IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 5, Issue 2, December 2025
Impact Factor: 7.67

Lumpi Skin Disease Virus in Animal

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Abstract: *Lumpy Skin Disease (LSD) is a significant transboundary viral infection caused by the Lumpy Skin Disease Virus (LSDV) of the genus Capripoxvirus and family Poxviridae. Primarily affecting cattle and water buffalo, the disease is vector-borne, transmitted mechanically by blood-feeding arthropods such as mosquitoes, ticks, and flies. LSD is characterized clinically by fever, lymphadenopathy, and the eruption of circumscribed cutaneous nodules, which may lead to ulceration and necrosis. The disease imposes severe economic burdens on the livestock industry due to emaciation, substantial loss in milk production, hide damage, infertility, and mortality rates up to 20%. Historically endemic to Africa, LSD has recently spread to the Middle East, Europe, and Asia, posing a global threat to animal health and international trade. This review synthesizes current knowledge on the etiology, epidemiology, pathogenesis, and clinical manifestation of LSD. It further discusses diagnostic methods, including Polymerase Chain Reaction (PCR) and Transmission Electron Microscopy (TEM), and evaluates control measures. As no specific antiviral treatment exists, management focuses on symptomatic support to prevent secondary bacterial complications and the implementation of effective vaccination programs using attenuated homologous or heterologous strains to achieve herd immunity.*

Keywords: Lumpy Skin Disease (LSD)Lumpy Skin Disease Virus (LSDV) Capripox virus Poxviridae Cattle and Water Buffalo Vector-borne Disease Livestock Epidemiology Viral Pathogenesis Vaccination Economic Impact

I. INTRODUCTION

Lumpy skin disease (LSD, Pseudo-urticaria, Neethling virus disease, exanthema nodularis bovis, and knopvelsiekte) is an infectious disease. It is caused by a virus (LSDV) in the family Poxviridae, genus Capripoxvirus. It is closely related antigenically to sheep and goat pox virus. However, these viruses cannot be differentiated using routine serological test [1]. LSD is a disease of cattle and water buffalo. It is a vector-borne disease transmitted by different biting and biting blood feeding arthropods. LSD Causes considerable economic losses due to emaciation, damage to hides, infertility, mastitis, loss of milk production, and mortality of up to 20%. The severity of clinical signs of LSD depends on the strain of capripoxvirus and the host cattle breed [2]. Until 1989, Lumpy skin disease is limited to African continent. However, the disease is moved outside Africa to Madagascar and the Middle East and causes serious economic loss to the livestock industry. The incubation period in the field is believed to be two to five weeks, and lesions first appear at the inoculation site in 4 to 20 days. Fever is the initial sign that is followed within two days by the development of nodules on the skin and mucous membranes [3]. A diagnosis of LSD is building upon the basis of the typical clinical patterns (morbidity and mortality). A confirmed diagnosis is based on transmission electron microscopic (TEM), immune peroxidase (IMP) staining, antigen trapping enzyme-linked immunosorbent assay (ELISA) and a polymerase chain reaction (PCR) test. There is no specific treatment for LSD. However, supportive treatment should be given to infected animals to relieve clinical signs and to control all secondary complications. Immunization of the susceptible animals is the effective methods to control the disease in South Africa, and the effective vaccines are produced from the Neethling strain virus. [4].

Lumpy skin disease is an infectious viral disease caused by Lumpy skin disease virus (LSDV) of Capri pox virus genus, subfamily Chordopoxvirniae, family Poxviridae. The disease is known by various names such as ―LSD‖, ―Pseudo-urticaria‖, ―Neethling virus disease‖, ―exanthema nodular is bovis‖, and ―knopvelsiekte‖ [5]. LSD is a non-zoonotic, vector borne and transboundary disease with limited host range and currently restricted to

