Lecture 37

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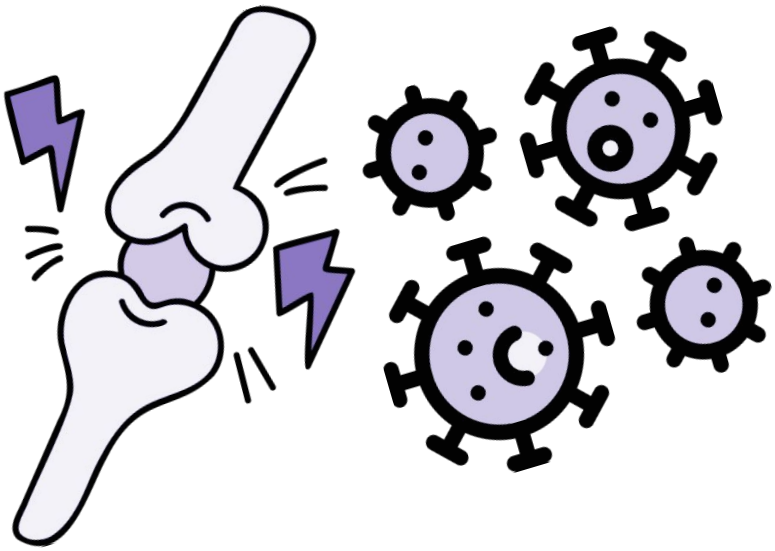

Reviewed By

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Malaria & Travel medicine

Objectives:

By the end of the lecture the student should be able to:

- ★ Learn the epidemiology and etiology of malaria
- ★ Know the clinical presentation
- ★ Know Risks to travelers
- ★ Know Malaria and pregnancy
- ★ Know Diagnostic work up
- ★ Learn the treatment and prophylaxis

Color index:

Original text Females slides Males slides
Doctor's notes Textbook Important Golden notes Extra

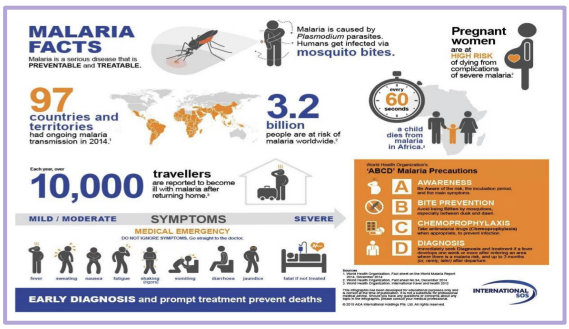
Malaria

Definition

Malaria is a protozoal infection caused by a few plasmodium species that spread by mosquito, once it enters the bloodstream it infects and destroys mainly liver cells and RBCs causes a variety of symptoms.

World facts

- **97** countries have malaria.
- **216 million** malaria cases worldwide in 2018.
- **445,000** malaria deaths worldwide in 2018.
- **10,000** world travelers become infected every year.

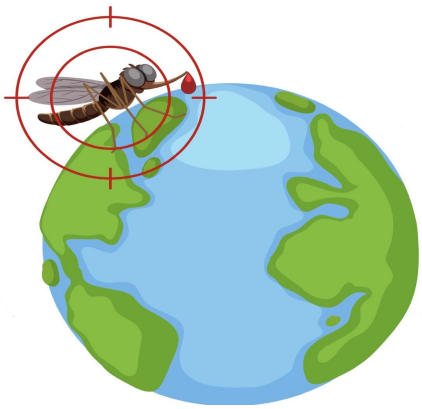


Goals, milestones and targets for the *Global technical strategy for malaria 2016–2030*

GOALS	MILESTONES		TARGETS
	2020	2025	2030
1. Reduce malaria mortality rates globally compared with 2015	At least 40% 18% reduction achieved 22% off track	At least 75%	At least 90%
2. Reduce malaria case incidence globally compared with 2015	At least 40% 3% reduction achieved 37% off track	At least 75%	At least 90%
3. Eliminate malaria from countries in which malaria was transmitted in 2015	At least 10 countries On track	At least 20 countries	At least 35 countries
4. Prevent re-establishment of malaria in all countries that are malaria-free	Re-establishment prevented On track	Re-establishment prevented	Re-establishment prevented

Global technical strategy for malaria 2016-2030.

Malaria in Travelers



- Non-immune travelers exposed to mosquito bites between dusk and dawn.
- This includes previously **semi-immune** travelers who have lost or partially lost their immunity during **stays of 6 months** or more in countries or **areas of no risk**.
- Travelers who have migrated to countries and areas of no risk are at particular risk when they travel to malarious areas to **visit friends and relatives.** Those travelers used to live in malaria endemic areas and got malaria during their childhood and think they have immunity against malaria and don't take proper precautions but there immunity is gone and they often get severe malaria when they get infected again.
- Most cases of falciparum malaria in travelers occur because of poor adherence or inappropriate prophylactic malaria drug regimens and failure to take adequate precautions against mosquito bites.
- Adherence to chemoprophylaxis can be improved if travelers are informed of the risk of infection and believe in the benefit of prevention strategies.
- **Late-onset vivax and ovale** malaria may occur despite effective prophylaxis.

Epidemiology

Stable transmission:
including much of sub- **Saharan Africa**, transmission occurs consistently year round. The bulk of the mortality is seen in **children**, while those who survive to adulthood acquire significant immunity; **low- grade parasitaemia** is still present but causes few symptoms.

Unstable transmission:
occurs when there is erratic, **seasonal or low- level transmission** (e.g. in the **Sahel belt**, where mosquitoes feed only in the rainy season). Little protective immunity develops and symptomatic malaria occurs at **all ages**. Changes in environmental or social conditions in such areas can lead to epidemics with substantial mortality in all age groups.

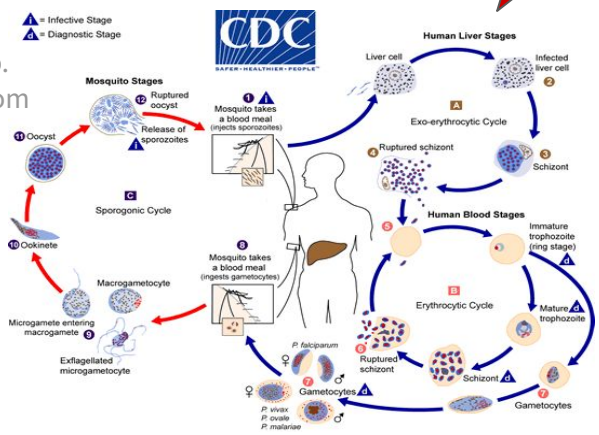
Malaria

◄ Incubation period & Life cycle

- Sporozoites reach the liver within 1-2 hours following Female Anopheles mosquito bite.
- Pt. asymptomatic for 12-35 days until RBCs stage of parasite life cycle.

Huge thanks to Microbiology teamwork!

Explanation for the picture:



1. Malaria is mainly carried by **female anopheles mosquito**.
2. The infected mosquito will bite and inject **sporozoites** from it salivary gland into the bloodstream of human.
3. Which then will travel through blood until it reaches the liver and enter the hepatocytes where it will multiply asexually to form **merozoites inside the schizont** (Exoerythrocytic schizont).
4. When the hepatic schizont rupture the merozoites will be released into blood, then it will enter the erythrocytes forming **immature trophozoites** (ring stage) which will have **2** pathways:

a. First pathway:

- It goes through the erythrocytic cycle starting from ring stage then into **Mature trophozoites**, then the merozoites will multiply inside the RBCs forming schizont (Erythrocytic schizont), which will rupture (hemolysis) and release the merozoites into the bloodstream (**Clinical attack** of malaria is due to this stage) and the cycle will repeat over and over again.

b. Second pathway:

- Some immature trophozoites will become **gametocytes** (male and female) those gametocytes will be ingested by another mosquito; in the mosquito:
  - ➔ There are Micro(Male) and Macro(Female) gametocytes, the microgametocytes will enter into the macrogametocytes in which they will form Ookinete then it will develop into Oocyst which will rupture releasing sporozoites in mosquito, then the cycle will go over and over again.

Pathogenesis

- The pathology of malaria is related to **anaemia**, **cytokine release** and, in the case of P. falciparum, widespread organ damage due to **impaired microcirculation**. The anaemia seen in malaria is multifactorial . In P. falciparum malaria, red cells containing schizonts **adhere to the lining of capillaries in the brain, kidneys, gut, liver** and other organs. As well as causing **mechanical obstruction**, these schizonts rupture, releasing toxins and stimulating further cytokine release.

