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A Short Investigation on the Frequency of Lumpy Skin Disease in the Cattle Population

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ABSTRACT

An infectious, eruptive, and sometimes lethal illness of cattle, lumpy skin disease (LSD) is caused by a virus related to the Neethling poxvirus of the genus Capripoxvirus of the family Poxviridae. Although LSD was first documented in Zambia, it is now known to occur in several nations throughout Africa and, on rare occasions, in the Middle East. The infectious agent responsible for Lumpy skin disease belongs to the family Poxviridae and is caused by the genus Capripoxvirus. Antigenically, LSDV is quite similar to poxviruses seen in sheep and goats. Risk factor evaluations, epidemiological considerations, seroprevalence, and financial implications have all received little attention in the scant literature on this illness in Ethiopia. Vectors of LSDV in cattle are arthropods known as mechanical haematophagus. During the rainy season, which spans from the tail end of summer until the first days of fall, LSD is prevalent. Vaccination, limiting animal mobility, and eliminating sick or exposed animals are ways to manage LSD.

Keywords: Prevalence, Lumpy skin disease, Morbidity, Epidemiology, Poxviridae, and Domestic animals.

INTRODUCTION:

It is believed that mechanical vector insects and wildlife both have significant roles to play in the epidemiology and maintenance of LSD, a skin disease that affects all cattle. The illness has a significant impact on the economies of many African nations, including Ethiopia. This is because of the high morbidity rate, the long-term loss of productivity caused by the disease, the limits on international traffic in live animals and animal products, and the high costs of control and eradication efforts. The introduction of additional cattle, shared watering holes and pasture areas, and other such practices all increase the likelihood of LSD. Clinical exams and laboratory testing are examples of the present diagnostic options (Virus to validate it, testing for isolation and identification as well as serology are required (Hailu et al., 2015). Emaciation, enlarged lymph nodes, leg and breast edema, fever, nodules on the skin, mucous membranes, and internal organs, high morbidity, low mortality, mastitis, orchitis, and occasionally death are all symptoms of this infectious viral disease, which can last anywhere from a few weeks to several years (Radostitis et al., 2007). It is thought that mosquitoes, biting flies, hard ticks, and other arthropods that feed on blood are the primary vectors of LSDV (Chihota et al., 2001). Typical clinical symptoms, epidemiology, histology, viral testing, and a final diagnosis of LSD are used for diagnosis. PCR and isolation (Tuppurainen and Oura, 2012). Due to a lack of knowledge about its epidemiological characteristics and the widespread

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belief that it is only a wound skin disease, this illness killed out a large number of cattle in my region last summer. Therefore, this seminar paper will address the following topics: the economic implications of lumpy skin disease at the farm and country level; and the epidemiological aspects of lumpy skin disease at the national and worldwide level.

Review of Literature

Historical Background of Lumpy Skin Disease

According to Morris (1931), the first account of LSD's clinical manifestations was published in 1929 in Zambia, which was once known as Northern Rhodesia. The infectious agent responsible for lumpy skin disease is the genus Capripoxvirus, which belongs to the family Poxviridae. The antigenically similar sheep and goat poxviruses are close relatives of lumpy skin disease virus (LSDV) (Woods, 1988). Despite their differences, most serological assays are unable to distinguish between these three viruses. There is a 55°C/2 hour and 65°C/30 minute window of vulnerability for LSDV. It has a 10-year shelf life when stored at -80°C and extracted from skin nodules. The tissue culture fluid that has been infected may be kept at 4°C for a duration of 6 months. Extremely acidic or strongly alkaline pH may infect the virus. Nevertheless, after 5 days at 37°C and pH 6.6–8.6, the titer remains unaffected. Ether (20%), chloroform (1%), formalin (1%), and some detergents (e.g., SDS) may all inhibit LSDV replication. Sodium hypo-chlorite (2-4%), iodine compounds (1:33 dilution), Virkon® (2%) and quaternary ammonium compounds (0.5%) are also known to be vulnerable to it (Woods, 1988). Particularly in dried scabs, LSDV remains viable for extended periods of time at room temperature. The deactivation of LSDV is a challenging task. Eight million cattle were afflicted by this parasite, which may live for 33 days or more in necrotic skin nodules. Thomas and Mare (1945), Von Backstrom (1945), and Diesel (1949) all note that the infections persisted until 1949 and caused significant economic losses. Kenya, in East Africa, was the site of the 1957 discovery of LSD. According to Ali and Obeid (1977), the illness was first reported in Sudan in 1972, and in 1974, it spread to West Africa. In 1983, when it was making its way into Somalia (Davies, 1999a and b). Over the course of many decades, the illness has persisted in spreading over the majority of Africa.

