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LUMPY SKIN DISEASE: A REVIEW

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Lumpy skin disease (LSD), Capri poxvirus, Skin nodules, Infection, Bovine

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ABSTRACT: Lumpy skin disease (LSD) causes enormous economic losses in the livestock business. Lumpy skin disease virus (LSDV) a member of the Poxviridae family causes the condition. The Neethling strain is the pro to type. LSDV is part of the Capri poxvirus genus which also includes sheep and goat poxviruses. LSD is an enzootic illness that causes skin nodules and is seldom lethal in cattle. Cattle and water buffalo are the only animals afflicted with substantial morbidity but moderate mortality rates. However, calves are more likely to die. LSD can lead to reduced milk and beef production, miscarriages in females and sterility in males. LSD originated from Zambia in 1929. LSD is considered an endemic disease in Africa. However, in 1984 the disease spread beyond Africa. It has been documented in Madagascar, as well as in various Arab Gulf Peninsula and Middle Eastern countries. The disease has recently been reported in LSD-free nations (Jordan, Syria, Lebanon, Turkey, Iran, and Iraq), potentially causing economic losses in the livestock business. This review article discusses LSD in light of recent worries about disease spread in LSD-free countries.

INTRODUCTION: Lumpy skin disease (LSD) also referred to as pseudo-urticaria, Neethling virus disease, exanthema nodularis bovis and knopvelsiekte is a contagious illness. The virus (LSDV) belongs to the Poxviridae family specifically the Capripoxvirus genus. It shares antigenic similarities with sheep and goat poxviruses. Routine serological tests cannot differentiate these viruses. LSD affects cattle and water buffalo. This disease is transmitted by biting arthropods that feed on blood. LSD causes significant economic losses including emaciation, hide damage, infertility, mastitis, loss of milk output and up to 20% mortality [2].

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The disease has spread beyond Africa to Madagascar and the Middle East causing significant economic loss to the livestock industry. In the field, the incubation period lasts two to five weeks with lesions appearing between 4 to 20 days of inoculation. Fever is the first indication, followed by nodules on the skin and mucous membranes after two days [3].

FIG. 1: LUMPY SKIN DISEASE

LSD is diagnosed based on common clinical patterns including morbidity and mortality rates. A

diagnosis is validated using transmission electron microscopy (TEM), immunoperoxidase (IMP) staining, antigen-trapping enzyme-linked immunosorbent assay (ELISA) and polymerase chain reaction (PCR) testing. There is no specific treatment for LSD. Infected animals should get supportive therapy to alleviate symptoms and prevent consequences. In South Africa, vaccinations based on the Neethling strain virus are efficient in controlling the disease [4].

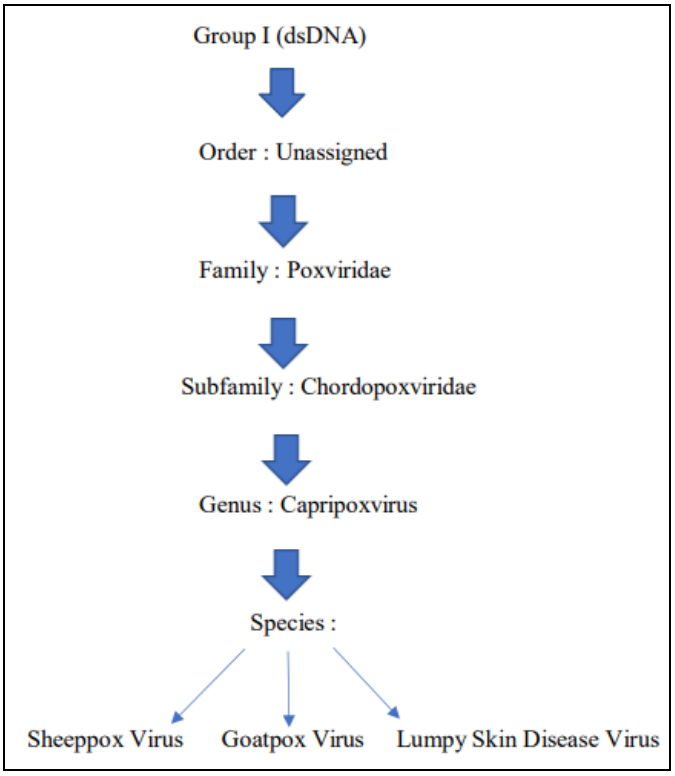

FIG. 2: CLASSIFICATION OF LUMPY SKIN DISEASE VIRUS

Epidemiology:

