PATHOGENICITY

- Disease is reticuloendotheliosis.
- Incubation Period- Varies 3-6 months.

PATHOGENICITY

Cellular & humoral proliferation.

Marked proliferation of macrophages .

In bone marrow , spleen, liver, lymph nodes, spleen

CLINICAL FEATURES

- Fever
- Malaise
- Headache
- Progressive enlargement of liver, spleen, lymph nodes.
- Skin is dry ,rough & pigmented .
- Hair.

VISCERAL LEISMANIASIS SHOULD

BE INCLUDED IN THE LIST

OF OPPORTUNISTIC INFECTIONS

LABORATORY DIAGNOSIS

- Non-Specific Laboratory Test
- Blood count
- Haemoglobin Estimation
- Estimation of Serum Proteins
- Parasitological Diagnosis
- Peripheral Blood Film By thick film Method
- Needle Biopsy
- Culture of blood and FNAC
- Animal Inoculation

LABORATORY DIAGNOSIS

- Immunological Test
- Non-specific Tests
- Aldehyde Test, Antimony Test

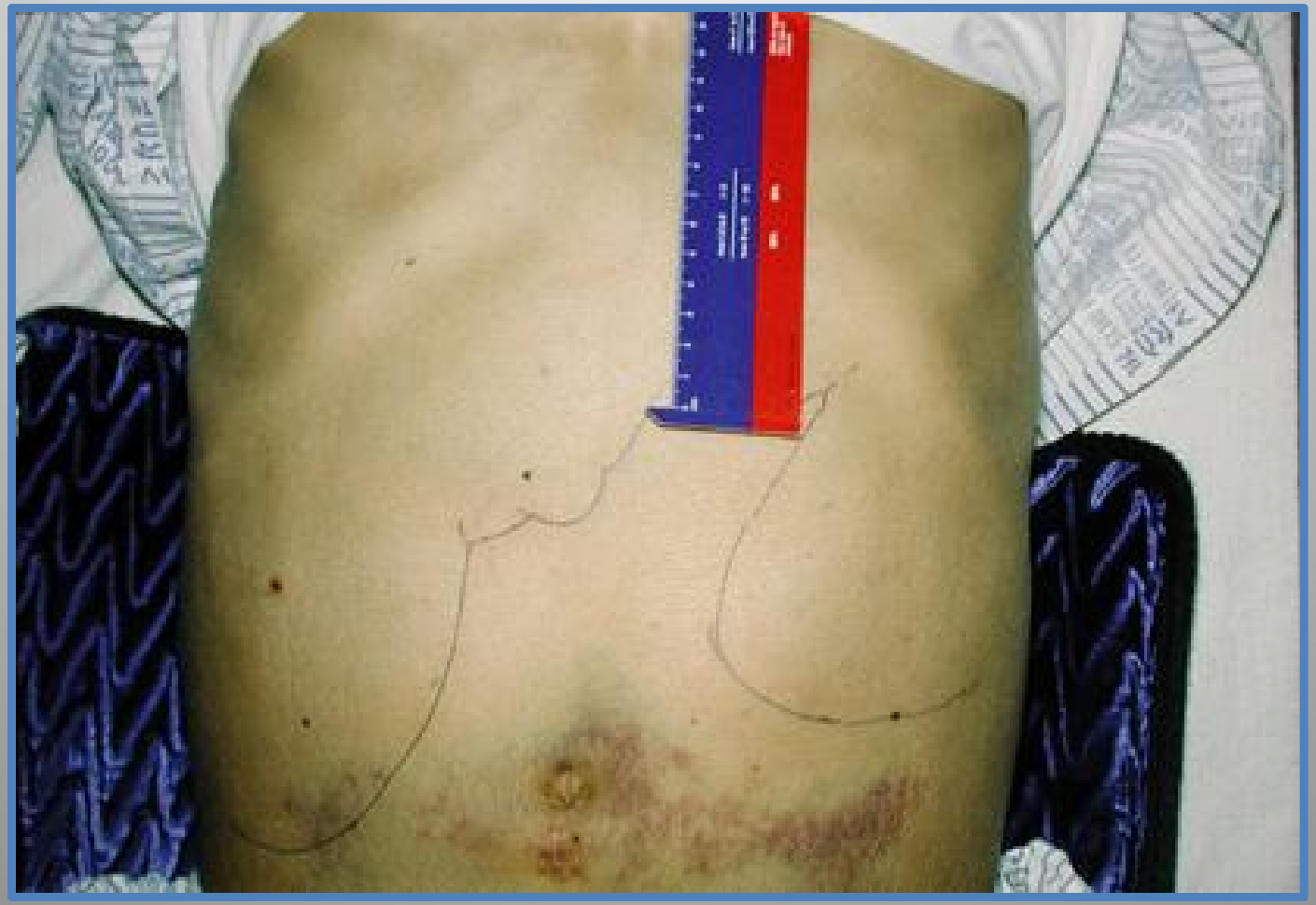
- Specific Tests
- DAT, IHA, IFAT, ELISA
- Leishmanin or Montenegro Skin Test

