



Plasmodium and Babesia

Presented by

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- **General characters:**

1- No special organs for locomotion (move by gliding) in some stages of their life cycle.

2- Multiply by alternation of sexual and asexual generations.

Plasmodia

Malaria parasites

Plasmodium vivax

Plasmodium ovale

Plasmodium malariae

Plasmodium falciparum

Causes

Causes

Causes

Causes

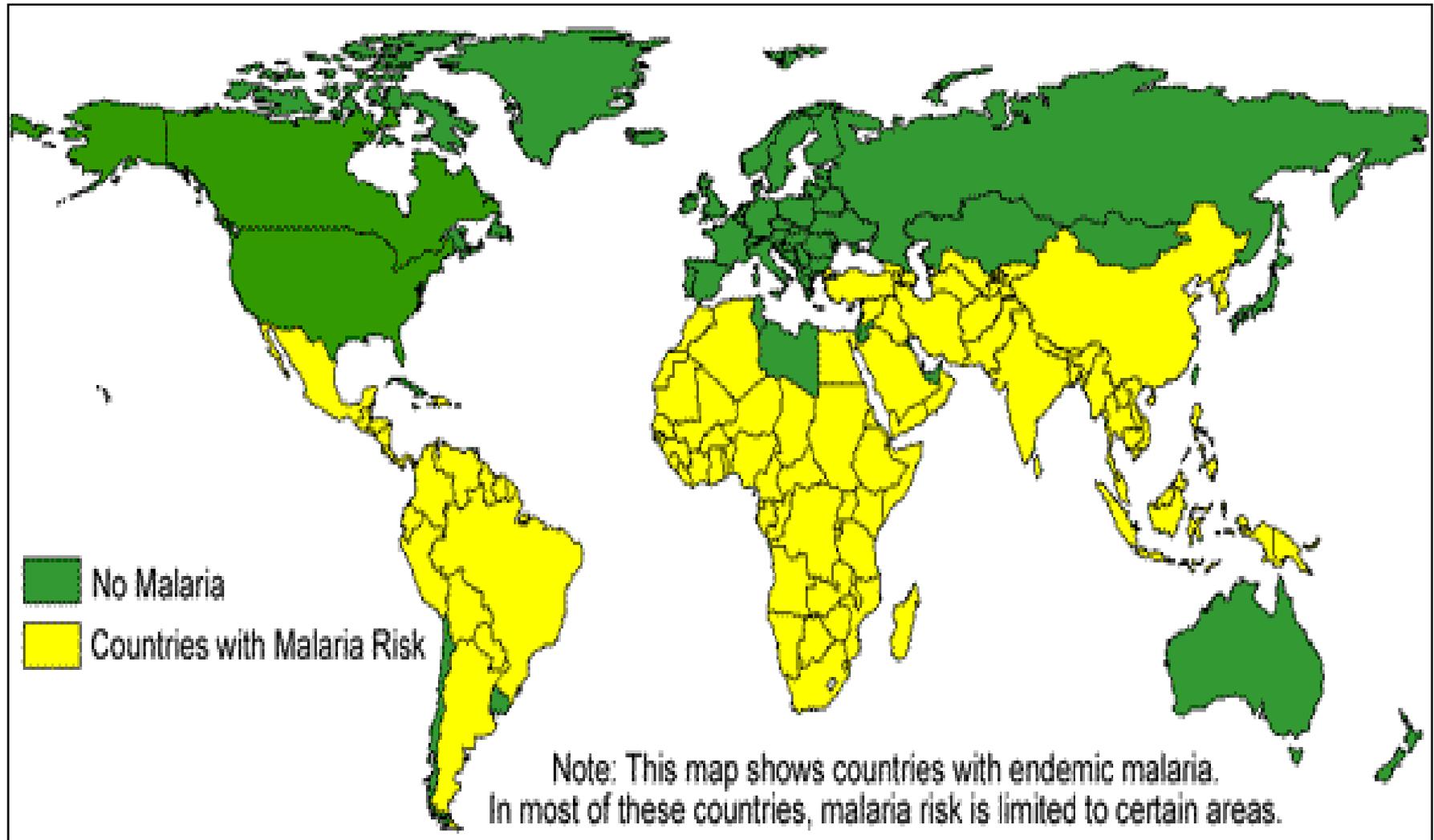
Benign tertian malaria

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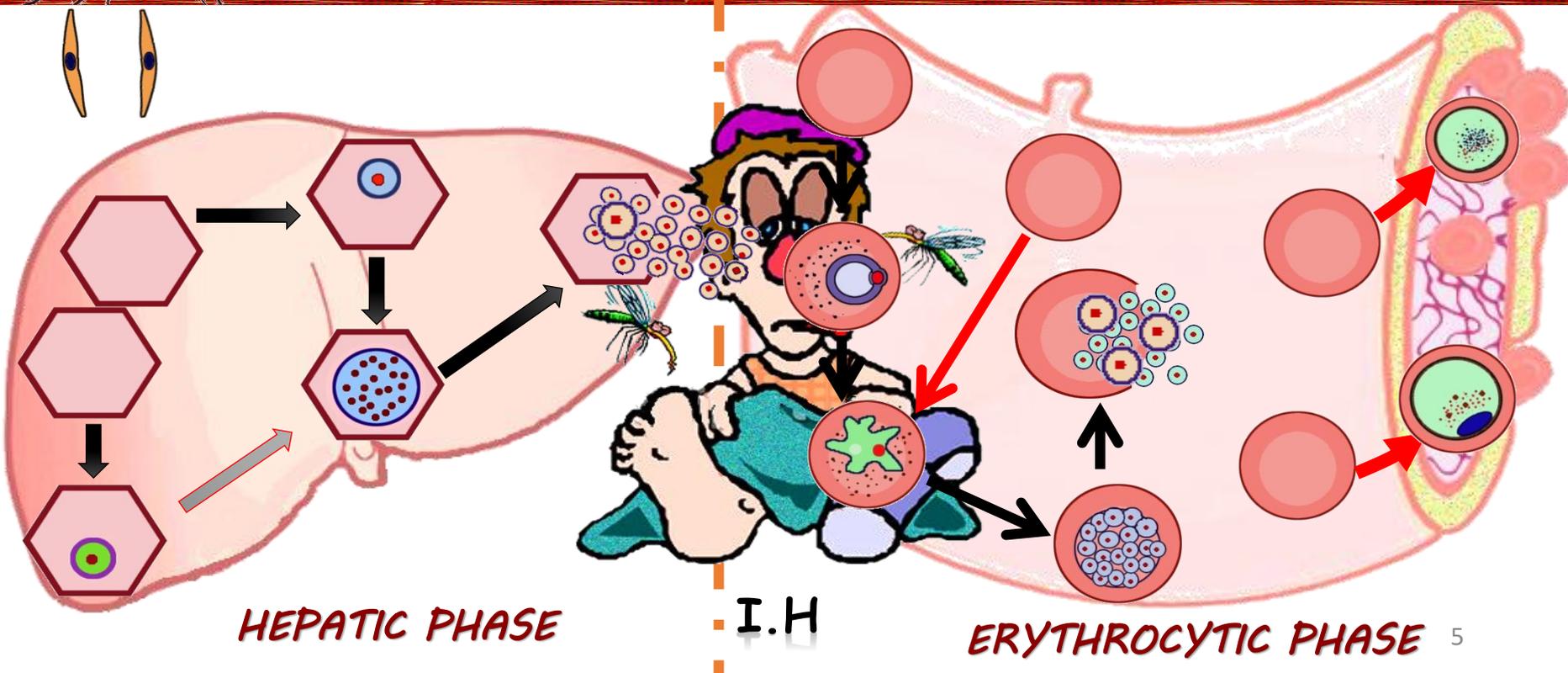
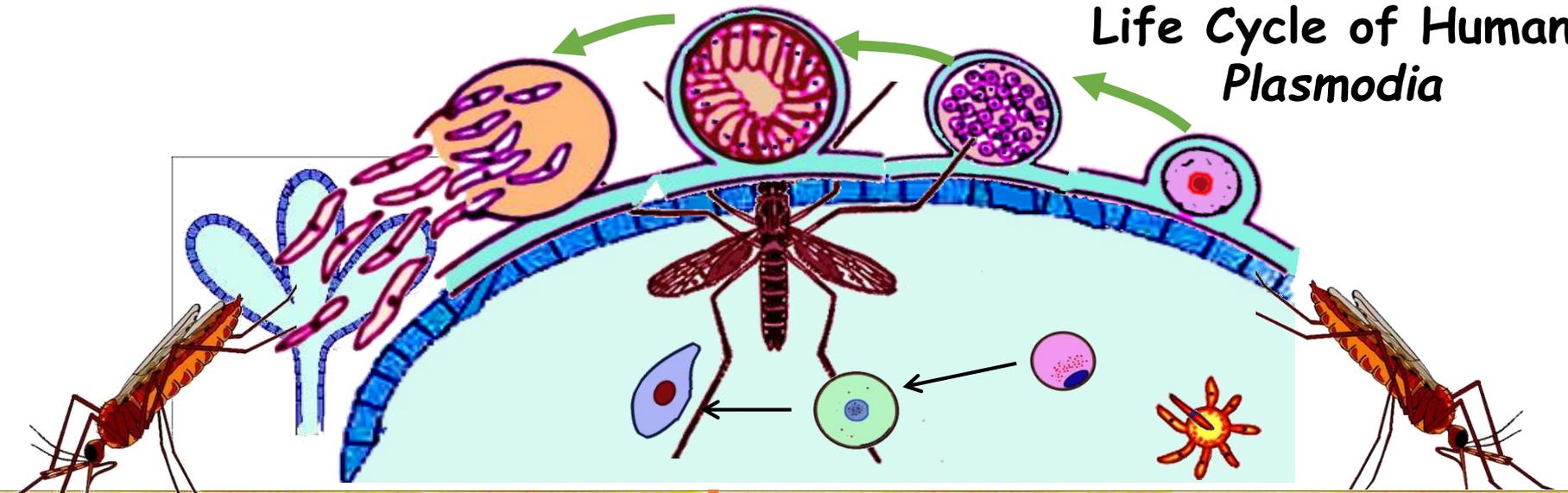
Benign quartan malaria