Morbidity and Mortality Rates: The morbidity and fatality rates associated with LSD outbreaks vary dramatically. It is determined by the following factors: geographic location and climate, management conditions, the nutritional status and general condition of the animal, the breed of cattle afflicted, immune status, population levels and distribution of potential insect vectors in various environments and virus virulence. The morbidity rate for LSD varies between 5 to 45 percent. However, morbidity rates of 1 to 5% are regarded more common. Higher rates have been observed in epizootics in Southern, West and East Africa as well as Sudan however far lower rates are possible within the same epizootic. In Oman in 2009, a farm population of Holstein cattle experienced high illness and mortality rates of 30-45% and 12%, respectively [8].

Animals that are Susceptible: LSD has a restricted vertebrate host range. Cattle and buffalo are the species that normally acquire sick during field epidemics. There have been five reported clinical cases of LSD in *Bubalus bubalis*, the Asian water buffalo. Other domestic ruminant species are not naturally affected during field outbreaks. Every cattle breed appears to be equally susceptible to the disease [9]. Young calves are particularly vulnerable to the disease and may acquire the distinctive lesion within 24 to 48 hours. However, all age groups of animals are susceptible [9].

Etiology: Mature capripoxvirions have a more oval shape and larger lateral bodies than orthopoxvirions. Their average dimensions are 320 x 260 nm.

The LSD virus spreads rapidly in several cell cultures including lamb and calf kidneys, adrenal and thyroid glands, muscle and testicles, Sheep embryonic kidneys and lungs, rabbit fetal kidneys and skin, chicken embryo, adult vervet monkey and baby hamster kidneys and primary cell cultures of bovine dermis and equine lungs are all used for this purpose. Cytopathic encephalopathy can occur within 11 days of primary encephalopathy [10].

The LSD virus has a single serotype that is closely related to the sheep and goat pox (SGP) virus and cannot be easily identified using virus assays. LSD virus strains have been discovered to interact with each other and with a Kenyan strain (O 240/KS) of sheep and goat pox virus (SGPV) through endonuclease tests of capripox virus. Kenyan SGPV strains differed from the O 240/KSGP strain but were comparable to those found on the Arabian Peninsula [11].

In comparison to strains from India, Iraq and Nigeria the Kenyan group of SGPV strains exhibited differences. The LSD virus is very resistant and tolerates a variety of physical and chemical treatments. The virus can survive in fresh skin for over a month and in air-dried hides for more than two weeks at room temperature [11].

