Review Article

Overview of Lumpy Skin Disease

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ABSTRACT

Lumpy skin disease, caused by the lumpy skin disease virus, is one of the major health issues affecting the livestock industry in most African countries. Although the virus is evacuated via various body secretions and excretions, including sperm, skin lesions are the most common sources of infection. As a result, susceptible hosts become infected with the virus. primarily through mechanical means from hematophagous arthropods such as biting flies, mosquitoes, and ticks Transstadial and transovarial persistence are also possible in various tick species. Following infection, under experimental conditions, typical lumpy skin disease lesions may erupt 7 to 14 days after infection. In natural cases, it takes 2 to 5 weeks. Lumpy skin disease is distinguished by a firm, skin nodules that are circumscribed, few (mild forms) to numerous (severe forms). can occasionally affect the mucous membranes of the urogenital system, respiratory system, and other internal organs. In turn, decreased milk production, abortions, temporary or permanent sterility, damage to the hide, and deaths will ensue, all of which add to a significant economic loss in nations that raise cattle. Therefore, the most efficient strategy to stop the spread of lumpy skin disease and its negative economic effects is through widespread vaccination along with other suitable control measures. This review was created with the intention of giving the most recent knowledge on the biology of the lumpy skin disease virus, its mode of transmission, and the clinical and pathological characteristics of the condition.

Keywords: Clinical symptoms; LSD; LSDV; lesions; pathogenesis; transmissions.

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INTRODUCTION

Capripoxvirus causes lumpy skin disease in cattle. It is distinguished by fever, enlarged lymph nodes, firm, circumscribed nodules in the skin, and ulcerative lesions, particularly of the mucous membrane of the mouth. Because of the decreased milk production, the disease is economically significant. yield, weight gain, infertility, and permanent damage to hides Lumpy skin disease is an OIE "List A" disease. because of the risk of rapid spread and serious consequences of losses. The European Economic Cooperation (EEC) / European Union was established in 1989. added LSD to Council Directive 82/894/EEC on animal notification diseases in the community, and thus it must be reported to all other member states and the commission within 24 hours of an outbreak. The disease's recent spread in disease-free countries emphasizes the importance of disease transmission, as well as control and eradication Lumpy skin disease virus (LSDV) is a double-stranded DNA virus with approximately 150 kilobase pairs (kbp) and relatively large sizes (230-260 nm) that is enclosed in a lipid envelope and belongs to the genus Capripoxvirus, which is genetically related to sheep pox (SPPV) and goat pox (GTPV) viruses. This virus, which affects domestic ruminants, is the most economically significant in the Poxviridae family. The virus's capsid or nucleocapsid is brick or oval in shape and contains the genome and lateral bodies.[1] However, the disease spreads beyond Africa to Madagascar and the Middle East, causing significant economic losses in the livestock industry. The incubation period in the field is thought to be two to five weeks, with lesions appearing four to twenty days after inoculation. Fever is the first symptom, followed two days later by the formation of nodules on the skin and mucous membranes.[2] Whole herd depopulation has been recommended on rare occasions, but in endemic situations, affected farms frequently isolate sick animals and provide supportive care, which may include wound dressings to prevent fly infestations and secondary infections Vaccination can be used in both reactive and proactive strategies. The majority of vaccines on the market today are live attenuated and contain either LSDV (homologous) or sheep and goat pox strains (heterologous) Heterologous vaccines are generally thought to be less effective but have fewer side effects, especially in European cattle breeds.[3]

HISTORY OF LUMPY SKIN DISEASE:

