Lumpy Skin Disease

*Neethling, Knopvelsiekte*

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**Importance**

Lumpy skin disease is a poxviral disease with significant morbidity in cattle. Although the mortality rate is generally low, losses occur from decreased milk production, abortion, infertility, loss of condition and damaged hides. Lumpy skin disease is endemic in parts of Africa, where outbreaks may be widespread. This disease has the potential to become established in other parts of the world.

**Etiology**

Lumpy skin disease is caused by a virus in the genus *Capripoxvirus* of the family Poxviridae. Lumpy skin disease virus (LSDV) is closely related antigenically to sheep and goat poxviruses. Although these three viruses are distinct, they cannot be differentiated with routine serological tests.

**Species Affected**

Lumpy skin disease is primarily a disease of cattle. *Bos taurus* breeds, particularly Jersey, Guernsey and Ayrshire, are more susceptible to clinical disease than zebu cattle (*Bos indicus*). A few cases have been reported in Asian water buffalo (*Bubalus bubalis*). Clinical cases or antibodies have been reported in other species such as oryx, but could have been caused by closely related poxviruses. Wild animals are not thought to play an important role in the spread or maintenance of LSDV.

**Geographic Distribution**

Lumpy skin disease is generally confined to Africa. Until the 1980s, this disease was only found south of the Sahara desert and in Madagascar, but in 1988, it spread into Egypt. It may also occur in other Middle Eastern countries. In 1989, an outbreak in Israel was eradicated by slaughter and vaccination.

**Transmission**

LSDV is thought to be transmitted primarily by biting insects. This virus has been found in mosquitoes in the genera *Aedes* and *Culex* during some outbreaks. Experimentally infected *Aedes aegypti* are infectious for 6 days and can transmit LSDV mechanically during this time. Flies (e.g. *Stomoxys calcitrans*) and other insects might also be involved in transmission, but this remains unproven.

Direct contact could be a minor source of infection. LSDV occurs in cutaneous lesions, saliva, respiratory secretions, milk and semen. Shedding in semen may be prolonged; viral DNA has been found in the semen of some bulls for at least 5 months after infection. Animals can be infected experimentally by inoculation with material from cutaneous nodules or blood, or by ingestion of feed and water contaminated with saliva. LSDV is very resistant to inactivation, surviving in desiccated crusts for up to 35 days, and can remain viable for long periods in the environment.

**Incubation Period**

The incubation period in the field is thought to be two to five weeks. In experimentally infected animals, fever can develop in 6 to 9 days and lesions first appear at the inoculation site in 4 to 20 days.

**Clinical Signs**

The clinical signs range from inapparent to severe. Host susceptibility, dose and route of virus inoculation affect the severity of disease. *Bos taurus* is more susceptible than *Bos indicus*, and young calves often have more severe disease than adults.

Fever is the initial sign. It is usually followed within two days by the development of nodules on the skin and mucous membranes. These nodules vary from 1 cm to 7 cm and penetrate the full thickness of the skin. They are particularly common on the head, neck, udder, genitalia, perineum and legs. Although the nodules may exude serum initially, they develop a characteristic inverted conical zone of necrosis, which penetrates the epidermis and dermis, subcutaneous tissue, and sometimes the underlying muscle. These cores of necrotic material become separated from the adjacent skin and are called “sit-fasts.” Secondary bacterial infections are
common within the necrotic cores. Some nodules, particularly those on the mucous membranes and udder, ulcerate rapidly. The superficial lymph nodes become enlarged and edematous. Nodules can also occur in the gastrointestinal tract, trachea and lungs; the latter may result in primary or secondary pneumonia.

In addition, feed intake decreases in affected cattle, milk yield can drop markedly, and animals may become emaciated. Rhinitis, conjunctivitis and keratitis can also be seen; ocular and nasal discharges are initially serous but become mucopurulent. Inflammation and necrosis of the tendons, or severe edema of the brisket and legs, can result in lameness. Secondary bacterial infections can cause permanent damage to the tendons, joints, teats and mammary gland. Abortions and temporary or permanent sterility may occur in both bulls and cows.

A few animals die, but the majority slowly recover. Recovery can take several months, and some skin lesions may take a year or two to resolve. Deep holes or scars are often left in the skin.

