Traveller's diarrhea:

Diarrhea is the leading cause of illness in traveller's and it is usually short lived (2-5 days), self limited condition occur commonly in visitors of developing countries.

The most common identified pathogen causing traveller's diarrhea is toxigenic E.coli, however campylobacter infection appear to predominate in other parts of the world. Other common causative organism include salmonella, Shigella, rota virus and the Norwalk agent. Other less common cause include giardiasis and to less extent vibriosis.

Clinical features:

1-The onset is usually abrupt.

2- The stool is watery.

3- Abdominal cramps.

4-Anorexia and vomiting.

5-Fever may be present.

6-Examination of the abdomen:

Shows no abnormality or occasionally diffuse tenderness.

Treatment:

1- The disorder usually resolve spontaneously.

2-Antidiarrhoeal agent are best avoided as they may occasionally cause toxic dilatation of the colon.

3-Loperamide 4mg and fluid replacement may be sufficient.

4-Antibiotics is useful in reducing the frequency of bowel movement and duration of illness in moderate to severe diarrhea only (3 day course of quinolone taken twice daily). Azithromycin may be a better choice in a quinolone resistant cases.

Prevention:

Involves good hygien, clean water and avoiding uncooked vegetables. Doxycyclin 100mg or trimetheprim 80gm or quinolone single daily dose is effective for susceptible individuals.

Malaria

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Malaria:

Malaria is a protozoan disease transmitted by the bite of infected anopheles mosquitoes. Human malaria is caused by plasmodium falciparum, P. vivax, P. ovale, P.malariae. malaria is a predominantly a disease of hot climates, however it may occur in other parts of the world such as Europe. Less frequent modes of transmission include blood transmission or injection and transplacental infection.

Pathogenesis and life cycle:

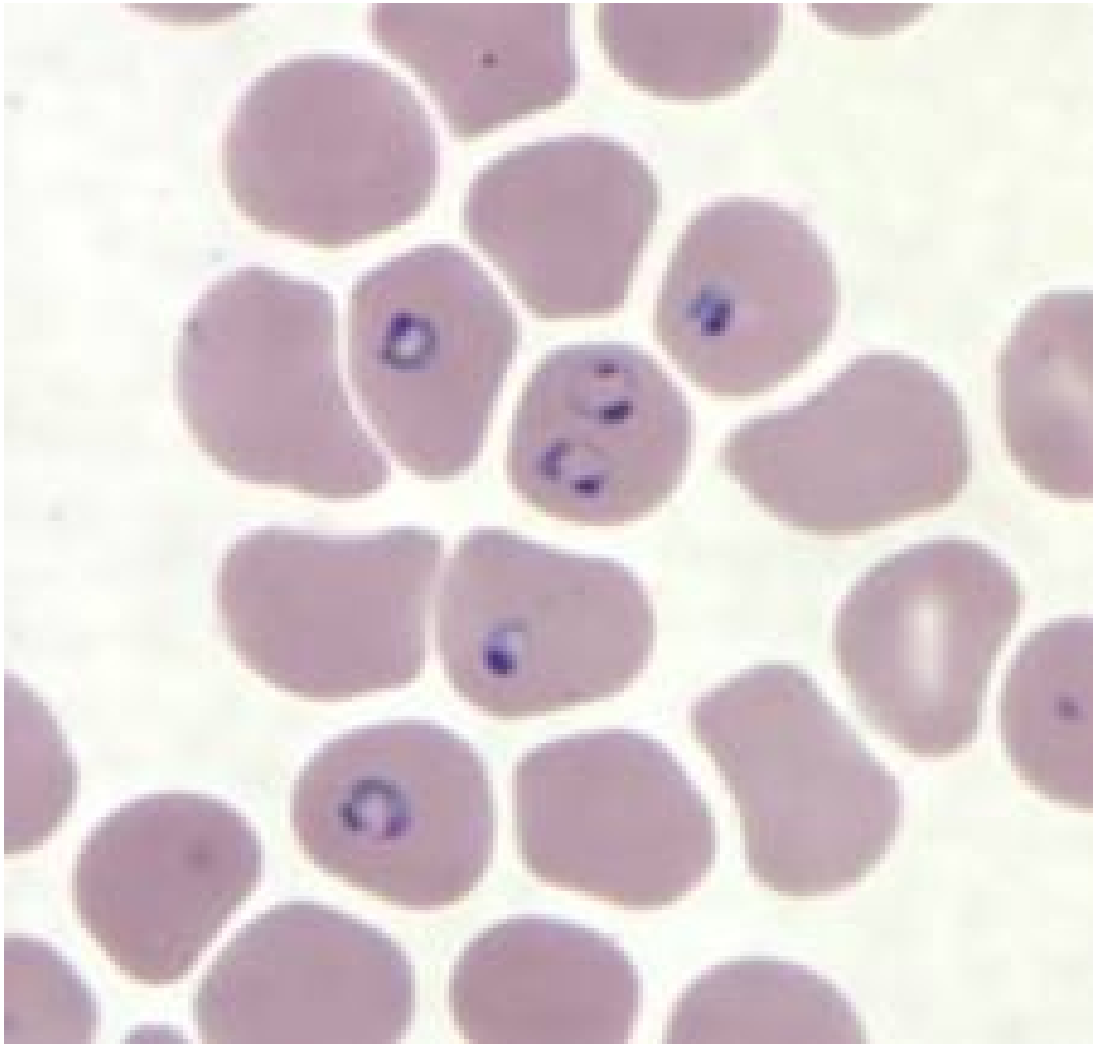
Almost all deaths are caused by falciparum malaria. Human infection begins when a female anopheline mosquito inoculates plasmodial sporozoites from its salivary gland during a blood meal which carried rapidly via the blood to the liver, invading hepatic cells and begin a period of asexual reproduction. This process producing pre-erythrocytic (schizogony) in which a single sporozoite eventually produce 10000-30000 daughter merozoites.

