University of Tikrit

College of nursing

Basic Nursing Sciences

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Parasitology

Cutaneous Leishmaniasis

By:

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Cutaneous Leishmaniasis (Oriental sore, Baghdad boil)

presents as skin lesions, which are generally localized, without involvement of the mucosa, and not generalized infection. They occur on exposed parts of the body accessible to sandflies: face, hands, forearms and lower limbs.

• Incubation period ranges from weeks to months

• Starts as small ,red papule at the site of the bite. This may disappear in afew weeks, but usually develops a thin crust that hides a spreading ulcer underneath. This lesion becomes irritated, with intense itching, and begins to enlarge & ulcerate.

• Gradually the ulcer becomes hard and crusted and exudes a thin, serous material. At this stage, secondary bacterial infection may complicate the disease It can be ulcerative or dry with papulo nodular lesion covered by scales.

Laboratory diagnosis:

1- diagnosed by direct observation of the parasites in skin scrapings, impression smears or skin biopsies stained with Giemsa.

2- Polymerase chain reaction assays (PCR) are often used for diagnosis in areas where they are available.

3- *Leishmania* spp. can also be cultured.

Mucocutaneous leishmaniasis (Uta) *Leishmania braziliensis*

It is the same as oriental sore. But some of the strains tend to invade the mucous membranes of the mouth, nose, pharynx, and larynx either initially by direct extension or by metastasis. The metastasis is usually via lymphatic channels but occasionally may be the bloodstream.

The lesions are confined to the skin in cutaneous leishmaiasis and to the mucous membranes, cartilage, and skin in mucocutaneous leishmaniasis. A granulomatous response occurs, and a necrotic ulcer forms at the bite site. The lesions tend to become superinfected with bacteria. Secondary lesions occur on the skin as well as in mucous membranes. Nasal, oral, and pharyngeal lesions may be polypoid initially, and then erode to form ulcers that expand to destroy the soft tissue and cartilage about the face and larynx.

Laboratory diagnosis:

• Demonstration of the amastigotes in properly stained smears from touch preparations of ulcer biopsy specimen.

• Serological tests based on fluorescent antibody tests.

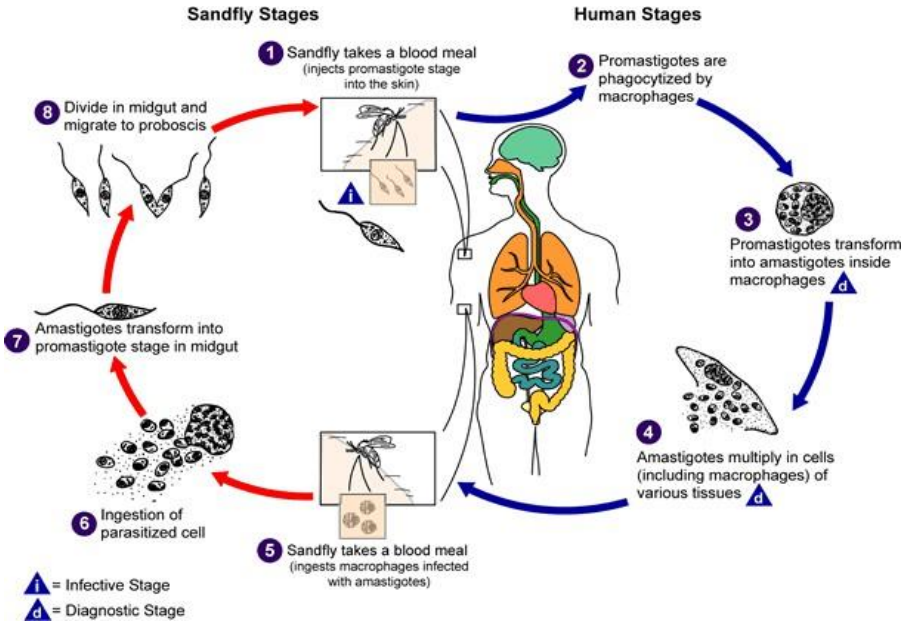
• Leishman skin test in some species.

Treatment

The drug of choice is sodium stibogluconate, a pentavalent antimonial Compound ,Alternative approaches include the addition of allopurinol and the use of amphotercin B.

Prevention

1- Prompt treatment of human infections and control of reservoir hosts.
2- Protection against infective bites of sand flies screening and insect repellents.
3- Health education.
4- Avoiding endemic areas especially during times when local vectors are most active.



