THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
Jan, 2025 Vol.5(1), 6053−6056

Popular Article

Bovine Trypanosomiasis: Pathogenesis, Diagnosis, and Vector Control Strategies

Pratistha Shrivastava[1], Manaswini Dehuri[2]
Assistant Professor, Department of Parasitology,
Institute of Veterinary Science & Animal Husbandry, SOA, Bhubaneswar-751029, India
Assistant Professor, Department of Veterinary Parasitology,
Odisha University of Agriculture and Technology, Bhubaneswar-751003, India
DOI:10.5281/ScienceWorld.14755939

Abstract

Trypanosomosis is transmitted by blood sucking arthropod vectors like tabanid flies and is distributed mostly in Asia, Africa and America with a wide host range. India serves as a significant source for the spread of *T. evansi* across Asia and nearby islands, with a higher incidence reported in northern and northwestern regions compared to the eastern and southern parts of the country. Antigenic variation leads to immune exhaustion and immunosuppression, which heightens vulnerability to secondary infections. Diagnosis of the disease typically relies on the presence of biting fly vectors, clinical symptoms, and laboratory tests such as direct examination of blood and body fluids, chemical tests, animal inoculation, and immunodiagnostic methods. Despite over 125 years of research, accurate diagnosis still faces challenges due to low sensitivity and specificity, with conventional parasitological techniques being the primary method.

Introduction

Trypanosomosis (Surra), caused by *Trypanosoma evansi*, is a major global veterinary issue. It leads to severe economic losses due to its impact on livestock health, including emaciation, anemia, fever, reduced productivity, abortions, and high mortality rates (20-90%). The disease is endemic in the Indian subcontinent, particularly affecting cattle and buffaloes. Humans are typically resistant, except in rare cases of genetic mutation. India is notable for the discovery of the first mammalian trypanosomes, with *T. evansi* being the commonest species in the region. T. evansi is a monomorphic, leaf-shaped parasite with dimensions ranging from 18–34 µm in length and 1.5–2.5 µm in width. It features a subterminal kinetoplast, a prominent undulating membrane, a centrally positioned vesicular nucleus, and a long anterior free flagellum measuring 5–6 µm.The disease is commonly referred to as "Surra" (meaning rotten or emaciation) in Hindi and is widely recognized by this name in many countries. Other regional names include "Dubla" (emaciated), "Purana" (chronic), and "Tibursa"

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Published 25/01/2025

(three-year disease). *T. evansi* is transmitted mechanically via blood-feeding Dipteran flies (e.g., *Tabanus, Stomoxys, Haematopota*). Fly activity increases transmission during the monsoon and post-monsoon seasons. The parasite does not develop inside the vector, and transmission efficiency depends on fly feeding intervals and intensity. Predisposing factors include host age, stress, inter-current infections, starvation, and animal movement between infected and healthy areas.

Distribution: Enzootic trypanosomosis caused by *T. evansi* covers a geographical area three times larger than tsetse-borne trypanosomes. It is endemic in India, China, Southeast Asia, North Africa, the Middle East, South America, and parts of the former USSR. India is a major source of *T. evansi* spread in Asia and surrounding islands. Incidence is higher in northern and northwestern India compared to eastern and southern regions.

Pathogenesis: The disease progresses through three stages:

Pyrexial stage shows intermittent fever, anaemic stage shows significant reduction in erythrocyte count (>25%) and in nervous stage Trypanosomes cross the blood-brain barrier, affecting the CNS.

Mechanism of Pathology:

Utilization of nutrients, excretion of toxic metabolites, tissue disruption, and immune-mediated injury (Host Damage). Anaemia caused by haemolysis, erythrophagocytosis, and immune complex-associated cyto-toxic reactions. Proteolytic enzymes and toxins lead to hypoglycemia, intravascular coagulation, and systemic effects.

Immune Response: Antigenic variation causes immune exhaustion, IgM hypergammaglobulinemia, and immunosuppression, increasing susceptibility to secondary infections.

Clinical and Cellular Changes:

Macrocytic anaemia, leucopenia/leucocytosis, thrombocytopenia, and clotting disturbances are seen. Lesions are found in connective tissues, blood, and lymphatic systems. Symptoms include oedema, urticaria, lymphadenopathy, keratitis, and fluid accumulation. Elevated serum enzymes (e.g., ALT, AST), reduced alkaline phosphatase, and hypoglycemia leading to liver dysfunction.

Fatal Outcomes: Hypoglycemia and liver failure, endotoxin release from lysed parasites, asphyxia due to lactic acid-induced oxygen absorption interference or lung capillary blockage by trypanosomes, CNS impairment caused by tryptophan metabolism byproducts.

