

# MALARIA

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Malaria is a life-threatening disease. Its typically transmitted through the bite of an infected *Anopheles* mosquito. Infected mosquitoes carry the *Plasmodium* parasite. When this mosquito bites you, the parasite is released into your blood stream.

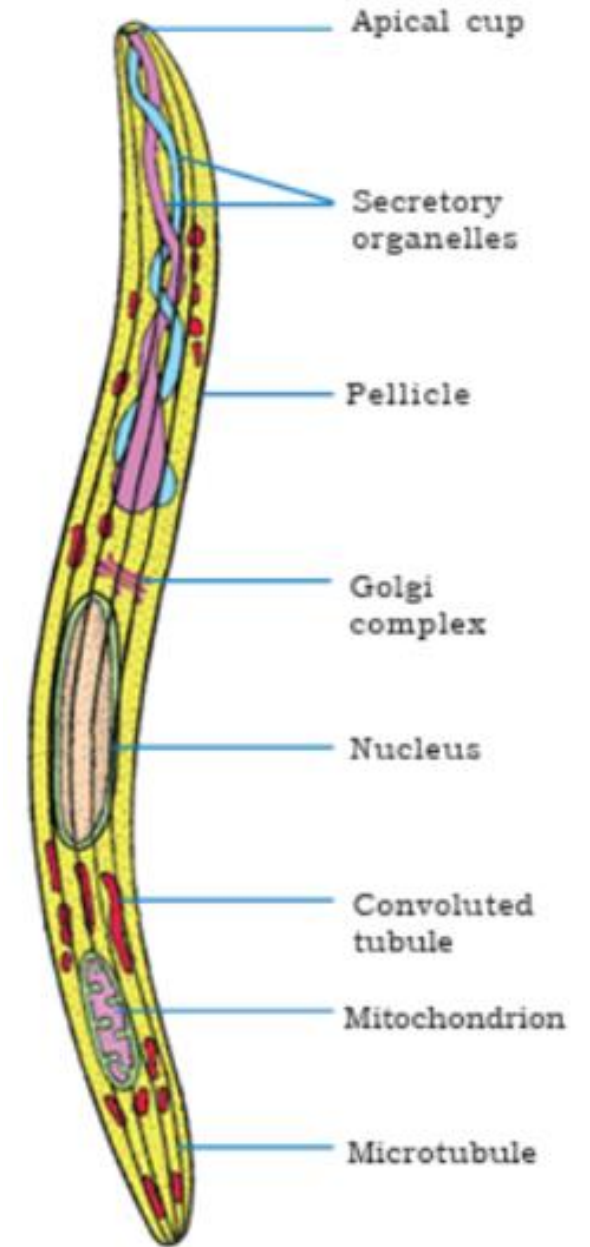
- i. Phylum : Protozoa
- ii. Subphylum : Apicomplexa (Sporozoa)
- iii. Class : Telosporea
- iv. Genus : *Plasmodium*
- v. Species : *vivax*

- Plasmodium is one of the most harmful parasites of man.
- It is a digenetic, intracellular parasite that lives in the liver cells and RBC of man. (It is extracellular in mosquito).
- Its primary host is the female Anopheles mosquito and the secondary host is man. Reservoir host is monkey.
- The infective stage is sickle shaped sporozoite and the mode of infection is inoculation.
- Four species of Plasmodium cause four types of malaria in man.
- They are
  - i) Plasmodium vivax – benign tertian malaria
  - ii) Plasmodium falciparum – malignant tertian malaria
  - iii) Plasmodium ovale – mild tertian malaria
  - iv) Plasmodium malariae – quartan malaria

Of all these four species, Plasmodium vivax is the most common and most widely distributed malaria parasite.

# Structure of sporozoite

- It is sickle shaped with a swollen middle part and pointed at both ends of its body.
- It measures about 15 microns in length and one micron in width.
- The body is covered by an elastic pellicle with 'microtubules' which help in the wriggling movements of the sporozoite.
- The cytoplasm contains cell organelles such as Golgi complex, endoplasmic reticulum, mitochondria and a nucleus.
- Cytoplasm also shows many convoluted tubules of unknown function throughout the length of the body.
- It contains a cup like depression called apical cup at the anterior end into which a pair of secretory organelles opens.
- They secrete a cytolytic enzyme, which helps in the penetration of sporozoite into the liver cells.



# Life cycle of Plasmodium in man (The human phase)

- The life cycle of Plasmodium is completed in two hosts as mentioned earlier.
- In man, the Plasmodium reproduces by asexual reproduction called schizogony.
- It occurs in liver cells (hepatocytes) as well as in RBC.
- In liver cells, it is called hepatic schizogony and in RBC it is called erythrocytic schizogony.