Sporozoa:

There are four species normally infecting humans:-

1. ***Plasmodium vivax*** (benign tertian malaria)
2. ***Plasmodium ovale*** **(**ovale tertian malaria**)**
3. ***Plasmodium falciparum*** **(**malignant tertian malaria**)**
4. ***Plasmodium malariae*** **(**quatrain malaria**)**

Life cycle

The life cycle of malaria is passed in two hosts and has sexual and asexual stage .

Vertebrate host - **man** (**intermediate host**), where the asexual cycle takes place.

The parasite multiplies by schizogony and there is formation of male and female gametocytes (gametogony).

Invertebrate host - **mosquito** (**definitive host**) where the sexual cycle takes place.

Union of male and female gametes ends in the formation of sporozoites (sporogony).

The life cycle passes in four stages:

Three in man:- Pre - erythrocyticschizogony (Exo- erythrocyticschizogony)

- Erythrocyticschizogony
- Gametogony One in mosquito - Sporogony

Introduction into humans- when an infective female Anopheles mosquito bites man, it inoculates saliva containing sporozoites (infective stage).

Exo- Erythrocyticschizogony- sporozoites reach the blood stream and within
30 minutes enter the parenchymal cells of the liver, initiating a cycle of schizogony. Multiplication occurs in tissue schizonts, to form thousands of tiny merozoites. Merozoites are then released on rupture of schizonts about 7^{th} – 9^{th} day of the bites and enter into the blood stream. These merozoites either invade the RBC's or other parenchymal liver cells. In case of *P. falciparum* and possibly *P. malariae*, all merozoites invade RBC's without re-invading liver cells. However, for *P. vivax* and *P. ovale*, some merozoites invade RBC's and some re-invade liver cells initiating further *Exo- erythrocytic* schizogony, which is responsible for relapses.

Erythrocytic cycle:
When merozoites leave liver cell to penetrate erythrocytes in the blood, they initiate an erythrocytic cycle. Merozoites invade erythrocytes and undergo a trophic period in which the parasite enlarges. The early trophozoite is often referred to as '**ring form**' because of its morphology. **Trophozoite** enlargement is accompanied by an active metabolism including the ingestion of host cytoplasm and the proteolysis of hemoglobin into amino acids. The end of the trophic period is manifested by multiple rounds of nuclear division without cytokinesis resulting is a **schizont**. Merozoites bud from the mature schizont, and the merozoites are released following rupture of the infected erythrocyte .when they will infect new RBC reinvadefresh RBC's repeating the schizogonic cycles or develop to Sexual stage – gamete formation, The development in this stage is characteristic to each species and is important in diagnosis.
The blood stage is responsible for the pathology associated with malaria, Erythrocyticschizogony (blood phase) is completed in 48 hrs in *P. vivax, P.ovale,* and *P. falciparum*, and 72 hrs in *P. malariae*.

Gametogony

Some merozoites that invade RBC's develop into sexual stages (male and female gametocytes). These undergo no further development until taken by the mosquito.

Sporogony (in mosquito)

When a female Anopheles mosquito vector bites an infected person, it sucks blood containing the different stages of malaria parasite. All stages other than gametocytes are digested in the stomach.

The microgametocyte undergoes ex-flagellation. The nucleus divides by reduction division into 6-8 pieces, which migrate to the periphery. At the same, time 6-8 thin filaments of cytoplasm are thrust out, in each passes a piece of chromatin. These filaments, the microgametes, are actively motile and separate from the gametocyte.

The macrogametocyte by reduction division becomes a macrogamete. Fertilization produce a **zygote**. The zygote changes into a worm like form, the **ookinete**, which penetrates the wall of the stomach to develop into a spherical **oocyst** between the epithelium and basement membrane. The oocystes increase in size. Thousands of **sporozoites** develop inside the oocysts. Oocysts rupture and sporozoites are liberated in the body cavity and migrate everywhere particularly to the salivary glands. Now the mosquito is infective.

1. *Plasmodium falciparum*

1. it invades young and old RBCs cells and The infected red blood cells also do not enlarge and become distorted.
2. Multiple sporozoites can infect a single erythrocyte, and show multiple infections of cells with small ring forms.
3. Occasionally, reddish granules known as Maurer's dots are observed
4. Mature (large) trophozoites stages and schizonts are rarely seen in blood films, because their forms are sequestered in deep capillaries, liver and spleen.
5. Peripheral blood smears characteristically contain only young ring forms and occasionally crescent shaped gametocytes.

