LEISHMANIASIS

Protozoan organisms of the genus Leishmania cause infections in humans and animals. Several pathogenic species of Leishmania are recognized. T he diseases, however, may overlap in clinical signs or lesions.

Leishmania occur in vertebrate hosts within parasitophorous vacuoles in **Macrophages and Reticulo-Endothelial Cells** as **Amastigotes (Leishmanial Forms)**. These are small oval protozoans without a flagellum. In Romanovsky-stained (Leishman, Giemsa, or Wright) preparations, they have pale blue cytoplasm containing, near the posterior end, a reddish nucleus. Anterior to the nucleus is a deep violet, rod-shaped body, the **Kinetoplast**. In the invertebrate hosts (Sand Flies), the organisms take shapes which vary from the Leishmanial Form to the **Leptomonad Form**. The Leptomonad Forms are slender and spindle-shaped and are Motile by means of a flagellum.

LIFE CYCLE:

Leishmania reproduce in the vertebrate host by binary fission. However, the complete life cycle and maintenance of virulence depend upon an intermediate host or vector and this MAIN VECTOR are blood-sucking **Sand Flies (Phlebotomus spp. etc)** where parasites undergo morphological transformation and Multiplication. So these vectors are involved in the transmission of Leishmania, and are necessary for their perpetuation. However, certain flies, such as ***Stomoxys calcitrans****, also* may transmit the infection mechanically.

Leishmania is ovoid-organism within Macrophages, which possesses a Rod-shaper Kinetoplast associated with rudimentary Flagellum, that does not extend beyond cell margin. This is called **Leishmanial Form or Amastigote Form**, which after ingestion by Sandfly (vector), transforms into a **Promastigote Form or Leptomonad Form** inside Insect-Gut in which Kinetoplast is situated at the Posterior End of the Body. These divide repatedly by Binary Fission and migrates to Proboscis – and when insect subsequently feed on host – these PromastIgotes are inoculated into a new host. Once reach within a Macrophage, the Promastigote reverts to Amastigote form and again starts to divide.

CLINICAL FORMS :-

Leishmaniasis is divided into three major clinical forms:

1. **Visceral Leishmaniasis** / "**Kala-Azar**" or "Dum-Dum Fever" [caused by *Leishmania donovani]* – Infection is Systemic, Hemolytic Anemia.
2. **Cutaneous Leishmaniasis** / 'Old World Cutaneous Leishmaniasis', **'Oriental Sore'**, **'Delhi Sore'**, or 'Baghdad Boil' (caused by *Leishmania tropica* or *Leishmania major),* - Lesions found at site of INSECT Bite, and
3. **Mucocutaneous**. Leishmaniasis / **'New World Cutaneous** Leishmaniasis' or 'American Leishmaniasis', [caused by *Leishmania braziliensis* or *Leishmania Mexicana].*

VISCERAL LEISHMANIASIS :-

Also known as "Kala-azar" and "Dum-dum fever", this form is caused by *Leishmania donovani.* It is more common form AND occurs naturally in humans, dogs, cats, cattle, horses, and sheep. It is quite prevalent in India.

Dogs initially develop "**Spectacles"** due to Depilation of Hairs around the Eyes, followed by Generalized Loss of Body Hair and Eczema.

Visceral leishmaniasis in animals occurs usually as a Chronic Debilitating Disease with periods of Fever, Cachexia, gradual weight loss, General Lymphadenopathy, Anaemia, and Leukopaenia.

Lymph nodes are enlarged, and there is splenomegaly and hepatomegaly. Usually the infection slowly progresses to death.

The lesions are characterized by massive infiltration of various organs with huge macrophages whose cytoplasm is filled with leishmaniae. The architecture of affected lymph nodes and spleen, may be completely obscured by the phagocytic cells and large numbers of plasma cells also. Liver, bone marrow, kidneys, lungs, gastrointestinal tract, and less often, other organs and skin, may be affected.

Gross lesions at postmortem consist of enlarged lymph nodes, spleen and liver, Pallor (Paleness) of Mucosal and Serosal Surfaces, soft red bone marrow, and Ulcers of the Intestine.

CUTANEOUS LEISHMANIASIS :-

This is caused by *Leishmania tropica* or *Leishmania major,* and occurs mainly in countries bordering Mediterranean region. It is also known as 'Old World cutaneous leishmaniasis', 'Oriental sore', 'Delhi sore', or 'Baghdad boil'. The reservoirs for human infection are various wild rodents.

The infection is characterized by single or multiple very **slowly developing, shallow nodules or ulcers of the skin, often on the Lips or Eyelids**. The lesions are characterized by infiltration of the skin with Macrophages accompanied by lymphocytes, plasma cells and rarely eosinophils. Numerous parasites are present within the Macrophages. Lesions of long-standing are surrounded by fibroblastic connective tissue. This gives them the appearance of a **Typical Granuloma**.

MUCOCUTANEOUS LEISHMANIASIS:

Also known as 'New World cutaneous leishmaniasis' or 'American leishmaniasis', mucocutaneous Leishmaniasis is caused by *L. braziliensis* or *L. mexicana.* It occurs in Mexico and Central and South America. Animals are usually not found infected, although dogs, cats, and monkeys are susceptible. The infection resembles cutaneous leishmaniasis, but in addition to the skin, chronic ulcers often occur at mucocutaneous junctions and may occur in oral and nasal cavities. The lesions are similar to those of cutaneous leishmaniasis.

DIAGNOSIS

- Demonstration of Amastigote parasite in Smears or Skin Scrappings or from Lymph Node aspirates.
- Infected macrophages may break-up mechanically due to proliferation of the parasite inside. The liberated organisms, are called **Leishman-Donovan Bodies**, and on staining with Giemsa stain - may be found free in Lymph or Plasma. Can be demonstrated in smears of deep tissues at edge of lesions.

DIFFERENTIAL DIAGNOSIS: Diseases which also cause proliferation of reticulo-endothelium, such as Toxoplasmosis, Histoplasmosis, Blastomycosis, and Epizootic Lymphangitis, present problems in differential diagnosis.

Final diagnosis must be based upon demonstration and identification of the causative organisms in tissue sections, smears or cultures. Identification of the kinetoplast and the nucleus, which are not present in the organisms of the diseases mentioned above, allows differentiation.

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Trichomonas foetus

Venereally transmitted, multiflagellated organism of reproductive tract of cattle.

Host: Cattle

Site: Prepucial Cavity & Penile Membranes in BULLS; The Uterus or Vagina in Cows

Transmission: Through COITUS

Signs:

In Cows, Early Abortions in 1st Trimester in characteristic feature; Retention of Placenta; Purulent Endometritis, Pyometra,

In Bulls, Small nodules on prepucial and penile membranes

Trichomonas gallinae

Host: Pigeon ; Turkeys; Chicken
Site: Yellow, Necrotic Lesions on Mouth, Oesophagus & Crop
Transmission: Through regurgitated crop contents of adult birds
Signs: Yellow, Necrotic Lesions on Mouth, Oesophagus & Crop in Pigeons.