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Lumpy Skin Disease (LSD): Etiology, Pathogenesis, Prevention and Control

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Abstract
Lumpy skin disease (LSD) is a disease that poses a threat to the livestock industry because it can cause large economic losses. This disease was first discovered in the country of Zambia in 1929. In Indonesia, LSD was first discovered in early 2022 in Indragiri Hulu Regency, Riau. This disease is caused by a virus belonging to the genus Capripoxvirus, subfamily Chordopoxvirinae, family Poxviridae. The LSD virus has a limited host and does not infect non-ruminant hosts. The characteristic clinical symptom of LSD is the appearance of nodules on the skin. Diagnosis of this disease can be done by looking at the typical clinical symptoms, laboratory tests such as virus isolation, serological tests (serum neutralization test, virus neutralization test (VNT), agar gel immune diffusion, indirect ELISA, and indirect fluorescent antibody technique (IFAT)), real time and conventional PCR, immunohistochemistry, LAMP, and IPMA. Prevention and control of LSD disease can be done in several ways such as vaccination, vector control, restrictions on livestock traffic, strict quarantine and stamping out.
Keywords: Diagnosis, Lumpy Skin Disease, Pathogenesis, Prevention and Control

INTRODUCTION

Lumpy skin disease (LSD) is a disease that poses a threat to the livestock industry. This disease was first discovered in Zambia in 1929, then spread widely to other countries such as Egypt, Lebanon, Turkey, Jordan, Iran, Azerbaijan and Cyprus. In 2015 this disease has spread to European countries such as Greece, Russia, Armenia, Albania, Bulgaria, Serbia, Montenegro and Kasovo (Anwar et al., 2022). Even in 2021 this disease has been found in Southeast Asian countries such as Thailand, Cambodia and Malaysia (BBVet Wates, 2021).

In Indonesia, LSD was first discovered in early 2022 in Indragiri Hulu Regency, Riau. At the end of 2022 there were reports of suspected LSD disease in Sleman (Kulon Progo Regency Agriculture and Food Service, 2023). Meanwhile, according to Yustendi et al (2022) stated that the results of a survey of 22.2% of cattle in Lam Urit Village, Simpang Tiga District, Aceh Besar District were infected with LSD. The spread of LSD is caused by livestock traffic from infected areas, so this disease is included in the transboundary animal disease (TAD) (Sendow et al., 2021).

Although LSD is not classified as a zoonotic disease, this disease can provide large economic losses for livestock entrepreneurs such as weight loss, decreased milk production, abortion, infertility and can even cause death (Nurjanah and Dharmayanti, 2022). The same

thing was also conveyed by Namzi and Tafti (2021) that LSD disease can cause a significant decrease in milk production (from 10% to 85%), skin damage, decreased growth in beef cattle, temporary or even permanent infertility, abortion, treatment costs. and vaccination and death in infected animals.

The morbidity rate for this disease varies from 5% to 45% and sometimes even reaches 100%. While the mortality is below 10% and even up to 40%. The severity of LSD disease is affected by the age of the livestock, race, immune status, and production period (Namazi and Tafti, 2021). The incubation period for LSD in infected experimental animals varies between 4-7 days. Meanwhile, naturally infected animals can take up to 5 weeks (Tuppurainen et al., 2017). According to OIE (2017) in Sendow et al (2021) states that the incubation period for LSD is 28 days.

This paper discusses Lumpy Skin Disease, especially its causes (etiology), pathogenesis, and prevention and control of this disease. Apart from that, clinical symptoms, susceptible animals, and transmission of the virus to animals will also be discussed.

Etiology

LSD disease is caused by a virus belonging to the genus Capripoxvirus, subfamily Chordopoxvirinae, family Poxviridae (Al-Salihi, 2014; Nikola et al., 2019). The virus that causes LSD has a diameter of 230-260 nm, is oval in shape, has a lipid envelope, replicates in the cytoplasm and has a double-stranded DNA genome with a length of about 151 kbp which consists of a central coding region bounded by identical 2.4 kbp inverted terminal repeats and is thought to contain 156 gen. Comparison of LSD viruses with Chordopoxviruses from other genera revealed that 146 conserved protein-coding genes are involved in transcription and mRNA biogenesis, nucleotide metabolism, DNA replication, protein processing, virion structure formation, virulence and host range (Ratyotha et al., 2022; Gumbe, 2018; Tulman et al., 2001). This virus has a close genome similarity (96%) with sheep pox and goat pox viruses but is phylogenetically different (Sendow et al., 2021). Under the electron microscope, the morphological structure of the LSD virus is similar to that of the Vaccinia virus. Viruses are able to reproduce in primary cells, such as kidney or testicular cells in lambs and cattle, lung and kidney cells of sheep embryos, and fibroblasts of chicken embryos. In addition, this virus is also able to reproduce in kidney cells of cows and baby hamsters, but pathological changes are slow, whereas in African green monkeys, this virus cannot reproduce (Liang et al., 2022).

