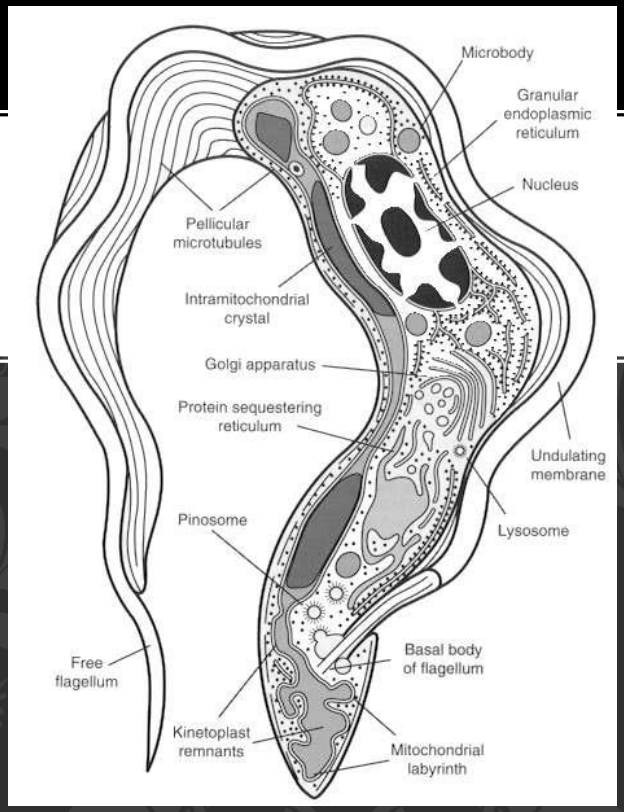
TISSUE PROTOZOA



Dr Sadia Ikram

LEARNING OBJECTIVES

By the end of this lecture students should be able to recall Tissue prtozoa

Toxoplasma gondii

Trypanosoma cruzii

Their life cycles, pathogenesis and clinical features of their diseses.

Should be able to diagnose their clinical casses.

