جامعة الانبار

كلية : الصيدلة

قسم : الصيدلة

اسم المادة باللغة العربية: الطفيليات الطبية

اسم المدة باللغة الإنكليزية: **Medical Parasitology**

المرحلة: الثانية

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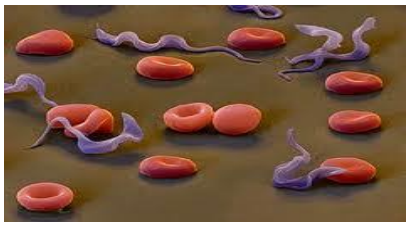
عنوان المحاضرة باللغة العربية: السوطيات الدموية

عنوان المحاضرة باللغة الإنكليزية: **Haemoflagelates**

محتوى المحاضرة:

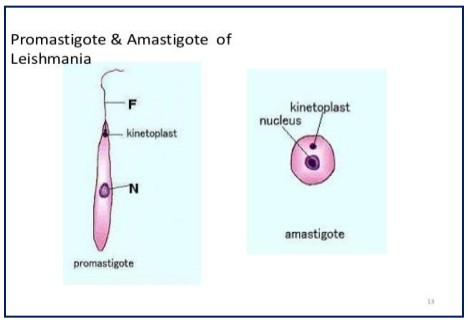
(HAEMOFLAGELATES)

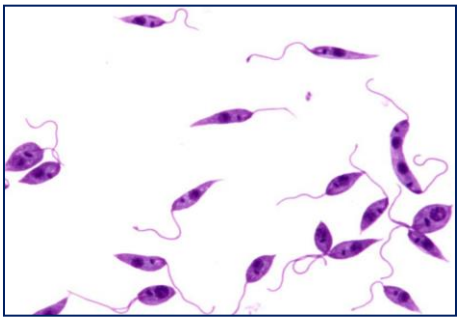


Leishmanaiasis:-

Leishmania Species:

