RESEARCH ARTICLE

Serum Biochemical and Histopathological Changes in Rats Experimentally Infected with *Trypanosoma evansi* Isolated from Dromedary Camels in Sudan

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ABSTRACT- The biochemical and histopathological changes in rats experimentally infected with *T. evansi* isolated from camels in El-Gadarif State, Sudan, were studied. A number of 18 adult male outbred albino rats, weighing between 133–137 g were used in this study. The rats were divided into 3 groups of 6 animals each (A, B and E). Group A and B were intraperitoneally infected with *T. evansi* (Showak stabilate) with 1×10^4 trypanosoma for the inoculum. Group B was given quinapyramine sulphate (20 mg/kg bwt) after parasitaemia was evident. Group E was left healthy, uninfected controls for the stabilate. There was significant reduction in serum glucose and phosphorus; compared to significant increase in Glutamate Oxaloacetate Transaminase (GOT), Glutamate Pyruvate Transaminase (GPT) and total protein in groups (A and B). Microscopically, the brain tissues of the infected rats revealed acute congestion of the meningeal capillaries, perivascular oedema, neuronecrosis (vaculation), gliosis and trypomastigotes in dilated capillaries. The lung revealed oedema, congestion, multifocal alveolar emphysema, hyperplasia of the peri-bronchiolar lymphoid tissues and haemorrhages. The spleen showed extensive haemorrhages, haemosiderosis and aggregation of histiocytes resulting in multinuclear giant cell formation. The kidneys showed acute congestion of the glomerular tufts. All tissues obtained showed exactly the same histopathological changes. No significant histopathological alterations were observed in the liver and heart. The most consistent histopathological changes were seen in the brain, lungs, spleen and kidneys. These changes were consistent with trypanosome infection and were confirmed by the presence of trypanosomes in most of the tissue sections examined.

Key-Words: biochemical, changes, dromedary camels, histopathological, *T. evansi*, Sudan

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INTRODUCTION

Trypanosomiasis is considered as one of the most common and serious disease problem in several camel breeding countries [1,2]. The pathology of *Trypanosoma evansi* infection was studied in Swiss albino mice using cattle isolate of the parasite. Gross post-mortem examination revealed enlargement of the spleen and petechial haemorrhages in the liver in the terminal stages of disease. Tissue sections revealed presence of numerous trypanosomes in blood vessels of the liver, spleen, brain and kidneys. Microscopically, the liver revealed lesions varying from vacuolar degeneration, coagulative necrosis along with congestion and haemorrhages [3]. Spleen showed extensive haemorrhages in red pulp area, haemosiderosis and aggregation of histiocytes resulting in multinuclear giant cell formation. Lungs revealed oedema, congestion and mild inflammatory changes. Brain revealed mild degenerative changes along with congestion of meningeal blood vessels. Kidneys showed tubular degeneration, congestion and cellular infiltration. Heart revealed mild degenerative changes along with interstitial oedema [3].

The biochemical changes associated with *T. evansi* infection in pregnant and non-pregnant camels were investigated [4]. Based on pregnancy diagnosis and serological findings, camels were classified into four groups as non-pregnant healthy camels, non-pregnant camels infected with *T. evansi*, pregnant healthy camels and pregnant camels infected with *Trypanosoma evansi*. The results revealed significant decreases in serum total proteins, albumin and globulin levels; and significant increases in serum total cholesterol and blood urea nitrogen (BUN) levels in pregnant camels infected with *T. evansi* compared with healthy pregnant camels. On the other hand, there were hyperproteinemia and hyperglobulinemia in healthy pregnant camels compared with non-pregnant camels. It was concluded that the biochemical changes associated with *T. evansi* infection in pregnant camels were hypoproteinemia, hypoalbuminemia, hypoglobulinemia and increased serum total cholesterol and blood urea nitrogen (BUN) levels [4].

MATERIALS AND METHODS

Ethics statement

The study protocol approved by the Faculty of Veterinary Medicine, Sudan University of Science and Technology, according to their guidelines for sampling domestic animals in Sudan and is in compliance with the animal welfare of the Sudan.

Study area

This parasite was isolated from a camel at a village within the vicinity of the Showak area, El-Gadarif State, Sudan. The study duration was one year.

Preparation of the inocula

A strain of *T. evansi* originated from a naturally infected camel from Showak, El-Gadarif State was used in this study. One albino rat was infected intraperitoneally with blood that was cryopreserved in liquid nitrogen, containing 1×10^4 parasites/animal to obtain a large amount of the parasite for blood inoculation of experimental groups.

