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REVIEW ON THE EPIDEMOLOGY AND ECONOMIC IMPORTANCE OF LUMPY SKIN DISEASE

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Abstract: The first description of the clinical signs of LSD was in 1929 in Zambia (formerly Northern Rhodesia) (Morris, 1931). Lumpy skin disease (LSD) is a generalized skin disease which is an infectious, eruptive, occasionally fatal disease of cattle caused by a virus associated with the Neethling poxvirus in the genus Capri poxvirus of the family Poxviridae. LSD was first described in Zambia and occurs in other most African countries and sporadically in the Middle East region. The genus Capri poxvirus of the family Poxviridae is the causative agent of Lumpy skin disease. Lumpy skin disease virus (LSDV) is closely related anti genially to sheep and goat poxviruses. In Ethiopia limited works has been done on this disease so far and few works have been reported on risk factors assessments, epidemiological aspects, seroprevalence and financial impacts. LSDV transmission among cattle is by the mechanical haematophagus arthropod vectors. LSD is common during wet season that is at the end of summer and beginning of autumn. The control of LSD can be achieved through vaccination, restriction of animal movement and eradication of infected and exposed animals.

Keyword: lumpy skin disease, lumpy skin disease virus, haematophagus arthropod vectors.

INTRODUCTION

Lumpy skin disease (LSD) is a generalized skin disease which is an infectious, eruptive, occasionally fatal disease of cattle caused by a virus associated with the *Neethling poxvirus* in the *genus Capri poxvirus* of the family Poxviridae. LSD was first described in Zambia and occurs in other most African countries and sporadically in the Middle East region (Hailu *et* al 2015).

Mechanical vector insects might play a major role in the epidemiology of LSD and wildlife plays a potential role in its maintenance. Factors associated with communal grazing and watering point and introduction of new cattle are significantly high risk of Lumpy skin disease occurrence. The transmission of LSDV is believed to occur mainly by blood-feeding arthropods vectors including hard ticks, biting flies and mosquitoes (Chihota *et al.*, 2001). The diseases is an acute to chronic infectious viral disease characterized by fever, nodules on the skin, mucous membranes and internal organs, high morbidity, low mortality, emaciation, enlarged lymph nodes, edema of leg and brisket, mastitis and orchitis and sometimes death (Radostits *et al*., 2007). Lumpy skin disease is usually diagnosed based on characteristic clinical signs, epidemiology, histopathology, virus isolation and PCR (Tuppurainen and Oura, 2012).

Lumpy skin disease is usually diagnosed based on characteristic clinical signs, epidemiology, histopathology, virus isolation and PCR (Tuppurainen and Oura,2012). The diagnostic tests currently available like the clinical examinations and laboratory tests (Virus isolation / identification and Serological tests) are needed to confirm it (Hailu et al 2015). The disease causes serious economic losses in most African countries including Ethiopia due to the prolonged loss of productivity from high morbidity, restrictions to the global trade of live animals and animal products, costly control and eradication measures (Hailu *et al,*2015). In Ethiopia limited works has been done on this disease so far and few works have been reported on risk factors assessments, epidemiological aspects, seroprevalence and financial impacts (Getachew *et al*., 2010). Also there was an outbreak of this disease in my area during last summer that affects many cattle even kill many of them due to lack of information regarding to its epidemiological aspects and considering it as simple wound skin disease.

LITERATURE REVIEW

Historical Background of Lumpy Skin Disease

The first description of the clinical signs of LSD was in 1929 in Zambia (formerly Northern Rhodesia) (Morris, 1931). In the beginning, LSD signs were considered to be the consequence of either poisoning or a hypersensitivity to insect bites. Same clinical signs were occurred in Botswana, Zimbabwe and the Republic of South Africa between 1943 and 1945, where the infectious nature of the disease was recognized in this outbreak. In South Africa, LSD occurred as a Causative Organism. The genus *Capri poxvirus* of the family *Poxviridae* is the causative agent of Lumpy skin disease. Lumpy skin disease virus (LSDV) is closely related antigenically to sheep and goat poxviruses (Woods, 1988). Although these three viruses are distinct, they cannot be differentiated with routine serological tests. LSDV is susceptible to 55°C/2 hours and 65°C/30 minutes. It can be recovered from skin nodules and kept at –80 °C for 10 years. The infected tissue culture fluid can be stored at 4°C for 6 months. The virus is susceptible to highly alkaline or acid ph. However, there is no significant reduction in titer when held at pH 6.6–8.6 for 5 days at 37°C. LSDV is susceptible to ether (20%), chloroform, formalin (1%), and some detergents, e.g., Sodium dodecyl sulphate. In addition, it is also susceptible to phenol (2% /15 minutes), sodium hypochlorite (2–3%), iodine compounds (1:33 dilution), Virkon® (2%) and quaternary ammonium compounds (0.5%), (Woods, 1988).