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ruminants viz. cattle and water buffaloes. The arthropod vectors responsible for the disease spread include biting flies, mosquitoes and ticks. Natural infection of sheep and goat has not been reported even in close contact with infected cattle and buffaloes but skin lesions have been seen after experimental infection in sheep, goat, giraffe, Giant gazalles, impalas LSD is associated with high morbidity but low mortality. The disease is characterized by fever, lymph node swelling, circumscribed nodules on skin causing severe emaciation, reduction in milk production, infertility. Overall, it affects the economic value of animal as it will affect the meat and milk production, hide quality, draft power of animals and reproductive efficiency (abortion and infertility) [6]. It is anotifiable disease having devastating effect on international livestock trade also. The disease is endemic in African countries but recently the disease has been reported from new territories around the world. The first case of LSD was reported from Zambia in 1929 and then in southern and northern African countries. Later on, it spread to Israel, Kuwait, Oman and Yemen. According to OIE, at present this disease is prevalent in countries including various African, European and Asian countries. The reasons of the disease spread to India are unknown but it may be due to livestock movement across international borders or may be due to vectors movement from the neighbouring countries. In recent years, LSD has been reported from countries neighbouring India like China and Bangladesh. Therefore, understanding the epidemiology of exotic diseases becomes necessary for timely planning the effective disease management. This review summaries the latest updates about the LSD [7].

Figure No. 1: Nodules on the skin of the animals Source: OIE [8], Iowa State University (B) and Gari et al. [9] (A).

1.1. Overview of Lumpy Skin Disease (LSD)

Lumpy skin disease (LSD) is a trans-boundary animal viral disease which causes considerable financial losses to the livestock industry. It was observed for the first time in Zambia in 1929 and spread rapidly in the cattle population across African countries [reviewed in reference. Until 1984, LSD was maintained within the countries of subSahara Africa. The firstconfirmed transcontinental spread of LSD from the African to Middle-East Asian countriesoccurred when the disease was reported in Israel in 1989. In 2013, it was confirmed in Turkey. By 2015–16, the disease was reported in South-East Europe, the Balkans and the Caucasus. Of late the disease was reported for the first time from India in November 2019 [10]. Clinically, LSD has been reported in cattle only. The incubation period of the disease is 4–12 days. The clinical picture starts with fever (40–41.5˚C) which persists for 1–3 days. This is accompanied by increased nasal and pharyngeal secretions, lachrymation, enlargementof lymph nodes, anorexia, dysgalactia, general depression and a disinclination to move. The skin nodules appear within 1–2 days, which gradually become harder and necrotic thereby inducing severe discomfort, pain and lameness. In 2–3 weeks, the nodules either regress, or necrosis of the skin results in hard, raised areas (sit-fasts) clearly separated from the surrounding skin. Some of the sit-fasts may slough away, leaving a full skin thickness hole in the skin which usually gets infected by bacteria or becomes liable to myasis.

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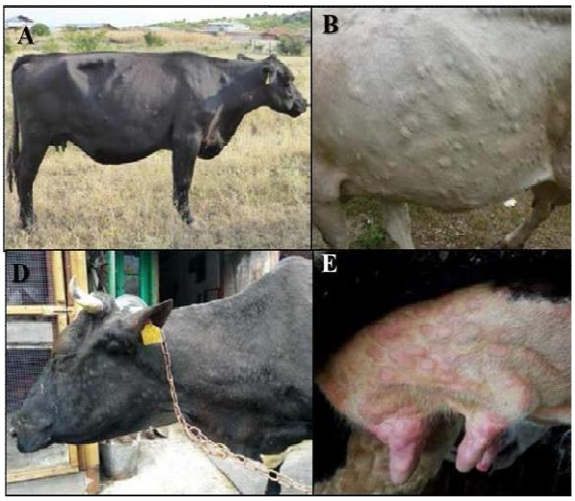

Figure No.2 Mild LSD With Full Body Lesoin 1.2. Virus and Classification

Lumpy skin disease virus (LSDV) is a virus in the family Poxviridae, subfamily Chordopoxviridae, genus Capri pox virus. The genus Capripox virus comprises three viruses; SPPV, GTPV, and LSDV. Lumpy skin disease virus is large–sized (230–260 nm) enclosed in a lipid enveloped with a genome of approximately 150 kilobase pairs (kbp) and shared 97% identity in the nucleotide sequences with SPPV and GTPV genome. The LSDV genome included at least 146 putative genes, which displayed proteins that play roles in virion structure, DNA replication, transcription and metabolism, protein.

