

Histological Changes in Organs of Rats Infected with *Trypanosoma congolense*

C.C. Ohaeri

Department of Biological Sciences, Michael Okpara University of Agriculture Umudike,
P.M.B. 7267, Umuahia Abia State, Nigeria

Abstract: Five albino rats were infected with *Trypanosoma congolense* and killed on post infection day 28 by ether anaesthesia. Liver and kidney were removed weighed and histologically processed. All the inoculated rats developed trypanosomiasis which was characterized by a fluctuating parasitaemia and marked changes in organs. The histological changes observed in the liver were enlarged hepatocytes that contained numerous vacuoles and dissociated hepatic cords when compared with uninfected rats that revealed normal morphology. Kidney section of infected rats showed thickening of basement membrane as compared to uninfected rats. It also showed cell infiltration by macrophages and lymphocytes around glomeruli and blood vessels. The observation of cell infiltrations in the organs suggests that trypanosome infection is associated with histopathological changes that contribute to chronic debilitation.

Key words: Histology, organs, *Trypanosoma congolense*, rats, infection, liver, kidney

INTRODUCTION

The pathology of trypanosomiasis has been reviewed (Fiennes, 1970; Murray, 1974). Proliferative changes in the lymphoid organs of *T. vivax* (Masake and Morrison, 1981) and *T. brucei* (Moulton and Sollod, 1976) infected cattle have been reported with little information as regards to infection with *T. congolense*.

Trypanosomiasis is characterized by chronic debilitation. In the advanced form, gross lesions, anaemia, oedema of subcutaneous tissues and lymphoid organs and serous atrophy of fat are commonly observed (Fiennes, 1970; Biryomumaisho *et al.*, 2003). Subcutaneous oedema is particularly prominent. The liver may be enlarged and oedema of lymph nodes is often seen in the acute disease condition but they may be reduced in size in the chronic disease state. The spleen and lymph nodes may be swollen, normal or atrophic in the course of the disease.

Moulton (1986) found accumulation of plasma cells and lymphocytes in medullary sinuses and cords of lymph nodes as well as haemolymph nodes of the spleen of *T. congolense* infected cattle.

Necrosis of the kidneys and heart muscle and sub serous petechial haemorrhages commonly occur. Gastroenteritis is common and focal polioencephalomalacia may be seen and this probably results from ischaemia (occlusion of arteries) due to enormous build-up of the parasites in the terminal capillaries of the brain.

A remarkable lesion is anaemia but with cellular invasion of various organs, inflammation, degeneration and necrosis become pronounced. Most body tissues show marked proliferative changes (Moulton, 1986). This study reports on the histological findings of the liver and kidney of rats infected with *T. congolense*.

MATERIALS AND METHODS

Experimental rats: About 16 clinically healthy male albino rats aged 7-9 weeks were purchased from National Institute for Trypanosomiasis Research (NITR), Vom Plateau State, Nigeria and used for the investigations. The rats were allowed to acclimatize for 1 week prior to start of experiment.

All the rats used in this study were screened and found negative for trypanosome infection before the experiment. The rats were divided into two groups of 8 rats per cage. (Group 1: infected and Group 2: not infected). The two groups were housed in separate plastic cages and fed growers mash (Top Feeds Nig. Ltd) and drinking water *ad-libitum* throughout the 28 days of study.

Trypanosome infection of experimental rats: At the end of acclimatization period, *T. congolense* (obtained from NITR, Vom) infected blood was taken from the donor rat with fulminating parasitaemia by cardiac puncture after anaesthetizing with ether. The number of parasites was

assessed by the haemocytometer technique (Sannusi, 1977) and was diluted with phosphate glucose buffered physiological saline solution. Group 1 rats were given 5×10^3 trypanosomes in 0.5 mL volume through intraperitoneal (i.p.) route. About 5 rats were retained as uninfected controls. The experiment was terminated 4 weeks post infection.

Collection of blood samples from experimental rats:

Blood smears from tail vein for determining parasitaemia were made at weekly intervals for a period of 4 weeks study. The number of trypanosomes was determined at designated times by the haemocytometer technique (Sannusi, 1977) and the results were expressed as mean number of trypanosomes per mL of blood.

