Original Research Article

Influence of Diaminazene Aceturate (Berenil®) on the Haematology of Yankasa Sheep Experimentally Infectedwith *Trypanosomaevansi*

ABSTRACT

Haematological profiles were determined in Yankasa sheep experimentally infected with *Trypanosoma evansi* (*T. evansi*) and treated with diaminazene aceturate (berenil®). A total of 30 animals were divided into 6 groups (A to F) (n=5). Animals from each group were either uninfected or infected with *T. evansi*, and treated with Berenil®. Infection of the infected groups (A, C and E) was done via intravenous inoculation of *T. evansi*, while the infected group C and E were treated with berenil® at 3.5 and 7 mg/kg BW, respectively, by day 16 post infection (PI). The infected groups had pre-patent period of 8 days, with similar levels of parasitaemia (4.7 ± 0.27). In group A, the mean parasite count rose significantly ($p < 0.05$) to 72.8 ± 1.07 by day 12 PI and continue to a peak value of 250.6 ± 1.98 by day 28 PI. In group C and E, the initial parasitaemia rose significantly ($p < 0.05$) to 80.8 ± 1.12 and 78.2 ± 1.11 by day 12 PI, following treatment with 3.5 and 7.0 mg/kg BW of berenil®, by day 20 PI, respectively, and was completely eliminated by day 9 and 5 post treatment (PT), respectively. As parasitaemia increased, PCV, RBC, Hb, WBC, platelets count, absolute lymphocytes and monocytes, significantly declined ($p<0.05$) in group A, leading to anaemia at day 16 PT. It is therefore,

demonstrated that both the two doses of berenil® were effective in the treatment of the disease under experimental conditions but 7.0 mg/kg cleared the parasitaemia faster.

KEYWORD: Diaminazene aceturate, Experimental, Haematology, *Trypanosoma evansi*, Yankasa breed of sheep

1. INTRODUCTION

Trypanosoma evansi, known as"surra", is a disease of most domestic and wild animals. The pathogenic effect of the disease varies according to the virulence of the trypanosome strain, the host, species, general stress and other concurrent infections. [1]. *Trypanosoma evansi* is the most widely distributed of the pathogenic animal trypanosomes affecting camel, cattle, buffalo, horses and donkeys [2], causing economic losses including; severe weakness, abortion, infertility, reduced milk yield, weight loss and reduced drought capabilities that later lead to neuropathy and immune suppression coupled with anemia and eventually death of the animal [3]. *Trypanosoma evansi* is wide spread in different ecological regions in Africa, Americas, some parts of Europe and Asia [4]. Various species of domestic livestock can be infected with *T. evansi*, including horses, camels, buffaloes, sheep, goats, cattle and pigs. It is primarily transmitted mechanically by biting flies from the species like *Tabanus*, *Stomoxys*, *Haematopota*, *lyperosia*, and *Chrysops spp*[5]. In South-America, next to flies, vampire bats, during certain seasons can transmit the disease [6]. The diagnosis of surra is usually based on the demonstration of the parasite in blood, supplemented by haematological, biochemical and serological tests [7]. In general, clinical sign of *T. evansi* infections include; pyrexia directly associated with parasitaemia together with a progressive anaemia, and loss of condition [8].

The field control of animal trypanosomosis has, over the years, relied on two broad strategies: using chemotherapeutic agents on infected animals and vector control. The chemotherapeutic approach is used much more widely acceptable than vector control because it is easier to eliminate the trypanosomes than the flies [9]. Chemotherapy, exerts its action by stopping the multiplication of the trypanosomes, thereby helping the immune system to overcome the infection [10]. Chemotherapeutic drugs are also known to be toxic and often have a similar

disruptive effect on the cells of the host [11], and are therefore always used with care and at the recommended dose level only [12]. The most widely used and preferred curative trypanocide is diaminazene aceturate [13]. Other trypanocidal drugs that can be used include isometamidium chloride (curative and preventive), cymelarsan (curative and used only in camels), suramin, and quinapyramine (curative and preventive) [14].In Nigeria, extensive work has been done on other animal trypanosomosis, but there remains paucity of information on infection due to *T. evansi* especially in Yankasa breed of sheep. Therefore, this research was designed to evaluate the haematological changes in Yankasa breed of sheep experimentally infected with *T. evansi* andwith different doses of diaminazene aceturate.

2. MATERIALS AND METHOD

2.1 STUDY LOCATION

The study was conducted at the Department of Veterinary Medicine, University of Maiduguri, Maiduguri, Borno State capital, which lies in the northeastern geopolitical zone of Nigeria. The state is situated within the semi-arid zone of west Africa. It lies on latitude 11^{O} 5'N and 13^{O} 5'E. It has a total area of 72,609 square km, with temperature ranging from 35-40^{o}C for most of the year

2.2 EXPERIMENTAL ANIMALS

A total of thirty (30) adult Yankasa breeds of sheep between 12-18 months of age with average weight of 30.22 ± 25kg were procured from Maiduguri livestock cattle market and used for this study. The animals were kept in fly-proof pens and acclimatized for a period of two weeks. During this period, they were screened for haemoparasites and helminths by collecting blood and fecal samples for standard clinical examination procedure, respectively. They were fed twice a day with hay and concentrate supplement while salt lick and water were given *ad libitum.*

2.3 EXPERIMENTAL DESIGN

The 30 adults Yankasa breed of sheep were randomly divided into six groups (A to F) of five animals each and labelled as follows:

Group A infected with 0.5ml of *Trypanosoma evansi,* but untreated control.