**Malignant malaria
tertian or sub
tertian**

Malaria endemic areas



Life Cycle of Human Plasmodia



HEPATIC PHASE

I.H

ERYTHROCYTIC PHASE

Life cycle

The life cycle of malaria passes in 2 alternate hosts

Man (I.H)

The asexual cycle
(Schizogony) takes
place ends by the
formation of male and
female gametocytes
(Gametogony).

Female *Anopheles* (D.H)

The sexual cycle
(Sporogony) takes place.



Life cycle of malaria parasites in man

Intrinsic cycle

Asexual cycle
in the liver

Asexual cycle
in RBCs

Gametogony

Exo-erythrocytic
schizogony
(liver phase)

Erythrocytic
schizogony
(blood phase)

Formation of **male** and
female gametocytes in
infected RBCs

Primary tissue
phase

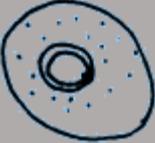
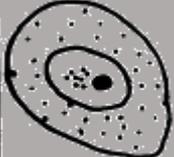
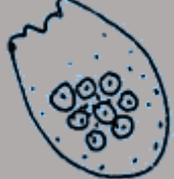
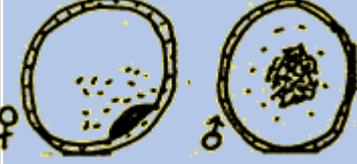
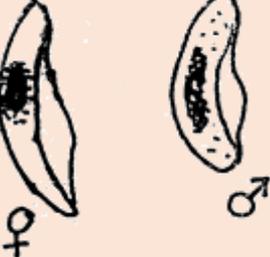
Secondary
tissue phase

