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HISTOPATHOLOGICAL CHANGES ASSOCIATED WITH EXPERIMENTAL *TRYPANOSOMA CONGOLENSE* INFECTION IN ALBINO RATS

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ABSTRACT

This work was carried out to evaluate the Histopathological lesions associated with experimental infection of *Trypanosoma congolense* in albino rats. Ten apparently healthy albino rats were randomly distributed into two groups (A and B). Group A (4 albino rats) which served as control while Group B (6 albino rats) to be infected with *T. congolense* organism. Each member of group B was inoculated intraperitonially with $1x10^6$ trypanosomes per 0.5 ml of blood obtained from donor mouse and monitored for changes in parasitemia. The peak parasitemia were observed at day 9 PI, the rats were humanely sacrificed and vital organs (brain, lungs heart, liver, spleen, kidney, and intestinal tract) were examined grossly and harvested for histopathological examination using H&E stain. The results revealed grossly pericarditis, hepatitis, nephritis, splenomegaly, petechiae haemorrhages at the intestinal mucosa and histopathological lesions observed were acute congestion of meningeal capillaries, perivascular Oedema, Neuronecrosis (vaculations), gliosis in the brain, congestion, Hyperplasia of the alveolar wall, perivascular infiltration of lymphocytes around small blood vessels (venules and arterioles) and thickened inter-alveolar septae infiltrated with mononuclear cells in the lungs, Vasculitis, mononuclear cellular infiltration, myocardial necrosis and haemorrhagic myocardium in heart, haemorrhages and acute congestion along with segregation of lymphoid follicles, hyperplasia, reticulo endothelial cells and considerable amount of amorphous haemosiderin granules in the spleen, Cellular infiltration of the portal triad and vasculitis in the liver, tubular necrosis, glomerulonephritis and destruction of Bowman's capsule in the kidney and petechae haemorrhages, necrosis of the intestinal villi, formation of pseudomembrane in the intestinal lumen and depletion of germinal centre of the intestinal tract. It can be concluded that experimental infection of albino rats with *Trypanosoma congolense* produced significant pathologic changes in vital organs.

Key words: *T. congolense*, albino rats, vital organs, gross lesion, histopathological lesions

INTRODUCTION

Trypanosomosis is a disease caused by extracellular haemoprotozoan parasites that survived in the blood stream of the host by complex evasion mechanism, including antigenic variation of the variant surface glycoprotein (VCG) (Adamu *et al.*, 2009), immunosuppression (Bissalla *et al.*, 2007). The disease in man and animal is characterized by acute, sub-acute and chronic courses with signs of fever, anemia, emaciation and high mortality (Wang *et al.*, 2002; Samuel *et al.*, 2016). Transmitted cyclically by tsetse fly (*Glossna spp*) (Prowse, 2005). It has also been reported that mechanical transmission of *T.vivax* and *T.evansi* does occur (Davies, 1997) while several

species of trypanosomes were reported to be transmitted transplacentally (Ijagbone and Agbede, 2002). Trypanosomosis has played an important role in tropical Africa, hence the World Health Organization listed major problem affecting man and animals (WHO, 1975).

Several pathological changes associated with Trypanosoma infection in have been reported in different species in rats (Doyle *et al*., 2017), Nagle *et al*., (1980), rabbits Ogunsanmi *et al*., (1994) in sheep, Mbaya *et al*., (2011) in camel, Morrison *et. al.,* (1981) in dog, Allam *et al*., (2011) in pigs and Samuel *et al.,* (2016) in donkeys. The aim of this study is to examined the Histopathological changes associated with *Trypanosoma congolense* infection in albino rats

MATERIALS AND METHODOLOGY

Experimental animals

Ten apparently healthy albino rats' sexes, weighing between 180 and 200 grams and aged between 8-9 weeks were used for this study. They were purchased from the stock of faculty of Pharmaceutical science, Ahmadu Bello University, Zaria. The rats were housed in standard cages bedded with wood shavings and acclimated for one week. Animals were fed with rat chow and water provided *ad-libitum.*

Source of parasites

The parasites, *Trypanosoma congolense,* were sourced from National Veterinary Research Institute (N.V.R.I) Vom. The parasites were maintained by continuous passage in donor mice. Parasitaemia was monitored by wet mount and viewed under × 40 magnification. Parasites were harvested from the blood of a donor mouse at peak parasitemia (10^9 parasites/ml) and were diluted with phosphate buffered saline. The preparation was used for *in vivo* (infection of experimental animals) studies (Herbert and Lumsden, 1976).

