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Pathology of Experimental *Trypanosoma evansi* Infection in Savannah Brown Buck

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Abstract: The pathology of experimental *Trypanosoma evansi* infection in Savannah Brown buck was studied using an isolate obtained from the blood of an infected camel slaughtered at Kano Abattoir, Nigeria. Gross pathological lesions observed included pale carcass with hydro peritoneum, generalized atrophy of the body fats, catarrhal enteritis, hepatomegally with bilaterally congested kidney, congested lungs with red hepatization of the liver. Histopathological lesions observed in the infected Savannah Brown buck included Zenkers necrosis of the myocardium with few mononuclear cellular infiltrations, focal areas and centrilobular necrosis of the liver, hemosiderin-laden macrophages involving the spleen and lymph nodes. There were degenerated seminiferous tubules and degenerated spermatids in the duct of the epididymis. The results of this study showed that the *T. evansi* isolate is pathogenic to the Savannah Brown buck.

Key words: *Trypanosoma evansi*, Savannah Brown buck, pathology

INTRODUCTION

Pathology of the trypanosomosis in different domestic animals has been well documented (Raisinghani *et al*., 1980; Saror, 1980). However, most of the reports were on *T. vivax, T. brucei* and *T. congolense* (Losos and Ikede, 1972; Saror, 1980) with little attention to pathological change occurring in *T. evansi* infection particularly in small ruminants. Some of the reported pathological changes in the organs of *T. evansi* infected rabbits, small east African goats and sheep included degenerative changes in the spleen and lymph nodes, which interfered with the immune response of the host (Uche and Jones, 1992). In goats infected with *T. evansi* lesions observed were hyperplasia of the lymphatic tissue, muscular atrophy, necrotic foci in the liver, kidneys, spleen and lungs with bronchopneumonia (Ngeranwa *et al*., 1993). Similarly slight splenomegaly, generalized congestion and petechae on liver, kidney, spleen lymph nodes heart and lungs in sheep infected with *T. evansi* (Saseendranath *et al*., 1995) orchitis and scrotal edema were also observed. Histopathological changes observed included hyperaemia, fatty degenerations, focal areas of necrosis in the liver, lungs had emphysema with collapse alveoli, mild gliosis in the brain and severe degenerative changes of the seminiferous tubules. Audu *et al*. (1999) had a similar observation in *T. evansi* infected Yankasa sheep with additional changes, which included centrilobular necrosis of the liver and haemosiderosis.

The present study was carried to investigate the pathological changes in Savannah Brown buck following infection with the *T. evansi* isolate.

MATERIALS AND METHODS

Nine Savannah Brown male goats aged between 12 and 18 months were obtained locally and conditioned for a period of 6 weeks. They were screened against the common haemoparasites and helminths prior to the experimental infections. The animals were kept in fly-roofed pens and maintained on hay, concentrates (cotton seed mixed with grain offal's) and salt licks. Water was supplied *ad libitum*. Baseline pre-infection data was obtained.

At the end of six weeks of conditioning the goats were divided into 2 groups. Four goats constituted the non-*T. evansi* infected control group and the remaining five goats formed the *T. evansi* infected group. Number tags were attached to each goat for the purpose of identification. The *T. evansi* isolate used in this study was obtained from the blood of a naturally infected camel slaughtered at Kano abattoir. Samples of the infected blood were obtained in EDTA. Transported over ice

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immediately to the laboratory in the Department of Parasitology and Entomology, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria and examined using wet film method. Ten Albino rats were inoculated with the infected camel blood in order to harvest the trypanosomes in sufficient number for subsequent infection of the experimental goats.

When the *T. evansi* infected rats develop parasitaemia of 3 + (three "pluses"; 20 trypanosomes per×40 field) for 3 consecutive days, they were bled and the pooled blood in EDTA was subsequently diluted with phosphate Buffered Saline Glucose (PSG) prior to inoculation in the experimental goats. Each of the goats in the infected group was intravenously inoculated via the jugular vein using 1 mL of the infected rat blood containing approximately 3×10^6 trypanosomes as estimated using the rapid "Matching" method of Herbert and Lumsden (1976). The inoculated goats were allowed to go through the full course of the infection.

All the goats were observed closely from the day of inoculation. Post-mortem examination was conducted in the Department of Pathology and Microbiology, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria on death of the experimental animals and specimens were collected in 10% buffered neutral formalin. Paraffin sections of 5mm thickness were prepared and stained with haematoxylin and eosin (H and E) for Histopathology. Samples obtained from control Savannah Brown bucks were similarly processed.

RESULTS

The carcasses of the infected Savannah Brown bucks were pale an indication of anaemia. Generally, there was hepatomegally with congestion of liver, kidney and lungs. Catarrhal enteritis of the intestine was also observed with areas of red hepatization of the liver.

Mesenteric and mandibular lymph nodes were grossly enlarged in most of the infected animals. There was also hydro peritoneum with atrophy of body fats in most of the infected goats.

Histopathological findings: Considerable histopathological changes occurred in most the tissues and organs of the infected Savannah Brown bucks. However such changes were not observed in the tissues of the control goats.

Heart: Histopathologically the myocardium of the infected Savannah Brown bucks showed Zenkers necrosis of the myocardial cells with few mononuclear cellular infiltrations (Fig. 1).

