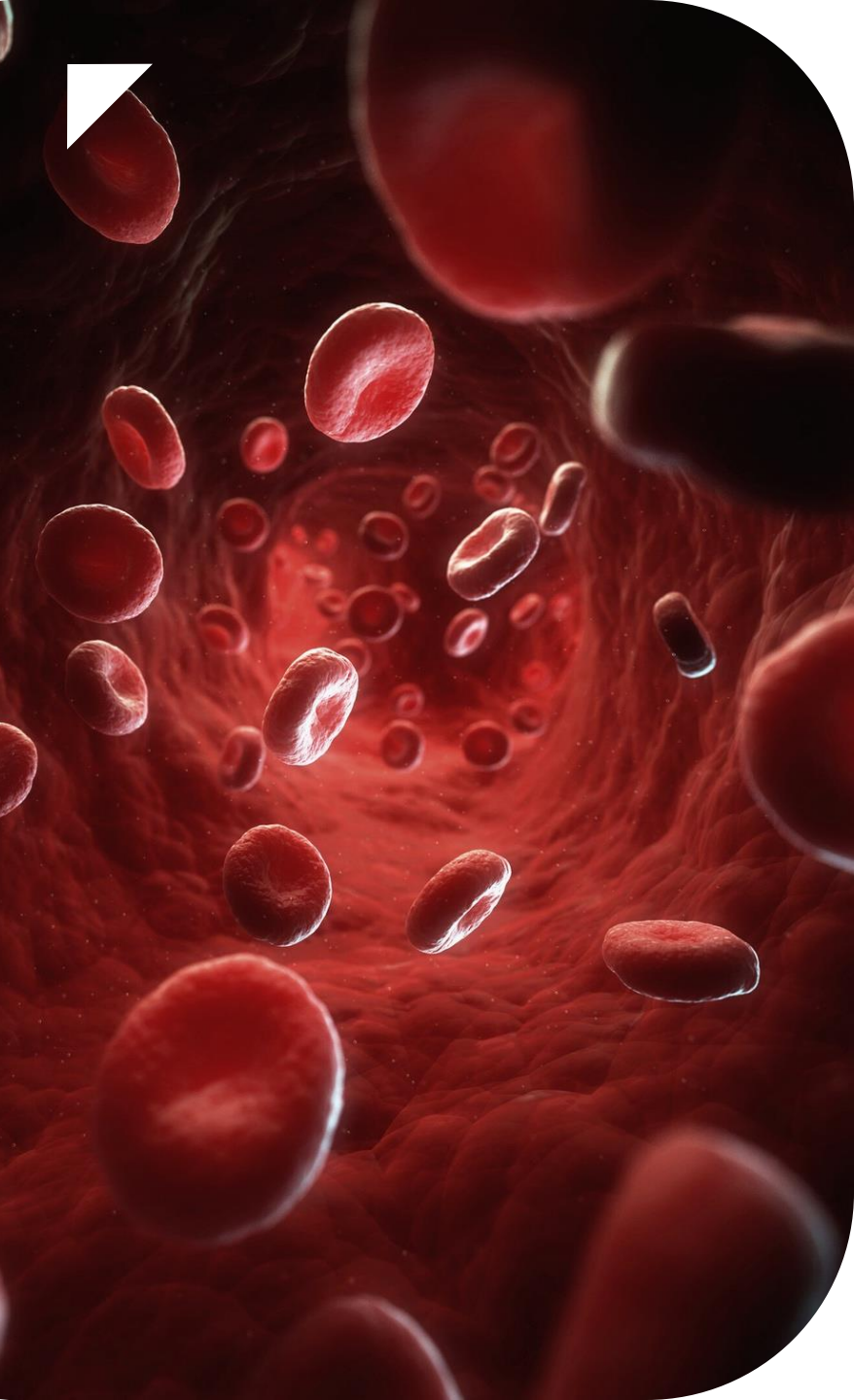




Updates In **Malaria** in Children and Pediatric **Retroviral** Infection

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Objectives



Review the epidemiology of Malaria and RVI.



Identify clinical presentation of uncomplicated and complicated malaria



List recommended treatment of malaria and RVI



OUTLINE

The burden of Malaria

Pathophysiology

Classification and Clinical Presentation of
Malaria

Congenital malaria

Malaria prevention strategies

The burden of Retroviral Infection

Some definitions

Pathophysiology

Clinical presentation and staging of RVI

Management of RVI

Prevention of Mother to Child transmission of RVI



MALARIA IN THE TROPICS

The disease burden



263 million cases of malaria in 2023 compared to 252 million cases in 2022 (representing about **11 million** more cases in 2023 compared to 2022)- WHO 2024 Report



The estimated number of **malaria deaths** - 597 000 (2023) and 600 000 (2022)

- Over half of these deaths occurred in four countries:



Nigeria (**30.9%**)



Democratic Republic of the Congo (**11.3%**)



Niger (**5.9%**)



Republic of Tanzania (**4.3%**)

AFRICA'S MALARIA BURDEN 2023

246M

MALARIA CASES

94%

OF GLOBAL MALARIA
CASES

95%

OF GLOBAL MALARIA
DEATHS

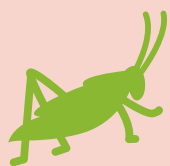
76%

OF MALARIA DEATHS IN
THE REGION WERE AMONG
CHILDREN UNDER 5

Data from Ghana.....



- National Malaria Strategic Plan introduced and implemented over a period of 7 years (2014-2020).
- Remarkable reduction in Malaria deaths from 2,200 (2014) to 308 (2020) – 86.0% reduction.
- Under 5 malaria case fatality rate reduction from 0.51% to 0.12% (75%) over the same period of time.
- National Malaria Strategic Plan (2021-2025) to reduce malaria mortality by 90% by 2025 (using 2019 as baseline).
- This can be achieved by the adherence to the recommended case management of Malaria as well as the availability of the resources.



Malaria is one of the **Commonest** Conditions in Ghana

12 million suspected cases of Malaria
in 2021 (41% OPD cases)

Over 5.7 million confirmed cases
2021 (20% OPD Cases)

21% of all admissions is due to
malaria



Malaria is **endemic** in Ghana/ entire population is at risk

High risk- Children 5yrs, Pregnant
women, Non-immune,
immunocompromised (HIV, DM
etc.), comorbidities-e.g., SCD