## 1. **Hepatic schizogony:**

Whenever, a mosquito infected by Plasmodium bites a man, nearly 2000 sporozoites are released into the blood of man through its saliva. Within half an hour, they reach the hepatocytes where they undergo pre-erythrocytic and exo-erythrocytic cycles.

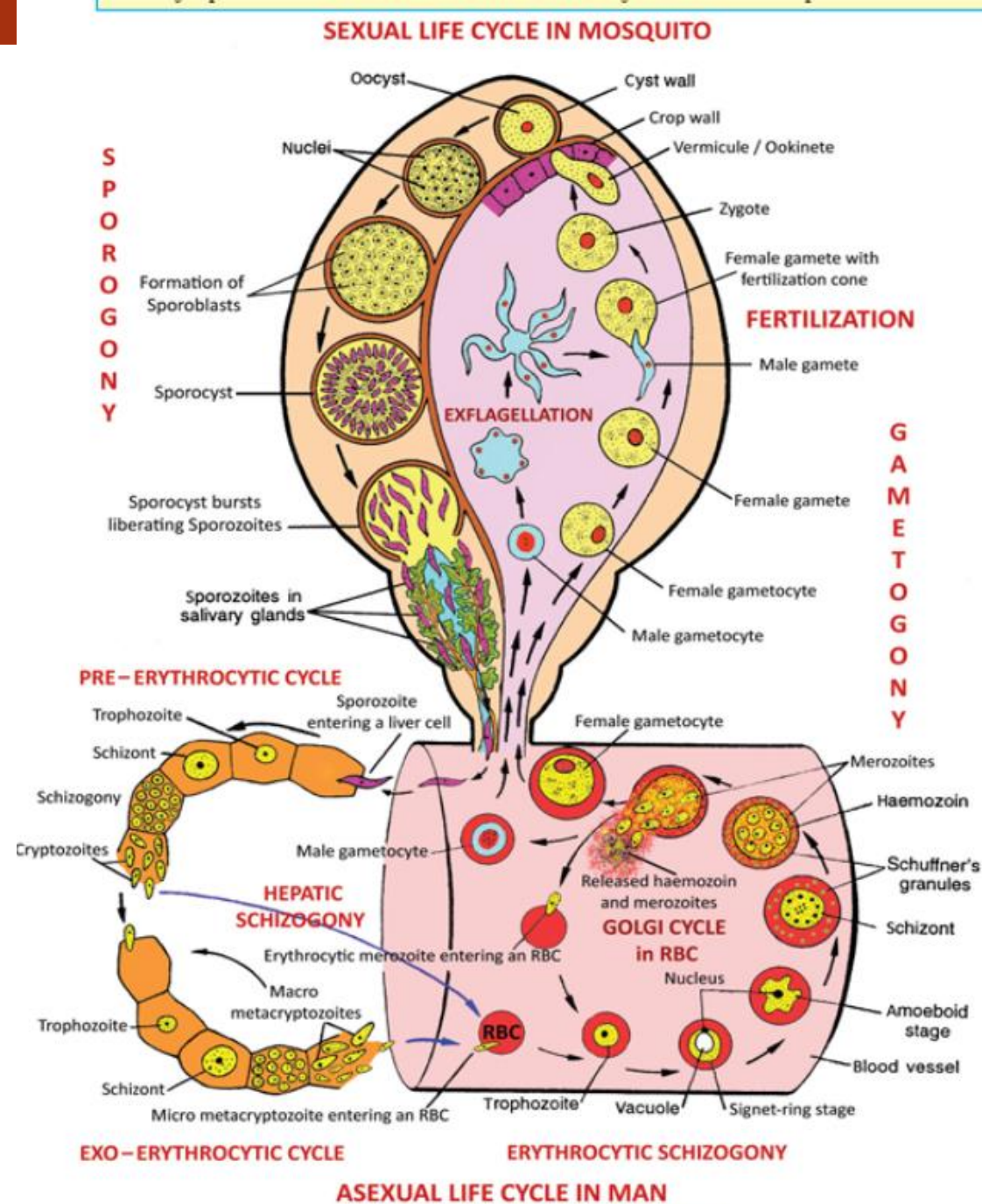


Fig. 6.6 Life cycle of *Plasmodium vivax*

## A. Pre-erythrocytic cycle

1. When the sporozoites reach the liver cells, they transform into trophozoites.
2. They feed on the contents of the hepatic cells, assume spherical shape and attain the maximum size.
3. This stage is called schizont stage.
4. Its nucleus divides several times mitotically, followed by the cytoplasmic divisions resulting in approximately 12,000 daughter individuals called cryptozoites or the 1st generation merozoites.
5. They enter the sinusoids of the liver by rupturing the cell membrane of the schizont and the liver cells. This entire process is completed approximately in 8 days.
6. Now these first generation merozoites have two options i.e. they can enter either fresh liver cells and continue exoerythrocytic cycle or they can enter RBC and continue erythrocytic cycle.

