**Diseases caused by Protozoa**

In adapting to their hosts, protozoa, like other animal parasites, have evolved many life-cycle patterns. While some species are parasitic during only one phase of their life cycle patterns, others have adapted to more than one host during the different phases of their life cycle.

The host in which parasite reaches sexual maturity and reproduction is termed **definitive host.** If no sexual reproduction occurs in the life cycle of a protozoan, such as trypanosome or an amoeba, the host that is believed to be the most important is arbitrarily identified as the definitive host.

An intermediate host is one in which the other stages of the life cycle occur. For example, for the malaria protozoa, the mosquito is the definitive host, and humans or other vertebrates are the intermediate hosts.

An animal (or human) that is routinely infected with a protozoa or parasite which can cause also infect human is termed as reservoir host.

**Malaria**

**Typical organisms –** Four species of plasmodia typically infect humans: ***Plasmodium vivax, P. ovale, P. malariae* and *P. falciparum.***

**Pathogenesis, Pathology & clinical findings-** Human infection results from the bite of an infected female anopheles mosquito, in which the **sporozoites,** resulting from the sexual and subsequent sporogenic cycle of developmentin the mosquito, are injected into the human bloodstream.

The **sporozoites** rapidly (usually within 1 hour) enter parenchymal cells of the liver, where the first stage of development in humans takes place (**exoerythrocytic** phase of the life cycle). Subsequently, numerous asexual progeny, the **merozoites,** rupture and leave the liver cell, enter the bloodstream and invade erythrocytes.

Parasites in the red cells multiply in a species characteristic fashion, breaking out of their host cells synchronously. This is the **erythrocytic** cycle, with successive broods of **merozoites** appearing at 48-hour intervals (***Plasmodium vivax, P. ovale, and***  ***P. falciparum*** ) or every 72 hours (***P. malariae***).

The incubation period includes the exoerythrocytic cycles (usually two) and at least one or two erythrocytic cycles. For ***P. vivax*** and ***P. falciparum***, this period is usually 10-15 days, but it may be weeks or months. The incubation period of ***P malariae*** averages about 28 days. There is no return of merozoites from red blood cells to liver cells. Without treatment, falciparum infection ordinarily will terminate spontaneously in less than 1 year unless it ends fatally.

The other three species continue to multiply in liver cells long after the initial bloodstream invasion, or there may be delayed multiplication in the liver. These exoerythrocytic cycles coexist with erythrocyctic cycles and, in ***P vivax*** and ***P ovale*** , may persist as nongrowing resting forms, or **hypnozoites,** after the parasites have disappeared from the peripheral blood. Resurgence of an erythrocytic infection (relapse) occurs when merozoites from hypnozoites in the liver break out, are not phagocytosed in the bloodstream, and succeed in reestablishing a red cell infection (clinical malaria).

Without treatment, ***P vivax*** and ***P ovale*** infections may persist as periodic relapses for upto 5 years. *P. malariae* infections lasting for 40 years have been reported, this is thought to be a cryptic erythrocytic rather than an exoerythrocyctic infection and is therefore termed a **recrudescence** to distinguish it from a relapse.

During the erythrocytic cycle, certain merozoites enter red cells and become differentiated as male or female gametocytes. The sexual cycle therefore begins in the vertebrate host, but for its continuation into the sporogonic phase, the gametocytes must be taken up and ingested by bloodsucking female anopheles as outlined in fig1.

Fig 1. Life cycle of malaria parasite.

***P vivax,*** *P. malariae* and ***P ovale*** parasitemias are relatively low-grade, primarily because the parasites favor either young or old red cells but not both; *P. falciparum* invade red cells of all ages, including the erythropoietic stem cells in bone marrow, so parasitemia may be very high.

*P. falciparum* also causes parasitized red cells to produce numerous projecting knobs that adhere to the endothelial lining of blood vessels, with resulting obstruction, thrombosis and local ischemia.

*P. falciparum* infections are therefore far more serious than the others, with a much higher rate of severe and frequently fatal complications (cerebral malaria, malaria hyperpyrexia, gastrointestinal disorders). Consequently, correct and prompt diagnosis of falciparum malaria is imperative and may be lifesaving.

**Symptoms**

Intial chill, lasting from 15 minutes to 1 hour, begins as a synchronously dividing generation of parasites rupture their host and escape into blood.

Nausea, vomiting and headache are common at this time. The succeeding febrile stage, lasting several hours, is characterized by a spiking fever that frequently reaches 40ºC or more. During this stage, the parasites presumably invade new red cells. The third or sweating stage concludes the episode. The fever subsides and the patient falls asleep and later awakes feeling relatively well.