Histology of rats tissues: Pairs of infected as well as pairs of uninfected rats were selected randomly and sacrificed on post infection day 7, 14, 21 and 28. Then samples of liver and kidney were weighed using weighing balance (Triple balance) preserved in 10% formalin and processed for light microscopy through a process of fixation, dehydration, clearing and embedding in paraffin wax and thereafter sectioned.

Sections were stained with haematoxylin and eosin (H and E) according to the method of Barker and Silvertown examined by light microscopy and photomicrographs taken with the Olympus microscope using 35 mm black and white film.

RESULTS AND DISCUSSION

Effect of *Trypanosoma congolense* infection on histology of liver and kidney in rats: Parasitaemia was confirmed during the course of the study ranging from $130 \pm 0.4 \times 10^6$ mL⁻¹ of blood on day 7- $320 \pm 1.2 \times 10^6$ mL⁻¹ of blood on day 14 post infection (Table 1) with an irregular

fluctuation but gradual fall to $132 \pm 0.5 \times 10^6$ mL⁻¹ of blood on day 28 post infection. The results of histological section of Liver of *Trypanosoma congolense* infected and uninfected rats stained by Haematoxylin Eosin (H and E) are shown in Fig. 1a, b, respectively. Infected rats liver revealed enlarged hepatocytes with numerous vacuoles and dissociated hepatic cords when compared with uninfected rats that showed normal morphology. The weight of the liver was greater in infected rats as compared to uninfected controls (Table 2).

Kidney section of infected rats showed thickening of basement membrane as compared to uninfected rats (Fig. 2a, b). Intense cell infiltration by macrophages and lymphocytes around glomeruli and blood vessels were also evident.

Information on the histopathology of kidney and liver cells in trypanosome infection is scarce in the literature, however Saror and Coles (1976) found accumulation of mononuclear cells around the glomeruli and blood cells in kidney, disorganized hepatic cords and hyperplastic lymph nodes in *T. congolense* infected cattle.

Table 1: Parasitaemia in rats infected experimentally with *Trypanosoma congolense*

Weeks (post infection)	Mean parasitaemia \pm SEM $\times 10^6$ mL ⁻¹ of blood
1	130 \pm 0.4
2	320 \pm 1.2
3	250 \pm 0.7
4	132 \pm 0.5

Table 2: Weight of liver and kidney of rats infected with *Trypanosoma congolense*

Organs	Infected rats of days post infection		Uninfected rats of days from start of experiment	
	14	21	14	21
Liver	3.5 \pm 0.05	5.0 \pm 0.06	2.1 \pm 0.15	2.3 \pm 0.04
Kidney	0.7 \pm 0.10	0.9 \pm 0.22	0.4 \pm 0.08	0.5 \pm 0.03

Values are mean \pm SEM in grams at experimental days 14 and 21

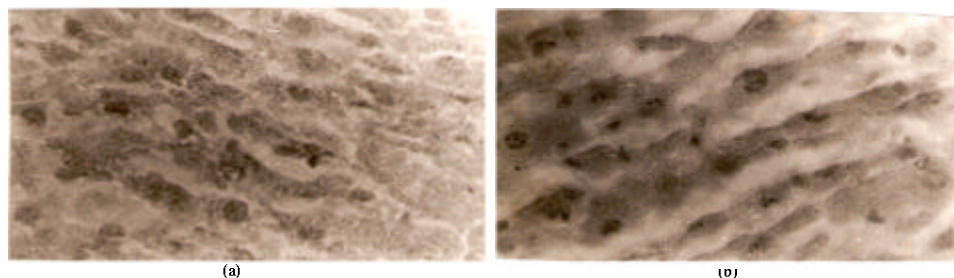


Fig. 1: Photomicrograph of rats Liver at 21 days post infection: (a) *Trypanosoma congolense* infected rat liver showing enlarged hepatocytes that contained numerous vacuoles and dissociation of hepatic cords. (b) Uninfected rat liver showing normal liver histology