The first clinical signs of LSD were described in 1929 in Zambia (then Northern Rhodesia). Initially, LSD symptoms were thought to be the result of poisoning or hypersensitivity to insect bites. Between 1943 and 1945, the same clinical signs were observed in Botswana, Zimbabwe, and the Republic of South Africa, where the

infectious nature of the disease was recognized. LSD emerged as a panzootic in South Africa, affecting eight million cattle. The disease lasted until 1949 and caused massive economic losses. LSD was discovered in East Africa in Kenya in 1957. The disease was first reported in West Africa in 1974. In 1983, it was spreading into Somalia.[4] In Kenya in 1957 (MacOwan, 1959), Sudan in 1971, Chad and Niger in 1973, Nigeria in 1974, and Somalia in 1983, lumpy skin disease was identified. Despite all control and eradication efforts, the illness has grown endemic in Egypt since the initial outbreak in Ismailia in May 1988. In Israel, an LSD epidemic broke out in 1989. It was hypothesized that insect vectors transported by the wind or housed in the vehicles of cattle traders helped the disease spread from the Egyptian epidemic. 50 km around the outbreak, ring vaccination preprogrammed slaughter.[5]

Transmission

Cattle, water buffalo, and wild ruminants are all susceptible to lumpy skin disease. It appears that sheep and goats are not infected with the virus. LSDV can survive in the environment for long periods of time at ambient temperatures, particularly in dried scabs. It has been reported that the virus can survive in necrotic skin nodules for up to 33 days or longer in desiccated crusts for up to 35 days, and in air-dried hides for at least 18 days.[6] .The virus that causes lumpy skin disease is not completely understood. The mechanical spread of the LSD virus has primarily been associated with flying insects, and all available evidence confirms field observations that LSD epidemics occur during peak biting insect activity. The majority of cases are thought to be the result of arthropod vector transmission. Because of differences in the active vector species found in different situations, attack rates range from 10-15% to nearly 100% in different epidemics.[7]

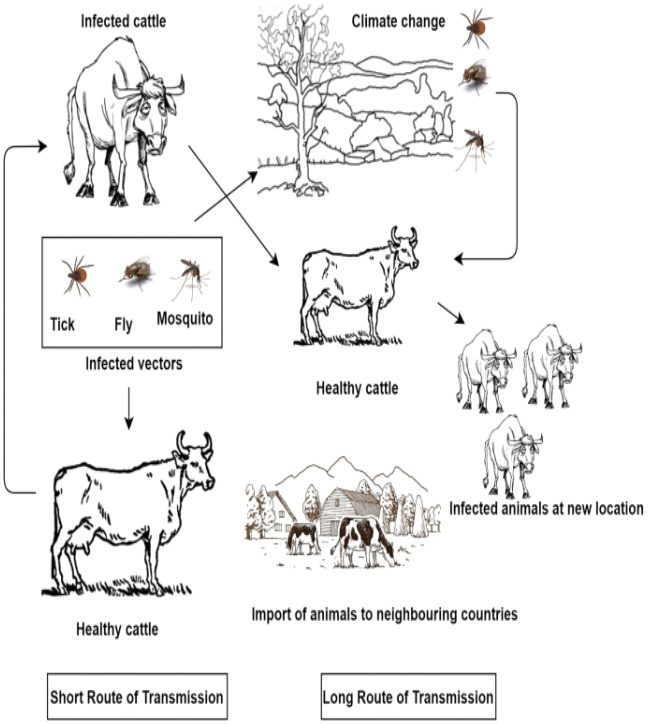

Figure 1: Transmission of LSD virus at a glance

The virus can spread through milk, nasal discharges, saliva, blood, and lachrymal secretions. According to a recent study, transmission via the intrauterine route could be a possibility.[8] The virus is thought to have originated from old skin lesions. The disease has been observed to appear following the seasonal rains in the majority of Sub-Saharan Africa. The population of various arthropod species is constantly increasing. Local disease spread in the presence of strict quarantines has been attributed to insect vectors flying in low-level air currents. The onset of frosts in South Africa and Egypt results in a significant decrease in the number of cases of LSD, which virtually disappears during the winter season before reappearing in the spring and summer. Despite total restrictions on animal movement, the disease spread throughout Egypt in the summer of 1989.[9]

