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Peste des Petits Ruminants (PPR): A Devastating Goat Disease

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Peste des petits ruminants (PPR) is caused by a Morbillivirus that belongs to the family Paramyxoviridae. Its name derived from French for "disastrous disease of small ruminants".PPR is an acute, highly contagious and fatal disease primarily affecting goats and sheep, whereas cattle undergo sub-clinical infection.PPR is classified as an OIE (Office International des Epizooties)-listed disease.

Considering the importance of sheep and goats in the livelihood of the poor and marginal farmers in Africa and South Asia, PPR is an important concern for food security and poverty alleviation. Clinically, the disease resembles rinderpest (RP) in cattle and is characterized by high fever (pyrexia), conjunctivitis, oculo-nasal discharges, necrotizing and erosive stomatitis, diarrhea and bronchopneumonia followed by either death of the animal or recovery from the disease. Pregnant animals may abort. The incubation period of the disease is 2–7 days. Death usually occurs 4–6 days after the onset of fever. PPRV infection leads to high morbidity (up to 100 %) and up to 90 % mortality.

Synonyms

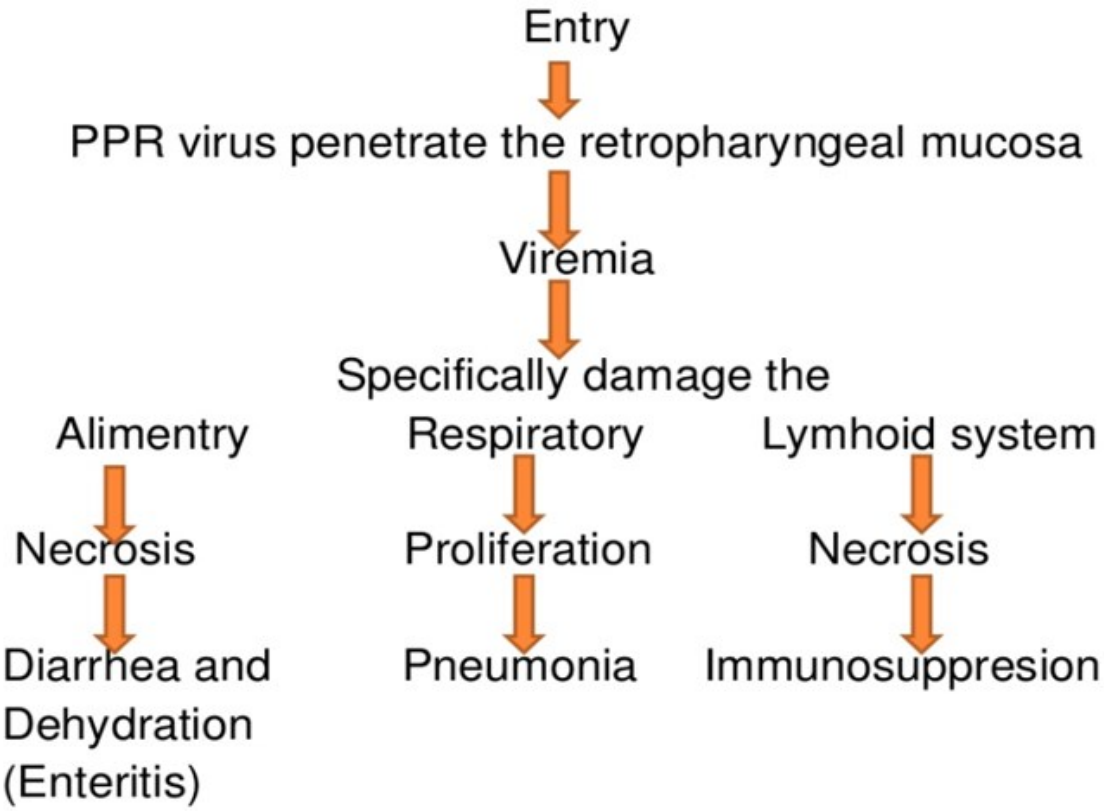
The disease used to be called 'Kata', 'Goat plaque', 'Psuedorinderpest', 'Pneumoenteritis complex' and 'Stomatitis pneumoenteritis syndrome'.

