

# Case Report

## HAEMATOLOGY AND SERUM BIOCHEMICAL ALTERATION IN STRESS INDUCED EQUINE THEILERIOSIS. A CASE REPORT

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### INTRODUCTION

Equine theileriosis is a tick borne haemoprotozoan disease caused by *Theileria equi*, a small piroplasm that is very pathogenic in horses. It is transmitted by various genera of ixodid ticks which include *Dermacentor* spp, *Hyalomma* spp and *Rhipicephalus* spp (Urquhart *et al.*, 1996). The disease is endemic in tropical and subtropical areas (Hailat *et al.*, 1997). It has been reported in Nigeria (Aliu, 1983), South and Central America (Barbosa *et al.*, 1995), some countries in the Middle East (Friedhoff, 1982) and Australia (Mahoney *et al.*, 1977).

Equine theileriosis occur in acute, sub acute and chronic form and hence the clinical manifestation is variable and could manifest as fever, anaemia, jaundice and haemoglobinuria in acute form (Urquhart *et al.*, 1996) and sometimes in chronic infection icterus and oedema of the ventral abdomen may be seen (Blood & Radostits, 1987). Carriers of the infection are usually asymptomatic and are responsible for the propagation and maintenance of the infection.

Identification of the parasites in the blood smears is the diagnostic mainstay but this bears certain limitations particularly when parasitaemia is low (Krause *et al.*, 1996). Serodiagnosis may give false positive test results in treated horses and false negative results especially in carriers as antibodies could be detected even four years after the parasites had been eliminated from the blood (Bruning, 1996). Polymerase chain reaction (PCR) proved very useful for the detection of haemoparasites (Caccio *et al.*, 2000) and combined with reverse line blot (RLB) offer possibility of simultaneous detection and identification of different species infecting horses (Nagore *et al.*, 2004). Imidocarb treatment of *Theileria equi* infected horses has been shown to clear the parasites from the circulation but the infected horses always remain carrier (Bruning, 1996).

Literature on equine theileriosis in Nigeria is scanty, but worse is the information on the associated effects of this disease condition on haematological, serum biochemical and serum chemistry of

infected horses. Presented in this case report are the haematological picture, serum biochemical and serum chemistry of horses naturally infected with *Theileria equi*.

### CASE PRESENTATION

Two locally bred stallions 14 and 18 years old weighing 270 kg and 250 kg respectively were presented to the Veterinary Teaching Hospital, University of Agriculture, Abeokuta, Nigeria with history of anorexia, dullness and polydipsia few days after polo tournament. Physical examination revealed pale ocular membrane, unilateral sub-mandibular lymphadenitis, mild dehydration, few ticks from the perineum area and copious ocular discharges. The rectal temperature at presentation were 41.2 °C and 40.9 °C, heart rates of 170m<sup>-1</sup> and 164m<sup>-1</sup> and respiratory rates of 11m<sup>-1</sup> and 13m<sup>-1</sup> respectively.

**Blood and Faecal examination:** Blood samples were collected by jugular venipuncture at presentation and one month following the last treatment. The blood samples were placed in two 10-ml tubes. One tube containing Ethylenediaminetetraacetic acid (EDTA) at 1 mg ml<sup>-1</sup> concentration as anticoagulant; the second tube contained no anti-coagulant. For examination of blood parasites, thin and thick blood smears were prepared at the time of sampling and stained with Giemsa stain. Sera were separated from the second tube and stored at -20 °C until required for examination.

Total red blood cells (RBC) and white blood cells (WBC) counts were made by a haemocytometer while packed cell volume (PCV) were determined using microhaematocrit method; the haemoglobin concentration (Hb) were spectrophotometrically determined (Cole, 1986). Mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) were calculated while complete serum biochemistry was carried out.

The faecal samples were screened using simple floatation and sedimentation methods while Mc-master egg counting technique was used to determine the helminths load of the horses.

The ticks were identified using the key described by Okello-onen, *et al.*, (1999).

