

**RENAL AND HEPATIC DYSFUNCTIONS IN EARLY PHASE OF
EXPERIMENTAL *TRYPANOSOMA BRUCEI* INFECTION OF NIGERIAN
PIGS.**

ABSTRACT

Aim: The effects of infection with *Trypanosoma brucei* on renal and hepatic functions in early phase of disease were studied in experimentally infected pigs. The aim of this study was to identify serum biochemical changes that could serve as biomarkers of early renal and hepatic dysfunctions and also serve as basis for therapeutic management of *T. brucei* infections in man and animals.

Study design: A total of 15 growing pigs aged between 6 and 12 months old were used for the study. The pigs were selected at random into two groups. Group one was made up of seven animals and served as the infected group. The pigs were each infected with 1×10^6 parasites in 2mls of normal saline subcutaneously. The second group, made up of six animals, served as the un-infected control group.

Place and duration of study: The pigs were housed in insect proof nets for two months while the experiment lasted.

Methodology: Blood for serum obtained through venipuncture of the anterior venacava was used for determination of the serum total proteins, albumin, creatinine and urea concentrations as well as activities of alanine and aspartate aminotransferase post infection.

Results: After infection there was a sharp increase in Total proteins($P = .05$)accompanied by decrease in albumin but increase in globulin concentrations on Day 5. Increases in, serum creatinine and Blood Urea Nitrogen(BUN) concentrations and, activities of Alanine and Aspartate amino transferases also occurred from this day.

Conclusion: It was concluded that decrease in serum albumin concentration alongside increase in creatinine and urea levels as well as those of alanine amino transferase and aspartate amino transferase are biomarkers of early onset of renal and hepatic pathology and determinants of ability to achieve self cure from anemia in *T. brucei* infections of man and animals. This underscores the relevance of erythropoietin use in chemotherapy of African trypanosomiasis and the roles of renal and hepatic integrity in trypanotolerance.

Key words: Renal, hepatic, function, *Trypanosoma brucei*, pigs.

INTRODUCTION

Variation in research results on serum biochemical studies in trypanosomiasis is influenced by both specie of infecting trypanosomes and animal hosts[1]. Such conflicting reports pose a problem in understanding of the pathogenesis and pathotology of the disease in animals and man as *T. brucei* infected animals serve as models for sleeping sickness in man[2 - 5]. Furthermore observations in pigs will help provide the needed consensus of opinion on the pathology of African trypanosomiasis and enhance its control. *T. brucei* is infective to all domestic animals with the exception of avian species; and antelopes. Disease course due to African trypanosomiasis had been described as being dependent on specie of infecting trypanosome, host and nutrition[6]. All trypanosome species exhibit antigenic variation, a phenomenon whereby blood stream trypanosomes switch from one variant surface glycoprotein (VSG) to another and

constitute a major setback to drug control and development of vaccine against African trypanosomiasis[7,8].

Infections by pathogenic trypanosomes cause diseases which vary in severity depending on the specie of infecting organism and susceptibility of the host[9]. Common clinical features of African trypanosomiasis have been summarized by several authors[6,10,11]. These include intermittent pyrexia, increased respiratory and heart rates, depression, pale mucous membranes (anemia), subcutaneous oedema, prominent jugular pulse, weight loss, enlargement of lymph nodes, hemorrhages, central nervous system disturbances, corneal opacity and disturbances in reproduction resulting to reduced libido, infertility and abortion; and death. In the human infective *T. gambiense* and *T. rhodesiense* and *T. brucei* infection of animals, two stages of clinical diseases have been recognized[2,12]. These are the early or haematolymphatic stage which is characterized by symptoms that occur before Central Nervous System (CNS) invasion by trypanosomes and the late or meningoencephalic stage. The early stage signs include the development of a chancre, an inflammatory swelling at the site of tsetse bite which is followed later by periodic pyrexia, joint pains, muscle aches, palpable enlargement of lymph nodes, generalized oedema which also extends to appendages, anemia, reduced libido, impotence, abortion, infertility and abnormalities of electro-cardiogram. The late stage signs are those associated with CNS involvement and which include ataxia, convulsions and other mental disorders.

Anaemia is a consistent feature of pancytopenia in trypanosomiasis of man and animals[1,6]. However self cure from anemia in early phase has been associated with antigenic diversity of *T. brucei* subspecies especially, *T. brucei brucei* infective to animals. Since not all antigenic strains exhibit this phenomenon, there are some unique differences in pathophysiology of haematological

derangement in *T. brucei* infections . The kidneys and liver play critical roles in erythropoietin biosynthesis and erythropoiesis[13]. Even though pathologies involving these organs are commonly reported in trypanosome infections, their roles in pathophysiology of anaemia has not been adequately investigated.

The aim of this study was to identify serum biochemical changes that could serve as biomarkers of early renal and hepatic dysfunctions and also serve as basis for therapeutic management of *T. brucei* infections in man and animals.

MATERIALS AND METHODS

A total of 15 growing pigs aged between 6 and 12 months old were used for the study. The animals were mixed breeds and aged as described by Kahn[14]. The animals were purchased from local pig farms within Zaria area and acclimatized for two months before the commencement of the experiment. During the acclimatization period, the pigs were dewormed using ivermectin (Kerpromec^(R), Deventer, Holland) a broad spectrum antihelminthic at the dose of 1ml/50kg body weight and administered subcutaneously. The animals were also administered orally, with amprolium chloride, a broad-spectrum anticoccidial. Diazintol^(R) (Diazinon dimpylate), a broad spectrum acaricide was occasionally administered topically for ectoparasite control fortnightly at the dose of 100ml/2000 litres of water. The animals were housed in insect proof pens. The pens were thoroughly washed with water containing antiseptic and sprayed with acaricides. The animals were fed growing pig ration as described by Eusebio[15].

