



Plasmodium vivax
cerebral MALARIA
CASE STUDY

Definition

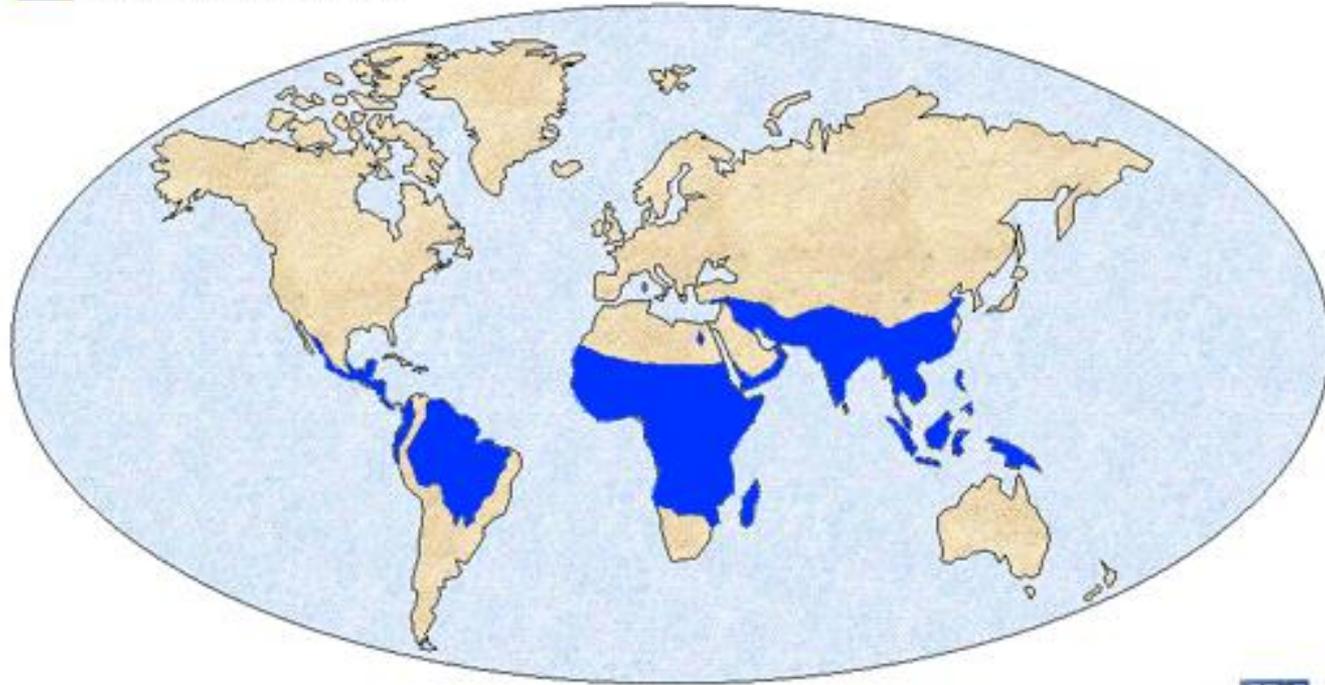
- Malaria is an infectious disease, which is caused by protozoan parasites of genus *Plasmodium*.

