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ORIGINAL ARTICLE

CLINICAL AND HEMATO-BIOCHEMICAL PROFILES OF DOGS DIAGNOSED OF NATURAL TRYPANOSOMOSIS AT UNIVERSITY OF NIGERIA VETERINARY TEACHING HOSPITAL, NSUKKA

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ABSTRACT

Hematological and serum biochemical profiles of animals are reflections of physiological disposition to their health status and production potentials. Findings in experimental studies on trypanosomosis may not translate directly to the expected findings in natural infections because of the effect of some variables like species and infecting dose of the parasite, nutritional status of the animal, concurrent infections, and stage of the trypanosomosis at the time of presentation to the clinic. *Trypanosoma brucei* and *T. congolense* infections in dogs have grave prognosis especially if the cases are not detected at their early stages. Clinical presentation, survivability of dogs treated with Diminazene aceturate as well as haematological and serum biochemical aberrations caused by natural infections with different *Trypanosoma* species were investigated with the view to identifying a variable or factor that could be used for prognostic evaluation in the treated dogs. Telephone numbers of the clients for dogs diagnosed with and treated against natural trypanosomes infections were collected and used to monitor the therapeutic outcomes. The clinical signs observed suggestive of late stage trypanosomosis were matched with the revelations from the case history. Significant ($p \leq 0.05$) reductions in PCV, HB, absolute neutrophil, and significant ($p \leq 0.05$) increase in monocytes count, serum urea, and creatinine were recorded in trypanosome infected dogs. Inability of clients to note the early clinical signs of infections with trypanosomes could have contributed to the low survival rate of the trypanosome-infected dogs even when treated.

Keywords: Trypanosome infections, dogs, hematology, biochemical changes, treatment outcome

INTRODUCTION

Hematological and serum biochemical profiles of animals give the general overview of their health status. Findings in experimental studies on canine trypanosomosis may not necessarily translate into the expected observations in natural trypanosome infections since some variables like infecting dose of the parasite, nutritional status of the animal, concurrent infections, stage of the trypanosomosis at the time of presentation to the clinic, among others, may affect the outcome of the clinical manifestations of the disease. Severity of the trypanosomosis is dependent on the strain of parasite species (Bengaly *et al.*, 2002; Liam *et al.*, 2010), the breed of affected dogs (Abenga *et al.*, 2005) and the dose and virulence of the infecting trypanosomes. Diminazene aceturate is the most used drug in treatment of canine trypanosomosis in the University of Nigeria Veterinary Teaching Hospital (UNVTH) and in Nigeria, generally. *Trypanosoma brucei* and *T. congolense* isolated from clinically infected dogs presented at the UNVTH were found to develop moderate to high level of resistance to Diminazene aceturate (Berenil) and isometamidium chloride in mice, respectively (Anene *et al.*, 1999). This may suggest poor prognosis for dogs diagnosed of natural trypanosome infection and treated with either of the two drugs in the area. However, it has been noted that mouse sensitivity tests should only serve as a guide to the parasite sensitivity and not to be used to predict curative doses for the large animals (Sones *et al.*, 1988). Quite often, dogs presented at UNVTH with trypanosomosis are treated with Diminazene aceturate with no deliberate effort made to ascertain the effectiveness of the treatment in relation to survivability of the dogs. Hematological and biochemical profiles may vary depending on factors such as disease condition, breed, sex, age, stress, season, physiological status,

and physical exercise (Kaneko *et al.*, 1997). Hematological and biochemical changes have been observed to be associated with trypanosome infection in animals, and several factors have been found to influence the nature and severity of these changes (Anosa, 1988; Anene *et al.*, 2011; Sivajothi *et al.*, 2015). These factors include the strain of the infecting agent and individual variability of the host in susceptibility to infection. Varying observations in serum biochemistry were reported in trypanosome-infected animals. Anene *et al.* (2011) reported that significant haematological and serum biochemical changes occurred in pigs naturally infected with trypanosomes in Nsukka area. Taiwo *et al.* (2003) reported elevated levels of total protein and globulin and decreases in cholesterol and glucose levels in sheep experimentally infected with *T. b. brucei* and *T. congolense*. Abenga and Anosa (2005) also reported increased serum protein, creatinine, and globulin levels in monkeys experimentally infected with *T. b. gambiense*. There is scarcity of reports on the treatment outcomes in dogs naturally infected with trypanosomes and treated at UNVTH. Also, there is dearth of information on the hematological and serum biochemical changes in dogs attributable to natural infections with trypanosomes. This study was aimed at investigating the clinical presentation, haematological and serum biochemical alterations and survivability of dogs diagnosed of natural trypanosomosis and treated with Diminazene aceturate with the view to identifying the factors or variables suggestive of poor prognosis, post-treatment.

MATERIALS AND METHODS

Study population

Dogs from Nsukka and its environs presented to the University of Nigeria Veterinary Teaching Hospital (UNVTH), Nsukka, between May 2015 and September 2016 and which were based on history and clinical examination findings suspected to be cases of trypanosomosis were used in this study. The Nsukka area where UNVTH is located has two seasons, the wet season that runs from April to October and dry season, which runs from November to March (Ozor *et al.*, 2015). Demographic data, such as age, sex and breed, time of signalment as well as the clinical signs observed during physical examination of the dog patients and treatment outcomes were recorded. The selection criteria used in this study included dogs manifesting clinical signs of trypanosomosis, such as corneal opacity, weakness, anorexia, pyrexia, enlarged superficial lymph nodes. Telephone numbers of the clients were obtained for calls to monitor the treatment outcomes of the dogs. Diminazene aceturate (at 7 mg/kg in the 13 cases out of 21, 5 mg/kg in the 4 cases out of 21, 3.5mg in the 2 cases out of 21, and dosages of 2 out of 21 cases are not recorded and are not known) was used in the treatment of the infected dogs. The clients were called from time to time to evaluate dog's recovery and survivability of treated dogs.

