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EDITORIAL

The Malaria Parasitology

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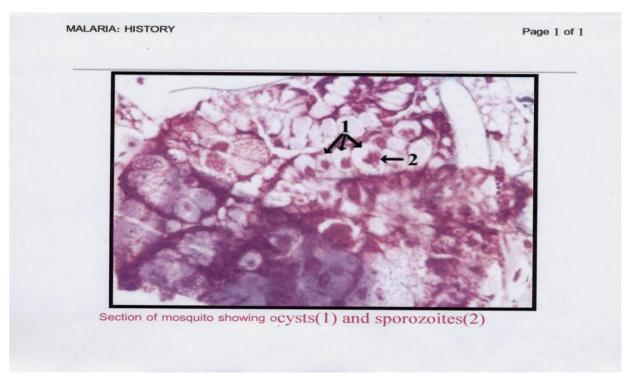
The microorganisms causing malaria are commonly referred to as malaria parasites, Plasmodium. In 1808 Laveran discovered plasmodium in blood in Algeria. There are nearly 120 species of plasmodia; 22 found in primate hosts, 19 found in rodents and bats and 70 found in birds and repliles. The human plasmodium has four groups classified according to the periodicity of their erythrocytic schizognoy:

- P. malaria; Quartan. (laveran 1881)
- P. vivax; Benign tertian. (Grassi and Feletti 1890)
- P. falciparum; Malignant tertian. (Welch 1897)
- P. ovale; ovale tertian. (stephens, 1992)

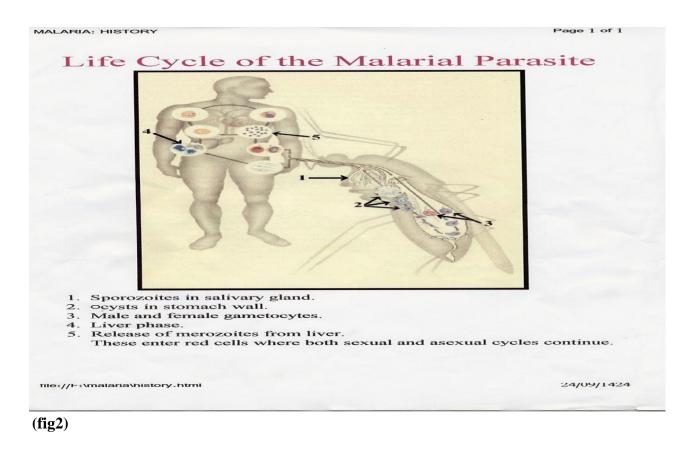
Life cycle. The life cycle of all species of human malaria parasites is essentially the same. It comprises an exogenous sexual phase (sporogony) with multiplication in certain Anopheles mosquitoes, and an endogenous asexual phase (schizogony)with multiplication in the vertebrate host. the latter phase includes the development cycle in the red corpuscles in the blood (erthrocytic schizogony) and the cycle taking place in the parenchyma cells of the liver (exo-erthrocytic schizogony-tissue stage).

The parasite in the mosquito host: When a female Anopheles mosquito ingests the blood of a human host (50 cum-med) with malaria parasites in the circulation, the infection ergthrocyles sets the parasites free in the mosquito stomach. The asexual parasites are digested together with the red blood cell (gametoeytes) the sexual cells undergo while mature further developmentmaturation process. The female gametocyte undergoes a maturation process and froms a female gamete macrogamete. The male gamete is called a microgamete. In the stomach of the mosquito a microgamete is attracted by a macrogamete; the latter forms a small projection through which the microgamete enters and thus completes the fertilization. The product of fusion of female and male gamete is called azygote. Then the zygote becomes an ookinete. The ookinete soon forces its way to the stomach wall to the outer surface of the stomach. There it becomes an oocyst. The oocyst gradually increases in size and devides to form sporozoites. The sporozoites burst through the weakened wall of the oocyst and invade the body cavity of the mosquito and then they reach the salivary glands of the female Anopheles which now becomes infective. When the mosquito feeds on blood after piercing the human skin the

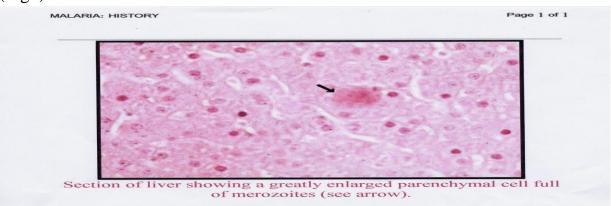
sporozoies are injected in the wound and pass into the blood stream(fig1,fig2)