The LSD virus can survive at a pH of 6.6-8.6 and even tends to be stable in an alkaline environment. In necrotic skin nodules the virus can survive for 33 days, dry crusts for 35 days, infected tissue protected from sunlight for 6 months, and 18 days on dry skin at room temperature. While their resistance to high temperatures is flexible, most LSD viruses can be deactivated at 55°C for 2 hours and 65°C for 30 minutes. The virus is also sensitive to highly acidic or alkaline solutions and detergents containing lipid solvents. In addition, this virus is also susceptible to ultraviolet light heating with a temperature of 55°C for 1 hour, chloroform, 1% formalin, ether, 2% phenol, 2-3% sodium hypochlorite, 0.5% quaternary ammonium, and dilution of iodine compounds (Das et al., 2021).

The LSD virus has a limited host and does not infect non-ruminant hosts. This virus is capable of infecting cattle, buffalo and other wild ruminants (Arjkumpa et al., 2022). According to Sendow et al (2021) stated that local cattle, Channel nation cattle and Friesland Holstain can be infected with LSD. Cattle have a higher morbidity rate than buffalo, this is because buffalo have thick skin making it difficult to be pricked by the vector that spreads LSD, besides that in the summer buffalo always wallow in mud or water thereby reducing the possibility of being attacked by insect vectors (Moudgil et al., 2023). In addition, wild animals can also be infected with the LSD virus, such as impala (Aepyceros melampus), Thomson's deer (Gazella thomsoni),

and giraffes. Experimentally the LSD virus is also capable of infecting sheep and goats, but natural infection has never been reported (Namazi and Tafti, 2021).

The LSD virus can attack all ages, but young animals are more susceptible to the disease (Gumbe, 2018). This is in accordance with a study conducted by Elhaig et al (2017) which stated that out of 450 cows examined in Egypt, there was no significant difference in the prevalence of LSD based on age and sex. Meanwhile, according to Modugil et al (2023) states that young animals have a higher level of vulnerability and severity compared to adults. This virus can cause high mortality in young animals compared to adults. The morbidity rate of this disease varies from 5% to 45% and sometimes even reaches 100%. While the mortality is below 10% and even up to 40%. Variations in mortality and morbidity of LSD depend on several factors such as geographical location, climate, maintenance management, nutritional status, immune status, general condition of animals, type of livestock, virulence, population level and distribution of insect vectors (Al-Salihi, 2014).

Transmission

The main source of LSD virus transmission comes from skin lesions, this is because the virus can survive for a long time on the skin or scabs. In addition, the LSD virus can also be excreted through blood, semen, nasal secretions and tears and milk (Namazi and Tafti, 2021). This is in accordance with a study conducted by Parvin et al (2022) which showed that skin lesions had a higher concentration of viral DNA than other samples, all samples in skin lesions tested positive for 100%, while LSD viral DNA was detected less frequently in blood (41, 17%), nasal secretions (20%), saliva (15.28%), milk (16.66%), and stool samples were not positive. The study of Shumilova et al (2022) experimentally proved that the LSD virus was detected in blood on days 18 and 29 and in nasal secretions from days 20 to 42. In addition, research conducted by Sudhakar et al (2020) on cattle in India Those suspected of being infected with LSD were identified using Polymerase Chain Reaction (PCR). The study showed that the LSD virus genome was detected more in scabs, namely 79.16%, blood around 31.81% and bovine frozen semen as much as 20.45%. In addition, this study also proves that the LSD virus can be transmitted naturally by the semen of infected males. Iron et al (2005) in their study on 6 bulls aged 11-20 months which were experimentally infected with LSD virus isolate strain V248/93 showed that the virus could be isolated in semen up to 42 days after infection and viral DNA could be detected up to 5 months using PCR.

