

Popular Article

Emergence of Lumpy skin disease in India

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Introduction

Emerging disease: “a new infection resulting from the evolution or change of an existing pathogenic agent, a known infection spreading to a new geographic area or population, or a previously unrecognized disease diagnosed for the first time and which has a significant impact on animal or public health”. Lumpy skin disease (LSD) is a transboundary, arthropod-borne viral disease of cattle and buffaloes. Lumpy skin disease (LSD, Pseudo-urticaria, Neethling virus disease, exanthema nodularisbovis, and knopvelsiekte) is an infectious disease. It is caused by a virus (LSDV) in the family Poxviridae, genus Capripoxvirus. Currently the disease has been emerged as a devastating threat in Europe, Middle East and the southeast Asia. The economic implications of the disease are high due to morbidity (5%-45%) rather than mortality (usually under 10%). OIE has categorized Lumpy skin disease as a notifiable outbreak considering its transboundary potential and threat as agro-terrorism disease.

Aetiology

Lumpy skin disease (LSD) caused by lumpy skin disease virus (LSDV) which is related to that of sheep pox. LSDV is a member of the genus Capripoxvirus within the subfamily Chordopoxvirinae, family Poxviridae. The LSDV is quite resistant against high ambient temperature and desiccation. The virus retains its ability to infect for long periods (18–35 days) in dry necrotic nodule laden hides even after the animal is slaughtered. But the virus is destroyed immediately when exposed to direct sunlight or lipophilic detergents. Virus becomes inactivated in 2 h at 55 °C as well as it takes 30 min at 65 °C temperature. Virus is prone to highly alkaline or acidic conditions. It can however, withstand minor pH fluctuations ranging between 6.6 and 8.6 at 37 °C for five days without any substantial decrease in titers. Other disinfectants such as iodine compounds (1:33 dilution), formalin (1%), quaternary ammonium compounds (0.5%), phenol (2% for 15 min), ether (20%), chloroform and sodium hypochlorite (2–3%) are highly effective against this virus.



Epidemiology in India

In India, first outbreak of the disease was noticed in Odisha (August 2019) and reported to the OIE on 18 November 2019. India faced three primary outbreaks of LSD in Odisha. The first incident started on 12 August 2019, in the Mayurbhanj districts of Orissa, where in a farm 9 cases (135 animals) were reported. Second outbreak was reported from Patalipura, where in a farm 20 cases (441 animals) were reported. Third case outbreak was reported on 20 August 2019 in Bhadrak, where in a farm 50 cases (356 animals) were reported. In 2020, the disease is prevalent in Maharashtra and Madhya Pradesh and now as of August 8, 2022, Rajasthan has reported 2,111 deaths of cattle, followed by Gujarat at 1,679, Punjab at 672, Himachal Pradesh at 38, Andaman & Nicobar at 29 and Uttarakhand at 26.

Table- Current situation in India

Year	No. of animals	Area	Reported By
19 May, 2022	120 cattle died	Jaisalmer district of Rajasthan	The Times of India
24 May, 2022	10 cattle died, 517 infected	Jamnagar and Dwarka, Gujarat	Ahmadabadmirror.com
31 May, 2022	120 cattle died	Jalore, Rajasthan	Dainik Bhaskar

Hosts

Cattle and buffalo are susceptible hosts. Exotic cattle breeds are more susceptible than indigenous cattle breeds. Animals of all ages are susceptible but calves are more susceptible and develop lesions within 24 to 48 hours. Wild animals under natural conditions, are resistant to infection but experimental infection produced clinical lesions in Giraffe, impala, Arabian oryx, springbok, oryx, and Thomson's gazelle. Normally the role of wildlife in the transmission and maintenance of LSDV has been found almost negligible. Humans are also resistant to the virus.