Parasites Used

Trypanosoma brucei, NITR/Federe strain obtained from the Nigerian Institute for Trypanosomiasis Research, Vom was used. The parasite was isolated from cattle and

characterized biologically using resistance to human serum[16] after which it was cryopreserved in liquid nitrogen. From here they were subpassaged once into rats and then into a donor pig before inoculation

Experimental Groups and Infection of Animals

The pigs were selected at random into two groups.

Group 1: This was made up of seven animals given identity numbers 04, 05, 06, 08, 10, 13 and 14 and served as the infected group. The pigs were each infected with 1×10^6 parasites in 2mls of normal saline subcutaneously. Dosage was calculated as described by Lumsden[17].

Group 2: This was made up of six animals given identity numbers 02, 03, 07, 09, 11 and 12 which served as the un-infected control group.

Daily Parasitaemia

Wet blood films taken daily from the ear veins of infected pigs was examined under x 40 objective of a light microscope. 100 microscopic fields were examined before the result was declared negative when parasites were not observed.

Separation of Serum

Blood for serum was obtained through venipuncture of the anterior venacava using 19 gauge hypodermic needles and 10ml syringes. The site for the venipuncture was aseptically prepared by cleaning with 70% ethanol. Blood samples for serum separation were collected into clean test tubes containing no anticoagulant and were allowed to coagulate at room temperature for

approximately 30 minutes and the clot gently removed with the help of a thin glass rod. After this the sample was centrifuged at 3000G for 10 minutes using an MSE centrifuge (CEK-300, Gallenkamp, England). Sterile pipettes were used to remove the separated serum into chemical-free and sterile plastic specimen bottles and then immediately transferred into a deep freezer at -20°C for storage before use.

Serum biochemical techniques

Determination of the serum total proteins, albumin, creatinine and urea concentrations in the pigs was carried out using Kodak DT60 auto-analyser (Eastman Kodak Co, New York). Serum globulin was determined as a difference between serum total protein and albumin level.

The activities of alanine and aspartate aminotransferase in the pigs were determined using commercial kits as described by Henry *et al.*[18].

Statistical Analysis

The data analysis was done using Graph Pad Prism Version 4.0 Software (Microsoft^(R)). All data were expressed as mean $\pm$ SEM. Values between infected and control animals were compared using student t – test. Daily values within the infected group were compared using one way Analysis of Variance (ANOVA) and Tukeys Multiple Comparism Test. In all cases values of $P < 0.05$ were considered significant.

Results

The pigs were readily infected with *T. brucei* with marked parasitaemia and clinical disease even though they were local pigs and therefore like ruminants, were expected to display levels of trypanotolerance. The mean total proteins concentrations of the animals were 63.0 ± 9.5 (g/L)

and $67.0 \pm 0.0 \pm 2.1$ (g/L) for the infected and control groups respectively while the total proteins concentrations of all animals on day 0 ranged from 51.0 to 75.0 (g/L). After infection the mean total protein concentrations of the infected group progressively increased in the first week to 67.5 ± 3.8 (g/L) by day 5 ($P = .05$) after which it progressively dropped to 63.2 ± 3.7 (g/L) on day 17 (Fig. 1). The total proteins however increased thereafter to 69.0 ± 1.0 (g/L) by day 21 followed by fluctuating decreases to 65.7 ± 2.5 (g/L) by day 40, the values still apparently above those of day 0. During this period, total protein concentrations of control animals showed slight decreases within normal range. The serum albumin concentrations of all experimental animals ranged from 31.0 to 45.0 (g/L) by day 0 while those of infected and control groups was 39.0 ± 4.0 and 40.0 ± 6.0 (g/L) respectively. After infection the serum albumin concentrations increased slightly to 39.5 ± 3.7 (g/L) by day 5 in the first week of infection. (Fig. 2). Thereafter the albumin concentrations showed fluctuating decreases to 33.7 ± 6.6 (g/L) by day 40. During this period, the serum albumin concentrations of control animals dropped slightly within normal range. The mean globulin values of experimental and control groups of the pigs was 24.0 ± 2.1 and 27.0 ± 4.9 (g/L) respectively on day 0 while the variation in the values for all the animals on the same day was between 23.0 and 34.0 (g/L). However after infection, the globulin levels of infected group slightly increased to 34.5 ± 6.2 (g/L) on day 11 in the second week of infection and then slightly declined in values above those of pre-infection in subsequent days followed by the second peak increase to the maximum value of 41.3 ± 1.5 (g/L) ($P < 0.05$) by day 21. Thereafter, the serum globulin values dropped only slightly to 36.2 ± 2.6 , 32.9 ± 4.2 and 32.0 ± 6.6 (g/L) on days 27, 33 and 40 respectively. These increases were however not statistically significant ($P > 0.05$) (Fig. 3). The mean globulin levels of control animals ranged within normal level.