epizootics, as noted earlier by House (1990) and Davies (1991 b). Three countries—Mauritus, Mozambique, and Senegal—reported LSD in 2001. These days, you can get LSD just about anywhere in Africa (Tuppurainen and Oura, 2012), with the exception of Libya, Algeria, Morocco, and Tunisia. Although it was suggested that the illness may expand beyond its original territory in the 1980s, it remained limited to nations in Sub-Saharan Africa from 1929 to 1984 (Davies, 1981). Reports of LSD outbreaks in Oman occurred in 1984 and 2009, respectively, throughout the Middle East (House et al., 1990; Kumar, 2011; Tageldin, 2014). Several countries have reported LSD invasions: Kuwait in 1986 and 1991, Egypt in 1988 and 2006, Israel in 1989 and 2006, Bahrain in 1993 and 2002-2003, Yemen, the United Arab Emirates in 2000, and the West Bank. References include Ali et al. (1990), House et al. (1990), Davies 1991a, Fayez and Ahmed (2011), Shimshony and Economides (2006), and APHIS (2006). The 2009 resurgence of LSD in a herd of 3,200 Holstein cattle in Oman was associated with a high death rate of 12% and a morbidity rate of 30% (Tageldin et al., 2014). May 1988 saw reports of LSD in Egypt's Suez Governorate (Ali et al., 1990). The local quarantine station in Egypt was the first point of entry for the illness in livestock brought in from Africa. During the summer of 1988, it made a localized spread and seemed to have survived the winter without causing any noticeable illness. The infections first arose in the summer of 1989 and persisted for five to six months, affecting twenty-two out of twenty-six Egyptian governorates. Because over two million cattle were vaccinated against sheep pox, the morbidity rate during this epidemic was quite low at 2%. Despite this, some 1,499 animals perished. There was a resurgence of the LSD epidemic in various Egyptian governorates in the summer of 2006, affecting cattle of both sexes and all ages, with severe and serious complications (Fayez and Ahmed, 2011; Hayle et al., 2020; Ali and Amina, 2013). One farm reported 30 cases on dairy cows. From 2007 to 2011, central Ethiopia was the site of

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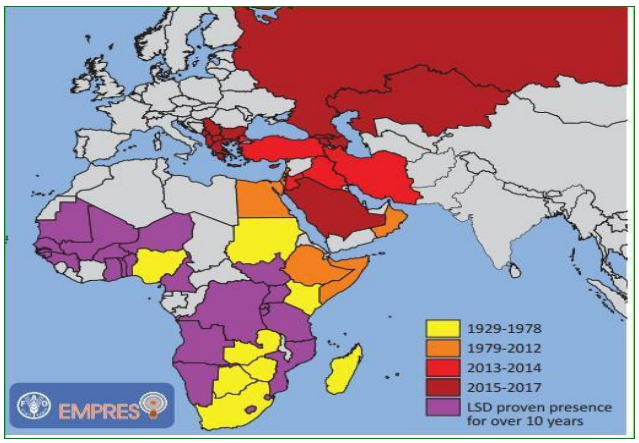
one of the continent's LSD epidemics. These active outbreaks were reported. Adama, Wenji, Mojo, and Welenchiti were the four districts that were examined. Between 2007 and 2011, there were a total of 1,675 recorded outbreaks, resulting in 62,176 illnesses and 4,372 fatalities. Between September and December, there was a spike in the frequency of outbreaks. Ayelet et al. (2014) reported a morbidity rate of 13.61% (296 cases) and a death rate of 4.97%. In 2012 and 2013, Syria, Lebanon, and Jordan were among the nations hit by LSD. The disease has been reported in Turkey in October 2013, Iran and Iraq in 2014 (Fig. 2) (Sherrylin et al., 2013).

