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Cerebral Trypanosomosis in buffalo - A case study

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Abstract
Trypanosoma evansi affects a wide range of host like cattle, buffalo, dog, horse and camel including man. Trypanosomosis in buffaloes is highly prevalent in Indian subcontinent. This communication deals with the clinical management of cerebral trypanosomosis in a4-year-old adult buffalo from Chirkhatta forest region of Lalkuan, District Nainital, Uttarakhand. The animal was presented with the history of dull and depressed appearance with generalised weakness, intermittent fever (39.5-40^0 C), nervous signs like circling movements, hyperaesthesia, twitching of muscles, pressing of head against the manger, staggering gait and stamping of feet. The case was suspected for nervous form of trypanosomosis. Confirmatory diagnosis was made using microscopic examination of thin blood smear stained with Giemsa stain that revealed the presence of intercellular flagellated hemoprotozoan, *Trypanosoma evansi*. The successful therapeutic management was attained by administration of single injection of Triquin @ 6 mg/kg body weight via subcutaneous route followed by supportive drugs. A gradual reduction was observed in nervous signs after 5 days post treatment. The clinical signs completely disappeared after a week. Peripheral blood smear was again examined 7 days post treatment and found negative for *Trypanosoma evansi*.

Keywords: Trypanosoma evansi, cerebral form, haematology, therapeutic management

1. Introduction
Animal trypanosomosis is one of the major economic yet neglected disease in tropical and subtropical regions of the world [5, 18].Among the various pathological trypanosomes,'Surra'caused by *Trypanosoma evansi* is considered as the most common and widely distributed both in terms of host range and the geographical distribution across the globe [6]. *T. evansi* is a flagellated hemoprotozoan which is transmitted by variety of flies *viz. Tabanus*, *Stomoxys*, *Haematopota*, *Chrysops* and *Lyperosia*. The disease is mainly prevalent in those areas which provide a suitable breeding ground and conditions for the propagation of vector flies. The pathological course of disease may vary from a symptomless carrier to per-acute, acute and chronic state. The various signs and symptoms of trypanosomosis include intermittent fever, progressive anaemia, weakness, oedema, conjunctivitis, marked depression, abortion, petechial haemorrhages, neurologic abnormalities, and sudden death [15]. Despite of being a blood and tissue parasite, it is also responsible for the various neurological abnormalities in cattle, buffalo, deer and pigs [2]. The neurological signs and symptoms mainly include circling, in-coordination, wobbling, restlessness and difficulty in rising. Keeping in view the severe pathogenic effect of nervous form of Surra, the present study was conducted to know is proper management.

2. Materials and Methods
A 4-year-old female buffalo was brought to Teaching Veterinary Clinical Complex, College of Veterinary and Animal Sciences, Pantnagar from Chirkhatta forest region of Lalkuan of District Nainital, Uttarakhand. The animal was presented with various signs and symptoms including dull and depressed appearance, generalised weakness, intermittent fever (39.5-40 ^{0}C), circling movement, hyperaesthesia, twitching of muscles, head pressing against the manger, staggering gait and stamping of feet. Based on the clinical history, animal was suspected for *Trypanosoma evansi* infection.

Microscopic examination of blood sample: Blood sample was collected in EDTA vial and brought to the Department of Veterinary Parasitology for confirmatory diagnosis.

Blood sample was examined by thin blood smear method using Giemsa stain [3]. The prepared blood smear was carefully examined under oil immersion microscope for the presence of organisms.

Haematological examination: The whole blood mixed with EDTA was thoroughly analysed for the study of various haematological parameters *viz*; haemoglobin(Hb), packed cell volume (PCV), total erythrocytic count (TEC), total leucocytic count (TLC), differential leucocytic count (DLC), mean corpuscular haemoglobin (MCH), mean corpuscular volume (MCV) and mean corpuscular haemoglobin concentration (MCHC).The haematological parameters were carried out as per the method described [9].

Therapeutic management: The infected animal was treated with a single dose of Triquin, a combination of Quinapyramine methyl sulphate and Quinapyramine methyl chloride @ 6 mg/kg body weight via subcutaneous route with other supportive drugs which included dextrose (5%) via intravenous route for 5 days, Tribivet 10ml via intra-muscular route for 7 days, anti-oxidant in form of Vitamin-C (Inj. Ascorvet) 10 ml via intra-muscular route for 7 days and 50 gm mineral mixture daily in feed.

3. Results

Thin blood smear examination revealed the presence of elongate flagellated organism in intercellular space of erythrocytes and identified as *Trypanosoma evansi* as per their morphological features (figure1a and 1b).