Relapse occurs with *P. vivax* and
P. ovale

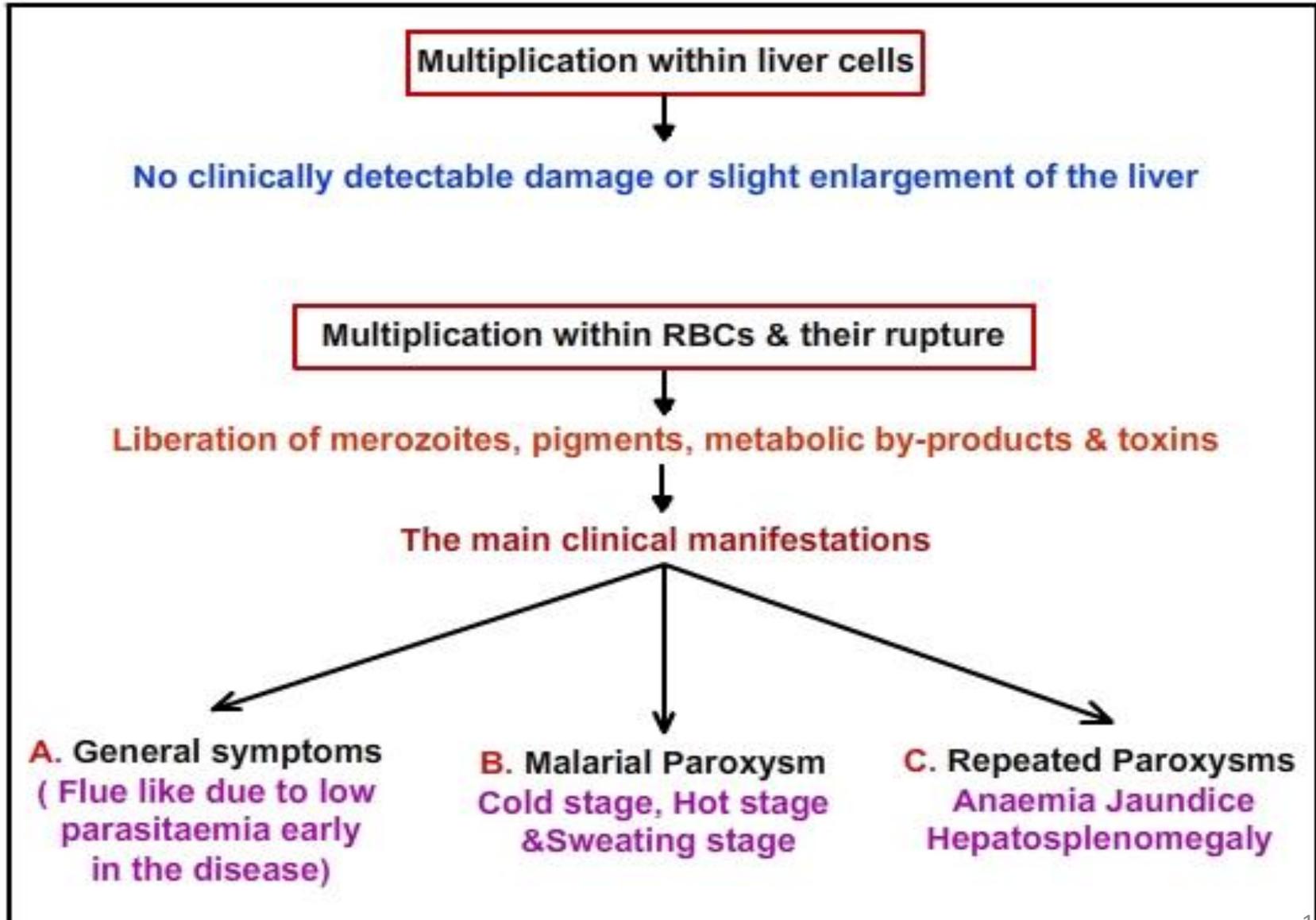
Life cycle of malaria parasites in mosquito

Extrinsic cycle or Sporogony

- Start when female *Anopheles* bites infected person for blood meal that containing all stages of malaria parasite. All the stages are digested in the stomach of the mosquito except **the gametocytes (I.S to mosquito)**.
- End by Fertilization between microgamete & the macrogamete forming **a zygote** ➔ elongated **ookinete** ➔ spherical **oocyst** ➔ sporocyst containing a large number of **sickle-shaped sporozoites (I.S to man)**.