Etiology

LSDV is a pleomorphic, enveloped, brick- or oval sha- ped dsDNA virus with a molecular size of 350x300nm and a molecular weight 73 to 91 (Kilodalton) KDa. An LSDV genome sequence is 145 to 152. The terminal genomic sequences contain a unique complement of at least 34 genes which are responsible for viral viru- lence, host range and/or immune evasion of host (Kara *et al*., 2003). All *Capripoxviruses* grow slowly on cell cultures and may require several passages. They can



be propagated on a variety of cells of bovine and ovine origin, causing easily recognizable cytopathic effects. In addition, the virus can be propagated in the cho- rioallantoic membranes of embryonated chicken eggs, causing macroscopic pock lesions. The replication of LSDV occurs in the cytoplasm of the host cell resul- ting in intracytoplasmic eosinophilic inclusion bodies (EFSA, 2015). LSDV is susceptible to sun light and detergents containing lipid solvents like ether (20%), chloroform, formalin (1%) and phenol (2%). The virus could be inactivated after heating for 1 hour at 55°C (Lefèvre and Gourreau, 2010). However, it withstands drying, pH changes, if not an extreme pH and can remain viable for months in dark room such as infected animal shade off their host. LSDV can persist in skin plugs for about 42 days (Sarkar *et al*., 2020; Babiuk *et al*., 2008b).

Epidemiology of lumpy skin disease

Lumpy skin disease is an important, economically devastating, notifiable disease that brought production loss in cattle due to generalized malaises and chronic debility (Tuppurainen and Oura, 2011). A good under- standing of epidemiological aspects of LSD related to pathogen, host and environment might aid in control & prevention mechanisms. Particular emphasis should be given to exposure of hosts to pathogen in suitable en- vironment that facilitate transmission and distribution of the disease. LSD is more prevalent during the wet summer and autumn months and occurs particularly in low-lying areas and along water courses (Bekere *et al*., 2022; OIE, 2010).

Geographic Distribution

LSD originated from Sub Sahara African countries in 1929 and spread to the north and south during the last seventy years. The geographic coverage of LSD has extended its range to include all countries in sub- Saharan Africa as well as Madagascar and it is en- demic to every African country and occurs in various ecological zones from temperate areas to dry semi-arid and arid areas (Kitching and Carn, 2000). Outbreaks outside the African continent have occurred in the Middle East in 2006 and 2007, in Mauritius in 2008 (OIE, 2014b), and Israel has reported with LSD outbreaks (Brenner *et al*., 2006). Epidemiological trend of LSD suggests that it is currently endemic in most of

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African countries and spreading further in to North Africa, Middle East countries and Mediterranean regions because of global trade movement in animals and animal products (Gammada, 2020; Tup- purinen and Oura, 2011, 2012).

Fig. 1: Countries that have reported of LSD; Source: (Tuppurainen *et al*., 2017).

Risk Factors

Host Risk Factors - Lumpy skin disease is a disease of cattle that causes several disorders. Though all breeds and age group are susceptible, *Bos taurus* is particularly more susceptible to clinical disease than zebu cattle and *Bos indicus* (Radostits *et al*., 2007). Among *Bos taurus*, fine-skinned, high-producing dairy Channel Island breeds are highly susceptible to LSDV (EFSA, 2015). Lactating cows appearing to be seve- rely affected and result in a sharp drop in milk production because of high fever caused by viral infection itself and secondary bacterial mastitis (Tuppurainen and Oura, 2011). Whereas indigenous breeds such as zebu and zebu hybrids are likely to have some natural resistance against the virus (Gari *et al*., 2011). It is not known what genetic factors influence the disease severity (Babiuk *et al*., 2008). High ambient temperatures, farming practices and cow which produce high milk yields, could be deemed to stress the animals and contribute to the severity of the disease in Holstein– Friesian cattle (Tageldin *et al*., 2014).

Young animals are severely affected and clinical sym- ptoms are rapid to appear. But traditional calf manage- ment practices that segregate calves from the herd might have contributed to a decreased exposure risk of calves to the source of infection. Calves in the endemic area can obtain certain protective passive immunity from their dam. An animal recently recovered from an attack is not susceptible to LSDV; because there is a solid immunity lasting for about 3 months (Gari *et al*., 2011). In local zebu cattle, male animals have higher cumulative incidence than females due to stress factor of exhaustion and fatigue rather than to a biological reason. The majority of male animals are draft oxen used for heavy labor, which might contribute to an increase in susceptibility. Another reason is that draft oxen cannot protect themselves well from biting flies when harnessed in the yoke, and the beat scratches on their skin induced while plowing may attract biting flies potentially capable of transmitting LSD infection (Gari *et al*., 2011). Generally, clinical severity of dis- ease depends on susceptibility, immunological status, and age of the host population and dose and route of virus inoculation (CFSPH, 2008).