Clinical features:

P. falciparum has the shortest incubation period, which ranges from 7 to 10 days. After the early flu-like symptoms, *P.falciparum* produces daily chills and fever as well as severe nausea, vomiting and diarrhea. The periodicity of the attacks then becomes tertian (36 to 48 hours), and fulminating disease develops. At the beginning the fever may occur irregularly, It takes some time for periodic and synchronized release of merozoties from RBC which gives the characteristics symptoms of periodicity.

Infects RBC to produce knobs that adhere to the endothelial lining of blood vessels ,This leads to obstruction, thrombosis, and local ischemia (decrease in blood supply) .

P. faclciparum infections are more severe with complications and higher mortality rate which can cause cerebral malaria resulting in coma and death , Renal dysfunction, respiratory failure,Gastrointestinal disorders, multiorgan failure . Liver involvement is characterized by abdominal pain, vomiting of bile, hepatosplenomegally, severe diarrhea, and rapid dehydration

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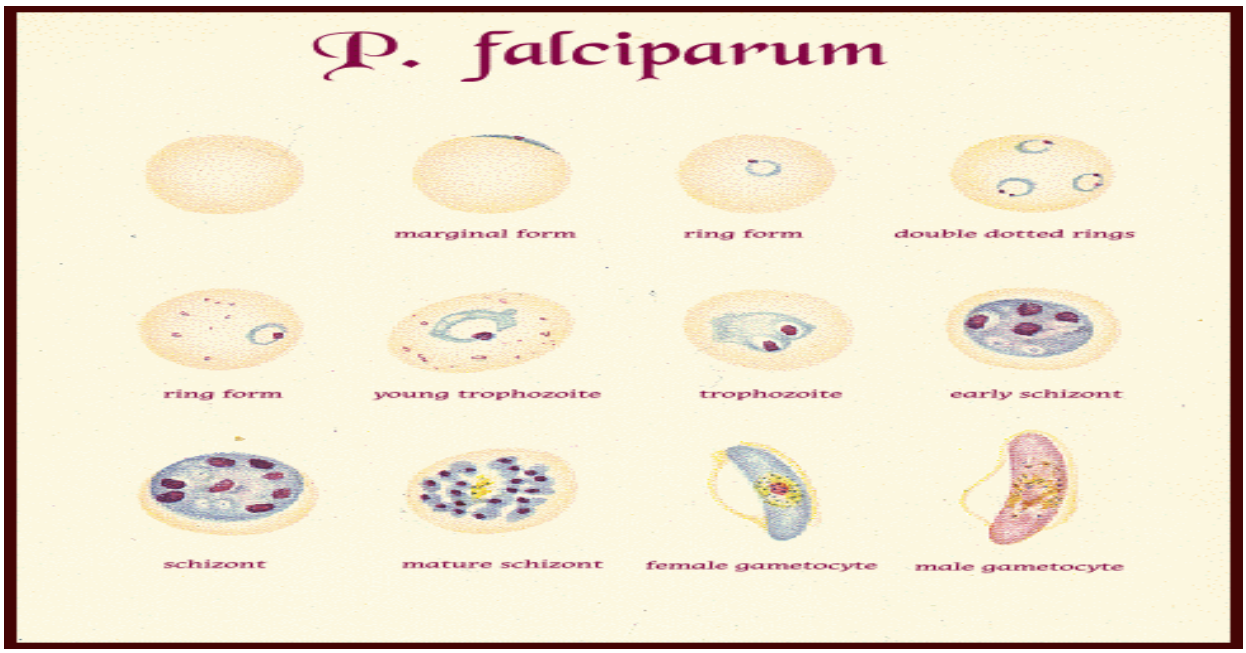


