AN OUTBREAK OF *PESTE DES PETITS RUMINANTS* IN GOATS AT DISTRICT LAHORE

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ABSTRACT

Peste des petits ruminants (PPR) is a highly contagious and economically important disease of small ruminants caused by a virus in the genus morbillivirus, family paramyxoviridae. This infection is responsible for high morbidity and mortality. In this study, an outbreak of PPR in goats was investigated in a well organized farm at district Lahore. Clinical signs, gross lesions and histopathological findings led to the suspicious of outbreak of PPR. Mesenteric lymph nodes and spleen samples collected from the dead animals were found positive for the PPR antigen by agar gel immunodiffusion (AGID) against hyperimmune anti-PPRV serum. Moreover, younger animals were more susceptible to PPR than adults.

Keywords: Peste des petits ruminants, PPR, agar gel immunodiffusion, hyperimmune anti-PPRV serum.

INTRODUCTION

Peste des petits ruminants (PPR) is OIE (Office International des Epizooties) notifiable disease of small ruminants caused by morbillivirus within the family paramyxoviridae. The disease occurs in goats and less often in sheep. The first clinical description of PPR was made in 1942 in West Africa and characterized by necrotic and erosive stomatitis, enteritis and pneumonia (Ismail *et al.,* 1995). Virus was initially isolated in Senegal in 1962 (Gilbert and Monnier, 1962). Since then, PPR virus has been recognized as occurring in most of the countries in equatorial Africa, Middle East (Lefevre *et al*., 1991), Turkey (Ozkul *et al.,* 2002), Saudi Arabia and Ethiopia (Roeder *et al*., 1994) and India (Shaila *et al.,* 1989). The existence of PPR has been recognized in Pakistan since 1991 when it gave rise to an epidemic in Punjab province (Athar *et al.,* 1995). Diagnosis of PPR is mainly based on conventional tests such as agar gel immunodiffusion, counter immunoelectrophoresis (Obi and Patrick, 1984), haemagglutination test (Wosu, 1991), immunocapture enzyme linked inmunosorbent assay (Singh *et al.,* 2004), polymerase chain reaction (Forsyth and Barrett 1995) or virus isolation (Brindha *et al.,* 2001).

In this paper, the available clinico-pathological features of this outbreak among goats in an organized farm and the identification of Peste des petits ruminants virus (PPRV) by AGID from clinical specimen are described.

MATERIALS AND METHODS

Description of outbreak: The outbreak was recorded in a small goat flock owned by a private entrepreneur in Lahore, Pakistan, during April 2006.The flock was comprised of 32 young ones (less than 6 month) and 26 adult goats. The animals had not been previously vaccinated against PPR. The flock had not previous any history of illness due to PPR. The flock was routinely vaccinated against enterotoxaemia-cum-lamb dysentery and contagious caprine pluropnemonia (CCPP) disease.

Symptomatic treatment was given to sick animals just after the start of clinical signs but no significant response was observed.

Gross and Histopathological examination: The affected flock was examined clinically and postmortem of the dead animals were performed. For histopathology, spleen, lungs, liver, intestine and lymph nodes were fixed in 10% formalin. Paraffin embedding technique and haematoxylin and eosin staining methods were used for histopathology (Bancroft and Stevens, 1990).

Serological examination: Detection of PPR virus antigen was carried out by agar gel immunodiffusion (AGID) test (White, 1958). For this purpose, mesenteric lymph nodes and spleen samples were taken and processed by the method as described in FAO animal health manual (Anonymous, 1996). Samples were minced with scissor and grounded in mortar and pestle. The resulting slurry was centrifuged at 1500*xg* for 15 minutes. Supernatant fluid was collected for the detection of antigen. The standard PPR antigen was also prepared in laboratory (Anonymous, 1996). In this, attenuated PPR virus Nigeria 75/I was grown on African green monkey kidney (Vero) cells until

cytopathic effects were observed through the cell sheet. The monolayer was then washed with phosphate buffer saline (PBS) and cells were scraped from the glass. The cells were disrupted to release antigen by suspending them in PBS and freeze and thawn several times at -20^{o}C. PPR antigen in oil adjuvant was prepared in laboratory. The inoculum was injected in rabbit at different intervals (Table-1) and hyperimmune serum was raised by the method as described in FAO animal health manual (Anonymous, 1996).

Table-1 Inoculation schedule of inoculum

Day of Injection	Quantity Of Inoculum	Rout Of Administration
Zero day	1 ml	Subcutaneous
21 day	1ml	Subcutaneous
35 day	2ml	Subcutaneous

The serum obtained was used in agar gel immunodiffusion test (AGID) to determine presence of PPR antigen in suspected samples.