Economic cost

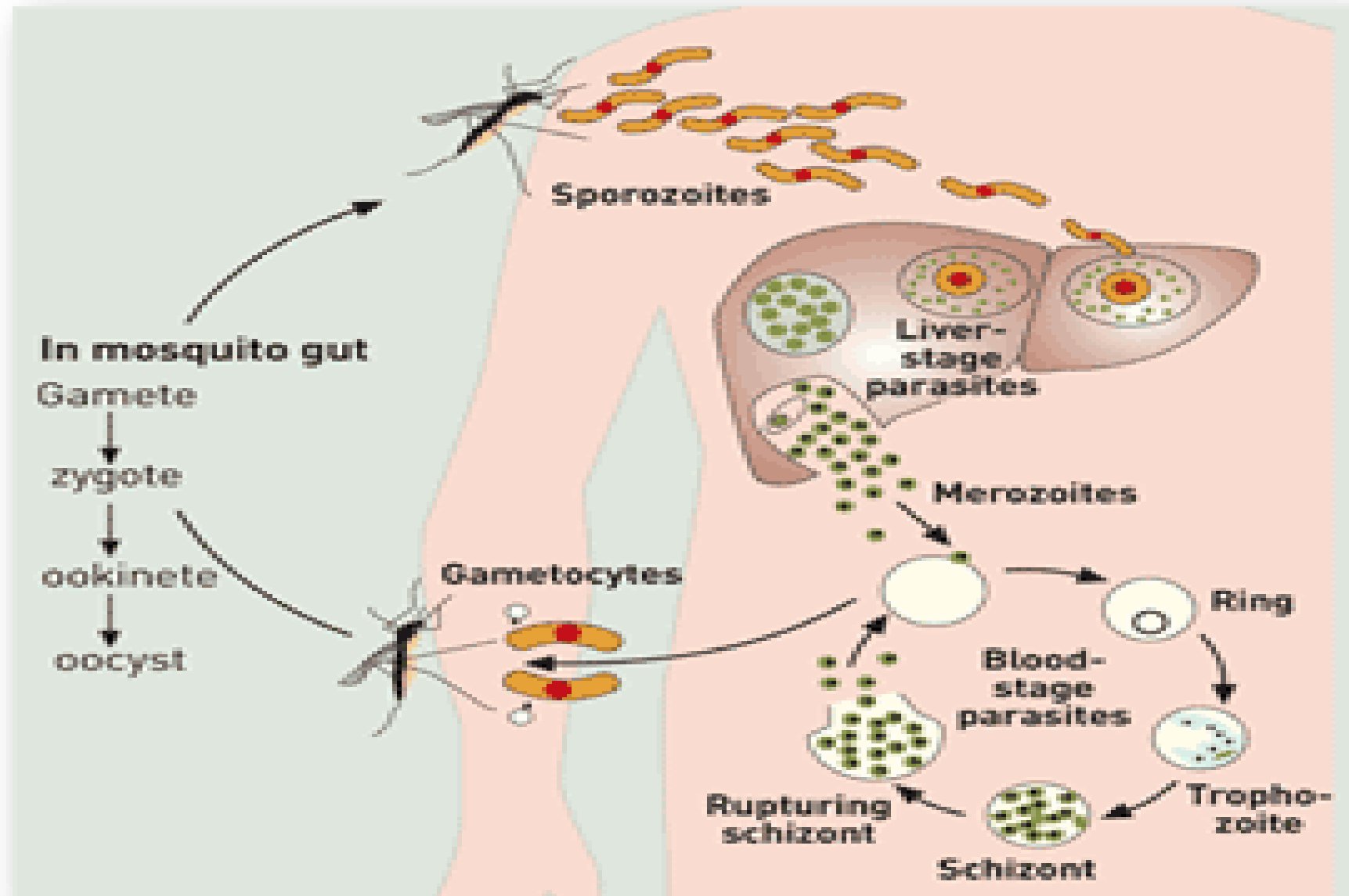
One of the Highest disease
expenditure- NHIS

GDP reduction by 0.25 - 6%

PATHOGENESIS OF MALARIA

- Caused by a parasite (plasmodium)
- 5 **plasmodium species** cause illness(malaria) in man:
 - **P. falciparum** mono infection 97.4%
 - **P. malariae** mono infection 1.1 %
 - **P. ovale** mono infection 0.8%
 - **P. vivax**
 - **P. knowlesi** has recently been identified in Malaysia but not yet identified in Ghana
- 3 main **modes of transmission**
 - Mainly bite of infective female **anopheles mosquito**
 - An accidental transmission via blood transfusion or needle stick injury
 - Congenital transmission from mother to child during pregnancy or parturition

Malaria Life Cycle



Pathogenesis of Malaria cont

The disruption of the tissue endothelial barrier integrity, local tissue inflammation and leucocyte infiltration into the tissues results in the following end organ damage:

1. Brain – Cerebral malaria.
2. Lungs – Pulmonary edema or acute respiratory distress
3. Placenta – Placental Malaria leading to fetal growth restriction and / or wastage
4. Kidneys – AKI with its attendant metabolic acidosis, hyperventilation and hypoxia. AKI can be worsened by anaemia, severe dehydration/shock and haemoglobinuria if present.
5. Hypoglycaemia – results from starvation (loss of appetite or vomiting), increased consumption of glucose by the parasite biomass and disordered glycogenolysis in the liver.

Classification of Malaria

- **Uncomplicated malaria**

- **Fever** or recent history of fever (past 2-3 days)
- No **severe disease** or evidence of **vital organ dysfunction**
- Positive malaria diagnostic **test** (RDT or Microscopy)

- **Severe Malaria**

- Fever
- **Severe disease** or evidence of **vital organ dysfunction** (**1 or more**)
- Positive malaria diagnostic test (RDT or microscopy)
 - *The delay in diagnosis and inappropriate treatment of uncomplicated malaria*

CASE MANAGEMENT

Strategy and core principles for treatment



Early diagnosis and prompt effective treatment



Rational use of quality-assured antimalarial medicines



Use of combination therapy



Appropriate weight-based dosing



Supportive care, monitoring and counselling



Prompt and appropriate referral

Case Management

The Diagnosis of malaria involves:

- Taking a good history
- Physical examination
- Laboratory investigation



Laboratory investigations

- Malaria (complicated or uncomplicated) is confirmed by either;
- **Microscopy (GOLD STANDARD)**
 - Quantitative test for both diagnosis and monitoring the progress of treatment
 - Both thick and thin films performed
 - Thick film – parasite detection, developmental stage and density estimation by the WBCs count method.
 - Thin film – Species of the malaria, percentage of infected RBCs, stage of parasite development and presence of malaria pigment.
 - It is done by reporting on species, stage and parasite density when positive
 - And reported No Malaria Parasites(No MPs) seen when Negative
- **mRDT (Malaria Rapid Diagnostic Test)**
 - Qualitative test
 - Reported as either Positive or Negative
- EITHER ONE CAN BE USED TO CONFIRM THE DIAGNOSIS

Laboratory investigations

- **mRDT (Malaria Rapid Diagnostic Test)**
 - Detects antigens of the parasite
 - Qualitative test, hence, cannot be used to monitor progress of treatment
 - Cannot be used for treatment follow up
 - Can be positive up to 28 days after parasite clearance
 - Histidine Rich Protein 2 (HRP2) based mRDTs used commonly in Ghana
 - Reported as either Positive or Negative
- EITHER ONE CAN BE USED TO CONFIRM THE DIAGNOSIS

Treatment of Malaria

- Depends on the classification of Malaria – Uncomplicated or severe disease
- Uncomplicated Malaria – Oral Antimalarials
- Severe Malaria – Parenteral antimalarials



CLINICAL EVIDENCE OF SEVERE MALARIA

CLINICAL FEATURE	DESCRIPTION
Severe Generalised weakness	Prostration
Impaired consciousness	Coma-GCS<11/15; BCS<3 or P/U in AVPU; Change in behaviour
Convulsions	1 seizure >30 minutes; 2 or more seizures in 24hrs, presents convulsing
Severe Pallor	
Hyperpyrexia	Axillary temperature $\geq 40.0^{\circ}\text{C}$ (for adults and children above 5 years)
Renal impairment	Urine output < 0.5mls/kg/hr for more than 6 hours
Significant Clinical Jaundice	
Respiratory distress	SPO2 < 92% ORA plus RR>30cpm, with labored breathing, crepitations
Significant abnormal bleeding	Including recent, recurrent or prolonged bleeding
Haemoglobinuria	
Shock	Compensated or decompensated