Transmission

Mechanical transmission by vectors is the prime route of spread of disease. In most of the endemic countries like sub-Saharan Africa, Egypt and Ethiopia, the disease incidences significantly increase with the onset of seasonal rains and summer season, coinciding with the peak activity of the vectors. Incidences decrease significantly with the onset of winters and reappears with arrival of spring and summer. It was



observed that despite restricted animal movements infection spreads to 80 to 200 km away through air movement of biting insects. The tick *Amblyomma spp.*, *Rhipicephalus decoloratus*, *Rhipicephalus appendiculatus* and *Amblyomma hebraeum* have been reported as a mechanical vectors and reservoirs of virus. The biting flies (*Stomoxys calcitrans* and *Biomyia fasciata*) and mosquitoes (e.g. *Culex mirificens* and *Aedes natrionus*), are also involved in mechanical transmission of disease. Virus is secreted in milk, nasal secretions, saliva, blood and lachrymal secretions forming indirect source of infection for animals sharing feeding and watering troughs. LSD virus transmission through intrauterine route has been documented in literature. The infection has been assumed to be transmitted from infected mother to calf via milk secretions and skin abrasions. The virus persists in the semen for up to 42 days post-infection and it has been established by experimental infection. Iatrogenic route can be another route of spread of virus when single needle used for mass vaccination that can acquire the virus from the skin scabs or crusts. Therefore, it suggests that quarantine could not be the only method to prevent the spread of LSD as movement of vector can blow out the disease.

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.

Clinical signs

- The clinical signs of LSD have two febrile phases (biphasic fever), which is appeared after variant incubation period 4-12 days (usually 7 days). The temperature of the infected animals raises to 40-41.5°C, which may persist for 6-72 hours or more and may rarely be up to 10 days. The infected animals also show lacrimation, increased nasal and pharyngeal secretions, anorexia, dysgalactia, general depression and a disinclination to move.
- The initial clinical signs of LSD are varied in severity that depends on the management system of the herd but do not relate to animal sex or age. Multiple firm circumscribed nodules are developed in the skin of the animals.
- These nodules are suddenly erupted within 1-2 days. The erupted nodules may be widespread or restricted to just a few lesions. The head, neck, the perineum, the genitalia, udder, and the limbs are the predilection sites. The whole of the skin of the infected animal is covered with lesions



infrequent cases. Typical LSD lesions are round, irregular, about 5-50 mm in diameter, and appear as circumscribed areas of erect hair over a firm and slightly raised area of skin.

- The healthy skin is clearly recognized by the adjacent skin reaction. The affected skin is hyperaemic, and there may be beads of serum exuded from them. The lesions are of full skin thickness and involve epidermis, dermis and sub-cutis, often with some oedema. They slowly harden and form a (dimple) indentation in the center.
- The regional lymph nodes are easily palpable and enlarged to 3-5 times their normal size. Some masses (lumps) may be detected in the subcutaneous tissues and are often distributed throughout the connective tissue and muscle in the body.
- The disease lesions are also developed on the muzzle in the nares and the oropharynx. The muzzle shows a typical ring-like lesion due to sloughing of the necrotic lesions from the healthy surrounding epithelium. Larynx, trachea, alimentary tract particularly the abomasum may also develop lesions (necrosis and ulceration) that lead to develop severe gastro-enteritis.
- Keratitis is a common complication. Mucopurulent discharges appear from the nares, persistent dribbling from the mouth, coughing and often stertorous and distressed respiration, if the larynx and trachea are involved. After 2-3 weeks, the skin lesions gradually become harder and necrotic.
- Several lesions associated with the formation of hard edematous plaques, cause severe discomfort and pain and inhibit movement. Later on, the "sit fast" of LSD are developed from harder lesions (core of necrotic tissue forms a plug).
- There is a distinct ring of living tissue around the lesions. Some of "sit fast" may peel off, leaving a full skin thickness hole in the skin, which heals by granulation. Bacteria may invade the hole. The limbs are swelled to several times their normal size due to inflammation, oedema and large areas of necrotic lesions.
- Hard skin over chronically edematous limbs may peel off, leaving large areas that can become infected or susceptible to myiasis. Lesions on the teats may falling away, predisposing animals to mastitis and loss of quarters. The common sequel of LSD is the pneumonia, associated with a large area of grey consolidation measuring 20-30 mm, which may be fatal.
- Abortion is a common sequel of the acute phase of the disease; aborted fetuses and live calves have been observed with skin lesions of LSD. Infertility is a problem following LSD infection; females remain in anestrous for several months and most infected cow suffering from cessation of ovarian activity mainly due to poor body condition.