Group B was uninfected and untreated control.

Group C was infected with 0.5ml of *Trypanosoma evansi* and treated with diaminazene aceturate (berenil®) at a single dose of 3.5mg/kg body weight at day 16 post infection (PI).

Group D was uninfected, treated with diaminazene aceturate at a single dose of 3.5mg/kg body weight at day 16 (PI).

Group E was infected with 0.5ml of *Trypanosoma evansi* and treated with diaminazene aceturate at a single dose of 7.0mg/kg body weight by day 16 (PI).

Group F was uninfected, treated with diaminazene aceturate at a single dose of 7.0mg/kg body weight by day 16 (PI).

2.4 INFECTIONS OF THE EXPERIMENTAL ANIMALS

Trypanasoma evansi used in this study was obtained from the Department of Veterinary Parasitology and Entomology, Ahmadu Bello University Zaria, Kaduna State, Nigeria. The organism was initially isolated from the blood of naturally infected Camel in Kano State Abattoir and maintained by serial passage in albino rats. Six (6) adult albino rats were used as donors. After patency, the donor rats were bled via the tail vein into a petri dish diluted with phosphate buffered saline glucose (pH 7.4). Each sheep in group A, C and E were inoculated through the jugular intravenous (IV) route with 0.5ml of blood containing 1.0×10^6 *Trypanosoma evansi.*The estimation of the number of infective trypanosomes was determined using the rapid matching method of Herbert and Lumsden [15].

2.5 ESTIMATION OF PARASITEMIA AND CLINICAL SIGNS

One milliliters of blood were collected from the jugular vein every 4 days into sample bottles containing EDTA. This blood was used to estimate parasitemia Parasitemia was determined using wet mount and hematocrit centrifugation techniques for the detection of trypanosomes as described by Adeyeye et al. [16] and Desquesnes et al. [17]. The number of parasites was estimated following the method described by Herbert and Lumbsden[18]. Post inoculation the animals were closely monitored for clinical signs of trypanosomosis such as pyrexia, palor of visible mucous membranes, anorexia, depression, lacrimation, nasal discharge, enlargement of lymph node.

2.6 SOURCE OF DRUG AND TREATMENT

Diaminazene aceturate berenil® used in this study was manufactured by InterchemieWerken "De Adelaar" B.V. Metaalweg and venry, Holland. It was administered at a single dose of 3.5 mg/kg body weight to groups C and D, and 7.0mg/kg body weight to groups E and F, by deep intramuscular (IM) route on day 16^{th} post infection at the peak of parasitaemia ($>45\times10^3$/uL).

2.7 BLOOD SAMPLE COLLECTION

About 1 ml of blood sample were obtained from the experimental animals via jugular vein, every 4 days (day 0, 4, 8, 12, 16, 20, 24 and 28) and dispensed into EDTA sample bottles. Blood samples collected were properly labelled according to group and used for determination of haematological parameters.

2.8 DETERMINATION OF HAEMATOLOGICAL PARAMETERS

Determination of packed cell volume (PCV) was carried out using microhaematocrit centrifuge as described by Coles, [19], Haemoglobin concentration (HB) were determined using cyanmethaemoglobin method [20], Red blood cell counts (RBC) and Total white blood cell counts (TWBC) were determined using Neubauer haemocytometer as described by Coles, [21], while thin blood smears stained with Giemsa stain were used to determine differential white blood cell counts. The Platelet counts were determined by the use of commercial kit (Randox lab Ltd, Ardmore, U.K.) according to the method of Cheesbrough, [22], via improved Neubauer counting chamber.

2.9 STATISTICAL ANALYSES

Data generated were expressed as mean ± standard deviation (S.D) using Analysis of variance (ANOVA). Turkey – Kramer multiple comparison test was used to compare within and between group means and $P< 0.5$ was considered significant [23].

3. RESULTS

3.1 Mean Parasite Counts ($\times 10^3$/ul)

Themean parasite counts of the sheep experimentally infected with *T. evansi* and treated with diaminazene aceturate and their controls are presented in Figure 1. All the infected groups (A, C, and E) had a pre-patent period of 8 days post infection (PI) with a uniform mean parasitaemia of 4.7± 0.27. In group A, the mean parasitaemia count of 4.7± 0.27 by day 8 (PI), rose significantly ($p<0.05$) to 100.2 ± on day 16 PI and continued without abatement to a peak value of 250.6 ± 1.98 by day 28 PI. At these points, the mucous (ocular and buccal) membranes of the animal in this group became pale. They became extremely weak, disinclined to move, anorectic and recumbent. They had to be humanly euthanized at this point (day 28 PI) to avoid painful death in accordance with international guidelines of using uninfected/uninfected animals for biomedical research. At this point, (day 28 PI), it was deemed that all the infected untreated animals had died of the infection. In the infected treated groups C and E, the initial parasitaemia of 4.7 ± 0.27, which occurred on 8 PI, reached a significant ($p<0.05$) peak count of 80.8± 1.12 and 78.2 ± 1.11 on day 12 (PI), respectively. Following treatment, the parasitaemia dropped significantly ($p<0.05$) to 20 ± 0.56 on day 9 post treatment and subsequently cleared from the blood stream. In group E the parasite was completely cleared from blood stream on day 5 post treatment. No death or relapse parasitaemia was encountered for 90 days after monitoring parasitaemia in the infected, treated groups (C and E) respectively.

