Lumpy Skin Disease

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Importance

Lumpy skin disease is a poxviral disease of cattle with significant morbidity. While the mortality rate is generally low, economic losses result from reduced condition, decreased milk production, abortions, infertility and damaged hides, and some severe illnesses are fatal. Occasionally, this disease may affect other species, such as water buffalo, yaks, camels and wild ungulates, during outbreaks in cattle. The causative virus seems to be spread mechanically by a variety of arthropod vectors, and outbreaks can be widespread and difficult to control. Lumpy skin disease was confined to Africa at one time, but later became established in the Middle East. It was recently introduced into Europe and Asia. While the virus was eradicated from some areas, including parts of Europe, it has become endemic in some countries and continues to spread.

Etiology

Lumpy skin disease virus (LSDV; formal species name *Capripoxvirus lumpyskinpox*) is a member of the genus *Capripoxvirus* and family Poxviridae. It is closely related antigenically to sheeppox virus and goatpox virus, and while the three viruses are considered to be distinct viral species, they cannot be distinguished with some diagnostic tests.

LSD viruses are currently divided into two clusters. Cluster 1 (subclusters 1.1 and 1.2) contains classical field and vaccine strains, while cluster 2 (2.1-2.6) consists of some recently emerged recombinant viruses that contain gene segments from both vaccine and field strains in cluster 1. They are generally referred to as vaccine recombinant viruses, and have diverse patterns of recombination. At least some of them appear to have originated from a poor quality live attenuated vaccine used in Kazakhstan, which was subsequently found to contain genetic sequences from multiple capripoxviruses. To date, all of the vaccine recombinant viruses identified have been at least as virulent as field strains.

Species Affected

Lumpy skin disease is mainly a disease of cattle, which seem to be its only maintenance host among domestic animals. Occasional clinical cases have also been reported in other ungulates including water buffalo (*Bubalus bubalis*), yaks (*Bos grunniens*), mithun (*Bos frontalis*), bantengs (*Bos javanicus*), gaur (*Bos gaurus*), dromedary camels (*Camelus dromedarius*), giraffes (*Giraffa camelopardalis*), Indian gazelles (*Gazella bennettii)*, springbok (*Antidorcas marsupialis*), impalas (*Aepyceros melampus*) and mainland serows (*Capricornis sumatraensis*). A putative case was reported in an Arabian oryx (*Oryx leucoryx*), but diagnosed by methods that cannot distinguish LSDV from other capripoxviruses, and viral nucleic acids were found in a nasal swab from an asymptomatic eland (*Taurotragus oryx*) in Namibia. Experimental infections of Thomson's gazelles (*Eudorcas thomsonii*), a neonatal giraffe and a young impala calf resulted in clinical signs, but two young African buffalo (*Syncerus caffer*) calves and two adult black wildebeest (*Connochaetes gnou*) remained asymptomatic. The absence of neutralizing antibody titers in the latter two species suggested that they probably did not become infected; however, protection from previous exposures and/or maternal immunity could not be entirely ruled out, as they were all captured from the wild, and not all animals develop significant antibody titers to this virus after exposure. Sheep and goats seem to be unaffected even when they are in close contact with cattle during outbreaks.

Whether any wildlife species might contribute to maintaining LSDV in the absence of cattle is not known. Anti-LSDV antibodies have been reported in a number of wild ungulates in Africa including wildebeest (*Connochaetes* spp.), springbok, eland, impalas, African buffalo and giraffes, but cross-reactivity to other capripoxviruses in serological tests makes this finding difficult to interpret.

Naturally-occurring infections have not been documented in any animals other than ungulates, but experimentally infected rabbits can develop skin nodules. While rodents are said to be refractory to this virus, one study reported lung lesions in experimentally infected, asymptomatic guinea pigs and a single “skin pustule” of unconfirmed etiology in a Syrian hamster.

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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 Asia, and Turkey. Russia has experienced repeated outbreaks in its eastern regions. Outbreaks also occurred in eastern and southern Europe around 2015-2017, but seem to have been successfully eradicated. Most outbreaks have been caused by cluster 1 viruses, but at least one vaccine recombinant virus was found in Africa, and cluster 2 vaccine recombinant viruses have been reported from outbreaks in eastern Russia and parts of Asia including China and Vietnam.