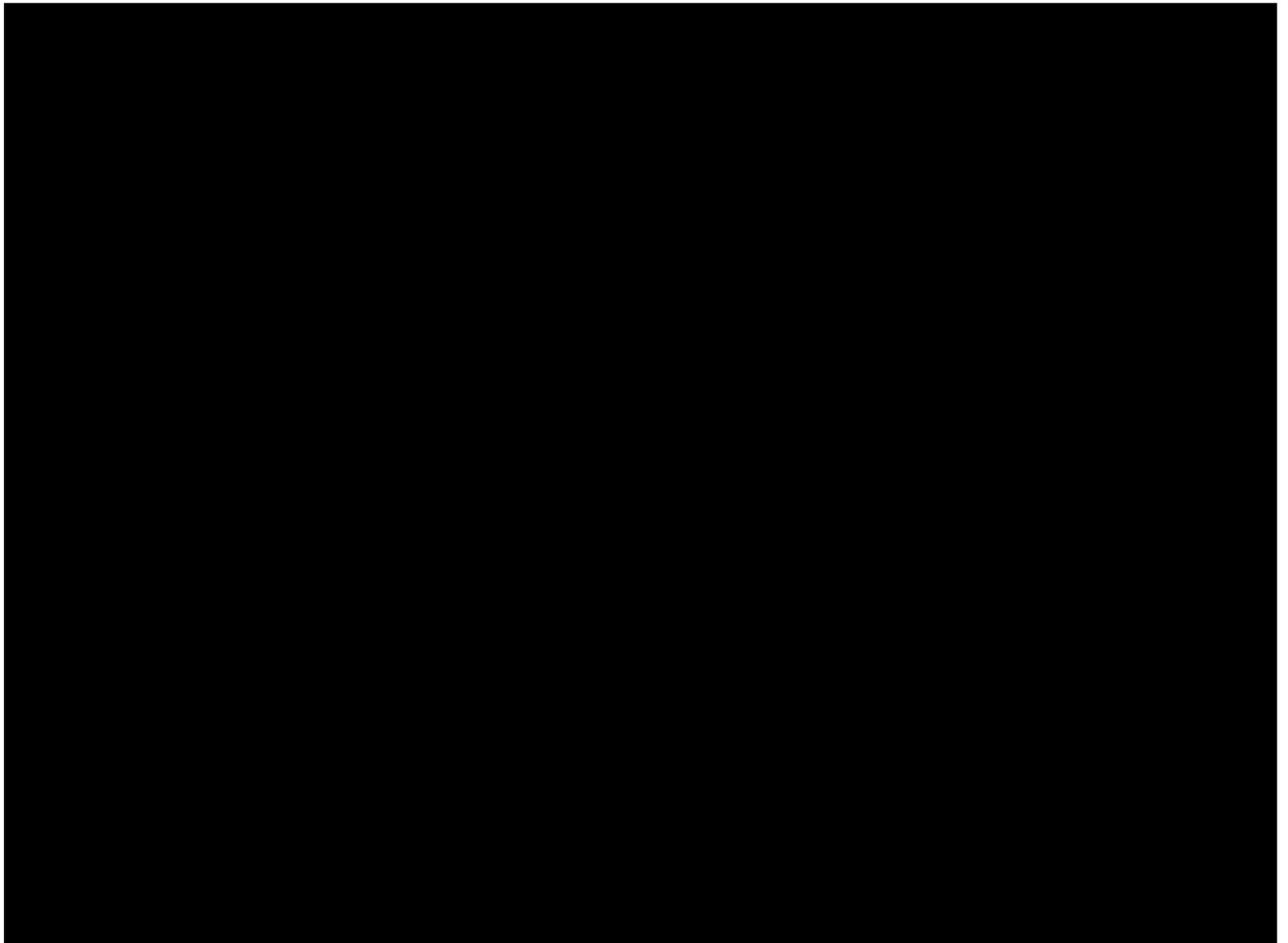
Causative Organism :

- ❖ *Plasmodium falciparum*
- ❖ *Plasmodium vivax*
- ❖ *Plasmodium ovale*
- ❖ *Plasmodium malariae*