Sample collection

Blood samples, 2 and 4 ml, were collected from each dog patient via the cephalic vein and dispensed into EDTA-coated and non-EDTA-coated tubes and used for haematological and serum biochemical evaluations, respectively EDTA blood samples were examined microscopically for detections and identification of trypanosomes using wet mount technique as

described by Murray *et al.* (1977). Three drops of uncoagulated blood were spotted on FTA® classic card Whatman®, which was air dried and stored in a dry place at room temperature until when needed. Blood and serum were also collected from 8 apparently healthy dogs in the age group of 4 months -2 years from among the dogs presented to UNVTH for general check-up and vaccination, which served as normal group.

Parasites Detection and Identification

The EDTA blood samples were examined microscopically for detections and identification of trypanosomes using wet mount technique as described by Murray *et al.* (1977).

Haematological Evaluations

The EDTA blood samples were used for the estimation of red blood cell count (RBC), packed cell volume (PCV), haemoglobin concentration (HB), total (WBC) and differential white blood cell counts using standard manual methods (Coles 1986; Jain 2002). Erythrocytic indices such as mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), and mean corpuscular haemoglobin concentration (MCHC) were calculated using the values of PCV and RBC, HB and RBC, and HB and PCV, respectively.

Measurement of serum biochemical parameters.

The blood in non-EDTA tube was used to harvest serum that was used to assay for levels of urea, creatinine, total protein, albumin,

globulin, bilirubin, total cholesterol, Alkaline phosphatase, Alanine amino transferase (ALT) and Aspartate amino transferase (AST) using RANDOX® test kits (Randox laboratories LTD, UK), according to the manufacturer's prescriptions.

Data Analysis

Data generated from the study were subjected to appropriate statistics using SPSS statistical package (version 16.0). The haematological and serum biochemical parameters of the trypanosome-infected dogs were compared with those of the apparently healthy dogs (control group) using Student's t- test. Probability (P) values ≤ 0.05 were considered statistically significant.

RESULTS

A total of 21 samples from 20 dogs (Nos. 7 and 11, were from one dog) presented to the UNVTH in the study period were diagnosed to be naturally infected with trypanosomes. The 20 dogs with natural trypanosome infections, made up of 6 males and 12 females, were of different breeds, which include Alsatian, West African Bull Mastiff, Bull Mastiff, Rottweiler, Caucasian, and Mongrels within the age range of 1 and 5 years. The cases were from six local government areas out of 17 LGA in Enugu state; they include Nsukka, Udenue, Igbo-Eze North, Igboeze- south, Isiuzo and Enugu East. The detail is shown in TABLE I below.

TABLE I: Demographic data of sex, age, and breed of the trypanosome-infected dogs, LGA, month and year of presentation and trypanosome species identified at UNVTH from May 2015-September 2016

Patient ID	Gender	Age (Years)	Breed	Contracted CAT from	Month and Year	PCR ID
1	Male	3	Caucasian	Obollo Afor (Udenue LGA)	November 2015	Tb
2	Male	1	West African Bullmastiff-	UNN (Nsukka LGA)	January 2016	Tb
3	Female	3	Caucasian	Mbu (Isi Uzo LGA)	January 2016	Tb
4	Female	2	Mongrel	Ekwueme Estate (Nsukka LGA)	March 2016	
5	Female	4	Rottweiler	Emene (Enugu East LGA)	March 2016	Tb
6	Male	5	Alsatian	UNN (Nsukka LGA)	April 2016	Tb
7	Male	5	Alsatian	Igugu (Udenue LGA)	April 2016	Tb
8	Female	1	Caucasian	Obollor Afor (Udenue LGA)	April 2016	Tb
9	Female	2	Mongrel	Ohom Orba (Udenue LGA)	May 2016	-
10	Female	1	-	-	May 2016	Tb
11	Male	5	Alsatian	-Igugu (Udenue LGA)	May 2016	Tb
12	female	4	Alsatian	-Enugu Road (Nsukka)	May 2016	Tc

13	female	4	Mongrel	Enugu-Ezike (Igbo Eze north)	June 2016	Tb
14	female	4	Caucasian	Obollor-Afor (Udenu LGA)	June 2016	Tb
15	Male	2	Bullmastiff	Nru (Nsukka LGA)	July 2016	Tb
16	Female	5	-	Iheaka (Igbo-Eze South LGA)	August 2016	-
17	Female	3	Rottweiler	Uwelu Owere (Nsukka LGA)	September 2016	Tb
18	Male	1	Rottweiler	Nru (Nsukka LGA)	May,2015	Tb
19	NK	NK	NK	UNN (Nsukka LGA)	July 2015	-
20	NK	NK	NK	NK	NK	Tb
21	Female	2	Alsatian	Uda (Igboeze-North)	September.2015	-

Tb = *Trypanosoma brucei*; Tc= *Trypanosoma congolense*; NK =Not Known; PCR = Polymerase chain reaction; ID = identification; CAT = Canine African Trypanosomosis

OBSERVATIONS FROM CASE HISTORY AND PHYSICAL EXAMINATION OF THE TRYPANOSOME-INFECTED DOGS

The chief complaints from the owners of trypanosome infected dogs were change in the eye colour of their dogs, distension of the abdomen, sudden weakness, anorexia, inappetence, gradual loss of appetite with eventual complete cessation of feeding for two weeks and cloudy eyes. The common clinical signs observed during physical examination of the infected dogs were fever (TABLE II), bilateral and unilateral corneal opacity, bilateral ocular discharges, sunken eyeball, swollen superficial lymph nodes mainly prescapular and popliteal lymph nodes, dullness, diarrhoea, rough hair coat, and purulent ocular discharge.

