Short Notes

J Vect Borne Dis 42, December 2005, pp 151-154

Malaria infection in HIV-seropositive and HIV-seronegative individuals in Jos-Nigeria

C.J. Uneke^a, O. Ogbu^a, P.U. Inyama^b & G.I. Anyanwu^b

^aDepartment of Medical Microbiology/Parasitology, Faculty of Clinical Medicine, College of Health Sciences (EBSUTH), Ebonyi State University, Abakaliki, Nigeria; ^bApplied Parasitology Unit, Department of Zoology, University of Jos, Nigeria

Key words Acquired immunodeficiency syndrome – co-infection – Jos-Nigeria – malaria – P. falciparum

Infections by malaria parasite and human immunodeficiency virus (HIV) represent major public health problems in many parts of the world. Both infections kill millions of people each year and both infections are scourges of developing nations in Africa, India, southeast Asia and South America¹. In recent years, it has been hypothesised that a possible deleterious interaction between both infections exists. The presented hypothesis suggested that chronic latent malaria infection prepares the niche where otherwise feeble HIV infection can thrive and cause acquired immunodeficiency syndrome (AIDS)². However, an earlier critical review from numerous available reports on this subject showed that there were hardly evidences to support this hypothesis³. Depending on the perspective it is viewed, malaria has either a lot or very little in common with HIV. With shared geography and demographics especially in sub-Saharan Africa, co-infection is common, yet surprisingly few obvious clinical associations between HIV and malaria are reported in this region¹.

Till date there is a dearth of information on the prevalence of malaria and HIV co-infection in Nigeria. The life expectancy in Nigeria was estimated to have dropped from 53 years in 1990 to 51 years in 2002, largely due to the AIDS epidemic. The epidemic in Nigeria has extended beyond the commonly classified high-risk groups and is now common in the general population with the adult prevalence rate at 5.8% in 2001⁴. Our objective in this study was to determine the prevalence of malaria infection in HIV-seronegative individuals as part of the preliminary investigation leading to a more advanced research on malaria and HIV co-infection in Nigeria.

The study was conducted from April 2002 through November 2003 in Jos-Plateau located in an area covering about 9,400 km² of the crystalline complex in Central Nigeria. The area has two seasons, the dry season (November–March) and the rainy season (April–October). Malaria transmission is usually high towards the end of the rainy season. The Central Nigeria is known to record the highest prevalence of HIV infection in the country⁴.

Patients who visited Jos University Teaching Hospital (JUTH) and the Plateau Specialist Hospital (PSH), who presented themselves for retroviral screening, were enrolled in the study. Informed consent was obtained from each patient with the assistance of their physicians. Thereafter, 5 ml of blood sample was ob-

tained by vene puncture from each of these patients for HIV and malaria parasite screening to identify the appropriate study group. The HIV serostatus of 490 of them (age range 17–60 yr) was confirmed by immunoblot analysis (Bio-Rad, Novapath Diagnostic Group, U.S.A.) at the International Centre for Scientific Culture (ICSC) Retroviral Laboratory, PSH, Jos. This was after an initial HIV screening using the Vironostica[®] HIV-1 microelisa system (Organon Teknika, Durham, U.S.A.) at the AIDS/Leishmaniasis Research Laboratory, University of Jos. These 490 individuals constituted the HIV-seropositive population. Thick and thin blood films were made 2–3 h of collection, stained with Giemsa according to standard protocol for the identification of malaria parasite⁵.

The HIV-seronegative population comprised of 578 individuals (10–59 yr age) who were HIV-seronegative by the Vironostica[®] HIV-1 microelisa system and were participants of the community targeted voluntary counseling test (VCT) for HIV-infection conducted in various areas of Jos-Plateau. These individuals willingly offered to be screened for HIV during these programmes. All the study participants were informed verbally and their consent duly obtained. Thick and thin blood films for malaria parasite diagnosis were performed as described earlier⁵, after the HIV-serostatus was established for each individual. Differences between proportions were evaluated by the chi-square test. Statistical significance was achieved if $p \le 0.05$.

