Peste des Petits Ruminants

Ovine Rinderpest
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 serious viral disease of sheep and goats, with morbidity and mortality rates than can approach 80-100% in naive populations. Clinical cases have also been recognized in various antelopes and wild relatives of sheep and goats, and occasionally in camels, some cervids and water buffalo. Some outbreaks have had a devastating effect on wildlife populations, raising concerns about the conservation of some endangered species. Since the 1980s, the PPR virus (PPRV) has expanded its boundaries in Africa, Asia and the Middle East, with recent incursions into parts of Europe as well. Some authors have speculated that this expansion might have been facilitated by the eradication of rinderpest virus, a closely related pathogen of cattle that can provide small ruminants with cross-protective immunity to PPRV. In 2015, international animal health groups began a project to eradicate PPRV, with the goal of complete elimination of this virus from the world by 2030. However, progress has been limited and uneven and faces significant barriers in some areas. The possibility of virus maintenance in wildlife also raises questions about its feasibility.

Etiology

Peste des petits ruminants virus (official species name: *small ruminant morbillivirus*) is a member of the genus *Morbillivirus* in the family Paramyxoviridae. PPRV has only one serotype, but four genetic lineages (lineages 1-4) and a number of viral strains, which may differ in virulence. Lineage 4 viruses, which are the only viruses with significant circulation outside Africa, are particularly common.

Species Affected

Peste des petits ruminants is primarily a disease of goats and sheep, which are its reservoir hosts. This virus has also been implicated, either alone or with other pathogens, in a few outbreaks in camels and water buffalo. Antibodies have been found in cattle, alpacas and free-ranging domestic yaks (*Bos grunniens*), and camels, water buffalo, cattle, pigs, llamas and alpacas can be infected experimentally.

Among wildlife, clinical cases have been seen in various relatives of sheep and goats, including members of the genera *Capra* and *Ovis* (e.g., wild goats, ibex, argali), aoudad/ Barbary sheep (*Ammotragus lervia*) and bharal (*Pseudois nayaur*); some antelopes, such as gazelles (*Gazella* spp.), saiga antelope (*Saiga tatarica*), impala (*Aepyceros melampu*), gemsbok (*Oryx gazella*), bushbuck (*Tragelaphus scriptus*), springbok (*Antidorcas marsupialis*), and four-horned antelope/ chowsingha (*Tetracerus quadricornis*); and certain cervids, including farmed water deer (*Hydropotes inermis)*. White-tailed deer (*Odocoileus virginianus)* can be infected experimentally. Serological and/or PCR-based evidence of infection, without reports of clinical signs, has been demonstrated in many other free-living or captive wild ungulates, including but not limited to African buffalo (*Syncerus caffer*), blue wildebeest (*Connochaetes taurinus*), waterbuck (*Kobus ellipsiprymnus*), topi (*Damaliscus lunatus*), and giraffes (*Giraffa camelopardalis*).

After domestic pigs and wild boar were found to be susceptible to experimental inoculation, a study examined free-living warthogs (*Phacochoerus africanus*) and found that some individuals were seropositive. Viral nucleic acids were reported in the tissues of an Asiatic lion (*Panthera leo persica*) that died of trypanosomiasis, and in nasal swabs from some dogs during PCR-based surveillance of animals visiting a veterinary clinic. However, it is possible that the nucleic acids in these dogs only represent contamination of the nares from viruses in the environment. Whether any wildlife can act as reservoirs for PPRV is unclear, but the virus can spread among wild populations during outbreaks, with the potential for transmission back to livestock.

Zoonotic potential

There is no evidence that humans are susceptible to PPRV.

Geographic Distribution

Peste des petits ruminants occurs in most of Africa and the Middle East and parts of Asia, with additional reports of outbreaks in Georgia, Albania, Turkey and Bulgaria in recent years. Lineage 4 viruses seem to account for all or nearly all virus circulation

outside Africa, though there have been rare reports of clinical cases that were apparently caused by other lineages. Some of these cases might have been caused by live attenuated vaccine strains.