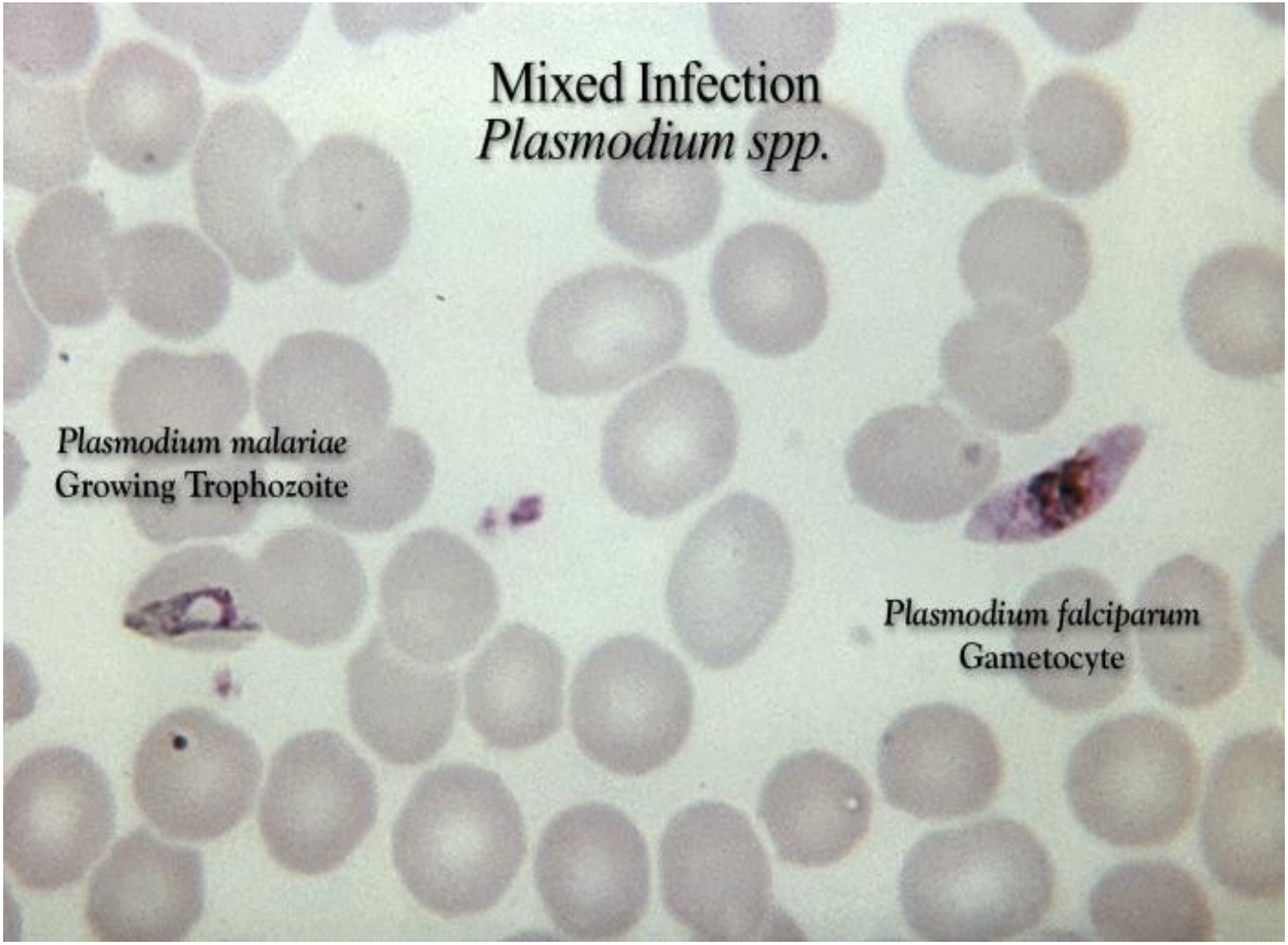
At this stage symptomatic infection begins. In P-vivax and P-ovale infections, a proportion of the intrahepatic forms do not divide immediately but remain dormant for months to years before reproduction begins. These dormant forms (hypnozoites) are the cause of the replaces that characterize infection with these 2-species. Merozoites invade erythrocytes and become trophozoites. Attachment is mediated via a specific erythrocyte surface receptor