2. *Plasmodium vivax*

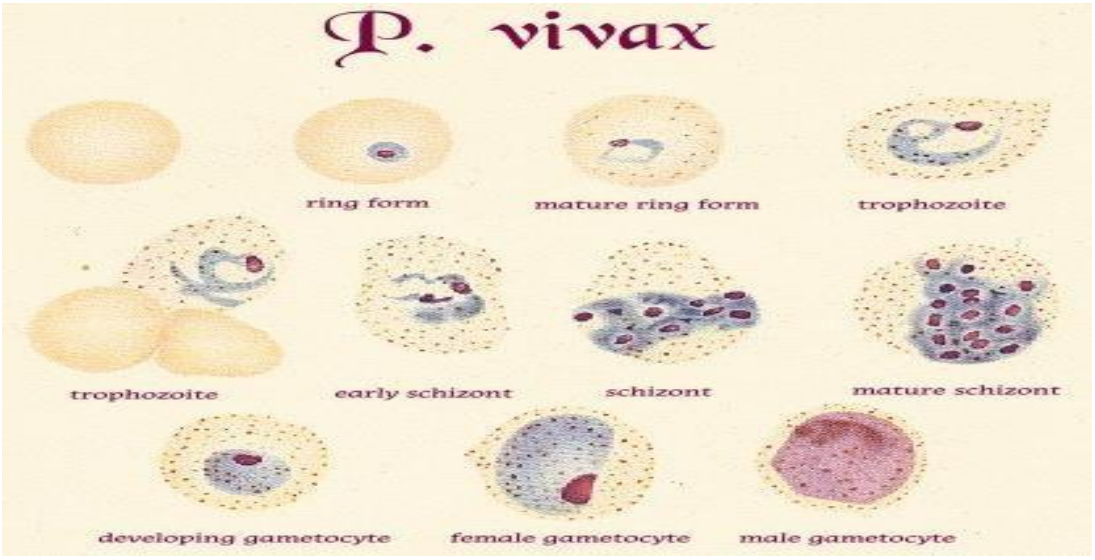
6. ***P.vivax*** is selective in that it invades only young immature erythrocytes.
7. Infected red blood cells are usually enlarged and contain numerous pink granules or schuffner's dots.
8. The trophozoite is ring-shaped but amoeboid in appearance.

9. More mature trophozoites and erythrocytic schizonts containing up to 24 merozoites are present.
10. The gametocytes are round
11. All developing forms are frequently present in peripheral blood.

Clinical features

After an incubation period (usually 10 to 17 days), the patient experiences flu-like symptoms, such as headache, muscle pains, photophobia, nausea and vomiting.

As the infection progresses, increased numbers of rupturing erythrocytes liberate merozoites as well as toxic cellular debris and hemoglobin into circulation. In combination, these substances produce the typical pattern chills, fever and malarial rigors. These paroxysms usually reappear periodically (generally every 48 hours) as the cycle of infection, replication, and cell lyses progresses.

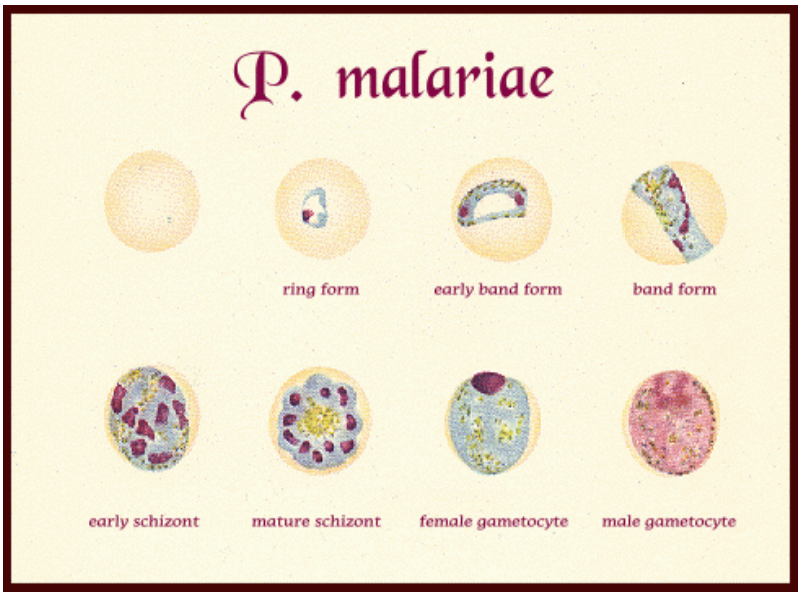


3. Plasmodium malariae

1. *P.malariae*can infect only mature.
2. This requirement produces no red cell enlargement or distortion.
3. Trophozoite may take band shape in this species as well as very compact dark staining forms.
4. Mature schizonts may have a 6-12 merozoites .
5. All forms can be seen in peripheral blood .
6. Oval or round gametocyte .
7. The schizont of *P.malariae*is usually composed of eight merozoites appearing in a rosette.

Clinical features

The incubation period for *P. malariae*is the longest of the plasmodia, usually 18 to 40 days, but possibly several months to years. The early symptoms are flu-like with fever patterns of 72 hours (quartan or malarial) in periodicity.