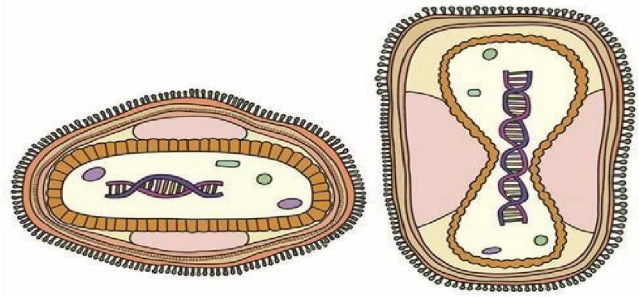

Figure No. 3: Schematic diagram of the poxvirus structure

1.3. Transmission, Spread, and Virus Stability

Lumpy skin disease is a vector-borne disease transmitted by mosquitoes (Aedes aegypti, Anopheles stephensi, Culex quinquefasciatus, and Culicoides nubeculosus), ticks (Rhipicephalus appendiculatus, Rhipicephalus decoloratus, and Amblyomma hebraeum), and Diptera (Haematopota spp. and Stomoxys calcitrans). The LSDV can survive in skin nodules for 1 month and at least 3 weeks in air–dried hides. The virus is excreted in the blood, nasal secretions, saliva, ear notches, semen, and milk and can be transmitted to suckling calves [16]. In general, vectors enhance the distribution of LSDV by mechanical and biological transmissions. Many studies have reported that after blood-sucking vectors (mosquitoes, ticks, glimpse, and flies) take a blood meal from infected cattle (viremia stage), the virus can propagate and shed in the salivary glands, head, body, and feces of insects. This allows the infected insects to become a reservoir for further transmission. The infectivity of LSDV has been studied in both egg and juvenile of ticks (R. decoloratus) in which it was found that the disease can be transmitted by transovarial transmission.

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II. HISTORY

The clinical syndrome of LSD was first described in Zambia in1929. Initially, it was considered to be the result of either poisoning or a hypersensitivity to insect bites. More cases also occur between 1943 and 1945 in Botswana (Bechuanaland), Zimbabwe (Southern Rhodesia) and the Republic of South Africa. A panzootic infection in South Africa affected approximately 8 million cattle till 1949 and consequently incurred enormous economic losses [19]. LSD was first found and diagnosed in East Africa (Kenya) in1957, Sudan in 1972, and in West Africa in 1974. Tanzania, Kenya, Zimbabwe, Somalia and the Cameroon, also reported an out breaks of epizootic LSD between 1981 and 1986 with mortality rates of 20% in affected cattle. The disease was restricted to some countries in sub-Saharan Africa between1929 to 1986 [20]. The LSD also reported in Asian countries such as Kuwait in 1986. Later on, other countries such as United Arab Emirates, Arab Republic of Yemen, and Democratic People's Republic of Yemen also confirmed or suspected some cases ofLSD [21]. The expectation of the travelling and invasion of the LSD to free neighbours countries are possible. LSD may invade north and west from Turkey into Europe and the Caucasus and East to Central and South Asia. In addition, Russian Federation to the north and Bulgaria and Greece to the west are considered to be at-risk countries [25].