The serum creatinine values of all experimental animal ranged from 72.0 to 88.0 (mmol/l) on day 0 while the mean values for infected and control groups on the same day was 81.0 ± 12.5 and 67.2 ± 8.9 (mmol/l) respectively. After infection, the creatinine values increased only slightly in the first week with a further increase to 86.2 ± 8.5 (mmol/L) on day 5 (Fig.4) which was not statistically significant ($P > 0.05$). Thereafter, the creatinine values progressively dropped to 66.0 ± 8.2 (mmol/L) on day 17 which was followed by further fluctuating drops to 65.2 ± 5.9 (mmol/L) by day 40 which was slightly below pre-infections values. During this period, no significant changes occurred in the serum creatinine concentrations of the control group. The serum urea concentrations of all experimental animal, ranged from 2.5 to 4.7 (mmol/L) on day 0 while the mean concentrations for infected and control groups were 3.6 ± 1.0 and 3.6 ± 0.7 (mmol/L) respectively. After infection, the serum urea concentrations of the infected group increased progressively and peaked at 4.1 ± 1.2 (mmol/L) on day 17. This was followed by slight drop to 3.7 ± 0.7 (mmol/L) on day 21 and a second peak increase to 4.3 ± 1.0 (mmol/L) by day 27 (Fig.5). These increases were not statistically significant ($P > 0.05$). Thereafter, the concentrations progressively dropped to 3.7 ± 1.0 and 3.3 ± 0.7 (mmol/L) on days 33 and 40 respectively. The serum urea concentrations of control group however, varied within normal range but a careful observation showed a more overall increase in urea concentrations compared to that of creatinine in the infected group.

The mean of serum alanine amino transferase activities of infected and control groups on day 0 were 24.5 ± 4.4 (I.U/L) and 24.6 ± 3.7 (I.U/L) respectively while those of all experimental animals ranged from 19.0 to 28.0 (I.U/L) on the same day. After infection, the serum alanine

amino transferase activities showed progressive increases to the peak value of 26.0 ± 6.3 (I.U/L) by day 5 in the first week of infection and thereafter progressively dropped to 22.0 ± 4.8 (I.U/L) on day 11. This was followed by another progressive increase with a second peak activity of 27.2 ± 0.5 (I.U/L) on day 17 followed by apparent drop to below pre-infection activities on day 21 and then a third peak of sustained increase to activities slightly above those of pre-infection until day 40 (Fig. 6). The mean serum alanine amino transferase activities for control animals however fluctuated within normal.

The mean serum aspartate amino transferase activities of infected and control groups were 12.0 ± 2.8 and 11.7 ± 3.8 (I.U/L) respectively on day 0 while the activities for all animals on the same day ranged from 9.0 to 16.0 (I.U/L). After infection, the levels of serum aspartate amino transferase showed slight progressive increase above pre-infection values in the first 21 days (Fig. 7). Thereafter the aspartate amino transferase activities increased to attain the peak level of 18.0 ± 1.4 (I.U/L) on day 27 which was statistically significant ($P = .05$). This was followed by a gradual drop to 13.5 ± 1.7 (I.U/L) on day 40. The mean aspartate amino transferase activities of the control group only fluctuated within normal range within the period.

Discussion

The level of total proteins in the infected pigs showed fluctuating increase during the period and was accompanied by hypoalbuminaemia and hyperglobulinaemia whose fluctuation patterns closely followed that of the changes in total proteins. Total protein may either be normal, increased or decreased in African trypanosomiasis depending on factors such as nutritional status of the host, host species and species of infecting trypanosome[1]. Elevation in the serum total

proteins as well as albumin and globulin levels in the pigs after infection support earlier observations in humans, goats and rabbits and monkeys infected with *T. rhodesiense*[19], *T. brucei* [20, 21] and *T. gambiense* [22] respectively and pigs infected with *T. brucei* [23] . Awoboade[24] observed that *T. gambiense* infected human populations showed higher plasma total proteins which was accompanied by decrease in albumin levels but increase in globulin levels. The increases in total proteins in African trypanosomiasis is believed to arise from hyperglobulinaemia and especially hypergamma-globulinaemia and has been associated with increase in immunoglobulin M (1gM) as well as dehydration which are a consistent finding in trypanosomiasis of man and animals[25]. Albumin is produced entirely in the liver and plays important roles in the regulation of flow of water between the plasma and tissue fluids by its effect on colloid osmotic pressure. Hypoalbuminaemia on the other hand is therefore the result of decreased protein synthesis due to hepatic dysfunction[25] in trypanosomiasis but may also be due to increased protein loss through the gut or kidney, malabsorption and plasma dilution directly or indirectly related to trypanosomiasis. Hypoalbuminaemia observed in this early phase of *T. brucei* infection in the pigs suggests the early setting in of hepatic pathology in the pig.

The serum creatinine concentrations only increased slightly in the first week of infection which agree with an earlier observation by Sadun *et al.* [26] in rhesus monkeys infected with *T. rhodesiense*. Abenga and Anosa[22] observed similar early slight increase by week 4 in *T. gambiense* infected vervet monkeys which thereafter dropped to pre-infection values while Awobode [24] reported higher plasma creatinine levels in *T. gambiense* infected population than their uninfected control group. Creatinine is formed during normal muscle metabolism and is believed to be excreted at a relatively constant rate regardless of diet, age or other factors while its increase in serum levels is associated with conditions that impair glomerular filtration[13].

Like the liver, the kidneys play roles in erythropoietin biosynthesis and regulation of erythropoiesis and ability to respond to anemia. Increase in the serum creatinine levels therefore signals the early setting in of renal pathology and eventual compromise of the role of the kidney in responding to anaemia in trypanosomiasis. Early increase in creatinine levels in the pigs therefore suggests there was renal pathology though not severe enough to impede erythropoietin biosynthesis.

