

## CASE REPORT

# Bilateral Ocular Affection Associated with *Trypanosoma evansi* Infection in German Shepherd Dog: A Case Report

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**T**rypanosomosis, an arthropod-borne infectious disease caused by a flagellate protozoan parasite, *Trypanosoma evansi*, poses significant economic challenges, particularly in the tropical regions such as India (Jaiswal *et al.*, 2015). The disease affects bovines, canines, felines, and camelids. It is primarily transmitted mechanically by the biting haematophagous flies, with outbreaks becoming more frequent during the rainy season due to increased vector population (Jaiswal *et al.*, 2015). Initially restricted to wild animals, over the time, disease has spread to the domestic animals (Sudan *et al.*, 2017). Clinically, trypanosomosis presents with symptoms such as anorexia, emaciation, intermittent fever, progressive anaemia, conjunctivitis, limb edema, and superficial lymphadenopathy (Pandey *et al.*, 2015). Neurological manifestations, like delirium, head pressing, ataxia, rear limb paralysis, and tonic-clonic seizures, have also been reported (Jaiswal *et al.*, 2015; Sudan *et al.*, 2017). Ocular involvement in canine trypanosomosis is rare, with limited literature available on the subject (Behera *et al.*, 2017; Abbas *et al.*, 2021). During disease development the parasites travel down from brain through optic nerve and causes deposition of immune complex in aquas humour which clinically results into development of corneal edema and opacity (Reddy and Sivajothi, 2017). This case report presents a clinical case of *T. evansi* induced ocular manifestations in a German Shepherd dog and its therapeutic management with diminazene aceturate in conjunction with supportive therapy.

### CASE HISTORY AND OBSERVATIONS

A two-and-a-half-year-old, 24.5 kg female German Shepherd (Reg. No. D-04-698) was brought to the Veterinary Clinical Complex of College of Veterinary Sciences, Rampura Phul, Bathinda, Punjab (India), with a history of inappetence, voice change, polydipsia, progressive weight loss, fever, bilateral corneal opacity for the past one month (Fig. 1), and vision loss for the past five days. Clinical examination revealed a fever of 104.5°F, pale mucous membranes, femoral and axillary lymphadenopathy, absence of menace reflex, and hyporeflexia in the gluteal muscles. Additionally, the dog exhibited tachycardia (148 beats/min), tachypnoea (52 breaths/min), and reduced pulse intensity. Dermatological assessment showed

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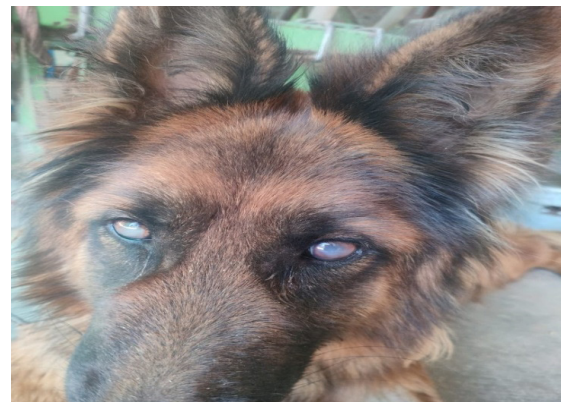
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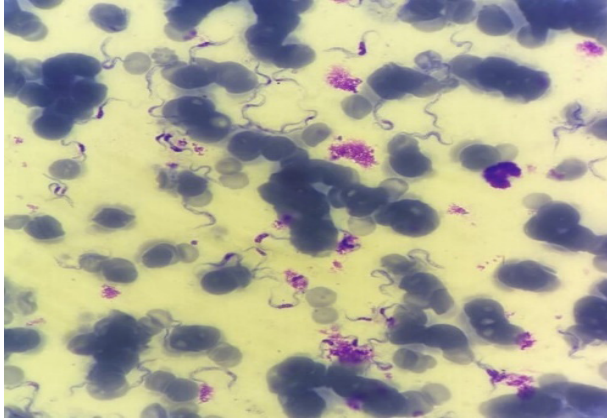
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no presence of ticks or fleas, and the hair coat appeared normal. Based on history and clinical examination the case was suspected for haemoprotozoan infection and accordingly wet blood film examination was performed. Furthermore, whole blood sample was collected in duplicate with and without EDTA for parasitological and haemato-biochemical analysis.



