Prevalence of suspected peste des petits ruminants infection and complicating bacteria in goats in Abeokuta, Ogun State, Nigeria

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Abstract

Peste des Petits Ruminants (PPR) is endemic in Nigeria and causes major economic losses due to the high rates of mortality and morbidity in infected domestic sheep and goats. The outbreak of PPR in some goat herds was observed in the Abeokuta area of Ogun State, Nigeria, between January and August, 2014. Amongst the herds affected were those of the research animal in the Federal University of Agriculture Abeokuta. The goat herds were located in the Directorate of the University Farms (DUFARMS), the University environments and Apakila. The goats were intensively managed on concentrates and Panicum maximum grass. The morbidity rates were 80-100 per cent in all the herds while mortality rates varied from 10% to 70%. Disease diagnosis was based on history, clinical signs and Post-mortem lesions. Complicating secondary bacteria were isolated from nasal and ocular discharges, which isolated, identified and sensitivity results obtained from laboratory tests. Treatments were carried out with sensitive antibiotics, analgesics and fluid therapy in a symptomatic approach. We conclude that PPR is endemic in this area of study and is responsible for a great loss in goat production and poverty of the rural goat farmers. We recommend that vaccination against PPR in this study area be instituted to avert the deleterious effects of the disease.

Keywords: bacteriology, goats, Pathology, PPR, prevalence

Introduction

Peste des petits ruminants (PPR) is an acute febrile viral disease of small ruminants, characterized by mucopurulent nasal and ocular discharges, necrotising and erosive stomatitis, enteritis and pneumonia (Singh et al., 2004). It is a contagious transboundary disease that is widely distributed across the Sub-saharan Africa, Middle East, Arabian Peninsula and the Indian subcontinent (Odo, 2003; Diallo, 2006). The disease causes serious economic losses and remains a major deterrent to a successful development of small ruminant production in the countries where it occurs (Dhar et al., 2002; Diallo, 2003; Yener et al., 2004). The disease is endemic in Nigeria and causes major economic losses due to the high rates of mortality and morbidity in infected domestic sheep and goats. The prevalence rates are highest in months of June to September and this correlates with the climatic conditions at this period of the year (Olayode et al., 2008). It causes death in more than 50 per cent of the affected animals due to high fever, pneumonia, diarrhoea and dehydration. It is followed by losses in production yield. Expenditure on medicine and infertility has been found to cause more than 80 per cent of the total cost, followed by Veterinary and labour services (Thombare and Sinha, 2009). A large number of bacteria invaders have been isolated in the course of the disease (Thombare and Sinha, 2009).
The viral agent belongs to the morbillivirus genus of the family *Paramyxoviridae* and possesses antigenic links with the viruses of rinderpest, canine distemper and human measles (Gibbs et al., 1979; Diallo, 1990). The PPR hydrated virus's half-life held at 37°C is about 2 hours and at 50°C, infectivity is destroyed within 30 min (Lefèvre, 1982). The virus is equally sensitive to lipid solvents and to low pH. A virus present in lymph nodes is protected from pH changes after death, and it can be recovered from the lymph nodes of carcasses held for 8 days at 4°C. PPR is spread through contact of infected and susceptible animals, there are several means of transmission which includes; inhalation of aerosols produced by sneezing and coughing of infected animals; direct contact with ocular, nasal or oral excretions; direct contact with faeces; fomites such as bedding, water, and feed troughs. Carrier state has not been established (Ozkul et al., 2002).

