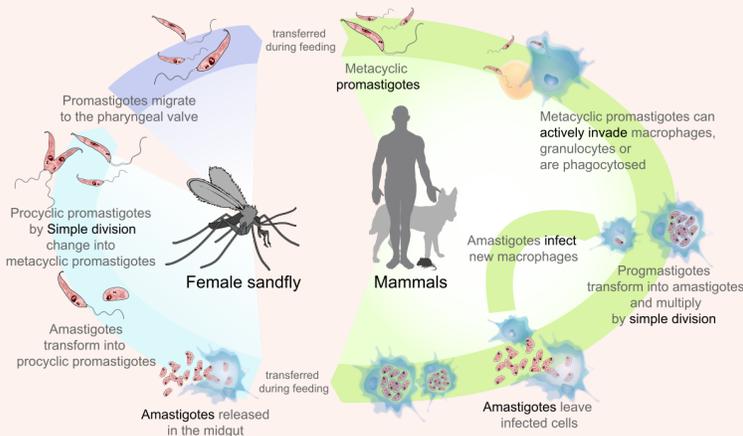


Lecture (2) : Leishmania & Trypanosoma

- Both parasites require 2 host (vertebrate < man and resevoir)& (invertebrate < vector).

Leishmania

- Information about life cycle:



D.S: Amastogoid in smear /Promastigote in culture.

I.S: promastigote with sanfly injection and Amastogoid in other method like mechancal transmittion (interrupted feeding , blood transfusion and coongenital transmission.

Habitat: reticuloendothelial cells.

vector: female sandfly

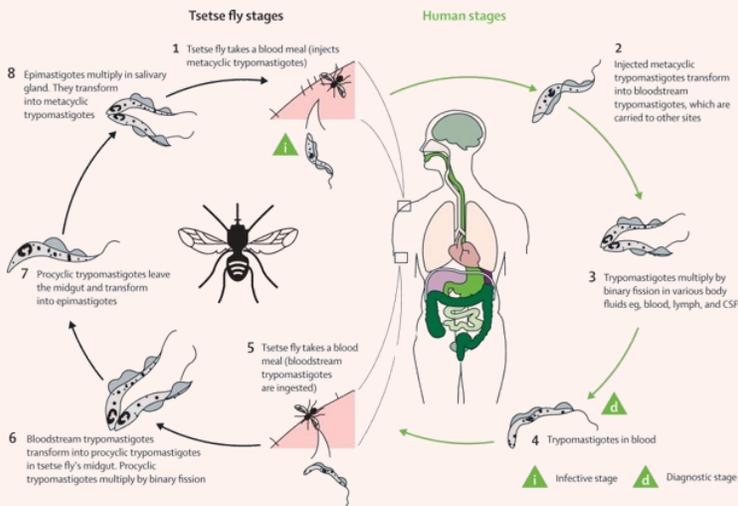
You have to know:

- It has other names like Kala azar , Dum. Dum fever & Black sickness.
- Female sand fly (Phlebotomus for OWVL & Lutzomyia In NWVL).
- **IT IS INTRACELLULAR PARASITE .**
- When we talk about clinical picture we should remember the fever (**intermitted with double daily rise**) & pigmented skin lesions (Kala azar and post kaka azar dermal leishmanoid).
- Most cause of death is **secondary bacterial infection.**
- In diagnosis we use smear, culture in NNN medium and leishmann. Or Montenegro (IDT) **not for screening but after recovery**
- Treatment is important especially **pentostam**(parenteral) and **miltefosine** (orally).



African trypanosomes:

Information about life cycle:



D.S : polymorphic trypanostigote and Epimastigote.

I.S: polymorphic trypanostigote (mechanical transmission , blood transfusion and congenital transmission) and metacyclic.

Vector : tse-tsefly (Glossina).

African Trypanosomiasis

(Polymorphic trypanosomes)



Trypanosoma brucei gambiense

Trypanosoma brucei rhodesiense

Chronic West African sleeping sickness (Gambian trypanosomiasis)

Acute East African sleeping sickness (Rhodesian trypanosomiasis)

Transmitted by *Glossina palpalis* (both male and female)

Transmitted by *Glossina morsitans* (both male and female)

Why *T. rodesian* is more dangerous than *T. gambian* ????

- it is resistant to treatment
- Acute >> death before reach CNS phase
- Difficult because it affect both man and animal (reservoir).

You have to know:



- Both female and male are vector.
- **IT IS EXTRACELLULAR PARASITE.**
- it is. Called **sleeping sickness.**
- this disease has 3 phases (chancer ,haemolyphatic and neurological stages).
- damage caused in the neurological is **irreversible.**
- in clinical picture we should remember the (**winterbottom sign**).
- in diagnosis we use microscopic examination , culture on NNN and in direct method we use **IgM serum:** always elevated in the blood and CSF due to antigenic variation of the trypanosome (**changing its antigenic coat**) to escape from host immune response
- (**evasion**).
- Treatment : in early stage (haemolympathic) >> **Suramin and Pentamidine .**
In late stage (cerebral)>> **Melarsoprol ,** tryparsamide and **Eflornithine** which is the new drug.



1) Concerning sleeping sickness, all the followings are correct EXCEPT?

1. insect is the vector of transmission.
2. Trypomastigotes multiply in the human blood.
3. In late stage of infection. there is invasion of CNS and CSF.
4. Winterbottom sign is characteristic.
5. Epimastigote (crithidial form) are found in the insect and human.

2) Vector transmitted leishmania diseases?

1. Cyclop.
2. Chryspos.
3. Lice.
4. Sand fly.
5. Ticks.

3) In visceral leishmaniasis which one is not a manifestation of the disease?

1. Promastigote is the infective stage.
2. Inside the macrophages the parasites are multiply as a mastigotes.
3. Bite reaction is clearly seen.
4. Sand fly transmit the disease.
5. Pentostam is the drug of choice.

Q	1	2	3
A	5	4	3

