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Lumpy Skin Disease (LSD), an Emerging Disease in India: A Review

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ABSTRACT

As per the literature scanned, outbreaks of LSD in cattle and Buffalo have been reported in certain pockets of world (endemically). Outbreaks of LSD have been reported in late 2019 (as per OIE report) in India. Recently, the outbreaks have been documented in certain parts of Maharashtra, especially Beed district and Marathwada (*Karyarambh* newspaper, 2020). Therefore, it was found pertinent to publish a review article, highlighting the recent updates of this disease. This would not only help field Veterinarians to tackle this field problem, but also to the laboratory scientists to undertake further research on control and prophylaxis strategies.

Key words: Endemically, Laboratory, Lumpy skin disease.

OIE defines an emerging disease as “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.” Whereas, a known or endemic disease is considered to be re-emerging if it shifts its geographical setting, expands its host range, or significantly increases its prevalence.

Emerging and re-emerging animal diseases in recent years have been associated with outbreaks that have serious consequences for animal and human health.

Lumpy skin disease (LSD) is highly infectious viral disease characterized by marked lymphadenopathy and appearance of nodules on all parts of the body.

The disease is mainly observed in cattle and buffalo. The disease is of non-zoonotic in nature. The disease in India is emerging disease and cases of which found in many parts of India. OIE listed this disease as List A disease. Recently, the outbreaks (sporadic) of this disease have been reported from certain parts of India. Therefore, considering its alarming emergence, this review has been prepared for reference of field Veterinarians.

World-wide Epidemiology

Lumpy skin disease is classified as a TAD (Transboundary Animal Disease) due to its significant economic impact on production and local livelihoods and to the international trade restrictions it entails in affected countries (OIE, 2016).

Clinical syndrome of lumpy skin disease was first described in Zambia in 1929 and is restricted to Africa (Gumbe, 2018). Then the disease spreads throughout southern Africa and north to Sudan. The lumpy skin disease is Enzootic in Africa and Israel.

In Ethiopia lumpy skin disease was first observed in 1983 (Gumbe, 2018). Outbreaks of LSD were reported in Turkey and Iraq in late 2013 (OIE World Animal Health Information Database [WAHID]; Wainwright *et al*. 2013). Iran recorded the outbreak of LSD in early 2014 (European Food Safety Authority Journal, 2015).

The incidence of Lumpy Skin Disease (LSD) occurrence is high during wet seasons when biting-fly populations are abundant and it decreases or ceases during the dry season (Gumbe, 2018).

Confirmed cases of LSD occurred in Azerbaijan in May 2014 (OIE, 2014). Thereafter, Armenia (2015) and Kazakhstan (2015), the southern Russian Federation (Dagestan, Chechnya, Krasnodar Kray and Kalmykia) and Georgia (2016) reported its outbreaks. Outbreaks of LSD in India occurred in late 2019 (OIE report on LSD, 2019).

Epidemiology in India

Outbreak of LSD in India were recorded at Chhotanagpur plateau region which covers parts of Orissa, Jharkhand, West Bengal and Chhattisgarh (Pashudhan Praharee, 2019). Nine cases of LSD were reported by Dr Chaturvedi to OIE from Khairbani, Betnoti, Mayurbhanj of Orissa on 12/08/2019 (Chaturvedi, 2019). Further, on 17/08/2019 Dr Chaturvedi reported twenty cases from Patalipura, Betnoti, Mayurbhanj, Orissa. Dr Hiresh Rajan Bhowmik (2019) reported 66 cases to OIE from Chittagong city, Bangladesh.

The outbreak of ‘lumpy skin disease’ in cattle in certain parts of Orissa has prompted China to issue a warning notification against imports of cattle and cattle products from

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India (The Hindu Business line, New Delhi, 2019). The disease has been reported very recently in cattle in Palakkad, Thrissur and Malappuram (The Hindu, January, 2020). In Beed district of Maharashtra, cases of lumpy skin disease were found in animals (Karyarambh newspaper, 2020).

