

Peste des Petits Ruminants

*Pseudorinderpest,
Goat Plague,
Pest of Small Ruminants,
Pest of Sheep and Goats,
Kata,
Stomatitis-Pneumoenteritis Syndrome,
Pneumoenteritis Complex*

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Importance

Peste des petits ruminants (PPR) is a highly contagious viral disease of sheep and goats. Heavy losses can be seen, especially in goats; all of the affected animals in some herds may die. At one time, peste des petits ruminants was thought to be restricted to West Africa, but it has since been recognized from the equator to the Sahara desert, as well as in Asia and the Middle East. Other nearby areas, such as southern Africa and central Asia, are threatened. Although increased recognition of PPR is one reason for the expanded geographic range, it is also possible that this virus is spreading.

The host range of peste des petits ruminants in wild animals is still unknown, and it is possible that this disease could threaten the conservation of some wildlife species. Severe outbreaks were reported in susceptible buffalo in 1995 and in captive gazelles in 2002. Nearly all of the affected animals died. Other species, such as deer and wild relatives of domesticated sheep and goats, may also be affected. In addition, PPR virus can infect some species asymptotically, which complicates surveillance for the closely related rinderpest virus.

Etiology

Peste des petits ruminants virus (PPRV) is a member of the genus *Morbillivirus* in the family Paramyxoviridae. Four genetic lineages (lineages 1-4) have been identified.

PPRV is closely related to rinderpest virus. Antibodies to PPRV and rinderpest are cross-protective, and vaccination for rinderpest can mask the presence of peste des petits ruminants. Serological cross-reactivity also complicates some diagnostic tests; the existence of a global rinderpest eradication campaign, which is in the final surveillance stage, makes it particularly important to differentiate these two viruses.

Species Affected

Among domesticated animals, peste des petits ruminants is primarily a disease of goats and sheep. PPRV is thought to have played a role in one epizootic in Ethiopia (1995-1996) that affected camels. Cattle are usually infected asymptotically, and are not known to transmit the disease to other animals. No clinical signs were reported in experimentally infected pigs, which also appear to be dead-end hosts.

Peste des petits ruminants can affect some wild ungulates, but there is very limited information on species susceptibility and the occurrence of disease. PPR was confirmed as the cause of two severe outbreaks, one in captive Dorcas gazelles (*Gazella dorcas*) and Thomson's gazelles (*Gazella thomsoni*) in Saudi Arabia in 2002, and the other in buffalo in India in 1995. Peste des petits ruminants is also thought to have caused another outbreak that affected both gazelles and deer in Saudi Arabia in the 1980s. White-tailed deer (*Odocoileus virginianus*) can be infected experimentally. In addition, peste des petits ruminants has been reported in captive Nubian ibex, Laristan sheep and gemsbok. Whether wild ruminants are important in the epidemiology of this disease is unknown.

Geographic Distribution

Peste des petits ruminants occurs south of the Sahara desert and north of the equator in Africa, in most of the Middle East, and in parts of Asia including much of the Indian subcontinent. The four virus lineages are found in different geographic regions. Lineages 1 and 2 occur in parts of Africa, and lineage 3 has been reported from parts of Africa, the Middle East, and southern India. It is not certain whether lineage 3 has persisted in India; one study reports that there is no evidence for this virus after 1992. Lineage 4 has been found in the Middle East and the Indian subcontinent, but as of 2008, this virus has not been reported from Africa.

Transmission

Transmission of PPRV mainly occurs during close contact. Inhalation is thought to be an important route of spread. PPRV is shed in nasal and ocular secretions, saliva, urine and feces. It probably occurs in milk. Although animals are not expected to become long-term carriers, one recent study reported that viral antigens were shed in

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the feces of clinically recovered goats for at least 11 to 12 weeks. Animals may also be contagious during the incubation stage. PPRV is relatively fragile in the environment, and long distance aerosol transmission is unlikely; in cool temperatures and in the dark, this virus has been shown to spread for approximately 10 meters.