Lumpy skin disease virus has remarkably stable, surviving for long periods at ambient temperature, especially in dried scabs. LSDV is very resistant to inactivation. It is surviving in necrotic skin nodules for up to 33 days or longer, desiccated crusts for panzootic, which affected eight million cattle. The disease continuous until 1949, and generate massive economic losses (Thomas and Mare, 1945; Von Backstrom, 1945; Diesel, 1949). In 1957, LSD was identified in East Africa in Kenya. In 1972, the disease was reported in Sudan (Ali and Obeid 1977) and West Africa in 1974. While it was spreading into Somalia in 1983 (Davies, 199 a and b). The disease has continued to spread over most of African continent in a series of epizootics as previously recorded by Davies (1991 b) and House (1990). In 2001, LSD was reported in Mauritius, Mozambique and Senegal. Nowadays, LSD occurs in most of African continent (except Libya, Algeria, Morocco and Tunisia) (Tuppurainen and Oura 2012). Unti

1980s (From 1929 to 1984) the disease was limited to countries in Sub-Saharan African continent, albeit it's probable to move beyond this range had been proposed (Davies 1981). In the Middle East, the outbreaks of the LSD were reported in Oman in 1984 and 2009 (House *et al.,* 1990; Kumar, 2011; Tageldin, 2014). Kuwait in 1986 and 1991, Egypt in 1988 and 2006 (Ali *et al.,* 1990; House *et al.,* 1990; Davies 1991a; Fayez and Ahmed, 2011; Ali and Amina, 2013), Israel in 1989 and 2006 (Shimshony 1989; APHIS, 2006; Shimshony and Economides, 2006), Bahrain in 1993 and 2002-2003, Yemen, United Arab Emirates in 2000 and the West Bank also reported LSD invasion (Shimshony and Economides, 2006; Kumar 2011; Sherrylin *et al.,* 2013). In Oman, Lumpy skin disease was re-emerged once again in 2009 in a farm population of 3200 Holstein animals with 9 high morbidity and mortality rates 30-45 % and 12% respectively (Tageldin *et al.,* 2014). In Egypt, Suez Governorate, the LSD was reported in May 1988 (Ali *et al.,* 1990). The disease was arrived in Egypt with cattle imported from-Africa and kept at the local quarantine station. It spread locally in the summer of 1988 and apparently overwintered with little or no manifestation of clinical disease. Twenty-two out of twenty-six Egyptian governorates were affected with diseases, and then the disease reappeared in the summer of 1989 and continuous for five to six months. This epizootic showed low morbidity rate (2%) due to the vaccination procedure that included nearly two million cattle with a sheep pox vaccine. However, approximately 1449 animals died. In the summer of 2006, in one farm with a total of 30 cases in dairy cows, LSD outbreak was re-emerged once again in several Egyptian governorates, where all age groups and both sex of Egyptian cattle were infected with severe and serious complications (Fayez and Ahmed, 2011; Ali and Amina, 2013). One of the outbreaks of LSD in African continent was occurred in central Ethiopia in 2007 to 2011. These outbreaks were described as active. It was investigated in four districts: Adama, Wenji, Mojo and Welenchiti. The totally 1,675 outbreaks were reported over 5 years period from 2007 to 2011, with 62,176 cases and 4,372 deaths. The Oromia represented the highest numbers of outbreaks (1,066), followed by Amhara (365) and the Southern Nations, Nationalities and People's Region (123). The 2010 were reported the highest number of outbreaks that were frequently seen between September and December. The morbidity and mortality rates were 13.61% (296) and 4.97 % respectively (Ayelet *et al* 2014). The disease has been reported in Turkey in October 2013, Iran and Iraq in 2014 (Sherrylin *et al* 2013).