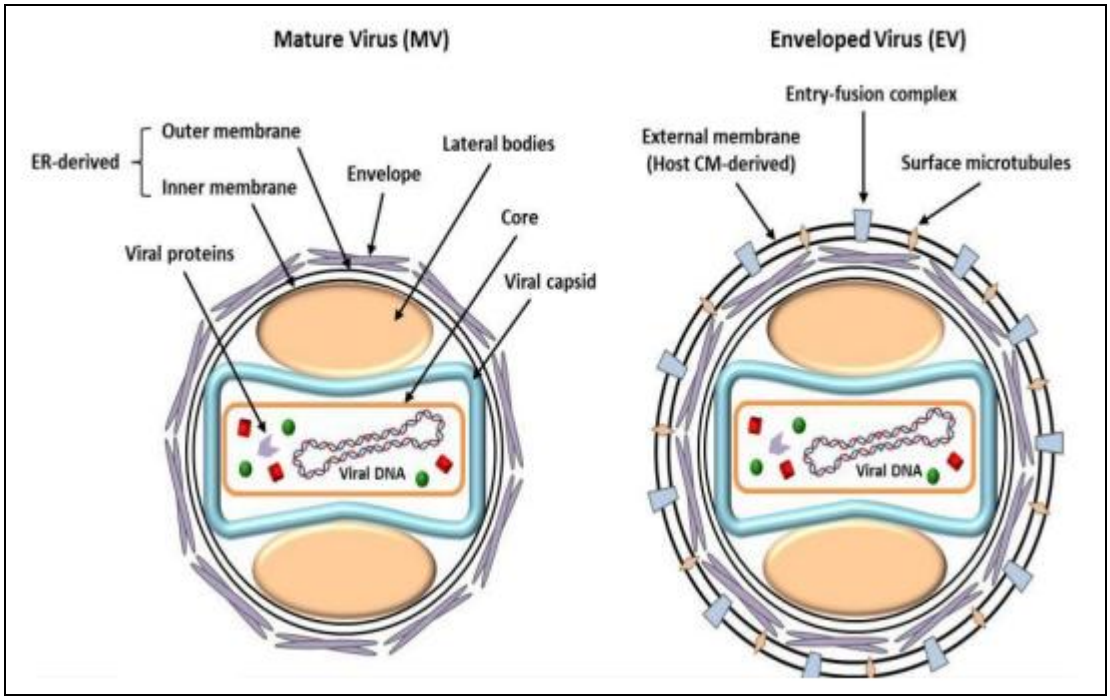

FIG. 3: THE STRUCTURE OF LSDV

Survival: LSDV exhibits remarkable stability as it may endure extended durations at room temperature particularly in desiccated scabs. LSDV is extremely resistant to inactivation; it can live for at least 18 days in air-dried hides, up to 33 days or longer in necrotic skin nodules and up to 35 days in desiccated crusts. The virus can survive for several months in dark environments, such as contaminated animal shelters but it is sensitive to sunshine and detergents that include lipid solvents [12].

Transmission: It is unclear how the lumpy skin disease virus spreads. The LSD virus spreads mostly through flying insects. Field observations show that epidemics occur during peak biting bug activity. Most cases are thought to have been transmitted by an arthropod vector [13].

Attack rates vary from 10-15% and nearly 100% depending on the active vector species present in each outbreak. Stomoxys such as tabanids and tsetse flies may be less effective in dry environments and have decreased transmission rates. Large mosquito breeding grounds contribute to increased morbidity rates following rain [13].

Direct & Indirect Mode of Transmission: Generally direct contact is considered an ineffective means of transmitting LSDV, although there is limited experimental evidence to support this. It was determined through early experimental work and field observations in South Africa that there is a possibility of LSDV transmission by direct touch, albeit at low rates and efficiency. Observations of LSD outbreaks that happen outside of the window of ideal insect activity temperatures lend credence to this [14].

Insect Transmission: The mechanical transmission of many poxviruses including swinepox, myxoma and poultry pox has been documented using arthropod vectors. The virus that causes rabbit fibroma is easily spread mechanically by fleas, mosquitoes and other biting arthropods. The viruses were linked to the mouthparts and head area of the arthropod in each of these cases but not to its body. Vector competency is contingent upon but not restricted [14].

Tick Transmission: The ability of a virus to resist histolysis in tick tissues and the sensitivity of tick cells to viral infection determine whether the virus survives in tick vectors.

Similar to how viruses are spread by insects, ticks can also mechanically transmit the virus if they feed multiple times and switch hosts in between. One instance of a poxvirus spread mechanically by ticks is the fowl pox virus [14].

