Clinical and epidemiological features of peste des petits ruminants in Sokoto Red goats

Aspects cliniques et épidémiologique de la peste des petits ruminants (PPR) chez la chèvre rousse de Sokoto - Les auteurs présentent les tableaux clinique, pathologique et épidémiologique de la PPR chez la chèvre rousse de Sokoto (Nigeria). Cliniquement la maladie se caractérise par de la fièvre, de la diarrhée et des sécrétions mucopurulentes abondantes de la sphère oculo-nasale, accompagnées de lésions des muqueuses vaginale et nasale et de graves désordres respiratoires. Globalement, avec une incidence de 0,7 p. 100, l'affection reste peu importante dans le tableau clinique des maladies reconnues dans la zone de Zaria, bien qu'au sein des foyers, les taux de morbidité (25 à 88 p. 100 selon les troupeaux) et de mortalité (23 à 56 p. 100) soient du même ordre que ceux de la zone humide. De même son incidence est peu élevée dans les deux zones en fin de saison sèche et en début de saison des pluies, ce qui pourrait résulter d'une corrélation avec les conditions éprouvantes de cette période de l'année. Escherichia coli et Pasteurella hemolytica ont été régulièrement trouvées en association avec la maladie. Les différences entre les systèmes d'élevage des zones de savane sèche et humide du Nigeria pourraient expliquer les tableaux épidémiologiques distincts observés dans ces deux milieux. Mots clés : Petits ruminants – Chèvre rousse de Sokoto – Peste des petits ruminants – Epizootiologie – Nigeria.

INTRODUCTION

Peste des petits ruminants (PPR) is a viral disease of sheep and goats first described in the Ivory Coast by GARGADENNEC and LALANNE (8). The disease was thought to be confined to West Africa (5, 6, 12, 14, 17) but has been described elsewhere recently (21). Breed susceptibility to PPR has not been clearly defined although there is a preponderance of clinical reports in the West African Dwarf (WAD) breeds (1, 9, 12, 14, 16, 19) compared with the sub-sahelian breeds such as the Sokoto Red (7) which are also common in West Africa. However, serological evidence indicates widespread infection in the Sokoto Red goats (22). The apparent lack of clinical reports in the Sokoto Red therefore suggests that they are less susceptible to clinical disease, or that clinical disease has unusual manifestations that make it difficult to recognise. This paper reports on the investigations that this breed shows typical clinical disease as described for the WAD, but the low frequency of disease outbreaks may be due to peculiar husbandry system and climatic conditions in this geographical zone.

MATERIALS AND METHODS

Field outbreaks

Two outbreaks were investigated. These involved two herds of Sokoto Red goats maintained in the Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria. The first outbreak occurred in March 1982, in a herd which had been maintained in the Faculty since 1975 without addition of new animals. The second occurred in 1983 in a herd which had been assembled from animals purchased from local markets. History, clinical signs, and other epidemiological parameters were recorded. Nasal and rectal swabs were taken from sick animals for bacterial culture. Animals that died were necropsied and specimen were taken for histopathology and serological diagnosis. Serum samples were obtained from animals during the febrile and convalescent stages. One year after the outbreak, all the animals in the first herd were bled and serum samples were examined for antibodies.

Serology

Laboratory diagnosis was done by the agar gel immunodiffusion (AGID) test using homogenates of lymph nodes and spleen from infected animals as antigen (15) and by the immunoelectroendoosmosis (IEOP) (13). Reference positive immune serum prepared in sheep was obtained from Dr. W.P. TAYLOR, Animal Virus Research Institute, Pirbright, England.

Clinical records

The case records of the Large Animal Clinic,

Faculty of Veterinary Medicine, Abu, Zaria, between 1980 and 1983 were examined for cases diagnosed as PPR and meeting the diagnostic criteria based on the field investigations. Cases were classified as PPR if during the course of clinical disease they showed fever, diarrhoea, mucopurulent oculonasal discharge and erosions of buccal or vaginal mucosa at one time or the other.

RESULTS

Epidemiology

Both outbreaks occurred in late dry season; the first began on March 11, 1982 and the second in May, 1983. The clinical records also showed that PPR cases clustered at the same period (March to May; tabl. I). The morbidity rate was 88 p. 100 in the first, and 25 p. 100 in the second; the mortality rates were 57 p. 100 and 25 p. 100 respectively, although the rates were higher in the young animals (under 1 year) in both cases (Tabl. II). The clinical records showed an overall prevalence rate of 0.7 p. 100 during the four year study period (Tabl. I), with a slightly higher prevalence in goats (1 p. 100 or 11 cases out of 1,042 animals) than sheep (8 cases out of 2,123 examined, or 0.4 p. 100).

Clinical disease

The clinical signs were similar to what has been described in the West African dwarf breeds (12, 16). The typical signs observed were diarrhoea, fever, mucopurulent oculonasal discharge, congestion at the commissures of the mouth, erosions of buccal and vaginal mucosae, (Figs 1, 2) and conjunctivitis without keratitis. Diarrhoea associated with fever, and oculonasal discharge were the most frequent clinical signs, and were seen in 86.4 p. 100 of cases. Erosions of buccal or vaginal mucosae, respiratory distress and conjunctivitis were less frequently observed.

