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Placental Malaria Parasitaemia and Neonatal Outcome at Owerri, Imo State, Nigeria

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Abstract: Introduction: Malaria remains endemic in many tropical African countries and continues to pose a significant public health challenge. Pregnant women and children are particularly vulnerable to malaria infection due to pregnancy-related immunological changes, with placental malaria being associated with adverse maternal and neonatal outcomes. **Objective:** This study aimed to determine the correlation between placental malaria parasitaemia and neonatal outcomes among term babies delivered in Owerri, the capital city of Imo State, Nigeria. **Methodology:** This was a descriptive correlational study conducted among consenting term parturients who delivered at the Labour and Delivery units of the Federal Teaching Hospital, Owerri; the Claretian University of Nigeria Hospital; and the Holy Family Sisters of the Needy Hospital, all located in Owerri, Imo State. Women with pre-existing medical conditions such as hypertension, cardiac or renal disease, diabetes mellitus, sickle cell anaemia, retroviral disease, as well as preterm parturients, were excluded. Data were obtained using interviewer-administered questionnaires and patients' case notes to collect information on socio-demographic characteristics, parity, gestational age, antenatal clinic attendance, use of intermittent preventive treatment (IPT) and insecticide-treated nets (ITNs). Immediately after delivery, placental blood samples were obtained from the maternal surface of the placenta for malaria parasite detection. Neonatal heel-prick blood samples were collected for malaria parasitaemia. In contrast, cord blood samples were used to estimate neonatal packed cell volume. Neonatal outcome measures assessed included birth weight, Apgar scores, jaundice, malaria parasitaemia, packed cell volume, and perinatal mortality. Data were analyzed using Chi-square and Fisher's exact with statistical significance set at $p \leq 0.05$. **Results/Findings:** Out of 431 term

parturients initially assessed, 400 were included in the final analysis. The prevalence of maternal peripheral malaria and placental malaria parasitaemia were 69% (276/400) and 14.0% (56/400) respectively. Placental malaria parasitaemia showed a statistically significant correlation with neonatal malaria parasitaemia (p = 0.0001), neonatal anaemia (p = 0.010), low birth weight (p = 0.0001) and neonatal mortality (p = 0.002). There was no significant association with Apgar scores (p = 0.425) and there was no record of Neonatal Jaundice in this study. **Recommendations:** Malaria prevention and control strategies should be further strengthened during pregnancy, with emphasis on consistent use of IPT and insecticide-treated nets, early antenatal booking, and routine screening for malaria to reduce placental infection and its adverse neonatal consequences. **Contributions to Knowledge:** This study provided local epidemiological evidence that placental malaria parasitaemia remains relatively common in Owerri and significantly affects neonatal outcomes. It reinforces the role of placental malaria as a contributor to neonatal morbidity. It adds to existing Nigerian data supporting intensified malaria control measures during pregnancy.
Keywords: Placental malaria; placental malaria parasitaemia; neonatal outcome

1. Introduction

1.1 Background of the Study

Malaria is an endemic Disease especially in the sub-Saharan African countries, with an enormous health burden. A lot of research has been done on Malaria with the bid to eradicate it. However despite the control measures put in place, malaria continues to be deadly. Studies have shown that pregnant women and children are more susceptible to the plasmodium falciparum parasite.[1,2]

It has been proven that malaria crosses the placenta barrier and sequester at the capillaries of the placenta[3]. Placenta being a channel that conveys foetal nourishment from the mother to the foetus and waste products from the foetus back to the mother, is capable of transferring toxins and infections such as malaria parasites from the mother to the foetus as well.[4]

Placental malaria parasitaemia defined as the sequestration of malaria parasites in the placental vascular space is more common in areas of stable malaria transmission.[5] There are different types of plasmodium species that cross the placenta namely, plasmodium falciparum, plasmodium vivax, plasmodium ovale and plasmodium malariae. In Nigeria and other sub-Saharan African countries, plasmodium falciparium is more prevalent.[6] A new growth of a sub-population of plasmodium falciparium is said to be attracted by the

presence of the Placenta leading to the sequestration of the falciparium at the placenta bed and vasculaturs.[7,8]

Placental malaria is also said to be more prevalent among primigravidas whose immunity against chondroitin sulphate A antigen is considered low but which builds up with successive pregnancies.[9,10]

Malaria infestation is hazardous to both the pregnant mother and her foetus. It is regarded as one of the most severe public health issues worldwide, accounting for a high number of maternal and infant mortality in Africa and other developing countries where it is considered endemic.[1,8,11-13] Although several studies exist on malaria in pregnanancy,[14-15] there is a paucity of research on placental malaria and neonatal outcomes in Imo State.

1.2 Statement of the Problem

Placental malaria continues to pose a significant risk to maternal and neonatal health, particularly in endemic regions such as Imo State. Despite the high burden, there is limited research specifically evaluating the correlation between placental malaria parasitaemia and neonatal outcomes in Owerri. This gap in knowledge hampers directed interventions to improve neonatal survival and maternal health.

1.3 Research Questions

1.What is the prevalence of maternal peripheral malaria parasitaemia in Owerri?

2.What is the prevalence of placental malaria among parturient women in Owerri?

3.How does placental malaria parasitaemia influence neonatal outcomes?

1.4 Objectives of the Study

General Objective

To determine the correlation between placental malaria parasitaemia and neonatal outcomes in Owerri,

the capital city of Imo State.

Specific Objectives include:

1.To measure the prevalence of maternal peripheral malaria parasitaemia in Owerri

2.To estimate the prevalence of placental malaria among parturient women in Owerri.

3.To measure neonatal outcome among those with positive placental malaria parasitaemia.

1.5 Research Hypotheses

Null Hypothesis (H_0): Placental malaria parasitaemia has no influence on neonatal outcomes.

Alternate Hypothesis (H_1): Placental malaria parasitaemia influences neonatal outcomes.

1.6 Justification of the Study

This study is justified because malaria remains a critical factor affecting maternal and neonatal health. By evaluating its prevalence and impact in Owerri, the study will provide essential data to inform public health policies and clinical management strategies, ultimately improving maternal and neonatal outcomes.

1.7 Significance of the Study

The findings of this study will:

1.Provide baseline data on placental malaria parasitaemia in Owerri.

2.Guide policymakers in implementing malaria control strategies for pregnant women.

3.Inform clinicians on the management of pregnancies complicated by placental malaria to improve neonatal outcomes.

1.8 Scope of the Study

This study was focused on parturient women in selected health facilities in Owerri, Imo State. It examined maternal peripheral malaria, placental malaria parasitaemia, and associated neonatal outcomes over a defined period of March to October, 2025.

1.9 Limitations of the Study

1.Variability in malaria detection methods may affect prevalence estimates.

2.Limited access to certain health facilities due to logistical challenges.

3.Potential underreporting or incomplete documentation of maternal and neonatal outcomes.

1.10 Operational Definitions of Terms

• **Placental Malaria Parasitaemia:** Presence and sequestration of malaria parasites in the placental beds and vascular space.

• **Neonatal Outcome:** Clinical status of the newborn, including birth weight, Apgar score, and incidence of complications.

• **Primigravida:** A woman who is pregnant for the first time.

• **Chondroitin Sulphate A Antigen:** Placental receptor that facilitates adhesion of malaria-infected red blood cells.

2. Literature Review

2.1 Introduction to the Review

A comprehensive review of relevant literature is crucial for any research as theoretical and empirical foundation for the study. In this research, the literature review serves to conceptualize placental malaria parasitaemia and its impact on neonatal outcomes in Owerri, Imo State, Nigeria, while highlighting existing gaps in knowledge, methods, and clinical practice.

Despite the global burden of malaria in pregnancy, few studies have systematically examined the correlation between placental malaria parasitaemia and neonatal outcomes in Imo State.[14-16] Moreover, routine examination of the placenta, neonatal malaria screenings and access to facilities capable of performing these tests remain limited in the study area. This review identifies these gaps and underscores the need for rigorous investigation.

2.2 Conceptual Review

This section establishes the theoretical and contextual foundation for the study, focusing on the biology, epidemiology, pathophysiology, and clinical significance of malaria in pregnancy particularly placental parasitaemia.