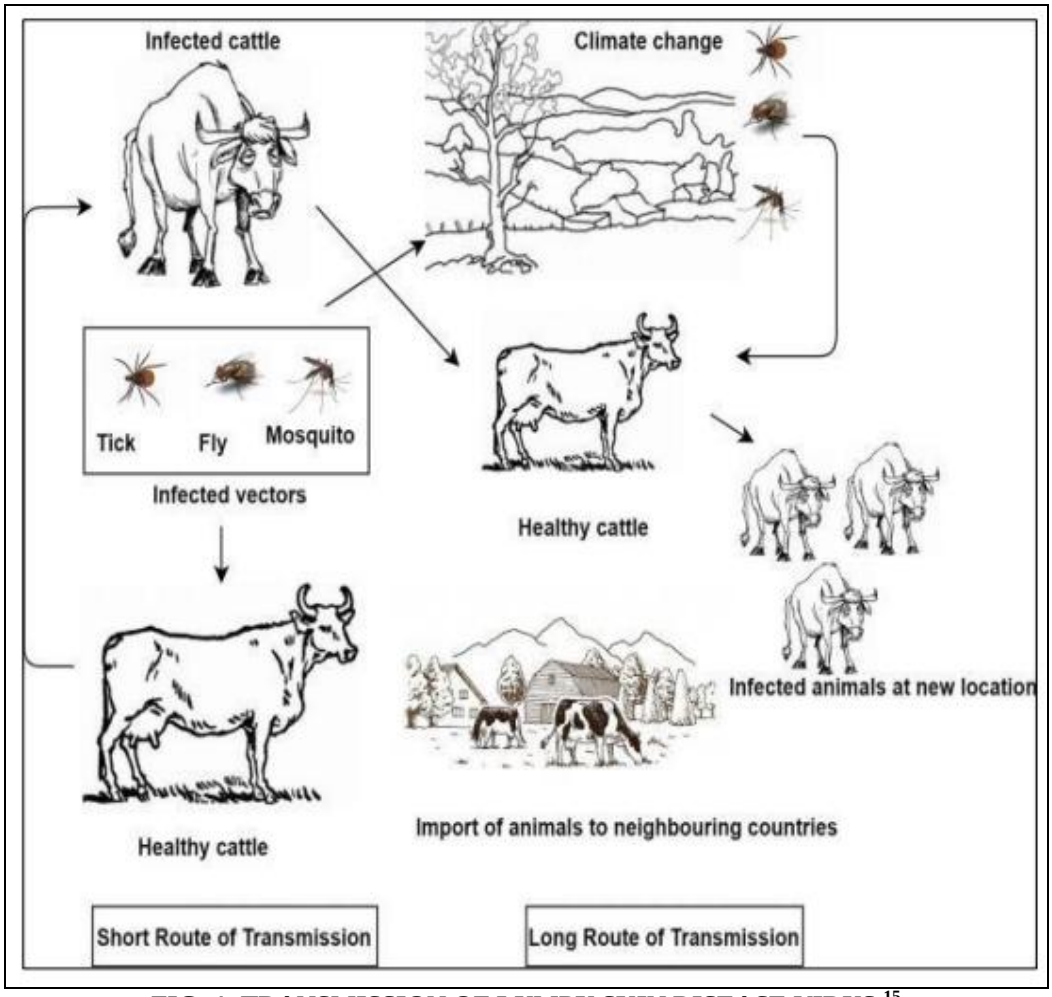

FIG. 4: TRANSMISSION OF LUMPY SKIN DISEASE VIRUS [15]

Risk Factors: Warm, humid weather circumstances that encourage a high population of vector populations such as those observed following periodic rains and the addition of additional animals to a herd are risk factors linked to the spread of LSD. Other risk factors that could raise the prevalence of the disease include the size of the herd, the number of vectors, the distance to the lake, the herd's migration, the movement of affected animals into areas free of the disease, common pastures and water sources. Furthermore, the wind's strength and direction may have a role in the virus's propagation. Cattle of all ages, breeds and genders are susceptible to the illness. The following are the risk factors:

Geographical Location: Regions with warmer climates and suitable vectors for transmission increase the risk of disease spread.

Movement of Livestock: Trade and movement of infected cattle can introduce the virus to new areas, accelerating its spread.

Vector Presence: Certain insect vectors, particularly biting flies like mosquitoes and ticks, play a crucial role in transmitting the virus between cattle.

Herd Density: High-density cattle populations increase the likelihood of disease transmission within and between herds.

Lack of Vaccination: Inadequate vaccination coverage leaves cattle susceptible to infection, allowing the virus to persist and spread more easily [16].

Clinical Sign: Cattle with lumpy skin disease are susceptible to infections, eruptive and sometimes deadly diseases. This viral illness which can range from acute to chronic is characterized by skin nodules on the skin and other body regions [23]. Large skin nodules covering the entire body, fever, enlarged lymph nodes, appetite loss, decreased milk production, mild depression, reluctance to move nasal discharge and lachrymation are the

main symptoms of the condition [24]. Compared to adults, young calves often have more serious illnesses [25]. The degree of LSD's clinical symptoms varies depending on age, sex, breed, and host immunity [26]. Clinical signs of LSD can be classified as moderate or acute depending on the lump count, vector load, host vulnerability and immunity, management, and environmental factors. The symptoms begin with biphasic fever and escalate to a peak of 40–40.5 °C, which can last for three days [27].

In the moderate form emaciation, anorexia, depression, hypersalivation, nasal and oral discharge, decreased production and a small number of nodular lesions with a diameter of 1 to 5 cm are discovered two to three days after the fever first appears. Usually seen on the skin of the nose, neck, back, legs, scrotum, perineum, eyelids, lower ear, naso-lacrimal mucosa and tail, the nodules are spherical, elevated, hard, painful and hyperaemic [28]. In the acute form, which manifests as persistent high fever, severe anorexia, depression and often several uniform nodules over the animal body within two to three weeks of the disease beginning the symptoms are more severe. The elevated nodular lesions are typically 1–7 cm in size most frequently seen in the legs, head, neck, genitalia and perineum. A hemorrhagic rim can readily distinguish the lesions from the surrounding healthy skin [27]. Serious loss of milk production, miscarriage and chronic anestrous behavior may emerge in affected cows however, clinical enlargement of the scrotum is noticed in male animals due to testicular tissue involvement which may result in either temporary or permanent infertility [29].