Parasitemia in the inoculated rat was regularly monitored by collecting blood from the tail vein and analyzing it by light microscopy. Blood samples showing actively motile organisms with characteristic flagellar movement were considered as positive for the presence of *T. evansi*. At the peak of parasitemia, the rat was anesthetized with chloroform inhalation, and with the help of a disposable syringe, blood was collected aseptically in EDTA anticoagulant by cardiac puncture. Using Neubaeur's counter, the trypanosome titre was determined in order to be diluted to $1x10^4$ trypanosoma for the inoculum.

Experimental animals

Eighteen (18) adult male outbred Albino rats, weighing between 133 to 137 g were used in this study. The rats were divided into 3 groups, each containing 6 of rats and were kept in a cage in the same environment with controlled temperature (25–30°C) and humidity around 60–70% RH.

Experimental design and grouping

The distribution of the experimental rats into 3 groups of 6 rats each group. Group A, the control group, was infected with *T. evansi* (Showak stabilate) and left without treatment. Group B was infected with *T. evansi* (Showak stabilate) and was treated with the quinapyramine sulphate (20 mg/kg bwt), after the parasite was seen (at the patency). Group E was uninfected healthy control for Showak Stock.

Trypanosome sub-inoculation

Sub-inoculation of the experiment group A and group B carried out intraperitoneally with the help of a sterile insulin syringe. Rat blood containing 1×10^4 trypanosomes in 0.2 ml volume was inoculated in each rat individually at day zero. The number of inoculated flagellates was estimated by Neubauer chamber and the dilutions obtain the titre of the inoculum were made in sterile phosphate buffer saline with glucose (PSG).

Table 1: The experimental design of the showak stabilate and protocol of treatment with Quinapyramine sulphate

Group	Stabilate	Parasite	Treatment protocol
A	Showak	*T. evansi*	Infected not treated
B	Showak	*T. evansi*	Infected and Treated with Q.S. (20mg/kgbwt)
E	Uninfected Healthy Control for Showak Stock		

Estimation of parasitaemia

All infected rats were bled daily as preferred by Eisler *et al.* [5] from the tip of the tail for trypanosomes detection using the following parasitological diagnostic methods:

Wet preparation

A drop of blood was mounted on a microscopic slide covered with 22x22 mm glass cover slip. Counts of parasite per field or per preparation were determined.

Haemocytometer count
The presence and degree of parasitaemia was determined daily for each rat by examining tail blood. A drop (5 µl) of blood was collected from the tail and mixed with trypanosome counting reagent (45 µl). Parasitaemia was counted as for WBC count using Neubeaur counter and the result was designated as a number of parasites per ml of blood. Parasitaemia was counted using 40x magnification during the 60 days of the experiment.

Drug dosages
Quinapyramine sulphate was used at a dose rate of 20 mg/kg bwt and dissolved in sterile water such that the required dose was contained in 0.2 ml of water for each rat and then inoculated intra-peritoneally.

Biochemical analysis
Blood for sera was collected in plain containers from the retro-orbital plexus. Serum samples were collected at four days intervals and were kept at -20°C until needed for biochemical analysis. All parameters were measured using commercial kits (Spinreact S.A./S.A.U. Ctra. Santa Coloma, Spain). The values obtained were read with a spectrophotometer (Jenway 6305 U.V./Vis. Spectrophotometer, U.K.) at the appropriate wavelengths and the values were calculated using standard formulae [6].

Histopathological Studies
Vital organs such as liver, kidney, lung, heart, spleen and brain were taken 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).

Data analysis
Data were presented as mean±standard error of mean (SE). The statistical analysis was performed using independent T-test using the Statistical Package for the Social Science (SPSS) software. P-values less than 0.05 were considered statistically significant.

RESULTS
The Overall Mean of Parasitaemia
Generally, the overall mean of parasitaemia in group A was 5.4 ±2.8 and in group B was 4.8 ±2.9 (Table 2).

Table 2: Overall means and Std. deviation of parasitaemia levels in rats infected-not treated (A group) Showak stabilate and rats infected-treated (B group)

Treatment	Strains	Mean	Std. deviation	N
Not treated	Showak	5.43	2.85	28
reated	Showak	4.79	2.94	61

The response of Showak stabilate to Quinapyramine Sulphate in group (A)

Rats inoculated by $1X10^4$ of the Showak stabilate of *T. evansi* but were not treated with Quinapyramine sulphate (A group) inflicted high mortalities during the experiment period where 1 died at day 16 post infection (pi), 1 at day 21, 1 at day 25, 2 at day 26 and 1 at day 28 with a mean survival period of 23.2 ±4.8.