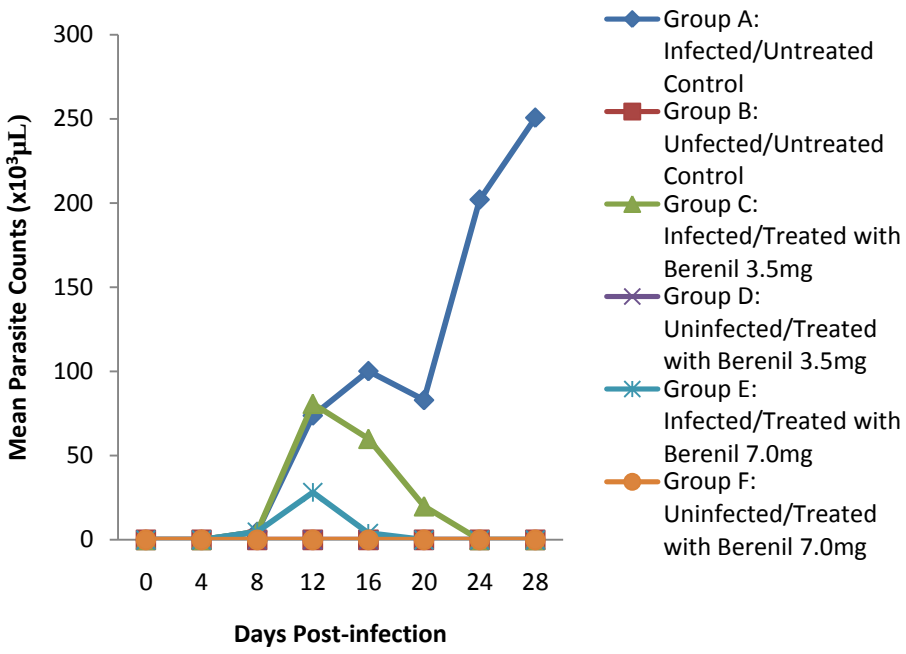

Fig. 1. Mean parasite counts ($x10^3/\mu L$) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

3.2 Packed cell volume (PCV)

The mean PCV changes of the sheep experimentally infected with *T. evansi* and their controls are presented in Figure 2. In group A, the pre-infection PCV value of 38.7 ± 0.78 experienced a gradual but significant decline ($p<0.05$) from day 8 PI, when parasitaemia became patent. By day 16 PI, parasitaemia had declined to 17.4 ± 0.52 and it continued unabatedly to 15.0 ± 0.48 on day 28 PI. In group B, D, and F, the pre-infection PCV of 38.8 ± 0.78, 38.5 ± 0.78 and 38.2 ± 0.77 all remained fairly constant ($p<0.05$) all throughout the period of the experiment. Meanwhile in group C, the pre-infected value of 38.4 ± 0.77 rose to a peak count of 20.7 ± 0.57. Following treatment with 3.5mg/kg BW of berenil® on day16 PI, the declined values began to rise significantly ($p<0.05$), until its pre-infection PCV of 38.4 ± 0.77 was attained on day 28 PI (day 13 PT). Similarly, in group E, the pre-infection PCV of 38.4 ± 0.78 also declined significantly ($p<0.05$) to 19.4 ± 0.56. Following treatment with 7.0mg/kg BW of berenil®, thevalues rose sharply ($p<0.05$), thereby attaining its pre-infection value on day 24 PI (day 9 PT)

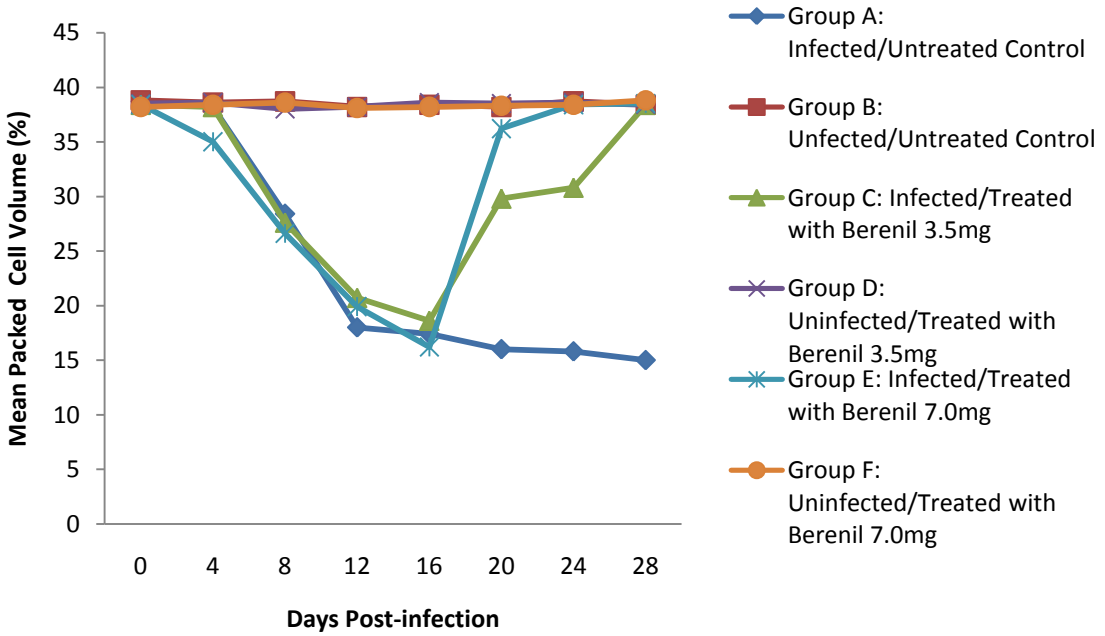

Fig. 2. Mean packed cell volume (%) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

