ORIGINAL ARTICLE

Prevalence of placenta *Plasmodium* parasitemia and pregnancy outcome in asymptomatic patients at delivery in a University Teaching Hospital in Nigeria

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Abstract

Background: Malaria is an important public health issue in pregnancy association with poor fetal and maternal outcome, especially in malaria endemic area like Nigeria.
Objective: The objective was to determine the prevalence of placental malaria in asymptomatic women in labor and to compare the fetal and maternal outcome between affected and unaffected women.
Subjects and Methods: A prospective cross-sectional study of 210 women who delivered at a tertiary health facility in Nigeria. Participants' peripheral venous blood, cord blood, and placental blood samples were examined microscopically for the presence of malaria parasite. Data collected were analyzed using SPSS version 16.
Results: Prevalence of placental malaria was 65.2%. Nulliparity was significantly associated with placental malaria (χ^2 = 21.32, P = 0.0000039, odds ratio [OR] =5.6). Poor compliance to intermittent preventive therapy was significantly associated with placental malaria (χ^2 = 16.67, P = 0.00004). The mean gestational age at delivery was 38.57 ± 1.7 weeks and 12.85% of women had preterm delivery. Sixty-seven (31.9%) women had anemia and malaria parasitemia was significantly associated with anemia (χ^2 = 8.34, P = 0.0039, OR = 2.6). Fourteen (6.67%) babies had low birth weight, but placental malaria was not significantly associated with low birth weight (χ^2 = 0.03, P = 0.87).
Conclusion: There is a high prevalence of placental malaria in the study population. Nulliparity, poor drug compliance, and maternal anemia were associated with placental malaria.

Key words: Malaria, Nigeria, placenta, pregnancy outcome, prevalence

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Introduction

Malaria infection is a major public health problem in sub-Saharan Africa, especially in pregnancy due to its association with poor fetal and maternal outcomes.[1,2] One of the targets of the millennium development goal (MDG) 6 is to reduce by 50% the prevalence of malaria in malaria endemic areas by 2015.[3] This target of the MDG is far from been achieved in sub-Saharan Africa due to the high prevalence of poverty, poor nutrition, illiteracy, and poor health care delivery. Consequently, the maternal and fetal morbidities and mortalities from this condition are still high.

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Malaria infection is caused by an obligate intracellular parasite of the genus *Plasmodium* and the recognized vector is the female anopheles mosquito, which strives well in dirty and swampy environments. The word "malaria" was derived from an Italian word, which means "bad air" as the disease is associated with bad air in swamps where mosquito breed freely.[4] Five species of the *Plasmodium* parasite are known to cause infection in humans. These are: *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, *Plasmodium malaria*, and *Plasmodium knowlesi* of which *P. falciparum* is the

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most deadly.[4,5] Knowledge of the life cycle of this parasite and its behavior is imperative in formulating preventive and treatment modalities.[6]

Successful control of malaria in pregnant women is a major step in curbing the burden of malaria in Africa. Control of malaria in pregnancy involves preventing infection as well as clearing parasitemia when it occurs.[6-13] Preventive measures as postulated by World Health Organization include keeping a clean environment, use of insecticide-treated nets (ITN), intermittent preventive treatment in pregnancy and effective case management of both complicated and uncomplicated cases.[10,13] The use of intermittent preventive treatment with sulfadoxine-pyrimethamine (SP) agent has been shown to be an effective in pregnancy and it has been adopted by the Federal Ministry of Health of Nigeria as the preferred chemoprevention method in pregnancy and has also postulated guidelines for its use.[12] Proguanil can also be used in women who are allergic to SP.[12] Despite adequate chemoprevention, placental malaria still occur because of sequestration of the malaria parasite in the placenta bed.[13] Placenta parasitemia incite release of inflammatory mediators, which affect placental function thus accounting for the adverse fetal outcome.[14-18] Clinical consequences of peripheral and placental malaria include maternal anemia, low birth weight/small for gestational age, preterm delivery, and increased perinatal mortality.[14,18-23] Although pregnant women in malaria endemic areas have higher rate of parasitemia compared to nonpregnant women, in some cases, infection is largely asymptomatic because some degree of preexisting immunity is retained during pregnancy.[15,17] However, malaria immune women are still susceptible to placental malaria because malaria parasites may become sequestered in the placenta and peripheral blood smears may fail to show evidence of infection.[14,17,18] This supports the fact that examining for malaria in the placenta is superior to peripheral blood film.[14,17]

This study seeks to determine the prevalence of placenta malaria and its adverse maternal and fetal outcomes among the study population with a view to developing strategies in reducing its menace.