Diagnosis & Management:

Presumptive Diagnosis: The clinical history, clinical symptoms, morbidity and death rate are the main factors that can support an assumption of LSD infection. When owners report cattle with distinctive necrotic skin nodules on the face, eyelid, neck, snout, nostrils, limbs, swollen lymph nodes, prolonged high fever and progressive emaciation a confirming diagnosis of LSD is recommended in every instance [30, 31]. Sometimes the clinical indications can be misinterpreted for other disorders that cause skin lesions necessitating laboratory confirmation. The diagnosis made after death is equally crucial. All things considered mouth mucous membranes, various gastrointestinal tract regions, the nasal cavity, the trachea and the lungs can develop pock-like lesions [32 33]. There is also a chance that the testicles and bladder will have lesions [33]. Larger mediastinal lymph nodes in dependent body parts are indicative of severe edema. The synovial fluid contains tenosynovitis and synovitis with fibrin production [34]. An animal's skin sample may be helpful for histopathological diagnosis [35].

Along with ballooning degeneration of the epidermal cell layers, eosinophilic intracytoplasmic inclusion bodies indicated in the keratinocytes, macrophages, endothelial cells and pericytes from skin nodules is regarded a pathognomonic lesion [36]. In the afflicted area other inflammatory cells like lymphocytes, macrophages and eosinophils are also invaded. Histologically widespread vasculitis, thrombosis, infarction and perivascular fibroplasia are seen [37]. Histologically severe coagulative necrosis (Zenker's necrosis) in subcutaneous muscle may be seen as a result of muscle involvement. Proliferation of lymph nodes is linked to oedema, congestion, bleeding and lymphoid tissue [38, 39].

Confirmatory Diagnosis: A great source of virus for confirmatory testing such as virus isolation and identification is a skin biopsy sample. When feasible samples should be gathered and sent to the laboratory via viral transport medium (VTM), such as phosphate buffer saline with 20 to 50% glycerol. For the diagnosis of viral agents, a number of laboratory diagnostic procedures are advised including electron microscopy, conventional or real-time polymerase chain reaction (PCR) and virus isolation.

The immunological response of an infected animal specific to LSDV can be detected by a variety of serological techniques including viral neutralization (VN), agar gel immunodiffusion (AGID), indirect fluorescent antibody test (IFAT), Western blot analysis and enzyme-linked immunosorbent assay (ELISA).40 Quantitative real-time PCR methods and traditional gel-based PCR methods are both regarded as quick, easy and sensitive assays. However, RT-PCR is thought to be more labor-saving, quicker and more sensitive than

conventional PCR [35]. Using a standard technique unique to the Capripoxvirus, PCR is the most widely used testing method for detecting the viral genome from skin lesions (scabs or nodules), unclotted blood, saliva, nasal swabs, semen, milk and tissue culture materials [41]. It is generally known that LSDV can be isolated and identified in cell or tissue culture. The most vulnerable cells for LSD virus proliferation are thought to be tissue cultures of bovine, ovine or caprine origin particularly primary or secondary cultures of bovine dermis cells, kidney cells (Madin-Darby Bovine Kidney, MDBK) cells, lamb testis (LT) cells etc. African green monkey kidney (Vero) cells and the chorioallantoic membrane of fertilized chicken eggs are suitable for LSDV development [35].

By employing a negative staining preparation method, the classic poxvirus virion can be identified via electron microscopy. Under an electron microscope, the capripox virion which has a brick-like morphology and is coated in short tubular elements, measures around 290 × 270 nm. It is well-suited for use with a pioloform carbon substrate. However, the effectiveness of this test method to differentiate LSDV from other orthopox species or variations is restricted [42]. Various serological tests can identify immune responses in infected or recovered animals; however, some methods are unable to differentiate LSDV from other capripoxvirus species [35].

Using the viral neutralization test (VNT), antibodies can be successfully detected in infected or re-covered cattle between two days and around seven months following infection. VNT's sensitivity has been reported to be about 70% and it has a limitation on antibody detection at low titers [43]. Using the capripox virus antigen fixed in the tissue culture plate, an indirect fluorescence antibody test (IFAT) can identify anti-body titers of up to 1/5000 in the serum of recovering animals [35]. It has the capacity to test a higher number of samples than VNT. However, it has a limitation of cross reaction with cowpox virus but not with parapox viruses [44]. The Agar gel immunodiffusion (AGID) test is a straightforward and reasonably priced method for identifying precipitated antigenic particles; however, it is not advised for use with live smallmouth virus (LSDV).