2.2.1 Malaria Pathogenesis of Various Plasmodium Species

Malaria is considered a hematological ailment with consequent hemolysis of the red blood cells (RBCs), leading to the manifestation of complicated or uncomplicated symptoms[17] It is a vector-borne unicellular protozoa parasite of the plasmodium genius that is transmitted by the bite of an infected female anopheles mosquito. There are about 200 Plasmodium species but only five species have been reported to infect humans: P. falciparum, P. Vivax, P. Malariae, P. Ovale and P. knowlesi.[18-19] Plasmodium Falciparum

is more prevalent in Nigeria and other sub-Saharan African countries.[6] It also has a high prevalence in South-East Asia, Eastern Mediterranean and Western Pacific regions. It is the most severe type of malaria that causes complex and complicated symptoms associated with red blood cell haemolysis and high parasitaemia[21]

The aetiology of Malaria begins with a bite of an infected *Anopheles* mosquito on a human host, and the deposition of sporozoites in its salivary gland in the wound, which enters the peripheral blood circulation, making its way into the liver, where it infects the hepatocytes. In the hepatocytes, it undergoes a change and matures into schizont, a process that takes up to two weeks.[22] The schizonts undergo through replication which leads to the formation and subsequent release of merozoites that infect the red blood cells. The merozoites then go through a serial cycle of asexual replications of ring stage, trophozoites, and schizonts that continue to release thousands of new motile merozoites that infect more red blood cells.[23] This leads to a barrage of plasmodium parasites in the blood circulation. Toxins are released, thereby stimulating the body's immune response by activating inflammatory pathways with subsequent manifestation of clinical symptoms.[21] A small fraction of intra-erythrocytic parasites switch to sexual development, producing morphologically distinct male and female gametocytes that reach the host's dermis and are ingested by a mosquito, rendering it infectious to humans.[24] These trigger the immune system and other pathological processes leading to the manifestation of clinical symptoms.

The complexity of the interaction of malaria parasite-induced red blood cells, shape modifications, sequestrations in the micro-vascular spaces, leading to micro-vascular obstruction, and endothelial damage and hypoxia, with the local and systemic immune responses that manifest clinical symptoms and disease severity defines the pathogenesis of malaria.[25] Malarial parasites have the exclusive ability to cause enormous changes in host cells, including loss of the normal red blood cells biconcave shape, increased plasma membrane rigidity which allows a wide spectrum of ions and other substances travel in and out of the cells.[26] There has been argument whether Inflammatory trigger of the malaria parasites in the host is sort of a defensive mechanism for the body or aggravating mechanism. However, Gowda et al and Deroost et al agree that inflammatory response in this case is protective, but that a prolonged pro-inflammatory response is just as lethal as it contribute to pathophysiology and disease progression.[27,28]

2.2.2 Definition and Epidemiological Basis

The Epidemiology of malaria refers to the distribution, determinants and control of malaria parasite diseases. It is a very complex disease with transmission being influenced by factors related to both human, populations, mosquito parasite and to the environment.[27] The nature, duration, and severity of malaria infection not only depend on the above mentioned factors, it also depends on the level of the individual's immunity. Hence, the most vulnerable groups affected by malaria in high transmission areas are children younger than five years (with 75%-76% of global malaria death in 2024) and primigravida.[29,30] This is because these categories of people are said to have low specific malaria acquired immunity which develops with age and gravidity respectively due to repeated infections. Also, in low transmission areas, all ages are at risk due to low infection exposure and low acquired immunity. Compared to non-pregnant women, pregnant women are three times likely to acquire malaria and have 50 % higher fatality rate.[37] They may be at higher risk due to factors including reduced immunity and the sequestration of infected erythrocytes to the placenta during pregnancy.[38] Despite this, pregnant women are said to be more attractive to Anopheles mosquitoes, because of probably hormonal and other physiological changes in pregnancy, thereby increasing their potentials of malaria transmissibility.[39]

Any one of four species of malaria parasite can cause illness, but *Plasmodium falciparum* causes almost all severe and complicated disease with sequestration of parasitized red blood cells seen in various human tissues, including brain, liver, kidney and placenta.[31] The mean incubation period for *P. falciparum* is usually between nine to fourteen days with an average of 12 days, with most patients presenting in the first or second month after exposure in endemic areas.[32] Majority of individuals in endemic region suffer uncomplicated disease.

2.2.3 Epidemiology: Global and Nigerian Perspectives

Malaria remains a serious global health threat,

particularly in the WHO African Region.[30] According to WHO's World malaria report, there were an estimated 282 million cases and 610,000 malaria deaths worldwide in 2024.[30] There is an increase of about 9 million cases (3%) compared with 2023 and 12,000 more deaths in 2024 compared with 2023.[30] The Sub Saharan African region shares the highest burden of 265million cases (95%) and 579,000 (95%) deaths. Children under five, accounted for about 75% of all Malaria deaths in the region.[30]

Malaria is transmitted throughout Nigeria, with 97% of the population at risk of being infected.[33] According to the 2025 World Malaria Report, Nigeria remains the global epicenter for malaria recording the highest percentage of the global malaria burden, with 24.3% of global malaria cases and 30.3% to 30.9% global malaria deaths.[30] In Nigeria, poverty, ignorance, poor health seeking behavior, lack of health insurance, overcrowding, poor sanitation, open and unkempt gutter system are some of the factors that favor mosquito breeding, thereby accounting for the persistent rise in malaria transmission.

2.2.4 Pathophysiology of Placental Parasitaemia

The mechanisms by which malaria infected red blood cells sequester at the placental beds have been variedly expressed. However, it is common knowledge now that the presence of receptors such as Chondroitin Sulfate A (CSA) antigen in the placenta attracts malaria infected red blood cells to congregate at the placenta perivascular spaces. This is mediated by the VAR2CSA protein on the infected erythrocyte plasma membrane binding to Chondroitin Sulfate A. (CSA)[3,34] receptors at the placenta perivascular spaces. The presence of chondroitin sulphate A and VAR2CSA complex result to influx of inflammatory factors such as monocytes, cytokins, and other pro inflammatory cells to congregate thereby, causing placental damage, hypoxia with subsequent materno-fetal insufficiency.[35,36] Consequently, this may lead to adverse foetal effects such as early miscarriage, stillbirth, intra uterine growth restrictions, low birth weight, and congenital malaria.[5,8,18]

2.2.5 Clinical Manifestation in Mother and Neonate

Majority of women with malaria parasitaemea in pregnancy may be asymptomatic. In endemic region, most pregnant women with falciparum parasitamea will have non complicated symptoms. However, those who present with clinical symptoms may be misinterpreted and misdiagnosed due to the development of similar pregnancy clinical manifestations such as nausea, vomiting, fatigue, chills, headache, fever and cough.[37]

In severe cases, it might present with severe illnesses such as high fever, convulsions, impaired consciousness, respiratory difficulty; other maternal complications such as anaemia and heart failure; fetal and neonatal complications such as intrauterine growth restrictions (IUGR), prematurity, low birth weight and congenital malaria.[30] Neonatal complex malaria may include jaundice, hepato-splenomegally and death. Therefore, it is important to get proper history so as to timely initiate proper management and to avert disastrous consequences for both the mother and the fetus/neonate.

Congenital Malaria is a potentially life-threatening infection of neonates occurring as vertical transmission of malaria during pregnancy or at birth. It is also defined as the presence of asexual stages of the parasite in cord blood or in the peripheral smear of the infant in the first seven days of life regardless of symptoms.[40,41] Clinical features of neonatal malaria are non-specific and overlap with those of neonatal sepsis. They include fever, refusal to suck, excessive crying and irritability, jaundice, convulsions, vomiting, diarrhoea, lethargy, anaemia and splenomegaly.[40,42] Although fever is a cardinal symptom of malaria, it may be absent in Congenital malaria[43]

2.2.6 Burden of Malaria in Pregnancy

Malaria in pregnancy presents a huge burden to both mother and the neaonate as a result of complications such as gestational hypertension, severe anaemia, anaemic heart failure which may lead to maternal death, premature births, still births, low birth weight and infant deaths. The burden is high in sub-Saharan Africa, especially in the West African region where approximately 25% of all pregnant women are vulnerable to malaria parasite infection each year.[11] In Nigeria, malaria affects 63-70% of pregnant women, with maternal and foetal complications.[44]

2.2.7 Evidence Linking Malaria Parasitaemia to Maternal and Neonatal Outcomes

In a systematic review of malaria in pregnancy, Minwuyelet et al, reported that Malaria prevalence can

reach 60% in sub-Saharan Africa and 36% globally, with placental malaria affecting up to 28% of cases.[11] The disease causes serious complications such as maternal anemia, premature birth, low birth weight, severe anemia and increased maternal and infant mortality.[11]

In another systematic review of thirty-one studies done on prevalence of adverse birth outcomes in malaria-infected pregnancies, Prakasini et al, reported high prevalence of low birth weight, preterm birth and small for gestational age in malaria-affected pregnancies.[45]

In Uyo, Akwaibom State, Johnson et al, highlighted a strong association between placental malaria especially early in pregnancy and low birth weight[46] A systematic review of 68 Literatures written between 2014 to 2025 and done by Ahma et al., assessing the maternal and fetal health risks associated with malaria infection during pregnancy and to evaluate the effectiveness of current prevention and management strategies demonstrated that malaria during pregnancy significantly increases the risk of maternal anaemia, placental malaria, preterm birth, and neonatal deaths, with highest burdens in sub-Saharan Africa and vulnerable groups.[47]

2.3 Empirical Review

2.3.1 Objective 1: Prevalence of Maternal Peripheral Malaria Parasitaemia

It is difficult to ascertain the actual prevalence of maternal peripheral malaria parasitaemia. This is because of varied determinant factors such as endemicity, gravidity, age, geographical location, diagnostic methods and other influencing factors such as HIV and use or none use of malaria preventive measures in pregnancy such as intermittent preventive treatment (IPT) and/or insecticide treated nets (ITN). Balogun et al recorded 14.3%[48] of maternal peripheral malaria parasitemia in their study in two secondary health facilities in Abuja, Nigeria. In a study done in the South west Nigeria, prevalence rates of 57.3%[49] and 40.8%[50] were recorded in Ile-Ife, in Oshon State and Abeokuta in ogun State respectively. A study done in South Eastern Nigeria shows a peripheral parasitaemia of 57.6%.[51] A study carried out previously at the Federal Teaching Hospitial Owerri (former FMC), one of the centers being studied, shows a prevalence of 65.6%.[52]Achu et al.[53] reported an 8.3% and 17.7% prevalence using RDT and Microscopy respectively in Delta State. Mohamud et al. and Madanitsa et al. recorded a prevalence rate of 59.8% and 48% in their respective studies in Uganda and Malawi using a Microscopy and RDT respectively.[54,55] The high prevalence in the various studies shows the vulnerability of pregnant women in malaria infection despite the mitigating measures taken.

