CLINICO-PATHOLOGICAL FINDINGS IN WEST AFRICAN DWARF GOATS WITH PESTE DES PETITS RUMINANTS INFECTION

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ABSTRACT

The clinical signs and pathological changes in West African dwarf goats naturally infected with peste des petits ruminants (PPR) were studied. Twenty one goats were housed in one pen throughout the duration of the study. One out of 21 goats was purchased with clinical manifestations suggestive of PPR and the goat served as the source of natural infection to the rest of the goats. By day 3 post aggregation of the animals, 5 out of the 21 goats showed clinical signs of PPR which include coughing, sneezing, pyrexia, starry hair coat, mucopurulent ocular and nasal discharges, watery diarrhea, head pressing, kyphosis and dehydration. By day 5 post aggregation all the goats had come down with the disease. Gross pathological findings included zebra stripes on the cecal mucosa, exudative pleurisy in the thoracic cavity, ecchymotic hemorrhages on the trachea with extensive froth, consolidation and congestion of the lungs. New findings of degenerative fatty liquefaction around the coronary region of the heart and within the ventricles, including ante-mortem clots within the ventricles were made in some cases. These manifestations were observed in varying degrees of severity in the goats and can be considered pathognomonic of PPR infection.

Keywords: clinical signs, pathological changes, peste des petits ruminants, West African Dwarf goats

INTRODUCTION

Peste des petit ruminants (PPR) is a highly contagious viral respiratory disease of small ruminants (Ogunsanmi, *et al.,* 2003; OIE, 2008). It is one of the major impediments to improving the productivity of small ruminants in regions where it is endemic (Chavan *et al*., 2009). PPR is caused by an RNA virus of the genus morbillivirus of the family paramyxoviridae similar to the rinderpest virus (Chavan *et al*., 2009). The occurrence of the disease has a seasonal variation, with outbreaks occurring mainly in the rainy season or very cold season (CIDRAP, 2003).

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Currently, there is no reported treatment for PPR. However, the disease could be managed using various broad spectrum antibiotics to prevent secondary pulmonary infections which most times precede death (Wosu, 1989; Ajala *et al.,* 1997; OIE, 2002). However, the few reports on the medical management of PPR available show that the infection is refractory to routinely used chemotherapeutic agents such as antibiotics, vitamin supplements and other supportive measures (Omamegbe *et al.*, 1984; Anene *et al*., 1987).

The clinical and pathological changes seen in the disease are considered to be useful tools in its diagnosis and subsequent management. This study, therefore, is designed with the objective of determining the clinical and pathological changes that occur in goats with natural infection of PPR.

MATERIALS AND METHODS

Twenty one West African Dwarf goats of mixed sexes, of varied ages and weighing 9.5-19.7 kg were used in this investigation. The goats were purchased in one batch from an open market in Mbano local government area of Imo state, in the forest zone of the southeastern parts of Nigeria. At purchase, all the goats appeared healthy except for a male buck which showed initial clinical signs suggestive of PPR and was regarded as the source of natural infection.

All the goats were housed together in one pen to simulate the condition normally encountered in the field. The goats were routinely screened for gastrointestinal and blood parasites. Positive cases for these infections were treated accordingly.

The clinical signs of the disease were observed in the goats on a daily basis. Post mortem examinations were conducted on dead animals soon after death and the pathological lesions were recorded. The pathological lesions present in different animals were evaluated using the “Score Method” as described by Jensen *et al.* (2002). Briefly, the range of variation in the severity of the lesions of the disease was divided into ordinal classes: Absent (0), Mild (+), Moderate (++) or Severe (+++). The study was conducted over a period of 2 weeks.

RESULTS AND DISCUSSION

The clinical signs manifested in all the experimental goats include coughing, sneezing, pyrexia of 41ºC, starry hair coat, mucopurulent ocular and nasal discharges, watery diarrhea, head pressing, kyphosis and dehydration (Table 1).

The gross pathological findings recorded include severe dehydration, scabby orf-like labial lesions, oral and vaginal erosions, severe occlusive oro-nasal and ocular discharges, matted eyelids, perineum soiled with watery feces, and cachexia (Table 2). Internally, there were frothy tracheal exudates, sero-hemorrhagic bronchial and bronchiolar exudates, pulmonary edema and consolidations, hydrothorax, fibrinous pleuritis, pulmonary hemorrhages, flabbiness of the left ventricular chamber, myocardial degeneration (Figure 1) and ante-mortem intra-

Table 1. Clinical signs observed in goats with peste des petits ruminants infection.

