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Popular Article

From Fly to Fatal: Decoding the Complex Life Cycle of *Equine Trypanosomiasis*

Bhawana Narnoliya[1], A. A. Vagh[1], Arpit Gupta[2], Atokali H. Yeptho[3]
[1]Department of Veterinary Medicine, College of Veterinary Science and Animal Husbandry, Junagadh, Kamdhenu University, Gujarat, India.
[2]Department of Veterinary Pharmacology, college of Veterinary Science, U.P. Pt. Deen Dayal Upadhyaya Pashu Chikitsa Vigyan Vishwa Vidyalaya
[3]Department of Veterinary Microbiology, College of Veterinary Science and Animal Husbandry, Junagadh, Kamdhenu University, Gujarat, India.
Corresponding author: bhawananarnoliya1999@gmail.com
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Abstract

Trypanosomiasis, commonly referred to as Surra and Dourine, is a major hemoprotozoan disease of horses, caused primarily by *Trypanosoma evansi*. The infection is transmitted mechanically by hematophagous flies, mainly *Tabanus* and *Stomoxys* species, and occurs predominantly in tropical and subtropical regions. Equine trypanosomiasis is characterized by intermittent fever, progressive anemia, weight loss, generalized weakness, edema of dependent parts (limbs, genitalia, and abdomen), urticarial plaques, petechial hemorrhages on mucous membranes, and neurological disturbances such as incoordination and lethargy in advanced cases The disease may also cause abortion or infertility in mares and reduced performance in working horses. Laboratory diagnosis involves detection of the parasite in stained blood smears, concentration techniques such as microhematocrit centrifugation, and molecular assays like PCR for confirmation. Serological tests like CATT/T. evansi are also employed for field screening. The infection induces marked anemia and leukopenia due to hemolysis and bone marrow suppression. Preventive measures include vector control, regular screening, and chemoprophylaxis in endemic zones.

Key words: - *Trypanosoma evansi*, *Tabanus* & *Stomoxys*, *Equine trypanosomiasis* (Surra), Anemia, vector control

INTRODUCTION

Trypanosomiasis is a parasitic disease caused by flagellated protozoa of the genus *Trypanosoma*, affecting both humans and a wide range of domestic and wild animals. It is one of the most economically important and widely distributed vector-borne diseases in tropical and subtropical regions of the world. The parasites are transmitted mainly through the bites of

blood-sucking flies such as *Tsetse* (in Africa), *Tabanus*, and *Stomoxys* (in Asia and Latin America), or through sexual contact in certain species. The epidemiology of *Trypanosoma evansi* infection in equines is formed by a combination of environmental, geographical, and management factors. These factors play a crucial role in influencing the occurrence, spread, and transmission dynamics of the parasite within equine populations. In animals, trypanosomiasis is commonly referred to as **Surra**, **Dourine**, or **Nagana**, depending on the causative species and geographical occurrence. In horses and other equines, the disease is most often caused by *Trypanosoma evansi* (Surra) and *Trypanosoma equiperdum* (Dourine). These infections lead to a range of clinical signs such as fever, anemia, edema, weakness, loss of body condition, and neurological disturbances.

FORM OF DISEASE

Surra has two forms: an **acute type** that mainly affects horses, dogs, and camels, and a **chronic type** mostly observed in cattle and water buffalo.

Acute form

In the acute stage of infection, affected animals typically exhibit a high fever ranging from 41.5°C to 44°C, accompanied by weakness, lethargy, anemia, marked weight loss, abortion, and disturbances in movement often linked to nervous involvement. Without treatment, death may occur within 2 weeks to 8 months.

Chronic form

The chronic form is characterized by recurrent fever spikes corresponding to peaks in parasitemia. Other common findings include edema, particularly in the lower limbs, urticarial skin plaques, and petechial hemorrhages on mucous membranes. Chronic infections may persist for up to two years, leading to progressive debilitation and poor body condition.