Routes of Transmission

Direct Transmission

LSDV is inefficiently transmitted between animals through direct contact, according to experimental and field evidence, though more experimental studies with a sufficient number of animals and modern investigation methods are needed to demonstrate direct transmission. [10].Iatrogenic transmission caused by the use of contaminated needles in various animals, as well as transmission to suckling calves via skin lesions in the teat (Tuppurainen et al., 2017), have been proposed to play a role in the virus's spread. Despite this, it is widely assumed that LSDV is spread ineffectively through direct contact between infected and susceptible animals[11] as in mother to calf via skin lesions on the mother's udder and teats, or via contaminated milk, the prevalence of milk Arthropod vectors may differ by region due to climate, season, temperature, and humidity variations Vegetation, and thus vectors in various areas are required to be investigated for their participation in LSDV transmission varies according to their abundance as well as feeding habits Vector transmission that is indirect is dominant over short distances, but movement Infected animals may play an important role in the disease's long-distance spread. Weather extremes and Natural disasters may influence the spread of vector-borne infectious disease, implying a link between climate change with these occurrences According to a recent study, precipitation Temperature is positively related to the risks while wind has a negative impact on LSDV [12].

Role of Vector

LSDV is mechanically transmitted by blood-feeding biting arthropod vectors such as hard ticks, biting flies, and mosquitoes. This vector-related transmission is mechanical as opposed to biological. This distinction is significant because infectious organisms do not typically survive in vectors for long enough to multiply. The virus is transmitted mechanically via contaminated mouthparts of vectors, with no actual replication of the virus in arthropod cells or tissues[13]. The virus has also been recovered from

Stomoxys, Biomyia, Musca, Culicoides, and Glossina species, all of which feed voraciously on domestic cattle. Although the virus was found in Anopheles Stephens, Culex quinquefascuatus, Stomoxy calcitrans, and Culicoides tuberculosis attempts to mechanically transmit LSD to susceptible animals were unsuccessful. Sevik and Dogan recently investigated the potential role of Culicoides spp. in the transmission of LSDV and discovered that Culicoides punctatus could have played a role in transmitting LSDV during the 2014-2015 outbreak in Turkey. As a result, it is obvious that various arthropods that feed on cattle can transmit LSDV and spread the virus[14].

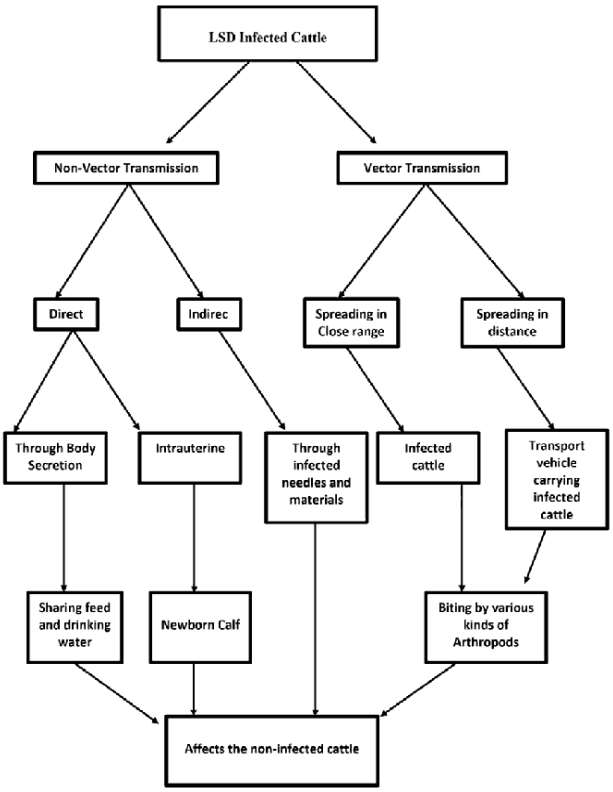

Figure 2: Mode of transmission of Lumpy skin Disease, with both vectors borne and non-vector borne [15]

