Old World cutaneous leishmaniasis (oriental sore) also called Delhi boil, Baghdad boil, Aleppo boil, is produced by Leishmanias belonging to *Leishmania tropica* complex. There are 3 serologically and biochemically distinct species of *L. tropica* complex:

1. *L. tropica*
2. *L. major*
3. *L. aethiopica*

All are transmitted by sand flies belonging to the genus *Phlebotomus*.

1. **L. tropica**: also known as *L. tropica minor* (causes dry sore or urban cutaneous leishmaniasis).
   - Produce chronic disease that if not treated, lasts for year or longer.
   - with 2month – 3year incubation period
   - It is characterized by the production of dry lesions that ulcerate only after several months
   - Lesions are usually single and occur primarily on the face.
   - It is found in urban areas, it is found in Iraq and around the Mediterranean basin and in Asia Minor, Afghanistan, India and Kenya.
   - The dog may be a natural host, but it is not thought to be an effective reservoir for humans.

2. **L. major**: also called *L. tropica major* (causes wet sore or rural cutaneous leishmaniasis).
• Produces an acute infection with duration of 3-6 months.
• With as little as 2 weeks incubation period.
• The lesions occur primarily on the lower limb.
• The lesions are moist and tend to ulcerate very early.
• There may be secondary or satellite lesions.
• *L. major* occurs in Asia Minor, Middle East (Iran, Syria, Palestine and Jordan) and in north and middle Africa.
• It is primarily a disease of rural areas.
• Reservoir hosts (Rodents) are important source of human infection.

3- *L. aethiopica* (causes diffuse cutaneous leishmaniasis):
• Produces a chronic disease similar to that caused by *L. tropica*.
• Seen in the high lands of Ethiopia and in Kenya and possibly in south Yemen.
• The Rock hyrax is a reservoir host of this specie

**Symptoms**
The first sign of the infection is a small red papule, which may itch intensely and grow to 2 cm or more in diameter. In *L. major* infection, the papule is covered with serous exudates (moist lesion) and ulcerates early. The papules are dry (dry lesions) and ulcerate only after several months in *L. tropica* and *L. aethiopica* infection.

In uncomplicated cases there are no systemic manifestations and since the disease is self-limiting, the patient seldom seeks medical assist. Ulcer may associate with local disfiguration, pyogenic complication, pain and some time septicemia. Although the usual cutaneous lesion (dry or moist) healssspontaneously, in certain instances such healing does not occur by itself.
These cases (no spontaneous healing) may be considered to represent the two poles of the spectrum of response:

1- Anergy
2- Hypersensitivity

1- The **anergic patient** is incapable to produce a response to infection, which therefore (amastigotes) can proliferate indefinitely and forming many lesions filled with parasites. This type of disease, known as diffuse cutaneous leishmaniasis is probably the results of:
   a) A deficient cell-mediated immunity.
   b) Some characteristics of the parasite itself, as it is seen primarily in infections caused by *L.aethiopica* and *L.Mexicana*.

2- The **hypersensitive patient** is capable of excellent antibody and cellular responses but cannot completely eliminate the parasites, so as the central lesion heals, active peripheral ones continue to
form. This type of response known as leishmaniasis recidiva, may be seen with any of the cutaneous leishmaniasis.

**Pathogenesis**
When the bite of infected sand fly liberates promastigotes into the skin → the parasite proliferates as amastigotes in the macrophages and other endothelial cells of the capillaries and small blood vessels of immediate area → lyses of the amastigotes occurs following activation of the macrophages by sensitized lymphocytes → a granulomatous reaction results in the formation of a localized nodule → which ulcerate when the blood supply to the area is compressed by the parasite-induced damage → a pyogenic infection develops in the open ulcer bed, and as host immunity increase → the ulcer heals.

**Resistance to re-infection**
Resistance to re-infection with the same species following primary infection is nearly absolute. Infection with *L. major* protects the host against subsequent *L. tropica* infection, but infection with *L. tropica* does not counter the same immunity to subsequent challenge with *L. major*.

**Mode of transmission**
1- Insect bite: a main mode of transmission (vector born transmission).
2- Blood transfusion: a rare mode of transmission.

**Diagnosis**
1- Usually made in endemic areas on clinical grounds
2- Microscopic detection of amastigotes (L.D. bodies) within large monocytic cells in Giemsa stained smear obtained by aspiration of fluid from beneath the ulcer bed, especially its active borders.
Scraping taken from the ulcer surface do not reveal the organisms, which are destroyed in areas secondarily infected with bacteria.

3- Culture: on NNN (Novy-MacNeal-Nicolle) media or other media. Culturing of material obtained by aspiration or biopsy may demonstrate promastigote forms.

4- Animal inoculation: aspirate or biopsy material may be inoculated subsequently into the nose of a hamster and the animal watched for nasal inflammation.

5- Leishmanin skin test (Montenegro test): involves the forearm intradermal injection of 0.1 ml suspension of killed promastigote, this test is used to measure delayed hypersensitivity. Positive result is indicated by an induration of 5mm or more in 48-72 hours. In cutaneous leishmaniasis this test is positive.

6- Immunological test (serology): has limited role in diagnosis because patient shows no detectable level of circulating antibodies.

**Treatment**

1- Sodium stibogluconate (Pentostam)

2- Pentamidine (isothionate)

3- Meglumineantimoniate

4- Amphotericin B
Dry sore (Urban cutaneous leishmaniasis)
Wet sore (Rural cutaneous leishmaniasis)