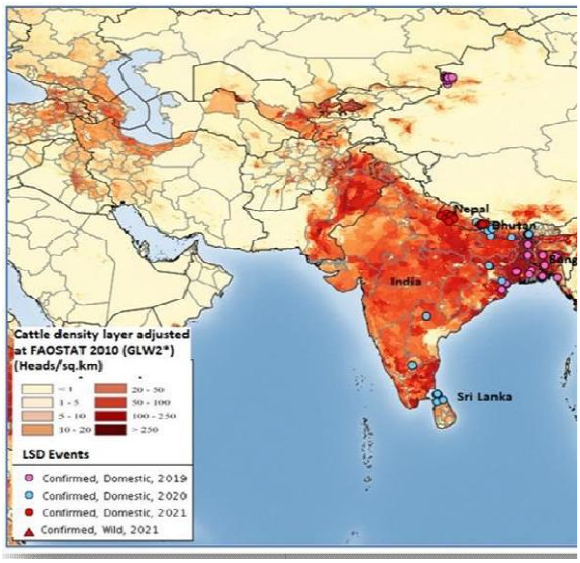

Figure No. 4: LSD outbreaks in Asia from 2019 to 2021

III. ETIOLOGY

Mature capri pox virions have a more oval profile and larger lateral bodies than ortho pox virions. Their average size is 320x 260 nm. The LSD virus grows and propagated to a high level in a wide variety of cell cultures such as lamb and calf kidneys, adrenal and thyroid glands, muscle and testes. Sheep embryonic kidneys and lungs, rabbit fetal kidneys and skin, chicken embryo fibroblasts, adult vervet monkey and baby hamster kidneys and primary cell cultures of bovine dermis and equine lungs are also used for that purpose [26]. The development of cytopathic effects may take up to 11 days during primary isolation. There is only one serotype of LSD virus which is very closely related serologically to the virus of sheep and goat pox (SGP), in which it cannot be distinguished easily by routine virus neutralization tests. It has been found that LSD virus strains are essentially identical with each other and with a Kenyan strain (O 240/KS sheep and goat pox virus (SGPV) using restriction endonuclease studies of capri pox virus.

DOI: 10.48175/568

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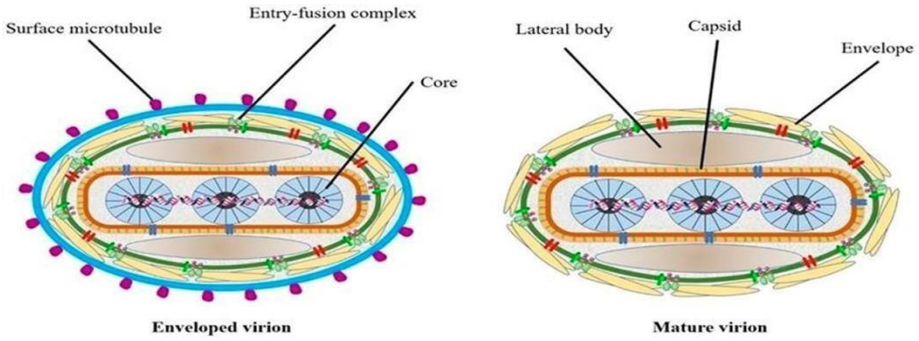

Figure No. 5: A diagram illustrating the predicted structure of LSDV [28].

IV. EPIDEMIOLOGY AND TRANSMISSION

Most of LSD virus infections are thought to be transmitted through insects. Pox viruses are highly resistant and can remain viable in infected tissue for more than 120 days or probably longer time The virus is also found in blood, nasal discharge, lacrimal secretion, semen and saliva, which considered as main sources for LSD transmission. The virus transmission is likely to be mechanical, although there is no enough data demonstrating a particular insect species as a vector of LSD virus transmission. However, the virus has been isolated from Stomoxys, Biomyia fasciata, Tabanidae, Glossina and Culicoides species. The role of all these insects in the transmission of LSD remains to be evaluated in the laboratory and under field conditions [29].

V. INCUBATION PERIOD

The incubation period is ranged between 2 to 5 weeks in the field while after experimental infection by intradermal inoculation a skin lesion containing virus more probably develops at the injection area within 1-3 weeks [31].