Effect of drug (Quinapyramine sulphate)%

$$= \frac{\text{Infected untreated–Inecfected Treated}}{\text{Infected untreated}} \text{x}100$$

= 8.3–6.6/ 8.3 x 100= 20.5%

The response of Showak stabilate to Quinapyramine Sulphate in group (B)
Only two rats died and this happened at day 52 and day 53 pi. with a mean survival period of 52.5±0.72 (Table 3).

Table 3: comparison between rats infected with *T. evansi* (Showak Stabilate) treated by Quinapyramine Sulphate a dose rate of 20 mg/kgbwt (after patency group B) and rats Infected-not-treated control (group A)

Time to death	Control of 6 Rats	Time to death	Infected Treated of 6 Rats
Day 16	1 rat, n= 5 rats	Day 52	1 rat, n= 5 rats
Day 21	1 rat, n=4 rats	Day 53	1 rat, n= 4 rats
Day 25	1 rat, n=3 rats	–	–
Day 26	2 rat, n= 1 rats	–	–
Day 28	1 rat, n= 0	–	–
X= 23.2±4.8		X= 52.5±0.72	

Treatment of rats in group (B), which were infected and treated with Quinapyramine sulphate was commenced at day 6 when the parasitaemia level was $\log_{10}$ 4.2, By day 8 the parasite was cleared from all rats in the group and remained so until day 10 during which period no protozoan can be detected in wet blood smears. Up to day 6, there was no significant difference between parasitaemia levels in both treated and control groups. By day 26, the treated group recorded a mean parasitaemia of $\log_{10}$ 6, while that of the control was $\log_{10}$ 8.3, which was significantly higher than the treatment group ($p < 0.05$). Control rats by day 17 parasitaemia fluctuated between $\log_{10}$ 7.8 to $\log_{10}$ 8.0 till the end of the study period. The drug has an effect on parasitaemia till day 26 (Fig.1).

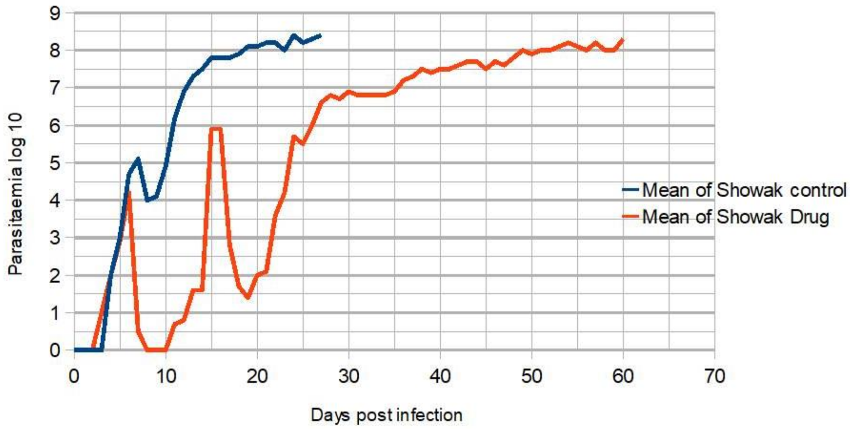

Fig. 1: Comparison of the means of parasitaemia levels (log_{10}), between rats infected with *T. evansi* (Showak Stabilate) treated by Quinapyramine Sulphate a dose rate of 20 mg/kg bwt and rats infected-not-treated control

Serum biochemical changes

Serum total protein

The mean serum values of total proteins in group A and group B were increased progressively during the study. The statistical analysis in group A showed a means of 8.2±1.3 g/dl and in group B showed a means of 8.7±1.3 g/dl (Table 4 a).

Serum glucose

The mean serum values of glucose in group A and group B were decreased. The statistical analysis in group A showed a means of 37.9±13.9 mg/dl and in group B showed a means of 46.2±12.6 mg/dl (Table 4 a).

Serum Albumin

The mean serum values of albumin in group A and group B were increased. The statistical analysis in group A showed a means of 5.9±0.97 g/dl and in group B showed a means of 5.8±1.7 g/dl (Table 4 a).

Serum creatinine

The mean serum values of creatinine in group A and group B were increased. The statistical analysis in group A showed a means of 2.8±1.1 mg/dl and in group B showed a means of 2.1±1.2 mg/dl (Table 4 a).

