

sheeppox and goatpox

Etiology

- Sheeppox, goatpox and lumpy skin disease of cattle are **members of the genus Capripoxvirus, one of six genera of poxviruses.**
- The **diseases produced by sheeppox and goatpox viruses are collectively called capripox infections.**
- They are named on the basis of their host specificity in natural outbreaks and are usually highly host specific in natural infections, although exceptions exist.
- For example, Kenya sheeppox and goatpox viruses, and Yemen and Oman sheep isolates, infect both sheep and goats, although the disease caused by the same isolate can vary dramatically between the two hosts.
- The viruses are closely related genetically, and hence they cross-react in serologic tests, and many can cross species barriers in experimental infections. Recombination may also occur naturally between isolates from different host species.

Epidemiology

- **The Capripoxvirus infections of small ruminants are the most serious of all the pox diseases in animals, characterized by fever and skin lesions.**
- In susceptible flocks and herds morbidity is 75% to 100%, with outbreaks often causing death in 10% to 85% of affected animals depending on the virulence of the infecting strain.

Methods of Transmission

Sheeppox and goatpox are highly contagious.

- The virus enters via the respiratory tract,

and transmission

- commonly is by aerosol infection associated with close contact with infected animals.
- **The virus is present in nasal and oral secretions for several weeks after infection and can live in scabs that have fallen off the animal for several months.**

-Spread can also occur from contact with contaminated materials and through skin abrasions produced iatrogenically or by insects.

- **Capripox has been shown to spread via the bites of *S. calcitrans* and the tsetse fly.** Experimental Reproduction The disease can be transmitted by intradermal, intravenous, and subcutaneous inoculation and by virus aerosols.

-Capripox antigen is detected 6 and 8 days postinfection in skin and lungs, respectively.

- **Risk Factors Animal Risk Factors Both sheeppox and goatpox affect sheep and goats of all ages, all breeds, and both sexes, but young and old animals and lactating females are more severely affected.**

-Young animals are more susceptible.

- Pathogen Risk Factors

The virus is resistant to drying and survives freezing and thawing. It is sensitive to extremes of pH and 1% formalin.

- Sensitivity to heat varies between strains, but most are inactivated at 60°C (140F) for 60 minutes. Isolates from most regions are host specific, but isolates from Kenya and Oman naturally infect both goats and sheep.

-Scabs shed by infected animals remain infective for several months.

- Economic Importance Loss is from mortality, abortions, mastitis, loss of wool, skin condemnation, and loss of exports.

- In ewes and does, severe losses may occur if the udder is invaded because of the secondary occurrence of acute mastitis.

- In some outbreaks, adult sheep are affected with the more severe form of the disease.

-Sheeppox is a potent threat to countries that have large sheep populations, and where the disease does not occur, because it is difficult to eradicate and has a high mortality rate.

In a natural outbreak in an intensive sheep dairy in Israel, losses accrued from acute illness, deaths, reduced milk production, and reduced fertility.

-Milk production declined for 8 weeks after the index cases and was accompanied by an increased somatic cell count.

Zoonotic Implications Human infections in people handling infected animals are not a consideration.

Pathogenesis

During an initial viremia, the virus is carried by infected monocytes/macrophages to many tissues, particularly the skin, respiratory tract, and gastrointestinal tract.

Syncytial cells are seen in skin, and these probably facilitate local spread of the virus.

- The development of typical pox lesions, as in vaccinia, is characteristic of the disease.

The virus is present in greatest quantities from 7 to 14 days after inoculation. Passive protection by serum will protect against challenge.

Circulating antibody limits spread of infection, but does not prevent replication of the virus at the site of inoculation.

CLINICAL FINDINGS

In sheep, sheeppox has an incubation period of 12 to 14 days, with the malignant form being the most common type in lambs.

There is marked depression and prostration, a very high fever, and discharge from the eyes and nose.

Affected lambs may die during this stage before typical pox lesions develop. When pox lesions develop, they appear on unwooled skin and on the buccal, respiratory, digestive, and urogenital tract mucosae.

-They commence as papules, then become nodular, occasionally vesicular, and pustular, then finally scab. Some progress from nodules to tumor-like masses.