LABORATORY EVIDENCE OF SEVERE MALARIA

DESCRIPTION	LABORATORY EVIDENCE
Severe Anaemia	Hb $\leq$ 5g/dl or HCT $\leq$ 15% (<12 years); Hb $\leq$ 7g/dl or HCT $\leq$ 20% (>12 years)
Renal Impairment	Blood Urea >20mmol/L(120mg/dl); Serum Creatinine >265umol/L(3mg/dl); Creatinine increase 15 fold from baseline within 7 days; Absolute increase of > 26.5umol/L(0.3mg/dL) in 48 hours
Hypoglycaemia	BG < 2.2 mmol/L(40mg/dl). Treat BG < 3mmol/L(54mg/dl)
Jaundice	(Bilirubin > 50umol/l) with P. falciparum count > 100 000/uL(2.5%) or P. knowlesi parasite count > 20 000/uL (0.5%)
Pulmonary edema	Radiologically confirmed
Acidosis	Base deficit of > 8mmol/L or Plasma HCO ₃ level < 15mmol/L; venous plasma lactate $\geq$ to 5mmol/L
Haemoglobinuria	Positive Hb on urine dipstick/chemistry; No RBCs seen on microscopy
Hyperlactaemia	Lactate $\geq$ to 5mmol/L
Hyperparasitaemia	Parasitaemia -P. Falciparum > 500,000/uL(10% RBC infected)NB (for non-immunes, use 100,000/uL); P. Knowlesi > 100,000/uL (2.5%)

Treatment of Uncomplicated Malaria

Treatment Objective

- The **clinical objectives** of treating uncomplicated malaria are to:
 - cure the infection as rapidly as possible
 - (Cure is defined as the elimination of all parasites from the peripheral blood)
 - prevent progression to severe disease
- The **public health objectives** of treatment are to:
 - prevent onward transmission of the infection to others
 - prevent the emergence and spread of resistance to antimalarial drugs

Treatment of Uncomplicated Malaria

General Population

- **1st line treatment options**
 - Artesunate Amodiaquine (AS-AQ)
 - Artemether Lumefantrine (A-L)
 - Artesunate- Pyronaridine (AP)
- **2nd line treatment option**
 - Dihydroartemisinin Piperaquine (DHAP)

Malaria Treatment Failure

- **Treatment failure (recrudescence)** operationally is the recurrence of asexual parasitemia within 4 weeks (28 days) after treatment often due to resistance of parasites to antimalarials or incomplete treatment.
- Treatment failure may be due to:
 - **Inadequate exposure** to the drug-
 - sub-optimal dosing
 - poor adherence
 - vomiting
 - Substandard medicines
 - Drug-drug interactions etc.
 - **True treatment failure (therapeutic failure)**- suspect drug resistance or unusual pharmacokinetics

Managing Malaria Treatment Failure

Remember: Need to prove treatment failure with Microscopy!! REFER

- *Look out for other causes of fever!!*
- **True treatment failure** (therapeutic failure):
 - Give **alternate 1st line ACT / 2nd line ACT** (i.e. DHAP)
- **Other forms of treatment failure** (i.e., vomiting, non-compliance, under dosing, poor quality medicine etc.):
 - Repeat **same ACT** (1st line) or
 - **Alternate 1st line ACT**

Reinfection

- **Reinfection** operationally, parasitemia occurring after 4 weeks (28 days) of previous treatment
- **Treatment:**
 - 1st Line: **ASAQ** or **AL** or **AP**

Treatment of P. Ovale, P. Vivax (Relapse Malaria)

- Treat with **ACTs + Primaquine**
 - To clear the hypnozoites in the liver
 - Primaquine dosage regimen depends on **G6PD status**
 - Primaquine is contra-indicated in:
 1. Infants less than 6 months old

Severe Malaria- Principles of Management

- Management of severe malaria comprises:
 - **Resuscitation** (Clinical assessment of the patient for urgent treatment of life- threatening problems)
 - Specific **antimalarial** treatment
 - Additional treatment for identified complication or co-morbidities
 - **Supportive** care
 - Counselling
 - **Prompt and appropriate referral and/or Follow-up** care

Initial assessment

- **Initial assessment** using the **ABCDE** approach
 - **Airway:** Secure the airway in an unconscious patient. Consider intubation
 - **Breathing:** Keep oxygen saturation >94%
 - **Circulation:** Assess for shock
 - **Disability** and Coma score
 - **Exposure:** Measure temperature, look at skin lesions and ensure safety
- Assess the patient for **clinical features of severe malaria**

Specific Antimalarial

- All patients with Severe Malaria Must be **ADMITTED** at a **HOSPITAL for PARENTERAL antimalarials**
- Parenteral antimalarials **MUST** be given for **at least 24 hours before switching to Oral medications** (recommended ACTs for 3 days)
- The Preferred order for antimalarials and their route of administration is:

- **IV Artesunate**

- **IM Artesunate**

- **Supp Artesunate (<25kg or <6years)**

- **IM Artemether**

- **IV Quinine infusion**

- **IM Quinine**

MANAGEMENT OF ACUTE COMPLICATIONS

Manifestation or complication	Immediate management ^a
Coma (cerebral malaria)	Maintain airway, place patient on his or her side, exclude other treatable causes of coma (e.g. hypoglycaemia, bacterial meningitis); avoid harmful ancillary treatments, intubate if necessary.
Hyperpyrexia	Administer tepid sponging, fanning, a cooling blanket and paracetamol.
Convulsions	Maintain airways; treat promptly with intravenous or rectal diazepam, lorazepam, midazolam or intramuscular paraldehyde. Check blood glucose.

Hypoglycaemia	Check blood glucose, correct hypoglycaemia and maintain with glucose-containing infusion. Although hypoglycaemia is defined as glucose < 2.2 mmol/L, the threshold for intervention is < 3 mmol/L for children < 5 years and <2.2 mmol/L for older children and adults.
Severe anaemia	Transfuse with screened fresh whole blood.
Acute pulmonary oedema	Prop patient up at an angle of 45°, give oxygen, give a diuretic, stop intravenous fluids, intubate and add positive end-expiratory pressure or continuous positive airway pressure in life-threatening hypoxaemia.
Acute kidney injury	Exclude pre-renal causes, check fluid balance and urinary sodium; if in established renal failure, add haemofiltration or haemodialysis, or, if not available, peritoneal dialysis.

Spontaneous bleeding and coagulopathy	Transfuse with screened fresh whole blood (cryoprecipitate, fresh frozen plasma and platelets, if available); give vitamin K injection.
Metabolic acidosis	Exclude or treat hypoglycaemia, hypovolaemia and septicaemia. If severe, add haemofiltration or haemodialysis.
Shock	Suspect septicaemia, take blood for cultures; give parenteral broad- spectrum antimicrobials, correct haemodynamic disturbances.

