The relationship between malaria and HIV

*SA TSWANA, **L NYSTROM, *SR MOYO, *M NZARA, ***P BOONE

Objective: To determine if there is an association between HIV and malaria infection
Design: A cross sectional survey.
Setting: Sanyati Rural District, a malarious endemic area of Zimbabwe.
Subjects: 338 volunteers aged 15 months to 76 years.
Main Outcome Measures: Prevalence of Malaria and HIV.
Results: The prevalence of malaria and HIV was 26.6% and 26.3% respectively. There was no association between prevalence of HIV and malaria.
Conclusion: There is no association between malaria and HIV.

Introduction

The marked immune suppression produced by human immunodeficiency virus (HIV) infection as a result of T cell dysfunction following loss of CD_4 + lymphocytic activity results in infection and disease due to viruses, bacteria, fungi and parasites. The quantitative loss of CD_4 + cells, the impairment of the clonal proliferation of CD_4 + cells, and a decline in interferon –γ markedly contribute to infection with organisms that require cell-mediated immunity to protect the host.[1]

Very early in the HIV epidemic, pneumonia due to pyogenic bacteria was reported in HIV infected individuals. Simberkoff *et al*[2] noted that AIDS patients developed hospital-acquired pneumococcal pneumonia in spite of having received pneumococcal vaccine against the specific serotype of *Streptococcus* pneumonia that caused illness. The literature is now full of documented cases of opportunistic organisms that are now reactivating due to the dysfunctioning of the immune system as a result of infection by HIV. Gram negative bacteraemia have also been frequently reported in patients with advanced HIV disease. Further, the association of *Mycobacterium* tuberculosis infection with HIV infection and AIDS was noted early in the AIDS epidemic.[3,4] A study conducted in Harare, Zimbabwe at the Infectious Diseases Hospital showed that TB was on the increase. At least 40% of the TB inpatients were positive for HIV antibodies.

Several organisms are now known to associate with HIV infection.[6] Of special attention are the sexually transmitted diseases. The correlation between STD and HIV is considered very high. Although correlation of bacterial, parasitic, fungal and viral infections with HIV has been noted, there are still some organisms which have not been established as whether they co-infect with HIV. There is no data to indicate malaria correlates with HIV infection, if that data is available it may be very scanty.

Our study was designed to determine if there is an association between HIV and malaria infection. The study was conducted in a malarious endemic area of Zimbabwe.

Materials and Methods

Study Area.

The study was conducted at Sanyati Baptist Mission Hospital. The hospital is about 90 km northwest of Kadoma in Sanyati tribal trust land under Kadoma district. Although the hospital is a mission institution with 70 beds it operates as a district hospital. This is due to the number of patients who attend the hospital. Several patients travel a distance of about 80 km. The other nearest district hospital (Gokwe Hospital) is about 85 km away, west of Sanyati Hospital. Most of the study subjects were from Gokwe, Chenjiri and surrounding areas of Sanyati Rural District. Local people around the hospital depend on subsistence farming. The area is malarious endemic zone, where malaria is one of the major diseases. Sanyati is one of the areas in Zimbabwe which records the highest mortality rate due to malaria.

Study Population.

The study population consisted of 338 volunteers. Of these 248 were females and 90 were males. The age ranged from 15 months to 76 years with a mean of 29 years. Most of the subjects were patients attending the hospital with malaria problems. More than 50% of the females were pregnant.

Specimen Collection.

Before blood specimens were collected all volunteers completed a questionnaire and signed a consent form. This was done after the research assistant nurse had clearly explained the objectives of the study to each study subject.