Fomites such as water, feed troughs and bedding can probably transmit PPRV for a short time, but do not remain infectious for long periods. There is very little information on the survival of PPRV in the environment, but this virus is very similar to rinderpest virus, which is inactivated by ultraviolet light and desiccation within four days, and normally survives for very short periods in carcasses. Temperatures above 70°C, as well as pH less than 5.6 or greater than 9.6, are also expected to inactivate PPRV. This virus may, like rinderpest virus, survive for a time in refrigerated meat, and for several months in salted or frozen meat. However, PPRV is unlikely to be transmitted to sheep or goats from this source, because pigs that might be fed meat are dead-end hosts. How the virus is maintained between outbreaks is not well understood.

Incubation Period

The incubation period can range from two to 10 days; in most cases, clinical signs appear in 2-6 days.

Clinical Signs

The severity of the disease varies with the species, as well as the animal's immunity to PPRV and its breed. Goats and sheep are not always affected to the same extent during an outbreak.

Peracute cases can be seen when PPR first occurs in naïve populations of sheep or goats. In this form, the clinical signs are generally limited to high fever, severe depression and death. More often, peste des petits ruminants occurs as a subacute or acute disease. In acute cases, the initial signs include a sudden high fever, inappetence, marked depression and somnolence. Serous nasal and ocular discharges appear soon after the onset of disease; these discharges generally become mucopurulent from secondary bacterial infections. Matting is common around the eyes, and the nose may become obstructed. Within a few days of the onset of fever, the gums become hyperemic, and small, gray, necrotic foci, covering shallow erosions, begin to appear in the mouth. (If these lesions are difficult to find, rubbing a finger across the gums and palate may recover foul-smelling exudates and shreds of tissue.) In some cases, the mouth lesions resolve rapidly. In others, they enlarge, spread and coalesce. Lesions are most common on the lips and gums, but they can also be found on the dental pad, palate, cheeks and their papillae, and tongue. In severe cases, the mouth may be completely covered in thick cheesy material. The oral lesions are painful, and animals may resist opening their mouths. The lips are often swollen, cracked and crusted, and the breath of animals with severe stomatitis is fetid. Increased

salivation is usually seen. Necrotic lesions may also be found on other mucous membranes, including those of the nasal cavity, vulva and vagina. Most animals develop profuse diarrhea, which may be watery, fetid and/or blood-stained, and sometimes contain shreds of tissue. Rapid respiration is common, and dyspnea, coughing and other signs of pneumonia may be seen. Some animals abort. In the late stages of the disease, small nodules resembling contagious ecthyma or sheep/goat pox can appear in the skin around the muzzle. The cause of these lesions is unknown. Severely affected animals become dehydrated and emaciated; hypothermia can precede death. Animals that do not die often have a prolonged convalescence. Subacute disease can also be seen in some animals; this form usually lasts 10-15 days. The symptoms are variable, but often include respiratory signs. Asymptomatic infections also occur.

Domesticated animals other than sheep and goats do not usually become ill. Cattle are usually asymptomatic; however, clinical signs have been reported in experimentally infected calves, and it is possible that some cattle in poor condition might become symptomatic. If they did, the syndrome would probably resemble rinderpest. Respiratory disease was reported in camels during an outbreak that may have been complicated by *Streptococcus equi*. Experimentally infected pigs remain asymptomatic.

Clinical signs have been described for a few exotic species. Deer can have symptoms similar to sheep and goats, but subclinical infections have also been reported. Captive gazelles became severely ill during one outbreak. The initial signs were anorexia and depression, followed by fever, lacrimation, congested mucous membranes, nasal discharges, salivation and diarrhea. All affected animals died. A highly fatal outbreak in buffalo was characterized by depression, profuse salivation and conjunctival congestion; however, the animals were not febrile. Experimentally infected 3-5-month-old buffalo calves developed a fever but no other clinical signs, and died in 30-35 days. Gastrointestinal lesions were found in these calves at necropsy.

Post Mortem Lesions

The postmortem lesions are characterized by inflammatory and necrotic lesions in the oral cavity and throughout the gastrointestinal tract.