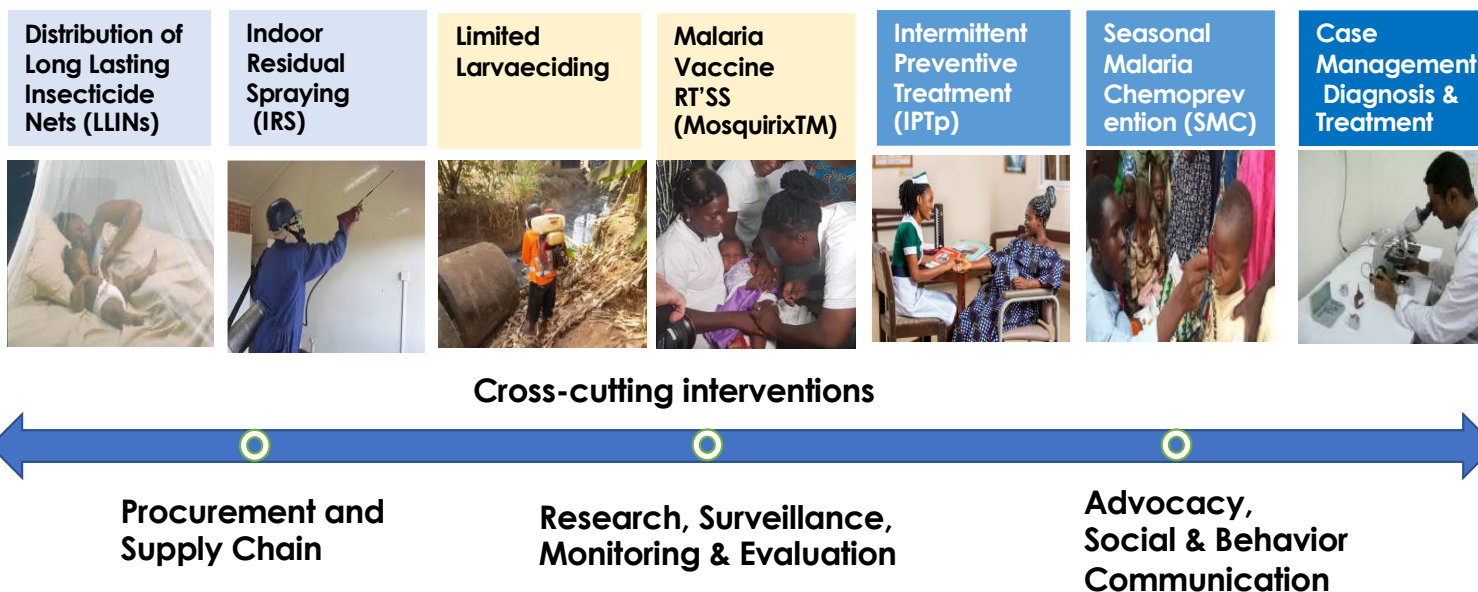
Supportive management

- Monitoring of vitals
- Blood glucose level
- Level of consciousness
- Fluid Intake and urine output
- Convulsions
- parasitemia

LONG TERM COMPLICATIONS

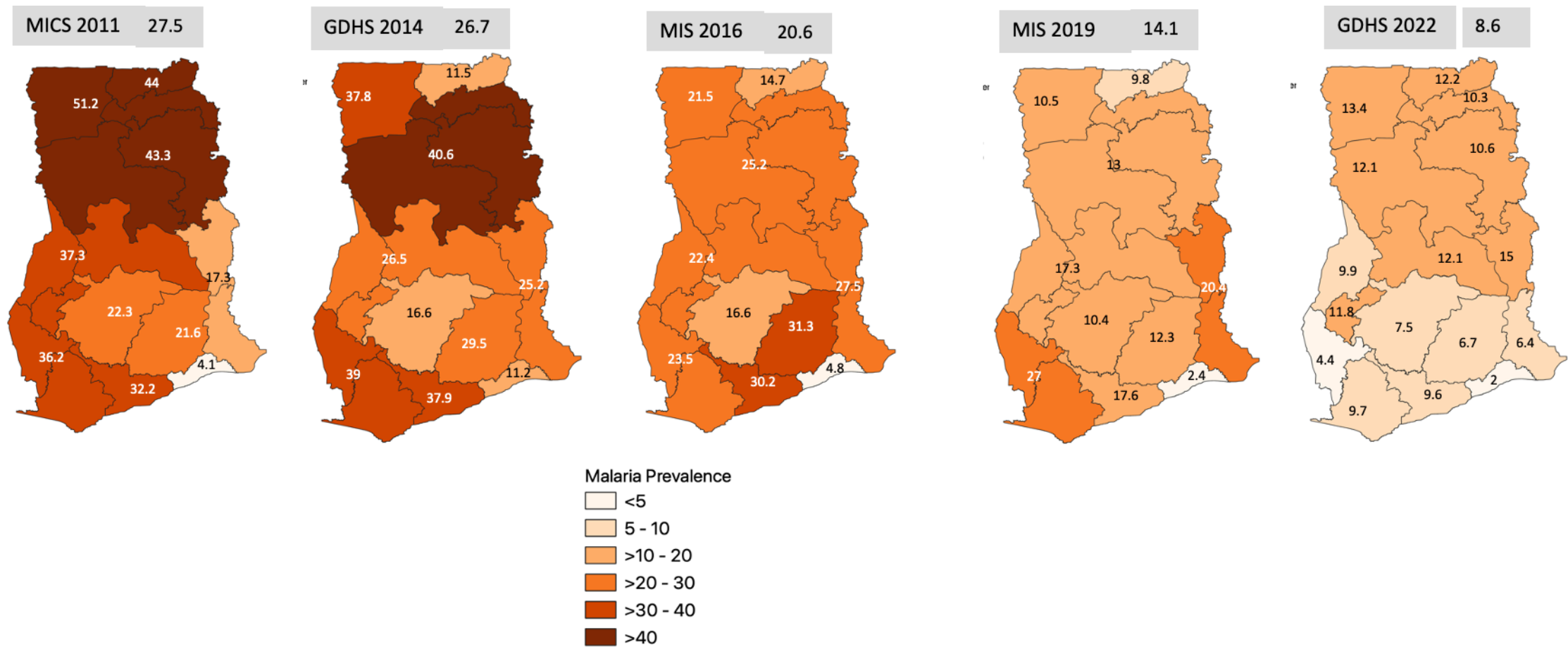
- **CNS impairment-** motor function, visual function, hearing and speech impairment, epilepsy
- **Late onset anemia;** delayed hemolysis (artesunate)
- **Recurrence;** recrudescence(persistence of merozoites that now become detectable), relapse(hypnozoites in p.Vivax and p. Ovale), reinfection
- **Nephrotic Syndrome**
- **Hyperreactive malarial splenomegaly**