TISSUE PROTOZOA

Toxoplasma gondii

Trypanosoma cruzii

TOXOPLASMOSIS

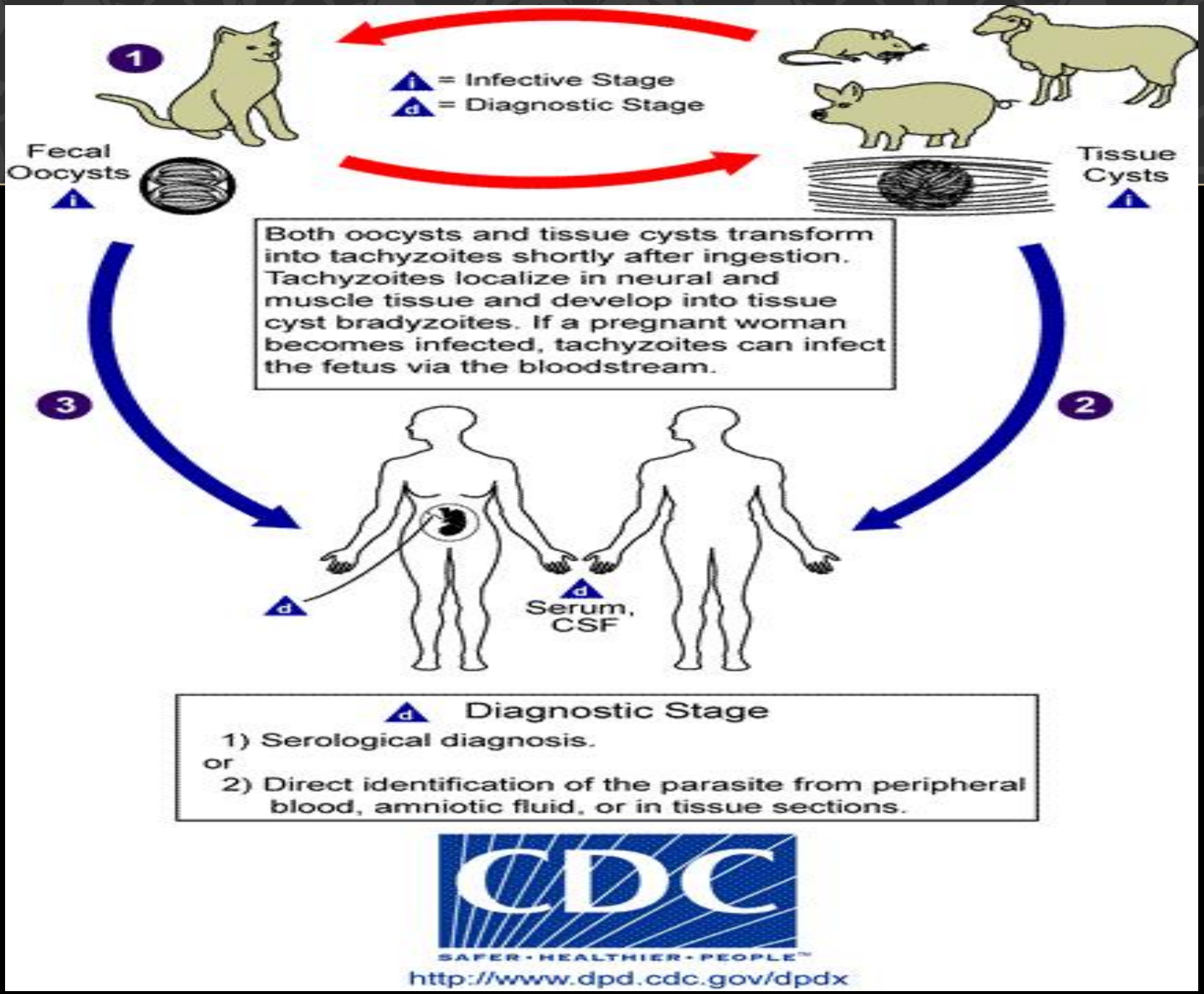
Toxoplasma gondii causes toxoplasmosis.

Congenital toxoplasmosis.

Definitive host: domestic cat.

Intermediate host: Humans & other mammals.

Transmission: Ingestion of cysts in undercooked meat or contact with cat feces.


1
i = Infective Stage
d = Diagnostic Stage
Fecal Oocysts
i
Tissue Cysts
i
Both oocysts and tissue cysts transform into tachyzoites shortly after ingestion. Tachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites. If a pregnant woman becomes infected, tachyzoites can infect the fetus via the bloodstream.
3
2
d
d
Serum, CSF
d Diagnostic Stage
1) Serological diagnosis.
or
2) Direct identification of the parasite from peripheral blood, amniotic fluid, or in tissue sections.
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Life Cycle

Cycle in cat begins with ingestion of cysts in raw meat (mice).

Bradyzoites released from cysts in small intestine, infect mucosal cells & differentiate into male & female gametocytes.

Gametes fuse to form oocysts, excreted in cat feces.

Cycle completed when soil contaminated with cat feces accidentally ingested.

Human infection occurs from eating undercooked meat (lamb and pork), from animals that grazed in soil contaminated with infected cat feces.

↓

In small intestine, cysts rupture & invade gut wall.

Ingested by macrophages and differentiate into **tachyzoites** (which kill cells & infect other cells).

↓

Cell-mediated immunity limits spread of tachyzoites, & parasites enter host cells in brain, muscle & other tissues converted in to **bradyzoites.**

↓

Tissue cysts

EPIDEMIOLOGY

Occurs worldwide.

Outbreaks due to ingestion of raw meat or contaminated water.

1% of domestic cats in United States shed *Toxoplasma* cysts.

PATHOGENESIS

After infection of intestinal epithelium, organisms spread to brain, lungs, liver & eyes.

Mostly asymptomatic.

When contained, organisms persist as cysts within tissues.

No inflammation & individual remains well unless immuno-suppression allows activation of organisms in cysts.