When comparing the report of a rural community and an urban community in Ghana, Ofori et al. and Glover-Amengor et al. reported peripheral malaria prevalence of 19.7%[56] and 35.5%[57] respectively. Ofori et al deduced that the reason for their much lower prevalence could be because of the drier coastal savannah region where their study was conducted compared to the Ashanti region located at the forest belt of Ghana which is regarded as a high malaria transmission area where Glover-Amengor et al did their studies.[56]

Researchers in Eastern Uganda, Ethiopia and Northwest Colombia recorded a much lower maternal peripheral malaria at 1.4%,[58] 6.4%[59] and 9.1%[60] respectively.

2.3.2 Prevalence of placental malaria

The actual prevalence of placental malaria parasitaemia may be hard to ascertain because of the different determining factors such as timing of the studies, diagnostic methods with their different sensitivities and specificities and population characteristics etc. However, a meta-analytic study of fifty different studies, forty-five of which were in sub-Saharan Africa showed a global prevalence rate of 17% using microscopy, and 23% with histological studies, majority being from Sub Saharan Africa.[61] Also Alemayehu et al. analyzed 178 observational studies published from 2000 to 2024 involving 93,809 parturient women from 23 countries with results showing a prevalence of 25.1% in Sub Saharan Africa, with 16.9%, 19.1%, 24.8% and 34.8% being RDT, microscopy, PCR, and histopathology, respectively.[62] From the above findings, it can be deduced that placental histopathology has the highest sensitivity followed by PCR diagnostic method. In Nigeria, the prevalence of placenta parasitaemia ranges from 10.5% to 65.8% using placental bloodsmears.[13,15-19] In other Sub-Saharan African countries, this range from 9.5% in Senegal,[63]3.9% in Southern Ethiopia,[64] 19% in

Cameroon,[65]19.6% in Burkina Faso[66] and 37.1% in The Gambia.[67]

Using placental histology, prevalence of 35.2%, 51.0% and 69.6% have been reported in Tanzania,[68] The Gambia[69] and Southeastern Nigeria[70] respectively.

In Burkina Faso, a study that evaluated the prevalence of placental malaria parasitaemia using histidine-rich-protein-2 (HRP-2) capture test and Polymerase chain reaction (PCR) recorded prevalence of 43.1% for HRP2 and 51.0% for PCR.[70] Also, in Ghana, the two techniques were used with prevalence rates of 41.0% for HRP2 and 59.0% for PCR.[71] The amenable result of the two separate studies shows that PCR and HRP2 have higher sensitivities for malaria detection than the other methods. However, in a study done in Malawi comparing PCR, histopathology and microscopy for the detection of placental malaria, there was no difference in the sensitivities of microscopy and PCR, but they noted a 100% specificity for PCR over placental histopathology.[72] However, Menendez C. and Mayo A. noted that caution should be exercised in the interpretation of the results of PCR detection because of factors that may affect its specificity like parasite macromolecules and dead parasites crossing the placental membrane giving a positive PCR result in cord blood.[73] Because of the cost, non-availability and high technicality of PCR, microscopy will continue to be the test of choice, especially in resource poor settings.

2.3.3 Neonatal Outcomes of Mothers with Positive Placental Malaria Parasitaemia

The high susceptibility of pregnant women to Plasmodium falciparum malaria infection may result to some distressing outcome to both the mother and fetus. The neonatal outcome of malaria in pregnancy include Congenital malaria, Low birth weight, still birth, neonatal death, preterm birth, intrauterine growth restriction, neonatal anaemia etc. These conditions are as a result of sequestered parasitized red blood cells that trigger inflammatory processes, damaging the placenta with subsequent placental insufficiency thereby leading to the above negative neonatal outcomes[68]These sequestered infected Red blood Cells along with the inflammatory reactions damage the endothelium of the placenta vasculatures, leading to obstruction of blood and nutrient flow to the fetus in utero with devastating consequences to the fetus.

2.3.4 Neonatal or Congenital malaria:

This is said to occur when malaria parasites cross the placenta either during pregnancy or at the time of delivery.[74] It is usually defined as the presence of the asexual forms of malaria parasites in cord blood smear or in the peripheral blood smear of the neonate within the first 7days of life irrespective of clinical symptoms.[3,74] In endemic areas, Congenital malaria may be asymptomatic and clear spontaneously.[75] Spontaneous clearance here implies that the infection may resolve without treatment which may be influenced by a combination of host and paarasite specific factors.[76] This is not so in the low malaria transmissible region where congenital malaria typically produces a more significant illness resulting in increased morbidity and mortality.

Congenital malaria was previously thought to be rare in endemic regions. However a multi- center studies done in the different parts of Nigeria, Kwara, Oyo and Plateau, analyzed by Falade et al.[77] reported an average prevalence of 5.1% of congenital malaria. In a systematic review of twelve different studies done in different parts of Nigeria, Kokori et al.[87] recorded prevalence as low as 5.1% to as high as 96.3%. Studies done in Minna, Ibadan, Lagos, Abuja, Enugu, and Ile Ife documented prevalence rates of 2.63%,[78] 14.0%,[79] 14.8%,[80] 18.1%,[48] 28.2%,[81] and 54.2%[82] respectively. In addition, reports from Moputu, Cameroon, Malawi and Zaire indicated prevalence of 1.5%,[83] 6.0%,[84] 7.8%,[85] and 9.0%[86] respectively. In some of these studies, there were strong associations between placental malaria parasitaemia and congenital malaria parasitaemia.[81,82] Kokori et al. deduced that the difference in the prevalence of congenital malaria could point at regional variability, reflecting differences in local malaria transmission rates; diagnostic practices, which is in consistent with world literatures hence, emphasizing the variation of congenital malaria prevalence according to endemicity.[87] It is important to note that congenital malaria may occur in the absence of maternal peripheral malaria parasitaemia.[86,88] In Zaire, 17.0% of the cases of congenital malaria occurred in neonates of women with negative maternal peripheral malaria parasitaemia at delivery and in the absence of placental malaria infection.[86]

The mechanism and timing of congenital transmission are poorly understood.[79,88] Proposed mechanisms include

maternal trans-placental transmission into the foetal blood circulation during the antenatal period, or at the time of delivery; direct penetration through the chorionic villi; or through premature detachment of the placenta.[79,89] It was previously vague whether the presence of plasmodium falciparum malaria parasites in umbilical cord blood was an infection acquired during intrapatum or as a result of contamination with infected maternal blood at delivery. In a study in Kenya, it was shown that malaria parasites identified in cord blood are acquired antenatally by trans-placental transmission of infected erythrocytes.[90]

2.3.5 Low Birth Weight (LBW)

In 2022, an estimated 12.7 million pregnant women in sub-Saharan Africa were exposed to malaria, resulting in 393,000 low birth weight (LBW) neonates.[91]

A baby born with a weight of less than 2.5kg is regarded as a low birth weight baby.[92] Low birth weight (LBW) contributes hugely to neonatal and infant mortality in Africa.[89,93] Infant mortality is said to be three times higher for LBW babies than for those of normal weight and a LBW baby is nine times more likely to die in the first month of life.[1] In Sub-Saharan Africa, the prevalence of low birth weight (LBW) babies born annually is estimated to be 10% of global low birth weight babies.[80,94] Malaria infection of the mother and trans-placental infection of the foetus accounts for 3.9% to 24.0% of this burden.[71,73,92]

It has been reported that a baby is twice likely to be born with a Low birth weight (LBW) if the mother has placental parasitaemia.[95] Primigravid women have the highest risk for placental parasitaemia and malaria-associated LBW babies.[94,96] Malaria is said to cause low birth weight babies. Some of the postulated mechanisms by which malaria is said to cause low birth weight are either by direct mechanical obstruction of the placenta by the parasites; fibronoid necrosis and thickening of the placental basement membrane; by the parasite and inflammatory system activation with the resultant poor materno-foetal circulation; or by indirect interference with placental functions through induction of pathological lesions in the trophoblasts.[95] Also, another mechanism by which malaria causes LBW may be through maternal malaria induced anaemia, and through trans-placental infection of the foetus.[93] However, there is still no agreement as to the main mechanisms mediating reductions in birth weight in placental malaria.[71]

Although the prevalence of placental parasitaemia ranges between 10.5% to 65.8%,[15-19,92] placental malaria infection associated low birth weight is elevated two to four times in various studies.[1,78,84] Balogun et al.[48] and Johnson et al.[46] noted a significant correlation between placental malaria and low birth weight. While there are documented findings on the correlation between placental malaria parasitaemia and low birth weight, some studies noted no significant correlation. Bassey et al[13] reported 6.67% of low birth weight neonates in Port Harcourt though this finding was not significantly associated with placental malaria parasitaemia. Also, a study in Zaire, and Rwanda found no correlation between placental parasitaemia and low birth weight.[97,98]

2.3.6 Neonatal Jaundice:

Jaundice in the context of an outcome of placental malaria parasitaemia is a significant sign of infection, which may result from parasite-induced red blood cell breakdown (hemolysis) or liver issues from the malaria, though it mimics other neonatal conditions like sepsis. Although clinical symptoms of congenital malaria are rare in endemic regions, neonatal jaundice is one of the clinical manifestations of congenital malaria.[99-100] There is scarcity of documented studies on the correlation between placental malaria parasitaemia and neonatal jaundice.