Experimental infection of the rats

Trypanosomes infected blood was obtained from the tail of the infected donor mice at peak of parasitaemia (10^9 parasites/ml) and used to maintain parasite suspension in phosphate buffer saline glucose solution, which was then inoculated into peritoneal cavity of uninfected albino rats (Experimental albino rats). The suspension contained 3 or 4 trypanosomes per microscopic field at × 40 magnification (approximately 10^6 trypanosomes per 0.5ml) as described by Ekanem and Yusuf, 2008).

Determination of Parasitaemia in Albino rats

Parasitaemia was monitored in the rats from blood obtained from the tail vein. The numbers of parasites were determined microscopically at × 40 magnification using rapid matching method (Herbert and Lumsden, 1976).

Experimental design

The 10 albino rats that were used for the study were randomly divided into two (2) groups; Group A = 4 albino rats which served as control and Group B = 6 albino rats which were infected intraperitoneally with with 1 x 106 trypanosomes contained in 1 ml of blood obtained from *T. congolense* infected donor mouse and monitored for 2weeks. Confirmation of infection was done starting from 4 days' post-infection using hematocrit method as described by Herbert and Lumsden, (1976).

Gross Examinations

The mucous membrane was pale, congestion of brain tissues, hepatomegaly, nephritis, pneumonia, pericarditis, splenomegaly and haemorrhagic intestinal mucosa were observed

Histopathological Examination

Vital organs such as liver, kidney, lung, heart, spleen and Gastro-intestinal tracts were harvested for histopathology. Samples from vital organs were preserved in 10% neutral buffered formal Saline for Histological examination. Histopathological slides were prepared following the conventional histopathological methods and finally stained with Haemotoxylin and Eosin Stain (H & E).

RESULTS

Histopathological Changes

The parasites were detected in the peripheral blood of the rats during the 4 days' post infection (PI). At necropsy, the carcases were generally pale with atrophy of body fats. Histopathology sections revealed the following:

Brain

Brain revealed acute congestion of meningeal capillaries; perivascular oedema, occluded capillaries parasitic emboli, neuronecrosis (vaculations), gliosis and trypomastigotes in dilated capillaries were also seen. (Plate I)

