

Original Research Article

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## Pathomorphological Studies of *Peste Des Petits Ruminants* (PPR) in Goats of Navsari and Valsad Districts, India

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### ABSTRACT

#### Keywords

C- ELISA,  
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#### Article Info

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During the seroprevalence study of PPR a total 210 serum samples were collected from goats of Navsari and Valsad districts of South Gujarat. Out of them 99 samples were found positive for PPRV antibody by C- ELISA kit and these PPR positive goats put under continuous observations. Out of these 99 PPR positive goats, 15 goats were died which were consider as a suspected death due to PPR disease. Post mortem was performed in death animals and gross lesions were noted as well as tissue samples were collected in 10% neutral buffer formalin for histological study. Tissue samples were processed, slide were prepared and subjected to histological examination. Characteristics microscopic lesions like syncytial cell formation, infiltration of mononuclear cells and necrosis were observed in lung, intestine, spleen, kidney and liver tissue.

### Introduction

*Peste des petits ruminants* (PPR) is an acute febrile viral disease of goat and sheep caused by RNA virus belonging to the genus *Morbillivirus* of *Paramyxoviridae* family (Gibbs *et al.*, 1979). PPR is also known as kata, stomatitis- pneumoentritis complex, goat plaque and pseudo - rinderpest (Jubb *et al.*, 2007). The disease is highly contagious causing varying degree of morbidity and mortality in susceptible animals (Radostits *et al.*, 2007).

PPR virus is normally transmitted by aerosol route, but may also spread through direct contact by means of contaminated water/feed. Substantial quantities of virus are found in ocular, nasal or oral secretions in early stage of sick goats and in the faeces in late stage of disease (Abegunde and Abu, 1977). PPRV was serologically confirmed from Gujarat by Hinshu *et al.*, (2001) followed by many researchers (Tiwari, 2004, Kanani *et al.*, 2006, Nagraj, 2006, Balamurugan *et al.*, 2011

and Chandrahas *et al.*, 2011). Recently, an outbreak of PPR was reported in Navsari and Valsad district of Gujarat (Sharma *et al.*, 2015 and Thakor *et al.*, 2016). Hence the present study was decided for pathomorphological study as well as use of latest techniques for diagnosis and detection of PPR antigen in-situ condition.

## Materials and Methods

For pathomorphological study only those animals were taken into consideration which represented PPR infection in flock according to C- ELISA result. Out of 210 serum samples 99 samples were shown positivity for PPR antibody in C- ELISA. Out of these 99 PPR infected animals, 15 animals were dead and these dead animals were subjected to Post mortem examination. During postmortem examination gross pathological lesions were recorded if any. After recording the gross lesions the tissues from various organs (e.g. lung, liver, intestine, spleen, kidney, heart and trachea) were collected and subsequently preserved in 10% neutral buffered formalin for histopathological examination.

Following fixation, tissue samples were processed for histopathology by paraffin embedding technique as per standard protocol (Luna, 1968). The blocks of tissue were cut in to section of four to six microns thickness, the sections were stained by Harri's Haematoxylin and Eosin staining method (Culling, 1969).

## Results and Discussion

Gross examination of respiratory tract revealed frothy exudates and haemorrhages in tracheal mucosa (Figure 1). Varying degree of congestion, haemorrhages, red hepatization, emphysema and consolidation was observed in right lobe of lung (Figure 2) along with firm consistency. Whereas in intestine,

hemorrhagic strips were found on mucosal surface of caecum (Figure 3) with raised nodules. Spleen revealed characteristics lesions of splenomegaly and edematous. Additionally, liver revealed haemorrhages on the surface, presence of serosanguinous fluid was also noticed in pericardial sac of heart (Figure 4) and minor haemorrhages on cortico- medullary junction of kidney.

The morbid pathomorphological lesions in lungs, trachea, caecum, spleen, liver, heart and kidneys observed in this study were partly in agreement with previous report made by El-Yuguda *et al.*, (2009), Al-Naeem *et al.*, (2009), Zahur *et al.*, (2009), Abd El-Rahim *et al.*, (2010), Chauhan *et al.*, (2011), Jagtap *et al.*, (2012), Rosemary *et al.*, (2013), Patel *et al.*, (2015) and Ekambaram *et al.*, (2015) in sheep and goat. The pneumonic lungs and hemorrhagic trachea probably due to descending upper respiratory infection of PPR complicated by secondary bacterial infection (Rosemary *et al.*, 2013). Moreover, congestion and red hepatization of lung were probably suggestive of PPRV involvement (Kumar *et al.*, 2004).

Most prominent histological changes were observed in lung, intestine, kidney, liver, spleen, lymph node and heart. Histopathological examination of lungs revealed alveolar dilatation, emphysema, thickening of alveolar septa, infiltration of mononuclear cells (Figure 5), eosinophilic intracytoplasmic inclusion body and interstitial pneumonia along with pink colour serous exudates. Presence of exudate in bronchioles, necrosis and shedding of bronchial epithelium (Figure 6) was also noted in present study. In trachea, degeneration and cystic lesions in tracheal gland (Figure 7) and infiltration of mononuclear inflammatory cells in tracheal sub-mucosa were observed. In intestine, necrosis of epithelium/crypts, haemorrhages

and infiltration of mononuclear cells into submucosa with sever loss of intestinal villi were common findings (Figure 8 and 9). Spleen and lymph node revealed haemorrhages and depletion of lymphoid cell in cortex and medullary cords. Liver shows lesions of degeneration, sinusoidal congestion and infiltration of mononuclear cells in the sinusoidal space along with syncytial cell.

