Comparative histopathological studies of experimentally infected Sokoto Red Goats with two isolates of *Trypanosoma evansi*


INTRODUCTION

*Trypanosoma evansi* affect a wide range of susceptible domestic and wild animals, and the cause of the disease in all species may be acute, sub acute or chronic, the outcome depending on strain, virulence, host species and duration of endemicity of infection in a region (Audu et al., 1999; Herrer et al., 2002; Lemonade, 1971). A parasite initially and invariably inflicts harm to its host. It is due to the overexploitation of one associate (host) by the other (parasite) and leads to severe injury or untimely death of the host. The central idea of ‘harm’ in host-parasite association can best be understood through pathogenic expression in the host. These pathogenic effects are macroscopic, microscopic, behavioral and or physiological manifestations. In *T. evansi* infection the histopathological lesions or alterations seen in kidneys might be due to either the effects of the parasites or their products and toxins (Onah et al., 1996). Mild glomerular and interstitial nephritis, tubular degeneration, and necrosis were observed in *T. evansi*-infected camels (Enwezor and Sackey, 2005). Ngeranwa et al. (1993) observed infiltration of inflammatory cells in the kidney and detected necrotic foci in the kidneys and lung of two goats infected with *T. evansi*. Kidney is susceptible to blood diseases. Toxins of the parasite and accumulation of immune complex are likely to impair the structure and function of the kidney. In such cases the nature of kidney damage indicates the severity of the disease (Ventura et al., 2002; Njiru et al., 2007). Pneumonia, necrotic foci and congestion of bronchial lymph nodes were reported from *T. evansi*-infected cattle (Bengaly et al., 2002). Lungs of bovine suffering from surra exhibited congestion, oedema, and extensive alveolar emphysema. Haemorrhage, haemosiderosis, alveolar emphysema and oedema and cellular infiltration were observed in rats and mice infected with *T. evansi*.
**Kano Isolate**

The *Trypanosoma evansi* isolate was obtained from the blood of naturally infected camel slaughtered at Kano metropolitan abattoir, North Western part of Nigeria. The Blood samples were obtained in sterilized Bijou bottles containing EDTA. The blood samples were examined by wet film examination. 0.5 milliliters of a blood sample that was positive for *T. evansi* was immediately inoculated intraperitoneally into a rat, the rats were monitored daily for two weeks, when it became positive, infected blood was collected from the rat and inoculated intravenously into a donor goat. This was to multiply the parasites for subsequent infection of the experimental animals.

**Sokoto isolate**

This was obtained from camels presented for slaughter at Sokoto metropolitan abattoir, North Western part of Nigeria. The Blood samples were obtained in sterilized Bijou bottles containing EDTA. The samples were processed at the Department of Parasitology and Entomology, Usmanu Danfodiyo University, Sokoto. Samples that were found positive for *T. evansi* by wet film were intraperitoneally inoculated into two rats; the rats were then transported to Zaria. Blood from positive rats was then inoculated into a donor goat. This was to multiply the parasites for subsequent infection of the experimental animals.

**Experimental animals**

Eighteen apparently healthy Sokoto red goats between the ages of 1 -2 years were purchased from markets around Zaria. The goats were acclimatized for 4 weeks, they were kept in a tick and fly proofed pen in the Department of Parasitology. The goats were dewormed using Lezamisole® they were also screened and found negative for *T. evansi* and other haemoparasites. They were fed grass hay, concentrates mixed with grain offal and water which was supplied ad libitum. The following parameters were monitored during the acclimatization period: daily rectal temperature, body weights taken once week, packed Cell Volume (PCV), and differential leukocytes counts. The eighteen goats were randomly divided into three groups of six animals each; they were tagged based on the following groupings.

- **Group A** infected with Kano isolates of *Trypanosoma evansi*
- **Group C** infected with Sokoto isolates of *Trypanosoma evansi*
- **Group B** non-infected control group.

When the *T. evansi* infected donor goats developed parasitaemia of +++ for two consecutive days they were bleed via jugular vein. The blood was obtained in a beaker containing EDTA as an anticoagulant. Each of the goats in group A and C were inoculated intravenously via the jugular vein with 2ml of blood from the donor goat of each of the isolates.

The goats in all the two groups were allowed to go through the full course of the infection. The goats were observed daily during the course of the experiment. Goats that died during the course of the experiment were sent for post mortem examination. Data obtained from the two groups and the controls were summarized as means. The collated data were compared statistically using student t-test.

