# PATHOLOGICAL STUDIES ON VISCERAL ORGANS OF EXPERIMENTALLY INFECTED ALBINO RATS.

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## Abstract

This study was carried out to investigate the pathological effects of *Trypanosoma evansi* infection on the visceral organs of albino rats. Twenty albino rats were used for this study and grouped into A, B, C, D. and E (control). Except those in control group, each animal was intraperitoneally infected with 0.5 ml containing 4.0 x$10^6$ trypanosomes/ml. Experimental animals were monitored daily and be given food and water *ad libitum.* The results showed that the isolates were virulent to the experimental animals. At the end of the experiment, animals were euthanized and visceral organs were collected. The organs were macroscopically examined and impression smear was made from each organ which revealed the presence of the parasites. From each organ, a tissue samples was collected and stored in 10 % neutral buffered formal saline for later use for histopathological study. The results revealed that there was congestions in the lungs of the infected rats but other organs shows normal structures. We conclude that the animals suffers acute form of the disease which is inconformity with characteristics of *T. evansi* infection. Thus, further research needs to be carried out with other species of the parasites to elucidate on other areas.



**Key Words: *T. evansi,* Albino rats, Pathological Studies**

## Introduction

Trypanosomes are parasites of both man and animals causes a disease known as trypanosomiasis. These wide range parasites include; *Trypanosoma brucei brucei, Trypanosoma brucei rhodesiense, Trypanosoma brucei gambiense, Trypanosoma vivax, Trypanosoma congolense* and *Trypanosoma evansi* (El-Amin *et al*., 1999). *T. evansi* causes a disease commonly known as ‘‘Surra’’ (Gill 1991), as a result of biting of flies of the Genus; *Tabanus, Stomoxys, Haematobia, Chrysops* and *Hippoboscids* thereby transmitting it mechanically (Desquesnes, 2004) to the host. In addition to these flies, it was also reported that the haematophagous bats play a role as natural hosts and vectors of *T. evansi* in Latin America (Quieroz *et al.*, 2000) and also, ingestion

of meat from infected carcasses by carnivores can result in infection, and in South America, vampire bats such as *Desmodus rotondus* (Hoare, 1972) are said to be of importance both as reservoirs of infection and as vectors (Luckins, 1998).

It was experimentally proved that, *T. evansi* is highly pathogenic to laboratory animals (rat, mice and rabbit) (Biswas et al. 2001; Singla et al. 2003) and cause severe histopathological damages to the infected animals. This occur as a result of utilizesation of glucose and oxygen by the parasite for its growth and multiplication, thereby causing depletion of these metabolites leading to degenerative changes in the host (Bal. *et al.,* 2012). Further changes develop in the organs either due to cellular damage caused by toxicants released by the parasite, or due to immunological reactions (Bal. *et al.,* 2012). Though *T. evansi* is a haemoprotozoa, visceral forms have been reported in heart, optic lobes, cerebrum, liver, kidney and lungs (Singla 2001). The cellular pathological lesions due to infection with *T. evansi* are mainly considered as a condition which results from the consequence of the immunological reaction triggered by trypanosomal antigens rather than the direct effects of the parasite (Dargantes *et al.*, 2005a). Immunologic products such as immune complexes, cytokines (i.e., interferons, interleukins, chemokines and tissue necrotic factors) and nitric oxide produced in response to presence of trypanosomes may mediate cellular damage in infected host (Baral *et al.,* 2007; Saleh *et al.,* 2009).

In addition, consolidation of the lungs, testicular enlargement and aspermia, splenic hyperplasia, enteritis, haemosiderosis in the spleen, liver, bone marrow and lungs, renal glomerular hypercellularity and cell depopulation on the spleen, bone marrow and lymph nodes in the late stage of infection have been observed (Dargantes *et al.,* 2005a).

The present study was designed to evaluate the gross and microscopical pathology of cattle strain of T. evansi in experimentally infected mice.