3.3 Red blood cell count

The mean total red blood cell counts of the sheep experimentally infected with *T. evansi* and their controls are presented in Figure 3. In group A, the pre-infection value of 11.84 ± 0.43, experienced a significant ($p<0.05$) decline which continued without abatement to 5.0 ± 0.28 on day 28 PI. In group B, D and F, their pre-infection values of 11.82 ± 0.43, 11.84 ± 0.4 and 11.80± 0.43 all remained fairly constant ($p<0.05$) throughout the study. However, in group C, the pre-infection value of 11.80 ± 0.43 declined significantly ($p<0.05$) to 7.0 ± 0.34 on day 16 PI. Following treatment with 3.5mg/kg BW of berenil® on day 16 PI, the declined values began to rise significantly ($p>0.05$) thereby, attaining its pre-infection value on day 28 PI or on day 13 PT. Similarly, in group E, the pre-infection value of 11.81 ± 0.43 declined significantly ($p<0.05$) to 7.0 ± 0.33 on day 16 PI. Following treatment with 7.0mg/kg BW of berenil® on day 16 PI, the declined values appreciated significantly ($p<0.05$) to its pre-infection value on day 24 PI or on

day 9 PT.

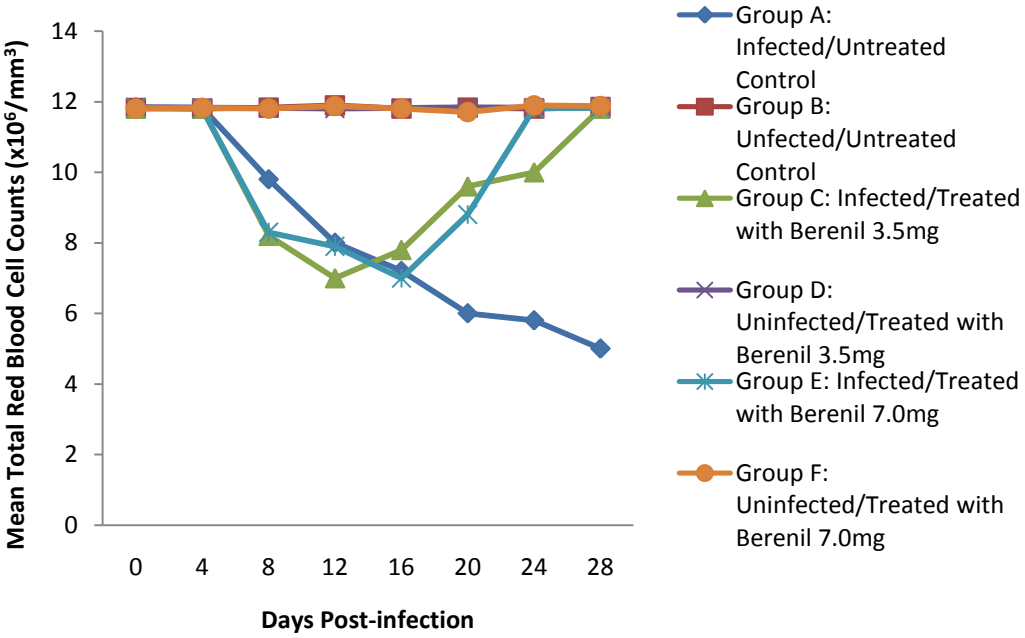

Fig. 3. Mean total red blood cell counts ($x10^6/mm^3$) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

3.4 Haemoglobin concentration

The mean haemoglobin concentrations of the sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls are presented in Figure 4. In group A, the pre-infected value of 9.5 ± 0.39 experienced a significant decline ($p<0.05$) to 6.0 ± 0.31 on day 16 PI. Thereafter, the value began to decline further to 4.0± 0.25 on day 28 PI. Meanwhile in group B, D, and F, the pre-infected values of 9.6 ± 0.39, 9.5 ± 0.39 and 9.7 ± 0.39 respectively, remained fairly constant ($p>0.05$) throughout the study. However, in group C the pre-infection value of 9.7 ± 0.39 declined significantly ($p<0.05$) to 5.8 ± 0.30 on day 16 PI. Following this treatment on day 16 PI, the declined values began to rise significantly ($p<0.05$),

thereby attaining its pre-infection value on day 28 PI or on day13 PT. Similarly, in group E, the pre-infection value of 9.7 ± 0.39 declined significantly ($p<0.05$) to 7.0 ± 0.33 on day 16 PI. Following treatment with 7.0mg/kg BW of berenil®, on day 16 PI, the value rose significantly ($p<0.05$), thereby attaining its pre-infection value on day 24 PI or on day 9 PT.

