Leishmaniasis

 A group of diseases caused by protozoa of the genus Leishmania.
 Leiahmaniasis classified into:
1) Cutaneous leishmaniasis.
2) Mucocutaneous leishmaniasis.
3) Visceral leishmaniasis.

1) Cutaneous leishmaniasis

 Is classified into:
a) Old world cutaneous leishmaniasis (oriental sore, Delhi boil)
b) New world cutaneous leishmaniasis.
 The insect (vector) is a sandfly called phlebotomus sandfly in old world cutaneous leishmaniasis and lutzomyia for new world cutaneous leishmaniasis.

a) Old world cutaneous leishmaniasis.

1) Leishmania tropica minor (dry or urban cutaneous leishmaniasis, oriental sore, Aleppo button, Jericho boil, Delhi boil, Baghdad boil).It is anthroponotic disease transmitted from human to human.
2) Leishmania tropica major (rural or wet cutaneous leishmaniasis).It is zoonotic disease.
3) Leishmania aethiopica (cutaneous and diffuse or disseminated cutaneous leishmaniasis of Ethiopia, anergic cutaneous leishmaniasis).

Incubation periods 2 weeks – 3 years
 In Leishmania tropica and Leishmania aethiopica as log as 3 years.
 In Leishmania major 2 weeks.

Life cycle: As in figure 5.

Clinical features and pathogenesis(*Leishmania tropica* and *Leishmania major*):
 The lesion occur at the dermis at the site of the inoculation of the promastigote. Mucous membrane are rarely involved.
 The lesion appears first as macule, then papule with slightly raised center covered by a thin blister like layer of epidermis.
 The lesion then breaks down with discharge of a small amount of clear or purulent exudate. At the ulcer crater like base in the dermis, a granulation layer is formed and the margin becomes indurated by infiltration of fibroblast. **In the dry type** , the disease is chronic ,occurs in the urban area , single lesion ,face is affected ,the ulceration is slow and may not occur with little surrounding tissue reaction ,the healing may take > 1 year. **In the wet type**, the disease is acute, occurs in the rural area, lower limb is affected, the ulceration is multiple and prone to early ulceration with high degree of surrounding tissue reaction and liable for secondary bacterial infection, the healing may take 3-6 months, gerbils and other rodent are the main reservoir.

Clinical types of cutaneous leishmaniasis

- ***Leishmania major*:** Zoonotic cutaneous leishmaniasis: wet lesions with severe reaction
- ***Leishmania tropica*:** Anthroponotic cutaneous leishmaniasis: Dry lesions with minimal ulceration

Oriental sore (most common) classical self-limited ulcer

b) New world cutaneous leishmaniasis (American cutaneous leishmaniasis).

They are caused by Leishmania mexicana complex and Leishmania braziliensis complex. The American strains of leishmania causing cutaneous leishmaniasis differ in their tendency to involve the mucous membrane of the mouth and nasopharynx by extension.

2) Mucocutaneous leishmaniasis

It is caused primarily by *Leishmania braziliensis* which cause **Espundia** start as papule at the site of bite and then metastatic lesion forms, usually at the mucocutaneous junction of the nose and mouth leading to disfiguring
granulomatous ulcerating lesion destroying the nasal cartilage but not adjacent bone. Death occurs from secondary bacterial infection.

Immunity to cutaneous leishmaniasis:

Host recovery in cutanous leishmaniasis depends on the development of cell mediated immunity. The usual cutaneous lesion heals spontaneously.

In certain instances, healing does not occur; these cases may represent the 2 poles of the spectrum of response. The first spectrum is the anergy as in leishmanial aethiopica. The second spectrum represent the hypersensitivity reaction in which the patient is capable for excellent Ab and cellular responses but cannot completely eliminate the parasites , so as the central lesion heals, active peripheral ones continue to form . This stage is called leishmaniasis recidiva or lupoid leishmaniasis (in *Leishmamnia tropica*).

Diagnosis:

1) **Specimens**: Lymph node aspirate, scrapings and biopsies from the margin of the lesion. The center of purulent discharge is of no value.

2) Microscopic examination: The specimen smeared onto a clean glass slide, the slide stained with Giemsa´s stain for demonstration of amastigote within the macrophage or spread out from ruptured cells.

3) Culture in NNN (Novy-MacNeal-Nicolle) medium or inoculation in hamster.

4) Leishmanin skin test (The test done by i.d injection of a suspension of killed promastigote) .

3) Visceral leishmaniasis

The three species are (*Leishmania donovani, Leishmania infantum, Leishmania chagasi*).

 The causative agent is a parasites of the RES not confined to the mucous membrane and subcutaneous tissue but throughout the body.

Dermal leishmaniasis:

Post kala-azar dermal leishmaniasis, present first as hypopigmented or erythematous macules on any part of the body or as nodular eruption especially on the face. The organism may be present in the lesion. It is a delayed hypersensitivity to parasite antigen and is interpreted as an indication of inadequate treatment with residuance of parasites that continue to propagate.

Diagnosis:

1) Specimens. Bone marrow, spleen and lymph node biopsies for detection of intracellular amasatigote (L-D bodies).Splenic aspirates are the most sensitive method for the diagnosis.

2) Culture in NNN (Novy-MacNeal-Nicolle) medium.

Note: The diagnosis is established by visualization of amastigotes in smear, biopsies, or by growth of promastigote in culture.

3) Serological tests: for detection of antigen or antibody.e.g.

I) indirect fluorescent Antibody test (IFAT).

II) Enzyme Linked Immunosorbant Assay (ELISA).

III) Direct agglutination test (DAT).

IV) Immunochromatographic K39 strip test
{Recombinant leishmanial antigens or synthetic
peptides (rK39)} (dipstick test).

The recombinant antigen is a 39 amino acid (rK39) cloned from the C-terminus of the kinesin protein of Leishmania species that cause visceral infecction. This test is used to detect antibodies against K39 antigen in patient with visceral leishmaniasis.

The test is simple, rapid (10 minutes), These tests (serological) remain positive for several months after cure has been achieved, so don’t predictresponse to treatment or relapse.

4) Leishmanin skin test is negative in the acute disease but positive 2 months after recovery.

7) Polymerase chain reaction (PCR).

Immunity:

Lifelong immunity.