Epidemiology

The disease is currently endemic in most of Africa, the Middle East, South Asia, and China. As one of the largest sheep (71.5 million) and goat (140.5 million) rearing countries in the world, India considers PPR as one of the major and priority of livestock diseases. Though there is no evidence of the first appearance of PPR in India, its presence was first reported in 1987. The first confirmed outbreak of PPR in sheep with 25 % mortality was reported in Arasur village, Villupuram district of Tamil Nadu during 1987, where characteristic clinical signs of PPR were noticed.

Economic Consequences

PPR is an OIE (Office International des Epizooties)-listed disease. Sheep and particularly the goats (also



known as poor man's cow) contribute significantly to the nutrition and cash income of small farmers in Africa and South Asia, the two regions with the largest concentration (about 72.90%) of the poor peoples in the world. According to the FAO estimates, the morbidity, mortality, production losses, and treatment cost of PPR altogether are likely to cause an economic loss of $2,972.5 million/year during 2012-2017 in the SAARC region among which, in India alone, it would be $2569.00 million/year.

Etiology

The causative agent, PPR virus (PPRV) is an enveloped RNA virus belongs to the genus Morbillivirus of the family Paramyxoviridae (subfamily Paramyxovirinae) under the order Mononegavirales with other members of the genus, which include rinderpest virus (RPV), measles virus (MV), canine distemper virus (CDV), phocine distemper virus (PDV) and dolphin and porpoise morbillivirus(DMV). The virus is a pleomorphic particle with a lipoprotein membrane enveloping a ribonucleoprotein core. The genome is a negative sense single stranded-RNA, approximately 16 Kilo bases (kb) long with negative polarity. The PPRV is genetically grouped into four lineages (I, II, III, and IV) based on the F and N gene sequences analyses. Lineages I–III circulate in Africa, while lineage IV is generally found in Asia. Till now, only lineage IV viruses have been reported in India. Three PPRV isolated from India of lineage IV that includes (PPRV/ Sungri/96, PPRV/Arasur/87,

