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### EVALUATION OF MALARIA TREATMENT AMONG PEOPLE LIVING WITH HIV

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#### ABSTRACT

An evaluation study was conducted on treatment of malaria among people living with HIV, reported cases obtained from United Nation Agency for International Development and Studies (UNAIDS) and World Health Organization (WHO) were used in the research work. It was observed that Malaria in people with HIV has recorded more than 4 million deaths yearly. The treatment of malaria for people living with HIV infection showed a synergistical interaction with each other. Patients with HIV infection are at high risk of an increase in the severity and burdens malaria infection which in turn facilitates the rate of malaria transmission. Malaria increases blood viral burden by different mechanisms. Therefore, high concentrations of HIV-1 RNA in the blood are predictive of disease progression, and correlate with the risk of blood-borne, vertical, and sexual transmission of the HIV virus. Furthermore, Malaria infection is also associated with strong CD4<sup>+</sup> cell activation and up-regulation of pro inflammatory cytokines and it provides an ideal microenvironment for the spread of the virus among the CD4<sup>+</sup> cells and for rapid HIV-1 replication. Therefore, this article aims to review information about HIV/malaria interactions, the effect of malaria on HIV transmission, treatment and the implications related to prevention of the disease.

**Keywords:** Human Immunodeficiency Virus, Transmission, Evaluation, Malaria, Treatment

#### INTRODUCTION

The diseases HIV and Malaria are among the two most important global health problems of developing nations, Nigeria inclusive (WHO, 2022). They cause more than 4 million deaths a year (Ariyoshi *et al.*, 2019). Malaria, sometimes called the “king of diseases”, is caused by protozoan parasites of the genus Plasmodium. The proportion increases each year because of deteriorating health systems, growing drug and insecticide resistance, climate change and atural disasters (Ayisi *et al.*, 2003). It is one of the leading causes of illness and death in the world. Nine out of ten of these deaths occur in Africa and the



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rest occurs in Asia and Latin America, being the world's most prevalent vector-borne disease. It is the fourth leading cause of death of children under the age of five years and pregnant women in developing countries (Ayoub *et al.*, 2008). HIV/AIDS is also one of the most destructive epidemics the world has ever witnessed. In 2007 an estimated 33.2 million people were living with HIV worldwide, while 2.5 million of these people were children under 15 years old. Furthermore, 420,000 children under 15 years were newly infected with HIV in 2007. Nearly 90% of all HIV-positive children live in sub-Saharan Africa (WHO, 2020).

In Ethiopia, while 66% of the population is at risk of malaria, 1.5 million people are infected with HIV (Francesconi *et al.*, 2001). In addition to this increased prevalence in developing countries Malaria and HIV/AIDS overlap geographically, primarily in sub-Saharan Africa, Southeast Asia and South America. While infection with either malaria or HIV/AIDS can cause illness and death, infection with one can make infection with the other worse and/or more difficult to treat (Bentwich *et al.*, 2012). The two diseases have particularly devastating effects for those living in malaria endemic regions throughout the world (Donovan *et al.*, 2000). Pregnant women suffer particularly serious consequences when infected with both HIV/AIDS and malaria. Thus, estimated number of malaria deaths stood at 409 000 in 2019. Children aged under 5 years are the most vulnerable group affected by malaria; in 2019, they accounted for 67% (274 000) of all malaria deaths worldwide (WHO, 2021). HIV/AIDS can increase the adverse effects of malaria, including anemia and placental malaria infection (Ariyoshi *et al.*, 2019). These two infections interact directionally and synergistically with each other. HIV infection can increase the risk and severity of malaria infection and the increased parasite burdens might facilitate higher rates of malaria transmission. Individuals in malaria-endemic areas that are considered semi-immune to malaria can also develop clinical malaria if they are infected with HIV. Also Malaria is a life-threatening disease caused by