## **B. Exo-erythrocytic cycle**

1. If the cryptozoites enter the fresh liver cells, they undergo the changes similar to that of the pre-erythrocytic cycle and produce the second generation merozoites called metacryptozoites.
2. These are of two types- the smaller micro-metacryptozoites and larger macro-metacryptozoites.
3. This entire process is completed approximately in two days.
4. The macro-metacryptozoites attack fresh liver cells and continue another exo-erythrocytic cycle, whereas the micro-metacryptozoites always enter blood stream and attack fresh RBC to continue repeated erythrocytic cycles producing merozoites.

### **Prepatent period**

1. The interval between 'the first entry of Plasmodium into the blood in the form of sporozoites and the second entry of Plasmodium into the blood in the form of cryptozoites is called prepatent period.
2. It lasts approximately 8 days.
3. During this period, the host does not show any clinical symptoms of the disease. It is only a means of multiplication.

## 2. Erythrocytic Schizogony

### A. Cycle of Golgi

1. This cycle is initiated either by the cryptozoites of pre-erythrocytic cycle or the micrometacryptozoites of exo-erythrocytic cycle.
2. In the fresh RBC, these stages assume spherical shape and transform into trophozoites.
3. It develops a small vacuole which gradually enlarges in size, pushing the cytoplasm and nucleus to the periphery.
4. Now the Plasmodium looks like a finger ring. Hence this stage is called signet ring stage.
5. Soon it loses the vacuole, develops pseudopodia and becomes amoeboid stage (late trophozoite stage).
6. With the help of pseudopodia, it actively feeds on the contents of the RBC and increases in size.
7. As a result, the RBC grows almost double the size.
8. This process is called hypertrophy.

9. The malaria parasite digests the globin part of the ingested haemoglobin and converts the soluble haem into an insoluble crystalline haemozoin.
10. It is called the 'malaria pigment' which is a disposable product.
11. During this stage, small red coloured dots appear in the cytoplasm of the RBC known as Schuffner's dots.
12. These are believed to be the antigens released by the parasite.
13. Now the Plasmodium loses the pseudopodia, increases further in size, occupies the entire RBC and becomes a schizont. It undergoes schizogony similar to that of the preerythrocytic cycle and produces 12 to 24 erythrocytic merozoites.
14. They are arranged irregularly in the RBC form. Finally the erythrocyte bursts and releases the merozoites along with haemozoin into the blood.
15. This cycle is completed approximately in 48 hours.

## **Incubation Period**

1. The period between 'the entry of Plasmodium into the blood in the form of sporozoite and the first appearance of symptoms of malaria in man' is called incubation period.
2. It is approximately 10 to 14 days.

## **B. Formation of gametocytes**

1. After repeated cycles of erythrocytic schizogony, when the number of fresh RBC decreases, some merozoites enter the RBC and transform into gametocytes instead of continuing the erythrocytic cycle.
2. This process generally takes place when the RBCs are present in spleen and bone marrow.
3. The gametocytes are of two types namely, smaller microgametocytes or male gametocytes and larger macrogametocytes or female gametocytes.
4. The gametocytes cannot undergo further development in man as the temperature and pH of the blood of man are not suitable for further development.
5. These gametocytes reach the blood circulation and wait to reach the next host. They degenerate and die if they are not transferred to mosquito within a week.

# Life cycle of Plasmodium in mosquito (The mosquito phase) – Ross cycle

- When a female *Anopheles* mosquito bites and sucks the blood of a malaria patient, the gametocytes along with the other stages of the erythrocytic cycle reach the crop of mosquito.
- All the stages are digested except the gametocytes. Further part of the life cycle consists of:
  - i) Gametogony
  - ii) Fertilization
  - iii) Formation of Ookinete & Oocysts
  - iv) Sporogony

## i) Gametogony:

The formation of male and female gametes from the gametocytes is called gametogony.

It occurs in the lumen of the crop of mosquito.