Malaria is Prevalent in Tropical Climates

■ Distribution of Malaria





Clinical IP
Prodromal ss
Paroxysms
Systemic



High fever



Severe sweating



Chills



Vomiting



Headache



Tiredness

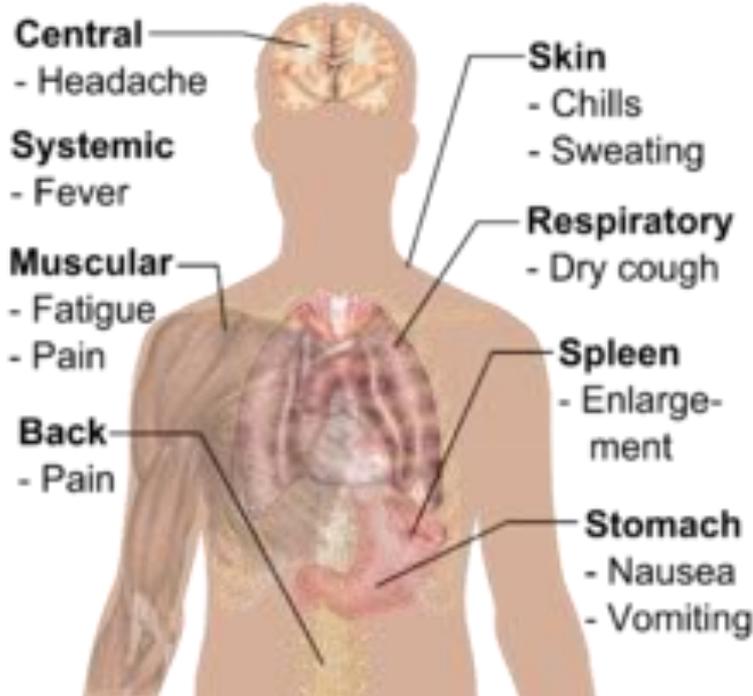


Muscle aches



Weakness

Symptoms of Malaria



Severe malaria

- Seizures
- Respiratory distress
- Anemia
- Organ failure
- Coma
- **Death**

- ❑ **Cerebral malaria** is a severe **malaria** presenting with neurological symptoms, including **coma**, or with a coma that lasts longer than 30 minutes after a seizure, OR it is any **impairment of consciousness** or **convulsions** in a patient of Malaria.
- ❑ Cerebral involvement is a frequent cause of mortality in malaria, especially in children, pregnant women, and nonimmune adults. Like other severe systemic complications, cerebral malaria is generally the result of infection by *Plasmodium falciparum*.
- ❑ Although *P. vivax* has been reported to cause cerebral symptoms in India and China, to our knowledge only 45 cases of central nervous system *P. vivax* malaria have been previously reported in the English language literature, about half of these cases have occurred in children.
- ❑ Here we report a unique case of ***P. vivax*** infection complicated by cerebral malaria in adult.

Pathogenesis

- ❑ The exact pathogenetic mechanism however remains elusive
- ❑ *P. vivax* can cause both sequestration-related and nonsequestration-related complications of severe malaria
