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## **Prevalence of Congenital Malaria in Ilorin, Nigeria**

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### **Abstract**

*A Seven months (March-September 2006) study on the prevalence of congenital malaria was carried out at the labour unit of three different hospitals within Ilorin metropolis: Eytayo Hospital, Surulere Medical Hospital and Children Specialist Hospital Centre Gboro Ilorin. A total of 130 blood samples were collected from the mothers and their newborn babies and examined for malaria parasite using both thin and thick films. Maternal packed cell volume (PCV), and genotype was also determined using haematocrit method and cellulose acetate electrophoresis respectively. The prevalence rate of maternal, fetal, placental and cord parasitaemia were 37(28.46%), 29(22.31%), 33(25.38%) and 30(23.08%) respectively. Malaria infected maternal blood had a mild reduction in PCV level ( $p<0.05$ ). Genotype showed strong correlation with maternal, fetal, placental and cord parasitaemia ( $p<0.05$ ). However, the effect of malaria prophylaxis was shown to be more protective for the placental parasitaemia ( $p<0.05$ ). Finally*

*maternal age and parity did not show strong correlation with Maternal, fetal, placental and cord parasitaemia ( $p>0.05$ ).*

Keywords: Malaria; Mothers; Newborns

## **Introduction**

Congenital malaria is defined as malarial parasites demonstrated in the peripheral smear of the newborn within the first twenty four hours to seven days of life [Ezechukwu et al., 2004]. This results from malaria parasites crossing the placenta from maternal into the fetal circulation, although the mechanism of transplacental passage is obscure. The localization of parasites within the placenta may jeopardize materno-foetal relationship, thus affecting the supply of nutrients to the foetus [Uko et al., 1999]. It has been postulated that pregnancy can make women more susceptible to malaria infection. This has been attributed to impairment of both humoral and cell mediated immunity as a result of pregnancy [Okonofua et al., 1990].

Malaria in pregnancy is a common cause of severe maternal anaemia and these results from haemolysis of both parasitized and unparasitized red blood cells [Jimoh, 2004]. This is common in primigravidae compared to multigravidae. This study was designed to determine the prevalence of congenital malaria at Ilorin, Kwara State using three hospitals: Eyitayo Hospital, Surulere Medical Centre and Children Specialist Hospital Centre Gboro. The first two representing private and the last public hospital respectively.

## **Materials and Methods**

### **Study Area**

The study was carried out at three different Hospitals; Eyitayo Hospital, Surulere Medical Centre and Children Specialist Hospital Centre Gboro, all in Ilorin, Kwara State over a period of seven months (March- September 2006). A hundred and thirty mothers and their babies delivered at the labour wards of these Hospitals were used after due parental and consultant consent. Questionnaire was applied to mothers and appropriate information filled such as maternal age, parity, past clinical history of malaria, anti malaria drug (such as chloroquine, amodiaquine in combination with sulphadoxine-pyrimethamine) used for treatment and prophylaxis were obtained from them. Laboratory test such as packed cell volume (PCV) and Haemoglobin Genotype were carried out on the maternal blood samples while malaria parasite density estimations was done on both maternal and fetal and cord

blood samples. Pregnant mothers presenting at the labour ward with history of fever were excluded from the study.

### **Blood Collection**

Aseptic procedures were adopted in the collection of the blood samples. The placenta was washed with clean water immediately after delivery before the blood was collected. The cord was cleaned with 70 percent alcohol to avoid maternal blood contamination before it was dispensed into EDTA bottles. Fetal blood was obtained from a peripheral vein on the dorsum of the hand using a 23G needle. Venous maternal blood was obtained using 5ml syringe and dispensed into EDTA bottles.

### **Preparation of Thick Blood Film**

Thick blood films for estimation of malaria parasite density was carried out according to the method of Greenwood and Armstrong [1991] described by Cheesbrough [2005]. After appropriate staining and drying, slides were examined microscopically using the x 100 (oil immersion) objectives.

### **Procedure for estimation of malaria parasite density**

This was carried out using the thick blood film method of Greenwood and Armstrong, [1991]. The average number of parasites counted per high power field ( 100 x objectives) was multiplied by 500. Between 10-15 fields were counted for each slide. The result is given per  $\mu$ l of blood.

