Possible Risk Factors for Congenital Malaria at a Tertiary Care Hospital in Sagamu, Ogun State, South-West Nigeria

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Summary

Congenital malaria, defined as the presence of malaria parasites in the erythrocytes of newborns aged <7 days, was considered rare in endemic areas until recent studies started reporting high prevalence rates. Various theories have been postulated to explain this phenomenon, but they are not proven conclusively from research. Against this background, a prospective study was designed with the following objectives. To determine the prevalence of congenital malaria parasitaemia and identify possible risk factors amongst newborns delivered in O.O.U.T.H Sagamu, Ogun State. Over a 6-month period, 192 live newborns and their mothers were consecutively recruited into the study. Within 3 days of life, neonatal peripheral blood samples were collected for malaria screening by blood film microscopy and detection of plasmodium lactate dehydrogenase (pLDH) with the OptiMAL/Rapid Malaria Test kit. Maternal peripheral blood samples were taken simultaneously, to check for malaria infestation by blood film microscopy, and questionnaires were administered on the mothers to identify possible factors associated with the development of neonatal parasitaemia. Neonatal clinical and laboratory data were recorded in a proforma designed for the study. Data analysis was done with Epi-info version 6 software and level of significance set at <5%. Twenty-one of 192 newborns delivered in O.O.U.T.H within the study period were diagnosed as having congenital malaria by blood film microscopy, giving a prevalence rate of 10.9%. The main identified innate neonatal risk factor for congenital malaria parasitaemia was prematurity. First-order pregnancy, history of fever within 3 months of delivery and peripheral parasitaemia at delivery (p < 0.001) were the variables that were significantly higher in the mothers of the parasitaemic newborns. We conclude that congenital malaria parasitaemia in tropical endemic areas is not rare. Pre-term neonates, infants of primigravidae, women with history of fever within 3 months of delivery and women with post-partum peripheral parasitaemia may benefit from routine screening for malaria.

Key words: congenital malaria; risk factors, newborns, Nigeria.

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Background

Congenital malaria, defined as the presence of malaria parasites in the erythrocytes of newborns <7-days old, was first described in 1876 [1, 2]. Since then, more than 350 cases have been reported in English literature. Most of these cases were reported from areas of unstable malaria, amongst offspring of non-immune women that had travelled to the tropics, or women from malaria endemic areas whose immunity had fallen [3]. In malaria endemic areas, however, the issue of the existence of congenital malaria was a subject of controversy. Although some researchers had reported relatively high rates of newborn peripheral parasitaemia in the first half of the twentieth century [4–8], the general
understanding and belief was that the incidence of congenital malaria in endemic areas was very low, with rates ranging from 0.18% to 0.95% [9–12]. These low rates were ascribed to various mechanisms; the placental barrier and the enhanced immunity of the endemic population with consequent passive transfer of maternal anti-malaria antibodies to the fetus during pregnancy [13], the high level of haemoglobin F which retards plasmodium maturation [14], and the para-amino benzoic acid (PABA) deficient breast milk diet, which deprives the parasites of folic acid essential for growth [15].

Since 1990, however, studies from various sites in Africa have reported high prevalence rates, and within Nigeria, rates range from 4.9% in Benin to 46.7% in Ile-Ife [1, 16–24]. Theories postulated to explain this increased prevalence include: lowering of maternal immunity from regular intake of anti-malarial drugs before and during pregnancy [25, 26], increased virulence of the malaria parasite [20], emergence of chloroquine resistance and changing patterns of drug sensitivity [19, 27] and increased awareness and investigation for malaria during the early neonatal period [28]. These theories have however not been proven conclusively by research. Against this background, this study was undertaken to ascertain the prevalence of congenital malarial parasitaemia and identify possible risk factors in newborns delivered in a tertiary care hospital, in South-West Nigeria.

Methods

The study was a descriptive, observational survey spanning a period of 6 months, August 2004 to January 2005. It was conducted in the maternity and neonatal wards of the Olabisi Onabanjo University Teaching Hospital Sagamu, Ogun State. This state lies within the rain forest belt, and within it, malaria is hyper-endemic, being responsible for >30–50% of febrile illnesses in children below the age of 5 years [29, 30]. The hospital is one of three tertiary health institutions in Ogun State and offers services to women and their newborns from Sagamu and environs, irrespective of their referral status. Approval to conduct the study was obtained from the Scientific and Ethical Review Committee of the hospital, and informed consent received from the mothers of the newborn babies prior to their enrolment in the study.

