

## Medical parasitology

### Hemoflagellates ( Blood & tissue flagellates )

Family : Trypanosomatidae

Genus : Trypanosoma

Trypanosomes are flagellated protozoa, trypomastigote in shape, with elongated body, central nucleus, posterior kinetoplast, long undulating membrane and free flagellum anterior.

#### Classification

- 1- Polymorphic trypanosomes, has different size & shape in blood  
e.g: T. brucei gambiense
- 2- Monomorphic trypanosomes has the same size & shape in blood  
e.g: T. cruzi.

#### **T. brucei gambiense & rhodesiense**

(**Disease** :African sleeping sickness )

**Transmitted** by Tsetse fly Glossina palpalis male & female

**Distribution:** Tropical west & central Afric

**Habitat:** Blood, lymphatic system & CNS

**Host:** Man

**Reservoir host:** May be cattle, goat, pig & monkeys

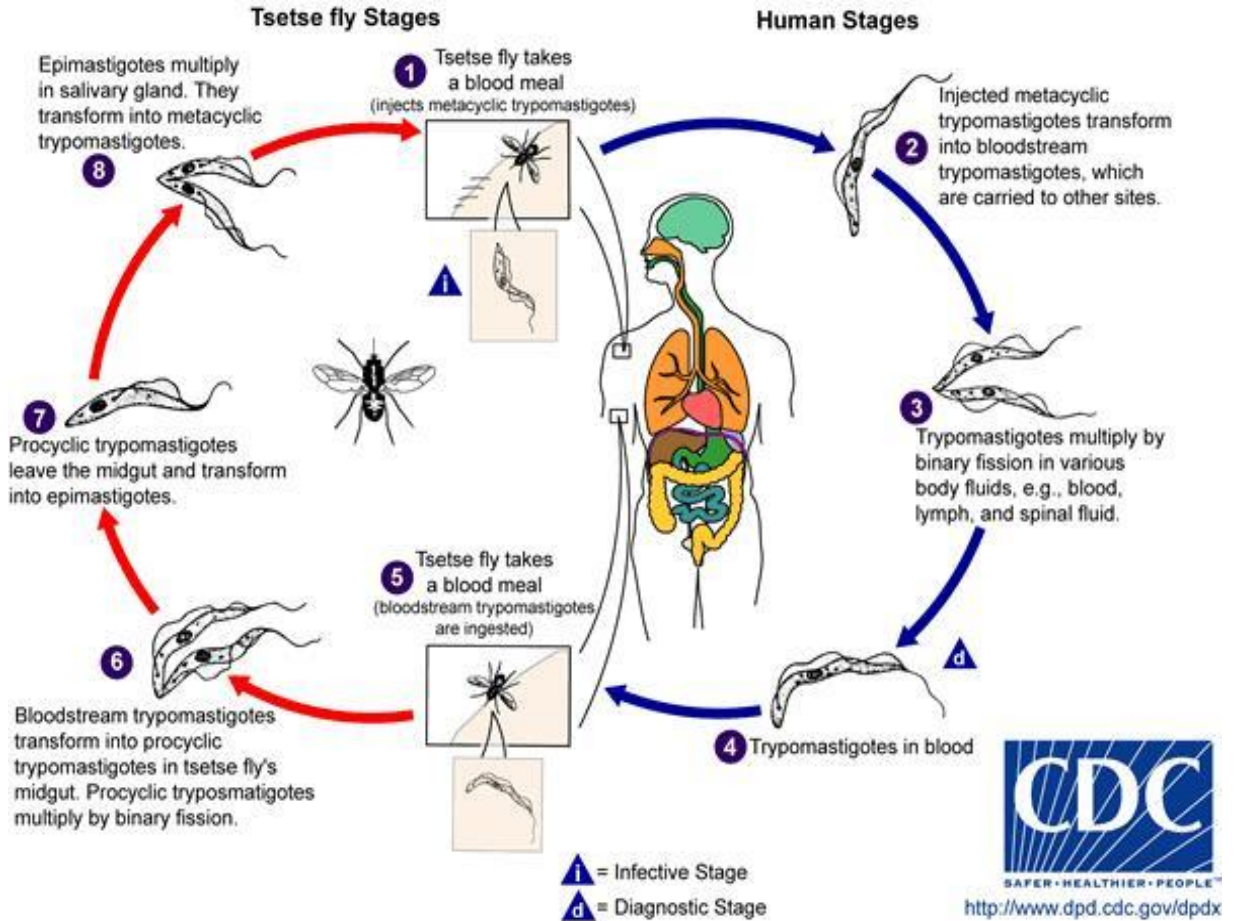
**Infective stage:** Metacyclic trypomastigotes