### **Procedure for genotype Determination and packed cell volume Estimation**

The procedure for the determination of haemoglobin genotype and packed cell volume (PCV) of the subjects were carried out according to the methods described by [Dacie and Lewis, 1991].

### **Statistical Analysis**

The data generated in this study were analyzed using student's t -test for equality of means while Chi-square test was used to test for independence among the categorical variables at  $\alpha$  level of 0.05 (level of significance).

### **Results**

The prevalence rates of maternal, fetal, placental and cord parasitaemia were 37(28.46%), 29(22.31%), 3(25.38%) and 30(23.08%) respectively (Table1). Maternal, fetal, placental and cord blood mean parasite density were  $32450 \pm$

128.46,  $12245 \pm 62.33$ ,  $25660 \pm 110.70$  and  $13355 \pm 65.06$  respectively (Table 2).

Mean maternal PCV was  $32.81 \pm 0.42$ , those with parasitaemia were  $30.86 \pm 3.56$  and those without parasitaemia were  $33.58 \pm 5.00$  (Table 3). Malaria infected maternal blood had a mild reduction in PCV level. Maternal PCV showed strong correlation with maternal parasitaemia ( $p < 0.05$ ).

With reference to Genotype in Table 4, it was discovered that parasitaemia was higher in individual with haemoglobin genotype AA. This was followed by individual with haemoglobin genotype AS. Only one patient had haemoglobin genotype AC but showed negative parasitaemia except for the placenta, which showed positive parasitaemia. Genotype showed strong correlation with maternal, fetal, placental and cord parasitaemia ( $p < 0.05$ ). Most of the women who took malaria prophylaxis still showed evidence of malaria parasitaemia. Malaria prophylaxis did not seem to affect maternal parasitaemia but was shown to be more protective for the placental parasitization ( $p > 0.05$ ) (Table 3).

The mean gestational age and parity of the patients were  $2.11 \pm 0.05$  years and  $1.63 \pm 0.04$  years respectively. The mean age of mothers with parasitaemia was  $1.97 \pm 0.50$  years and those without parasitaemia were  $2.16 \pm 0.56$  years (Table 3). Age group 14-20 years had the highest prevalence of 38.5% maternal, 30.8% fetal, 30.8% placental and 30.8% cord parasitaemia which was closely followed by age group 21-30 years which had 31.1%, 24.4%, 27.8%, 25.6% maternal, fetal, placental and cord parasitaemia respectively while the lowest prevalence of 14.8% maternal, 11.1% fetal, 11.1% placental and 11.1% cord parasitaemia was seen in age group 31-40 years (Table 5). Maternal age showed no strong correlation with maternal, fetal, placental and cord parasitaemia ( $p > 0.05$ ). The lowest parasitaemia was seen in age group 31-40 years (Table 5).

Mean parity of mothers with parasitaemia was  $1.59 \pm 0.50$  and those without parasitaemia were  $1.65 \pm 0.48$  (Table 3). Parasitaemia was higher in primigravidae (Table 5). Maternal age and parity showed no strong correlation with maternal, fetal, placental and cord parasitaemia ( $p > 0.05$ ).

## **Discussion**

Congenital malaria is defined as malarial parasites demonstrated in the

peripheral smear of the newborn within the first twenty four hours to seven days of life [Ezechukwu et al., 2004]. Congenital malaria hitherto thought to be rare is becoming increasingly prevalent.

The prevalence rates of maternal, fetal, placental and cord parasitaemia recorded in this study were 28.4%, 22.31%, 25.38% and 23.08% respectively (Table 1). In Maputo, Mozambique 17.3% of screened mothers was infected and so were 1.5% of their newborn babies [Bergstrom et al., 1993]. In the highlands of Jos, northern Nigeria, 44.51% of mothers and 28.2% of their newborn were infected [Egwunyanga et al., 1995]. Higher prevalence of congenital malaria in some other communities in Nigeria have been reported by [Jimoh, 2004; Mackay, 1934; Obiajunwa et al., 2005; Rienhardt et al., 1978] which were 32%, 55%, 26.61% and 46.7% respectively. However, this present study has also established that congenital malaria is a common occurrence amongst the newborns in Ilorin, Kwara State, Nigeria

The mean parasitaemia density in maternal, placental, fetal and cord blood were  $32450 \pm 128.46$ ,  $12245 \pm 62.33$ ,  $25660 \pm 110.70$  and  $13355 \pm 65.06$  respectively (Table 2). The highest parasite density was seen in maternal blood while the least was seen in fetal blood. The low prevalence rate of parasite density in fetal blood could be as a result of barrier offered by the placental to the foetus. This is in agreement with the finding of Ezechukwu et al., [2004].