Etiology

Lumpy skin disease is caused by *Lumpy Skin Disease virus* (LSDV), which is a member of family *Poxviridae*, subfamily *Chordopoxvirinae,* genus *capripoxvirus*, the prototype strain *Neethling Virus*. LSDV is pleomorphic, enveloped, brick- or oval-shaped dsDNA virus with a molecular size of 350*300nm 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 in viral virulence, host range and/or immune evasion of host (Kara *et al.*, 2003).

All Capri poxviruses 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 chorioallantoic membranes of embryonated chicken eggs, causing macroscopic pock lesions. The replication of LSDV occurs in the cytoplasm of the host cell resulting in intracytoplasmic eosinophilic inclusion bodies (EFSA, 2015).

Lumpy skin disease 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 its host. LSDV can persist in skin plugs for about 42 days (Babiuk *et al*., 2008b).

Epidemiology of lumpy skin disease

Lumpy skin disease is an important, economically devastating, notifiable disease which brought production loss in cattle due to generalized malaises and chronic debility (Tuppurainen and Oura, 2011). Good understanding of epidemiological aspects of LSD related to pathogen, host and environment might aid for control and prevention mechanisms. Particular emphasis should be given to exposure of hosts to pathogen in suitable environment 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 (OIE, 2010).

Geographic Distribution

Lumpy skin disease was originated from Sub Sahara Africa 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 endemic 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 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 (Tuppurinen and Oura, 2011, 2012).

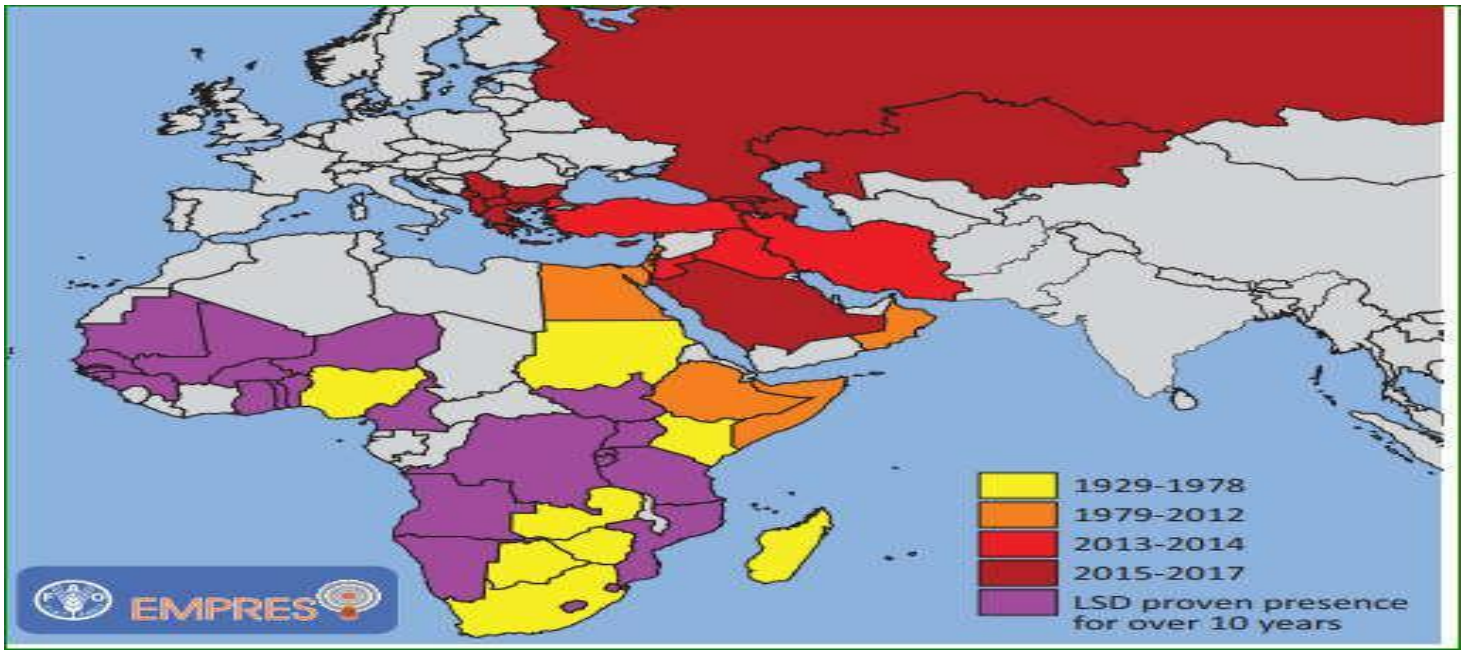

Figure 1: Countries that have reported of LSD.