### a. Formation of male gametes:

1. During this process, the nucleus of microgametocyte divides into eight daughter nuclei called pronuclei which reach the periphery.
2. The cytoplasm is pushed out in the form of eight flagella like processes.
3. Into each flagellum like process, one pronucleus enters and forms a micro gamete or male gamete.
4. These male gametes show lashing movements like those of flagella and get separated from the cytoplasm of microgametocyte. This process is called exflagellation.

### b. Formation of female gamete:

1. The female gametocyte undergoes a few changes and transforms into a female gamete. This process is called maturation.
2. The nucleus of the female gamete moves towards the periphery and the cytoplasm at that point forms a projection.
3. This projected region is called the fertilization cone.

## ii. Fertilization:

1. The fusion of male and female gametes is called fertilization.
2. It also occurs in the lumen of the crop of the mosquito.
3. When an actively moving male gamete comes into contact with the fertilization cone of the female gamete, it enters it.
4. The pronuclei and cytoplasm of these two gametes fuse with each other, resulting in the formation of a synkaryon.
5. Since the two gametes are dissimilar in size, this process is known as anisogamy.
6. The female gamete that bears the synkaryon is called the zygote which is round and non-motile.

## iii) Formation of ookinete and oocysts:

1. The zygote remains inactive for some time and then transforms into a long, slender, motile, vermiform ookinete or vermicule within 18 to 24 hours.
2. It pierces the wall of the crop and settles beneath the basement membrane. It becomes round and secretes a cyst around its body.
3. This encysted ookinete is now called oocyst. About 50 to 500 oocysts are formed on the wall of the crop and appear in the form of small nodules.

#### **iv) Sporogony:**

1. The formation of sporozoites in the oocysts is called sporogony.
2. the nucleus of the oocyst first undergoes reduction division followed by repeated mitotic divisions resulting in the formation of sporoblasts.
3. The nucleus of the sporoblast divides and forms several nuclei.
4. Each nucleus is surrounded by a little bit of the cytoplasm and transforms into a sickle shaped sporozoite. Oocyst with such sporozoites is called sporocyst.
5. When this sporocyst ruptures, about 10,000 sporozoites are liberated into the haemocoel of the mosquito.
6. From there, they travel into the salivary glands and are ready for infection.
7. The life cycle of Plasmodium in mosquito is completed in about 10 to 24 days.

# Clinical Presentation

**TABLE 119-4** Clinical Presentation of Malaria

## Initial presentation

Nonspecific fever, chills, rigors, diaphoresis, malaise, vomiting<sup>15,49</sup>

Orthostatic hypotension

Electrolyte abnormalities

## Erythrocytic phase

Prodrome: headache, anorexia, malaise, fatigue, myalgia

Nonspecific complaints such as abdominal pain, diarrhea, chest pain, and arthralgia

Paroxysm: high fever, chills, and rigor<sup>2,3,9,49,54,55</sup>

Cold phase: severe pallor, cyanosis of the lips and cutis anserina ("goose flesh")<sup>9,49,55</sup>

Hot phase: fever between 40.5°C (104.9°F) and 41°C (105.8°F).

Sweating phase:

Follows hot phase by 2–6 hours

Fever resolves

Marked fatigue and drowsiness, warm, dry skin, tachycardia, cough, severe headache, nausea, vomiting, abdominal pain, diarrhea, and delirium

Lactic acidosis and hypoglycemia (with falciparum malaria)<sup>48,49,54,55</sup>

Anemia

Splenomegaly

## *P. falciparum* infections

Hypoglycemia, acute renal failure, pulmonary edema, severe anemia, thrombocytopenia, high-output heart failure, cerebral congestion, seizures and coma, and adult respiratory syndrome<sup>49,54,55</sup>

## LABORATORY DIAGNOSIS

## Malaria

### Microscopic tests:

❑ **Peripheral blood smear**—gold standard

➤ **Thick smear**—more sensitive

➤ **Thin smear**—speciation can be done based on the following features:

◆ *P. vivax*—amoeboid ring form and schizont

◆ *P. falciparum*—ring forms (multiple ring form, accolé form, headphone shaped ring forms), banana shaped gametocytes

◆ *P. malariae*—band forms

◆ *P. ovale*—enlarged fimbriated oval RBC with ring forms

❑ **Fluorescence microscopy** (Kawamoto's technique)

❑ **Quantitative buffy coat examination**—parasitized RBCs appear as brilliant green dots.

