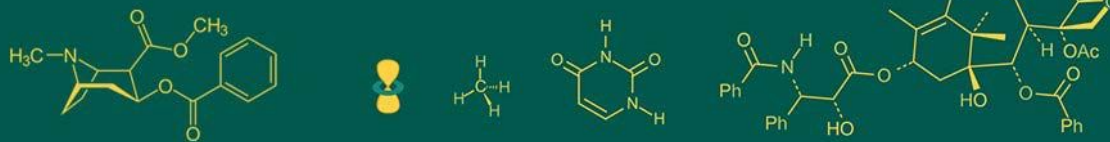


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
 ISSN Online: 2617-4707
 NAAS Rating: 5.29
 IJABR 2025; SP-9(3): 494-497
www.biochemjournal.com
 Received: 15-01-2025
 Accepted: 19-02-2025

Shalini AS
 Wildlife Research and Training
 Centre, Opp. Hindustan Lever
 Godown Factory Square,
 Mahurzari Road, Fetri,
 Nagpur, Maharashtra, India

SM Kolangath
 Wildlife Research and Training
 Centre, Opp. Hindustan Lever
 Godown Factory Square,
 Mahurzari Road, Fetri,
 Nagpur, Maharashtra, India

Dhoot VM
 Wildlife Research and Training
 Centre, Opp. Hindustan Lever
 Godown Factory Square,
 Mahurzari Road, Fetri,
 Nagpur, Maharashtra, India

SV Upadhye
 Wildlife Research and Training
 Centre, Opp. Hindustan Lever
 Godown Factory Square,
 Mahurzari Road, Fetri,
 Nagpur, Maharashtra, India

MD Pawshe
 Wildlife Research and Training
 Centre, Opp. Hindustan Lever
 Godown Factory Square,
 Mahurzari Road, Fetri,
 Nagpur, Maharashtra, India

BK Bhadane
 Wildlife Research and Training
 Centre, Opp. Hindustan Lever
 Godown Factory Square,
 Mahurzari Road, Fetri,
 Nagpur, Maharashtra, India

Corresponding Author:
SV Upadhye
 Wildlife Research and Training
 Centre, Opp. Hindustan Lever
 Godown Factory Square,
 Mahurzari Road, Fetri,
 Nagpur, Maharashtra, India

Successful management of *Trypanosoma* spp. in a Royal Bengal tigress (*Panthera tigris tigris*)

Shalini AS, SM Kolangath, Dhoot VM, SV Upadhye, MD Pawshe and BK Bhadane

DOI: <https://www.doi.org/10.33545/26174693.2025.v9.i3Sg.4036>

Abstract

A female Tigress, approximately three years old, was rescued from C.N. 1168, Halda beat, Brahmapuri Division after being trapped in a man-animal conflict. She was rehabilitated and placed in a quarantine cell at the Gorewada Rescue Centre for further care and management. During the quarantine period, the Tigress exhibited lethargy, loose feces, and frequent yawning. Clinical examination revealed a high-grade fever of 104.5°F, hyperemia of the vaginal and anal mucous membranes, and mild rectal bleeding. Hematological analysis indicated a low platelet count of $71 \times 10^3/\mu\text{l}$, moderately low hemoglobin levels at 9.4 g/dl, and macrocytic, normochromic anemia. Serum biochemistry showed elevated globulin levels of 4.7 g/dl, mildly low sodium levels, and low glucose levels at 25 mg/dl. Blood smear examination was positive for *Trypanosoma* spp. The tigress was treated with 10% Dextrose intravenously, followed by Triquin at 4.4 mg/kg body weight subcutaneously. The tigress made an uneventful recovery.