Economic Impact

LSD is classified as a List A disease by the International Epizootics Office. the potential for rapid spread and the ability to cause significant economic loss (OIE, 2010). The disease's morbidity and mortality rates vary. a lot depends on the breed of cattle, the population's immunological status, as well as insect vectors involved in the Morbidity rates, which are generally varied. a lot depends on the breed of cattle, high. outbreaks, it was reported that there were more than 50%, despite the fact that mortality rates are usually lower. fewer than 10% of Cows appear in 1% to 7% of .cases could abort [16]The morbidity and mortality rate of LSD vary greatly depending on the presence of insect vectors and host susceptibility. When compared to indigenous African and Asian animals, high-milk-producing European cattle breeds are highly susceptible and severely afflicted. The disease's morbidity rate can range from 3% to 85%, with an average of 10% in endemic areas. Although the disease is not associated with high mortality (1-3%), the economic losses associated with LSD eruption are greater. It causes significant economic losses due to decreased feed intake, milk production, weight conversion, abortion and infertility, and damaged hides. Furthermore, the disease is a significant notifiable disease that impedes international trade. [17] The indirect economic impact of LSD includes trade restrictions, immunization, quarantine and treatment costs, feed and labor costs, stamping out, farm biosecurity, and so on. Farm owners must pay an additional fee for sic feed supplements. animals during the recovery period, as well as the extended period for fattening The cost of LSD in Jordan, which included treating affected cattle with broad-spectrum antibiotics and anti-inflammatory drugs, was estimated to be US$ 35.04.Sometimes a large number of affected animals must be stamped out, as was done in Greece and Bulgaria, where Bulgaria faced the highest economic disaster of around US$ 8000 per herd. As a transboundary infectious disease, the likelihood of rapid spread via the production marketing channel is high.

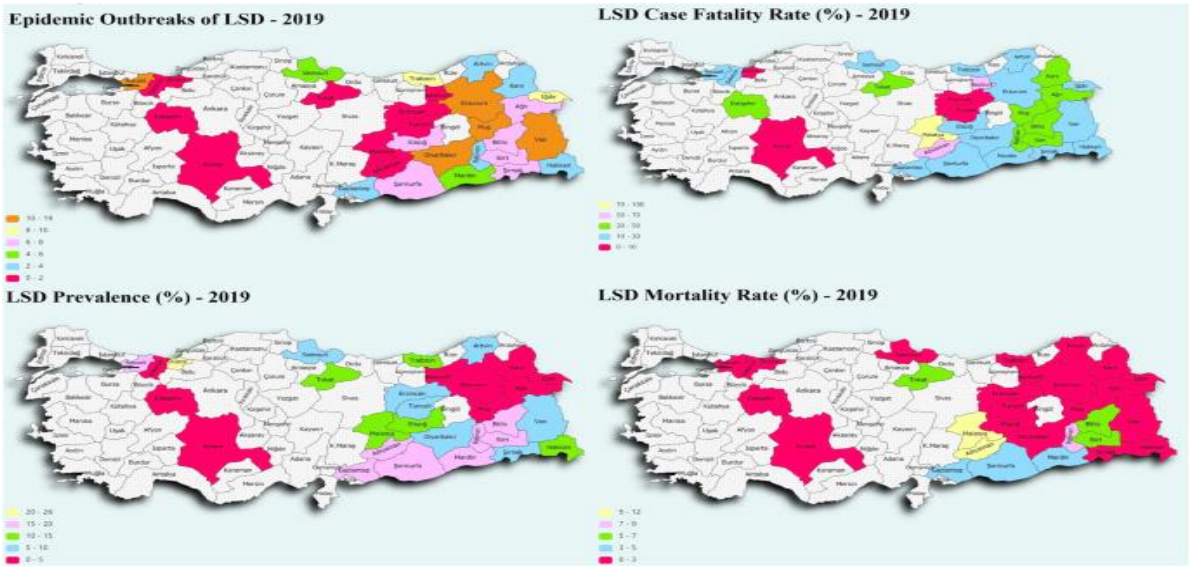

Figure 3: Map of epidemic outbreaks, mortality, LSD prevalence, and fatality rate by province