Serum phosphorus

The mean serum values of phosphorus in group A and group B were decreased. The statistical analysis in group A showed a means of 3.8±2 mg/dl and in group B showed a means of 5.4±1.9 mg/dl (Table 4 a).

Serum glutamate oxaloacetate transaminase

The mean serum values of GOT in group A and group B were increased. The statistical analysis in group A showed a means of 105.2±36.1 U/l and in group B showed a means of 83.6±0.35 U/l (Table 4 a).

Serum glutamate pyruvate transaminase

The mean serum values of GPT in group A and group B were increased. The statistical analysis in group A showed a means of 39.8±9.2 U/l and in group B showed a means of 34.8±7.9 U/l (Table 4 a).

Table 4 a: Means serum levels of biochemical changes in rats infected experimentally with *T. evansi* rats infected-not-treated control and treated with Quinapyramine Sulphate at a dose rate of 20 mg/kgbwt

Parameters	units	Group A	Group B
Total proteins	g/dl	8.2±1.3	8.7±1.3
Glucose	mg/dl	37.9±13.9	46.2±12.6
Albumin	g/dl	5.9±0.97	5.8±1.7
Creatinine	mg/dl	2.8±1.1	2.1±1.2
Phosphorus	mg/dl	3.8±2	5.4±1.9
GOT	U/L	105.2±36.1	83.6±0.35
GPT	U/L	39.8±9.2	34.8±7.9

GOT= Glutamate Oxaloacetate Transaminase; GPT= Glutamate Pyruvate Transaminase. Values were expressed as Mean ±SD

Table 4 b: Rat Biochemical Reference Normal Ranges

parameters	Ranges Values	units
Total proteins	5.6 -7.6	g/dl
Glucose	50 – 135	mg/dl
Albumin	3.8 - 4.8	g/dl
Creatinine	0.2 – 0.8	mg/dl
Phosphorus	3.11-11 mg/dl	mg/dl
GOT	45.7 – 80.8	U/L
GPT	17.5 – 30.2	U/L

Histopathological changes

Representative tissue sections of the liver, kidney, heart, spleen, lungs and brain from the groups A and B showed the followings: all tissues obtained showed exactly the same histopathological changes. No significant histopathological alterations were observed in the liver and heart. The most consistent histopathological changes were seen in the brain, lungs, spleen and kidneys.

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. Trypanosomes were observed in congested blood vessels of brain in rat died of teaming parasitaemia (Fig. 2).