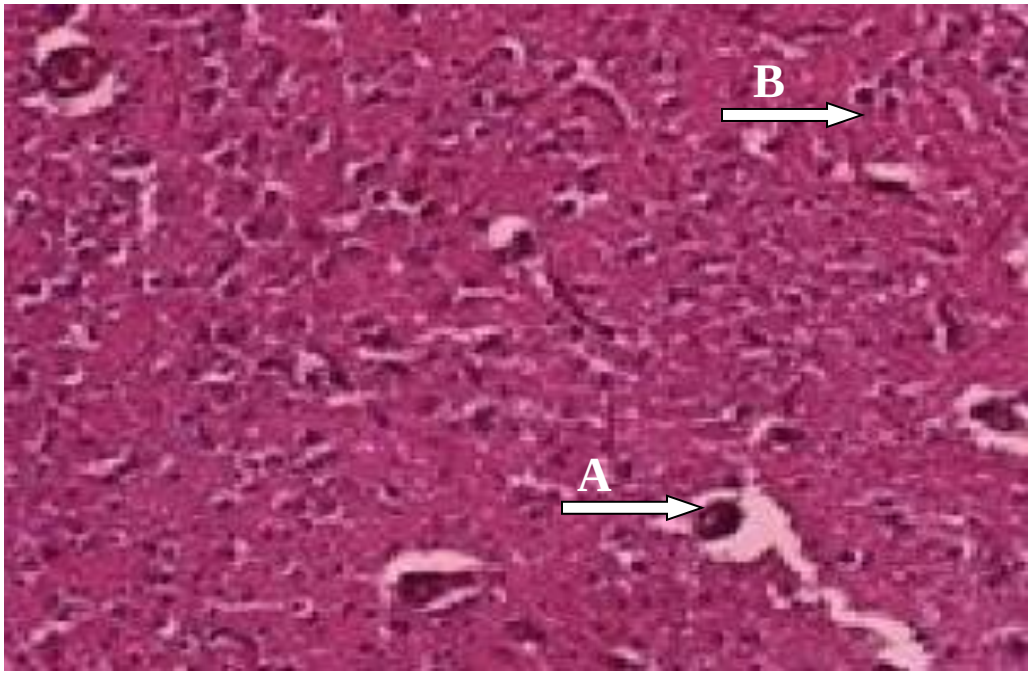

Plate I: Showing neuronecrosis (vaculations) and gliosis (A), infiltration by mononuclear cells H and E Stain. X 400.

Lungs

Lungs revealed congestion, oedema, multifocal alveolar, focal areas of atelectasis, hyperplasia of the alveolar wall and peri-bronchiolar lymphoid tissues, perivascular infiltration of lymphocytes around small blood vessels (venules and arterioles) and thickened inter-alveolar septae infiltrated with mononuclear cells (Plate II).

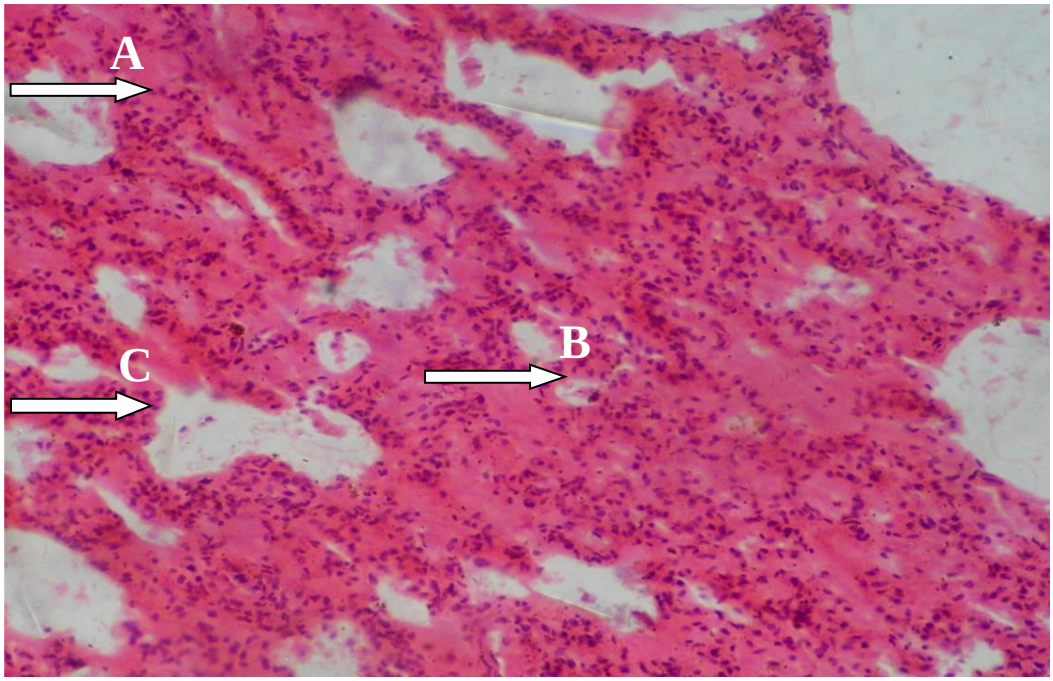

Plate 1I: A section of lung from an albino rat infected with *T. congolense*. Note the normal alveoli (A), compressed alveoli (B) and the thickened inter-alveolar septae infiltrated with mononuclear cells (C). H and E Stain. X 200.

