PPR outbreak in goats: Epidemiological and therapeutic studies

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ABSTRACT

An outbreak of PPR occurred in newly purchased goats (30 males of 5-6 months age and 45 adult females). The disease appeared on 12th day of quarantine. Anorexia, fever diarrhoea, nasal discharge and pneumonia were the constant features of the disease, but buccal lesions, conjunctivitis and corneal opacity were observed only in few cases. The overall morbidity, mortality and case fatality rates were 32.1, 5.3 and 16.6% respectively. Influence of disease on haematological profile revealed anaemia and mild leucopenia. Bacteriological study of nasal and faecal swabs revealed organism indistinguishable to Escherichia spp, Corynebacterium spp. and Staphylococcus spp. Necropsy findings consisted of necrotic and ulcerative lesions on lips, gums, buccal mucosa, pharynx, oesophagus and nasal mucosa. Larynx, trachea and bronchi showed congestion and pulmonary parenchyma revealed consolidation and emphysema. Erosive and haemorrhagic abomasitis and enteritis, enlargement of spleen and lymphnodes were mainly observed. Antimicrobial and rehydration therapy showed recovery in 55% animals with enrofloxacin, 25% with trimethoprim sulphadiazine, 20% with ampicillin-cloxacillin combination and 20% with cefotaxime-amikacin combination. Overall therapeutic survivability was 85.7% with overall recovery rate of 10 days. Disease was confirmed as PPR by serum neutralization (for Morbilli virus) and PPR specific IC-ELISA test.

Key words: Bacteriology, Epidemiology, Goat, Necropsy, PPR, Treatment

After control of rinderpest in the country, emergence and continued outbreaks of Peste des Petits (PPR) since 1994 in the goat and sheep population is alarming, and is a serious threat to the growing goat industry in India. Diversification of disease to buffaloes and presumptive zoonotic indication are also cause of great concern (Govindrajan et al. 1997, Wekhe and Berepubo 1994). Absence of specific vaccine and ban on use of tissue culture rinderpest vaccine (TCRP) have put the entire small ruminant population at constant risk. Therefore, PPR outbreaks are now a regular feature in different parts of the country since 1994 (Nayak et al. 1997, Rana et al. 1998, Anjaneyalu and James 1999). Disease has now become endemic in goats and sheep. This paper investigates some of the epidemiological features of PPR infection and management of outbreak with intensive therapeutic intervention in newly purchased Jamunapari goats under quarantine.

MATERIALS AND METHODS

Animals

Jamunapari goats (45 adult females, and 30 male kids of 5-6 months age) were purchased from the Jamunapari field project area of the institute (CIRG, Makhdoom), (village Jagtoli and Kathodi ka Nagla, in Chakarnagar block, Etawah district UP) in March 2000. Animals were inhabitant of subsistence grazing system of management. Detailed clinical examination as per standard techniques (Smith and Sherman 1994) and mouth examination of each goats was performed before purchase. Goats were apparently healthy and did not show any overt clinical signs at the time of purchase. Animals were transported 250km from Etawah to the quarantine shed of the CIRG, Mathura.

Meteorological observations

Climatic conditions during the outbreak were 19.53°C minimum and 41.44°C maximum mean daily temperature and 27.92% mean relative humidity.

Housing

Goats were housed in quarantine shed of institute and were provided sufficient green fodder and concentrate along with free access to freshwater. Coproscopical examination of 30 randomly selected animals revealed very low endoparasitic and coccidial oocyst load. However, prophylactic dose of anthelmintic doramectin @ 200 µg/kg body weight, s/c, was given to all goats. On day 6, enterotoxaemia vaccination (B P Division, IVRI, Izatnagar) was followed by FMD vaccination on day 11 post arrival. Goats were examined daily...
for any apparent clinical disease. First clinical case of PPR was reported on 12th day of quarantine. All clinical cases of PPR were examined along with other laboratory investigations.

**Treatment**

Sick goats were treated with combinations of antimicrobials, viz. enrofloxacin, @ 3-5 mg/kg body weight im, daily, ampicillin-cloxacillin 0.5-1 g im, or iv twice a day, trimethoprim sulphadiazine, 150 mg/kg body weight and cefotaxime-amikacin 250-500 mg twice daily to combat secondary bacterial infections. Antihistaminics, vitamins and extensive rational rehydration and electrolyte therapy was provided for restoration of body fluid and ionic balance.