- ❑ *P. vivax* infected RBCs form rosettes, like those seen in *P. falciparum* infections.
- ❑ Although *P. vivax* is seldom fatal, it is a major cause of morbidity.

Case Study

- ❑ A 28-years-old apparently healthy **Indian** female.
- ❑ Two months post normal vaginal delivery, she came to Saudi Arabia for Hajj on October 2011.
- ❑ Two weeks later, she presented to a polyclinic with a 4 days history of **intermittent fever** and **shortness of breath** and she was drowsy and confused.
- ❑ Initially diagnosed and treated as community acquired pneumonia

Case Study, cont.

- ❑ However, her condition didn't improve and she returned back next day with temperature of **40°C**, respiratory distress, hemodynamic instability and deterioration of renal function.
- ❑ CBC revealed normocytic normochromic **anemia**
- ❑ Chest X ray showed bilateral infiltrate
- ❑ Echocardiography showed EF 35%.
- ❑ **Diagnostic impression ??**
- ❑ She received supportive treatment with no improvement

Case Study, cont.

❑ Five days later, she transferred to our hospital KAMC, non conscious with severe hypotension.

❑ Physical Examination she appeared ill

Nonconscious

Temp- **40°C**

Bp ↓, Pulse ↑, RR ↑

HSM

No focal neurological signs

❑ Treatment started as noradrenaline infusion, ARDS ventilation protocol and continuous renal replacement therapy (CRRT).

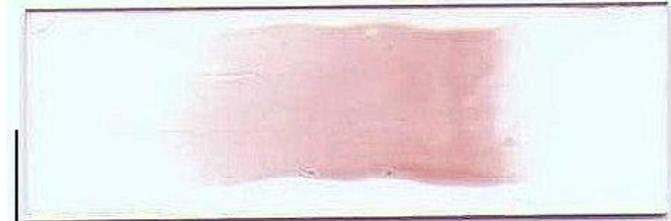
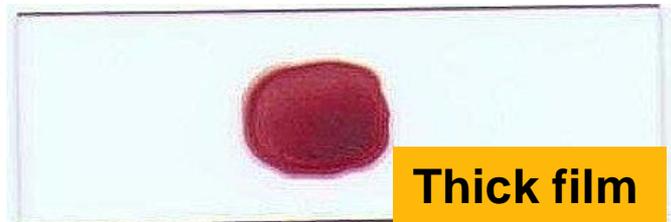
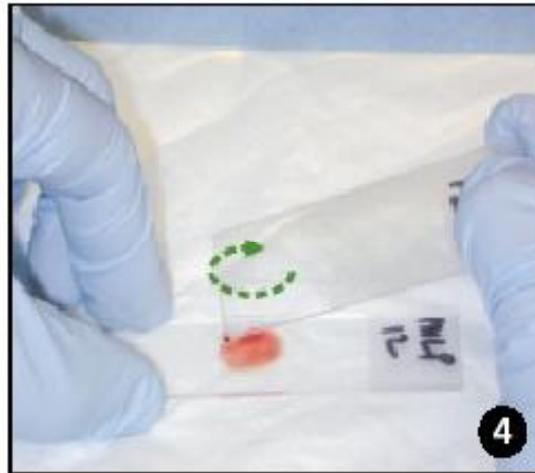
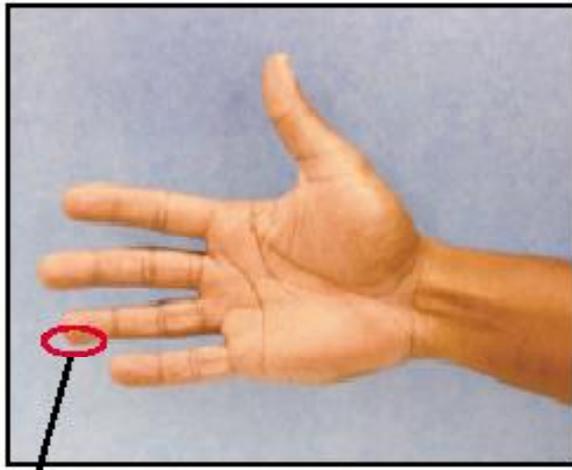