Transmission

Small ruminants can shed PPRV in nasal and ocular secretions, saliva, urine, feces and milk. While long-term carriage is not thought to occur, some studies have detected viral antigens and/or nucleic acids in the feces of clinically recovered goats for up to 4 months. How long live virus is present in the feces is uncertain. Transmission mainly occurs during close contact, often via inhalation. Long distance aerosols are unlikely: in cool temperatures and in the dark (conditions maximizing virus survival), PPRV was only able to spread about 10 meters. Fomite-mediated transmission is also possible, but this virus is not thought to survive very long in the environment. The closely-related rinderpest virus was inactivated by ultraviolet light and desiccation within 3-4 days or less, and normally remained viable for very short periods in carcasses. Rinderpest virus was, however, reported to survive for a time in refrigerated meat, and for several months in salted or frozen meat. PPRV RNA has been detected in *Culicoides* midges, but the significance of this finding, if any, is unclear.

Experimentally infected cattle, camels, South American camelids and pigs, most of which had few or no clinical signs, shed PPRV inconsistently and only at very low levels, suggesting that while they might transmit this virus occasionally, this is probably uncommon. The only animals that transmitted PPRV in the laboratory were pigs with clinical signs and camels, which both infected goats sharing their pens. Outbreaks in water buffalo, camels and wildlife suggest that animal-to-animal transmission is probably possible in incidental hosts if they become ill.

Disinfection

PPRV can be destroyed by many common disinfectants including sodium hypochlorite, phenolic agents, citric acid, alcohols, iodophors and alkalis (e.g., sodium carbonate, sodium hydroxide). This virus is also expected to be inactivated by pH less than 5.6 or greater than 9.6, or temperatures above 70°C (158°F).

Incubation Period

The incubation period ranges from 2 to 10 days. In most cases, clinical signs appear in about 3-6 days.

Clinical Signs

Sheep and goats

Classically, peste des petits ruminants begins as a high fever with nonspecific signs of illness (e.g., inappetence, marked depression) and serous oculonasal discharge, which eventually becomes mucopurulent. Matting is common around the eyes, and the nares may become obstructed. Within a few days, the gums become hyperemic and small, gray, necrotic foci, which cover shallow erosions, begin to appear in the mouth, particularly on the lips and gums. (If 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, with much of the mouth becoming covered with necrotic material in severely affected animals. Mouth lesions are usually accompanied by increased salivation, the lips are often swollen, cracked and crusted, and the breath of animals with severe stomatitis is fetid. Necrotic lesions may also be found sometimes on other mucous membranes, such as those of the nasal cavity, vulva and vagina.

Many affected sheep and goats also develop profuse diarrhea, which may be watery, fetid and/ or blood-stained, and sometimes contains shreds of tissue. Some have respiratory signs (e.g., coughing, dyspnea), and the illness can be complicated by secondary bacterial pneumonia. Pregnant animals may abort, and immunosuppression caused by PPRV can exacerbate concurrent infections, contributing to the clinical signs. Deaths are usually the result of dehydration and/or pneumonia. Animals that recover often have a prolonged convalescence.

Peracute cases, which are mainly seen when PPRV first infects naïve small ruminants, are generally limited to a sudden high fever and severe depression, followed rapidly by death. Mild cases are characterized by variable signs that often include respiratory involvement. Apparently healthy animals with subclinical infections may have PPRV-associated lesions in the lungs at slaughter.

Wild ungulates

Clinical cases that have been described in antelope, cervids and the wild relatives of small ruminants resemble those in sheep and goats, though some signs may be less prominent. Oral lesions were minimal or absent during outbreaks in some species, especially those with a rapid course. An acute illness in captive Nubian ibex (*Capra nubiana*) was characterized mainly by nonspecific signs of illness and watery-to-bloody diarrhea, which was usually fatal within 24 hours, while an outbreak in captive four-horned antelope appeared as an acute respiratory disease with frothy nasal discharge. Most of these antelope died within 2 days, with no diarrhea or obvious intestinal lesions at necropsy. Ulcerative keratitis and conjunctivitis has been seen in some species (e.g., wild goats, *Capra aegagrus*, and farmed Chinese water deer), in addition to the typical PPR signs, and lameness has been reported in some wildlife, though not definitively linked to PPR.

Camelids, water buffalo, cattle and pigs

Published information about outbreaks in camels is limited. Many animals might be infected subclinically, as PPRV antigens have been found in lung lesions from apparently healthy camels at slaughter. One outbreak in Iran was similar to the disease in small ruminants, with sudden death in some animals, and fever, nonspecific signs of illness, ecthyma-like lesions, ulcerative keratitis/

conjunctivitis, yellowish diarrhea and respiratory signs (pneumonia, respiratory distress) in others. However, oral erosions and necrosis were much less prominent than in small ruminants. Yellowish diarrhea, which later became bloody, and abortions were the major signs in another incident in camels, but nonspecific signs of illness, subcutaneous edema and infrequent coughing, as well as sudden deaths, were also seen. In this outbreak, fatalities were most common in animals that were pregnant or had recently given birth. Another outbreak attributed to PPRV, which may have been complicated by *Streptococcus equi*, was mainly characterized by acute respiratory disease with fever, dyspnea and lacrimation. Experimentally infected camels usually have few or no clinical signs, though one study reported fever and mild respiratory disease with coughing and nasal discharge.

