International Journal of Advanced Research in Biological Sciences
ISSN : 2348-8069
www.ijarbs.com

Research Article

Hemato- biochemical prognostic indicators for *Trypanosoma* infection in a captive asiatic wild dog

Boon Allwin[1*], Thirumurugan R [2], Senthil N.R [3], Kalaignan, Pa[4], Vairamuthu.S[5]

[1]Ph.D Scholar, Dept. of Wildlife Sciences, Madras Veterinary College, Chennai-07
[2]Zoo Veterinarian, Arignar Anna Zoological park, Vandalur, Chennai-51
[3]Assistant professor, Centralized Clinical Laboratory, Madras Veterinary College, Chennai-07
[4]Dept. of Wildlife Sciences, Madras Veterinary College, Chennai-07
[5]Professor and Head, Centralized Clinical Laboratory, Madras Veterinary College, Chennai-07
*Corresponding author

Abstract

Trypanosomiasis (Surra) is an important protozoan disease caused by *Trypanosoma sp* . It is widely distributed in tropical and subtropical regions. It is insect borne and their epidemiology is determined by the ecology of insect vector. The course of Trypanosomiasisinfection in a Asiatic wild dogs was followed from incidence to convalescence, the hematological and biochemical findings were studied. Infected animal showed some alteration in serum biochemistry including hypoglycemia with progressive decrease in red blood cell count and hemoglobin concentration, leading to anemia which persisted. Leucopenia and neutropenia were also observed during the course of the infection. The affected wild dog developed hyperproteinemia and a decrease in the albumin:globulin ratio was observed. Aspartate aminotransferase and alamine aminotransferase levels increased significantly. The present study was to evaluate the hematological and biochemical alterations in a Wild dog infected with *Trypanosoma sp.*

Keywords: Trypanosoma-haematology-biochemistry-death.

Introduction

Trypanosoma is an extra erythrocytic hemoprotozoan parasite, transmitted by biting flies and infected meat, causing fever, corneal opacity, anaemia, and myocarditis. *Trypanosoma evansi* is the causative agent of surra , an important disease widely distributed in tropical and subtropical regions. Surra affects a great variety of domestic and also wild mammals [1]. Infections with manifestation of severe clinical symptoms have been reported [2],[3][4]. Surra is characterized by high morbity and mortality rates and anemia has been recorded as a consistent finding in naturally infected dogs and Anemia is a cardinal feature of the disease in which red blood cells are removed from the circulation by the expelled mononuclear phagocytic system. Later, in infection of several months duration, when the parasitaemia become low and intermittent, anemia may resolve to a variable degree [5].

Despite the importance and the worldwide distribution of surra, very little is known about the pathogenesis of this trypanosomiasis. Moreover, there are few reports about the disease which refers to isolated cases of natural infection, what justifies additional investigation and the present paper describes the hematological, biochemical alterations in a wild dog infected with trypanosomiasis and the possible causes of death due to trypanosomiasis.

Materials and Methods

A male captive wild dog aged 3 years weighing 15 kg, of Arignar Anna Zoological Park, Vandalur

was presented with a complaint of anorexia, lethargy, edema of forehead, staggering gait and reduced activity in enclosure. The wild dog was chemically immobilized with xylazine @ 1mg/kg and ketamine @ 10 mg/kg and taken to the zoo veterinary hospital for detailed examination and treatment. Physical examination revealed high rise of temperature (40.8^{o}C), pale mucous membrane, bilateral lacrimation, and generalized debility. On thoracic auscultation, exaggerated breath sounds on both sides were observed. The wet film was observed immediately using microscope, the peripheral blood smear, whole blood and serum were analysed. Hematology examination was done in Auto-haemoanalyser (BC-Vet 2800) and serum biochemistry in A15 auto analyzer (Biosystems).

