Parasitology

Lecture: 3

**New world cutaneous Leishmanianiasis**

It is called American cutaneous leishmaniasis, which is caused by:

1- *L. mexicana* complex
2- *L. braziliensis* complex

The vector is sand fly genus *Lutzomyia*

1- **Lesions caused by *L. mexicana* complex**

*L. mexicana* lesions are usually single, self-limiting cutaneous papules, nodules or ulcers on face and ears (about 60%), they are painless and usually heals within few months.
Ulcerative lesions on the ears (Chiclero’s ulcer) may cause destruction of the ear cartilage.
Reservoir hosts are rodents and dogs.
Sometimes lead to diffuse cutaneous leishmaniasis.

2- **Lesions caused by *L. braziliensis* complex**

(*MucocutaneousLeishmaniasis (Espundia)*)

Produced by species belongs to *Leishmania braziliensis* complex. It is distributed in South Africa. The cutaneous lesions develop exactly as does the oriental sore, but they are more frequently multiple and may become large. Secondary infection plays a prominent role in the persistence of these ulcers, sometimes ulceration of the adjacent areas extend to involve mucosal surfaces, but more frequently the lesions seem to develop by metastasis.
Nasal mucosa and that of the hard and soft palate is affected.
The nasal septum is destroyed but does not involve the bone, ulceration may result in loss of all the soft parts of the nose, the lips, and the soft
palate and may lead to severe facial disfiguration. Death usually occurs from secondary infection. Various rodents and dogs are infected; sand flies belonging to the genus Lutzomyia are vectors.

**Visceral Leishmaniasis (Kala-azar) (Dum-Dum fever, black fever)**

It involves different age groups in various geographical areas. VL is divided into four clinical and epidemiological (ecological) forms:-

1- **Mediterranean (Infantile) type**
   - Sporadic cases.
   - Distributed in the Mediterranean basin, Middle East, former southern USSR and northern China (this type is found in middle and south of Iraq).
   - Affects chiefly infants and young children (especially under 5 years).
   - Reservoir hosts are domestics and wild dogs (fox and Jackal), so it is a zoonotic disease.
   - Caused by *L. Infantum*.

2- **Indian (classical) type**
   - Epidemic disease.
   - Distributed in Indian subcontinent.
   - Man is the reservoir host, lack of an animal reservoir host (not a zoonotic disease).
   - Seen in young adults and adolescents.
   - Frequent presence of amastigote in peripheral blood (circulating macrophages).
   - Caused by *L. donovani*.

3- **African type (Sudanese) type**
   - Seen in adults men.
• Rodents and dogs are the reservoir hosts (zoonotic disease).
• Distributed in Sudan and East Africa.
• Presence of the parasites in peripheral blood is less common.
• Caused by *L. donovani* (as Indian type).

4- **South America type**
• Distributed in central and south America.
• Affects mainly children.
• Reservoir host are foxes, domestic dogs and cats (zoonotic disease).
• Caused by *L. chagasi*.

**Mode of transmission**
1- Vector born transmission (by insect bite): represents the most common mode of transmission, while the others are rare.
2- Blood transfusion.
3- Needle sharing.
4- Congenital transmission (not transplacental transmission) but during the exchange of the mother’s blood at the time of passage of the fetus through birth canal → transmission occurs.
5- Sexual transmission.
6- Laboratory acquired transmission: from needle stick injuries, handling contaminated specimens and some to oral exposure.
7- Person to person transmission: via contact with infected fluid (nasal and oral secretions, tonsillopharyngeal mucous and urine) of patient with VL.

**Clinical presentation**
Incubation period vary from 2 weeks to 18 months.
Visceral leishmaniasis characterized by pentad of irregular faver, hepatosplenomegaly, pancytopenia (leucopenia, anemia and thrombocytopenia), hypergammaglobulinemia and weight loss.
However after infection most cases remain asymptomatic or associated with symptoms that resolved spontaneously, but the disease (VL) may be fetal (95%) if left untreated. Other clinical manifestations may include paleness, ecchymosis, epistaxis and gingival bleeding, oedema, ascitis, skin hyper-pigmentation, jaundice, abdominal distension and lymphadenopathy.

Pathogenesis

- The parasite (amastigotes of various species of *L. donovani* complex) infect RE cells throughout the body.
- Proliferation of the RE cells, particularly of the spleen and liver leads to massive hepatosplenomegaly (hypertrophy of these organs).
- The bone marrow may be involved, resulting in anemia and leucopenia.
- Hypoalbuminemia → oedema.
- Gamma globulins may constitute 60% to 70% of the serum proteins → reverse albumin/globulin ratio.
- Splenomegaly with stasis of blood in the sinusoids may results in increased destruction of both red and white blood cells resulting in anemia and leucopenia.
- Recovery from leishmanial infection is generally believed to involve interaction between T lymphocytes and macrophages.
- Melanocyte stimulation → hyperpigmentation → so it is called Kala-Azar which means black fever.
- Thrombocytopenia → lead to severe mucosal bleeding, epistaxis and ecchymosis.
- Death occurs usually due to secondary infection as dysentery, pneumonia and tuberculosis, due to disturbed immunity.
Post Kala-azar dermalleishmaniasis (PKDL) (Dermal Leishmaniod)

It is a dermatotropic form of leishmaniasis, it is found in areas where *L. donovani* is the causative agent of VL (Indian and African types) and develops after 2-10 years from cure of VL.

Three clinical type of PKDL are encountered:

1- Depigmented macules.
2- Erythematous patches.
3- Nodular lesions.

Mucocutaneous Leishmaniasis
(Espundia)
L.D. bodies in tissue section
Patient with kala-azar, hepatosplenomegaly