Days after aggregation. of herd	Number showing clinical signs	Most characteristic sign seen in the goats	Comment
1-2	1	Coughing and sneezing, serous to muco-purulent oculo-nasal discharges.	As purchased from market. Source of natural infection
3-4	5-8	Anorexia and variable appetite, Coughing, diarrhoea, serous and catarrhal nasal discharges, variable rectal temperature (37.8 – 41.5^{o}C).	Very slow spread of infection in-spite of close contact of inmates
5	20	2 sudden deaths, matted eye lids, oral ulcers, orf-like lesions, kyphosis, head pressing and a few cases of diarrhoea.	Several sudden deaths typical of per-acute PPR infections. Sudden rapid spread
11	20	Signs more severe – serous discharges becoming catarrhal, more incidence and profuse diarrhoea. Temperature range 36.4 -41^{o}C	Sudden deaths

cardiac clots (Figure 2).

The gastro-intestinal tract showed labial and oral erosions or ulcers, orf-like labial scabs, relative emptiness of the intestines but full fore-stomachs, ecchymotic and petechial intestinal hemorrhages and prominent Zebra crossings in the terminal ileum, cecum and proximal colon. The gall bladder was grossly distended in all cases (Figure 3). Deaths occurred early in the course of the disease as some goats died as early as day 5 post aggregation of the herd with mean death time of 5±5.0 days.

Although there was no viral isolation of morbilivirus to confirm the manifested clinical signs and pathologic lesions, the diagnosis is suggestive of PPR because the closely related rinderpest has been eradicated in Nigeria. FAO (1999) confirmed that rinderpest in small animals is relatively a rare event, even in Asia. Other differential diagnoses such as contagious caprine pleuropneumonia (CCPP), coccidiosis, contagious ecthyma and cowdriosis were eliminated due to the absence

Table 2. Pathological changes observed in goats with peste des petits ruminants infection evaluated by score method as described by Jensen *et al.* (2002).

Assessment	Number showing clinical signs	Score
Exudative pleurisy	21/21	+++
Copious ocular and nasal discharges	21/21	+++
Ecchymotic haemorrhage in the trachea	15/21	+++
Zebra stripes on the mucosa of the ileu, caecum and colon	18/21	+++
Haemorrhage on the mucosa of the oma-sum	10/21	+++
Copious froth in the trachea and bronchus	21/21	+++
Froth in the lungs	21/21	+++
Consolidation and congestion of the lungs	21/21	+++
Adherence of blood clots on the walls of the ventricle and valves	6/21	+++
Fatty liquefaction on the coronary region of the heart	5/21	+++
Engorged gall bladder	21/21	+++
Catarrhal secretions in the intestine	15/21	+++
Watery diarrhoea	21/21	+++
Ulceration of the vagina	1/21	+++
Depraved appetites in a goat	1/21	++

of either diarrhea, nervous or respiratory compromise in their clinical signs. For instance, classical CCPP, cowdriosis and contagious ecthyma do not show any sign of diarrhea in the infected goat. Also there was no nervous sign in the goats in the present study, which is characteristic of cowdriosis in goats. Goats with coccidiosis do not manifest copious ocular and nasal discharges, as was observed in the goats in the present study.

All the infected goats had cough and sneezed randomly in an attempt to dislodge the muco-purulent discharges that clogged up the respiratory system. The cough was also sequel to the existing heart disease observed in the animals. In the respiratory system, it appeared that the copious froth in the trachea and the lungs in most of the infected goats played a major role in the death of the animals.

Virtually all the 21 infected goats show extensive accumulation of froth in the trachea and within the lungs tissue. The froth invariably prevented proper respiration

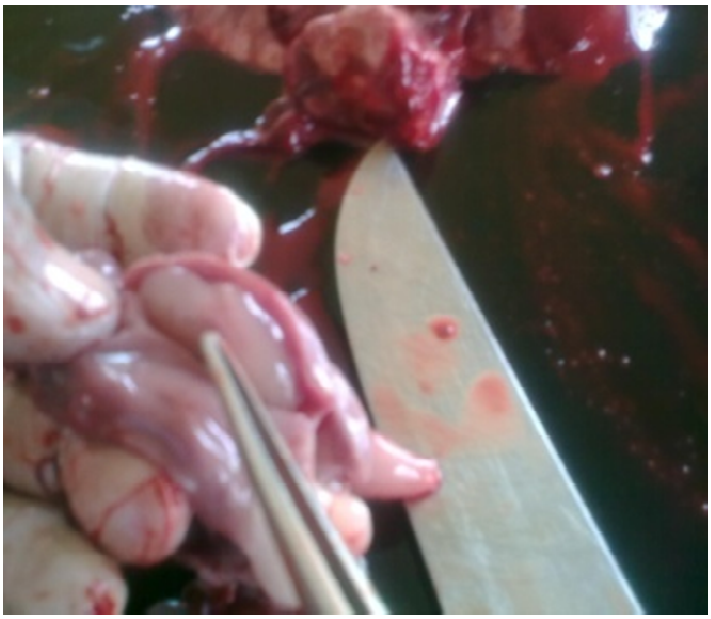

Figure 1. Fatty degeneration in the ventricles seen in a goat with peste des petits ruminant infection.

