Review Article

Congenital malaria: Is it really rare?

Mandliya JC[1], Gupta R[2]

[1]Dr Jagdish Chandra Mandliya, Associate Professor, [2]Dr Rajesh Gupta, Assistant Professor; both from the Department of Pediatrics, Ruxmaniben Deepchand Gardi Medical College, Ujjain, M.P., India

Address for correspondence: Dr Jagdish Chandra Mandliya. Email: drjagdish1@yahool.co.in

Abstract

Congenital malaria is acquired from the mother prenatally or perinatally. In African countries congenital malaria is mainly caused by Plasmodium falciparum while in Asia and Europe, P.vivax is common. Postulated mechanism for congenital transmission of malaria parasites include maternal transfusion into the foetal circulation either at the time of delivery or during pregnancy, direct penetration through the chorionic villi or penetration through premature separation of placenta. Signs and symptoms include fever, restlessness, pallor, jaundice, poor feeding, vomiting, diarrhoea, cyanosis, hepatosplenomegaly and convulsions mostly between 10 and 30 days of age. Examination of peripheral blood by thick smear using light microscopy (LM) is the gold standard. Treatment of the congenital vivax malaria requires a blood schizonticide, like chloroquine. However, additional efforts should be made to establish safety profile, the correct dosage and formulation of Artemisinin-based combination therapy (ACT) in infants with a body weight of less than 5 kg.

Keywords: Congenital malaria, Plasmodium falciparum, Pregnancy, Hepatosplenomegaly

Background

Malaria in infants is classified according to the time of infection. Congenital malaria, defined as asexual parasites detected in the cord blood or in the peripheral blood during the first week of life [1],while neonatal malaria, which can occur within the first 28 days of life, is due to an infective mosquito bite after birth.[2] In many malaria-endemic countries, infants and children frequently die at home[3] and the cause of death remains undetermined and unrecorded [5,6,4].In endemic areas , distinguishing malaria acquired congenitally from that acquired by transmission from mosquitoes is difficult.[7] Congenital malaria can be acquired by transmission of parasites from mother to child during pregnancy or perinatally during labour.[8]

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Pregnant women are more susceptible to malaria than non-pregnant women, especially in first and second pregnancy[9]. In the past, congenital malaria was thought to be extremely rare both in endemic and non – endemic areas[10] but more recent studies however suggest that incidence has increased.[11] In African countries congenital malaria is mainly caused by Plasmodium falciparum[12]which is an important cause of abortions , miscarriages , premature births , intrauterine growth retardation and neonatal deaths,[13] while in Asia P.vivax is common[32]. In European countries most cases are due to P.malariae and P. Vivax[14] .

Problem areas

Congenital malaria was first described in 1876.[15] It is acquired from the mother prenatally or perinatally and is a serious problem in tropical areas[13].

Prevalence rate of congenital malaria between 0.3% and 46.7% have been obtained from both endemic and non-endemic areas [16-18]. In most cases of congenital malaria ,the baby is likely to have been infected while in–utero.[19] More than 2000 million people live in the areas where malaria transmission occurs and are therefore at risk of being infected.[8] Although congenital malaria develops in 01 percent of immune and 10 percent of nonimmune mothers in endemic areas , placental infection occurs in as many as one third of pregnant women.[70] It follows that 1000 million people are exposed to the risk of malaria when pregnant. Globally between 75000 and 2,00,000 infant deaths are attributed to malaria infection in pregnancy every year.[20,21]

The newborns of primigravidae are more susceptible to congenital malaria than those of the multigravidae.[71] Congenital malaria cases are rarely reported in the USA.[13] For the 81 cases of the congenital malaria reported in the USA in the past 40 years, the predominant infecting species was Plasmodium vivax.[23]

Most studies come from Sub-Saharan Africa, where approximately 25 million pregnant women are at risk of P.falciparum infection every year and one in four woman have evidence of placental infection at the time of delivery. P.falciparum infection during pregnancy in Africa rarely result in fever and therefore remain undetected and untreated. [24]