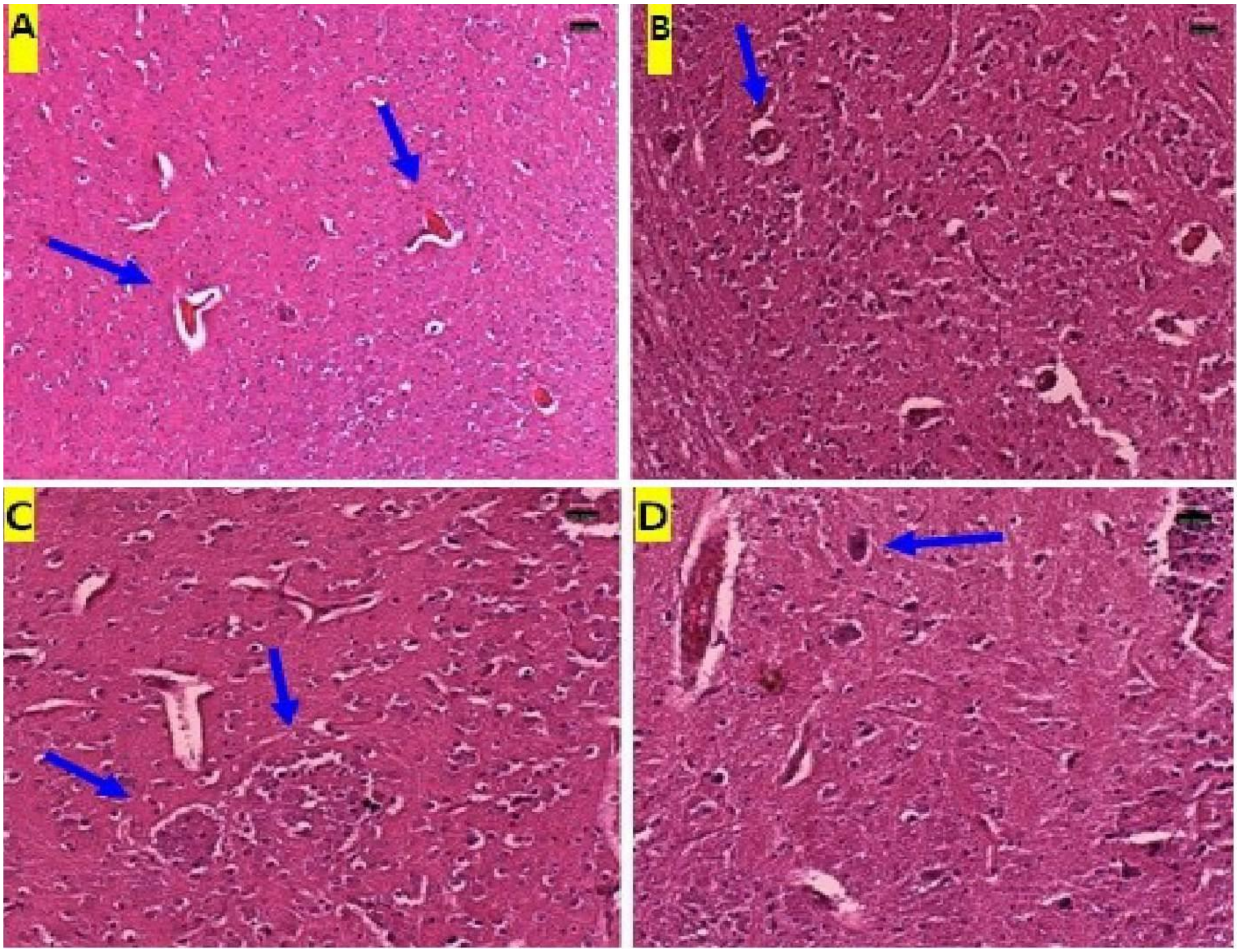

Fig. 2: Brain sections: showing congestion, perivascular edema (A: Arrows); occluded capillaries, parasitic emboli (B: Arrow); neuronecrosis (vaculations) and gliosis (C: Arrows) and Trypomastigotes in dilated capillaries (D: Arrow) (H & E stain)

Lungs

Lungs revealed oedema, congestion, multifocal alveolar emphysema, focal areas of atelectasis, increased cellularity of the alveolar wall, hyperplasia of the peri-bronchiolar lymphoid tissues, perivascular infiltration of lymphocytes around small blood vessels (venules and arterioles) and haemorrhage was also seen (Fig. 3).

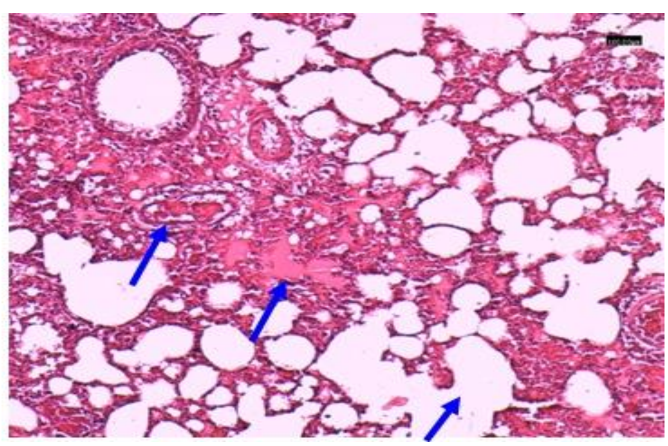

Fig. 3: Lung section showing congestion, oedema, hemorrhages and emphysema (H & E stain)

Spleen

Spleen exhibited extensive haemorrhages and acute congestion along with segregation of lymphoid follicles, hyperplasia, reticuloendothelial cells and hypertrophy. A considerable amount of amorphous haemosiderin granules was evident in most of the sections of the spleen (Fig. 4).

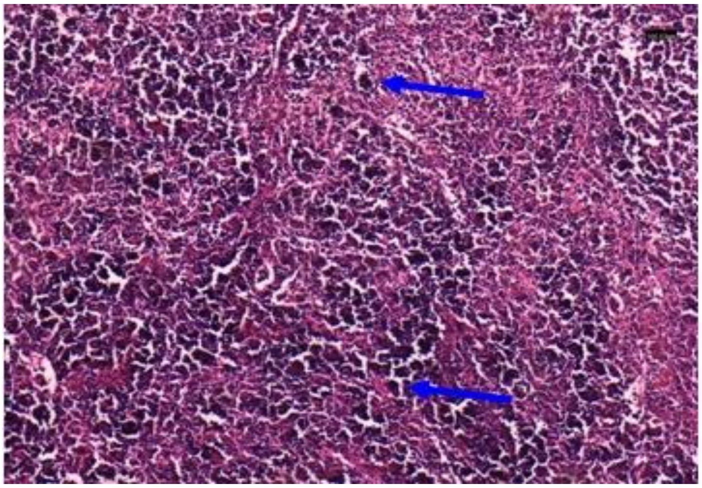

Fig. 4: Spleen: Histopathological section showing amorphous haemosiderin
granules (arrows) (H & E stain)

Acute congestion of the glomerular tuft (Fig. 5).

