

Seroprevalence of Malaria Infection among Human Immunodeficiency Virus Patients in Ondo State, Nigeria

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Abstract: Blood samples from 1410 patients within the age groups of 0-9, 10-19, 20-29, 30-39, 40-49 and 50-59, in Okitipupa, Akure, Owo, Ondo and Ikare-Akoko all in Ondo state Nigeria, were screened for the presence of human immunodeficiency virus (HIV-1 and HIV-2 antibodies). The subjects were made up of 570 (40.43%) males and 840 (59.57%) females. An overall prevalence of 206 (14.61%) was confirmed positive for HIV antibodies with HIV-1 accounting 75.24% (155/206), HIV-2 16.51% (34/206) and HIV- 1+2 accounting for only 8.25% (17/206). Out of the 206 seropositive samples, only 69 (33.50%) were positive for malaria. The highest prevalence of HIV antibodies was observed among age groups 20-29 (16.2%) and 30-39 (16.2%). There was an association between HIV and malaria infection. The highest prevalence of malaria infection in HIV seropositive patients was 66.7% and was observed among children within the age group of 0-9 years. Also, the prevalence rate of malaria in Okitipupa (50%) was highest, followed by Akure (35%), Owo (20%), Ikare (16.7%) and lastly Ondo town (12.5%).

Key words: Seropositive, seronegative, seroprevalence, malaria, HIV

INTRODUCTION

The Human Immunodeficiency Virus (HIV), a non-oncogenic retrovirus and a member of lentivirinae subfamily, is the etiologic agent of Acquired Immune Deficiency Syndrome (AIDS) and AIDS-Related Complexes (ARC). The term "Syndrome" has been used because AIDS does not constitute a single illness but rather encompasses a wide range of clinical diseases including specific life threatening infections and neoplasm associated with a profound and irreversible acquired disorder of cell-mediated immunity^[1,2].

AIDS has been identified in over 200 countries and an estimated 42 million people are living with HIV/AIDS worldwide. In addition, more than 3 million die every year from AIDS related illnesses. For infection to be established, HIV must contact and bind to the Cluster Differentiation (CD⁺4) cells; so that it can enter a cell, multiply and spread. CD⁺4 are present on T- lymphocytes, macrophages and dendritic cells. HIV causes a depletion of these protective cells, available to protect the body from invading diseases^[3].

HIV is mostly transmitted via sexual routes, either by homosexual or heterosexual contact^[4,5]. Parental transmission is another mode of spread^[6] in countries that lack adequate medical resources and this may occur via reuse of inadequately sterilized injection needles^[7]. The

risk of contracting HIV infection from a unit of infected blood is estimated to be over 95%. HIV is also readily transmitted through infected blood products. Mother-to-child transmission, via pregnancy, labour, delivery or breastfeeding, is the most significant source of HIV infection in children below the age of 15 years^[4,8,9].

HIV is not spread through casual contact or by mosquitoes or other insect vectors^[10]. However, malaria and AIDS share some similarities. One common feature between malaria and AIDS is that there is no effective vaccine for immunization against these diseases, which have continued to threaten the existence of man in Africa. Just like AIDS, malaria parasite can be transmitted through blood transfusion and communal use of syringes by drug addicts^[11].

However unlike AIDS, malaria is transmitted through the bite of an infected *Anopheles* mosquito. Malaria is the most important of all tropical diseases and it constitutes the most public health problem facing people in the tropical Africa including Nigeria. The United Nations Populations Division has statistically shown in 1990 that malaria is the only disease apart from AIDS that has a significant rising tendency. Each year at least 300 million cases of malaria result in more than a million deaths worldwide. Ninety percent of these deaths are in sub-Saharan Africa and most are children under five years old^[12]. Malaria is caused by protozoan parasite of the

genus *Plasmodium*. Four species of *Plasmodium* can produce the disease in its various forms: *P. falciparum*, *P. vivax*, *P. ovale* and *P. malariae*. *P. falciparum* is the most widely spread of the four^[2,13,14].

S The present investigation was designed to provide information on the prevalence of malaria parasite in HIV infected patients in Ondo State, South-western Nigeria.

MATERIALS AND METHODS

A total of 1410 samples from 570 males and 840 females aged 0-59 years were collected from different government hospitals at Okitipupa, Akure, Owo, Ondo and Ikare-Akoko all in Ondo state Nigeria between January and September 2006. In all 361 samples were collected from Akure, 344 samples from Okitipupa, 275 samples from Ondo town, 138 from Ikare-Akoko and 292 samples from Owo. A survey questionnaire was designed asking for sex, age and marital status of the patients (respondents). The samples were tested serologically.

Blood samples were collected with sterile disposable syringes and needles into different anticoagulant bottles and spurned down using bench centrifuge at 3000 rpm for 5 min. Serum obtained was stored in clean tube at 2-8°C until needed for screening. The resulting plasma samples were transferred separately into sterile tubes. Assay was done using Trinity Biotech USA Capillus HIV-1/ HIV-2 test kit^[15]. The Genie 11 HIV-1/ HIV-2 is a dual recognition enzyme immunoassay kit.