Malaria Control Interventions in Ghana

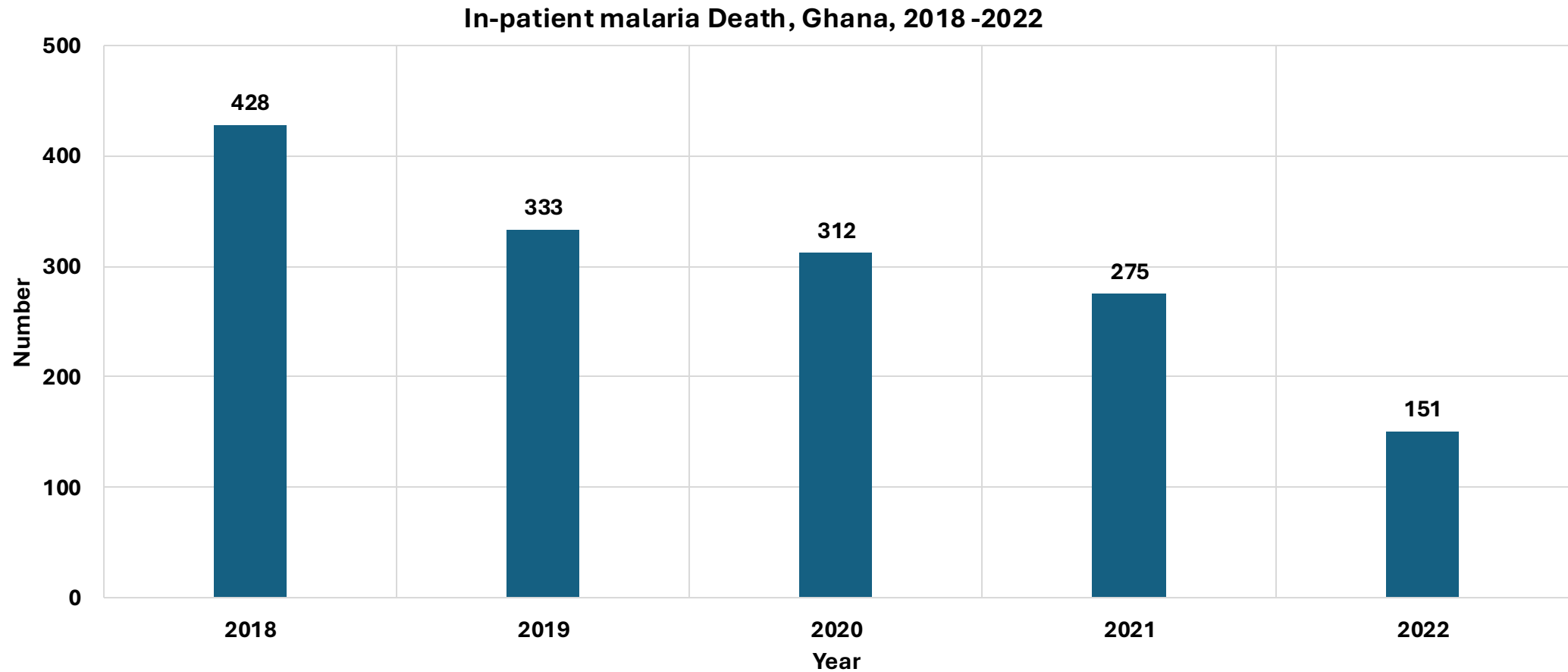


Progress with Malaria Control in Ghana (1)

Malaria Parasite Prevalence
Among Children 6-59months in
Ghana, 2011-2022



Progress with Malaria Control in Ghana (2)



MALARIA CHEMOPROPHYLAXIS FOR NON-IMMUNE TRAVELLERS 1/2

- Different drugs available
- Antimalarials should be started 1-21 days before arriving in the malaria-endemic area and continued for 7-28 days after leaving the area.
- Not 100% protective but it reduces the risk of severe disease
- If a person develops Malaria while on any malaria chemopreventive medicine, that particular drug should be excluded from their treatment regimen.

MALARIA CHEMOPROPHYLAXIS FOR NON-IMMUNE TRAVELLERS 2/2

DRUG	ADMINISTRATION	CONTRAINDICATIONS
Atovaquone-Proguanil	Taken daily with food Begin 1-2 days before exposure, during exposure and for 7 days following exposure	Pregnant women Infants weighing less than 5kg Women breastfeeding infants weighing less than 5kg
Doxycycline	Taken daily with food or fluids Begin 1-2 days before exposure, during exposure and for 7 days following exposure NB:Do not lie down immediately (within 30 minutes) of taking Doxycycline to prevent heartburns (esophagitis)	Children less than 8 years Pregnant and breastfeeding women
Mefloquine	Taken weekly beginning at least 2 weeks before exposure, during exposure and for 4 weeks post exposure Safe in pregnancy and puerperium	Known hypersensitivity to Mefloquine History of seizures Major psychiatric disorder

MALARIA VACCINE

- As of October 2023, both the RTS,S/AS01 and R21/Matrix-M vaccines are recommended by WHO to prevent malaria and significantly reduce life-threatening severe disease in children.
- The RTS,S malaria vaccine was first recommended by WHO to prevent malaria in children in October 2021. The vaccine reached more than 2 million children in Ghana, Kenya and Malawi through the Malaria Vaccine Implementation Programme (MVIP) from 2019 to 2023.
- Recommended for use in children under 5 years living in Malaria-endemic settings.
- Both vaccines reduce malaria cases by 75% when given seasonally in areas of highly seasonal transmission where seasonal malaria chemoprevention is provided.
- Given as an intramuscular (IM) injection. 4 doses required given at 6, 7, 9 and 18 months of age.

Special considerations for RTS,S Malaria vaccine

Children who are unable to receive their dose of the vaccine at these scheduled times should be given the vaccine soon thereafter.

Vaccine catch up for dose 1 is to be given at age 6-11 months and that for dose 4 at age 18-59 months.

CONGENITAL AND NEONATAL MALARIA (CNM) 1/2

- Neonatal malaria is defined as the presence of a positive parasitaemia with asexual forms of plasmodium associated with at least one symptom (fever, jaundice, anaemia, splenomegaly, vomiting, hepatomegaly, diarrhoea, restlessness, drowsiness, convulsions, poor feeding, cyanosis, pallor, respiratory distress) in a child less than or equal to 28 days
- These same symptoms occurring in a neonate up to 7 days is considered as congenital malaria
- Prevalence in Africa and outside Africa – 39.5% and 56.3% respectively

CONGENITAL AND NEONATAL MALARIA (CNM) 2/2

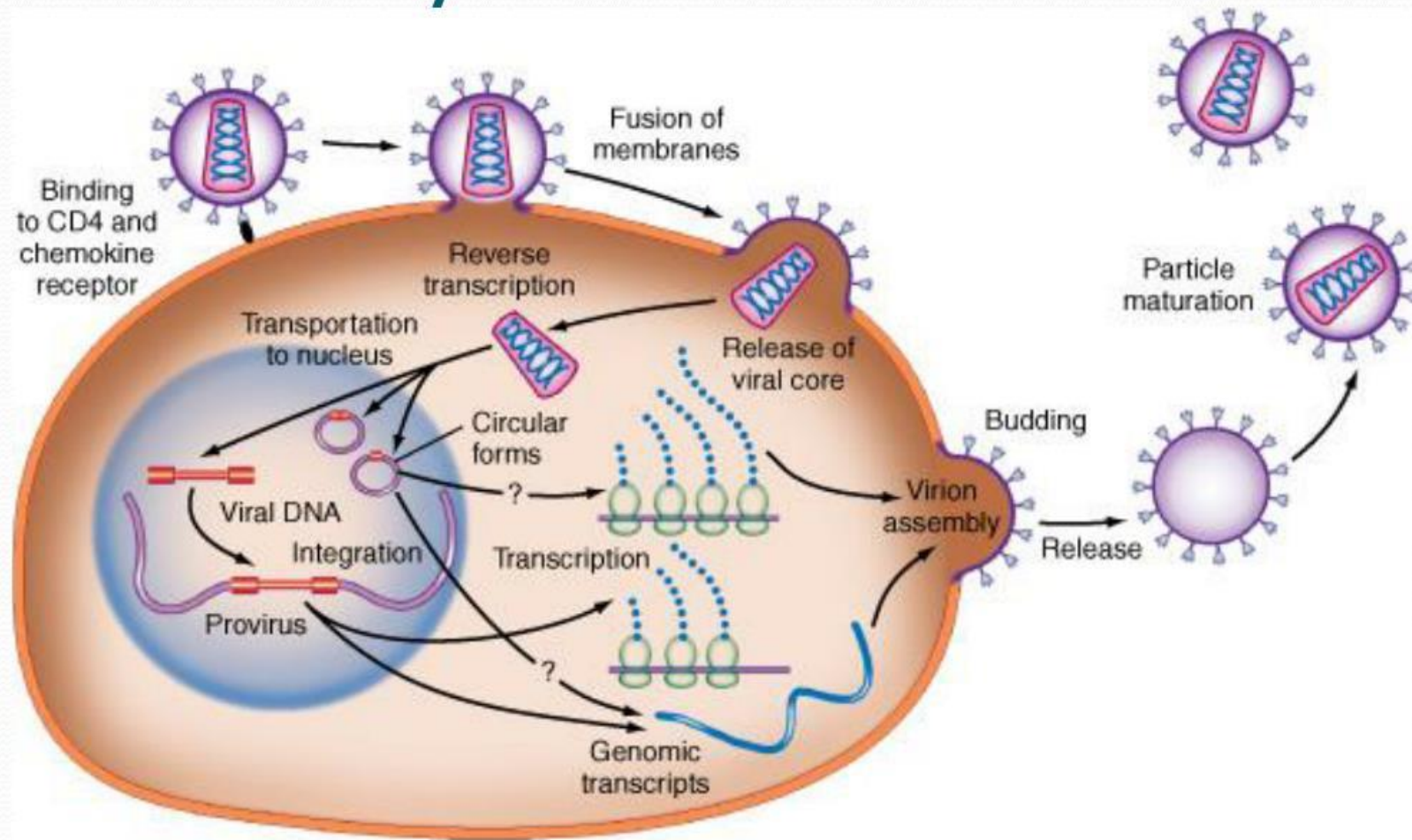
- Congenital malaria arises from transplacental transmission of Plasmodium, primarily Plasmodium falciparum, during pregnancy or delivery, leading to significant neonatal health consequences
- Neonatal Malaria results from an infective mosquito bite after birth.
- For infants weighing less than 5 kg with uncomplicated P. falciparum, the World Health Organization (WHO) recommends treatment with ACT at the same mg/ kg body weight dose as for children weighing 5 kg.
- Due to the physiological immaturity and rapid changes that occur in neonates, the pharmacokinetic and dynamic (PK/PD) profiles of anti-malarial drugs are likely to be different to older children. Slow gastric emptying, villous formation and intestinal motor activity, which do not mature until week 20 of life, affect the enteral absorption of most medications. Hence, parenteral treatment is preferable for neonates and young infants.

