HLS-Microbiology 2+3

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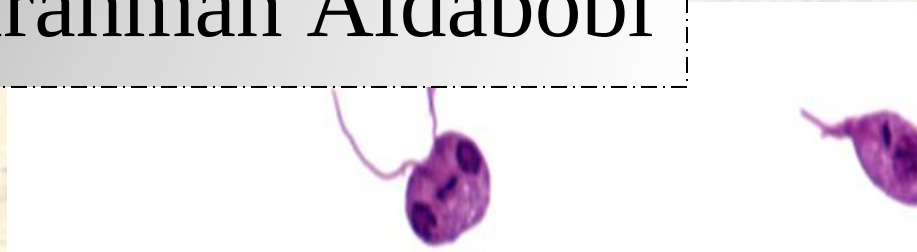
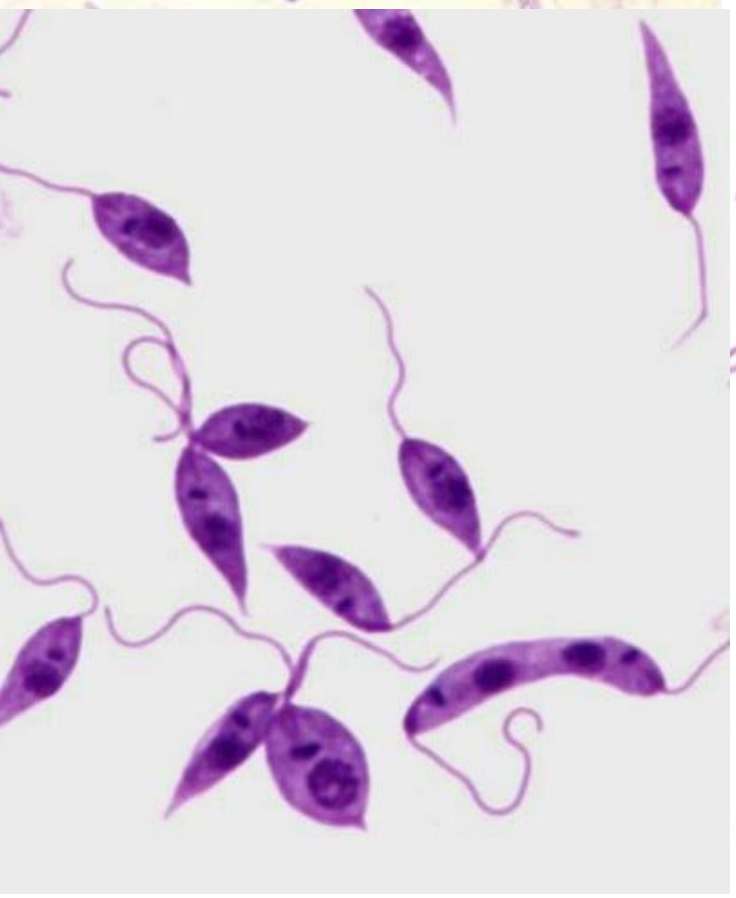
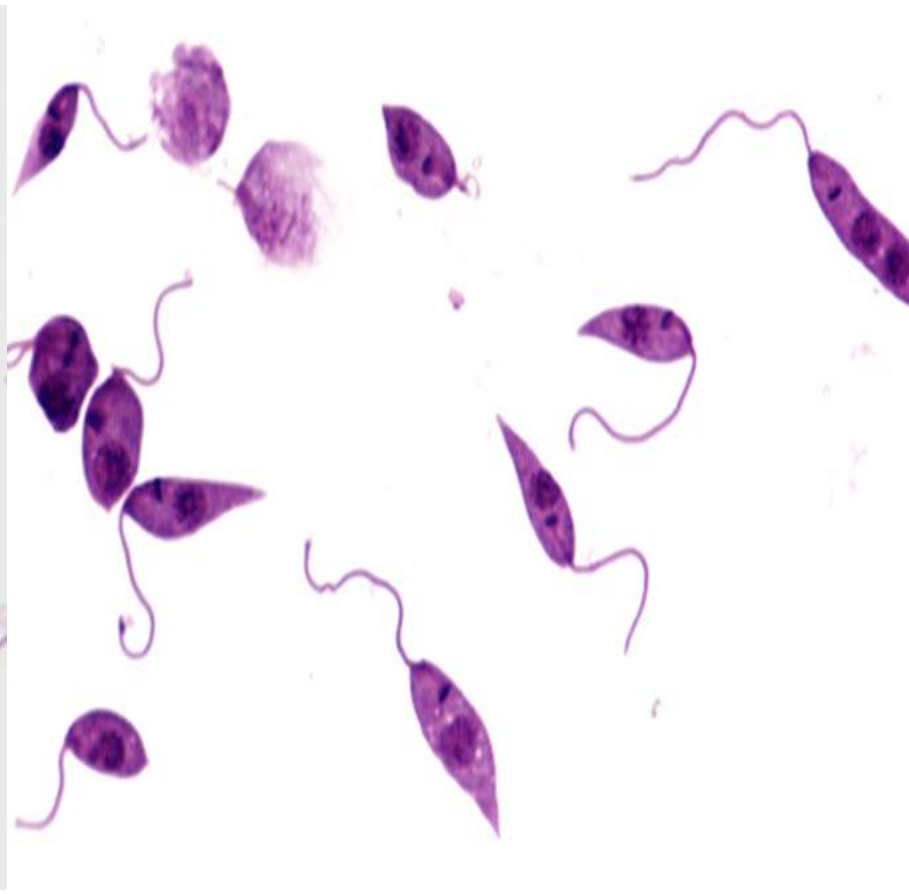