Lab Investigation

Data's	Observed Value	Normal Value
Hb	8.3 gm/dl(↓)	12-15
RBC	3.2 X10 ¹² /L (↓)	4.3-5.7
WBC	14.7 X10 ⁹ /L(↑)	3.9-11
Platelet	71 X10 ⁹ /L (↓)	150-400
Creatinine	1.6 mg/dl (↑)	0.6-1.3
Albumin	2.2 g/dl (↓)	3.5-5.2
Bili (T)	1.7 mg/dl(↑)	0.0-1
Bili (D)	0.7 mg/dl (↑)	0.0-0.3
Bili (ID)	0.7 mg/dl	Total-Direct

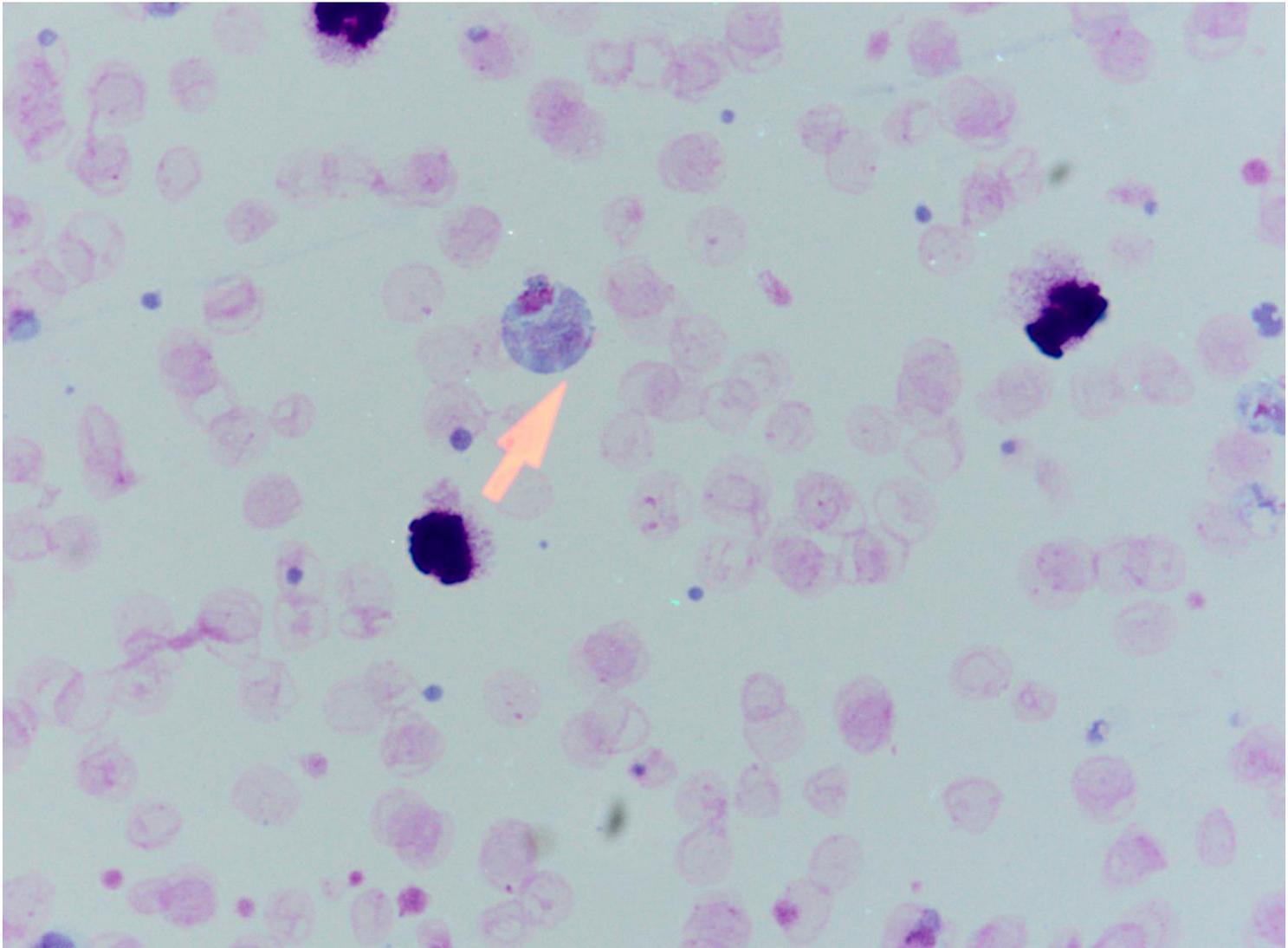
- ❑ Serology for **Dengue** fever proved negative.
- ❑ **Septic screen sent.** Came **positive** for **MRSA**. She started oral Meropenem, Vancomycin and Levofloxacin and adjusted dose with CRRT.
- ❑ In view of **non-improvement** since started treatment in previous hospital plus her country of origin from **India**, **malaria** was suspected