Because LSDV can provide false-positive results when it reacts with antibodies to the viruses that cause pseudo cowpox and bovine popular stomatitis [45]. For the purpose of detecting capripoxvirus antibodies, Western blot analysis is a sensitive and more specific configuration however, because pure antigen is needed for this method, it is costly and time-consuming [46].

For the purpose of detecting blood antibodies against LSDV, a new commercial enzyme-linked immunosorbent test (ELISA) kit that has been endorsed by WOAH is now accessible. In tests, ELISA exhibits excellent specificity and does not cross-react with parapox viruses nevertheless, it is unable to distinguish between serum and plasma antibodies against the LSDV, SPPV and GTPV viruses [45].

Particularly in prickle cells immunohistochemistry studies can reveal LSDV antigen particles within the cytoplasm of the epidermal basal cell layer. The reactions manifest as a granular, golden brown immunoperoxidase staining of viral antigen [47].

Differential Diagnosis: Differential diagnosis is crucial since there are a few other illnesses that cause skin lesions that resemble those caused by LSD.48 Bovid herpesvirus 2 (BHV2) which causes superficial skin lesions and has a brief course of illness is the cause of pseudo lumpy skin disease. Because the para poxvirus which causes pseudo cowpox primarily affects the teat and udder, it may be distinguished from LSD lesions.

Unlike LSD, vaccine viruses and cowpox viruses (Ortho poxviruses) also cause site-specific lesions on the teat, udder and muzzle. Because of its similarities to LSD, dermatologists brought on by infections of Hypoderma bovis and Dermatophilosis congolensis may be confusing. On the other hand, the animal's rear skin has exposed larvae due to the significant swelling and erosion of the sores. When the spinal cord is damaged the lower body and legs may become paralyzed [49]. The parasitic skin conditions onchocercosis and demidocosis are typically site-specific and are frequently characterized by the presence of parasites. PCR and other antigen- or antibody-specific tests can be used to differentiate between these disorders [50].

Sample Collection:
Preferred Sample Types:

A. Skin lesions and scabs

B. Saliva or nasal swabs

C. EDTA blood for PCR assay

D. Whole blood for serum samples

Pathogenesis: There is a significant range in the clinical presentation of LSDV infections, including both short and long-term subclinical infections as well as fatalities. The virus takes between 7-28 days to fully incubate after it has successfully infected its natural host [52, 53].

After the Incubation Period, Clinical Signs can be Categorized into four Distinct Phases: The first phase known as the acute phase, animals experience fever up to 41°C for approximately 7 days. They also experience anoxia, depression, lacrimation, increased nasal discharge, saliva secretions, lack of milk and multinodular lesions around the skin and mucous membrane. There are non-febrile cases. In phase two, there is a significant 3-5 times normal growth of the subscapular and precrural lymph nodes as well as an increase in multiple nodules most of which are on the head, neck, limbs, udder, mucosal membrane, nasal and oral cavities or plaques at the site of inoculation. The nodular lesions, which range in size and quantity from a few to several lesions encompassing the whole animal have a diameter of 0.5–5 cm. Nodules burst after one to two days, sometimes releasing virus as well depending on the virus concentration. It is occasionally discovered that vasculitis and lymphangitis are the causes of limb edema. In phase three, nodules lesions develop ulcerated and necrotic after two to three weeks. Moreover, beaded serum leaks, particularly from the extremities and results in discomfort, lameness and immobility. In severe cases, there is profuse salivation, lachrymation and nasal discharge; an ulcerative lesion occurs in the mucous membranes at numerous sites, such as the eye and nasal cavities. Animal secretions could include LSDV. In phase four, Complete healing of ulcerations, thickening of the skin and darkening of the lesion occur after at least one month. The immediate eruption of several confined skin nodules and a feverish response are the symptoms of LSD. Viral particles travel via blood and cause widespread lymphadenitis. Following the early-febrile state viremia lasts for around four days. Lesions are created in such sites after skin lesions brought on by the virus replicating in specific cells such as fibroblasts, pericytes and endothelial cells of lymphatic and blood arteries [5].