- **Causes of anemia :** Haemolysis of infected red cells ,Haemolysis of non-infected red cells (blackwater fever) , Dyserythropoiesis , Splenomegaly and sequestration ,Folate depletion.

Malaria

◄ Modes of Transmission

1. Human to Human by **anopheles mosquito** (between dusk and dawn)
2. Blood transfusion
3. Contaminated needles
4. Congenital

◄ Predisposing factors:

- **non- immune individuals:**
  - children in any area
  - adults in areas of unstable transmission
  - visitors from a non- malarious region.
- People living **near airports** in Europe have acquired malaria from accidentally imported mosquitoes.

◄ Protective factors:

- **Certain genetic traits:**
  - P. falciparum : **sickle cell, thalassaemia, G6PD deficiency** and HLA-B53, does not grow well in red cells that contain haemoglobin F, C or especially S. Haemoglobin S heterozygotes (AS) are protected against the lethal complications of malaria.
  - P. vivax: People who **lack the Duffy antigen**[1] (a common finding in West Africa).

01 ***Plasmodium Falciparum (Pf)*:** The most serious and common

02 ***P. Vivax (Pv)***

03 ***P. Ovale (Po)***

04 ***P. Malariae (Pm)***

05 Zoonotic malaria: ***P. Knowlesi (Pk), P. Simium (Ps)***

- P. vivax and P. ovale may persist in liver cells as dormant forms, hypnozoites, capable of developing into merozoites months or years later. Thus the first attack of clinical malaria may occur long after the patient has left the endemic area, and the disease may relapse after treatment if drugs that kill only the erythrocytic stage of the parasite are given.
- P. falciparum and P. malariae have no persistent exo-erythrocytic phase but recrudescence

1. The Duffy blood group antigen serves not only as blood group antigen, but also as a receptor for a family of proinflammatory cytokines termed chemokines, and as a **receptor for Plasmodium vivax malaria parasites.**

Malaria

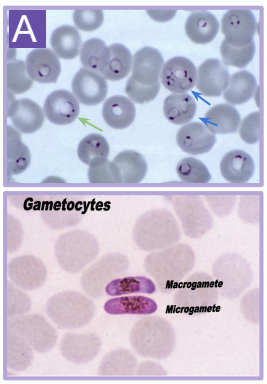
1 Plasmodium Falciparum[1]

❖ **General Information:**

- Most **highly pathogenic** species.
- **Resistant** to many antimalarial drugs.
- ★ The species that causes the **most morbidity and mortality** worldwide.
- Represents the major cause of malaria in **tropical** countries.
- Responsible for the sporadic great regional **pandemics** that sometimes occur in **subtropics**.

❖ **Pathogenesis:**

01. The pre-erythrocytic cycle starts immediately after injection of the **sporozoites** by the mosquito.
02. Infects **mature and young erythrocytes**. The surface of erythrocytes infected with late stage **trophozoites** or **schizonts** is altered so they stick to endothelial cells in various tissues (cytoadherence) causing multi-organ failure.
03. **Schizogony** is particularly prolific in all stages (pre-erythrocytic, erythrocytic and sporogony) that may be the cause of its success as a species and its virulence.



❖ **Characteristics**

- Infection in the peripheral blood is characterized by the presence of **ring forms** and **gametocytes**, whereas late trophozoites and schizonts are only seen exceptionally.
- The level of **parasitaemia may be high** and **multiple infection** in a single erythrocyte is common.
- The gametocytes are characteristically **crescent-shaped** and unlike the **gametocytes** of other species, are very slow to reach maturity (up to 10 days) and early forms of gametocytes are sequestered.
- **A:** rings in peripheral blood thin film of P.Falc showing double dotted ring and multiple parasites invading one RBC. These findings are **characteristic for P.Falciparum** .

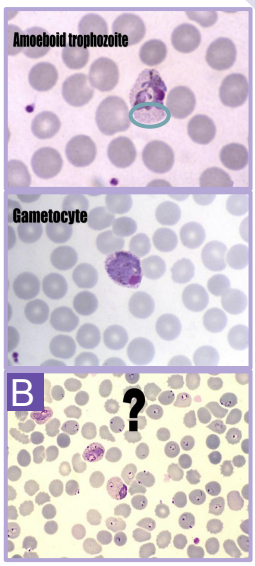
2 Plasmodium Vivax

❖ **General Information:**

- Occurs throughout most of the temperate zones as well as large areas of the tropics (but is mainly absent from tropical West Africa).
- It causes 'benign' **tertian malaria.**
- Polymorphic and the subspecies status proposed for some strains may be justified.

❖ **Characteristics :**

- Restriction of erythrocyte invasion to **reticulocytes bearing Duffy blood group determinants** (explains why RBCs infected with trophozoites of P. vivax are sometimes described as **larger** than normal).
- The presence of caveolar structures on the surface of the infected erythrocyte membrane take up stain, and are described as **Schüffner's dots**.
- After invading the hepatocyte some of the sporozoites may transform into **hypnozoites**, then remain latent for months or years and be responsible for **subsequent relapses**.



- **B:** Co-infection with p.falc and p.vivax.

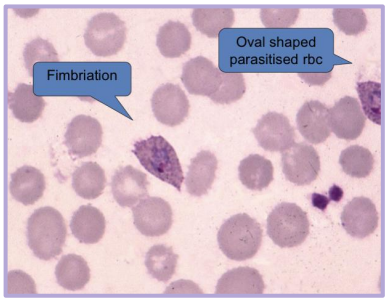
1. The fever has no particular pattern. **Jaundice is common** due to haemolysis and hepatic dysfunction. The liver and spleen enlarge and may become tender. **Anaemia develops rapidly, as does thrombocytopenia**.

Malaria

3 Plasmodium Ovale

❖ **General Information:**

- Distinct from P. vivax in minor morphological differences, antigenic and molecular differences. Most of the biological and **clinical features are identical**.
- The major biological difference is that P. Ovale **can infect Duffy negative reticulocytes**, whereas P. Vivax cannot.



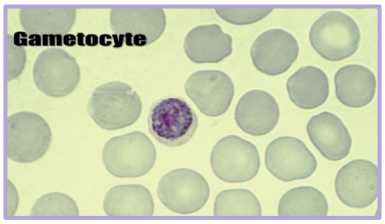
4 Plasmodium Malariae[1]

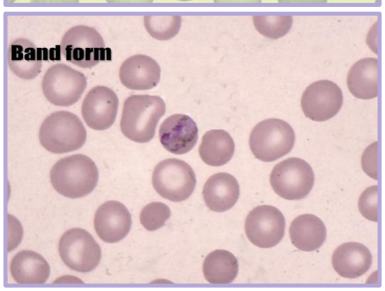
❖ **General Information:**

- Differs from the other three human malaria parasites in its **slow development** and its longer asexual cycle.
- Development is slow in both the vector and the human host because of less efficient schizogony.
- The asexual cycle is **72** hours instead of 48 hours – hence '**quartan malaria**' – because fever paroxysms occur every 4th day (according to the Roman custom of regarding day 0 as day 1).

❖ **Characteristics:**

- Infects **old erythrocytes**, explaining why infected cells are often described as '**smaller**' by microscopic.
- The presence of 'knobs' at the surface of infected erythrocytes, which are similar to P. Falciparum, but the cells **do not** exhibit any cytoadherence (and so no sequestration).
- The surface of infected erythrocytes **does not** exhibit any caveolar/vesicle complexes and **Schüffner's dots are absent**.
- **Sporozoites** of P. Malariae **do not transform into hypnozoites**, and so there are **no relapses**.
- P. malariae can survive for a very long time in the peripheral blood (**10 years or more**) at a very low level of parasitaemia occasionally producing detectable **peaks** with a recrudescence of clinical symptoms.





5 Plasmodium Knowlesi & Simium

❖ **General Information:**

- Simian Monkey to Human transmission (zoonotic malaria)
- **P. Knowlesi:** in South East Asia, it looks like P. Malariae.
- **P. Simium:** in South America, it looks like P. Vivax.
- **Need PCR** for diagnosis.
- Can cause severe disease

1. In children, P. malariae infection is associated with glomerulonephritis and nephrotic syndrome.

Malaria

◀ Malaria Paroxysm (attack) 437 dr slides

- **3** successive stages:

1st. Rigors, Headache associated with pale **COLD** skin (1-2 Hr).

2nd. Delirium, Tachypnoea, **HOT** Skin Fever (Several hours).

3rd. Fever (**SWEATING** & fatigue).