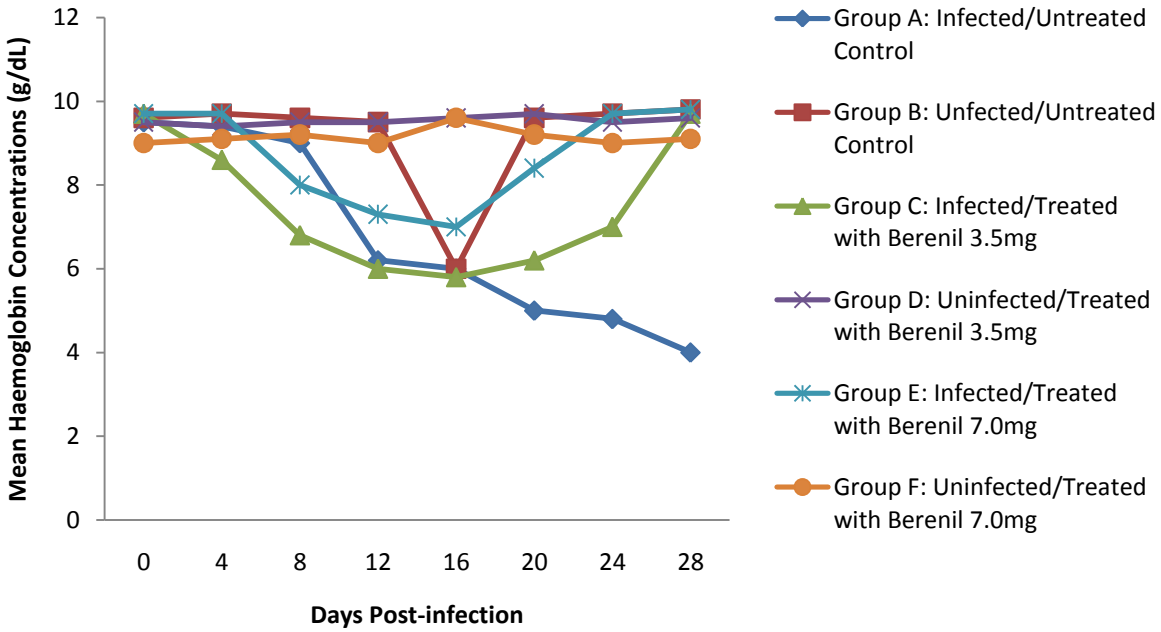

Fig.4. Mean haemoglobin concentrations (g/dl) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

3.5 Total white blood cell count

The mean white blood cell counts (WBC) of the sheep experimentally infected with *T. evansi* and their controls is presented in Figure 5. In group A the mean pre-infection value of 10.0 ± 0.39 experienced significant ($p<0.05$) decline to 7.2 ± 0.34 on day 16 PI. This decline in values continued unabatedly to 5.0 ± 0.28 on day 28 PI. For group B, D, and F, the pre-infection WBC counts of 10.2 ± 0.39, 10.0 ± 0.39, and 10.3± 0.39, respectively, remained fairly constant ($P>0.05$) throughout the study. Meanwhile, in group C, the pre-infection value of 10.3 ± 0.39, declined significantly ($p<0.05$) to 7.0 ± 0.33 on day 16 PI. Following treatment with 3.5mg/kg

BW of berenil®, by day 16 PI, the pre-infection value was attained on day 28 PI (day 13 PT). Similarly, in group E, the pre-infection value of 10.2±0.39 declined significantly ($p<0.05$) to 7.4±0.35 on day 16 PI. Following treatment with 7.0mg/kg BW of berenil®, on day 16 PI, the values began to increase significantly ($P<0.05$), until its pre-infection value was attained on day 24 PI (day 9 PT).

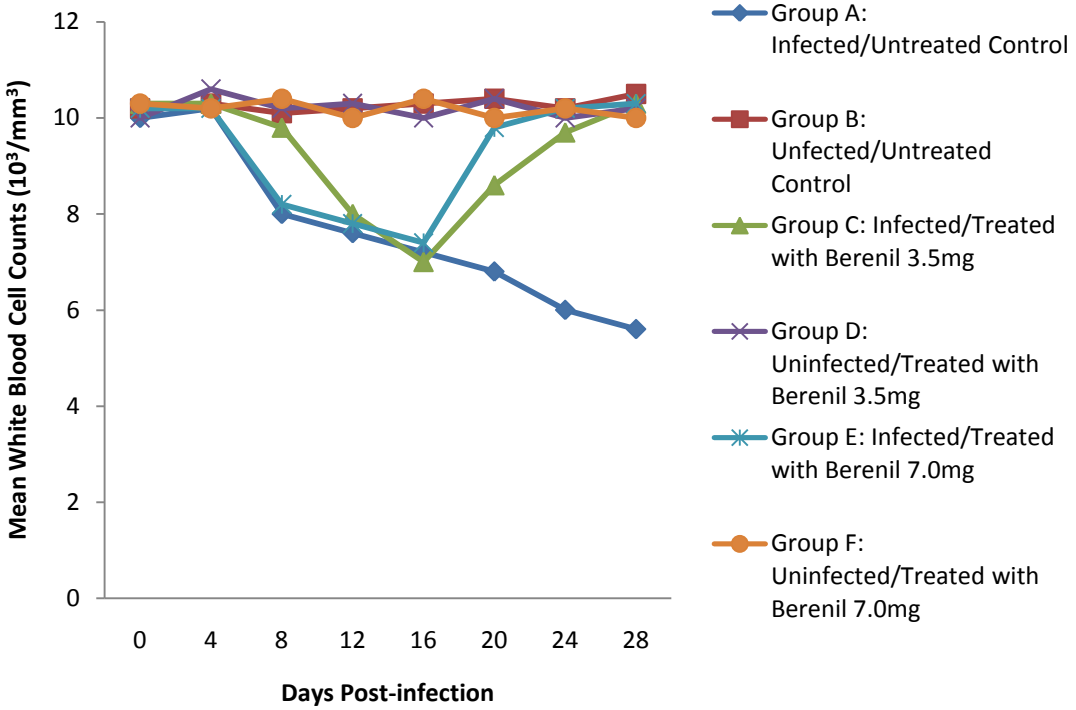

Fig.5. Mean white blood cell counts (10^3/mm^3) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