2.3.7 Apgar scores:

The Apgar score is a standardized assessment of a neonate's status immediately after birth and the response to resuscitation efforts and remains the gold standard for evaluating neonates.[101] It was originally designed in 1952 by Dr. Virginia Apgar, an anaesthesiologist to assess the color, heart rate, reflexes, muscle tone, and respiration in order to determine the need for intervention to establish breathing at 1 minute. However, the guidelines for the Neonatal Resuscitation Program state that Apgar scores should not be used to determine the initial need for intervention, what interventions are indicated, or when to initiate them, as resuscitation must be commenced before the 1-minute Apgar score is assigned.[102]

Apgar scoring is generally done at one and at five minutes post birth, and may be repeated later if the score is and remains low. Apgar score is said to be normal if it is 7 and above, fairly low if it is 4 to 6,

and critically low if it is 3 and below.[103] A low score at one-minute may show that the neonate requires medical attention, however, it is not necessarily a pointer for long-term problems; especially if there is an improvement at the five-minute score. If the Apgar score remains below 3 at later times such as 10, 15, or 30 minutes post- delivery; this may lead to an increased risk of cerebral palsy in population studies but not necessarily with an individual neurologic disability.[104] Emechebe et al recorded a significant effect on APGAR score between women with placental parasitaemia and those who were negative. They noted 16.5% of babies with low Apgar scores from women with placental parasitization versus 8.4% in women who didn't have placental malaria.[105] Datta et al. reported 70% of low APGAR score (< 7) in their study.[106] Balogun et al.[48] did not record poor APGAR score among studied neonates of placental malaria positive parturients. There is paucity of documented studies on the correlation of Apgar score and placental parasitaemia.

2.3.8 Neonatal anaemia:

Anaemia is defined by the World Health Organization as a reduction in the amount of red blood cells in circulation, which causes the hemoglobin (Hgb) concentration to drop below the necessary level for age or gender.[107] It may also be defined as a hemoglobin or hematocrit value that is more than two standard deviations below the mean for age.[108]

Newborns are usually born with an average hemoglobin count of 17 g/dl.[109] While there is no certain value to define neonatal anaemia, Brabin et al defined neonatal anaemia as cord haemoglobin level of less than 12.5g/dl or Packed cell volume of less than 37.5,[110] while Tiruneh et al defined it as cord haemoglobin less than 13.5g/dl.[111]. Its prevalence is reported to be high in sub-Saharan Africa.[112,113] A study done in Ethiopia reported 26.4% prevalence,[113] while in two separate studies in southern Malawi, neonatal anaemia prevalences of 23.4%[110] and 23.3%[114] were recorded.

Placental malaria parasitaemia and neonatal anaemia have been evaluated with varying results. A study done in Calabar Teaching Hospital reported a prevalence of 22.3% from placental malaria positive mothers.[105] Nwali et al did not record any neonatal anaemia in their study at Federal Teaching Hospital, Abakaliki.[115] Balogun et al[48] noted that only 33 out of 210 participants in their study had neonatal anaemia and they reported that this finding was not considered significant. A study conducted in Blantyre Malawi, to establish whether placental malaria affected neonatal haemoglobin status found that neontal haemoglobin levels were unchanged by maternal malaria and anaemia during pregnancy.[110] However, maternal malaria parasitaemia was observed to induce neonatal inflammatory response and elevated cord ferritin which was associated with LBW and shorter gestation period[110].

Although the aetiology of neonatal anaemia is complex and multifactorial; placental malaria could play either a major or minor role, depending on the local epidemiological situation.[114] It has been suggested that exposure of the fetus to malaria antigens due to damage of the placental barrier may make the newborn more susceptible to immunologically mediated haemolysis or to dyserythropoiesis.[116-119]

Symptoms of neonatal anaemia include pallor, tachycardia, jaundice and poor feeding, with treatment ranging from conservative management to blood transfusions.

2.3.9 Perinatal mortality:

Placental parasitaemia and its effects on perinatal mortality have been investigated in various parts of Sub-Saharan Africa.[3,5,120] The impact is still debatable with conflicting results from various studies. A study done in Ethiopia Eastern Africa, reported a seven-fold increased risk of stillbirth in association with placental parasitaemia in areas with unstable malaria transmission.[121] In The Gambia, researchers observed some seasonal differences in stillbirth rates, with the lowest rate occurring during the three months of the late dry season when placental malaria prevalence was low.[122] Conversely, another study in The Gambia, reported a two-fold increased risk of stillbirths among mothers with malaria-infected placenta and noted that placental malaria infection was independently associated with a higher risk of delivering stillbirths in the population studied.[67] However, in some studies in other countries,[121,123] this association was not always found to be statistically significant.

2.4 Methods of detecting Placental Malaria parasitaemia:

There are different methods of detecting placental

malaria parasitaemia. This contributes to the difficulties in determining the actual prevalence of placental malaria parasitaemia. The methods include microscopy of placental blood smears (BS), Rapid Diagnostic Tests (RDT), Deoxyribonucleic Acid (DNA) and Ribonucleic Acid (RNA) detection techniques and Placental Histopathology.

2.4.1 Blood Smears and microscopy (BS):

A blood smear with thick and thin preparations is the commonly available method used in detecting malaria parasites, especially in low resource countries.[14-16] It involves staining blood smears with stains such as Giemsa, Wright or Romanowsky stains and viewing under the microscope with up to 100magnification power. Its Sensitivity ranges between 42% and 95% with a specificity of 61% to 100%.[124-125] It is fairly easy to do and has been used for a long time in clinical practice. Time consumption and labour intensiveness are some of its draw backs. Secondly, its sensitivity decreases in parallel with the density of malaria parasites in the blood.[125] The interpretation of the smear, especially in malaria-endemic areas, may be difficult. This is because a negative blood film does not necessarily exclude malarial illness, just as a positive blood film does not necessarily confirm a diagnosis of malarial illness. This is because many individuals with positive films may have no symptoms. However; till date, it is still the standard technique of malaria parasite diagnoses in clinical use in malaria endemic countries of the world, Nigeria inclusive.[14-16]

2.4.2 Rapid Diagnostic Tests:

The Rapid Diagnostic Tests (RDTs) are immunochemical tests that are based on the detection of parasite antigens in the blood with the use of specific monoclonal antibodies. Rapid Diagnostic Tests for malaria detect one or more of the following antigens: Histidin rich protein 2 (HRP-2), Plasmodium Lactate Dehydrogenase (pLDH) or parasite Aldolase.[127,128] There are many RDTs kits in the market; namely, the Optimal test, which is based on the detection of pLDH, Parasite F [Becton Dickinson, Franklin Lakes, N.J.], ICT *Pf* or *Pf/Pv* [Amrad ICT, Sydney, Australia], and PATH Falciparum Malaria IC test [PATH, Seattle, Wash.] The last three are used for the detection of HRP-2. The parasite is specific for only plasmodium falciparium while the rest can detect other malaria species. The WHO guideline recommends RDT assays to achieve 90% specificity and 95% sensitivity in order to be recommended for malaria diagnosis.[129] In general, RDT HRP2 tests have higher sensitivities than the RDT pLDH tests, although the specificity is better for pLDH-based tests.[128, 129]

Laboratory trials of the Parasite F immunochromatographic test for HRP-2 for the detection of Plasmodium falciparum in blood samples have shown an overall average sensitivity of 84% to 94.5% when greater than 100 parasites/μl are present (0.002% parasitaemia), and a specificity of 95.2% to 99% for Plasmodium falciparum when compared with thick blood film microscopy.[120-131] The RDTs detecting HRP-2 are most commonly used because they are more stable across a wider temperature range and they have a lower detection threshold than pLDH-bases tests.[132]

The advantages of RDTs in general include, rapid diagnosis (results are available within 15-20minutes), less expensive, easy to do and do not need expert interpretations. The setbacks of RDT-HRP2 based tests include the inability to show the severity of the disease since they only indicate the presence of infection and not the extent. They also have a limited role in the monitoring of the therapeutic response because malaria antigens persist for several weeks after effective treatment, thereby reducing their specificity.[132,133]

