

A case of *Trypanosoma congolense* savannah type infection and its management in a dog

Peter Kimeli*, Ambrose Ngeno Kipyegon, Willy Edwin Mwangi and John Demesi Mande

Department of Clinical Studies, Faculty of Veterinary Medicine, University of Nairobi, PO Box 29053-00650, Kangemi, Kenya.

*Corresponding author's e-mail: kimeli08@yahoo.com

ABSTRACT

A case of *Trypanosoma congolense* savannah type infection in a 4-year old German shepherd dog weighing 26-kg was presented to the Small Animal Clinic, University of Nairobi, Kenya, with the history of anorexia and difficulty in breathing. The clinical manifestations were fever, pale mucous membrane, dyspnea and wasting. Blood examination revealed the existence of trypanosome parasites, and showed mild anemia. Internal Transcribed Spacer (ITS) based polymerase chain reaction confirmed the presence of *Trypanosoma congolense* savannah type. Along with supporting therapy, the case was successfully managed using diminazene aceturate injection (dosed at 3.5 mg/kg body weight) through intramuscular route. Complete recovery of the case was observed on day 6 of post-treatment.

Keywords

Dogs, German shepherd, Natural infection, Recovery, *Trypanosoma congolense* savanna type

Received : 08 July 2014,

Revised: 31 July 2014,

Accepted : 05 August 2014,

Published online: 08 August 2014.

INTRODUCTION

Animal trypanosomosis constitutes a serious impediment to livestock production and economic development in tsetse infested regions of Sub-Saharan Africa (Matete, 2003; Nimpaye et al., 2011; Abakpa et al., 2013). *Trypanosoma congolense* (*T. congolense*) is considered to be the most important cause of animal trypano-

somosis in East Africa (Matete, 2003), which is transmitted cyclically by *Glossina* sp. (Gillingwater et al., 2010), and mechanically by other biting flies. Dogs have been shown to be highly susceptible to *T. congolense* which makes them an important sentinel for infection (Greene, 2006; Museux et al., 2011). Dogs have also been reported to be infected following consumption of trypanosome infected carcasses or ingestion of insect vectors (Montenegro et al., 2002).

Four different types of *T. congolense* found in different ecological zones were identified using deoxyribonucleic acid (DNA) probes; these were *T. congolense* forest type, *T. congolense* kilifi type, *T. congolense* savannah type, and *T. congolense* tsavo type (Majiwa et al., 1993; Clausen et al., 1998). All these types are pathogenic, and their infection in dogs is characterized by parasitaemia, leucopenia, and anemia (Bengaly et al., 2002; Sidibe et al., 2002).

Although, *T. congolense* infection is common in dogs, only savannah (Gow et al., 2007) and forest (Desquesnes et al., 2012) types have been reported to cause natural infection in dogs. However, the dogs died during the disease management period. A number of trypanosomacidal agents have been used effectively against trypanosomosis in canines (Rani and Suresh, 2007). However, there are scanty reports on response of therapy to a natural infection of *T. congolense* savannah type in dogs.

The current report described a case in a dog that was naturally infected with *T. congolense* savannah type, and its successful therapeutic management.

CASE HISTORY, FINDINGS AND MANAGEMENT

A 4-year old German shepherd dog weighing 26-kg was presented to the Small Animal Clinic, University of Nairobi, with the history of anorexia and difficulty in breathing. The dog had been to the coastal region of Kenya for over a year; the area was reported to be endemic for trypano-somosis. Clinical examinations revealed fever (40.2°C), pale mucous membrane, dyspnea, and general body weakness. A blood smear from the ear vein stained with giemsa revealed *Trypanosoma* parasites, and hematology showed mild anemia (Table 1). A polymerase chain reaction (PCR) confirmed the presence of *Trypanosoma congolense* savannah type (Figure 1) in blood. The PCR amplification was done using the primers ITS 1 BR: 5'-TTG CTG CGT TCT TCA ACG AA -3', and ITS 1 CF: 5'-CCG GAA GTT CAC CGA TAT TG -3', as described by Thumbi et al. (2008).

Table 1: Hematology results of a 4-year old German shepherd dog that was naturally infected with *Trypanosoma congolense* savannah type as observed at the Small Animal Clinic, University of Nairobi.