TABLE II: Physiological parameters, date of presentation and treatment outcome of the trypanosome-infected dogs at UNVTH during the period of May 2015-September 2016.

Patient ID No.	T (°C)	HR	PR	RR	Weight (Kg)	DOP	Outcome of treatment
1	40.4	136	136	76	25.5	24/11/15	Died
2	39.3	ND	ND	ND	45	12/01/16	Alive as at 6 th Sep 2018
3	40.6	156	152	P	25.5	14/01/16	NK
4	39.6	168	84	104	14	01/03/16	Survived but died after 3months
5	40.1	144	116	72	23.5	24/3/16	Dog died after several relapse/re-infection
6	39.4	ND	ND	ND	ND	8/4/16	Alive 4/9/2018
7	39.6	144	140	124	21	9/4/16	Recovered but relapsed
8	40.3	144	108	120	29	11/4/16	Died
9	40.3	112	116		11.7	16/5/16	Recovered but died of other sicknesses
10	39.5	128	72	P	19.5	16/5/16	Died
11	39.9	124	120	48	17	25/5/16	Died
12	41.0	112	108	140	20.5	31/5/16	Died after 80days
13	40.2	ND	ND	ND	29.5	1/6/16	NK
14	39.6	132	ND	ND	21.5	13/6/16	Alive August 2018
15	40.6	128	136	16	32	29/7/16	Died after one months

16	39.5	96	100	44	12.5	15/8/2016	Died
17	38.6	112	132	12	19.7	26/9/2016	Died
18	39.9	140	132	120	20.3	2/5/2015	Recovered
19	40.6	140	132	104	21	9/7/2015	Recovered*
20	NK	NK	NK	NK	NK	NK	NK
21	39.1	140	140	60	13.8	9/11/2015	Was sold when clinical sign reoccurred.

ND = not done; NK =not known; T =Temperature (°C); HR =Heart rate (BPM); PR= Pulse rate (BPM); RR = Respiratory rate (CPM); DOP = Date of presentation; NK= Not Known

HAEMATOLOGICAL PROFILES OF THE TRYPANOSOME-INFECTED DOGS

The means PCV, RBC, HB concentration and absolute neutrophil count in the trypanosome-infected group were significantly ($p \leq 0.05$) lower than those of the apparently healthy group (TABLE III). The mean of absolute monocyte count in the trypanosome-infected group was significantly ($p \leq 0.05$) higher than that of the apparently healthy group. The mean values of MCV, MCH and MCHC in the two groups of dogs were not significant ($p \leq 0.05$). Also, there were no significant ($p \leq 0.05$) differences between the mean absolute eosinophil, basophil, lymphocyte, and total WBC counts of the trypanosome-infected and those of the apparently healthy dogs.

TABLE III: Hemogram of the trypanosome-infected dogs and apparently healthy dogs presented at the UNVTH between May 2015 and September 2016

Parameters/Indices	Trypanosome-infected dogs	Apparently healthy dogs (n=8)
PCV (%)	23.75 ± 2.22 ^b	40.63 ± 3.48 ^a
RBC (X10 ¹² /L)	2.90 ± 0.51 ^b	5.87 ± 0.75 ^a
HB (g/dl)	9.31 ± 0.82 ^b	14.48 ± 1.77 ^a
MCV (fl)	98.45 ± 15.51 ^a	74.79 ± 9.30 ^a
MCH (pg)	38.21 ± 5.42 ^a	26.06 ± 2.66 ^a
MCHC (g/dl)	39.70 ± 2.66 ^a	35.43 ± 2.18 ^a
WBC (x 10 ⁹ /L)	12.59 ± 2.57 ^a	13.37 ± 1.97 ^a
Lymphocyte count (X10 ⁹ /L)	4.35 ± 1.02 ^a	5.68 ± 0.93 ^a
Monocyte count (X10 ⁹ /L)	6.44 ± 1.91 ^b	0.06 ± 0.05 ^a
Basophil count (X X10 ⁹ /L)	0.02 ± 0.16 ^a	0.00 ± 0.00 ^a
Eosinophil count (X10 ⁹ /L)	0.45 ± 0.27 ^a	0.00 ± 0.00 ^a
Neutrophil count (X10 ⁹ /L)	1.30 ± 1.31 ^b	7.56 ± 1.24 ^a

Values with different superscripts horizontally are statistically different ($p \leq 0.05$)

SERUM BIOCHEMICAL PROFILES OF THE TRYPANOSOME-INFECTED DOGS

The mean serum urea and creatinine concentrations, and ALT activity of the trypanosome-infected dogs were significantly ($p \leq 0.05$) higher than those of the apparently healthy group (TABLE IV). There were no significant ($p \leq 0.05$) differences between the mean AST and ALP activities, bilirubin, total protein, albumin, globulin, and cholesterol concentrations in the trypanosome-infected group and those in the apparently healthy group. Though statistically insignificant, means of AST activity of trypanosome infected group was higher when compared to the apparently healthy group.

TABLE IV: Serum biochemical parameters of trypanosome-infected and apparently healthy dogs presented at the UNVTH between May 2015 and September 2016.

