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Biochemical Changes in Rats Experimentaly Infected with *Trypanosoma evansi*

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Introduction

Trypanosomosis is a group of diseases caused by flagellated protozoan parasites of the genus *Trypanosoma*, Family *Trypanosomatida*. They are widely distributed in Africa, South America, Asia and Middle East (Hoare, 1972; Molyñeux and Ashford, 1983). *Trypanosoma evansi* causes a disease referred to as surra. It is an important disease of livestock in Africa and Asia causing great economic losses in camels and water buffalo. *Trypanosoma evansi* is mechanically transmitted during the feeding of blood sucking *Diptera* especially *Tabanid* flies (Ford, 1971). Infectious diseases as well as toxic materials cause biochemical changes in animal tissues and fluids therefore study of the concentrations of minerals and other blood constituents is important.

Materials and Methods

Materials: Swiss albino rats (Albino Wister) were obtained from the Central Veterinary Research Laboratory, Soba, Khartoum, Sudan. They were fed with pellets and fresh vegetables and were watered *ad libitum* throughout the experimental period. *T.evansi* was isolated from naturally infected camels at Soba, Khartoum Town. The rapid matching wet-count technique described by Herbert and Lumsden (1976) was used for *Trypanosomes* counting.

Methods: GOT and GPT were determined according to the method of Reitman and Frankel, (1957); Schmidt, (1963). Total protein and albumin according to the method described by Doumas, (1972) and Walsh, (1983); Glucose by Tinder, (1969), Urea by Berthelol, (1859); Alkaline phosphatase and creatinine by Young et al (1979). The electrolytes, Sodium and Potassium were detected by flame photometer (Corning 400), Zinc, Copper and Magnesium by atomic absorption spectrometer (Unicam 929).

Statistical Analysis: The results were expressed as means ± standard error (SE). The significance of differences between the means was analyzed by the studen't's t- test (Snedicor and Cochran, 1967).

Experimental design: Three groups of ten rats each aged 4-6 weeks average weight 150g were used in this experiment. The groups were divided as follows: Group 1 infected with *T.evansi*. Group 2 infected with *T.evansi* and treated with Cymelarsan at a dose rate of 0.25 mg/kg Body weight. Group 3 left as control. Trypanosomes were injected intraperitonially at a dose rate of $5x10^5$. Cymelarsan was administered subcutaneously at a dose rate of 0.25mg/kg BW as prescribed by the manufacturers. Blood from ocular vein was collected once a week till the end of the experiment for serum. The serum was kept at −20°C for analysis. Parasitaemia was checked daily.

Results

There was slight increase in the activity of SGOT in the infected and untreated group of rats (Table1) during the third week of infection as compared to the control group (Table 3). The group that received Cymelarsan showed no change in the activity of SGOT. There was gradual increase in the activity of SGPT in the infected and untreated group of rats started from slight increase in the second week of infection till the third week where the increase was significant ($p<0.05$) compared to the control group. With Cymelarsan treated group of rats, the activity of SGPT showed no increase during the whole period of treatment. The concentrations of both total protein and globulin were increased but the increase was not significant. The concentration of albumin showed gradual decrease where the decrease was marked during the third week of infection. Urea and creatinine concentrations increased gradually where the increase was significant ($p<0.05$) in the third week of infection. The concentration of sodium and magnesium in blood decreased slightly during the second week of infection. The decrease of magnesium was found to be significant ($P<0.05$) during the third week of infection. There was also gradual decrease in the concentration of potassium but the decrease was highly significant during the third week of infection ($P<0.01$). The concentration of copper showed a gradual decrease where the decrease was found to be significant at the third

Table 1: Biochemical changes in rats infected with *T.evansi* (5 x 10^5)

	Week 1	Week 2	Week 3	Week 4
Glucose	5.77 ± 0.02	5.16 ± 0.03	2.84 ± 0.04	0.41 ± 0.02*
GOT	19.0 ± 0.32	18.75 ± 0.41	20± 0.56	25.25 ± 0.07
GPT	29.0 ± 0.41	32.75 ± 0.35	45.5 ± 0.48	59.5 ± 0.82*
Total Prot	7.26 ± 0.22	6.98 ± 0.32	7.16 ± 0.26	8.53 ± 0.41
Albumin	2.23 ± 0.03	2.12 ± 0.09	1.98 ± 0.08	0.87 ± 0.03*
Globulin	5.08 ± 0.04	4.86 ± 0.01	5.18 ± 0.02	7.66 ± 0.32
Potassium	10.60 ± 0.01	7.03 ± 0.05	7.9 ± 0.12	2.93 ± 0.41**
Magnesium	0.74 ± 0.11	0.65 ± 0.03	0.59 ± 0.04	0.51 ± 0.02*
Sodium	100.70 ± 0.39	106.5 ± 0.42	104.33 ± 0.55	97.8 ± 0.38
Copper	0.87 ± 0.05	0.84 ± 0.01	0.5 ± 0.01	0.29± 0.03**
Zinc	0.45 ± 006	0.47 ± 0.02	0.31 ± 0.23	0.25± 0.08
Urea	4.06 ± 0.07	4.27 ± 0.24	5.92 ± 0.38	8.12± 0.24*
Creatinine	0.58 ± 0.10	0.61 ± 0.21	1.03 ± 0.01	2.03± 0.07*

