Peste des Petits Ruminants (PPR)

Rayne Joseph, Geetha Sanal Kumar, Swapna Susan Abraham, M.C.Mohan

Dr. Rayne Joseph,
Dr. Geetha Sanal Kumar,
Dr. Swapna Susan Abraham
&
Dr. M.C.Mohan
Chief Disease Investigation Office, Palode

Peste des petits ruminants (PPR) is a contagious acute or sub acute viral disease of goats and sheep characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, and pneumonia. Goats are usually more severely affected than sheep.

Etiology

PPR is caused by a *Morbillivirus* from the Paramyxoviridae family. It is very closely related to RP virus antigenically. The genus also includes the Rinder Pest virus, Measles virus, Hedgehog distemper and Canine distemper virus. These are RNA viruses and the single RNA strand is tightly coiled in a helical nucleocapsid. These viruses are structurally, physically, chemically and antigenically very similar. Since these viruses are enveloped they are easily destroyed. Heat, desiccation, light, ultraviolet radiation, pH extremes and common disinfectants rapidly destroy these viruses.

The PPR virus and the RP virus have an affinity for lymphoid tissue and epithelial tissue of the gastrointestinal and respiratory tracts which results in their characteristic (and very similar) lesions

Epidemiology:

Goats and sheep are affected by PPR in the field, with goats appearing to be more susceptible. Captive wildlife killed by PPR include wild ungulates from three families:

- *Gazellinae* (*Dorcas gazelle)*
- *Caprinae* (*Nubian ibex and Laristan sheep*)
- *Hippotraginae* (*Gemsbok*)

Experimentally, the American white-tailed deer (*Odocoileus virginianus*) is fully susceptible. Cattle and pigs can be infected sub clinically, experimentally. Oral lesions observed in calves indicate that cattle may be a source of infection for sheep and goats. Such subclinical infections result in seroconversion, and cattle are protected from challenge with virulent RPV. Cattle have been considered dead end hosts for the disease; however, this has not been proven.

Transmission

Viral shedding occurs in ocular secretions, nasal secretions, saliva, urine and feces for approximately 7 days after fever is initiated. Transmission requires close contact. Inhalation of aerosols is the most common route of transmission of the disease. Nuzzling, licking, feed and water troughs can also be sources of infection.

Fomites such as bedding may also contribute to the onset of an outbreak. As in rinderpest (RP), there is no known carrier state. Infected animals may transmit the disease during the incubation period.

Incubation Period

PPR has an incubation period of 4 to 5 days.

Morbidity and Mortality

The incidence of PPR in an enzootic area may be similar to that of rinderpest (RP) in that a low rate of infection exists continuously. Such epizootics may be characterized by almost 100 percent mortality among affected goat and sheep populations.

The prognosis of acute PPR is usually poor. The severity of the disease and outcome in the individual is correlated with the extent of mouth lesions. Prognosis is good in cases where the lesions resolve within 2 to 3 days. It is poor when extensive necrosis and secondary bacterial infections result in an unpleasant, fetid odor from the animal's breath. Respiratory involvement is also a poor prognostic sign. A morbidity rate of 80-90 percent and a casefatality rate of 50-80 percent are not uncommon — particularly in goats.

Young animals (4 to 8 months) have more severe disease, and morbidity and mortality are higher. PPR is less severe in sheep than in goats. Poor nutritional status, stress of movement, and concurrent parasitic and bacterial infections enhance the severity of clinical signs.

Clinical Signs

The disease usually appears in the acute form, with an incubation period of 4 to 5 days followed by a sudden rise in body temperature to 104-106° F (40-41° C). The temperature usually remains high for about 5 to 8 days before slowly returning to normal. Affected animals appear ill and restless and have a dull coat, dry muzzle, and depressed appetite. From the onset of fever, most animals have a serous occulo nasal discharge, which progressively becomes mucopurulent. The discharge may remain slight or may progress, resulting in a profuse catarrhal exudate, which crusts over and occludes the nostrils. At this stage, animals have respiratory distress, and there is much sneezing in an attempt to clear the nose.

Small areas of necrosis may be seen on the visible nasal mucous membranes. The conjunctiva usually becomes congested. As with the nose, there may be profuse catarrhal conjunctivitis resulting in matting of the eyelids.

