Malaria & Travel Medicine

Objectives:

- Epidemiology & Etiology.
- Clinical presentation.
- Risk to travelers.
- Malaria and pregnancy.
- Diagnostic work up.
- Treatment & Prophylaxis.

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Resources: 435 team + Davidson + kumar + Medicine 500 Single Best Answers

- Editing file
- Feedback

Malaria

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.

★ Epidemiology: (Grey text here is included in the lecture but you're not expected to memorize it)

- In 2017, an estimated 219 million cases of malaria occurred worldwide, compared with 239 million cases in 2010 and 217 million cases in 2016.
- Most malaria cases in 2017 were in the WHO African Region (200 million or 92%), followed by the WHO South-East Asia Region with 5% of the cases and the WHO Eastern Mediterranean Region with 2%.
- Fifteen countries in sub-Saharan Africa and India carried almost 80% of the global malaria burden. Five countries accounted for nearly half of all malaria cases worldwide: Nigeria (25%), Democratic Republic of the Congo (11%), Mozambique (5%), India (4%) and Uganda (4%).
- The 10 highest burden countries in Africa reported increases in cases of malaria in 2017 compared with 2016. Of these, Nigeria, Madagascar and the Democratic Republic of the Congo had the highest estimated increases, all greater than half a million cases. In contrast, India reported 3 million fewer cases in the same period, a 24% decrease compared with 2016.
- The incidence rate of malaria declined globally between 2010 and 2017, from 72 to 59 cases per 1000 population at risk. Although this represents an 18% reduction over the period, the number of cases per 1000 population at risk has stood at 59 for the past 3 years.

- **Plasmodium falciparum is the most prevalent malaria parasite** in the WHO African Region, accounting for 99.7% of estimated malaria cases in 2017, as well as in the WHO regions of South-East Asia (62.8%), the Eastern Mediterranean (69%) and the Western Pacific (71.9%). P. vivax is the predominant parasite in the WHO Region of the Americas, representing 74.1% of malaria cases.
- **In 2017, there were an estimated 435,000 deaths from malaria globally**, compared with 451,000 estimated deaths in 2016, and 607,000 in 2010.
- Children aged under 5 years are the most vulnerable group affected by malaria. In 2017, they accounted for 61% (266,000) of all malaria deaths worldwide.

- Endemic disease.
- Usually does not occur at altitudes above 1500 m.
- World wide ease of travel.
- Most important parasitic disease of humans.
- Transmitted in over 100 countries.
- Affecting more than 3 billion people world wide.
- Causing 1-2 billion deaths per year.
- Prevalent in Tropical Climates, Parts of Africa and Middle East.
- **Transmitted Via female Mosquito Bite.**
- The mosquito require moderate temperature, it can't live in extreme hot or cold weather. Can't live in Riyadh, Al Baha, and Khamis Mushait.
- People living in endemic malaria areas have an some kind of immunity against malaria but its not permanent[1].

[1] they develop an acquired semi-immunity, to read more about it Click here.

★ Etiology:

Malaria is caused by the **Plasmodium parasite**. The parasite can be spread to humans through the **bites** of infected mosquitoes (**Female** Anopheles mosquitoes). There are many different types of plasmodium parasite, but only **5 types** cause **malaria** in humans:

1. **Plasmodium falciparum** – mainly found in Africa, it's the **most common** type of malaria parasite and is responsible for most malaria deaths worldwide.

Any patient suspected to have Malaria, we assume and treat him as if its caused by P. Falciparum. Because it's the worst and most serious type.

