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Studies on the serum electrolyte changes in Trypanosoma. Congolense infected New Zealand White rabbits

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Introduction Trypanosomosis is a protozoan disease of domestic animals and man characterised by dehydration weight loss, stunting, drop in milk yield, infertility, abortion and death (ILRAD, 1988). It ranks as one of the most economically important disease of livestock in Africa (Anosa, 1980). Trypanosomosis is caused by an haemoflagellate parasite called Trypanosoma spp transmitted by tsetse flies (Glossina spp). Although several studies have been done on the serum electrolyte changes accompanying trypanosomosis in different animal species. (Singh and Guy, 1983 and Zia-ur Rahmani et al, 1996). There are however very few reports in the literature on the serum electrolyte profile in the rabbit. This work is aimed at studying the serum electrolyte profile during a course of experimental infection by T. Congolense in New Zealand White rabbit.

Materials and Method. Twenty New Zealand White rabbits of both sexes, aged between 8 to 10 months were used for this study. The animals were clinically stabilised for 2 weeks before the infecting with T Congolense. Blood samples were collected from each animal for evaluation of serum electrolytes pre-infection. The twenty rabbits were afterward infected intraperitoneally with 10^6 T. Congolense as described by Lumsden et al (1973). Mice were used for the passage and maintenance of the trypanosomes as well as for the sub-inoculation for defection of trypanosomes in the infected rabbits. Blood samples were collected again on the eighth and fifteenth days post-infection for serological studies. Flame photometer was used to determine serum sodium (Na^+) ion and potassium (k^+) ion; Calcium (Ca^{2+}) ion by atomic absorption photometry; Chloride ion (cl^-) by spectrophotometric; phosphate ion ($HP0_4^{2-}$) by colorimeter and bicarbonate ion (HCO_3^-) by continuos flow colometric method. Student's t-test was applied to determine significance between pre-infection and 8 days post-infection (P.I) values,between pre-infection and 15 days post-infection (P.I) value.

Results. A decrease in the values of serum electrolytes was observed at the 8th day P.I for Na+, K+,Ca2+ and $HP0_4^{2-}$. This decrease is statistically significant ($P<0.05$) for k^+,Ca^{2+} and $HP0_4^{2-}$ when compared to the pre-infection values. While statistically significant ($P<0.05$) increase was observed for chloride ion and bicarbonate ion at 8th day P.I. (Table 1). As at 15th day P.I., there was increase in the serum concentration of Na^+, Ca^{2+}, HCO_3^- and $HP0_4^{2-}$ above what was recorded for 8th day P.I and pre-infection values, however the difference in the values between the pre-infection and 15th day P.I was only significant in Serum Ca^{2+} and HCO_3^-. While the values for potassium and chloride ions levels were the same as that of pre-infection values.

Table 1: Mean Serum electrolyte concentrations at pre-infection, 8 days post-infection and 15 days post-Infection

Parameters	Pre-infection (I)	8 days Post-infection (Iia)	15 days Post-infection (IIb)	P Value Ivslla	Ivsllb
Ca^{2+} mg/dL	10.94 ± 0.40	9.67 ± 0.73	11.93 ± 0.62	$p < 0.01$	$p < 0.05$
HPO_4^{2-} mg/dL	4.93 ± 0.25	4.33 ± 0.33	4.43 ± 0.28	$p < 0.01$	$p < 0.05$
Na^+ mEq/L	138.29 ± 1.19	138.00 ± 1.15	139.67 ± 2.19	$p > 0.05$	$p > 0.05$
Cl^- mEq/L	104.00 ± 1.02	105.67 ± 0.88	104.00±1.15	$p < 0.01$	$p > 0.05$
K^+ mEq/L	5.00 ± 0.13	4.70 ± 0.06	5.00 ± 0.15	$p < 0.05$	$p > 0.05$
HCO_3^- mEq/L	20.57 ± 0.61	23.33 ± 0.33	23.43 ± 0.32	$p < 0.01$	$p < 0.01$

Discussion. Hypocalcaemia was observed at the early stage 8 days P.I. The significant drop in the value of serum calcium ion level is most expected. Hypoalbuminaemia and haemodilution have been reported commonly in trypanosomiasis especially in cattle (Anosa, 1988) . Aliyu et al , (1997) commented that the increase in plasma volume is to compensate for the fall in red cell mass, this haemodilution is expected to cause a relative decrease in most of the Serum electrolytes values. However, calcium ion level specifically has been reported to have a direct relationship with Serum albumin, hence hypoalbuminaemia is accompanied with hypocalcaemia (Meuten, 1982). The hypercalcaemia observed at the later stage is due to the mobilising effect of parathormone and vitamin D activities on calcium. The increased activity of parathormone is suspected to be responsible for increased tubular reabsorption of calcium and decreased blood phosphorus concentration (Rasmussen, 1974). The result of this study showed a consistent hypophosphataemia which is also in line with the finds of Rainsinghani et al, (1981). Plasma expansion in response to decreased circulating RBC must have been due to neurohormonal responses which serve to increase water consumption via increased thirst and enhanced renal conservation of water (Rose, 1984). This is mostly achieved by increased antidiuretic hormone and mineralo corticoid (aldosterone) activity through the renin-angiotensin systems in the body. The hypokalaemia earlier observed therefore must have been mediated by increased aldosterone-induced renal potassium loss as sodium and water are preferentially reabsorbed in the renal tubules (Rose, 1984). Hypochloraemia and hypernatraemia occurred simultaneously in this study though values are not statistically significant. Divers et al, (1986) explained that changes in chloride concentration which are not associated with similar change in sodium concentration are usually associated with acid-base imbalances. Rose (1984) had earlier declared that excessive hydrogen ions loss associated with mineralocorticoid (aldosterone) excess may cause or contribute to generation of metabolic alkalosis. Metabolic alkalosis is clinically represented by increased bicarbonate and low chloride ions (Whitlock et al, 1975). Metabolic alkalosis is therefore implicated for the increased bicarbonate and low chloride ions recorded in this work. The findings in this study clearly indicates that electrolytes and acid-base imbalances could be as much important as the haematological effect in the pathology of trypanosomosis and of course the therapeutic measures to be taken for the treatment of the infected animals.

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