Heart

Vasculitis, mmononuclear cellular infiltration, myocardial necrosis and haemorrhagic myocardium (Plate III)

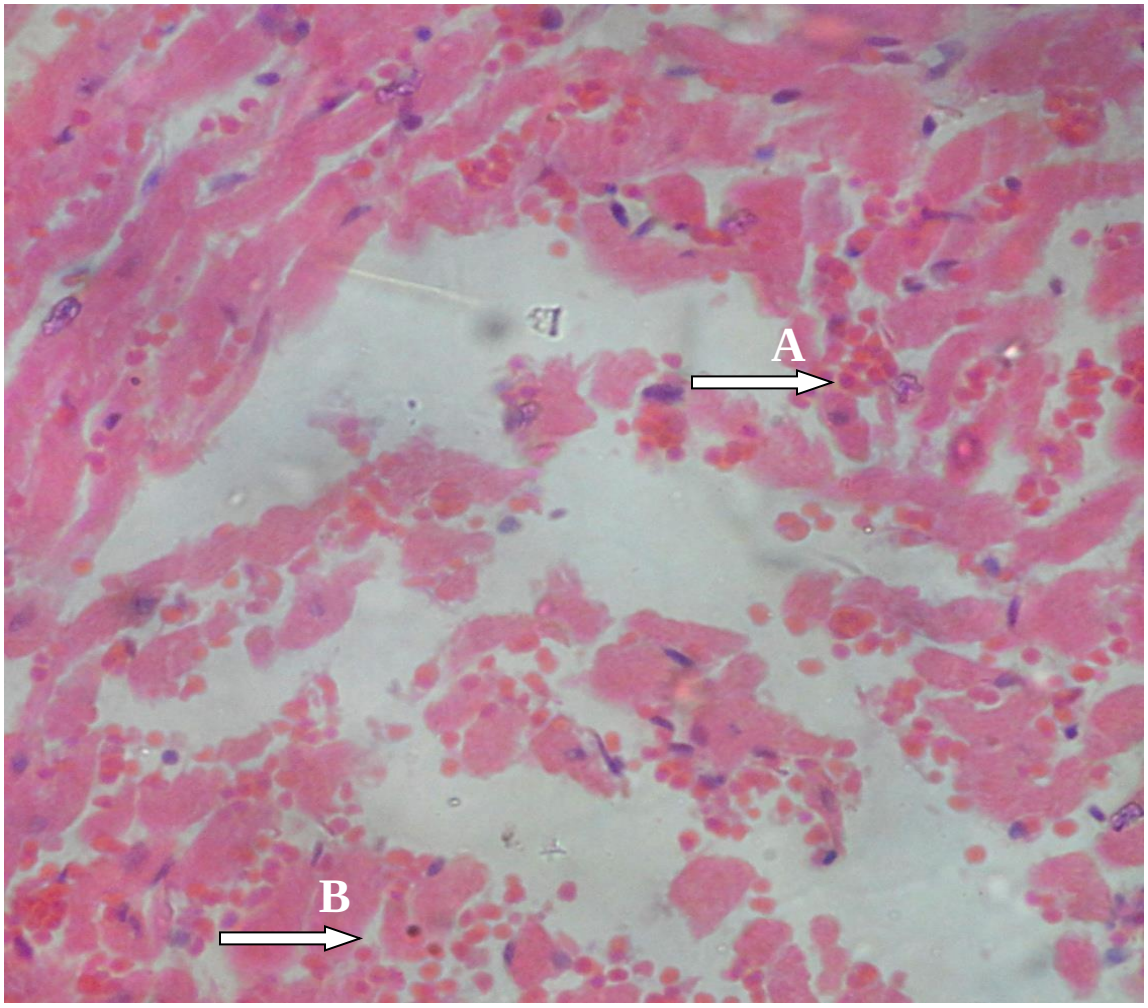

Plate III: section of cardiac muscle from an albino rat infected with *T. congolense.* Note the area with haemorrhage (A) and myocardial necrosis (B). H and E Stain. X 400.

Spleen

The spleen revealed extensive haemorrhages and acute congestion along with segregation of lymphoid follicles, hyperplasia, reticuloendothelial cells and Considerable amount of amorphous haemosiderin granules (Plate IV).