5.1. Host susceptibility

Host susceptibility, dose and route of virus inoculation affect the severity of disease. Both male and female, all age groups and various species and breeds of cattle are considered to be at risk and can get LSD infection, which may follow by severe and serious complications. Among more famous breeds, Bostaurus breeds of cattle are more susceptible for the disease than Bosindicus breeds, although younger animals often affecting and show more severe disease than adult ones. The disease is started with the onset of fever almost 1 week after entering the virus. It has been found that infection with LSD virus is not leading to the characteristics in cattle [32].

5.2. Pathogenesis

Intradermal or subcutaneous inoculation of cattle with LSD virus results in the swelling at the site of injection after about 1 week and enlargement of the regional lymph nodes, while generalized eruption of skin nodules usually occurs 7-19 days after injection Following intradermal inoculation of cattle with LSDV, about 40-50% of animals will only develop a localized lesion at the site of inoculation or no clinical signs at all, whereas those that have been inoculated intravenously are more inclined to develop generalized lesions and more severe disease . LSD virus in experimentally infected cattle was demonstrated in saliva 11 days after the development of fever, in semen after 22 days, and in skin nodules after 33 days, while the virus not found in urine or faces. Viremia occurred after the initial febrile reaction and persisted for at least 4 days [33]. Various types of cells such as Pericytes, fibroblasts, epithelial and endothelial cells can be infected by the virus. Viral replication in pericytes, endothelial cells and probably some cells in blood vessel and lymph vessel walls results in severe vasculitis and lymphangitis in affected areas. In severe cases infraction may also result. Viral concentrations at the skin nodules, lymph nodes, liver, kidneys, skeletal muscle, saliva and semen of infected animals however, have not been determined. Immunity after recovery from a natural infection is life-long in most survivor cattle; calves from immunized dam acquire maternal antibody and are resistant to clinical disease for about 6 months [34].

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5.3. Clinical signs and pathological observations

Skin nodules about 0.5-5 cm in diameter in whole skin or subcutaneous tissue and swollen superficial lymph nodes especially subscapular and precrural lymph nodes are the main symptoms of LSD infection in most animals. These nodules can also affect the nasal, oral, ocular, and genital mucosa. Their number may range from a few to several hundreds. Cutaneous lesions may resolve rapidly or may indurate and persist as hard lumps, or become sequestrated to leave deep ulcers partly filled with granulation tissue, which often suppurates. Papules most easily seen in hairless areas of perineum, udder, inner ear, muzzle and eyelids, which leads to the development of ulcerative lesions with excessive salivation, lacrimation and nasal discharge that may contain LSD virus [35]. Some of the infected cattle may develop oedematous swelling of one or more legs and show lameness. This virus infection is more severe in cows at the peak of lactation and causes a sharp drop in milk production due to high fever (40-41°C) and secondary bacterial mastitis. If extensive necrosis occurs in the upper respiratory tract, secondary infected necrotic tissue may be inhaled, resulting in pneumonia. Stenosis of the trachea following healing of lesions with scar tissue information few weeks or even months after infection has been described [36].