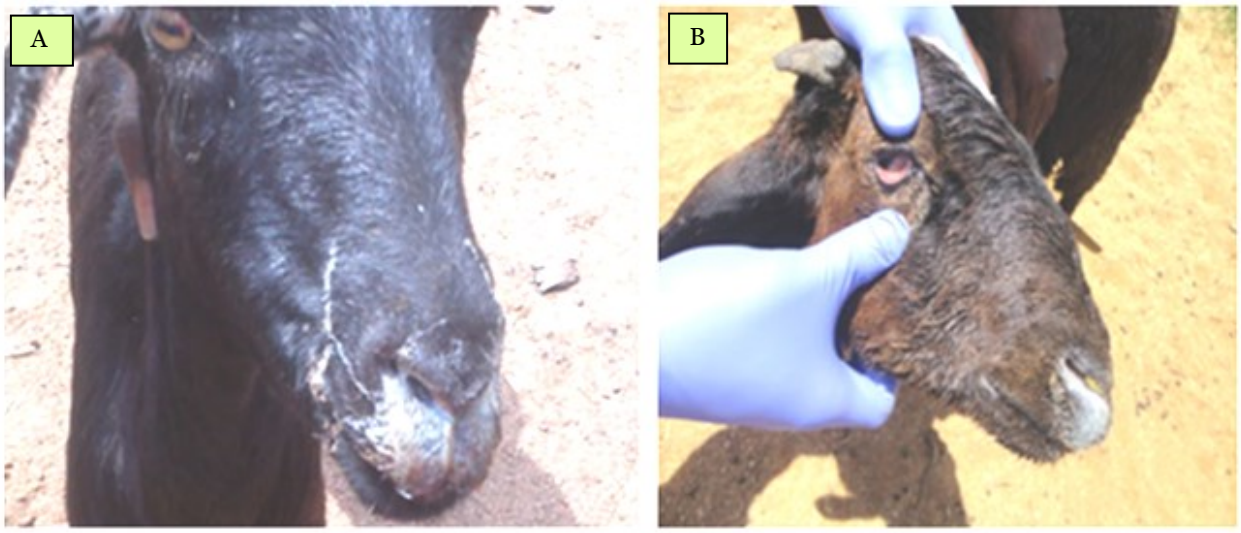


Fig.1 (**A**) PPR virus-infected goat showing nasal discharge, (**B**) congestion of conjunctiva (Balamurugan *et al*., 2014).

and PPRV/Coimbatore/97) (Palaniswami et al., 2005).

Pathogenesis

PPRV is both lymph- and epithelio-tropic and infection usually result in conjunctivitis, rhinotracheitis, ulcerative stomatitis, gastroenteritis, and pneumonia.

Clinical sign:

The clinical signs, pathogenesis, and lesions of the disease in sheep and goats, in general, are similar to those of rinderpest except that the disease is more acute in onset, especially in goats, and follows a more rapid course. Another difference is the marked involvement of the respiratory tract; affected animals have dyspnea, hyperpnea, and cough. There is also a marked serous to mucopurulent nasal and ocular discharge. Erosion/ ulceration of the oral and pharyngeal epithelium may be diffuse and pseudomembranes are characteristically observed in the oral cavity. The different stages of the disease are (i) incubation period, (ii) Prodromal phase (febrile), (iii) mucosal phase (ocular and nasal discharges, hyperemia of conjunctiva and mucosa of anterior nares, and erosions on the tongue, palate, lips (iv) diarrhoeal stage and (v) in non-fatal cases, 'recovery stage' in which, sheep and goats that recover from PPR develop active lifelong immunity.

Gross Pathology

The pathology of PPR is characterized and dominated by retrogressive and necrotic changes in lymphoid tissues and epithelial cells of gastrointestinal and respiratory systems. The prominent lesions include, consolidation, changes in the colour of lungs and sometimes, frothy mucus is observed in cut pieces of a lung on squeezing, anteroventral areas of right lung are frequently involved; areas of lungs become dark red or purple, firm to touch mainly in the anterior and cardiac lobe. Secondary bacterial pneumonia is