## MATERIALS AND METHODS

### Study Area

The study was conducted in Sokoto, state capital of Sokoto State located in latitude 13°04′N and longitude 5°14′E in the extreme northwest of Nigeria, near the confluence of the Sokoto River and the Rima River. The city covered land mass area of 125,971 square kilometers with 5,806,952 (NPC, 2020) population. Sokoto is the capital of Sokoto State (and its predecessor, the North-western State) (FAO, 2006).



### 3.2 Sample Collection

Blood samples from two hundred (200) camels at Sokoto central abattoir were collected using EDTA bottle. Ten (10) mls of blood was collected from each camel and transferred into EDTA (Ethylene Diamine Tetra Acetate) bottle. The blood samples were transported to Parasitology Laboratory, Department of Biological Sciences (Usmanu Danfodiyo University, Sokoto) immediately to detect the presence of the parasite (*T. evansi*) using wet mount, thin and thick blood smears.

### Experimental Animals

Twenty albino rats containing both sexes (male and female) were obtained from Animal Garden, Usmanu Danfodiyo University, Sokoto and kept to acclimatize for two weeks.

Experimental rats were kept in cages and fed water and pelleted food *ad libitum*. They were divided into groups; A, B, C, D and E.

**Animal Inoculation**

Inoculation was achieved with the aid of syringe and needle as described by Onyeyili, (1994). One (1ml) ml of fulminating blood from an infected camel was diluted with 9mls of normal saline. Out of this, 0.5 ml was pulled containing 4.0 $x10^6$trypanosomes/ml and inoculated intraperitoneally into the rats in Group A. Methylated spirit was used to clean the inoculation area before and after inoculation to avoid secondary infections (Onyeyili, 1994).

**Post Infection Monitoring**



All the experimental rats were monitored daily and given food and water *ad libitum.* Post infection study begins immediately six hours post inoculation (P.I), to detect the emergence of parasite. In determining the infection take-up, a drop of blood was collected from tail of each rat pre-sterilized with methylated spirit onto a clean glass slide, covered with cover slip, and then viewed under a microscope (x40).

**Impression Smear/Histopathology**

Impression smears of the internal organs of experimental rats of Group A from heart, lungs, kidneys, spleen, and testes indicated the presence of trypanosomes in peripheral blood of these organs, but the result of histopathology showed absence of any trypanosome in heart, lungs, kidneys, spleen, and testes but the lungs showed congested alveoli with numerous inflammatory cells within alveoli sac. For animals in Group B, the impression smears from the internal organs of heart, lungs, kidneys, spleen, and testes showed the presence of trypanosomes in peripheral

blood of these organs. Histopathology results showed the absence of any trypanosome parasite in heart, lungs, kidneys, spleen, and testes in all the groups (Plates 5-10).