PATHOGENESIS

Congenital infection of fetus:

When mother infected during pregnancy.

If infected before pregnancy: Organism in cyst form & no trophozoites pass through placenta.

One-third of mothers infected during pregnancy give birth to infected infants. (10% of these infants symptomatic).

CLINICAL FINDINGS

Primary infections in immuno-competent adults asymptomatic.

Congenital infection: Abortion, stillbirth, or neonatal disease with encephalitis, chorioretinitis, hepatosplenomegaly, fever, jaundice & intracranial calcifications.

Most infected newborns asymptomatic.

Some develop chorio-retinitis or mental retardation.

One of leading causes of blindness in children.

Reduced cell-mediated immunity (AIDS patients) cause life-threatening disseminated disease, encephalitis.

COMPLICATIONS

Encephalitis.

Hydrocephalus.

Uveitis

Pneumonia

Myocarditis

Abortion

LABORATORY DIAGNOSIS

1. **Microscopic examination:**

- Giemsa-stained preparations shows crescent-shaped trophozoites during acute infections.
- Cysts may be seen in tissue.

2. Culture:

- Can be done in cell culture.

3. Serology:

- Immunofluorescence assay for **IgM antibody**.
- **IgM** for diagnosis of congenital infection & acute infection. (If significant rise in antibody titer in paired sera).
- **ELISA:** For titre of IgM & IgG.

4. PCR: Detects DNA of Toxoplasma.

TREATMENT & PREVENTION

- **Congenital & disseminated toxoplasmosis in immuno-compromised patients:**
- Combination of sulfadiazine & pyrimethamine.

- **Acute toxoplasmosis in immuno-competent:**
- usually self-limited.
- Cooking meat thoroughly kills cysts.
- Pregnant women avoid under cooked meat
- Avoid Contact with cats & refrain from emptying cat litter boxes.
- Not to feed raw meat to cats.

TRYPANOSOMA

T. CRUZI IS THE CAUSE OF CHAGAS DISEASE (AMERICAN TRYPANOSOMIASIS)

Three major species:

Trypanosoma cruzi.

Trypanosoma rhodesiense

Trypanosoma gambiense.

LIFE CYCLE

VECTOR: REDUVIID BUG (CONE-NOSE OR KISSING BUG).
RESERVOIR HOSTS: HUMANS AND ANIMALS (ANIMAL RESERVOIRS INCLUDE DOMESTIC CATS, DOGS, ARMADILLO, RACCOON & RAT).

Cycle in **reduviid bug** begins with entry of trypomastigotes in blood of reservoir host through bite.

↓

form nonflagellated amastigotes within host cells. Myocardial, glial & reticuloendothelial cells most frequent sites.