Serum biochemical parameters	Trypanosome-infected dogs (n=8)	Apparently healthy dogs (n=8)
Urea (mg/dl)	53.13 ± 13.49 ^b	22.82 ± 5.25 ^a
Creatinine (mg/dl)	1.99 ± 0.48 ^b	0.72 ± 0.08 ^a
AST (IU/L)	80.00 ± 22.86 ^a	32.42 ± 6.15 ^a
ALT (IU/L)	36.88 ± 6.03 ^b	15.34 ± 4.58 ^a
ALP (IU/L)	31.75 ± 4.52 ^a	53.08 ± 11.90 ^a
Total Bilirubin (mg/dl)	0.44 ± 0.09 ^a	0.47 ± 0.05 ^a
Total Protein (g/dl)	5.83 ± 0.37 ^a	6.20 ± 0.74 ^a
Albumin (g/dl)	2.25 ± 0.30 ^a	2.82 ± 0.64 ^a
Globulin (g/dl)	3.56 ± 0.30 ^a	3.38 ± 0.91 ^a
Cholesterol (mg/dl)	1.27 ± 15.20 ^a	1.00 ± 20.43 ^a

Values with different superscripts horizontally are statistically different ($p \leq 0.05$)

DISCUSSION

The treated dogs' cases of trypanosomosis studied came from six Local Government Areas (LGA) of Enugu State: Udenu, Nsukka, Igbo Eze North and Igbo Eze-South were from the Enugu North Senatorial Zone, while two other LGAs, Enugu East and Isi Uzo, are from Enugu East senatorial zone. Nsukka local government had the highest proportion (42.11%) and was followed by Udenu LGA with 31.58% of canine trypanosomosis cases within the study period. One of the possible reasons to this could be the relative nearness of these two

LGAs to the UNVTH, even though canine trypanosomosis was reported to be very common in the Nsukka Agricultural zone of Southeastern Nigeria (Onamegbe *et al.*, 1984; Anene and Onamegbe, 1984; Obidike *et al.*, 2005; Umeakuana *et al.*, 2016). Higher number of cases of canine trypanosomosis was recorded in the wet season (75%) than in the dry season (25%). Although the study period had more cases of canine trypanosomosis in wet season than dry season, however the highest frequency of the cases was recorded in June 2016. Such seasonal variations with a higher prevalence of

canine trypanosomosis during wet than dry months of the year had earlier been reported (Omamegbe *et al.*, 1984) and in cattle (Majekodumi *et al.*, 2013). This could be attributed to the favorable condition offered by the wet season to the vectors especially tsetse fly to reproduce and multiply (Majekodumi *et al.*, 2013). Female dogs were more infected with trypanosomes in the study area than the male dogs. A possible explanation could be that these dogs were used for breeding and breeders generally prefer having female dogs to male dogs as we observed during history taking. Most of the infected dogs were exotic breeds (Caucasian, Alsatian, West African bull Mastiff, Rottweiler, Alsatian, and Bull mastiff) while the Nigerian local breed of dog had list number of infected with the trypanosomes. This may underscore the superiority of the local dog's resistance to trypanosome infection over that of the exotic breeds or it could be because more attention is usually paid to the health of exotic breeds than the local breeds of dogs for economic reasons which may form the reason the local dogs are not presented to clinics for testing and treatment. Infections with *Trypanosoma brucei* and *Trypanosoma congolense* were responsible for most canine African trypanosomosis in the study area, given the proportions recorded in this study. Enlarged superficial lymph nodes, corneal opacities and pyrexia were common clinical signs in most of the dogs infected with the *T. brucei* group. Although enlarged superficial lymph nodes and pyrexia were also observed in the *Trypanosoma congolense*-infected dog, there was no corneal opacity. This association between corneal opacity and infection with *Trypanosoma brucei* subspecies in dogs has been reported earlier (Lisulo *et al.*, 2014). The corneal opacity observed in the infections with *T. brucei* subspecies of dogs could be due to invasive nature of this parasite and therefore not seen in the *T. congolense*-infected dogs because the parasite is restricted to the

circulatory system. Corneal opacity may be an indication of chronic /late-stage canine trypanosomosis, which is associated with trypanosome invasion of central nervous system and its associated treatment failure due to reported inability of trypanocide to cross blood brain barrier to clear the parasite (Chukwu *et al.*, 1990). Relapses were noticed in some of the *T. brucei* infected- and treated dogs and most of the treated dogs still died. Development of resistance to trypanocides and the ability of *T. brucei* to survive in the cryptic foci inaccessible or poorly accessible to drugs are some of the factors reported to be responsible for the poor response to chemotherapy and chemoprophylaxis (Chukwu *et al.*, 1990; Anene *et al.*, 1999; Anene *et al.*, 2006; Ezech *et al.*, 2009). Parasites from cryptic foci, usually the brain (Chukwu *et al.*, 1990), continue to serve as infection source to the dog once the trypanocide in the systemic circulation wanes and this cycle may continue till the dog dies of multiple organ and tissue damages. The finding of *T. brucei* subspecies and *T. congolense* as the main cause of canine trypanosomosis in this study agrees with the earlier reports by Omamegbe *et al.* (1984) and Umeakuana *et al.* (2016), who also reported that infections with *T. brucei*, majorly, and *T. congolense* were responsible for most morbidities and mortalities in dogs within the study area. It is noteworthy that these earlier studies were based on microscopy and morphological characterization of the trypanosomes. *T. brucei* and *T. congolense* infections in dogs carry a grave prognosis, especially if the cases are not detected at their early stages (Jennings *et al.*, 1977a; Morrison *et al.*, 1981; Greene 2006). However, two of the three cases that survived and had no relapse infection during a period of one year after treatment could be attributed to early detection