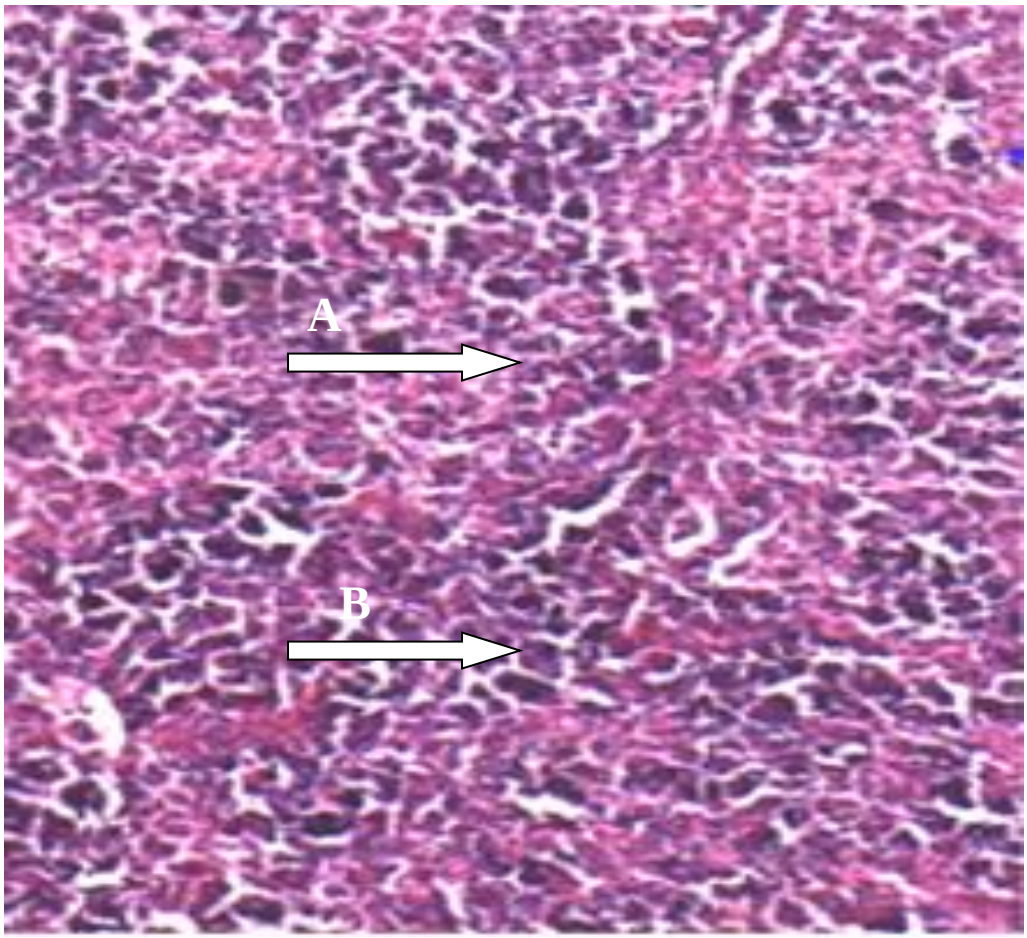

Plate IV: section of Spleen showing hyperplasia of germinal centres, (A) and amorphous haemossidecetriionn granules (B) H&E x400