POST KALA-AZAR DERMAL DISEASE (PKDL) OR DERMAL LEISHMANOID

- It is a common consequence of theraputic cure from visceral Leishmaniasis caused by L. donovani.
- Develops 1-2 years after completion of antimonial treatment.

LEISHMANIASIS

LEISHMANIASIS



LEISHMANIA TROPICA

- It causes cutaneous leishmaniasis.

HABITAT

- Inside reticulo endothelial cells (dermatocytes) of skin.

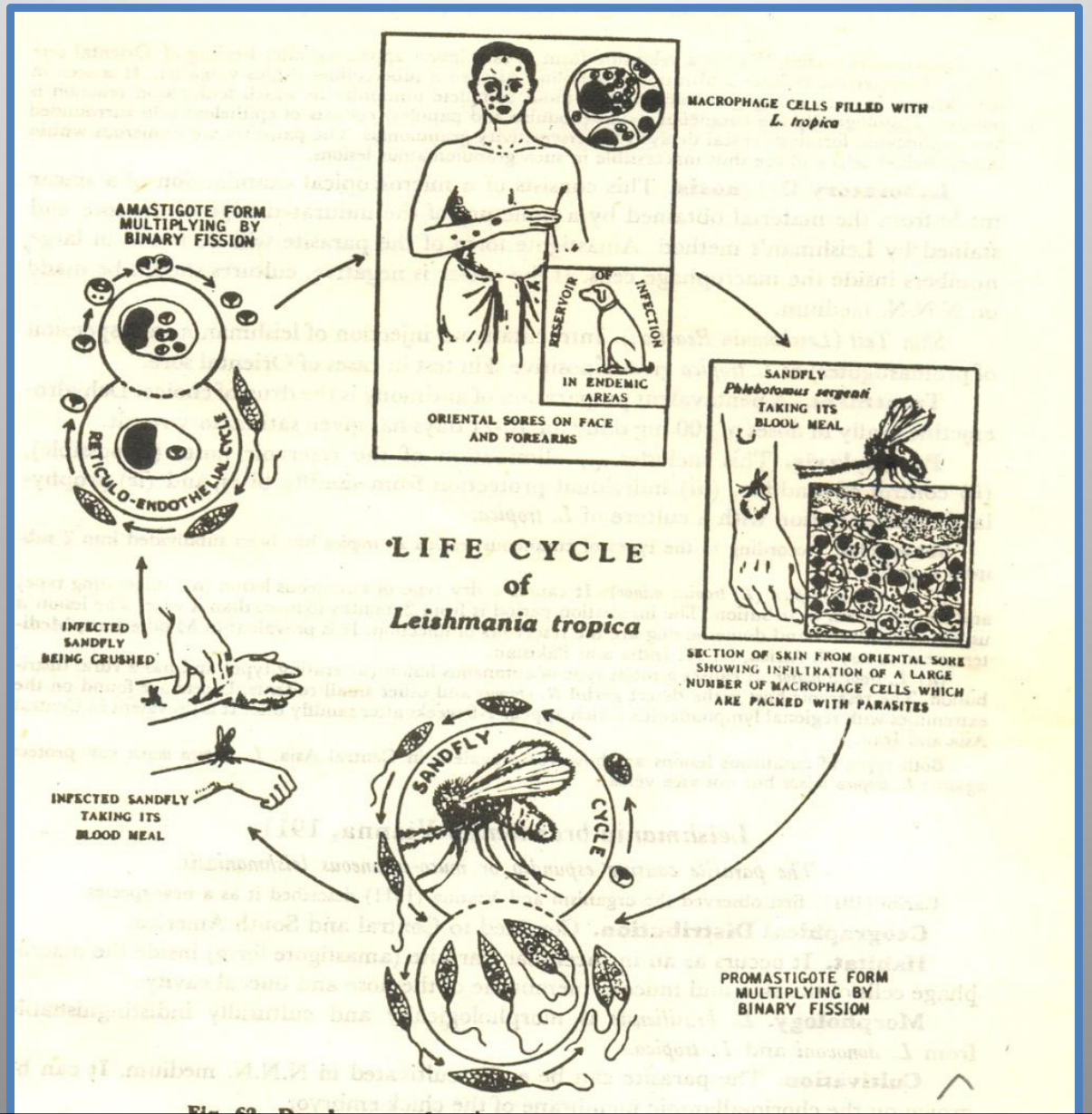
MORPHOLOGY

Same as that of Leishmania donovani.

LIFE CYCLE

- Vertebrate host; man.
- Invertebrate host; P. sergenti.
- Life cycle as that of Leishmania donovani except that amastigote forms reside in reticulo endothelial cells of skin and not in the viscera.

LIFE CYCLE OF LEISMANIA TROPICA



PATHOGENICITY

- Incubation period; A few weeks to six months.
- Leishmania tropica causes urban orthoponotic cutaneous leishmaniasis or oriental sore or Delhi boil.
- At the site of Inoculation promastigotes are phagocytosed by RE cells of skin and are transformed into amastigotes→ leishmanioma develops → delayed hypersensitivity.

CLINICAL FEATURES

- A raised papule is formed, it ulcerates and heals in about six months leaving a depressed scar.
- The sores are distributed on the exposed parts of body.
- It causes dry lesions.

LABORATORY DIAGNOSIS

- Microscopic examination shows amastigote forms of parasites.
- Biopsy
- Promastigotes may be isolated in NNN culture medium and Hockmeyer’s medium.
- Leishmanin skin test is positive.

TREATMENT

- **Pentavalent antimonials**;
  - Sodium stibogluconate
  - Meglumine antimoniate
- **Aromatic diamidines**