of the parasites and the subsequent chemotherapy before the trypanosomes migrate to other tissues especially the brain. It has been reported that treatment of experimental *T. brucei* infection in animals became less effective as the interval between infection and treatment increased (Jennings *et al.*, 1977a), and a possible explanation for this phenomenon was the presence of trypanosomes in drug inaccessible sites like the brain (Jennings *et al.*, 1979). Clients in trypanosome infections-endemic areas, like our areas of study, should be educated on the need to present their dogs to veterinary hospitals/clinics for early diagnosis and management of the cases once they observe any change in the behavior, especially inappetence or anorexia. In this study, it was evident that dog owners neglected the early warning signs (anorexia and inappetence) as revealed in their primary complaints, which could have contributed to poor treatment outcomes and high mortalities recorded in this study. Trypanosomal resistance to the trypanocide (diminazene aceturate) used could be another reason for the observed relapse infections in some of the infected dogs in this study. Since trypanosomosis is endemic in areas sampled, there is need to adopt chemoprophylaxis for every uninfected dog and curative chemotherapy for early diagnosed cases of canine trypanosomosis as these approaches could help in reducing the trypanosome-infection related morbidities and mortalities. The alterations in erythrocytic parameters (low PCV, RBC count and HB concentrations) recorded in dogs with trypanosomosis were indicative of anaemia, a consistent clinical sign of trypanosomosis. The erythrocytic alterations are also a common finding in acute trypanosome infections of animals (Anosa, 1988; Ihedioha and Chineme, 2004). The high MCV values recorded in the trypanosome-infected dogs shows the anaemia was macrocytic, which is an indication of good bone marrow response to the

deficit in circulating erythrocyte mass. It signifies the presence of immature red blood cells (reticulocytes) in peripheral circulation (Jain, 2002; Stockham & Scott, 2008). In this study, total bilirubin is within range for both apparently healthy and trypanosome infected group. This may be because in small animals, a healthy liver has the capability of conjugating large amounts of bilirubin hence haemolysis may in most cases be accompanied by normal bilirubin levels, or that the rate of haemolysis has not exceeded the rate of conjugation by hepatocytes (Stockham and Scott, 2008). Monocytosis seen in the trypanosome infected group may be due to a need for increased phagocytic function (Stockham and Scott, 2008). In trypanosomosis, monocytes transform into macrophage and function in phagocytosis of trypanosomes and lysed RBCs (Ihedioha and Chineme, 2004). The serum biochemical alterations seen in the trypanosome-infected group include azotemia (high urea and creatinine levels). This may be prerenal following a reduction in its glomerular filtration rate due to dehydration or fever or renal dysfunction (Abenga and Anosa, 2005; Gunaseelam *et al.*, 2009; Latimer, 2012; Sivajothi and Sudhakara Reddy, 2017; Bakari *et al.*, 2017). Elevation of serum creatinine has been associated with damage to host tissues as well as renal and hepatic malfunction (Abenga and Anosa, 2005). The causes of elevated BUN levels include kidney disease such as glomerulonephritis, excessive protein catabolism and febrile conditions. Nwoha *et al.*, (2013) reported initial increase in BUN in trypanosome-infected dogs, which later declined during the disease. However, similar fluctuation has been reported by other researchers (Jerry and Victor 2007). Elevation of BUN has also been reported in man (Anosa

1988; Awobode, 2006). Fever and glomerulonephritis are common features of trypanosomosis and presumably act together to elevate BUN (Poltera, 1985). The elevation in serum ALT activity seen in the trypanosome-infected group may be due to an alteration in hepatocellular membrane permeability resulting from tissue anoxia/circulatory hypoxia (due to anaemia) or due to hepatic dysfunction) (Gunaseelam *et al.*, 2009; Latimer, 2012; Sivajothi & Sudhakara Reddy, 2017). Decreasing albumin levels and elevation of globulin level due to hyper gamma globulinaemia have been reported in trypanosomosis (Anosa, 1988; Omeje and Anene, 2012). Wassel (2000) reported increase in plasma globulin and attributed it to tissue destruction by *Trypanosoma brucei*. Globulin increase has been reported in trypanosome infected sheep (Taiwo *et al.*, 2003) and monkey infected with *T.b. gambiense* (Abenga and Anosa, 2004). Hypoproteinaemia in mice infected with *Trypanosoma brucei* due to decrease in hepatic biosynthesis and progressive loss of albumin in urine has been reported (Agu and Egbuji, 2002). Hypoproteinaemia has also been reported in dog (Nwoha *et al.*, 2013), pig (Otesile *et al.*, 1991), goats (Witola and Lovelace, 1997) sheep (Taiwo *et al.*, 2003), rabbit (Orhue *et al.*, 2005; Sivajothi *et al.*, 2015), cattle (Sadique *et al.*, 2001) and rat (Umar *et al.*, 2009). However, increase in blood total protein has also been reported in trypanosome-infected rabbit (Takeet and Fagbemi, 2009), monkeys (Abenga and Anosa, 2004), Sheep (Taiwo *et al.*, 2003). Surprisingly, no significant change was observed in *T. brucei* infected pigs (Anene *et al.*, 2011), velvet monkey infected with *T.b. rhodesiense* (Ngure *et al.*, 2008) and human being naturally infected with *T. b. gambiense* (Awobode, 2006). Increase in blood protein in trypanosomosis has been linked to increase in gamma globulins produced as part of host immune response (Sow *et al.*, 2014; Sivajothi *et al.*, 2015).