Keywords: Tigress, Trypanosomiasis, loose faeces, pyrexia, Triquin

Introduction

Natural infections of *T. evansi* have been reported in 48 countries, including 20 in Asia, 17 in Africa, seven in South America, and four in Europe, with no reports in North America, Australia, or Antarctica. Since surra was classified as an OIE notifiable disease in 2009, 27 countries have confirmed its presence at least once (Aregawi *et al.*, 2019) [2]. Surra originated in Africa, where *T. evansi* evolved from *T. brucei brucei* through partial or complete loss of kinetoplastic DNA (Lun *et al.*, 1995; Lai *et al.*, 2008) [18, 17]. In Asia, numerous outbreaks linked to high morbidity and mortality have been recorded in Timor rusa deer (*Rusa timorensis*) and hog deer (*Axis porcinus*). Clinical cases and fatalities due to surra have also been observed in endangered species, including the Asian black bear (*Ursus thibetanus*), Asian elephant (*Elephas maximus*), leopard (*Panthera pardus*), tigers (*Panthera tigris*), jaguar (*Panthera onca*), and Sumatran rhinoceros (*Dicerorhinus sumatrensis*) (More *et al.*, 2017) [19]. An outbreak of *T. evansi* in circus tigers was reported by Bhaskararao *et al.* (1995) [4], following the feeding of infected meat. The prevalence of *T. evansi* in wild ruminants and the potential for oral transmission to wild carnivores, particularly endangered species, constitutes a significant threat (Herrera *et al.*, 2011) [11].

Tigers are unique animals that play a critical role in maintaining ecosystem balance. According to IUCN, approximately 3,900 tigers remain in the wild, with 2,967 found in India—accounting for about 80% of the global tiger population. Their habitat is restricted to a few national parks and zoological gardens. Various health-related issues hinder population maintenance, emphasizing the need to understand diseases affecting tiger health (Deka *et al.*, 2021) [7].

Trypanosomiasis (surra), caused by *T. evansi*, is a significant disease, transmitted mechanically through the bites of blood-sucking flies, including tabanids (Tabanus, Haematopota, Haematobia). The rainy and post-rainy seasons are particularly conducive to the development of tabanid larvae, increasing disease propagation during these times. However, cases of surra can occur year-round. Despite its wide distribution, current information on the prevalence and economic impact of feline trypanosomiasis in India remains limited.

Recently, it has emerged in non-endemic areas, infecting new hosts and raising concerns about its zoonotic potential, including occasional human infections (Joshi *et al.*, 2005; Powar *et al.*, 2006) [12, 24].

The spillover of parasites from wildlife to domestic livestock and humans, and vice versa, compromises health (Thompson, 2013) [28]. While most trypanosome infections in wildlife do not cause significant harm to their hosts (Auty, 2012) [3]. However, there is a high risk of infection spillover from game parks and conservation areas where parasites and vectors are concentrated in high numbers, and domestic livestock pose risks to wildlife-protected species. The foundation of trypanotolerance in wildlife species remains inadequately comprehended. The extensive genetic variety of *Trypanosoma* spp. poses a difficulty by heightening the likelihood of enhanced virulence (Kasozi *et al.*, 2021) [15].

Clinical Observation and Treatment

A 3-year-old female tigress exhibited signs of lethargy, persistent yawning, and loose feces during the monsoon season. She was placed in a treatment cage for further evaluation, which indicated a high-grade fever of 104.5°F, hyperemia of the vaginal and anal mucous membranes, and mild rectal bleeding. Blood samples were collected via venipuncture of the coccygeal vein. The results of hematology and serum biochemistry are detailed in Table No. 1. A blood smear examination revealed the presence of trypomastigotes, confirming a diagnosis of trypanosomiasis. The tigress received treatment with 10% Dextrose, followed by subcutaneous administration of Triquin (Quinapyramine sulphate and Quinapyramine chloride) at a dosage of 4.4 mg/kg body weight, along with supportive therapy including Caripill (2 tablets), Raricap (2 tablets), Becozyme C Forte (2 tablets), Silybon (2 tablets), Zincovit tablets (2 tablets), and Rinifol tablets (2 tablets) orally once a day for 15 days. The animal subsequently made a full recovery.