In the case of P-vivax this receptor is related to the duffy blood group antigen. Most west Africans and people that carry the duffy negative phenotype are therefore resistant to P-vivax malaria, this stage of development appear as a small ring forms intraerythrocytic under .light microscopy

By the end of the 48 hours intraerythrocytic life cycle (72 hours for P-malaria) the parasite grown to occupy most of the red cell. Multiple nuclear divisions take place (merogony), and the red cells rupture to release 6-30 daughter merozoites, each capable of invading a new red cell and repeating the cycle. Some of the parasite develop into a morphologically distinct long lived sexual forms (gametocytes) that .can transmit malaria

:Effects on red blood cells




Mixed Infection
Plasmodium spp.
Plasmodium malariae
Growing Trophozoite
Plasmodium falciparum
Gametocyte

Malaria is always accompanied by haemolysis and in a severe or prolonged attack anaemia may be profound. The causes of anaemia in malaria may be due to:

1- Haemolysis of infected RBC.

2- Haemolysis of uninfected RBC.

3- Dyserythropoiesis.

4- Splenomegally causing erythrocyte sequestration.

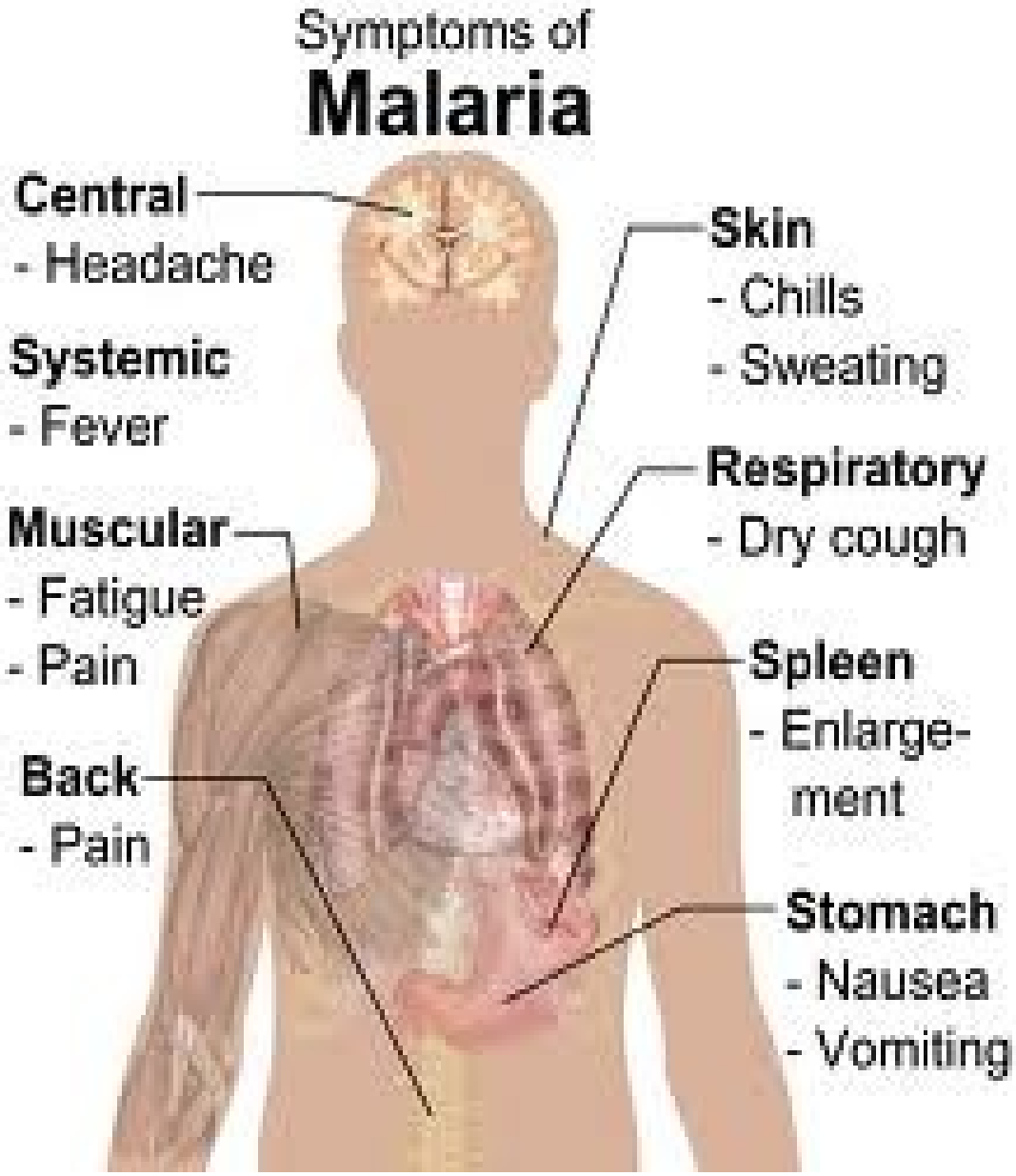
5-Depletion of folate stores.

Haemolysis is most severe with P-falciparum, which invades red cells of all ages but especially young cells. P-vivax and ovale invade reticulocyts and P-malaria invade normoblast, so that infections remain lighter. Sequestration of red cells containing mature parasite in P-falciparum occur which interfere with microcirculation especially in the vital organs which defence the parasite and as a consequence only the younger ring forms of the parasites are seen in .the peripheral blood in falciparum malaria

In the other three (benign) malarias, sequestration doesn't occur and all stages of the parasites development are evident on peripheral blood smears.

P-falciparum doesn't grow well in red cells that contains HbF, C or especially S. Hbs hetrozygotes are protected against the lethal complications of malaria which might be related to the low oxygen tensions.

:Clinical features



1- The first symptoms of malaria are nonspecific:

The lack of a sense of well being, headache, fatigue, abdominal discomfort and muscle ache followed by fever.

2- Headache may be severe in malaria however there is no neck stiffness or photophobia as that seen in meningitis.

3-Nausea, vomiting and orthostatic hypotension are common.

The classical malaria paroxysms in which fever occur at regular- 4 intervals suggest infection with P-vivax or P-ovale. Fever starts with a rigor ,the patients feels cold and the temperature rises to about 40C° after half an hour to an hour the hot or flush phase begin. It last several hours and gives way to profuse perspiration and gradual fall in temperature. The cycle is repeated 48 hours later. Gradually the spleen and liver enlarge and may become tender. Anaemia develops slowly. Replaces are common two years after leaving the endemic .area

5- P-malaria infection: fever occur in bouts every third day. (parasitaemia may persist for many years with or without symptoms). P-malaria causes GN and nephritic syndrome in children.