LSD disease transmission can occur directly or indirectly. Directly occurs due to contact with infected animals while indirectly occurs due to contamination by secretions of animals infected with the LSD virus. During an LSD outbreak on a dairy farm in Israel in 2006, researchers used mathematical modeling to investigate different possible routes of transmission (Magori-Cohen et al., 2012). The results of this study indicated that the spread of the LSD virus in an infected population could hardly be attributed to direct animal-to-animal contact, therefore it was concluded that transmission occurred mostly through indirect contact, possibly via blood-sucking insects.

Although direct and indirect contact (without vectors) is considered less effective as a source of infection, it can happen. Issimov et al (2020) which stated that transmission of LSD disease within a population occurs through aerosols when infected animals exhale and through contaminated food or drink. Meanwhile, according to Mishchenko (2015) in Issimov et al (2020) that the transmission of LSD in Iran, Azerbaijan, the Republic of Dagestan, Georgia and Russia occurred due to direct and indirect contact. This statement is supported by the results of a study conducted by Shumilova et al (2022) using bulls as experimental animals and providing feed that was previously inoculated with the LSD virus (classical strain Dagestan/2015 and recombinant Saratov/2017) to determine indirect spawning routes. . The

results showed that of the 5 bulls that had been treated (Dagestan/2015) they remained healthy and not seroconverted at the end of the experiment. Whereas in the treatment (Saratov/2017) 2 bulls experienced a mild infection.

These results prove that recombinant virus transmission occurs through digestion due to contaminated feed. In addition, in this study the results obtained were that control cattle also experienced clinical symptoms such as fever on days 10 and 20, lesions on days 13 and onwards, and seroconverted on day 31, so it was suspected that there was transmission of the virus through the air so that it could cause cows to infected control. Alexandr et al (2020) stated that LSD can be transmitted to healthy animals by direct contact with infected animals. In fact, his research proved that in an insect repellent facility, evidence was obtained that a new type of LSD virus was capable of transmitting to animals it came in contact with. Tuppurainen et al (2017) stated that transmission of LSD can occur through direct contact, feed or water contaminated by infected animal secretions (saliva, nose and eye discharge), and artificial insemination.

In addition, the LSD virus can also be transmitted through carcasses or other livestock products, intrauterine (from an infected mother to her child) and through breast milk (Sendow et al., 2021). Rouby and Aboulsoud (2016) in their research proved that premature calves born to LSD-infected mothers were confirmed positive by PCR containing LSD viral DNA, while the results of the Enzyme Linked Immunosorbent Assay (ELISA) and serum neutralization test (SNT) confirmed that the calf developed precolostral serum antibodies against the LSD virus. Thus indicating the existence of intrauterine transmission of the virus. LSD disease can also be transmitted through anthropod/insect vectors such as mosquitoes, ticks and flies (Seerintra et al., 2022). The vectors most likely to be capable of spreading LSD are blood-sucking anthropods such as stable flies (Stomoxys calcitrans), mosquitoes (Aedes aegypti) and hard ticks (Rhipicephalus and Amblyomma species). New evidence suggests that the house fly (Musca domestica) may be able to play a role in the transmission of LSD disease, but this has not been clinically tested (Sprygin et al., 2019). Issimov et al (2020) in their research proved that three Stomoxys species (Stomoxys calsitrans, Stomoxys sitiens, and Stomoxys indica) can act as mechanical vectors in LSD transmission, even animals infected due to Stomoxys spp bites mostly show moderate degrees of severity and only one severe case.

Research conducted by Tuppurainen et al (2011) proved that the LSD virus can be transmitted transstadially and transovarially by the tick Rhipicephalus decoloratus, while Rhipicephalus appendiculatus and Amblyomma hebraeum mechanically or intrastadially. Chihota et al (2001) through their research proved that female Aedes aegypti mosquitoes are capable of transmitting LSD mechanically. These mosquitoes are capable of transmitting LSD from infected animals to susceptible animals during a period of 2-6 days after infection. The Aedes aegypti mosquito has been involved in long-distance airborne transmission of LSD in an area, making it difficult to control the disease (Mulatu and Feyisa, 2018). Other blood-sucking insects capable of spreading LSD are mosquitoes (Anopheles stephensi, Culex quinquefasciatus) and flies (Haematopota spp, Culicoides nubeculosus, Cilicoides punctatus) (Chihota et al., 2003; Al-Salihi, 2014; Sohier et al., 2019 ; Namzi and Tafti, 2021).