Pathogen Risk Factors- LSDV is one of the species of *Capripoxviruses* that is resistant to different che- mical and physical agents (Murphy *et al*., 1999). Cap- ripoxviruses have lipid-containing envelopes and sus- ceptible to a range of detergents containing lipid sol- vents like ether (20%), chloroform, formalin (1%), phenol and sunlight. They are also susceptible to sunlight, but survive well at cold temperatures. LSDV is susceptible to temperature of 55°C/two hours, 65°C/ 30 minutes, alkaline or acid pH. No significant reduction in titer when held at pH 6.6–8.6 for five days at 37°C (OIE, 2014b). LSD virus is present in nasal, lachrymal and pharyngeal secretions, semen, milk and blood. However, the virus may persist in saliva for up to 11 days, in semen for 22 days, in necrotic tissue re- maining at the site of a skin lesion for 33 days and for 6 months on fomites, including clothing and equipment but there is no evidence that virus can survive more than four days in insect vectors. There is no evidence of the virus persisting in meat of infected animals, but it might be isolated from milk in early stages of fever (Babiuk *et al*., 2008a).

Capripoxviruses are very resistant in the environment and can remain viable for long periods on or off the animal host. They may

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persist for up to 6 months in a suitable environment, such as shaded animal pens. Can be recovered from skin nodules kept at −80 °C for 10 years and infected tissue culture fluid stored at 4°C for six months (Aus-vetplan, 2009).

Environmental Risk Factors- Environmental deter- minants play a great role in the epidemiology of lumpy skin disease. It has major impact on the agent, host and vectors as well as interaction between them. These predisposing factors have a great role in maintenance of arthropod vector and transmission of the virus to susceptible animals. Animals sharing the communal grazing lands and watering points, uncontrolled cattle movements across different borders due to trade and pastoralism, rainfall and wet climate which favor insect multiplications, other reasons of cattle move- ment from place to place and presence of water bodies are some of potential risk factors of LSD (Tuppurainen and Oura, 2011). LSD is associated with increased number of insects which are mechanical vectors (Ma- goricohen, 2012). It is more prevalent during the wet and warmer condition of summer and autumn months and occurs particularly in low lying agro-climate zone and along watercourses (OIE, 2010).

The warm and humid climate in midland and lowland agro-climates has been considered as more favorable environment for the occurrence of large populations of biting flies than the cool temperature in the highlands (Tuppurainen *et al*., 2012).

Source of Infection

Clinically sick animals are the main source of infection to other healthy animals. However, LSD virus can be present in blood, cutaneous lesions, saliva, nasal dis- charge, lachrymal secretions, milk, semen and very rarely in drinking water, which may be sources for transmission (Babiuk *et al*., 2008b; Irons *et al*., 2005; Zelalems *et al*., 2015).

Morbidity and Mortality

The morbidity of the disease is highest in wet, warm weather and decreases during the dry season (OIE, 2008). In outbreaks of the disease, the morbidity rate varies widely depending on the immune status of the hosts and the abundance of mechanical arthropod vec- tors and averagely ranges from 3% to 85% (CFSPH, 2008, Tuppurainen *et al*., 2012). But it can reach as high as 100% in natural outbreaks while mortality rate rarely exceeds 5% but may sometimes reach 40% (Babiuk *et al*., 2008; Irons *et al*., 2005).

Mechanism of Transmission

Direct Transmission- Direct transmission can occur when the animals share the same feeding and drinking trough due to contamination by nasal and salivary discharges from infected animals or ingestion of the already contaminated food or by iatrogenic agents (Lefèvre and Gourreau, 2010). Suckling calves may be infected through infected milk. Transmission of LSDV through semen has been experimentally demon- strated (Annandale *et al*., 2013). The more recent study demonstrated that persistence of the live virus in bovine semen for up to 42 days post infection and viral DNA was detected until 159 days post infection (Irons *et al*., 2005). During the natural outbreak of LSD in Egypt in 2006–2007, 25 % of cows had been found with infected ovarian by LSDV and 93 % of cows were suffered from ovarian inactivity and showed no signs of estrus (EFSA, 2015). There is an assumption that virus is also secreted in vaginal secretions. Generally transmission of the virus by contact is inefficient and field evidence reported that the disease is not contagious (Salib and Osman, 2011).