VI. DIAGNOSIS FOR LUMPY SKIN DISEASE

At present time, no commercial diagnostic test kits for LSD virus detection are available yet. Thus, the tentative diagnosis of LSD is usually based on the characteristic clinical signs, differential diagnosis, and the clinical diagnosis which is confirmed by laboratory tests using conventional polymerase chain reaction (PCR) techniques. LSD should be suspected clinically when there are characteristic skin nodules, fever and enlargement of superficial lymph nodes. The lumps on the skin follows within 2 days which may appear anywhere on the body from the nose to the tail. Same characteristic lesions appear in the mucosa of the mouth, vagina and conjunctiva. A purulent nasal and ocular discharge are not rare. Laboratory confirmation of LSD virus can be done very rapidly using a PCR method specific for Capri poxviruses or by the demonstration of typical Capri pox virions in biopsy material or desiccated crusts using the transmission electron microscopy (TEM). Routine diagnostic techniques are described in the OIE Manual of Diagnostic Tests and Vaccines. Capri poxvirus is distinguished from Para poxvirus, which causes bovine popular stomatitis and pseudo cowpox, but cannot be distinguished morphologically from cowpox and vaccine virus infections of bovine. Confirmation of LSD in a new area requires virus isolation and identification. LSD virus can propagate in bovine, caprine or ovine cell cultures; especially lamb testis cells. The cytopathic effects and the intra-cytoplasmic location of inclusion bodies can be used to distinguished LSD virus from the herpes virus, the causative agent of pseudo lumpy skin disease. Recently, direct immunofluorescence, virus neutralization test, enzymelinked immunosorbent assay (ELISA) and immune blotting (Western blotting) can be used for the identification of LSD virus antigens in infected animals. However, the immunity to LSD infection is predominantly cell mediated, thus the virus Neutralization test is not sufficiently sensitive to identify animals with LSD virus due to low level of neutralizing identify development. Genome detection using Capri pox virus-specific primers for the attachment protein and fusion protein a gene has been reported, and several conventional and real-time PCR methods have been established to be used on blood, tissue and semen specimens. Crossreaction occurs with bovine papular stomatitis and pseudo cowpox virus when agar gel immune diffusion test is used. Indirect Fluorescent Antibody Test (IFAT) demonstrated to be suitable for use in retrospective serological surveys in a study carried out in Ethiopia, and it was evaluated test for accuracy [37].

6.1. Differential diagnosis

Misdiagnosis of skin lumps and misreporting of infection have probably been common over the years due to veterinarians not having previous experience of the disease. Although severe LSD is highly characteristic, but milder forms can be confused and misdiagnosed with numerous diseases and infections such as pseudo lumpy skin disease (Bovine Herpes virus), bovine papular stomatitis (Para poxvirus), pseudo cowpox (Para poxvirus), Vaccinia virus and Cowpox virus (Orthopoxviruses) infections, dermatophilosis, insect or tick bites, besnoitiosis, rinderpest, demodicosis, Hypoderma bovis infection, photosensitization, urticaria, cutaneous tuberculosis and onchocercosis [38].

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VII. SYMPTOMS OF LUMPY SKIN DISEASE

Lumpy Skin Disease (LSD) is a viral disease primarily affecting cattle, caused by the Lumpy Skin Disease Virus (LSDV), a member of the Capripoxvirus genus in the Poxviridae family.

The disease is characterized by a variety of symptoms, and its clinical presentation can range from mild to severe. Below are the main symptoms:

7.1. Skin Lesions

• Lumps or nodules: These are the most characteristic signs of LSD. They appear on the skin, particularly on the head, neck, limbs, and body. The nodules are typically round, raised, firm, and range in size from a few millimeters to several centimeters.

• Ulceration or necrosis: In severe cases, the nodules may ulcerate, leading to the formation of scabs or necrotic areas, particularly in more advanced cases.

7.2. Fever

A sudden rise in body temperature is common, often reaching 40°C or higher. This fever can last for several days.

7.3. Swelling

• Edema: Swelling of the skin, particularly around the eyes, neck, and brisket, is common. This swelling can be particularly noticeable in the face and limbs.

• Lymph node enlargement: Lymphadenopathy (swollen lymph nodes) is frequently observed, especially in the neck and jaw area.

7.4. Respiratory Signs

• Nasal discharge: Clear or serous discharge may be present, which can later become mucopurulent in more severe cases.

• Coughing: Respiratory distress can sometimes occur, particularly in the case of secondary bacterial infections.

7.5. Digestive Symptoms

• Reduced appetite: Infected animals may show a lack of appetite or reduced feed intake, leading to weight loss.

• Diarrhea: This is less common but can occur in severe cases.

7.6. Reproductive Symptoms

• Abortion: Infected pregnant cows may experience spontaneous abortion, stillbirths, or birth of weak calves.

• Reduced fertility: LSD can reduce the fertility of infected animals.

7.7. General Malaise

• Depression: Infected animals often appear lethargic or unwell.