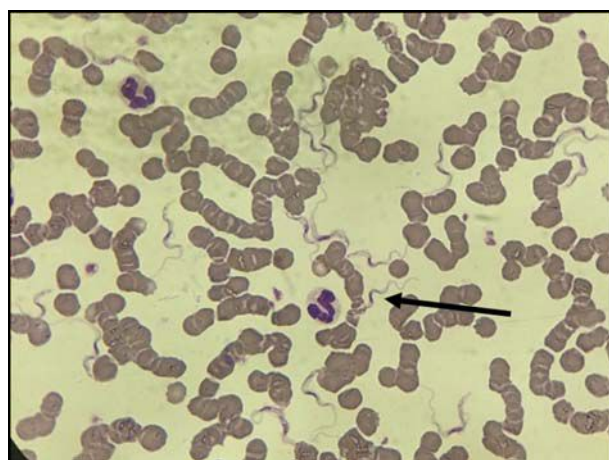


Fig 1: Blood smear examination showing trypomastigotes in a female Tiger

Discussion

Felids in their natural environments typically traverse large territories, leading to lower parasitic exposure and, consequently, lower genetic resistance to such infections (Raja *et al.*, 2014) [25]. In captivity, especially in zoological settings, the prevalence of parasitic infections can increase significantly, creating a serious threat (Muoria *et al.*, 2005)

[21]. Parasitic infections not only contribute to morbidity in these animals but can also lead to fatal outcomes in severe cases (Chhabra and Pathak, 2013) [5]. *Trypanosoma evansi* has diverse origins, host species, and clinical manifestations. *T. evansi* was reported natural infections in four Himalayan charming bears, in Pakistan; the animals exhibited high fever, anaemia, high pulse and respiration rate, tachypnoea, ataxia and depression (Muhammad *et al.*, 2007) [20]. Elephants are affected the same as camel. Affected elephants show pyrexia, anaemia, loss of appetite, oedema of the face, neck, brisket, trunk, limbs and lower abdomen, dry and hard skin, restlessness, reluctance to work, dullness, listlessness and sleepy appearance. Urine is scanty and progressively becomes viscous and sometimes marked greenish in colour. Swelling between the jaws and the lower part of the abdomen is often seen (Desquesnes *et al.*, 2013) [8]. Clinical signs in affected tiger intermittent temperature (102-105°F), anorexia, lacrimation and pale mucous membrane, enlargement of prescapular lymph node, nervous signs like seizures, uncontrolled movements in the body and sometimes a transitory change in consciousness (Khan *et al.*, 2015) [16]. In the present case, the tiger showed signs of lethargy, loose faeces and high fever.

Its transmission occurs through various means, such as biting and sucking insects, vampire bats, as well as vertical, horizontal, iatrogenic, and oral routes, with different epidemiological significance based on season, location, and host species. Leeches may also transmit trypanosomes, a factor worth examining, particularly in relation to the buffalo leech (*Hirudinaria manillensis*) in Asia (Desquesnes *et al.*, 2013) [8]. Blood glucose level of donkeys with surra fluctuates with initial transient hyperglycaemia and later persistent hypoglycaemia that extends to chronic stage of *T. evansi* infection. Acute injury to pancreas might have reduced insulin activity to control blood glucose level (Sun *et al.*, 2016) [27]. Host glucose reserve is increasingly utilized and depleted by host animal body cells in febrile condition (Habla *et al.*, 2012) [10] blood stream form trypanosome, trypomastigote and in presence of hepatocytes degeneration in trypanosomiasis due to increased demand for metabolism resulting in hypoglycemia (Garba and Mayaki, 2018) [9]. Quinapyramine, part of the amino quinaldine derivatives, can be administered as quinapyramine methyl-sulphate through subcutaneous injection at a dosage of 5 mg/kg body weight to treat infections. A more effective combination, quinapyramine sulfate and quinapyramine chloride (Triquin), can be used as a therapeutic and preventive measure against *T. evansi* in horses and camel. While local tolerance may be low, the drug is generally effective, offering chemoprophylactic effects for up to four months (Desquesnes *et al.*, 2013) [8].