3.6 Absolute platelet counts

The absolute platelet counts of sheep infected with *T. evansi* and their controls are presented in Figure 6. In group A, the pre-infection value of 151.6 ± 1.54 experienced a significantly ($p<0.05$) decline in value to 60.0 ± 0.97 on day 16 PI. Thereafter, it fluctuated on day 20 PI and continued to decline further, to 45.6±0.84 on day 28 PI. In group B, D, and F, with pre-infection values of 152.6 ± 1.54, 152.9 ± 1.55 and 150.3 ± 1.53 all remained fairly constant respectively ($p>0.05$)

throughout the study. For group C, the pre-infection value of 151.8± 1.54 declined significantly ($p<0.05$) to 70.0± 1.05 on day 16 PI. Following treatment with 3.5mg/kg BW of berenil® on day 16 PI, the pre-infection value was attained on day 28 PI (day 13 PT). Similarly, in group E, the declined in values of 75.0 ± 1.08 on day 16 PI increased significantly ($p<0.05$) following treatment with 7.0mg/kg BW of berenil® on day 16 PI to its pre-infection value on day 24 PI (day 9 PT).

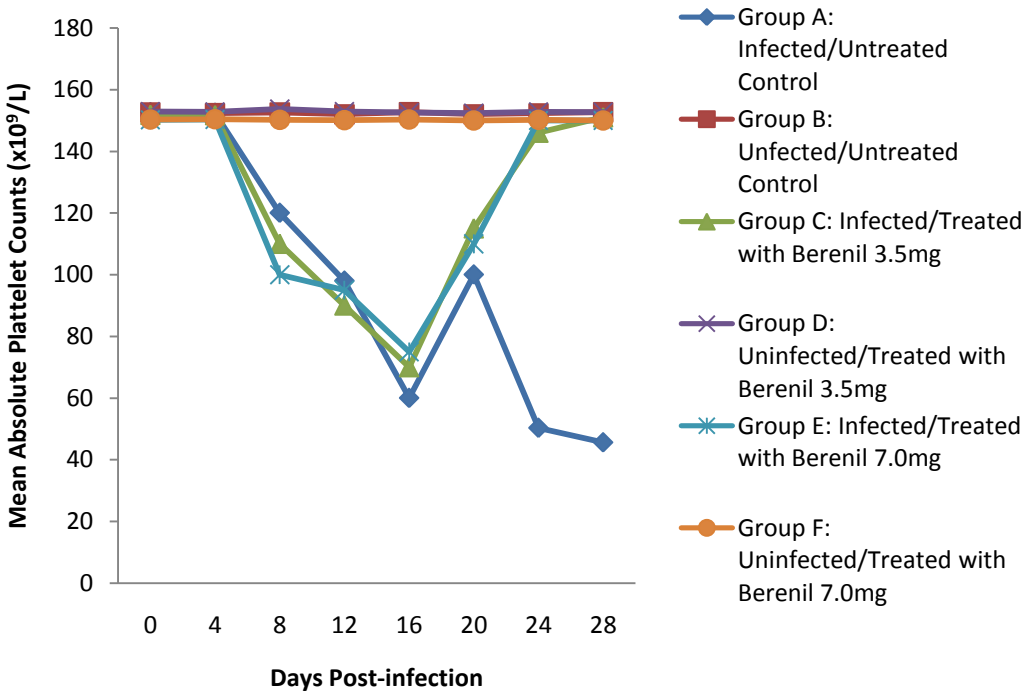

Fig.6. Mean absolute platelet counts ($x10^9$/L) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

3.7 Absolute Neutrophil counts

The absolute neutrophil counts of the sheep experimentally infected with *T. evansi* and their controls are presented in Figure 7. In group A with a pre-infection value of 7.36 ± 0.34 experienced an unabated but significant ($p<0.05$) decline of values to 4.3 ± 0.26 on day 16 PI.

Thereafter, a further decline continued unabated to 3.0 ± 0.22 on day 28 PI. In groups B, D and F, the pre-infection values of 7.80 ± 0.34, 7.80 ± 0.35 and 7.08 ± 0.33 remained fairly constant ($p>0.05$) throughout the period of the experiment respectively. In group C, the pre-infection value of 7.82 ± 0.35 continued to decline ($P<0.05$) without abatement to 4.0 ± 0.25 on day 16 PI. Following treatment with 3.5mg/kg BW of berenil®, on day 16 PI the values increased gradually until the pre-infection value was attained on day 28 PI (day 13 PT). In group E, the pre-infection value of 7.06 ± 0.33 experienced a significant decline ($p<0.05$) to 4.2 ± 0.28 on day 16 PI. Following treatment with 7.0mg/kg BW of berenil® on day 16 PI, the values increased slightly ($p>0.05$) without attaining its pre-infection value on day 24 PI (day 9 PT).