**Post mortem evaluation**

Animals that died during the course of the experiment were posted. Gross examination of organs was done and samples of the spleen, kidney, lungs, brain, testis, epididymis, intestines, liver and lymph nodes, were preserved in 10% formal saline processed and embedded in paraffin wax. Sections were later cut at 5µm and stained with haematoxylin and eosin (H&E).

**RESULTS AND DISCUSSION**

Red Sokoto goats infected with the Sokoto isolate of *T. evansi* showed signs of passing large volumes of urine and became recumbent before death; this might be due to reported kidney damage in *T. evansi* infected goats (Dargantes et al., 2005b). The tubular degeneration and the mononuclear cellular infiltration noticed in this in the kidney of the infected goats in this study lend credence to this inference (Plate I).

The pathological response which was largely immunological in this study lending support to the report of Dargantes et al., (2005a), where mostly it was the lymphatic tissues that showed significant changes, the generalized cellular reaction was essentially one of lymphocytes and macrophages both within the lymphatic system itself and in other organs. In this study however, moderate changes were seen in non-lymphatic tissues (Plates I-XIII). These included renal glomerular hypercellularity and testicular aspermia. The testicular aspermia and similar changes in the epididymis suggest that *T. evansi* infection in Sokoto Red goats could affect fertility as earlier reported (Ngeranwa et al., 1991).

The liver has been reported to be one of the most important organs that are largely affected by *T. evansi* infection in goats. Pathological changes such as congestion, haemorrhagic lesions, cellular infiltration in the portal tract, and fatty degeneration of hepatocytes have been reported by various authors (Biswa et al. 2001;
Plate I. Photomicrograph of *T. evansi* (Sokoto isolate) in the blood of a Red Sokoto goat. (Giemsa stain x 400).

Plate II: Photomicrograph of spleen of a Red Sokoto goat infected with Sokoto isolate of *T. evansi*, showing the absence of germinal centers and depletion of lymphoid cells in the white pulp. H &E stain X 400.

Dargantes et al., 2005b). In this study however, most of the reported pathological changes in the liver were not seen. This may be because most of the changes occur during the chronic phase of the disease. The experiment in this study was terminated at early phase of the chronic stage; this is because the goats especially those infected
with Sokoto isolate died at this stage. In lymphatic tissues, the spleen is the most important organ and serves as first line of defense in response to parasitic invasion (REF). The pattern of spleen damage...
Plate V: Photomicrograph of kidney of a Red Sokoto goat infected with Sokoto isolate of *T. evansi* showing tubular degeneration and slight infiltration of mononuclear cells. H & E X 400.

Plate VI: Photomicrograph of testis of Red Sokoto goats infected with Sokoto isolate of *T. evansi* showing generalized testicular degeneration and absence of spermatids. H & E X 400.

Plate VII: Photomicrograph of epididymis of Red Sokoto goats infected with Sokoto isolate of *T. evansi* showing degenerated epididymal epithelium and total absence of spermatozoa in the ducts. H & E X 400.

Plate VIII: Photomicrograph of lungs of Red Sokoto goats infected with Kano isolate of *T. evansi* showing mild congestion and cellular infiltration, thickened alveolar walls, atelectasis, infiltration by mononuclear cells. H & E X 400.

Plate IX: Photomicrograph of small intestine of Red Sokoto goats infected with Kano isolate of *T. evansi* showing diffuse mild inflammatory cells infiltration and a slight submucosal oedema. H & E X 400.

Plate X: Photomicrograph of kidney of Red Sokoto goats infected with Kano isolates of *T. evansi* showing mild congestion and degeneration of the convoluted tubules. H & E X 400.

varies in different parasitic infection.
Plate XI. Photomicrograph of testes of a Red Sokoto goat infected with Kano isolate of \textit{T. evansi} showing generalized testicular degeneration and absence of spermatids. H &E X 400.

Plate XII. Photomicrograph of epididymis of Red Sokoto goat infected with Kano isolate of \textit{T. evansi}. Showing absence of spermatozoa in the epididymal ducts and squamous metaphase of the mucosa. H &E X 400.

Plate XIII. Photomicrograph of lymph node of Red Sokoto goats infected with Kano isolate of \textit{T. evansi} showing depletion of cells from lymphoid follicles and from the medulla. H &E X 400.

Conclusion

In this study, the spleen of goats that died early due to the infection, showed some early pathologic changes such as absence of cells in the germinal centers and depletion of cells in the white pulp.

REFERENCES