Subjects and Methods

This study was a cross-sectional study involving 210 consecutive booked pregnant mothers who presented in labor, at the maternity section of the University of Port Harcourt Teaching Hospital. Ethical clearance for the conduct of this study was obtained from the Ethics Committee of the hospital, and written informed consent was obtained from all participants. A selected number of residents who were in charge of the labor ward during the period of the study were trained on important aspects of the research work, and they assisted in conducting the research. A designed proforma was used to obtain sociodemographic information from the participants such as compliance to malarial chemoprophylaxis and use of ITN. Other useful information regarding the participants was obtained from the participant's antenatal case notes. About 5 ml of venous blood was obtained from a peripheral vein of each participant and transferred into an EDTA bottle. At delivery, 5 ml of umbilical cord blood was also collected into an EDTA bottle and following delivery of the placenta, an incision of about 1 cm was made on the maternal surface of the placenta and 5 ml of blood was collected from the incision site into an EDTA bottle.[14] All specimens were correctly labeled and at the end of each working day the samples were sent to the microbiology and hematology laboratories of the hospital for analysis. Thick and thin smears of the peripheral (maternal), umbilical cord, and placenta blood were prepared for malaria parasite. In addition, the hemoglobin concentration of the peripheral and cord blood samples were also determined. Two senior microbiologists reviewed the microbiology slides, and a consensus decision was reached on each specimen before the results were released. Clinical correlates and outcome measures for each participant were documented. The socioeconomic status of the women that took part in this study was calculated using the participant's level of education and her husband's occupation as described by Olusanya *et al*.[24] Statistical analysis of generated data was carried out using SPSS for windows version16, (SPSS Inc , Armout, Chigaco USA.) Software Package (IBM, Armont USA) and means, percentages, standard deviations were also calculated. Statistical comparisons and test of significance between positive and negative groups were calculated using the Chi-square test for categorical variables and Student's "*t*"-test for means of continuous variables. Differences were considered significant if $P < 0.05$.

Determination of sample size

The sample size was calculated from the formula:[25] $n = z^2p\ (1 - p)/d^2$ with a prevalence of placenta malaria of 14%[23] and a relative precision rate of 5%, and allowing an attrition rate of 10%. The allowed minimum sample size for this study was 204. Therefore, 210 women were recruited for this study over a 3-month period from March 1, to May 2012.

Exclusion criteria

Women who were excluded are those who did not receive standard malaria preventive treatment in pregnancy, diagnosed or treated for malaria in the index pregnancy or had features suggestive of malaria in labor. Also excluded were mothers with multiple gestation, sickle cell disease, retroviral disease, and preterm prelabor rupture of fetal membranes.

Determination of malaria parasitemia

The prepared blood smears were air dried and subsequently stained with freshly prepared Giemsa stain at pH 7.2. The

stained smears were examined under × 100 oil immersion lens of a light microscope. Malaria diagnosis was based on identification of asexual stages of *Plasmodium* species on the thick blood film while thin smears were used for specie identification. Parasite density was determined by counting the number of parasites per high power field and ranged from + (1–10 parasites per 100 high power fields), ++ (>10 parasite per 100 high power fields), +++ (1–10 parasites in one high power field), and ++++ (>10 parasites in one high power field).[26] The slides are reported as negative if no parasite is identified per 100 power fields.

Determination of hemoglobin concentration

Hemoglobin concentration was determined by the cynmethemoglobin method.[27] About 20 µl of blood was mixed with 4 ml of Drabkins solution in a clean test tube and allowed to stand for 5 min for conversion. The optical density of the solution formed, which was read off the spectrometer at 540 nm, was proportional to the concentration of hemoglobin and this was compared to known hemoglobin standard calibrated charts to obtain the hemoglobin concentration of the tested sample.