UPDATES IN PAEDIATRIC RETROVIRAL INFECTION

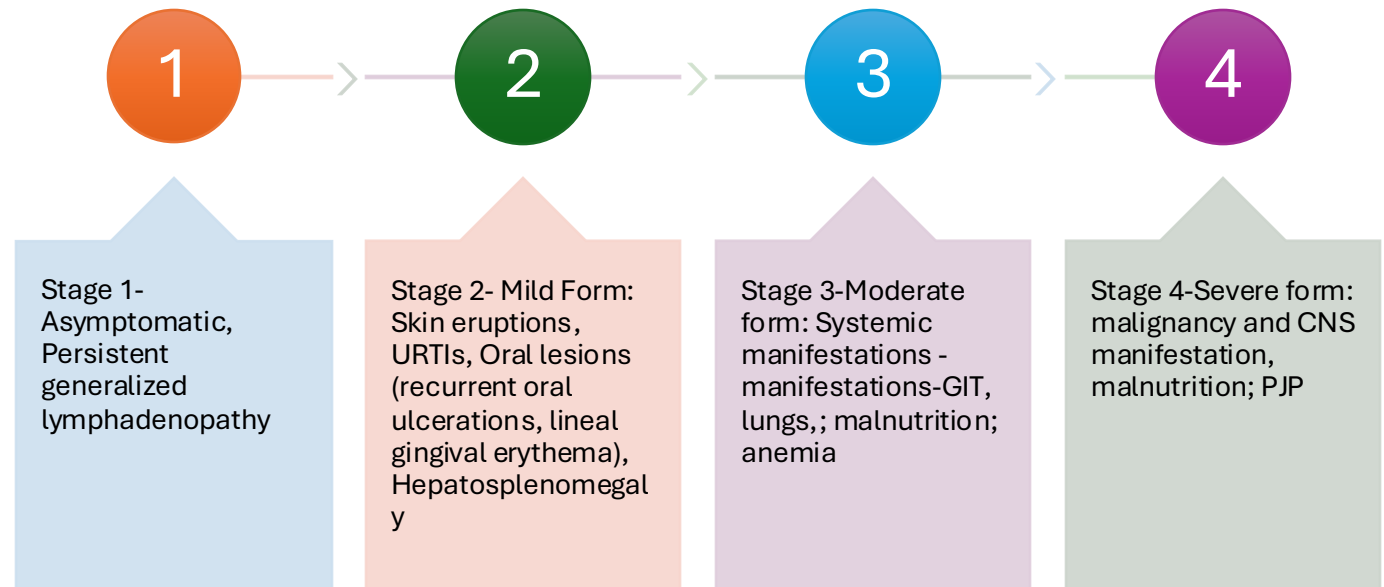
Introduction

- ❖ HIV is a retrovirus.
- ❖ Structurally, a lipid bilayer envelope surrounds the cylindrical core of HIV, which contains the RNA genetic information and the machinery that promotes viral replication and integration during initial cellular infection.
- ❖ Mother- to child transmission of HIV is the most common mode of transmission in children.
- ❖ Approx 1.5 million children living with HIV in 2022 (WHO global report).
- ❖ Prevalence in Ghana is low.

HIV life cycle



Clinical Manifestation



Investigations

Three step diagnostic algorithm:

First response HIV 1 and 2

Oraquick HIV 1 and 2

SD Bioline HIV 1 and 2

Point-of-care nucleic acid testing to diagnose HIV among infants and children younger than 18 months of age.

CLASSES OF ARVS

Class	Mechanism of Action	Examples
NRTIs (Nucleoside/Nucleotide Reverse Transcriptase Inhibitors)	Block reverse transcription of viral RNA into DNA	Abacavir, Lamivudine, Zidovudine, Tenofovir, Emtricitabine
NNRTIs (Non-Nucleoside Reverse Transcriptase Inhibitors)	Bind directly to reverse transcriptase and inhibit its function	Nevirapine, Efavirenz, Rilpivirine, Etravirine
PIs (Protease Inhibitors)	Prevent viral protein processing, producing immature virus	Lopinavir/ritonavir, Atazanavir, Darunavir
INSTIs (Integrase Strand Transfer Inhibitors)	Block integration of viral DNA into host genome	Dolutegravir, Raltegravir, Bictegravir
Entry Inhibitors	Prevent HIV from entering host cells	Maraviroc (CCR5 antagonist), Enfuvirtide (fusion inhibitor)
Boosters	Enhance levels of other ARVs by slowing metabolism	Ritonavir, Cobicistat

ARTS AND THEIR EFFECT ON HIV PATHOPHYSIOLOGY

HIV Stage	Pathophysiological Role	ART Class That Acts Here	Examples
Attachment & Entry	HIV binds to CD4 and co-receptors (CCR5/CXCR4)	Entry inhibitors	Maraviroc (CCR5 antagonist), Enfuvirtide (fusion inhibitor)
Reverse Transcription	Viral RNA is converted to DNA	NRTIs & NNRTIs	Zidovudine, Lamivudine, Efavirenz
Integration	Viral DNA integrates into host genome	Integrase inhibitors (INSTIs)	Dolutegravir, Raltegravir
Transcription & Translation	Host cell produces viral proteins	No direct ART class targets this step	—
Assembly & Maturation	New virions are assembled and matured	Protease inhibitors (PIs)	Lopinavir/ritonavir, Darunavir

