Visceral Leishmaniasis

The disease is caused by *L. donovani*, *L. infantum* and *L. chagasi* in different geographical regions. The natural habitat of these species in man is reticuloendothelial system especiallyspleen, liver, bone marrow, intestinal mucosa. It may be found in endothelial cells of kidneys.

- The parasite exist in two forms:
- Amastigote Form
- In this form this parasite is found in the cells of reticulo endothelial system of vertebrate hosts like man, dog, hamster.



- Promastigote Form
- They are found in sandfly and in cultures.



- Mode of Transmission Natural transmission is by the bite of sandfly (*Phlebotomus*).
- Mother to fetus (verticaltransmission).
- Blood transfusion.
- Accidental inoculation of culture in the laboratory.



Pathogenicity

- *L. donovani* causes visceral leishmaniasis or kalaazar.
- Spleen:
- The spleen is the most affected organ. It is enlarged and the capsule is thickened
- Liver:
- The liver is enlarged. The Küpffer cells and vascular endothelial cells are heavily parasitized, but hepatocytes are not affected. Liver function is, therefore, not seriously affected.
- The bone marrow
- Is heavily infiltrated with parasitized macrophages, which may crowd the hematopoietic tissues.
- **Peripheral lymph nodes** and lymphoid tissues of the nasopharynx and intestine are hypertrophic.

Anemia

- with hemoglobin levels of 5–10 g/dL may occur
- infiltration of the bone marrow with the parasite
- increased destruction of erythrocytes due to hypersplenism.
- Autoantibodies to red cells may contribute to hemolysis



Clinical Features of Kala-azar

- The clinical illness begins with fever, which may be continuous, remittent, or irregular.
- **Splenomegaly** starts early and is progressive and massive.
- Hepatomegaly and lymphadenopathy also occur but are not so prominent.
- Skin becomes dry, rough, and darkly pigmented (hence, the name Kala-azar).
- **Cachexia** with marked by anemia, emaciation, and loss of weight.
- **Epistaxis** and bleeding from gums are common.



Post Kala-azar Dermal Leishmaniasis

- About 3–10% cases of patients of visceral leishmaniasis in endemic areas develop PKDL, about year or 2 after recovery from the systemic illness. The lesions are of 3 types.
- **Depigmented macules:** These commonly appear on the trunk and extremities and resemble tuberculoid leprosy.
- Erythematous patches: These are distributed on the face in a 'butterfly distribution'.
- Nodular lesion: Both of the above mentioned lesions may develop into painless yellowish pink Nonulcerating granulomatous nodules. The parasite can be demonstrated in the lesions.





Post kala- azar dermal leishmaniasis



Cutaneous leishmaniasis

- Leishmania tropica, L. major and L. aethiopica
- Amastigote in reticuloendothelial cells of skin. Promastigote form in sandfly (*Phlebotomus*).
- Pathogenicity
- Promastigote enters through punctured wound and transform into amastigote form inside histiocytes and endothelial cells.
- Inflammatory granulomatous reaction occurs with infiltration of lymphocytes and plasma cells.
- As a result of disturbance of blood supply, necrosis and then ulcer formation occur.

Clinical Features

- It causes disease called oriental sore, Delhi boil, Baghdad boil. Lesions are cutaneous (exposed parts).
- Three distinct patterns of old world cutaneous leishmaniasis have been recognized.
- The anthroponotic urban type causing dry ulcerating lesions, leading to disfiguring scars, caused by the species *L. tropica*. It begins as a raised papule, which grows into a nodule that ulcerates over some weeks. The dry ulcers usually heal spontaneously in about a year.
- **The zoonotic rural type** causing moist ulcers which are inflamed, often multiple, caused by *L. major*. Lesions due to *L. major* heal more rapidly than *L. Tropica*
- The non-ulcerative and often diffuse lesions caused by *L. aethiopica* and seen in the highlands of Ethiopia and Kenya are known as diffuse cutaneous leishmaniasis.