### Nonmicroscopic tests:

❑ **Antigen detection tests** (RDTs) or ICTs—detect pan malarial Ag (LDH, aldolase), falciparum specific Ag (HRP-II)

❑ **Culture**—RPMI 1640 medium

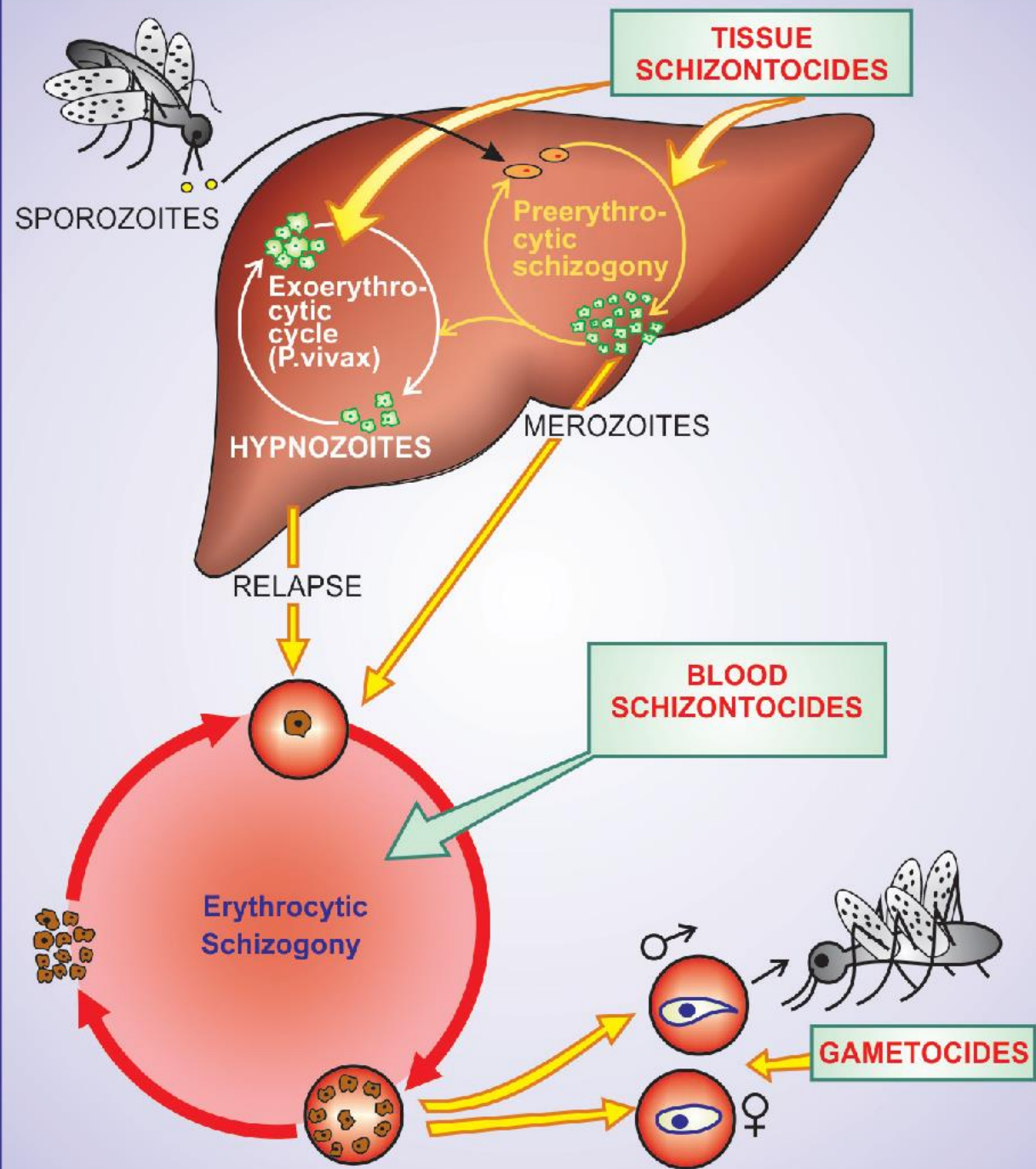
❑ **Molecular diagnosis**—PCR targeting 18S rDNA.