Of the 490 HIV-seropositive individuals studied 103 (21%, 95% CI 17.41–24.63) had malaria infection, while 67 (11.6%, 95% CI 8.98–14.2) of the 578 HIV-seronegative individuals also had malaria infection. *Plasmodium falciparum* was identified in all the cases. Mixed infections of *P. falciparum* and *P. malariae* occurred in 2.6% of all the infected cases. Amongst the HIV-seropositive individuals, the males were significantly more infected than the females (27.4%, 95% CI 21.2–33.52 vs 16.6%, 95% CI 12.32–20.9); ($\chi^2 = 8.198$, p ≤ 0.05 , df =1) (Table 1) The prevalence of malaria infection increased with age

amongst the HIV-seropositive individuals with the highest rate of 32% (95% CI 13.71–50.29) at the 51– 60 yr age group, although there exist no significant difference in the trend ($\chi^2 = 5.697$, $p \le 0.05$, df = 4). The prevalence of malaria infection was slightly higher amongst the males (12.7%, 95% CI 8.76–16.58) than the females (10.4%, 95% CI 6.84–14.02) in the HIVseronegative population (Table 1). There was no significant difference statistically ($\chi^2 = 0.693$, $p \le 0.05$, df = 1). Amongst the HIV-seronegative individuals, the highest prevalence of malaria infection of 14.8% (95% CI 8.68–21) was observed at the 31–40 yr age category (Table 1). There was also no sigificant difference statistically (χ^2 =4.987, $p \le 0.05$, df = 4).

 Table 1. Malaria infection amongst HIV-seropositive and seronegative individuals in Jos-Nigeria

Parameter	No. examined	No. with malaria infection	95% confidence interval
HIV-seropos	itive		
Sex			
Male	201	55 (27.4)	21.2-33.52
Female	289	48 (16.6)	12.32-20.9
Total	490	103 (21)	17.41-24.63
Age (yr)			
<u><20</u>	15	2 (13.3)	3.87-30.53
21–30	184	27 (14.7)	9.56-19.78
31-40	188	44 (23.4)	17.35-29.45
41–50	78	21 (26.9)	17.08-36.76
51-60	25	8 (32)	13.71-50.29
Total	490	103 (21)	17.41–24.63
HIV-seroneg	ative		
Sex			
Male	300	38 (12.7)	8.76–16.58
Female	278	29 (10.4)	6.84-14.02
Total	578	67 (11.6)	8.98-14.2
Age (yr)			
≤ 20	215	20 (9.3)	5.42-13.18
21–30	195	26 (13.3)	8.56-18.1
31-40	128	19 (14.8)	8.68-21
41–50	35	2 (5.7)	1.98-13.4
51-60	5	0 (0)	_
Total	578	67 (11.6)	8.98-14.2

Figures in parentheses are percentages.

This investigation demonstrated that HIV-seropositive individuals had a higher prevalence of malaria infection than the HIV-seronegative individuals in the study area. Jos-Plateau is considered as an area of stable *P*. *falciparum* malaria transmission although considerable urbanisation processes are going on. Nevertheless, the inhabitants have developed a high degree of immunity to the parasite through repeated exposures, and thus are not generally susceptible to the effects of severe clinical malaria. This may have accounted for the lower prevalence of malaria infection amongst the HIV-seronegative population.

The humoral and cell-mediated immunity to malaria parasite are reported to be developed in such an area of stable transmission⁶. This immunity can be altered in HIV-infected persons and could influence the frequency and course of malaria infection^{7, 8}. This may explain the higher prevalence of malaria infection in the HIV-seropositive population. This submission may not be conclusive as HIV-disease progression co-factors such as : presence of opportunistic infections, nutritional status, age, viral load and genotype⁹, could also have influenced the malaria infection in the HIV-infected population. Our inability to record the parasite density is a major drawback to this research. Future studies integrating the parasite density are advocated.