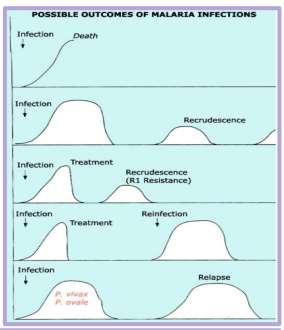
Notes Regarding Malaria Paroxysm:

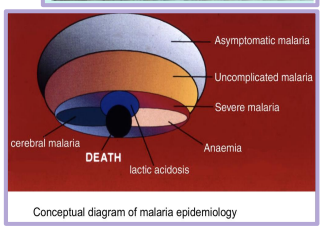
- Paroxysms associated with **synchrony of merozoite release**.
- **Between paroxysms temperature is normal and patient feels well and Asymptomatic**.
- Falciparum may not exhibit classic paroxysms. (continuous fever)
- Classically the attacks (Periodicity):
  - ➔ Occur every **48** hours with (P. vivax, and P. ovale) "tertian parasites".
  - ➔ Occur every **72** hours with (P.malariae) "quartan parasite".
  - ➔ Whereas (P.falciparum) show IRREGULAR attacks , or hectic (especially in non-immune).

◀ History

- Travel to malaria endemic area
  - ➔ **P. Falciparum**: up to 1 year from travel (usually first 3 months).
  - ➔ **P. Malariae**: up to 10 years.
  - ➔ The main symptom that is usually present is fever which is non specific.

- **Non specific: Fever**
- **Sweats, chills**
- **Myalgia**
- **Headache**
- **Diarrhea**
- **Cough**
- **Jaundice, dark urine**
- **Confusion, Seizures**





Conceptual diagram of malaria epidemiology

◀ Signs

01 **Fever, HR tachycardia, low BP, low O2**

02 **Level of consciousness**

03 **Evidence of seizures**

04 **Splenomegaly the most common clinical sign**

05 **Jaundice**

06 **Anemia**

Does this patient have malaria?	
Acquired during travel:	**Living in endemic region:**
• Fever +LR 5.1 • Splenomegaly +LR 6.5 • Hyperbilirubinemia +LR 7.3 • Thrombocytopenia +LR 5.6	• Splenomegaly +LR 3.3 • Hepatomegaly +LR 2.4

Malaria

❖ **Malaria clinical spectrum:**

- Most fever or flu-like illness
- Fatalities with (Pf) (occasionally with splenic rupture).
- **Cerebral malaria** (Pf) - may be focal or generalized symptoms.
  - ★ RISK FACTORS FOR POOR PROGNOSIS IN CEREBRAL MALARIA: High bilirubin, High creatinine, Hight lactase.
- **Hypoglycemia** - from disease and treatment (quinine leads to insulin release).
- **Risk Acute Respiratory Distress Syndrome** (Pf) due to capillary leak - need to limit fluids.
- **Acute renal failure** (Pf) - common complication of severe malaria.

WHO Criteria – Severe Malaria

History of recent possible exposure and no other recognized pathology.

EITHER

Asexual forms of (Pf) on blood smear.

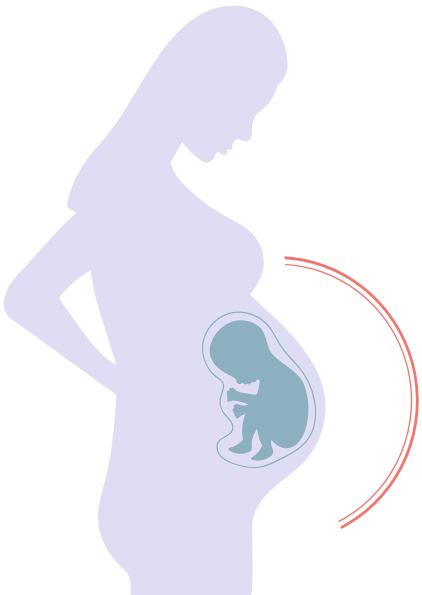
AND

Any one or more of the following 11 features:

01 Impaired consciousness or coma.
02 Severe normocytic anemia.
03 Renal failure.
04 Pulmonary edema or adult respiratory distress syndrome (ARDS).
05 Hypoglycemia.
06 Circulatory collapse, shock
07 Spontaneous bleeding/disseminated intravascular coagulation.
08 Repeated generalized convulsions.
09 Acidemia/acidosis.
10 Hemoglobinuria.
11 Parasitemia of > 5% (> 250 000/microlitre) in non-immune individuals.

◀ Pregnancy & Malaria

The extra is from 437 dr slides.



01 | Mortality, Abortion, Stillbirth.

02 | Anemia, hypoglycemia, pulmonary edema: more common.

03 | Premature delivery high infant mortality.

04 | Low birth weight ,Placental insufficiency.

05 | High parasitemia? placenta favorable site for P. Falciparum.

06 | Primigravidae , more susceptible to placental malaria

◀ Diagnosis

- Detailed targeted history including **travel history** and clinical examination together with High Index of Suspicion (HIS).

Thin and Thick films[1]	**Urea and Creatinine**	**ABG**
CBC, Coagulation profile	**LFT, Bilirubin**	**CXR**
Random Blood glucose	**Lactic acid**	**Urine analysis**

01. Blood film

Extra from 437 dr slides

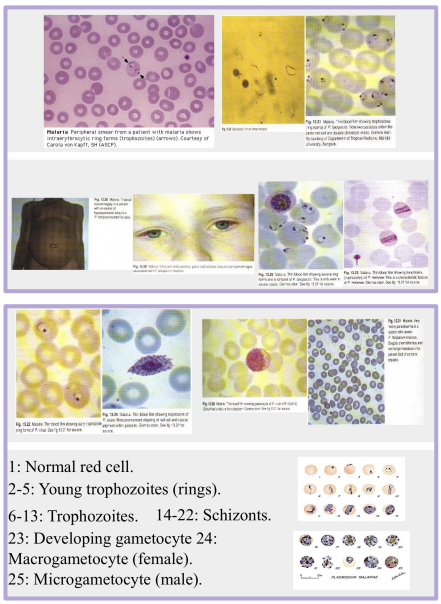
- **Giemsa stain** or **wright's stain**.
- Correct identification of malarial Species is essential for treatment because of P. Falciparum is resistant to Chloroquine & others.

➔ **On Giemsa stain:**
Cytoplasm: light blue, Nucleus: dark blue.

➔ **In P. Falciparum:**

1. **Only ring stage asexual parasite and gametocytes seen in Peripheral Blood.**
2. While **RBC with Trophozoites or Schizonts** stage **sequestered** in peripheral microvasculature, and **NOT circulating Peripheral Blood.**

➔ **In P. Vivax, Ovale & malariae:**
All asexual erythrocytic stages circulate in peripheral blood, thus seen on Blood Smear.



❖ **Acutely ill patients** | DDX: P.F. vs P. Vivax, because:

A. P. Ovale – Vivax – clinical, morphological.
B. P. malariae - ch. Infection

Differential diagnosis of malaria in acutely ill patients based on peripheral blood smear:		
Findings	**P. Falciparum**	**P. Vivax & P. Ovale**
Multiple infected RBCs	Common	Rare
Mature (trophozoite & schizont) parasites	Absent	Common
RBC enlargement with later parasite stages	Absent	Common

➔ Mature (trophozoites & schizont) stage **P. falciparum** typically sequestered in the peripheral microvasculature.

➔ RBC enlargement in **P. vivax** typically occurs with later stage parasites that do NOT circulate in **P. falciparum** infection.

1. In the **thick** film, erythrocytes are lysed, releasing **all blood stages of the parasite**. This, as well as the fact that more blood is used in thick films, **facilitates the diagnosis of low-level parasitaemia**.
A **thin** film is essential to **confirm** the diagnosis, to **identify the species** of parasite and, in P. falciparum infections, to quantify the **parasite load** (by counting the percentage of infected erythrocytes).

Malaria

02. Serology

Extra from 437 dr slides

- **NOT** useful in managing acutely ill patient.

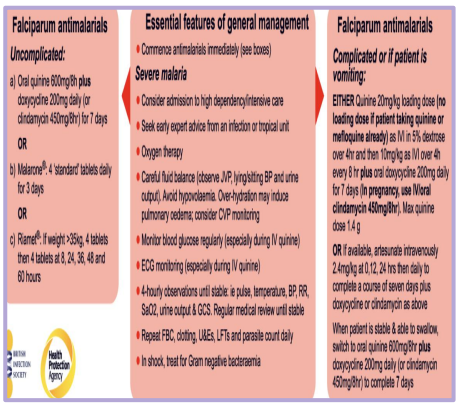
03. DNA probe (PCR)[1]

- Limited utility for the diagnosis of acutely ill patients in the standard healthcare setting
- Most useful for confirming the species of malarial parasite after the diagnosis has been established by either smear microscopy or RDT.