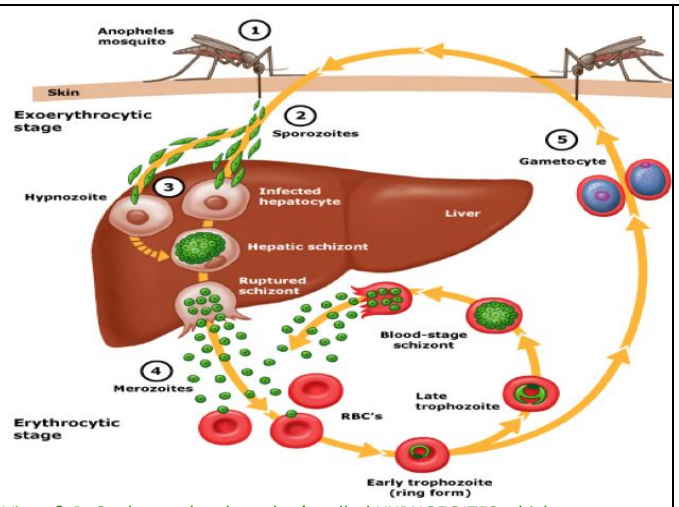
2. **Plasmodium vivax** – mainly found in Asia and South America, this parasite causes **milder** symptoms than Plasmodium falciparum, but it can stay in the liver for up to 3 years, which can result in relapses.
3. **Plasmodium ovale** – fairly uncommon and usually found in West Africa, it can remain in your liver for several years without producing symptoms. P. Vivax and P. Ovale look the same under the microscope and their way in causing the disease.
4. **Plasmodium malariae** – this is quite rare and usually only found in Africa. Usually does not cause a serious disease.
5. **Plasmodium knowlesi** – this is very rare and found in parts of southeast Asia.

★ Incubation Period and Life Cycle

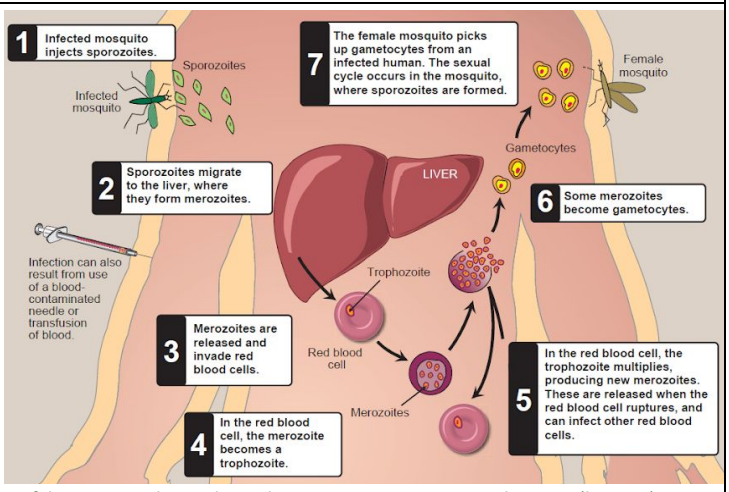
- Sporozoites reach the liver within 1-2 hours following Female Anopheles mosquito bite.
- Pt. asymptomatic for 12-35 days until RBCs stage of parasite life cycle.

Life cycle of Malaria:

1. Infected female Anopheles mosquito feeding on human → injects **Sporozoites (infective stage)**
2. In the bloodstream **Sporozoites** go to the LIVER & live inside hepatocytes (hypnozoite)/ Or infect the liver cell and kills it.
3. Mature into **Schizonts** → bursting into tons of **Merozoites**
4. Back to the bloodstream → infects healthy RBCs go 2 ways, **1.(Ring -> Trophozoites -> RBC schizonts ->** bursting into many **merozoites)** → massive RBCs destruction. **2.(Ring -> Gametocytes)**
5. Another female Anopheles mosquito bites infected human → picking plasmodium **Gametocytes**
6. Inside the mosquito the **Gametocytes** mature into sporozoites & so on..



P.Vivax & P. Ovale can develop what's called HYPNOZOITES which can **stay dormant (sleeping)** in the liver & result in **reinfection** up to years after the parasites have been cleared from blood!!



P. falciparum and P. malariae have NO persistent exoerythrocytic (hepatic) phase. (no hypnozoites)

★ Pathogenesis

RBCs invasion	- P. F[2]: invades RBC at all ages. - P. Mal[3]: only old RBC. - P. ovale and P. vivax invade young RBC's.
Microvascular pathology	Secondary Ischemia Adherence of Non-deformable parasitized RBC to endothelium. Normal RBCs can be squeezed through small vessels whereas RBCs that are filled with parasites become rigid & cannot be squeezed → causing occlusion of vessels & ischemia. In P. F, infected RBC adhere to vascular endothelium in post-capillary venules in brain, kidney, liver, and lungs causing vessel congestion which results in organ damage
Renal failure	Hemolysis, Ischemia secondary microvascular pathology.
Deep coma[4]	Hypoglycemia, microvascular adherent parasitized RBC.
Pulmonary edema	Secondary to capillary leak Syndrome[5] (without congestive cardiac failure).
Immune-Complex Nephrotic Syndrome	Secondary to P. Malariae, this is only in chronic cases.