**Treatment:** Each horse received three treatments of imidocarb (Imidox<sup>R</sup>, Pfizer, Nigeria) at 72 hrs intervals in a dose rate of 4 mg kg<sup>-1</sup> body weight (BW) and repeated one month later in a single

dose of 4 mg kg<sup>-1</sup> body weight. Glucose and methionine administered by intravenous infusion at 150 mg kg<sup>-1</sup> and 20 mg kg<sup>-1</sup> respectively.

**Clinical and haematological findings:** Clinical examination revealed that the two horses suffered from fever, anorexia, weakness and icterus. Examination of the stained blood smears from the two horses revealed few parasitized red blood cells with *T. equi*. Both horses had mild anaemia that was macrocytic normochromic in nature. One of the horses had normal value (WBC) while the other had non-significant ( $p>0.05$ ) decrease in the value of (WBC) but both with significant ( $p<0.05$ ) decrease in the level of lymphocytes. All the haematological parameters except the lymphocytes of the horse 2 returned to normal range after one

month and no intra-erythrocytic parasite was found in their blood smears (Table 1).

**Serum chemistry profile:** There were increases above normal ranges in serum urea, globulin, lactate and fibrinogen; and decrease below normal ranges in the serum conjugated bilirubin and iron in horse I while horse II shows increase above normal ranges in serum total protein, globulin, fibrinogen, globulin, lactate and bile acid with significant decrease in serum glucose and conjugated bilirubin. Both horses also had significant ( $p<0.05$ ) increase in sodium (Na) electrolytes but significant decrease in organic phosphate in horse 2 (Table 2).

The ticks were identified as *Rhipicephalus evertsi* Neuman, 1897.

**TABLE 1. HAEMATOLOGICAL PARAMETERS OF HORSES NATURALLY INFECTED WITH *T. equi***

Parameters	Horse 1		Horse 2		Normal range	Unit
	BT	AT	BT	AT		
RBC	06.43	08.24	06.30	07.40	06.50-12.00	$\times 10^{12} l^{-1}$
Hgb	11.10	14.40	10.80	13.20	11.00-16.00	g dl <sup>-1</sup>
PCV	32.30	41.50	32.40	40.90	33.00-49.00	%
MCV	50.10	46.20	52.60	48.00	36.00-49.00	fl
MCHC	34.60	33.40	34.50	32.60	30.00-36.00	g dl <sup>-1</sup>
WBC	05.17	07.20	05.96	05.92	05.40-12.00	$\times 10^{12} l^{-1}$
S/NEU	72.00	64.00	73.00	68.00	39.00-71.00	%
B/NEU	01.00	01.00	00.00	00.00	00.00-2.00	%
Lymph	22.00	28.00	24.00	26.00	27.00-59.00	%
Mono	03.00	01.00	00.00	00.00	00.00-04.00	%
Eosino	02.00	01.00	00.00	00.00	00.00-03.00	%
Basophil	00.00	00.00	00.00	01.00	00.00-03.00	%

BT: Before treatment, AT: After treatment, S/NEU: Segmented neutrophil, B/NEU: Banded neutrophil, Lympho: Lymphocyte, Mono: Monocyte, Eosino: Eosinophil.