Management

❖ **Basic Principles of Malaria Management:**

- Think of the diagnosis and do **thick and thin blood films**.
- Ask lab about **species** of malaria – falciparum versus non-falciparum (vivax, ovale, malariae).
- Ask about percent parasitemia
  – If told **greater than 1% parasitemia** – think PF!.
- Verify no evidence of **severe or complicated malaria** (no need for parenteral therapy).
- Medical Emergency.
- Consider admission to hospital (especially for falciparum) at least observe tolerance of meds in ER.
- FOLLOW UP.
- **Drug of choice:** Artesunate based therapy.



- **Oral therapy:**
  - Artemether-Lumefantrine[2]
  - Atovaquone / Proguanil (Malarone)
  - Quinine plus Doxycycline[3] / Clindamycin

  * Avoid Mefloquine for treatment.

- **Parenteral therapy**
  - Artesunate
  - Quinine (or quinidine): needs telemetry.

Box 20.57 Drug treatment of uncomplicated malaria

Parasite	Drugs	Regimen	Plus	Regimen
Plasmodium vivax *P. ovale* *P. malariae*	Chloroquine	600 mg[a] 300 mg[b] 6 h later 300 mg 24 h later 300 mg 24 h later	Primaquine	0.25–0.5 mg/kg per day for 2–3 weeks
	or (if known resistance to chloroquine, or dual infection with P. falciparum*)*			
	ACT (not artesunate + SP)	3 days	Primaquine	0.25–0.5 mg/kg per day for 2–3 weeks
P. falciparum (adults, endemic zone) ★	ACT	3 days	Primaquine	0.75 mg/kg single dose
	or (if not available)			
	Quinine + doxycycline	7 days	Primaquine	0.75 mg/kg single dose
P. falciparum (pregnant women) ★	All trimesters: Artesunate			
P. falciparum (infants)	ACT	3 days; appropriate dose for body weight	Primaquine	0.75 mg/kg single dose
P. falciparum (returning travellers)	Atovaquone–proguanil *or* quinine + doxycycline	7 days		

[a]10 mg/kg in children. [b]5 mg/kg in children. [c]Use ACT only if quinine is not available. ACT, artemisinin-based combination therapy; SP, sulfadoxine–pyrimethamine.

Box 20.59 Drug treatment of severe *falciparum* malaria in adults and children

Drug/route	Immediate dose	Subsequent doses
Severe malaria is an emergency: after rapid assessment and confirmation of diagnosis, if possible, treatment should be started with whatever parenteral treatment is available. The options, in order of preference, are:		
1. Intravenous artesunate	2.4 mg/kg	2.4 mg/kg at 12 and 24 h, then daily (up to 7 days)
2. Intravenous quinine	20 mg/kg[a]	10 mg/kg 8-hourly (up to 7 days)
3. Intramuscular artesunate	2.4 mg/kg	2.4 mg/kg at 12 and 24 h, then daily (up to 7 days)
4. Intramuscular artemether	3.2 mg/kg	1.6 mg/kg daily
5. Rectal artesunate	10 mg/kg	Transfer to centre where parenteral therapy available
Continue parenteral treatment for at least 24 h, regardless of improvement in condition. After this, if the patient is improving, switch to oral therapy to complete 7 days with:		
ACT		
Or	*Plus*	Primaquine 0.75 mg/kg single dose
Quinine + doxycycline		

[a]10 mg/kg if patient has already received oral quinine or mefloquine.
ACT, artemisinin-based combination therapy.

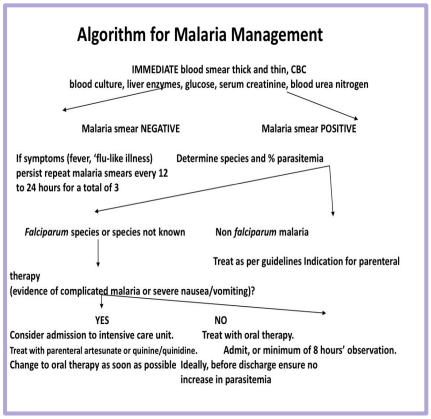
1. DNA detection (PCR) is used mainly in research and is useful for determining whether a patient has a recrudescence of the same malaria parasite or a re-infection with a new parasite.
2. ★ P. falciparum is now resistant to chloroquine and sulfadoxine-pyrimethamine (Fansidar) almost world-wide, so an artemisinin-based treatment is recommended.
3. We usually **avoid doxy in pregnancy,** we don't use it in malaria as often as we used to. **Artesunate is the main therapy**

Malaria

◀ Management cont.

❖ **Non-P. Falciparum:**

- **Chloroquine** (base) 600mg followed by 300mg at 6, 24 and 48 hours.
- **primaquine** (30mg base/day for vivax, 15 mg/day for ovale) for 14 days in **vivax and ovale** after treatment of acute infection use to **eradicate liver parasites**; **G6PD must be measured** before primaquine is given – seek expert advice if low.



13.59 Severe manifestations/complications of *falciparum* malaria and their immediate management

Coma (cerebral malaria)
- Maintain airway
- Nurse on side
- Exclude other treatable causes of coma (e.g. hypoglycaemia, bacterial meningitis)
- Avoid harmful ancillary treatments such as corticosteroids, heparin and adrenaline (epinephrine)
- Intubate if necessary

Hyperpyrexia
- Tepid sponging, fanning, cooling blanket
- Antipyretic drug (paracetamol)

Convulsions
- Maintain airway
- Treat promptly with diazepam or paraldehyde injection

Hypoglycaemia
- Measure blood glucose
- Give 50% dextrose injection followed by 10% dextrose infusion (glucagon may be ineffective)

Severe anaemia (packed cell volume < 15%)
- Transfuse fresh whole blood or packed cells if pathogen screening of donor blood is available

Acute pulmonary oedema
- Nurse at 45°, give oxygen, venesect 250 mL of blood, give diuretic, stop intravenous fluids
- Intubate and add PEEP/CPAP (p. 193) in life-threatening hypoxaemia
- Haemofilter

Acute renal failure
- Exclude pre-renal causes
- Fluid resuscitation if appropriate
- Peritoneal dialysis (haemofiltration or haemodialysis if available)

Spontaneous bleeding and coagulopathy
- Transfuse screened fresh whole blood (cryoprecipitate/fresh frozen plasma and platelets if available)
- Vitamin K injection

Metabolic acidosis
- Exclude or treat hypoglycaemia, hypovolaemia and Gram-negative septicaemia
- Fluid resuscitation
- Give oxygen

Shock ('algid malaria')
- Suspect Gram-negative septicaemia
- Take blood cultures
- Give parenteral antimicrobials
- Correct haemodynamic disturbances

Aspiration pneumonia
- Give parenteral antimicrobial drugs
- Change position
- Physiotherapy
- Give oxygen

Hyperparasitaemia
- Consider exchange or partial exchange transfusion, manual or haemophoresis (e.g. > 10% of circulating erythrocytes parasitised in non-immune patient with severe disease)

Specific therapy
- Intravenous artesunate
- Mefloquine should be avoided due to increased risk of post-malaria neurological syndrome

From WHO. Severe falciparum malaria. In: Severe and complicated malaria. 3rd edn. Trans Roy Soc Trop Med Hyg 2000; 94 (suppl. 1): S1–41.

◀ Drug toxicity

- **Quinine**:
  - ➔ **Hypoglycemia**, **arrhythmias**, bitter taste , GIT upset , nausea, vomiting , tinnitus, **high tone deafness.**
- **Doxycycline**:
  - ➔ GI upset, vaginal candidiasis.
- **Mefloquine**[1]:
  - ➔ **Neuropsychiatric symptoms** (mood changes), encephalopathy… transient.
- **Artemether-Lumefantrine**:
  - ➔ H/A, anorexia, dizziness, arthralgia and myalgia.

1. Contraindicated in the first trimester of pregnancy, lactation, cardiac conduction disorders, epilepsy, psychiatric disorders.

Malaria

◀ Prevention

❖ **When travelling to malaria endemic areas:**

- Avoid mosquito bites .
- Wear long sleeved shirts and long trousers.
- Sleep in well–screened or air-conditioned rooms.
- Permethrin impregnated bed nets.
- Use bednetting of good quality with small mesh that is not damaged and impregnated with permethrin.
- Apply insect repellent containing no more than 30% DEET, or use 20% or greater Picaridin.
- At dusk, spray aerosolized insecticides (such as those containing pyrethrins) in living and sleeping areas.
- Chemoprophylaxis.