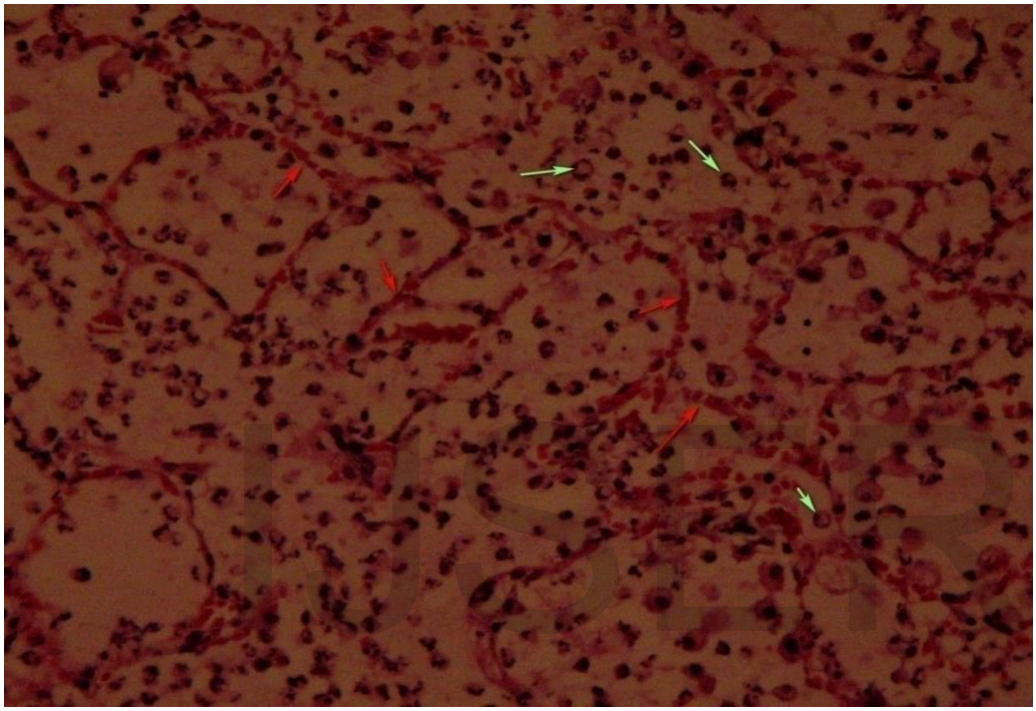

Plate 5: Transverse section photomicrograph of Lungs showings congested alveoli (Red arrow) with numerous inflammatory cells within the alveoli sac (Green arrow) and no parasite seen. H&E (x200)

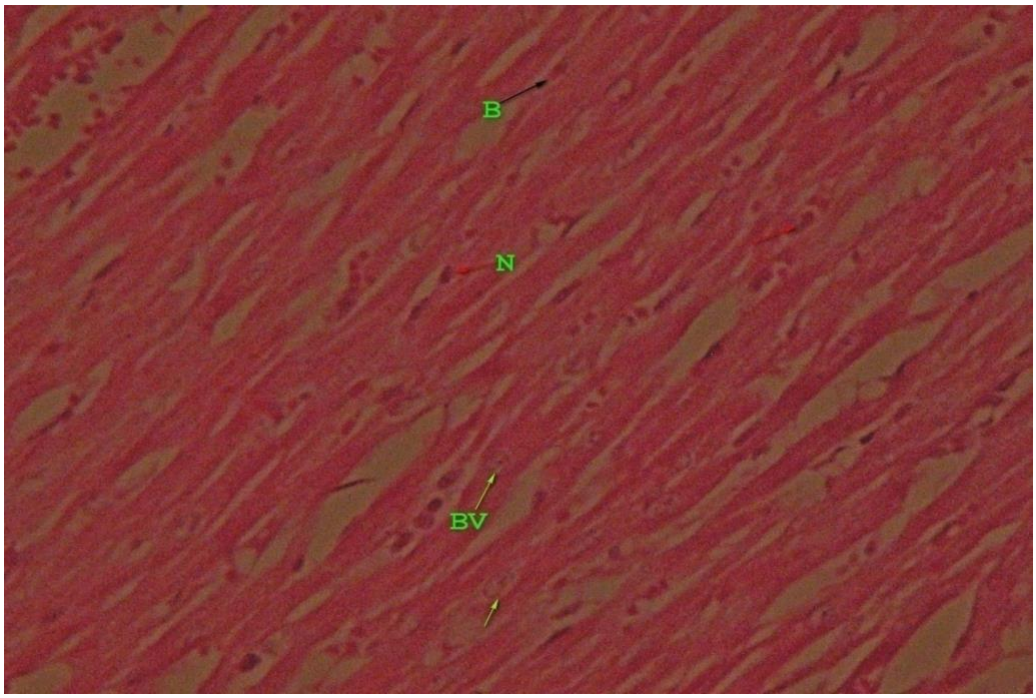


Plate 6: Transverse section photomicrograph of cardiac muscle showings normal muscular orientation (Red arrow) with normal nucleus (N) and blood vessels (BV) and no parasite seen. H&E (x200)