All live newborns of consenting mothers irrespective of gestational age, that were delivered in the study centre qualified for inclusion in the study. Babies with gross congenital anomalies or those requiring blood transfusion prior to recruitment were excluded.

To achieve results at the 95% confidence level for a finite population, the sample size was calculated in two phases. First, a general formula was applied to obtain the sample size required to achieve results at the 95% confidence level in an infinite population. A second formula was then used to correct for a finite population [31, 32].

1. General formula: 
\[
N = \left( \frac{Z^2}{d^2} \right) \left( p \cdot q \right)
\]

Where 
- \(N\) = minimum sample size representative of an infinite population (>10,000); 
- \(Z\) = 1.96 (at 95% confidence interval); 
- \(p\) = estimated prevalence of parasitaemia (50%); 
- \(q\) = 1 - \(p\); 
- \(d\) = The maximum tolerable difference between the true population and sample rate (5%).

The sample size thus calculated was 384.

2. Correction formula for a finite population
\[
n' = \frac{n}{(1 + (n/N))}
\]

Where 
- \(n'\) = new sample size based on inclusion of finite population correction factor; 
- \(n\) = The previously calculated sample size (384) for infinite populations; 
- \(N\) = the estimated total reference population size = 360 (30 births/month × 12 months).

The corrected sample size was thus 186.

An additional 10% was added to allow for attrition from withdrawals, non-respondents, spillage of blood samples, etc. [19], thus, a total of 205 babies and their mothers were enrolled in the study. Of these, 192 babies (168 singletons, nine sets of twins and two sets of triplets) and their mothers (179) were studied; hence, the attrition rate was 6.3%.

Data Collection

After resuscitation of the newborns at delivery, physical examination was done and anthropometric measurements taken. For each anthropometric parameter, mean values to the nearest one decimal place were recorded after three consecutive readings, and the data were entered into collection forms. The gestational age of the subjects was determined by a combination of mother’s last menstrual period and Dubowitz scoring [33]. Where there was a discrepancy of 2 weeks or more, the assessed gestational age by Dubowitz scoring was upheld [34]. The newborns were then classified as being appropriate for gestational age (AGA), small for gestational age (SGA) or large for gestational age (LGA), based on their gestational age and birth weight using Olowe’s chart [35]. Within 72 h of delivery, neonatal and maternal peripheral blood samples were collected directly into an EDTA bottle for malaria screening by blood film microscopy at the hospital’s service laboratory.
Laboratory analysis
Identification, quantification and speciation of malaria parasites and neonatal haematocrit estimation were done within 48 h of blood collection using standard procedures [36–38], the details of which are described in a previous report [39].

Administration of questionnaires and data analysis
Semi-structured questionnaires were administered on the mothers to identify possible factors associated with the development of neonatal parasitaemia. Information sought included personal biodata such as age, parity, address and accommodation type, educational attainment and occupation. Information about probable maternal risk factors for congenital malaria infestation such as history of malaria in pregnancy, use of malaria preventive strategies, blood transfusion in pregnancy, fever in last 3 months of pregnancy were also sought. Maternal socio-economic status was determined by allocating index scores based on educational level and occupation as described by Oyedeji [40].

Data analysis was done with Epi-info version 6 software. To ensure the accuracy of data entry, a ‘CHECK’ file was created to verify entries and ascertain that data were entered within specified ranges, and the ‘VALIDATE’ option of the EPI-INFO menu was used to validate another series of the same records. Discrepant entries were identified and corrected. For the descriptive aspects of analysis, frequency distributions were generated for all categorical variables. Mean and standard deviations and frequency distributions were generated for all categorical variables in parasitaemic and aparasitaemic newborns. The parasitaemic babies had lower mean Apgar scores, gestational ages and anthropometric indices than their aparasitaemic counterparts, though the differences were not statistically significant. The parasitaemic babies also had lower mean packed cell volumes (37% ± 6.92 versus 40.3% ± 6.97, t = 1.88, p = 0.06). The only neonatal variable that was significantly associated with congenital malaria parasitaemia was prematurity. Figure 1 illustrates the frequency of parasitaemia by maturity group.