2.4.3 Placental histology:

Placental histology has been referred to as the 'gold standard' in the diagnosis of malaria prasitaemia because of its ability to detect sequestered parasites when none are detected in the peripheral circulation.[134] Both malaria parasites and pigments are detected in the placenta biopsy specimen. The sensitivity of placental histology ranges between 65.2% to 85.0%, while its specificity ranges between 87.5% to 100.%.[61,135] The advantages of Placental histology over the other methods include the identification of features of malaria in pregnancy that are significant to clinical outcomes, such as inflammatory infiltrates and hemozoin depositions.[136-137] Hemozoin depositions is an evidence of past infection. Inflammatory infiltrates may occur in a subset of women with active malaria infection, particularly first-time mothers, and is strongly linked to Low Birth weight (LBW) babies.[138-139] The disadvantages of placental histology include the fact that the placenta specimen is available

for histology only after delivery, there is the possibility of erythrocytes alteration during histological processing thereby preventing true recognition of parasites or artifacts may falsely appear as parasites, increase cost compared to microscopy and RDTs, expertise analysis required and women with confirmed peripheral malaria and who had effective treatment often have no histological changes at delivery[140,141] Other challenges include limited standardization between laboratories in processing and scoring tissues, and limited infrastructure to properly collect, process and analyze placental samples in some tropical areas.

2.4.4 Deoxyribonucleic Acid and Ribonucleic Acid (DNA/RNA) parasite antigen detection technique:

Deoxyribonucleic Acid and Ribonucleic Acid parasite antigen detection technique in the diagnosis of malaria include: Polymerase Chain Reaction (PCR), quantitative PCR (qPCR) and Loop-mediated isothermal amplification (LAMP). Polymerase Chain Reaction method was first described and is the most widely used.[142] It is generally more sensitive in the detection of malaria parasite antigens than Blood Smear microscopy. [143-144] In fact, whether applied to peripheral blood or placental blood samples, PCR methods yield positivity rates of 20% or more above those of BS microscopy.[145-146] Real-time quantitative PCR (qPCR) followed shortly after PCR with good sensitivity and range that allow monitoring parasitaemia levels.[147-148] Loop-mediated isothermal amplification (LAMP) is a newer alternative to PCR and qPCR. This method does not require DNA purification but utilizes simple instrumentation (water bath or a heat block), and can be completed in less than one hour.[149] Several studies that compared the LAMP method to microscopy, PCR and RDT, reported high sensitivities and specificities in non-pregnant populations.[150-151] However, the performance of the LAMP method for malaria in pregnancy diagnosis has scanty studies but the result so far is showing high sensitivity and specificity as in non-pregnant population. [152]

Although, these tools are more sensitive for parasite detection compared with Blood Smear and microscopy, they require highly trained staff, specialized equipment and increase cost thereby making them unrealistic in clinical use, especially in low resource settings. In addition, the test format and the time to obtain results are not suitable for use in primary care settings.

Considering the sensitivities, specificities, labour intensiveness, time consumption and economic involvement of the different methods, Blood Smear and microscopy still remains valuable in the investigation of malaria parasitaemia in endemic regions.[125,153-158] This is the basis for using blood smear and microscopy as the method of choice for this study.

2.5 Synthesis and Research Gap

From the conceptual and empirical literature reviewed, it is evident that:

- While placental malaria is well-documented globally, limited studies exist in Imo State.
- Routine placental examination and neonatal malaria screening are not widely implemented.
- The relationship between maternal parasitaemia, placental malaria and neonatal outcome is not fully explored locally.

This review therefore justifies the current study as it seeks to generate local evidence on placental malaria parasitaemia and its impact on neonatal outcomes, providing a reference for future research, clinical interventions and policy-making.

3. Research Methodology and Materials

This chapter describes the methodological framework adopted for the conduct of this study. It outlines the study design, area, population, sampling procedures, data collection methods, laboratory analyses, ethical considerations, and statistical analysis.

3.1 Materials

The materials utilized in this study included interviewer-administered structured questionnaires, patients' case notes, sterile syringes and needles, EDTA sample bottles, microscope slides, sterile lancets, Giemsa stain, light microscope with ×100 oil immersion objective, Uniscope micro-haematocrit centrifuge (Model SM 124), and laboratory consumables required for malaria microscopy and packed cell volume estimation.

3.2 Area of the Study

The study was conducted in Owerri, the capital and largest city of Imo State, Nigeria, between 15 March and 31 October 2025. Owerri comprises three Local Government Areas (LGAs): Owerri Municipal, Owerri North, and Owerri West, with an estimated population

of 1,405,873 as of 2016 census data and a total land area of approximately 551 km²[159].

Owerri Municipal is predominantly urban, while Owerri North and Owerri West are largely semi-urban.[158] The city experiences two major seasons: the rainy season (April–October) with an annual rainfall of 1,500–2,200 mm, and the dry season (November–March).[160] The hottest period occurs between January and March, with an average annual temperature of 20°C and an average annual relative humidity of 75%, rising to 90% during the rainy season.[160]

3.3 Research Design

This study adopted a descriptive correlational research design, aimed at determining the relationship between placental malaria parasitaemia and neonatal outcomes among term parturients in Owerri, Imo State.

3.4 Population of the Study and Sample Size Determination

3.4.1 Study Population

The study population consisted of consenting term parturients who delivered at the Labour and Delivery units of:

- Federal Teaching Hospital, Owerri (Owerri Municipal LGA)
- Holy Family Hospital, Owerri (Owerri North LGA)
- Claretian University Hospital, Owerri (Owerri West LGA)

These hospitals were so chosen because of their high antenatal clinic attendance and delivery rates.

3.4.2 Sample Size Determination

The minimum sample size was calculated using the Cochran formula:

$$n = \frac{Z^2(p\,q)}{d^2}$$

Where n = sample size

Z = a constant given as 1.96

P = Prevalence put at 0.5(50.0). Since the sample size is less than 10,000.

d = Level of precision put at 0.05

q = 1-p

$$n = \frac{(1.96)^2 0.5 \times (1-0.5)}{0.05^2}$$

$$n = \frac{3.8416 \times 0.5 \times 0.5}{0.0025}$$

n = 384 (minimal sample needed)

A total of 400 term parturients and their neonates were studied. Although 431 parturients were initially recruited, 31 were excluded due to failure to meet inclusion criteria, refusal of neonatal heel prick, or sample clotting.

3.5 Sample and Sampling Technique

A **consecutive sampling technique** was employed. All eligible and consenting term parturients presenting in labour during the study period were recruited until the required sample size was attained.

3.6 Research Instruments

Data were collected using:

- Structured interviewer-administered questionnaires
- Patients' clinical case notes
- Laboratory diagnostic tools for malaria microscopy and packed cell volume estimation

The questionnaires captured information on socio-demographic characteristics, parity, gestational age, use of insecticide-treated nets (ITNs), intermittent preventive treatment (IPT), and antenatal clinic attendance.

3.7 Validation of Instruments

The questionnaire was reviewed by experts in Obstetrics, Pathology, and Public Health to ensure content and face validity. Necessary adjustments were made prior to data collection.

3.8 Reliability of Instruments

Consistency in laboratory analysis was ensured by using standardized procedures and having all samples analyzed by one haematologist and one microbiologist, thereby minimizing inter-observer variability

3.9 Method of Data Collection

Three milliliters of maternal peripheral venous blood were collected into EDTA bottles for packed cell volume (PCV) estimation and malaria parasite detection using thick and thin blood films.

Within 30 minutes of placental delivery, placental blood was obtained from the incised maternal surface or pooled blood and analyzed for placental malaria parasitaemia.

Neonatal heel-prick blood samples were collected using sterile lancets for malaria parasite detection, while cord blood was collected into EDTA bottles for neonatal PCV estimation.

Neonatal parameters including birth weight, Apgar scores, neonatal jaundice, and perinatal mortality were recorded by the accoucher. All samples were properly labelled and processed under strict quality control.

3.10 Analytical Tools / Methods of Data Analysis

Data analysis was performed using the chi square and Fisher's exact. Descriptive and inferential statistics were applied as appropriate. The level of statistical significance was set at $p \leq 0.05$.

3.11 Laboratory Analysis

Maternal peripheral, placental, neonatal heel-prick, and cord blood samples were analyzed at the Holy Family Hospital Laboratory, Owerri.

Thin and thick blood films were stained with Giemsa and examined microscopically using a ×100 oil immersion objective. Parasite density was graded as mild (+), moderate (++), or severe (+++).

Packed cell volume was determined using the Uniscope micro-haematocrit centrifugation method (Model SM 124).

4. Result

4.1 Result

Four hundred and thirty one term parturients who gave their consents were assessed for eligibility in the study. Seven parturients were excluded because retroviral screening of two turned out positive in labour, one had Hepatitise C, while four had pre-eclampsia. Thirteen women refused heel prick of their babies and so were also excluded from the study. Eleven samples could not be analyzed by the laboratory scientists because of coagulation issues. Therefore 400 patients' samples were finally analyzed.