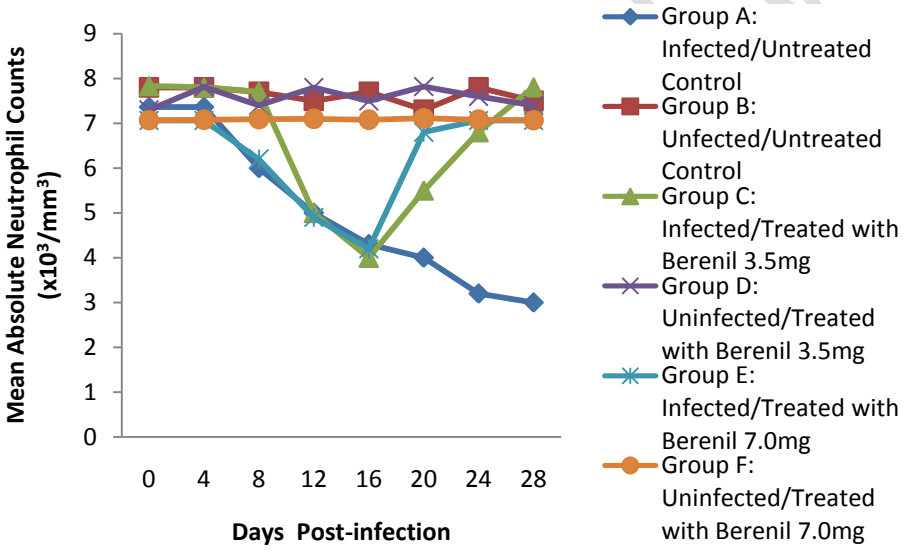

Fig.7. Mean absolute neutrophil counts ($x10^3/mm^3$) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

3.8 Absolute Lymphocyte

The mean absolute lymphocyte changes of Yankasa breed of sheep and their controls are presented in Figure 8. In group A, the pre-infection value of 3.26 ± 0.23 continued to declined significantly ($p<0.05$) without abating to a value as low as 1.0 ± 0.3 on day 28 PI. In group B, D and F, their pre-infection values of 3.45 ± 0.23, 3.04 ± 0.22 and 3.60 ± 0.24 remained fairly

constant (p>0.05) throughout the experiment. In group C, the pre-infection value of 3.27 ± 0.23 declined significantly (p<0.05) to 1.9 ± 0.17 on day 16 PI. Following treatment with 3.5mg/kg BW of berenil® on day 16 PI, the values increased significantly (P<0.05), thereby attaining its pre-infection value on day 28 PI (day 13 PT). In group E the pre-infection value of 3.67 ± 0.24 declined significantly (p<0.05) to 1.6 ± 0.16 on day 16 PI. Following treatment with 7.0mg/kg BW of berenil® on day 16 PI, the pre-infection value was attained on day 24 PI (day 9 PT).

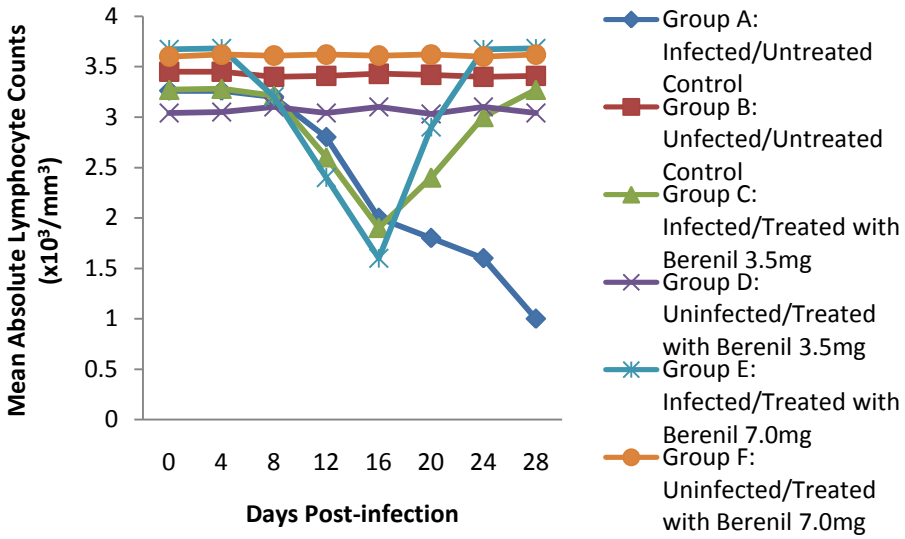

Fig.8. Mean absolute lymphocyte counts ($x10^3/mm^3$) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

3.9 Absolute monocyte counts

The mean absolute monocyte changes of the sheep and their controls are presented in Figure 9. In group A, the pre-infection value of 4.5 ± 0.27 experienced a significant increase (p<0.05), which continued without abating to 47.8 ± 0.86 on day 28 PI. In group B, D and F, their pre-infection values of 4.6 ± 0.27, 4.8 ± 0.27 and 4.0± 0.25 remained fairly constant (p>0.05) throughout the study. In group C, the pre-infection value of 4.7± 0.27, rose significantly (p<0.05) to 20.7 ± 0.57 on day 16 PI. Following treatment with 3.5mg of berenil® on day 16 PI, the values

began to decline significantly (P<0.05) to its pre-infection value on day 28 PI (day 13 PT). In group E, the pre-infection value of 4.5 ± 0.27 rose significantly (p<0.05) to 23.2 ± 0.60 on day 12 PI. Following treatment with 7.0mg/kg BW of berenil® on day 16 PI, the value declined significantly (p<0.05) to its pre-infection value on day 24 PI (day 9 PT).