Clinical disease

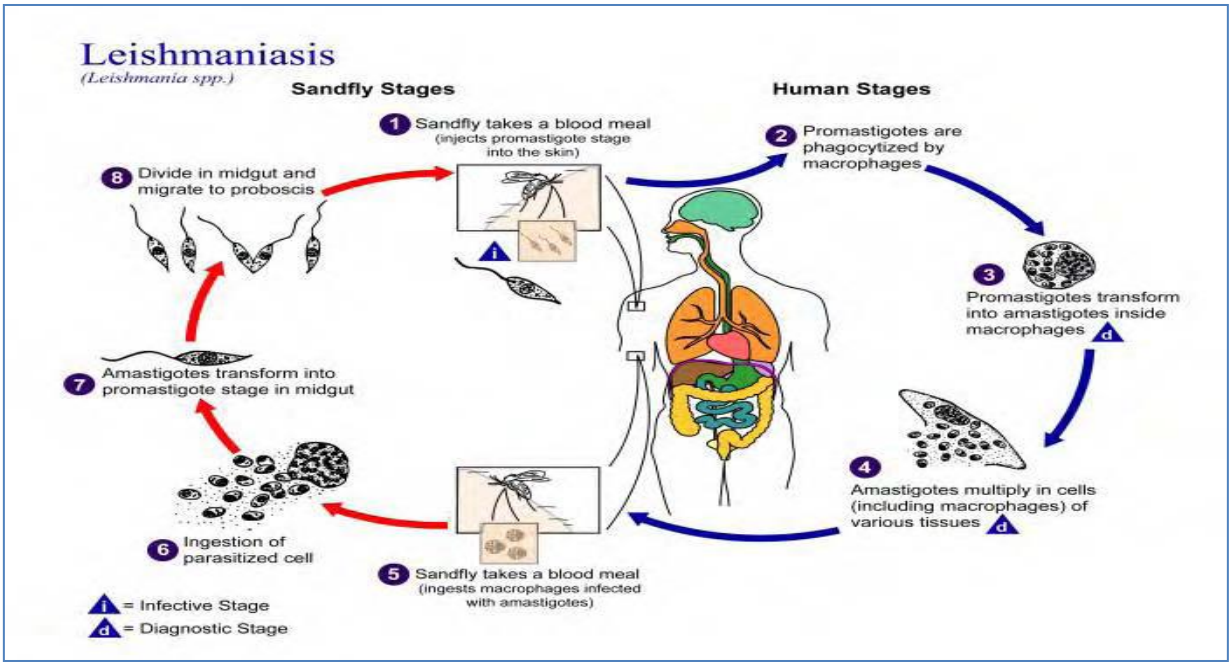




- **Visceral leishmaniasis**
- **Cutaneous leishmaniasis**
- **Mucocutaneous leishmaniasis**

The species of leishmania exist in two forms, **amastigote** (aflagellar) and **promastigote** (flagellated) in their life cycle. They are transmitted by certain species of sand flies (Phlebotomus & Lutzomyia).

Life cycle: All forms of infection starts when a female sandfly (*Phlebotomus* species) takes a blood meal from an infected host. Small amounts of blood, lymph and macrophages infected with *Leishmania* **amastigotes** are ingested. Once ingested the **amastigotes** transform to **promastigotes** in the sandfly, the non-infective **promastigotes** divide and develop into infective **metacyclic promastigotes**. These are formed in the midgut of the sandfly and migrate to the proboscis. When the sandfly bites, the extracellular inoculated promastigotes at the site of the bite are phagocytosed by macrophages. After phagocytosis, transformation to dividing **amastigotes** occurs within 24 hours. Reproduction at all stages of the lifecycle is believed to occur by binary fission. No sexual stage has been identified.



Visceral leishmaniasis

Leishmania donovani

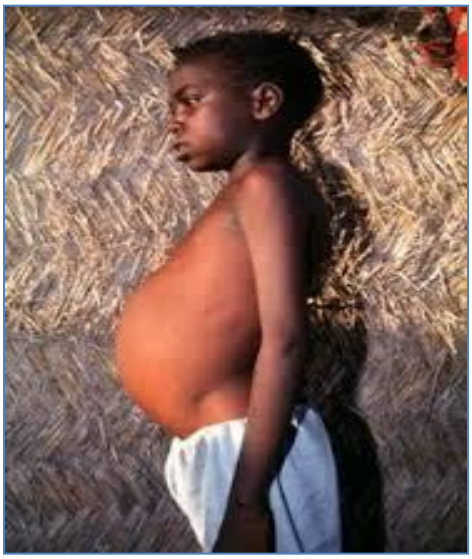
Important features: The natural habitat of *L.donovani* in man is the reticuloendothelial system of the viscera, in which the amastigote multiplies by 48 simple binary fission until the host cells are destroyed, whereupon new macrophages are parasitized. In the digestive tract of appropriate insects, the developmental cycle is also simple by longitudinal fission of promastigote forms. The amastigote stage appears as an ovoidal or rounded body, measuring about 2-3µm in length; and the promastigotes are 15-25µm lengths by 1.5-3.5µm
breadths.

Pathogenesis

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver, spleen and bone marrow) are the most severely affected organs. Reduced bone marrow activity, coupled with cellular distraction in the spleen, results in anaemia, leukopenia and thrombocytopenia. This leads to secondary infections and a tendency to bleed. The spleen and liver become markedly enlarged, and hypersplenism contributes to the development of anaemia and lymphadenopathy also occurs. Increased production of globulin results in hyperglobulinemia, and reversal of the albumin-to-globulin ratio.

Clinical features

Symptoms begin with intermittent fever, weakness, and diarrhea; chills and sweating that may resemble malaria symptoms are also common early in the infection. As organisms proliferate & invade cells of the liver and spleen, marked enlargement of the organs, weight loss, anemia, and emaciation occurs. With persistence of the disease, deeply pigmented, granulomatous lesion of skin, referred to as post-kala-azar dermal leishmaniasis, occurs. Untreated visceral leishmaniasis is nearly always fatal as a result of secondary infection.