★ Clinical Features:

Clinical features vary with: Geography, Epidemiology, and Age.

High risk population includes: 1. Children
2. Pregnant women
3. Non-immune travelers to malaria endemic areas[6]

Major clinical Features:

- Recurring Fever, depends on the type of parasite.
- Chills. (Associated with RBC lysis – mature schizonts).

Severe vs Chronic

Severe P. Falciparum infection: (> 10 parasite/ mcl)
Acute complications:

- Renal failure
- Coma secondary to hypoglycemia, TNF, or microvascular pathology
- Pulmonary Edema
- Thrombocytopenia
- G. Enteritis – especially diarrhea.

Chronic P. Falciparum infection:
P. Falciparum is usually acute but can be chronic when the mature parasite stays in the spleen causing splenomegaly.

- Splenomegaly typically resolves after treatment with anti-malarial medications (6-12 months).
- P. Malariae associated Immune complex nephrotic syndrome.
- P. Vivax – late splenic rupture with trauma 1-3 mon. after initial infection.

[2] Plasmodium falciparum
[3] Plasmodium malariae
[4] Those with coma or seizures have the worst prognosis and treatment won't improve mortality.
[5] As deformed infected RBCs congest and block pulmonary vasculature (Microvascular pathology) → Leaking of proteins and fluids form small capillaries to the surrounding tissue leads to capillary leak → Pulmonary Edema.
[6] Non-immune travelers = Travelers Not on prophylaxis medications.

Malaria Paroxysm (attack):VERY IMPORTANT				
Three successive stages:				
Rigors, Headache associated with pale **COLD** skin (1-2 Hr) Not specific	→	Delirium, Tachypnoea, **HOT** Skin (Several hours) Due to the vasoconstriction	→	Fever (Marked **SWEATING** and fatigue) patient usually goes to sleep in this stage

▸ **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"
  - and every 72 hours with (P.malariae) "quartan parasite"
  - whereas (P.falciparum) show IRREGULAR attacks ,or hectic (especially In non-immune)

★ Diagnosis:

- Detailed targeted **history** including **travel history** and clinical examination together with **High Index of Suspicion (HIS).**
- **Blood film**
- Serology: not useful in managing acutely ill patient.
- DNA probe(PCR): similar thick film sensitivity.
- Infection with more than one parasite spp: 5-7%.

★ Blood film:

- Usually done by **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**:

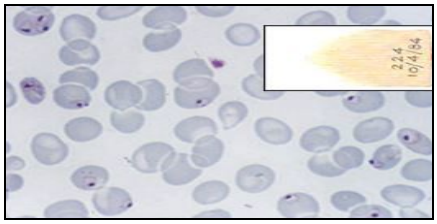
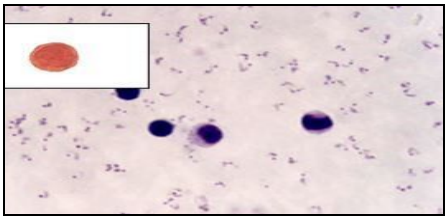
- Only **ring stage** asexual parasite and gametocytes seen in peripheral Blood.
- While RBC with Schizonts stage: sequestered in peripheral, Microvasculature, and NOT circulating peripheral blood.

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

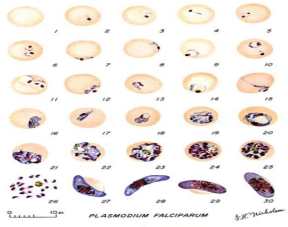
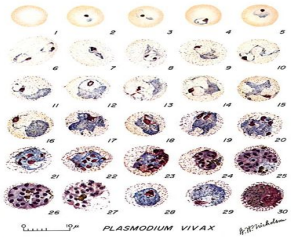
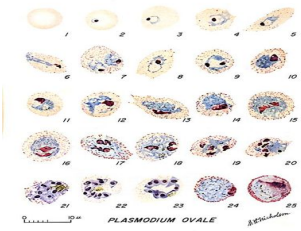
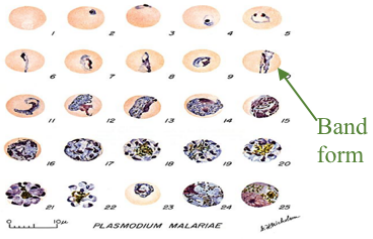
➔ Acutely ill Patient Differential Diagnosis is P. Falciparum Or P. Vivax Because:

- P. Ovale & P. Vivax present as acute infection but clinically and morphologically are the same.
- P. malariae: present as a chronic Infection not acute.

