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, economic losses result from loss of condition, decreased milk production, abortions, infertility and damaged hides. The causative virus seems to be spread mainly by insects, and outbreaks can be widespread and difficult to control. Lumpy skin disease was confined to Africa at one time, but it has now become endemic in parts of the Middle East, and the virus is continuing to spread. Recent outbreaks were reported as far north as Russia, Armenia, Azerbaijan, Turkey and southern and eastern Europe. The virus was eradicated from some countries, but not others. It has multiple arthropod vectors, including biting flies, midges, mosquitoes and ticks, and could become established in other parts of the world.

Etiology

Lumpy skin disease virus (LSDV) is a member of the genus *Capripoxvirus* and the family Poxviridae. It is closely related antigenically to sheeppox virus and goatpox virus. Although these three viruses are considered to be distinct viral species, they cannot be distinguished by routine serological tests.

Species Affected

Lumpy skin disease is primarily a disease of cattle, but clinical cases have also been reported in Asian water buffalo (*Bubalus bubalis*). Sheep and goats seem to be unaffected even when they are in close contact with cattle during outbreaks. The situation in wild ungulates is currently unclear. Viral nucleic acids were detected in unspecified skin lesions from springbok (*Antidorcas marsupialis*), while a putative clinical case in an Arabian oryx (*Oryx leucoryx*) was diagnosed by methods that cannot distinguish LSDV from other capripoxviruses. Impala (*Aepyceros melampus*), giraffe (*Giraffa camelopardalis*) and Thomson's gazelle (*Eudorcas thomsonii*) developed clinical signs after experimental inoculation, but there have been no reports of illnesses in these species during outbreaks in cattle. Anti-LSDV antibodies have been reported in a number of wild ungulates in Africa, including wildebeest (*Connochaetes* spp.), springbok, eland (*Taurotragus oryx*), impala, African buffalo (*Syncerus caffer*), giraffe and other species; however, the serological tests would also have detected antibodies to other capripoxviruses.

Zoonotic potential

There is no evidence that LSDV can infect humans.

Geographic Distribution

Lumpy skin disease is currently endemic in most of Africa, parts of the Middle East and Turkey. Outbreaks also occurred recently in parts of Asia and Europe. Lumpy skin disease was eradicated from some of the affected countries, but the status of others is not yet resolved.

Transmission

LSDV is thought to be transmitted primarily by arthropod vectors. Mosquitoes, biting flies (e.g., *Stomoxys calcitrans, Biomyia fasciata*), *Culicoides* midges and hard ticks (e.g., *Amblyomma hebraeum*, *Rhipicephalus* spp.) are currently thought to be mechanical vectors. Ticks probably play little or no role when LSDV is spreading rapidly during epizootics; however, they might be involved in transmission and maintenance in endemic regions. Prolonged virus survival has been reported in some vectors. Experimentally infected *Aedes aegypti* were infectious for 6 days, and transovarial and transstadial transmission were demonstrated in some species of ticks. Some flying arthropods, such as *Culicoides*, might introduce LSDV to new areas when they are carried by the wind.

Direct contact seems to be a minor source of infection. Early studies suggested that transmission between animals was inefficient in insect-free environments, although some cattle became infected when they were allowed to share a water trough with severely affected animals. Contaminated feed is also likely to be a source of

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exposure, and cattle can be infected experimentally by inoculating them with material from cutaneous nodules or blood. In cattle, LSDV has been detected in cutaneous lesions, saliva, respiratory secretions, milk and semen, but some studies found only small amounts of virus. Infected animals can transmit LSDV whether or not they develop skin lesions. Shedding in semen may be prolonged; viral DNA has been found in the semen of some bulls for at least 5 months after infection, and live virus for up to 42 days. Transmission via artificial insemination has been demonstrated experimentally. Cows can also infect their fetuses *in utero*. There is limited information about LSDV transmission and shedding in water buffalo; however, some animals shed viral nucleic acids in milk.