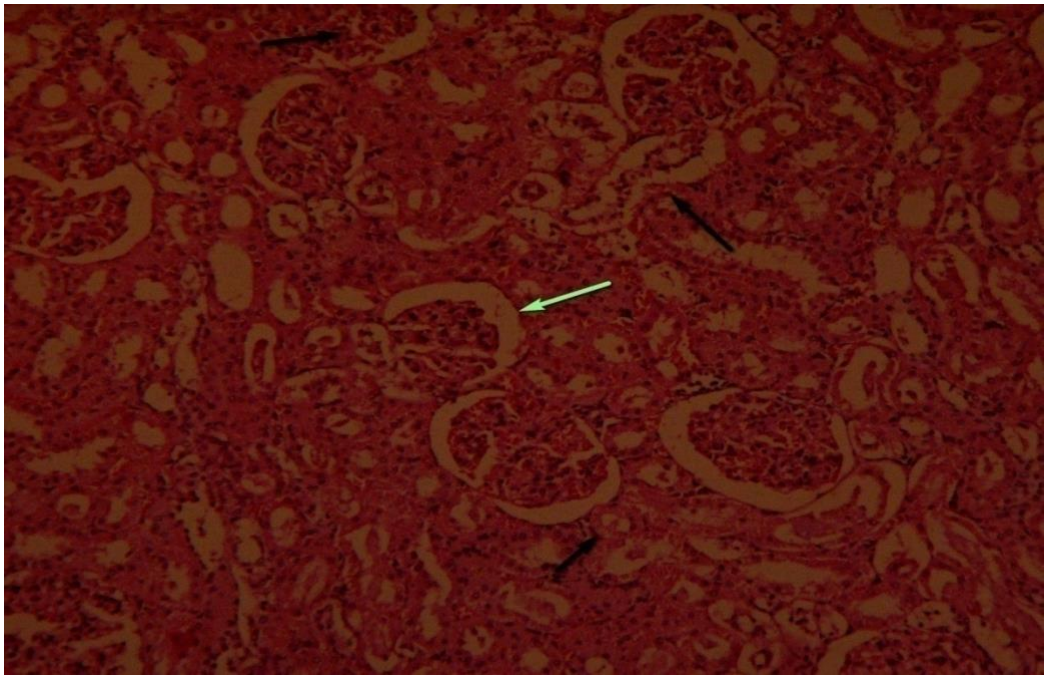

Plate 7: Transverse section photomicrograph of Kidney showings normal glomerulus (Green arrow) with normal collecting ducts (Black arrow) and no parasite seen. H&E (x200)