# TREATMENT

| Stage of Malaria                                   | Clinical Use       | Drug                                                                                                                                                 |
|----------------------------------------------------|--------------------|------------------------------------------------------------------------------------------------------------------------------------------------------|
| Pre erythrocytic stage<br>(Tissue Schizonticide)   | Causal Prophylaxis | <ul style="list-style-type: none"><li>• Proguanil</li><li>• Primaquine</li><li>• Pyrimethamine</li><li>• Sulfadoxine</li></ul>                       |
| Erythrocytic stage<br>(Erythrocytic Schizonticide) | Clinical Cure      | <ul style="list-style-type: none"><li>• Mefloquine</li><li>• Atorvaquone</li><li>• Chloroquine</li><li>• Halofantrine</li><li>• Artemisins</li></ul> |
| Exo Erythrocytic Schizonticide                     | Radical cure       | <ul style="list-style-type: none"><li>• Primaquine</li></ul>                                                                                         |
| Gametocides                                        | Gametocidal        | <ul style="list-style-type: none"><li>• Proguanil, Primaquine, Primaquine</li></ul>                                                                  |

# Therapeutic Classification of Anti malarial drugs

|                              |                                                                                                                                                                                             |
|------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Tissue schizonticides</b> | Primaquine, pyrimethamine, sulfonamides                                                                                                                                                     |
| <b>Hypnozoiticides</b>       | Primaquine, tafenoquine                                                                                                                                                                     |
| <b>Blood schizonticides</b>  | <i>Type 1, quick onset:</i> Chloroquine, mefloquine, quinine, halofantrine, artemisinin<br><i>Type 2, slow onset :</i> Pyrimethamine, sulfonamides, sulfones, other antibiotics, atovaquone |
| <b>Gametocides</b>           | Primaquine for Plasmodium falciparum<br>Quinine for <i>P. vivax</i> , <i>P. malariae</i> , <i>P. ovale</i> .                                                                                |
| <b>Sporontocides</b>         | Primaquine, chloroquine.                                                                                                                                                                    |



# OBJECTIVES AND USE OF ANTIMALARIAL DRUGS

The aims of using drugs in relation to malarial infection are:

- (i) To prevent clinical attack of malaria (prophylactic).
  - (ii) To treat clinical attack of malaria (clinical curative).
  - (iii) To completely eradicate the parasite from the patient's body (radical curative).
  - (iv) To cutdown human-to-mosquito transmission (gametocidal).
- These are achieved by attacking the parasite at its various stages of life cycle in the human host .
  - Antimalarials that act on erythrocytic schizogony are called *erythrocytic schizontocides*
  - Antimalarials that act on pre erythrocytic as well as exoerythrocytic (*P. vivax*) stages in liver are called *tissue schizontocides*,
  - Antimalarials which kill gametocytes in blood are called *gametocides*.

## Antimalarial therapy is given in the following forms.

### 1. Causal prophylaxis

- A. The **pre erythrocytic phase** (in liver), which is the *cause* of malarial infection and clinical attacks, is the target for this purpose.
- B. **Primaquine** is a causal prophylactic for all species of malaria, but has not been used in mass programmes, because of its toxic potential.
- C. **Proguanil** is a causal prophylactic, primarily for *P.f.*, but is not used in India, because of weak activity against liver stages of *P.v.*, and rapid development of resistance when used alone.
- D. A combined formulation of **atovaquone (250 mg) + proguanil (100 mg)** is commonly used as a prophylactic by Americans and other western travellers visiting malaria endemic areas.
- E. Atovaquone also is active against preerythrocytic stage of *P.f.*, but is **not approved** in India.

## 2. Suppressive prophylaxis

The schizontocides which suppress the erythrocytic phase and thus attacks of malarial fever can be used as prophylactics.

### A. Chloroquine (CQ) 300 mg (base\*) or 5 mg/ kg weekly.

- In travellers, start one week before with a loading dose of 10 mg/kg and continue till one month after return from endemic area.
- However, it can be used as a prophylactic only in areas with CQ-sensitive *P.f.* (Mexico, Argentina, etc.). Since CQ-resistant *P.f.* is now widespread in India, and there are no exclusively *P.v.* areas, CQ is no longer employed as prophylactic in India.

### B. Mefloquine 250 mg started 1–2 weeks before and taken weekly till 4 weeks after return from endemic area, has been used for areas where CQ-resistant *P.f.* is prevalent.

- In India, use of mefloquine for prophylaxis is not allowed among residents, but may be used by travellers.

### C. Doxycycline 100 mg daily starting day before travel and taken till 4 weeks after return from endemic area for CQ-resistant *P.f.*, is an alternative regimen for short-term (maximum 6 weeks) visitors and those unable to take mefloquine.

- It is contraindicated in pregnant women and children < 8 yr.

**D. Proguanil 200 mg daily with chloroquine 300 mg weekly** affords substantial protection against moderately Cqresistant *P. falciparum*.

This has been successfully used in Africa, but found ineffective, and not employed in India.