Source: (Tuppurainen *et al*., 2017).

Risk factors

host risk factors

Lumpy skin disease is a disease of cattle and causes several disorders. Though all breeds and age group are susceptible, Bos taurus is particularly more susceptible to clinical disease than zebu cattle and Bosindicus (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 severely 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 symptoms are rapid to appear. But traditional calf management 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 disease depends on susceptibility, immunological status, and age of the host population and dose and route of virus inoculation (CFSPH, 2008).

pathogen risk factors

Lumpy skin disease is one of the species of Capri poxviruses that is resistant to different chemical and physical agents (Murphy et al., 1999). Capri poxviruses have lipid-containing envelopes and susceptible to a range of detergents containing lipid solvents 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).

Lumpy skin disease 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, in necrotic tissue remaining 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). Capri poxviruses are very resistant in the environment and can remain viable for long periods on or off the animal host. They may 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 (AUSVETPLAN, 2009).

Environmental risk factors

Environmental determinants 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 movement from place to place and presence of water bodies are some of potential risk factors of LSD (Tuppurainen and Oura, 2011).

Lumpy skin disease is associated with increased number of insects which are mechanical vectors (Magori-cohen, 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).

Host susceptibility

Lumpy skin disease is a disease of cattle and causes several disorders. Though all breeds and age group are susceptible, Bos taurus is particularly more susceptible to clinical disease than zebu cattle. Among Bos taurus, fine-skinned Channel Island breeds develop more severe disease (OIE, 2010). Lactating cows appearing to be severely 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). Young animals are severely affected and clinical symptoms are rapid to appear. Apart from these animals, few cases have been reported in Asian water buffalo (Bubalus bubalis).Natural cases have not been seen in an impala and a giraffe but, demonstrated in both of them after experimentally inoculation with LSDV. The absence of reservoir host for LSD virus might lead us to the assumption that infection might persist in the endemic areas at a low level as unapparent or mild form in the cattle population (OIE, 2008).

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 discharge, lachrymal secretions, milk, semen and Very rarely 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 vectors 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 demonstrated (Annandale *et al*., 2013). A more recent study demonstrated the 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 arthropods vectors including hard ticks, biting flies and mosquitoes (Chihota *et al.*, 2001; Getachew *et al*., 2010; Magori-cohen, 2012). This vector related transmission is apparently 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 transmission, 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 susceptible 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 transstadial and trans

ovarian transmission of LSD virus by *Boophilus decoloratus* and mechanical transmission by *Repicephalus appendiculatus* and *Ambyloma hebraeum* (Tuppurainen *et al*., 2011).

Mosquitoes (female *Aedes egypti and Culex quinquefasciatus)* and other flies such as *tabanids* (horse flies), biting midges (*Culicoides nubeculosus*), 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 suggests, outbreaks of LSD is highly associated with prevalence 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).