However, decrease in total protein was also associated with acute infection where feed intake is reduced due to illness (Sow *et al.*, 2014). Increase in blood cholesterol has been reported in goats (Adejinmi and Akinboade, 2000). Hypercholesterolemia has been associated with hepatic malfunctioning resulting from impairment of liver lipid metabolism in African trypanosomosis (Adejinmi and Akinboade, 2000), rabbit (Arowolo *et al.*, 1988). However, contrasting result (hypochloresteraemia) was reported in cattle experimentally infected with *T. brucei rhodesiense* (Wellde *et al.*, 1989), pigs (Adamu *et al.*, 2009; Omeje and Anene, 2012), sheep (Adamu *et al.*, 2008) and in goats infected with *T. brucei* (Biryomumaishe *et al.*, 2003). Trypanosomes require lipoproteins to multiply in axenic culture. Thus, the lowering of cholesterol could be partly due to utilization of the cholesterol by the trypanosomes since the blood stream forms are unable to synthesize cholesterol but requires it along with phospholipids for synthesis of their membranes and growth (Nok *et al.*, 2003). However, there were no significant change in serum protein, globulin, albumin and cholesterol of trypanosome infected dogs and apparently healthy dogs.

In summary, the finding that *Trypanosoma brucei* was the major cause of canine trypanosomosis in the dogs presented at the UNVTH and Nsukka area corroborated with results of earlier reports. The clinical symptoms suggested late stage trypanosomosis and negligence on the part of the dog owners to act on the early warning signs of the disease.

Dog owners should be educated on the need to present their dogs for medical attention once change in feeding is observed in their dogs as this is one of the early warning signs of the

sickness so that treatment outcome will be improved since the time interval between infection and treatment inversely affects the treatment outcome.

REFERENCES

- ABENGA, J. N. AND ANOSA V.O. (2005). Serum total proteins and creatinine levels in experimental Gambian trypanosomosis of vervet monkeys. *African Journal of Biotechnology*. 4 (2):187-190
- ABENGA, J. N., AND ANOSA, V. O. (2004). Serum biochemical changes in experimental Gambian trypanosomiasis. I: Enzymes and electrolytes. *Journal of Protozoology Research* 14: 32-42
- ABENGA, J.N., DAVID, K., EZEBUIRO, C.O.G., LAWANI, F.A.G. (2005). Observations on the tolerance of young dogs (puppies) to infection with *Trypanosoma congolense*. *African Journal of clinical and experimental microbiology*. 6:28–33.
- ADAMU, S., BARDE, N., ABENGA, J. N., USEH, N. M., IBRAHIM, N. D. G., ESIEVO, K. A. N. (2009). Experimental *Trypanosoma brucei* infection-induced changes in the serum profiles of lipids and cholesterol and the clinical implications in pigs. *J Cell Animal Biol*. 3(2):15–20.
- ADAMU, S., IGE, A. A., JATAU, I. D., NEILS, J. S., USEH, N.M., BISALLA, M., IBRAHIM, N.D.G., NOK, A. J., ESIEVO, K. A. N. (2008) Changes in the serum profiles of lipids and cholesterol in sheep experimental model of acute African trypanosomosis. *AfrJ Biotechnol*. 7(12):2090–2098
- ADEJINMI, J. O. AND AKINBOADE, O. A. (2000). Serum biochemical changes in WAD goats with experimental mixed *Trypanosoma brucei* and *Cowdria ruminantium* infection. *Trop. Vet*. 18: 111-120.
- AGU, W. E., AND EGBUJI, A. N. (2002). Urine albumin Levels in mice infected with *Trypanosma brucei*. *Veterinaski Archiv* 72(2): 101-108.
- ANENE, B. M. AND OMAMEGBE, J. O. (1984). Abortion Associated with *Trypanosomabrucei* Infection in an Alsatian Bitch. *Tropical veterinarian* 2:211-213.
- ANENE, B. M., CHUKWU, C. C. AND ANIKA, S. M. (1999). Sensitivity to diminazene aceturate and isometamidium chloride of trypanosomes isolated from dogs in Nsukka area, Nigeria. *Revue d'Elevage et de Medecine Veterinaire des Pays Tropicaux* 52, 129–131.
- ANENE, B.M, IFEBIGH, A.O., IGWILO I.A., UMEAKUANA., P.U. (2011). Prevalence and haemato-biochemical parameters of trypanosome-infected pigs at Nsukka, Nigeria. *Comp Clin Path*.20:15–8.
- ANENE, B., EZEOKONKWO, R., MMESIRIONYE, T., TETTEY, J., BROCK, J., BARRETT, M. AND DE KONING, H. (2006). A diminazene-resistant strain of *Trypanosoma brucei*

brucei isolated from a dog is cross-resistant to pentamidine in experimentally infected albino rats. *Parasitology*, 132:(1), 127-133. doi: 10.1017/S0031182005008760