The serum urea level in the infected group showed fluctuating increase most part of the infection period. These increases were consistent with the previous observations in *T. brucei* infected rabbits[27] and goats [20] and *T. gambiense* – infected vervet monkeys [28]. Elevated serum urea levels have been similarly associated with kidney diseases such as glomerulonephritis, urinary tract obstruction and excessive protein catabolism associated with severe toxic and febrile conditions [25]. Fever and glomerulonephritis are consistent features of African trypanosomiasis [2]. Serum alterations in urea level therefore suggest too that *T. brucei* caused early onset of renal pathology in the infected pigs. However these increases were inferior to those described by Abenga *et al.* [29] in acute *T. vivax* infection in Red Sokoto goats in which the serum urea level in the infected animals increased by 50% in the second week followed by early death of the animals. These observations suggest that in the early phase *T. brucei* as well as *T. gambiense* infection of animals and man, the kidneys still retain ability for effective erythropoietic response as they are less affected and therefore able to overcome early or crippling effect of anaemia. Anosa[1] and Anosa *et al.*, [30] had described erythropoietic response to anaemia in African trypanosomiasis which usually sets in during the first wave of parasitaemia in the acute or early phase of disease. These responses are characterized by marked erythroid

hyperplasia with reticulocytosis and gross expansion of red bone marrow in the long bones in the early phase but bone marrow hypoplasia and drop in reticulocyte counts due to low erythropoietic response in the chronic phase. They further concluded that superior reticulocyte response in *T. brucei* infections suggests that erythropoietin plays a major role in recovery from anaemia in early phase of *T. brucei* infected animals[1], but this activity is hindered in the chronic stage due to advanced pathological changes in the liver and kidney.

The mean serum levels of alanine amino transferase showed fluctuating increase from day 5 in the infected group. Even though these increases were not significant, they were more sustained towards the last half of the forty days observation period. A similar mild increase and pattern were observed in monkeys infected with *T. gambiense* [31] while Awobode[24] reported that the alanine aminotransferase levels in *T. gambiense* disease of humans were about twice those of the un-infected human population. The serum aspartate aminotransferase levels were however remarkably increased towards the half of the observation period. These observations were however at variance with those of Olowe[32] who reported normal levels of alanine and aspartate amino-transferases in a case of congenital *T. gambiense* infection in an infant. These increases however agreed with the observations of Abenga and Anosa[31] in *T. gambiense* infected vervet monkeys where the serum aspartate aminotransferase concentrations increased by more than a hundred percent by week four post infection and tripled by the tenth week when the animals died. Alanine and aspartate aminotransferases are found primarily in tissues that have high rates of protein metabolism especially kidney, liver and muscle[13] and damage to body cells result to alteration of membrane permeability and consequent release of these enzymes into the extracellular fluid. Although increase in the serum activities of these enzymes have been

described as a good marker of soft tissue damage, their increases in trypanosomiasis have been attributed to hepatocellular and renal damage[25]. However elevation in serum levels of these enzymes may also result from trypanosome lyses due to the host's defence mechanisms[33].

It was concluded that decrease in serum albumin concentration alongside increase in creatinine and urea levels as well as those of alanine amino transferase and aspartate amino transferase are biomarkers of early onset of renal and hepatic pathologies and determinants of ability to achieve self cure from anemia in *T. brucei* infections of man and animals. This underscores the important role of erythropoietin administration in the management of anemia in African trypanosomiasis and the place renal and hepatic integrity in achieving tolerance to the disease.

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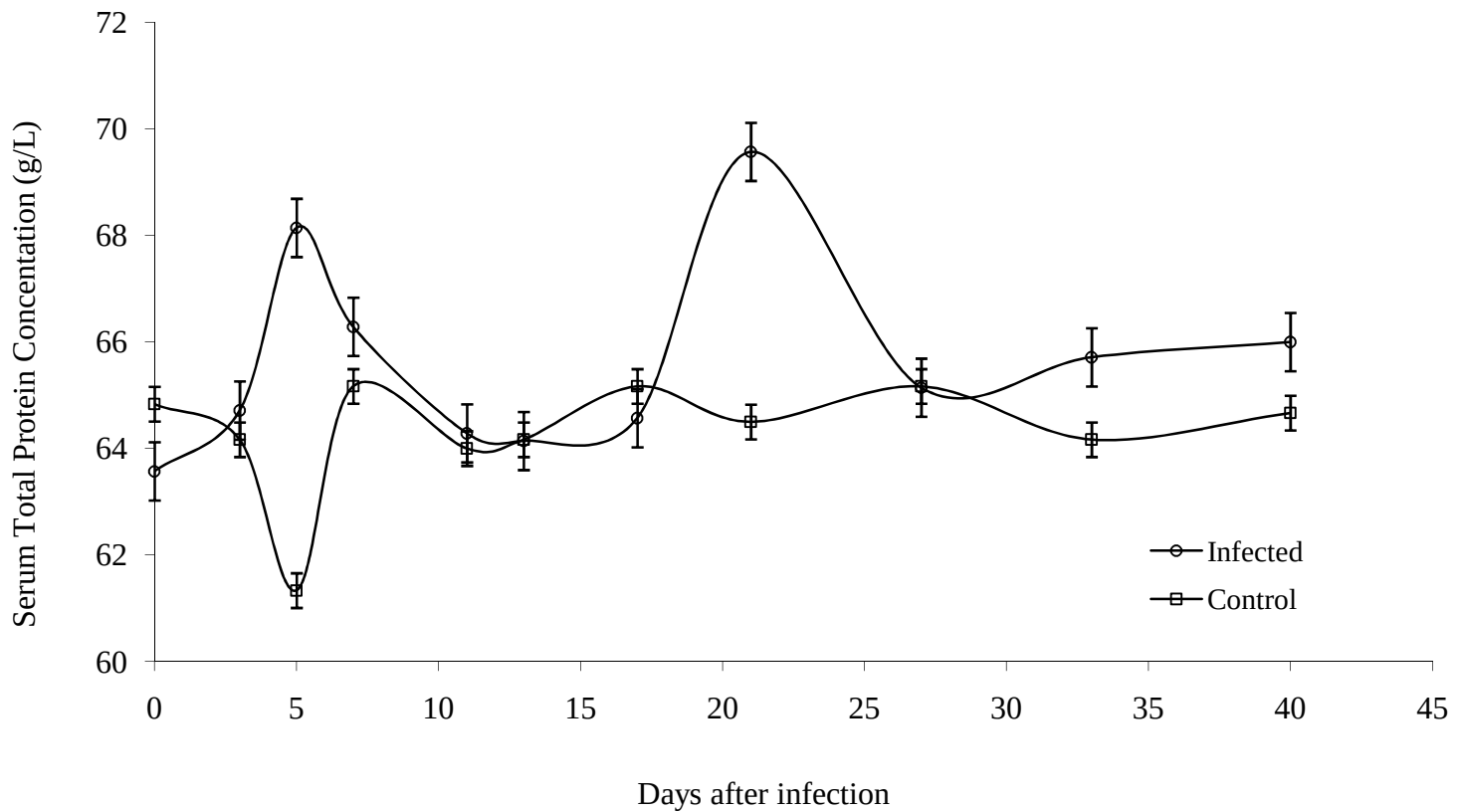