(fig1)

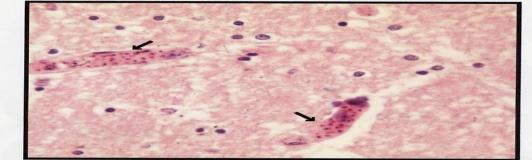


The parasite in the vertebrate host: Tissue or exo-erythrocytic schizogony following the inoculation of sporozoites there is a brief period of about half an hour when the blood is infected. Then the sporozoites disappear from the blood. Then they enter the paraenchymal cels of the liver (Hepatocytes) via the Kupffer cells and undergo a process of development and multiplication known as pre-erythrocytic schizogony. The sporozoites develop in young chizonts, deviding schizonts and eventually into mature schizonts. The mature schizonts enlarge the Hepatocytes and then they burst to release into the blood thousands of merozoites. In relapsing type of malaria infection (P.vivax and propably P.ovale) some of the sporozoites do not from hyponzoites but develop directly into pre-erythrocytic schizionts(fig3-4) (Fig3)



MALARIA: HISTORY

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Section of brain showing blood vessels blocked with developing *P. falciparum* parasites (see arrows)

(fig4)

Erythrocytic phase: the merozoites released from tissue schizont invade the erythrocytes. The youngest stage in the red blood cels are ring forms. As these grow in size they become more irregular in shape. All these early stags of the parasite are called trophozoites. Afer a period of growth the trophozoite undergoes an asexual deivding process of erythrocytic schizogony. The nucleus of the parasite devids 3-5 times into a variable

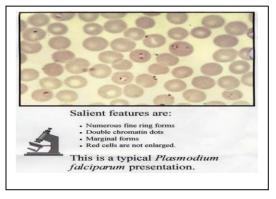
number of small nuclei. This is soon followed by the division of the eytoplasm forming a schizont. Mature schizonts include merozoites. When the process of schizogony is completed the red blood cell bursts and the merozoites are relased into the blood stream. The merozoites then invade fresh erythrocytes in which another generation of parasites is produced by the same process. This erythroeytic eycle of chizogony is repeated over and again the course of infection leading to a progressive increase of parasitaemia until the process is slowed down by the immune response of the host. After several generations of merozoites have been produced some of these give rise to sexually differentiated froms (gametoeytes)

Malaria laboratory Diagnosis Blood examination for malaria paracites (Blood film):

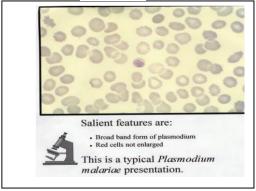
The only certain means of diagnosing malaria infection is the detection of the plasmodium by microscopical examination of the blood. This examination should be a routine in medical practice. Blood is obtained from the fourth finger of the left hand then athick and thin films are prepared and stained with Giemsa stain(fig5,6,7,8)

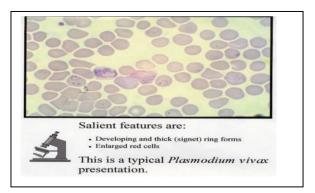
(Fig5)

(Fig6)

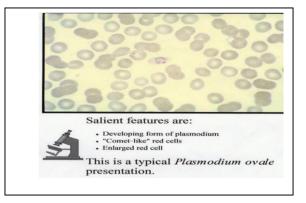


(fig7)





(fig8)



New techniques:

1. Dipstick tests

These include; (ICT) P.Falciparm, optimalr and Kat-quick kits.

These methods are based on the detection of plasmodial histidine rich prokin-2(HRP-2) or parasite specific lactate dehydrogenase present in P. falciparum infection. They are specific and sensitive-approaching ^{100%}, however ^{6%} cross react with sera positive for rheumatoid factor. These tests are accurate, speedy for the diagnosis of P.falciparum infection. And easy to use. They ara used as screening or confirmatory. However they cannot indicate the parasite load and have false positive results (cirallating antigens may be detected for 2 weeks.) Moreover these tests are expensive.

2. detection of antibodies

These include immunofluorscence techniques and the ennzymatic immunoassays They are of limited use for the diagnosis of acute malaria infection

3. Brcton-Dickisons Quantitative Buffy coal (QBC) method.

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