TRYPANOSOMA & LEISHMANIA

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Trypanosoma

leishmania
Trypanosoma

• Causative agents of African trypanosomosis (sleeping sickness) and American trypanosomosis (Chagas disease).

• Trypanosoma brucei gambiense and Trypanosoma brucei rhodesiense cause African trypanosomosis (sleeping sickness) in humans.

• Trypanosoma cruzi, the causative agent of American trypanosomosis (Chagas disease) occurs in humans and many vertebrate animals in Central and South America.
African trypanosomiasis: African sleeping sickness
American trypanosomiasis (Chagas’ disease)
Morphology

- The morphologically differentiated forms include spindly, uniflagellate stages (trypomastigote, epimastigote, promastigote) and a rounded, amastigote form.
• A unique feature of African trypanosomes is their ability to change the antigenic surface coat of the outer membrane of the trypomastigote, helping to evade the host immune response.

• The trypomastigote surface is covered with a dense coat of variant surface glycoprotein (VSG)

• Each time the antigenic coat changes, the host does not recognize the organism and must mount a new immunologic response
AFRICAN TRYPANOSOMIASIS

- Is caused by 2 sub spp.:
  - T. brucei gambiense: West African trypanosomiasis
  - T. brucei rhodesiense: East African trypanosomiasis

- Vector: tsetse fly (Glossina spp.)
- Which is found only in rural Africa
- Glossina palpalis transmits T. b. gambiense
- Glossina morsitans transmits T. b. rhodesiense
Epidemiology.

• There are epidemiological differences between T. gambiense and T. rhodesiense), the main one being that T. rhodesiense persists in a latent enzootic cycle in wild and domestic animals and is normally transmitted by Glossina from animal to animal, more rarely to humans.

• T. gambiense, on the other hand, is transmitted mainly from human to human by the tsetse flies, although various animal species have also been identified as reservoir hosts for T. gambiense strains.
Epidemiology
• **Clinical feature:**

After the host has been bitten by an infected tsetse fly, a **nodule or chancre** at the site may develop after a few days.

• **stage I:** The patient have **systemic trypanosomiasis** without CNS involvement.

• The trypomastigotes enter the bloodstream and invade the lymph nodes

• The first symptoms appear and include: irregular fevers with night sweats, enlargement to liver and spleen, **Winterbottom's sign**.
• **Stage II**: organisms invade the CNS, the sleeping sickness stage of the infection is initiated

• The patient becomes emaciated and progresses to profound coma and death
• **Specimen:** blood, serum, CSF, aspiration from lymphnode

• **Routine Methods:** thick and thin blood films

• **Antigen Detection:** simple and rapid test card indirect agglutination

• **Antibody Detection:** Serologic by using ELISA Serum or CSF IgM concentrations

• **Molecular Diagnostics:** PCR-based methods to detect infections and differentiate species, but these methods are not routinely used
• All drugs used in the therapy of African trypanosomiasis are toxic and require prolonged administration

• anti parasitic drug selected depends on whether the CNS is infected

• Suramin or pentamidine isethionate can be used when the CNS is not infected

• Melarsoprol, a toxic trivalent arsenic derivative, is effective for both blood and CNS stages but is recommended for treatment of late-stage sleeping sickness
1. Preventing flies from biting through the use of insecticide will reduce the transmission of the parasite.

2. Screening of people at risk helps identify patients at an early stage.

3. Treatment cases and should be monitored for 2 years after completion of therapy.
• Trypanosoma cruzi (Chagas’ disease)
• Zoonosis
• Transmitted by vector: reduviid bugs.
• Reduviid bug defecates while taking a blood meal

• **Definitive host:**
  • Human, dog, cat, rats...etc.
• **Habitat** in the Definitive host:
  • Trypomastigote in blood
  • Amstigote in tissue
Through out central and south America
Triatomine Bug Stages

1. Metacyclic trypomastigotes
   (passes metacyclic trypomastigotes in feces,
   trypomastigotes enter bite wound or
   mucosal membranes, such as the conjunctiva)

2. Monkey
   - Metacyclic trypomastigotes penetrate various cells at bite
     wound site. Inside cells they transform into amastigotes.

3. "A" Amastigotes multiply
   by binary fission in cells of infected tissues.

4. "d" Intracellular amastigotes transform into trypomastigotes,
   then burst out of the cell and enter the bloodstream.