Potential Therapeutic Target: E/S proteases and other hydrolytic enzymes released by trypanosomes may degrade host tissues. Immune targeting of these enzymes could mitigate disease progression.

Clinical Signs

Sudden onset of nervous signs (convulsions, ataxia, blindness, circling) and rapid death. Neurological symptoms may mimic anthrax or brain disorders. Cause of death often involves cerebral vessel occlusion and anoxia are observed in per-acute form, dullness, staggering gait, fever, circling,

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and death within 6–12 hours are observed in acute form, intermittent fever, depression, reduced milk production, conjunctivitis with white discharge, swollen pre-scapular lymph nodes, anaemia, weight loss, abortion, and fetlock joint knuckling are observed in sub-acute form, weakness, emaciation, anaemia, corneal opacity, and production losses are seen in chronic form.

Immunity: Trypanosomes evade the host immune system through antigenic variation, releasing sequential sets of antigens to escape antibody-mediated responses, immune evasion.

Immune Modulation: Levamisole improves immunity by stimulating hypergammaglobulinaemia, increased Ig production, leucocytosis, and lymphocytosis and Dexamethasone exacerbates the infection.

Immunopathology: Impaired blood-brain barrier (BBB) and increased intra-cerebral immunoglobulin biosynthesis are observed in infected cattle.

Overview of Diagnostic Methods

a) **Parasite Detection Techniques:** Direct smear method, Wet Blood Examination (effective for motile *T. evansi),* Mouse Sub-Inoculation, Micro Hematocrit Technique, Quantitative Buffy Coat (Rapid field method for asymptomatic carriers), Miniature Anion Exchange Centrifugation Technique (Extremely sensitive for detecting low parasite levels).
b) **Chemical tests:** Mercuric Chloride Test, Thymol Turbidity Test, Jone's Nitric Acid Test.
c) **Immunodiagnostic Techniques:**
d) **Antibody Detection**: ELISA, IFAT (Indirect Fluorescent Antibody Test), Card Agglutination Test (CATT).
e) **Antigen Detection**: ELISA-Based Antigen Detection, Latex Agglutination Tests (e.g., Surratex).
f) **Nucleic Acid Detection Techniques:** DNA Probes, PCR (Polymerase Chain Reaction), PCR-ELISA.

Treatment, control, and prevention

Control primarily relies on chemotherapy and chemoprophylaxis. However, challenges include drug resistance, high costs, limited availability of effective drugs, and potential toxicity.

Curative Drugs

Diminazene aceturate (Berenil): 3.5–5.0 mg/kg via deep intramuscular injection, Isometamedium (Samorin): 0.5–2.0 mg/kg intramuscular injection, Cymalarsan: Effective for the nervous form of surra; crosses the blood-brain barrier. Dosage: 0.75 mg/kg intramuscular injection, Suramines (Antrypol, Nagapol, Gilpol): 12 mg/kg (10% solution) intravenously.

Prophylactic Drugs: Used in high-risk scenarios or when access to animals is difficult.

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Quinapyramine sulphate/chloride combination (e.g., Antrycide prosalt): 3.0–5.0 mg/kg subcutaneously.

Vaccine development has been unsuccessful due to the ability of trypanosomes to frequently change surface antigens, rendering previous antibodies ineffective.

Vector Control: Insecticides: Synthetic pyrethroids are preferred due to their effectiveness and reduced environmental impact. Organochlorines and organophosphates may also be used. Use of traps or insecticide-impregnated targets (screens) are the alternative methods.

Reference

Desquesnes, M., Dargantes, A., Lai, D.-H., Lun, Z.-R., Holzmuller, P., & Jittapalapong, S. (2013). *Trypanosoma evansi and Surra: A Review and Perspectives on Transmission, Epidemiology and Control, Impact, and Zoonotic Aspects. BioMed Research International, 2013, 1–20.*

Gill, B. S. (1991). Trypanosomes and trypanosomiases in Indian livestock. Indian Council of Agricultural Research, New Delhi, pp. 191.

Jayabal, L., Chaudhri, S.S., Singh, A. and Kumar, D. (2003). *Detection of circulating antigens in immune complexes of Trypanosoma evansi infected cattle and buffaloes by ELISA*. J. Vet. Parasitol., 17: 85-88.

Shrivastava P, Dehuri M, Mohanty B, Mishra C, Venkatesh KM, Biswal SS.(2022). *Molecular characterization and prevalence of bovine hemoprotozoan and rickettsial organism from Bhubaneswar, Eastern India*. Animal Biotechnology, 34(7):2917-2927.

Singh V and Singla L.D. (2013). Veterinary Parasitology in Indian Perspective. 1st Edition, Satish Serial Publisher, New Delhi, pp. 227-302.