Clinically, PPR resembles rinderpest and is characterised by mucopurulent ocular and nasal discharges, necrotizing and erosive stomatitis, enteritis and pneumonia. These signs may not all be exhibited by infected animals during an outbreak, as symptomless infections have been reported (Diop et al., 2005; Couacy-Hymann et al., 2007a and 2007b). It has been observed that PPR outbreaks occur one or two weeks after purchase and stocking of susceptible animals. The incubation period is about six days. The onset of the disease is marked by a sudden dullness and pyrexia, with high body temperature (40°-41°C). Whole blood collected could be used for serological diagnosis. Serological tests include Virus Sero-neutralisation, competition ELISA, Counter immunoelectrophoresis, Agar gel immunodiffusion and Immuno-diffusion inhibition test. Samples can also be submitted for identification of the agent; techniques include detection of the antigen by immunological method (counter immunoelectrophoresis, ELISA) virus identification, and virus RNA detection using PPR-specific DNA probes or amplification by polymerase chain reaction (PCR) (OIE, 2002). For these methods specimen can be collected on live animals: swabs of the eye (conjunctival sac), nasal secretions, and mouth and rectal lining, clotted and whole blood (with EDTA anticoagulant) or at post-mortem: fresh and preserved samples of tonsil, tongue, spleen, lymph nodes, affected areas of the alimentary tract mucosa (Geering, 1995). PPR is one of the reportable diseases according to OIE. The prevalence and outbreaks of the disease has been reported in many parts of the world such as Turkey (Yener et al., 2004), Pakistan (Ahmad et al., 2005), Iran (Abdollahipour et al., 2006), Iraq (Dosky et al., 2006), Tajikistan (Kwiatek et al., 2007), Congo (OIE, 2006) and some others. Also the disease outbreak has been reported in some parts of Nigeria such as Maiduguri (El-Yuguda et al., 2009), Osun State (Olayode et al., 2008), Oyo State (Obi, 2012). But the report of PPR prevalence and outbreak in Abeokuta and its environ is scarce. Therefore in this paper we report the prevalence of PPR among West African dwarf (WAD) goats in Odeda Local Government, Abeokuta, Nigeria.

**Materials and methods**

**Farm sites**

The disease outbreak was observed in six herds. The first herd affected had 32 West African Dwarf (WAD) goats which were all female animals at the DUFARMS, Federal University of Agriculture while the others were in Apakila, all in Odeda Local Government, Abeokuta, Nigeria.
History and management system
The goats were purchased from markets and were brought in and kept for research purposes. The disease outbreak was observed during the period of quarantine and adaptation to the new environment. The herds were managed intensively, the goats were fed concentrate diets in the mornings and fresh *Panicum maximum* grass was provided afterwards. Feed and water was provided ad libitum.

Disease diagnosis
The diagnosis of the disease was based on history, the clinical signs and Postmortem lesions of the posted dead animals (carcasses) at the Pathology Department of the College of Veterinary Medicine, Federal University of Agriculture, Abeokuta. The disease was confirmed from the gross pathological lesions found which were pathognomonic for PPR infections.

Clinical signs
There was pyrexia, copious nasal and ocular mucopurulent discharges, oral necrotising and ulcerative stomatitis, severe diarrhoea, dehydration, emaciation, anorexia, lethargy, foaming in the mouth, curdled unsteady gait, dyspnoea, congested ocular mucous membrane, catarrhal conjunctivitis, corneal opacity, abortion, depression, palpitation, tachycardia and panting.

Gross lesions
Gross post-mortem findings in the present study were severe mucopurulent ocular nasal discharges, encrustation and scab formations around the mouth commissure, ulcerations of the oral mucous membrane, suppurative bronchopneumonia in 60% of the cases and enteritis in 100%.

Plan
Ocular and nasal discharge were collected and sent to with sterile swab sticks and sent to Microbiology laboratory for isolation, identification and sensitivity tests of complicating bacteria. Carcasses of dead goats were taken to the Pathology Laboratory for confirmation of the disease and likely cause of death.

Isolation and Identification
Samples of ocular and nasal swabs were aseptically collected from affected goats and inoculated on 7% sheep blood agar and MacConkey agar without salt (Oxoid CM 516, UK), and were incubated at 37°C for 18-24 hours. Each isolate obtained was examined for colonial morphology according to Cowan and Steel (1993) and further characterized using API enterobactericaea kit and interpreted accordingly.