Plasmodium

1-Sporozoa → Non-Motile in adult stage and alternates between sexual and asexual reproductive phases + **Intracellular** (All of them)

2-Inside the human body → Plasmodium only have ASEXUAL (aka. Schizogony) life cycle

3-Two Hosts with two cell cycles (Dipteran Insect + Vertebrae Host)

4-Steps Of infection (Number one Killer in parasitic infections)

a) **Female Anopheline Mosquito** takes a blood meal from the human
b) **Sporozoites** spread from the salivary glands of the mosquito to the blead
c) **Sporozoites** reach the liver → Replicate asexually (Schizogony) in the hepatocytes
d) This asexual reproduction results in **Exoerythrocytic Merozoites** → This is called the extra-erythrotic (Intrahepatic) life cycle
e) **Exoerythrocytic Merozoites** reach circulation and invades RBCs
f) Inside the RBC, Merozoites are also called **Young Trophozoites** → Uninuclear
g) **Young Trophozoites** starts to divide its nucleus → **Developing Schizont**
h) **Mature Schizont** contains Merozoites that will be spilled in the bloodstream (The number of **Merozoites** within the **Mature Schizont** is used to differentiate between plasmodium types)

5-Extra-Erthythrocytic cycle is one of the most important ways to differential between malaria and Babesia

Plasmodium Vivax

1-The most common cause of malaria

2-Cause Benign Tertian Malaria → Fever every three days → Fever in the first day, then two normal days, then fever and so on

3-Infect Young RBCs → Reticulocytes

4-Low number of Reticulocytes in the blood → Lower load of vivax in the blood → Benign

6-**Hypnozytes** → Dormant form in the liver that can relapse after 1-5 Years (Only in Vivax and Ovale → Different treatment regimen)

7- **Mature Schizont** contain (12-24) merozoites, used to differentiate between it and Ovale (more than 8 → Vivax, Less than8 → Ovale)

8-Contains **Schuffner's Dots** → Granulations (stipplings) within the infected RBC

9-May have symptoms of photophobia, anorexia, muscle aches, nausea, and vomiting

10-Cause Typical Rigors → Fever with the feeling of cold and shivering

11-In case of prior exposure to malaria → Parasite may be present in the blood few days before symptoms

12-Plasmoduim Ovale is the same of Vivax, but less common, less relapsing, less severe, no typical rigors and with <8 merozoites within the mature schizont (same treatment and same Schuffner's dots)

Plasmodium Malariae

1-Cause the classical malaria (now it is endemic in some regions)

2-Quartan Malaria → Cycle repeats in 72 hours → Fever, normal three days then fever

2-Infect Old RBCs, No **Schuffner's Dots**

3-Most common complication → **Membrane Proliferative Glomerulonephritis** (Immune complex deposition within the glomeruli → **Proteinuria**)