**Microbiological investigations**

Faecal swabs (5) were randomly collected from diseased animals. Swabs collected with sterile precautions, brought immediately to laboratory, were streaked on blood agar and Mac Conkey’s lactose agar plate and incubated at 37°C for 24 hr.

**Necropsy**

Detailed necropsy examination of dead animals was conducted. Serum samples, mesenteric lymphnodes and pieces of tongue, spleen and lungs were collected and sent on ice to the IVRI, Mukteshwar, Kumaon, for confirmatory diagnosis.

**RESULTS AND DISCUSSION**

First clinical symptom noticed was diarrhoea in 10 male kids (33.3%), on day 12th of quarantine. Since faecal samples from diarrhoeic kids were heavily positive (+4) for coccidiosis therefore, amprolium in therapeutic doses at the rate of 15 mg/kg body weight was given to all kids (males), irrespective of their clinical symptoms. Kids showed transient clinical recovery from coccidiosis. Disease was later confirmed as PPR by serum neutralization test (morbilli virus infection), and by PPR specific IC-ELISA test on pooled and individual tissue samples. Sudden flareup of clinical coccidiosis in these earlier coccidiosis negative kids can be correlated with the immunosuppressive effect of PPR virus, which is a well documented phenomenon.

**Morbidity and mortality**

Morbidity, mortality and case fatality rates in adult goats were 8.9, 2.2 and 25.0% and in kids, 66.6, 10.0, and 15.0% respectively. The overall morbidity, mortality and case fatality rates were 32.1, 5.3 and 16.6% respectively. The result (Table 1) indicate higher susceptibility of kids to PPR infection as compared to adult goats (Rana et al. 1998). Kids usually harbour normal subclinical load of coccidia in this group, which exerts immunosuppressive effect, which might have triggered the endemic infection of PPR. Similarly Akakpo et al. (1996) suggested that strains of certain E. coli strains augment the effect of PPR infection which is because of its fimbrial adhesions to the intestinal mucosa. Therefore, the concurrent intestinal infections have triggering effect on infections like PPR which further enhances the susceptibility of younger animals to infection. Role of other enteroviruses and gastrointestinal parasites (like Haemonchus) in increasing the host susceptibility to PPR can not be ruled out. Adult goats were less susceptible as only 4 goats (8.9%) were affected. It may be because of strong and developed immune system in adults.

After the initialization of disease, epidemic gradually built up which peaked after 1 week of first case. Epidemic gradually subsided after 28 days, when each animal recovered completely from the infection.

**Clinical symptoms**

Initially kids and later adult goats were off-feed, dull, depressed and had high temperature (up to 107°F). Nasal discharge, which was initially serous later became mucopurulent. This resulted in typical thick deposit around nares which was obstructing respiratory passage; foetid diarrhoea, mild to shooting in nature. Frequency of symptoms has been displayed in Table 2. Anorexia, fever, diarrhoea, nasal discharge and pneumonia were the constant features of the disease, but buccal lesions has not been a regular clinical feature in goats and were observed in 4 cases only (16.6%, 3 kids and 1 adult goat). Whereas, hyperaemic lesions were more common. Buccal lesions comprised whitish yellow deposition on dorsum of tongue mucosa and patches of erythema on gums and either side of tongue. Conjunctivitis

<table>
<thead>
<tr>
<th>Table 1. Mortality and morbidity rates in PPR outbreak in quarantine Jamunapari goats</th>
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<tr>
<td><strong>Age group</strong></td>
</tr>
<tr>
<td>Adult</td>
</tr>
<tr>
<td>Kids</td>
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<td>(5-6 months)</td>
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<td>Overall</td>
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Table 2. Frequency of predominant symptoms and treatment response in PPR outbreak in goats