- ANOSA, V.O. (1988). Haematological and biochemical changes in human and animal trypanosomiasis part II. *Revue d'Elev. Med. Vet. Trop.*, 41: 151-164
Trypanosoma brucei infection in dogs. *Veterinary Parasitology*, 151: 139 – 149.
- AROWOLO, R. O. A., ELHASSAN, E. O. AND AMURE, B. O. (1988). Assessing hepatic dysfunction in rabbits experimentally infected with *Trypanosoma brucei*. *Rev. Elev. Med. Vet. Pays Trop.* 41: 277-281.
- AWOBODE, H. O. (2006). The biochemical changes induced by natural human African trypanosome infections. *African Journal of Biotechnology* 5(9): 738-742.
- BAKARI, S. M., OFORI, J. A., KUSI, K. A., AMING, G. K., AWADARE, G. A., CARRINGTON, M. & GWIRA, T. M. (2017). Serum Biochemical Parameters and Cytokine Profiles Associated with Natural African Trypanosome Infections in Cattle. *BiomedCentral*. 10: 312.
- BENGALY, Z., SIDIBE, I., BOLY, H., SAWADOGO, L. AND DESQUESNES, M. (2002A). Comparative pathogenicity of three genetically distinct *Trypanosoma congolense*-types in inbred Balb/c mice. *Veterinary Parasitology* 105, 111–118
- BIRYOMUMAISHO, S., KATUNGUKA-RWAKISHAYA, E., RUBAIRE-AKIIKI, C. M. (2003). Serum biochemical changes in experimental *Trypanosoma brucei* infection in small East African goats. *Vet. Archiv.* 73(3): 167-180.
- CHUKWU, C. C., ANENE, B. M., ONUKOWUSI, K.O. AND ANIKA, S.M. (1990). Relapse infection after chemotherapy in dogs experimentally infected with *Trypanosoma brucei*. *Journal of Small Animal Practice* 31: 141-144
- COLES, E. H. (1986). *Veterinary Clinical Pathology*, 3rd ed. W.B. Saunders Company, Philadelphia, pp: 145 – 151.
- EZEH, I.O., AGBO, L.I., EMEHELU, C.O., NWEZE, E.N., EZEOKONKWO, R.C. AND ONAH, D.N. (2009). Berenil-resistant *Trypanosoma brucei brucei* infection in hunting dog in Nsukka area, Enugu state, Nigeria. *Nigerian veterinary journal*, 29:34-42
- GREENE, C.E. (2006). *Infectious diseases of the dog and cat*. 3rd ed. St Louis: Elsevier.
- GUNASEELAN, L., SENTHIL KUMAR, K., SELVARAJ, P. & KATHIRESAN, D. (2009). Haemato Biochemical Changes in a Case of Canine Trypanosomosis. *Tamilnadu Journal of Veterinary & Animal Sciences*. 5 (3): 122-1213.
- IHEDIOHA, J. I., AND CHINEME, C.N. (2004). Hematopoietic System. In: *Fundamentals of Systematic Veterinary Pathology* Vol. 1 Great AP Express Publishers Limited, Nigeria, and pp 107-160.

- JAIN, N. C. (2002). Schlam's Veterinary Haematology. *Lippincott Williams & Wilkins*. Philadelphia. Baltimore. New York, London. Buenos Aires. Hong Kong. Sydney. Tokyo.
- JENNINGS, F. W., WHITELOW, D. D. AND URQUHART, G. M. (1977A). The relationship between duration of infection with *Trypanosoma brucei* in mice and the efficacy of chemotherapy. *Parasitology* 75, 143–153.
- JENNINGS, F. W., WHITELOW, D. D., HOLMES, P. H., CHIZYUKA, H. G. B. AND URQUHART, G. M. (1979). The brain as a source of relapsing *T. brucei* infection in mice after chemotherapy. *International Journal for Parasitology* 9, 381–384.
- JERRY, N.A., VICTOR, O.A. (2007). Serum biochemical changes in experimental *Gambian* Trypanosomiasis. II. Assessing hepatic and adrenal dysfunction. *Turk. J. Vet. Anim. Sci.* 31 (5):293-296 (TUBITAK)
- KANEKO, J. J., J. W. HARVEY, M. L. BRUSS (1997): *Clinical Biochemistry of Domestic Animals*,
- LATIMER, K. S. (2012). *Duncan and Prasse's Veterinary Laboratory Medicine: Clinical Pathology*. 5th ed. *John Wiley and Sons*. Hoboken.
- LIAM, J.M., SARAH, M., LINDSAY, S., CHI, N.C., ANNETTE, M., ANDY, T., TURNER, C. M. R. (2010). Role for parasite genetic diversity in differential host responses to *Trypanosoma brucei*. *Infection and immunity*. 78 (3): 1096-1108.
- LISULO, M, SUGIMOTO, C., KAJINO, K., HAYASHIDA, K., MUDENDA, M., MONGA, L., NDEBE. J, NZALA, S, NAMANGALA, B. (2014). Determination of prevalence of African trypanosome species in indigenous dogs of Mambwe district, eastern Zambia, by loop mediated isothermal amplification. *Parasites & Vectors*, 7: 19
- MAJEKODUNMI, A.O., AKINYEMI, F., CHARLES, D., PICOZZI K., THRUSFIELD, M.V., WELBURN, S.C. (2013). A Longitudinal survey of African animal trypanosomiasis in domestic cattle on the Jos Plateau, Nigeria. Prevalence, distribution, and risk factors. *Parasite Vectors*, 6:239
- MORRISON, W.I., MURRAY, M., SAYER, P.D., PRESTONE, J.M. (1981). The pathogenesis of experimentally induced *T. brucei* infection in dog: Tissue and organ damage. *Am. J. Pathol.* 102, 168–181.
- MURRAY, M., MURRAY, P.K., MCINTYRE, W.I.M. (1977). An improved parasitological technique for the diagnosis of African trypanosomiasis. *Trans. R. Soc. Trop. Med. Hyg.* 71, 325–326.
- NGURE, R. M., NDUNGU, J. M., NGOTHO, J. M., NANCY, M. K., MAATHAI, R. G., AND GATERI, L. M. (2008). Biochemical changes in the plasma of vervet monkeys (*Chlorocebus aethiops*) experimentally infected with