Immunity

Host cellular and humoral defense mechanisms are stimulated.

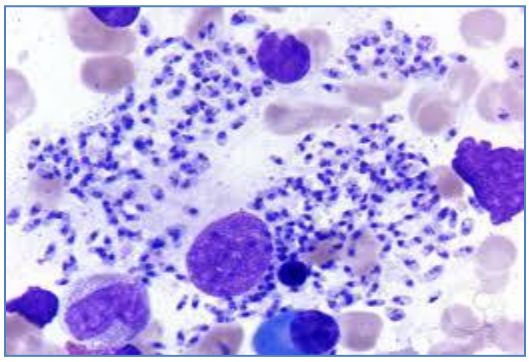
Laboratory diagnosis

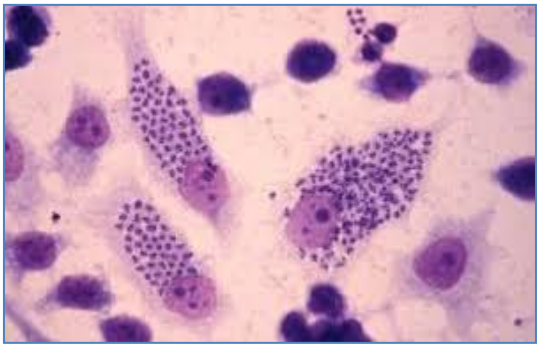
• Examination of tissue biopsy, spleen aspiration, bone marrow aspiration or lymph node aspiration in properly stained smear (e.g. Giemsa stain).

• The amastigotes appear as intracellular & extra cellular L. donovan (LD) bodies.

• Culture of blood, bone marrow, and other tissue often demonstrates the promastigote stage of the organisms.

• Serologic testing is also available.





Treatment

The drug of choice is sodium stibogluconate, a pentavalent antimonial compound. Alternative approaches include the addition of allopurinol and the use of pentamidine or **amphotercin B.**

Prevention

• Prompt treatment of human infections and control of reservoir hosts.
• Protection from sand flies by screening and insect repellents.

Old World Cutaneous Leishmaniasis (Oriental sore)

Clinical disease

L.tropica minor - dry or urban cutaneous leishmaniasis

L.tropica major - wet or rural cutaneous leishmaniasis

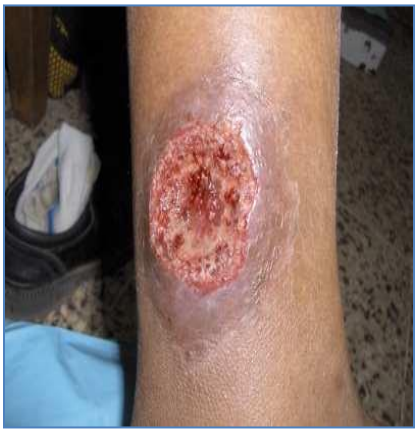
L.aethiopica - cutaneous leishmaniasis

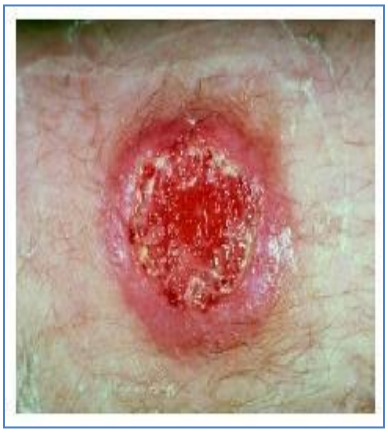
Important features

These are parasites of the skin found in endothelial cells of the capillaries of the infected site, nearby lymph nodes, within large mononuclear cells, in neutrophilic leukocytes, and free in the serum exuding from the ulcerative site. Metastasis to other site or invasion of the viscera is rare.