**Post Mortem Lesions**

The post mortem lesions can be extensive. Characteristic grayish-pink deep nodules with necrotic centers are found in the skin; these nodules often extend into the subcutis and underlying skeletal muscle, and the adjacent tissue exhibits congestion, hemorrhages and edema. The regional lymph nodes are typically enlarged.

Flat or ulcerative lesions may also be found in the mucous membranes of the oral and nasal cavities, pharynx, epiglottis and trachea. Nodules or other lesions can occur in the gastrointestinal tract (particularly the abomasum), udder, urinary bladder, lungs, kidneys, uterus and testes. In the lungs, the lesions are difficult to see and appear as focal areas of atelectasis and edema. The mediastinal lymph nodes are enlarged in severe cases, and pleuritis may be seen. Some animals may have synovitis and tendosynovitis with fibrin in the synovial fluid.

Aborted fetuses do not always have the characteristic external lesions, but some may be covered in nodules.

**Morbidity and Mortality**

Lumpy skin disease can occur as sporadic cases or in epizootics. The incidence of disease is highest in wet, warm weather, and decreases during the dry season. New foci of disease can appear at distant sites; in these cases, the virus is thought to be carried by insects.

The morbidity rate varies widely, depending on the presence of insect vectors and host susceptibility, and ranges from 3% to 85%. More severe disease is seen in *Bos taurus*, particularly Channel Island breeds, than zebu cattle. Calves and lactating cows tend to be most susceptible to disease. In addition, the disease signs can vary widely among the cattle in a group, with some animals having apparent infections and others developing severe disease.

The mortality rate is low in most cases (1–3%), but has been as high as 20% in some outbreaks. Unusually high mortality rates of 75–85% have been reported but remain unexplained.

**Diagnosis**

**Clinical**

Lumpy skin disease should be suspected when the characteristic skin nodules, fever and enlarged superficial lymph nodes are seen. The mortality rate is usually low.

**Differential diagnosis**

Differentials include pseudo-lumpy skin disease/bovine herpesvirus, pseudocowpox, but not from orthopoxviruses (cowpox virus). LSDV can be distinguished from the herpesvirus that causes pseudo-lumpy skin disease by the cytopathic effect and the intracytoplasmic location of inclusion bodies. LSDV antigens can be identified with direct immuno-fluorescence, virus neutralization or enzyme-linked immunosorbent assay (ELISA). Nucleic acid recognition methods have also been described. LSDV can be detected in cell cultures or directly in tissues by polymerase chain reaction (PCR) assays.

Typical capripox virions can be seen in biopsy samples or desiccated crusts using transmission electron microscopy. This finding, in combination with a history of generalized nodular skin lesions and lymph node enlargement, can be diagnostic in cattle in endemic areas. Electron microscopy can distinguish capripoxviruses from the parapoxviruses that cause bovine papular stomatitis and pseudocowpox, but not from orthopoxviruses (cowpox and vaccinia virus).

**Laboratory tests**

Confirmation of lumpy skin disease in a new area requires virus isolation and identification. LSDV will grow in bovine, caprine or ovine cell cultures; the best growth is seen in lamb testis cells. LSDV can be distinguished from the herpesvirus that causes pseudo-lumpy skin disease by the cytopathic effect and the intracytoplasmic location of inclusion bodies. LSDV antigens can be identified with direct immuno-fluorescence, virus neutralization or enzyme-linked immunosorbent assay (ELISA). Nucleic acid recognition methods have also been described. LSDV can be detected in cell cultures or directly in tissues by polymerase chain reaction (PCR) assays.

**Samples to collect**

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should
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Live vaccines help control losses from lumpy skin disease in endemic areas. Insecticides and repellents may also be helpful. Antibiotics are used to control secondary infections.

Public Health

LSDV does not infect humans.

Internet Resources

http://www.spc.int/rah/

United States Animal Health Association.
Foreign Animal Diseases

The Merck Veterinary Manual
http://www.merckvetmanual.com/mvm/index.jsp

World Organization for Animal Health (OIE)
http://www.oie.int

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/

OIE Terrestrial Animal Health Code
http://www.oie.int/international-standard-setting/terrestrial-code/access-online/

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