↓

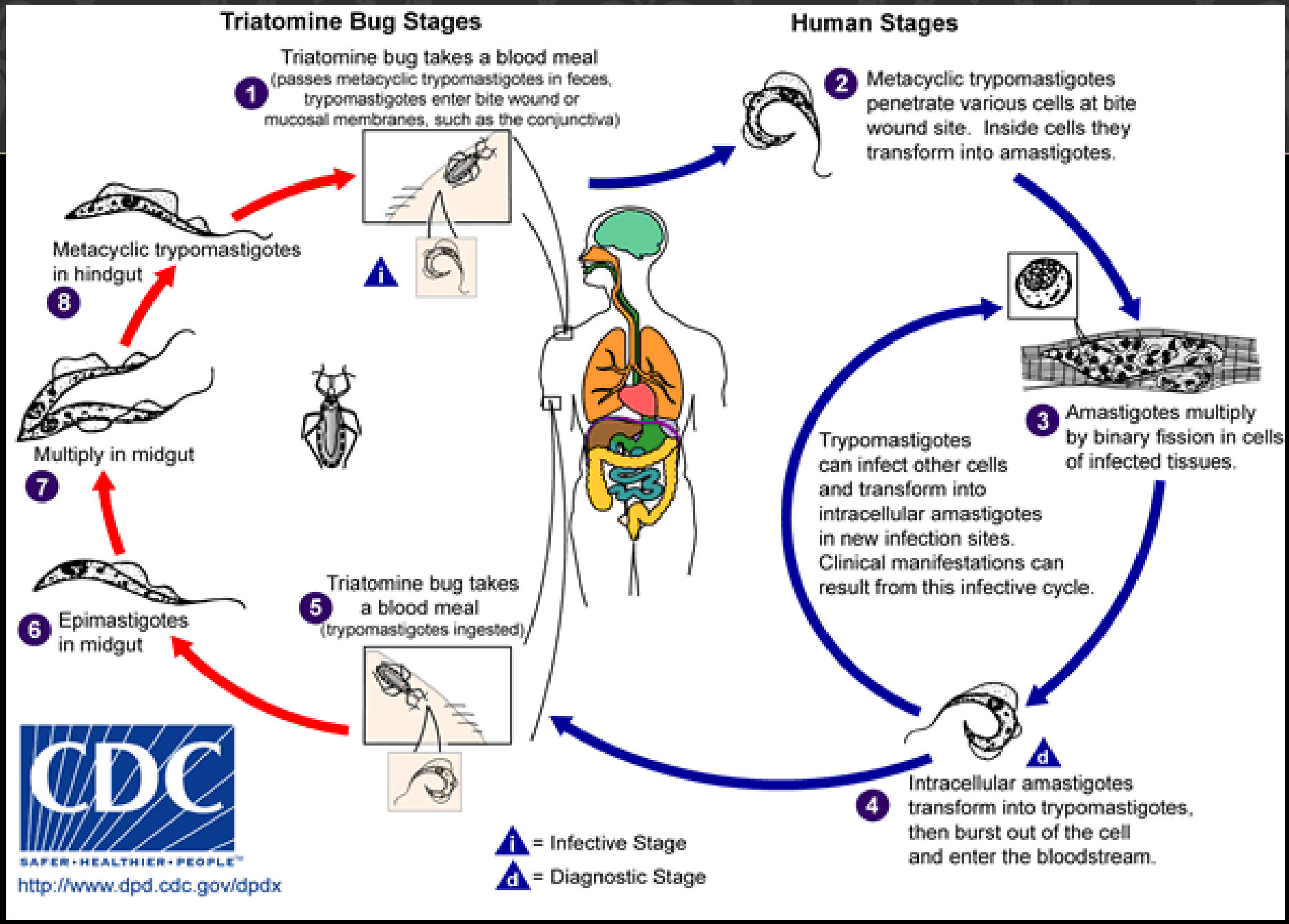
Intracellular amastigotes transform in to trypomastigotes. Cells burst, trypomastigotes released.

↓

Bug takes blood meal, trypomastigotes multiply in mid gut & differentiate into epimastigotes & then into trypomastigotes in hind gut.

↓

Trypomastigotes, enter blood of host when reduviid bug bites.


Triatomine Bug Stages
Human Stages
Triatomine bug takes a blood meal
(passes metacyclic trypomastigotes in feces, trypomastigotes enter bite wound or mucosal membranes, such as the conjunctiva)
1
i
Metacyclic trypomastigotes in hindgut
8
Multiply in midgut
7
Epimastigotes in midgut
6
Triatomine bug takes a blood meal
(trypomastigotes ingested)
5
2
Metacyclic trypomastigotes penetrate various cells at bite wound site. Inside cells they transform into amastigotes.
3
Amastigotes multiply by binary fission in cells of infected tissues.
Trypomastigotes can infect other cells and transform into intracellular amastigotes in new infection sites. Clinical manifestations can result from this infective cycle.
d
4
Intracellular amastigotes transform into trypomastigotes, then burst out of the cell and enter the bloodstream.
i = Infective Stage
d = Diagnostic Stage
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EPIDEMIOLOGY

Occurs primarily in rural Central and South America.

Seen in rural areas as reduviid bug lives in walls of rural huts & feeds at night.

It bites around mouth or eyes. (kissing bug).