Pathogenesis

LSD's pathophysiology has been thoroughly described. firm, precisely define Skin nodules that are 5 to 50 mm in diameter form. There could be some of either a few in serious cases or a lot in mild cases. The thickness of the complete skin, subcutaneous tissues beneath the skin, and even the skeletal system There are muscles used. There may be soft, yellow-gray nodules or ulcerative lesions. also manifest on the muzzle, nose, and oral mucous membrane intestinal, respiratory, vulvar, and prepuce systems. Nodules on the skin are firm. and the chopped section has a whitish-grey look. The central necrotic eventually became A plug that could peel off and produce a granulating, raw sore. Edema subcutaneously of the ventral regions of the body, including the limbs, and widespread lymphadenopathy are traits of the acute illness [18] In experimental infections, intravenous, intradermal, and subcutaneous methods are employed. While only 40% to 50% of animals may experience localized lesions or severe generalized illness following intraepidermal inoculation no visible signs of any sickness. four to six weeks following vaccination, a little swelling at the injection site seven days, and regional lymph node localizedent occur following generalized us or intradermal LSDV vaccination of cattle [19].

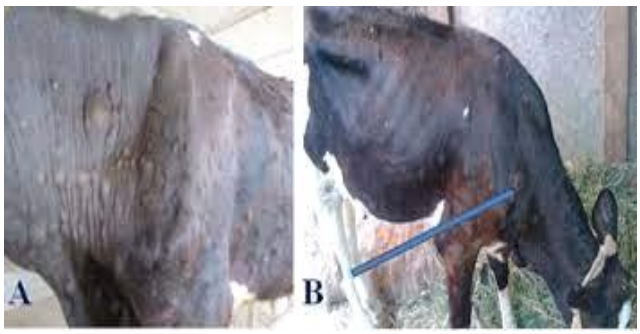

Figure 4: a lesion with few skin nodules in a mild form (A) and a lesion covering the entire body in a severe form (LSD) are characteristic nodular lesions that indicate severity (B).

Figure 5: Cow infected with LSD reveals multiple skin nodules

Very young calves, lactating cows, and underweight animals appear to suffer more severe disease in natural infection, which may be caused by defective humoral immunity. Using serum, antibodies may be detected at 21 DPI. Immunity following recovery from a natural infection, as determined by neutrneutralizationys is permanent; immunological calves of cows acquire maternal antibodies and are for roughly six months, clinical illness is resistant Eventually, Affected animals recover from the infection, and no known carrier condition exists. as LSDV [20]

Gross Pathological Findings

LSD causes gross lesions that are well-described. Ordinarily, skin nodules are homogeneous in size, firm, spherical, and elevated; nevertheless, some nodules may swell into vast, asymmetrical plaques that are bounded on all sides. In addition, a buildup of reddish material can be seen on the nodules' sliced surfaces. Subcutis layer edema and grey serous fluid. Resolution of the lesions is visible Deep ulcers can develop from indurated, also known as "sitfasts," seclusion, or indurated. The typical On the larynx, nasal cavity, and muzzle, circular necrotic alimentary lesions may be detected. uterus, forestomach, abomasum, trachea, bronchi, inner lips, gingiva, and dental pad testicles, udder, vagina, teats, and (Ali et al 1990). Unacceptably large regional lymph nodes 3-5 times larger than normal, oedematous, and with pyelonephritic foci, in local cellulitis in addition.[21]The musculature and fascia of the leg are also covered with the LSD characteristic nodular lesions, which are grey-white in color and bordered by red inflammatory tissue. Additionally, the lesions produce an ulcer that gradually heals by granulation since they are cut off from the healthy tissue and necrotic epithelium. Animals with severe infections may exhibit secondary bacterial pneumonia, tracheal stenosis, acute and chronic orchitis, mastitis with secondary bacterial infection, and other lesions in the female reproductive tract.[22]

Figure 6: Internal lumpy skin lesions: Ulcerative lesions in the oral cavity (A) and cross-section of skin lesion (B); lesions in the trachea (C) and gall bladder (D),

Histopathological Changes

Acute skin lesions' histological changes include vasculitis, perivasculitis, lymphangitis, thrombosis, edema, necrosis, and infarction. Neutrophils, macrophages, and occasionally eosinophils infiltrate necrotic areas before being replaced by lymphoblasts, lymphocytes, plasma cells, macrophages, and fibroblasts. In addition to macrophages, endothelial cells, period ema, and keratinocytes in the skin, eosinophilic intracytoplasmic inclusion bodies can also be found in smooth muscle cells, epithelial cells associated with hair follicles, skin glands, and endothelial cells [23].

