

Trypanosoma

- Multiply asexually and they move by flagella
- Belong to a class called “Kinetoplastida”
- This class has four morphological forms:
 - Promastigote + Epimastigote are found in the vector
 - Trypomastigote + Amastigote (non-motile, intracellular) are found in the vertebrate host (humans)

1. African Trypanosomiasis (Sleeping sickness):

- Infective stage: metacyclic trypomastigote (in the saliva of the vector)
- Diagnostic stage: trypomastigote in the blood (extracellular, there is no intracellular stage in African Trypanosomiasis)
- Intermediate host: the vector
- Definitive host: Human

a. East African Trypanosomiasis

- Causative agent: *T. brucei rhodesiense*
- Vector: *Glossina* (tsetse) morsitans
- Reservoir: Humans and wild animals (Anthroponotic & zoonotic)
- More acute, fast progression, severe and fatal

b. West African Trypanosomiasis

- Causative agent: *T. brucei gambiense*
- Vector: *Glossina palpalis*
- Reservoir: Mainly humans (anthroponotic) but also domestic animals can act as a reservoir
- Begins subacute, with slow progression and not fatal as East African Trypanosomiasis
- * African Trypanosomiasis is always extracellular
- * Shows Antigenic variation → able to change the antigenic surface coat (variant surface glycoprotein) of the outer membrane of the trypomastigote, helping to evade the host immune response (become hard to be detected in blood)
- * Both sexes of Tsetse fly can transmit the parasite

Clinical feature:

1. chancre (painless) → an acute local reaction at the site of the bite
2. Stage 1 → Winterbottom's sign (enlargement of the posterior cervical lymph nodes), hepatosplenomegaly, irregular fever and night sweats but there are **no CNS symptoms**
3. Stage 2 → trypomastigote crosses brain blood barrier **causing CNS symptoms:**
meningoencephalitis → coma (uncontrollable urge to sleep and that's where the name of the disease came from) → death (if it was *T. brucei rhodesiense* since it's fatal)

Therapy

Stage 1 → Suramin or pentamidine

Stage 2 → Melarsoprol (effective for both blood and CNS stage)

2. American Trypanosomiasis (chagas' disease)

- Causative agent: Trypanosoma cruzi
- Vector: reduviid bugs or triatomine bug or kissing bug
- Infective stage: metacyclic trypomastigote
- Diagnostic stage: trypomastigote in blood in the acute phase / amastigote intracellularly inside tissues' cells mainly cardiac muscle cells in the chronic phase
- * Reduviid bug defecates while taking a blood meal, it does bite the host like Tsetse fly but doesn't inject the infective stage it rather leave it in their feces
- * The incubation period in humans is about 7-14 days.

Clinical feature:

1. Chagoma → the site of the bite and the site where feces get access to the body after it becomes itchy (raised hard nodule surrounded by hard edema" induration")
2. In the Acute Phase (starting 1 week after infection) → Irregular fever/night sweats / Lymph node enlargement / HSM / Unilateral swelling of eyelids Romana's sign, in case the trypomastigote got access through mucus membranes mostly conjunctiva / Acute myocarditis, (due to the presence of the parasite in the blood).
3. In the Chronic phase → Disturbance in holo organs' functions (including the heart) / enlargement of the heart + arrhythmia (the most frequent) / Enlargement of the colon and esophagus / Patients die because of functional disability

Therapy

Nifurtimox and benznidazole reduce the severity of acute Chagas' disease

Leishmania

- flagellated protozoa
- Belong to a class called "Kinetoplastida"
- Obligate intracellular organism
- Common in our region especially cutaneous Leishmaniasis
- Vector-mediated infection, life cycle requires two hosts
- vertebrate host: mammalian
- Invertebrate vector: female sand fly (Phlebotomus)
- Reservoir host: dogs and wild foxes
- Infective stage: promastigote (engulfed by a macrophage where there it becomes amastigote)
- Diagnostic stage: amastigote
- Transmission 1. Bite of sand fly 2. Transfusion blood, transplantation and sharing syringes 3. Mother to baby 4. Direct contact; from man to man through nasal secretions

Leishmania Species

1. Cutaneous Leishmaniasis (L.tropica, L. major)

- * Most common form of leishmania and common in our region

- Habitat: Skin only

- * Clinical feature: Lesions start as macule (red, soft, itchy, painless) → papule (raised, firm, serious discharge) → ulceration → granulomatous reaction → scars

- * most of them heal spontaneously -depending on the patient's immune status

- * Leishmania infantum; it's common in our region and a part of L. tropica complex so originally causes cutaneous leishmaniasis but might progress to visceral leishmaniasis

2. Mucocutaneous leishmaniasis (L. braziliensis ,L. mexicana)

- * Another name: nasopharyngeal leishmaniasis

- * The primary lesions are similar to those found in cutaneous leishmaniasis, but the difference is that it involves mucocutaneous membranes most commonly those of nasopharyngeal region

- * Do not heal spontaneously, and secondary bacterial infections are common and may be fatal.

- * It might progress into visceral leishmaniasis.

3. Visceral Leishmaniasis (L. donovani)

- * Called “kala azar” which means “black fever”

- * The most severe form of leishmaniasis

- * The incubation period: 10 days to 2 years

- * Clinical signs: enlarged liver and spleen, swollen lymph nodes occasional acute abdominal pain if left untreated, will almost always result in the death of the host.

- * Special sign in visceral leishmaniasis patients >> abdominal distention, dark pigmentation of the skin of the abdomen and they become anaemic as well

- * Some of the patients after recovery from visceral leishmaniasis develop cutaneous leishmaniasis, we call this condition Post-Kala-azar Dermal Leishmaniasis (PKDL)

Therapy

- In simple cutaneous leishmaniasis, lesions usually heal spontaneously but it takes months, some conditions might require treatment
- Antimony, sodium stibogluconate drugs of choice for the treatment of visceral and mucocutaneous leishmaniasis

There are NO Vaccines for Trypanosoma & Leishmania.