Values = means ± standard error * = significant ** = Highly significant

Table 2: Biochemical changes in rats infected with *T.evansi* (5 x 10^5) and treated with Cymelarsan (0.25 mg/kg BW)

	Week 1	Week 2	Week 3	Week 4
Glucose	5.37 ± 0.11	4.51 ± 0.53	4.32 ± 0.42	5.06 ± 0.38
GOT	18.15 ± 0.65	17.0 ± 0.64	15.75 ± 0.55	14.25 ± 0.64
GPT	17.25 ± 0.98	18.5 ± 0.78	19.0 ± 0.62	20.0 ± 0.55
Total Prot	6.88 ± 0.51	6.61 ± 0.25	7.15 ± 0.19	6.84 ± 0.12
Albumin	2.63 ± 0.02	2.28 ± 0.05	2.51 ± 0.04	2.31 ± 0.05
Globulin	4.25 ± 0.06	4.33 ± 0.12	4.64 ± 0.08	4.53 ± 0.07
Potassium	5.3 ± 0.11	5.47 ± 0.23	5.08 ± 0.16	5.03 ± 0.21
Magnesium	0.74 ± 0.01	0.71 ± 0.06	0.73 ± 0.09	0.72 ± 0.07
Sodium	108.6 ± 9.84	108.6 ± 8.54	106.5 ± 6.25	106.5 ± 7.28
Copper	0.69 ± 0.06	0.69 ± 0.09	0.65 ± 0.07	0.68 ± 0.05
Zinc	0.24 ± 0.04	0.28 ± 0.05	0.29± 0.04	0.23 ± 0.06
Urea	4.23 ± 0.11	4.77 ± 0.21	4.13 ± 0.32	4.04 ± 0.28
Creatinine	0.65 ± 0.02	0.65 ± 0.005	0.67 ± 0.01	0.55 ± 0.02

Values = means ± standard error

Table 3:- Biochemical changes in control rats

	Week 1	Week 2	Week 3	Week 3
Glucose	5.55 ± 0.08	5.51 ± 0.08	4.86 ± 0.07	5.06 ± 0.06
GOT	17.15 ± 0.34	18.25 ± 0.52	16.75 ± 0.43	15.25 ± 0.66
GPT	17.75 ± 0.31	17.25 ± 0.44	18.0 ± 0.35	19.0 ± 0.45
Total Prot	6.48 ± 0.34	6.5 ± 0.16	7.08 ± 0.22	6.84 ± 0.21
Albumin	2.85 ± 0.06	2.48 ± 0.06	2.55 ± 0.04	2.41 ± 0.05
Globulin	4.45 ± 0.03	4.82 ± 0.05	4.46 ± 0.04	4.52 ± 0.06
Potassium	5.55 ± 0.05	5.27 ± 0.08	5.08 ± 0.12	5.05 ± 0.09
Magnesium	0.69 ± 0.02	0.72 ± 0.09	0.73 ± 0.06	0.71 ± 0.08
Sodium	106.46 ± 6.32	108.82 ± 6.25	107.5 ± 7.11	106.25 ± 5.84
Copper	0.68 ± 0.02	0.69 ± 0.07	0.66 ± 0.08	0.68 ± 0.05
Zinc	0.25 ± 0.04	0.28 ± 0.03	0.26± 0.01	0.24 ± 0.02
Urea	4.54± 0.11	4.68 ± 0.07	4.33 ± 0.06	4.14 ± 0.05
Creatinine	0.65 ± 0.09	0.64 ± 0.14	0.67 ± 0.08	0.58 ± 0.07