Predisposing factors

- Presence of growing numbers of naive animals, abundance of active blood-feeding vectors and uncontrolled animal movements are usually drivers for extensive LSD outbreaks.
- The primary case is usually associated with the introduction of new animal(s) into, or in close proximity to, a herd.
- 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 (Alaa *et al*. 2008).

Etiology

LSDV (lumpy skin disease virus) belongs to genus Capripox, family Poxviridae. The prototype strain of this virus is also known as Neethling poxvirus. Further, it has been observed that, the LSD virus has antigenic relationship with Sheep pox and Goat pox virus (Vegad and Katiyar, 2008).

Virus susceptibility

Virus is found susceptible to Phenol 2% as a disinfectant, Sodium hypochlorite (2–3%) and iodine compounds.

LSDV is very stable and survives well in extremely cold and dry environments within the pH range 6.3-6.8. It is found susceptible to alkaline or acidic pH. Also, the virus is susceptible to temperature of 55°C/2 hours, 65°C/30 minutes (OIE).

Host

The virus is highly host specific. It is reported that, LSD virus causes disease only in Cattle and Buffalo. The more susceptible breeds to LSDV infection are related to fine-skinned breeds such as Holstein Friesian (HF) and Jersey breeds (Gumbe, 2018). Further, extensive serological surveys of wild ruminant species in Africa have not identified a wildlife reservoir of LSDV (World Organization for Animal Health, 2017). LSDV is not zoonotic in nature.

Morbidity and mortality

Morbidity varies between 2 and 45 per cent, whereas, Mortality rate is usually less than 10 percent. Average 2% case fatality rate has been recorded but it may vary with outbreaks (Radostits *et al*. 2006).

The incubation period of virus is 2 to 4 weeks. Susceptibility of the host to this disease depends on immune status, age and breed (FAO Manual, 2017).

Transmission of the disease

Infection occurs mainly indirect through insects. Insects involved in LSD are probably haematophagous flies *viz. Tabanus spp., Stomoxys spp., Culicoides spp.*, mosquitoes and several species of ticks. The transmission is by mechanical way and hence flies such as *Tabanus* and *Stomoxys* are preferred vector owing to the fact that these flies are interrupted feeders (Abdulqa *et al*., 2016). However, the main vectors are still unknown (Hunter and Wallace, 2011). The principal vector is likely to vary between geographical regions and ecosystems (FAO Manual, 2017).

Direct transmission is also possible through drinking water, saliva, milk, sperm, or through contact with lesions of infected animals. The virus persists in the semen of infected bulls so that natural mating or artificial insemination (AI) may be a source of infection for females. Further, infected pregnant cows are known to deliver calves with skin lesions. The virus may be transmitted to suckling calves through infected milk, or from skin lesions in the teats.

Experimentally, the disease can be transmitted by injecting blood collected prior to the eruption of nodules and also by injecting emulsified tissue of nodules (Sastry, 2001).

Pathogenesis

On Subcutaneous or intradermal inoculation of cattle with LSDV, there is inflammation and development of a localized swelling at the site of inoculation after 4 to 7 days.

⬇

After development of localized swelling there is enlargement of the regional lymph nodes and generalized eruption of skin nodules within 7 to 19 days after inoculation

⬇

Viral replication occurs in pericytes, endothelial cells, cells in blood vessel and lymph vessel walls causes vasculitis and lymphangitis

⬇

In severe cases infarction may occurs which results in to edema and necrosis of different organs

⬇

Immunity after recovery from natural infection is life-long in most cattle

⬇

Calves of immune cows acquire maternal antibody and are resistant to clinical disease for about six months (Coetzer, 2014).

Molecular Pathogenesis

Molecular pathogenesis of LSD is not been well studied.