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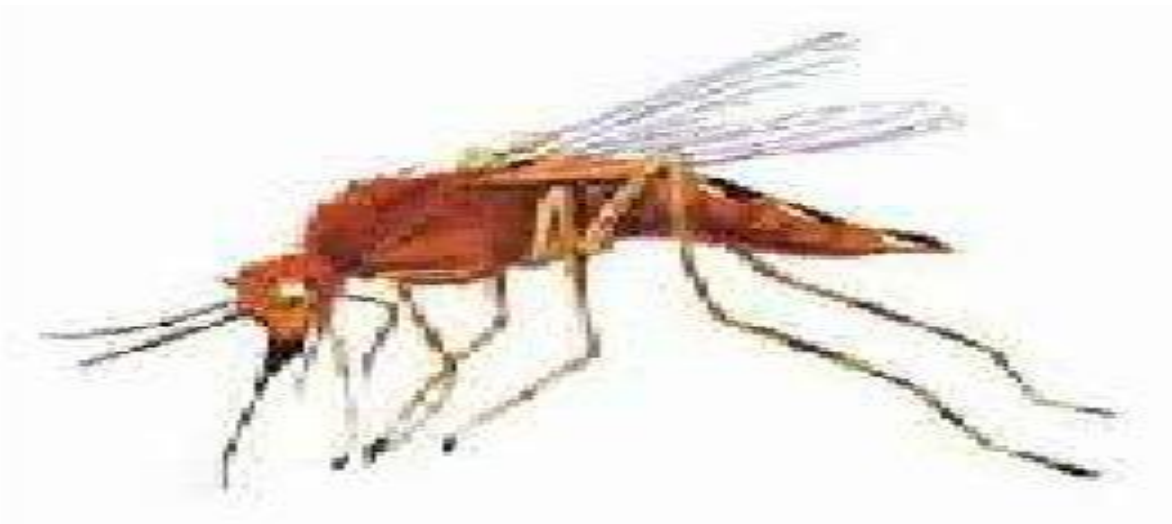
parasites that are transmitted to people through the bites of infected female *Anopheles* mosquitoes. It is preventable and curable. In 2019, there were an estimated 229 million cases of malaria worldwide (Ayoubu *et al.*, 2008).

### **Epidemiology of Malaria**

According to (WHO, 2017), an estimated 200 million cases, or 92% of global cases. In 2017, five countries accounted for nearly half of all malaria cases worldwide. Four of these were in Africa: Nigeria (25%), the Democratic Republic of the Congo (11%), Mozambique (5%), and Uganda (4%). Africa is the region most affected by malaria in the world. Around 170.9 million cases of the disease were reported in the continent in 2019. From a country perspective, Nigeria registered the highest number of cases, around 23.4 million, followed by the Democratic Republic of the Congo, with 20.5 million cases (Ariyoshi *et al.*, 2019). However, the estimated number of cases decreased by 7.9%, between 2015 and 2019 (from 370 per 1000 population to 343 per 1000 of the population at risk) and the number of deaths decreased by 25.9% in the same period (from 0.919 per 1000 population to 0.730 per 1000 of the population at risk) (Corbett *et al.*, 2000). On like many other parts of Nigeria, malaria is endemic in Niger State, with year-round transmission. In 2010, 45% of febrile under-fives tested in the North Central zone were RDT-positive (Wilson *et al.*, 2006). Malaria constituted the highest proportion of childhood febrile illnesses presenting to health facilities.

### **Causes of Malaria**

Malaria is an acute febrile illness caused by *Plasmodium* sp, which are spread to people through the bites of infected female *Anopheles* mosquitoes (WHO, 2014). Malaria is a serious disease that spreads when an infected mosquito bites human, there are many factors that can cause malaria such as: Bitten by a malarial vector (*Anopheles stephensi*), Use of shared and infected syringes, Organ transplantation, transfusion and from an infected mother to her baby during birth (Ariyoshi *et al.*, 2019).



**A female anopheles mosquito**

### **CAUSATIVE AGENTS OF MALARIA**

Malaria is a mosquito-borne infectious disease caused by various species of the parasitic protozoan microorganisms called Plasmodium (Ayoub *et al.*, 2008).