This is shown in the flow chart in **Figure 1**. below.

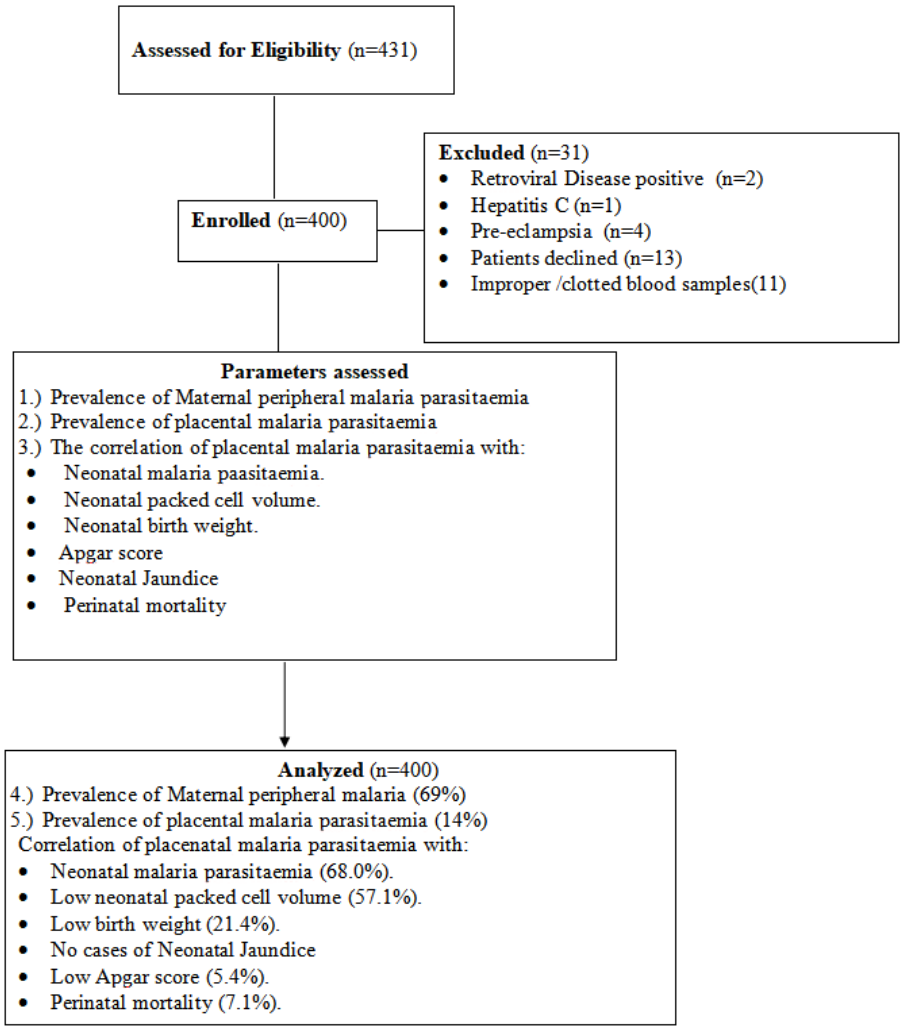

Figure 1. The flow pattern of the parturients recruited in the study.

4.1.1 Biosocial Characteristics of Study Parturients

Their age range was between 16 and 44 years with a mean of 26.2 ± 7.71years. Married women were 97.3% (389/400) while single women accounted for 2.7% (11/400). Majority of the respondents, 56.2% (225/400), had tertiary education with good proportion 43% (172/400) having secondary education. Also, majority of the women were self-employed, 53.8% (215/400), while civil servants accounted for 38.5% (154/400). This is shown in **Table 1**.

Table 1. Biosocial Characteristics of the respondents

Biosocial characteristics	Frequency (N = 400)	Percentage (%)
Age (Years)		
< 20	59	14.7
20 – 27	**176**	**44.0**
28 – 35	144	36.1
36 –44	21	5.2
Total	**400**	**100.0**
Marital Status		
Married	**389**	**97.3**
Divorced/widowed	0	0.0
Single	11	2.7
Total	**400**	**100.0**
Level of education		
No formal education	1	0.3
Primary	2	0.5
Secondary	172	43.0
Tertiary	**225**	**56.2**
Total	**400**	**100.0**
Occupation		
Self Employed	215	**53.8**
House wives	8	2.0
Civil servants	**154**	**38.5**
Students	**23**	**5.7**
Total	**400**	**100.0**
Use of ITN	**54**	**13.5**
None of ITN	346	86.5
Total	**400**	**100**
Use of IPT		
1-3 Doses	**145**	**36.2**
4 or more doses	88	22.0
No IPT	167	41.8
Total	**400**	**100**
Maternal PCV		
≥ 36	**286**	**71.5**
<36	114	28.5
Total	**400**	**100**

4.1.2 Parity of Studied Parturients

The parturients ranged from nulliparity to grand multiparity. The mean parity was 1.3±2.15, with nulliparity constituting the majority, 49.3% (197/400) while para≥3 accounted for 10.5% (42/400). This is shown in **Table 2**.

Table 2. Parity of studied parturients

Parity	Frequency (N = 400)	Percentage (%)	Mean
Para 0	**197**	**49.3**	**1.3±2.15**
Para 1	110	27.5	
Para 2	51	12.7	
Para ≥3	42	10.5	
Total	**400**	**100.0**	

4.1.3 Gestational Age of The Parturients

All the respondents were of term gestation. The mean gestational age was 38.9 ±2.46 weeks; Gestational age 37^{+0}-38^{+0}weeks accounted for the majority with 37.0% (148/400) while ≥41^{+1} weeks accounted for 5.2% (21/400). This is shown in **Table 3** below.

Table 3. Gestational age of the parturients

Gestational Age	Frequency (N = 400)	Percentage (%)	Mean
37^{+0}-38^{+0}	**148**	**37.0**	**38.9±2.46weeks**
38^{+1}-39^{+0}	87	21.8	
39^{+1}-40^{+0}	111	27.8	
40^{+1}-41^{+0}	33	8.2	
≥41^{+1}	**21**	**5.2**	
Total	**400**	**100.0**	

4.1.4 Antenatal Attendance

The mean frequency of antenatal attendance was four visits with more than half of the parturients, 61.7% (247/400), attending between one to four times while those who attended care for nine times or more accounted for 14.3% (57/400) as shown in **Table 4** below.

Table 4. Antenatal attendance

Antenatal attendance	Frequency (N = 400)	Percentage (%)	Mean frequency
None	0	0.0	**4.45**
1-4	**247**	**61.7**	
5-8	96	24.0	
≥9	57	14.3	
Total	**400**	**100.0**	

4.1.5 Mode of Delivery

Most of the respondents, 86% (344/400), had spontaneous vaginal deliveries. Up to 14% (56/400) were delivered by caesarean section. This is shown in **Table 5**.

Table 5. Mode of delivery

Mode of delivery	Frequency (N = 400)	Percentage (%)
SVD	**344**	**86**
C/S	56	14
Total	**400**	**100.0**

4.2 Prevalence of Maternal Malaria Parasitaemia

Out of 400 parturients studied, 276/400(69.0%) had positive maternal peripheral malaria parasitaemia while 124/400 (31%) were negative. This is shown on **Table 6** below.

Table 6. Prevalence of Maternal Malaria Parasitaemia

Parturients	Total (%)
Positive MP	**276 (69%)**
Negative MP	124(31%)
Total	**400 (100%)**

4.2.1 Use of Malaria Control Measures

The percentage use of malaria control measures (ITN and IPT) are 13.5% (54/400) and 58.3% (233/400) respectively. This is shown on **Table 7** below.

Table 7. Use of Malaria Control Measures

Paturients	Use/%	NON Use/%	Total/%
ITN	**54(13.5)**	**346(86.5)**	**400(100)**
IPT	**233(58.3)**	**167(41.7)**	**400(100)**

4.2.2 Correlation of ITN and Maternal Peripheral MP

Out of the 276/400 who had maternal malaria parasitaemia, 271/276 (98.2%) parturients were among those that didn't use ITN while only 5/276 (1.8%) of those who had maternal malaria peripheral parasitaemia used ITN as shown on **Table 8** below.

Table 8. Correlation of ITN and Maternal Peripheral MP

Malaria Control measures	MP Peripheral blood		Total (%)
	Positive(%)	Negative (%)	
ITN use	**5(1.8)**	**49 (39.5)**	54(13.5)
ITN non use	271(98.2)	75 (60.5)	346(86.5)
Total	**276(100.0)**	**124(100.0)**	**400 (100.0)**

Chi-square =104.15, df = 1, p-value = 0.0001

4.2.3 Correlation of ITN and Placental Parasitaemia

Among those who had Placental malaria parasitaemia, only 5.4% (3/56) used ITN while 94.6% (51/54) of the parturients who had placenta malaria parasitaemia didn't use ITN as shown on **Table 9** below.