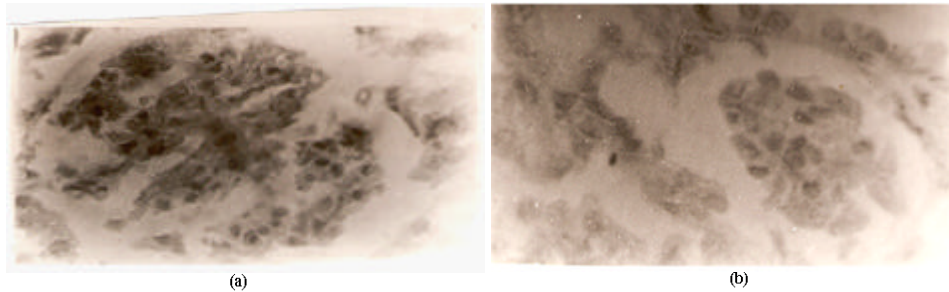


Fig. 2: Photomicrograph of rats kidney at 21 days post infection: (a) *Trypanosoma congolense* infected rat kidney showing thickened basement membrane and infiltration of macrophages and lymphocytes. (b) Uninfected rat kidney showing normal kidney histology

CONCLUSION

The results of these histological findings indicate that there is massive macrophage and lymphocyte proliferation thus disrupting the structural integrity of the cells of these organs. The observation revealed continuous massive proliferation of macrophages and lymphocytes around the glomeruli and blood vessels of the kidney throughout the course of the study which suggest a depletion of lymphoid cells. These proliferative changes were disorganized and the organs of both the kidney and liver were therefore distorted.

This is most likely the cause of derangement in many blood proteins like albumins, fibrinogen and coagulation proteins found in trypanosome infected animals (Ohaeri, 2005) since, hepatocytes are responsible for their synthesis. This will also result in lagging behind of the uptake of bilirubin by hepatocytes which have been impaired by trypanosome infection against its rate of production by erythrocyte phagocytic destruction, thus leading to increased concentration of bilirubin in the blood. Histopathological changes has also been demonstrated (Moulton, 1986) in lymph nodes of cattle infected with *T. congolense* in which the medullary cords were thickened and contained numerous lymphocytes and plasma cells. Similar changes were also seen in the spleen. The liver increased in weight on post infection day 14, attaining maximum weight on day 21 post infection and the weight was greater than that of the uninfected group throughout the period of the experiment.

ACKNOWLEDGEMENTS

The researches is grateful to the Federal Government of Nigeria for funding part of this study under the Federal Government postgraduate scholarship (Ref. FSBA/FGSS: PG/027GA). This study formed part of a Ph.D thesis submitted to Michael Okpara University of Agriculture, Umudike, Abia State Nigeria.

REFERENCES

- Biryomumaisho, S., E. Katunguka-Rwakishaya and C.M. Rubaire-Akiiki, 2003. Serum biochemical changes in experimental *Trypanosoma congolense* and *Trypanosoma brucei* infection in Small East Africa goats. *Veterinarski Arhiv*, 73: 167-180.
- Fiennes, R.N.T.W., 1970. Pathogenesis and Pathology of Animal Trypanosomiasis. In: *The African Trypanosomiasis*, Mullingan, H.W. (Ed.). Allen and Unwin, London, pp: 729-950.
- Masake, R.A. and W.L. Morrison, 1981. An evolution of the structural and functional changes in the organs of boran cattle infected with *Trypanosoma vivax*. *Am. J. Vet. Res.*, 42: 1738-1746.
- Moulton, J.E. and A.E. Sollod, 1976. Clinical, serologic and pathogenic changes in calves with experimentally induced *Trypanosoma brucei* infection. *Am. J. Vet. Res.*, 37: 791-802.
- Moulton, J.E., 1986. Structural and functional changes in the organs of cattle infected with *Trypanosoma congolense*. *Tropical Veterinarian*, 4: 97-106.
- Murray, M., 1974. The pathology of African trypanosomiasis. *Prog. Imm.*, 11: 181-192.
- Ohaeri, C.C., 2005. Tsetse and trypanosomiasis in parts of Abia State, Nigeria. Ph.D. Thesis, Michael Okpara University of Agriculture, Umudike, Abia State Nigeria.
- Sannusi, A., 1977. Enumeration of trypanosomes in the haemocytometer, problem and prospects. *Proceedings of 1st Annual Conference of the Nigerian Society of Parasitology*, Aug. 30, Ibadan, Nigeria, pp: 4-4.
- Saror, D.I. and E.H. Coles, 1976. Clinicopathological aspects of *Trypanosoma congolense* infection in zebu cattle. *J. Nig. Vet. Med. Assoc.*, 5: 41-44.