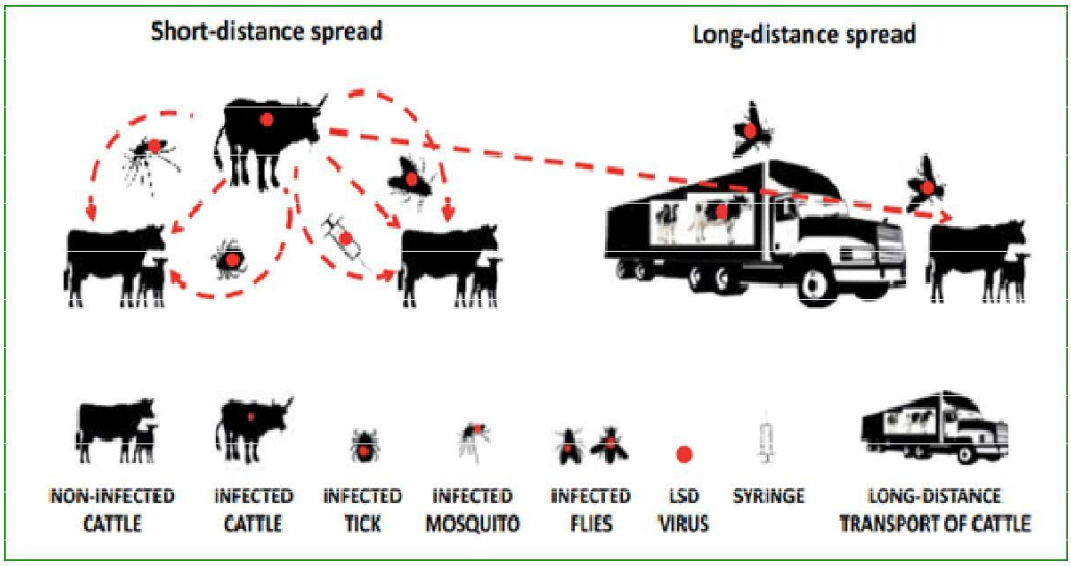

Figure 2 : Schematic illustration of the spread of LSDV

Source (Tuppurainen *et al*.2017).

Pathogenesis

Lumpy skin disease is developed by 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 with development of inflammatory nodules on skin (Vorster, 2008).

Lumpy skin disease is generalized and epitheliotrophic disease that cause localized and systemic reaction and results in vasculitis and lymphadenitis which result in to edema 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).

Lumy skin disease 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 factors 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).

Clinicall sign

Course of lumpy skin disease may be acute, sub-acute and chronic. The virus causes from unapparent infection 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 depression 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).

Figure 3: Lumpy skin disease randomly distributed nodules on the skin**.**

Source: (Coetzer *et a.,l*2004).

In severe cases, ulcerative lesions may develop in mucous membrane of mouth, trachea, and larynx and esophagus (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 lesions, greatly reduce mobility as indicated by (Murphy *et al.* 1999).

Figure 4: Inverted conical zone' of necrosis and so called a sit-fasts les.

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 Capri poxvirus 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, bronchi, 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 Histo pathological findings of the LSD disease are very characteristic and provide a basis for diagnosis. The lesions vary considerably depending on the stage of development. In the acute stage of the disease, it is mostly characterized by lesions of vasculitis, thrombosis, infarction, perivascular fibroplasia. Inflammatory cell are infiltrated the infected areas, which includes macrophages, lymphocytes 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 macrophage 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. Endothelial 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 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).

Microscopic Lesion

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Diagnosis

According to OIE (2010), LSD can be diagnosed based on epidemiology, clinical signs, necropsy findings and laboratory diagnose. 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 (Tuppur Histo pathological findings of the LSD disease are very characteristic and provide a basis for diagnosis. The lesions vary considerably depending on the stage of development. In the acute stage of the disease, it is mostly characterized by lesions of vasculitis, thrombosis, infarction, perivascular fibroplasia. Inflammatory cell are infiltrated the infected areas, which includes macrophages, lymphocytes 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 macrophage type cells (OIE, 2010).

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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; 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.

Streptotrichosis (Dermatophilosi):-Lesions are superficial, often moist and appear as crusts or 0.5- to 2-cm diameter accumulations of keratinized material. 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 consisting of local wound dressing to discourage fly worry and prevent secondary infections bacterial infection (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 Capri poxvirus 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 Capri poxvirus derived from either of the sheep or goats as used in Egypt by Romanian sheep pox strain (OIE, 2010). Live, attenuated vaccines against LSD are commercially available. These have antigenic homology and there is cross protection among them. Local strain of Kenyan sheep and goat pox virus has been shown to effectively immunize sheep, goats and cattle against infection with Capri poxvirus 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, quarantine 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; AUSVETPLAN, 2009). 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 prevented by restriction of the importation of the animals and their products but in those nations which experience 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 vaccinated 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 recommendation on vaccines must be carefully followed and practiced. Antibiotics also given to prevent the secondary bacterial complication as the defense mechanism of the body weakened which can prolong the complete recovery of the diseased animals (CSFPH, 2008).