Results
Prevalence of congenital malaria
Twenty-one (10.9%) of 192 newborns had positive blood films, while 171 (89.1%) had negative blood films. Thus, the prevalence of congenital malaria in the study population was 10.9%. The predominant species identified in 20 (95.2%) blood films was Plasmodium falciparum. Plasmodium malariae was identified in one blood film. Parasite counts ranged from 17 to 2940 parasites/μl, with a mean count of 793 ± 845 parasites/μl.

Possible factors associated with congenital malaria
Table 1 shows the results of the comparison of demographic, anthropometric and haematological variables in parasitaemic and aparasitaemic newborns. The parasitaemic babies had lower mean Apgar scores, gestational ages and anthropometric indices than their aparasitaemic counterparts, though the differences were not statistically significant. The parasitaemic babies also had lower mean packed cell volumes (37% ± 6.92 versus 40.3% ± 6.97, t = 1.88, p = 0.06). The only neonatal variable that was significantly associated with congenital malaria parasitaemia was prematurity. Figure 1 illustrates the frequency of parasitaemia by maturity group.

Demographic and obstetric profiles of mothers
The mean ages of mothers in the comparison groups were 31.3 ± 6 and 30.3 ± 5 years, respectively, thus, they were not significantly different (t = 0.76, p = 0.45). Their socio-demographic profiles were also similar, as majority of the mothers in both groups were from the Yoruba tribe. None of the Ibo or Hausa women studied delivered babies with parasitaemia. As shown in Table 2, about a third of the mothers of parasitaemic babies (33.3%) were primiparous, in contrast to the mothers of non-parasitaemic babies, of which only 13% were primiparous. Analysis of the occurrence of neonatal parasitaemia in relation to maternal parity revealed that 25% of the primiparous women in the study population delivered parasitaemic babies in contrast to 9% of the multiparous women. These differences were statistically significant (p = 0.04). Other obstetric variables such as booking status, mean gestational age at booking, and mean number of hospital visits that were compared in the two groups of women were however found to be similar.

Use of malaria preventive strategies by mothers
The use of malaria prevention strategies was similar in mothers of babies with and without parasitaemia. The most common strategies employed by mothers in the study population were insecticides (82.1%) and window nets (80.4%). Anti-malarial prophylaxis came in third place (51%) as only ninety-one of the mothers in the study population used anti-malarial prophylaxis during pregnancy. The use of anti-malarial prophylaxis was lower among mothers of parasitaemic babies (42.9%) than mothers of non-parasitaemic babies (51.9%). Ten percent of women who took anti-malarial prophylaxis delivered parasitaemic babies in contrast to 13.6% of those who did not, but these differences were not statistically significant (p = 0.6).
As shown in Table 3, 31 mothers (17.3%) had a history of hospitalization during the index pregnancy, and the frequency of this variable was higher in mothers of parasitaemic babies than mothers of aparasitaemic babies (33.3 versus 15.2%). Similarly, a history of prolonged rupture of membranes was obtained more often amongst mothers of parasitaemic babies (23.8 versus 15.8%), but these differences were not statistically significant ($p = 0.06$ and $p = 0.34$, respectively). None (0%) of the mothers of parasitaemic babies had a history of blood transfusion in pregnancy, but such history was obtained in seven (4.4%) of the other group. This difference was also not statistically significant (Fisher’s exact $p = 0.4$). A history of fever in pregnancy featured in 112 (62.6%) of the 179