<table>
<thead>
<tr>
<th>Clinical symptoms</th>
<th>No. of animals</th>
<th>Per cent</th>
<th>Treatment response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Off feed</td>
<td>24</td>
<td>100</td>
<td>Late</td>
</tr>
<tr>
<td>Fever</td>
<td>24</td>
<td>100</td>
<td>Early</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>24</td>
<td>100</td>
<td>Last</td>
</tr>
<tr>
<td>Pneumonia/coughing</td>
<td>24</td>
<td>100</td>
<td>Quick</td>
</tr>
<tr>
<td>Buccal lesion</td>
<td>4</td>
<td>16.6</td>
<td>Slow</td>
</tr>
<tr>
<td>Conjunctivitis</td>
<td>6</td>
<td>25.0</td>
<td>Slow</td>
</tr>
<tr>
<td>Corneal opacity</td>
<td>1</td>
<td>4.16</td>
<td>Slow</td>
</tr>
<tr>
<td>Severe respiratory distress (asthma like)</td>
<td>2</td>
<td>8.3</td>
<td>Quick</td>
</tr>
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was noted in only 6 cases (25.1%), leading to corneal opacity in 1 case, which may be related to auto-immune reaction. However, exact reason is unexplainable. In 2 animals severe respiratory distress, similar to asthma was observed, which has not been reported earlier, which was managed by use of antihistaminics. As a result of first acute and then chronic illness (mainly diarrhoea), animals became weak, emaciated with rough hair coat and poor appetite. It may be associated with extensive damage to intestinal epithelium leading to malabsorption-digestion syndrome. Three kids which showed recurrent attack of diarrhoea, could be said to belong to this class. Temperature pattern showed a sharp rise (up to 107°F) within 2-3 days of illness which persisted for next 2-3 days with mild deviation. Later on it started declining and come to normal on day 6-7. The probable reason for such a high rise in body temperature is the super infection by secondary invaders. Of all the clinical lesions diarrhoea was last to respond.

Necropsy findings

Carass showed signs of dehydration, emaciation and soiling of the posterior part with fluid faces. Lips, gums, buccal mucosa, pharynx and oesophagus revealed necrotic and ulcerative lesions. Nasal mucosa also showed necrotic lesion. Larynx, trachea and bronchi revealed congestion, consolidation of pulmonary parenchyma, atelectasis and emphysema. Abomasum revealed erosive and haemorrhagic lesions. Spleen and lymphnodes were enlarged. Broadly, lesions of erosive haemorrhagic enteritis were prominent and pathognomic of the etiological agent.

Laboratory investigations

Influence of disease on haematological profile (Table 3) revealed anaemia, mild leucopenia related with blood loss through intestine and destruction of lymphoid tissue (Smith and Sherman 1994). Microbiologically, on the basis of haemolysis, morphology, cultural and staining characteristic, revealed haemolytic and lactose fermenting colonies. Microscopically organism were non-capsulated, Gram negative cocco-bacilli, indistinguishable to Escherichia spp. Similarly, nasal swabs screening revealed Gram positive bacilli, bipolar organism granular cytoplasm with Chinese letter arrangement mixed with Gram positive cocci, indistinguishable to Corynebacterium spp. and Staphylococcus spp. Findings are in correlation with Ugochukwu and Agwu (1991), who identified many bacterial isolates in cases of PPR in goats, viz. Staphylococcus spp., Streptococcus spp. Neisseria spp., Pasteurella spp., Pseudomonas spp., Corynebacterium spp. etc.

Being viral infection, there is no specific treatment but to combat secondary bacterial invaders, sick animals were treated with antibiotics/antibiotics/antibacterial drugs. Antibiotic therapy revealed efficacy of 55% (11 animals) with enrofloxacin, 25% (5 animals) with trimethoprim sulphadiazine, 20% (4 animals) with ampicillin and cloxacillin combination and 20% (4 animals) responded with cefotaxime amikacin combination. Overall therapeutic survivability was 85.7%. The response to drugs varied due to variable susceptibility of different strains involving more than 1 species of bacteria present in intestine and lungs. The intra venous administration of metronidazole along with antibiotics was given to check anaerobic infection and was useful. Besides antibiotics, antihistaminics, and vitamins were given as supportive therapy, along with 5% dextrose normal saline and ringers lactate solution as and when required. Respiratory distress, pneumonia and fever were signs which resoluted early, while conjunctivitis, diarrhoea and buccal lesions were last to resolve. Increased survival rate and decreased mortality with intensive therapy was noted by Paritosh (1997).

Variable recovery rates were observed in kids, viz. 15 days (2 animals), 11 days (8 animals), 8 days (5 animals), 6 days (9 animals). Average recovery rate was 10 days. This could be associated to the individual resistance of animal to disease.

Appearance of PPR in apparently healthy animals could be associated with herding of animals from different locations, various degrees of stresses and sudden change in environment and feeding habits. Transport stress has significant effect as free grazing animals were suddenly herded for more than 12 hr without feed and fodder. The animals could not get acclimatized due to their sudden movement to intensive management system and change in feed and fodder. Jamnapari being a free grazing breed of goats, helped in predisposing of animals to infection. Transport stress had also been seen related to PPR outbreaks by Nayak et al. (1999) as has been other diseases. Environment stress, particularly hot and humid climate also favours precipitation of disease (Wous 1995).

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