Transmission

LSDV is mainly thought to be transmitted by arthropods acting as mechanical vectors. Potential vectors include biting flies (e.g., the stable fly *Stomoxys calcitrans* and other *Stomoxys* species, various tabanids), mosquitoes, *Culicoides* midges, hard ticks and possibly even some non-biting flies such as *Biomyia fasciata and Musca domestica*. The relative importance of different arthropods is still uncertain, but the life cycles of ticks suggest that they probably play little or no role in spreading this virus rapidly during outbreaks. However, ticks might be involved in virus transmission and/or maintenance in endemic regions, and transovarial and transstadial transmission have been demonstrated in some tick species. Some authors have suggested that flying insects such as *Culicoides* might introduce LSDV to new areas when they are carried by the wind, but very large numbers of insects would probably have to be transferred, as viral contamination on mechanical vectors is low.

Direct contact between animals seems to be a minor source of infection for cluster 1 viruses. Transmission of these viruses is reported to be inefficient in insect-free environments, though some cattle became infected when they were allowed to share a water trough with severely affected animals. However, a cluster 2 vaccine recombinant virus readily infected cattle that shared an arthropod-free room but had no direct contact and used separate feed or water troughs. While viruses in the skin, especially cutaneous lesions, are probably responsible for most transmission to arthropod vectors, LSDV can also be shed in saliva, respiratory secretions, milk and semen, at least in small amounts. Animals can transmit this virus to arthropods whether or not they develop skin lesions, though this occurs much more readily from symptomatic than subclinical cattle for cluster 1 viruses. Shedding in semen may be prolonged: viral DNA has been found in the semen of some bulls for at least 5 months, and live virus for up to 42 days. Viruses in semen can by transmitted to cows by artificial insemination, and can also infect embryos produced by *in vitro* fertilization. Infected cows can pass the virus to their fetuses *in utero*.

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, where it is protected from sunlight. At 4°C (39°F), LSDV remained viable for 6 months in tissue culture fluid. Prolonged virus survival, ranging from two days to a week or more, has been reported in some arthropod vectors, though to date, the only demonstration of transmission to animals during this time was from experimentally infected *Aedes aegypti* mosquitoes, which were infectious for 6 days. Overwintering of cluster 2 viruses has been seen in cold climates (e.g., Russia).

Disinfection

LSDV is susceptible to a number of disinfectants including sodium hypochlorite, iodides, quaternary ammonium disinfectants, ether, chloroform, formalin, phenols, and detergents that contain lipid solvents. LSDV and other capriviruses can be inactivated by heating at 56°C (133°F) for 30 minutes or 60°C (140°F) for 10 minutes, though longer periods (e.g., one hour at 56°C) were recommended by some authors as a precaution in case of unusually heat resistant strains.

Incubation Period

The incubation period in the field is thought to be 1-5 weeks, but some experimentally infected cattle may develop clinical signs as soon as 4 days.

Clinical Signs

LSDV infections in cattle range from inapparent to severe. The signs in subclinically infected animals are usually limited to elevated body temperature and regional lymph node enlargement, which may be subtle and difficult to detect. In animals with overt disease, the illness often begins with a period of nonspecific clinical signs, which may include fever, increased nasal secretions, lachrymation, reduced appetite and lethargy, before the development of the characteristic lesions on the skin and mucous membranes. 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. They are sometimes difficult to observe in animals with a long hair coat, but detectable by palpation.

Although they may exude serum in the early stages, many nodules subsequently 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.” They may become secondarily infected with bacteria or attract myiasis flies, and sometimes slough, leaving a hole in the skin. Mucosal lesions

are also round initially, with a ring-like margin of separation from healthy tissues, 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 causing respiratory signs (e.g., coughing) and leading to primary or secondary pneumonia. Conjunctivitis, keratitis and/or ulcerative ocular lesions can be seen in some animals, and may result in visual impairment.

Feed intake decreases in affected cattle, and severely affected animals may become emaciated and/or dehydrated. Milk yield can drop markedly. Diarrhea has also been observed in some outbreaks. In addition, some animals develop edematous ventral swellings, which can involve the brisket, legs and mammary gland, and the sheath in bulls. In severe cases, the skin on edematous legs or the udder may become necrotic and slough. Permanent damage to the tendons, joints, teats and mammary gland is possible. Temporary or permanent sterility may be seen in bulls, and pregnant cows may abort or give birth prematurely. Aborted fetuses and stillborn calves are sometimes covered in nodules, but they may also appear normal. Severely affected cattle with lumpy skin disease can die, but most animals slowly recover. However, this may take several months, and some skin lesions can take a year or two to resolve. Deep holes or scars are often left in the skin.