Algorithm for early infant prophylaxis and diagnosis

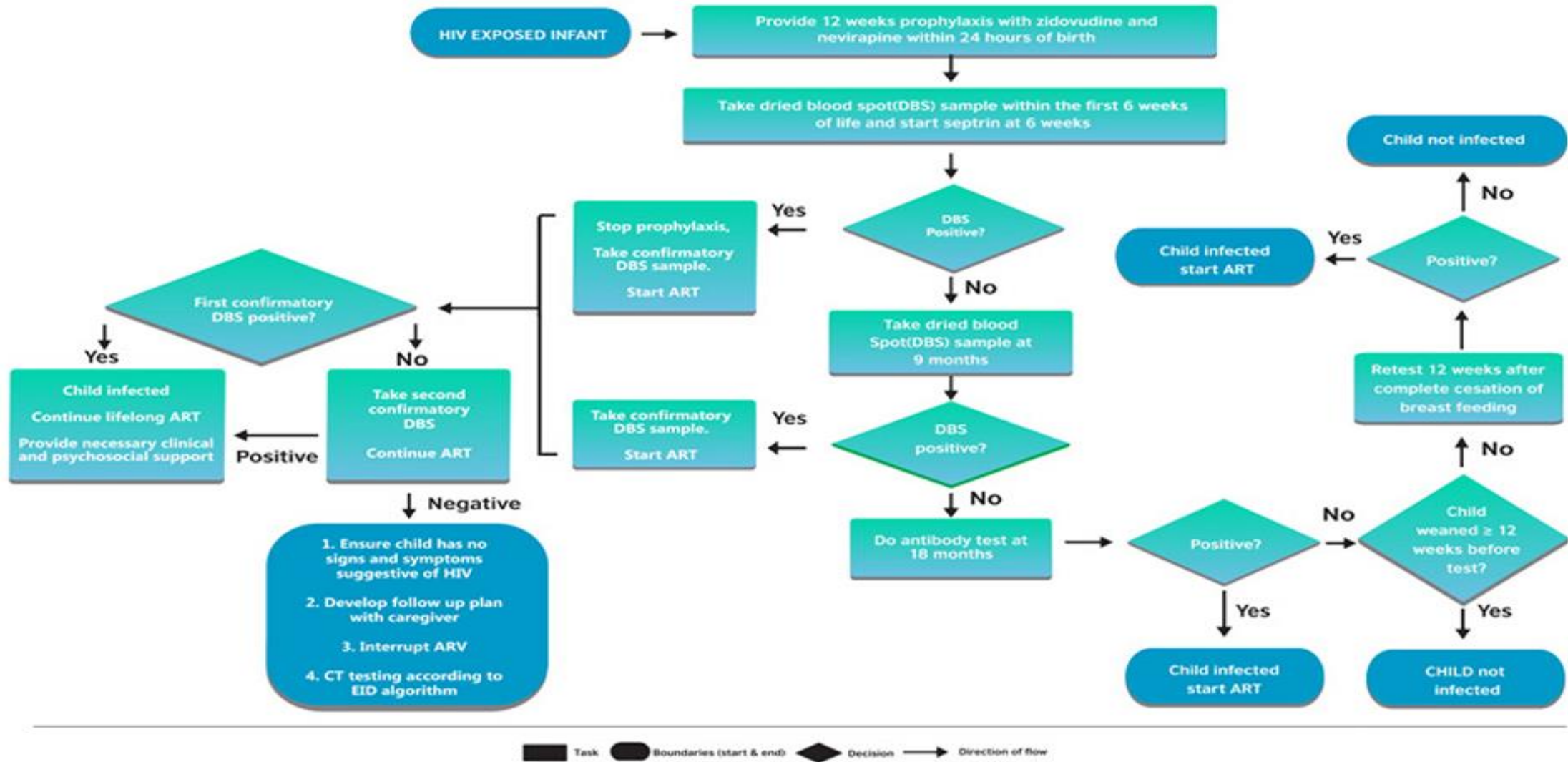
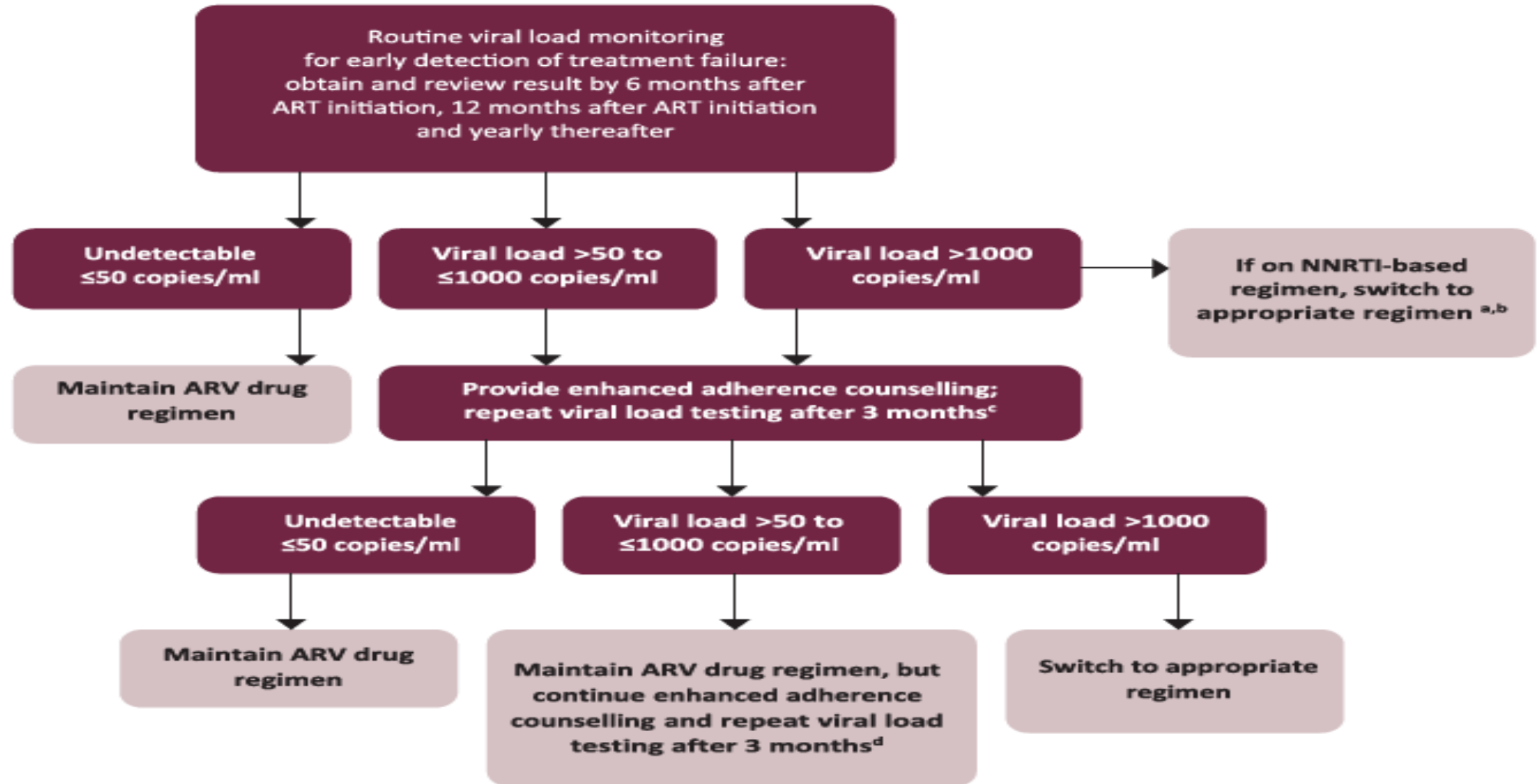


Figure 5.2
Algorithm for Early infant prophylaxis and diagnosis

Algorithm for treatment monitoring



Signs of treatment success

- ❖ At least one suppressed viral load result within the past six months (if viral load is not available: CD4 count >200 cells/mm³ or CD4 count >350 cells/mm³ for children 3-5 years)
- ❖ Weight gain
- ❖ Absence of symptoms and concurrent infections.

Signs of treatment failure

- ❖ A lack of growth among children who show an initial growth response to therapy
 - ❖ A loss of neurodevelopment milestones
 - ❖ Development of encephalopathy.
 - ❖ Recurrence of infections, such as oral candidiasis refractory to treatment.
-
- Before an ARV regimen is thought to be failing, based on clinical criteria, the child should have had a reasonable time on the ART (i.e. must have received the ART for at least 6months).
 - A switch to a second line regimen is recommended when virological failure is recognized.