★ Thin Vs. Thick Blood Film (THICK smear for detection, THIN smear for speciation)

Thin blood film	**Thick** blood film
○ **RBC morphology preserved.** ○ In P. Vivax, infected RBC enlargement with parasite maturation. ○ Schuffner's dots (Eosinophilic dots in RBC cytoplasm). ○ May see Maurer's clots in RBC cytoplasm. 	○ **RBC lysed.** ○ You may examine 10X. Blood more than in thin film. ○ More diagnostic in lower degree of parasitemia. 

For further reading:

Organism	Blood Stage Parasites	Thin Blood Smears
1-Plasmodium falciparum:	1: Normal red cell 2-18: Trophozoites (2-10: ring-stage trophozoites) 19-26: Schizonts (26 is a ruptured schizont) 27-28: Mature Macrogametocytes (female) 29-30: Mature microgametocytes (male)	
2- Plasmodium vivax:	1: Normal red cell. 2-6: Young trophozoites (ring stage parasites). 7-18: Trophozoites. 19-27: Schizonts. 28-29: Macrogametocytes (female). 30: Microgametocyte (male).	
3- Plasmodium ovale:	1: Normal red cell. 2-5: Young trophozoites. 6-15: Trophozoites. 16-23: Schizonts. 24: Macrogametocytes (female). 25: Microgametocyte (male).	
4- Plasmodium malariae	1: Normal red cell. 2-5: Young trophozoites (rings). 6-13: Trophozoites. 14-22: Schizonts. 23: Developing gametocyte. 24: Macrogametocyte (female). 25: Microgametocyte (male).	

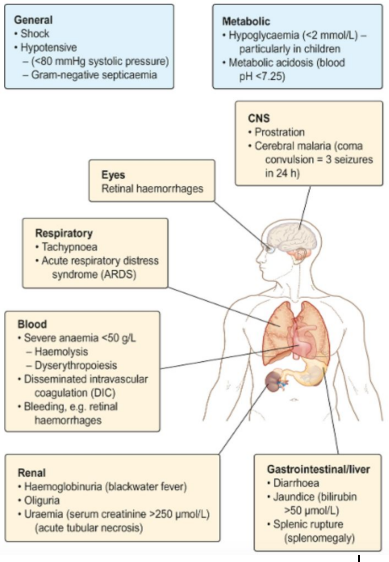
★ Differential Diagnosis Of Malaria In Acutely ill Patients Based On P.B. Smear

	P. Falciparum	P. Vivax/P. Ovale
Multiple Infected RBC	common	rare
Mature (Trophozoite and 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.

★ Malaria Complications: VERY IMPORTANT

1. Major clinical features of malaria are those of the complications.
2. Majority of complications (apart from anemia) are associated with **P. Falciparum.**



★ Anemia related complications:

Anemia presents in most severe infections and **parallels** parasitaemia, and it's **due to:**
- ➔ Hemolysis of infected RBCs
- ➔ Delayed reticulocyte release from bone marrow
- ➔ Immune-mediated hemolysis of non-infected RBCs

In non-immune patients (Primary infections):	- Black water fever (Haemoglobinuria) - Exaggerated haemolytic response to quinine[7]-sensitized RBCs
Jaundice	- Mild unconjugated jaundice is common, and **parallels hemolysis.** Hepatocellular dysfunction may contribute to jaundice.
Tissue hypoxia	Hypoxia results from anemia + altered microcirculation (Maturation of erythrocyte schizonts in P. falciparum takes place in tissue capillaries and venules).
P.falciparum parasitized RBC sequestered in microcirculation	because of: - Altered deformability of parasitized RBC - Adhesion involving parasite-derived proteins within RBC and glycoproteins on vascular endothelium.

[7] Quinine is a medication used to treat malaria and babesiosis.