6- P-falciparum infection: more dangerous than other forms of malaria. The onset is often insidious with malaise, headache and cough. Cough and mild diarrhea are common.

Fever has no particular pattern, jaundice is common due to haemolysis and hepatic dysfunction. The liver and spleen enlarged and become tender. Anaemia develops rapidly. Splenoctomy increases the risk of severe malaria.

Complications of malaria due to P-falciparum

1- Sever anaemia.

2- Organ damage due to anoxia.

- Brain (confusion, coma).
- Kidney's (oligurea, uraemia due to acute tabular necrosis).
- Lungs (cough, pulmonary oedema).
- Intestine (diarrhoea, congestion).
- Liver (jaundice, encephalopathy which is rare).

Intravascular haemolysis (black water fever): this is associated- 3 with chronic falciparum malaria, most commonly in those who take antimalarial treatment irregularly or are deficient in G6PD. Haemolysis is unpredictable and severe, destroying uninfected as .well as infected red cells. The urine is dark or black

4- Hypoglycemia especially with quinine treatment.

5- Shock secondary to septicaemia.

6- Hypotensive shock.

7- Metabolic acidosis.

8- Splenic rupture.

9- Complication during pregnancy (maternal death, abortion, still birth, low birth weight).

Tropical splenomegally syndrome (hyperreactive malarial splenomegally):

In some hyperendemic areas gross splenomegaly is associated with an exaggerated immune response to malaria and is seen more common in adult. The condition is more common in female and in certain ratial and family groups and is characterized by over production of IgM. IgM aggregates are phagocytosed by reticuloendothelial cells in the spleen and liver. Light microscopy of the liver usually shows sinusoidal lymphocytosis. Anaemia and lymphocytosis can be confused with leukaemia. Portal hypertension .may develop

Investigations:

Malaria should be considered if febrile patients is in or has recently left a malarias locality.

1- Thick and thin blood films should be repeated if necessary. In P-falciparum only ring forms are normally seen in the early stages. (Gametocytes may persist despite treatment and are harmless).

2- Immunochromatographic (Dip sticks test for P-falciparum antigen are non microscopic means of diagnosis) they should be used in parallel with blood film examination.

D.DX:

Other causes of fever with splenomegally in the tropics should be considered such as T.B, visceral leishmaniasis, schistosomiasis, chronic brucellosis in addition to leukaemia and lymphoma.

Management:

1- For all infections with P-vivax, P-ovale and P-malaria the drug of choice is chloroquin (600 mg base followed by 300mg base in six hours then 150 mg base twice daily for 2 more days). P-falciparum is now resistant to chloroquin and so not used to treat this type of malaria except in an area of chloroquin sensitivity.

2- Quinine dihydrochloride or sulphate 600 mg eight hourly until the blood is free of parasite (3-5 days) then followed by a single dose of sulfadoxine with pyrimethamine (3 tablets of fansidar). In pregnancy a seven day course of quinine should be given.

3- Alternative to quinine + fansidar are mefloquin.

4- In complicated P-falciparum quinine is given as an I.V infusion over 4 hours (10mg/kg up to 700 mg repeated 8-12 hourly until the patient can take drug orally). Intramuscular quinine may cause necrosis of muscle.

5- In comatose patient L.P may be indicated to exclude co-existing bacterial meningitis.

6-Severe anaemia requires transfusion, careful attention to fluid balance is essential. Oligurea can be treated by frusemide or infusion of manitol. Exchange transfusion is life saving in complicated very heavy infection (> 10% of red cell infected). Hypoglycaemia and septicaemia should be treated. Dialysis may be needed if renal failure is developed.

Prevention:

Clinical attacks of malaria may be preventable by drugs such as proguanil which attack the preerythrocytic form or by drugs such as chloroquin or mefloquine after it has inter the erythrocyte. Chemotherapy is begun one week before entering the malarias area and is continued until 4-weeks after leaving it. Chloroquine over 5 years may cause irreversible retinopathy. (proguanil and chloroquine is safe during pregnancy). Mefloquine is contraindicated in the first trimester of pregnancy. Fansidar should not use for chemoprophylaxis as it may cause death from agranulocytosis or Stevens Johnson's syndrome. Mefloquine is useful in areas of multiple drugs resistant.