Clinical Article

DIAGNOSIS AND TREATMENT OF CANINE TRYPANOSOMIASIS – A CASE STUDY

P Ramesh¹, CH Sudha Rani Chowdary¹ and Y Chaitanya¹
¹Assistant Professor, Dept. of TVCC, NTR CVSc, Gannavaram
Krishna District, Andhra Pradesh Pin-521 101
E-mail: rameshvety777@gmail.com (*Corresponding Author)

Abstract: Trypanosomiasis was diagnosed in seven dogs of different breeds, age groups and sex with clinical signs like chronic inappetance, gradual loss of physical condition, dyspnoea, lymphadenopathy, excitability, tremors and seizures. Clinical examination of all the dogs revealed pale mucous membranes, weak pulse, enlarged lymph nodes, rough hair coat and loss of skin elasticity. Wet blood film and stained blood smears showed large number of trypomastigotes of trypanosomal organisms. Hemato-biochemical findings showed moderate to severe anemia, lymphocytosis, hypoglycemia, hypoproteinemia, hypoalbuminemia, elevated ALT, BUN and creatinine levels. All the dogs were treated with single dose of Diminazine aceturate @ 3.5 mg per kg body weight IM along with supportive therapy. Complete clinical recovery was recorded in 85.7 % (6/7) of dogs.

Keywords: Canines, lymphadenopathy, trypomastigotes, anaemia, diminazine aceturate.

Introduction

Trypanosomiasis is an important and widely prevalent hemoproteozoan disease caused by T.evansi which affects a wide variety of domestic, wild and zoo animals and transmitted by biting flies particularly Tse tse, Tabanus, Stomaxys, Culicoides etc (Green, 2006). Severity of canine trypanosomiasis ranges from acute, subacute to chronic. In dogs an acute and fatal type is commonly seen and death possibly occurs in 2-4 weeks (Soulsby, 1982). Lakshmi Prasad et al., (2015) reported prevalence of 2.28 % in male, 2.40% in female dogs and highest prevalence in young dogs of less than 2 years of age. Trypomastigote form of trypanosome enters host cells soon after infection, multiplies sub clinically, escapes the immune system and spread throughout the body primarily within macrophages. Parasitaemia develops within a few days and peaks 2 to 3 weeks post infection, coinciding with clinical symptoms (Barr et al. 1991). Clinical signs are characterized by weight loss, progressive weakness, anorexia, anaemia, intermittent fever, conjunctivitis, swelling of limbs, enlarged superficial lymph nodes and bilateral corneal opacity which is a characteristic finding in chronic trypanosomiasis (Thirunavukkarasu et al. 2004).
Materials and Methods

Trypanosomiasis was diagnosed in seven dogs of different breeds and age groups of either sex that were presented to the Teaching Veterinary Clinical Complex, NTR College of Veterinary Science, Gannavaram during the period from June, 2014 to November, 2014. All the cases were subjected to detailed clinical examination, microscopic examination of wet blood films, hematological and serum biochemical studies. Single dose of Diamaminizine aceturate @ 3.5 mg/kg body weight intramuscularly is used as therapeutic regimen along with the supportive therapy in the present study.

Results and Discussion

In the present study, incidence of trypanosomiasis observed in male dogs was 57.14% (4/7) of which 71.42% (5/7) were kept near dairy farms and fish ponds for guarding purpose. Greene, (2006) stated that transmission of infection in nature occurs primarily around water sources frequented by hosts and vector flies. Singh et al., (1993) also reported an incidence of 4.68% of subclinical trypanosomiasis in dogs kept near dairy farms during rainy season. No correlation was found based on age, breed and sex, the distribution of which is shown in table 1. In the present study clinical findings recorded were inappetance to anorexia from one to two weeks, gradual loss of physical condition, weakness and depression in four dogs corneal opacity, epistaxis, excitability, tremors and seizures in two dogs and circling in one dog (Fig.1&2). Pyrexia, enlarged lymph nodes, pale mucous membranes (Fig. 5), increased CRT (capillary refill time) were noticed upon physical examination in all cases. Similar findings were reported by Thirunavukkarasu et al., (2004), Rani and Suresh (2007) and Rashid et al., (2008).

In all the cases, examination of wet blood film and stained blood smears showed large number of trypomastigotes of trypanosomal organisms (Fig. 3& 4). Irwin and Jefferies (2004) also reported that presence of trypomastigotes in thick or thin blood films, buffy coat smears was diagnostic finding in trypanosomiasis in dogs. Hemato-biochemical findings showed moderate to severe anemia, hypoglycemia, hypoproteinemia, hypoalbuminemia, elevated ALT, bilirubin, BUN and creatinine levels (Table 2).

Anemia was a consistent finding as reported previously in different hosts infected with T. evansi due to hemolysis as a result of erythrophagocytosis, hemodilution and depression of erythropoiesis (Jaktar and Purohit, 1971). In the present study, neutropenia and reactive lymphocytosis were recorded which are in agreement with Aquino et al. (2002).
Hypoglycemia was a consistent feature in all the cases of this study which is due to utilization of blood glucose by parasites in circulation thereby lowering blood glucose levels.