Plasmodium Falciparum

1-Most serious one → Affects all ages of RBCs

2-Causes Malignant Tertian Malaria → Very serious fever (>41) (Malignant)

3-Infected RBCs will develop some membranous projections → More adherent to the endothelium → Stuck and prevent blood flow → Ischemia

4-These are the basis of Cerebral Malaria (most serious complication)

5-Black water fever → The hemolysis of RBCs increases the hemoglobin concentration in the urine and body fluids → Discoloration

6-Pateints later-on will suffer from Hypovolemia and shock (Dr. did not explain)

7-Maurer's dots (instead of Shcuffner's Dots) → Larger, single, and more bluish

8-Banana-Shaped (Crescent-shaped) Gametocytes + Double dotted chromatin rings

Plasmodium Simian

1-Simian Malaria / Human Fifth Malaria - affects all ages of RBCs

2-Affect East Asia more commonly (Near Monkeys)

3-24 Hour Cycle (Day with fever and day without fever)

4-Early Blood stages resemble Falciparum, Mature stages resemble Malariae, misdiagnosed with Malariae and can be fatal

5-No **Schuffner's Dots**

Clinical Features (For All)

1-Incubation period from one week to five weeks

2-All start with non-specific symptoms (Fatigue, malaise, arthralgia, …)

3-All start with irregular fever then stick to pattern as were illustrated

4-All can cause anemia (more common with falciparum due to parasitemia)

Diagnosis

Definitive diagnosis is to see the plasmodium in the Blood

a) Thick blood film → To see if there is plasmodium in the blood
b) Thin Blood film → To identify the species of the plasmodium
c) Most common used stain is Giemsa stains (Wright's Stain and Fluorescent nucleic acid stains, such as acridine orange can also be used)

d) Serological methods →Look for antigens in the plasmodium such as Histidine-rich protein 2 (HRP2) and specific parasite lactate dehydrogenase (pLDH)
e) PCR can also be used

Treatment

1-Ovle + Vivax → Quinolones + Primaquine (The last to kill hypnozoites and prevent relapsing- Quinolones alone are NOT ENOUGH)

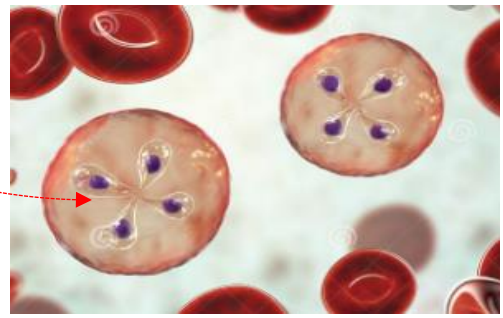
2-Falciparum → Artemisinin Based combination

3-Malariae → Quinolones

*Prevention is the most important + No vaccine

Babesiosis

Very similar to malaria, so the most important thing is to distinguish between both and here are some differences:



1-Can be extracellular + Tetrads of merozoites are distinguishing features

2-The most common causative agent is B. Microti → Transmitted by Ixodes scapularis (the same transmitter agent of Lyme Disease)

**B.duncani by Ixodes Pacificus, B.divergens (most common in Europe) by Ixodes dentatus.

3-NO INTRAHEPATIC PHASE

4-Fever with no cyclic patterns

5-Severes Disease is seen with >50-year-old, neonatal prematurity, Male gender, immunocompromised, malignancy and hemoglobinopathy

6-Pathogensis is all around RBC Lysis → Anemia + Increased Hb + RBC depri accumulation in the kidney → Renal Failure

7-Treat with Atovaquone + Azithromycin (If severe → Clindamycin + Quinones)

8-Prevent by avoidance of tick

Trypanosoma

1-Flagellated + Asexual Production (Along with leishmania)

2-Developmental Stages (Amastigote → Promastigote →Epimastigote →Trypomastigote), first two in vector and last two in human

African Trypanosomiasis

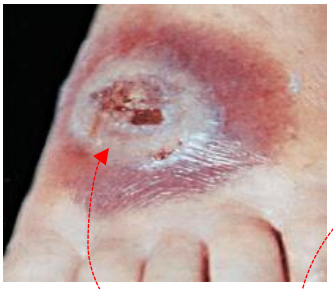
1-Sleeping Sickness

2-Causative agents → Trypanosoma brucei (Gambiense-West/Rhodiense-East)