★ Other complications:

Early complications	
Cerebral Malaria	- Most severe common complication. caused by P. Falciparum **V.Imp: What are the risk factors for poor prognosis in cerebral malaria?** if the patient has all these indicators, treat as **inpatient.** Mnemonic> CBL 1. Increased Creatinine > Nephropathy 2. Increased Bilirubin > Hepatic dysfunction 3. Increased Lactate. **Factors that DO NOT modify outcome in cerebral malaria:** - Depth of coma - Temperature - Vomiting - Seizures, Due to the obstruction of blood flow to the brain. - Parasite load - Anemia - HIV infection (HIV infection also did not affect clinical or biological presentation of cerebral malaria)
Renal Failure	- Most severe common complication - ATN (acute tubular necrosis) - Dehydration - Hypotension - Hyperviscosity
Pulmonary Edema	- ARDS – may complicate acute phase of severe malaise. Fluid overload may contribute.
Hypoglycemia We should give dextrose even if the patient is diabetic	Because of : - Glucose consumption by the host and By plasmodium. - Lactic acidosis. - Quinine/quinidine increase insulin secretion.
Bleeding	- Thrombocytopenia[8] - Consumption coagulopathy.
Other	- Shock: Endotoxemia - Diarrhea - Hyponatremia (due to SIADH[9])
Late complications	
- Tropical splenomegaly in P. Falciparum endemic areas. → caused by repeated attacks - Nephrotic Syndrome with P. malariae. - Burkitt's lymphoma (caused by Plasmodium falciparum or EBV)[10]	

[8] Patient may present with general flu like symptoms like runny nose & fever but with conjunctival bleeding due to the thrombocytopenia. although malaria doesn't affect the platelets, platelets are destroyed by immune complexes.

[9] No one knows why it just happened to be associated with severe malaria.

[10] P. F can activate EBV leading to BL. but, both individually can cause BL.

★ Malaria And Hemoglobinopathies: Malaria is serious in all types of haemoglobinopathies.

- **Heterozygous sickle cell trait[11]:** children are less likely to contract P. falciparum.
- **Hemoglobin S-C disease**: No such protection, rather mortality is higher than normal!
- **Thalassemics**: partially protected (Fetal Hb).
- **G-6-phosphatase RBCs**: less prone to P. falciparum.

★ Malaria & Pregnancy NEVER treated as outpatient

- Mortality reached 30%
- Anemia, hypoglycemia, pulmonary edema: common
- Abortion
- Stillbirth
- Premature delivery high infant mortality
- Low birth weight
- Placental insufficiency
- **High parasitaemia? placenta is a favorable site for P. falciparum.**

★ Congenital Malaria:

- Transplacental infection:
  - ➔ Can be all 4 species
  - ➔ Commonly *P.v.* and *P.f.* in endemic areas
  - ➔ *P.m.* infections in nonendemic areas due to long persistence of species
- Neonate can be diagnosed with parasitemia within 7 days of birth or longer if no other risk factors for malaria (mosquito exposure, blood transfusion)
- Fever, irritability, feeding problems, anemia, hepatosplenomegaly, and jaundice
- Be mindful of this problem even if mother has not been in malarious area for years before delivery

★ Treatment:

Principles of treatment:

- Treatment should be guided by three main factors **(CDC):**
  1. The infecting Plasmodium species.
  2. The clinical status of the patient.
  3. The drug susceptibility of the infecting parasites as determined by the geographic area where the infection was acquired and the previous use of antimalarial medicines.
- If treatment must be initiated before the species is known treat as **P .falciparum.**
- P. falciparum should be presumed to be chloroquine resistant, except in a few areas of Central America and the Middle East.
- Primaquine should be given if Plasmodium vivax or Plasmodium ovale is likely.

Note that:

- Nearly all p. Falciparum today is resistant to **Chloroquine[12]**!!! (except few areas of Central America & Middle East)
- **Primaquine** should be given if Plasmodium vivax or Plasmodium ovale is likely.

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[11] P. vivax uses "Duffy antigen" on RBC. in Sickle cells trait, Lack of the duffy coat protects them from P. vivax.

[12] Due to mutations in the parasite and the mosquito making them resistant.