Whereas in kidney, degenerative changes accompanied by desquamation of epithelium lining of tubules, haemorrhages, mild glomerular atrophy and interstitial nephritis (Figure 10). While mild pathological changes were observed in heart like congestion, haemorrhages, degenerative changes, infiltration of mononuclear cells and focal area of necrosis.



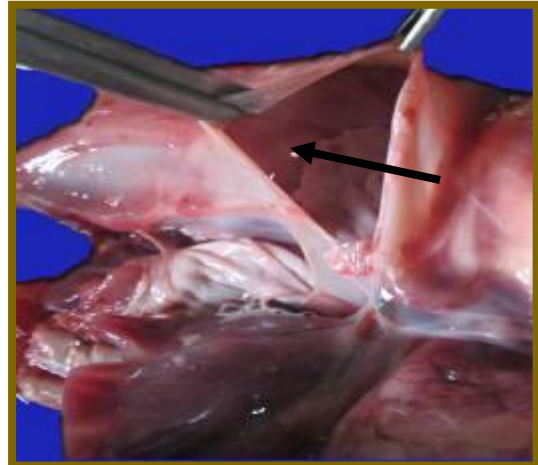
**Fig.1** Frothy exudates and hemorrhages in trachea



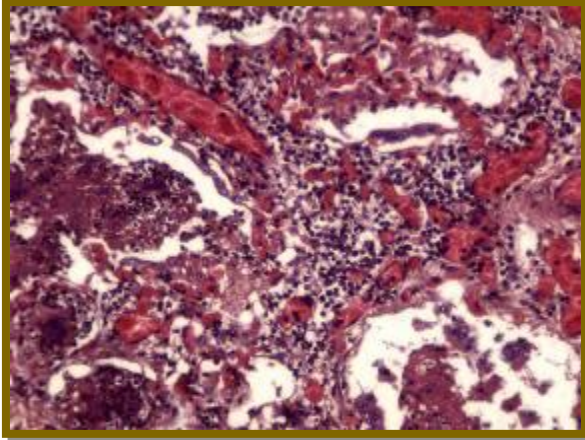
**Fig.2** Congestion, hemorrhages, red hepatization and consolidation in right lobe of lung



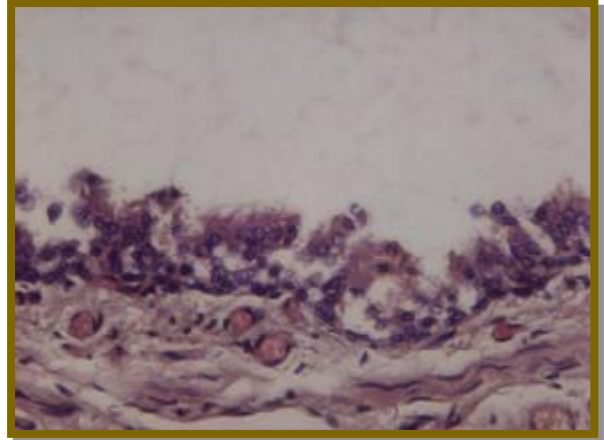
**Fig.3** Hemorrhagic strips in mucosal surface of caecum



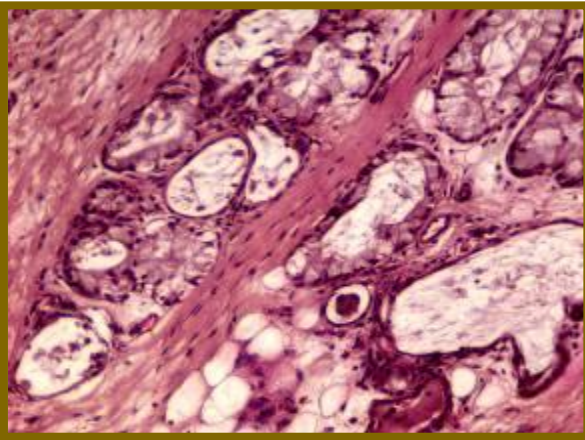
**Fig.4** Presence of serosanguinous fluid (arrow) in pericardial sac of heart



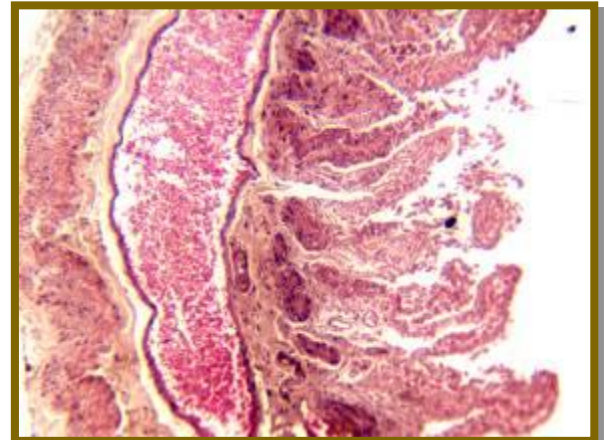
**Fig.5** Lung, infiltration of mononuclear inflammatory cells in inter alveolar septa and alveoli lumen along with alveoli capillary congestion (H and E x 20)