Thick and thin blood films were done

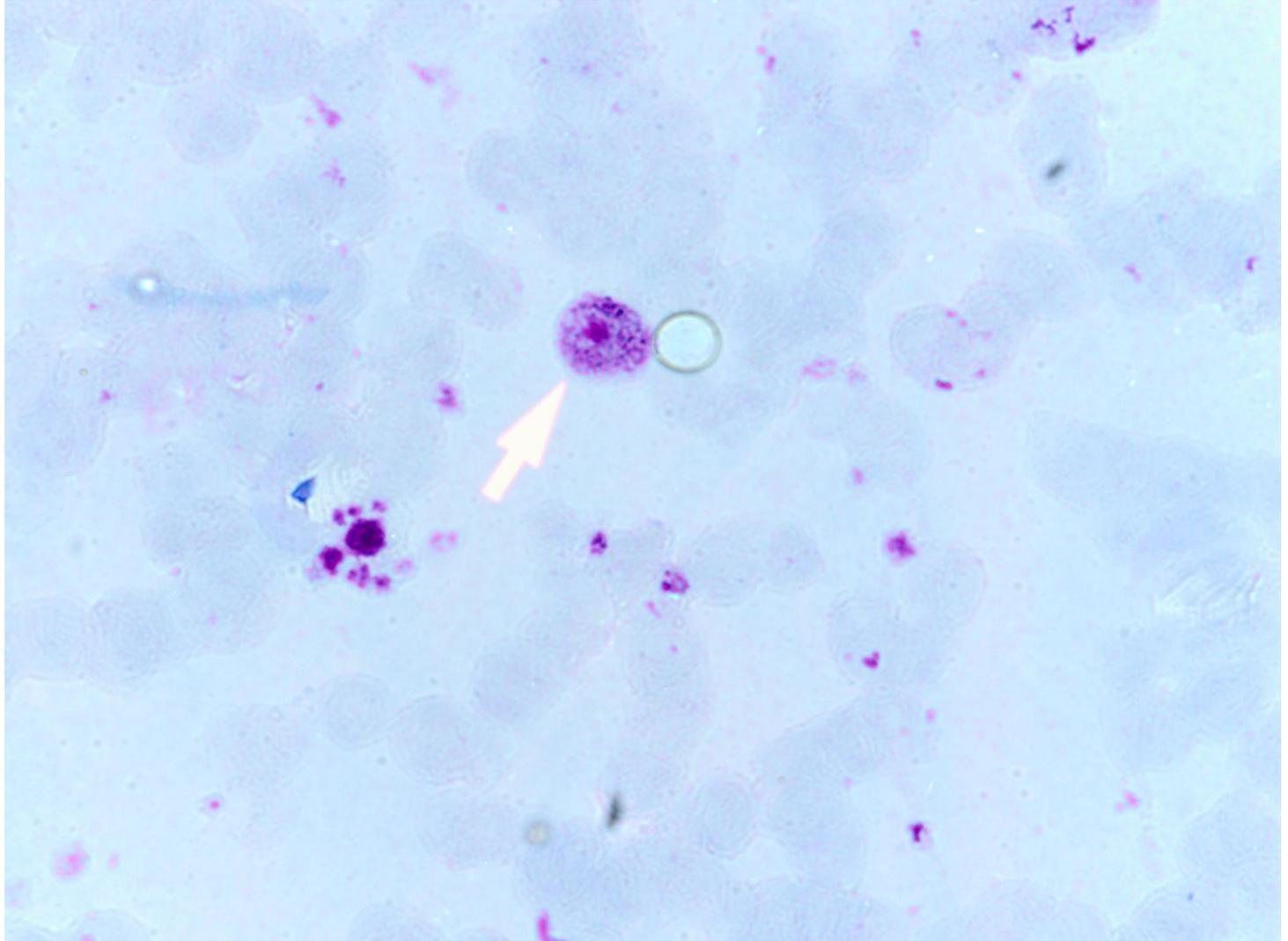


Thin film

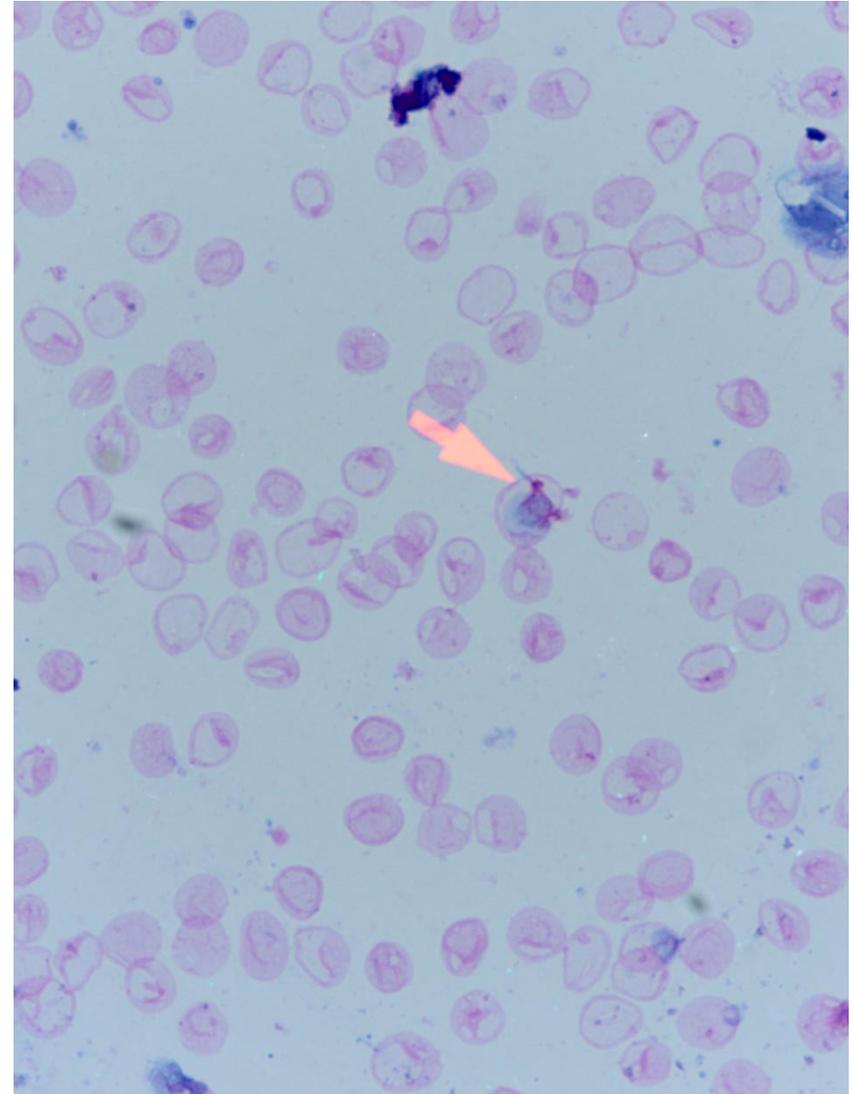
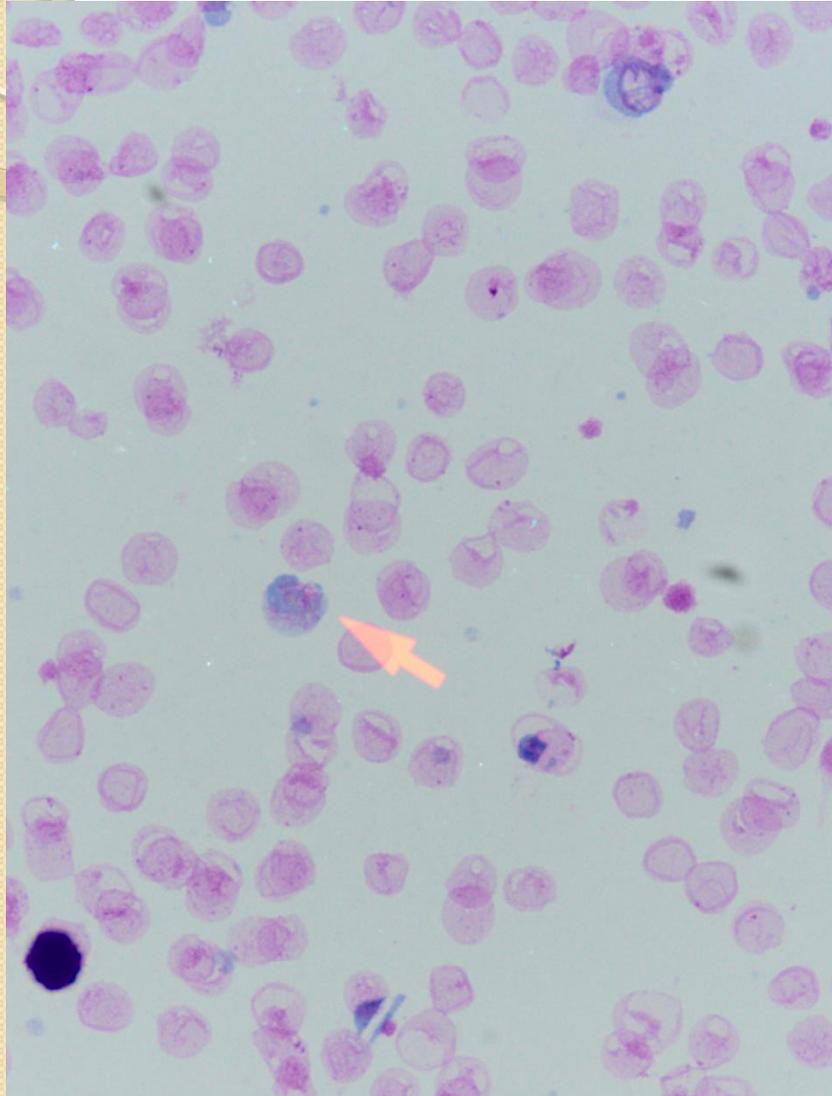
Thin film showed that infected RBCs are slightly enlarged, **macrogametocytes** appeared rounded in shape with homogeneous cytoplasm; diffuse delicate light brown pigment throughout the parasite; eccentric pink compact chromatin and it fill the red blood cell.