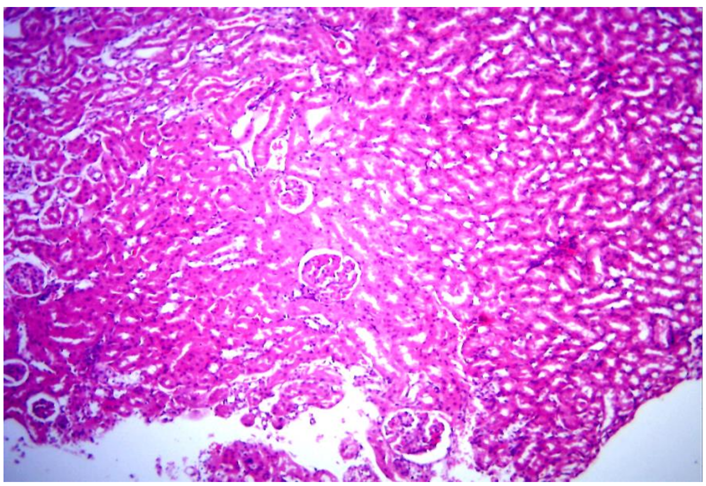

Fig. 5: Kidney: Acute congestion of the glomerular tuft (H&E X100)

No significant histopathological alterations were observed in the liver (Fig. 6).

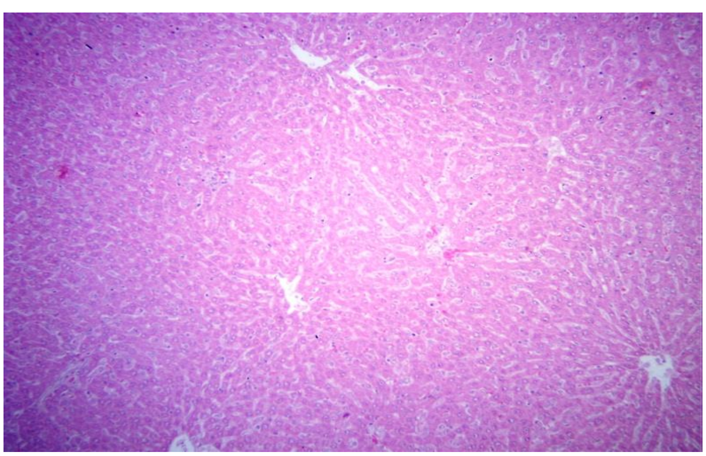

Fig. 6: Liver: No significant histopathological changes (H&E X100)

No significant histopathological alterations were observed in the heart (Fig. 7).

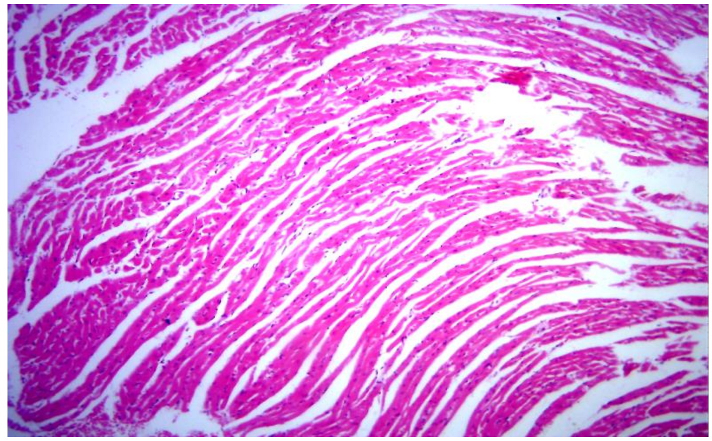

Fig. 7: Heart: No significant histopathological changes (H&E X100)