Results

Wet film revealed numerous motile organisms that were suggestive of *Tryps* sp., the blood smear stained with Leishman-Giemsa stain revealed as many as 4-7 trypanosomes per field which is indicative of severe infection. Haematological parameters and Serum biochemistry was found to be altered (Table 1, 2&3)

Table 1. Haemotological examination

Component	Recorded value	Normal range*
Hb (g/dl)	9.2	(16.69 ± 0.26)
RBC*10^6	4.92	(7.49 ± 0.05)
PCV (%)	26.7%	(46.88 ±1.31)
WBC*10^3	7.2	(7.91 ± 4.79)
MCV	54.43	62.50
MCH	29.0	28.0
Anaemia-microcytic normochromic		

Table.2 Differential count

Cell	Recorded value	Normal range*
Neutrophils	74	(72.63 ± 2.81)
Lymphocytes	20	(25.25 ± 2.76)
Monocytes	02	(00.75 ± 0.31)
Eosinophils	04	(01.38 ± 0.41)
Basophils		NIL

Table 3. Serum Biochemistry

Parameters	Recorded value	Normal range*
BUN	20.40	(20.56 ± 0.74)
Creatinine	2.87	(1.44 ± 0.04)
Total protein (g/dl)	7.03	(6.09 ± 0.30)
Albumin (g/dl)	3.01	(2.38 ± 0.22)
Globulin (g/dl)	4.02	(3.71 ± 0.33)
Calcium (mg/dl)	2.70	(9.10 ± 0.64)
Phosphorus(mg/dl)	6.07	(5.86 ± 0.48)
Glucose	33.99	(53.63 ± 1.28)
ALT	102	(19.76 ± 0.79)

Normal range * [17**]**

Discussion

The results of the present study are in accordance to who studied clinical hematology of canine chages disease and reported hypoproteinemia, hypoalbuminemia, anemia, decrease packed cell volume and slight thrombocytopenia. The leucogram indicated leucopenia with no change in differencial count was in regard to some authors [2][4], while others registered no change in total white blood cell count [6][7]. Some alterations in blood biochemistry, including hypoglycemia and decrease in albumin:globulin rate, were verified in naturally infected dogs [2], [7].

The anemic changes are attributable to extravascular destruction of RBC which may be through the process of erythrophagocytosis or metabolic product and toxins liberated from the parasites. Anemia was a consistent finding as reported previously in different hosts infected with *Trypanosomiasis*[8],[9],[10] including dogs[2],[4],[6]. Despite being considered a significant pathological feature of the disease, the origin of anemia is not completely elucidated but anaemia can be attributed to some causes like the whip like motion of their flagellum, causing mechanical damage to the RBC's membrane, the increase in temperature because of the infection causes an increased osmotic fragility, the sialidase enzyme cleaves Sialic acid on RBC membrane and destroys it, toxins and metabolites released when Trypanosomes are lysed further Disseminated Intravascular Coagulopathy (DIC), clogs the capillaries, the RBC's going through get damage and are Phagocytized by Mononuclear phagocytic system MPS, Lysed Trypanosome (Antigen) may coat the RBCs further Phagocytized by MPS, Hyperactivity of MPS Phagocytise the normal RBCs, damage of haemopoietic organs by Trypanosome toxins reduces the haemopoiesis and causes Anaemia.

