Class: Sporozoa.

Subclass: Haemosporidia.

Family: Plasmodiidae.

Genus: Plasmodium.

Species: Plasmodium vivax.

Plasmodium malariae.

Plasmodium falciparum.

Plasmodium ovale.

In Algeria 1880 – infected RBCs by the parasite.

In 1894 – mosquito-transmitted disease.

It is one of the greatest killers in the world in addition to cancer and heart diseases.

Geographical distribution:

- As far north as 64°N latitude (Russia).
- As far south as 32°S latitude (Argentina).
- Dead sea 400 meters below sea level.
- At 2600 m. above sea level Kenya.
- At 2800 m. above sea level Bolivia.

^{*} Malaria → bad air plasmodioses.

- P. vivax= most extensive in distribution.
 P. falciparum= tropical & subtropical.
 P. malariae= Less common but wide distribution.
- P. ovale= East & West Africa.

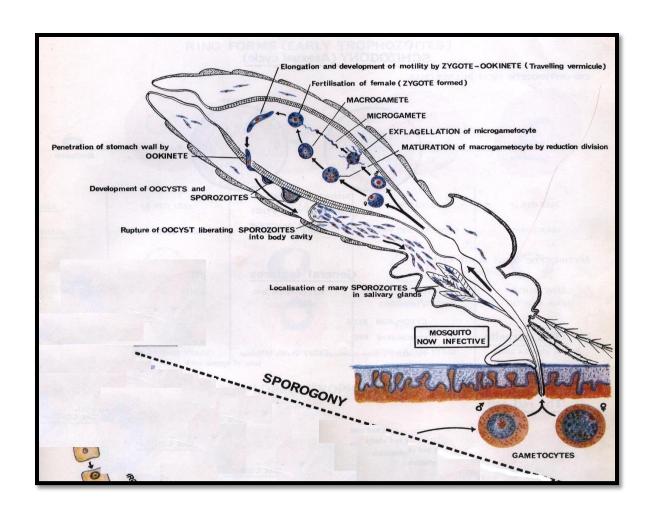
Risk factors for wide distribution:

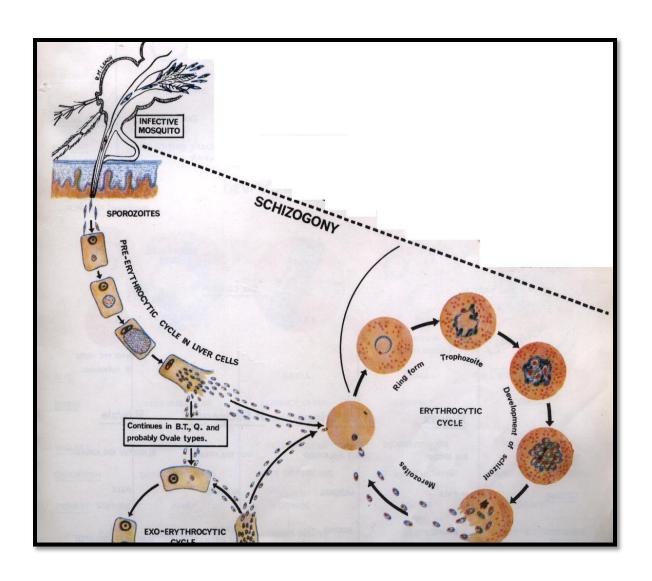
- 1) The impact of the Korean & Vietnam wars.
- 2) Air transpory among countries.

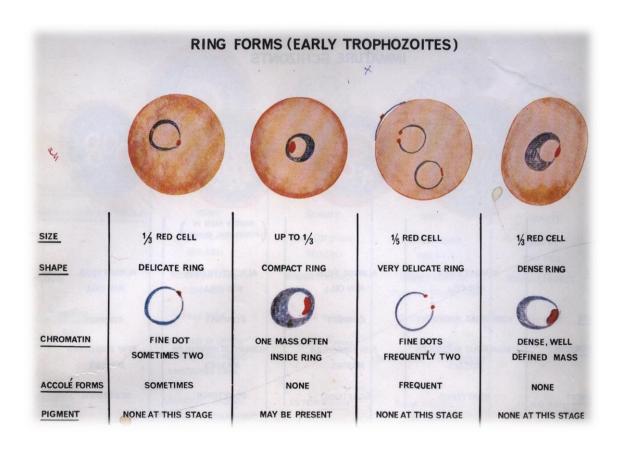
Methods of infections & transmission:

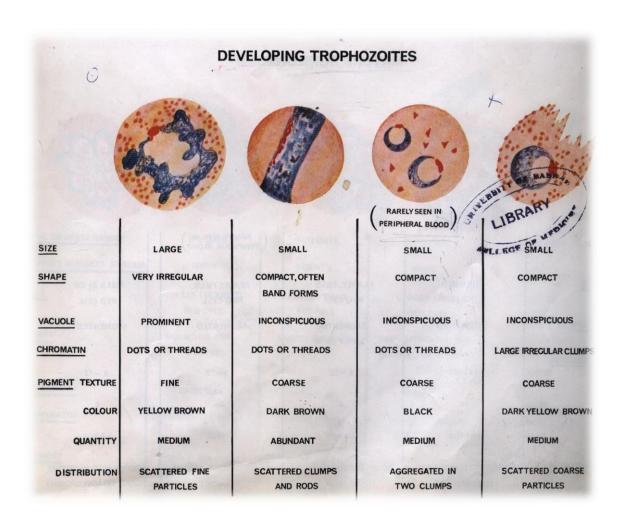
- 1) \bigcirc *Anopheles* bits.
- 2) Blood transfusion.
- 3) Organ trasplantation.
- 4) Congenital route.
- 5) Hypodermic needle.

Life cycle:

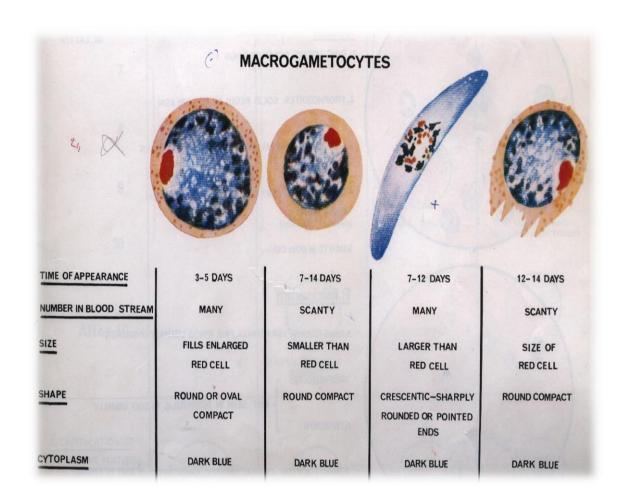


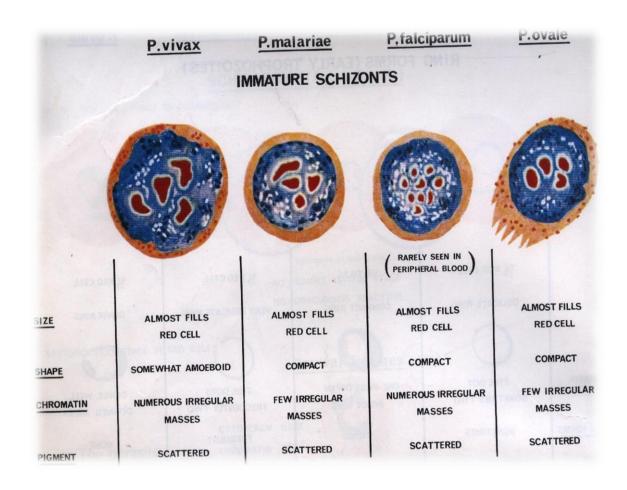


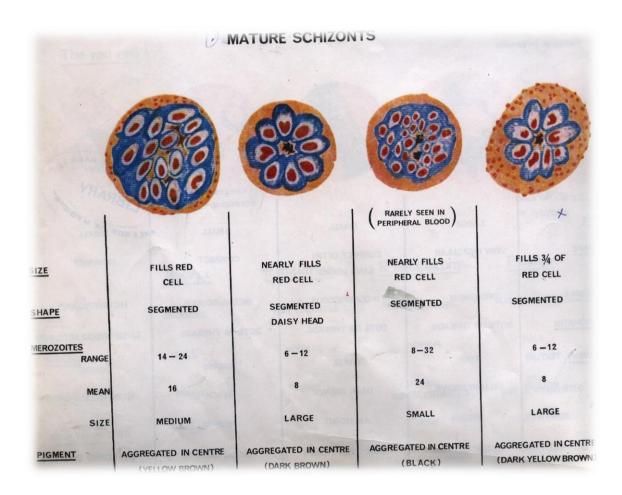




Morphology of Malaria parasites (cont.) P. vivax P.malariae P.falciparum P. ovale **MICROGAMETOCYTES** ty TIME OF APPEARANCE 3-5 DAYS 7-14 DAYS 7-12 DAYS 12-14 DAYS NUMBER IN BLOOD STREAM MANY SCANTY MANY SCANTY FILLS ENLARGED SIZE SMALLER THAN LARGER THAN SIZE OF RED CELL RED CELL RED CELL RED CELL SHAPE ROUND OR OVAL ROUND COMPACT KIDNEY SHAPED ROUND COMPACT COMPACT BLUNTLY ROUND ENDS CYTOPLASM PALE BLUE PALE BLUE REDDISH BLUE PALE BLUE CHROMATIN FIBRILS IN SKEIN AS FOR Pivivax FINE GRANULES AS FOR P.vivax WITH SURROUNDING SCATTERED THROUGHOUT UNSTAINED AREA PIGMENT ABUNDANT BROWN AS FOR P.vivax DARK GRANULES AS FOR P. vivax GRANULES THROUGHOUT THROUGHOUT







P. vivax = vivax tertian malaria (benign).

P. malariae = Quartan malaria.

P. falciparum = malignant tertian malaria.

P. ovale = ovale tertian malaria

The periodic febrile response is related to the time of rupture of a sifficient No. of mature schizonts and consequent discharge of merozoites into the blood stream (Synchronised schizogony).

THE SEVERITY OF THE ILLNESS DEPENDS ON:

1. The degree of parasitaemia.

- 2. The extent of RBCs destruction.
- 3. The defence responses of the host.

Relapse (Delayed primary attack): Re-ocurrence of malarial attack after apparent cure due to persistent exo-erythrocytic stages or dormont sporozoites (hypnozoites). It occurs in P.vivax (2-3 years) & P.ovale (longer than a year).

Recrudescence: Renewal of clinical manifestation due to subclinical population of the parasites. It occurs in P. falciparum (1 year) & P. malariae (many years).

MALARIA IN HYPERENDEMIC AREAS

The typical malarial manifestation occurs in young children only. Older children & adults who survive previous infection develop tolerance to the disease.

CONGENITAL MALARIA

The mechanism of the transplacental passage of the parasite is obscure. It has not been reported in laboratory animals.

Pathology:

Anemia: Acute = normocytic & normochromic.

Chronic & relapses = Pernicious anemia.

Falciparum malaria = Sludging RBCs.

C.N.S: 1. Congestion of meninges & brain.

- 2. Occlusion of the capillaries of the cortex.
- 3. Necrotic lesions in midzonal brain

(malarial granuloma).

Spleen: 1. Dark, congested & enlarged.

- 2. Hyperplasia of red & white pulp.
- 3. Erythropoiesis & lymphopoiesis.
- 4. Haemosiderosis.

Liver:

- 1. Enlarged & dark in colour.
- 2. Hypertrophy of the kupffer cells with ingested malarial pigments.
- 3. Degeneration & necrosis in the centro- lobular regions.

Kidney:

- 1. Falciparum malaria = congestion & Punctate haemorrhages in the Cortex & medulla.
- 2. Malariae malaria = nephrotic syndrome (hyalinisation of the tuft of the glomeruli & segmental cells.

Thickening basal membrane due to

Deposition of Ag-Ab complexes).

Heart: Embolic blockage of the coronary vessels.

Placenta: Falciparum malaria, mature schizonts in the intervillous spaces with histiocytes in the maternal side of the placenta.

Complications:

Quartan malaria → nephrosis.

Falciparum malaria → Cerebral malaria.

Gastrointestinal malaria.

Hyperpyrexia.

Algid malarial.

Black water fever.

Immunity:

I) Natural immunity:

- Duffy group determinants among west African & American blacks.
- Sickle cell anemia.
- Haemoglobin C & E.
- G 6 PD deficiency.

II) ACQUIRED IMMUNITY:

Premunition immunity.

Stable & unstable malaria.

Why *Plasmodium falciparum* is malignant:

- 1. It invades erythrocytes of all ages.
- 2. Schizogonic cycle requires 36-48 h.
- 3. Several parasites in a single RBC.
- 4. Adherence of RBC one to another & to the lining of the blood vessels.
- 5. The toxic products interfere with oxygen utilization by the host cells.
- 6. Autoimmunity in destruction both the parasitized & the non-parasitized RBCs.

Diagnosis:

- 1. Clinical picture.
- 2. Thick blood film.
- 3. Thin blood film.
- 4. Serological tests.