	<i>Plasmodium vivax</i>	<i>Plasmodium ovale</i>	<i>Plasmodium malariae</i>	<i>Plasmodium falciparum</i>
Trophozoite (ring stage)				
Mature trophozoite				
Schizont				
Gametocyte				
	Infect young RBCs		Old RBCs	All RBCs ⁹ ages

Pathogenesis



Clinical pictures

1) General symptoms in all types of malaria

A- Prodromal symptoms
(Low parasitemia and asynchronous RBCs rupture)

Low grade fever, malaise, headache and pain in bone and joints.

B- Malarial paroxysm

➤ Due to **synchronous** rupture of a large number of parasitized RBCs and liberation of merozoites with malarial pigment and toxin into the circulation.

Cold stage (1/2- one hour): Sensation of cold, shivering and the patient is feverish. The skin is pale and cyanotic.

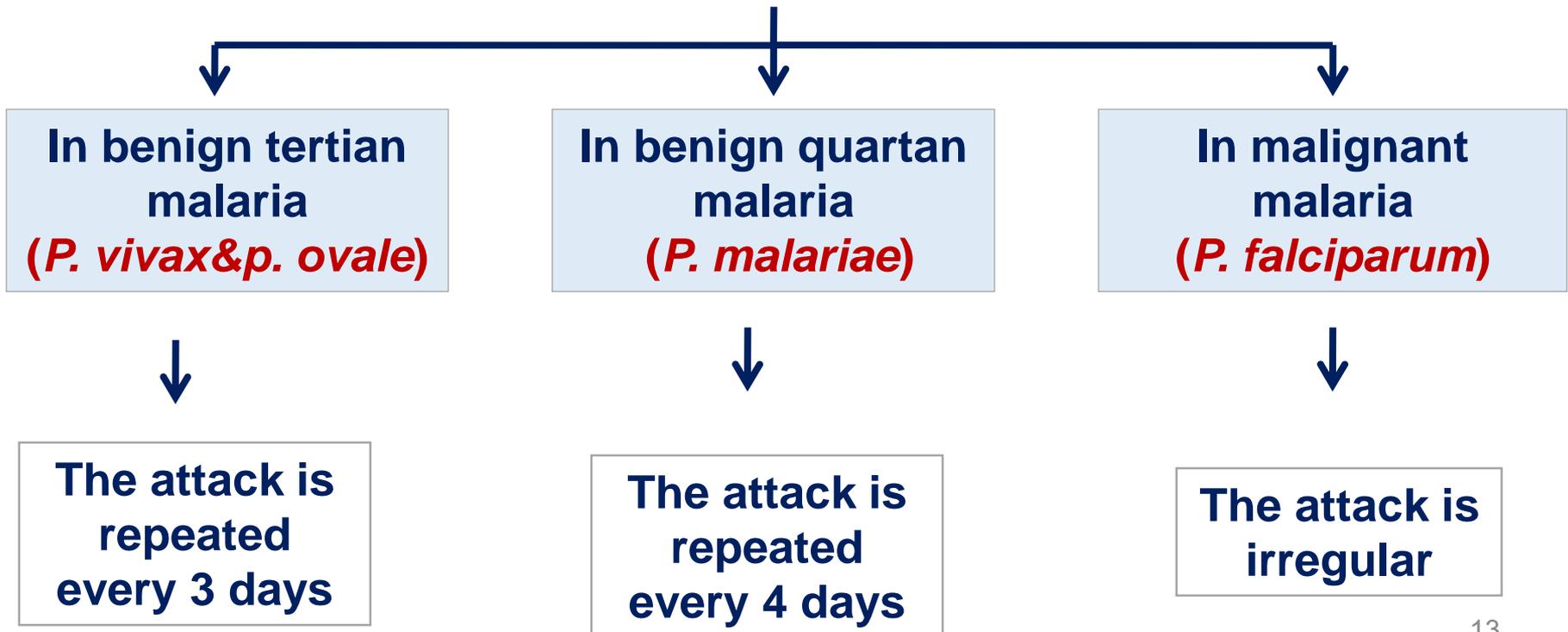
Hot stage (1-4 hours): High fever (40 C° or more), hot dry skin, flushed face, headache and pain in limbs and back .

C - Repeated paroxysms
(Anemia, jaundice, hepatosplenomegaly)

Sweating stage (1-4 hours): Profuse sweating with ↓ temperature, moist and coal skin).