Liver
Cellular infiltration of the portal triad and vasculitis Plate VII),

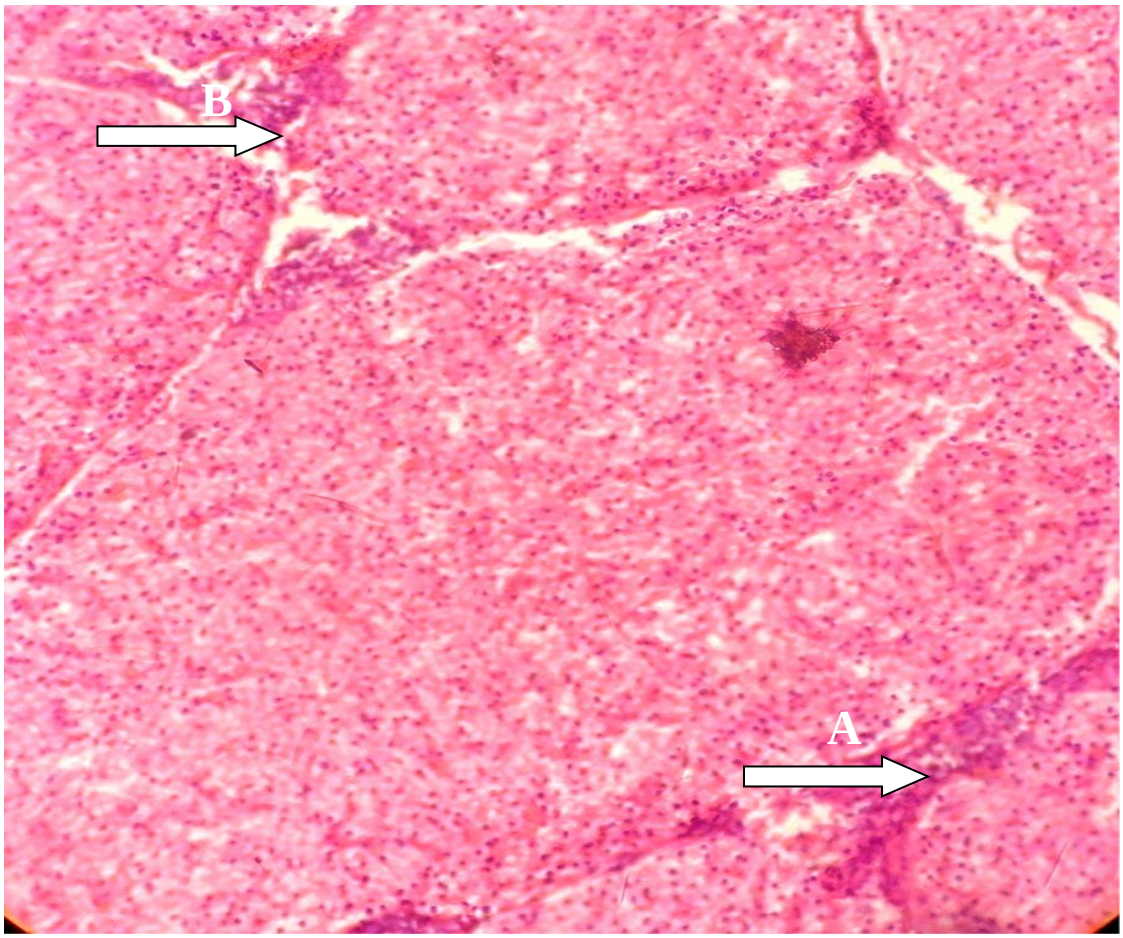

Plate 4.70: Gross section of liver from a *T. congolense* infected albino rat with mononuclear infiltration of the portal of triad (A) and vascullitis (B) (H and E.X 400)

Kidney

The kidney revealed areas of tubular necrosis, glomerulonephritis and destruction of Bowman's capsule (Plate V).

