Peste des Petits Ruminants (PPR)

Author: Prof Moritz van Vuuren


Licensed under a Creative Commons Attribution license.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Due to similarities to other mucosal and diarrhoeal diseases, diagnosis is dependent upon the submission of specimens to a diagnostic laboratory. Specimens in the live animal can include ocular swabs, whole blood and serum, gum debris and lymph node aspirates. Necropsy material in formalin should include mesenteric lymph nodes, spleen, tonsil and third eyelid.

Diagnostic tests are available for detection of viral antigens, viral genome, virus-induced antibodies and viral isolation. Antigen detection can be accomplished with the aid of direct immunostaining, agar-gel immunodiffusion or immunocapture ELISA.

Detection of viral genome can be done by means of reverse transcription polymerase chain reaction assays. A variety of serological tests has been developed that include virus neutralization, competitive ELISA and the indirect fluorescent antibody test. Morbilliviruses are so closely related antigenically that serum raised against any 1 of them cross-reacts with all others in the genus in a variety of tests. Histopathological examination of tissues may reveal lympholytic lesions and inclusion bodies.

Clinical signs and pathology

An incubation period of 3-6 days following entry through the respiratory system is followed by duration of disease of 2 weeks or more.

The clinical signs are very similar to those that were associated with rinderpest and that are seen in dogs with canine distemper.
Conjunctivitis and nasal discharge in sand gazelles suffering from PPR

Hyperaemia and congestion of the conjunctiva

The first clinical sign is a rise in temperature that reaches 40 to 41 °C within two or three days. This is followed by depression, inappetence and development of serous nasal and ocular discharges that progressively become purulent and more profuse. The nasal discharge may block the nares and encrust the muzzle, causing the animal to snort and sneeze and the ocular discharge may mat the eyelids together.

Mucopurulent discharges from the eyes and nasal cavities

Severe purulent nasal exudate

One to two days after the start of pyrexia the buccal mucosa becomes congested and pale areas of necrosis become visible on the gums.
Erosions and ulcerations on the mucous membranes of the lips

Necrotic tissue accumulating on the buccal mucosa

In severe cases these increase in size and extend to involve the cheeks, dorsum of the tongue, hard palate and dental pad. The caseous epithelium easily sloughs, leaving shallow red erosions.

Accumulation of necrotic material and ulcerations on the dorsal surface of the tongue

Diarrhoea begins two or three days after the onset of pyrexia and death may occur some three to seven days later.
Less severe and mild cases also occur in which all or some of the signs develop to a lesser degree and the mortality rate is much lower.

Infected goats develop leukopenia, the degree of which may correlate with the severity of clinical disease. Eosinopenia, monocytosis and a rise in the packed cell volume, presumably due to haemoconcentration resulting from the diarrhoea, have also been reported.

The carcass is frequently emaciated, soiled with faeces, and the eyelids, nares and lips encrusted with discharges.

Erosions are found throughout the buccal cavity and pharynx, and, less frequently, the oesophagus.
Erosions and ulcerations on the soft palate in a sand gazelle

Necrosis and erosions on the side and at the base of the tongue in a sand gazelle

Erosions and ulcerations on the tongue and soft palate

As with rinderpest, erosive lesions in the forestomachs are often absent but may occasionally be found on the ruminal pillars and the leaves of the omentum. The abomasum and small intestine are usually dark red and congested rather than eroded and haemorrhages have been reported.
Marked congestion in the wall of the small intestine

Peyer's patches may be necrotic, and erosions and congestion can be found on the ileocaecal valve. Marked congestion of the apices of the longitudinal folds of mucosa (zebra striping) may be seen in the large intestine and rectum.

Congestion and haemorrhage in the intestines

The upper respiratory tract mucosa is usually congested, erosions in the nares may be present, and tracheitis has been described. The lungs may be partly or diffusely congested, the affected areas...
appearing red and firm due to interstitial pneumonia. Changes in the trachea and lungs are more severe if there is concurrent bacterial infection.

Peripheral and visceral lymph nodes may be oedematous and friable, occasionally enlarged or haemorrhagic. The spleen is often congested and firm. The female genital mucosa may be congested and show epithelial erosions.

