (Das *et al*., 2021) [8].

Treatment, prevention, and control

Antibiotics against secondary skin infections and pneumonia, together with some anti-inflammatory medications, can be administered to infected animals as supportive care, as there are no particular antiviral medications available (Vinothraj *et al*., 2020) [29]. Additionally, the skin lesions can be treated. Gautam and co-investigators (2020) [12] observed that sulphonamide applied to nodular lesions and diclofenac gel used in the swelling region worked well. According to Feyisa (2018) [11], LSD virus infection was successfully treated with a combination of three days of dexamethasone and broad-spectrum antibiotics. Fly-repelling antiseptic ointment applied in tropical areas can be a wise decision (Islam *et al*., 2021) [15].

Lumpy skin disease is prevented and controlled by vaccination, isolation of livestock, killing of infected and exposed animals, killing of insect vectors during the early stages of an outbreak to reduce mechanical transmission of the virus, cleaning and disinfection of the area, and an awareness campaign to encourage industry and community cooperation (Radostits *et al*., 2006) [24]. The disease is most likely spread by biting flies and specific tick species; movement restriction and quarantine are typically ineffective measures of management. Therefore, vaccination or immunoprophylaxis is practically the only form of control in endemic locations (Rushton and Leonard, 2009) [25].

There are currently only live-attenuated LSD vaccinations on the market. In endemic locations, live vaccinations aid in the management of lumpy skin disease losses. Vaccines based on attenuated sheep pox viruses are less effective than homologous LSD vaccines. Vaccinations using heterologous live-attenuated viruses have the potential to produce mild to severe side effects. Since live vaccinations could potentially spread infection to vulnerable sheep and goat populations, this vaccination is not recommended in nations where sheep and goat pox is nonexistent (Al-Salihi, 2014) [2]. Consequently, immunization is the only effective way to manage the disease in endemic nations; nevertheless, the safety of using live attenuated vaccines in non-endemic locations is seriously questioned (OIE, 2010) [23].

Conclusion and Recommendations

A virus connected to the Neethling poxvirus of the genus *Capripoxvirus* of the family *Poxviridae* causes lumpy skin disease, a contagious, eruptive, and occasionally fatal skin condition in cattle. Depending on the age, sex, and breed of the cattle, the clinical indications of LSD may be acute or sub-acute. Large-scale transmission of LSDV occurs through the movement of cattle and their byproducts, with blood-feeding insects serving as the main vectors of infection. Appropriate molecular and serological methods can be used to diagnose LSD. Additionally, employing efficient preventative measures like immunization and closely monitoring the various facets of the disease, including epidemiology and transmission, may lead to improved disease control.

Based on the above conclusion, the following recommendations are forwarded

- It is emphasized to make prompt and accurate diagnosis in endemic areas.
- To prevent further spread, it is strongly advised to vaccinate against the homologous strain of the LSDV, restrict animal movement, and screen bulls intended for breeding for the LSDV virus.

Contribution of authors

All the authors contributed for the preparation of the manuscript.

Conflict of interest

No conflict of interest was observed.

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