- Chemoprophylaxis of malaria should be limited to short-term use in special risk groups, such as
  - nonimmune travellers,
  - nonimmune persons living in endemic areas for fixed periods (army units, labour forces),
  - infants, children and
  - pregnant women (falciparum malaria has serious consequences in the pregnant).
    - Intermittent preventive therapy (IPTp) in the form of one dose pyrimethamine (75 mg) + sulfadoxine (1500 mg) each is 2<sup>nd</sup> and 3<sup>rd</sup> trimester (gap not < 1 month) is recommended by WHO only in areas with high *P.f.* endemicity (*P.f.* >30%) for pregnant women.

### 3. Clinical cure

The erythrocytic schizontocides are used to terminate an episode of malarial fever.

The available drugs can be divided into:

- (a) **High-efficacy drugs:** Artemisinin, CQ, amodiaquine, quinine, mefloquine, halofantrine, lumefantrine and atovaquone. These drugs can be used singly to treat attacks of malarial fever, but are now generally combined
  - (b) **Low-efficacy drugs:** Proguanil, pyrimethamine, sulfonamides, tetracyclines and clindamycin. These drugs are used only in combination for clinical cure.
- The faster acting drugs are preferred, particularly in falciparum malaria where delay in treatment may result in death even if the parasites are cleared from blood by the drug.

## 4. Radical cure

- A radical curative is needed in relapsing malaria, while in falciparum malaria — adequate treatment of clinical attack leaves no parasite in the body.

Drug of choice for radical cure of vivax and ovale malaria is:

1. Primaquine 15 mg daily for 14 days.
2. Tafenoquine, a new long-acting 8-aminoquinoline exoerythrocytic schizontocide, is being developed as a single dose antirelapse drug for vivax malaria.

## 5. Gametocidal

- This refers to elimination of the male and female gametes of *Plasmodia* formed in the patient's blood.
- Gametocidal action is of no benefit to the patient being treated, but will reduce the transmission to mosquito.
- **Primaquine** is gametocidal to all species of *Plasmodia*, while artemisininins have weak lethal action on early-stage but not mature gametes.
- Gametes exposed to **proguanil or pyrimethamine** may fail to carry on the life cycle normally in the mosquito.
- Adequate control of clinical attacks will reduce formation of gametes.
- A **single 45 mg (0.75 mg/kg) dose of primaquine** is employed immediately after clinical cure of falciparum malaria to kill the gametes and cut down transmission to mosquito.
- This should be given even when an **artemisinin** is used for clinical cure because artemisininins do not kill all the gametes.
- **Primaquine** used for radical cure of vivax malaria eliminates *P.vivax* gametes as well.

**A. Vivax (also ovale, malariae) malaria**

1. Chloroquine 600 mg (10 mg/kg) followed by 300 mg (5 mg/kg) after 8 hours and then for next 2 days (total 25 mg/kg over 3 days) + Primaquine 15 mg (0.25 mg/kg) daily × 14 days

*In occasional case of chloroquine resistance*

2. Quinine 600 mg (10 mg/kg) 8 hourly × 7 days + Doxycycline 100 mg daily × 7 days or  
+ Clindamycin 600 mg 12 hourly × 7 days  
+ Primaquine (as above)  
or  
Artemisinin-based combination therapy (see below)  
+ Primaquine (as above)

**B. Chloroquine-sensitive falciparum malaria<sup>§</sup>**

1. Chloroquine (as above) + Primaquine 45 mg (0.75 mg/kg) single dose (as gametocidal)

**C. Chloroquine-resistant falciparum malaria**

- 1.\* Artesunate 100 mg BD (4 mg/kg/day) × 3 days +  
Sulfadoxine<sup>#</sup> 1500 mg (25 mg/kg) + Pyrimethamine 75 mg (1.25 mg/kg) single dose  
or
2. Artesunate 100 mg BD (4 mg/kg/day) × 3 days +  
Mefloquine<sup>#</sup> 750 mg (15 mg/kg) on 2nd day and 500 mg (10 mg/kg) on 3rd day.  
or
3. Artemether 80 mg + Lumefantrine 480 mg twice daily × 3 days (child 25–35 kg BW  $\frac{3}{4}$  dose;  
15–25 kg BW  $\frac{1}{2}$  dose; 5–15 kg BW  $\frac{1}{4}$  dose)  
or
4. Arterolane (as maleate) 150 mg + Piperaquine 750 mg once daily × 3 days  
or
5. Quinine 600 mg (10 mg/kg) 8 hourly × 7 days  
+ Doxycycline 100 mg daily × 7 days or + Clindamycin 600 12 hourly × 7 days

\*First line ACT under NVBDCP

<sup>#</sup>Sulfadoxine-pyrimethamine (S/P) alone and mefloquine alone are also used, but should preferably be combined with artesunate.