Indirect Transmission - The transmission of LSDV occurs mechanically by blood-feeding biting arthro- pods vectors including hard ticks, biting flies and mos- quitoes (Chihota *et al*., 2001; Getachew *et al*., 2010; Magori-cohen, 2012). This vector related transmission is apparently

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mechanical, rather than biological. This distinction is important because infectious organisms do not generally survive in vectors for long periods for multiplication. In the mechanical mode of transmis- sion, the virus is transmitted via contaminated mouth parts of vectors without actual replication of the virus in arthropod cells or tissues. Study by (Chihotas *et al*., 2001) indicated that the virus can survive 2-6 days post feeding from infected cattle and transfers this to sus- ceptible cattle by female mosquito, Aedes egypti during experimental infection. Recently, new evidence has been published reporting a possible role of hard ticks in the transmission of LSDV. The study showed molecular evidence of trans stadial and trans ovarian transmission of LSD virus by Boophilus decoloratus and mechanical transmission by Repicephalus ap- pendiculatus and Ambyloma hebraeum (Tuppurainen *et al*., 2011). Mosquitoes (female *Aedes egypti* and *Culex quinquefasciatus*) and other flies such as taba- nids (horse flies), biting midges (*Culicoides nubeculo- sus*), and *Glossina* species like tsetse fly are among the other arthropod vectors that play a great role in the transmission of the virus. Non biting flies, including housefly (Muscidae), bush fly (Hippoboscidae) and blowflies (Calliphoridae) are also very commonly associated with sucking of infected lachrymal, nasal or other secretions and transfer the virus to another susceptible animal. Vermin, predators and wild birds might also act as mechanical carriers of the virus (Ausvetplan, 2009).

Epidemiological evidence sug-gests, outbreaks of LSD is highly associated with pre-valence of high insect vectors population and with upcoming of rainy season. Epidemics of LSD are associated with rainy seasons, river basins and ponds during which cattle grazed and humid areas that is conductive insect multiplication (OIE, 2010).

Pathogenesis

LSD is developed by the entry of infectious LSDV through skin or GIT mucosa then viremia accompanied by a febrile reaction. Then the virus reaches and causes swelling of regional lymph nodes (Gari *et al*., 2011). Mechanism by which the virus causes skin lesions is due to replication of the virus in specific cell such as endothelial cells of lymphatic and blood vessels walls

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with development of inflammatory nodules on skin (Vorster, 2008). LSD is epitheliotrophic diseases that cause localized and systemic reaction and results in vasculitis and lymphadenitis which result in to oedema and necrosis. In some severe cases thrombosis and other symptoms will be observed. Nodules of LSD may be changed to grey-pink with caseous necrotic cores. Circumscribed necrotic lesions may ulcerate. Skin localization is due to epitheliotrophic property of LSDV (Radostitis *et al*., 2007).

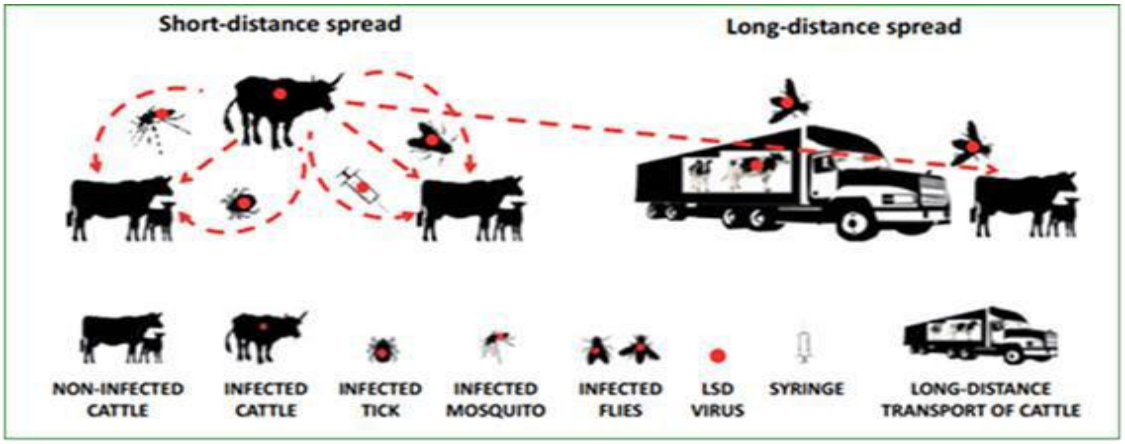

Fig. 2: Schematic illustration of the spread of LSDV; Source: (Tuppurainen *et al*., 2017).