- Trypanosoma brucei rhodesiense*. *Journal of Cell Animal Biology* 2: 150-7.
- NOK, A. J., NOCK, A. H., BONIRE, J. J. (2003). The cholesterol pathway of *Trypanosoma congolense* could be a target for triphenylsiliconsalicylate inhibition. *Appl. Organomet. Chem.* 17: 17-22.
- NWOHA, R. I. O., EZE, I. O. AND ANENE, B. M. (2013). Serum biochemical and liver enzymes changes in dogs with single and conjunct experimental infections of *T. brucei* and *Ancylostoma caninum*. *African Journal of Biotechnology*, 12(6): 618-624. 2.
- OBIDIKE, R. I., SHOYINKA, S. V. O., AKPEDE, T. O. (2005). Haematology and Urinalysis of *Trypanosoma brucei* infected dogs in Trypanosomosis endemic area. *Nigerian Journal of Experimental and Applied Biology*. 6, 49 -54
- OMAMEGBE, J. O., ORAJAKA, L. J. E., AND EMEHELU, C. O. (1984). The incidence and clinical forms of naturally occurring canine trypanosomiasis in two Veterinary clinics in Anambra State of Nigeria. *Bulletin of Animal Health and Production in Africa* 32, 23–29.
- OMEJE, J. N., ANENE, B. M. (2012). Comparative serum biochemical changes induced by experimental infection of *T. brucei* and *T. congolense* in pigs. *Veterinary Parasitology*. 2012 Dec; 190(3-4):368-374. DOI: 10.1016
- ORHUE, N. E. J., NWANZE, E.A.C., OKAFOR, A. (2005). Serum total protein, albumin, and globulin levels in *Trypanosoma brucei* infected rabbits: effect of orally administered *Scoparia dulcis*. *Afr. J. Biotechnol.* 4(10):1152-1155.
- OTESILE, E. B., AKPAVIE, S .O., EGBEMI, B. O., OGUNREMI, A. O. (1991). Pathogenicity of *Trypanosoma brucei brucei* in experimentally infected pigs. *Revue d' Elevage et de Medecine Veterinaire des Pays Tropicaux* 44, 279-282.
- OZOR, N., OZIOKO, R., ACHEAMPONG, E. (2015). Rural-Urban Interdependence in Food Systems in Nsukka Local Government Area of Enugu State, Nigeria. *J. Agr. Ext.*, 19(2): 157-183.
- POLTERA, A. A. (1985). Pathology of human African trypanosomiasis with reference to experimental African trypanosomiasis and infection of the Central Nervous System. *Br. Med. Bull.* 41: 169-174.
- SADIQUE, N.A., ADEJIMI, J. O., ARIRI, H. (2001). Haematological and plasma protein values of Zebu cattle in trypanosome –endemic zone. *Tropical Animal Production Investment* 4:219-223.
- SIVAJOTHI, S. & SUDHAKARA REDDY, B. (2017). Therapeutic Management of Anaemia Due to Trypanosomosis in Dogs. *International Clinical Pathology Journal*. 5 (3): 244-256.
- SIVAJOTHI, S., RAYULU, V. C., & SUDHAKARA REDDY, B. (2015). Haematological and biochemical

- changes in experimental *Trypanosoma evansi* infection in rabbits. *Journal of parasitic diseases: official organ of the Indian Society for Parasitology*, 39(2), 216–220.
- SONES, K.R., NJOGU, A.R., HOLMES, P.H., (1988). Assessment of sensitivity of *Trypanosoma congolense* to isometamidium chloride: a comparison of tests using cattle and mice. *Acta trop.*, 45: 153-164
- SOW, A., ZABRÉ, M., MOUICHE, M., KOUAMO, J., KALANDI, M., BATHILY, A., *ET AL.* (2014) Investigation of biochemical parameters in Burkinabese local small ruminants breed naturally infected with trypanosomosis. *Int J Biochem Res Rev.* 4:666–79.
- STOCKHAM S.L., AND SCOTT M.A. (2008). *Fundamentals of Veterinary Clinical Pathology*, 2nd ed. Blackwell Publishing, Iowa, U.S.A.
- TAIWO, V.O., OLANIYI, M.O. AND OGUNSANMI, A.O. (2003) Comparative Plasma Biochemical Changes and Susceptibility of Erythrocytes to in Vitro Peroxidation during Experimental *Trypanosoma congolense* and *T. brucei* Infections in Sheep. *Israel Journal of Veterinary Medicine*, 58, 112-117.
- TAKEET, M. I. AND FAGBEMI, B. O. (2009). Haematological, pathological and plasma biochemical changes in rabbits experimentally infected with *Trypanosoma congolense*. *Science World Journal* 4, 29-36.
- UMAR, I. A., MAVYOMS, N. G., DAIKWO, E., ABUBAKAR, G., BURATAI, L. B., IGBOKWE, I. O., AND IBRAHIM, M. A. (2009). The effect of aqueous extract *hibiscus sadariffa* (sorrel) calyces on haematological profile and organ pathological changes in *Trypanosoma congolense*-infected rats. *African Journal of Traditional Complementary and Alternative Medicine* 6(4): 585-591.
- UMEAKUANA, P.U., MOHAMMED, B.R., ANENE, B.M. (2016). Canine trypanosomosis in the University of Nigeria Veterinary Teaching Hospital (UNVTH), Enugu state, Nigeria, sub – Saharan Africa. *Journal of veterinary advances.* 2016; 6(11):1350-1356.
- WASSELL, J. (2000). Haptoglobin: function and polymorphism. *Clinical Laboratory.* 46(11–12):547–552.
- WELLDE, B. T., PRESTON, J. M., KOVATCH, R. M., HIGGS, J., AND CHUMO, D. A. (1989). *Trypanosoma congolense*: Erythrocytic indices, Plasma iron turn-out and effects of treatment in infected cattle. *Annals Tropical Medical Parasitology* 83(1): 201-206.
- WITOLA, W. H., LOVELACE, C. E.A. (1997). Serum protein in indigenous Zambian goats with trypanosomosis. Meeting abstract FASEP Journal 11:9.