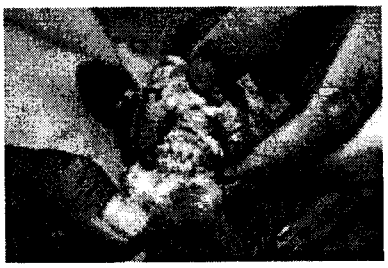
Necrotic stomatitis is common. It starts as small, roughened, red, superficial necrotic foci on the gum below the incisor teeth. These areas may resolve within 48 hours or progressively increase to involve the dental pad, the hard palate, cheeks and their papillae, and the dorsum of the anterior part of the tongue.

Early mouth lesions

Necrosis may result in shallow irregular nonhemorrhagic erosions in the affected areas of the mouth and deep fissures on the tongue. There may be excessive salivation but not to the extent of drooling.

Mouth lesions in later stages

At the height of development of oral lesions, most animals manifest severe diarrhea, often profuse but not hemorrhagic. As it progresses, there is severe dehydration, emaciation, and dyspnea followed by hypothermia, and death usually occurs after a course of 5 to 10 days. Bronchopneumonia, evidenced by coughing, is a common feature in the later stages of PPR. Pregnant animals may abort. Secondary latent infections may be activated and complicate the clinical picture.



Gross Lesions

PPR causes severe inflammatory and necrotic lesions in the mouth and in the gastrointestinal tract. Unlike RP, there is also a definite, albeit inconstant, respiratory system component; hence, the synonym stomatitis-pneumoenteritis complex.

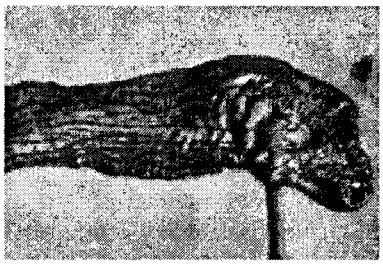
Emaciation, conjunctivitis, erosive stomatitis involving the inside of the lower lip and adjacent gum, cheeks near the commissures, and the free portion of the tongue are frequent lesions.

In severe cases, lesions may also be found on the hard palate, pharynx, and upper third of the esophagus. The abomasum is a common site of regularly outlined erosions and often oozes blood.

Lesions in the small intestine are generally moderate, being limited to small streaks of hemorrhages and, sometimes, erosions in the first portion of the duodenum and the terminal ileum. Payer's patches are the sites of

extensive necrosis, which may result in severe ulceration. The large intestine is usually more severely affected with congestion around the ileocecal valve and in the rectum. In the posterior part of the colon and the rectum, discontinuous streaks of congestion known as "zebra stripes" can be found on the mucosal folds.

Zebra stripes in Intestine



In the respiratory system, small erosions and petechiae may be visible on the nasal mucosa, turbinates, larynx, and trachea. Bronchopneumonia may be present, usually confined to the anteroventral areas and is characterized by consolidation and atelectasis.

Pneumonia complicated by Pasteurella

There may be pleuritis, which may become exudative and results in hydrothorax. The spleen may be slightly enlarged and congested. Most lymph nodes throughout the body are enlarged, congested, and edematous. Erosive vulvovaginitis similar to the lesions in the oral mucocutaneous junction may be present.

epizootiological findings.

Diagnosis In the field, a diagnosis can be made on the basis of clinical, pathological, and Laboratory confirmation is an absolute requirement — particularly in areas or countries where PPR has not previously been reported.

Specimens for Laboratory

- Specimens to be collected for diagnosis include
- Blood in EDTA anticoagulant
- Clotted blood or paired serum
- Mesenteric lymph nodes, spleen, lung, tonsils, and sections of the ileum and large intestine.
- Swabs of serous nasal and lachrymal discharges may also be useful.

NOTE: All samples should be shipped fresh (not frozen) on ice within 12 hours after collection. The above samples should be collected in the acute phase of the disease. Ideally, samples should be collected from several animals in an outbreak.

Laboratory Diagnosis

A wide range of laboratory procedures have been described for detecting virus or viral antigen, viral nucleic acid, and antibody.