Present result showed that males were more infected with the malaria parasite than the females in both of the study population. This was not unexpected because analysis of sociobehavioural factors revealed that males expose themselves more to the risk factors of malaria acquisition. It is a common habit of males in Jos-Plateau to stay long hours into the night outside the houses or sleep outdoors without appropriate coverings. There is also high level of indifference to the use of insecticide-treated bednets by the males.

Malaria infection prevalence increased with age amongst the HIV-seropositive individuals, although a somewhat similar pattern was observed amongst the HIV-seronegative population but it was less obvious in the latter. A review of a number of studies in different transmission groups confirmed that age is a cofactor for disease progression⁹. The immunity to malaria and HIV-1 infection has been shown to be age dependent¹⁰. It is possible that the older HIV-infected individuals may be more likely to develop infections including malaria than the younger HIV-infected individuals.

In conclusion it is imperative to state that people who have grown up in malaria endemic regions especially in the sub-Saharan Africa often retain partial immunity to malaria, and there is no solid evidence that this immunity is lost, as HIV disease progresses¹. However, it is suggested that the roots of the HIV outbreak and AIDS pandemic lay in the urbanisation processes in many developing countries of the sub-Saharan Africa including Nigeria, which have resulted in the eradication of the Anopheles vector from previously endemic areas, with consequent loss of natural immunity thereby enhancing vulnerability to opportunistic infections, HIV among them². Our findings have provided additional baseline information on the burden of malaria and HIV coinfection in Nigeria. Further research is advocated on this subject especially in the sub-Saharan Africa, the region with the highest prevalence of both infections.

References

- 1. Huff B. HIV and malaria: two intertwining epidemics. *Am Found Aids Res* 2000; *6*: 1–5.
- 2. Yaffe I. AIDS possibly caused by HIV and latent malaria coinfection. *Hypothesis* 2001; 57: 419–22.
- Chandramohan D, Greenwood BM. Is there an interaction between human immunodeficiency virus and *Plasmodium falciparum? Int J Epidemiol* 1998; 27: 296–301.
- 4. National HIV/AIDS and reproductive health survey. Abuja, Nigeria: Federal Ministry of Health 2003; p. 1–4.
- 5. *Manual of basic techniques for a health laboratory.* Geneva : WHO 2003; p. 172–82.
- Dean JA, Cohen S. Immunology of malaria. Ann Rev Microbiol 1983; 37: 161–7.
- 7. Stekette RW, Wirima JJ, Bloland PB, Chilima, B, Mermin JH, Chitsulo L, Breman JG. Impairment of a pregnant woman's acquired ability to limit *Plasmodium falciparum*

154

by infection with human immunodeficiency virus type-1. Am J Trop Med Hyg 1996; 55: 42–9.

- Chaisavaneeyakorn S, Moore JM, Otieno J, Chaiyaroj SC, Perkins DJ, Shi YP, Nahlen BL, Lal AA, Udhayakumar V. Immunity to placenta malaria III. Impairment of interleukin (IL)-12, not IL-18, and interferon-inducible protein-10 responses in the placental intrevillious blood of human immunodeficiency virus/malaria-co-infected-women. J Infect Dis 2002; 185: 127–31.
- Osmond H. HIV disease progression from infection to CDC defined AIDS. In: Cohen PT, Sande MA, Volberding PA editors. *The AIDS knowledge base*. New York : Little, Brown & Company 1994; p. 1.7:1–1.7:19.
- Wabwire-Mangen F, Shiff CJ, Vlahov D, Kline R, Serwadda D, Sewankambo NK, Mugerwa RD, Quinn TC. Immunological effects of HIV-1 infection on the humoral response to malaria in an African population. *Am J Trop Med Hyg* 1989; *41:* 504–11.

Corresponding author: C.J. Uneke, Department of Medical Microbiology/Parasitology, Faculty of Clinical Medicine, College of Health Sciences (EBSUTH), Ebonyi State University, P.M.B. 053, Abakaliki, Nigeria e-mail: unekecj@yahoo.com