

Haemoflagellates (Leishmaniasis & Trypanosomiasis)

GIT & HAEMATOLOGY BLOCK



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LEISHMANIASIS

kinetoplast A disease caused by leishmania nucleus kinetoplast parasites, and transmitted by sand fly bite as **PROMASTIGOTES** (infective stage). Inside humans they are found in macrophages and tissue as **AMASTIGOTES** (diagnostic phase). amastigote promastigote **Infective Stage – in insects** Diagnostic Phase – in human lives inside macrophages so it > Pathogen: Leishmania affects the reticuloendothelial system (visceral leishmaniasis) *Leishmania major**(main species in KSA) Leishmania tropica* **Cutaneous Leishmaniasis** Leishmania aethiopica (common form) Leishmania Mexicana Leishmania donovani* Visceral Leishmaniasis Leishmania infantum* (severe form) *Leishmania chagas* Mucocutaneous leishmaniasis Leishmania braziliensis

♦ Life Cycle:

* <u>Female</u> sand fly is the vector for the disease.

Found in humans and rattans as **AMASTIGOTE** \rightarrow sand fly takes a blood meal \rightarrow amastigote grow to **promastigote** in sand fly \rightarrow sand fly takes blood meal and infect humans by **promastigote** \rightarrow promastigote <u>phagocytizied</u> by macrophages \rightarrow promastigote transform to **amastigote** \rightarrow **amastigote** multiply in cells (including macrophages) of human tissue \rightarrow sand fly transmit the disease again.



CUTANEOUS LEISHMANIA :

Scommon Clinical types of cutaneous Leishmaniasis:

- *Leishmania major*: Zoonotic (from animal to human) cutaneous Leishmaniasis : <u>wet</u> lesions with **severe** reaction.
- *Leishmania tropica*: Anthroponotic (from human to human) cutaneous Leishmaniasis : <u>Dry</u> lesions with minimal ulceration.

Sclinical presentation:

Oriental sore (most common) classical self-limited ulcer.

Uncommon types of cutaneous Leishmaniasis:

• Diffuse cutaneous leishmaniasis (DCL):

Caused by L. aethiopica, **diffuse** nodular non-ulcerating lesions. Low immunity to Leishmania antigens, numerous parasites.

Leishmaniasis recidiva (lupoid leishmaniasis):

Severe immunological reaction to leishmania antigen leading to persistent dry skin lesions, few parasites.

LD = Leishman-Donovan

Diagnosis:

- Smear: <u>Giemsa stain</u> microscopy for LD bodies (amastigotes)
- Biopsy: microscopy for LD bodies or culture in NNN medium for promastigotes.

➡ <u>Treatment:</u>

- No treatment self-healing lesions
- Medical:
 - Pentavalent antimony (Pentostam), Amphotericin B
 - +/- Antibiotics for secondary bacterial infection.
- Surgical:
 - Cryosurgery (cryotherapy) is the application of extreme cold to destroy abnormal or diseased tissue.
 - Excision Excision : Surgical removal by cutting.
 - Curettage : Surgical scraping, usually of the lining of a body cavity, to clean it of foreign matter, to remove tumours or other growths or diseased tissue.

VISCERAL LEISHMANIA : (kala-azar)

$\stackrel{\text{W}}{\Rightarrow}$ <u>Etiology:</u>

- Leishmania infantum mainly affect children
- Leishmania donovani mainly affects adults

Presentation:

- Fever (2 times per day)
- Splenomegaly, hepatomegaly, hepatosplenomegaly
- Weight loss
- Anaemia
- Epistaxis (Bleeding from nose)
- Cough
- Diarrhea

- Untreated disease can be fatal >< (affect the reticuloendothelial system)
- After recovery it might produce a condition called **post kala-azar dermal leishmaniasis (PKDL)**

Diagnosis:

- Parasitological diagnosis:
- Bone marrow aspirate
- Splenic aspirate (very dangerous)
- Lymph node
- Tissue biopsy (not done)

Methods :

- 1. Microscopy -> Amastigotes (LD bodies)
- 2. Culture in NNN medium -> Promastigotes

• Immunological diagnosis:

- Specific serologic tests: Direct Agglutination Test (DAT), ELISA, IFAT
- Skin test (leishmanin test) for survey of populations and follow-up after treatment.
- Non specific detection of hypergammaglobulinaem by formaldehyde (formol-gel) test or by electrophoresis.