- Agar gel immunodiffusion
- Counter immuno electrophoresis
- Indirect fluorescent antibody test
- Competitive ELISA
- Immunohistopathology

Differential Diagnosis

1. Rinderpest.
2. Pasteurellosis.
3. Contagious caprine pleuropneumonia.
4. Bluetongue.
5. Heartwater.
6. Contagious ecthyma (contagious pustular dermatitis, orf).
7. Foot-and-mouth disease.
8. Nairobi sheep disease
9. Coccidiosis.
10. Plant or mineral poisoning

Treatment

There is no specific treatment for PPR. However, drugs that control bacterial and parasitic complications may decrease mortality.

Vaccination

PPR vaccine at a dose of 1ml per animal subcutaneously protects goats for at least 12 months against PPR.

Control and Eradication

Eradication is recommended when PPR appears in new areas. Methods that have been successfully applied for RP eradication in many areas would be appropriate for PPR. These include

- Quarantine
- Slaughter
- Proper disposal of carcasses and contact fomites
- Decontamination of facilities and equipment

❖ Restrictions on importation of sheep and goats from infected areas

PPR can be financially a very "costly" disease, particularly in cases of epidemics, but also in endemic areas. As a result the method of control and prevention is very important.

Public Health

Peste des petits ruminants is not infectious for humans.

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from the farmers side the most possible reasons for not being able to carry on the recommended buffalo management practices are-

a)lack of awareness b)lack of financial resources c)lack of adequate services from government.

Number of prevailing and emerging diseases are the main health barriers causing impact t on buffalo poduction.Most of the diseases recorded in cattle are also recorded in buffaloes. However,there are differences in clinico-epidemiological profile, particularly in relation to severity and manifestations.

Opportunities

Now a days there is increasing reliance on crop byproducts for dairy production. Buffalo is the best choice in this condition compared to crossbred cattle. In recent years, there is a occurrence of draught which has decreased the reliability of crop production. So people are shifting towards animal husbandry which provides employment and income throughout the year. In such cases, buffalo rearing is the best option among AH activities. Not only in rural areas, Buffaloes can also be made popular in urban areas through its unique products. Because of difference in composition and physiochemical properties compared to cow milk, buffalo milk has cretin processing advantages.It is preferred for consumption as fluid milk, as tea/coffee whitener and for making dairy products because of high fat content. As they produce amount of milk without intensive feeding and their resistance to diseases like mastitias,minimizes the need for antibiotic treatment. So , buffalo can be focused as the producer of "organic milk". Carabeef can bring valuable foreign exchange as there is no report of BSE and less incidence of FMD.

To harness all the above opportunities, some shortcomings are to be rectified like—

❖ Improvement in the current available technologies for semen freezing and also in the adoption of AI for buffaloes by the farmers.

❖ The comparative efficiency of digestion in cattle and buffaloes in relation to different feed resources need further investigation.

❖ The economic and technical feasibility of modern techniques should be further studied.

❖ Need to identify more promising breeds/ strains(production. reproduction and adoption) by determining intra and inter allelic genetic group differences.

❖ Need to develop systems for producing superior male germplasm for milk(Progeny testing,ONBS with MOET).

❖ Need to develop crossbreds(Godavari and Mehsana) by combining good characters of two or more breeds.

Threats

Germplasm loss is a major threat to buffalo production system. This occurs mainly due to mortality in young stock, slaughter of adults at the end of lactation period. The high producing she buffaloes can be transferred to the nearby rural areas for subsequent breeding. Transport cost and good salvager value from slaughter stops middlemen from sending them back to rural areas. So there is an urgent need to enact a legislation banning slaughter at least up to the age of 10 years. Germplasm loss can be avoided by collecting ovaries after slaughter,oocyte maturation and IVF and transplantiong such embryos in low producing surrogate mothers.

In the export of carabeef, FMD could act as hindrance. So it is necessary to take steps for prevention and lower incidence of FMD.

To conclude, there is a need for a new approach which is farmer centred,pro-poor and need based with due importance to sociocultural and gender aspects.

In the Indian context, the greatest virtues of the buffalo are its relatively resistance to common tropical diseases, ability to thrive on poor quality crop byproducts, and still yield 2-5 liters of milk that is truly a nature's gift to mankind.So,the buffalo can be aptly called as Cinderella of Indian dairying.