CLINICAL FEATURES

Acute phase of Chagas disease:

- Facial edema & nodule (chagoma) near bite and fever, lymphadenopathy & hepatosplenomegaly.
- Bite around eye: Unilateral palpebral swelling (Romaña's sign).
- Acute phase resolves in 2 months.

Chronic form:

- Myocarditis and megacolon.
- Death: Due to cardiac arrhythmias & failure.

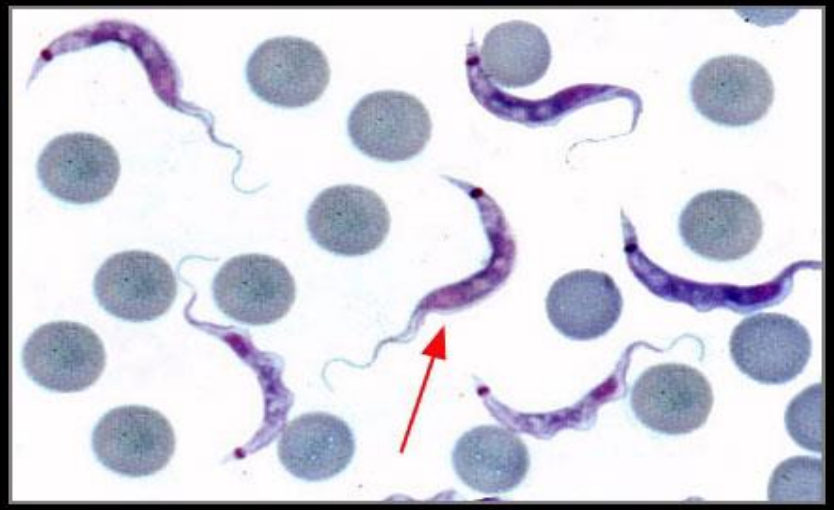
Complications:

- Cardiomyopathy, Megacolon, Degeneration of autonomic nervous system.

LABORATORY DIAGNOSIS

1. **Microscopy:**

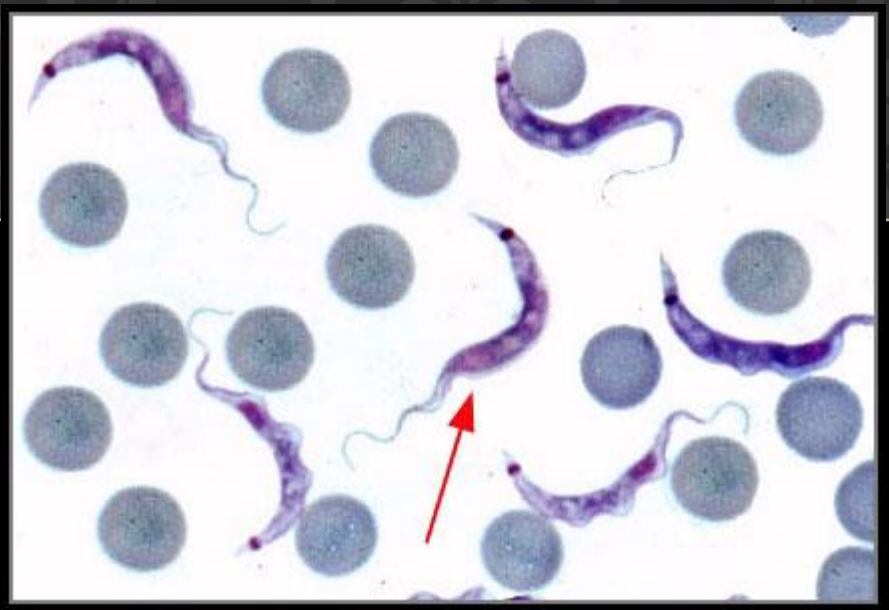
- Trypomastigotes in thick or thin films of patient's blood.
- Stained and wet preparations showing motile organisms.
- Stained preparation of a bone marrow aspirate or muscle biopsy specimen (Reveal amastigotes)