Clinical signs and symptoms

Fever which may exceed 42°C along with anorexia, depression, lethargy is observed. Snoring, oculo-nasal discharge and ptyalism are the additional signs (Fig 1). Nodular lesions on legs results in Lameness. Skin nodules may rupture and get invaded by secondary bacteria which causes mastitis. Nodular lesions on trachea, pharynx and bronchi causes respiratory distress. Corneal opacity and blindness may be observed in some cases. Pregnent animals may abort.

Gross lesions

Dermatological lesions appear as round circumscribed lesions varying from 0.5 cm to 5.0 cm in diameter and they

may be surrounded by a ring of hemorrhages (Fig 2). Lymph nodes are enlarged and oedematous (Lymphadenopathy). Occasionally, ulcers are found in respiratory tract. Nodular lesions are seen on trachea, pharynx and bronchi. The fate of nodules may vary, nodules may resolve quickly or persisted as a hard lump for 12 months or longer (Sastry, 2001). Secondary bacterial infection complicates the disease.

Morbid specimen collection

On the basis of lesions, material useful for laboratory diagnosis of this disease is Blood/serum of affected animal (for serological test); Skin or Dermal lesions- For HP and for Virus isolation; Enlarged lymph node- For HP and for Virus isolation; Affected lung and trachea - For HP and for Virus isolation; Semen of affected Bull and Milk of affected cattle and buffalo (with udder lesions).

Microscopic lesions

- Nodules are present, which involve all layers of the skin, the subcutaneous tissue and sometimes the adjacent musculature.
- Granulomatous lesions are found on various mucous membranes and in some organs (particularly the lungs) and in the upper respiratory and digestive tracts.
- Acanthosis (thickened epidermis), parakeratosis (thickened stratum corneum containing pyknotic nuclei) and hyperkeratosis (thickened stratum corneum) of epidermis occurs
- Eosinophilic intracytoplasmic inclusion bodies found in keratinocytes, fibroblasts and macrophages, *etc*. (Fig 3) -cited by (Tageldin *et al*. 2014).

Diagnosis

- **Field diagnosis** is often based on characteristic clinical signs and lesions of the disease.
- **Biopsy for inclusion bodies**: Eosinophilic intracytoplasmic inclusion bodies are found in keratinocytes, fibroblasts and macrophages, *etc*. (Brenner *et al*. 2006).

Laboratory diagnosis

- **ELISA:** It is the test that detects and measures of antibodies in blood.
- **FAT:** Detects the presence of a particular antigen.
- **Polymerase Chain Reaction (PCR):** It is the quickest and accurate method of detecting LSDV. Skin nodules, saliva and nasal secretions and blood are the suitable samples for the detection of LSDV.
- **Virus neutralization test:** Detects antibody that can block virus replication.

Pathological diagnosis

- **Histopathology:** Tissues (Lungs, trachea, dermal lesions) collected in 10% buffered formal saline and processed by routine histopathological technique. Necrosed epidermis, ballooning degeneration of squamous epithelial cells and eosinophilic intracytoplasmic inclusion bodies observed (Brenner *et al*. 2006).



Fig 1: Note copious thick yellow occulo-nasal discharge and thick copious oral discharge (ptyalism) from LSD affected cattle.



Fig 2: Note discriminate nodules of varied size and shape throughout the body of LSD affected cattle.

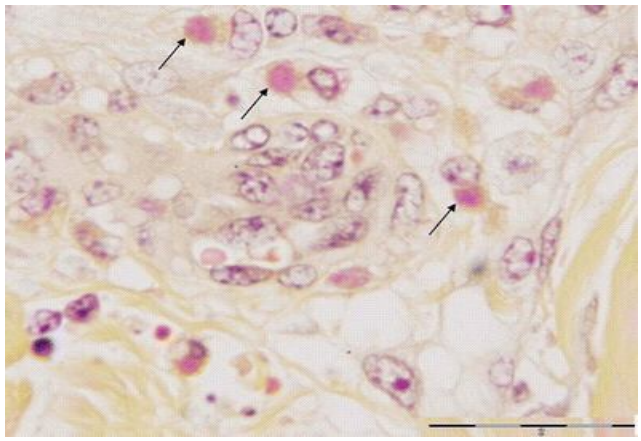


Fig 3: Mononuclear cells displaying intracytoplasmic inclusions (arrows).