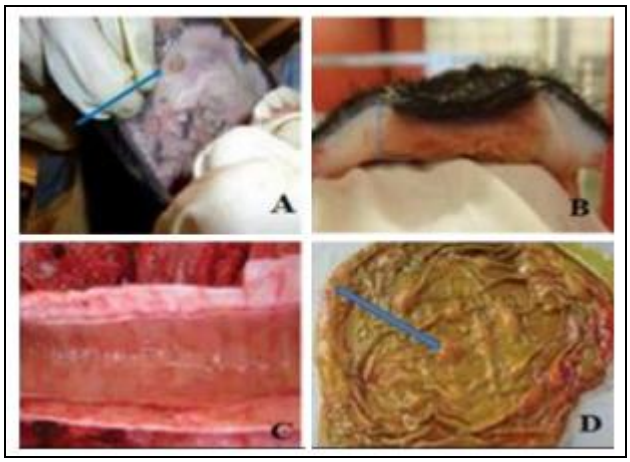

FIG. 6: INTERNAL LUMPY SKIN LESIONS: (A) ULCERATIVE LESIONS IN THE ORAL CAVITY AND (B) CROSS-SECTION OF SKIN LESION; (C) LESIONS IN THE TRACHEA (D) GALLBLADDER)

The immediate eruption of several confined skin nodules and a feverish response are the symptoms of LSD. Viral particles travel via blood and cause widespread lymphadenitis. Following the early-febrile state, viremia lasts for around four days. Lesions are created in such sites after skin lesions brought on by the virus replicating in specific cells such as fibroblasts, pericytes and endothelial cells of lymphatic and blood arteries. Virus replication, viremia, fever, cutaneous location of the virus and nodule growth happen after LSDV infection [55]. Following the virus's intradermal inoculation in an

experiment, the following outcomes were noted: 4 to 7 days after infection (DPI): localized swelling as 1-3 cm plaques or nodules at the site of inoculation; 6 to 18 DPI: viremia and viral shedding through nasal and oral discharge; 7 to 19 DPI: regional lymphadenopathy and the formation of generalized skin nodules; 42 days following fever: virus found in semen [56]. Animals that have recovered from the virus's natural infection have displayed a lifetime immunity. Because of the acquired maternal antibodies, calves from infected dams are resistant to clinical illness for about six months. Affected animals recover from the infection and there is currently no known LSDV carrier condition [57, 58].

Immunization: When an illness occurs naturally the immunity it produces usually manifests two weeks after the initial infection. Antibodies from experimental infection can be found 6 to 8 days after infection. Up to 5 months following infection, the peak level of immunity can still be seen 3 to 4 weeks later. Despite the fact that antibodies can stop extracellular organisms from spreading, the majority of LSDV are primarily intracellular. Humoral immunity is insufficient to stop viruses from proliferating inside of cells. As a result, cell-mediated immunity is necessary for animals to effectively control infection [59]. An animal's humoral immune response, which helps prevent sickness, can extend longer than 7 months after vaccination. It is advised that animals in endemic locations receive an annual booster shot [60]. In endemic locations, live attenuated vaccines have proven to be an effective means of immunizing against LSD. Cross-protection of immune response against antigenic homology of Capripoxvirus, encompassing Sheep Pox Virus (SPPV), Goat Pox Virus (GTPV), and Lumpy Skin Disease Virus (LSDV) was advantageous. There is a live attenuated vaccination for LSD eradication on the market. In nations where LSD outbreaks are common, the sheep pox vaccine from SPPV and GTPV is utilized as a control measure [52].

There are the following Kinds of LSD Vaccines: The most effective vaccine available right now to prevent LSD in cattle is the attenuated LSDV vaccine also known as a Neethling vaccine. In cattle, the potential for effective control success is 80%; in SPPV and LSDV outbreak areas, attenuated SPPV vaccines are appropriate; and in GTPV and SPPV outbreak areas, attenuated Gorgan GTPV vaccine is appropriate [53]. In nations that were formerly LSD-free, there is frequently serious concern about the safety of live homologous vaccinations. As per the vaccine manufacturers, it takes around 2 to 3 weeks for the formation of protective immunity after immunization [61]. Adverse reactions often manifest 1 to 2 weeks following immunization and consist of localized skin lesions at the vaccination site. After vaccination an animal's milk output may temporarily drop [62].

Control & Prevention: As of yet, no efficient remedy for LSD has been created. Antibiotics and anti-inflammatory drugs are used to treat symptoms. Effective control and preventive measures must be put in place in order to control the disease.

Limit Movement: To stop the spread of transboundary disease, it should be completely prohibited for animals with LSD to migrate. If animals with these kinds of lesions are found within a country, they need to be isolated for examination in order to stop the disease from spreading quickly.