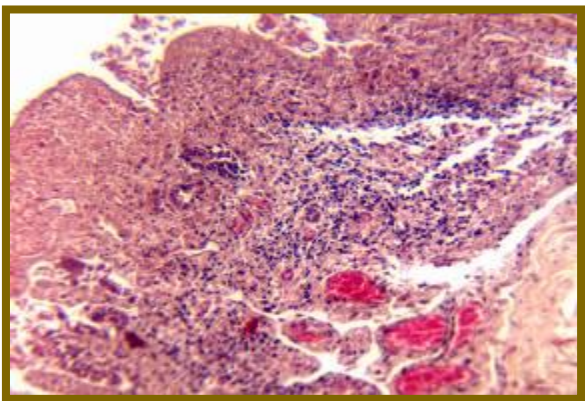
**Fig.6** Lung, necrosis and shedding of bronchial epithelium (H and E x 20)



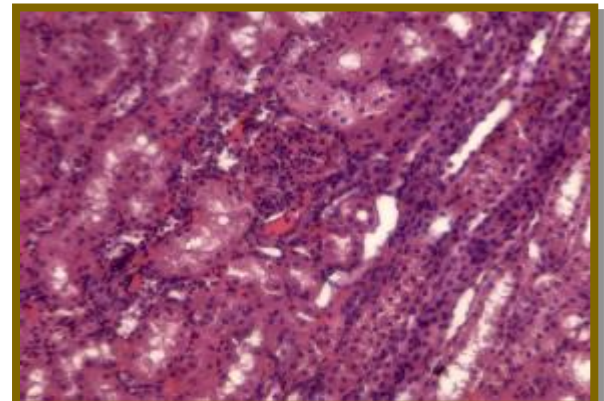
**Fig.7** Trachea, degeneration and cystic lesions in tracheal gland (H and E x 20)



**Fig.8** Intestine, hemorrhages in submucosa, blunting, necrosis and sloughing of intestine villi (H and E x 20)



**Fig.9** Intestine, infiltration of mononuclear inflammatory cells in submucosa of intestine (H and E x 20)



**Fig.10** Kidney, infiltration of mononuclear inflammatory cells in interstitial space (H and E x 20)

Histopathological examination of lungs revealed alveolar dilatation, emphysema and infiltration of mononuclear cells along with pink colour serous exudate which was shown similarity with the earlier observations of various researchers (Aktas *et al.*, 2011; Jagtap *et al.*, 2012; Patel *et al.*, 2015 and Maina *et al.*, 2015). Presence of syncytial cells and eosinophilic intracytoplasmic inclusion body in bronchial epithelium cells was supported by the earlier reports of Brown *et al.*, (1991), Islam *et al.*, (2001), Kumar *et al.*, (2004), Toplu (2004), Kul *et al.*, (2007) and Saglam and Temur (2009). Though interstitial pneumonia was evident and partially agreement with the findings of Kumar *et al.*, (2004) and Kul *et al.*, (2007). Presence of exudate in bronchi and hyperplasia of bronchial epithelium was noted in present study which was also same as the findings of Aktas *et al.*, (2011) and Maina *et al.*, (2015).

Necrosis of epithelium/crypts in intestine was also found by Kumar *et al.*, (2004), Aktas *et al.*, (2011) and Patel *et al.*, (2015). Whereas haemorrhages and infiltration of mononuclear cells into submucosa were partially agreement with the report of Maina *et al.*, (2015).

Loss of intestinal villi might be due to the epitheliotropic nature of PPR virus (Maina *et al.*, 2015) may lead to necrosis of epithelium cells of villi.

According to present and earlier reports of Islam *et al.*, (2001), Kul *et al.*, (2007), Maina *et al.*, (2015) and Patel *et al.*, (2015) lymphoid cell depletion was observed in cortex and medullary cords of spleen and lymph node. It might be due to the severe lymphocytolysis in lymphoid tissues by PPR virus (Kul *et al.*, 2007). In agreement with the present findings Aktas *et al.*, (2011) and Madboli and Ali (2012) also reported degeneration and infiltration of mononuclear cells in the sinusoidal space in liver.

Histopathological lesions of kidney included degenerative changes, desquamation of epithelium lining of tubules, interstitial nephritis and mild glomerular atrophy were partly in accordance with the observations of Jagtap *et al.*, (2012) and Patel *et al.*, (2015). Further, in present study histopathological changes of heart included congestion, haemorrhages, degenerative changes and focal area of necrosis were supported by the observation of Jagtap *et al.*, (2012). Although the type of lesions was more or less similar in all the animals, there was variations in the severity and involvement of the organs.

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