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Parasitaemic babies $n = 21$</th>
<th>Aparasitaemic babies $n = 171$</th>
<th>$\chi^2$ or $t$-test</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>9 (42.9)</td>
<td>86 (50.3)</td>
<td>0.46</td>
<td>0.5</td>
</tr>
<tr>
<td>Female</td>
<td>12 (57.1)</td>
<td>85 (49.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age (h)</td>
<td>$14.1 \pm 12.28$</td>
<td>$17.4 \pm 14.36$</td>
<td>0.95</td>
<td>0.35</td>
</tr>
<tr>
<td>Mean apgar score ± SD</td>
<td>$6.1 \pm 2.2$</td>
<td>$6.3 \pm 2.03$</td>
<td>0.36</td>
<td>0.7</td>
</tr>
<tr>
<td>1 min</td>
<td>$8.7 \pm 1.8$</td>
<td>$8.8 \pm 2.0$</td>
<td>0.3</td>
<td>0.7</td>
</tr>
<tr>
<td>Maturity Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-term</td>
<td>7 (33.3)</td>
<td>24 (14)</td>
<td></td>
<td>0.03$^a$</td>
</tr>
<tr>
<td>Non-pre-term</td>
<td>14 (66.7)</td>
<td>147 (86)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean birth weight (kg)</td>
<td>$2.75 \pm 0.8$</td>
<td>$2.93 \pm 0.6$</td>
<td>1.08</td>
<td>0.3</td>
</tr>
<tr>
<td>Birthweight category</td>
<td></td>
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<tr>
<td>LBW &lt;2.5 kg</td>
<td>7 (33.3)</td>
<td>45 (26.3)</td>
<td>0.2</td>
<td>0.7</td>
</tr>
<tr>
<td>NBW ≥2.5 kg</td>
<td>14 (66.7)</td>
<td>126 (73.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean length (cm)</td>
<td>$44.4 \pm 5.2$</td>
<td>$46.1 \pm 3.5$</td>
<td>1.75</td>
<td>0.08</td>
</tr>
<tr>
<td>Mean occipito-frontal circumference (cm)</td>
<td>$33.4 \pm 2.6$</td>
<td>$33.8 \pm 2.1$</td>
<td>0.8</td>
<td>0.4</td>
</tr>
<tr>
<td>Weight for gestation</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Small for gestational age</td>
<td>5 (23.8)</td>
<td>22 (12.9)</td>
<td></td>
<td></td>
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<tr>
<td>Appropriate for gestational age</td>
<td>15 (71.4)</td>
<td>143 (83.6)</td>
<td>2.01</td>
<td>0.37</td>
</tr>
<tr>
<td>Large for gestational age</td>
<td>1 (4.8)</td>
<td>6 (3.5)</td>
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<td></td>
</tr>
<tr>
<td>Survival outcome</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Alive</td>
<td>19 (90.5)</td>
<td>167 (98.0)</td>
<td></td>
<td>0.13$^a$</td>
</tr>
<tr>
<td>Dead</td>
<td>2 (9.5)</td>
<td>4 (2.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Fig 1. Prevalence of parasitaemia by maturity group of newborns.

*Pregnancy history.* As shown in Table 3, 31 mothers (17.3%) had a history of hospitalization during the index pregnancy, and the frequency of this variable was higher in mothers of parasitaemic babies than mothers of aparasitaemic babies (33.3 versus 15.2%). Similarly, a history of prolonged rupture of membranes was obtained more often amongst mothers of parasitaemic babies (23.8 versus 15.8%), but these differences were not statistically significant ($p = 0.06$ and $p = 0.34$, respectively). None (0%) of the mothers of parasitaemic babies had a history of blood transfusion in pregnancy, but such history was obtained in seven (4.4%) of the other group. This difference was also not statistically significant (Fisher’s exact $p = 0.4$). A history of fever in pregnancy featured in 112 (62.6%) of the 179
mothers in the study population. The frequency of such history was higher in mothers of parasitaemic babies than mothers of babies without parasitaemia, (76.2 versus 60.8%), but the difference was not statistically significant ($\chi^2 = 1.28, p = 0.26$). Similarly, more of the mothers with a history of fever in pregnancy delivered parasitaemic babies than mothers without such history (14.2 versus 7.5%, $\chi^2 = 1.88, p = 0.17$). This difference was also not statistically significant. On further enquiry about the timing of fever, 67 (37.4%) mothers gave a history of fever in the third trimester, and this variable was significantly higher in mothers of babies with congenital malaria in comparison with their counter-parts whose babies did not have congenital malaria [66.7 versus 33.5%, odd ratio (OR) = 3.96, 95% confidence interval (CI) = 1.39–11.63, $p = 0.007$].