Epidemiology

Epidemiological outbreaks of lumpy skin disease, which spread rather quickly over a region or nation, typically happen at regular intervals in endemic locations.LSD is not very contagious, according to laboratory and field studies. Natural epidemics can have morbidity rates of 3 to 85%. Only 40 to 50 percent of infected animals in infections that were created experimentally had clinical symptoms. Typically, less than 10% of people die. Despite the fact that no alterations between the capripoxvirus strains collected over 20 years were discovered, it was proposed that the Different pathogenic strains, the effectiveness of disease transmission via the vector, and infection routes could all contribute to variations in mortality and morbidity rates [24]. It can survive for months in a dark area, though, like an infected mammal, and it can endure drying and pH fluctuations, if not an extreme pH. a canopy over its host. Skin can retain LSDV.for around 42 days. The likelihood that the body protein with viral-type insertion in infected cells could shield the virion once the scab has healed. dissolved, however, it hasn't, been proven [25]

Geographical Distribution

LSDV was initially identified in 1929 in Zambia before being reported in various other parts of Africa. In Saudi Arabia, Lebanon, Jordan, Iraq, Israel, Turkey, and Iran, the illness has been confirmed. In addition to Albania, Kosovo, Serbia, and Montenegro, it has also expanded to Russia, Azerbaijan, Armenia, Greece, and Bulgaria since 2015. Because of this, it is important to take into account the increased risk of disease ououtbreaks cross the remainder of Europe and Asia. From 2014 to 2016, the OIE reported on the number of outbreaks of lumpy skin disease in various nations. For instance, in several Middle Eastern nations with wide borders, there were 6, 8, 1,294, 1, 16, 1, and 330 LSD outbreaks, which corresponded to Iran, Iraq, Turkey, Kazakhstan, Azerbaijan, Armenia, and Russia, respectively[26]. Madagascar and Africa both. Most African nations are home to it, and it can be found in a variety of ecoregions, with the exception of Libya, Algeria, Morocco, and Tunisia, which are still regarded as having no cases of the disease Outside of the African continent, outbreaks occurred in the Middle East in 2006 and 2007, in Mauritius in 2008, and Israel has also recorded LSD outbreaks (OIE, 2014b).[27]

Figure 7: Geographical distribution of LSD in the world

Morbidity and Mortality

As a result of uncomfortable swellings in the intestines, oropharynx, and subsequent bacterial infections, Morbidity, hunger, and dehydration are the main causes of cattle mortality. N.odular infinflammatory ellings that cause permanent damage to the skin cause it to, become necrotic and flake off, leaving a raw, sore lesion overall body weakness and lesions that make mobility difficult cause a loss of strength. Emaciation, cachexia, and wasting caused by feeding disruption led to the loss of beef Anorexia also affect the ability to produce milk Fever and intrauterine infections both contribute to abortions. Reproduction is hampered by painful sores on a bull's sheath and weak females[28]. The rates of morbidity and mortality during LSD outbreaks vary enormously. It depends on the following variables: geographical location, climate, management practices, nutritional state, and overall health of the affected breed of cattle; population size, immunological state, and the spread of potential insect vectors in different environments; viral virulence. Between 5 and 45% of LSD users experience morbidity However, the morbidity raterare1 of 5% of regarded as more typical. Enhanced rates have been observed in zoonosis outbreaks in Southern, West, and East Africa, as well as that. even if there may be instances of the same epizootic at far lower rates. Additionally, hig Also reported for Oman were death and morbidity rates of 30-45% and 12%, respectively. a herd of Holstein cattle on a farm in 2009 [29]. Similar to this, during 2014 and 2015, additional cases of LSDV were recorded in Iran, Azerbaijan, Iraq, Greece, and Cyprus. The first LSDV incursion was recorded in the northern Caucasus region of Russia in 2015 and in the European Union in 2016 [30].