LSDV may remain viable for long periods in the environment. It has been reported to survive for up to 35 days in desiccated crusts, for at least 18 days in air-dried hides, and for months in sheds protected from sunlight. At 4°C (39°F), LSDV remained viable for 6 months in tissue culture fluid.

Disinfection

LSDV is susceptible to a number of disinfectants including sodium hypochlorite, iodine, quaternary ammonium disinfectants, ether, chloroform, formalin, phenol, and detergents that contain lipid solvents. It is reported to be susceptible to heating at 55°C (131°F) for 2 hours or 65°C (149°F) for 30 minutes.

Incubation Period

The incubation period in the field is thought to be 1-4 weeks, but experimentally infected cattle may develop clinical signs as soon as 4 days.

Clinical Signs

LSDV infections in cattle range from inapparent to severe. In addition to fever and enlarged superficial lymph nodes, animals typically develop lesions on the skin and mucous membranes. On the skin, lesions initially appear as firm, round, slightly raised, circumscribed areas of erect hair, which are often separated from the surrounding normal skin by a narrow hemorrhagic band, and develop into full-thickness skin nodules that range in diameter from < 1 cm to 8 cm. Some animals have only a few nodules, but others develop large numbers. Nodules are particularly common on sparsely haired areas such as the head, neck, udder, genitalia, perineum and legs, but may cover the entire body. Although they may exude serum in the early stages, many nodules subsequently develop the 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." They sometimes slough, leaving a hole in the skin, or become secondarily infected with bacteria. Mucosal lesions are initially round, but quickly ulcerate. Ulcers in the oral and nasal cavities can result in nasal discharge and excessive salivation. Lesions can also occur in the oropharynx, gastrointestinal tract, upper respiratory tract and lungs, sometimes resulting in primary or secondary pneumonia. Some animals develop conjunctivitis and/ or keratitis.

Feed intake decreases in affected cattle, and severely affected animals may become emaciated. Milk yield can drop markedly. Some animals also develop edematous ventral swellings, which can involve the brisket and legs, as well as the sheath in bulls. In severe cases, the skin on edematous legs or the udder may become necrotic and slough. Secondary bacterial infections can cause permanent damage to the tendons, joints, teats and mammary gland. Temporary or permanent sterility is possible in bulls, and pregnant cows may abort. Some aborted fetuses and premature calves have been covered in nodules; no nodules were found in other cases. Most animals with lumpy skin disease slowly recover, although severely affected animals can die. 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.

Similar clinical signs, including nodules, decreased milk production and weight loss, have been reported in water buffalo; however, this species is reported to be less severely affected. Some water buffalo seroconverted without signs of illness.

Post Mortem Lesions

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The postmortem 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 be found on 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 and lungs, and sometimes in other tissues such as the urinary bladder, kidneys, uterus and testes. Lesions in the lungs are difficult to see and often appear as focal areas of atelectasis and edema. The mediastinal lymph nodes may be enlarged in severe cases, and pleuritis may be evident. Some animals have additional complications, such as synovitis and tendosynovitis.

Some aborted fetuses and premature calves may have large numbers of skin nodules. They can also have lesions on internal organs.

Diagnostic Tests

LSDV, its nucleic acids and antigens can be detected in biopsy or necropsy samples of skin nodules, lymph nodes and lesions on internal organs, as well as in scabs, nodular fluid and skin scrapings. This virus can sometimes be isolated from skin nodules for up to 3-4 weeks, although samples are preferably collected during the first week of illness. It may also be isolated from blood samples during

the early, viremic stage of disease, but this is unlikely to be successful after generalized lesions have been present for more than a few days.

PCR assays can detect viral nucleic acids directly in tissue samples. Some tests are reported to distinguish LSDV from sheeppox and goatpox viruses. Dot blot hybridization and loop-mediated isothermal amplification assays (LAMP) have been published. Capripoxvirus antigens can be detected in tissues with immunostaining methods. LSDV can be isolated in various bovine, caprine or ovine cell cultures; it is reported to grow best in primary or secondary lamb testis or bovine dermis cells, but other cells can be used. Virus growth can be slow, and may require several passages. Isolated viruses can be identified by PCR tests or other genetic assays, and they can be recognized as capripoxviruses with direct immunofluorescence, virus neutralization and other tests.

