EFFECT OF ACUTE CAPRINE TRYPANOSOMIASIS ON HAEMOGLOBIN, UREA AND SERUM ELECTROLYTES

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The effect of acute caprine trypanosomiasis on haemoglobin (Hb) concentration, urea and serum electrolytes was studied in Red Sokoto goats infected with Trypanosoma vivax. The course of infection lasted only two weeks when the infected goats died of fulminating parasitaemia and high fever. Haemoglobin concentration of the infected goats was only slightly decreased. However, the serum urea level was significantly increased (P < 0.05) while CI, K+ and HCO3 levels were slightly increased above pre-infection values by week two post infection (PI). Serum Na+ increased only in the first week PI but returned to pre-infection values by the second week.

INTRODUCTION

African animal trypanosomiasis is a debilitating parasitic disease of livestock in sub-humid Africa resulting to huge economic losses annually (1,2). The disease has been described as probably the single most devastating disease in Africa in terms of poverty and lost agricultural production (2). The disease causes not less that 3 million livestock deaths each year and reduces calving rate, livestock numbers, milk yield, meat supply, work efficiency of draft animals and mixed farming (1). Despite the impact of the disease on man and his domestic animals, the exact factors involved in the pathogenesis or trypanosomiasis is not yet fully understood. The severity of pathology is dependent on the specie of infecting trypanosome and the host (3,4).

Trypanosoma vivax is highly pathogenic and a major threat to ruminants in West Africa (3,5). The disease in small ruminants hitherto, was believed to be of less economic importance. However, reports on both natural and experimental infections in sheep and goats (1,6) show that the impact of trypanosomiasis in small ruminants is substantial. Anaemia and other haematological changes and serum biochemical changes associated with the disease has been described (4,7). In this investigation we report haemoglobin levels, urea and serum electrolytes associated with acute T. vivax infection in Red Sokoto goats.

MATERIALS AND METHODS

Six adult female Red Sokoto goats weighing 12.3 to 20.7 kg body weight (BW) were used for the study. All the animals were purchased from local markets around Kaduna and screened for haemoarcsises before use; all the animals were negative for trypanosomes by either haematocrit centrifugation technique (HCT) or by Buffy Coat Method (BCM) (8). During the acclimatization period which lasted three weeks, each animals was treated with Ivomec®, MSD-AGVET, U.S.A. at the does of 1.0 ml/50 kg B.W. subcutaneously for internal and ecto parasite control. Oxytetracycline Long Acting (Pfizer, Ikeja, Nigeria) was also administered at the does of 1.0 ml/10 kg BW through the intramuscular (IM) route. Each goat was treated with diazinazene acetauvate (Benelir ®), vetimex, Bladel-Hollan, at 7.0 mg/kg BW, 1 ml. The animals were fed fresh grass, maize brain mixed with concentrates and water ad libitum. Four of the goats served as experimental (infected) group while the remaining two goats, served as control group. T. vivax (NITR/Federe) isolated from cattle and chyopreserved once, into a donor female Red Sokoto goat from where they were harvested from the jugular blood for inoculation 2 x 10⁶ parasites were inoculated into the goats intravenously. Blood samples collected weekly by jugular venipuncture were put into ethylene diamine tetracetic acid (EDTA) bottles and sterile Universal bottles for Hb determination and serum separation respectively. Sera samples were stored at 20°C till analysed. Haemoglobin estimation was done using the cyanomethaemoglobin method (9) to determine the Hb concentration. Sodium (Na+) and Potassium (K+) concentrations were determined using the flame photometer (Corning Model 400, Corning Scientific Limited, England). Chloride (Cl-) and bicarbonate (HCO3) were measured according to Toror and Ackerman (10) while serum urea level was determined as described by Harrison (11).

RESULTS

The infected goats became parasiteaemic 3 to 4 days post infection (PI). The infection in the Red Sokoto goats was virulent, characterized by fulminating parasitaemia and high fever. By week 2, PI, all the infected goats had died. The mean Hb level dropped slightly from 10.44 g/dl ± 1.34 before infection to 8.97 g/dl by week 2 PI (P > 0.05, fig. 1).