**Peripheral parasitaemia rates at delivery**

Table 4 shows that 57 (31.8%) of the 179 mothers in the study population had positive blood films for malaria parasites. Eighteen (85.7%) women who delivered malaria-infested babies had peripheral parasitaemia in contrast to 39 of 158 (24.7%) mothers of non-parasitaemic babies. Thus, peripheral parasitaemia was almost four times more prevalent in mothers of parasitaemic babies than mothers of non-parasitaemic babies and this difference was highly significant. (RR = 12.84, 95% CI = 3.94–41.4, $p < 0.0001$).

In summary, prematurity, first-order pregnancy, history of fever within three months of delivery and presence of peripheral parasitaemia at delivery were significantly associated with congenital malaria infestation.

**Discussion**

This study has confirmed the existence of congenital malaria amongst newborns delivered in this centre, and identified some possible risk factors for congenital malaria. The prevalence rate (10.9%) of congenital malaria obtained by blood film microscopy was within the range of 4.9–46.7% documented.
in other Nigerian studies over the last decade [17, 20, 23, 24]. The disparity in prevalence rates observed at different sites may be due to differences in susceptibility to malaria infestation amongst populations in the diverse geographic zones within the country, or to differences in methodology, skill and experience amongst researchers and microscopists in the various research centres.

The identification of *P. falciparum* as the main aetiologic agent of congenital malaria in this study corroborates findings in earlier reports [16, 17, 19, 20]. It was not unexpected since *P. falciparum* accounts for about 96% of malaria infestations in the West African sub-region [30]. Furthermore, the range of parasitaemia observed in the studied newborns was similar to ranges reported in previous research from other malaria endemic areas [16, 19, 20, 41]. Using standard criteria for classifying parasitaemia levels, these rates are low [42], and can be attributed to three transient mechanisms that combine to restrict replication of the malaria parasite. They include the temporary cessation of erythropoiesis that occurs as infants adjust from intra-uterine to post-natal life [43], the presence of fetal haemoglobin that retards the intra-erythrocytic development of the parasite [14, 44], and the relative deficiency of PABA, a consequence of breastfeeding that deprives malaria parasites of folic acid, thereby suppressing their growth [15].

The poor neonatal indices of the parasitaemic babies that were documented in this study are in agreement with studies done in Zambia and Ile-Ife [19, 24]. In Zambia, parasitized newborns weighed 469 g less than the uninfected newborns, while the Ile study reported that mean lengths of babies with congenital malaria parasitaemia were significantly lower than those of their non-parasitaemic counterparts. The observed differences in the Apgar scores, mean anthropometric indices and mean haematocrit values of parasitaemic and non-parasitaemic babies in this study were not statistically significant but they were too consistent to be ignored. One major reason for taking note of them is because the observed disadvantages may represent a setting for poorer outcome should another adverse factor supervene.

The relatively poor early neonatal profile of the parasitaemic babies in this study was not unexpected. Congenital malaria is a sequel of both gestational and placental malaria, both of which have been associated with some degree of placental insufficiency leading to growth restriction in the fetus, and pre-term delivery of low birth weight babies [32, 45]. Given the fact that prematurity (which is usually accompanied by low birth weight) was significantly higher in the parasitaemic group of babies in this study, one would also have expected a significantly higher proportion of low birth weight babies in the parasitaemic group. Surprisingly, this was not the case. The observed lack of statistical significance was probably because some of the low birth weight babies in the study population were both term and non-parasitaemic, suggesting that maturity rather than size was the more important factor. This hypothesis is supported by the observation that the higher prevalence of SGA babies in the babies with parasitaemia was also not statistically significant. The non-significant difference in the mean gestational age of the two groups of babies was probably because one of the parasitaemic babies was post-term, thus increasing the mean gestational age in the parasitaemic group.

The relatively higher proportion of pre-term babies in the parasitaemic group and higher parasitaemia rates in the pre-term babies suggests that prematurity could be a risk factor for congenital malaria. This could be related to the transfer of maternal IgG to the fetus, which occurs mainly during the third trimester. A shortened gestation period would result in lower levels of maternal anti-malarial antibodies in the fetus, thus allowing the optimal growth of malaria parasites and their subsequent detection in peripheral blood film. On the other hand, placental parasitization may have contributed to the pre-term delivery [34].