DISCUSSION

In this study, a stabilated *T. evansi* parasitic protozoan strain, which was isolated from a camel at a village within the vicinity of Showak area, Gedarif State, North eastern Sudan (named as Showak stock; resistant to Quinapyramine Sulphate) was investigated and studied. During this study, the local isolate of *T. evansi* stock was compared in experimentally infected rats. The prepatent period of infection by *T. evansi* was found to be variable depending on the host and the parasite isolate. Rats inoculated by $1X10^4$ of the Showak stabilate of *T. evansi* but were not treated with Quinapyramine sulphate (A group) in this paper, showed a pre-patent period of 4-6 days post infection which disagreed with the result reported by Da Silva *et al.* [7]. However, this result was in agreement with that reported by Garba *et al.* [8] and Habila *et al.* [9] who reported a prepatent period of 3–7 days in a donkey infected by *T. evansi*. Group A has inflicted high mortalities during the experiment period, which was similar to the results of Samah [10]; Hoare [11] and Dargantes *et al.* [12]. In the rats infected-treated with Quinapyramine Sulphate at a dose of 20 mg/kgbw (B group), the rats showed a prepatent period of 3–5 days post infection, which was similar to the result reported by Da Silva *et al.* [13] in cats experimentally infected with *T. evansi* as well as with rats infected by *T. evansi* [14] and with goats infected by *T. evansi* [15].

Biochemical evaluation of the body fluids gives an indication of the functional state of various body organs and biochemical changes in body fluids that result from infections depending on the species of the parasite and it's virulence [16]. The serum total proteins in the groups A and B were increased progressively during the study which disagreed with the result reported by Hussain *et al.* [17]; Sivajothi *et al.* [18]; Biryomumaisho *et al.* [19]; Katunguka-Rwakishaya [20]; Allam *et al.* [21] and Megahed *et al.* [4]. This increase of total protein was in agreement with the result reported by Arora and Pathok [22] and Samia *et al.* [23] who found that the concentration of total protein was increased in rats experimentally infected with *T. evansi.* Also, it was in agreement with the result reported by Orhue *et al*. [24]; Ekanem and Yusuf [25] and Sow *et al*. [26], who found that the concentration of total protein was increased in rats experimentaly infected with *T. brucei*. and *T. brucei*-infected rabbits. The increase in protein levels during the chronic phase of the infection is usually attributed to the increase in globulin levels, as a result of the immune response by the animals to the infection [27-29]. In the present study, the serum glucose in the infected groups (A and B), has decreased during the study, which is similar to the result reported by Sivajothi *et al*. [18]; Sinha *et al*. [30]; Arora and Pathok [22] and Samia *et al*. [23], who found that the concentration of glucose was decreased in rats experimentally infected with *T. evansi*. This situation could be explained by the parasites' need for glucose for their cellular metabolism through their glycolytic pathway [31]. However, this finding was not in agreement with that reported by Youssif *et al.* [15] who found that goats infected by *T. evansi* had increased level of glucose.

The serum values of creatinine in the infected groups (A and B), have not increased progressively during the study. This non-progressive increase of creatinine is in agreement with the results obtained in a *T. cruzi* infection in mice [32], *T. brucei* infected animals [18,33,34] and *T. b. brucei* infected rats [21,35]. However, these results were not in agreement with those obtained by Luckins [36]; Chaudhary and Iqbal [37] and Youssif *et al*. [15]. The increase of creatinine due to increase in skeletal muscle disease, myocardial injury or necrosis and cerebral cortical necrosis, also, could be due to destruction of kidney cells resulting in the inability of the kidneys to excrete creatinine [35]. The serum values of albumin in the groups (A and B), were increased during the study. The increase of albumin disagreed with the results reported by Arora and Pathok [22] and Samia *et al*. [23] who found that the concentration of albumin was depressed in rats experimentally infected with *T. evansi*. Also, the result reported by Megahed *et al.* [4] found that the concentration of albumin was decreased in pregnant camels infected with *T. evansi* compared with healthy pregnant camels and, also, a decrease of albumin in camels infected by *T. evansi* was further reported by Hussain *et al*. [17].

In the present study the serum phosphorus in the groups A and B, were decreased during the study, which is similar to the result reported by Youssif *et al*. [15] in goats infected by *T. evansi* but is not similar with that reported in sheep infected with *T. congolense* [38,39]. This decrease of phosphorus might be due to renal excretion.