Results

A total of 137 of the 210 placenta blood films examined were positive for malaria parasite giving a prevalence of placental malaria of 65.2%. A total of 122 (58.1%) of the 210 maternal blood films examined were positive for malaria parasite while 106 (50.5%) of the 210 cord blood examined were positive for malaria parasite. A total of 109 (89.3%) of the 122 positive peripheral blood films had mild (+) parasitemia, while moderate (++) parasitemia was observed in 13 (10.7%) women. Mild and moderate parasitemia were observed in 135 (98.5%) and 2 (1.5%) of the 137 positive placental blood films respectively, whereas all the 106 positive cord blood films had only mild parasitemia. *P. falciparum* was the only *Plasmodium* specie detected in all the positive blood films.

Table 1 shows the relationship between age and malaria parasitemia in peripheral, placental, and cord blood samples of the participants. The mean age of the women was 30.4 ± 4.8 years and ranged from 18 to 43 years. All of the six women that were below 20 years of age had placental malaria while 131 of the 204 women that were aged 20 years and above had placental malaria ($P = 0.07$).

Table 2 shows the sociodemographic characteristics of the women.

The mean parity of the study population was 1.5 ± 1.4 and ranged from 0 to 6. Fifty-two (85.2%) of the 61 nulliparous women had placental malaria, while 76 (51.0%) of the 149 women with higher parity, that is, Para 1 and above had placental malaria ($\chi^2 = 21.32$, $P = 0.0000039$, odds ratio [OR] =5.55). Table 3 shows the relationship between the parity of the women and malaria parasitemia. The mean gestational age at booking was 20.7 ± 6.1 weeks, while the mean gestational age at delivery was 38.6 ± 1.7 weeks and range from 33 to 42 weeks. About 27 (12.9%) women delivered before 37 weeks of gestation, while 183 (87.1%) women delivered at term. A total of 18 (13.1%) of the 137 women who had placenta malaria had preterm delivery, while 9 of the 73 women who did not have placental malaria had preterm delivery ($\chi^2 = 0.03$, $P = 0.86$) [Table 4].

One hundred and eighty (85.7%) women took SP for malaria chemoprophylaxis while 30 (14.3%) women had proguanil for malaria chemoprophylaxis. One hundred and forty-nine (82.8%) of the 180 women who had SP were compliant while 5 (16.7%) of the 30 women who

Table 2: Sociodemographic characteristics

Characteristics	Frequency (%)
Marital status	
Married	208 (99)
Single	2 (1)
Total	210 (100)
Educational level	
Tertiary	155 (73.8)
Secondary	55 (26.2)
Primary	0 (0)
Total	210 (100)
Socioeconomic status	
Upper class	98 (47.1)
Middle class	78 (37.5)
Lower class	32 (15.4)
Total	208 (100)

Table 1: Relationship between maternal age and malaria parasitization

Age (years)	Frequency number (%)	Placental blood film (*n* (%))		Maternal blood film (*n* (%))		Cord blood film (*n* (%))		χ^2	*P* value
		Positive	Negative	Positive	Negative	Positive	Negative		
<20	6 (2.9)	6 (100)	0 (0)	6 (100)	0 (0)	5 (83.3)	1 (16.7)		0.07
20-24	8 (3.9)	4 (50)	4 (50)	2 (25)	6 (75)	1 (12.5)	7 (87.5)		0.02
25-29	87 (41.5)	59 (67.8)	28 (32.2)	55 (63.2)	32 (36.8)	42 (48.3)	45 (51.7)	0.44	0.50
30-34	67 (31.9)	45 (67.2)	22 (32.8)	41 (61.2)	26 (38.8)	43 (64.2)	29 (35.8)	0.16	0.68
>35	42 (20.0)	23 (54.8)	19 (45.2)	18 (42.9)	24 (57.1)	20 (47.6)	22 (52.3)	2.54	0.11
Total	210 (100)	137	73	122	88	106	104		

Table 3: Relationship between parity and malaria parasitization

Parity	Frequency number (%)	Placental blood film (*n* (%))		Maternal blood film (*n* (%))		Cord blood film		χ^2	*P* value
		Positive	Negative	Positive	Negative	Positive	Negative		
Primigravida (Para 0)	61 (29.0)	52 (85.2)	9 (14.8)	47 (77)	14 (23)	33 (54)	28 (46)	21.32	<0.01
Primipara (Para 1)	59 (28.1)	32 (54.2)	27 (45.8)	28 (47.5)	31 (52.5)	22 (37.3)	37 (62.7)	4.38	0.036
Multipara (Para 2-4)	85 (40.5)	50 (58.8)	35 (41.2)	45 (53)	40 (47)	49 (57.6)	36 (42.4)	2.59	0.10
Grandmultipara (Para≥5)	5 (2.4)	3 (60)	2 (40)	2 (40)	3 (60)	2 (40)	3 (60)		0.57
Total	210 (100)	137	73	122	88	106	104		