LSD skin nodules may exude serum initially but develop a characteristic inverted grayish pink conical zone of necrosis. Adjacent tissue exhibits congestion, hemorrhages and edema. Enlarged lymph nodes are found and secondary bacterial infections are common within the necrotic cores. Multiple virus-encoded fac- tors are produced during infection, which influence pathogenesis and disease (Tuppurainen *et al*., 2012). Incubation period of LSD can vary under field and experimental conditions. It varies from 4 and 14 days in experimentally inoculated animals and 2 – 4 weeks in naturally infected animals (OIE, 2010).

Clinical Sign

Course of lumpy skin disease may be acute, sub-acute and chronic. The virus causes from in apparent in- fection to severe clinical symptoms and those animals which develop clinical disease may have a biphasic febrile reaction. The major visible clinical signs are; fever of 40-41.5^{o}C which may last 6 - 72 hours, lacrimation , increased nasal and pharyngeal secretion, loss of appetite, reduced milk production, some de- pression and movement reluctance, nodule in the skin, mucous membrane and internal organs and swelling of superficial lymph nodes. Diameter of nodular lesion may be up to 1-7 cm diameter appears as round, firm, intradermal and circumscribed areas of erected hair (OIE, 2010; Tuppurinen and Oura, 2011).

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Fig. 3: Lumpy skin disease randomly distributed nodules on the skin; Source: (Coetzer *et al*, 2004).

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In severe cases, ulcerative lesions may develop in mucous membrane of mouth, trachea, larynx and eso- phagus (Radostitis *et al*., 2007). The necrotic cores become separated from the adjacent skin and are referred to as 'sit-fasts'. It might be exacerbated by Secondary bacterial complication and infestation of fly worms (CFSPH, 2008). Lesions in skin, subcutaneous tissue, and muscles of limbs, together with severe skin inflammation caused by secondary infection of les- ions, greatly reduce mobility as indicated (Murphy *et al*., 1999).

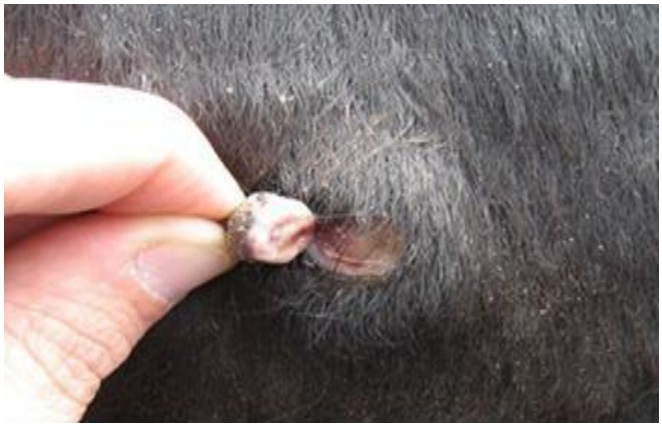

Fig 4: Inverted conical zone' of necrosis and so called a sit-fasts lesion; Source: (Blackwell, 2013).

The most common sites of nodules are head, neck, perineum, genitalia, limb and udder; involve skin, cutaneous tissues and sometime underlying part of the muscle. Severity of clinical signs depends on strain of *Capripoxvirus* and breed of the host cattle and in case of experimental infection route of transmission and dose of the virus also has determinant factor (OIE, 2010).

Pathological lesion

Gross Lesions - On autopsy, nodules may be found in the subcutaneous tissue, muscle fascia and in muscles, which are grey-pink with caseous necrotic cores, congestion, hemorrhage and edema. The subcutis is infiltrated by red, watery fluid. Similar nodules may be scattered through the nasopharynx, trachea, bron- chi, lungs, rumen, abomasum, renal cortex, testicles and uterus (Ausvetplan, 2009). Bronchopneumonia may be present and enlarged superficial lymph nodes are common. In severe cases there is synovitis and tendosynovitis with fibrin in the synovial fluid (CFS- PH, 2008).