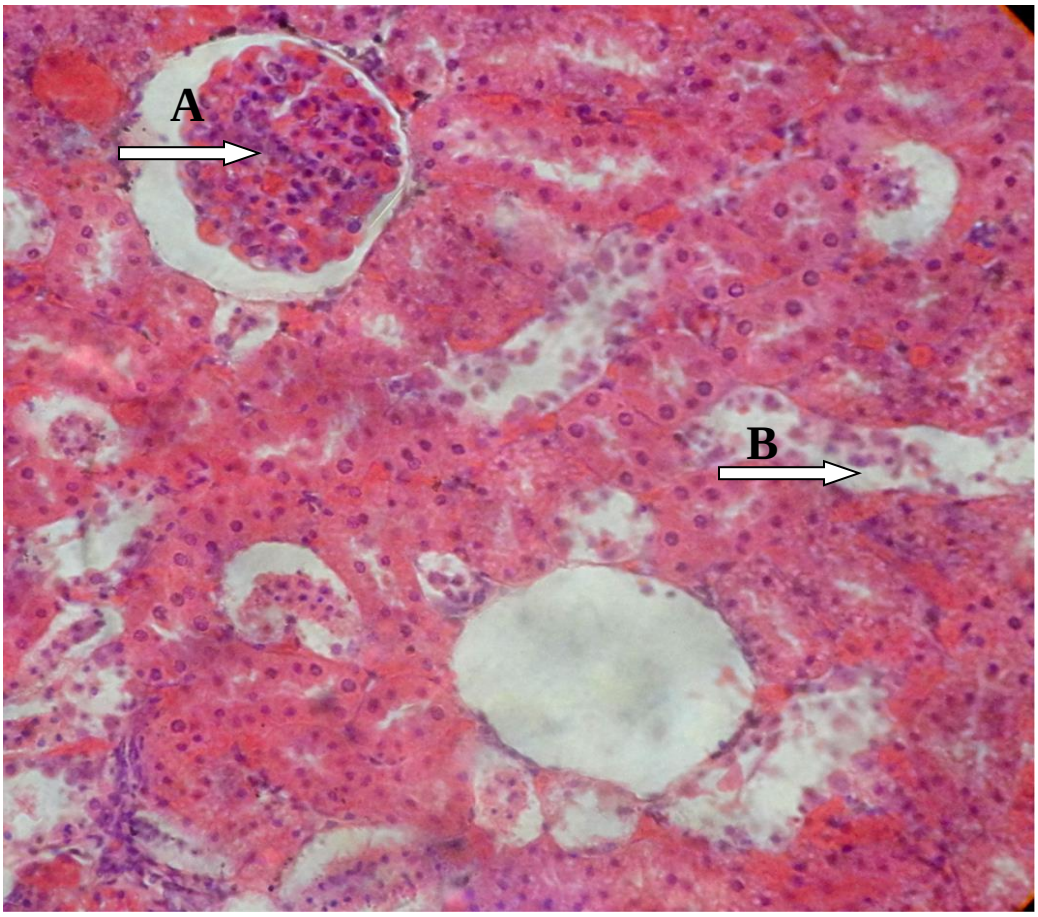

Plate V: Kidney section of albino rat infected with *T.congolense* glomerulonephritis (A) and renal tubular necrosis (B). H and E Stain. X 400.

Intestine

The intestinal revealed petechae haemorrhages, necrosis of the intestinal villi, formation of pseudomembrane in the intestinal lumen and depletion of germinal centre (Plate VI)