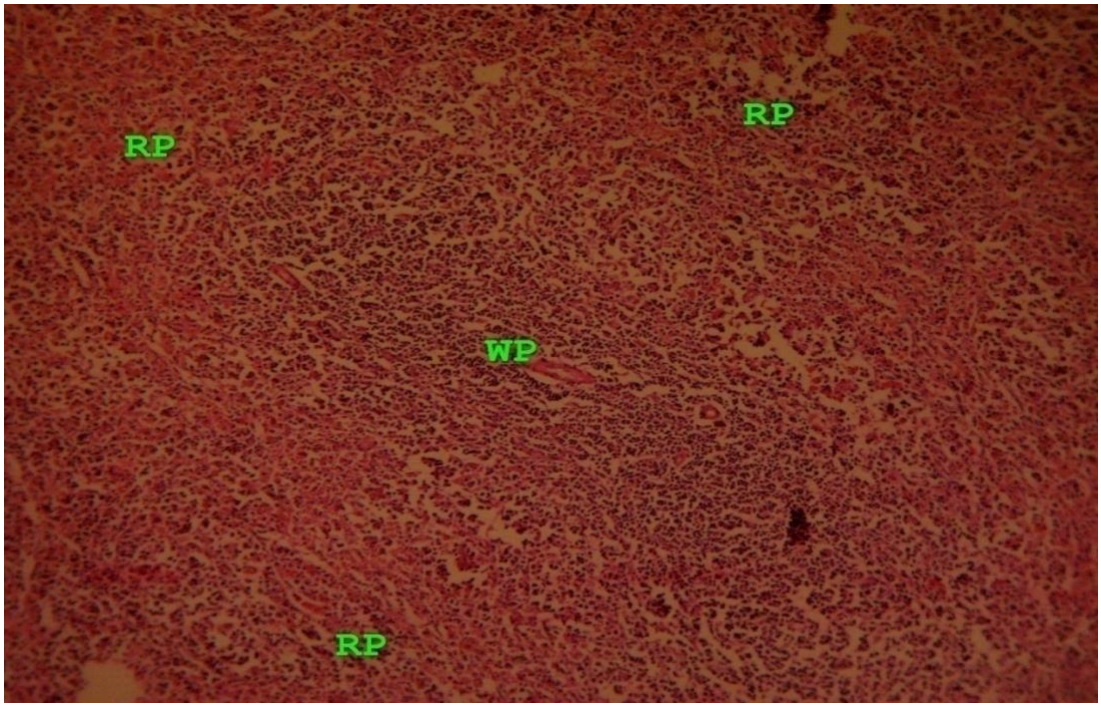


Plate 8: Transverse section photomicrograph of Spleen showing normal cells with two zone Red pulp (RP) and white Pulp (WP) with normal blood vessels, no parasite seen. H&E (x200)



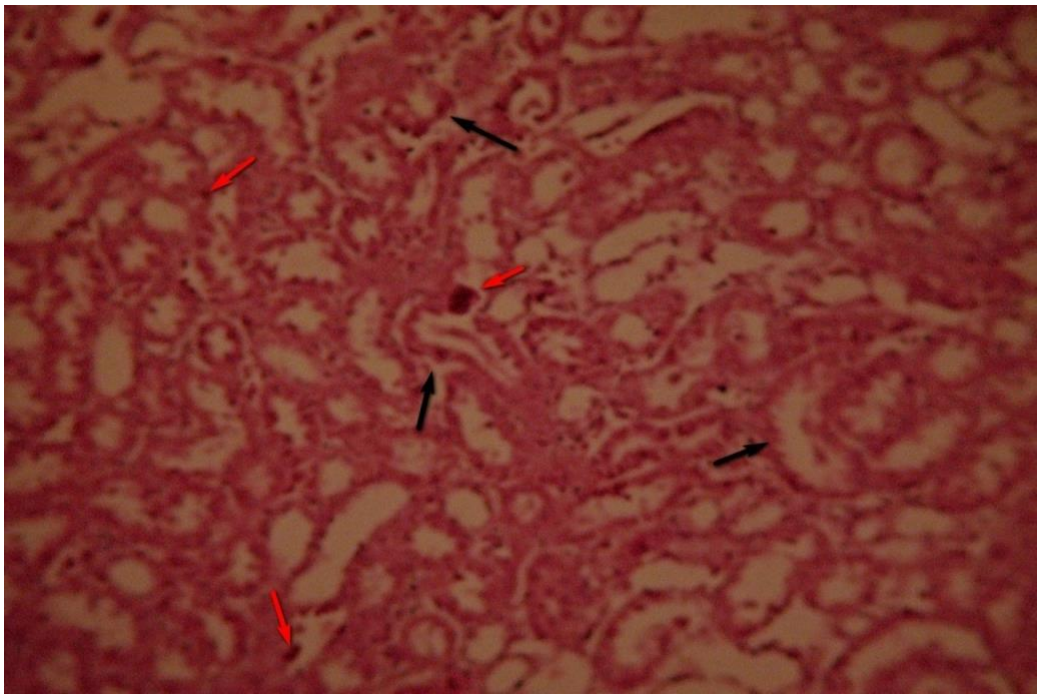

Plate9: Transverse section photomicrograph of Testis showings normal seminiferous tubules with cells at various stage of mitotic differentiation (Black arrow) and numerous blood vessels (Red arrow) and no parasite seen. H&E (x200)

