LEISHMANIASIS

Etiology: Several species of Leishmania are pathogenic for man:

1-visceral leishmaniasis (Kala-azar, black disease, dumdum fever); L. donovani

2- cutaneous leishmaniasis (oriental sore, Delhi ulcer, Aleppo, Delhi or Baghdad boil); L. tropica (L. t. major, L. t. minor and L. ethiopica)

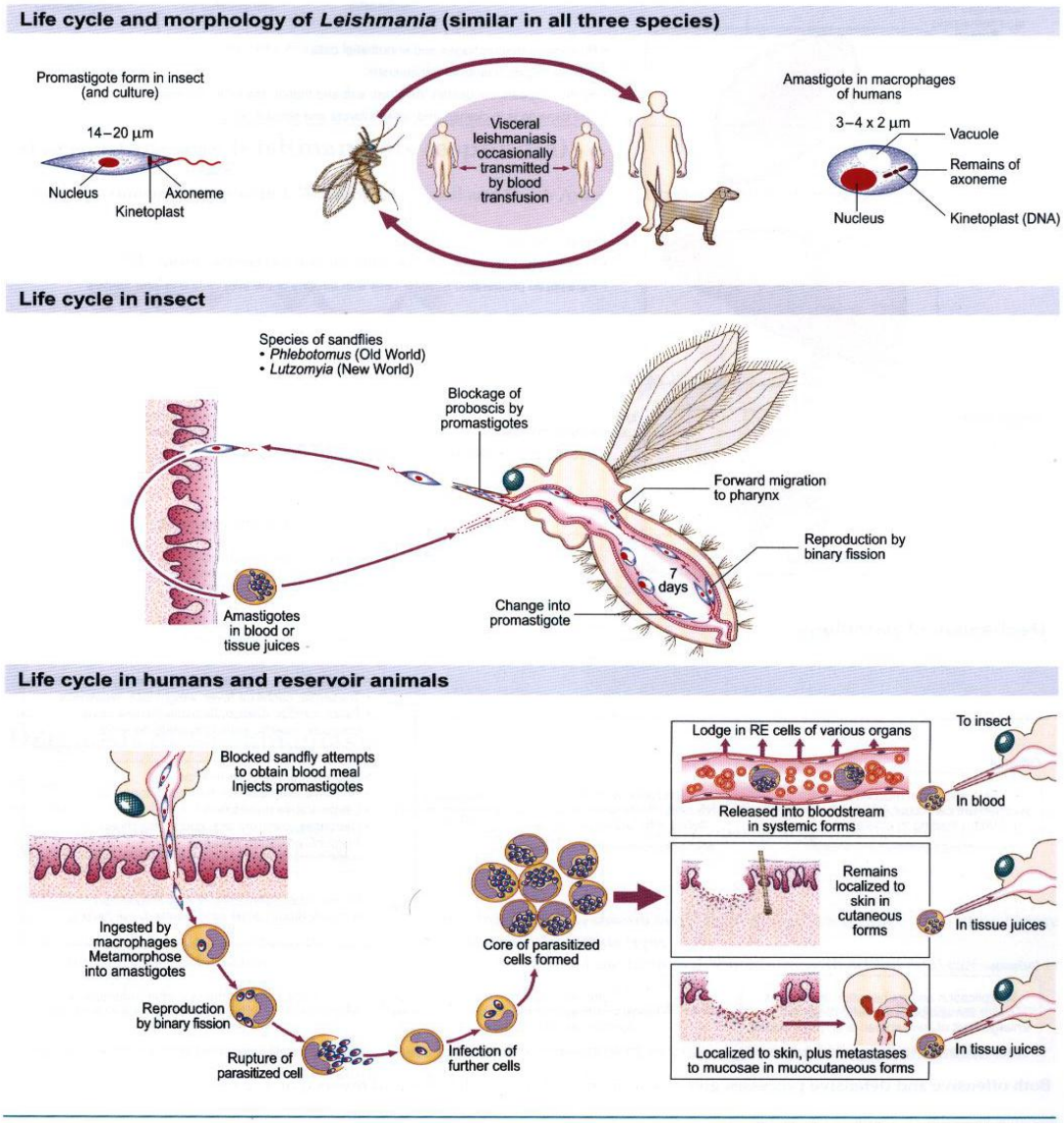
3- mucocutaneous leishmaniasis (espundia, Uta, chiclero ulcer). L. braziliensis (also, L. mexicana and L. peruviana) are etiologic

Epidemiology: Leishmaniasis is prevalent world wide: ranging from south east Asia, Indo-Pakistan, Mediterranean, north and central Africa, and south and central America.

Morphology: Amastigote (leishmanial form) is oval and measures 2-5 µ by 1-3 µ whereas the leptomonad measures 14-20 µ by 1.5-4 µ, same as trypanosome promastigote.