3-Vector → Tsetse (Glossina) fly → East → Glossina Morsitans, West →Glossina palpalis

4-No intracellular amastigote

5-Have a dense coat of variant surface glycoprotein → Ability to change the antigenic surface coat



6-East → Acute and fast (less affect)

7-West → Chronic and slower

8-Infective Stage → Metacyclic Trypomastigotes

9-Diagnostic Stage →Extracellular Trypomastigotes

10-Start a painless nodule at the site of the infection called **Trypanosomal Chancre**

11-Stage 1→ parasitemia with no CNS symptoms → Fever, night sweats, enlargement of the lymph nodes and spleen + **Winterbottom's Sign** (enlargement of the posterior cervical lymph nodes)

12-Satge 2 → CNS Symptoms → The parasite exits the blood and enters the CSF → Coma+ Uncontrollable urge to sleep

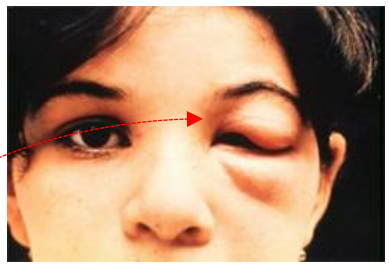
13-Diagnosis is by usual laboratory

14-The outcomes of the treatment are best before the CNS symptoms + No vaccine

15-Tx

a) Without CNS symptoms (Stage1) → Suramin / Pentamidine isethionate
b) With CNS symptoms (Stage 2) → Melarsoprol

American Trypanosomiasis



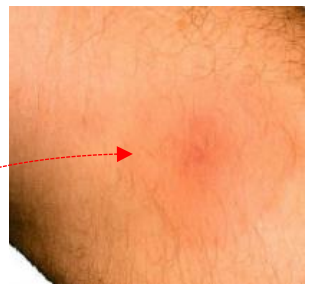
1-Chagas Disease

2-Causative Agent → Trypanosoma Cruzi

3-Vector → Triatoma infestans (Reduviid bugs) 'Kissing bugs'

4-Have intracellular amastigote and prefer cardiac muscle

5-They leave their feces in the face of the patient → Patient rub his eyes → Unilateral swelling of the eyelids → **Romana's Sign**, it can also be introduced to the body by an open wound.



6-Trypmastigoite in the blood and Amastigote in the tissue

7-Infective Stage: Trypomastigote (By feces or by wound)

8-Diagnostic Stage: Intercellular amastigote (Needs biopsy)

9- Red indurated swelling at the site of the bite→ **Nodule Chagoma**

10-Acute phase is characterized by fever, lymph nodes enlargement, Hepatosplenomegaly + Romana's Sign

12-Chronic phase → The amastigote is found intracellularly and prefers the cardia myocytes → Cardiomyopathies and heart failure, may also cause Megacolon and Megaesophagus

14-Tx →Nifurtimox, Benznidazole

Leishmania

1-Falgellated + Obligate intracellular

2-Vector → Females Sandfly

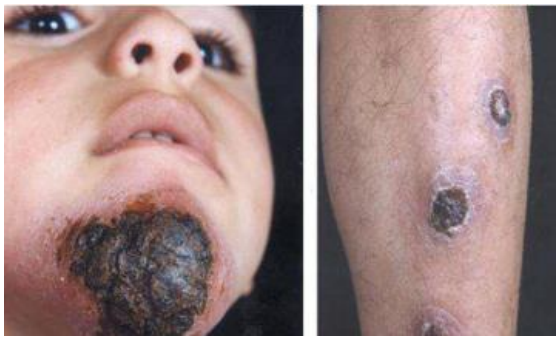
3-Infects phagocytic cells and macrophages

4-Infective Stage → Promastigote

5-Diagnostic Stage → Intercellular Amastigote

6-Transmitted by the bite of the sand fly, Transfusion blood, Mother to baby, Direct contact