Treatment:

- Pentavalent antimony- sodium stibogluconate (Pentostam)
- Amphotericin B
 - Treatment of complications:
- Anaemia
- Bleeding
- Infections etc.

N.B the following 2 infections aren't endemic here; there for the doctor said don't concentrate on the lifecycle

TRYPANOSOMIASIS

- In Africa → African sleeping sickness.
- In Latin America → Chagas disease.

African Sleeping Sickness:

✤ <u>Etiology:</u>

- Trypanosoma brucei rhodesiense:
 East Africa, wild and domestic animal reservoirs.
- **Trypanosoma brucei gambiense:** West and Central Africa, mainly <u>human</u> infection.

$\stackrel{\text{life Cycle:}}{\to}$

Human Stage:

The tsetse fly taking blood from a mammalian host, an infected tsetse fly injects **metacyclic trypomastigotes** into skin tissue \rightarrow parasites first enter the **lymphatic system** and then pass into the **bloodstream** \rightarrow they transform into bloodstream **trypomastigotes** \rightarrow reach other body fluids (e.g., lymph, spinal fluid), and continue to replicate by binary fission.

• Tsetse fly stage:

A tsetse fly becomes infected with bloodstream trypomastigotes when taking a blood meal on an infected mammalian host \rightarrow In the fly's midgut, the parasites transform into procyclic trypomastigotes \rightarrow multiply by binary fission \rightarrow leave the midgut \rightarrow transform into epimastigotes \rightarrow epimastigotes reach the fly's salivary glands and continue multiplication by binary fission.



- 1. Skin stage: chancre. (painless ulceration at the site of entry of a pathogen)
- 2. Haematolymphatic stage (Winterbottom's stage) : generalized lymphadenopathy (swollen lymph nodes due to infection and inflammation), anaemia, generalized organ involvement.
- 3. Central nervous system stage (CNS): Meningoencephalitis.

(Involvement of the CNS also cause the sleeping sickness)

Development of the disease more rapid in Trypanosoma brucei rhodesiense.
 (They die before reaching the sleeping sickness)

➡ <u>Diagnosis:</u>

- Lymph node aspiration (in Winterbottom's stage) → Microscopy
- **CSF aspiration** (in CNS stage) → Microscopy

➡ <u>Treatment:</u>

For early infection : Pentamidine , Suramin . For late infection : eflornithine (Diflouromethylornithine- DFMO)

Chagas Disease: usually transmitted through the conjunctiva

Other name is American TRYPANOSOMIASIS

$\stackrel{\text{W}}{\Rightarrow}$ <u>Etiology:</u>

Trypanosoma cruzi

$\stackrel{\text{life Cycle:}}{\Rightarrow}$

Scratching the site of the bite causes the trypomastigotes to enter the host through the wound, or through intact mucous membranes \rightarrow inside the host, the trypomastigotes invade cells \rightarrow they differentiate into intracellular amastigotes \rightarrow amastigotes multiply by binary fission and differentiate into trypomastigotes \rightarrow released into the bloodstream. This cycle is repeated in each newly infected cell. Replication resumes only when the parasites enter another cell or are ingested by another vector.



Sclinical Presentation:

- Chagoma

 cutaneous stage (local swelling where the parasite entered the body)
- Ocular lesion → Romana's sign (swelling of the eyelids on the side of the face near the bite wound)
- Heart damage → Myocarditis (in chronic stage)

♦ <u>Diagnosis:</u>

- Blood film
- Serology: IFAT (Immunofluorescence Antibody Test)
- Xenodiagnosis: feeding bugs on a suspected cases.

Xenodiagnosis is a process to

diagnose an infectious disease by exposing tissue to a vector and then examining the vector for the presence of a microorganism or pathogen.

➡ <u>Treatment:</u>

- benznidazole
- nifurtimox

N.B doctor Adele said don't worry about the drugs for TRYPANOSOMIASIS BECAUSE THERE'RE NOT VERY COMMON



Q1- what is the most common etiology for cutaneous Leishmaniasis in saudia Arabia ?!

A-Leishmania major

C- Leishmania infantum

B- Leishmania aethiopica*D-* Leishmania chagas

Q2-what acts as the vector for the transmission of Chagas Disease?!

A-Tsetse fly

B- sand fly

C-Mammals

D- Triatomic bugs

Answers; A,D