N.B. The patient becomes normal and temperature is normal till the second paroxysm occurs. Malaria paroxysms occur for at least 2 weeks or more with decreasing intensity then stop.

The attacks recur at the following interval



2) Specific symptoms

In *P. malariae*

Antigen antibody complex is deposited in the kidney glomeruli ⇒ **nephrotic syndrome** with the occurrence of edema and protein and cast in urine.

In *P. falciparum*

➤ **Adhesion phenomena (pernicious syndrome)** occurs in infected RBCs containing **trophozoites and schizonts**. RBCs clump together and stick to the endothelium of the blood vessels ⇒ **vascular obstruction of small blood vessels of internal organs** ⇒ tissue anoxia of organs including brain, kidney, GIT, heart, lung, liver and adrenal

➤ **Black water fever**

Acute intravascular haemolysis of infected RBCs ⇒ severe anaemia, fever, jaundice, haemoglobinuria (dark red urine) and acute renal failure due to inadequate quinine treatment.

➤ **Cerebral malaria:** headache, high fever, convulsion, paralysis, coma and death.

➤ **Algid malaria:** shock, collapse, sudden ↓ in blood pressure due to adrenal insufficiency

➤ **Gastrointestinal malaria:** diarrhoea or dysentery with bloody loose stool ⇒ malabsorption and dehydration

➤ **Congenital malaria** ⇒ abortion.

• Why *Plasmodium falciparum* causes malignant malaria and is more dangerous ????????????

• Differences between malarial relapse and recrudescence ??????

Laboratory Diagnosis

Direct

Microscopic: Thin and thick blood films stained with **Giemsa and Leishman** reveal different stages (rings, trophozoites, schizonts and gametocytes) in all types of malaria except in *P. falciparum* only rings and gametocytes are seen in the peripheral blood (due to adhesion phenomena).

Indirect

1-Serological tests: CFT, IHT, ELISA, FAT, or rapid strip or dip stick test to detect circulating antigens using monoclonal antibodies.
2-Molecular diagnosis: by PCR.

Treatment

Tissue schizonticide and hypnozoitocides

Blood schizonticides

Treatment of complications

Chemoprophylaxis

➤ **Primaquine**

It is also gametocidal

Chloroquine-sensitive

➤ **Chloroquine**

Chloroquine-resistant

Fansidar: Pyrimethamine & sulphadoxine
Artemisinin-based: Pyrimethamine/ sulphadoxine + artesunate
Mefloquine:

➤ **Blood transfusion** in severe anaemia.
➤ **Haemodialysis** in renal failure.

➤ **Weekly** mefloquine
➤ **Daily** doxycycline

When and How long?????

Malaria- Prevention

Key methods to prevent malaria transmission are:

- Long lasting insecticide impregnated nets (LLINs)
- Indoor residual spraying
- Mosquito repellents
- Preventative drug treatments



Babesia

❖ *Babesia* species cause a disease known as babesiosis.

❖ **Causative organisms:**

-*Babesia microti*

-*Babesia divergens* (commonly affects splenectomized-
persons)

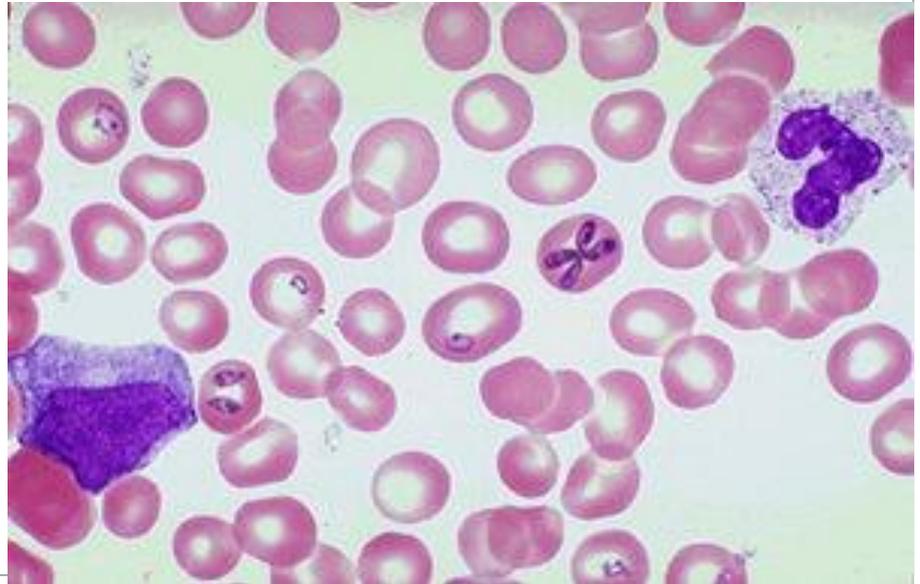
❖ **Habitat:** RBCs.