Similar clinical signs of varying severity have been reported in water buffalo, yaks, mithuns, gaur, camels, bantengs and free-living or captive wild ungulates. Water buffalo seem to be less severely affected than cattle and often have subclinical infections or seroconvert without signs of illness. The few cases documented in camels were characterized by small, self-limited nodules, which resolved within a few days and did not develop into pustules, form scabs or have necrotic cores. The reported illnesses in giraffes, Indian gazelles and some other wild ungulates were severe and/or fatal; however, milder cases might have gone unnoticed. Skin lesions might also be subtle in some individuals. Experimental infections in a newborn giraffe and a few-week-old impala calf resulted only in isolated lesions until shortly before death, which occurred soon after the lesions generalized and the animals became visibly ill. While both animals had severe stomatitis at this stage, neither displayed excess salivation. Most of the lesions in the impala were in the metacarpal and metatarsal region, while visible lesions in the giraffe occurred mainly in the mouth and at the inoculation site. An adult giraffe, however, developed the characteristic skin nodules, mainly on the legs and neck.

Post Mortem Lesions

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While the gross lesions in subclinically infected animals may be limited to lymph node enlargement, cattle that die of lumpy skin disease usually have characteristic grayish-pink deep cutaneous nodules with necrotic centers. The nodules often extend into the subcutaneous tissues and underlying skeletal muscle, and adjacent tissues exhibit congestion, hemorrhages and reddish-yellow edema. 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 also 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 may be difficult to see and often appear as focal areas of atelectasis and edema, but some animals may have areas of consolidation and lesions typical of bronchopneumonia. The mediastinal lymph nodes may be enlarged in severe cases, and pleuritis may be evident. Additional complications such as synovitis and tenosynovitis are also possible. Aborted fetuses and premature calves may have large numbers of skin nodules, but can appear normal. They may also have lesions on internal organs. One study, in a limited number of cattle, reported no obvious differences in gross lesions between animals infected with cluster 1 and cluster 2 (vaccine recombinant) viruses.

The lesions in species other than cattle appear to be similar. One naturally-acquired fatal case in an adult giraffe was characterized by large numbers of skin nodules but few lesions in internal organs.

Diagnostic Tests

LSDV, its nucleic acids and antigens can be detected in biopsy samples from skin nodules, scabs, nodular fluid, skin scrapings, lymph nodes, the spleen and lesions on various internal organs. Live virus can sometimes be recovered from skin nodules for up to 3-4 weeks, although samples are preferably collected during the first week of illness. Viral nucleic acids may also be found by PCR in nasal or oral secretions and ocular swabs. LSDV may be isolated from blood samples during the early, viremic stage, but this is unlikely to be successful after generalized lesions have been present for more than a few days.

PCR is often used for diagnosis. Tests that have been developed include some that can distinguish LSDV from sheeppox and goatpox viruses, or cluster 1 field viruses from cluster 1 vaccine strains (DIVA tests). Identifying cluster 2 vaccine recombinant viruses can be difficult unless sequencing is done, as the commonly used PCR tests may identify them as either vaccine strains, field strains or both, and sometimes don't detect these viruses at all. However, some recently published PCR tests or combinations of tests are reported to identify these viruses. Dot blot hybridization tests, loop-mediated isothermal amplification assays (LAMP) and recombinase polymerase amplification assays have also been published for LSDV.

Capripoxvirus antigens can be detected in tissues by immunostaining. Antigen-detection ELISAs have also been described and might be available in some laboratories. Virus isolation can be accomplished in various bovine, caprine or ovine cell cultures. LSDV is reported to grow best in primary or secondary lamb testis or bovine dermis cells, but other cells or cell lines (e.g., MDBK cells) can be used. This virus will also grow in embryonated chicken eggs; however, this method

is not sensitive enough for primary virus isolation. Isolated viruses can be recognized as capripoxviruses with direct immunofluorescence, virus neutralization and other tests, and they can be identified as LSDV with PCR tests or other genetic assays.

Histopathology can be helpful, and other tests have been used occasionally. Transmission electron microscopy can detect the typical capripoxvirus morphology in biopsy samples or desiccated crusts, and distinguishes these viruses from the parapoxviruses that cause bovine papular stomatitis and pseudocowpox. Together with a history of consistent clinical signs, it can provide a presumptive diagnosis in endemic areas. Electron microscopy cannot distinguish capripoxviruses from orthopoxviruses (cowpox and vaccinia virus), but orthopoxvirus infections do not usually resemble lumpy skin disease.

