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Research Paper

Haematological changes in *Trypanosoma evansi* infected cattle

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Abstract

During the three years period of study twelve cross bred cattle were identified that they were suffering with clinical *Trypanosomiasis*. Up on clinical examination cattle had anorexia, fever, ocular discharges, dullness and enlarged lymph nodes. Stained peripheral blood examination revealed the presence of *Trypanosoma evansi*. Haematological abnormalities include decreased TEC, PCV, Hb and TLC values ($P<0.01$). The present leucopaenia was characterized by neutrophilia, eosinophilia and lymphocytopaenia ($P>0.05$).

Keywords: *Anaemia - Cattle - Haematology - Leucopaenia - T. evansi*

1. Introduction

Trypanosomiasis is a protozoan disease of livestock commonly called as Surra caused by *Trypanosoma evansi*. The disease is transmitted by *Tabanid* flies. It affects most of the livestock and wild animals. It is distributed throughout the India and found as endemic. The disease is seen as an outbreak in pre monsoon and in monsoon seasons due to active breeding of the *Tabanid* flies. The disease is characterized by anaemia, nervous complications, emaciation and death. It causes severe loss to the farmers by the way of poor milk production, reduced ability to work and high mortality. The disease also interferes with immunity and causes immunosuppression of bacterial and viral infections and sometimes also causes vaccination failure. The incidence and the severity of the disease vary with the strain of the parasite as well as the species of host affected [1]. Clinical signs may vary based on the severity of the infection. Prevalence of *T.evansi* was recorded in different species of animals by different diagnostic methods in India [2, 3]. Haematological and biochemical abnormalities were also recorded in experimental animal infection with *T.evansi* [4]. Little is known about the haematological changes associated with the local strain of *T.evansi* in cattle in Andhra Pradesh. Present study was conducted to study the haematological changes in cattle affected with natural infection with *T.evansi.*

2. Materials and methods

Present study was carried out on the cross bred cattle presented to the College Hospital of College of Veterinary Science, Tirupati during the three years period i.e. from 2009 to 2012. Cattle with inappetance, dullness, pyrexia, bilateral ocular discharges, chronic emaciation, and enlarged lymph nodes were screened for the presence of haemoprotozoans. Initially all the cattle were tested for the presence of haemoprotozoans by peripheral wet blood film examination, followed by stained blood smears examination for confirmation of trypanosomes species [5]. Whole blood was collected from the suspected cattle and ten cross bred cattle (control group) in the age group of 4 to 8 years from the dairy farm in College was served as control group. Blood was collected in 10% EDTA coated vials was used for estimation of packed cell volume (PCV), total leucocyte count (TLC), total erythrocyte count (TEC) and haemoglobin (Hb). The peripheral blood smears were stained by Leishman's stain to study the differential count (DLC) by battlement method following which; the absolute counts were also calculated. Statistical analysis was done by Student's t-test to

compare between infected and control cattle were obtained; *P* value of 0.05 or lower was considered to be significant [6].

3. Results and discussion

Based on the laboratory examination, twelve cross bred cattle diagnosed that they have *T.evansi*. Wet blood film examination revealed the presence of motile *Trypanosoma* organisms in between the RBCs of collected blood from the suspected animals. Up on stained peripheral blood smear examination; organisms were confirmed as *T.evansi* based on morphology (Figure-1).

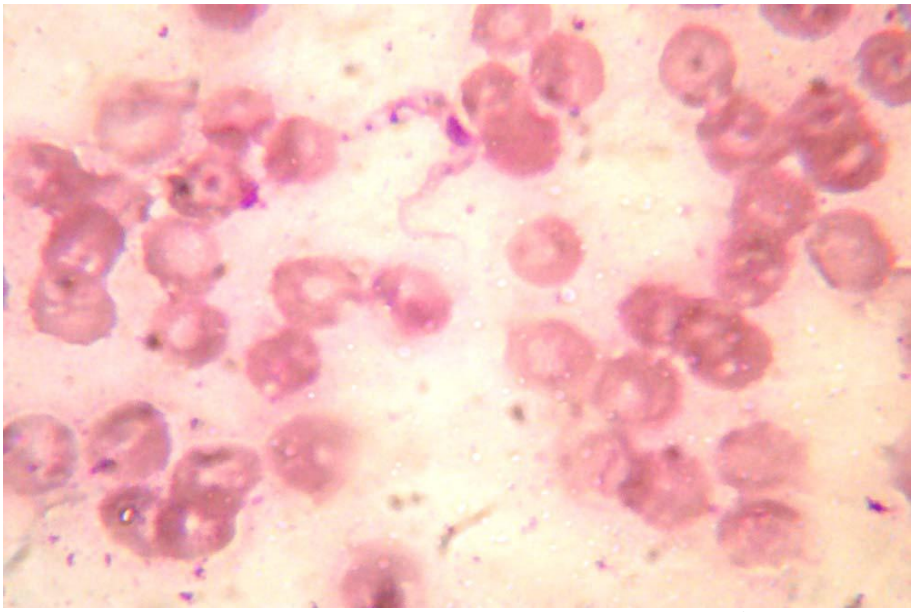

Fig. 1: *T.evansi* in the stained blood smears (100X with 4X Camera magnification)

All the cattle exhibited high rise of intermittent temperature (103.2^0 F to 105.2^0 F), dullness, enlarged lymph nodes, conjunctivitis, corneal opacity, anorexia, sunken eye balls, congested conjunctival mucus membranes, bilateral lacrimation, labored breathing, sudden drop of milk yield and emaciation (Figure-2).