Control primarily relies on chemotherapy, yet this approach presents challenges, including high costs and limited drug availability. Diminazene and quinapyramine are the primary agents used in India, with most trials focusing on these two. Other effective drugs face supply issues, like suramin, and cymelarsan has yet to receive commercial approval. Singh and Joshi (1991) [26] evaluated diminazene, suramin, quinapyramine, and isometamidium in naturally infected buffaloes, finding both quinapyramine (4.4 mg/kg body weight) and isometamidium (0.5 mg/kg body weight) effective therapeutically, while quinapyramine proved superior for prophylaxis. A subsequent study (Joshi and Singh, 2000) [13] confirmed all four drugs were therapeutically effective against clinical *T. evansi* in

buffaloes, but their prophylactic efficacy varied: quinapyramine (66.6%), suramin (66.6%), isometamidium (33.3%), and diminazene (0%). Improvements in haemoglobin, PCV, and TEC values, along with significant increases in blood glucose, total serum protein, and cholesterol, were noted post-treatment (Joshi and Singh 2001) [14]. In camels, quinapyramine pro-salt proved effective as both a therapeutic and prophylactic agent against natural *T. evansi* infections (Pathak *et al.*, 1997a) [23]. A case in Andhra reported successful treatment of a goat infected with *T. evansi* using quinapyramine chloride (Rao and Hafeez, 1999) [4]. De and Mukherjee (2006) [6] successfully managed trypanosomiasis in a 2-year-old male horse with quinapyramine sulfate. Muraleedharan (2015) [22] noted that diminazene diaceturate and Anttrycide curative are common treatments for trypanosomiasis in livestock and tigers. Ananda *et al.* (2014) [1] reported clinical cures in trypanosome-positive cases treated with Triquin along with supportive therapy.

Table 1: Haematological Alterations in Tigress Before and After Treatment

Haematology				
Tiger GT-35 (H2-SAF-1) (Female)				
Values	Pre-Treatment	Post-Treatment		
	Day 1	Day 2	Day 7	Reference Value
WBC (10 ³ /μl)	6.71	29.79	13.93	5.5-19.5
LYM (10 ³ /μl)	0.71	2.22	2.32	1.5-7.0
MON (10 ³ /μl)	0.37	0.22	0.64	0-1.5
NEU (10 ³ /μl)	5.34	26.78	10.68	2.5-14.5
EOS (10 ³ /μl)	0.29	0.56	0.28	0-1
BAS (10 ³ /μl)	0.00	0.01	0.02	0-0.2
LYM (%)	10.5	7.4	16.6	18-35
MON (%)	5.5	0.7	4.9	2-6
NEU (%)	79.6	89.9	76.6	57-75
EOS (%)	4.3	1.9	2.0	2-6
BAS (%)	0.1	0.0	0.1	0-4
RBC (10 ⁶ /μl)	6.13	5.60	6.45	5-10
HGB g/dl	9.4	8.9	11.0	9.8-15.4
HCT (%)	35.77	31.40	37.09	30-45
MCV (fl)	58	56	58	39-55
MCH (pg)	15.3	15.9	17.1	13-17
MCHC (g/dl)	26.2	28.4	29.7	30-36
PLT (10 ³ /μl)	71	58	268	300-800

Table 2: Serum Biochemical Alterations in Tigress Before and After Treatment

Serum Biochemistry			
Tiger GT-35 (H2-SAF-1) (Female)			
Value	Pre-Treatment	Post-Treatment	Reference
	Day 1	Day 7	range
BUN (mg/dL)	40	43	10-30
CRE (mg/dL)	2.1	1.8	0.3-2.1
ALT (U/L)	42	34	20-100
ALP (U/L)	8	22	10-90
AST (U/L)	191	54	12-43
TBIL (mg/dL)	0.4	0.5	0.1-0.6
GLU (mg/dL)	25	91	70-150
CA (mg/dL)	9.6	9.9	8.0-11.8
TP (g/dL)	7.1	7.8	5.4-8.2
ALB (g/dL)	2.4	2.6	2.2-4.4
GLOB (g/dL)	4.7	5.2	1.5-5.7
NA ⁺ (mmol/L)	137	146	142-164
K ⁺ (mmol/L)	4.1	5.1	3.7-5.8
CL ⁻ (mmol/L)	107	113	99-122
tCO ₂ (mmol/L)	11	16	15-24

Conclusions

Early diagnosis and prompt treatment are crucial for managing *Trypanosoma* infections in wild felids. Quinapyramine-based therapy, combined with supportive care, proved effective in this case. Regular health monitoring of rescued carnivores is essential to prevent fatal disease progression. Further research on vector control and disease prevention strategies is necessary to mitigate future outbreaks.