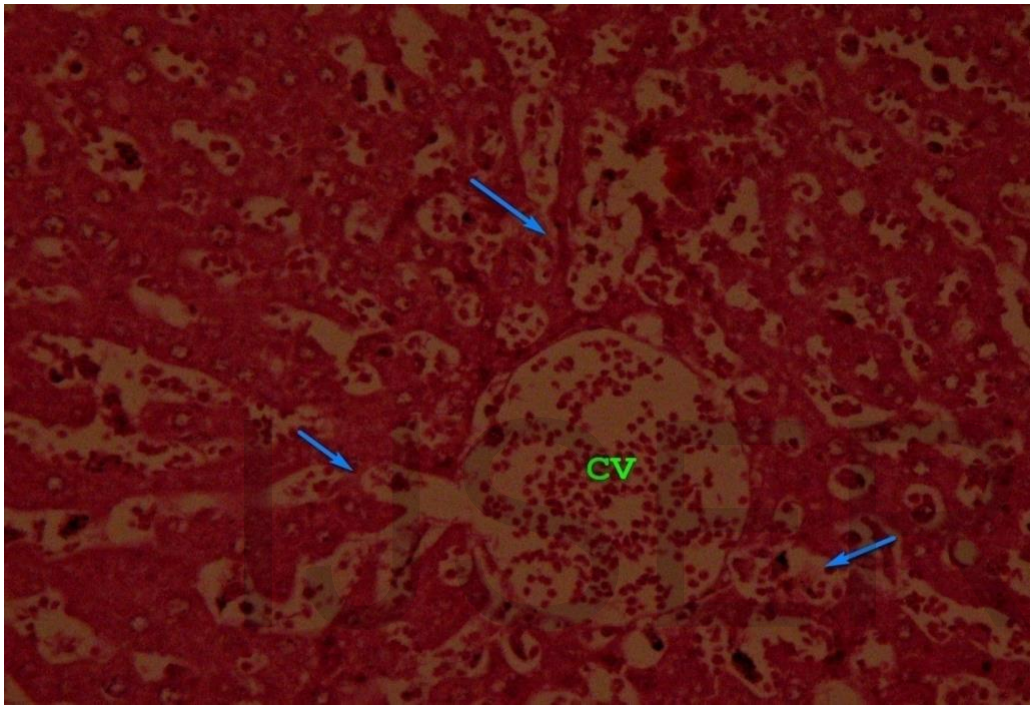


Plate 10: Transverse section photomicrograph of liver showing normal hepatocyte (black arrow), with sinusoidal congestion (blue arrow) with normal clear central vein (CV), no parasite seen. H&E (x200)

Impression smears showed the presence of *T. evansi* in the peripheral blood of internal organs (heart, lungs, kidneys, liver, spleen, and testes) of infected rats in both groups. This agrees with previous findings of Jatau (2010), who also detected (*T. evansi*) in impression smears of brain, heart, lung, liver, spleen, kidneys, and testes, in rat experimentally infected with *T. evansi*. In this study, no lesions were detected in any of the sampled organs in both groups (A and B).This observation agrees with the finding of Ogbaje *et al*., (2011) who reported that, the parasites (*T.*

*evansi*) were not able to cause any lesion in infected West African dwarf goats. The occurrence of congestions in the lungs of the infected rats has also been reported in other trypanosome infections (Audu *et al.,* 1999) in which *T. brucei* were located in tissues and in the blood vessels during acute infections (Losos and Ikede, 1972).

The absence of histopathological lesions in the organs of the infected rats contrasted findings by Audu *et al.* (2004) who found gross pathological and histopathological changes in organs of giant rats infected with *T. brucei*. Similarly, Ikede and Losos, (1972), Omotainse and Anosa, (1992) observed histopathological changes in *T. brucei* infected cattle, donkeys, horses, dogs, and rabbits.

We came to the conclusion that, the presence of *T. evansi* parasites in the impression smear of these organs coupled with the absence of clear histopathological changes of such organs, may be an indication that the rats suffered acute form of the disease, having died before the invasion of the parasites into the organs. Hence, this affirmed one of the characteristics of *T. evansi* infection. There's need for further research to be carried out using same or different species on same/different animals with the aim to elucidate on other areas based on the effects of *T. evansi* on experimentally infected albino rats.

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