The serum values of GOT and GPT in the infected groups (A and B), were increased during the study. This increase was in agreement with the results obtained during an infection in sheep by *T. brucei* [21,40], *T. vivax* infection of cattle and sheep [41], *T. congolense* infection of goats [42] and in dogs infected with *T. brucei* [43]. Other studies have reported elevated serum enzymes [22,33,44,45]. However, these findings contradict the observations of Taiwo *et al*. [40] during an infection of sheep with *T. congolense*. The causes of elevation of GOT and GPT levels in the serum were attributed, mainly, to the necrosis of the liver, skeletal muscles and kidneys [46] or, partly, due to cellular damage caused by lyses or destruction of the trypanosomes [34].

The main histopathological changes in the brain which revealed acute congestion of meningeal capillaries with perivascular oedema agreed with the result reported by Dargantes *et al.* [12], Doyle *et al.* [47] and Reham and Magdi [48]. Moreover, the presence of occluded capillaries, parasitic emboli, neuronecrosis (vaculations), gliosis and trypomastigotes in dilated capillaries were also reported by Biswas *et al*. [49] in rats infected by *T. evansi*. The changes in the brain might be due to toxic substances released by the parasite. Also, the pathological changes in the brain could be attributed to the constant irritation caused by the presence of the parasites.

The main histopathological changes in the lungs revealed oedema, congestion and multifocal alveolar emphysema which is in agreement with the results reported by Takeet and Fagbemi [50], Reham and Magdi [48] and Sivajothi *et al*. [51]. The congestion and oedema in the lungs were mainly due to the inflammatory response to the parasite resulting in vasodilatation and exudation in the focal areas, atelectasis, increased cellularity of the alveolar wall, hyperplasia of the peri-bronchiolar lymphoid tissues and perivascular infiltration of lymphocytes around small blood vessels (venules and arterioles) and haemorrhages. Similar type of changes were also observed in the lungs of rats experimentally infected with *T. evansi* [49,52]. However, these findings were not in line with those reported by Nagle *et al.* [53] who observed no changes in the lungs of *T. rhodesiense* infected rabbits.

The main histopathological changes in the spleen which included extensive haemorrhages and acute congestion along with segregation of lymphoid follicles, hyperplasia, reticuloendothelial cells and hypertrophy were similar to the results reported by Sivajothi *et al.* [51]. Considerable amount of amorphous haemosiderin granules was evident in most of the sections of spleen which agrees with the findings of Reham and Magdi [48] as well as with the findings reported by Bal *et al*. [3] in rats infected with *T. evansi*. Initial changes in the spleen might be due to immediate hypersensitivity to *T. evansi*.

The main histopathological changes in the kidneys which included acute congestion of the glomerular tuft agreed with the result reported by Bal *et al*. [3] and Sivajothi *et al.* [51] in the rats infected with *T. evansi* and similar, also, with the result reported by Onah *et al*. [54] and Auduo *et al*. [55]. 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 [56,57].

The lack of significant histopathological alterations observed in the liver in this study was similar to the result reported by Adewale *et al*. [58]; but, however, it was not similar to the result reported by Reham and Magdi [48]; Bal *et al.* [3]; Sivajothi *et al.* [51]; Onah *et al.* [54] and Audue *et al.* [55]. In the rats infected by *T. evansi* where the liver revealed lesions varying from vacuolar degeneration, coagulative necrosis along with congestion and haemorrhages, these effects might be due to hypoglycemia leading to cell starvation. No significant histopathological alterations were observed in the heart which was similar to the result reported by Adewale *et al.* [58]; but was not in line with the result reported by Reham and Magdi [48]; Sivajothi *et al.* [51] and Bal *et al.* [3] in rats infected with *T. evansi*.

CONCLUSIONS

The biochemical and histopathological changes of *T. evansi* isolated from camels in Sudan were studied in experimentally infected rats. The infection resulted into significant reduction in serum glucose and phosphorus; compared to significant increase in Glutamate Oxaloacetate Transaminase, Glutamate Pyruvate Transaminase and total protein. Microscopically, the brain tissues of the infected rats revealed acute congestion of the meningeal capillaries, perivascular oedema, neuronecrosis (vacula-tion), gliosis and trypomastigotes in dilated capillaries. The lung revealed oedema, congestion, multifocal alveolar emphysema, hyperplasia of the peri-bronchiolar lymphoid tissues and haemorrhages. The spleen showed extensive haemorrhages, haemosiderosis and aggregation of histiocytes resulting in multinuclear gi-ant cell formation. The kidneys showed acute congestion of the glomerular tufts. No significant histopathological al-terations were observed in the liver and heart. The most consistent histopathological changes were seen in the brain, lungs, spleen and kidneys. These changes were consistent with trypanosome infection and were confirmed by the presence of trypanosomes in most of the tissue sections examined.

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