TRANSMISSION

Trypanosoma evansi is transmitted mechanically by the bites of hematophagous flies, mainly *Tabanus* (horse flies) and *Stomoxys* (stable flies). These flies carry the parasite in their mouthparts after feeding on infected animals and transfer it to healthy ones during subsequent bites. Because this transmission does not involve parasite multiplication in the vector, Surra can spread rapidly in areas with dense fly populations.

PATHOGENESIS

1. **Entry and Early Dissemination**

   Transmission occurs through the **bite of blood-sucking flies** such as *Tabanus* and *Stomoxys*, which mechanically inoculate *Trypanosoma evansi* into the bloodstream during

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interrupted feeding. The **trypomastigotes swiftly invade the blood and lymphatic systems**, spreading throughout the body.

2. **Hemolymphatic Phase (Acute Systemic Stage)**

   During this phase, parasites multiply rapidly in the bloodstream, leading to **high parasitemia**. The **immune system mounts an intense response**, activating macrophages, neutrophils, and complement, accompanied by a surge of cytokines that induce **high fever.** To evade host defenses, the parasites undergo **antigenic variation** by altering their Variable Surface Glycoproteins (VSGs), resulting in **recurrent waves of parasitemia**.

3. **Vascular and Immune-Mediated Tissue Injury**

Parasite toxins and immune complexes trigger **endothelial damage**, increasing vascular permeability. This leads to dependent edema, particularly in the limbs, ventral abdomen, scrotum, and sheath. **Petechial and mucosal hemorrhages** develop due to platelet dysfunction and fragile capillaries. The skin may show **urticarial plaques or wheals**, a hallmark of hypersensitivity and vascular leakage.

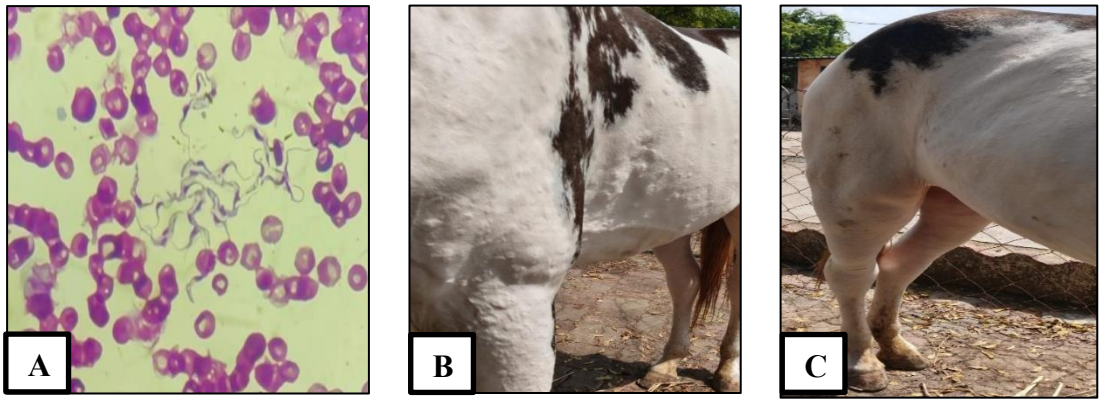

Fig. A The small, elongated, purple-stained bodies (**trypomastigotes**) are clearly visible swimming among the circular **red blood cells**. This visual confirms the presence of **parasitemia**, where high numbers of the infectious agents are circulating in the blood. **Fig. B** In severe cases, the skin may even show **urticarial plaques or "hives,"** which are a hallmark of the body's **hypersensitivity and inflammatory reaction** to the circulating parasites and their toxins. **Fig B & C** Both images show the **lower limbs and ventral (under) abdomen** of the infected horse. This is **edema**, caused by inflammation and vascular leakage. The damaged capillaries and blood vessels allow fluid to seep out into the surrounding tissues, causing the characteristic, often pitting, swellings or "wheels."

Diagnosis

Accurate diagnosis is vital for controlling trypanosomiasis. A combination of **clinical**, **parasitological**, **serological**, and **molecular** methods is used.

