PREVALENCE OF MALARIA AND PRACTICE OF PREVENTION AMONG HIV POSITIVE PREGNANT WOMEN AT AMINU KANO TEACHING HOSPITAL (AKTH), KANO.

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ABSTRACT
A total of 360 primigravidae were studied at AKTH. 180 each of similar age and socioeconomic status were used as cases and controls in a prospective study.
There was no statistically significant difference in their practice of malarial prevention at booking. The prevalence of malaria in their blood smears at booking was 32.22%, with the HIV infected cases accounting for 23.33%, whilst the HIV uninfected accounted for 8.89%The prevalence of clinical malaria was significantly eight times higher and the prevalence of recurrent malaria was twenty six times higher in the HIV infected primigravidae as compared to the HIV uninfected primigravidae. Review of malarial prophylaxis in HIV infected primigravidae is recommended.

Keywords: *HIV infected, HIV uninfected, Malaria*, *Multigravidae, Primigravidae.*

INTRODUCTION

Malaria and Human Immunodeficiency Virus (HIV) infection are two of the most common infections in Sub Saharan Africa,[1]where it is estimated that 36.1 million adults and children are living with HIV/AIDS.[2] Sub Saharan Africa contributes only about 10% of the world's population, but account for over 80% of the world's HIV infected women.[2] Without intervention, it is estimated that about 25 to 45% of the HIV infected women will transmit the infection to their children.[2] Over 30 million women in Africa become pregnant in malaria-endemic area each year.[3] Up to 20% of pregnant women in endemic area will have asymptomatic malarial parasitaemia.[4]Even though malaria is a curable and preventable disease, it kills over a million people a year, majority of whom are children and pregnant women.[5] In the tropics, Falciparum malaria during pregnancy is a major cause of maternal, fetal and neonatal morbidity and mortality.[6] It is a major public health issue in Africa. The economic cost of Malaria in pregnancy and HIV is enormous and weighs heavily on the economies of poor African countries.[7,]

Pregnant women, especially primigravidae and secundigravidae are a specific risk group to malaria infection.[9] HIV infection in pregnancy appeared to impair a pregnant woman's ability to resist malaria infection. The protection expressed by multigravida to malaria infection disappears when HIV infection co-exist with malaria. The exact mechanism for increased susceptibility to malaria during pregnancy is unknown. However, it is attributed to suppressed immunity in pregnancy.[6,10] It is suggested that the increased protein demand in pregnancy is not usually met by nutritional supply alone and therefore; protein from the immune system is diverted to fill in the deficit thereby causing a partial alteration of immunity.[10]The vast majority of these infections are sub-clinical and in most women are asymptomatic and therefore undetected and untreated. There is specific expression of variants of P. falciparum erythrocyte membrane protein-1 in pregnancy that occurs in the syncytiotrophoblastic cells[11]. The parasites adhere

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PROSTAGLANDIN LEVELS AND SEMEN QUALITY IN MALE PARTNERS OF INFERTILE COUPLES IN ILE-IFE, NIGERIA.

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Table I: Prostaglandin profile of all Respondents

Prostaglandin	PGF2α	PGE
Mean± SEM	2.77±0.23	248.79±13.88
Median	1.98	250.35
Range	0.15-11.05	21.8-652.0

Table II: Prostaglandin profile by semen status, sperm count and socio economic status

PG/Status	N	Mean± standard error of mean (SEM)	Median	Range
PGF2α(µg/ml)	27	2.1 ±0.32	1.64	0.25-7.83
Normal semen	87	3.0±0.28	2.3	0.15-11.05
Subnormal semen		**P<0.05**		
Total PGE(µg/ml)		325.1± 28.3	356.5	50.7-628.8
Normal semen	27	225.1 ±15.1	219.6	21.8-652.0
Sub-normal semen	87	**P<0.01**		
PGF2α(µg/ml)	12	2.06 ±0.43	2.29	0.15-4.23
Azoospermic	52	3.45±0.42	2.43	0.2-11.05
Oligospermic	50	2.23±0.23	1.9	0.24-7.83
Normospermic		**P<0.05**		
Total PGE(µg/ml)		168.7±29.7	2.43	0.2-11.05
Azoospermic		216.8±19.1	224.6	21.8-503.2
Oligospermic		301.3± 21.5	334.8	45.3-652.0
Normospermic		**P<0.01**		
PGF2α(µg/ml)	38	3.15±0.42	2.66	0.31-10.69
High Social Class	42	2.74±0.34	1.64	0.21-11.05
Middle Social class	34	2.37±0.36	1.71	0.15-10.30
Low Social class		**P> 0.05**		
Total PGE(µg/ml)		243.64±35.6	250.35	21.8-480.8
High Social Class		217.14±30.5	220.0	25.9-538.0
Middle Social class		293.65±28.9	285.5	29.5-652.0
Low Social class		**P> 0.05**		