The carcass is often emaciated and/or dehydrated, and may have evidence of diarrhea and serous or mucopurulent oculonasal discharges. The lips often have prominent crusty scabs, and necrotic stomatitis is common. Erosions, which are shallow and sharply demarcated from normal epithelium, may be found in the mouths of some animals. In severe cases, the hard palate, pharynx and upper esophagus can also be involved. Similar lesions may be found on the vulva and vaginal mucous membranes. The rumen, reticulum and omasum are not significantly involved, although erosions are occasionally found on the pillars of

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the rumen. Erosions, which may ooze blood, are common in the abomasum. Hemorrhagic streaks and erosions sometimes occur in the duodenum and the terminal ileum. The Peyer's patches often have extensive necrosis, which can lead to ulceration. The most severe lesions are seen in the large intestine, particularly around the ileocecal valve, at the cecocolic junction and in the rectum. "Zebra stripes" or "tiger stripes" of congestion, hemorrhage or darkened tissue can sometimes be seen in the posterior part of the colon on the mucosal folds. (Zebra stripes can also be seen in animals with diarrhea and tenesmus from other causes). Respiratory lesions are also common. Congestion, small erosions and petechiae may be found in the nasal mucosa, turbinates, larynx and trachea, and blood-tinged, frothy exudates have been reported in the tracheas of some experimentally infected goats. Many animals have bronchopneumonia. The lymph nodes, particularly those associated with the respiratory and gastrointestinal tracts, are generally congested, enlarged and edematous. In peracute cases, the lesions may be limited to congestion of the ileocecal valve and bronchopneumonia.

Similar lesions have been reported in buffalo and gazelles. Hemorrhagic and edematous gastroenteritis (involving the abomasum and all segments of the intestines) was reported in infected buffalo. In gazelles, small erosions were found on the tongue, and the esophagus contained thick mucoid deposits along the walls. The papillae of the rumen were congested. The abomasum was severely affected, with tiny hemorrhagic erosions, marked congestion and edema in the pyloric region. Congestion, hemorrhages and small erosions were found in the duodenum, and congestion was seen in the jejunum. The Peyer's patches appeared shallow and were hyperemic at their edges. Congestion was seen around the ileocecal valve. The mucosae of the colon and rectum were congested, with a 'zebra stripe' pattern. Congestion was also reported in the liver, kidney, pancreas and brain. Froth was found in the trachea and bronchi, and the lungs were congested. The lymph nodes and spleen were small. Unilateral corneal opacity was reported in one animal.

Morbidity and Mortality

Peste des petits ruminants is highly contagious when it first occurs in a naïve population. Periodic outbreaks may also be seen in endemic regions, particularly when animals are mixed or new animals are introduced into a herd. Some epizootics are associated with changes in weather, such as the beginning of the rainy season or a cold, dry period. In endemic regions, animals between three months and two years of age are most severely affected; young animals that are still nursing and older animals tend to be spared.

The severity of the disease varies with the host's species, immunity and breed. Clinical signs are reported to be more common in goats than sheep in Africa, but the opposite has been reported in some parts of Asia. Breed differences are also seen: some isolates can affect one breed

of goats severely, while causing mild disease in another. The morbidity and mortality rates can reach 100%, particularly in naïve herds; however, these rates tend to be lower in endemic areas and the reported mortality rates in some individual flocks are as low as 20%.

High case fatality rates have been reported when PPRV infected herds of exotic ungulates. In an outbreak among buffalo in India, the case fatality rate was 96%. Fifty of 385 buffalos were affected; most (38) of these cases occurred in animals that had been recently introduced into the herd and were not yet vaccinated against rinderpest. In captive gazelles, the morbidity rate was 51% and the case fatality rate was 100%. During a countrywide outbreak among camels in Ethiopia, the morbidity rate was greater than 90%, and the mortality rate ranged from 5% to 70%.

Diagnosis

Clinical

Peste des petits ruminants should be considered in sheep, goats or gazelles with any acutely febrile, highly contagious disease characterized by oral necrosis with ocular and nasal discharges, respiratory disease and/or gastrointestinal signs.

Differential diagnosis

The differential diagnoses include rinderpest (although many reports of 'rinderpest' among small ruminants may have been PPR), bluetongue, contagious ecthyma, foot and mouth disease, heartwater, coccidiosis and mineral poisoning. The respiratory signs can resemble contagious caprine pleuropneumonia (CCPP) or pasteurellosis; pasteurellosis can also be a secondary complication of peste des petits ruminants.

Laboratory tests

Peste des petits ruminants can be confirmed by virus isolation, but recovery of the virus is not always successful. PPRV can be isolated in B95a cells (a marmoset cell line). Primary lamb kidney or African green monkey kidney (Vero) cell cultures have also been used. Cultures are examined for cytopathic effect (CPE); the identity of the virus can be confirmed by virus neutralization or other methods.