The hypoproteinemia and hypoalbuminemia reported in this study are in agreement with findings of other workers on trypanosomosis (Nwoha et al. 2013, Bisalla et al. 2007 and Orhue et al. 2005). The decrease in serum albumin and thereby serum protein could be attributed to decreased liver biosynthesis and progressive loss of albumin in urine (Agu and Egbuji, 2002).

Uremia and elevated creatinine levels in this study are in agreement with Kwem et al (2000). However, Nwoha et al. 2013 reported initial increase and subsequent decrease in BUN values in their study on trypanosomosis. Uremia and elevated creatinine levels observed in this study could be due to kidney dysfunction due to tissue damage caused by parasitemia. David and Michael, 2003 attributed severe creatinemia to sequestration of the trypanosomes in the muscle tissues of the heart leading to damage of the cardiac muscles and release of creatine kinase into circulation.

Hyperbilirubinemia in this study is in accordance with many authors (Nwoha et al. 2013, Kwem et al. 2000, Jerry and Victor 2007). This could be due to the hemolysis caused during severe parasitemia. Increased ALT in this study is in accordance with Nwoha et al. 2013 which could be due to hepatic damage caused by parasitemia.

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Breed</th>
<th>No. of dogs affected</th>
<th>Age</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Grate Dane</td>
<td>1</td>
<td>11/2 Year</td>
<td>Male</td>
</tr>
<tr>
<td>2</td>
<td>Doberman</td>
<td>2</td>
<td>2 Year</td>
<td>Male</td>
</tr>
<tr>
<td>3</td>
<td>Rottweiler</td>
<td>1</td>
<td>9 months</td>
<td>Male</td>
</tr>
<tr>
<td>4</td>
<td>Spitz</td>
<td>2</td>
<td>5 Year</td>
<td>Female</td>
</tr>
<tr>
<td>5</td>
<td>Mongrel</td>
<td>1</td>
<td>3 Year</td>
<td>Female</td>
</tr>
</tbody>
</table>

Table 2:

<table>
<thead>
<tr>
<th>Hematobiochemical parameter</th>
<th>Mean±SE</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEC (millions/cmm)</td>
<td>3.15 ± 0.16</td>
<td>4.8-9.3</td>
</tr>
<tr>
<td>TLC (thousands/cmm)</td>
<td>6.66± 0.55</td>
<td>4.0-15.5</td>
</tr>
<tr>
<td>Hb(g%)</td>
<td>6.16 ± 0.47</td>
<td>12-15</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>19.16 ± 1.2</td>
<td>37-55</td>
</tr>
<tr>
<td>Lymphocytes (%)</td>
<td>59.33 ± 3.38</td>
<td>12-30</td>
</tr>
<tr>
<td>Neutrophils (%)</td>
<td>39.16 ± 3.62</td>
<td>60-77</td>
</tr>
<tr>
<td>Monocytes (%)</td>
<td>0.33 ± 0.21</td>
<td>3-10</td>
</tr>
<tr>
<td>Eosinophils (%)</td>
<td>1.16 ± 0.30</td>
<td>2-10</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>1.56 ± 0.13</td>
<td>2.7-4.4</td>
</tr>
<tr>
<td>ALT/SGPT (u/L)</td>
<td>109.66 ± 3.48</td>
<td>5-107</td>
</tr>
</tbody>
</table>
All the dogs were treated with a single dose of diminazine aceturate @ 3.5 mg / kg deep IM, Meloxicam @ 0.5 mg/kg SC, 20% dextrose @ 5ml/kg IV followed by DNS until correction of dehydration and oral supplementation of hematinsics was given (Fe-folate) for two weeks to correct anemia. Uneventful recovery was noticed in 85.7 % of cases (6/7), but one dog with acute neurological signs died on the day of therapy, complete absence of parasitemia was recorded on day two in rest of the cases (Fig.6). Clinical recovery was noticed during second week of therapy (disappearance of corneal opacity, pale mucosa etc.). Single dose of diminazine aceturate is effective for complete elimination of parasitemia on day two of therapy. Which was in agreement with Rashid et al (2008) and Rani and Suresh (2007) who stated that Diminazene diaceturate given IM once at a dosage of 3.5 mg/kg was effective for trypanosome infections in dogs showed good clinical improvement after treatment.

**Conclusions**

No correlation was found with age, sex and breed wise, but dogs kept at dairy farms and near fish ponds for guarding purposes will have a higher risk for the infection due to increased fly activity especially in the rainy season. Pyrexia, enlarged lymph nodes and anaemia are consistent findings where as corneal opacity and nervous sings might appear in chronic form of the disease. Single dose of diaminizine aceturate is effective in eradicating infective forms of the trypanosomal organisms on day two of the therapy.
Figures

**Fig.1:** Photograph showing monitoring of severely ill dog under recumbency with acute form of trypanosomiasis

**Fig.2:** Photograph of dog with corneal opacity affected with trypanosomiasis

**Fig.3:** Photomicrograph of a wet blood film showing trypomastigotes (400X)

**Fig.4:** Photomicrograph of a blood smear showing large number of trypomastigotes. Leishman's stain, 1000X

**Fig.5:** Photograph of the affected dog showing pale penile mucosa

**Fig.6:** Photograph of dog showing clinical improvement following therapy on day one
References