to this syncytiotrophoblast more, through this protein, leading to intervillous parasitaemia. The intervillous parasitaemia causes "leakage" through the placental barrier which is thought to increase vertical transmission of HIV where the infection co-exists with malarial infection. However, with subsequent pregnancies, protective immune mechanism gradually develops and expands, and manifestation of infection declines. With the binding of parasites in the intervillous spaces, there is placental sequestration, placental infiltration by inflammatory cells and release of pro-inflammatory mediators. The effects of these are maternal anaemia, low birth weight and preterm delivery with increased perinatal mortality[11].

Some components of immune response to P. falciparum are modified by HIV infection. P. falciparum stimulates HIV replication by increased cytokine production (interleukin 6 and tumour necrosis factor alpha) by activated lymphocytes. It also increases potential HIV reservoir in the placenta by increasing the Chemokine receptor 5(CCR 5+) receptors in the placenta[12]. These changes result in HIV positive women being more likely to develop malaria in pregnancy than seronegative women, and also have rapid progression of their HIV infection during pregnancy[12]. Dual infection therefore, has detrimental effect on maternal and infant survival. Effective malaria control will reduce the prevalence of malaria infection in the community and will reduce pregnancy associated malaria complications. In addition, there will be a reduction in the severity and complications of HIV in pregnancy. The huge economic burden saddled in the treatment of malaria can be channeled to provision of other health and social needs of the community that will improve quality of life.

The study was embark upon in order to ascertain the Prevalence of asymptomatic malaria among HIV infected and HIV uninfected primigravidae who registered in AKTH for antenatal care.

MATERIALS AND METHODS

Aminu Kano Teaching Hospital (AKTH) is a 500-bed hospital established in 1988. It is located in Kano, the largest commercial centre of northern Nigeria. The hospital receives clients from within Kano and the neighboring States of Jigawa, Katsina, Kaduna, Bauchi and Zamfara. The majority of patients are Hausa or Fulani, although substantial numbers are Ibo or Yoruba. Most are traders, farmers, businessmen or civil servants. The hospital operates a specialist antenatal clinic (ANC) four times per week, attending to an average of 120 clients per day; The PMTCT programme has been fully integrated into the hospital's obstetrics services. In the ANC, voluntary group counseling is offered to all new clients, along with routine HIV testing, with a right to 'opt out.' HIV-positive patients are then offered full PMTCT services.

Routinely two doses of Sulphadoxine-pyrimethamine combination (Fansidar^{R}) is given to the HIV uninfected pregnant women at four weeks interval after 18 weeks of gestation, while the HIV infected receives three doses[14].

All ANC clients are also supplied with ITN and are advised to sleep under it and are also advised to clear their surroundings of mosquito breeding sites.

STUDY DESIGN:

The study was a prospective study conducted among pregnant women attending AKTH for their antenatal care.

Study Population:

The study population is pregnant women attending the ANC clinic of AKTH.

INCLUSION CRITERIA:

Primigravidae who register for ante natal care at gestational age of 16 weeks or less during the study period, gave their consent to participate in the study and consented to HIV screening were recruited. Where the patient was not sureof her LMP, ultrasound scan dating was employed.

The reason for selection of only primigravidae as cases and control is because primigravidae have lowered immunity to malaria infection compared to multigravidae[9], and this variation in the level of immunity may interfere with the outcome of the study.