The mortality rate in this form of the disease may reach 50%. In the benign form, more common in adults, only skin lesions occur, particularly under the tail; there is no systemic reaction, and animals recover in 3 to 4 weeks.

Abortion and secondary pneumonia are complications. In sheep, infection with goatpox is more severe than with sheeppox, with lesions on the lips and oral mucosa, teats, and udder.

Goatpox in goats is very similar clinically to sheeppox in sheep. Young kids suffer a systemic disease, with lesions spread generally over the skin and on the respiratory and alimentary mucosae.

Adult goats may have systemic disease and extensive lesions, but in adult goats the disease is usually mild, and lesions are as described previously for the benign form in sheep.

A flat hemorrhagic form of capripox is seen in some European goats, and this form has a high case-fatality rate.

CLINICAL PATHOLOGY

Antigen Detection Diagnosis is based on typical clinical signs combined with laboratory confirmation of the presence of the virus or antigen. Using electron microscopy, large numbers of characteristic “sheeppox cells” containing inclusion bodies and typical capripox virions can be seen in biopsies of the skin.

The virus can be cultured in tissue culture, but virus isolation as a method of rapid diagnosis is limited by the extended time it takes for virus cytopathic effects to develop and the need, with some strains, for several blind passages before this occurs.

Direct fluorescent antibody testing is used to detect the presence of poxvirus in the edema fluid, and the antigen can be detected in biopsies of lymph glands by AGID using specific immune sera.

An antigen detection ELISA is also available .

Serology : Serologic testing can be by virus neutralization, which is 100% specific, or by an indirect fluorescent antibody or an agar gel precipitation test (AGPT), both of which cross-react with antibody to orf virus. An indirect ELISA has a similar diagnostic sensitivity and slightly lower specificity than the virus neutralization assay.⁶ Virus-specific analysis of antibody response by Western blot can differentiate the infections.

PCR and melt-point analyses for the detection of capripox antigen have been developed, some as duplex or multiplex assays to differentiate capripox infections from orf virus.

These are suitable for use in countries that do not have the disease and do not hold live capripox virus.

Loop-mediated isothermal amplification (LAMP) assays are potentially a cost-effective test to rapidly differentiate sheeppox and goatpox during outbreaks.

NECROPSY FINDINGS

In the malignant form, pox lesions extend into the mouth, pharynx, larynx, and vagina, with lymphadenopathy and a hemorrhagic spleen.

- Lesions may also appear in the trachea. Lesions in the lung are severe, manifesting as lentil-sized white pox nodules to a consolidating and necrotizing pneumonia.

Lesions occasionally reach the abomasum and are accompanied by hemorrhagic enteritis. Histologically, cells infected with capripox virus have a characteristic appearance with vacuolated cytoplasm and nuclei, marginated chromatin, and multiple inclusion bodies (“sheeppox cells”).

With the use of double immunohistochemical labeling, the viral antigen appears in cells of the monocyte/macrophage lineage within 6 to 8 days of infection, and later in pneumocyte .

DIFFERENTIAL DIAGNOSIS

1-Contagious ecthyma (orf)

2-Bluetongue

TREATMENT

No specific treatment is advised, but palliative treatment may be necessary in severely affected animals.

CONTROL

Control in countries or regions that are free of this disease centers around prohibiting the importation of live animals and unprocessed produce from infected areas and, if the infection is introduced, ring vaccination, the destruction of affected flocks, and the quarantine of infected premises.

Vaccination with natural lymph has been used in some affected areas, but it can spread the disease.

Natural infection with one capripox strain imparts immunity to all capripox infections, and vaccination with a single capripox vaccine will give protection across all species and against all capripox infections.

A variety of commercial vaccines are available, and there is no easy basis for comparison.

- Killed virus vaccines elicit only temporary protection, but live attenuated vaccines protect against infection for more than 1 year.

-Colostrum antibody can interfere with vaccination until 6 months of age.

-Vaccination programs in endemic areas recommend vaccination of lambs at 2 and 10 weeks, followed by an annual booster.⁵

-A subunit capripox virus vaccine has been developed.

-Vaccination in the face of outbreak is unlikely to prevent deaths during the subsequent 2 weeks and, if needle hygiene is poor, may facilitate the spread of the disease.