Fig. 1. Variation in the mean serum Total proteins concentrations of pigs infected with *T. brucei*..

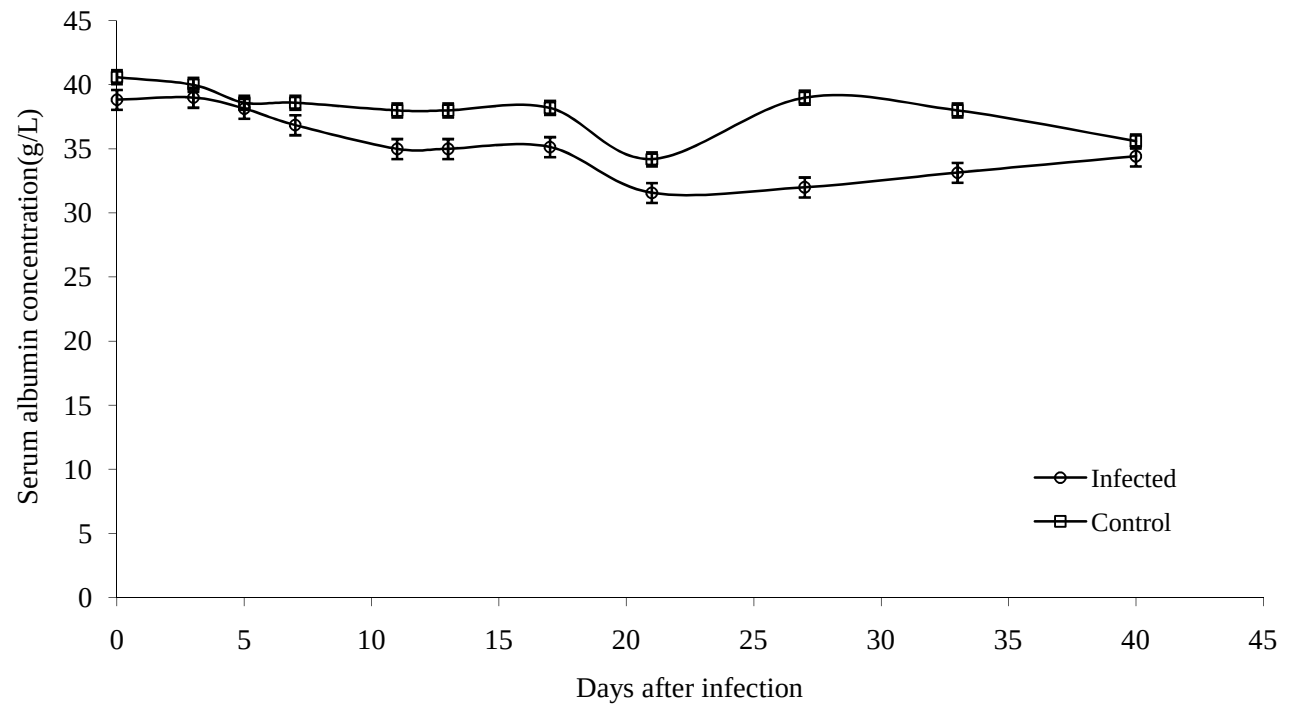


Fig. 2: Variation in the mean serum albumin concentrations of pigs infected with *T. brucei*.