Haematological findings are given in Table 1. In the present study, decrease in haemoglobin (6.8gm/dl), total erythrocytic count ($4.5x10^3/\mu l$) was observed as compared to reference value. However, total leucocyte count, mean corpuscular haemoglobin, mean corpuscular volume and mean corpuscular haemoglobin concentration were recorded within normal range. The differential leucocyte count revealed the increase in neutrophils (46%) and decrease in monocytes (1%) while eosinophil, lymphocyte and basophil counts were recorded within normal range. The haematological variations clearly indicated that the animal was suffering fromnormocytic normochromic anaemia. The blood profile also showed lymphocytopaenia with neutrophilia pointing towards a degenerative shift to left (Table 1).

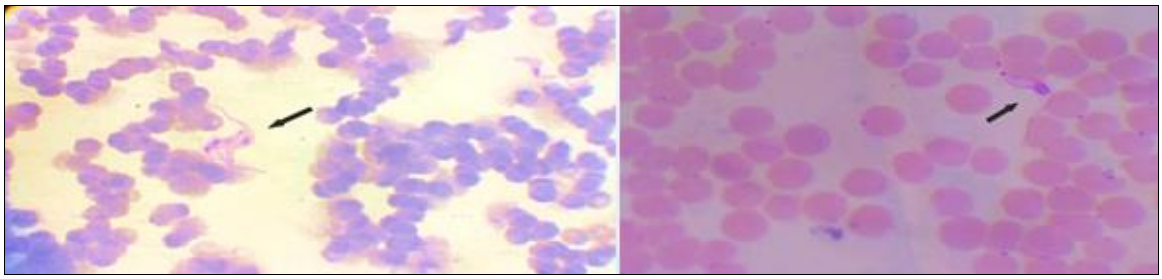

Fig 1ab: showing the presence of *Trypanosoma evansi* in peripheral thin blood smear examination (1000x)

Table 1: Haematological findings of infected buffalo

S. No.	Haematology	Infected Buffalo	Reference range [20]
1.	Haemoglobin (g/dl)	6.8	8-15
2.	Packed Cell Volume (%)	21.2%	26-46
3.	Total erythrocyte count (x $10^6/\mu l$)	4.50	5.0-10.0
4.	Total Leucocyte Count (x $10^3/\mu l$)	7.9	4.0-12.0
5.	Differential Leucocyte Count (%)		
	Neutrophils	46	25-30
	Lymphocyte	51	60-65
	Basophils	0	0-1
	Eosinophils	2	2-5
	Monocytes	1	5-7
6.	Mean Corpuscular Haemoglobin Concentration (gm %)	32.1	30-36
7.	Mean Corpuscular Volume (fl)	47.1	40-60
8.	Mean Corpuscular Haemoglobin (pg)	15.1	11-17

The animal showed considerable recovery after therapy. The gradual and progressive decrease in the nervous signs like circling movements and head pressing were recorded after 5days post treatment. The clinical signs completely disappeared after a week following treatment indicating a complete recovery. A peripheral thin blood smear examination was again conducted after a period of one week for the presence or absence of the haemoprotozoan parasite. The smear was found to be negative for the presence of *Trypanosoma evansi*. Hence, it clearly proves that Triquin is a very effective drug against Surra.

4. Discussion

In the present study, the buffalo was found positive with *T. evansi* responsible for nervous form of the diseases which is inconsonance with the findings of other workers [4]. The organisms utilise glucose and oxygen in the host and lead to various haematological and inflammatory changes in the host body [19]. The mechanism of development of anaemia in trypanosomosis is quite complex and multifactorial in origin [11]. The most common factor for the development of anaemia is erythrocytic injury caused by lashing of trypanosome flagella; erythrocytic haemolysis due to haemolysins produced by trypanosomes, lipid peroxidation and increased erythrophagocytosis caused by mononuclear phagocytic system [10, 13, 14]. Inadequate energy supply to erythrocytes may cause alteration in erythrocytes membrane surface leading to weakening of the cell membrane, increased osmotic

fragility and haemolysis [8]. Erythrocyte membrane damage has also been associated with adhesion of erythrocytes, platelets and reticulocytes to sialic acid receptors of trypanosome surfaces [16]. There are, mainly, 3 phases of anaemia development in trypanosomosis *viz.*, phase of acute crises, chronic phase and phase of recovery [1]. Phase of acute crises begins with initial appearance of trypanosomes in peripheral circulation. In this phase, macrocytic normochromic anaemia is seen. However, in chronic phase, normocytic and normochromic type of anaemia is observed [12]. The neurological symptoms may be attributed to the release of toxins or various immunological reactions [6]. In order to counter the adverse effects caused by the parasite, trypanocidal or trypanostatic drug along-with supportive therapy is required. The quinapyramine prosalt (a combination of quinapyramine methyl sulphate and quinapyramine chloride) is both therapeutic and prophylactic drug for the treatment of Surra in cattle, buffaloes, horses and camels. The subcutaneous injection of quinapyramine prosalt leads to the formation of depot at the site of injection and provides protection for around 3 months [17].