Table 9. Correlation of ITN and Placental Parasitaemia

Malaria Control measures (ITN)	MP Placental blood		Total (%)
	Positive (%)	Negative (%)	
ITN use	**3(5.4)**	51(14.8)	54(13.5)
ITN non use	**53(94.6)**	293(85.2)	346(86.5)
Total	**56(100.0)**	**344(100.0)**	**400 (100.0)**

Chi-square =3.70, df = 1, p-value = 0.054

4.2.4 Correlation of IPT and Maternal Peripheral MP

The total number of parturients who used intermittent preventive therapy (IPT) was 233/400 (58.2%) while those who did not take IPT were 167/400 (41.8%). Among the 276/400 (69%) who had peripheral malaria parasitaemia, 114/276 (41.3%) used IPT while 162/276 (58.2%) did not use IPT. Out of the 167 who didn't use IPT, 162/167 (97.0%) had peripheral malaria parasitaemia. Out of the 124/400 who were negative for peripheral malaria parasitaemia, 96% (119/124) were of the women who used IPT. This is shown on **Table 10** below.

Table 10. Correlation of IPT and Maternal Peripheral MP

Malaria Control measures	MP Peripheral blood		Total (%)
	Positive (%)	Negative (%)	
IPT Use	114(41.3)	**119 (96.0)**	233(58.2)
IPT Non use	**162(58.7)**	5 (4.0)	167(41.8)
Total	**276(100.0)**	**124 (100.0)**	**400 (100.0)**

Chi-square =105.13, df = 1, p-value = 0.0001

4.2.5 Correlation of IPT and Maternal Placental MP

Among those who had placental malaria parasitaemia, 12.5% (7/56) were from the parturients who received IPT while 49/56 (87.5%) of those who had placental

parasitaemia did not receive IPT. This is shown on **Table 11** below.

Table 11. Correlation of IPT and Maternal Placental MP

Malaria Control measures (IPT)	MP Placental blood		Total (%)
	Positive (%)	Negative (%)	
IPT Use	**7 (12.5)**	226(65.7)	233(58.2)
IPT Non use	**49 (87.5)**	118(34.3)	167(41.8)
Total	**56(100.0)**	**344(100.0)**	**400(100.0)**

Chi-square =56.04, df = 1, p-value = 0.0001

4.3 Prevalence of Placental Malaria Parasitaemia

The prevalence of placental malaria parasitaemia in this study was 14.0% (56/400) as shown in **Figure 2** below.

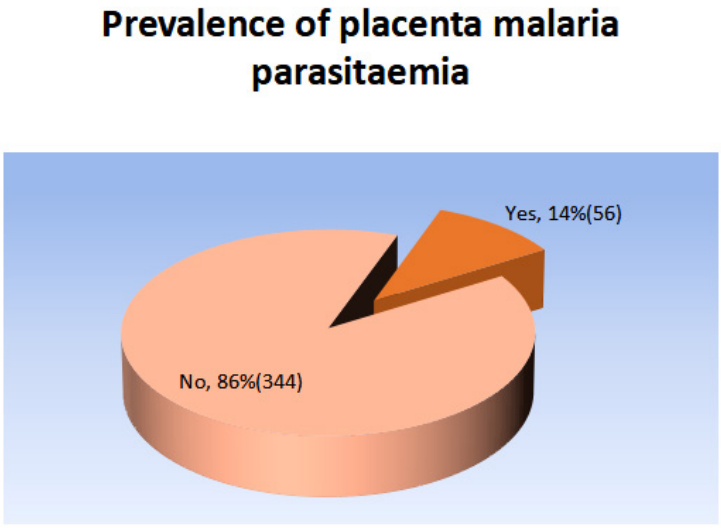

Figure 2. The prevalence of placental malaria

4.3.1 Correlation between Maternal Peripheral malaria Parasitaemia and Placental malaria Parasitaemia

The prevalence of Peripheral Malaria Parasitaemia in this study was 276/400 (69.0%). Approximately, 79% (44/56) of those with placental malaria parasitaemia also had maternal peripheral malaria. 21% (12/56) of those with placental parasitaemia did not have peripheral malaria. This is shown on **Table 12** below.

Table 12. Correlation of Maternal malaria and placental malaria falciparum

Maternal Peripheral malaria parasitaemia	MP Placenta blood		Total (%)
	Positive (%)	Negative (%)	
Positive	**44(78.6)**	232(67.4)	276(69.0)
Negative	**12(21.4)**	112 (32.6)	124(31.0)
Total	**56 (100.0)**	**344 (100.0)**	**400 (100.0)**

Chi-square =2.79, df = 1, p-value = 0.095

4.3.2 Correlation between placental Malaria Parasitaemia and neonatal Malaria

Neonatal malaria parasitaemia accounted for 12.0% (48/400) of the studied population. While 68% (38/56) of those with placental malaria parasitaemia had neonates who also had malaria parasitaemia, only 3.0% (10/344) of those without placental malaria parasitaemia had neonatal malaria parasitaemia as shown in **Table 13**.

Table 13. Correlation between Placenta malaria parasitaemia and Neonatal malaria

Neonatal malaria parasitaemia	MP Placenta blood		Total (%)
	Positive (%)	Negative (%)	
Positive	**38(68.0)**	**10(3.0)**	**48(12.0)**
Negative	18(32.0)	334(97.0)	352(88.0)
Total	**56(100.0)**	**344 (100.0)**	**400 (100.0)**

Chi-square =192.38, df = 1, p-value = 0.0001

4.3.3 Correlation between Placental Malaria Parasitaemia and Neonatal PCV

The proportion of studied babies with low packed cell volume was 41.5% (166/400). Those from placental parasitaemic mothers accounted for 57.1% (32/56) while neonates of non-placental malaria parasitaemic mothers accounted for 39% (134/344) as shown in **Table 14**.

Table 14. Correlation between Placenta parasitaemia and Neonatal PCV

Neonatal Packed cell volume(PCV)	Placenta blood Malaria parasite		Total (%)
	Positive (%)	Negative (%)	
PCV ≤36	**32(57.1)**	134(39.0)	**166(41.5)**
PCV>36	24(42.9)	210(61.0)	234(58.5)
Total	**56(100.0)**	**344(100.0)**	**400(100.0)**

Chi-square = 6.56, df =1. p-value =0.010

4.3.4 Correlation between Placental Malaria Parasitaemia and Low Birth Weight

Low birth weight constituted 3.8% (15/400) in this study. Neonates from the placental malaria parasitaemic mothers accounted for 21.4% (12/56) while those without placental malaria parasitaemia accounted for 0.9% (3/344). This is shown in **Table 15**.

Table 15. Correlation between placental parasitaemia and Birth weight

Neonatal Birth Weight(kg)	Placenta blood Malaria parasite		Total (%)
	Positive (%)	Negative (%)	
≤2.5	12(21.4)	3(0.9)	15(3.8)
>2.5	44(78.6)	341(99.1)	385(96.2)
Total	**56(100.0)**	**344(100.0)**	**400(100.0)**

Chi-square = 56.39, df =1. p-value =0.0001

4.3.5 Correlation between Placental Malaria Parasitaemia and APGAR Scores

The prevalence of Low Apgar scores is 3.0% (12/400). Neonates from the placental malaria parasitaemic mothers accounted for 5.4% (3/56) while those of the non-placental malaria parasitaemic mothers accounted for 2.6% (9/344). This is shown in **Table 16.**

Table 16. Correlation between placental parasitaemia and Apgar Scores

Neonatal Apgar Scores	Placenta blood Malaria parasite		Total (%)
	Positive (%)	Negative (%)	
Low	**3(5.4)**	9(2.6)	**12(3.0)**
Normal	53(94.6)	335(97.4)	388(97.0)
Total	**56(100.0)**	**344(100.0)**	**400(100.0)**

Fisher's exact = 1.25, df =1. p-value = 0.425

4.3.6 Correlation between Placental Malaria Parasitaemia and Perinatal mortality

The prevalence of perinatal mortality in this study was 1.3% (5/400). It constituted 7.1% (4/56) among those with placental malaria parasitaemia, while it was 0.3% (1/344) among those without placental parasitaemia.

This is shown in **Table 17**.

Table 17. Correlation between placenta parasitaemia and neonatal mortality

Neonatal Mortality	MP Placenta blood		Total (%)
	Positive (%)	Negative (%)	
Yes	**4(7.1)**	1(0.3)	**5(1.3)**
No	52(92.9)	343(99.7)	395(98.7)
Total	**56 (100.0)**	**344 (100.0)**	**400(100.0)**

Fisher's exact chi-square = 18.32, df = 1, p-value = 0.002

5. Discussion, Conclusion and Recommendations

5.1 Discussion

5.1.1 The Prevalence of Peripheral malaria parasitaemia

The prevalence of maternal peripheral malaria parasitaemia in this study was 69%. This is in congruent with the study previously done in FMC Owerri by Iwuchukwu et al. who recorded 65.2% prevalence. Fehintola et al.[49] in Ile-Ife and Mohamud et al.[54] in Somalia recorded high prevalence of 57.7% and 59.8% respectively. However, Achu et al. in Delta State and Balogun et al. in Abuja recorded lower prevalence of 17% and 14. 3% respectively. The reason for the high prevalence in this study may be because of the environmental factors and pathogenicity of malaria falciparum in the studied environment. In Owerri, mosquito is said to be high especially during the months of March to October which is considered the raining season. Therefore the timing of this study could contribute to the high prevalence recorded in this study.