Acknowledgements

The authors are thankful to Hon'ble PCCF (WL) and Chief Wildlife Warden, Maharashtra State, the FDCM Pvt. Ltd and Divisional Manager, Gorewada Project, Nagpur for granting permission to manage the Trypanosomiasis in a Female Tiger at Wildlife Research and Training Centre, Nagpur and also for providing the facilities required for its successful management.

References

- Ananda KJ, Ganganaik S, Kavitha Rani B. Epidemiological studies on haemoprotozoan diseases in bovines in and around Shimoga: A malnad region of Karnataka. Indian Vet J. 2014 Oct;91(10):29-31.
- Aregawi WG, Agga GE, Abdi RD, Buscher P. Systematic review and meta-analysis on the global distribution, host range, and prevalence of *Trypanosoma evansi*. Parasites Vectors. 2019 Jan;12:67.
- Auty H, Anderson NE, Picozzi K, Lembo T, Mubanga J, Hoare R. Trypanosome diversity in wildlife species from the Serengeti and Luangwa Valley ecosystems. PLoS Negl Trop Dis. 2012 Oct;6:e1828. doi: 10.1371/journal.pntd.0001828.
- Bhaskararao T, Raju PB, Das JH, Hafeez M. Some observations on an outbreak of surra in circus tigers. Indian Vet J. 1995;72:1210-21.
- Chhabra MB, Pathak KML. Parasites and parasitic diseases of wildlife in India. 2. Carnivores and birds. Indian J Anim Sci. 2013 Jun;83(6):567-78.
- De UK, Mukherjee R. Trypanosomiasis in equine and its management-A case report. Indian Vet J. 2006;83:72.
- Deka A, Goswami S, Hazorika M, Devi P, Baishya BC, Barman U, Dutta TC. A case report of trypanosomiasis in a tiger. J Entomol Zool Stud. 2021 Feb;9(1):991-992.
- Desquesnes M, Dargantes A, Lai D, Lun ZR, Holzmüller P, Jittapalapong S. *Trypanosoma evansi* and Surra: A review and perspectives on transmission, epidemiology and control, impact, and zoonotic aspects. Biomed Res Int. 2013 Sep;2013:321237. doi: 10.1155/2013/321237.
- Garba UM, Mayaki AM. Fluctuations in blood glucose levels of donkeys infected with *Trypanosoma evansi*. ARC J Anim Vet Sci. 2018 Dec;4(1):14-21.
- Habila N, Inuwa MH, Aimola IA, Udeh MU, Haruna E. Pathogenic mechanisms of *Trypanosoma evansi* infection. Res Vet Sci. 2012 Aug;93(1):13-7.
- Herrera HM, Rocha FL, Lisboa CV, Rademaker V, Mourão GM, Ansen AM. Food web connections and the transmission cycles of *Trypanosoma cruzi* and *Trypanosoma evansi* (Kinetoplastida, Trypanosomatidae) in the Pantanal Region, Brazil. Trans R Soc Trop Med Hyg. 2011 Jul;105(7):380-7.