SAMPLE SIZE DETERMINATION:
The minimum sample size was determined using the formula for sample size estimation as follows:
$n=(z_{\alpha}+z_{\beta})^2(p_1q_1+p_2q_2)/(p_2-p_1)^2$

where; n= minimum sample required,
z_{α} = standard normal deviate at 95%
Confidence level, =1.96
z_{β} =standard normal deviate corresponding to the power of the test to detect differences, 95% power was used for this study. The value obtained from the normal distribution table is 1.64.
p_1= prevalence of malaria among HIV
Positive pregnant women of 56.3%[15] =0.56
q_1= complimentary probability to p_1 = 1-p_1 (1-0.56=0.44)
p_2=prevalence of malaria among HIV negative pregnant women of 36.5% = 0.37.[15]
q_2= complimentary probability to p_2 = 1-p_2 (1-0.37=0.63)
Therefore, $n= (1.96 + 1.64)^2 [(0.56x0.44) + (0.37x0.63)] / (0.37-0.56)^2$
Thus; n= 172.The sample was approximated to 180.

SAMPLING TECHNIQUE:
For every HIV infected primigravida recruited, the next HIV uninfected primigravida of similar age and socioeconomic status was recruited as control till the total number of 180 was obtained in both groups. The socioeconomic status of the women was derived from their level of education and their husband's occupation.

INSTRUMENT OF DATA COLLECTION:
A proforma that was designed for this study was used to record the data obtained from the patients. The proforma had two sections, A and B. Section A asked questions on practice of malaria prevention of the enrollees while section B was the result of laboratory investigation at booking and follow up of the patients to detect clinical and recurrence of malaria fever.

Blood samples taken for routine ante natal investigations at booking were used for malaria parasite determination. Giemsa staining technique was used to stain the thin film for malaria parasite. Patients that came with features of clinical and recurrent malaria had blood smear for malaria parasite done to confirm malaria infection.

DATA ANALYSIS:
Data obtained was entered into a personal computer for analysis. Analysis was done using Minitab Inc 1998 statistical software. Qualitative data was presented as percentages while quantitative data was presented as mean and standard deviation. A chi square test was used to determine significant association between qualitative variables. A P- value of 0.05 or less was considered significant.

ETHICAL CLEARANCE:
Ethical clearance for the study was obtained from the ethical committee of Aminu Kano Teaching Hospital

RESULTS:
A total of 360 primigravidae were recruited for the study. Among them 180 were infected while 180 were uninfected by HIV.

Table 1.
Practice of Malarial prevention by HIV uninfected and HIV infected Primigravidae before booking

A. ITN USE					
VARIABLE	YES (%)	NO (%)	P-VALUE	OR	CI
Uninfected	97(53.89)	83(46.11)		1.00	
Infected	86(47.78)	94(52,22)	0.25	1.28	0.83-1.97
Total	183(50.83)	177(49.17)			
B. USE OF MALARIA PROPHYLAXIS					
Uninfected	85(47.22)	95(52.78)		1.00	
Infected	96(53.33)	84(46.67)	0.27	0.78	0.51-1.21
Total	181(50.28)	179(49.72)			
C. USE OF INSECTICIDES					
Uninfected	98(54.44)	82(45.56)		1.00	
Infected	83(46.11)	97(53.89)	0.11	1.40	0.90-2.16
Total	181(50.28)	179(49.72)			
D. ENVIRONMENTAL SANITATION					
Uninfected	102(56.67)	78(43.33)		1.00	
Infected	93(51.67)	87(48.33)	0.34	1.22	0.79-1.89
Total	195(54.17)	165(45.83)			

Table 2.
Prevalence of Malaria Parasites in blood smears of HIV uninfected and HIV infected Primigravidae at booking.

STATUS		Blood smear			
	Present (%)	Absent (%)	P-value	OR	CI
Uninfected	16(8.89)	164(91.11)		1.00	
Infected	42(23.33)	132(76.69)	0.0001	3.26	1.62-6.36
Total	58(16.11)	296(83.89)			

Table 3.
Prevalence of clinical Malaria among HIV infected and HIV uninfected Primigravidae.

STATUS	CLINICAL	MALARIA			
	YES (%)	NO (%)	P-VALUE	OR	CI
Uninfected	3(18.75)	13(81.25)		1.00	
Infected	28(66.67)	14(33.33)	0.001	8.67	1.87-46.37
Total	31(53.45)	27(46.55)			

Table 4.
Prevalence of recurrent malaria among HIV infected and HIV uninfected Primigravidae.