5.1.2 Prevalence of Placental Malaria Parasitaemia

The prevalence of placental malaria parasitaemia observed in this study was 14.0%. This finding is comparable to the 10.5% reported by Falade et al.[76] in Ibadan, 14.0% reported by Mokuolu[92] et al. and 16.9% by Johnson et al.[46] in Uyo. The similarity in prevalence may be attributed to the hospital-based nature of the studies and the fact that they were conducted in semi-urban or urban settings, where access to antenatal care services and malaria preventive measures are relatively better.

However, the prevalence recorded in this study is lower than the 18.0%, 20.5%, 29.9% and 35.9% recorded by Ibanga et al.,[82] Balogun et al.,[48] Ukaga et al.[16] and Ofori et al.[55] respectively. This difference may be related to the timing of the studies and different environmental factors. Also, in recent years, increased emphasis has been placed on malaria prevention during pregnancy, particularly through intermittent preventive treatment (IPT) and the use of insecticide-treated nets (ITNs) during antenatal health education. Consequently, pregnant women in more recent studies may be more inclined to utilize these preventive measures, resulting in a declining trend in placental malaria prevalence as noted in this study where more than 58% of women were on IPT and 13.5% on ITN.

5.1.3 Placental Malaria and Maternal Peripheral Malaria

This study did not show a significant correlation between maternal and placental malaria parasitaemia. This was in accordance with studies done by Madanitsa et al.[55], Babalola et al.[50], Fehintola et al.[49] and ezebialu et al.[69] These noted a high degree of discordance in their results, meaning that, the acquisition of placenta malaria is not dependent on peripheral malaria parasitaemia.

5.1.4 Placental Malaria Parasitaemia and Neonatal Malaria Parasitaemia

A statistically significant correlation was observed between placental malaria parasitaemia and neonatal malaria parasitaemia ($p = 0.0001$). This finding indicates an increased likelihood of neonatal malaria when placental parasitisation is present. Similar observations were reported by Mukhtar et al.,[75] Balogun et al.[48] and Ibanga et al.[82]

Although neonatal malaria parasitaemia was detected in the present study, none of the affected neonates exhibited clinical symptoms such as fever or jaundice.

Interestingly, 3.0% (10/48) of neonates born to mothers without placental parasitaemia also had detectable neonatal malaria parasitaemia, though this finding was not statistically significant. This finding agrees with the observation of Nirjesy et al.[85] in Zaire where 17% of those with congenital malaria did not stem from women with peripheral nor placental malaria parasitaemia. This observation suggests that the

absence of placental parasitaemia does not necessarily preclude neonatal malaria. This perhaps implies that there may be other alternative mode of transmission or it may point to the limitations in the sensitivity of microscopic diagnostic method used.

5.1.5 Placental Malaria Parasitaemia and Neonatal Anaemia

A strong and statistically significant correlation was found between placental malaria parasitaemia and neonatal anaemia ($p = 0.010$). This finding is consistent with the report by Ibanga et al.[82] in Uyo. However, it contrasts with the findings of Falade et al.[76] and McGregor et al.[120], who found no significant association between placental malaria and neonatal anaemia.

Ibanga et al.[82] attributed their findings to high maternal anaemia resulting from poor utilization of malaria preventive measures, particularly ITNs. In the present study, only 13.5% of participants slept under ITNs, while 58.0% received at least one dose of IPT. The strong association observed may also be explained by immunopathological mechanisms. Exposure of the fetus to malaria antigens following damage to the placental barrier may predispose neonates to immunologically mediated haemolysis or dyserythropoiesis.[119] Additionally, poor antenatal compliance and inadequate access to comprehensive antenatal care services may have contributed to the high prevalence of neonatal anaemia, as evidenced by the finding that nearly over a half of neonates born to mothers without placental parasitaemia had low packed cell volume.

5.1.6 Placental Malaria Parasitaemia and Birth Weight

Placental malaria parasitaemia showed a statistically significant association with low birth weight ($p = 0.0001$). Neonates born to placental parasitaemic mothers had lower mean birth weights compared with those born to non-parasitaemic mothers. This finding agrees with reports from Ibanga et al.[82], Fehintola et al.[48],Tako et.[65] al and Quadrago et al.[66]

Conversely, studies conducted in Minna (Nigeria), Zaire, and Rwanda did not demonstrate a significant association between placental malaria and low birth weight.[73],[91],[92] The significant association observed in this study may be explained by poor antenantal compliance with low usage of haematinics and malaria preventive measures leading to possible maternal anaemia with subsequent fetal anaemia and low birth weight. It may also be explained by poor materno-fetal insufficiency resulting from mechanical obstruction of placental blood flow by parasitised erythrocytes and fibrinoid necrosis, which is the proposed mechanism underlying low birth weight in placental malaria.[89]

5.1.7 Placental Malaria Parasitaemia and Neonatal Mortality

The correlation between placental malaria parasitaemia and neonatal mortality was statistically significant at $p = 0.002$. In contrast, Omalu et al.[77] and McGregor et al.[112] reported no association between placental malaria and neonatal mortality. Although neonatal mortality related to placental malaria is often linked to low birth weight,[93,139] the mortalities recorded in this study did not show a direct association with low birth weight.

5.1.8 Placental Malaria Parasitaemia and Apgar Scores

No statistically significant association was observed between placental malaria parasitaemia and Apgar scores ($p = 0.425$). This finding is consistent with reports by Mokuolu et al.[92] and Ibanga et al.[82] Although some neonates born to placental parasitaemic mothers had low Apgar scores, the association was not significant. Given that Apgar scores primarily assess neonatal birth asphyxia, this suggests that placental malaria may not be a major determinant of immediate post-delivery neonatal adaptation.

5.1.9 Clinical Implications

The findings of this study have important clinical implications. First, placental malaria parasitaemia is not uncommon among pregnant women delivering in Owerri. Second, although neonatal jaundice was not observed, several adverse neonatal outcomes were significantly associated with placental malaria. Third, the detection of neonatal malaria parasitaemia among neonates born to mothers without placental parasitaemia indicates that negative placental microscopy does not necessarily exclude fetal malaria exposure.

This observation highlights the limitations of blood smear microscopy compared with more sensitive diagnostic methods such as placental histology and polymerase chain reaction (PCR). Furthermore, the high prevalence of neonatal low packed cell volume

suggests that factors beyond placental malaria, including poor antenatal attendance and inadequate use of haematinics, may contribute significantly to neonatal anaemia in the studied environment.

5.1.10 Strengths and Limitations of the Study

A major strength of this study is that it represents, to the best of the researcher's knowledge, the first multi-center investigation in Owerri assessing the relationship between placental malaria parasitaemia and neonatal outcomes.

However, the study has limitations. Owerri is a large and densely populated city, and findings from the three selected hospitals may not be fully generalizable to the entire population. In addition, while microscopy is sensitive and specific, it is inferior to placental histology and PCR for detecting placental malaria. Finally, although maternal packed cell volume was measured, its direct influence on neonatal packed cell volume was not assessed.

5.2 Conclusion

Placental malaria parasitaemia is not uncommon among parturient women in Owerri. It has a significant impact on neonatal outcomes, with positive correlations observed with neonatal malaria parasitaemia, neonatal anaemia, low birth weight, and neonatal mortality.

5.3 Summary of Significant Findings

1.The prevalence of maternal peripheral and placental malaria parasitaemia in Owerri was 69% and 14.0% respectively.

2.Placental malaria parasitaemia was significantly associated with neonatal malaria parasitaemia.

3.A strong association existed between placental malaria parasitaemia and neonatal anaemia.

4.Placental malaria parasitaemia was significantly associated with low birth weight.

5.A significant association was observed between placental malaria parasitaemia and neonatal mortality.

6.No significant association was found between placental malaria parasitaemia and Apgar scores.

5.4 Contributions to Knowledge

This study provides multi-center evidence on the burden of placental malaria parasitaemia and its effects on neonatal outcomes in Owerri. It contributes to existing knowledge by demonstrating that neonatal malaria and anaemia may occur even in the absence of detectable placental parasitaemia on microscopy, thereby highlighting diagnostic limitations and the need for more sensitive methods.

5.5 Recommendations

1.Malaria control measures during pregnancy, including IPT and ITN use, should be strongly encouraged.

2.Antenatal attendance should be improved to ensure full utilization of preventive and therapeutic interventions.

3.Future studies should adopt placental histology and molecular diagnostic techniques such as PCR for improved detection of placental malaria.

5.6 Suggestions for Further Studies

Further research is recommended to:

- Assess the influence of maternal anaemia on neonatal packed cell volume among placental malaria parasitaemic pregnancies.
- Examine the impact of antenatal attendance and malaria preventive practices on neonatal outcomes.
- Compare microscopy with placental histology and PCR in diagnosing placental malaria parasitaemia.

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