**TABLE 2. SERUM CHEMISTRY OF HORSES NATURALLY INFECTED WITH *T. equi***

Parameters	Horse 1		Horse 2		Normal range	Unit
	BT	AT	BT	AT		
Sodium	145.00	137.00	147.00	142.00	134.00-143.00	mmol l <sup>-1</sup>
Potassium	4.90	3.80	4.50	4.60	3.00-5.00	mmol l <sup>-1</sup>
Chlorides	102.00	100.00	102.00	101.00	89.00-106.00	mmol l <sup>-1</sup>
Inorg Phosph	1.29	1.28	0.96	1.04	1.10-1.50	mmol l <sup>-1</sup>
Magnesium	1.10	0.90	1.20	1.10	0.60-1.20	mmol l <sup>-1</sup>
Iron	2.20	16.00	1.90	18.60	18.00-40.00	$\mu\text{mol l}^{-1}$
Urea	7.90	6.20	7.00	5.60	3.50-7.30	mmol l <sup>-1</sup>
Creatinine	160.00	161.00	157.01	159.00	80.00-180.00	$\mu\text{mol l}^{-1}$
Total Protein	64.00	63.00	71.00	65.00	50.00-67.00	gl <sup>-1</sup>
Albumin	27.00	26.00	31.00	28.00	24.00-33.00	gl <sup>-1</sup>
Globulin	37.00	32.00	40.00	36.00	20.00-35.00	gl <sup>-1</sup>
Triglycerides	0.40	0.39	0.20	0.20	0.10-0.64	mmol l <sup>-1</sup>
Glucose	4.20	4.00	2.50	4.20	3.40-5.90	mmol l <sup>-1</sup>
AST	207.00	236.00	397.00	372.00	100.00-700.00	$\mu\text{l}^{-1}$
ALP	148.00	143.00	131.00	128.00	120.00-250.00	$\mu\text{l}^{-1}$
GGT	26.00	23.00	41.00	36.00	10.00-70.00	$\mu\text{l}^{-1}$
LDH	924.00	942.00	1225.00	1105.00	520.00-1480.00	$\mu\text{l}^{-1}$
CK	195.00	156.00	195.00	161.00	20.00-400.00	$\mu\text{l}^{-1}$
Total bilirubin	27.00	31.00	39.00	38.00	20.00-50.00	$\mu\text{mol l}^{-1}$
Conj bilirubin	1.00	6.00	1.00	3.00	2.00-15.00	$\mu\text{mol l}^{-1}$
Lactate	4.80	1.50	6.00	1.80	1.00-1.50	mmol l <sup>-1</sup>
Serum bile	9.47	9.42	16.09	14.20	0.00-15.00	$\mu\text{mol l}^{-1}$
Fibrinogen	11.00	4.30	13.00	4.20	0.50-4.00	gl <sup>-1</sup>

AST: Aspartate aminotransferase, ALP: Alkaline phosphatase, GGT: Gamma-glutamyl transpeptidase, LDH: Lactic dehydrogenase and CK: Creatin kinase.

## DISCUSSION

This report presents the dynamics of haematological alterations and serum biochemical imbalance in horses naturally infected with *T. equi*. The disease was characterised by fever, anorexia, weakness and icterus. The disease has been reported in Nigeria (Aliu *et al.*, 1983) but event of biochemical alteration in natural equine theileriosis has not been recorded.

The two horses responded satisfactorily to treatment with imidocarb at 4mg kg<sup>-1</sup> body weight repeated three times at 72 hrs interval. Vial *et al.*, (2006) observed that imidocarb given at 2 mg kg<sup>-1</sup> body weight two times at 24-hrs interval and 4 mg kg<sup>-1</sup> body weight four times at 72 hrs intervals appears effective for *Babesia caballi* and *T. equi* in horses respectively. The significant decrease in blood Hg, PCV and RBC observed in this report conform with the findings of Hailat *et al.*, (1997) in Jordan and Butler *et al.*, (2005) in France but the anaemia in this study was macrocytic normochromic in nature. The lymphopaenia recorded in the two horses could be as a result of the destructive effect of merozoites that could have resulted from pre-erythrocytic schizogony in the lymphocytes (Schein *et al.*, 1981).

The hypernatremia recorded in this report could be as a result of pre-renal dehydration which could lead to hypertonicity. The significant elevation of serum proteins; globulin and fibrinogen could be as a result of chronic inflammatory disorder of the liver such as cirrhosis. Though the total serum bilirubin level was normal, the serum conjugated bilirubin was low probably due elevated serum bile acid level which may have increased the dialysability of conjugated bilirubin. All the serum enzymes levels were within the normal ranges in both cases reported, contrary to the reports of Hailat *et al.*, (1997). The excess serum lactate recorded in both horses could not be unconnected with tissue hypoxia because they both became ill after strenuous exercise.

The satisfactory responses of the horses and return of haematological parameters to normal ranges after treatment with imidocarb preclude diseases such as equine viral arteritis (EVA), equine infectious anaemia (EIA) and purpura haemorrhagical (PH) which were considered as differential diagnosis of equine theileriosis (Blood & Radostits, 1987).

This report suggest that this is a stress induced equine theileriosis and that the horses had been carriers before clinical manifestation of the disease, hence screening of the horses for *T. equi* with more sensitive method such as polymerase chain reaction should be carried out regularly especially on imported breeds .

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