Many studies have revealed that after an insect vector sucks the blood of an infected animal, the virus can spread and is present in the salivary glands, head, body and faeces of the insect. The LSD virus can live in Aedes aegypti mosquitoes for at least 2-8 days. Even a recent study in England confirmed that the LSD virus can survive for 9 days in the mouths of the insects Stomoxys calcitrans, Aedes aegypti, Culex quinquefasciatus, and Cubicoides nubeculosus (Liang et al., 2022). Insects infected with the LSD virus can become a reservoir in transmitting the disease to other animals. Most LSD illnesses are discovered in the summer when the vector is

active. The increased risk of spreading LSD occurs during warmer and more humid summers which are able to support the breeding of vector populations (Ratyotha et al., 2022).

This is in accordance with the statement of Namzi and Tafti (2021) that the risk factor for the spread of LSD is a warm and humid climate which is able to support the breeding of vector populations, as seen in the rainy season and the introduction of new animals to the herd. In addition to herd migration, transport of infected animals to areas free of LSD, pastures and drinking water sources have been considered as factors that could increase the incidence of the disease. Wind direction and strength also affect the spread of the LSD virus. While the risk factors associated with LSD virus seropositivity include age, gender, maintenance management, rainfall and source of drinking water. Until now transmission is still reported through mechanical vectors, so that the spread of the disease from one area to another can be through the transportation of infected livestock, then bitten by insects that are thought to act as mechanical vectors. Meanwhile, the mechanism as a biological vector is still unclear (Horigan et al., 2018). Transmission of the LSD virus to humans has never been reported, this is in accordance with the statement of Choudari et al (2020) which states that LSD cannot be transmitted to humans (non zoonotic).

Pathogenesis and Clinical Symptoms

Experimentally, the incubation period for LSD ranges from 4-7 days after infection, whereas naturally occurring infections have an incubation period of up to 5 weeks (Tuppurainen et al., 2017). Meanwhile, according to Moudgil et al (2023) stated that after transmission of the LSD virus to its natural host, the incubation period varies between 7-28 days. The course of this disease can be acute, subacute and chronic. The virus undergoes intracellular replication in fibroblasts, macrophages, pericytes, and endothelial cells then undergoes viremia and causes vasculitis and lymphangitis in infected tissues. Kumar et al (2021) stated that the duration of LSD viral viremia varied from 1 to 10 days. Animals that recover from LSD will acquire antibodies for about 6 months.

Clinical symptoms that appear after the incubation period can be grouped into 4 phases. The first phase is the acute phase, in which animals show symptoms of high fever of 41°C which occurs for 7-10 days and is followed by other symptoms such as anorexia, lacrimation, depression, increased nasal discharge, decreased milk, saliva secretion, multinodular lesions found around the skin and mucous membranes, but some cases of animals do not show symptoms of fever. The second phase is swelling of the subscapular and precrural lymph nodes as well as an increase in multiple nodules with a diameter of 0.5-5 cm which mostly occur in the head, neck, trunk, genitals, udder, mucous membranes, nasal cavity, mouth, and plaque areas. inoculation. After 1-2 days the nodules burst and are able to spread the virus to the surrounding environment. Sometimes found lymphangitis and vasculitis. The third phase of the nodule will turn into ulceration and necrosis after 2-3 weeks. In severe cases, ulcerated lesions appear on the mucous membranes of the eye and nasal cavities, excessive salivation, lacrimation, and nasal discharge. The animal's secretions may contain the LSD virus which is capable of infecting other animals. The fourth phase, after at least 1 month, there is healing of the ulceration and also skin thickening and hyperpigmentation of the lesions (Ratyotha et al., 2022).

The virus is in the seminal fluid after 42 days after the fever (Namazi and Tafti, 2021). Infected animals also often have secondary infections with pneumonia bacteria and fly bite nodules will cause deep wounds. In addition, complications from LSD cause abortion, infertility, decreased lactation, and anastrus. Some infected animals show no clinical symptoms (Sendow et al., 2021).

Diagnosis

LSD can be diagnosed by looking at the typical clinical symptoms of the disease. However, this is difficult to do if the symptoms are mild or even asymptomatic, so laboratory tests are needed to detect and confirm the disease, such as virus isolation, serological tests (serum neutralization test, virus neutralization test (VNT), immune diffusion gel agar, indirect ELISA, and indirect fluorescent antibody technique (IFAT)), real time and conventional PCR. Virus isolation and PCR are the most sensitive methods for detecting the LSD virus on the skin. Zeedan et al (2019) in their research concluded that the use of the Real Time PCR method detected more positive samples containing the LSD virus than conventional PCR and FAT.