1. **Clinical Examination:** Fever (Intermittent or Remittent), edema of ventral parts of the body (lower chest, abdomen, sheath/udder, and legs). This is largely due to hypoproteinemia, skin Plaques (Urticarial Lesions), weight loss (cachexia) despite a good

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appetite, weakness, lethargy, ocular signs and sometimes neurological symptoms (due to CNS involvement) are seen as the disease progresses to the chronic stage.

2. Microscopic Detection

Microscopy is the gold standard for rapid confirmation, particularly in the acute phase, but its sensitivity is limited in chronic, low-parasitemia infections (**fig A)**.

3. Serological Tests (Antibody Detection)

Serology detects the horse's immune response to the parasite, making it effective for chronic cases and large-scale screening where parasites may not be present in the blood at all times.

- **CATT/ *T. evansi* (Card Agglutination Test)**

  This is a **rapid, simple, and inexpensive** test that can be performed in the field without extensive laboratory equipment.

  - **Mechanism:** It uses stained, freeze-dried *T. evansi* antigen which, when mixed with serum from an infected animal, will clump together (**agglutinate**) due to the presence of specific antibodies. It's excellent for large-scale **surveillance and control programs**.
- **ELISA (Enzyme-Linked Immunosorbent Assay) and IFA (Indirect Fluorescent Antibody Test):** These are high-throughput, sensitive, and specific lab tests used for **large-scale screening** and **international trade certification**.
  - **Mechanism:** They measure the levels of specific anti-*trypanosomal* antibodies in the horse's serum, indicating current or past exposure. They are particularly valuable for identifying **asymptomatic carriers**.

4. Molecular Diagnosis (PCR)

Molecular methods offer the highest level of sensitivity and specificity by detecting the parasite's unique genetic material.

- **Polymerase Chain Reaction (PCR):**
  - **High Sensitivity & Specificity:** PCR can amplify a tiny, unique segment of the parasite's **DNA** to detectable levels. This means it can confirm infection even when there are extremely low levels of parasitemia.

5. Hematological Findings (Blood Profile)

- **Marked Anemia: Normocytic, normochromic anemia** (normal cell size and color) caused by a combination of:
- **Leukopenia (Low White Blood Cells):** Often observed in the acute phase, reflecting immune system suppression or exhaustion.

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TREATMENT AND CONTROL

Early treatment is essential to prevent chronicity and death. Common drugs include:

- **Diminazene aceturate** (3.5–7 mg/kg body weight, IM) – effective in early infections.
- **Suramin sodium** (5–10 mg/kg IV) – used for more advanced or resistant cases.
- **Quinapyramine salts** (sulfate and chloride combination) – used both therapeutically and prophylactically in endemic areas.

However, the emergence of **drug resistance** and **relapse cases** remain serious challenges, emphasizing the need for accurate dosing and veterinary supervision.

Vector Control

Reducing vector contact is crucial to prevent transmission of *T. evansi*. Measures include:

- Regular **spraying of insecticides** and use of **repellents** on horses.
- **Fly-proof stables** with netting and proper manure disposal.
- Avoiding grazing during peak fly activity (early morning and evening).

PREVENTION AND MANAGEMENT

1. **Regular Screening:** Routine blood examination and serological testing in endemic zones help detect carriers and prevent outbreaks.
2. **Movement Control:** Transport of infected animals should be restricted. Quarantine measures are essential for newly introduced horses.
3. **Good Nutrition and Hygiene:** Proper feeding, rest, and parasite control improve immunity and reduce susceptibility.
4. **Chemoprophylaxis:** In high-risk regions, periodic administration of trypanocidal drugs under veterinary guidance helps protect valuable equines.
5. **Awareness and Training:** Educating horse owners, breeders, and veterinarians about disease signs and transmission routes ensures early reporting and effective control.

CONCLUSION

Equine trypanosomiasis remains a silent yet devastating threat to horse health and rural livelihoods across tropical regions. Understanding its transmission, recognizing early clinical signs, and adopting regular screening and vector control can turn the tide against *T. evansi*. With vigilant management and scientific awareness, Surra need not remain a fatal sentence-it can be a preventable disease.

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