Pathogenesis

In neutrophilic leukocytes, phagocytosis is usually successful, but in macrophages the introduced parasites round up to form amastigote and multiply. In the early stage, the lesion is characterized by the proliferation of macrophages that contain numerous amastigotes. There is a variable infiltration of lymphocytes and plasma cell. The overlying epithelium shows acanthosis and hyperkeratosis, which is usually followed by necrosis and ulceration.





Clinical features

The first sign, a red papule, appears at the site of the fly's bite. This lesion becomes irritated, with intense itching, and begins to enlarge & ulcerate. Gradually the ulcer becomes hard and crusted and exudes a thin, serous material. At this stage, secondary bacterial infection may complicate the disease. In the case of the Ethiopian cutaneous leishmaniasis, there are similar developments of lesions, but they may also give rise to diffuse cutaneous leishmaniasis (DCL) in patients who produce little or no cell mediated immunity against the parasite. This leads to the formation of disfiguring nodules over the surface of the body.

Immunity

Both humoral and cell mediated immunity (CMI) are involved

Treatment

The drug of choice is sodium stibogluconate, with an alternative treatment of applying heat directly to the lesion. Treatment of *L.aethopica* remains to be a problem as there is no safe and effective drug.

Prevention

- Prompt treatment & eradication of ulcers
- Control of sand flies & reservoir hosts.

New World Cutaneous and Mucocutaneous Leishmaniasis

(American cutaneous leishmaniasis)

Clinical disease:

Leishmania mexicana complex- Cutaneous leishmaniasis.

Leishmania braziliensis complex- mucocutaneous or cutaneous leishmaniasis

Important features:

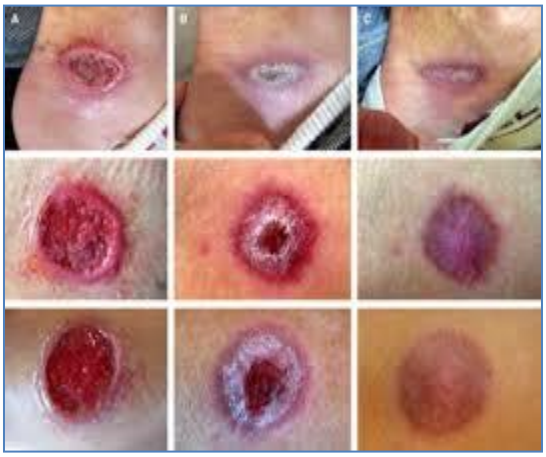
The American cutaneous leishmeniasis is the same as oriental sore. But some of the strains tend to invade the mucous membranes of the mouth, nose, pharynx, and larynx either initially by direct extension or by metastasis. The metastasis is usually via lymphatic channels but occasionally may be the bloodstream.

Pathogenesis

The lesions are confined to the skin in cutaneous leishmaiasis and to the mucous membranes, cartilage, and skin in mucocutaneous leishmaniasis. A granulomatous response occurs, and a necrotic ulcer forms at the bite site. The lesions tend to become superinfected with bacteria. Secondary lesions occur on the skin as well as in mucous membranes. Nasal, oral, and pharyngeal lesions may be polypoid initially, and then erode to form ulcers that expand to destroy the soft tissue and cartilage about the face and larynx. Regional lymphadenopathy is common.

Clinical features

The types of lesions are more varied than those of oriental sore and include Chiclero ulcer, Uta, Espundia, and Disseminated Cutaneous Leishmaniasis.



Laboratory diagnosis

- Demonstration of the amastigotes in properly stained smears from touch preparations of ulcer biopsy specimen.
- Serological tests based on fluorescent antibody tests.
- Leishman skin test in some species.

Immunity

The humoral and cellular immune systems are involved

Treatment

The drug of choice is **sodium stibogluconate**.

Prevention

- Avoiding endemic areas especially during times when local vectors are most active.
- Prompt treatment of infected individuals.

In general the list of Leishmaniasis treatment:

Treatment:

- Pantavalent antimony sodium gluconate (**Pentostom**).
- Pentamidine isothionate (Lomidine)
- Amphotericin B
- Allopurinol + Pentostom
- Gamma interferon + Pentostom.