The indirect ELISA method detects more antibody-positive samples than IFAT from bovine serum samples. The study concluded that the RT-PCR test is a simple, sensitive, fast, and reliable method for detecting LSD virus in blood and skin nodule biopsies of suspected infected bovines. The samples used to detect LSD virus DNA by PCR or RT-PCR methods were skin nodules, secretions, semen and blood from infected animals. While the target genes that are often used are P32, RPO30, and GPCR (Ratyotha et al., 2022). Yimer (2021) stated that the use of molecular methods (RT PCR and PCR) in diagnosing the LSD virus was only able to detect viruses belonging to the genus Capripoxvirus in general and was unable to differentiate between the LSD virus and sheep pox virus and goatpox virus. So that the use of the RT PCR and PCR methods needs to be continued with sequencing (Sendow et al., 2021).

The gold standard for diagnosing LSD virus is virus isolation, however using this method requires time to isolate and culture the virus from tissue or chorioallantoic membranes from embryonated chicken eggs (Amin et al., 2021). This is in accordance with the statement of Cavalera et al (2022) which stated that virus isolation is the gold standard for diagnosing LSD, but it takes several weeks to isolate the virus. However, according to the World Organization for Animal Health (WOAH) recommends using the VNT method as the gold standard in detecting antibodies to the LSD virus. The VNT test is a highly sensitive and specific test method that can measure viral antibody titers after infection or vaccination. However, the VNT test requires skill and experience in using and interpreting the results and requires a level 3 biosafety laboratory (BSL3). In addition, WOAH also suggests using ELISA as an alternative serological method (Sthitmatee et al., 2023). However, this test can provide false detection caused by non-specific binding between Parapoxvirus and Capripoxvirus (Ratyotha et al., 2022).

In addition, several researchers used other methods in diagnosing LSD, such as those carried out by Mwanandota et al (2018) in their research proving that the use of Loop-mediated isothermal amplification (LAMP) has good sensitivity and specificity to support routine LSD diagnoses, while its ability to detect the LSD virus in apparently healthy animals demonstrates its usefulness in identifying populations at risk for exposure to LSD. Haegeman et al (2020) tested the use of the Immunoperoxidase Monolayer Assay (IPMA) to detect LSD virus antibodies. This study concluded that the use of the IPMA method was able to detect LSD virus antibodies earlier in infected, vaccinated, and vaccinated/infected animals than VNT and ELISA. Amin et al (2021) conducted a study to diagnose LSD that occurs naturally in cattle using virological, molecular and immunohistopathological assays. Histological examination of the skin of various cases revealed various changes depending on the stage of infection. Immunohistochemistry is used as a confirmatory test to detect LSD virus antigen in nodule tissue of infected bovine skin using LSD virus specific antibodies.

In making the diagnosis, it is also necessary to consider the differential diagnostic of the disease, this is because several other diseases show clinical symptoms similar to LSD (Sendow, et al., 2021) such as pseudo LSD caused by bovine herpesvirus 2 (BoHV2) which is characterized by the presence of lesions that involves only the epidermis and produces a scab

after the peeling occurs. The disease causes superficial nodules similar to early-stage LSD. The histopathological features of BoHV-2 infection that are not found on LSD are intranuclear inclusion bodies and viral syncytia. While other diseases that are differential diagnostics of LSD are photosensitization, dermatophilosis, dermatophytosis, bovine farcy, actinobacilosis, actinomycosis, urticaria, insect bites, nocardiasis, besnoitiosis, demodicosis, onchocerciasis, cowpox, and pseudo cowpox (for integumentary lesions). Bluetongue, foot and mouth disease, malignant catarrhal fever, bovine viral diarrhea, popular bovine stomatitis, and infectious bovine rhinotracheitis (Guyasa, 2022).

Prevention and Control

Prevention and control of LSD can be done in several ways, such as vaccination, vector control, restrictions on livestock traffic, strict quarantine and stamping out (Sendow et al., 2021). Vaccination is an effective and successful option for controlling LSD. Tuppurainen et al., (2020) stated that vaccination is the only way to prevent the spread of the LSD virus both in endemic areas and in newly affected areas. Currently most of the commercially available vaccines for LSD are live attenuated vaccines based on LSD virus strains, sheeppox virus (SPPV), or goatpox virus (GTPV). However, the first inactivated vaccine has recently hit the market (Tuppurainen et al, 2021). GTPV/SPPV-based vaccines have lower efficacy than live attenuated LSD virus vaccines but these vaccines do not cause fever and the appearance of post-vaccination clinical symptoms (Sprygin et al., 2020). Live vaccines are capable of producing a strong and long-lasting immune response and are effective in disease prevention, but these vaccines are capable of causing local inflammation and mild symptoms with skin lesions (Datten et al., 2023).