• Dehydration: Due to fever, reduced appetite, and fluid loss, animals can become dehydrated.

7.8. Conjunctivitis

• Eye discharge and irritation are common, and the eyes may become swollen and red.

7.9. Lesions in Internal Organs (in severe cases)

In severe or prolonged infections, internal lesions may develop in organs like the lungs, liver, and gastrointestinal tract, although this is less commonly observed than skin lesions.

VIII. TREATMENT, PREVENTION AND CONTROL

The treatment of LSD is only symptomatic and targeted at preventing secondary bacterial complications using antimicrobial therapy [41]. Treatment trials performed by Salib and Osman, with the aim of preventing LSD

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complications and saving life has been successful using combination of antimicrobials, anti-inflammatory, supportive therapy and antiseptic solutions. The complications encountered during the trial including corneal opacity (keratitis), mastitis, dysentery, lameness, pneumonia and myasis have been recovered within 3 days to 2 weeks. However, the treatment of LSD (its complications) is costly as well as does not ensure full recovery therefore; prevention is more beneficial to avoid the substantial economic losses due to hide damages, loss of milk due to mastitis and loss of animal product due to death, abortion, fever and myiasis. Gariet al. study on epidemiological aspects and financial impact of lumpy skin diseases in Ethiopia illuminates the importance of vaccination in controlling LSD in endemic areas. He authors also enumerates vaccination can enable the financial costs due to LSD to be reduced by 17% per head in local zebu herds and 31% per head in Holstein Friesian or crossbred herds. Therefore vaccination is the only effective method to control the disease in endemic areas as movement restrictions and removal of affected animals alone are usually not effective. Effective vaccines against LSD exist and the sooner they are used the less severe the economic impact of an outbreak is likely to be. Members of the capri pox virus are known to provide cross protection. Hence, homologous (Neethling LSDV strain) and Heterologous (sheep pox or goat pox virus) live attenuated vaccines can all be used to protect cattle against LSD infection [42]. Commercially available capri pox virus (CaPV) vaccine strains include LSDV Neethling strain, Kenyan sheep and goat pox virus (KSGPV) O-240 and O-180 strains, Yugoslavian RM65 sheep pox (SPP) strain, Romanian SPP, and Gorgan goat pox (GTP) strains. Recently, a study by Gari et al. on efficacy of three CaPV strains against LSD in Ethiopia revealed that the Gorgan GTP vaccine can effectively protect cattle against LSDV and that the Neethling and KSGP O180 vaccine were incompetent and suggests the need for further molecular characterization for those ineffective vaccines. In countries previously free of LSD and which use sheep pox vaccine to protect sheep against sheep pox, it is recommended to use the same vaccine during LSD outbreaks, because of potential safety issues associated with the live attenuated LSDV vaccine use. In addition, rapid confirmation of a clinical diagnosis is essential so that eradication measures, such as quarantine, slaughter-out affected and in-contact animals, proper disposal of carcasses, cleaning and disinfection of the premises and insect control can be implemented as soon as possible during the eruption. Moreover, rigorous import restrictions on livestock, carcasses, hides, and semen from endemic areas must be in place in disease free areas [43].

IX. CONCLUSION

For effective control of LSD in a camp setting, vaccinate cattle at 3–6 months of age with a live attenuated vaccine. Mass vaccination should be prioritized in camps to achieve herd immunity, and boosters may be necessary depending on local epidemiological conditions. Proper coordination and monitoring will help ensure the success of the vaccination campaign and prevent the spread of LSD. To control and prevent the spread of Lumpy Skin Disease, vaccination is crucial. Early Vaccination of calves, followed by annual boosters, is recommended. Pregnant animals Should avoid vaccination, and strict biosecurity measures should be enforced to reduce. The spread of the virus. Consulting with a veterinarian is essential to establish the most. Appropriate vaccination protocol for a specific region or outbreak situation

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IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 5, Issue 2, December 2025 Impact Factor: 7.67

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