Low parity order, peripartal fever and peripheral parasitaemia at delivery in mothers are possibly predictive of congenital malaria infestation as observed in this study. The higher prevalence of gestational malaria amongst primigravidae than multigravidae is well documented [21, 45–47], and has recently been explained by the acquisition of variant surface antigen/chondroitin sulphate A (VSA\textsubscript{CSA}) specific antibodies in women of child-bearing age with each pregnancy. These antibodies inhibit and reverse the cytoadherence of placental parasites to the human synciotrophoblast, thus reducing the susceptibility to malaria as parity increases [48–51].

Majority of mothers in the study population claimed to have used one form of malaria preventive strategy or the other, and the use of insecticides and bed-nets was surprisingly but insignificantly higher in the mothers of parasitaemic babies. The insecticides may have been fake, however (Nigerian factor), and impregnation of the bed-nets with pyrethrum doubtful. Unfortunately, it was not possible to assess the quality of the products used. There are other interesting speculations however, including incorrect use of chemical insecticides used in net treatment, time of tucking in the nets and bites due to exposure outside the nets. It is also possible that some mothers did not use any preventive measures at all, but claimed to have done so.

The more frequently obtained history of fever in pregnancy in mothers of parasitaemic babies in this study was consistent with the observations of Ibhanasebhor and Okolo and Runsewe-Abiodun [52, 53]. In their studies, however, the occurrence of
fever was within a shorter duration prior to delivery (48 h to 2 weeks) and it is not certain if the fever in all cases was a consequence of malaria infestation. The occurrence of high fever in pregnancy is known to precipitate labour, and may explain the observation of higher rates of prematurity in parasitaemic babies compared to non-parasitaemic babies. The fever in pregnancy may also have contributed to a breakdown in the placental barrier, predisposing to the transfer of infected maternal erythrocytes to the fetus with resultant congenital malaria infestation in the newborn baby.

It is also possible that the high prevalence of history of fever in pregnancy in the mothers was due to recall bias, since the information was obtained after delivery. However, its higher frequency in the mothers who delivered parasitaemic babies relative to their counterparts who did not, suggests that this constraint did not influence the quality of information significantly. These reported episodes of fever in pregnancy were probably bouts of gestational malaria, reflecting the increasing prevalence of malaria worldwide due to the emergence of \( P. falciparum \) strains of increased virulence and resistance to anti-malarial drugs. Although the history of fever in pregnancy was not identified as a possible risk factor for congenital malaria infestation in this study, the finding of a significantly more frequent history of fever in the last trimester amongst mothers of parasitaemic babies suggests that the timing of gestational infestation may be more relevant to materno-fetal transmission than its occurrence alone. The discovery of peripheral parasitaemia in majority of the mothers of parasitaemic babies in this study supports this opinion.

The discovery of peripartum peripheral parasitaemia (PPP) as the major maternal factor associated with congenital malaria in this study, provides evidence for the theory that materno-fetal transmission of malaria parasites also occurs at birth during abruption of the placenta [35, 43]. Thus, intrapartum transmission of parasites may probably be a more important time of transmission than during pregnancy. The observation of some babies being delivered to women who did not have parasites in their blood film was not unexpected, as other researchers have also reported this phenomenon [22, 54]. In such babies, the source of infection may have been the placenta with sequestered parasites. The mothers themselves may have had parasites sequestered in their internal organs with clear blood films at the time of blood sampling.

Thus, this study has confirmed the existence of congenital malaria as an entity in newborns delivered in the study centre. There is increasing evidence that congenital malaria infestation occurs more frequently now than in the past, but the public health significance of this observation is yet to be evaluated [23, 24]. The importance of this study lies in the fact that it has identified some possible risk factors for congenital malaria infestation in malaria endemic areas. It would be interesting to observe if those neonatal and maternal associations identified with congenital malaria in this study will be identified in other malaria endemic areas.

In conclusion, congenital malaria parasitaemia in tropical endemic areas is not rare and pre-term neonates, infants of primigravidae, women with a history of fever within 3 months of delivery and women with post-partum peripheral parasitaemia may benefit from screening for malaria. Further prospective studies to determine the natural progression of congenital malaria infestation are however required to determine its clinical significance.

References