PPR antigen was demonstrated in the lymph nodes of dead animals from both outbreaks by the AGID test with both PPR and rinderpest hyperimmune sera. Serum samples obtained from febrile animals had no demonstrable antibodies whereas antibodies to PPR were present in convalescent sera from both

| TABLE I Monthly distribution of PPR cases seen in the Large Animal Hospital, Faculty of Veterinary Medicine, A.B.U., Zaria, between 1980 and 1983. |
|-------------------------------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|
| Nb. of animals seen           | 330    | 303    | 223    | 306    | 253    | 238    | 272    | 360    | 257     | 423     | 393     | 413     | 3,871   |
| Nb. and p. 100 of PPR cases   | 0 (0)  | 1 (0.3)| 4 (1.7)| 5 (1.6)| 2 (0.8)| 2 (0.8)| 2 (0)  | 0 (0)  | 0 (0)   | 0 (0)   | 1 (0.3)| 2 (0.5)| 19      |
| p. 100 of PPR cases per month | 0      | 5.3    | 21.1   | 26.3   | 10.5   | 10.5   | 10.5   | 0      | 0       | 5.3     | 10.5    | 100     |

| TABLE II Age-specific morbidity and mortality rates in PPR outbreaks in two herds of Sokoto Red goats in Zaria, Nigeria. |
|-------------------------------|--------|--------|--------|--------|--------|
| Age group                     | Morbidity * (p. 100) | Case fatality * (p. 100) | Morbidity ** (p. 100) | Case fatality ** (p. 100) |
| 0-6 months                    | 7/14 (50.0) | 3/7 (42.9) | 0/0 (0) | 0/0 (0) |
| 6 months - 1 yr               | 2/5 (40.0)  | 0/2 (0)   | 22/22 (100.0) | 14/22 (63.6) |
| 1-2 yrs                       | 3/15 (20.0) | 0/3 (0)   | 8/12 (66.7) | 3/8 (37.5) |
| 2-3 yrs                       | 0/6 (0.0)   | 0/0 (0)   | 0/0 (0)   | 0/0 (0)   |
| Over 33 yrs                   | 1/32 (4.5)  | 0/1 (0)   | 0/0 (0)   | 0/0 (0)   |
| Total                         | 13/52 (25.0)| 3/13 (23.1)| 30/34 (88.2)| 17/30 (56.7)|

* Herd I
** Herd II
outbreaks obtained 4 weeks, 8 weeks and 1 year after recovery.

Cultures of nasal swabs from clinical cases consistently yielded *Pasteurella hemolytica*, *beta*-hemolytic *Streptococci*, *Staphylococcus aureus*, and occasionally non-hemolytic streptococci. Cultures of rectal swabs yielded *Escherichia coli* which were not further characterised.

**Pathology**

Gross and histopathological findings were consistent with those described for in other breeds (12, 16, 19).

**DISCUSSION**

This study indicates that PPR occurs in the Sokoto Red (SR) goats with clinical and epidemiological features very similar to those reported in the West African Dwarf breeds (1, 5, 6, 16, 19). The susceptibility and the severity of the clinical disease, including morbidity and mortality rates seen in the Sokoto Red goats were at par with those reported for the WAD breeds. This is in contrast with the suggestions of DUROJAIYE (6) and AKEREJOLA (3) that the Sahelian breeds are more resistant to infection and clinical disease than the WAD breeds.

Although there is no breed-specific resistance, the low prevalence rate (0.7 p. 100) and the relative absence of clinical reports in the literature suggest that clinical PPR is less common in the SR goats than in the WAD breeds.

The husbandry system may provide some explanation for this apparent paradox. Goats (and sheep) in the Zaria area are generally allowed to roam free, finding forage and water where they can. This extensive system of rearing allows indiscriminate mixing of animals. Young animals may therefore be infected at an age when they still have some maternal immunity, suffer a mild infection, and acquire active immunity. Subsequent infections re-enforce this immunity and the animals will subsequently avoid (clinical) disease. Susceptible animals may accumulate in isolated villages as suggested by TAYLOR (22), or as a result of husbandry practices such as tethering or confinement to compounds as practiced in southern Nigeria. Such susceptible animals when exposed to contact with infected animals in or from the market may experience explosive epidemics. This pattern has been reported in WAD breeds in southern Nigeria by OPASINA.

The higher prevalence of PPR during late dry and early rainy season is most likely attributable to increased stress to the respiratory system due to climatic changes, and reduced fodder. The dry harmattan period preceeding the end of dry season imposes direct stress on the animal; in addition drying up of vegetation forcee animals to wander great distances in search of fodder and water. Congregation of animals at the few available watering holes also increases the chances of transmission of the disease.
The isolation of *Pasteurella* species and *E. coli* in the present study supports the suggestion by other investigators (2, 11, 15, 19) that bacterial pathogens may be involved in the pathogenesis of PPR. The exact role, if any, of bacteria in the disease needs to be more clearly defined.

**REFERENCES**