Life cycle: The organism is transmitted by the bite of several species of blood-feeding sand flies (Phlebotomus) which carries the pro mastigote in the anterior gut and pharynx. It gains access to mononuclear phagocytes where it transform into a mastigotes and divides until the infected cell ruptures. The released organisms infect other cells.

The sand fly acquires the organisms during the blood meal, the amastigotes transform into flagellate promastigotes and multiply in the gut until the anterior gut and pharynx are packed. Dogs and rodents are common reservoirs.



Symptoms:

Visceral leishmaniasis (kala-azar, dumdum fever): L. **donovani, organisms in visceral leishmaniasis are rapidly eliminated from the site of infection hence there is rarely a local lesion, although minute papules have been described in children.**

They are localize and multiply in the mononuclear phagocytic cells of spleen, liver, lymph nodes, bone marrow, intestinal mucosa and other organs.

- **1-4 months after infection there is occurrence of fever, with a daily rise to 39-41 C, accompanied with chills and sweating.**
- **Spleen and liver progressively become enlarged**
- **exhibits splenomegaly, distended abdomen and severe muscle wasting**

- **Eyelashes and eyebrows thickening, stiffening and darkening of the Profile view of a boy**
- **With progression of the diseases, skin develops hyperpigmented granulomatous areas (kala-azar: black disease).**
- **Chronic disease renders patients susceptible to other infections.**
- **Untreated disease results in fatal termination.**

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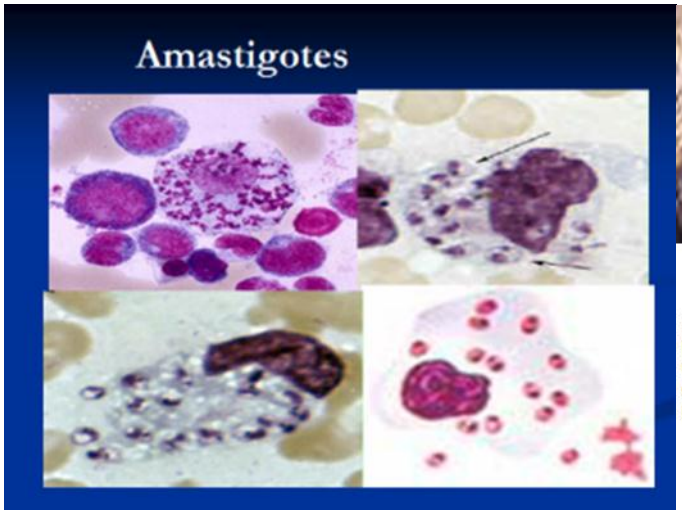


Figure 11A **Many children suffering from visceral leishmaniasis develop a noticeable thickening, stiffening and darkening of the eyelashes and eyebrows.** WHO/TDR/Crump

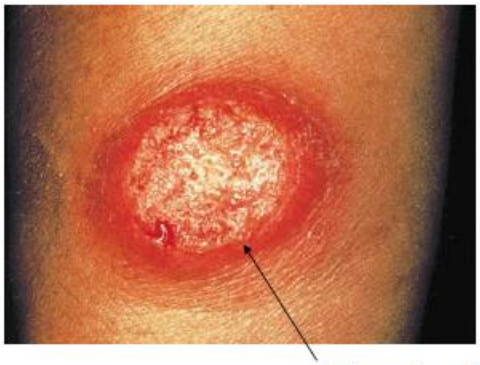
Figure 11B Profile view of a teenage boy **suffering from visceral leishmaniasis. The boy exhibits splenomegaly, distended abdomen and severe muscle wasting.** WHO/TDR/Kuzoe

Figure 11C A 12-year-old boy suffering from visceral leishmaniasis. The boy exhibits splenomegaly and severe muscle wasting. WHO/TDR/El-

Figure 11D Jaundiced hands of a visceral leishmaniasis patient. WHO/TDR/El-Hassan

Cutaneous leishmaniasis (Oriental sore, Delhi ulcer, Baghdad boil):

In cutaneous leishmaniasis, the organism (L. tropica) multiplies locally, producing of a papule, 1-2 weeks (or as long as 1-2 months) after the bite, which gradually grows to form a relatively painless ulcer. The center of the ulcer encrusts while satellite papules develop at the periphery. The ulcer heals in 2-10 months even if untreated but leaves a disfiguring scar. The disease may disseminate in the case of a depressed immune function



Diagnosis

•Biopsy edge of ulcer

•Impression smear -> geimsa

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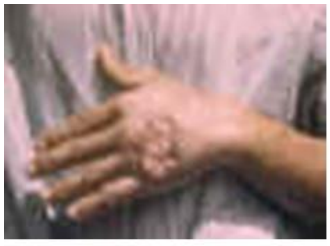

Figure 12A
Skin ulcer due to leishmaniasis, hand of Central American adult.
CDC/Dr. D.S. Martin