Values = means ± standard error

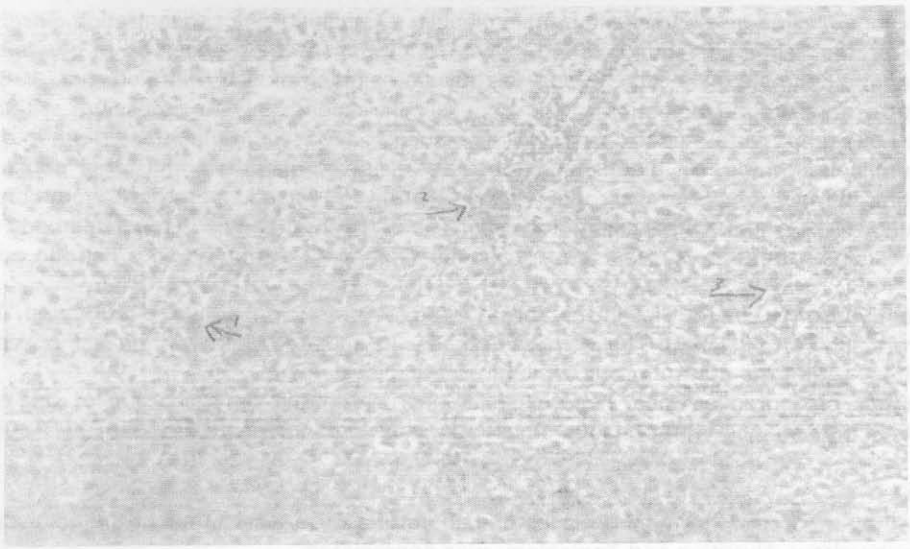

Fig. 1: Liver of rat experimentally infected with *T. evansi*
Note: Mild vascular degeneration and necrosis (H & E x 10)

week of infection ($P<0.05$). There was also gradual decrease in the concentration of zinc as compared to the control group. The group that treated with Cymelarsan showed no biochemical changes during the whole period of experiment (Table 2).
The histopathological changes recorded for the infected group was found to be degenerative changes in the liver and the kidney characterised by hemorrhage, focal hepatocellular necrosis and haemosiderosis (Fig. 1).

Discussion

The result of albumin showed that there was a significant decrease in the concentration of albumin in blood in the group which was infected and untreated. The decrease was marked when the mean parasitaemia count was high. There was no significant change in the other groups of rats where treatment was achieved with Cymelarsan. Albumin is synthesized in the liver therefore decrease in albumin concentration might be attributed to the damage in liver where there could be less synthesis of albumin. This result agreed with that of Arora and Pathok, (1995) who studied the biochemical changes in acute infection of *T. evansi* in dogs. He found that there was a significant decrease in the level of albumin in blood of infected dogs. He suggested that the edema might be the first sign in chronic disease caused by *T. evansi*. Results obtained here agreed with that of Ogunsanmi and Anosa (1994) who studied the serum biochemical changes in West Dwarf sheep experimentally infected with *T. brucei*. They found that the serum albumin value was depressed. The findings suggest that there might be a hepatic and/or renal malfunction. Their result also agreed with the present finding in the increase in the concentration of total protein found in the control group of rats. The total serum globulin is sometimes used as a crude measure of the severity of liver diseases. The increase in the concentration of globulin in the infected group of rats indicated that the immune system did not affected by the parasite. The concentration of glucose decreased in the infected group of rats where the decrease was found to be proportional to the trypanosomes count in blood. This decrease in glucose might be due to the high carbohydrate consumption of the parasite.
The low concentration of copper in the infected group of rats was found to be indicative for the presence of anemia, which is the main symptom of trypanosomosis. Copper in the form of ceruloplasmin is the factor responsible for the binding of Ferric (Fe^{++}) to Apoferritin to form Ferritin + Albumin and hence Transferin. This mechanism is usually carried out in the intestine which is the organ where copper is absorbed mainly in the duodenum (Milne, 1994). Another factor is that the liver is an important organ for copper storage and any type of hepatic damage will result in its inability to synthesize ceruloplasmin. This result agreed with that of Ogunsanmi and Arora, (1994) who found that the concentration of copper was depressed in sheep experimentally infected with *T. brucei*.

Zinc is transported in blood plasma by albumin and $á_2$-macrglobulin. Circulating zinc concentration closely correlate with the major carrier protein, albumin. Thus lowered plasma zinc concentration observed in hypoalbuminemia conditions, such as hepatic necrosis, may reflect depressed plasma binding of zinc (Simmer *et al*, 1987).
Magnesium is the fourth most abundant cation in the body and is second only to potassium within the cell. Magnesium and potassium are found to be decreased and the decrease was clear in the infected group of rats. The decrease in magnesium concentration in blood might be as a result of disrupt in the renal conservation of magnesium due to defect in renal tubular reabsorption (Milne, 1999). Decreased serum potassium concentration (hypokalemia) was found to accompany magnesium decrease. The primary organ which regulates body water and exracellular Na^+ is the kidney therefore the decrease in sodium might be attributed to a hepatic and/or renal malfunction.
The increase in the activity of GOT and GPT in the control group of rats suggest liver dysfunction. This result agreed with that of Arora and Pattok (1995). The increase in these enzymes together with the ALP enzyme activity was found to be significant with the control group which indicate liver damage.
The pathological changes presented as focal hepatocelullar necrosis in the control group of rats where the parasitaemia is high, is in consonance with previous observations in goats suggested by Saror, (1980).

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