Status of lumpy skin disease in Ethiopia

In Ethiopia, LSD was first observed in 1983 in the western part of the country around southwest of Lake Tana (Gari, 2011). After its first appearance, an explosive sudden epidemic spread from the north through the central to the southern part of the country. The national disease report showed LSD has spread virtually to all the regions in the country and in different agro-climatic zones. Because of the wide distribution of the disease and the size and structure of the cattle population in Ethiopia, LSD is one of the most economically important livestock diseases in the country (Gari *et al.,* 2010, 2012). A recent study across different agro-ecological zones in Ethiopia showed an overall observed LSD prevalence of 8.1% and a mortality of 2.12%. The case fatality is estimated

to be 2% (Gari *et al*., 2010). The highest frequency of LSD outbreaks in the country have been reported between September and December, with the highest numbers in October and November; which is the end of the main rainy season in most parts of the midland and highland agro-ecological zones and the lowest number is reported in May (Ayelet *et al*., 2014). Among indigenous local zebu cattle Fogera breed located in the northwest of the Ethiopia is reported to manifest severe clinical disease in epizootic occurrence of LSD (Gari *et al.*, 2011; OIE, 2008). A study in Ethiopia also shows that communal grazing, watering points and movement of infected stock have been found to be associated with the occurrence of LSD (Getachew *et al*., 2010) According to Gari *et al.,* (2010), LSD is one of reported diseases in Ethiopia which deserves outbreak notification to the National veterinary services. According to Ayelet *et al*., (2014) analysis of retrospective data between January 2007 and December 2011 indicated that LSD is reported from all regions of the country except Harari and Dire Dawa. The majority of outbreaks are frequently reported from midland agro-climate zone of Oromia, Amhara and the Southern Nations, Nationalities and People's Region, which is known to be favorable for the breeding of the blood feeding insect vectors of LSD and has the highest population density of livestock in Ethiopia (Gari *et al.,* 2010). In Ethiopia limited works has been done on this disease so far and few works have been reported on risk factors assessments, epidemiological aspects, seroprevalence and financial impacts (Getachew *et al*., 2010). Control of LSD in Ethiopia relies mainly on ring vaccination carried out at the onset of an LSD outbreak. In Ethiopia both Kenyan SGPV and Neethling strain vaccines are produced at the National Veterinary Institute (NVI) and the Kenyan SGPV strain are widely used for all cattle, sheep and goats. The vaccine protection lasts for a minimum of three years (Gari *et al.*, 2011).

Economic importance of lumpy skin disease.

Lumpy skin disease is one of the economically significant diseases in Africa and the Middle East countries that cause severe production loss in cattle. The economic importance of the disease is mainly due to having high morbidity rate rather than mortality (Tuppurainen and Oura, 2011). The impact of lumpy skin disease can broadly be divided into direct losses, i.e. the direct impact on animal health and productivity, and indirect losses, which include mitigation or control efforts, lost export opportunities (EFSA, 2015).

Direct losses include visible losses such as animal death and illness or stunting that result from disease or subsequent control methods. Invisible losses, on the other hand, include less immediate impacts of animal disease, such as reduced productivity or changes in herd fertility, which result in the need to have a higher proportion of animals in a breeding group rather than in production. In resource-limited countries, the slaughter of infected and in-contact animals is usually seen as a waste of a valuable source of food and is not usually feasible. This kind of loss primarily affects the stakeholders of the agriculture sector, for example farmers (EFSA, 2015). Among indirect losses, forgone revenues should be considered, namely the indirect economic impact of animal diseases resulting from ban on international trade of livestock, losses in consumer confidence and negative effects on other sectors of the economy. The dynamics of supply and demand of animals and animal products can be disturbed by large outbreaks and their impact can be much larger than combining the impacts observed on single farms. Furthermore, the mitigation and control costs should be also considered, i.e. the costs of the drugs, vaccines, surveillance and labor needed to carry out control measures. These costs may also have an impact on tax payers because of the supplementary resource that may be needed for the implementation of control program (EFSA, 2015).