<sup>§</sup>In India (including under NVBDCP) all *P.f.* cases, irrespective of CQ-resistance status, are treated with artemisinin-based combination therapy (ACT).

## ***Severe and complicated falciparum malaria***

- This includes *P. falciparum* infection attended by any one or more of—
  1. Hyperparasitaemia,
  2. Hyperpyrexia,
  3. Fluid and electrolyte imbalance,
  4. Acidosis,
  5. Hypoglycaemia,
  6. Prostration,
  7. Cardiovascular collapse,
  8. Jaundice,
  9. Severe anaemia,
  10. Spontaneous bleeding,
  11. Pulmonary edema,
  12. Haemoglobinuria,
  13. Black water fever,
  14. Renal failure
  15. Cerebral malaria.
- Parenteral (i.m./i.v.) drugs have to be used; oral drugs may be substituted when the condition improves.

## **Treatment of severe and complicated falciparum malaria**

Artesunate: 2.4 mg/kg i.v. or i.m., followed by 2.4 mg/kg after 12 and 24 hours, and then once daily for 7 days. Switchover to 3 day oral ACT in between whenever the patient can take and tolerate oral medication.

or

Artemether: 3.2 mg/kg i.m. on the 1st day, followed by 1.6 mg/kg daily for 7 days. Switchover to 3 day oral ACT in between whenever the patient is able to take oral medication.

or

Arteether: 3.2 mg/kg i.m. on the 1st day, followed by 1.6 mg/kg daily for the next 4 days. Switchover to 3 day oral ACT inbetween whenever the patient is able to take oral medication.

or

Quinine diHCl: 20 mg/kg (loading dose) diluted in 10 ml/kg 5% dextrose/dextrose-saline and infused i.v. over 4 hours, followed by 10 mg/kg (maintenance dose) i.v. infusion over 4 hours (in adults) or 2 hours (in children) every 8 hours, until patient can swallow. Switchover to oral quinine 10 mg/kg 8 hourly to complete the 7 day course.

# Treatment for Prevention of Malaria in travelers

**Table 35–5.** Drugs for the prevention of malaria in travelers.<sup>1</sup>

| Drug                     | Use <sup>2</sup>                                                                                                                        | Adult Dosage (all oral) <sup>3</sup>                                                                                                                                       |
|--------------------------|-----------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Chloroquine              | Areas without resistant <i>Plasmodium falciparum</i>                                                                                    | 500 mg weekly                                                                                                                                                              |
| Malarone                 | Areas with multidrug-resistant <i>P falciparum</i>                                                                                      | 1 tablet (250-mg atovaquone/100-mg proguanil) daily                                                                                                                        |
| Mefloquine               | Areas with chloroquine-resistant <i>P falciparum</i>                                                                                    | 250 mg weekly                                                                                                                                                              |
| Doxycycline              | Areas with multidrug-resistant <i>P falciparum</i>                                                                                      | 100 mg daily                                                                                                                                                               |
| Primaquine <sup>4</sup>  | Terminal prophylaxis of <i>Plasmodium vivax</i> and <i>Plasmodium ovale</i> infections; alternative for <i>P falciparum</i> prophylaxis | 30-mg base daily: for terminal prophylaxis take for 14 days after travel; for chemoprevention begin 1–2 days before travel, take during travel and for 7 days after travel |
| Tafenoquine <sup>4</sup> | Alternative for <i>P falciparum</i> prophylaxis                                                                                         | 200 mg once daily for 3 days and then weekly until 1 week after last exposure                                                                                              |

## *Falciparum malaria during pregnancy*

- An attack of falciparum malaria occurring during pregnancy has serious implications both for the mother as well as the foetus.
- It must be treated promptly and aggressively.

Drugs recommended are:

1. Quinine 600 mg TDS × 7 days + clindamycin 300 mg TDS/QID (20 mg/kg) for 7 days; can be used during all trimesters, especially the 1st.
2. Artemisinin-based therapy is a better tolerated 3 day regimen, which may be used during the 2nd and 3rd trimester as an alternative to 7 day quinine + clindamycin therapy.

# PREVENTION

- Spraying of DDT, BHC, etc., insecticides at intervals in mosquito breeding places like pools, ponds, ditches and stagnant water.
- Introduction of larvivorous fishes like Gambusia, insectivorous plants like Utricularia into the places where mosquitoes breed.
- Avoiding mosquito bite by using mosquito nets, mosquito repellents, etc.
- Spraying of kerosene, pyrethrum oil, etc., on stagnant water.