The test kit is made up of Port A in which plasma containing antibody is added and port B which already has antigens to HIV-1 and HIV-2 adsorbed on the absorbent material. When anti HIV antibodies are present in the tested plasma or serum, they will migrate to port B where the reaction takes place. Fifty microlitre of plasma was diluted with 3 drops (150 µL) of reagent 1 (specimen diluents). It was introduced to port A and allowed to stay for 3 min. Three drops of reagent 2 (streptavidin alkaline phosphate) was added to port B and allowed to stay for 3 min. Port B was then filled with reagent 3 (washing solution) and mixed with chromogenic substrate after which it was allowed to react for 3 min. The reaction was stopped with stop solution and the result was read after 4 min. The reaction was then visualized as gray-blue spots by reaction with chromogenic substrate.

Thin and thick blood films were made from the HIV seropositive blood samples for malaria screening. The films were allowed to air dry. The thin film was fixed with absolute methyl alcohol for 30 sec after which the methyl alcohol was poured off and the 3% diluted Giemsa stain was immediately added. The stain was allowed to stand for 30 min after which it was flushed off with tap water.

The slide was thereafter flooded with buffer solution (pH 7.2) and allowed to differentiate for about 30 sec. It was flushed, drained and air-dried. The thick film was stained in a beaker containing 5% Giemsa stain for one h, after which tap water was gently poured on the slide and air-dried. The slides were observed microscopically using the oil immersion objective.

RESULTS

The results of the analysis showed that of the 1410 samples screened for HIV, 60 (4.25%) were confirmed seropositive for HIV antibodies with HIV-1 accounting for 155 (11.0%) and HIV-2 strain 34 (2.4%) while HIV-1+2 stain accounted for only 17 (1.2%) of samples. Of the 206 HIV-positive samples, only 60 (29.1%) had malaria parasite.

The prevalence of HIV antibodies according to age group, sex and marital status is shown in Table 1. There was strong association between sex and HIV infectivity. There was also a strong association between marital status and HIV infectivity. The age group mostly infected is those within 20-30 years of age (16.20%), followed by those within the age group of 10-19 years (15.6%) while those within the age group of 0-9 years were 15%. The least infected were those in the age group of 50-59 years. On the basis of sex, the male had the highest prevalence of HIV infection compared with the female.

HIV-1 occurred mostly in children within the age group 0-9 years (100%) while those in the age group of 30-39 years are the second most affected by HIV-1 strain (82.4%). This was followed by the age group of 40-49 years (77.5%) and then by the age group of 20-29 years (71.4%) and lastly 10-19 years (57.1%). In all HIV-1 had 75.24% prevalence while HIV-2 had a total infection rate of 16.51% and the age group mostly infected was that of 50-59 years (33.3%). HIV-1+2 had a total infection rate of 8.25% with those patients within the 10-19 years of age being the most infected Table 2.

Table 3 shows the distribution of malaria infection among different age groups and sex of HIV seropositive patients. Children within 0-9 years were mostly infected with malaria (66.7%), followed by those within the age group of 40-49 years (44.4%). On the basis of sex, females had the prevalence of 38.2% while the males had infection rate of 26.9%. In all, malaria had infection rate of 33.50%.

The infection rates of HIV and malaria infection in the different areas studied are presented in Table 4. Akure had the highest rate of individuals with HIV (19.1%), while Ondo had the least (10%). Okitipupa had the highest malaria infection rate (50%), followed in a decreasing order by Akure, Owo, Ikare-Akoko and Ondo town.

Table 1: Prevalence of HIV antibodies by age group, sex and marital status
% Infection+S.E.

Age group	Male single	Male married	Female single	Female married
0-9	12.4+0.2	0	16.5+0.1	0
10-19	14.8+0.1	0	15.2+0.2	0
20-29	15.1+0.2	14.2+0.1	12.8+0.1	31.5+0.3
30-39	8.2+0.1	25.3+0.2	19.9+0.2	12.4+0.2
40-49	12.5+0.2	25.1+0.1	14.7+0.3	9.8+0.2
50-59	0	50.1+0.2	0	19.4+0.1

Table 2: Prevalence of HIV-1, HIV-2 and HIV-1+2 antibodies among the various age groups
% Infection of HIV strains +S.E.

Age group	HIV-1	HIV-2	HIV-1+2
0-9	100	0	0
10-19	57.1+0.3	24.5+0.1	14.3+0.1
20-29	71.4+0.2	15.6+0.2	9.8+0.2
30-39	83.3+0.1	9.5+0.1	9.8+0.4
40-49	77.5+0.3	9.2+0.1	5.8+0.3
50-59	70.1+0.2	36.4+0.3	0

Table 3: Distribution of malaria infection according to age group and sex of HIV positive patients
% infection of malaria

Age group	Male	Female	Total
0-9	100	51.6	66.7
10-19	51.2	40.8	43.1
20-29	24.3	44.9	31.4
30-39	13.1	17.8	15.1
40-49	32.7	50.5	44.4
50-59	0	49.7	32.6

Table 4: Prevalence of HIV and malaria in different parts of Ondo state, Nigeria
% Infection