- The infected bulls, which suffer from lesions on the genitalia, may also be infertile for months. Deterioration in the general condition occurs in the severely affected animals and under range conditions the mortality can be high. The recovered animals suffered from weakness and debility for up to 6 months. The majority of affected animals develop comparatively few nodules and recover uneventfully.



Treatment

No registered treatment for virus is present, although symptomatic treatment can be given to the animal which includes NSAIDS & antibiotics (topical +/- injectables) to protect from secondary infection.

Following proposed treatment protocols might help in lowering animals' suffering:

- Antihistaminic drug (Chlorpheniramine maleate) @ 0.4-0.5 mg / kg b. wt I.M./I.V. Anti-inflammatory (Inj. Meloxicam) @ 0.5 mg / kg b. wt I.M. in case of fever
- Antibiotics (Inj. Enrofloxacin @ 2.5 to 5 mg / kg b.wt I.M for 4 to 5 days and Amoxicillin @ 10 mg / kg b.wt I.M for 5 to 6 days.
- Supportive therapy- Vitamin A, D and E 10 ml I.M. Inj on alternate days for 4 times. Vitamin C 10 g per day per adult animal P.O Tab for 7 to 8 days. Liver tonics @ 50 ml per day orally.

Control and Prevention

To control the disease, effective control and preventive measures need to be implemented, which include: -

a) Restrict movement: Movement of infected animals with LSD should be strictly prohibited to prevent the spread of transboundary disease. Within countries, if animal with such lesions are observed, they should be quarantined for inspection to prevent the rapid spread of disease.



b) Restrict vector movements: Vectors movement due to prevailing winds may cause disease transmission. Vector control methods like use of vector traps, use of insecticides can also be used for preventing the disease.

c) Vaccination: A live attenuated vaccine is available for LSD. LSD virus closely resembles to sheep and goat poxviruses, hence vaccines against these two diseases can be used for LSD. For effective control and prevention of disease, long term vaccination with 100% coverage should be made mandatory as LSD virus being stable survives in environment for long time. Before introducing new animals to the affected farm, they should be immunized. Calves should be immunized at the age of 3 to 4 months raised from mothers, who are vaccinated or naturally infected. Adult animals should be vaccinated annually. Immunity starts to develop about 7 to 10 days after vaccination. Sheep Pox Vaccine is a live attenuated Sheep Pox Vaccine using an indigenous strain (SPPV Srin 38/00) was developed by the ICAR-IVRI and technology was transferred to the Hester Biosciences. Raksha Goat Pox is a live attenuated vaccine (Uttarkashi strain) was developed by ICAR-IVRI and technology was transferred to Indian Immunologicals Ltd (IIL). Live attenuated LSD Neethling strain.

ICAR-National Research Centre on Equines (ICAR-NRCE), Hisar (Haryana), in collaboration with ICAR-Indian Veterinary Research Institute (IVRI), Izatnagar, Uttar Pradesh has developed a homologous live-attenuated LSD vaccine Lumpi-ProVac^{Ind} (Ranchi strain).

Conclusions

- Until 19th century, the disease was endemic in Africa.
- But this disease now outstretched into the Middle East, Eastern Europe, and Russia and recently in south east Asia at a faster rate.
- Hence, it is needless to say, this is the high time to anticipate emergency preparedness to limit this trans-boundary disease from spreading enormously.
- Attention should be concentrated on vector control, movement restriction, harsh quarantine, improved vaccination programs, proper veterinary care, and overall farm sanitary management.

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