Table 4: Gestational age at delivery and malaria parasitization

Gestational age (weeks)	Frequency number (%)	Placental blood film (*n* (%))		Maternal blood film (*n* (%))		Cord blood film (*n* (%))		χ^2	*P* value
		Positive	Negative	Positive	Negative	Positive	Negative		
28-36	27 (12.9)	18 (66.7)	9 (33.3)	18 (66.7)	9 (33.3)	11 (40.7)	16 (59.3)	0.03	0.86
37-42	183 (87.1)	119 (65.0)	64 (35.0)	104 (56.8)	79 (43.2)	95 (52.0)	88 (48.0)		
Total	210 (100)	137	73	122	88	106	104		

Table 5: Fetomaternal outcome

Outcome	Frequency number (%)	Placental blood film (*n* (%))		Maternal blood film (*n* (%))		Cord blood film (*n* (%))		χ^2	*P* value
		Positive	Negative	Positive	Negative	Positive	Negative		
Maternal anemia (Hb<10 g/dl)	67 (31.90)	53 (79.1)	14 (20.9)	49 (73.1)	18 (26.9)	43 (64.2)	24 (35.8)	8.34	0.0039
Low birth weight (birth weight<2.5 kg)	14 (6.67)	10 (71.4)	4 (28.6)	10 (71.4)	4 (28.6)	8 (57.1)	6 (42.9)	0.03	0.87
Birth asphyxia (Apgar scores<7 at 1st min)	12 (5.71)	6 (50)	6 (50)	5 (41.7)	7 (58.3)	5 (41.7)	7 (58.3)		
Perinatal death	2 (0.95)								

Hb=Hemoglobin

were on proguanil were compliant with the drug, and the difference was statistically significant (χ^2 = 57.47, *P* = 0.0000000). Forty-nine (87.5%) of the 56 women in the entire study population who were not compliant with their preventive therapy had placental malaria, while 88 (57.1%) of the 154 women in the entire study population who were compliant with their chemoprophylaxis had placental malaria. The difference was statistically significant (χ^2 = 16.67, *P* = 0.00004). Forty-seven (22.4%) women used ITN in the index pregnancy, while 163 (77.6%) did not use ITN.

Sixty-seven (31.9%) had hemoglobin concentration <10 g/dl (anemia) at delivery. Fifty-three (38.7%) of the 137 women with placental malaria had anemia while 14 (19.2%) of the 73 women without placental malaria had anemia. The difference was statistically significant (χ^2 = 8.34, *P* = 0.0039, OR = 2.6). Seven (3.5%) babies had cord blood hemoglobin concentration <15 g/dl.

The mean birth weight of the study population was 3.4 ± 0.5 kg and ranged from 2.3 to 4.6 kg. The mean birth weight in mothers with placental malaria was 3.3 ± 0.5 kg, while the mean birth weight of mothers without placenta malaria was 3.5 ± 0.5 kg, and the difference was not statistically significant (*t* = 1.76, *P* = 0.081). Fourteen (6.67%) of the 210 babies delivered had a birth weight <2.5 kg (low birth weight). Ten (7.3%) of the 137 women with placental malaria had low birth weight babies while 4 (5.5%) of the 73 women without placental malaria had low birth weight babies, and the difference was not statistically significant (*P* = 0.43). Twelve (5.7%) babies had Apgar scores of 6 or less (birth asphyxia) at the 1st min of birth. There were two cases of perinatal death from severe birth asphyxia giving a perinatal mortality rate among the study populations was 9.52/1000 total births. The two babies that died did not have cord blood malaria parasitemia. Table 5 shows the fetomaternal outcome.