Various serological tests, including virus neutralization, an indirect fluorescent antibody test, ELISAs and immunoblotting (Western blotting), can detect antibodies to capripoxviruses. Most of these tests, including standard virus neutralization, cannot determine whether the antibodies are to LSDV, goatpox virus or sheeppox virus. Samples from mildly affected cattle sometimes give false negatives in serological tests, and one study suggested that infected water buffalo might have low or no neutralizing titers but can be recognized more readily with ELISAs.

Treatment

There is no specific antiviral treatment for lumpy skin disease. Affected animals are treated supportively, including with antibiotics as necessary for secondary bacterial infections. 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, on contaminated fomites, or by insects. Limited outbreaks recognized early have sometimes been controlled with quarantines, depopulation of affected herds, and cleaning and disinfection of infected premises, but vaccination was an important component of some eradication campaigns, particularly when the outbreak was larger. Culling of exposed, or at least affected, animals is still recommended when mass vaccination is used, but its extent may vary. Insect control is usually included, though its effectiveness is still unclear. Some authors note that insecticide treatment of carcasses helps prevent flies from acquiring the virus, which is particularly important if the carcasses are transported through uninfected areas. Quarantines and movement controls help prevent live animals from introducing LSDV to distant foci.

Live attenuated vaccines can control losses in areas where LSDV is endemic, and also appear to decrease or eliminate virus shedding in some secretions and excretions. Vaccine reactions occur in a minority of recipients, and seem to be more prominent in regions where the virus is newly introduced and animals are being vaccinated for the first time. These reactions (the “Neethling response”) are usually characterized by skin lesions that mimic but are milder than lumpy skin disease, and contain the vaccine virus. Possible vaccine-associated abortions were also reported during the European mass vaccination campaigns. Killed vaccines are rarely available.

Morbidity and Mortality

Lumpy skin disease tends to be more common in warm, humid areas, but it is not limited to these regions. Overwintering and winter outbreaks have been demonstrated in cold climates, at least for cluster 2 vaccine recombinant viruses. Clinical cases can occur either sporadically or in epizootics. The speed at which an outbreak propagates, as well as the morbidity and mortality rates, are thought to be influenced by the density and efficiency of the local arthropod vectors. In endemic areas, the number of cases typically rises during wet, warm weather, when these vectors are more abundant. New foci of disease that appear at distant sites are mostly thought to be associated with the movement of infected animals rather than insects.

Individual cattle differ significantly in their susceptibility to lumpy skin disease and the severity of the clinical signs. Some infected herds contain only a few symptomatic animals, though other herd members may be infected subclinically. Even in laboratory experiments where the animals all receive the same dose, clinical signs often appear in only 30-70% of the recipients. *Bos taurus* breeds, particularly Channel Island breeds, are reported to be more susceptible than zebu cattle (*Bos indicus*). Young calves and lactating cows also tend to be more severely affected.

Reported morbidity rates in outbreaks are highly variable, ranging from 1-2% to 80-90%. During the period when lumpy skin disease was mostly confined to Africa, estimates of morbidity were often around 2-20% and mortality rates in the range of 1-10%. Morbidity was less than 30%, and sometimes much lower, during some of the recent outbreaks in Europe and Asia, and mortality was less than 1% in many areas, though higher rates were also seen. Vaccination and other control measures might have played a role in limiting disease severity in some countries. In Jordan morbidity and mortality rates were 43% and 10%, respectively, in unvaccinated cattle, with 5% morbidity and 1% mortality in vaccinated cattle. Reported case fatality rates in symptomatic animals mostly vary from 2% to 23%, with occasional reports of higher rates, particularly in small groups of animals, where just a few deaths can significantly influence this value.

Lumpy skin disease seems to be much milder in water buffalo than cattle, with a high proportion of subclinical infections or seroconversion without clinical signs, and few deaths. Limited evidence suggests that mithuns might also have relatively mild signs, and the few reported cases in camels were mild, self-limited and occurred in animals that had close contact with cattle during a major outbreak. The situation in yaks is unclear: while this species had similar morbidity and mortality rates as cattle during some outbreaks in India, they were more severely affected on one mixed farm in China. There are only a few reports of clinical cases in captive or free-living wildlife, and the effect of LSDV on these species is still poorly understood.

Internet Resources

European Food Safety Authority (EFSA). Lumpy Skin Disease

EFSA. Scientific Opinion on Lumpy Skin Disease

Food and Agriculture Organization of the United Nations (FAO). Lumpy Skin Disease Field Manual

The Merck Veterinary Manual

United States Animal Health Association. Foreign Animal Diseases

World Organization for Animal Health (WOAH)

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

WOAH Terrestrial Animal Health Code

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