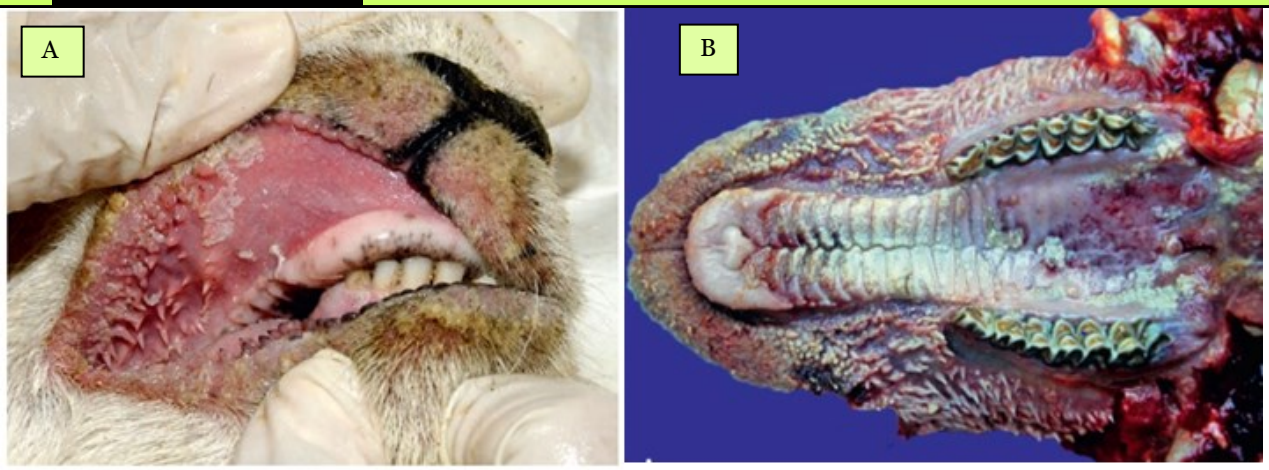


Fig.2 (A) Oral ulceration and fibronecrotic inflammation and (**B**) Ulceration and fibrinonecrotic pseudomembrane on the oral mucosa of a sheep (Jubb, Kennedy & Palmer 6th edition)

common. The congested alveolar border is found to be one of the most characteristic clinical and pathological changes of PPR in goats. Bronchopneumonia is a constant lesion, with the possibility of pleuritis and hydrothorax (Sreenivasa *et al*., 2000).Lymph nodes associated with lung (mediastinal) and intestine (mesenteric) are most commonly affected which are generally enlarged, oedematous and congested. Necrotic or hemorrhagic enteritis or congestion around the ileocaecal valve, at the caeco-colic junction and in the rectum are seen usually. In the posterior part of the colon and rectum, discontinuous streaks of congestion ("Zebra stripes" or "Zebra markings") on the mucosal folds are observed, which are typical of PPR.

Histopathology

Microscopically the prominent lesions in the palatine tonsils, which included necrosis of surface and crypt epithelium with infiltration of neutrophils, the formation of syncytial cells and scattered intranuclear inclusion bodies. Lymphocyte depletion (primarily in the cortical lymphatic nodules) and numerous multinucleated syncytial cells and apoptotic cells. In the spleen, the white pulp areas depleted of lymphocytes and the red pulp appears hypercellular. Intestinal lesions are most severe and observed within the duodenum, jejunum, and ileum,

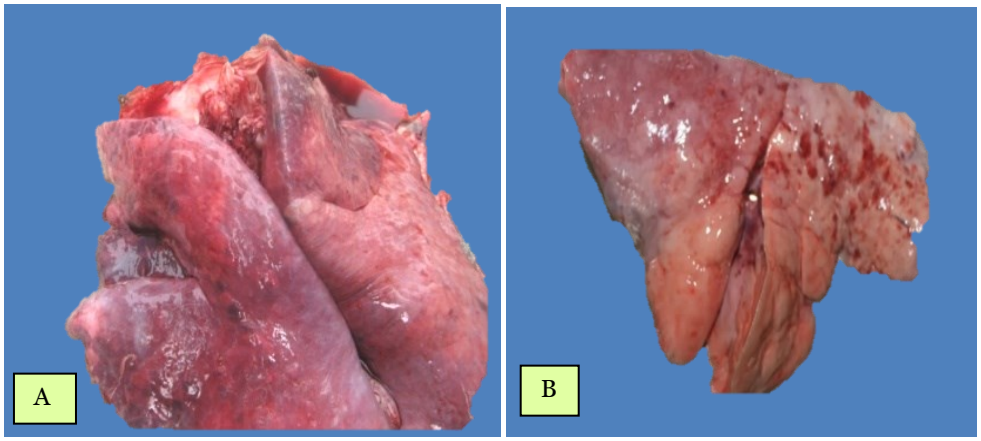


Fig.3. Lungs showing apical and cranial lobe consolidation which extend up to diaphragmatic lobe and note the presence of hemorrhage (**A&B**).