- **Electron microscopy:** Primary diagnosis can be done by this method. Electron microscopy reveals typical capripox virus.

Differential diagnosis

The disease must be differentiated from the following disease conditions.

- **Pseudo-lumpy skin disease (BHV-2) (Allerton strain):** In which lesions are more superficial and course of the disease is shorter and less severe than that of Lumpy skin disease. Pseudo lumpy skin disease can be differentiated from lumpy skin disease by PCR.
- **Pseudo cowpox (Para poxvirus):** In pseudo cowpox lesions occurs mainly on teat and udder and disease can be differentiated from Lumpy skin disease by PCR.
- **Vaccinia virus and Cowpox virus (Ortho poxviruses):** Cutaneous lesions usually develops on teat and udder and muzzle. Cowpox is zoonotic disease unlike Lumpy skin disease and it can be differentiated from Lumpy skin disease by PCR.
- **Dermatophilosis:** Lesions of Dermatophilosis are superficial (often moist and appear as crusts of keratinized material).
- ***Hypoderma bovis* infection:** Swelling or eroded skin on the back of animal and larvae protrude from skin of back. paralysis lower body and legs may occur if spinal cord is involved. Lumpy skin disease can be ruled out by detecting LSDV by PCR.
- **Photosensitisation:** Dermal lesions resembles with that of Lumpy skin disease, but in photosensitisation lesions are more superficial and can be differentiated by PCR.
- **Insect bites or tick bites, Urticaria:** Multifocal oedema seen which leads to formation of diffuse wheals over dermis. Swelling over face, limbs and ventral side of abdomen are seen in urticaria unlike that of Lumpy skin disease and can be differentiated by PCR.
- **Cutaneous tuberculosis:** Subcutaneous single or multiple hard painless nodules formed on body of animal. Lumpy skin disease can be differentiated from cutaneous tuberculosis by detecting LSDV by PCR.
- **Onchocercosis:** Lesions are mainly present on ventral midline as intradermal nodules; Lumpy skin disease can be differentiated from onchocercosis by PCR.
- **Demidocosis:** Parasites lives in hair follicles and they are associated with skin glands causing formation of papules and nodules over skin of animal. Lumpy skin disease can be ruled out by detecting LSDV by PCR.

Prophylaxis

- Only live attenuated vaccines against LSD are currently commercially available.
- The attenuated Neethling strain (LSDV) vaccine is used to vaccinate cattle in Africa (Coetzer, 2004).
- It is possible to use the sheep pox/goat pox vaccine for cattle (Capstick and Coackley, 1961; Ganguly, 2016).
- Antibodies appear 10 days after vaccination and reach the highest level in 30 days post inoculation.

Treatment

- Symptomatic treatment of affected animals may be carried out.
- Administration of antibiotics for 5-7 days to check secondary infection.
- Administration of anti-inflammatory and anti-histamine preparations may also be considered.
- In case of pyrexia, paracetamol can be given.
- Application of antiseptic ointment with fly-repellent property over the eroded skin is recommended.
- Parenteral / oral multivitamins are advised.
- Feeding of liquid food, soft feed and fodder and succulent pasture is recommended for the infected animals.

Control

- Control of vectors by following integrated management practices such as physical, biological, cultural and chemical control. The practices such as change in grazing timings by avoiding bright sunshine hours; use of biocontrol agents against flies and ticks are also recommended. Use of herbal pesticides as repellants and last resort as spray of chemical insecticides are the options available.
- Vaccination in endemic areas.
- Quarantine of new arrived and infected animals.
- Disinfection of animal houses and premises with phenol 2%, sodium hypochlorite (2-3%), *etc*.
- Avoid movement/transport of infected animals and avoid trading during outbreaks.

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