STATUS	RECURRENCE				
	YES (%)	NO (%)	P value	OR	CI
Uninfected	1(33.33)	2(66.67)		1.0	
Infected	26(92.86)	2(7.14)	0.037	26.0	1.05-1360
Total	27(87.07)	4 (12.93)			

There was no statistically significant difference in the practice of malarial prevention between the HIV infected and the HIV uninfected Primigravidae before booking as shown in table 1

The prevalence of malaria Parasitaemia was 23.33% among the HIV infected primigravidae, and 8.89% among the HIV uninfected primigravidae. The odds of occurrence of malaria Parasitaemia among the HIV infected primigravidae was three times higher than that among the HIV uninfected primigravidae. (OR= 3.26, CI = 1.62-6.36, p< 0.05) as shown in Table 2.

Table 3 showed the prevalence of clinical malaria was 66.67% among the HIV infected primigravidae compared to 18.75% among the HIV uninfected primigravidae. The odd of occurrence of clinical malaria among the HIV infected primigravidae was eight times higher than among HIV uninfected primigravidae, which is statistically significant. (OR= 8.67, CI= 1.87-46.37, p< 0.05) Recurrence of clinical malaria occurred in 92.86% of the HIV infected primigravidae, while among the HIV uninfected primigravidae it was 33.33%.The odd of recurrence of clinical malaria was twenty six times higher among HIV infected women compared to HIV uninfected primigravidae. Recurrence of clinical malaria was found to show statistically significant difference between HIV infected and HIV uninfected primigravidae with a P value of 0.037 as shown in Table 4.

DISCUSSION:
The prevalence of malaria Parasitaemia in the study population was found to be 32.22%. The HIV infected primigravidae accounted for

23.33%, while the HIV uninfected primigravidae accounted for 8.89%. This finding was similar to that from Awka in Nigeria[9,22] and from Chikwawa District, Southern Malawi[15] all of which reported high prevalence of malaria parasitaemia among primigravidae at booking. The prevalence was however lower than that in malaria non-endemic regions[16,17]. This difference could be because of partial immunity to malaria in malarial endemic areas, which is lowered by HIV infection. This can be appreciated in this review where the prevalence of clinical malaria was about eight times higher among HIV infected primigravidae and the risk of having recurrence of clinical malaria was twenty six times higher than among HIV uninfected women despite using three doses of IPT for HIV infected and two doses for HIV uninfected women during their antenatal period. This was similar to the finding of other workers from malaria endemic regions[18,19].

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The practice of malaria prevention before booking among the cases and controls, which did not show any statistically significant difference between the two groups, was necessary in order to confirm that the difference in malarial parasitaemia in their blood smears that were taken at booking, which was three times higher among the HIV infected was not as a result of differences in the practice of prevention of malarial infection among the two groups before booking[20,21,23].

The prevalence of clinical malaria which was eight times higher and recurrence of clinical malaria which was twenty six times higher among the HIV infected women despite three doses of IPTp was probably due to their higher susceptibility to malarial infection as a result of lowering of their partial immunity[10]. This call for a review of their malarial prophylaxis, in order to control the high prevalence of malarial parasitaemia among them, as it has been found to be associated with high feto-maternal morbidity and mortality, and failure of PMTCT of HIV[13,24].

This study agrees with the recommendation of other similar studies[18] that the malarial prophylaxis that is given to HIV infected women should be increased from three doses to four doses to correspond with the WHO recommended four antenatal visits in the focused antenatal care in order to ensure compliance and to reduce feto-maternal morbidity and mortality associated with malarial infection in pregnancy and also to ensure success of prevention of mother to child transmission of HIV infection.

CONCLUSION:

The prevalence of malaria parasitaemia among HIV infected primigravidae at booking was found to be three times higher than among the HIV uninfected primigravidae. Likewise the prevalence of clinical malaria and recurrent malaria infection was found to also be significantly higher in them despite the routine antimalarial prophylaxis.

Recommendation:

With the increased risk of clinical and recurrent malarial infection in HIV infected primigravidae, it is recommended that IPT_p should be increased from 3 to 4 doses among the HIV infected primigravidae. This should correspond with the four antenatal visits in the focused antenatal care in order to ensure compliance.

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