Microscopic Lesion - Histopathological findings of the LSD disease are very characteristic and provide a basis for diagnosis. The lesions vary considerably de- pending on the stage of development. In the acute stage of the disease, it is mostly characterized by les- ions of vasculitis, thrombosis, infarction, perivascular fibroplasia. Inflammatory cell are infiltrated the in- fected areas, which includes macrophages, lympho- cytes and eosinophils. Keratinocytes, macrophages, endothelial cells and pericytes may be revealed Intra- cytoplasmic eosinophilic inclusions. The epidermis and dermis layers of the infected animal are showing edema and infiltrated with large epithelioid macro- phage type cells (OIE, 2010). There are an edema and infiltration of the epidermis and dermis with large epithelioid macrophage type cells, which have also been well described for sheep pox. They are found with plasma cells and lymphocytes in early lesions and in older lesions, fibroblasts and polymorph nuclear leucocytes with some red cells predominate. Endo- thelial proliferation is seen in the blood vessels of the dermis and sub cutis, with lymphocytic cuffing of the blood vessels, which lead to the thrombosis and necrosis. Specific intra cytoplasmic inclusions may be found in the

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various epithelial elements, sebaceous glands and follicular epithelium. These are largely eosinophilic-purple and appear to have a clear halo surrounding them, which is probably a processing artefact. The lesions are substantially the same throughout the body (Burdin, 1959; Ali *et al*., 1990; El-neweshy *et al*., 2012; Ali and Amina, 2013).

Diagnosis

According to OIE (2010), LSD can be diagnosed based on epidemiology, clinical signs, necropsy findings and laboratory diagnosis. Clinically it is diagnosed by its pathognomic nodular lesions like multiple skin nodules with circumscribed areas of erected hair, nodules around nostrils, turbinate, mouth, vulva and prepuce that can persist as hard lumps or become moist, necrotic and slough (Gari *et al*., 2011).

Also there is edema of leg and swelling of the superficial lymph nodes (Tuppurinen and Oura, 2011). At necropsy, LSD can be diagnosed by looking at the nodules on the skin, in mouth, nostrils, vulva and prepuce and, on mucous membranes, swelling of the superficial lymph nodes and systemic involved symptoms (CFSPH, 2008). Rapid laboratory diagnoses are needed to confirm the disease. Laboratory diagnosis of LSD can be made by transmission electro microscopic isolation and identification of the agent, Serological tests, routine histopathological examination and immune histological staining (Tuppurainen, 2005; OIE, 2010). Isolation of virus can be made from collected biopsy or at post-mortem from skin nodules, lung lesions or lymph nodes within the first week of the occurrence of clinical signs, before the development of neutralizing antibodies (OIE, 2010; CFSPH, 2008). Primary cell cultures are bovine skin dermis and equine lung cells, but growth of such viruses is slow and requires several passages (Tuppurainen, 2005). Serological tests are used for retrospective confirmation of lumpy skin disease but they are much more time consuming to be used as primary diagnostic methods and limited presence of detectable antibodies in serum (Vorster, 2008). Real-time PCR for the diagnosis of LSD has high sensitivity and good specificity and it is most appropriate technique (OIE, 2010; Haile, 2020; Tuppurainen and Oura, 2011).

Differential Diagnosis

Lumpy skin disease can be suspected whenever clinical signs indicate towards persistent fever which may exceed 105.8°F, wide spread skin nodules (lumps), enlarged peripheral lymph nodes, conjunctivitis, keratitis, corneal opacity, edema in the brisket and legs (Radostits *et al*., 2007). Histopathology can be an important tool to exclude viral, bacterial or fungal causes of nodular development in clinical cases and characteristic cytopathic effects which are eosinophilic intra cytoplasmic inclusion bodies in cases of LSD are well known (Brenner *et al*., 2006).

According to AUSVETPLAN, (2009) and OIE, (2010) listed below are differential diagnosis of LSD.

- Bovine herpes mammillitis: The lesions are superficial (involving only the epidermis) and occur predominantly on the cooler parts of the body such as teats and muzzle. There is no generalized disease.
- Hypodermal bovis: The parasitic fly larvae of this parasite have a predilection to migrate to the dorsal skin of the back. They cause a nodule with a small central hole through which the larva exits the body, which results in significant hide damage.
- Photosensitization: Dry, flaky, inflamed areas are confined to the unpigmented parts of the skin.
- Ringworm (dermatophytosis): The lesions of ringworm in cattle are grayish, raised, plaque-like, and often pruritic. The organism can be demonstrated with a silver stain.