Chemoprophylaxis[1]:

❖ **Start 2 days pre-travel, continue 7 days after return:**

➔ Atovaquone/ Proguanil[2] (Malarone): 1 tab/d (250 mg atovaquone /100 mg proguanil)

❖ **One or 2 weeks pre-travel, continue 4 weeks after return:** less preferred

★ Mefloquine 250 mg once/wk.

➔ Doxycycline 100 mg daily.

➔ Primaquine 30 mg base daily.

➔ Chloroquine[3] sensitive areas: 500 mg (300 mg base) : once/wk.

1. Choice of regimen is determined by area to be visited, length of stay, level of malaria transmission, level of drug resistance, presence of underlying disease in the traveler concomitant medication taken.
2. **Pregnant** and lactating women may take **proguanil** or **chloroquine** safely. Avoid Malarone in pregnancy.
3. **Chloroquine** should not be taken continuously as a prophylactic for more than 5 years without regular ophthalmic examination, as it may cause **irreversible retinopathy**.

❖ **Malaria Vaccine:** skipped by the dr

- RTS,S/AS01 (Mosquirix).
- Engineered from T-cell epitope in the pre-erythrocytic circumsporozoite protein (CSP) of PF malaria parasite and the envelope protein of HBsAg with an adjuvant AS01.
- Efficacy of 25-50% in infants and young children.

Travel

◀ Fever in Returning Travelers

❖ The **most common** etiologies of fever in returning travelers are listed first:

01 Dengue (flavivirus):

- **4-7 day** incubation, widespread in tropics (increasing in Africa), vector borne (Aedes).
- **Diagnosis**: PCR, NS1 antigen; IgM only after day 4.

02 Malaria (Plasmodia sp.):

- **7-30 day** incubation for P. falciparum, widespread but very high risk in Africa, vectorborne. (Anopheles).
- **Diagnosis**: blood smear, PCR, malaria RDT (rapid diagnostic test).

03 Typhoid fever (Salmonella sp.):

- Incubation **6-30 days**, mostly South and Southeast Asia, fecal-oral transmission.
- **Diagnosis**: blood culture. Avoid Widal test can give a false -ve or false +ve.

04 Chikungunya virus:

- **4-7 day** incubation, widespread in tropics, vector borne (Aedes)
- **Diagnosis**: PCR; IgM only after day 4.

05 Zika virus:

- Incubation **3-14 days**, mostly Southeast Asia, little risk in Americas and Caribbean since 2016, vector borne (Aedes).
- **Diagnosis**: PCR of blood or urine, IgM (high false-positive rate), Viral Neutralization is definitive, no testing available in someone with previous flavivirus infection of any kind.

06 COVID-19 (SARS-CoV-2):

- Incubation most commonly **3-7 days**, present in almost all countries, can be acquired in-flight or after return.
- **Diagnosis**: PCR more sensitive than antigen, very sensitive during febrile phase.

07 Leptospirosis:

- Incubation **2-29 days**, South and South Asia, South America, transmitted from urine of infected rodents.
- **Diagnosis**: IgM only after day 5; PCR earlier.

08 Rickettsial disease:

- Scrub typhus:
  - Incubation **6-20 days**, Asia and northern Australia, chigger mites. Murine typhus worldwide.
  - **Diagnosis**: PCR (blood or eschar), IgM.
- Spotted fever group:
  - Incubation **2-14 days**, widespread but highest risk with R. Africae in southern Africa, mostly ticks.
  - **Diagnosis**: PCR (blood or eschar), IgM/IgG.

09 Rabies:

- Incubation **20-60 days** most common, widespread, animal or **bat bite**.
- **Diagnosis**: PCR of saliva or skin, serum antibody detection.

10 East African trypanosomiasis:

- Incubation **7-21 days**, eastern and southern African game parks, **tsetse fly**.
- **Diagnosis**: microscopy of blood films, lymph node aspirate, or chancre.

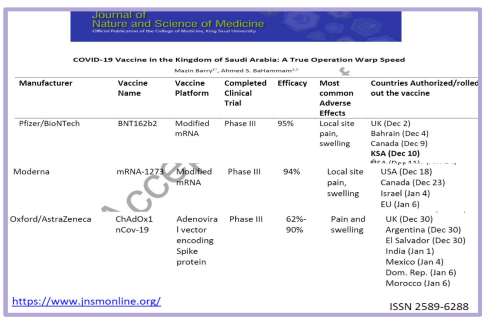
◀ Fever in Returning Travelers

- Other **frequent diseases** in travelers:
  - influenza A (seasonal).
  - Acute HIV
  - Mononucleosis.
  - Measles.
  - Varicella.
  - Tuberculosis.
- **Infrequent diseases** in travelers to consider:
  - Avian influenza (H5N1 & H7N9).
  - African hemorrhagic fevers (Ebola incubation is 8-12 days).
  - CCHF (Crimean-Congo haemorrhagic fever).
  - Yellow fever.
  - Japanese encephalitis.
  - Monkeypox (Nigeria and neighbors).
  - Relapsing fever.
  - Acute toxoplasmosis.
  - Arboviruses including Ross River.
  - Tickborne encephalitis.
  - West Nile virus (emerging in Europe).
  - Hantavirus (both hantavirus cardiopulmonary syndrome and hemorrhagic fever with renal syndrome).

◀ Vaccines

Required:

- Yellow Fever in Africa.
- Meningococcal, for all non saudies to enter SA.
- COVID-19 soon, for international travel.



Journal of Nature and Science of Medicine

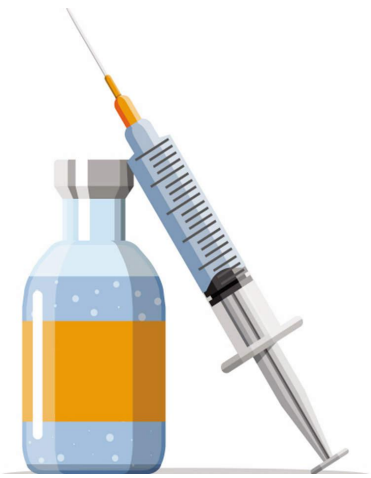
COVID-19 Vaccine in the Kingdom of Saudi Arabia: A True Operation Warp Speed

Manufacturer	Vaccine Name	Vaccine Platform	Completed Clinical Trial	Efficacy	Most common Adverse Effects	Countries Authorized/rollec out the vaccine
Pfizer/BioNTech	BNT162b2	Modified mRNA	Phase III	95%	Local site pain, swelling	UK (Dec 2) Bahrain (Dec 4) Canada (Dec 9) **KSA (Dec 10)**
Moderna	mRNA-1273	Modified mRNA	Phase III	94%	Local site pain, swelling	USA (Dec 18) Canada (Dec 23) Israel (Jan 4) EU (Jan 6)
Oxford/AstraZeneca	ChAdOx1 nCov-19	Adenoviral vector encoding Spike protein	Phase III	62%-90%	Pain and swelling	UK (Dec 30) Argentina (Dec 30) El Salvador (Dec 30) India (Jan 1) Mexico (Jan 4) Dom. Rep. (Jan 6) Morocco (Jan 6)

https://www.jnsmonline.org/ ISSN 2589-6288

Recommended :

- Polio
- Influenza
- Hepatitis A/B
- Measles
- Tetanus/Diphtheria/Pertussis
- Typhoid
- Rabies
- Japanese Encephalitis
- Tick-borne Encephalitis



* How Do You Know What Vaccines are Needed for Travel? The CDC's Health Information Website for Clinicians and Travelers (Published every 2 years, The "yellow book") Available online.

Yellow Fever

◀ Yellow fever[1]

- Mosquito-borne hemorrhagic fever caused by a flavivirus.
- **Rare fatalities in travelers** since vaccine introduction.
- ❖ **Epidemiology**:

~200,000 cases per year, 90% in Africa.

Indigenous case fatality rates vary 20-60% .

◀ Stages

- Has 3 stages:

Infection (3-4 days)[2]	Remission (48 hours)	Intoxication[3]
Fever, malaise, leukopenia.	Abatement of symptoms, 15% progress.	Return of symptoms, organ dysfunction, hemorrhage.

◀ Transmission

1. From primates or humans
2. Mosquito vector

◀ Disease Prevention

- Avoid mosquito bites: DEET, Clothing, Mosquito nets, Eliminate standing water.
- Vaccination

◀ Yellow Fever Vaccine

- Live-attenuated vaccine. Developed in 1936.
- Seroconversion >95%.
- Single 0.5ml subcutaneously.
- Revaccination at 10-year intervals required by World Health Organization.
  - Protection from one vaccine. However, may last 30 or more years.