**Mode of infection:** Bite of infected *Glossina palpalis*

### **Life cycle**

- 1-**Infected tsetse fly bites a host to take a blood meal & inoculates metacyclic trypomastigotes with saliva into the bite wound.
- 2-**The organisms rapidly transform into trypomastigotes and divide by binary fission in the interstitial spaces at the site of the bite punom
- 3-**Parasites spread via blood to lymphatic system and CNS.
- 4-**The tsetse fly takes blood meal from an infected host containing trypanosomes.
- 5-**In the midgut of the fly, trypanosomes multiply by longitudinal binary fission & pass forward to hypo-pharynx where they change to epimastigotes.
- 6-**Parasites migrate to the salivary gland sand change to short stumpy metacyclic trypomastigotes (infectivestage).
- 7-**The cycle inside the vector takes about 3 weeks .

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**Pathogenesis & clinical picture** (Incubation period:1-3 weeks )

**1-Trypanosoma chancre** (skin) Trypomastigotes multiply at the site of bite a local Inflammatory nodule painful , red , rubbery ,may or may not ulcerat

**2-Haemolympathic stage** (lymph node) Trypomastigotes invade blood & lymphatic system & multiply toxic manifestations & lymphocytic hyperplasia.

**3- Patient gets** irregular fever, headache, joint & muscle pain and rash. enlarged liver & spleen, generalized lymphadenopathy especially in posterior triangle of neck

**4-Meningoencephalitis stage** (sleeping sickness stage;brain): Patient suffers of: severe headache, mental apathy, slow speech, tremors, involuntary movements & convulsions. Sleeping stage develops>>> coma & death [from the disease or from intercurrent infections as malaria, dysentery or pneumonia].

## **Diagnosis of African trypanosomiasis**

### **I-Clinical diagnosis**

- \* History (residence or traveling to endemic area).
- \* Clinical picture.

### **II-Laboratory Diagnosis**

- \* Direct
- \* Indirect

#### **Direct methods**

To demonstrate parasite early in aspirate from chancre, blood, lymph node, bone marrow a) Microscopic examination of fresh unstained or Giemsa stained films to demonstrate the polymorphic trypomastigote. b) Culture (NNN media): >>>epimastigote. c) Animal inoculation: Intraperitoneal injection of mice or hamsters

followed by blood examination >>> polymorphic trypomastigotes.

### **Indirect methods [Serologic tests]**

Detecting an increase in total IgM level and, anti-trypanosoma antibodies in serum: e.g. IFAT, IHAT, ELISA

### **Treatment**

1-Suramin (Antrypol): I. V. injection of 1 gram for 5 doses; 1-

Eflornithine: I.V infusion of 100 mg/kg /6 hours for 2 weeks;

followed by 3-4 weeks oral treatment with 75 mg/kg every 6 hours.

### **Trypanosoma cruzi**

**Disease:** A merican trypanosomiasis or Chagas 'disease.

**Distribution:** Central & South America.

**Habitat:** Blood, reticulo-endothelial cells & muscle fibers (heart).

**D.H.:** Man.

**Vector:** Triatoma (reduviid, winged bug).

**Infective stage:** Metacyclic trypomastigotes [in feces of vector, posterior station transmission].

**Mode of transmission** Contamination of bite wound, skin abrasion or mucous membrane by faeces of infected vector; may be transmitted by -blood transfusion, organ transplantation & congenitally. Morphology