Resistance patterns: (Grey text here is included in the lecture but you're not expected to memorize it)

Chloroquine-resistant P. falciparum	*Chloroquine-sensitive P falciparum*
Eastern Hemisphere: All of sub-Saharan Africa, Saudi Arabia, Yemen, Iran, Pakistan, Afghanistan, China, Nepal, and all of Southeast Asia **Western Hemisphere:** Panama, Haiti, Brazil, Peru, Bolivia, Colombia, Venezuela, Ecuador, French Guiana, Guyana, and Suriname	**Eastern Hemisphere:** Turkey, Iraq, Syria, Georgia, Azerbaijan, Tajikistan, Turkmenistan, and Kyrgyzstan **Western Hemisphere:** Argentina, Paraguay, Mexico, Guatemala, Costa Rica, Honduras, Nicaragua, El Salvador, and Dominican Republic
Mefloquine-resistant P falciparum	*Chloroquine-resistant P vivax*
Southeast Asia: Regions of Vietnam, Laos, Thailand, Burma, and Cambodia	Papua New Guinea and Indonesia

Treatment Of Uncomplicated Malaria

Uncomplicated (meaning creatinine, bilirubin and lactate levels are normal > no end organ damage) **P. Falciparum** infection:	**One of the following:** ➔ Artemether-Lumefantrine AKA, Artesunate ➔ Atovaquone-proguanil ➔ Quinine, If Artemether is not available, Quinine can be used but it causes cardiotoxicity and insulin secretion. ➔ Mefloquine
Uncomplicated P. Malariae, P. Knowlesi, P vivax or P. ovale infection. **chloroquine-sensitive** P. Falciparum infection:	➔ Chloroquine phosphate or ➔ Hydroxychloroquine
Uncomplicated P vivax infection, expected to be chloroquine-resistant:	**One of the following:** ➔ Quinine ➔ Atovaquone-proguanil ➔ Mefloquine ➔ Amodiaquine

Treatment of Complicated Malaria: (Will not ask about the doses)

1. Quinidine gluconate 10 mg/kg loading dose over 1-2h, then 1.2 mg/kg/h for at least 24h
2. Once parasitemia is < 1% and patient can take oral medication, switch to quinine 650 mg PO TID to complete 3-d course (7d course if malaria was acquired in southern Asia) to prevent the relapse. In addition, give doxycycline 100 mg IV or PO BID for 7d.
3. For **pregnant** women, instead of doxycycline, give clindamycin 20 mg base/kg/day PO divided TID for 7d.

Chemoprophylaxis: One of the following:

- ➔ Atovaquone-proguanil
- ➔ Chloroquine phosphate
- ➔ Doxycycline
- ➔ Mefloquine, safe for pregnant women.
- ➔ Primaquine[13], usually a 6 week course. 4 weeks before traveling, continue there and 1 week after.

[13] Given to prevent relapse, Contraindicated in: Children, Pregnant women, G6PD deficiency.

Other Measures in Treating Severe Malaria	
Antibodies against TNF-a	Steroids **Never use steroids in malaria patient**
- They reduce fever - They have no effect on mortality & morbidity, Why? Effects of other cytokines as IL – 1, TNF- b on pathogenesis of complicated severe malaria.	- Harmful, by controlled trials. - Dexamethasone > longer duration of coma + worse outcomes than patient receiving quinine alone.
Reducing mosquito-human contact	Malaria vaccine (No vaccines for malaria)

Additional Supportive Measures:

- Blood Tx[14] / Exchange Tx in children.
- Hypoglycemia treatment and prophylaxis especially in pregnant women.
- Avoidance of IVF overload.
- Dialysis, In cases of ATN caused by malaria.
- Heparin for consumption coagulation and DIC caused by malaria.
- Pregnant woman & Non-immune traveler should receive prophylaxis.

★ Future Perspective:

Success to control or eradicate malaria faced by obstacles:

- Increasing drug resistance in P. falciparum and appearing resistance in P. vivax.
- Basis of protection against infection and disease not understood.
- Biologic basis of vector capacity responsible for mosquito-borne malaria transmission is unknown.
- Increasing anopheline mosquito resistance to insecticide.