Contraindications

- Age <9 months old
  - (Can consider at 6-9 months old during outbreaks).
- Pregnant women
  - (Yellow fever can cross placenta).
- Severe egg allergies.
- Severe immunocompromise.
- Immunomodulatory drugs.

1. Diagnosis of yellow fever can be confirmed by viral isolation from blood in the first 24 days of illness, the presence of IgM or a fourfold rise in IgG antibody titre. Leucopenia is characteristic.
2. Humans are infectious during the viraemic phase, which starts 3–6 days after the bite of the infected mosquito and lasts for 4–5 days.
3. In more severe disease, fever recrudescence is associated with lower back pain, abdominal pain and somnolence, prominent nausea and vomiting, bradycardia and jaundice. Liver damage and DIC lead to bleeding with petechiae, mucosal haemorrhages and gastrointestinal bleeding. Shock, hepatic failure, renal failure, seizures and coma may ensue.

Yellow Fever

◀ Yellow Fever Vaccine

Side Effects
Adverse Reactions (10-30%):
Local soreness, Mild fever, Headache, Myalgia.

Rare Severe Reactions

- **Anaphylaxis**:
  - ➔ Risk 1/131,000.

- **Yellow fever associated neurotropic disease (YEL-AND)**:
  - ➔ Risk 1:150,000 - 200,000.
  - ➔ Multiple neurologic conditions:
    - Encephalitis (esp. infants <9 months), Guillian-Barre, Bell's Palsy.
  - ➔ Onset 2-28 days after vaccination.
  - ➔ Rarely fatal.

- **Yellow fever associated viscerotropic disease (YEL-AVD)**:
  - ➔ Mimics severe yellow fever infection.
  - ➔ Major organ system failure occurs:
    - Hepatic, renal, circulatory failure.
    - 50% or greater fatality rate.
  - ➔ Occurs 1-8 days (average 3 days) after initial vaccination.
  - ➔ Risk 1:200,000 - 300,000 (Greater risk if over age 60).

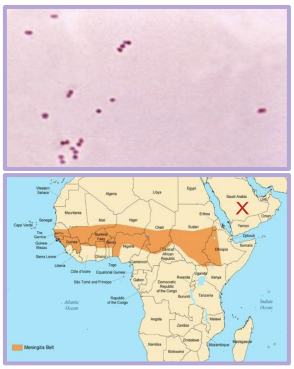
- Certification of vaccination required:
  - International Certificate of Vaccination or Prophylaxis for Yellow Fever form (ICVP).
  - Must be signed by licensed physician or designee.
- Waiver form for medical contraindication to vaccine (e.g. pregnancy).

- Angola
- Benin
- Bolivia (or signed affidavit at point of entry)
- Burkina Faso
- Burundi
- Cameroon
- Central African Republic
- Congo, Republic of the
- Côte d'Ivoire
- Democratic Republic of Congo
- French Guiana
- Gabon
- Ghana
- Liberia
- Mali
- Niger
- Rwanda
- São Tomé and Príncipe
- Sierra Leone
- Togo
- Always check up to date list at www.cdc.gov/travel

Yellow Fever Vaccination Proof Required for Entry

Meningococcal

◀ Meningococcal Disease



- 1st Picture: **Neisseria Meningitidis** (Gram negative diplococci).
- 2nd Picture: Sub-Saharan Africa.
- Youngest children = highest risk.

❖ **Epidemiology**:
- Greatest risk: dry season (Dec. - June).
- Hajj pilgrimage to Saudia Arabia associated with outbreaks.

0.5-10/100,000 in non-epidemic areas.

Risk of travelers: **0.4/100,000**.

Up to 1,000/100,000 in epidemic areas.

◀ Meningococcal vaccine

- Vaccine required to attend the Hajj (annual pilgrimage to Mecca)
  - If under age 15, polio vaccination needed also.
  -

❖ **Available vaccines**:

MCV4
(Menactra™)
- Quadrivalent meningococcal polysaccharide–protein conjugate vaccine.
- 2-55 years old.
- Preferred in <11 year olds.

MPVS4
(Menomune®)
- Quadrivalent meningococcal polysaccharide vaccine
- 2 years and older.
- Use for >55 years old.

MenACWY-CRM
(Menveo®)
- Quadrivalent meningococcal oligosaccharide diphtheria CRM197 Conjugate Vaccine.
- 11-55 years old.

❖ **Revaccination**: If high-risk (epidemic area or travel).

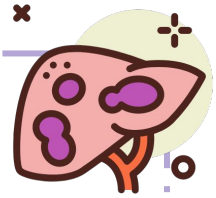
If vaccine given at **2-6 years** old:
Repeat after **3** years, then every **5** years.

If vaccine given **>6 years** old:
Repeat every **5** years.

An area for your notes

Hepatitis A

◀ Hepatitis A



- Worldwide prevalence.
- Adults often contract from asymptomatic children.
- Incubation 28 days (range 15-50 days).
- Viral shedding 2 weeks before to 1 week after symptoms.
- Usually self-limited disease.

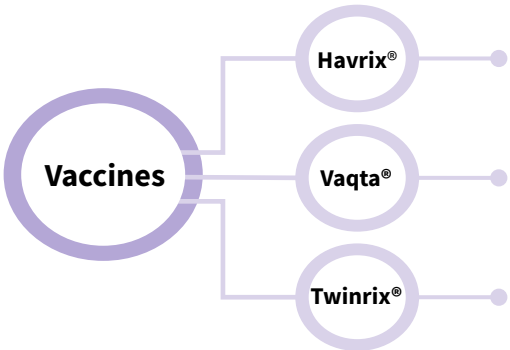
❖ **Mode of transmission**:
- ➔ Fecal/oral transmission (Associated poor hygiene or sanitation).

❖ **Symptoms include**:
- ➔ Jaundice
- ➔ Fatigue
- ➔ Abdominal pain
- ➔ Anorexia
- ➔ Nausea

◀ Hepatitis A Vaccine

- Inactivated Hep A virus (**Havrix**® or **Vaqta**®).
- Combined with Hepatitis B (**Twinrix**®).

❖ **Travel vaccine indications**:
- ➔ Anyone **>1 year old** traveling anywhere other than to:
  - ◆ U.S. and Canada, Western Europe, Scandinavia, Japan, Australia and New Zealand.
- ➔ For healthy patients **<40 years** old, one dose before travel confers adequate protection.



01
➔ Dose at **0** and booster at **6-12 months**.

02
➔ Dose at **0** and booster at **6-18 months**.

03
- Combination of Inactivated **Hepatitis A** with Recombinant **Hepatitis B**.
- Indicated for **18 years** old and **older**.
- Better choice if both vaccines are indicated
- 3-dose series:
  - ➔ **0**, **1**, **6 months**.
- 4-dose accelerated series:
  - ➔ **0**, **7 days**, **21-30 days** and **12 months**.

◀ Hepatitis A treatment

❖ **Consider immunoglobulin treatment for patients**:
- ➔ Leaving in less than two weeks.
- ➔ Older.
- ➔ Immunocompromised.
- ➔ Chronic medical conditions.
- ➔ Under 12 months of age.

Influenza

Skipped by the dr

◀ Risk factors

- **Risk depends on timing and destination**:
  - Tropics: year round risk.
  - Temperate climates: risk generally April-September.
- **Avian subtype risks**:
  - Visiting poultry farms.
  - Visiting open markets where live poultry are present.
  - Eating undercooked poultry products (eggs, meat, etc.).

◀ Preventative measures

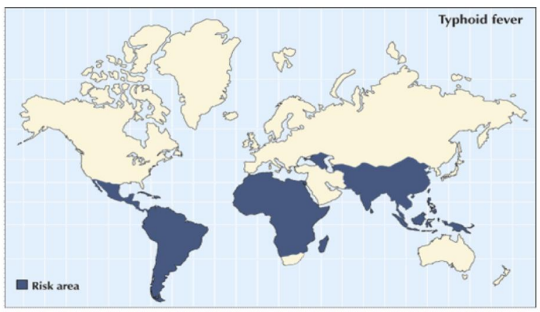
Hygiene: washing hands.

Annual vaccination.

Typhoid Fever

Definition Acute life-threatening illness.Caused by Salmonella typhi[1].

◀ Epidemiology



22,000,000 cases worldwide/year.
200,000 deaths.

