

Congenital Malaria Presenting as Neonatal Sepsis

Sahana Devadas, Manu Malenahalli Ashok*, Sarala Sabapathy, Rekha Takkalakki Devendrappa

Abstract

Congenital malaria is a rare disease that occurs through vertical transmission of malarial parasites from the mother to the neonate, either during pregnancy or delivery.

A 23-day-old male neonate presented with fever, refusal of feeds, and hurried breathing for the past 3 days. On enquiry, it was found that antenatally the mother had fever with chills intermittently throughout gestation but had not taken any treatment. The neonate was hemodynamically stable but pale and had grade 3 splenomegaly. Peripheral smear revealed ring and trophozoite forms of *Plasmodium vivax* and a few ring forms of *Plasmodium falciparum*, suggestive of congenital malaria. The peripheral smear of the mother also revealed trophozoite forms of *P vivax*.

Clinical features of congenital malaria include anemia, fever, hepatosplenomegaly, poor feeding, jaundice, and thrombocytopenia. The present case had most of the features. As the condition was confirmed with peripheral smear, the treatment for the same was initiated with blood schizonticides. As congenital malaria is a rare condition, it should be included in the differential diagnosis of neonatal sepsis.

Key Words: Congenital malaria, neonatal sepsis, peripheral smear, vertical transmission, blood schizonticides, *Plasmodium vivax*, *Plasmodium falciparum*

*Correspondence

Dr Manu Malenahalli Ashok

Junior Resident

Department of Pediatrics

Bangalore Medical College and Research
Institute

Bengaluru 560002, Karnataka
India

E-mail: manu.fulloflife@gmail.com

Introduction

Congenital malaria is a delayed complication of maternal malaria. Very few cases have been reported in the literature. Congenital malaria occurs when parasites are transmitted vertically from the mother to the child during pregnancy or perinatally during labor. The condition is rarely diagnosed in infants of asymptomatic mothers if it is not detected earlier both in the mother and the neonate.¹ Nevertheless, the incidence of congenital malaria is on the rise currently because of high resistance and virulence levels of parasites due to altered antigenic determinants.² Although malaria is endemic in India, studies related to this condition are rare from India.²⁻⁴

Case Report

A 23-day-old male neonate presented with fever and refusal to feed for the past 3 days, to the NICU of Vani Vilas Hospital (Bengaluru, Karnataka, India). Antenatally, the mother had fever with chills intermittently throughout the pregnancy but did not take any treatment.

On examination, the neonate was pale and tachypnic, had grade 3 splenomegaly, and was hemodynamically stable; other systems were normal. Based on the history and further examination, the neonate was provisionally diagnosed to have sepsis and hence started on IV antibiotics.

In view of pallor and splenomegaly, peripheral smear test was conducted, which showed ring and trophozoite forms of *Plasmodium vivax* and a few ring forms of *Plasmodium falciparum* and normocytic normochromic anemia with thrombocytopenia. All these indicated malaria of mixed infections. Considering the age of the neonate, the condition was suspected as congenital malaria. Hence, the mother was also screened. The peripheral smear test of the mother revealed the trophozoite form of *P vivax*. Anemia was treated with packed RBC transfusion in the neonate.

The neonate was treated with quinine 25 mg/kg/d for 7 days, TID. As parasitemia was persistent even after 7 days of treatment with quinine, IV artesunate

(3 mg/kg, 5 doses at 0, 12, 24, 48, and 72 h) and IV clindamycin (10 mg/kg/d, BID, for 7 d) were administered. After completion of this course of combination therapy, peripheral smear showed no hemoparasites. At the time of discharge, the neonate was pink and playful with improved hemoglobin level, normal platelet count, and reduced serum total bilirubin, and he was feeding well (Table and Figures 1–3).

Table. Hematological and Biochemical Parameters of the Neonate Before and After the Treatment

Parameter	At Admission	After Treatment
Hemoglobin, g%	5.8	9
Total Count, cells/mm ³	6600	8200
Platelet Count, cells/mm ³	52,000	2.57 lakh
Hematocrit, %	21	26
STB, mg/dL	7.2	2.1
SGOT/SGPT, IU/L	62/42	54/46

SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase; STB, serum total bilirubin.



Figure 1. Newborn With Grade 3 Splenomegaly

Discussion

Malaria during pregnancy and in newborns results in a significant disease burden and is estimated to cause > 3,00,000 fetal and neonatal deaths and 2500 deaths of pregnant women, worldwide, annually.^{5,6}

It can be acquired by transmission of parasites from the mother to the child during pregnancy or perinatally