**Fig. 1:** Bilateral corneal opacity in *T. evansi* infected dog

Microscopic examination of Geimsa-stained peripheral blood smear revealed presence of extracellular flagellated organisms with undulating membrane and sub-terminal kinetoplast. Based on morphological characteristics, the organism was identified as *Trypanosoma evansi* (Fig. 2). The results of haemato-biochemical analysis revealed presence of anaemia, thrombocytopenia and hypoproteinemia along with mild hepatic damage (Table 1).



**Fig. 2:** Blood smear showing *Trypanosoma evansi* (Oil-immersion)

## TREATMENT AND DISCUSSION

The treatment regimen included diminazene aceturate (3.5 mg/kg body weight, intramuscular, with two doses given at weekly intervals) and a broad-spectrum antibiotic with beta lactamase inhibitor (Ceftriaxone + Sulbactam @ 25 mg/kg body weight, intramuscular for three days) was implemented to avoid secondary bacterial infection, if any. Supportive therapy comprised parenteral administration of isotonic fluids (DNS and Ringer's lactate each at 20 mL/kg body weight), and intramuscular inj. of Pheniramine maleate (1 mg/kg b.wt.), Meloxicam (0.2 mg/kg b.wt.), and Vitamin B<sub>12</sub> (1 mg/kg b.wt.) for three days. Additionally, a hepatoprotectant (°Hepamust @ 5 mL twice daily orally for 15 days), a platelet enhancer (°Imu Lat @ 10 mL once daily orally for 10 days), and a hematinic (°Hemobest Per 10 mL twice daily orally for three days) were also administered. For the ophthalmic

condition, herbal eye drops (4 drops twice daily for 7 days) and fresh extract of human placenta (gel for local application) were prescribed. After ten days post-therapy, dog started showing ocular improvement and fully recovered 21 days post-therapy (Fig. 3).



**Fig. 3:** Resolution of corneal opacity after therapy

Dogs are highly susceptible to *T. evansi* infection and exhibit various clinical signs such as intermittent fever, edema of the head and larynx, swelling of the abdominal wall and legs, anaemia, lymphadenopathy, tachycardia, weakness, muscle spasms, emaciation, hindquarter paralysis, and myocarditis (Jaiswal *et al.*, 2015). Hepatic damage (Varshney *et al.*, 1998) and jaundice (Baby *et al.*, 2000) have also been reported due to this infection in dogs. Additionally, ocular symptoms including conjunctivitis, lacrimation, keratitis, and often bilateral corneal opacity have also been reported (Panigrahi *et al.*, 2015; Behera *et al.*, 2017). Haemato-biochemical changes observed in affected dog like hypoglycemia, hypoalbuminemia, hyperglobinaemia, and hyperkalaemia have also been recorded previously (Sarvanan *et al.*, 2005). Amongst various drugs used for the treatment of infection, diminazene aceturate has shown the most promising results, particularly at a dosage of 3.5-5.0 mg/kg b.wt. administered in multiple doses at weekly intervals (Panigrahi *et al.*, 2015).

**Table 1:** Haemato-biochemical parameters in affected dog

Parameters	Pre-treatment values	Post-treatment values	*Reference value	Interpretation
RBC count (millions/mm <sup>3</sup> )	3.38	3.23	5-10	Anemia
Haemoglobin (g/dL)	6.2	6.3	8-15	
WBC count (10 <sup>3</sup> cells/mm <sup>3</sup> )	8600	12000	4-12	NA
Neutrophils (%)	74	72	15-33	Neutrophilia
Lymphocytes (%)	26	23	45-75	Lymphopenia
Platelets (×10 <sup>9</sup> /mm <sup>3</sup> )	67000	1,20,000	211-621	Thrombocytopenia
SGPT (IU/L)	152	53	10-109	Hepatic damage
Total Protein (g/dL)	8.4	8.1	5.4-7.5	Hyperproteinemia
Albumin (g/dL)	2.5	3.2	2.3-3.1	NA
BUN (mg/dL)	14.2	7.4	8-28	
Creatinine (mg/dL)	0.46	0.42		Normal renal function

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