Compared to P.falciparum, P.vivax has a much wider distribution outside Africa and it extends far into the temperate zones.[22] P.vivax is common in Asia, America[24] , Europe [25], Singapore [26] and Thailand[27]. In 2012, first case from Latin Amarica has been reported in which findings show that maternal P.vivax infection still occurs in areas in the pathway towards malaria eliminalation.[28]

Causative agents

Congenital malaria usually occurs in the offspring of a non- immune mother with P.vivax and P.malariae infection, although it can be observed with any of the human malaria species.[13]

A well documented risk factor for developing neonatal and congenital malaria is maternal 3rd trimester malaria infection.[29] The higher prevalence of congenital malaria due to P. vivax than to P. falciparum in non-endemic countries is well established.[30,36] The most likely explanation is represented by the longer incubation time and the relatively wider clinical presentation in P.vivax malaria which allows for more maternal episodes to go undiagnosed and untreated . A potential additional determinant is represented by the contraindication during pregnancy of drugs that can eradicate the liver stage of parasite thus increasing the likelihood of late relapse.[25] P.falciparum has been documented in 6 out of 7 case studies of congenital malaria reported from Sri Lanka .[31]

Mechanism of transmission

Postulated mechanism for congenital transmission of malaria parasites include maternal transfusion into the foetal circulation either at the time of delivery or during pregnancy , direct penetration through the chorionic villi or penetration through premature separation of placenta.[31] The remarkable capacity of the foetus to resist infection has been demonstrated.[32]

This resistance can reflect the physical barrier of the placenta to infected red cells, the passive transfer of maternal IgG antibodies and the unfavourable environment offered by foetal erythrocytes for plasmodial replication due to their foetal haemoglobin composition and low free oxygen tension.[31,33]

In pregnancy, reduced lymphoproliferative response sustained by elevated levels of serum cortisol, loss of cell-mediated immunity in the mother, the presence of placenta, a new organ in the primigravidae, allows the parasite to bypass the existing host immunity, or allows placenta-specific phenotypes of *P. falciparum* to multiply. Pregnant women display a bias towards type-2 cytokines

and are therefore susceptible to diseases requiring type-1 responses for protection like TB, malaria. *P. falciparum* has the unique ability of cytoadhesion.

Chondroitin sulfate A and hyaluronic acid are the adhesion molecules for parasite attachment to placental cells.[34]

How it presents?

The first sign or symptom most commonly occurs between 10 and 30 days of age (range 14 hours to several months of age).[13] Signs and symptoms include fever, restlessness, pallor, jaundice, poor feeding, vomiting, diarroea, cyanosis, hepatosplenomegaly [13] and convulsions[35].

Severe thrombocytopenia with[15] or without bleeding is also a frequently reported feature of congenital malaria.[36,37,38] Infants with congenital or neonatal malaria may have a different clinical presentation than older children, and diagnosis may be confused with other neonatal diseases due to an overlap of clinical manifestation.[39]

Because of its non specific presentation with fever during the first 3 months of life, it is an important differential diagnosis when evaluating such infants with fever in the pediatric emergency department.[40] Some studies show atypical presentation of congenital malaria with no fever.[41]

In an article published in Archives of Pediatrics (2000), Balaka B. *et.al.* differentiated congenital malaria disease (CMD) from congenital malaria infection (CMI) in an endemic area.[42](Table 1)

Table 1: Clinical differentiation of Congenital Malaria

Congenital Malaria disease (CMD)	clinical manifestations associated with positive thick and thin blood films in a mother and her newborn
Congenital Malaria infection (CMI)	positive parasitemia but no clinical manifestations

An association between obstructive jaundice and neonatal malaria was noted by Patwari.[43] Hepatomegaly is rather less commonly noted than splenomegaly.[44] Younger the age of malarial onset , greater the severity of hepatic damage (and therefore jaundice).[45]

In a case study by Hewson MP, illustrates the difficulty of early diagnosis and the atypical nature of presentation in a preterm infant.[46]

Analysing the morbidity due to the congenital malaria in early infancy , Poespoprodjo JR *et.al.* noted that the case fatality rate was similar for inpatients with P.falciparum malaria and P. vivax whereas severe malarial anaemia was more prevalent among those with P.vivax malaria.[47]

How to diagnose?