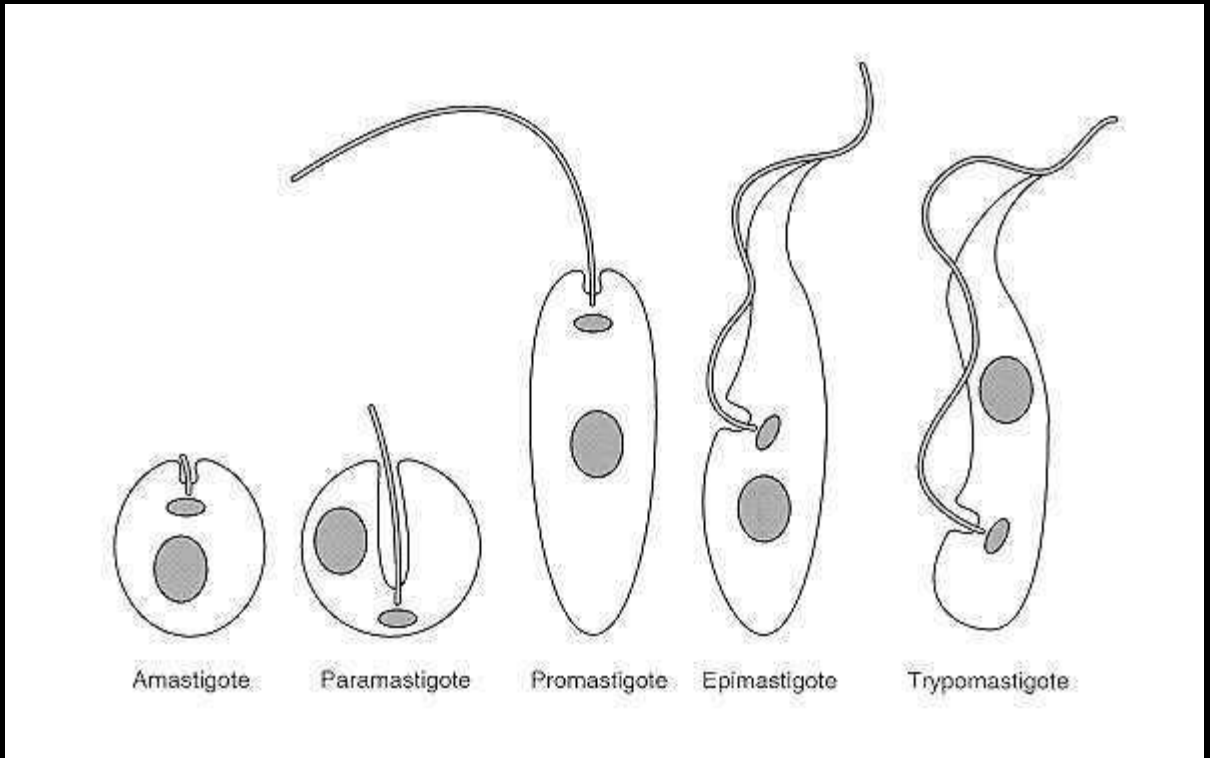


LABORATORY DIAGNOSIS

2. **Serologic tests:**
- **Become positive after 1 month of infection.**
- Indirect fluorescent antibody test.
- Indirect hemagglutination and complement fixation tests.
- ELISA

3. **PCR:** Detects The DNA.




Amastigote
Paramastigote
Promastigote
Epimastigote
Trypomastigote

MCQ # 1

Trypanosoma cruzi initially penetrates through the mucous membranes on the skin and then multiplies in a lesion known as a chagoma. In the chronic stage of the disease, the main lesions are often observed in the

a. Spleen and pancreas

b. Heart and digestive tract

c. Liver and spleen

d. Digestive tract and respiratory tract

e. Heart and liver

MCQ # 2

A butcher, who is fond of eating raw hamburger, develops chorioretinitis; Giemsa-stained preparations shows crescent-shaped trophozoites. This patient is most likely infected with

a. Trichinosis

b. Schistosomiasis

c. Toxoplasmosis

d. Visceral larva migrans

e. Giardiasis