

## LUMPY SKIN DISEASE: AN EMERGING BOVINE VIRAL INFECTION IN INDIA

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### Abstract

Lumpy skin disease (LSD) is a highly infectious emerging viral disease of cattle and buffaloes in India. The main source of transmission of the disease is arthropods (mosquitoes, biting flies, *Culicoides* midges and three blood sucking hard ticks) which act as mechanical vector. Major clinical signs include skin nodules all over the body, fever, lacrimal discharge, nasal discharge, anorexia, decreased milk yield, emaciation, depression and reluctance in movement. Since the first report of lumpy skin disease in India, it is spreading rapidly across the country causing a possible threat to the cattle and buffalo population in terms of productivity and mortality.

**Key words:** Emerging, India, Lumpy skin disease, Productivity

### Introduction

Lumpy skin disease (LSD) is an important infectious transboundary infection of cattle and buffaloes of all age groups and breeds. This is an emerging viral infection having important economic significance across the world. LSD is also called as neethling virus disease, pseudo urticaria, exanthema nodularis bovis and Knopvelsiekte causing temporary to permanent damage to the skin characterized by distinctive nodular lesions leading to detrimental effect on the commercial value of hides (Amenu *et al.*, 2018; Feyisa, 2018). Moreover, LSD causes severe loss to the farmers in terms of chronic debility, decreased milk yield, poor growth rate, infertility, abortion and even death (Babiuk *et al.*, 2008; Abutarbush *et al.*, 2013). As per 20<sup>th</sup> Livestock Census, the total cattle and

buffalo population of India is 192.49 and 109.85 million respectively. Most of the Indian population is directly involved in dairy industry. Recently, lumpy skin disease has been reported in India and is spreading rapidly across the country which may indirectly correlate the productivity due to stress by associated viral infection. In this context, the present article describes the details of LSD along with possible way of prevention and control measures to overcome the production loss.

### History of outbreaks

Lumpy skin disease (LSD) had been reported for the first time in North Rhodesia (Zambia) in the year 1929, which was initially thought to be due to an allergic reaction in cattle caused

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by biting insects (MacDonald, 1931). Similar clinical signs were recognized in Botswana, Republic of South Africa and Zimbabwe in between 1943 and 1945 (Al-Salihi, 2014). In 1972, LSD was reported in Sudan (Ali and Obeid, 1977). It was transmitted to Somalia in 1983 (Davies, 1991). By the year 1974, the disease was spread to west as far as Nigeria and in 1977 it was transmitted to Mauritania, Mali, Ghana and Liberia (OIE, 2010). LSD was restricted to countries in sub-Saharan Africa from 1929 to 1986 (Gumbe, 2018). Epidemic of lumpy skin disease outside the sub-Saharan African continent had reported in Egypt (1988), Israel (1989), Kuwait (1991), Lebanon (1993), Yemen (1995), United Arab Emirates (2000), Bahrain (2003), Israel (2006-2007) and Oman (2010) (Tageldin *et al.*, 2014). In India, lumpy skin disease had been first reported from Mayurbhanj and Bhadrak Districts in Odisha in August, 2019 (Sudhakar *et al.*, 2020).

### Causative agent and its properties

Lumpy skin disease (LSD) is caused by lumpy skin disease virus which has epitheliotropic property (Gari *et al.*, 2010). It is a member of the genus *Capripoxvirus* of the family *Poxviridae*. Sheep pox and Goat pox are the two other virus species of genus *Capripoxvirus*. Genetically, LSD virus is very similar to the other Capripox species such as Sheep pox and Goat pox virus. The virus has a double-stranded DNA genome of about 151 kbp. It is enveloped, linear, ovoid shaped virion measuring 220-450 nanometer (nm) by 140-266 nm (Stram *et al.*, 2008).

Lumpy skin disease (LSD) virus is susceptible to temperature (55°C for 2 hours; 65°C for 30 minutes) and detergents containing lipid solvents like ether (20%), chloroform, formalin (1%), phenol (2%), sodium hypochlorite (2-3%), iodine compound (1:33 dilution) and quaternary ammonium compounds (0.5%). However, the virus can withstand drying, pH shift (if not an extreme pH) and can remain

viable for months in dark room such as infected animal shades. It can persist in skin plugs for about 42 days (Abeya *et al.*, 2018). The virus can be isolated from the nodules kept at -80°C for 10 years and infected tissue culture fluid stored at 4°C for 6 months.