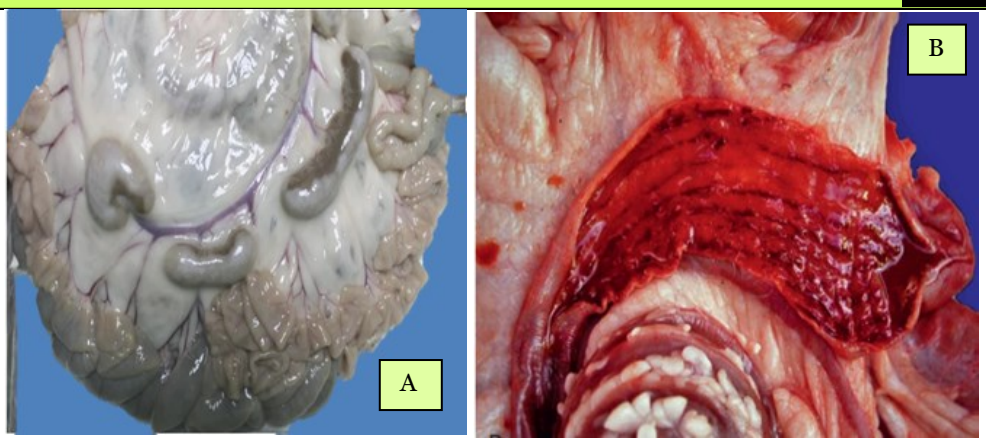


Fig.4. (**A**) Intestinal mucosa showing congestion with enlarged and hemorrhagic mesenteric lymph nodes and (**B**) Colonic mucosa revealed hemorrhagic streaks (Zebra stripes) over mucosal fold (Bamouh *et al*., 2019)

with the ileum showing the most severe changes characterized by blunted villi, degeneration of surface and crypt epithelial cells, expansion of lamina propria by a primarily mononuclear infiltration with scattered syncytial cells and severe depletion of lymphocytes within Peyer's patches. In the lungs, severe bronchointerstitial pneumonia, observed in the cranial and middle lobes. Multifocal suppurative and necrotizing bronchiolitis, with variable epithelial attenuation to hyperplasia and occasional intracytoplasmic inclusion bodies. The alveolar walls are expanded by inflammatory cells and hyperplastic type II pneumocytes. Multifocal consolidation with infiltrates of mixed inflammatory cells also present.

Diagnosis

PPRV is routinely diagnosed based on case history, clinical signs, gross and histopathological findings but more accurately by using molecular techniques like Immunocapture ELISA (ICE), Cell culture in Vero cell line (Diallo *et al*. 1989), Transcription polymerase chain reaction (RT-PCR) (Sannat *et al*., 2014), Loop-Mediated Isothermal Amplification (LAMP) Assay (Li *et al*., 2010).

Differential diagnosis

Rinderpest, contagious caprine pleuropneumonia (CCPP), bluetongue, Pasteurellosis, contagious ecthyma, foot and mouth disease (FMD), heartwater, coccidiosis, and Nairobi sheep disease and have similar outcomes.

Treatment

Since PPR is a viral disease, there is no specific treatment for this disease. However, treatment of affected animals by administration of antibiotics (long-acting oxytetracycline, chlortetracycline) to prevent secondary bacterial infections and antidiarrhoeal medicines has been practiced with supportive therapy (B-complex and Dextrose saline) for 5–7 days, which may be useful to reduce the severity of the disease. Treatment and management of clinical cases of PPR or in the event of outbreaks in sheep and goats are necessary to minimize the economic losses to farmers.

Vaccination

The only way to control PPR is by vaccination. For the prevention of PPR, OIE since 1972 recommended the use of the Tissue culture rinderpest

