Subject: HLS – microbiology

Topic: hemoflagellates

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Hemoflagellate (Trypanosoma, leishmania -- move by the mean of single flagella, asexual only)

General developmental stages: amastigote [contains **kinetoplast** which is the origin of the flagellum] → promastigote → epimastigote → trypomastigote [contains **flagellum** + **undulating membrane** on the same axis of the protozoa]

Trypanosoma

Causes trypanosomiasis = African trypanosomiasis [sleeping sickness] caused by ***Trypanosoma brucei*** complex + American trypanosomiasis [Chagas disease] caused by ***Trypanosoma cruzi*** ➔ *both are vector borne, can be transmitted through blood transfusion and transplacental*

Morphology

Inside humans: **trypomastigote** + **amastigote** (round intracellular form presents only in American trypanosomiasis)

Inside vectors: **promastigote** + **epimastigote**

Antigen variation

A unique feature of **African trypanosomes** is their ability to change the antigenic surface coat of the outer membrane of the trypomastigote (VSG), helping to evade the host immune response.

1- African trypanosomiasis

Caused by: **T. brucei gambiense -> west African trypanosomiasis** [chronic, slowly, reservoir: humans], **T. brucei rhodesiense -> east African trypanosomiasis** [acute, faster, reservoir: animals, less frequent]

Vector: Tsetse fly (Glossina spp.)

Infective stage: metacyclic trypomastigotes

Diagnostic stage: trypomastigotes

In human bloodstream: trypomastigotes / inside the vector: procyclic trypomastigotes -> metacyclic trypomastigotes

Clinical features: after the host has been bitten by an infected tsetse fly, a painless nodule (**chancre**) at the site may develop – **stage 1**: the patient have systemic trypanosomiasis **without CNS involvement +** The trypomastigotes enter the bloodstream and invade the lymph nodes + irregular fever with night sweats, enlargement of liver and spleen, **Winterbottom's sign – stage 2:** organisms **invade the CNS**, the sleeping sickness stage of the infection is initiated, coma, death

Laboratory diagnosis: **trypomastigotes inside the blood**, antigen detection, antibody detection, molecular diagnostics

Therapy: if the CNS isn't affected → **Suramin**, if the CNS is affected (bad prognosis) → **Melarsoprol**

Prevention: preventing flies from biting, screening of people at risk, treatment cases

2- American trypanosomiasis:

Caused by Trypanosoma cruzi (Chagas' disease)

Vector: reduviid bugs (kissing bugs) / defecate while taking a blood meal / enter through wounds or mucosal membranes

Epidemiology: through out central and south America

Infective stage: metacyclic trypomastigotes

Diagnostic stage: amastigote (in tissues)

trypomastigote (in blood)

Clinical features: nodule chagoma – **acute phase:** start after 1 week of infection, fever, lymph node enlargement, enlarge liver and spleen, unilateral swelling of eyelids **Romana's sign**, acute myocarditis – **chronic phase:** involve the heart, where enlargement of the heart, including cardiac changes (can affect any organ – enlargement of the colon)

Therapy: **Nifurtimox**

Prevention: vector control, transfusion control, testing of organ, tissue or cell donors and receivers

Leishmania:

Vector: female sand fly

Obligate intracellular organism

Infects primarily phagocytic cells and macrophages

Infective stage: promastigotes

Diagnostic stage: amastigotes

Transmission: bites of sand fly, transfusion blood and transplantation, mother to baby, direct contact from man to man through nasal secretion

A. **Cutaneous leishmaniasis :** Leishmania tropica, L major, L infantum / lesions / **Leishmania major** is the major species of Leishmania parasite in Jordan .

B. **Mucocutaneous leishmaniasis (naso-pharyngeal):** L. braziliensis

C. **Visceral leishmaniasis (kala- azar / black fever):** L. donovani / liver, spleen, bone marrow / enlarged liver and spleen / India + Sudan

Laboratory diagnosis: amastigotes inside the macrophages / **intradermal Montenegro test [type 4 / delayed hypersensitivity reaction]**

Therapy**:** cutaneous leishmaniasis -> lesions usually heal spontaneously / mucocutaneous + visceral leishmaniasis → ***sodium stibogluconate***

Prevention: vector control