### Host range

Lumpy skin disease (LSD) virus has narrow and specific host range. It does not have any non ruminant host (Shen *et al.*, 2011). It causes natural infection in cattle (*Bos indicus* and *Bos taurus*) and Asian water buffaloes (*Bubalus bubalis*) (Ali *et al.*, 1990; Tuppurainen *et al.*, 2017). However, susceptibility rate is significantly higher in cattle (30.8%) than buffaloes (1.6%) (El-Nahas *et al.*, 2011). All cattle appear to be equally susceptible to the disease irrespective of breeds (Al-Salihi, 2014). However, some researchers have reported that *Bos taurus* is more prone to LSD than *Bos indicus* which might be due to exotic breeds are less resistant against diseases. Coetzer (2004) had observed that the losses were more in severe cases of the disease in *Bos taurus* or imported breeds with relatively thin skin than *Bos indicus* or indigenous breeds with thicker skin diameter. Cattle of both sexes and all age groups are susceptible to the infection, however there is evidence that young animal may be more susceptible to the severe form of the infection (Al-Salihi, 2014). The disease had been demonstrated after experimental infection in impala, giraffe and Thomsons gazelle (Young *et al.*, 1968). It had also been reported in an Arabian oryx and springbok (Greth *et al.*, 1992; Tuppurainen *et al.*, 2017).

### Transmission

The principal way of transmission of LSD virus is arthropods which act as mechanical vector. Albeit rare transmission occurs through direct contact and contaminated feed and water (Ali *et al.*, 2012). Incidence is more prevalent in low lying agro-climate zone and along water courses

during the wet and warmer condition of summer and autumn months, which favours arthropod multiplication (OIE, 2010). The mosquitoes (*Culex mirificens* and *Aedes aegypti*), biting flies (*Stomoxys calcitrans* and *Biomyia fasciata*), *Culicoides* midges and three blood sucking hard ticks viz. *Rhipicephalus (Boophilus) decoloratus* (blue tick), *Rhipicephalus appendiculatus* (brown ear tick) and *Amblyomma hebraeum* are identified as vector as well as act as reservoir of the virus (Sarma, 2009; Lubinga *et al.*, 2014). The virus is present in all secretions of the infected animals such as blood, saliva, semen, nasal discharge, lachrymal discharge and milk, and cutaneous lesions (Babiuk *et al.*, 2008). Moreover, infected pregnant cows are known to deliver calves with skin lesions (Tuppurainen *et al.*, 2017).

### Pathogenesis

Lumpy skin disease (LSD) virus enters the host body through skin or gastro intestinal tract mucosa resulting in viraemia accompanied by febrile reactions which persist for two weeks. The virus reaches the regional lymph nodes and causes lymphadenitis. The virus causes skin lesions due to its rapid replication in specific cells such as endothelial cells of lymphatic and blood vessel walls with development of inflammatory nodules on the skin (Vorster and Mapham, 2008). The disease is a generalized epitheliotropic disease causing localized and systemic reaction resulting vasculitis and lymphadenitis that result to edema and necrosis. In some severe cases, thrombosis and infarction may be observed. Skin nodules may be changed to grey-pink with caseous necrotic cores. Circumscribed necrotic lesions may ulcerate referred as 'sit-fasts'. Lymph nodes are enlarged and secondary bacterial infections are common in the necrotic cores. Multiple virus-encoded factors are produced during infection, which influence pathogenesis and disease (Tuppurainen *et al.*, 2017).

### Clinical signs

Lumpy skin disease (LSD) can occur in acute, sub acute and chronic forms (Gari *et al.*, 2010). The incubation period of the disease varies from 4 to 14 days in experimental conditions whereas in natural infection, the incubation period is 2 to 5 weeks (Carn and Kitching, 1995; Tuppuraine *et al.*, 2005). The different clinical signs visible in the disease are skin nodules all over the body, fever (may exceeds 41°C) which persists for 6 to 72 hours, lacrimal discharge, nasal discharge, anorexia, decreased milk yield, emaciation, depression and reluctance in movement (Al-Salihi, 2014). Skin nodules (Fig. 1) can be observed over the muzzle, nares, back, limbs, scrotum, udder, perineum, eyelids, ears, nasal mucosa, oral mucosa and tail (Salib and Osman, 2011). Skin nodules may disappear, but sometimes these may persist as hard lumps or become moist, necrotic and ulcerated (Fig. 2). The ulcerated lesions may create a hole in the skin (Abutarbush *et al.*, 2013). These ulcerated lesions may be contaminated by secondary bacterial complication and infestation of fly worms (Amenu *et al.*, 2018). Tuppurainen *et al.*, (2017) observed that subscapular and prefemoral lymph nodes had become enlarged to 3-5 times of their normal size and easily palpable (Fig. 3). Edematous and inflammatory swellings of the face, brisket and one or more limbs (Fig. 4, 5), may be seen leading to difficulties in movement. In some cases, unilateral or bilateral keratitis may be observed in infected animals (Jameel, 2016). Pneumonia is a common sequel in animals affected by LSD with lesions in the mouth and respiratory tract (Al-Salihi, 2014). Females infected with the disease may remain in anestrus for months or become sterile due to poor body condition and other stress (Al-Salihi, 2014). In some cases, the affected pregnant animals may abort. In infected bulls, there may be orchitis leading to infertility to sterility (Sarma, 2009).