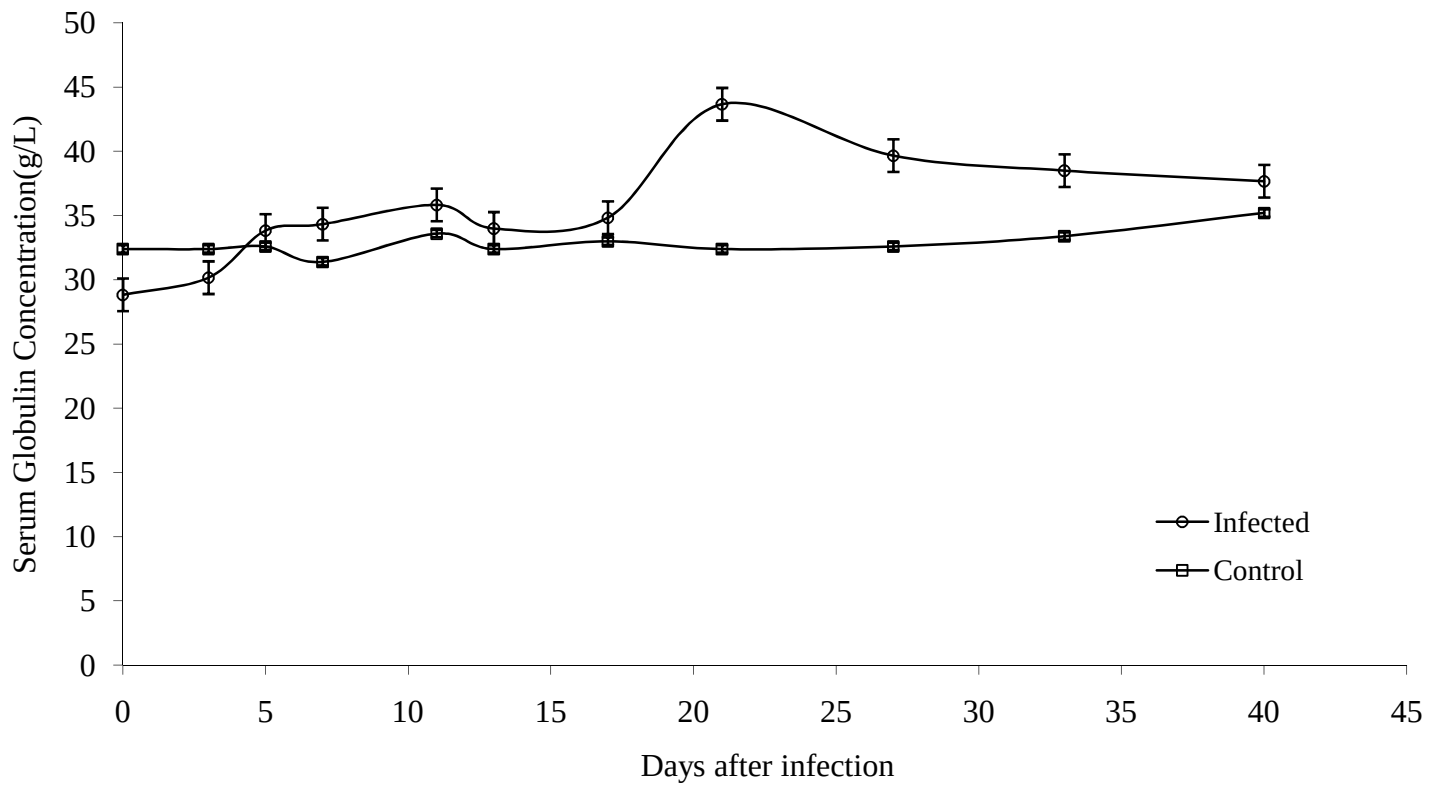


Fig.3: Variation in the mean serum globulin concentrations of pigs infected with *T. brucei*

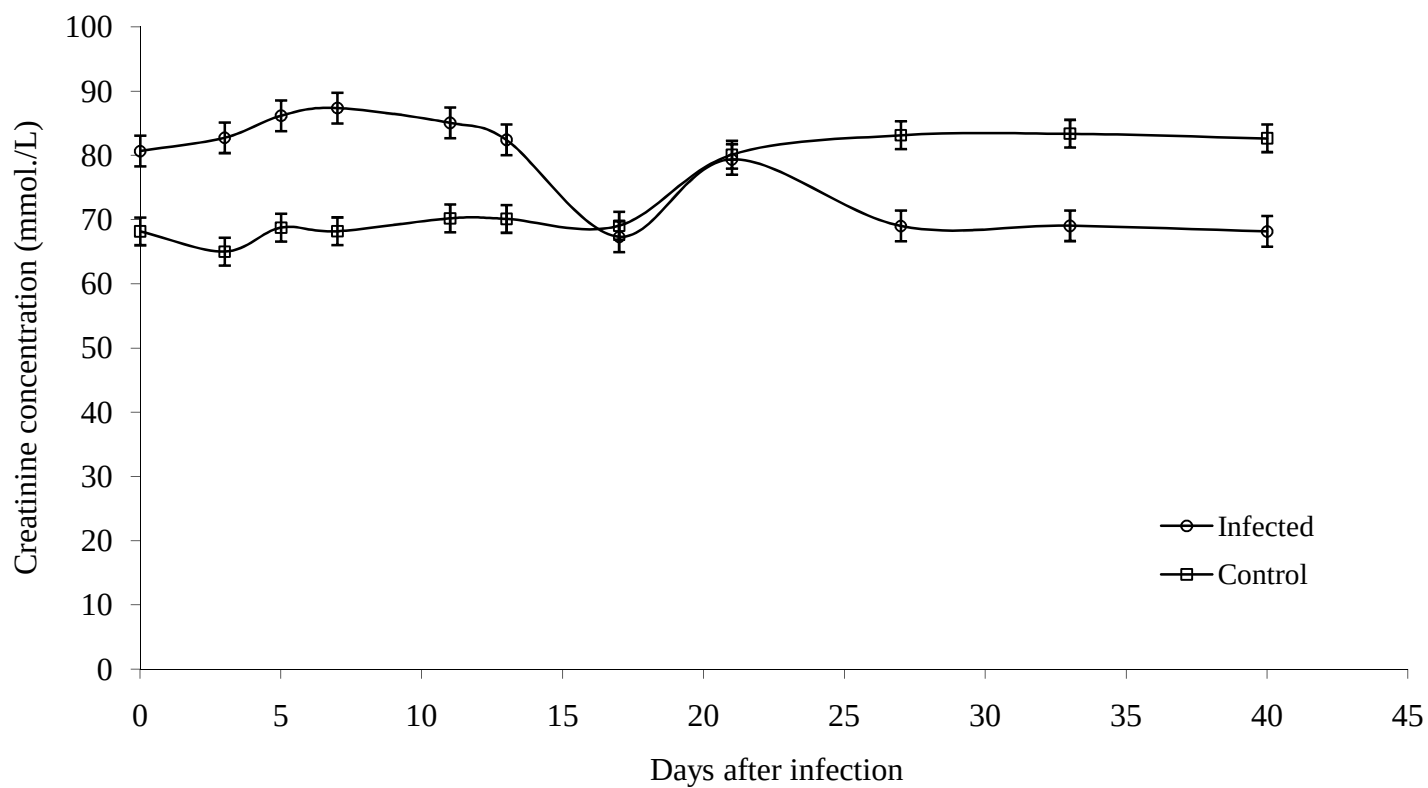


Fig. 4: Variation in the mean creatinine concentrations of pigs infected with *T. brucei*.