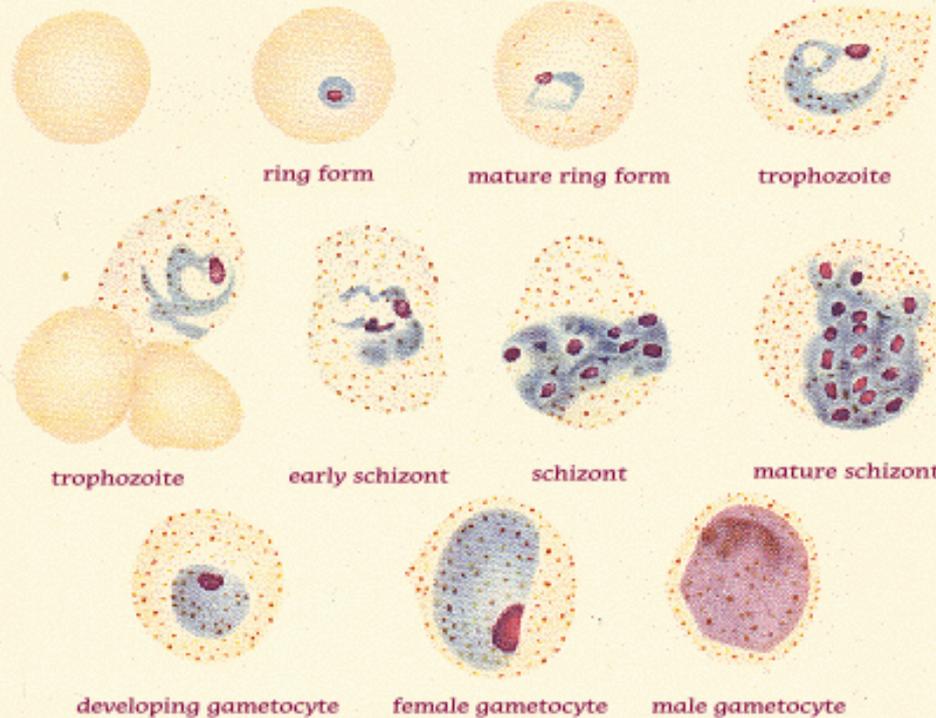
Thin film showed that infected RBCs are slightly enlarged, **Microgametocytes** appeared rounded in shape with large central pink to purple chromatin mass surrounded by pale or colorless halo and evenly distributed pigment and it fill the red blood cell.



Thin film showed that infected RBCs are slightly enlarged, Multishaped irregular ameboid parasite; large chromatin dot and a vacuole were seen



P. vivax



Diagnostic points:-

- 1.Red cells containing parasites are usually enlarged.
- 2.Schuffner's dots are frequently present in the red cells as shown above.
- 3.The mature ring forms tend to be large and coarse.
- 4.Developing forms are frequently present.

Diagnosis

- ❑ Thick and thin blood films: Positive *P. vivax*
- ❑ Rapid Antigen detection test: Positive *P. vivax* but
Negative *P. falciparum*
- ❑ Antifalciparum antibodies: Negative

Started **Quinine** in a dose of 600 mg I.V. TID on 11/11/11, plus other medical support. There was a **good response**

The patient's parasitemia became undetectable after 48 hours of treatment and she improved neurologically. Peripheral blood films were repeatedly reviewed to document a mixed malarial infection with *P. falciparum*, but this species could not be detected.

- ❑ During her stay in the hospital, on 24/11/11, the patient had **tonic-clonic seizures** treated with midazolam and phenytoin. Urgent CT brain done revealed signs suggestive of **cerebral malaria**. MRI brain was done on 27/11/11, also it was suggestive of **extensive vasculitis related to malaria**.
- ❑ Kidney function **improved**, starts to pass urine with improved creatinine and urea, D/C CRRT. Patient became fully **conscious**, extubated and transferred from ICU to ward on 29/11/11. She started **primaquine** for 14 days to avoid relapse.
- ❑ The patient was discharged one month after hospitalization, having made a full clinical and microbiologic recovery.

Treatment

- ❑ **Chloroquine** (4 Aminoquinolines)
- ❑ **Primaquine** (8 Aminoquinolines)
- ❑ **Quinine, Mefloquine** (Aryl-amino alcohols)
- ❑ **Fansidar** (Drug Combination)= sulfadoxine 500 mg+
Pyremethamine 25 mg
- ❑ **Mefloquine**
- ❑ **Artimesinine** (derived from leeches)
- ❑ **Pyremethamine**
- ❑ **Atovaquona**

Curative treatment (Radical cure):

- *Falciparum & malariae* malaria:=**blood schizonticides**

Chloroquine

Chloroquine resistant:

Quinine (650 mg/8h for 10 days)

Quinine +fansidar (to potentiate the action)

Mefloquine

Artimesinine

- *Vivax & ovlae* malaria:

Chloroquine+primaquine (Chloroquine alone is inefficient)

Prevention and control:

- Treatment of patients (source of infection)
- Chemoprophylaxis
- Vector control:
 - Insecticides
 - Destruction of breeding places
 - Avoid exposure to bite by:
 - Repellants
 - Clothes
 - Nets
- Vaccine trials

Chemoprophylaxis:

one week before travelling, during and 4 weeks after leaving endemic area

True prophylaxis= In healthy persons= Tissue schizonticides

- **Pyremethamine (daraprim)** 25mg/week (one tablet)
- **Mefloquine (drug of choice)**

Clinical prophylaxis=Suppressant treatment:

Elimination of asexual erythrocytic forms

Chloroquine 600 mg/week, 1 week before travelling