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Serum urea levels and electrolyte changes in Red Sokoto goats are shown on table 1. The Urea level of infected animals increased significantly (P<0.05) from preinfection value of 4.9 ± 2.50 (mmol/L) to 10.8 ± 1.7 (mmol/L) by week 2 PI. The serum Cr, K+ and HCO3 levels increased slightly above pre-infection values (P>0.05) by week 2. However Na+ increase only on week 1 PI but returned to pre-infection values by the second week.

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>PRE-INFECTION</th>
<th>POST-INFECTION</th>
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<tbody>
<tr>
<td></td>
<td>WEEK 1</td>
<td>WEEK 2</td>
</tr>
<tr>
<td>Urea (mmol/L)</td>
<td>4.9±2.5</td>
<td>7.12±0.3</td>
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<tr>
<td>Na+ (mmol/L)</td>
<td>143.08±5.8</td>
<td>160.0±10.5</td>
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<tr>
<td>Cr (mmol/L)</td>
<td>101±1.7</td>
<td>92.66±8.5</td>
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<tr>
<td>K+ (mmol/L)</td>
<td>3.60±0.6</td>
<td>4.4±1.0</td>
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<tr>
<td>HCO3 (mmol/L)</td>
<td>23.00±0.5</td>
<td>25.0±0.8</td>
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Table 1: UREA AND SERUM ELECTROLYTES IN T. VIVAX INFECTED RED SOKOTO GOATS

**DISCUSSION**

The acute nature of T. vivax infection in goats resulting to death without significant fall in the haemoglobin levels in infected animals suggest that Red Sokoto goats are highly susceptible to T. vivax and death may result from other pathogenic factors beside anaemia. The course of T. vivax infection observed in this study differs from the observations of Kalu et al (7) and Akinwale et al (12) in the same breed of goats. This is probably as a result of high virulence of the strain of parasite used. High urea levels recorded in this study has previously been observed in acute and sub acute trypanosomiasis in cattle caused by T. vivax (13) and T. rhodesiense (14) and T. gambiense infected monkeys (15). Urea levels are elevated during periods of high parasitaemia and fever which occur in acute infection (4). The causes of elevated Blood Urea Nitrogen (BUN) include kidney disease such as glomerulonephritis, urinary tract obstruction and excessive protein catabolism associated with severe toxic and febrile conditions (4). Fever and glomerulonephritis, are consistent features of trypanosomiasis and acute disease course in Red Sokoto goats which was characterized by fulminating parasitaemia and high fever. These factors therefore may have acted together to precipitate very high increase in blood urea level, and perhaps with accompanying early renal damage. The slight increase in serum bicarbonate level is in agreement with previous observations in T. brucei-infected bicarbonate ions by the kidney. The observed increase in sodium ions are also attributable to renal dysfunction (12). Slight decrease in serum chloride recorded in this study doest not agree with previous report on T. vivax infected goats (7). This might have arisen from the acute nature of the disease in the Red Sokoto goats. Kadima et al (17) however reported fluctuating levels of serum sodium and chloride ions which was associated with fluctuating parasitaemia in T. vivax infected cattle. Serum potassium cations also increased in the T. vivax infected Red Sokoto goats. A similar increase was reported in T. brucel and T. equiperdum infections of rats(4) and T. gambiense — infected Monkeys (15). Anosa, (4) reported that increases in the serum K+ levels of trypanosome infected animals correlated with decreased in Red blood cell (RBC) values. He attributed it to release of K+ from RBC and damaged tissue coupled with the effects of kidney damage. The mild anaemia observed in this study may therefore have been responsible for the mild increase in the serum K+ level in the infected goats.

The findings of this study suggest that kidney damage occur probably very early in T. vivax infected goats and may be one of the factors in the pathogenesis of trypanosomiasis in animals resulting to early death.

**REFERENCES**