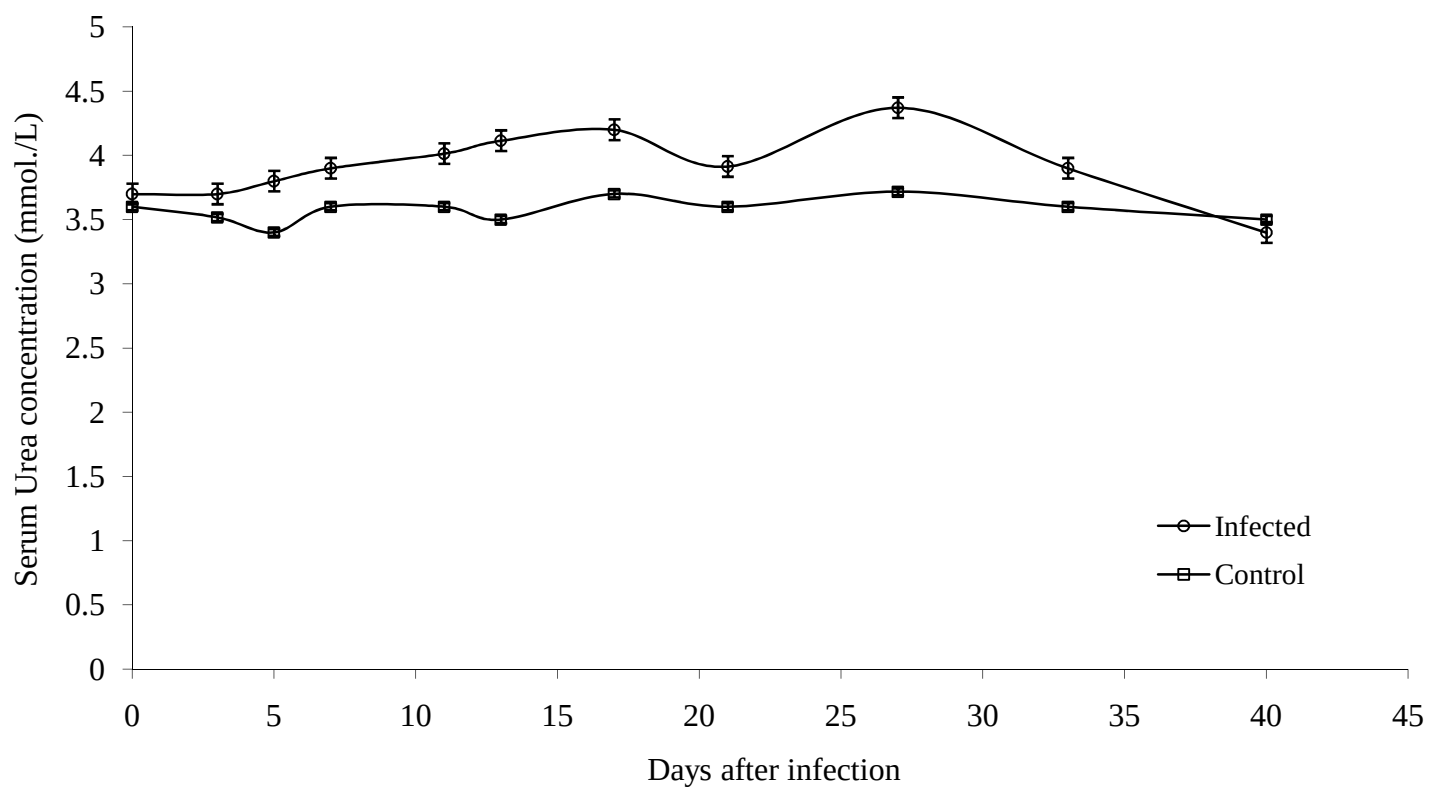


Fig. 5. Variation in the mean serum urea concentrations of pigs infected with *T. brucei*.