4. Plasmodiumovale

1. *P. ovale* is similar to *P. vivax* in many respects, including its selectivity for young erythrocytes.
2. The host cell becomes enlarged and distorted, usually in an oval form.
3. Schuffner's dots appear as pale pink granules.
4. Mature schizonts contain about 10 merozoites.
5. All developmental stages can be seen in peripheral blood .
6. Oval or round gametocyte.

Clinical features

The incubation period for *P.ovale*is 16-18 days but can be longer. Clinically, ovale malaria resembles vivax malaria with attacks recurring every 48-50 hours. There are however, fewer relapses with P.ovale. Less than 2% of RBCs usually become infected.

Treatment

Chloroquine the drug of choice that kills merozoites, Resistance is becoming common among *P. falciparum* and also reported in *P. vivax.*

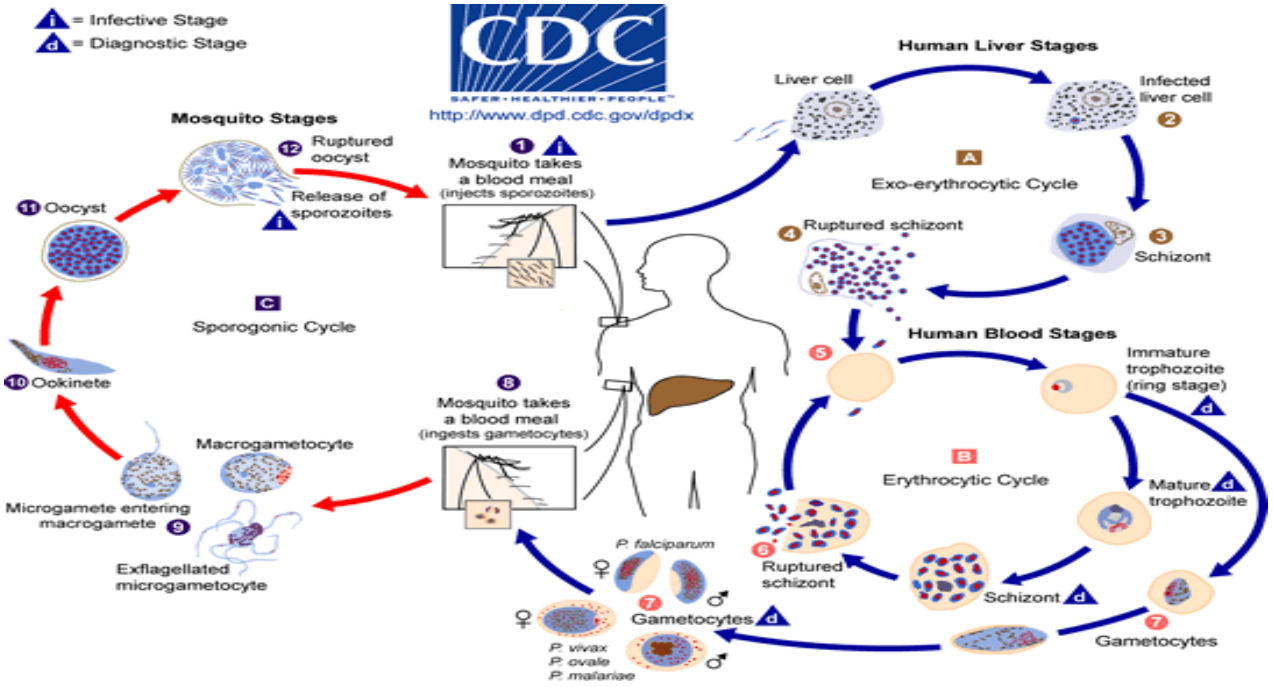
Resistant infections are usually treated with more than one drug:

- Quinine + pyrimethamine-sulfadoxine sulfate
- Quinine + tetracycline
- Quinine + clindamycin

Laboratory diagnosis

Microscopic examination of thick and thin films of blood is the method of choice for confirming the clinical diagnosis of malaria and identifying the specific species responsible for disease.
Malaria parasites in thick and thin blood films are best stained at pH 7.1 – 7.2 using a Giemsa stain .
The thick film is a concentration method that may be used to detect the presence of organisms. The thin film is most useful for establishing species identification.

• Protection of insect bite by screening, netting and protective clothing
• Use of insect repellents.



Life cycle of *Plasmodium* spp.