5. Human
   - Triatomin bug takes a blood meal
     (trypomastigotes ingested)

6. Epimastigotes
   in midgut

7. Multiply in midgut

8. Metacyclic trypomastigotes
   in hindgut

Legend:
- Infective Stage
- Diagnostic Stage
- Chagas’ disease are categorized as acute, indeterminate, and chronic
- Nodule chagoma: near the bite

- The **incubation period** in humans is about 7-14 days
• **Acute phase:**

• Start 1 week after infection
• Fever
• Lymph node enlargement
• Enlarge liver and spleen
• Unilateral swelling of eyelids romana’s sign
• Acute myocarditis
• Chronic phase:

• Develop years after the diagnosis of acute disease

• Most frequent clinical signs of chronic Chagas’ disease involve the heart, where enlargement of the heart, including cardiac changes

• Enlargement of the colon
• Nifurtimox and benznidazole reduce the severity of acute Chagas’ disease.

• Both medicines are almost 100% effective in curing the disease if given soon after infection at the onset of the acute phase including the cases of congenital transmission.
1. Vector control
2. Transfusion control and screening of blood donors
3. testing of organ, tissue or cell donors and receivers
• It is a flagellated protozoan
• Life cycle requires two hosts:
  a) **vertebrate**; mammalian host
  b) **Invertbrate vector**; female sand fly
• Obligate intracellular organism
• Infects primarily phagocytic cells and macrophages
• The incubation period ranges from 10 days to 2 years,
Leishmaniasis is divided into clinical syndromes according to what part of the body is affected most.

1. Cutaneous Leishmaniasis (L.tropica, Leishmania major)
2. Mucocutaneous leishmaniasis (L. braziliensis)
3. Visceral Leishmaniasis (L. donovani).
1. Bite of sand fly
2. Transfusion blood and transplantation
3. Mother to baby
4. Direct contact; from man to man through nasal secretion.
Cutaneous Leishmaniasis: Leishmania tropica, L major, L infantum

- **Habitat**: skin
- **Disease**: Cutaneous leishmaniasis
- **Clinical feature**: first sign is a lesion (generally a firm, The lesions begin as reddish, soft itchy papular, gradually enlarges, raised and firm, with serous discharge at the bite site.)
- **Epidemiology**: the Middle East, South America
In Jordan there are several species of Leishmania; Leishmania infantum, Leishmania tropica, and Leishmania major.

- Leishmania major is the major species of Leishmania parasite in Jordan.
• The primary lesions are similar to those found in cutaneous leishmaniasis.

• Dissemination to the nasal or oral mucosa may occur from the active primary lesion or may occur years later after the original lesion has healed.

• These mucosal lesions do not heal spontaneously, and secondary bacterial infections are common and may be fatal.
Visceral Leishmaniasis (L. donovani)

- Is the most severe form of leishmaniasis
- The parasite migrates to the internal organs such as the liver, spleen (hence "visceral"), and bone marrow

- **The incubation period**: 10 days to 2 years, usually
- **Symptoms**: fever, anorexia, malaise, weight loss, and, frequently, diarrhea
- **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

**Epidemiology**: Bangladesh, Brazil, Ethiopia, India, South Sudan and Sudan.
1) **Stained blood smear**: aspiration, scraping

2) **Cultured**: cultured using special techniques

3) **ELISA, IFA or direct agglutination** give useful indication of active or recent kala-azar.

4) **PCR** methods have excellent sensitivity and specificity for direct detection
5-Intradermal Montenegro test:
Injection of intradermal antigen prepared from cultured promastigotes of Leishmanian spp.
This produces a typical cell-mediated response.

6-Histologic examination by biopsy from tissue to demonstrate the presence of organism in the tissue.
• The patient response varies depending on the Leishmania species and type of disease.

• In simple cutaneous leishmaniasis, lesions usually heal spontaneously

• **Antimony, sodium stibogluconate** drugs of choice for the treatment of visceral leishmaniasis.
**PREVENTION**

- **Reduction of sand fly population**
  - by insecticides mainly DDT, dieldrin, malathion

- **Reduction of reservoir**
  - by killing all the infected dogs in the cases of zoonotic kala-azar.

- **Education in the community**
  - About the causes and modes of transmission of leishmaniasis.

- **Prevention of exposure to sand fly**
  - using insect repellent, bed nets and window mess as needed.

*There are No Vaccines* to prevent leishmaniasis.
The End