Additional mechanisms include hemolysis as a result of erythrophagocytosis, hemodilution and depression of erythropoiesis. Increase in serum unconjugated bilirubin levels in infected animals occurred at week four after inoculation, coinciding with the lowest RBC values observed, suggesting the occurrence of an hemolytic crisis in this period of the infection. Hyperbilirubinemia has been reported in naturally infected dog as consequence of an increase in unconjugated bilirubin [7] and conjugated bilirubin [6]. Increase in serum bilirubin levels was not a consistent finding during the infection and did not exceed normal limits suggesting that extravascular destruction of red cells is a more likely the explanation for anemia. The wild dog showed leucopenia with neutropenia. Leucopenia [2] and normal leucograms [6],[7] have been reported in naturally infected dogs. The marked rise in AST levels, compared to the rather modest increase in ALT observed in infected animals, indicate that little of the former is derived from the liver. It is likely that the AST originated from heart muscle since myocarditis was found in three infected dogs and Increases in ALT and AST activity have been also observed in a *Trypanosomiasis* naturally infected dog [7]. A significant increase in serum protein levels, as consequence of globulin rise, and a parallel decrease in albumin concentrations were observed in infected dogs. The decrease in albumin:globulin ratio has been frequently reported in *Trypanosomiasis* infection in various studied hosts [2],[8], [10],[11], [12]. It is suggestive that fall in albumin levels was secondary to hyperglobulinemia as a compensatory mechanism for the maintenance of normal blood viscosity increased by high globulin levels. Hypoalbuminemia as consequence of liver damage may be rejected since serum albumin levels fall only after extensive and chronic liver malfunction, what was not a feature in this experimental infection, as evidenced by liver function tests and anatomopathological findings. There is evidence to suggest that increase in immunoglobulin levels was responsible for hyperglobulinemia observed in infected dogs. Study of electrophoretic patterns of serum proteins in *Trypanosomiasis* infected camels [8] [11][13][14] showed a marked increase in ɣ-globulin fraction which is coinciding with findings of this case

The anaemia ultimately ends up in anoxia, all organs need oxygen for efficient and effective functioning for maintenance and regulation. CNS is most susceptible to anoxia leading to various nervous signs like ataxia, staggering gait mimicking blindness. Most importantly, anoxia leads to decreased mitochondrial function interfering with the Glycolysis, leading to lactic acid build up leading to tissue damage. Most significant case of tissue damage is Myocardial Damage [6] [15].Separation and degeneration of the muscle fibres of the heart occur which, if extensive enough, will be fatal. Also organs like spleen, liver and bone marrow are usually damaged. Bone marrow

damage: Aggravates anaemia, Immunosuppression. Liver damage that is evident by increased ALT and AST levels in affected animals as in this case coinciding with the recordings of [7]. Spleen damage: active Immunosuppression. Secondary infections may also be fatal to the animal due to immunosuppression. (as immune system is exhausted and bone marrow is insulted) This explains the existence of concurrent infections with other microbes and also the effects of drug action nullified with a failing immune system.

Variant Surface Glycoprotein (VSG) [16] causes the intermittent and high fever usually fatal if animal but depends on the status of the animal. Immune system does kill most trypanosomes but when compromised the toxins flood the circulatory system leading to toxaemia ultimately death. This VSG is also a factor that hinders the invent of a vaccine for trypanosomiasis. Trypanosomes are generally dependent on glucose for their nutrition and drain the glucose content from the host tissue. Glucose is an essential energy source for all tissues. Trypanosomes being glucose dependant for their nutrition absorb large amount of glucose from blood causing hypoglycaemia. Liver glycogen depleted, muscle glycogen depleted. Protein is the next bio molecule attacked and finally fat is attacked. However, not efficient due to lack of Oxygen (Anoxia).Liver cells are overworked and anoxic causing damage to liver. RBC (from lack of glucose) have less Oxygen intake leading to asphyxia and acidosis

Conclusion

This paper indicates the changes in the haematology and serum biochemical quotients that occur during the course of trypanosome infection. Further the various reasons for the the cause of death due to trypanosomes are opined with scientific justification. Intense research with the species level specifications and experimental infections in simulated models may be helpful in pin pointing pathology and related diagnosis and treatment. This paper concludes that Pyrexia, Toxaemia (Aggravated by less efficient liver), Secondary infections due to immunosuppression, Anaemia (PCV < 30%, decreased blood viscocity) Anoxia, Hypoglycaemia (reduced blood glucose by 30%), failure of liver cells to compensate, Heart failure due to Myocardial damage (by toxins, anoxia), Strain on heart to pump watery blood are cause of death by trypanosomes.

Acknowledgments

The authors sincerely thank the Dean, Faculty of basic sciences, Madras Veterinary College, Dr.S. A. Asokan and Thiru. K.S.S.V.P.Reddy I.F.S., Additional Principal Chief Conservator of Forests and director, Arignar Anna Zoological park, Vandalur, for the facilities provided and to all the personnel involved in this from logistics to keeping.

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