Lumpy Skin Disease (LSD)

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LSD is one of the economically most important viral diseases of cattle and Asian water buffaloes after foot-and-mouth disease (FMD). LSD typically reduces milk yield, causes severe emaciation, permanent damage to hides.

Due to the impacts of global climate change and changes in trading patterns of animals and animal products, LSD has become an emerging disease threat. Initially the LSD was thought to be an allergic response to insect bites which was supported by the prevalence of the condition after the rains, when biting insect populations were at their highest. Subsequent studies showed that a transmissible agent was involved. Lumpy skin disease (LSD) is one of the most serious poxvirus diseases of cattle caused by lumpy skin disease virus (LSDV) within the genus *Capripoxvirus*. It causes acute to subacute systemic disease characterized by mild to severe symptoms including fever, nodules on the skin, in the mucous membranes and in the internal organs, skin edema, lymphadenitis and occasionally death.

Host

Lumpy skin disease virus can infect cattle and possibly other closely related wildlife such as the Arabian oryx, but does not cause clinical disease in sheep or goats. LSDV has a limited host range and does not infect non-ruminant hosts. Both sexes and all ages of cattle breeds are susceptible to LSDV. However, younger animals may be more susceptible to the severe form of the disease. Breeds of *Bos taurus* with high milk production are more susceptible than African/Asian indigenous cattle. Even in close contact with infected cattle, sheep and goats never developed LSD

Vectors and other environmental risk factors

LSDV can remain viable for long periods in the environment at ambient temperatures, especially in dried scabs. It is reported that the virus persists in necrotic skin nodules for up to 33 days or longer, in desiccated crusts for up to 35 days and for at least 18 days in air-dried hides.

The main sources of infection are considered to be skin lesions as the virus persists in the lesions or scabs for long periods. The virus is also excreted via the blood, nasal and lachrymal secretions, saliva, semen and milk (transmissible to suckling calves) which is responsible for direct transmission.

Pathogenesis

The incubation period of the LSD is 2-5 weeks. The major clinical signs of the disease are characteristics circular skin nodules of over the body, fever, palpable enlarged subscapular and prefemoral lymph nodes, lacrimation, keratitis, nasal discharge, drop in milk yield, off-fed, emaciation, depression and reluctance movement. Morbidity and mortality of the disease is 5-45 and 1-5%, respectively.

The illness can affect cattle of any breed and age. However, depending on the virulence of the strains and the sensitivity of the cow breed, the severity of the clinical indications of LSD ranges from asymptomatic to lethal.

Viremia (fever) is seen usually after 6 days of infection as the virus continues to multiply. Peaks of viremia are reflected as intermittent rise and fall in body temperature. The virus exhibits a tropism towards keratinocytes and brings about various pathological changes. Initially, there is hyperplasia and ballooning degeneration, later epidermal micro-vesicles develop and the released chemokines attract inflammatory cells to the site.

At around the 7th day, nodular lesions start to appear on the skin of the affected animal. Such lesions are also evidenced in various tissue containing epithelial cells. Later on, the micro-vesicles join each other and form large vesicles, and the lesions begin to ulcerate and exude serous discharge. These nodular sites become necrotic enclosing a typical grey to a pink-colored conical area that gets separated from the

adjoining tissue and these lesions are known as 'sit-fasts'. There are congestion, hemorrhages, and edema in the neighboring zone of tissue. The necrotic zones of tissue are invaded by bacteria in the mean course of time which further complicates the pathogenesis. The lymph nodes become enlarged. The host factors such as younger animals may be more susceptible to the severe form of the disease. Breeds of *Bos taurus* with high milk production are more susceptible than African/Asian indigenous cattle.

Diagnosis

Clinical Diagnosis: Lesions include skin nodules observed on the forehead, eyelids, ears, muzzles, nostrils, udder, limbs. A sample taken from the skin can used in biopsy for further confirmation of the disease

Post-mortem findings: The epidermal and mucosal lesions described above will be apparent at post-mortem. Ulcerations may be found in the lining of the trachea and gastro-intestinal tract. Lung lesions consisting of pale grey nodules may also be seen

Gross pathological findings: LSD has well-described gross lesions. Skin nodules are usually uniform in size, firm round and raised, but some may fuse into large irregular and circumscribed plaques. The cut surface of the nodules is reddish-gray, in addition, to the accumulation of the reddish grey serous fluid and edema in the subcutis layer. The resolved lesions appear as indurated which is called "sitfasts" or seclude or may form deep ulcers. The typical circular necrotic alimentary lesions may also be seen on the muzzle, nasal cavity, larynx, trachea, bronchi, inside of lips, gingiva, dental pad, forestomach, abomasum, uterus, vagina, teats, udder and testes (Ali et al 1990). Regional lymph nodes are grossly enlarged and can be 3-5 times their usual size, oedematous and having pyaemic foci, in addition to local cellulitis. Muscle tissue and the fascia over limb muscle may be show nodular lesion that are grey-white surrounded by red inflammatory tissue. The same nodules are distributed throughout the carcass. It is about 10-30 mm diameter in the kidney. Interstitial or bronchopneumonia associated with 10-20 mm diameter lesions are also scattered in the lungs. These lesions result from infiltration of the large epithelioid 'celles claveleuses', described by Borrel for sheep pox. The lesions are separated from the necrotic epithelium far from the healthy tissue. The necrotic tissue sloughs away to leave an ulcer that slowly heals by granulation. Severely infected animals may show secondary bacterial pneumonia, tracheal stenosis, acute and chronic orchitis, mastitis with secondary bacterial infection, and similar lesions in the female reproductive tract.

Confirmative diagnosis of LSD

Diagnosis of LSD mainly depends on the typical clinical signs, differential diagnosis, and application of various diagnostic laboratory techniques for detection and confirmation of the disease, such as electron microscopy examination, virus isolation (VI), serological tests (serum neutralization test, agar gel immune diffusion, indirect enzyme-linked immunosorbent assay, and indirect fluorescent antibody technique [IFAT]), and real-time or conventional polymerase chain reaction (PCR). Viral isolation and identification as well as PCR methods are the most sensitive methods for detecting LSDV in skin samples. However, viral isolation is a gold standard for LSDV diagnosis; it is time-consuming as the protocol takes several weeks to isolate LSDV in tissue cultures or chorioallantoic membrane (CAM) of embryonated chicken egg (ECE). Immunohistochemistry (IHC) is an essential tool for diagnosing many animal diseases, including LSDV; several authors have reported it as a direct method for detecting the pathogenic antigen distribution using specific anti-LSDV antibodies in skin nodules of infected cattle

Differential diagnosis

There are many diseases causing similar signs of LSD. It is important to obtain a definite diagnosis to ensure the best preventative and control measures for susceptible herds. LSD can be confused with the following diseases:

- Pseudo-lumpy-skin disease
- Bovine virus diarrhoea/mucosal disease
- Demodicosis (Demodex)
- Bovine malignant catarrhal fever (Snotsiekte)
- Rinderpest
- Besnoitiosis
- Oncocercariasis
- Insect bite allergies.

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