Fig. 2: Sunken eye balls due to *T.evansi* infection in cattle

In four cattle nervous symptoms like walking in circles, beating the head on walls was also noticed. Mean haematological values in *T. evansi* infected cattle as well as control cattle were given in Table-1.

Table 1: Mean haematological values of control and *Trypanosoma* infected cattle (Mean±S.E)

Parameters	Control group (n=12)	*Trypanosoma* infected cattle (n=12)	t - test	P
Hb (g/dl)	10.81 ±0.49	8.37 ±0.31	4.52**	0.0007
PCV (%)	31.71 ±0.89	28.71 ±5.7	2.45*	0.0305
TEC $x10^6$/cumm	5.70 ±0.20	4.76 ±0.80	4.22**	0.0012
TLC /cumm	8000.0±310.9	7328.57 ±71.8	1.86^{NS}	0.0871
Lymphocytes /cumm	5454.57 ±160.4	4828.57 ±312.2	1.78^{NS}	0.0998
Neutrophils /cumm	2071.86±112.7	2310.29±119.8	1.45^{NS}	0.1728
Eosinophils /cumm	259.57 ±15.67	325.86±63.5	1.01^{NS}	0.3314
Monocytes /cumm	388.43 ±11.99	399.29 ±28.48	0.35^{NS}	0.7315

NS – Non Significant (P>0.05)
* Significant (P<0.05)
* * Highly Significant (P<0.01)

The mean TEC, PCV and Hb reduced significantly (P<0.01) compared to control group, indicating anemia. The patho physiology of anaemia in *Trypanosomiasis* is complex and multi factorial in origin [7]. It initiates a cascade of events leading to haemolytic anaemia and cardiovascular collapse [8]. Anaemia caused by mechanical injury to erythrocyte occurs by the lashing action of the powerful locomotory flagella and microtubule reinforced bodies of the millions of the organisms during parasitaemia [9]. Erythrocyte membrane damage has also been associated with adhesion of erythrocytes, platelets and reticulocytes to trypanosome surfaces via sialic acid receptors leading to damages to erythrocyte cell membranes [10]. *Trypanosomiasis* may cause a drop in feed intake hence there is energy deficit and loss of tissue associated with catabolism of body fat, deficiencies of vitamin C, B and essential amino acids [11]. Inadequate energy supply to erythrocytes may alter the erythrocyte membrane surface therefore leading to weakening of the cell membrane, increased osmotic fragility and haemolysis [12]. Igbokwe and Nwosu explained that anaemia due to massive erythrophagocytosis by an expanded and active mononuclear phagocytic system (MPS) of the host [13]. Low PCV observed in the infected group may be as a result of acute haemolysis due to growing infection. Previous studies have shown that infection with trypanosomes resulted in increased susceptibility of red blood cell membrane to oxidative damage probably as a result of depletion of reduced glutathione on the surface of the red blood cell [14]. Severity of anaemia usually reflects the intensity and duration of parasitaemia.

Reduction in the mean total leucocytes count and mean lymphocyte count was noticed but, it was statistically not significant from the control group (P>0.05). Increased in the mean neutrophil count, monocyte count and eosinophils count of the infected animals which were statistically not significant from the control groups (P>0.05). The reduction in leukocyte levels could be a result of immuno-suppression. Abd El-Baky and Salem observed significant leucocytosis, neutrophilia, monocytosis and eosinophilia in naturally infected camels with *Trypanosomiasis* [15]. The eosinophilia observed is a feature of parasitic infections and is associated with immediate-type hypersensitivity reactions. Significant decrease (P<0.05) in RBC, Hb, PCV and lymphocyte count was observed by Chaudhary and Iqbal. They also recorded significant (P<0.05) increase in WBC and neutrophils in natural *Trypanosomiasis* in racing dromedary camels in their studies [16]. Leucopaenia in animal *Trypanosomiasis* has been reported to be due largely to ineffective or depressed granulopoiesis in the bone marrow [17]. The lower counts of WBC, lymphocytes observed in the infected group may be attributed to the immunosuppressive actions of *Trypanosome* infection [18, 19]. Sulaiman and Adeyemi, reported lymphocytosis in their studies which may be due to generalized lymphoid tissue hyperplasia, characteristic of the acute phase of the disease, during which period, lymph nodes and spleen are remarkably reactive while in chronic infection, the immune system becomes depleted of lymphoid cells [20].

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