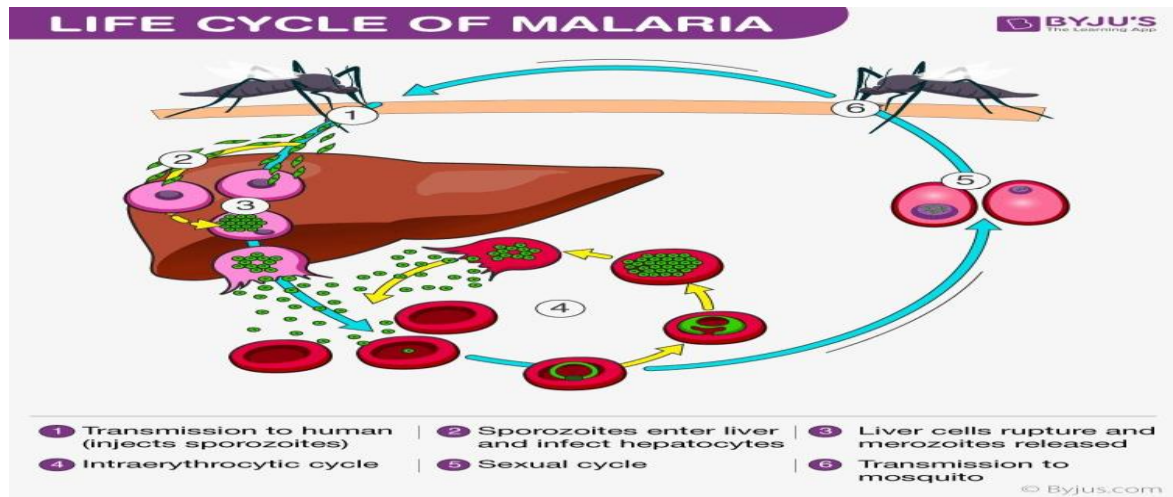
### **Pathogenesis of Malaria**

After a mosquito takes a blood meal, the malarial sporozoites enter hepatocytes (liver phase) within minutes and then emerge in the bloodstream after a few weeks (Ayoub *et al.*, 2008). These merozoites rapidly enter erythrocytes, where they develop into trophozoites and then into schizonts over a period of days (during the erythrocytic phase of the life cycle). Rupture of infected erythrocytes containing the schizont results in fever and merozoite release (Wilson *et al.*, 2006). The merozoites enter new red cells, and the process is repeated, resulting in a logarithmic increase in parasite burden (WHO, 2021).

### **Malaria Life Cycle**

Malaria parasite exists in the form of a motile sporozoite. The vector of malaria i.e. the female Anopheles mosquito transmits the malarial sporozoites into the hosts. When an infected mosquito bites a human, the sporozoites are injected into the blood

through the mosquito's saliva. The sporozoites travel into our body and accumulate in the liver (Bentwich *et al.*, 2012). These parasites initially multiply within the liver, by damaging the liver and rupturing the blood cells in the body. Malaria kills by causing the destruction of the red blood cells in the host (Toure-Bolde *et al.*, 2016). The parasites reproduce asexually in the RBCs, bursting the cells and releasing more parasites to infect more cells. The rupture of red blood cells by the malaria parasite releases a toxin called hemozoin which causes the patient to experience a condition known as the chills (Ariyoshi *et al.*, 2019).



### Mode of Transmission

**Mother to the growing fetus (Congenital malaria):** Transfer of parasitized red cells from infected mother to the child either transplacentally or during labor can lead to malaria in the newborn, called as congenital malaria (Ticconi *et al.*, 2003). Congenital malaria seems to be rarely reported and has always been considered to be more frequent in the no immune population than in the endemic areas. In recent years, however, higher prevalence of congenital malaria ranging from 8% to 33% has been reported from both malaria-endemic and non endemic areas, including the United States, Europe, India, etc (WHO,



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2017). Congenital malaria has been reported due to all four plasmodium species that commonly infect humans, although most cases are reported following *P. falciparum* or *P. vivax* malaria in the mother. In non endemic countries, *P. malariae* may cause a disproportionately higher number of congenital malaria cases due to its longer persistence in the host. Congenital malaria occurs more often during the first pregnancy.

**Transfusion Malaria:** Malaria can be transmitted by transfusion of blood from infected donors. First reported in 191, the risk of acquiring transfusion malaria is very low (1 case per 4 million) in non-endemic countries such as the United States, whereas in the endemic countries, it is much higher (>50 cases per million donor units). Following a malaria infection, the individual may remain infective for weeks to months, or even years, in case of *P. malariae* infection (Bentwich et al., 2012). Therefore, those who have suffered from malaria should not donate blood for at least 3 years after becoming asymptomatic and proven carriers of *P. malariae* should never donate blood (Ariyoshi et al., 2019). The risk of transmission is higher in transfusion of fresh, whole blood, particularly when the blood has been stored for less than 5 days and the risk is considerably lesser after 2 weeks. Most donors implicated in transfusion-transmitted malaria are predominantly semi-immune with very low parasite loads and the infectious dose is estimated to be 1 to 10 parasites in a unit of blood (Ayoub et al., 2008).