**Department of Medical Microbiology*
University of Zimbabwe Medical School
P O Box A178, Avondale
Harare, Zimbabwe
Phone/Fax: 263-4-792245
***Department of Epidemiology and Public Health*
Umea University, Umea
Sweden
****Baptist Hospital*
Kadoma, Zimbabwe

Correspondence to:
Professor Sam Tswana

For subjects below 18 years of age parental consent was obtained. Before blood was taken each volunteer completed a brief questionnaire; 5mls of venous blood was then collected into plain tubes. For the two infants less than 24 months old blood was collected by heel pricking. Specimens were immediately taken to the laboratory for processing. In the laboratory thin and thick slides were prepared after which all specimens were centrifuged for three minutes at 2 000xG and sera was separated and stored at -20°C.

Examination for Malaria Parasites.

Smears were stained using the Giemsa stain technique and were read by two microscopists. Results were recorded independently and where there were differences these slides were referred to the parasitologist for confirmation. Further, the parasitologist randomly examined the positive slides for further confirmation.

Examination for HIV Antibodies.

Detection of HIV antibodies was done using the enzyme linked immunoserbent assay (ELISA) Abbott Laboratory diagnostics, Chicago, and the Cambridge Biotech, Galway, Ireland and were read spectrophotometrically. Any sera positive for the presence of any border-line, indeterminants or those which disagreed on the two different ELISA were confirmed using the Western Blot.

Statistical Methods.

To test whether sex and age may confound the association between the prevalence of malaria and HIV, the chi squared test with Yates' correction and the Kruskal-Wallis (H) test were used, respectively. Logistic regression models were fitted to the data in a step wise fashion. A result yielding a p value of less than 5% was considered statistically significant.

Results

Data was available on 338 persons of whom 93 (27.7%) were males. The prevalence of malaria was 26.6%. It was significantly (p<0.001) higher in females (41.9%) than males (20.6%). The prevalence of malaria among males was higher (x^2=20.77; df=1; p<0.001) in those aged less than 30 years (28/40 or 70.0%) than in those of age 30 years or more (10/50 or 20.0%). Among females, the prevalence of malaria was highest among those aged less than 20 years (34.5%) as shown in Table I.

Table I: Prevalence of malaria and HIV by age group among female subjects.

Age Group (Years)	Total	Malaria n (%)	HIV n (%)
<20	55	19 (34.5)	6 (10.9)
20-29	94	19 (20.2)	16 (27.7)
30-39	61	6 (9.8)	13 (21.3)
40+	32	6 (18.8)	8 (25.0)

The prevalence of HIV was 26.3% and was significantly (p=0.009) higher in males (36.6%) as compared with females (21.8%).

Although among males the prevalence of HIV was higher in those of age 30 years or more (20/50 or 40.0%) than in those of less than 30 years (11/40 or 27.5%), this was not statistically significant (x2=1.03, df=1, p=0.309). Meanwhile, among female subjects, the prevalence of HIV was highest in the age group 20 to 29 years (27.7%) as shown in Table II.

Table II: Prevalence of both malaria and HIV by age group.

Age group (years)	Total	n (%)
<20	79	4 (5.1)
20-29	112	6 (5.4)
30-39	77	6 (7.8)
40-49	35	2 (5.7)
50+	31	1 (3.2)

The prevalence of both malaria and HIV was 5.9% (20/338) and it was significantly (x^2=5.01, df=1; p=0.025) higher in males (10.8% or 10/93) than in females (3.7% or 9/243). However, no association was observed between prevalence of both malaria and HIV and age (x^2=0.88; df=1; p=0.831).

Confounding Factors in the Association of Malaria and HIV.

Sex was associated with both malaria (x^2=14.68; df=1; p<0.001) and HIV (x^2=6.88; df=1; p=0.009).

The median (Q_1, Q_3) ages were significantly different (H=22.79; df=1; p=0.001) between those with malaria [22 (14, 29)] and those without malaria [29 (22, 38)], and significantly different (H=5.10; df=1, p=0.024) between those with HIV [28 (24, 38)] and those without HIV [26 (19, 36)].