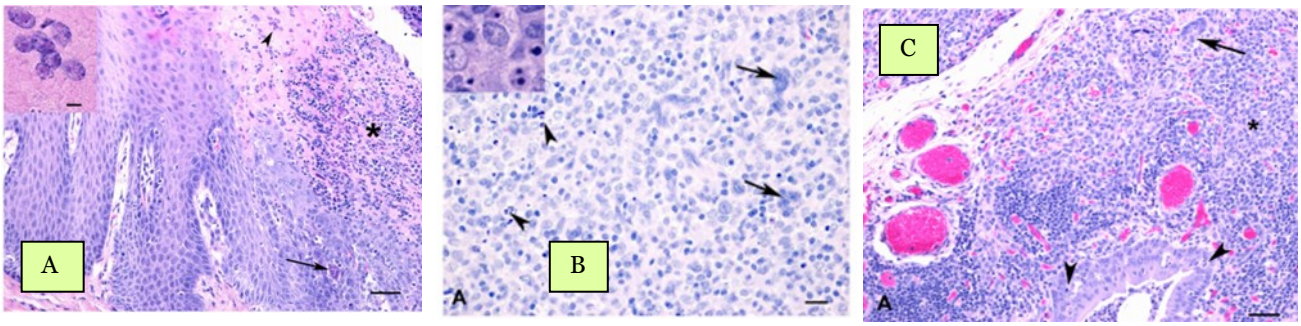


Fig 5.(A) There is necrosis of surface epithelium and extensive neutrophilic infiltrate (*) as well as occasional syncytial cells (arrow) and intranuclear inclusion bodies in upper epithelial layers (arrowhead) (H&E, X200). **(B)** Lymph node revealed Multinucleated syncytial cells (arrows) and degenerating or apoptotic lymphocytes (arrowheads) were observed. Inset: Higher magnification showing detail of apoptotic lymphocytes (H&E, X200). **(C)** Hyperplasia of bronchiolar epithelium is evident with scattered epithelial degeneration (arrowheads) and abundant neutrophils within the lumen. Surrounding parenchyma is consolidated (*) with severe infiltration of mononuclear inflammatory cells. Note large syncytial cell (arrow) (H&E, X100).

vaccine. It was successfully used to control PPR in West African and other African countries. The first homologous PPR vaccine was developed using live attenuated Nigerian strain PPRV Nig 75/1 after 63 passages in Vero cells produced a solid immunity for 3 years. Nowadays recombinant vaccine also developed to control PPR.PPRV/Sungri/96 strain (Sreenivasa *et al*., 2000) is the only vaccine virus strain used in India under mass PPR vaccination campaigns as all the commercial manufacturers in India, both at private and public sector, are currently using only this Lineage IV virus. The recommended age for a vaccination with this vaccine has been estimated to be 5 months and above as the maternal antibodies from vaccinated dams wane by about 4 months (Balamurugan *et al*., 2012). Single-dose immunization protocol is sufficient for the protection of small ruminants.

Prevention and control

For the proper control of PPR, there is a need for strong support of diagnostic methods and proper, timely vaccination of the susceptible population. Hence, the availability of attenuated cell culture vaccine and various diagnostic techniques/kits for the diagnostic of PPR favors strong recommendations put forward for the control program. The control of PPR can be ensured only through the implementation of effective prophylactic measures. All the sheep and goats of the affected flock should be under quarantine for at least 1 month after the last clinical case. Animal movements have to be strictly controlled in the area of the infection. The only effective way to control PPR is by mass vaccination of the animals and quarantine measures and the use of an effective vaccine against PPR is the only solution to control the disease effectively.

Some of the other control measures (sanitary prophylaxis) are strict quarantine and control of animal movements, quarantine of newly purchased or newly arriving goats/sheep for at least 2–3 weeks and know the health

status and the source of any new animal (s) brought into the flock, migratory flocks are a threat to local sheep and goat, therefore, contact may be avoided, effective cleaning and disinfection of contaminated areas of all premises with lipid solvent solutions of high or low pH and disinfectants including physical perimeters, equipment, and clothing, dead animal/carcasses should be burnt/buried deeply, monitor animals closely and frequently for any developing illness or signs of disease, isolate any sick animals from the flock and contact the Veterinarian immediately to examine sick animals in the herd/flock, use separate facilities and staff to handle isolated animals, educate and train the employees about PPR and the signs of illness and monitoring of wild and captive animals, especially in contact with sheep and goats.

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