**Needle stick injury:** Cases of malaria transmission through needle-stick injuries, accidentally among health care professionals (some even fatal) or due to needle sharing among drug addicts, have also been reported (Ter et al., 2004).

### MECHANISMS OF HOW MALARIA AFFECTS HIV TREATMENT

#### **Immunological Mechanisms**

Scientific speculation has it those malaria-induced changes in viral load result from “a series of immunological mechanisms” (Fana et al., 2011). For example, *P. falciparum*



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infection might promote macrophages and CD4<sup>+</sup> cells to activate viral transcription. A high parasite density likely elicits a strong immune response, leading to a high turnover of HIV-1 RNA and fever indicates a cytokine response that might raise HIV-1 RNA concentrations. A number of studies suggest more effective sexual transmission of HIV and an accelerated progression to clinical disease is seen in people with elevated concentrations of HIV-1 RNA in the blood (Berger et al., 2019).

Many inter current infections and even immunizations can lead to a short-term increase in plasma viral load in HIV-infected people, often accompanied by a fall in the CD4<sup>+</sup> cell count (Corbett et al., 2000). This has been interpreted as being due to the activation of T cells by the cytokines generated in response to the infecting organisms, which in turn increases the population of susceptible cells for HIV replication. In vitro studies have demonstrated that exposure of HIV-infected T-cell cultures to malarial antigens leads to significantly enhanced viral replication. Increased HIV replication was associated with high levels of cellular proliferation and activation and could be blocked by antibodies to TNF- $\alpha$  (Greenberg et al., 1995). The massive out-pouring of TNF- $\alpha$  and other pro inflammatory cytokines in response to acute malaria infection, together with the activation of CD4<sup>+</sup> cells, would be expected to generate ideal conditions for HIV replication, especially as TNF- $\alpha$  can act directly on the HIV long terminal repeat to up-regulate viral replication.

Additionally, HIV replication in CD4<sup>+</sup> cells requires that cells express an activated phenotype and inter current infections that induce immune activation have been found to increase HIV disease progression (Spina et al., 2006). In large areas of the tropics, HIV and malaria coexist with considerable potential for co-infection (Berger et al., 2019). In non-immune individuals, malaria infection leads to a pro-inflammatory (T helper 1- type) immune response with activation of CD4<sup>+</sup>, CD4<sup>+</sup> + 5RO + T cells to produce interferon gamma (IFN- $\gamma$ ). T cell-derived signals synergize with parasite-derived stimuli to activate



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monocytes and macrophages to produce interleukin-1 (IL-1) and IFN- $\gamma$ . As the CD4+ CD4+ 5RO+ T cell subset is a preferred target for HIV replication, these would seem to be ideal conditions for rendering individuals susceptible to HIV infection and for re-activation of viral replication in infected individuals, raising the possibility that malaria infection may lead to more rapid progression to AIDS and death (Corbett et al., 2000).

*P. falciparum* malaria causes increased CD4+ cell activation, increasing the number of susceptible target cells for HIV-1 infection, potentially resulting in increased HIV-1 RNA levels and continued new infection of susceptible activated CD4+ cells. CD4+ cell activation associated with malaria may persist despite therapy. The fact that malaria subjects with the highest CD4+ cell percentages at enrollment experienced the largest RNA declines is also consistent with the hypothesis that CD4+ cell activation is partly responsible for raised HIV RNA levels. Alternatively, malaria patients with higher CD4+ cell percentages may be better able to control either infection, resulting in a more rapid decline in RNA (Harms et al., 2002). More than 95% of HIV-1 in the plasma is produced by infected-activated CD4+ lymphocytes (Harms et al., 2002). The elevated RNA levels observed for 4 weeks after therapy are not therefore surprising.

High parasite density is likely to elicit strong immune activation, leading to a high turnover of HIV-1 RNA (Richman et al., 1997). Increases in HIV-1 RNA related to malaria might be mediated through a series of immunological mechanisms. *P. falciparum* infection might promote macrophages and CD4+ cells to activate viral transcription. These mechanisms could explain the differences observed between the groups classified according to parasitaemia, fever, and CD4+ count in some studies. Fever indicates a cytokine response that might increase concentrations of HIV-1 RNA. The modest increase in HIV-1 RNA concentration among people with lower CD4+ counts and higher concentrations HIV-1 RNA at baseline might indicate poor CD4+ cell function, which restricts the otherwise normal response to the cytokine stimuli of





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malaria, leading to a reduced increase in HIV-1 RNA turnover (Ariyoshi et al., 2019).