Discussion

The prevalence of placental malaria of 65.2% detected in asymptomatic women in this study is higher than both reported by Mokuolu *et al*.[23] and Mockenhaupt *et al*.[14] in a malaria endemic regions. Sarr *et al*. have reported a lower prevalence of 10.9% from a malaria low transmission community.[28] These variations in prevalence may be due to variations in community-acquired immunity, sociodemographic characteristics of the study population, case selection, use and resistance to malaria chemoprophylaxis and the diagnostic tools employed in the detection of the parasite.[14,15,17-19,23,28] It was demonstrated in this study that despite the absence of malaria parasite in peripheral and cord blood, placenta parasitemia was demonstrated in some patients. This is in support of the findings by Mockenhaupt *et al*.[14] but

at variance with the findings by Mokuolu *et al*.[23] As observed in the study by Tako *et al*.,[15] nulliparity in this study was significantly associated with placental malaria. This study demonstrated that primigravidae are 5.6 times more likely to have placental malaria than women with higher parity. Primigravidae are prone to placental malaria because they express specific placental receptors that facilitate binding of parasitize erythrocytes to the placental tissue, but this is less likely in the multigravidae due to subsequently acquired blocking antibodies that prevent such binding from occurring.

The prevalence of preterm delivery among women with placental malaria was comparable with that reported by Mokuolu *et al*.[23] but higher than that by Sarr *et al*.[28] whose study was carried out in a malaria low transmission area suggesting that regions with high malaria transmission are more likely to be associated with adverse fetal outcome.

In terms of malaria prevention therapy, a significant number of placental malaria was observed in women who were not compliant with their drugs and compliance was better with SP compared to proguanil. Proguanil is not used as a first line chemoprophylaxis in the study population, but mostly indicated in women who are allergic to or do not tolerate SP and this explains why only 14.3% of women were on proguanil. Proguanil is administered daily, and the fact that proguanil is more expensive may account for its poor compliance compared to SP, which is normally taken twice throughout the course of the pregnancy. The implementation of the direct observed treatment as recommended by the Federal Ministry of Health of Nigeria[7] for the administration of SP, which is currently not practiced among the study populations may improve compliance with SP, and this may reduce the prevalence of placental malaria in the study population. Most of the women in this study do not use ITN.[8] Educating our women on the role of ITN in preventing malaria will impact positively in reducing the prevalence of malaria. Furthermore, government should intensify efforts in ensuring that ITN are readily available at all health facilities and ITN should be distributed to all antenatal women at booking in order to encourage its utilization.

Placental malaria in this study was associated with a 2.6-fold increased risk of maternal anemia. The prevalence of anemia in this study was 31.9% with most been mild anemia and none needed blood transfusion during the period under review. The causes of anemia in pregnancy are multifactorial.[29-31] Poor nutrition and poverty were unlikely causes of anemia in the study population as most of the women that took part in this study had tertiary level of education and were of middle or upper socioeconomic class. Efforts were made in this study to exclude other causes of anemia in pregnancy as women with sickle cell anemia, multiple pregnancy and retroviral disease were excluded from this study. However, it is still difficult to attribute this prevalence of anemia solely to the effect of malaria parasitemia as other causes such as helminthiasis, malabsorption syndrome and food idiosyncrasies were not excluded.

The prevalence of cord blood malaria parasitemia noted in this study, which was 50.5%, was rather high. Congenital malaria occurs due to transplacental transmission of parasitized erythrocytes.[32-34] This was supported in this study by the fact that all cases of positive cord blood smears also had positive placental blood smears. It is worthy to note that none of the babies with positive cord blood had clinical manifestation of malaria at birth. This may be due to the fact that all the positive cases had mild parasitemia as well as the effect of maternal IgG antibodies transferred to the fetus. These antibodies may delay or modify the onset of clinical manifestation of congenital malaria.[32,33] It is therefore important that babies with positive cord blood should be followed-up closely as some cases of congenital malaria have been reported several weeks after birth.[33] Therefore, further study to correlate the relationship between cord blood parasitemia and the baby's peripheral blood parasitemia as well as follow-up of the babies over a period of time to determine the prevalence of clinical malaria in positive cases is required.

The mean birth weight as well as the prevalence of low birth weight between women with placental malaria and those without placental malaria were not significantly different, and the prevalence of low birth weight of 6.7% recorded in this study was similar to the 6.8% reported by Mokuolu *et al*.[23] The perinatal mortality rate of 9.52/1000 total births recorded in this study was not due to placenta malaria.

Conclusion

There is a high prevalence of placental malaria in the study population. Nulliparity, poor drug compliance and maternal anemia were significantly associated with placental malaria, but preterm delivery and low birth weight were not significantly associated with placental malaria. Case selection may account for this fetal outcome. The implementation of direct observation therapy and provision of ITN may reduce the prevalence of placental malaria and improve the fetal and maternal outcome in the study population.

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