Transmission electron microscopy, which can detect the typical capripoxvirus morphology in biopsy samples or desiccated crusts, is sometimes employed in diagnosis. Used together with a history of consistent clinical signs, it can provide a presumptive diagnosis 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). However, orthopoxvirus infections do not usually resemble lumpy skin disease in cattle. Histopathology can also be helpful.

Various serological tests, including virus neutralization, an indirect fluorescent antibody test, ELISAs, and immunoblotting (Western blotting), can detect antibodies to LSDV, but some of these tests have not yet been validated. Agar gel immunodiffusion was employed in the past, but it is not currently recommended by the World Organization for Animal Health (OIE), due to issues with cross-reactivity (bovine papular stomatitis and pseudocowpox virus) and sensitivity. Most serological tests, including standard virus neutralization, cannot distinguish antibodies to different species of capripoxviruses. False negatives may be seen in mildly affected animals, and the OIE considers serology to be most appropriate as a herd test.

Treatment

There is no specific treatment for lumpy skin disease, but supportive care, including antibiotics as necessary for secondary bacterial infections, can be helpful. Wound dressings have been used to reduce fly strike and secondary infections.

Control

Disease reporting

Veterinarians who encounter or suspect lumpy skin disease should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

Lumpy skin disease could be introduced into a new area by infected animals, contaminated hides and other animal products, or infected insects. Outbreaks recognized early have sometimes been eradicated with quarantines, depopulation, and cleaning and disinfection of infected premises, but vaccination was an important component of eradication plans in some large outbreaks. Quarantines and movement controls are unlikely to prevent transmission completely when LSDV is being spread by vectors; however, they can stop infected animals from introducing the virus to distant foci. Insect control is generally employed during lumpy skin disease outbreaks, although its effectiveness is still unclear. Some authors note that insecticide treatment of carcasses helps prevent flies from acquiring the virus. This is especially important if the carcasses are transported through uninfected areas.

Live attenuated vaccines can control losses in areas where LSDV is endemic, and also appear to decrease or eliminate virus shedding in semen. Killed vaccines may also be available in some areas. The currently available vaccines differ in quality and efficacy. Vaccine reactions as well as vaccine failures have been reported.

Morbidity and Mortality

Lumpy skin disease can occur as sporadic cases or in epizootics. It tends to be more common in warm, humid areas, but is not limited to these regions. In endemic areas, the number of cases typically rises during wet, warm weather, when more insect vectors are present, and decreases during the dry season. New foci of disease can appear at distant sites.

Lumpy skin disease may affect only a few cattle in a herd, although other animals can be subclinically infected. Some individual cattle have only a few lesions; others develop severe clinical signs. *Bos taurus* breeds, particularly Channel Island breeds, are reported to be more susceptible to clinical signs than zebu cattle (*Bos indicus*). Young calves and lactating cows also tend to be more severely affected. Reported morbidity rates during outbreaks range from 1-2% to 80-90%, while mortality rates are often between 1% and 6%, but are occasionally higher or lower. The overall morbidity rates during recent outbreaks in the Middle East and Europe were generally < 30%, and sometimes much lower; however, control measures may have limited the number of cases. Jordan reported morbidity and mortality rates of 43% and 10%, respectively, in unvaccinated cattle, compared to 5% morbidity and 1% mortality in vaccinated cattle. Case fatality rates ranged from 2% (in an endemic region of Egypt) to 23%. The limited information on water buffalo suggests that morbidity is much lower in this species than in cattle.

Internet Resources

European Food Safety Authority (EFSA). Lumpy Skin Disease
https://www.efsa.europa.eu/en/topics/topic/lumpy-skin-disease

EFSA. Scientific Opinion on Lumpy Skin Disease
http://onlinelibrary.wiley.com/doi/10.2903/j.efsa.2015.3986/pdf

United States Animal Health Association.
Foreign Animal Diseases
http://www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf

The Merck Veterinary Manual
http://www.merckvetmanual.com/

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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