Maternal PCV had a strong correlation with maternal parasitaemia ( $p < 0.05$ ) (Table 3). The significant reduction in PCV level indicates a relationship between malaria parasitaemia and anaemia. This may be due to the fact that pregnant women with malaria parasitaemia are likely to be anemic though may or may not be severe. This is in line with Chimsuku et al., [1994] report.

Women with haemoglobin genotype AA showed high prevalence of parasitaemia with about 41.6% as compared with 5% recorded in haemoglobin genotype AS women (Table 4). This is in accordance with the findings of Cheesbrough, [2005] who reported that sickle cell trait carriers have lower parasite densities and are more protected against death from severe malaria and the development of hyper reactive malaria splenomegaly compared with haemoglobin genotype AA (HbAA) and haemoglobin genotype SS (HbSS) individuals. Genotype showed strong correlation with maternal, fetal, placental and cord parasitaemia ( $p < 0.05$ ).

However, malaria prophylaxis was shown to be more protective for the placental parasitization only (Table 4). This disagrees with Jimoh [2003] who reported that malaria prophylaxis was more protective for the placental parasitization and fetal parasitaemia.

Furthermore, it was discovered that age group 14-20 and 21-30 years were more susceptible than older ones (Table 5). This confirmed that as women get older, their resistance to malaria becomes higher due to improvement in host immunity. This is in consonance with the work of McGregor et al., [1983] who reported a decline in malaria prevalence as age increase and that with improved host immunity thus reducing susceptibility in later years.

Finally, prevalence of malaria parasitaemia was higher in primigravidae compared to multigravidae (Table 5). This could be as a result of immunosuppressant action of hormone notably cortisone on cell mediated immunity produced regularly during pregnancy by primigravidae. This is in accordance with the work of [9].

Therefore in order to reduce the incidence of congenitally acquired malaria, the need for adequate antenatal care geared towards providing early diagnosis and prompt treatment of malaria should be practiced. There is need for Government at all levels to provide should distribute antimalaria drugs free of charge to all pregnant women.

### **Conclusion**

In Conclusion, pregnant women should be encouraged to register for antenatal care early and they should be placed on malaria prophylaxis immediately.

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Table 1: Prevalence rates of malaria parasitaemia in maternal, fetal, and placental cord blood.

GROUP	Positive Parasitaemia (%)	Negative Parasitaemia (%)	Total (%)
Maternal	37 (28.46%)	93 (71.54%)	130(100%)
Fetal	29 (22.31%)	101(77.69%)	130(100%)
Placental	33 (25.38%)	97 (74.62%)	130(100%)
Cord blood	30 (23.08%)	100 (76.92%)	130(100%)

Table 2: Prevalence of malaria parasitaemia and parasite density in maternal, fetal, placental and cord blood

Nature of sample	No of sample	No of positive sample	Mean parasite density ( $\mu$ l )
Maternal blood	130	37(28.46%)	32450+128.46
Fetal blood	130	29(22.31%)	12245+ 62.33
Placental blood	130	33(25.38%)	25660+ 110.70
Cord blood	130	30(23.08%)	13355+ 65.06

Table 3: Mean values of maternal and fetal indices in all patients, those with positive and negative parasitaemia.

_Variable (means)	All patients	Positive parasitaemia	Negative parasitaemia	p-value
Maternal Gestational age	2.11± 0.05	1.97± 0.50	2.16±0.56	0.076
Parity	1.63±0.04	1.59±0.50	1.65±0.48	0.059
Maternal PCV	32.81± 0.42	30.86±3.56	33.58±5.00	0.003

P>0.05 showed no strong correlation P<0.05 showed strong