Major consequences of the disease are retarded genetic improvement, inability of the animal to work, draught power and traction loss due to lameness, decreased milk production, abortion, infertility, chronic debility in beef cattle and loss of condition and damaged hides cause enormous economic losses (Babiuk *et al.*, 2008). If LSD became endemic, continuing economic loss and poor productivity would occur due to stock losses, reduced production in cattle industries, ban on international trade of livestock and costs of annual mortality, treatment and vaccination. Lesions in skin, subcutaneous tissue, and muscles of limbs, together with severe skin inflammation caused by secondary infection of lesions, greatly reduce mobility (Murphy *et al.,* 1999). According to Gari *et al*., (2011) annual financial loss following an LSD outbreak in Ethiopia is calculated as the sum of the values of the annual production losses due to

morbidity and mortality and the costs for treatment and vaccination. Treatment cost represents the expenses incurred by farmers for medication.

C=Md + (B+M+Wop) +V+T

where **C** is the total financial costs, **M** is the milk production losses, **B** is the beef production losses, **Wop** is the work output losses, **Md** is the mortality losses, **V** is the vaccination costs, **T** is the treatment costs .

Lumpy skin disease incidence interferes with normal herd dynamics, causing a reduction of surplus in the case of mortality, or a reduction of stock for the market in affected herds because of long term morbidity that can lower weight gain. The valuation of the draft power loss depends on the point in the crop season that an ox fell sick and on the corresponding demand for draft power during that specific season. The reduced work output of draft oxen due to LSD is an important loss for the mixed crop-livestock farming system. Morbidity of draft oxen leads to reduce crop production through a reduction in cultivation and lower yields due to inefficient land preparation and timing (Gari *et al*., 2011). According to Gari *et al*., (2011), the financial loss impact between local zebu and HF/crossbreds shows that HF/crossbreds have far higher production losses in most parameters compared with local zebu cattle; the financial loss impact thus has a linear relationship with the incidence of the disease in each breed type. Milk production losses of up to 50% per lactation have been reported in infected herd. This shows that lumpy sin disease infection is very important in high producing exotic breeds. High economic losses were also incurred by feedlot owners for extra feed bought to assist sick animals during their recovery and the lengthened period required for fattening. Further, animals that recovered were no longer fit for export purposes and were therefore sold at local markets at a lower price. Lastly, the survey found that animals that had recovered from LSD produced less milk and suffered a loss in draught power (Ayelet *et al*., 2014). Overall, LSD is considered as a disease of high economic pressure because of its ability to compromise food security through protein loss, draft power, reduced output of animal production, increase production costs due to increased costs of disease control, disrupt livestock and their product trade, result of reduced milk yield, weight loss, abortion, infertility in cows, mastitis and infertility in lactating cows, infertility in bulls (Kumar, 2009). Permanent damage to the skin and hide greatly affect leather industry. It causes ban on international trade of livestock and causes prolonged economic loss as it became endemic and brought serious stock loss (AUSVETPLAN, 2009; Getachew *et al*., 2010).

CONCLUSION AND RECOMMENDATIONS

Lumpy skin disease is one of the most economically significant trans boundaries, emerging viral diseases of domestic cattle caused by viruses of the genus *Capri poxvirus*. It has significant economic importance in animals, due to chronic debility, reduction in milk production and weight, damaged hides, abortion and death. LSD is now endemic in most African and Middle Eastern countries. LSDV transmission among cattle is by the mechanical haematophagus arthropod vectors. The importance of different mechanical vectors in the transmission of LSDV is likely to vary in different geographical regions, depending on the environment, temperature, humidity and abundance of the vectors. LSD is common during wet season that is at the end of summer and beginning of autumn. The control of LSD can be achieved through vaccination, restriction of animal movement and eradication of infected and exposed animals.

- Based upon the above conclusion the following recommendation are forwarded
- A better control of illegal livestock and animal product movements should be considered.
- The government and/ NGO should facilitate awareness creation and training for farmers and veterinary staff in recognizing the disease under field conditions,
- If LSD entered the disease free country, rapid detection and prompt culling of infected
- In order to effectively control LSDV in endemic countries, a comprehensive understanding of the ecology of different blood-feeding and biting arthropod species in the cattle farming setting is important.
- Animals suspected with LSD should be isolated and the farm should be quarantined until definitive diagnosis is determined.

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