However, it was reported that short stimulations with *P. falciparum* antigens down-regulated CCR5 but not CXCR4 (Hoffman et al., 1999). Conflicting results have been obtained from in vitro studies (Mapbase et al., 2004). Firstly, an increase in HIV replication in blood mononuclear cells exposed to either malaria antigens or malaria pigment has been observed, and this was shown to be associated with increased production of TNF- $\alpha$ . However, longer stimulation up-regulated CCR5 through the induction of IFN- $\gamma$  and this IFN- $\gamma$  production blocked HIV replication (Riley 2019). More recently, mononuclear cell activation by malaria antigens was shown to render these cells more susceptible to HIV infection and to reactivate replication of endogenous HIV in cells from HIV-infected adults. Differences in viral strain, host cell type, and culture conditions might account for these conflicting reports (Berger et al., 2019).

Moreover, in a recent report using rodent malaria, DCs from infected animals were shown have impaired cross-presentation activity (Chandramohan et al., 2018). It is therefore possible that this inhibitory effect might influence the establishment and/or maintenance of HIV anti-immunity (Perelson et al., 1997).

### **SIGNS AND SYMPTOMS OF MALARIA**

A high temperature of 38 C or above.

- Feeling hot and shivery
- Fever, fatigue, chills, vomiting, and headaches
- Diarrhea, anemia and muscle pain
- Profuse sweating and convulsions
- Bloody stools
- In severe cases, malaria can be devastating; it can lead to seizures, coma and *eventually death* ( *Djabanor et al., 2017*)



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### **Treatment of Malaria/HIV**

Treatment for malaria should start as soon as possible. To treat malaria, your provider will prescribe drugs to kill the malaria parasite. Some parasites are resistant to malaria drugs. The type of medication and length of treatment depend on which parasite is causing your symptoms. Antimalarial drugs include:

- Artemisinin drugs (artemether and artesunate)
- Atovaquone (Mepron).
- Chloroquine.
- Doxycycline
- Mefloquine.
- Quinine.
- HIV treatment involves taking of drugs that reduces the amount of HIV in our body
- Antiretroviral therapy (ART)
- There is no effective cure for HIV but with proper medical care we can control HIV (Djabanor et al., 2017)

### **Prevention and Control of Malaria**

- When it comes to controlling the disease, keep an eye out for the symptoms like fever with high temperature. As soon as you find any possible signs of malaria, consult your doctor immediately
- Currently there is no over-the-counter medication available for malaria. So the only way you can treat this disease is by taking all the necessary precautions and safety measures
- Wear full sleeve protective clothing
- Make sure you don't keep your windows and doors open at night as mosquitoes get active during the night and pose a higher risk. You can either use a mosquito net or any net to seal your window and then open for the whole day (Chandramohan et al., 2018)
- Use a mosquito net over the bed if your bedroom isn't air-conditioned or screened. For additional safety, you can treat the mosquito net with the insecticide permethrin.
- Depending on the condition and prescription, you can take anti-malarial tablets



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- If you are a regular user of sunscreen, make sure you apply sunscreen first and then use an insect repellent. Also, go for a sunscreen with SPF of 30-50
- Follow the prescriptions of the doctor. This means that if your doctor has requested you to follow a 2-week course, follow the prescription and medications for two weeks
- Keep your home and surroundings clean without any junks or wastes (Fana et al., 2011)

### CONCLUSION

The viral disease HIV infection affects the clinical presentation, severity and response to treatment of malaria cases in every individual irrespective of the age, it usually depends on the intensity of malaria transmission in the area, consequent level of host immunity and the individual affected (E.g. adult, child or pregnant women) (Chandramohan et al., 2018). However in different malaria HIV co-endemic countries, there has been little or no research conducted, infection of HIV and malaria are among the two most important global health problems in developing countries including Nigeria which is reported to have caused more than 4 million deaths yearly especially in patients with HIV infection, thus increasing the risk of severity of malaria and burdens.

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