Very few outbreaks have been reported in other domestic animals. An outbreak with a high case fatality rate in water buffalo was characterized by depression, profuse salivation and conjunctival congestion, but the animals were not reported to be febrile. Experimentally infected 3-5-month-old water buffalo calves developed a fever but no other clinical signs, and died in about a month. Few or no signs that could definitively be attributed to PPRV have been seen in experimentally infected cattle, alpacas and llamas, though some animals had mild nonspecific signs such as increased lethargy or mild ocular or nasal discharge. Pigs inoculated with one virus were asymptomatic, but some pigs and wild boar that received a different virus had mild to moderate signs including fever, reduced appetite and activity, diarrhea and conjunctivitis. Whether the diarrhea was caused by PPRV or a concurrent *Balantidium coli* infection was uncertain.

Post Mortem Lesions

Small ruminants that die of peste des petits ruminants are often emaciated and/or dehydrated. There may be evidence of diarrhea, serous or mucopurulent oculonasal discharges, crusted scabs on the lips, and stomatitis with necrotic areas and shallow, sharply demarcated erosions. Similar necrotic lesions and/or erosions may also be found on other mucous membranes, including those of the pharynx, upper esophagus and genital tract, and small erosions and petechiae may be noted on the nasal mucosa, turbinates, larynx and trachea.

The abomasum frequently contains erosions, Peyer's patches can have extensive necrosis, and the large intestine is often severely affected, particularly around the ileocecal valve, at the cecocolic junction and in the rectum. In some cases, there may be "zebra stripes" of congestion, hemorrhages and/or darkened tissue on the mucosal folds in the posterior colon. (These stripes can also be seen in animals with diarrhea and tenesmus from other causes). While the duodenum and the terminal ileum sometimes have hemorrhagic streaks and erosions, and there may occasionally be erosions on the pillars of the rumen, the rumen, reticulum, omasum and small intestine are otherwise generally spared.

Respiratory lesions may include congestion of the lungs or signs of bronchopneumonia, sometimes with evidence of secondary bacterial infection. Froth, which may be blood-tinged, is occasionally found in the trachea. The lymph nodes, particularly those associated with the respiratory and gastrointestinal tracts, are usually congested, enlarged and edematous, and the liver and spleen may have necrotic lesions. In peracute cases, lesions may be limited to congestion of the ileocecal valve and bronchopneumonia.

Similar lesions may be found in other hosts; however, some common lesions in small ruminants, such as necrosis and erosions in the upper intestinal and respiratory tracts, might be less prominent or absent. Hemorrhagic and edematous gastroenteritis involving the abomasum and all segments of the intestines was the major feature in naturally infected water buffalo, and similar gastrointestinal lesions were found in experimentally infected water buffalo calves. Conversely, an outbreak among captive four-horned antelope included hemorrhagic lesions in the trachea and severe congestion of the lungs, but no obvious intestinal lesions.

Diagnostic Tests

PPRV, its nucleic acids and antigens may be detected in the blood, ocular and nasal discharges, and/or swabs of the buccal and rectal mucosa from live animals, and in various tissues, particularly the mesenteric and bronchial lymph nodes, lungs, spleen, tonsils and affected sections of the intestinal tract at necropsy. While multiple tissues may be diagnostic in severely affected animals, the mesenteric lymph nodes are the most consistently positive site in animals with mild cases, and nasal swabs are more likely to detect the virus than ocular or oral swabs, especially when viral titers are low. Samples for virus isolation should be collected during the acute stage of the disease, preferably from animals with a high fever that have not yet developed diarrhea.

Clinical cases are often diagnosed by detecting viral nucleic acids with RT-PCR or reverse transcription-loop mediated isothermal amplification (RT-LAMP) assays. Virus isolation can also be employed, using Vero cells or other cell lines, including some new derivatives of Vero or CV1 cells that may recover the virus sooner and with less need for blind passages. The identity of the virus can be confirmed by RT-PCR or other genetic methods, or by virus neutralization.