Risk area:

- Southeast Asia
  - 6-30 times more common.
  - Highest risk of FQ (Fluoroquinolones) drug resistance.
- Africa, Caribbean, Central and South America.
- Length of stay = increased risk.

◀ Mode of transmission

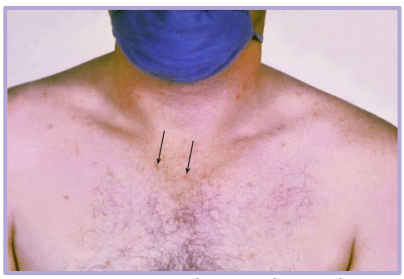
- Humans – only source:
  - ➔ Acquired through fecal contamination of food and water.

1. After a few days of bacteraemia, the bacilli localise, mainly in the lymphoid tissue of the small intestine, resulting in typical lesions in the Peyer's patches and follicles. These swell at first, then ulcerate and usually heal. After clinical recovery, about 5% of patients become chronic carriers (i.e. continue to excrete the bacteria after 1 year); the bacilli may live in the gallbladder for months or years and pass intermittently in the stool and, less commonly, in the urine.

Typhoid Fever

Signs & symptoms[1]

- Incubation period: **6-30 days**.
- Headache.
- Malaise.
- Fever.
- Increasing in severity.
- Low-grade septicemia.
- "**Rose spots**" on trunk.



Rose spots on the chest in a patient with typhoid.

At the end of the first week, a rash may appear on the upper abdomen and on the back as sparse, slightly raised, rose-red spots, which fade on pressure.

Complications

- Serious complications (**2-3 weeks**):
  - ➔ **Hepatosplenomegaly.**
  - ➔ **Intestinal hemorrhage/perforation.**

Treatment

- Oral rehydration.
- Antibiotics:
  - ➔ **3rd generation cephalosporin** (10-14 days).
  - ➔ Azithromycin Ciprofloxacin only if no resistance (7-10 days).
- Steroids in severe cases.

Prevention

- Avoid contaminated food and water.
- Hygiene.
- Local cuisine.

Vaccines

- **2** available vaccines:

Vivotif®

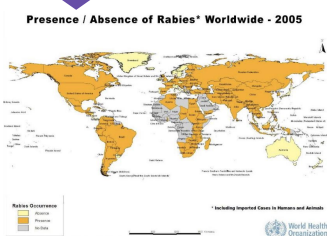
- Oral, live-attenuated.
- Ages **6** and **older**.
- 50-80% protection.
- 4 pills – one every other day.
- Completed **1 week** before potential exposure.
- Revaccination **every 5 years**.

Typhim Vi®

- Capsular polysaccharide (IM).
- Ages **2** and **older**
- 50-80% protection.
- Single 0.5ml injection.
- **2 weeks** before exposure.
- Booster **every 2 years**.

Rabies[2]

- Found globally.
- ❖ **Consider vaccination**:
  - ➔ If potential exposure to wild animals (especially dogs).
  - ➔ Prolonged exposure where endemic.



Pre-exposure prophylaxis[3]

- Series of 3 at **0**, **7** and **21-28 days**.
- 2 vaccines available:
  - ➔ Imovax®
  - ➔ Rabavert®

Post-exposure

- **Rabies Immunoglobulin** (RIG) plus **vaccine**:
  - ➔ RIG days **0**, **4**.
  - ➔ Vaccine days **0**, **3**, **7**,**14**.
- If had vaccine:
  - ➔ No RIG needed.
  - ➔ Vaccine days **0** and **3**.

1. The temperature rises in a stepladder fashion for 4 or 5 days with malaise, increasing headache, drowsiness and aching in the limbs. Constipation may be caused by swelling of lymphoid tissue around the ileocaecal junction, although in children diarrhoea and vomiting may be prominent early in the illness.
2. Rabies is caused by a rhabdovirus that infects the central nervous tissue and salivary glands of a wide range of mammals, and is usually conveyed by saliva through bites or licks on abrasions or on intact mucous membranes. Humans are most frequently infected from dogs and bats.
3. Pre-exposure prophylaxis is required by those who handle potentially infected animals professionally, those who work with rabies virus in laboratories and those who live at special risk in rabies-endemic areas.

Japanese Encephalitis

◀ Japanese Encephalitis Virus (JEV)

- Most common cause of encephalitis in Southeast Asia.
- Carried by mosquitoes
- ❖ **Risk**:
  - ➔ Little risk in urban areas.
  - ➔ Mostly rural areas.
    - Not recommended for short-term travel to urban area.

Geographic distribution in Southeast Asia.

◀ Signs & symptoms[1]

- Incubation **5-15 days**.
- Most infections asymptomatic, <1% develop clinical disease:
  - Headache, fever, vomiting, diarrhea.
    - Most recover in 1 week.
- 1:300 severe symptoms with 30% fatality:
  - Mental status changes.
  - Focal neurological deficits.
  - Parkinsonian syndrome.
  - Seizures (especially children).

◀ vaccine

- Inactivated **Vero cell culture** (JE-VC):
  - For people **over 17 years** old.
  - Duration of protection unknown.
  - Need for boosters undetermined.
  - Pregnancy Category B.

Travelers' Diarrhea Self-treatment

Antibiotic +/- loperamide

- **Quinolones**: single dose, max 3 days.
- **Azithromycin**: single dose of 500mg max 3 days or one time dose of 1000 mg.
- **Rifaximin**

Giardiasis (giardia duodenalis): rotten eggs burping, diarrhea.

- **Metronidazole**: 250 mg po TID 7 d.
- **Nitazoxanide**: 500 mg po bid 3 days

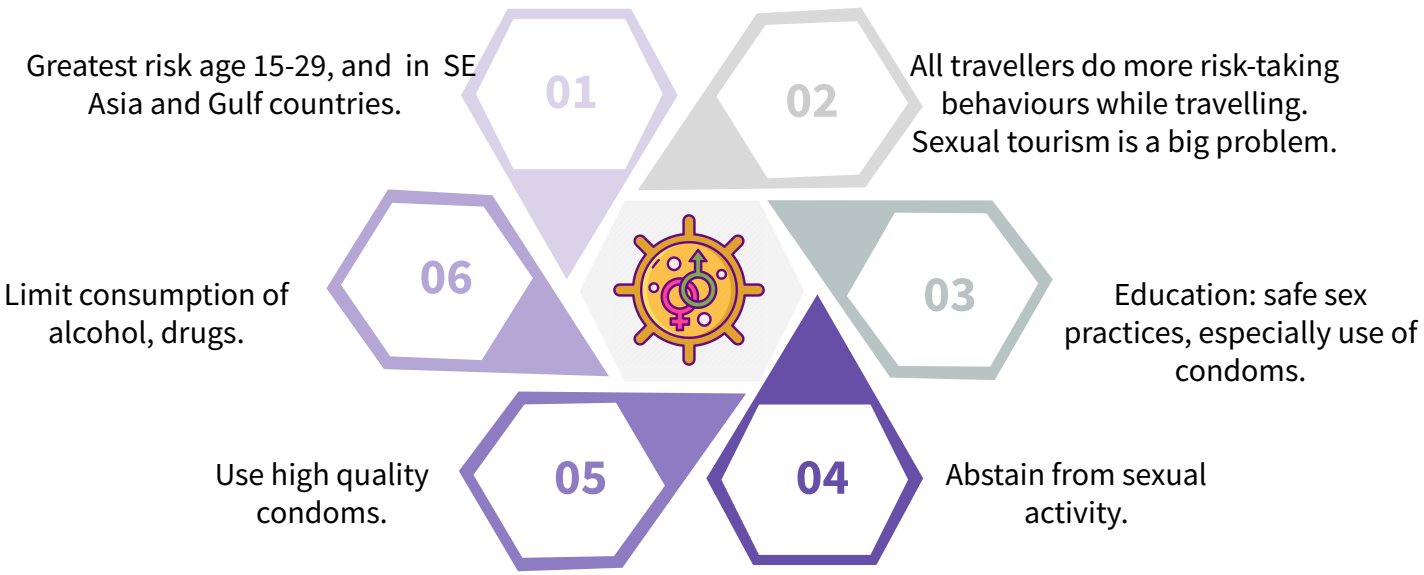
.

1. Initial systemic illness with fever, malaise and anorexia is followed by photophobia, vomiting, headache and changes in brainstem function. Neurological features other than encephalitis include meningitis, seizures, cranial nerve palsies, flaccid or spastic paralysis, and extrapyramidal features. Mortality with neurological disease is 25%.