The most significant model with one factor to consider for adjustment was that with sex (Table III). Age was not significant once sex was in the model (x2= 1.78; df=1; p,0.05).

After adjusting for sex, there was no association between malaria and HIV (p=0.077).

Table III: Models for association between malaria and HIV.

Model	x^2	df	p value
No adjustment	0.80	1	0.372
Age adjusted	2.50	2	0.287
Sex adjusted	10.60	2	0.005
Age and sex adjusted	8.82	3	0.032

Discussion

Malaria and HIV infection are probably the greatest challenges facing developing countries today. Because of the emerging of HIV, organisms which laid latent in the human host are now reactivating. There are several reporting cases of opportunistic organisms that are now reactivating due to the disfunctioning of the immune system as a result of infection by HIV. There are ample evidences on the association of HIV infection with other micro-organisms.[6] However, there is scanty data on the correlation between HIV and malaria.

Our study showed no interaction between malaria and HIV. This is in agreement with studies by Allen *et al* (1991)[7] and Atozori *et al* (1993).[8] However, it should be noted that the

study was cross sectional, not longitudinal and it was conducted during the malaria peak season.

Although the prevalence of HIV was higher (26.3%) than the national rate (20%), (personal communication with AIDS Co-ordinator Ministry of Health and Child Welfare, Harare, Zimbabwe), we would not find any association at all between *Plasmodium falciparum* and HIV. Even though malaria infection was more common in HIV seropositives than in seronegatives, the intensity of the *Plasmodium faciparum* infection was not associated with HIV infection.

Acknowledgements

This work was supported by the Block Allocation from the University of Zimbabwe. The authors would like to sincerely thank the Research Board of the University of Zimbabwe. Further our sincere thanks go to Sanyati Hospital staff for their unlimited assistance in specimen collection. We would also like to express our special gratitude to both Ms L Muteyaunga and Ms T Ndasiyiwa for typing this manuscript. Finally our deepest thanks are extended to Dr S Siziya for his assistance in statistical analysis.

References

1. Bowen DL, Lane HC, Fauci AS. Immuno pathogenesis of the acquired Immunodeficiency syndrome. *Ann Intern Med* 1985;103:704-9.
2. Simberkoff MS, EL Sadr W, Schiffman G, Rahal JJ Jr. *Streptococcus pneumoniae* infections and bacterimia in patients with acquired immunodeficiency syndrome with report of pneumococcal vaccine failure. *Am Rev Resp Dis* 1984;130:1174-6.
3. Handwerger S, Mildvan D, Sene R, Mckinley FW. Tuberculosis and the acquired immunodeficiency syndrome at a New York City Hospital. *Chest* 1987;91:176-80.
4. Barnes PF, Bloch AB, Davidson PT, Snider DE Jr. Tuberculosis in patients with human immunodeficiency virus infection. *N Engl J Med* 1991;324:1644-50
5. Houston S, Ray S, Mahari M, Neil P, Tswana SA, Legg W *et al.* The association of tuberculosis and HIV infection in Harare, Zimbabwe. *Tubercle and Lung Dis* 1994;75(3): 220-6.
6. Neu HC, Levy JA, Weiss RA. Focus on HIV Proceedings of the International Symposium Brocket Hall, Hertfordshire Sept. 23-26, 1992.
7. Allen S, Van de Perre P, Serufilira A, Lepage P, Carael M, DeClercq A, *et al.* Human immunodeficiency virus and malaria in a representative sample of child bearing women in Kigali; Rwanda. *J Infect Dis* 1991;164:67-71.
8. Atzori C, Bruno A, Chichino G, Cevini C, Bernuzzi AM, Gatti S *et al.* HIV-1 and parasitic infections in rural Tanzania. *Ann Trop Med Parasitol* 1993;87:585-93.

Correspondence to:
Dr OA Fadiran
Department of Surgery
College of Health Sciences
Obafemi Awolowo University
Ile-Ife, Nigeria