  - Pentamidine
- **Others**
  - Monomycin
  - Paromomycin
  - Aminosidine
  - Amphotericin B
  - Allopurinol

LEISHMANIA INFANTUM

- Main vertebrate host –domestic dog which may develop acute /chronic disease.
- Dog carries abundant parasites in the skin.
- Infantile visceral leishmaniasis.
- Age of child <2 years.
- May also infect adults with HIV .

LEISHMANIA AETHIOPICA

- Mammalian hosts are rock hyraxes.
- Sand fly host is P. longipes.
- In man may cause cutaneous leishmaniasis & DCL.
- Lesions are more swollen & less necrotic than those of L. tropica.
- Gradual scaling / exfoliation of dermis at the center of lesion.
- Because of appearance of lesions they resemble lepromatous leprosy.
- This condition is difficult to treat and my last for the rest of life .

LEISHMANIA MAJOR

- Mammalian host –great gerbil & fat sand rat.
- Sand fly vector –P papatasi & P .salehi.
- This infection occurs in epidemics in people inhabited in zoonotic focci.
- It causes rural zoonotic cutaneous leishmaniasis or oriental sore.
- Wet lesions > 100 in number.
- Produces delayed type of hypersensitivity response.
- Diagnostic methods are same as that of L . tropica.

PROPHYLAXIS

- Active case detection & treatment.
- Elimination of sand flies by spraying of insecticides.
- Insect repellents applied to exposed parts of skin.
- Use of fine mesh bed nets.
- Sleeping at night on the roof.
- Destruction of desert rodents.
- Elimination of dogs .
- To prevent transmission to other persons cover the lesions /ulcers with gauze pieces.
- Avoidance of visit to areas of high risk.