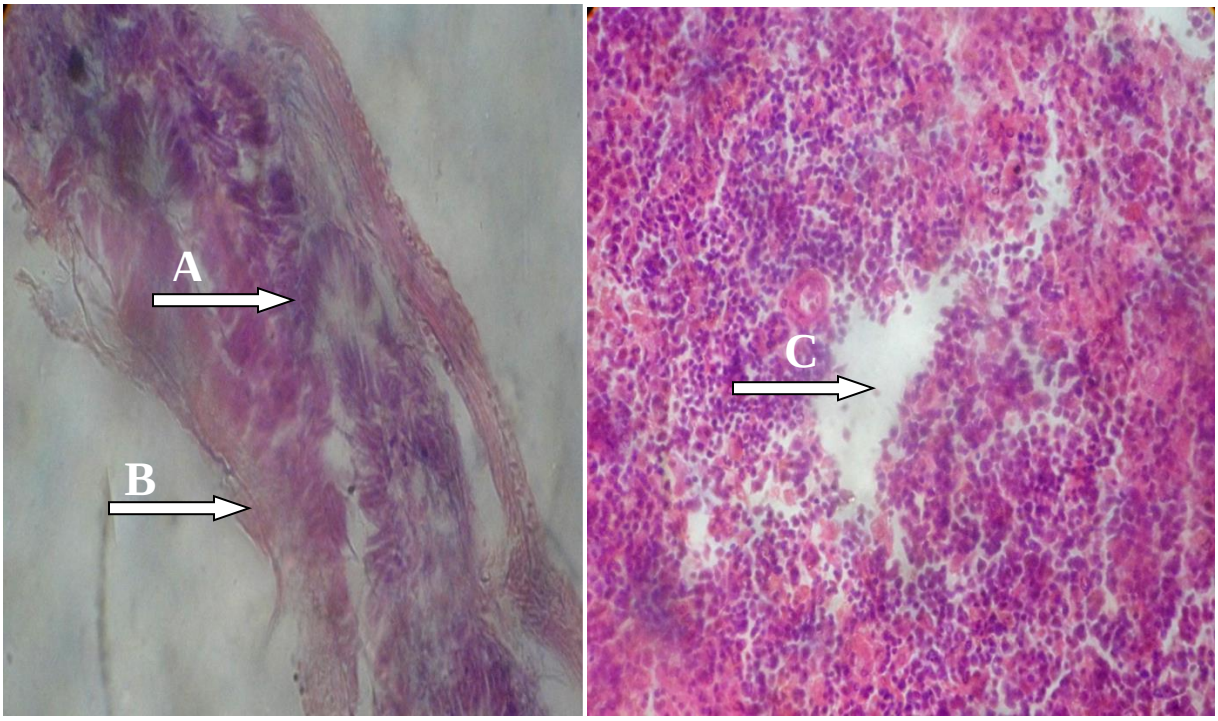

Plate 4.50: Gross section of intestine from *T. congolense* infected albino rat with necrosis of villi (A) and pseudo membrane in the lumen (B) and depletion of the germinal center (C). H and E Stain. X 200

DISCUSSION

This study revealed that experimental infection of albino rats with Trypanosoma congolense produced gross and pathological lesions in vital organs. The main changes in the brain which revealed acute congestion of meningeal capillaries with perivascular Oedema agreed with the result reported by Doyle *et al*., (2007) and Reham and Magdi (2013) in rats. Moreover, the presence of occluded capillaries, parasitic emboli, neuronecrosis (vaculations), gliosis and trypomastigotes in dilated capillaries were also reported by Biswas *et al*., (2010) in rats infected by T. evansi. The pathological changes seen in the brain could be attributed to the elaboration of toxic substance by the parasite and also due to the constant irritation caused by the presence of the parasites (Abuessailla *et al*., 2017). The lungs lesions observed in this present study were in agreement with the results reported by Sivajothi *et al*., (2015a) in trypanosome infected rats. The lung lesions could be attributed to the irritation and inflammatory response to the parasite which resulting in vasodilatation and exudation (Biswas *et al.*, 2010). The result obtained in this work does not agree with the work of Nagle *et al*., (1980), who reported no changes in the lungs of *T. rhodesiense* infected rabbits. These differences could be due to differences in species, level of parasitemia etc. The lesions observed in the heart in this present study were consistent with the findings of Nok *et al*., (1992) in rats, Ogunsanmi *et al*., (1994) in sheep, Mbaya *et al*., (2011) in camel. The

mechanisms through which trypanosomes cause tissue injuries include mechanical damage, increased permeability of the vascular wall, immunological mechanisms and damage by trypanosome toxin (Losos and Ikede, 1972). The finding in this work disagreed with the finding of Abuessailla *et al*., (2017) in rats. The spleenic lesions were in agreement with the work of Abuessailla *et al*., (2017) in rats infected experimentally with *T. congolense* and the work of Sivajothi *et al*., (2015a) and Bal *et al*., 2012 in Trypanosoma evansi infection in rats. The changes observed in the spleen might be due to anaphylactic reaction to the parasite, increase stimulation and activities of the spleen in filtering the blood of the parasite, as a result of extravascular haemolysis which occur mainly in the spleen and immunological mechanisms and damage by trypanosome toxin (Pentreath and Kennedy, 2004). The liver pathologies observed in this study were consistent with the findings of Onah *et al*., (1996), Audue *et al*., (1999) and Sivajothi *et al*., (2015a) in the rats infected by *T. evansi*. The liver pathology could be attributed to mechanical damage, immunological mechanisms, damage by trypanosome toxin and hypoglycemia leading to cell starvation (Pentreath and Kennedy, 2004). This finding was however different from that reported by Adewale *et al*., (2016) and Abuessailla *et al*., (2017) who observed no significant changes in liver of rats infected with Trypanosome parasite. The kidneys lesions were in agreement with the report of Bal *et al*., (2012) and Sivajothi *et al*., (2015a) in the rats infected with *T. evansi* and it has been reported that changes in the kidneys are mainly due to the toxins produced by the parasite and the accumulation of immune complexes which impair the structure and function of the kidney (Morrison *et al*., 1981; Ngeranwa *et al*., 1993). The intestinal pathology was consisted with the findings of Nok *et al*., (1992).

CONCLUSION

It can be concluded that experimental infection of albino rats with Trypanosoma congolense produced significant pathologic changes in vital organs.

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