Parameter	Recorded value	Normal range
WBC	13.02 M/mm ³	6.0-17 M/mm ³
Lymphocytes	18.9%	10-40%
Monocytes	2.5%	2-10%
Granulocytes	78.6%	50-80%
Erythrocytes	4.13 M/mm ³	5.5-8.5 M/mm ³
MCV	71.5 fl	58-73 fl
Hematocrit	29.5%	35-55%
MCH	29.5 pg	19.5-24.5 pg
MCHC	41.3 g/dL	28-40 g/dL
Hb	12.2 g/dL	10-18 g/dL

WBC = white blood cell, MCV = Mean Corpuscular Volume, MCH = Mean Corpuscular Hemoglobin, MCHC = Mean Corpuscular Hemoglobin Concentration, Hb = Hemoglobin.

The patient was treated using diminazene aceturate (Veriben®, CevaSanteAnimale - LaBalastiere, France) dosed at 3.5 mg/kg b.wt., administered intramuscularly, and the drug was repeated once after 14 days. In addition, long acting amoxicillin trihydrate (Betamox® Norbrook veterinary Pharmaceuticals, Nairobi-Kenya) was administered once dosed at 10 mg/kg b.wt., and a total volume of 2L of 5% dextrose solution was given twice a day for 2 days. Re-evaluation for parasitaemia was done daily for 2 weeks, and was found as negative on day 5 of the initial treatment. Complete clinical recovery was observed day 6 of post-treatment.

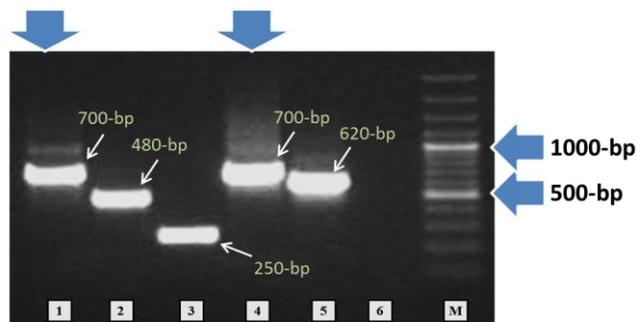


Figure 1: Gel electrophoresis of the infected animal and reference DNA samples amplified with ITS 1 BR and ITS 1 CF; **Lane 1:** dog sample (700-bp), **Lane 2:** *T. brucei* (480-bp), **Lane 3:** *T. vivax* (250-bp), **Lane 4:** *T. congolense* savannah type (700-bp), **Lane 5:** *T. congolense* kilifi type (620-bp), and **Lane 6:** negative control. **Lane M:** 100-bp ladder.

DISCUSSION

Several studies in dogs have reported *T. congolense* infections of unknown types (Harrus et al., 1995; Matete, 2003; Abakpa et al., 2013). On the other hand, there were reports of *T. congolense* savannah type (Gow et al., 2007) and forest type (Desquesnes et al., 2012) infections in dogs. However, in all cases, the dogs died despite attempted management. The current report illustrates a favorable outcome following successful therapeutic management of *T. congolense* savannah type infection in a dog.

The clinical signs in the reported case were fever (40.2°C), anemia, and wasting. These observations were in agreement with those of Rani and Suresh (2007) and Rashid et al. (2008) who reported trypanosomosis in dogs. Bilateral corneal opacity is a characteristic finding in chronic trypanosomosis (Rani and Suresh, 2007). However, this was not observed in the current case.

The hematological findings in the current study revealed mild anemia (Table 1). Other studies have reported severe anemia in dogs associated with trypanosomosis (Gow et al., 2007; Desquesnes et al., 2012). It was postulated that the mild anemia observed in the current case might be due to low parasitemia.

Diagnosis by microscopy does not allow the identification below the subgenus level, and fails to detect mixed infections or immature trypanosome infections. The molecular biology method could overcome the limits of sensitivity and specificity. The PCR thus allowed the *in-vitro* amplification of specific DNA sequence facilitating the accurate detection of many parasites (Morlais et al., 1998). In this study, PCR was successfully used to identify *T. congolense* savannah type in dog.

The Internal Transcribed Spacer (ITS) based assay made the PCR diagnosis more accurate and faster (Thumbi et al., 2008). However, microscopy was considered as the most appropriate method for clinical diagnosis in the field condition.

Diminazene aceturate was administered as an antiprotozoal drug of choice because of its availability and reported positive outcome in dogs (Rashid et al., 2008). This drug was used for both curative and prophylactic reasons; the latter was the reason for our repeat treatment. A broad spectrum antibiotic was used to provide protection against secondary bacterial infection predisposed by the lowered immunity. Dextrose solution (5%) was administered to counter the perceived hypoglycemia.