The Capripoxvirus genus has been recognized as capable of providing cross-protection, therefore cattle can be protected from LSD virus infection by using live attenuated vaccinations that are homologous (strain Neethling virus LSD) or heterologous (sheep pox or goat pox viruses). Commercially accessible vaccine strains such as Neethling virus LSD strain, KSGPV O-240 and O-180 strain spox (GTP) strains, Kenyan sheep and goat pox virus, Capripoxvirus (CaPV) including Gorgan goat, Romanian SPP, and Yugoslav RM65 sheep pox (SPP) strains.Tekilegiorgis and Tamir (2019) state that in the Middle East and the Horn of Africa the sheep poxvirus (SPPV) vaccine provides incomplete protection and adverse reactions in post-vaccinated cattle.

Meanwhile, Gary et al (2015) through their research proved that the Ethiopian Neethling virus vaccine, the Kenyan goat and sheep pox vaccine ((KSGP) O-180 strain vaccines) were unable to protect against LSD in cattle. Meanwhile, Goran goat pox is able to protect cows from LSD. However, these results are different from a study conducted by Hakobyan et al (2023) that the heterologous vaccine (sheep poxvirus) used in Armenia provided immunity to the cattle population of 86.09% and did not cause any side effects in the cattle. Some of the factors identified as causing vaccine failure are differences between vaccine strains and virus strains that infect animals in the field, low vaccine titers, vaccination of animals that are temporarily experiencing an incubation period of disease, and errors in vaccine handling and storage (Datten et al., 2023).).

Vaccination along with vector control measures, strict quarantine, restrictions on livestock traffic can be effective in preventing the spread of LSD. Vectors can play a role in mechanical disease transmission, in fact most of the spread of the LSD virus is caused by vectors of blood-sucking insects, so it is necessary to control the spread and reproduction of these vectors. Several methods are used for vector control by using vector traps, insecticides, and ectoparasites (Gupta et al., 2020; Sacharia and Deepa, 2021). Tuppurainen et al (2017) stated

that efficient vector control can reduce the rate of mechanical disease transmission, but this does not apply if livestock are reared extensively.

It further states that the use of insecticides on a large scale can disrupt the ecological balance and beneficial insects such as honey bees. So it is necessary to carry out environmentally friendly vector control such as limiting vector breeding by cleaning feces and preventing standing water in the cage and the surrounding environment and members. In addition to vectors, disease transmission can occur by infected animals and contaminated equipment. The earlier the outbreak can be controlled if the animal population is quarantined and sanitizes the cages and equipment. Controlling the movement of animals or quarantine of animals to be imported for at least 3-4 weeks can prevent the spread of LSD (Ratyotha et al., 2022).

CONCLUSION

Lumpy skin disease (LSD) is a disease caused by a virus belonging to the genus Capripoxvirus, subfamily Chordopoxvirinae, family Poxviridae. LSD disease transmission can occur directly or indirectly, besides that it can be transmitted through blood-sucking insect vectors such as stable flies (Stomoxys calcitrans), mosquitoes (Aedes aegypti) and hard ticks (Rhipicephalus and Amblyomma species). The course of this disease can be acute, subacute and chronic. Clinical symptoms caused by this disease are high fever of 41°C, anorexia, lacrimation, depression, increased nasal discharge, decreased milk, saliva secretion, the appearance of nodules with a diameter of 0.5-5 cm which mostly occur in the head area, neck, trunk, genitals, udder, mucous membranes, nasal cavity, mouth, and plaque at the inoculation site. In addition, complications from LSD cause abortion, infertility, decreased lactation, and anastrus. Diagnosis of this disease can be done by looking at the typical clinical symptoms, laboratory tests such as virus isolation, serological tests (serum neutralization test, virus neutralization test (VNT), agar gel immune diffusion, indirect ELISA, and indirect fluorescent antibody technique (IFAT)), real time and conventional PCR, immunohistochemistry, LAMP, and IPMA. Prevention and control of LSD disease can be done in several ways such as vaccination, vector control, restrictions on livestock traffic, strict quarantine and stamping out.

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