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Fig. 1. Characteristic nodular skin lumps of lumpy skin disease (a, b)



Fig. 2. Ulcerated skin lesion

Fig. 3. Easily palpable enlarged subscapular lymph node



**Fig. 4. Edematous swelling of brisket**



**Fig. 5. Swelling of the right forelimb**



**Fig. 6. Insect proof housing for cattle in endemic area**

### **Morbidity and mortality**

The morbidity and mortality rates vary based on the status of the host's immune system and seasons due to abundance of arthropod vectors. Morbidity rate of the disease is highest in wet and warm weather and lowest during the dry season (OIE, 2008). Morbidity rate varies between 5 - 45 percent and mortality rate is 1- 5 percent (Al-Salihi, 2014). However, in natural outbreaks, morbidity rate may reach as high as 100% and mortality rate may sometimes reach 40% (Amenu *et al.*, 2018).

### **Diagnosis**

Lumpy skin disease (LSD) can be diagnosed based on peculiar clinical signs of the disease and the incidence of disease outbreak in nearby areas. However confirmatory diagnosis is furnished by laboratory tests with the presence of viral antigen or antibody (Amenu *et al.*, 2018). Non necrosed skin nodules biopsy samples or post mortem skin nodules, lung lesions and lymph nodes are collected within first week of onset of clinical signs of the disease before the development of neutralizing antibodies for isolation and identification of the viral antigen (Abeya *et al.*, 2018). The viral antigen can also be isolated from buffy coat of the blood sample collected into EDTA or heparin during the viraemic stage of the disease (Al-Salihi, 2014). LSD virus grows slowly on cell cultures and may require several passages. They can be recognizable by cytopathic effects. The virus can propagate in the chorioallantoic membranes of embryonated chicken eggs and African green monkey kidney (Vero) cells, causing macroscopic pock lesions. The replication of the virus occurs in the cytoplasm of the host's cell resulting in intracytoplasmic eosinophilic inclusion bodies (Abeya *et al.*, 2018).

Commonly used method of diagnosing LSD is detecting viral DNA using the polymerase chain reaction. Other laboratory tests carried out for

diagnosis of LSD are electron microscopy, virus neutralization test, indirect fluorescence tests, agar gel immunodiffusion test, Western blot and enzyme-linked immunosorbent assay (OIE, 2016).

It is important to differentiate the LSD from other similar skin diseases *viz.* pseudo lumpy skin disease, pseudo cow pox, bovine papular stomatitis, insect bites, urticaria, photosensitization, dermatophilosis, demodicosis, besnoitiosis, ringworm etc. (Tuppurainen *et al.*, 2017).

### **Treatment**

As LSD is a viral disease, there is no specific treatment till date. However, the infected animals are treated with antibiotics to prevent other secondary bacterial invasion and other supportive medications to subside the clinical signs (Al-Salihi, 2014). Local dressing of the skin with antiseptic is also suggested to prevent flies and bacterial infection (Amenu *et al.*, 2018). Antibiotics like penicillins, cephalosporins, tetracyclines, fluoroquinolones etc. are indicated for 5 to 7 days depending on severity of the disease (Feyisa, 2018). Administration of antihistaminic and non steroidal anti-inflammatory drugs are also indicated. Antipyretic drug like paracetamol is given to subside the fever. To overcome the anorexia and speedy recovery liver supportive and multivitamins should be administered regularly.

### **Prevention and control**

In endemic areas, isolation of the diseased animals are carried out to prevent spread of the disease into the healthy herd. Moreover, newly purchased animals must be quarantined before introduction to the healthy herd. Purchase of animals from the endemic areas should strictly be restricted. In addition, Abeya *et al.* (2018) had suggested that in newly spread areas the infected animals are to be slaughtered and

properly disposed along with contacted equipments and premises must be thoroughly cleaned and disinfected. Control of insect vectors to minimize the spread of LSD by using insecticides, insect repellents and providing insect proof housing (Fig. 6) for animals (Babiuk *et al.*, 2008) are recommended. In endemic areas, vaccination of the animals (above 6 months age) every year is the method of control of the disease. Four live attenuated strains of capripoxvirus are currently used as vaccines to control the lumpy skin disease outbreak *viz.* Kenyan sheep and goat pox strain (KS-1), Yugoslavian RM 65 sheep pox strain, Romanian sheep pox strain and South African

neethling lumpy skin disease virus strain.

### Conclusion

Lumpy skin disease (LSD) is a highly infectious viral disease of cattle and buffaloes. As the disease is newly reported in India, it becomes a serious threat to the entire bovine population in India. Though the mortality is less in LSD but negligence and lack of knowledge may cause great production loss or even death of the animals. Better understanding and awareness for LSD are of utmost necessity to tackle the production loss due the disease in India.

**Conflict of interest:** The authors declare that they have no competing interests.

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