Just go through them:

World Health Organization

وزارة الصحة
Ministry of Health

Kingdom of Saudi Arabia
Ministry of Health
Public Health Agency
Infectious Diseases Control General Directorate
Malaria Department

The national policy of malaria case management in The Kingdom of Saudi Arabia

1. Treatment of simple uncomplicated falciparum malaria:

1.1 First-line Treatment: ***Artesunate (AS) + Sulfadoxine – Pyrimethamine* (SP)**

Age in years	Weigh in Kgs	Day 1		Day 2	Day 3
		SP (500 S+25 P mg tab)	AS (50mg tab)	AS (50mg tab)	AS (50mg tab)
5 - 11 Months	5 - 10 Kgs	½	½	½	½
1 - 6 years	11 - 24 Kgs	1	1	1	1
7 - 13 years	25 - 50 Kgs	2	2	2	2
> 13 years	> 50 Kgs	3	4	4	4

❖ A single dose of primaquine (0.25 mg base/kg bw, maximum dose 15 mg) should be added on the first day of treatment to ACT for uncomplicated falciparum malaria as a gametocytocidal medicine.

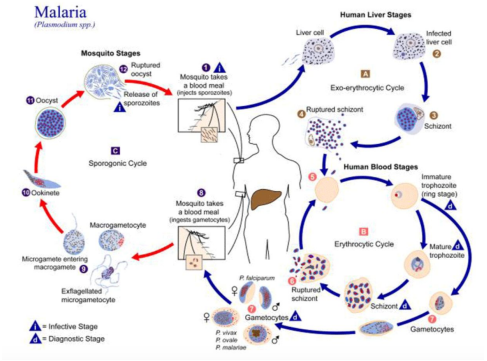
1.2 Second-line Treatment: ***Artemether* 20mg + *Lumefantrine* 120mg**

Age in years	Weigh in Kgs	Day1		Day2		Day3	
		AM	PM	AM	PM	AM	PM
< 5		Not recommended					
<3 years	5 - 14	1	1	1	1	1	1
3 - 8 years	15 - 24	2	2	2	2	2	2
9 - 14 years	25 -34	3	3	3	3	3	3
>14 years	> 34	4	4	4	4	4	4

❖ A single dose of primaquine (0.25 mg base/kg bw, maximum dose 15 mg) should be added on the first day of treatment to ACT for uncomplicated falciparum malaria as a gametocytocidal medicine.

2. Treatment of malaria caused by P. vivax, or P. ovale and malariae:

Chloroquine 25mg base / kg divided over three (3) days, *(Chloroquine 4 tablets day 1, 4 tablets day 2, 2 tablets day 3)* combined with *Primaquine* 0.25 mg / kg bw taken daily with food for 14 days *for vivax and ovale , (Primaquine 15 mg tabs daily for 14 days for adult)*



3.Treatment of severe malaria:

Treatment		Day 1		Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
		Time 0	12 hrs						
First option	*Artesunate* I.V / I.M	2.4mg/kg	2.4mg/kg	2.4mg/kg	2.4mg/kg	2.4mg/kg	2.4mg/kg	2.4mg/kg	2.4mg/kg
Second option	*Artemether* I.M	1.6mg/kg	1.6mg/kg	1.6mg/kg	1.6mg/kg	1.6mg/kg	1.6mg/kg	1.6mg/kg	1.6mg/kg
Third option	*Quinine* I.V	20mg/kg in 5% Glucose(loading dose)		After 8hrs of loading dose start the maintenance dose as, 10mg/kg /8 hourly till the patient can take by mouth then shift to the oral.					

4. Treatment of malaria in pregnancy: **N.B.** Malaria in pregnancy should be considered severe and treated in hospital

Pregnancy in weeks	Uncomplicated malaria	Severe malaria
0-12 (1st trimester)	*Quinine + Clindamycine*	*Quinine + Clindamycine*
13- delivery (2nd &3rd trimester)	* First option: (AS + SP) * Second option: *Quinine + Clindamycine*	*Artesunate* Or *Quinine + Clindamycine*
Puerperium	AS + SP	*Artesunate* Or *Quinine + Clindamycine*