Limit Vector Movement: The movement of vectors put on by the dominant winds may result in the spread of disease. The disease can also be avoided by using vector control techniques including using insecticides and setting up vector traps.

Vaccination: There is a live attenuated LSD vaccine available. Different strains of the LSD virus were used by companies to create vaccinations [63]. In addition to global climate change, poverty in farming communities in endemic locations, restricted access to effective vaccines and an increase in both legal and illicit live animal trading all appear to be contributing to the spread of capripoxviruses. In addition to mobility limitations and the removal of afflicted animals, vaccination is the only strategy that effectively controls the disease in endemic areas. LSD is solely treated symptomatically, using a combination of antimicrobials, anti-inflammatory, supportive care and antiseptic treatments intended

to prevent further bacterial problems [64] [65]. As control measures, the elimination of afflicted animals, limitations on movement and mandatory and regular vaccinations have all been suggested. But because arthropods are the disease's primary vectors, getting rid of the illness will probably be challenging and delaying getting rid of sick animals will make it more likely that LSD will spread. Furthermore, risk considerations must to be made in control operations. Veterinary professionals and livestock workers might diagnose clinical situation. Cross-protection is a known feature of capripoxvirus members. Therefore, cattle can be protected against LSD infection using both homologous (Neethling LSDV strain) and heterologous (Sheeppox or Goatpox virus) live attenuated vaccines [66, 67].

Live vaccinations effectively stop the spread of disease by eliciting a potent and durable immune response [68]. Live vaccinations however may result in skin sores and moderate illness with localized inflammation [69].

Treatment: The most effective way to stop the spread of LSD in cattle is to vaccinate them with a vaccine that has been shown to be effective, especially if done beforehand. It is very advised to get vaccinated against LSD when it is found in neighboring nations. An instantaneous response to an outbreak inside a nation is emergency vaccination. As soon as the first case is discovered, it needs to be done. Vaccinations for emergencies can be administered as targeted, ring, barrier or blanket treatments [73].

Vaccines: As of the now, the only vaccinations against the LSD virus are live attenuated vaccines. The development of inactivated vaccines is still underway. For cattle, there are three vaccination classes that provide effective protection against LSDV.

Attenuated LSDV Vaccines: Attenuated homologous LSDV vaccines are currently being produced by three different vaccine manufacturers. In cattle live attenuated LSDV vaccinations offer reasonable protection if 80 percent coverage can be achieved. The "Neethling response" is a modest side effect of attenuated LSDV vaccinations that has been documented. Furthermore, there was no discernible difference in routine culling, immediate culling or in-farm mortality for animals vaccinated for the first time between the pre- and post-vaccination periods following. Vaccination with the live attenuated Neethling LSD vaccine, according to recent studies. Neither was there a significant change in mortality or milk production during the 30-day period following vaccination.

Attenuated SPPV Vaccines: In areas where both SPP and LSD are prevalent cattle have been immunized against LSDV using sheep pox virus vaccines. Given that SPPV vaccinations are thought to offer only a partial protection against LSDV, vaccination choice should always be made in light of the vaccine's proven efficacy against LSDV as determined by a controlled challenge study.

Attenuated GPPV Vaccines: It has been shown that the commercially available strain of GTPV offers equivalent protection against LSD as the LSDV vaccinations. In those nations where GTP and LSD are overlapping, the GTPV vaccination is a good, affordable substitute.

Therapeutic agent for LSD Treatment: Currently, no effective treatment for LSD has been identified. Sick animals should be evacuated from the herd and treated with antibiotics, vitamins and other medications. These medicines reduce the risk of subsequent bacterial infections and fevers leading to better animal health. Most animals infected with LSD recover with fatality rates below 3%. Secondary bacterial infection may require more than 6 months for full healing.

CONCLUSION: Lumpy skin disease (LSD) caused by the Neethling poxvirus of the Capripoxvirus genus in the Poxviridae family affects cattle and can be fatal. Initially discovered in Zambia, LSD is now widespread across Africa and has spread to the Middle East, Asia and Europe. First recorded in Ethiopia in 1983. LSD is transmitted mainly through arthropod vectors with wildlife as a reservoir particularly in warm and rainy seasons. Severity varies by cattle breed, age and sex with young and lactating cows being more affected. Diagnosis is possible with molecular and serological tests.

Control measures include vaccination, restricting animal movement and culling infected animals.

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CONFLICT OF INTEREST: Nil

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