Lymphoid tissues are depleted of lymphocytes with germinal centres particularly being affected. Pyknosis and karyorrhexis of lymphocytes are present in the cortex, and there is proliferation of reticuloendothelial cells along the medullary cords and in the sinuses. Comparable changes are seen in the Malpighian bodies of the spleen. Necrosis throughout the Peyer's patches has been described, although a mere reduction in the numbers of lymphocytes without necrosis may be all that is evident in this tissue.

Isolated areas of epithelial necrosis occur in the deep glands of the abomasum, and similar but more widespread changes are seen throughout the intestine together with atrophy of villi and accumulation of debris in the glandular crypts. The lamina propria is infiltrated with lymphocytes, macrophages and eosinophils.

The respiratory tract mucosa may have areas of necrosis and hyperplasia, and inclusion bodies in epithelial cells. Interstitial pneumonia characterized by the infiltration of lymphocytes and neutrophils, and proliferation of pneumocytes is common. A striking feature that was seen in rinderpest but is characteristic of measles, is severely enlarged multinucleate cells often containing intracytoplasmic and intranuclear viral inclusions in the alveoli and terminal bronchioles. It seems probable that the virus is responsible for a primary pneumonia that is readily superinfected with secondary pathogens.

**Laboratory confirmation**

A presumptive diagnosis of PPR can be made from clinical signs and lesions supported by epidemiological evidence such as the introduction of new and possibly diseased stock. In epidemic or
virgin areas the diagnosis must be confirmed in the laboratory, either by the detection of virus specific antigen or nucleic acid, or by the isolation of virus in cell culture. Demonstrating a rise in specific antibody in paired sera gives retrospective confirmation.

Specimens should be collected from up to ten acutely affected animals with fever and early mucosal lesions. Clotted blood and whole blood in anticoagulant, together with ocular, nasal and oral swabs should be collected from live animals. If possible two or three animals should be slaughtered and 20 to 30 grams each of spleen, lymph nodes, lung and gut mucosa also collected. The specimens should be chilled on ice, but not frozen, and despatched to the laboratory as soon as possible.

Viral antigens can be detected by immunohistochemical staining of tissues, and by monoclonal antibody-based enzyme-linked immunosorbent assay (ELISA) techniques.

Specific nucleic acid sequences can be detected by DNA probes and by polymerase chain reaction (PCR) techniques. The latter test has the advantage of producing large quantities of nucleic acid that can be sequenced to provide extra information about the lineage, or sub-type of PPRV involved. This information is particularly important for tracing the origin of epidemic outbreaks.

The virus is isolated by inoculating swab material, buffy coat or ten per cent tissue suspensions onto young monolayers of primary sheep or goat kidney cells. Vero cells, some lines of primary bovine kidney and bovine lymphoblasts are also sensitive. Cytopathic effects develop after four days and are characterized by rounding and shrinking of cells, strand formation and large flat syncytia. The identity of the virus is confirmed as PPRV by serological tests in which hyperimmune or convalescent sera are used.

If antigen or infectious virus cannot be detected, survivors should be bled two to four weeks after the first sampling and the paired sera assayed for antibody levels to the virus in neutralization tests. A four-fold or greater increase in titre confirms the presumptive diagnosis. It is essential to use the homologous virus since titres to heterologous virus may be too low to show a four-fold rise. Other techniques for detecting and quantifying specific antibody to PPRV that have been developed include haemagglutination inhibition and ELISA. Recent ELISAs take advantage of highly specific and pure antigens prepared by recombinant technology.

**Differential diagnosis**

PPR can be confused with several diseases that cause respiratory problems and mortality in small stock. To facilitate early detection and response in the event of introduction into a previously free country, it should be included in differential diagnostic lists for respiratory tract disease. Such a list includes mycotic stomatitis, bluetongue, epizootic haemorrhagic disease, pneumonia pasteurellosis, contagious caprine pleuropneumonia (not present in southern Africa), coccidiosis, sheep and goat pox (absent in southern Africa) and contagious pustural dermatitis (Orf).