5. Conclusion

Cerebral trypanosomosis is a highly fatal disease condition. Hence, considering the findings of the present study it is essential to take suitable measures to counter prevention and treatment of the infected animal as early as possible otherwise it may lead to huge economic losses in terms of death of the infected animals. These findings also suggest that cerebral trypanosomosis can be managed through early diagnosis and appropriate therapeutic management.

6. Author's declaration: Authors declared no conflicts of interest.

7. References

1. Anosa VO. Haematological and biochemical changes in human and animal trypanosomosis, Parts I & II, Revue d' Elevage et de Medicine Ve'te'rinaire des pays Tropicaux 1988;41(2):65-78.
2. Bharadwaj RK, Randhawa CS. Chronic trypanosomiasis in crossbred cattle. Indian Veterinary Journal 2010;87:408.
3. Bhatia BB, Pathak KML, Banerjee DP. Text book of Veterinary Parasitology 2004, 431.
4. Chandratre GA, Rath AR, Jakhar KK. Pathogenesis of nervous form of trypanosomosis in buffalo: A case study, Journal of Entomology and Zoology Study 2019;7(3):88-91.
5. Da Silva AS, Wolkmer P, Costa MM, Tonin AA, Eilers TL, Gressler LT *et al*. Biochemical changes in cats infected with *Trypanosoma evansi.* Veterinary parasitology, 2010; 171(1):48-52.
6. Da Silva CB, Wolkmer P, Paim FC, Da Silva AS, Siqueira LC, De Souza CL *et al*. Iron metabolism and its relationship to anemia and immune system in *Trypanosoma evansi* infected rats. Experimental Parasitology 2013;133(3):357-364.
7. Desquesnes M, Dargantes A, Lai DH, Lun ZR, Holzmuller P, Jittapalapong S. *Trypanosoma evansi* and surra: a review and perspectives on transmission, epidemiology and control, impact, and zoonotic aspects. Biomed Research International 2013;2013:321237. DOI: 10.1155/2013/321237.
8. Jennings FW. The anaemia of parasitic infections, Proceedings of the 7th International Conference of the World Association for the Advancement of Veterinary Parasitology, Thessalonica, Greece 1975, 15-19, 41-67.
9. Jain NC. Schalm's Veterinary Hematology (No. Edition 4). Lea & Febiger 1986.
10. Igbokwe IO, Nwosu CO. Lack of correlation of anaemia with splenomegaly and hepatomegaly in *Trypanosoma brucei* and *Trypanosomacongolense* infections of rats. J Comp Pathol 1997;117:261-265.
11. Naessens J, Kitani H, Yagi Y, Sekikawa K, Iraqqi F. TNF-α mediates the development of anaemia in a murine *Trypanosoma brucei rhodesiense* infection, but not the anaemia associated with a murine *T. congolense* infection, Clinical and Experimental Immunology 2005;139(3):403-410.
12. Maxie MG, Losos GJ. Release of *Trypanosoma vivax* from the microcirculation of cattle by Berenil®. Veterinary Parasitology 1979;3:277-281.
13. Morrison WI, Murray M, Sayer PD, Preston JM. The pathogenesis of experimentally induced Trypanosoma brucei infection in the dog, Tissue and organ damage. American Journal of Pathology 1981;102:168-181.
14. Murray M, Morrison WI. Parasitaemia and host susceptibility in African trypanosomiasis, Proceedings of a workshop, Nairobi, Kenya, 1978,20-23,71-81.
15. Ranjithkumar M, Saravanan BC, Yadav SC, Kumar R, Singh R, Dey S. Neurological trypanosomiasis in quinapyramine sulfate-treated horses-a breach of the blood–brain barrier? Tropical Animal Health and Production 2014;46(2):371-377.
16. Shehu SA, Ibrahim NDG, Esievo KAN, Mohammed G. Role of erythrocyte surface sialic acid inducing anaemia in Savannah Brown bucks experimentally infected with *Trypanosoma evansi*. Veterenarski Archiv, 2006;26(6):521-530.
17. Singh V, Raisinghani PM, Lodha KR, Manohar GS. Trypanocidal value of a drug in experimental Trypanosoma evansi infection in camels. Indian Vet. J 1987;64:467-470.
18. Singla LD, Aulakh GS, Juyal PD, Singh J. Bovine trypanosomosis in Punjab, India. In Animal health: a breakpoint in economic development? The 11th International Conference of the Association of Institutions for Tropical Veterinary Medicine and 16th Veterinary Association Malaysia Congress, August Petaling Jaya, Malaysia, 2004, 23-27, 283-285.
19. Sivajothi S, Reddy BS, Kumari KN, Rayulu VC. Haematological changes in *Trypanosoma evansi* infected cattle. International Journal of Scientific World 2014;2(1):27-30.
20. Reece WO, Swenson MJ. The composition and functions of blood. In: Dukes' Physiology of Domestic Animals, 12th edition, pp 122. Cornell University Press, Ithaca, NY. Reproduced with permission from Cornell University Press 2004.