New drug 'on the block'



Dolute-gravir	Dis-persible Tablet: 10mg	3-<6 kg, 5mg once daily 6-<10 kg, 15mg once daily 10-<14 kg, 20mg once daily 14-<20 kg, 25mg once daily	Insomnia, weight gain/ clinical obesity	Neural tube defects, depression and suicide ideation	
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A sneak peek at Dolutegravir

- ❖ DTG-containing regimens are preferred for all patients, including those on TB treatment.
- ❖ All ART naïve patients and patients re-initiating ART after previously interrupting ART should be initiated on a DTG-containing regimen.
- ❖ Patients on ART who have not yet been transitioned to a DTG-containing regimen should be evaluated and transitioned as a matter of urgency.

DTG EFFECT ON OTHER MEDICATIONS

Drug/Class	Effect on Dolutegravir or Vice Versa	Clinical Action
Rifampicin (used for TB)	↓ DTG levels (induces UGT1A1/CYP3A4)	Double DTG dose: 50 mg twice daily
Metformin	↑ Metformin levels (DTG inhibits OCT2/MATE1)	Limit metformin dose to <1000 mg/day and monitor renal function
Antacids / Calcium / Iron supplements	↓ DTG absorption (chelation)	Separate doses: DTG 2 hrs before or 6 hrs after
Carbamazepine, phenytoin, phenobarbital	↓ DTG levels (enzyme induction)	Avoid or increase DTG dose to 50 mg BID

What is trending?

- ❖ Long acting injectibles-**Cabotegravir + Rilpivirine**: Approved for adolescents aged 12–18 years as a **bi-monthly injection**, offering an alternative to daily pills
- ❖ New second line regimen-**Tenofovir alafenamide (TAF) + Dolutegravir (DTG)**
- ❖ Child friendly formulations-**Dispersible DTG/ABC/3TC tablets** now available for children ≥ 6 kg
- ❖ Single tablet-**Bictegravir/TAF/FTC** approved for children ≥ 2 years and ≥ 14 kg
- ❖ Personalized HIV care-**Pharmacogenomics** with the help of AI to guide individualized treatment plans based on genetic profiles

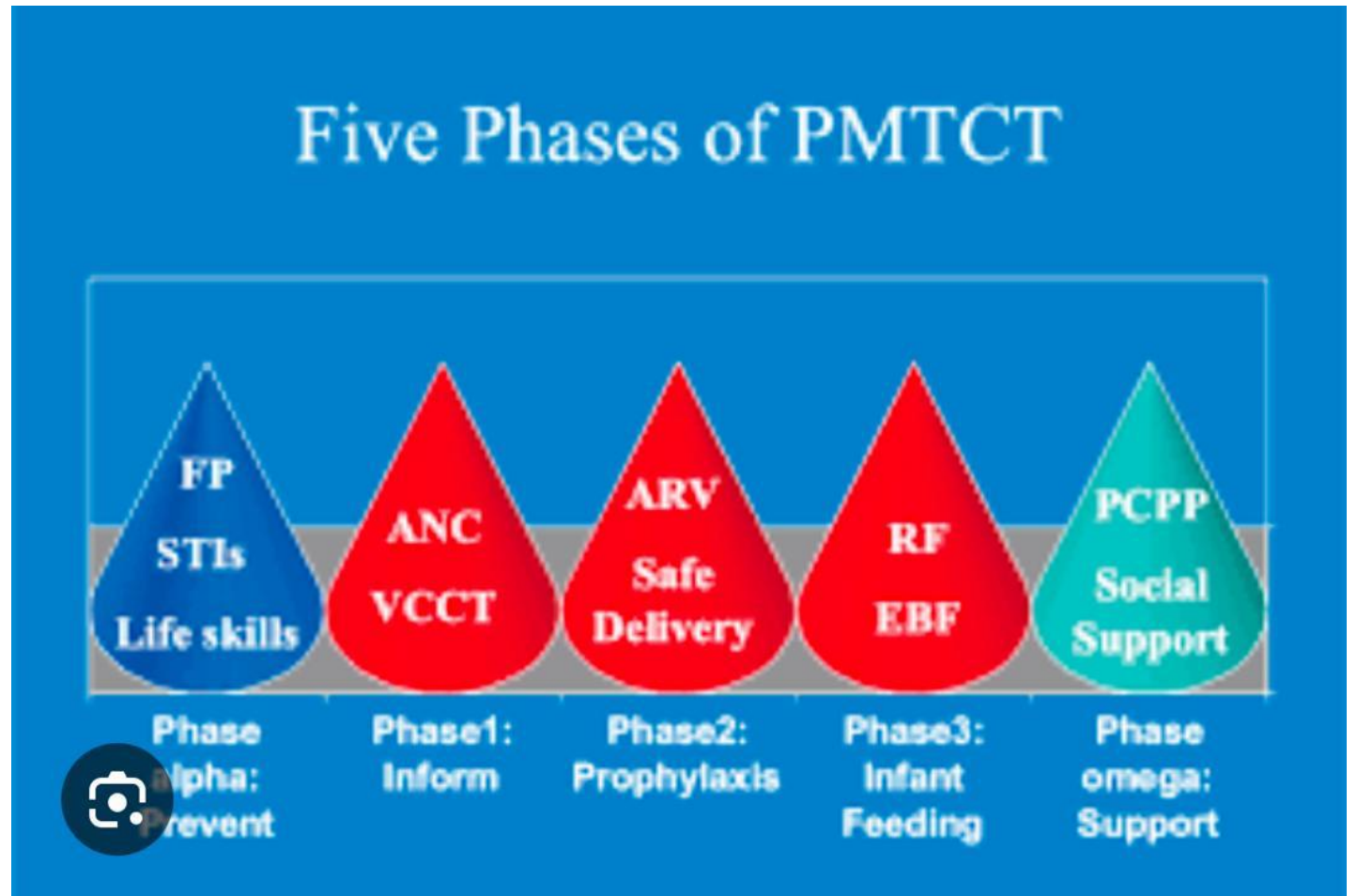
Lenacapavir

- ❖ Class: Capsid inhibitor
- ❖ Dosing: Only two injections per year
- ❖ Pre-exposure prophylaxis (PrEP): WHO now recommends LEN as a long-acting option for HIV prevention
- ❖ Treatment: Being studied as part of long-acting ART regimens, especially for people with multidrug resistance or poor adherence
- ❖ Early Adopter Countries: Kenya and South Africa among the first to roll out LEN nationally

Therapy

- ❖ In February 2025, the FDA expanded the indication for a three-drug combination of emtricitabine (FTC) and tenofovir alafenamide (TAF), both HIV nucleoside analog reverse transcriptase inhibitors (NRTIs), and rilpivirine (RPV), a non-nucleoside reverse transcriptase inhibitor (NNRTI), to include pediatric patients weighing at least 25kg. The indication for children is as follows:
- ❖ As initial therapy in those with no antiretroviral treatment history with HIV-1 RNA less than or equal to 100,000 copies/mL; or
- ❖ To replace a stable antiretroviral regimen in those who are virologically-suppressed (HIV-1 RNA less than 50 copies/mL) for at least 6 months with no history of treatment failure and no known substitutions associated with resistance to the individual components of ODEFSEY.

PREVENTION OF MOTHER TO CHILD TRANSMISSION



CONCLUSION

Malaria and Retroviral infection contribute significantly to paediatric mortality and morbidity especially in the African population.

Clinical development of non-artemisinin based combination therapies underway in clinical trials (shown very positive results) for both severe and uncomplicated Malaria; as well as improved dosing with single doses to improve compliance.

Great strides are being made in the management of Malaria and Retroviral infection.

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THANK
YOU