12. Joshi PP, Shegokar VR, Powar RM. Human trypanosomiasis caused by *Trypanosoma evansi* in India: the first case report. *Am J Trop Med Hyg*. 2005 Sep;73(3):491-5.
13. Joshi SS, Singh B. Evaluation of therapeutic and chemoprophylactic efficacy of certain drugs against clinical surra in buffaloes. *Indian Vet J*. 2000;77:895-7.
14. Joshi SS, Singh B. Clinicopathology and biochemical alterations in clinical cases of *T. evansi* infection in buffaloes and effect of treatment thereon. *Indian Vet J*. 2001;78:642-644.
15. Kasozi KI, Zirintunda G, Ssempijja F, Buyinza B, Alzahrani KJ, Matama K, *et al*. Epidemiology of trypanosomiasis in wildlife—Implications for humans at the wildlife interface in Africa. *Front Vet Sci*. 2021 Jun;8:621699.
16. Khan BN, Ali Z, Yasmeen R, Bibi F, Ziaulah, Mehboob N. *Trypanosoma* infestations in Royal Bengal tiger (*Panthera tigris tigris*) at Lahore Zoological Gardens and its therapy. *J Anim Plant Sci*. 2015 Jan;25(3 Suppl 2):477-482.
17. Lai DH, Hashimi H, Lun ZR, Ayala FJ, Lukes J. Adaptations of *Trypanosoma brucei* to gradual loss of kinetoplast DNA: *Trypanosoma equiperdum* and *Trypanosoma evansi* are petite mutants of *T. brucei*. *Proc Natl Acad Sci USA*. 2008 Feb;105(6):1999-2004.
18. Lun ZR, Desser SS. Is the broad range of hosts and geographical distribution of *Trypanosoma evansi* attributable to the loss of maxicircle kinetoplast DNA? *Parasitol Today*. 1995;11(4):131-133.
19. More S, Bøtner A, Butterworth A, Calistri P. Assessment of listing and categorization of animal diseases within the framework of the Animal Health Law (Regulation (EU) No 2016/429): *Trypanosoma evansi* infections (including surra). *EFSA J*. 2017 Jul;15:1-34.
20. Muhammad G, Saqib M, Sajid MS, Naureen A. *Trypanosoma evansi* infections in Himalayan black bears (*Selenarctos thibetanus*). *J Zoo Wildl Med*. 2007 Mar;38(1):97-100.
21. Muoria PK, Muruthi P, Rubenstein D, Ogue NO, Munene E. Cross-sectional survey of gastrointestinal parasites of Grevy's zebras in southern Samburu, Kenya. *Afr J Ecol*. 2006 Jan;43(4):392-5.
22. Muraleedharan K. An update on *Trypanosoma* infection in livestock of Karnataka State, India. *Vet Res Int*. 2015 Jul-Sep;3(3):46-54.
23. Pathak KML, Kapoor M, Shukla RC. Evaluation of therapeutic and prophylactic efficacy of Quinapyramine methyl sulphate-chloride in camels naturally infected with *Trypanosoma evansi*. *Indian J Anim Sci*. 1997;67(2):132-133.
24. Powar RM, Shegokar VR, Joshi PP, *et al*. A rare case of human trypanosomiasis caused by *Trypanosoma evansi*. *Indian J Med Microbiol*. 2006 Jan;24(1):72-74.
25. Raja MMRU, Dey AR, Begum N, Kundu UK, Ashad FA. Coprological prevalence of gastrointestinal parasites in carnivores and small mammals at Dhaka Zoo, Bangladesh. *J Threat Taxa*. 2014 Mar;6(3):5574-5579. doi: 10.11609/JoTT.o3569.5574-9.
26. Singh B, Joshi SJ. Epidemiology, clinicopathology and treatment of clinical *Trypanosoma evansi* in buffalo (*Bubalus bubalis*). *Indian Vet J*. 1991;68:975-979.
27. Sun Q, Nie S, Wang L, Yang F, Meng Z, Xiao H, *et al*. Factors that affect pancreatic islet cell autophagy in adult rats: Evaluation of a calorie-restricted diet and a high-fat diet. *PLoS ONE*. 2016 Mar;11(3):e0151104. doi: 10.1371/journal.pone.0151104.
28. Thompson RCA. Parasite zoonoses and wildlife: one health, spillover, and human activity. *Int J Parasitol*. 2013 Jun;43(12):1079-1088. doi: 10.1016/j.ijpara.2013.06.007.
29. World Health Organization. Tropical disease research: progress 1975-1994, highlights 1993-94, twelfth programme report of the UNDP/World Bank/WHO Special Programme for Research and Training in Tropical Diseases (TDR). Geneva: World Health Organization; 1995. Available from: WHO TDR Publications.