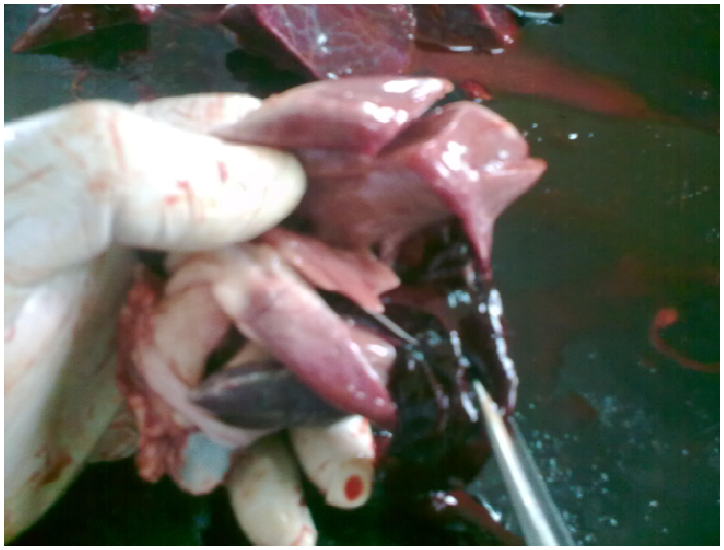

Figure 2. Adhesion of blood clots on the walls of the ventricles seen in a goat with peste des petits ruminant infection.

Figure 3. Engorged gall bladder seen in a goat with peste des petits ruminant infection.

which resulted to suffocation and death. Also, all the infected goats had copious nasal and ocular discharges which suggested evidence of sinusitis. The initial serous nasal and ocular discharges later became muco-purulent due to invasion of secondary bacterial infection. *Bordetella bronchoseptica* has been isolated in most cases of secondary bacterial invasion in PPR infection. The pneumonic lungs and hemorrhagic trachea were seen in all the infected animals probably due to descending upper respiratory infection complicated by secondary bacterial infection. All the infected goats either present fibrinous or exudative pleurisy. It seems that those that died early in the disease had exudative pleurisy whilst those that had stronger immunity and tried to throw away the virus developed fibrinous pleurisy. This was due to the considerable time lag during which the immune system fought the virus which afforded fibrocytes opportunity to invade the exudates in the thoracic cavity and laid down fibrous tissues. The fibrinous exudation may be confused with post mortem findings in contagious CCPP. However, the absence of diarrhea in CCPP eliminates it as the cause of the disease. These respiratory signs agree with the findings of FAO (1999). The consistent observation of these lesions in infected animals could be regarded as pathognomonic for PPR.

In previous investigations of PPR, the circulatory lesions have been overlooked as major causes of death in PPR infections as was observed in the EAZWV Transmissible Disease Fact Sheet (Geerts, 2009). Nevertheless, this study presents a novel discovery of pathological changes associated with the heart which are believed to be contributory to the high mortality associated with PPR in goats. The ante-mortem blood clots (Figure 2) were either firmly or loosely attached to the

walls of the ventricles, some clots were actually difficult to dislodge from the walls of the heart. This could mean occurrence of heart disease that progressed to heart failure which rapidly led to the death of the animals. The presence of fatty materials around the coronary borders and within the ventricular walls (Figure 1) compromised the pumping activities of the heart. This resulted to a decrease cardiac output and an increased venous return which compounded the stress on an ailing heart (unpublished report). Also, the exudates and the froth in the respiratory system decreased the oxygen tension in the lungs which, in turn, reduced perfusion and available oxygen for the cardiomyocytes. All these facilitated the high mortality rate associated with PPR.

The changes observed in the gastrointestinal system, are consistent with the findings of Ahmad *et al*. (2005). The presence of catarrhal secretions and profuse watery diarrhea in the infected animals signified the localization of PPR virus in the digestive system. Invariably, attempts to eliminate the virus lead to profuse diarrhea, dehydration and death. Most cases of death in PPR were as a result of dehydration following diarrhea. Consistent engorged gall bladder was also observed in all the infected animals which explains maldigestion and malabsorption in the intestine. However, there was no record of engorged gall bladder in the literature by early researchers. There were ecchymotic hemorrhages on the mucosa of the abomasum, cecum and the colon. The hemorrhages on the mucosa of the colon appeared in stripes giving it the characteristic name “Zebra stripes” usually pathognomonic for PPR (FAO, 1999; OIE, 2008).

The results of this study show that apart from the already established respiratory and gastrointestinal changes in PPR observed in infected goats, circulatory derangement occurred and are also contributory to high mortality associated with PPR. Manifestation of these classical signs in goats in less developed countries with less sophisticated laboratory for viral isolation could be used for diagnosis of PPR and can signal the commencement of treatment pending viral isolation.

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