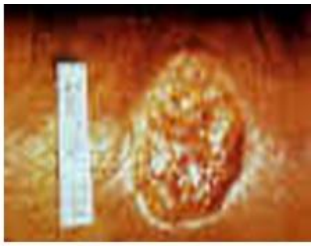

Figure 12B
Crater lesion of leishmaniasis, s

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Mucocutaneous leishmaniasis (espundia, Uta, chiclero):

- The initial symptoms of mucocutaneous leishmaniasis are the same as those of cutaneous leishmaniasis, except that in this disease the organism can metastasize and the lesions spread to mucoid (oral, pharyngeal and nasal) tissues and lead to their destruction and hence sever deformity.
- The organisms responsible are L. braziliensis, L. mexicana and L. peruvianaMucocutaneous leishmaniasis (espundia, Uta, chiclero):

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Pathology: Clinical feathers Pathogenesis of leishmaniasis is due to immune reaction to the organism, particularly the cell mediated immunity. Laboratory examination reveals a marked leukopenia with relative monocytosis and lymphocytosis, anemia and thrombocytopenia. IgM and IgG levels are extremely elevated due to both specific antibodies and polyclonal activation.

Diagnosis: is based on the history of exposure to sand fly, symptoms and isolation of the organisms from the lesion aspirate or biopsy, by direct examination or culture. Skin test (delayed hypersensitivity: Montenegro test) and detection of anti-leishmanial antibodies by immuno-fluorescence are indicative of exposure

Treatment and Control: Pentostam (Sodium stibogluconate) is the drug of choice Pentamidine isethionate is used as an alternative drug.

Control measure involves the vector control and avoidance Immunization has not been effective.

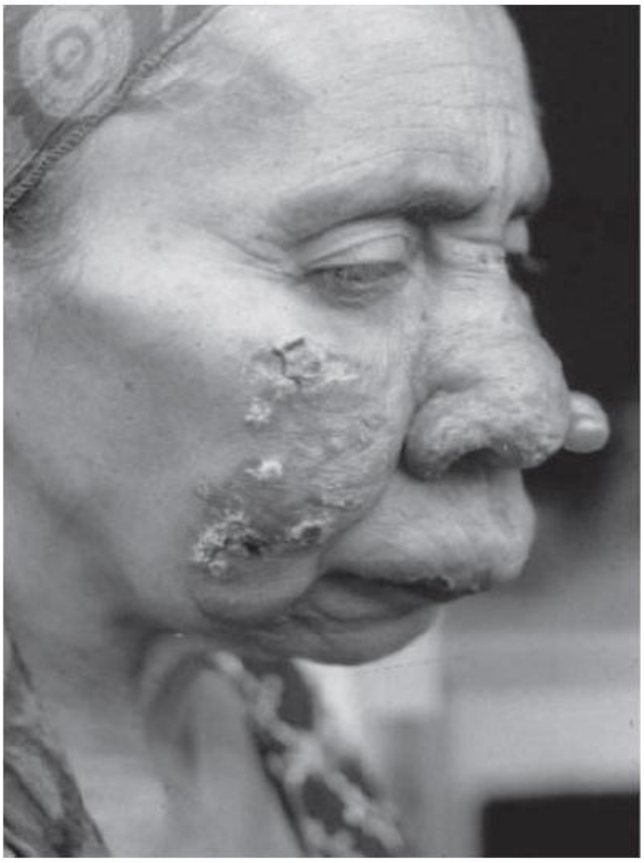

Figure 25.3. Clinical manifestations in a patient suffering from mucocutaneous leishmaniasis caused by *L. brasiliensis*. Courtesy Dr. P. Marsden.

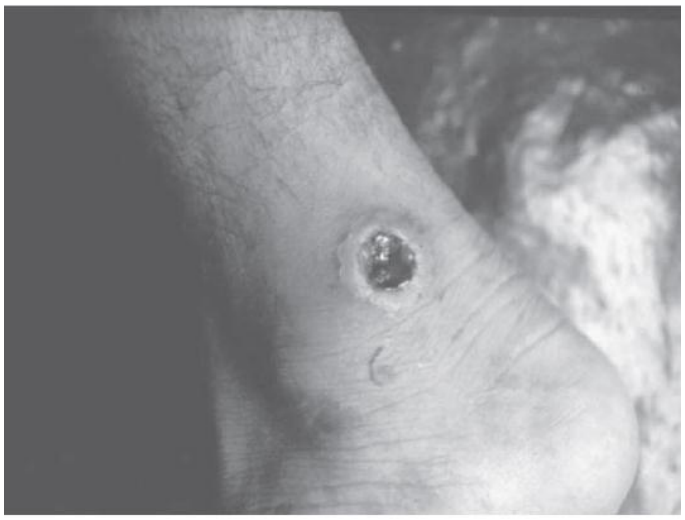

Figure 25.2. A typical ulcerating skin lesion in a patient suffering from localized cutaneous leishmaniasis (LCL). Courtesy Dr. J.R. David.