NEW WORLD LEISHMANIASIS

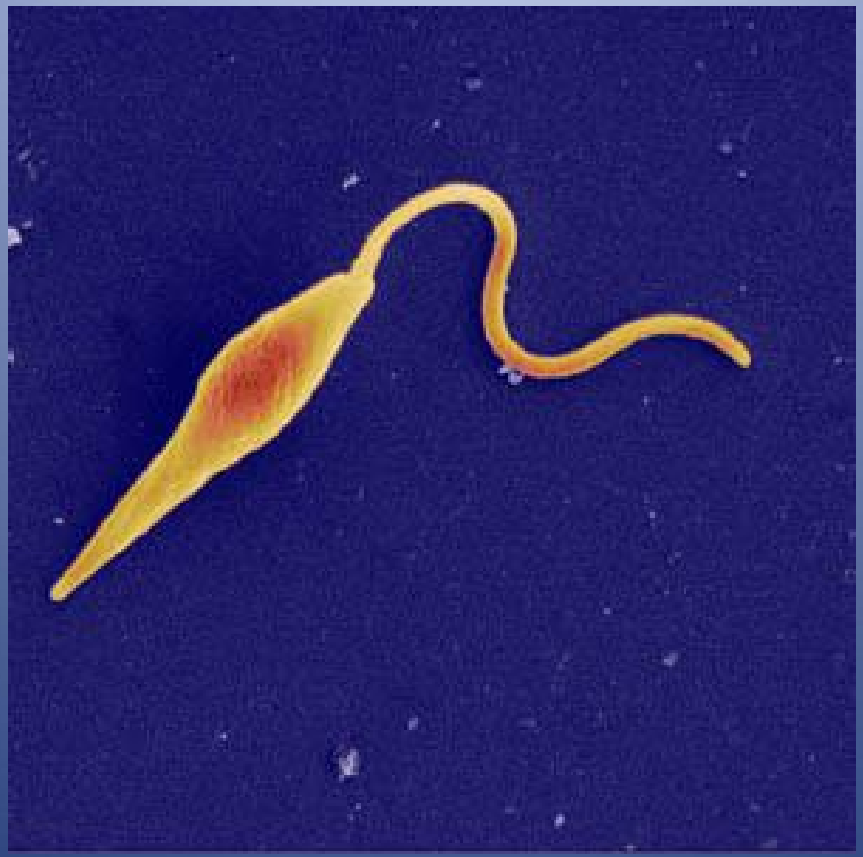
- LEISHMANIA BRAZILLIENSIS COMPLEX
- LEISHMANIA MEXICANA COMPLEX

• HABITAT

- Occurs as intracellular parasite (amastigote form) inside macrophages of skin and mucous membranes of nose & buccal cavity.

MORPHOLOGY

- Same as that of other species.



LIFE CYCLE

- It is same as that of other species of Leishmania except that ;

i. Amastigote form occurs inside macrophages of skin and mucous membranes of nose & buccal cavity.

ii. Vector is sand fly of genus Lutzomyia & Psychodopygus.

PATHOGENICITY

- Both cause zoonotic disease.
- L .braziliensis complex causes muco cutaneous or espundia ulcers.
- Initial lesion enlarges radially forming a weeping ulcer with a clean cut margin.
- L .braziliensis sub species form metastatic lesions in nasal , pharyngeal & laryngeal mucosa.
- Papulo pustular swellings → cause destructive and mutilating erosions which heal by scarring → producing camel nose.

PATHOGENICITY

- In L. mexicana complex parasite provokes an intense reaction at the site of bite.
- After 7-10 days a tiny papule appears which produces a crater-like lesion with inflamed borders
- Chiclero's ulcer.
- It produces secondary lesions.

PATHOGENICITY

- L. braziliensis complex causes espundia ulcers.
- Metastatic lesions in nasal, laryngeal and pharangeal mucosae.

LEISHMANIASIS

Disseminated Cutaneous Leishmaniasis: A Patient with 749 Lesions

LABORATORY DIAGNOSIS

- Amastigote forms in punctured material from nodule or edge of an ulcer.
- NNN culture medias are used.
- IFA in 89% - 95% is positive.
- ELISA is also positive in 85% cases.
- Montenegro skin test is positive.

TREATMENT

- Treatment and prophylaxis of new world leishmaniasis is same as that of old world leishmaniasis.