Differentiating between congenital and acquired neonatal malaria can be difficult, especially in areas of intense malaria transmission.[66] In areas with limited resources, the capacity to diagnose malaria in young infants may be limited[67,68] and any issues with quality or accuracy of the diagnostic technique may result in the diagnosis of malaria being missed.[69] For many years, the diagnosis of plasmodium infection has been based on the examination of peripheral blood by thick smear using light microscopy (LM). This is a very specific test that performs when parasitemias are >1000 parasites /L.[48-50] Other technique used in the diagnosis of malarial infection include rapid diagnostic test (RDTs).[51]

Nucleic acid based amplification tests such as nested polymerase chain reaction (nPCR) have been widely used in major laboratories.[52] Both microscopy and PCR allow species discrimination, but both also identifies different parasite stages.[53]

Histopathology (HP) is also suitable for diagnosis of plasmodial infection in placental tissue. This is the gold standard in such cases .[54] Histopathology can detect parasites, malarial pigment (hemozoin) or both, any of which can establish the diagnosis.[55,56] (Table 2)

Table 2: Histopathological classification of Placental tissue inflammation due to Malaria parasite

Acute active infection	presence of parasites with pigment scarce or absent
Chronic active infection	presence of parasites and pigments relatively abundant
Past infections	exclusive presence of pigments

Out of above mentioned investigative modalities, LM has the best operational – economical qualification. nPCR and HP perform better compared with LM but field implementation of these two techniques remain a problem.[51]

Efforts are being made to design nucleic based test suitable for field amplification in rural settings, including isothermal loop amplification (LAMP).[57-60] The LAMP reactions are easy to set up and results can readily be assessed by detection of turbidity or more importantly , simply through the naked eye.[61]

How to treat?

Treatment of the congenital vivax malaria requires a blood schizonticide, like chloroqine.[25] Due to the absence of an exoerythrocytic life cycle in congenitally acquired malaria, chloroquine is the drug of choice for treatment. Unnecessary administration of primaquine phosphate for P.vivax infections was found in a review of cases from 1966 to 2005 in the United States.[23] Infection with chloroquine resistant strain require multiple drug therapy.[62] There are no official data on how to use ACT in this age group, despite the fact that malaria can occur at a very young age and that ACT offers greater efficacy and tolerability compared with quinine. At this time, a clear recommendation on the use of ACT can be difficult, due to a lack of data in infants with a body weight of less than 5 kg .[39]

Prevention strategy

Pregnant women in malaria endemic region may experience a variety of adverse consequences from malaria infection including maternal anemia , placental accumulation of parasites , low birth weight (LBW) from prematurity and IUGR, foetal parasite exposure , congenital infections and infant mortality linked to preterm LBW and IUGR- LBW.[20]

Preventive strategies include regular chemoprophylaxis, intermittent preventive treatment (IPT) with antimalarials and insecticide treated bednets.[63] Intermittent preventive treatment in pregnancy at antenatal visits with two doses of Sulfadoxime - Pyrimethamine (SP) is recommended for population groups in areas of high transmission who are particularly vulnerable to *Plasmodium* infection and its consequences.[64]

The WHO 20th malarial committee designated IPT as the preferred approach to reduce the adverse consequences of malaria during pregnancy.[65]

Conclusion

Congenital malaria was supposed to be rare in the past but recent data suggests its higher prevalence in endemic regions of the world especially in Africa and Asia. High suspicion of index should be considered diagnosing malaria in pregnancy to prevent congenital malaria. Apart from conventional preventive measures, it is recommended to develop evidence based diagnostic criteria and treatment guidelines for congenital malaria.

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