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- Streptotrichosis (Dermatophilosi): Lesions are sup- erficial, often moist and appear as crusts or 0.5- to 2-cm diameter accumulations of keratinized mate- rial. Lesions are common in the skin of the neck, axillary region, inguinal region, and perineum. The organism can be demonstrated by Giemsa staining.

Treatment

There is no specific antiviral treatment available for LSD infected cattle. Sick animals may be removed from the herd and given supportive treatment con- sisting of local wound dressing to discourage fly worry and prevent secondary infections bacterial in- fection (Tuppurainen *et al.,* 2012).

Prevention and Control

Vaccination in endemic area - Immunity acquired from natural infection of the disease might be life-long and vaccination has been successfully used. LSD could be kept under control by vaccination of cattle every year (Thomas, 2002). All strains of Capripox- virus examined so far, whether of bovine, ovine or caprine origin, share a major neutralizing site, so that animals that have recovered from infection with one of the strains are resistant to infection with any other strain. Consequently, it is possible to protect cattle against LSD using strains of *Capripoxvirus* derived from either of the sheep or goats as used in Egypt by Romanian sheep pox strain (OIE, 2010). Live, atten- uated vaccines against LSD are commercially avail- able. These have antigenic homology and there is cross protection among them. Local strain of Kenyan sheep and goat pox virus has been shown to effect- ively immunize sheep, goats and cattle against infec- tion with *Capripoxvirus* with a remarkable success. The next one is attenuated South African LSD virus Neethling strain) vaccine derived from cattle, freeze dried product is also available (OIE, 2010).

Vaccination in new areas- Risks of introduction of the disease in to the new areas are by the introduction of infected animals and contaminated materials (Davies, 1991; Kitching, 1995). If the occurrence of LSD is reported or confirmed in new areas, before the spread of the disease to other areas extensively, qua- rantine of the area, slaughtering of the diseased and in contact animals and contacted equipment must be cleaned and disinfected (Davies, 1991; Netherland contingency plan of LSD, 2002). Ring vaccination of cattle within the foci of infection with a radius of 25- 50 Km, quarantine and animal movement should be restricted to eradicate the disease from the area, but if the area coverage of the disease is large, the most convenient techniques for the control of the disease is mass vaccination of the cattle. These two techniques, slaughter and vaccination were practiced in Israel and Egypt since the first outbreak of the disease occurred and it was effective for the time being (Yeruham *et al*., 1995).

Other control techniques - For countries free of the disease, the introduction of the disease can be pre- vented by restriction of the importation of the animals and their products but in those nations which experi- ence the infection can limit the spread of the lumpy skin disease by restriction of the animal movement from one place to another, quarantine, keeping of sick animals well apart from the rest of the herd and must not share drinking or feeding troughs by making awareness creation of the farmers (Thomas, 2002). Animals older than six months must be vaccinated against lumpy skin disease during spring. It is safe to vaccinate pregnant cows. All animals must be vacci- nated once a year. When vaccinating the animals during a disease outbreak, it is important to use one needle per animal so that the virus is not spread from sick to healthy animals. Professional help and recom- mendation on vaccines must be carefully followed and practiced. Antibiotics also given to prevent the secon- dary bacterial complication as the defense

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mechanism of the body weakened which can prolong the complete recovery of the diseased animals (CSFPH, 2008).

CONCLUSION AND RECOMMENDATIONS:

Emerging viral illnesses of domestic cattle caused by viruses of the genus Capripoxvirus include one of the most commercially relevant ones, lumpy skin disease. The economic impact on animals is substantial because of symptoms such as chronic weakness, decreased milk supply and weight, damaged skins, miscarriage, and mortality. Nowadays, LSD can be found in almost every country in Africa and the Middle East. Vectors of LSDV in cattle are arthropods known as mechanical haematophagus. Environment, temperature, humidity, and vector abundance all have a role in determining which mechanical vectors are most important for the transmission of LSDV. During the rainy season, which spans the end of summer until the beginning of fall, LSD is prevalent. It is possible to manage LSD by vaccinating animals, limiting their mobility, and eliminating diseased or exposed animals.

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