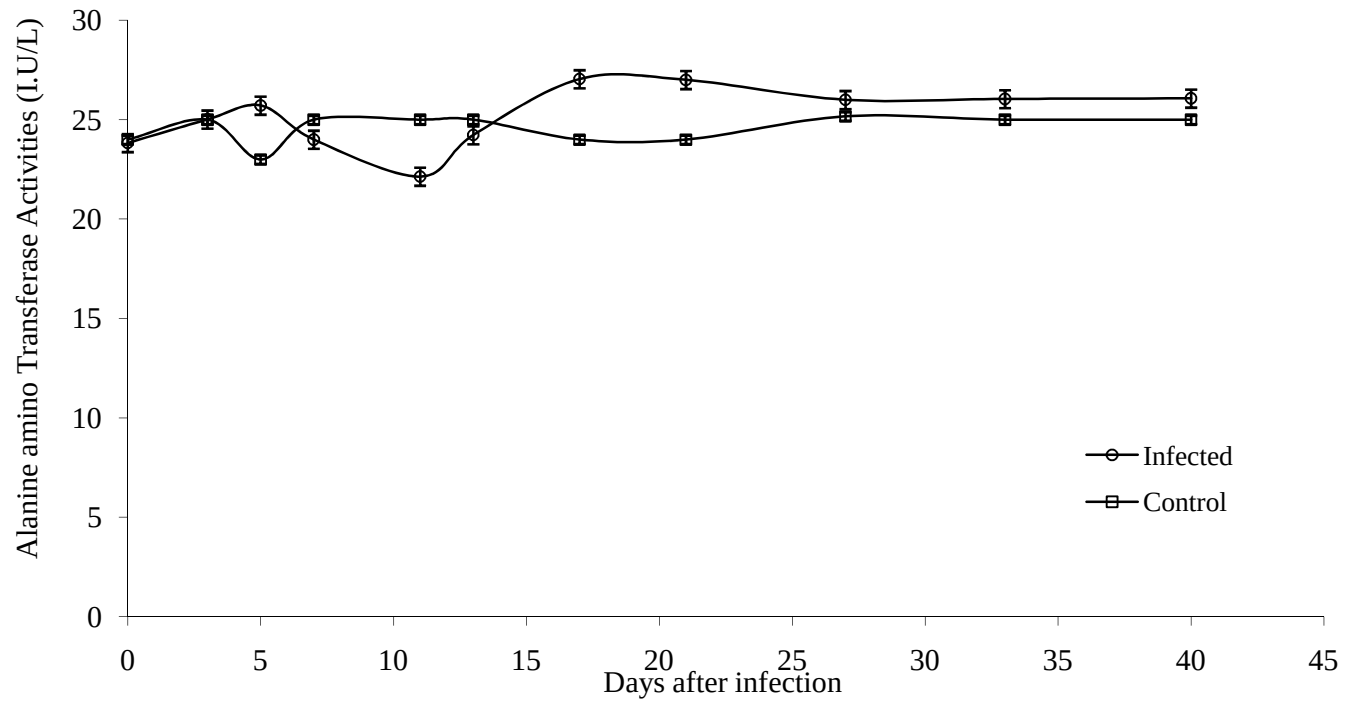


Fig. 6. Variation in the mean serum alanine amino transferase activities of pigs infected with *T. brucei*.

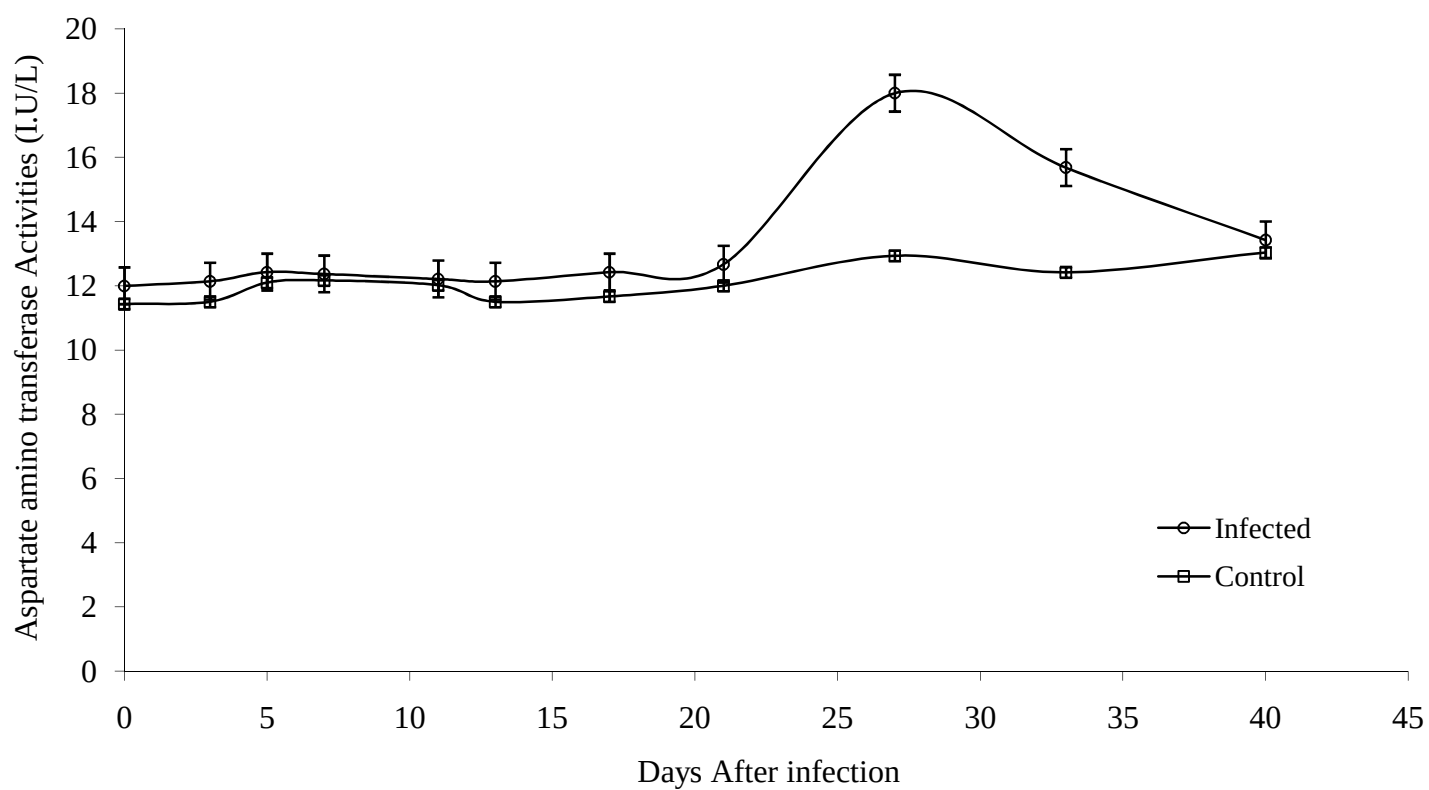


Fig. 7. Variation in the mean serum aspartate amino transferase activities of pigs infected with *T.brucei*