For any queries; ***Please call:*** ***0112917743 or 0114738275***

[14] Transfusion

Summary

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.	
Organisms causing malaria	○ Plasmodium falciparum *most common ○ Plasmodium vivax ○ Plasmodium ovale ○ Plasmodium malariae ○ Plasmodium knowlesi
Malaria life Cycle	1. Infected female Anopheles mosquito feeding on human → giving **Sporozoites (infective stage)** 2. **Sporozoites** goes to the LIVER --> living in/destroying hepatocytes. 3. Mature into **Schizonts** → bursting into tons of **Merozoites** into blood stream. 4. Back to the bloodstream → infecting healthy RBCs **(Ring -> Trophozoites -> schizonts ->** bursting into many **merozoites)** → massive RBCs destruction. Or (Ring -> release of Gametocytes). 5. Another female Anopheles mosquito bites infected human → picking plasmodium **Gametocytes** that mature into sporozoites etc...
Malaria Paroxysm (attack)	1. Rigors, Headache associated with pale **COLD** skin (1-2 Hr) 2. Delirium, Tachypnoea, **HOT** Skin (Several hours) 3. Fever (Marked **SWEATING** and fatigue)
Investigations	○ Detailed targeted history including travel history and clinical examination together with High Index of Suspicion (HIS). ○ Blood film (**Giemsa stain or wright's stain)** ○ Serology ○ DNA probe(PCR): similar thick film sensitivity.
Imp Complications	- Hypoglycemia - Cerebral malaria - Hemoglobinuria - Thrombocytopenia - Splenomegaly
Risk factors for poor prognosis in cerebral malaria	1. Increased Creatinine. 2. Increased Bilirubin. 3. Increased Lactate.
Treatment	- Treatment should be guided by three main factors (CDC): - The infecting Plasmodium species - The clinical status of the patient - The drug susceptibility of the infecting parasites as determined by the geographic area where the infection was acquired and the previous use of antimalarial medicines. *If treatment must be initiated before the species is known treat as P .falciparum. **Uncomplicated P.F, One of the following:** ➔ Artemether-Lumefantrine ➔ Quinine **Prophylaxis:** Primaquine, Mefloquine. **Pregnant:** in cases of relapse instead of doxycycline, give clindamycin.

Questions

1. A 27-year-old woman, who has recently returned from holiday in Africa, presents to accident and emergency with a 7-day history of fevers, sweats, headache, malaise and lethargy. On examination, her temperature is 39°C. Cardio respiratory and gastrointestinal examinations are unremarkable. What is the most likely differential diagnosis?

A. Malaria
B. Tuberculosis
C. Influenza
D. Typhoid

2. A 30-year-old man, who has recently returned from holiday in Africa, presents to accident and emergency with a 7-day history of fever, sweats, malaise and lethargy. Thick and thin blood films detect Plasmodium falciparum. What is the most appropriate treatment?

A. Conservative management
B. Acyclovir
C. Quinine
D. Chloroquine

3. What primary or specific tests should be performed before giving a patient primaquine for the treatment of relapsing malaria?

A- Test for glucose-6-phosphate activity
B- Test for sickle cell anemia
C- Test for blood hemoglobin abnormalities
D- B&C

4. Which of the following is a risk factor for poor prognosis of cerebral malaria?

A- Low hemoglobin level
B- Decreased bilirubin
C- Seizure
D- Increased lactic acids

5. Which of the following is the first line treatment of simple falciparum uncomplicated malaria?

A- Artemether + Lumefantrine
B- Atovaquone-proguanil
C- Mefloquine
D- Artesunate + Sulfadoxine - Pyrimethamine

6. Malaria patients are symptoms-free between paroxysms:

A- True

B- False

7. Which of the following is in high risk for malaria:

A- Five year old travelling to malaria endemic country

B- Third trimester pregnant women travelling to malaria endemic country

C- Non-immune 50 years old travelling to malaria endemic country

D- All at high risk

8. Which of the following is NOT a character for neonates of malaria infected mothers?

A- High birth weight

B- Placental insufficiency

C- Low birth weight

D- High mortality

9- Malaria patients may have a shock because of endotoxemia.

A- True

B- False

10. Which of the following types of plasmodium prefers old RBCs?

A- P. Vivax

B- P. Malaria

C- P. Falciparum

D- P. Ovale

Answers:

1. A 2. C 3. A 4. D 5. D 6. A 7.D 8. C 9.A 10. B

Q2 explanation: Falciparum malaria may develop into a potentially life-threatening disease if left untreated. Therefore, conservative management (A) is not the appropriate answer. Aciclovir (B) is a commonly used antiviral drug that is not used in the treatment of malaria. Widespread chloroquine (D) resistance means that this is not the first-line drug of choice for patient with falciparum malaria. Quinine (C) is used to treat falciparum malaria and can be given orally or intravenously.