Advice for Acclimatization

- Tell climbers to avoid abrupt ascent to altitudes above 9850 ft.
- Spend 2-3 nights at 8200-9850 ft before further ascent.
- Add an extra night of acclimatization for every 2000-3000 ft of ascent.
- Make day trips to higher elevation with return to lower elevation for sleep.
- Avoid alcohol and sedatives for the first 2 nights at altitude.
- Moderate exercise (Extreme exercise at altitude may be harmful).
- acetazolamide 125-250 mg bid starting 24 hours before ascent and continue for 48 hrs at maximum altitude.
- Side effects: paresthesias, polyuria, nausea, drowsiness, impotence, myopia, bitter taste.

Sexually transmitted illnesses and Travel

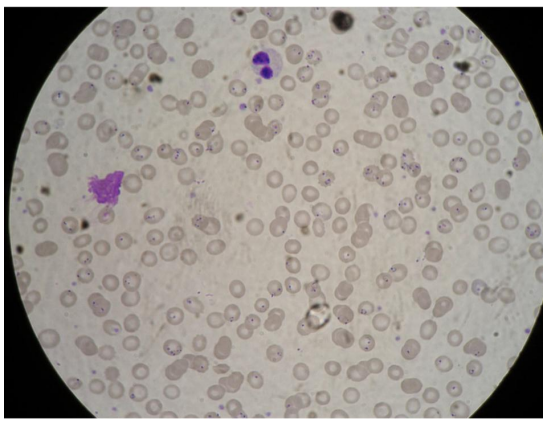


◀ Case study 1:

❖ 18 year old pregnant woman returned from **Jazan** one week ago. Fever and headache for 3 days.
Physical examination: Pale, Temp. 39°C Spleen enlarged.
Investigations: WBC 8, Hb 9.0, MCV 93, Plt 90, bilirubin 52

- **What is your diagnosis?**
  P.falc malaria showing double chromatin dotted ring and multiple invasions in one RBC

- **What are the risks in her condition?**
  - Severe disease.
  - Primigravidae.
  - Risk of low birth & abortion.
  - Risk of low glucose , pulmonary oedema.

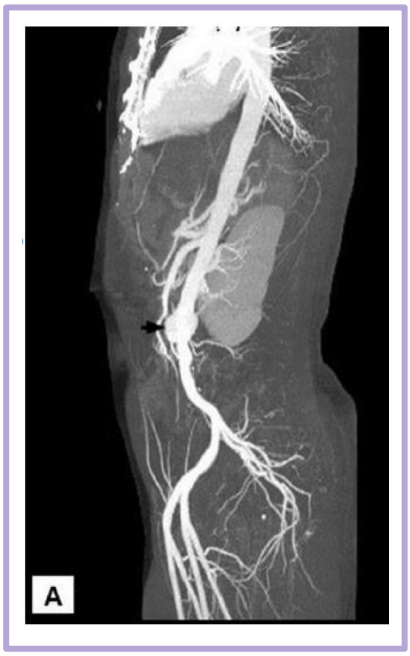


◀ Case study 2:

❖ 42 Saudi Man with continuous fever for one month Chills, rigors, weight loss 2 weeks of mid-back pain radiating to flanks 1 month prior to onset of fever traveled to Thailand, visited rice fields. He diagnosed with **Melioidosis**.

- **What do you see in the picture?**
  Infrarenal abdominal aortic pseudoaneurysm.

- **What do you expect to see in the blood culture?**
  Burkholderia pseudomallei causative agent of melioidosis.



Summary

Epidemiology	• **Stable transmission:** including much of **sub- Saharan Africa**, transmission occurs consistently year round. The bulk of the mortality is seen in **children**, while those who survive to adulthood acquire significant immunity; **low-grade parasitaemia** is still present but causes few symptoms. • **Unstable transmission:** **seasonal or low- level transmission** (e.g. in the **Sahel belt**, where mosquitoes feed only in the rainy season). **Little protective immunity** develops and symptomatic malaria occurs at **all ages**.
Pathogenesis	• RBCs invasion. , Microvascular pathology, Renal failure, Deep coma, Pulmonary edema , Immune-complex Nephrotic syndrome.
Plasmodium Falciparum	- **Resistant** to many antimalarial drugs. - **Most morbidity and mortality.** - Infects **mature** and young erythrocytes. - Cytoadherence. - **Schizogony** is particularly prolific in all stages. - Infection in the peripheral blood > **ring forms** and **gametocytes "**crescent-shape". - Travel history up to 1 year (usually first 3 months), may present as continuous fever.
p.Vivax	- Benign' **tertian malaria.** - Restriction of erythrocyte invasion to **reticulocytes bearing Duffy blood antigen** - **Schüffner's dots**. - **Hypnozoites** > remain latent responsible for subsequent relapse.
p.Ovale	- Can **infect Duffy negative** reticulocytes. - **Late-onset vivax and ovale** malaria may occur despite effective prophylaxis. - **Hypnozoites** >remain latent responsible for subsequent relapse.
P. Malariae	- **Slow development.** - The asexual cycle is **72** hours '**quartan malaria**'. - Infects old erythrocytes >'**smaller**' infected cells. - **Do not transform into hypnozoites** > **no relapses.** - The peripheral blood (**10 years or more**) at a very low level of parasitaemia >detectable peaks with a recrudescence of clinical symptoms. - Travel history up to 10 years.
Treatment	• **Oral therapy:** ➔ Artemether-Lumefantrine. ➔ Atovaquone / Proguanil (Malarone). ➔ Quinine plus Doxycycline/Clindamycin -Avoid Mefloquine for treatment. - • **Parenteral therapy:** ➔ Artesunate. ➔ Quinine (or quinidine): needs telemetry. • **Non-P. Falciparum:** - **Chloroquine** at 6, 24 and 48 hours. - **Primaquine** for 14 days: In **vivax and ovale** after treatment of acute infection use to **eradicate liver parasites**; G6PD must be measured before primaquine.
Chemoprophylaxis	• **start 2 days pre-travel, continue 7 days after return:** ➔ Atovaquone/ Proguanil (Malarone): 1 tab/d. • **One or 2 weeks pre-travel, continue 4 weeks after return:** ➔ Mefloquine: once/wk. ➔ Doxycycline: 100 mg daily. ➔ Primaquine: base daily. ➔ Chloroquine: sensitive areas.

Lecture Quiz

Q1: The region with the greatest morbidity and mortality from malaria in the world is?

A. Africa.
B. Southeast Asia.
C. South America.
D. Oceania.

Q2: A young American adult consults with you before travel to Kenya. Appropriate regimens for the prevention of malaria in travelers from a developed country to areas with chloroquine-resistant P. falciparum malaria include

A. Malarone, meloquine, or artemisinin.
B. Malarone, meloquine, or doxycycline.
C. Coartem, Malarone, or doxycycline.
D. quinine, artemisinin, or Fansidar.

Q3: A child from Ghana is admitted with fever, altered consciousness, acute renal failure, and 7% parasitemia with P. falciparum. Severe malaria should be treated with:

A. Oral artemisinin-based combination therapy
B. Malarone or meloquine
C. Quinine or quinidine
D. Intravenous artesunate, if available, and as a back-up intravenous quinine or quinidine

Q4: Reasons for the predilection for P.falciparum to cause severe malaria include all of the following excepT

A. cytoadherence of P. falciparum–infected erythrocytes to vascular endothelium.
B. infection of erythrocytes of all ages.
C. a high prevalence of resistance to available antimalarial drugs.
D. increasing pathogenicity with increasing age of the patient.

Q5: A 55-year-old woman presented in May with fever and headache of 1 day's duration. She returned 7 days earlier from a 3-week trip to game parks in Tanzania, East Africa. On examination, her temperature is 40° C, blood pressure is 120/85 mm Hg, heart rate is 120 beats per minute, and respiratory rate is 18 breaths per minute. She has no rash, her neck is supple, chest is clear to auscultation and percussion, and abdomen is soft and nontender with normal bowel sounds. he laboratory technician on call has never done a malaria smear, and the hospital does not offer rapid diagnostic tests. Results from a reference laboratory will be available in 24 hours. Which of the following would be the best course of action?

A. Treat with meloquine
B. Treat if parasites are identified at the reference laboratory
C. Treat with chloroquine
D. Treat with artemether/lumefantrine (Coartem)

Q6: The region with the most highly resistant malaria parasites in the world is:

A. Africa
B. Southeast Asia
C. South America
D. Oceania

Answers: Q1:A | Q2:B | Q3:D | Q4:D | Q5:D| Q6:B

Answers Explanation File!

THANKS!!

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Send us your feedback:
We are all ears!