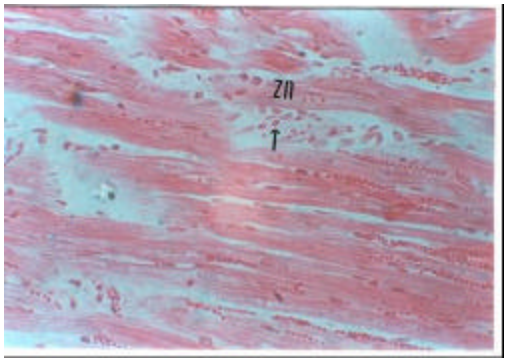

Fig. 1: Photomicrograph of the heart of *Trypanosoma evansi* infected Savannah Brown bucks that died 27 days post-infection showing Zenker's necrosis (Zn) of myocardial cells with mononuclear cellular infiltrations (Arrows) H and E×400

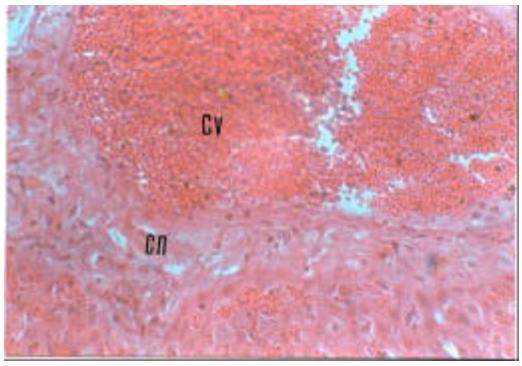

Fig. 2: Photomicrograph of the liver of *Trypanosoma evansi* infected Savannah Brown bucks that died 37 days post-infection showing centrilobular necrosis (Cn) and congested blood vessel (Cv) H and E×400

Liver: The liver of the infected Savannah Brown bucks showed centrilobular necrosis of the hepatic cells with congested blood vessels (Fig. 2).

Kidney: The kidneys of the infected goats showed atrophied glomeruli, dilated bowman's space, renal tubular necrosis and congested blood vessels (Fig. 3).

Spleen: The spleen of the *T. evansi* infected goats showed hyperplastic splenic follicle (Fig. 4).

Other findings: The testis of the infected goats showed degenerated spermatogenic cells and the epididymis showed degenerated spermatids in the epididymal duct with degenerated epithelial lining of the duct.

The lymph nodes of the infected Savannah Brown bucks showed a massive proliferation of lymphocytes

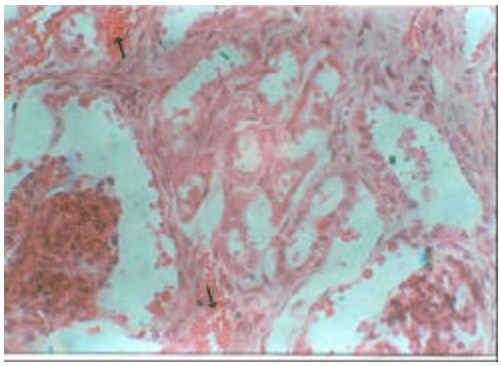

Fig. 3: Photomicrograph of kidney of *Trypanosoma evansi* infected Savannah Brown Buck that died 37 days post-infection showing atrophied glomeruli, dilated Bowman's space, renal tubular necrosis and congested blood vessels (Arrows) H and E×400

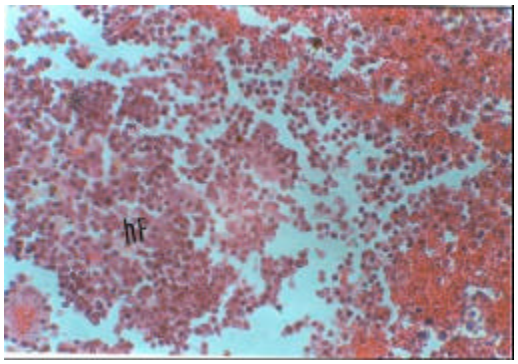

Fig. 4: Photomicrograph of the spleen of *Trypanosoma evansi* infected Savannah Brown Buck that died 37 days post-infection showing hyperplastic splenic follicle (hf) H and E×400

with hemosiderin-ladened macrophages. In the pituitary glands there was necrosis of the chromophil and chromophobe cells while the adrenal glands focal areas of necrosis were observed in the zona glomerulosa, zona fasciculata and zona reticularis. Similarly no significant changes were observed in tissues of the control goats.

DISCUSSION

The results of this study have shown that the major gross pathological features of *T evansi* infection in Savannah Brown buck were paleness of the carcasses, hydro peritoneum, hepatic congestion, pulmonary congestion and congestion of the kidneys, hepatomegally, catarrhal enteritis and generalized atrophy of the body fats. These findings were in agreement with previous reports of Strivastava and Ahluwalia (1973) on *T. evansi* infection in pigs, Ikede *et al.* (1977) on *T. vivax* of different domestic animals, Maikaje *et al.* (1991) on *T. evansi* infection in Uda sheep, Saseendranath *et al.* (1995) on *T. evansi* infection in sheep and Audu *et al.* (1999) on *T. evansi* infection in Yankasa sheep.

The histopathological lesions in this study included Zenkers necrosis of the myocardial cells with few mononuclear cellular infiltrations, centrilobular necrosis of the hepatic cells with focal areas of necrosis of the adrenal and pituitary glands. Splenic hyperplasia with haemosiden-ladened macrophages in the spleen and lymph nodes, epididymal and testicular degenerative changes. These observations agreed with earlier reports of Losos and Ikede (1972) on *T. congolense, T. brucei, T. rhodensiense* and *T. gambiense* in domestic and laboratory animals, Strivastava and Ahluwalia (1973) on *T. evansi* infection in pigs, Raisinghani *et al.* (1980) on *T. evansi* infection in pigs and Audu *et al.* (1999) on *T. evansi* infected Yankasa sheep.

With these findings, we can conclude that the *T. evansi* isolate used in this study was pathogenic to the Savannah Brown buck.

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