PPRV antigens can be detected by immunocapture ELISA (ICE), counter immunoelectrophoresis (CIEP) or agar gel immunodiffusion (AGID). CIEP and ICE can distinguish PPRV from rinderpest virus, but the AGID test cannot differentiate these two viruses. AGID is also relatively insensitive, and may not be able to detect small quantities of viral antigens in milder forms of PPR. Immunofluorescence and immunochemistry can be used on conjunctival smears and tissue samples collected at necropsy. Viral nucleic acids can be detected with reverse transcription PCR (RT-PCR). Other forms of PCR, including a multiplex RT-PCR and a RT-PCR-ELISA, have also been described.

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Serological tests include virus neutralization and competitive ELISA assays. Both tests can distinguish peste des petits ruminants from rinderpest; this is not always possible with older serological tests such as complement fixation.

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 only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease.

In live animals, swabs of ocular and nasal discharges, and debris from oral lesions should be collected; a spatula can be rubbed across the gum and inside the lips to collect samples from oral lesions. Whole, unclotted blood (in heparin or EDTA) should be taken for virus isolation and PCR. Biopsy samples of lymph nodes or spleen may also be useful. Samples for virus isolation should be collected during the acute stage of the disease, when clinical signs are present; whenever possible, these samples should be taken from animals with high fever and before the onset of diarrhea. At necropsy, samples can be collected from lymph nodes (particularly the mesenteric and mediastinal nodes), lungs, spleen, tonsils and affected sections of the intestinal tract (e.g. ileum and large intestine). These samples should be taken from euthanized or freshly dead animals. Samples for virus isolation should be transported chilled on ice. Similar samples should be collected in formalin for histopathology. Whenever possible, paired sera should be taken rather than single samples. However, in countries that are PPR-free, a single serum sample (taken at least a week after the onset of clinical signs) may be diagnostic.

Recommended actions if peste des petits ruminants is suspected

Notification of authorities

State and federal veterinarians should be informed immediately of any suspected cases of peste des petits ruminants.

Federal: Area Veterinarians in Charge (AVIC):

www.aphis.usda.gov/animal_health/area_offices/

State Veterinarians:

www.usaha.org/Portals/6/StateAnimalHealthOfficials.pdf

Control

Peste des petits ruminants is usually spread by direct contact, and it is usually introduced into a herd by an infected animal. Asymptomatically infected animals can shed the virus, and one report suggests that virus shedding may be possible for up to 12 weeks or longer in recovered animals. Quarantine and testing decrease the risk of infection.

Peste des petits ruminants can be eradicated with a combination of quarantines, movement controls, euthanasia

of infected and exposed animals, and cleaning and disinfection of infected premises. Ring vaccination and/or vaccination of high-risk populations can also be helpful. The rapid inactivation of PPRV in the environment aids eradication; this virus is thought to remain viable for less than four days outside the animal. PPRV can be inactivated by many disinfectants including alkalis (sodium carbonate, sodium hydroxide), halogens (sodium hypochlorite), phenolic compounds, citric acid, alcohols and iodophores. Carcasses are generally buried or burned. Care should be taken to prevent the virus from spreading to susceptible or potentially susceptible wild populations such as deer, gazelles, wild sheep or feral goats.

Peste des petits ruminants is controlled in endemic areas by vaccination. Animals that recover develop good immunity, which persists for at least four years and possibly lifelong. To prevent infections in susceptible wildlife and captive wild animals such as gazelles, they should be prevented from having contact with sheep and goats. Vaccination might also be possible in these species. Good nursing and treatment for bacterial and parasitic complications can help to decrease mortality in endemic regions.

Public Health

There is no evidence that peste des petits ruminants virus infects humans.

Internet Resources

Food and Agriculture Organization (FAO) of the United Nations. Recognizing peste des petits ruminants. A field manual

<http://www.fao.org/docrep/003/x1703e/x1703e00.htm>

The Merck Veterinary Manual

<http://www.merckvetmanual.com/mvm/index.jsp>

United States Animal Health Association. Foreign Animal Diseases

http://www.vet.uga.edu/vpp/gray_book02/fad/index.php

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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