Prevention and Control

Unfortunately, the lumpy skin condition cannot yet be treated with any specific antiviral medications. To cure secondary bacterial infections or to increase the animal's appetite, sick animals may be separated from the herd and given supportive care such as antibiotics, anti-inflammatory medications, and vitamin injections. Since it is believed that blood-feeding insects are the main source of LSD transmission, quarantine and mobility restrictions alone are not particularly efficient at containing them unless they are accompanied by widespread immunization· [31,32].In order to minimize additional bacterial problems, LSD is solely symptomatically treated with a combination of supportive care, anti-inflammatory medications, and antiseptic treatments. As control measures, the killing of afflicted animals, enforcing mobility restrictions, and mandatory, ongoing immunization have all been suggested. Eliminating the disease, however, is expected to be challenging due to the role of arthropod vectors, and any delays in removing infected animals raise the chance of LSD spread.[33].In endemic areas, live vaccines aid in the control of lumpy skin disease losses. According to the OIE, four people are still alive. Capripoxvirus attenuated strains have been used as vaccines for LSD management This is a strain of Kenyan sheep and goat pox virus that has been passed through lamb 18 times. Yugoslavian RM 65 sheep pox strain, testis (LT) cells or fetal calf musclfetals Romanian sheep pox virus strain and South African lumpy skin disease virus strain 60 times through lamb kidney cells and 20 times through the chorioallantoic membranIt[34]. is important to carefully follow and put into effect professional advice and vaccination recommendations. As the body's defensive mechanisms are compromised, antibiotics are also given to prevent additional bacterial complications. This can delay the afflicted animals' full recovery. The following vaccines were used to protect the animal:

- Homologous live attenuated virus vaccine (Neethling strain: conferred immunity lasts up to three years
- Heterologous live attenuated virus vaccine (similar to sheep or goat pox vaccine, but can cause localized sometimes severe reactions). This vaccine is not recommended in countries free of sheep and goat pox because live vaccines could otherwise infect susceptible sheep and goat populations.
- There are no new generation recombinant capripox vaccines on the market.

Clinical Signs

Both naturally occurring and artificially synthesized LSD exhibit some clinical symptoms. The disease's progression could be acute, subacute, or chronic. 40 to 50 percent of experimentally widespread skin lesions appear on infected animals; many cases are subclinical Under natural conditions, the incubation period for LSD is 2-4 weeks, while in experimentally produced sickness, it is between 4 and 14 days [35.36]. Lachrymation and nasal expulsion are also clinical signs, as are enlarged subscapular and pre-femoral lymph nodes and decreased milk yield. Infected animals may also experience abortion, prolonged fever, infertility, emaciation, and lameness.

Diagnosis

Based on the clinical indications, the condition can be presumed to exist. However, a slow-moving, mild illness may be challenging to identify and quickly spread Laboratory techniques are required to validate the diagnosis. LSD can be diagnosed in a laboratory by identifying the substance [37.38.] Bovine herpes mentalities, stomatitis, vaccine virus, pseudocowpox, and vaccinia virus should all be considered in the differential diagnosis of the illness. Insect bites, cutaneous tuberculosis, and dermatophilosis ring worm. Additionally, serological tests using enzyme-linked immunosorbent assays might be used to identify the illness. indirect fluorescent antibody test, ELISA assay (IFAT), virus, and indirect immunofluorescence test Serum Neutralization and the Neutralization Test (VNT) Test (SNT) (SNT). The ELISA, however, has been experimentally verified, demonstrating greater sensitivity in compared to IFTA or VNT, and specificity. A relatively recent test known as immuno-peroxidase Monolayer Assay (IPMA) has been noted as a potential application in the diagnosis of LSD. It is an affordable and practical exam with better resistance to low biosafety levels and greater sensitivity and specificity compared to VNT and commercial ELISA. During an autopsy, tiny nodules resembling pox knobs are visible in the mucosal membrane of many cavities and viscera, including the tongue, oro-nasal cavities, etc [39].