❖ **D.H and vector of transmission:** Hard tick

❖ **I.H:** Cattle and rodents and occasionally man.

Differences between *Babesia* and *Plasmodium*

- No hepatic stage.
- Merozoites arranged in pairs or Maltese cross
- No pigments
- Vector is the hard tick



Mode of transmission:

1- Bite of hard tick (sporozoites)

2- Blood transmission (erythrocytic stages)

Pathogenesis & symptomatology

❖ Disease : Babesiosis.

The parasite invades the RBCs where it multiplies by budding ⇒ the cell ruptures and the released parasites invade other cells leading to anaemia, jaundice, and hepatosplenomegaly.

Laboratory diagnosis

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graph TD; A[Laboratory diagnosis] --> B[Direct]; A --> C[Indirect];
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Direct

➤ **Stained blood film by Giemsa or Leishman** to detect the intraerythrocytic parasites.

➤ **Animal inoculation** : blood sample is injected intraperitoneally in hamster, the parasites can be detected in animal blood after 2 weeks in positive cases.

Indirect

➤ **Serological tests**

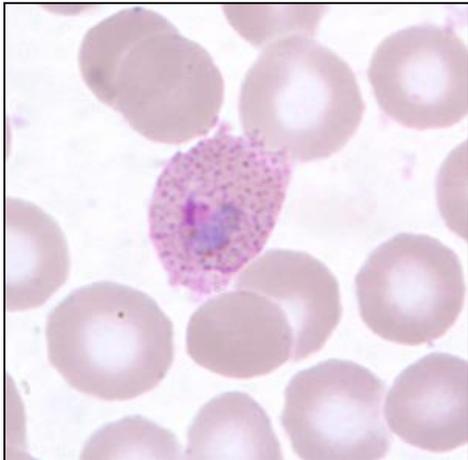
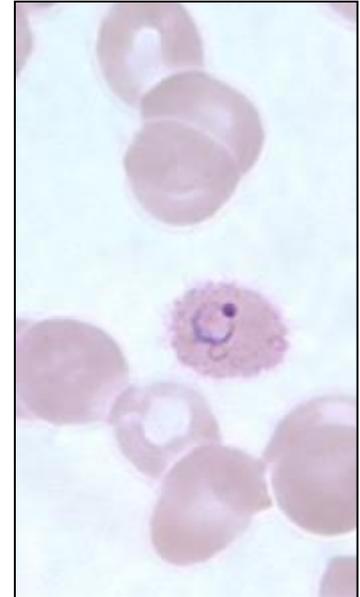
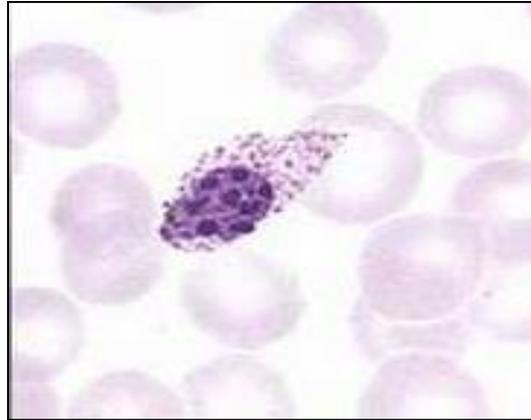
➤ **PCR to detect *Babesia* DNA**

Treatment

- 1. Quinine + clindamycin.**
- 2. Exchange blood transfusion in severe cases**



Guess the stage?



Guess what is this?