Antimicrobial susceptibility testing
The susceptibility pattern of each isolate was tested against commonly used antibiotics by the disk diffusion method on Mueller Hinton agar according to Bauer et al. (1966). The following antibiotic disks were used: ampicillin (10µg), Amoxicillin (10µg), Tetracycline (30µg), Cefuroxime (30µg), Gentamicin (10µg), Ofloxacin (10µg), Chloramphenicol (30µg), Cotrimoxazole (5/25µg) and Ciprofloxacin (10µg). Pure isolates of 0.5 McFarland was spread on Mueller Hinton agar and the antibiotic disks were placed. The plates were incubated at 37°C for 18–24 hours. The inhibition zones were measured and interpreted as sensitive, intermediate, or resistant according to Clinical and Laboratory Standards Institute guidelines (2009).

Results and discussion

Microbiology
For ocular sample examination, *Staphylococcus aureus* (haemolytic) and B-haemolytic streptococci were identified while *Staphylococcus aureus* was identified for nasal sample.
Table 1: Sensitivity tests

<table>
<thead>
<tr>
<th>Drug</th>
<th>Staphylococcus areus</th>
<th>B-haemolytic streptococci</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin (10µg)</td>
<td>Sensitive</td>
<td>Resistant</td>
</tr>
<tr>
<td>Gentamycin (10µg)</td>
<td>Sensitive</td>
<td>Sensitive</td>
</tr>
<tr>
<td>Tetracycline (30µg)</td>
<td>Sensitive</td>
<td>Sensitive</td>
</tr>
<tr>
<td>Ofloxacin (10µg)</td>
<td>Sensitive</td>
<td>Sensitive</td>
</tr>
<tr>
<td>Cotrimaxazole (25µg)</td>
<td>Resistant</td>
<td>Resistant</td>
</tr>
<tr>
<td>Cefuroxime (30µg)</td>
<td>Sensitive</td>
<td>Resistant</td>
</tr>
<tr>
<td>Ciprofloxacin (10µg)</td>
<td>Sensitive</td>
<td>Sensitive</td>
</tr>
<tr>
<td>Chloramphenicol (30µg)</td>
<td>Resistant</td>
<td>Resistant</td>
</tr>
</tbody>
</table>

Post-mortem
The carcasses were dehydrated and soiled with faeces, necrotic and ulcerative lesions in the nose and mouth, the lungs were severe congested and oedematous; and cranial lobes were consolidated. The entire length of the intestine was distended with dark watery faecal materials; the mucous membrane of the small intestine were hyperemic and there were 'Zebra Stripping'.

Treatment/disease management
A symptomatic treatment approach was adopted. Combination of antibiotics, multivitamins, bronchodilators and analgesics were used at therapeutic doses. The drugs used for the management of the cases were Metronidazole infusion used as a systemic antibiotic IV infusion, Tetracycline Injectables used as a combined systemic antibiotic, Chloramphenicol eye drop used as an antibiotic for conjunctivitis, Multivitamin Injectables used as a reboants, Dichlorphenac Caplets used as an analgesic and Salbutamol tablets used as a bronchodilator.
Prevalence rate observed in this study was 80% and dissimilar to the reports of El-Yuguda et al. (2009) who reported a prevalence rate of 63%. This dissimilarity in the prevalence rate may be attributed to the breed difference as El-Yuguda et al. (2009) case report was on Sahel goats in Maiduguri while this present work was on WAD goats. Obi et al. (1988) also reported similar clinical signs and lesions of the disease as was observed in this study. The gross post-mortem lesions in the present study were highly suggestive for PPR as it agreed with the reports of other workers (Ozkul et al., 2004; Yener et al., 2004).

Conclusion
The study showed that PPR was endemic in Abeokuta and do recommend that adequate education be given to rural farmers in order to get their goats vaccinated so as to avert the losses incurred from expenditures of Veterinary drugs, Veterinary Services, loss in production from emaciation, abortion, infertility and deaths. These losses have always translated to depression of farmers, food insecurity and poverty of the rural farmers. 'Prevention is always better than cure. There is the need to isolate and characterize the PPR virus in the study area, so that effective vaccine against the disease can be produced.

References
Abdollahipour, G., Raoofi, A., Najafi, J.,


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