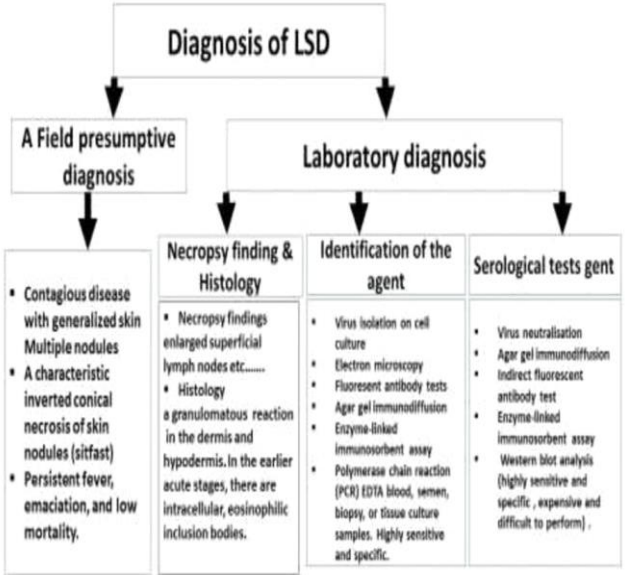

Figure 4: The diagnostic procedures of the LSD [40.]

Differential Diagnosis

It is possible to mistake LSD for the skin lesions caused by pseudo lumpy skin disease (caused by the bovine herpesvirus 2), insect bites, Demodex infection, onchocerciasis, besnoitiosis, and dermatophilosis. diseases that can be mistaken for LSD and cause mucosal ulcers include feverBovine LSD can occasionally imitate allergic symptoms like urticaria and bug bites [41]. The differential

diagnosis for LSD includes pseudocowpox, besnoitiosis, demodicosis, vaccinia virus, bovine papular stomatitis, dermatophilosis, vesicular stomatitis, cutaneous TB, photosensitization, onchocerciasis, and ringworm.[42]

Risk Factors

Warm and humid weather, circumstances that foster a plethora of vector populations, such as those seen following seasonal rains, and the introduction of additional animals to a herd are risk factors linked to the spread of LSD. Other risk factors that may enhance the occurrence of the disease include the herd size, vector populations, distance to the lake, herd migration, transport of diseased animals into disease-free areas, common pastures, and water sources.[43] Most clinical symptoms are most commonly seen in young animals who are clinically impacted. Traditional calf care techniques, however, that separate calves from the herd, may have reduced the danger of calves becoming infected. A specific passive immunity that is protective can be acquired by calves from their dam in an endemic location. Because there is a strong immunity to LSDV that lasts for about three months, an animal that has recently recovered from an attack is not susceptible to it .[44]

CONCLUSION

Prior to now, only sub-Saharan Africa has been affected by lumpy skin disease (LSD), a vector-borne illness brought on by the genus CPV. It is, however, now gradually encroaching on new lands, including Europe. Clinically, the illness manifests as discrete nodular lesions, which primarily affect the skin and underlying tissues of infected animals. Occasionally, other body organs, like as the conjunctiva, alimentary, respiratory, and urogenital tracts, may also CPVffected Because of the decreased hide quality, chronic debility, decreased milk output, weight loss, infertility, miscarriage, and mortality caused by the lesions, there are enormous economic losses. A dramatic impact on rural livelihoods that are heavily dependent on cattle, as well as major productivity losses, may also result from this. Since the disease's presence has led to tight trade restrictions, the effects are equally catastrophic on a national level. Therefore, the following advice is provided in order to recognize these alarming situations: In addition to the conventional clinical indicators, it is important to identify the clinical-hematological and biochemical profile of calves affected by LSD For control measurements, accurate on-time diagnosis is required. In endemic locations, immunization with a homologous strain of the LSDV is required annually. During the peak of bug activity, vector management and animal movement restrictions are crucial LSDV detection is necessary for bulls used for breeding.

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