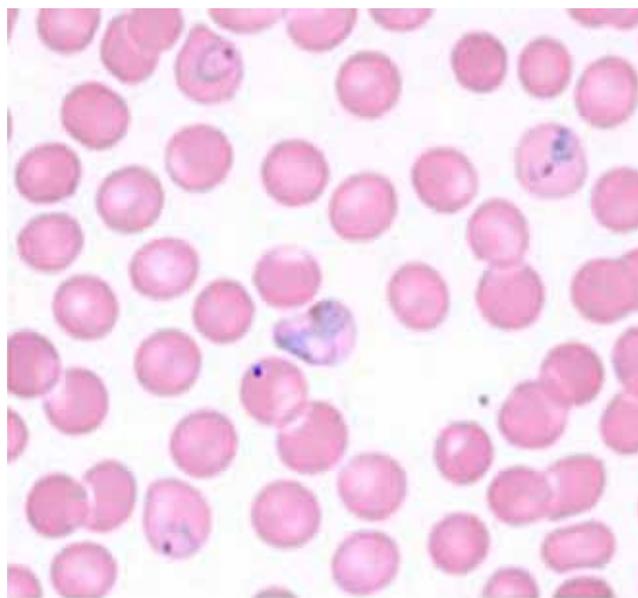


Figure 2. Peripheral Smear Showing the Trophozoite Form of *Plasmodium vivax* and a Few Ring Forms

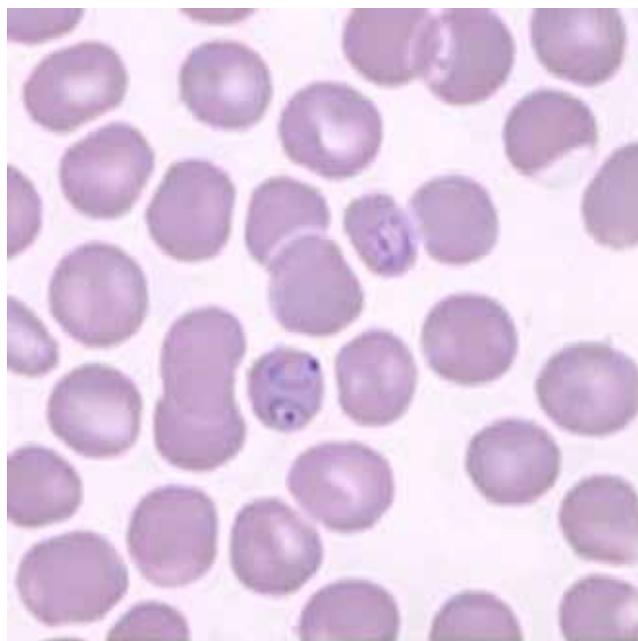


Figure 3. Peripheral Smear Showing Ring Forms of *Plasmodium falciparum*

during labor. Syncytiotrophoblast, villous disruption, syncytial knot formation, and fibrin-type fibrinoid deposition are characteristics of malarial infection.

Syncytial destruction causes low birth weight and congenital infections.⁷

During pregnancy, malaria may have 2 kinds of effects on the fetus: (1) it can result in premature labor, intra-uterine growth retardation, high perinatal mortality, anemia, miscarriage, low birth weight, and maternal deaths (both maternal and neonate death if infected by *P vivax* and *P falciparum*)⁶ and (2) it may also protect the neonate against malaria and severe diseases via acquired maternal immunity.⁸ Although both IgG and IgM antimalarial antibodies can be detected in the maternal blood, only IgG crosses the placental barrier.

Although congenital malaria can rarely present within 7 days, one such case was reported by Sotimehina et al.⁹ More commonly, onset of symptoms may be prolonged. The explanation for this varied presentation is based on transmission of infection in the antenatal period or during delivery and presence of transplacentally acquired maternal antibody (IgG). Based on the available literature, the mean age of onset of symptoms of congenital malaria is between 3 and 12 weeks after birth, which coincides with the half-life of maternal IgG antibody in neonates.¹⁰

Once the maternal antibodies start to wane, the symptoms show up in the neonate, who is healthy at birth. Fever may be occasionally absent. The other associated symptoms include irritability, refusal of feeds, hepatosplenomegaly, hemolytic anemia, jaundice, and thrombocytopenia. The present case had many of the aforementioned features. Peripheral smear examination is a simple test to diagnose congenital malaria.

The treatment of congenital malaria involves blood schizonticides.¹¹ Primaquine can be avoided because there is no hepatic stage of plasmodium. In this case, we initially treated the neonate with quinine, then switched to parenteral artesunate and clindamycin because of persistent parasitemia.

Conclusion

Congenital malaria is relatively a rare condition that should be included in the differential diagnosis of sepsis, fever without focus, unexplained anemia with jaundice,

and hepatosplenomegaly in malaria-endemic regions. The neonate may be healthy at birth and it can also occur in a neonate whose mother is asymptomatic. Hence, a high index of suspicion and timely interventions are crucial to prevent the untoward outcome. This case is being reported for its rarity and to create awareness.

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Author Affiliations

Dr Sahana Devadas, Professor; **Dr Manu Malenahalli Ashok**, Junior Resident; **Dr Sarala Sabapathy**, Professor and Head; **Dr Rekha Takkalakki Devendrappa**, Department of Pediatrics, Vanivilas Hospital, Victoria Hospital Campus, Fort Road, KR Market, Bengaluru 560002, Karnataka, India