Locality	HIV	Malaria
Akure	19.1	35
Ikare-Akoko	15	16.7
Okitipupa	16	50
Ondo	10	12.5
Owo	11.2	20.1

DISCUSSION

The overall prevalence of malaria infection among the HIV seropositive patients was 33.50 with 26.90 and 38.20% being observed in males and females respectively. Peak infectivity was observed among children within the age group of 0-9 years (66.7%). This strongly with Joklik *et al.*^[13] who observed and reported in the United States of America that children suffer most from malaria infection. The susceptibility of children to malaria infection could be attributed to their low immune *status quo*, which in this context might be due to HIV infection, which has degenerative effect on the immune system. It can also be attributed to the non-challant attitude of their parents towards them, which may be the direct consequence of poverty, or inadequate knowledge about the mode of transmission of malaria parasite.

The high rate of malaria infection in females could be attributed to their social behavior such as their dressing code, which in most cases involves short sleeves and shorts skirts, thereby exposing parts of their bodies to mosquito attack. Females often stay out late during mosquito-biting hours carrying out domestic activities. The findings also agree with that of Warren and Mohmoud^[11], who observed that females are at higher risk of malaria infection compared to their male counterparts. The low infection rate of malaria among age group 30-39 years could be attributed to an updated knowledge about the mode of transmission of malaria parasite or due to preventive measures among this age group.

The high rate of malaria infection in Okitipupa and Akure could be attributed to water lodge around resident houses, poor drainage system, bushes around houses, which are good breeding grounds for mosquitoes. Inadequate preventive and control measures also contribute to high rate of malaria infection in these areas. Drug resistant malaria parasite could also contribute to the high infection rate since most of the patients in these areas cannot afford hospital bills thereby resorting to inadequate medication.

The low rate of malaria infection in Ondo town may be due to awareness about the nature of transmission of malaria parasite since most people living in this area are enlightened, hence control and preventive measures must have been put in place. In Ikare-Akoko, the respondents were more of the low- class individuals and this explains the slightly high rate of malaria infection in this area; because the rich in this area patronize mainly private hospitals and are well informed on the control and preventive measures of malaria parasite. Conclusively, the study has shown that the prevalence of malaria infection among HIV patients is about one third (1/3).

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REFERENCES

1. Centre For Disease Control, (CDC), 1982. Origin of the Epidemic in AIDS. Images of the Epidemic. World Health Organization, Geneva, P.J.
2. Bhatia, R. and R.L. Ichhpujani, 2004. Essentials of Medical Microbiology, 3rd (Edn). Jaypee Brothers Medical Publishers, New Delhi, India, pp: 384-390.

3. Stern, B.S., D.D. Gowda, J.D. Lifson, R.C. Penhallow, K.G. Bench and E.G. Engleman, 1989. pH independent HIV entry into CD4 + positive T-cells via virus envelop fusion to the plasma membrane. *Cell*, 49: 659-668.
4. World Health Organisation, 1994. HIV transmission: Matters of Fact. In: *AIDS-Images of the Epidemic*. WHO Geneva, pp: 9-12.
5. Decock, K.M., M.G. Fowler and E. Mercier, 2000. Prevention of mother-to-child HIV transmission in resource-poor countries, *Translating Resource into Policy and Practice*. *JAMA*, 283: 1175-1182.
6. Temmerman, M., 2001. HIV in pregnancy. *Verhandelingen-Koninklijke Academic Voor Geneeskunde van Belgie*, 63: 493-510.
7. Patrascu, I.V., S.T.N. Constrantin and A. Dublanchet, 1990. HIV-1 Infection in Romanian Children. *Lancet*, pp: 1-672.
8. Centre for Disease Control, (CDC), 1987. Recommendation for Prevention of HIV transmission in health care setting. *MMWR*, 36: 25-27.
9. Offor, E., J. Onakewhor and F.E. Okonofua, 2000. Maternal and neonatal seroprevalence of HIV antibodies in Benin-city, Nigeria. *J. Obstetrics and Gynaecol.*, 20: 589-591.
10. Friedland, G.H. and K.S. Klien, 1987. Transmission of HIV. *N. Engl. J. Med.*, 317: 117-125.
11. Warren, K.S. and A.F. Mohmoud, 1990. *Malaria*. In: *Tropical Geographical Medicine*. 2nd (Edn). McGraw-Hill Co, pp: 295-263.
12. Jones, C., 2006. Hitting malaria where it hurts; Household and community response in Africa. *Id21 insights #9*, August 2006, www.id21.org, pp: 1-2.
13. Joklik, W.K., H.P. Willet, D.B. Amos and C.M. Wilfert, 1992. *Human Retrovirus in zinsser microbiology*. 20th (Edn). Appleton and Lange, California, pp: 1048-1055.
14. Blacklock, D.B. and T. Southwell, 1997. *A Guide to Human Parasitology*. 10th (Edn). H.K. Lewis and Co Ltd. London, pp: 47-67.
15. Onifade, A.K., 2003. Incidence of Human Immunodeficiency Virus among pregnant women attending antenatal clinics in Ondo state Nigeria. *J. Gender and Health Studies*, 1: 218-222.