1. Two forms in man

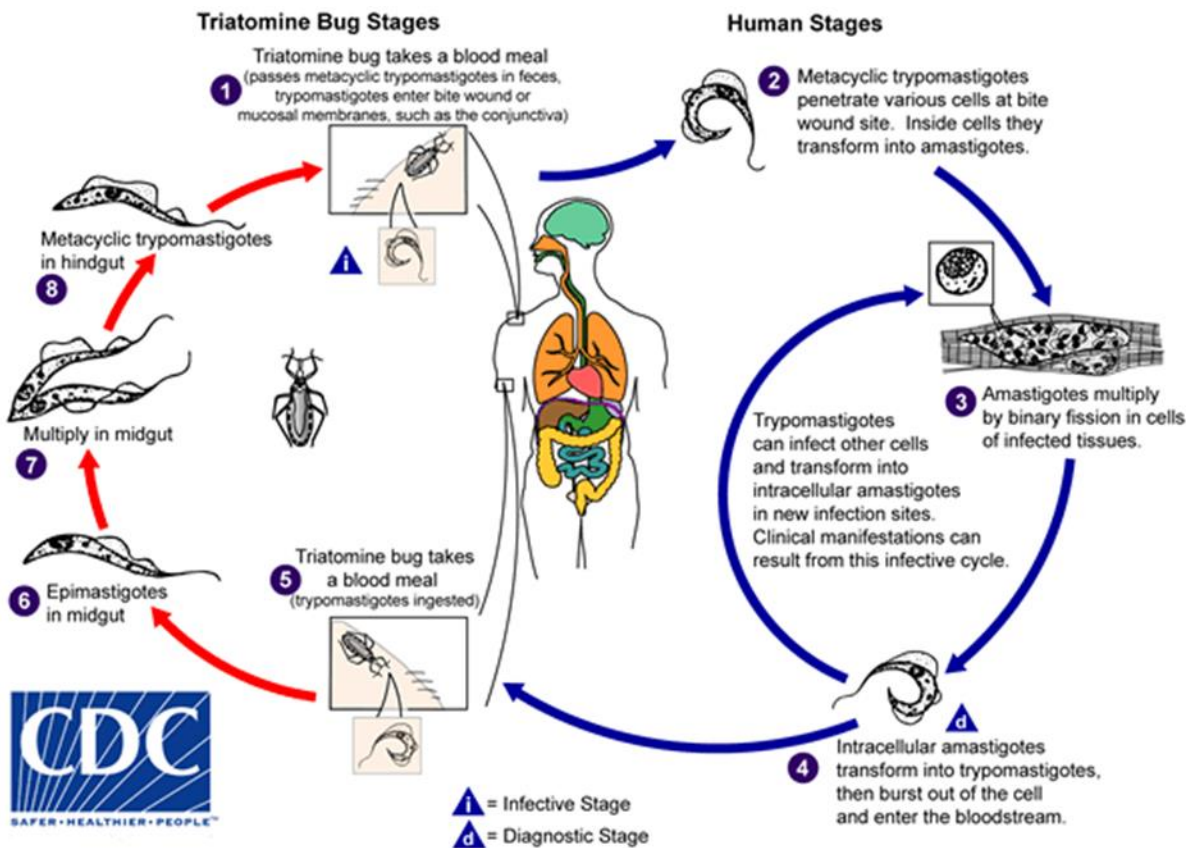
**a-Amastigote (multiplying form) :** intracellular in cardiac muscle & other tissues [smooth muscle cells, cells of brain & macrophage]

**b-Trypomastigote (none multiplying form):** in blood, monomorphic, C or U shape with short undulating membrane

2. Two forms in the vector

**a-Epimastigote** in the midgut.

**b-Metacyclic** or short stumpy trypanosomes (infective stage) in hindgut and stool.



## **Life cycle**

1-Infected winged bug bites a host for a blood meal & defecates on skin.

2-Metacyclic trypomastigotes found in feces enter through the bite wound.

3-Metacyclic forms are taken up by macrophage cells, where they change to amastigotes& multiply asexually by binary fission.

4-Rupture of cells releases the amastigotes that change to C-shaped trypomastigotes in the circulation, infect new host cells or they remain in blood.

5-When a winged bug bites an infected host, it sucks blood containing C-shaped mono-morphic trypomastigotes.

6-In midgut, the parasites change to epimastigotes & pass backward to hindgut where they change to metacyclic trypomastigotes [infective stage].

7-The cycle in vector takes about 10 days.

## **Pathogenesis & clinical picture**

**Acute Chagas' disease** It is more common in infants & children.

## 1-Primary lesion

At the site of bite, parasites multiply inside macrophage cells (erythematous,) (**Chagoma** occurs more on the face.)

2-Romana's sign: When chagoma occur around the eye>>>unilateral conjunctivitis, edema of eye lids & cheek.

3-Parasite reaches regional lymphnode, blood, organs and tissues producing fever, enlarged lymph nodes ,skin rash, enlarged liver and spleen.

4-In severe infections signs of meningoencephalitis and heart failure.

5-Death may occur or patient may recover or pass to chronic stage.

## Chronic Chagas' disease

Parasite attacks:

1-Heart muscle fibers: congestive lead to heart failure.

2-Colon muscle fibers: Megacolon lead to constipation.

3-Less commonly CNS or thyroid gland involvement.

Diagnosis==\* Clinical &\* Laboratory:

1-Blood film >>(C-shaped T.cruzi).



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2-Examination of biopsy from chagoma, lymph node, liver, spleen, bone marrow, or muscle biopsy by culture or animal inoculation.

3-Serological tests

4-Molecular techniques: e.g. IFAT, IHAT, ELISA & PCR.

### **Treatment:**

1-Primaquine orally (destroys trypomastigotes in blood and decreases tissue invasion).

2-To inhibit intracellular development of T.cruzi:

- Nifurtimox (Lampit): 8-10 mg/kg/day for two months. Or
- Benznidazole (Radanil): 5mg/kg/day for two months.

### **Prevention and control**

#### **African sleeping sickness T.gam.**

Treatment of patients Control of vectors (Glossina) Pentamidine as prophylactic drug

#### **Chagas' Disease**

Treatment of patients Control of vectors (Triatoma)

Elimination of reservoir hosts