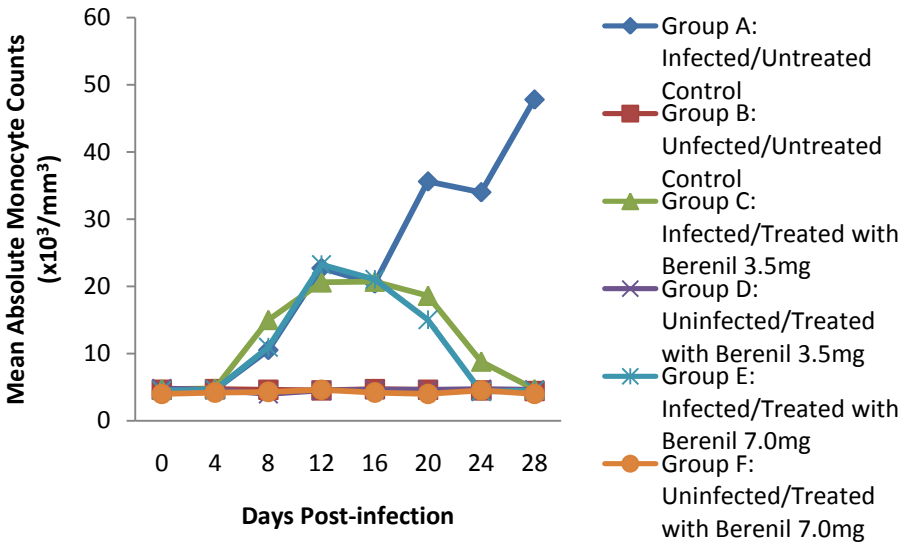

Fig.9. Mean absolute monocyte counts ($x10^3/mm^3$) of Yankasa breed of sheep experimentally infected with *T. evansi* and treated with two different doses of berenil® and their controls.

4. Discussion

This study observed a short pre-patent (PP) of 8 days in the infected animals, which agrees and comparable to 7-11days observed in Yankansa sheep infected with *T. evansi*[24, 25]. However, the result of PP in present study deviates from Wada et al. [26], who observed a much longer pre-patent period of 20 days in Yankasa breed of sheep infected with *T. evansi*. The variations in the pre-patent periods may be attributed to dose of infection, immunity, virulence of the strain of the parasite [27]. Uniform patency and parasitaemia was observed following inoculation of the animals, irrespective of the host susceptibility. Similar observation has been reported in *T. evansi* infected buffaloes, cow, and goats [28, 29], in *T. brucei gambiense* infected baboons (*Papio anubis*) [30]. Fluctuating parasitaemia were also observed generally, among the *T. evansi*infected

sheep.Fluctuations in parasitaemia are known features of trypanosomosis commonly caused by antigenic variation [31]. The ability of the host to limit the peak and number of each wave of parasitaemia is however, dependent on whether the infection is acute, sub-acute or chronic [32], and this may explain the reason why parasitaemia in sheep appreciated and declined following treatment on day 16 PI.

Evaluation of haematological parameters reveal that the infected sheep (group A, C and E) showed a significant decline in red blood cells (RBC), Packed cell volume (PCV), and haemoglobin concentrations (Hb) as the infection progressed. This was indicative of anaemia, which started at the onset of parasitaemia, from day 8 PI. This is in agreement with several reports that the anaemia in trypanosomosis often starts during the 1st wave of parasitaemia and is haemolytic in nature [33]. However, the haemolytic nature of the anaemia in most cases would depend on the species of trypanosomes involved as suggested by Mbaya et al.[34]. Trypanosome infection may cause haemolytic anaemia due to massive erythrophagocytosis by an expanded and active mononuclear phagocytic system (MPS) of the host[35, 36]. The presence of the MPS might have been associated with increased demand on the system to remove dead red blood cells, tissue cells, trypanosomes, antigen-antibody complexes and to participate in immune responses [37]. The fact that the red cell parameters (PVC, RBC, Hb) decreased sharply during wave of parasitaemia, but maintained a gradual increase during the period of low, or no parasitaemia, showed an inverse relationship between parasitaemia and anaemia [38, 39].

Also, the infected sheep exhibited leucopenia which was indicative of immunosuppression commonly encountered in African trypanosomosis [40]. Similar findings were observed in *T. evansi* infection in camels [41]. Lymphopenia was encountered among African giant rats infected with *T. brucei*[42]. This is probably associated with an increased demand on the system for lymphocytes, which is a common requirement in both immune and inflammatory responses in trypanosomosis [43]. Meanwhile, the significant neutropenia encountered among the infected sheep might be associated with splenic sequestration of leucocytes, which often suppressed the raising of neutrophil numbers [44].

The thrombocytopaenia encountered among the sheep is associated with coagulation defects due to low platelet numbers, which have been reported to be associated with disseminated intravascular coagulation in trypanosomosis [45]. This has been reported commonly in acute and chronic *T.brucei* infection of sheep [46] but it does not agree with the reports of Wada et al. [47], which perhaps is the only report of thrombocytosis in cattle trypanosomosis.

The infected sheep showed significant monocytosis. This has been reported as a common feature in trypanosomosis [48]. Monocytosis in trypanosomosis has been associated with a proliferation of tissue macrophages likely due to an increased demand on the system to remove dead red cells, antigen/antibody complexes and to participate in immune responses. Since macrophages are formed from blood monocytes, this increased need for macrophages may have been responsible for the consistent monocytosis encountered in this study. The result suggests that administered drugs improved blood components possibly by depletion of proliferating parasites, and all haematological parameters modulated to their pre-infection status with zero mortality.

CONCLUSION

In conclusion, our finding has provided evidence that the administered drug (berenil®) has the potentials for modulating the state of anaemia, immunosuppressive conditions induced by trypanosome infected sheep in a dose dependent manner.

ETHICAL APPROVAL

Experimental animals were used according to all local laws, guidelines, and policies at University of Maiduguri, Nigeria.

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