Cutaneous Leishmaniasis



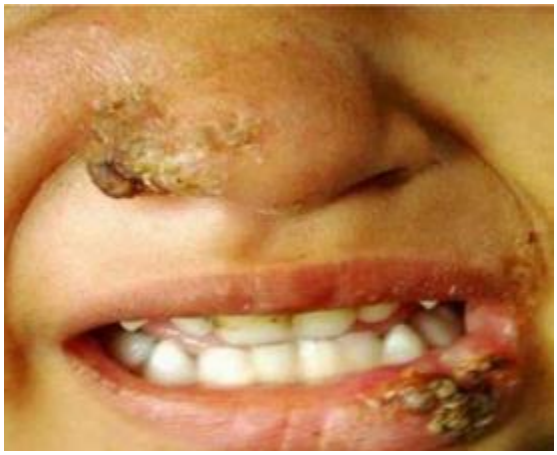
1-Caused by Leishmania Tropica, Major, Infantum

2-Leishmanis Major is the most common in Jordan

3-Only skin ulceration (Usually painless)

4-Heals spontaneously, may take months and my leave a scar

Mucocutaneus Leishmaniasis



1-Caused by Leishmania Braziliensis (Mexicana in central and south America)

2-Also called nasopharyngeal leishmania

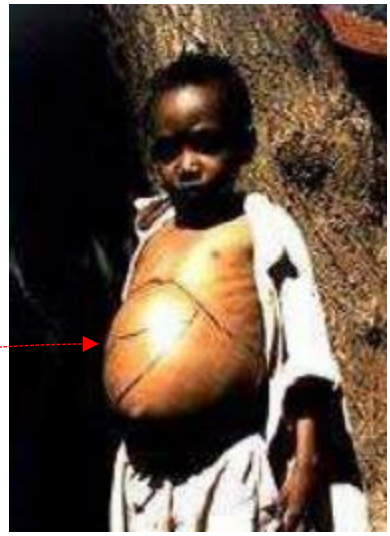
3-Primary lesion (same in Cutaneous) → Disseminates to the nasal/oral mucosa either from the active lesion or years after it has healed

4- Does not regress spontaneously (may cause nasal septum perforation of not treated)

5-Sample for diagnosis are taken from erosions

Visceral Leishmaniasis

1-Caused by Leishmania Donovani



2-Also called Kala Azar – Black Fever (Indian name since it is endemic in India)

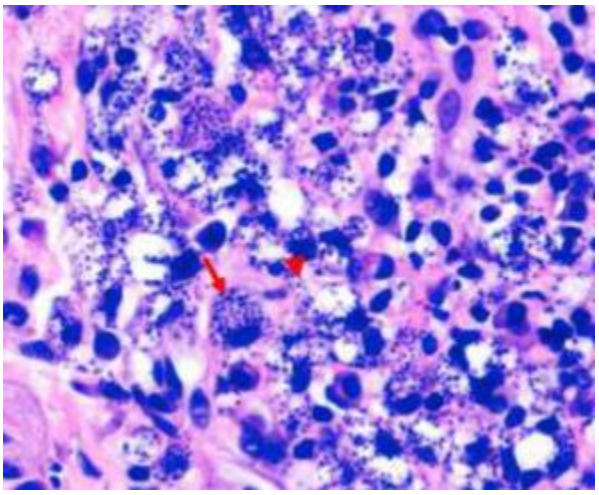
3-Targets Macrophages and APCs → Reach the lymph nodes → Lymphadenopathy → and reach internal organs (Spleen, Liver, Bone marrow)

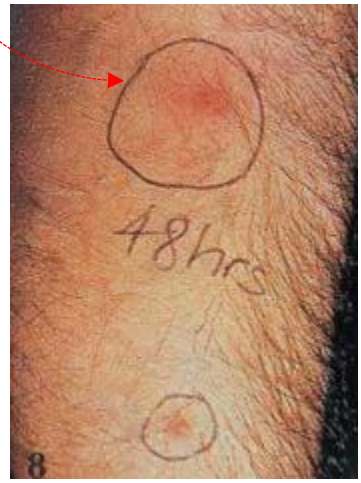
4-Causes Skin pigmentation, fever (Black-Fever), Abdominal Distension and Diahhrea

5-Diagnose by visualizing the parasite → Bone marrow aspiration from the sternum (look for the amastigote), may take blood sample or biopsy form different organs

6-Intradermal Montenegro test → Similar to PPD test → Depend on delayed type hypersensitivity (Type IV)

7-Tx → Antimony, Sodium stibogluconate





It is ALL about Time.