RESULTS AND DISCUSSION

Clinical Signs: Most frequent clinical findings in diseased animals were high body temperature (39.6 to 41.4 ^{o}C), severe muco-purulent nasal and ocular discharges, stomatitis, respiratory distress and diarrhoea. Mortality was higher in younger than adults. The overall morbidity rate in flock was 68.9 %(n = 40) while case fatality rate in youngers and adults was 43.7% (n = 14) and 23% (n = 6), respectively. The rectal temperature, pulse and respiratory rates were elevated (Table 2). Discrete areas of erosion and ulceration were detected on the lips, gingival, buccal mucosa and tongue.

Table 2: Rectal Temperature, Pulse and Respiratory rates of youngers and adults goats with Peste des petits Ruminants

Goats	Rectal Temperature (^{o}C)	Pulse Rate (bpm)	Respiratory Rate (breaths/min)
Youngers	41.4	110	40
	40.4	120	36
	40.1	116	40
	39.6	130	29
	40.3	100	38
Adults	41.2	112	58
	40.4	120	34
	40.1	132	40
	41.4	124	50

Gross and Histopathological examination: Postmortem examinations of five dead animals were carried out which revealed discrete area of erosion on the gums, lips and nasal cavity. Lungs were consolidated with patches of red zones on costal surface. Lymph nodes were swollen. Severe serosal congestion and erosion extended throughout the length of small and large intestine in mucosae. Spleenomegaly was observed in most of the cases.

Spleen, lungs, liver, intestine and lymph nodes were subjected to histopathological studies (Bancroft and Stevens 1990). In Lungs, atelectasis was observed with polymorph nuclear infiltration. Perivascular cuffing of mononuclear cells were the most pronounced feature. The alveolar walls were thickened. Mucosal hemorrhages were observed along with congestion of blood vessels in intestine. Hepatic necrosis was observed in some of cases with pyknotic nuclei. In spleen, the changes were not much pronounced except fibrosis of trabeculae

Serological examination: By agar gel immunodiffusion (AGID) test, clear precipitation lines were observed between the wells containing known PPR hyperimmune serum and suspected PPR antigen, suggesting the presence of PPR virus in the suspected sample.

Peste des petits ruminates poses a serious threat to the development of small ruminants production. In present study, an outbreak was recorded in a small flock, comprised of 32 young ones and 26 adult goat. Observed clinical signs in diseased animals were high rise of body temperature, severe muco-purulent nasal and ocular discharge, stomatitis, respiratory distress and diarrhoea. Similar clinical signs and symptoms were observed by earlier workers (Taylor and Abegunde, 1979, Taylor, 1984, Furley *et al.,* 1987, Aruni *et al.,* 1998, Amjad *et al.,* 1996 and Gul *et al.,* 2001) who reported purulent oculonasal discharge, severe cough and diarrhoea at terminal stages.

Overall morbidity rate due to PPR was 68.9 % while case fatality rate in youngers and adults was 43.7 % and 23 % respectively. Abu-Elzein *et al*., (1990) reported a high morbidity rate (90%) and case mortality rate (70%) in Saudi Arabia. This may be due to breed susceptibility to PPR disease or climatic condition of area. High morbidity due to disease is in accordance to Saravaran *et al*., (2007) who reported 63.2 % morbidity in goats with infection of peste des petits ruminants.

Grossly, consolidated lungs, enlarged lymph nodes, spleenomegaly and erosive intestines were observed which was in accordance to Aruni *et al*., (1998) who studied 48 PPR positive animals from ten different outbreaks and notified severe congested and consolidated lung parenchyma, enlarged retropharyngeal lymph nodes and spleen, and severe supporative inflammation of intestinal mucosa.

Similarly, on histopathological examination, atelectasis of lung, mucosal haemorrhages of intestine along with hepatic necrosis and fibrosis of trabeculae of

spleen were observed. These observations are inline with Aruni *et al*., (1998) and Yener *et al*., (2004) who observed oedematous and thickened alveolar septae with mononuclear inflammatory infiltrate, hyperaemic oedematous mucosa with focal ulcerated areas in intestine, focal necrosis of hepatic tissues and infiltration of spleen trabeculae with mononuclear cells.

Regarding the presence of PPR virus in suspected samples, spleen and mesenteric lymph nodes were subjected to AGID. Clear and distinct precipitation lines between the wells containing suspected PPR antigen and known PPR hyperimmune serum were observed. This observation correlated to Furley *et al.,* (1987) who collected the samples of spleen and mesenteric lymph nodes from *Borcas Gazelle* dying at Al Alin Zoo and employed AGID test.

In present study, it was observed that young animals were more susceptible to PPR than adults. This is in accordance to Aubaker *et al.,* (2008) who reported that goats were more prone to PPR than sheep and young animals of both species were at high risk than adults. Similar finding were observed by Kulkarni *et al.,* (1996) who reported significantly higher case fatality rate in kids.

In conclusion, clinical findings, gross and histopathological examination along with results of AGID test suggested that it was a serious outbreak of PPR disease in goats. Furthermore study showed that younger animals are more prone to PPR disease than adults.

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