CONCLUSION

The patient was in an early stage of infection due to the mild anemia and low parasitemia observed. This might have contributed to the positive response to the successful management of the case. Moreover, full recovery was therefore attributed with early diagnosis and prompt treatment.

REFERENCES

- Abakpa SAV, Takeet MI, Makinde AF, Adeleye AI, Adebayo OO (2013). Molecular diagnosis of natural infection of *Trypanosoma congolense* in a dog in Abeokuta, Nigeria: A Case Report. *IOSR Journal of Agriculture and Veterinary Science*, 5: 3-6.
- Bengaly Z, Sidibé I, Ganaba R, Desquesnes M, Boly H, Sawadogo L (2002). Comparative pathogenicity of three genetically distinct types of *Trypanosoma congolense* in cattle: clinical observation and haematological changes. *Veterinary Parasitology*, 108: 1-19.
- Clausen PH, Wiemann A, Patzlt R, Kakaire D, Poetzsch C, Peregrine A, Mehlitz D (1998). Use of PCR assay for the specific and sensitive detection of *Trypanosoma* spp in naturally infected dairy cattle in peri-urban Kampala, Uganda. *Annals of the New York Academic Science*, 29: 21-31.
- Desquesnes M, Ravel S, Deschamps JY, Polack B, Roux F (2012). Atypical hyperpachymorph *Trypanosoma* (nannomonas) *congolense* forest-type in a dog returning from senegal. *Parasite*, 19: 239-247.
- Gillingwater K, Mamabolo MV, Majiwa PAO (2010). Prevalence of mixed *Trypanosoma congolense* infections in livestock and tsetse in KwaZulu-Natal, SouthAfrica. *Journal of the South African Veterinary Association*, 81: 219-223.
- Gow AG, Simpson JW, Picozzi K (2007). First report of canine African trypanosomosis in the UK. *Journal of Small Animal Practice*, 48: 658-661.
- Greene CE (2006). Trypanosomiasis. In *infectious disease of the dog and cat*. 3rd Edn., WB Saunders; pp 676-685.
- Harrus S, Harmelin A, Presenty B, Bark H (1995). *Trypanosoma congolense* infection in two dogs. *Journal of Small Animal Practice*, 36: 83-86.
- Majiwa PAO, Maina M, Waitumbi JN, Mihok S, Zwegarth E (1993). *Trypanosoma* (Nannomonas) *congolense*: molecular characterization of a new genotype from Tsavo, Kenya. *Parasitology*, 106: 151-162.
- Matete GO (2003). Occurrence, clinical manifestation and the epidemiological implications of naturally occurring canine trypanosomosis in western Kenya. *Onderstepoort Journal of Veterinary Research*, 70: 317-323.
- Montenegro VM, Jiménez M, Dias JC, Zeledón R (2002). Chagas disease in dogs from endemic areas of Costa Rica. *Mem Inst Oswaldo Cruz*, 97: 491-494.
- Museux K, Boulouha L, Majani S, Joirnaux H (2011). African trypanosoma infection in a dog in France. *Veterinary Record*, 168: 590. DOI: 10.1136/vr.d888
- Nimpaye H, Njiokou F, Njine T, Njitchouang GR, Cuny G, Herder S, Asonganyi T, Simo G (2011). *Trypanosoma vivax*, *T. congolense* "forest type" and *T. simiae*: prevalence in domestic animals of sleeping sickness foci of Cameroon. *Parasite* (Paris, France), 18: 171-179.
- Rani NL, Suresh K (2007). Canine trypanosomiasis. *Indian Veterinary Journal*, 84: 186-187.
- Sidibe I, Bengaly Z, Boly H, Ganaba R, Desquesnes M, Sawadogo L (2002). Differential pathogenicity of *Trypanosoma congolense* subgroup: implication for the strategic control of trypanosomiasis. *Newsletter on Integrated Control of Pathogenic Trypanosomes and their Vectors*, 6: 33-35.
- Thumbi SM, McOdimba FA, Mosi RO, Jung'a JO (2008). Comparative evaluation of three PCR base diagnostic assays for the detection of pathogenic trypanosomes in cattle blood. *Parasite Vectors*, 1: 46.
- Rashid A, Rasheed K, Hussain A (2008). Trypanosomiasis in dog; A case report. *Journal of Arthropod-Borne Diseases*, 2: 48-51.
- Morlais I, Grebaut P, Bodo JM, Djoha S, Cuny G, Herder S (1998). Detection and identification of trypanosomes by polymerase chain reaction in wild tsetseflies in cameroon. *Acta Tropica*, 70: 109-117.