PPRV antigens can be detected with immunocapture ELISAs or commercial fast lateral flow assays, as well as older assays such as counter immunoelectrophoresis (CIEP) and agar gel immunodiffusion (AGID). AGID is relatively insensitive, and may not be able to find small amounts of antigens in mild illnesses or early in the course of the disease. Immunofluorescence and immunohistochemistry can reveal antigens in conjunctival smears or tissue samples collected at necropsy.

Serological diagnosis in an endemic region requires paired serum samples and a rising titer, but a single sample may be sufficient in PPR-free countries. PPRV antibodies are usually detected with ELISAs or virus neutralization, though older tests such as complement fixation, AGID or CIEP have also been used. Serological tests used in wildlife or domestic animals other than small ruminants should be validated for that species. Camel antibodies, in particular, may not be detected by some commonly used tests.

Treatment

There is no specific treatment for peste des petits ruminants, but supportive care and treatment of bacterial and parasitic coinfections may decrease mortality.

Control

Disease reporting

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

Prevention

Peste des petits ruminants is generally controlled by vaccination in endemic regions. Avoiding contact between domestic small ruminants and captive or free-ranging wildlife, including contact via fomites, is expected to be helpful in preventing wildlife outbreaks. This virus can be excluded from disease-free regions with import testing and quarantines, and outbreaks can be eradicated by the standard methods, i.e., movement controls, euthanasia of infected and exposed animals, and cleaning and disinfection of infected premises, possibly aided by vaccination. The possibility that PPRV may circulate in wildlife, at least for a time, must also be considered in control and eradication plans.

Morbidity and Mortality

Exposure to PPRV can be common among small ruminants in some endemic regions, with seroprevalence rates ranging from < 2% to greater than 50%. Antibodies have also been found in a wide variety of wild ungulates and significant numbers of camels, cattle and water buffalo, with some studies from the Indian subcontinent showing higher seroprevalence in water buffalo than either goats or cattle.

The severity of the illness can be influenced by the animal's species, level of immunity, breed, age and concurrent illnesses or infections, as well as the virulence of the PPRV strain. Although clinical cases usually seem to be more severe in goats than sheep, there have been outbreaks in sheep where goats were only mildly affected. PPRV can spread widely when it first infects a population of small ruminants, and morbidity and case fatality rates may reach 80-90%. However, morbidity and mortality rates can be as low as 10-20% in endemic regions, where young animals between three months and two years of age are most severely affected and nursing lambs and kids or older animals tend to be spared. Endemic areas may have periodic outbreaks, particularly when animals are mixed or introduced into a herd. Seasonality has also been reported in some areas, associated with changes in the weather such as the beginning of the rainy season or a cold, dry period. Immunity is reported to persist for at least 4 years in recovered small ruminants, and for at least 3 years in animals immunized with some live attenuated vaccines.

Information about peste des petits ruminants in other domestic animals is limited. While many infections in camels might be subclinical, an outbreak in a group of camels in Iran was severe, and a country-wide epidemic in this species in Ethiopia had a morbidity rate of >90%, and mortality rates ranging from 5% to 70%. Mortality in camels varied from 0% to 50% during outbreaks in Sudan, and was highest in animals that were pregnant or had recently given birth. No clinical cases have been noted in cattle, and the one reported outbreak in water buffalo had a morbidity rate of 13% and case fatality rate of 96%. Most of the cases occurred in buffalo that had been recently introduced into the herd and were not yet vaccinated against rinderpest.

Outbreaks described in captive wildlife are often severe, with case fatality rates as high as 67-100% in some species. Disase severity is more difficult to quantify in free-living wildlife, but one estimate of an outbreak among saiga antelope (*Saiga tatarica*) in Mongolia suggested that 80% of this endangered population was lost to PPR. This disease also killed an estimated 25-40% of the wild goat population in one national park in Iran in 2001, and 77% of the wild sheep (*Ovis orientalis*) and wild goats in another Iranian national park in 2015-2016. During an outbreak in China, the estimated mortality rate was 65–70% in wild aoudad but 1-2% in other susceptible species. Whether this was the result of differences in their susceptibility or different population structures and habits that made the virus less likely to spread was unclear.

It is still uncertain whether PPRV continues to circulate in wildlife after an outbreak, and for how long, but one analysis of an ecosystem in the Serengeti seemed to be consistent with repeated introductions of the virus from domestic animals rather than maintenance in wildlife. This pattern would be similar to rinderpest, which persisted for a few years in wildlife but eventually disappeared.

Internet Resources

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