Congenital malaria presenting as late-onset sepsis in infant with mother treated with antimalarial therapy: A case report

Prachi Bhageria¹, Anuj Dhyani², Vipul Gupta³, Arvind Gupta⁴, Seshendra Sharma³

From ¹Junior Resident, ²Senior Resident, ³Post Graduate Student, ⁴Head, Department of Pediatrics, Asian Institute of Medical Sciences, Faridabad, Haryana, India

Correspondence to: Dr. Prachi Bhageria, Department of Pediatrics, Asian Institute of Medical Sciences, Faridabad, Haryana, India.
E-mail: prachibhageria@gmail.com
Received - 19 August 2019 Initial Review - 30 September 2019 Accepted - 06 November 2019

ABSTRACT

Incidence of congenital malaria has been reported to be in the range between 0.3% and 30% in both the endemic and non-endemic areas. Malaria in pregnancy and congenital malaria are epidemiologically less investigated in India. Timely intervention and awareness are necessary for desirable outcome and prevention of the condition and to prevent perinatal and maternal morbidity and mortality. Herein, we report a case of a 1-month-old male baby, presenting with high-grade fever, feeding difficulty, and hepatosplenomegaly on examination. Mother had a history of chills and rigors in her first trimester and was found to be smear positive for Plasmodium vivax. Investigations of the child revealed thrombocytopenia, raised C-reactive protein and positive peripheral smear for P. vivax. She was treated with Inj. artesunate and cured. Thus, congenital malaria should be considered as a differential diagnosis in newborn presenting with clinical features of sepsis even when mother has been adequately treated.

Key words: Artesunate, Congenital malaria, Placenta, Plasmodium vivax

Congenital malaria is commonly defined as the presence of Plasmodium spp. parasites in the peripheral blood during the first 7 days of life where transmission occurs transplacentally [1]. Parasites are identified in the peripheral blood of neonate and not the umbilical cord blood or placenta [1]. Although the use of umbilical cord blood has been proposed as an alternative to peripheral blood, it does not represent active infection. Falade et al. noted spontaneous clearance of parasitemia in 62% of neonates born, before day 2 of birth, and maternal and placental parasitemia were the most important risk factors for patent neonatal parasitemia [2]. The incidence of congenital malaria depends on various factors such as geographical area, time period of reporting, and clinical definition [1]. Incidence of congenital malaria has been reported to be in the range between 0.3% and 30% both in endemic and non-endemic areas [3-8]. Most of these studies were conducted in areas with Plasmodium falciparum as the predominant species, whereas in India, it is Plasmodium vivax [4,9-15].

The rate of transmission of the parasite is low during pregnancy due to the placenta acting as a barrier to maternally infected red blood cells, passive immunity by maternal antibodies, and the protective role of fetal hemoglobin (Hb) [1]. Placental malaria is asymptomatic and silently causes fetal wasting. Among the firstborn, it can significantly affect the birth weight, the differences usually around 150–300 g [5]. Malaria during pregnancy is an unestablished risk factor for increased infant morbidity and mortality in endemic areas. Maternal malaria during pregnancy may cause serious anemia and adversely affect the placental circulation interfering with nutrition and oxygenation of the fetus. It is associated with abortion, stillborn, prematurity, fetal growth retardation, and neonatal deaths. Neonate can present with fever, irritability, feeding problems, hepatosplenomegaly, anemia, and jaundice [5].

CASE REPORT

A 1-month-old male baby was admitted to our institute with complaints of high-grade fever for 5 days and feeding difficulty for 1 day. The baby was born vaginally at term with birth weight 2.3 kg. Postnatal period was uneventful. On examination, the baby was irritable but consolable, febrile with axillary temperature of 101°F. Systemic examination was unremarkable except for mild splenomegaly. Liver was palpable 1.5 cm below costal margin, normal in consistency, and texture. Spleen was palpable 1 cm below costal margin and was firm in consistency. Pending investigation reports, the baby was started on intravenous (IV) fluids and empirical antibiotics (Inj. cefotaxime).

Investigations were done which showed Hb 12.0 g/dl, total lymphocyte count 8200/mm³, platelet count 0.28 lacs/mm³, and C-reactive protein 20.55 mg/dl. Peripheral smear showed the presence of schizonts of P. vivax, normocytic normochromic picture, and white blood cell showing left shift with increased immature forms (Fig. 1). Malaria parasite rapid test came out to be positive for the parasite.

Antenatal history of mother was reviewed and she was found to have a history of chills and rigors in her first trimester and was
smear positive for *P. vivax*. She was treated with Inj. artesunate in adequate dose (120 mg IV for three doses) over 2 days followed by a combination therapy of artemether and lumefantrine (80 mg/480 mg) twice daily for 3 days. Based on the history, clinical examination, and presentation of baby, the diagnosis of congenital malaria was made. The child was treated with Inj. artesunate (10 mg once daily for 7 days) and Inj. clindamycin (40 mg twice daily for 7 days). The child showed clinical improvement and repeated investigations after 48 h of therapy, showed a prompt increase in platelet counts (206,000/mm$^3$) along with the clearance of malarial parasites in blood.

**DISCUSSION**

It is estimated that the incidence of congenital malaria is 0.3–30%, as reported by Fischer [3]. Although malaria continues to be a major public health problem in India and pregnant women have been described as the vulnerable group, there have been very few epidemiological studies to determine disease burden in pregnancy and infants [4-6]. The pathophysiology of congenital malaria is due to maternal transfusion into the fetal circulation during pregnancy or at delivery or due to direct penetration of chorionic villi. The incidence of congenital malaria is low in pregnancy as the placenta acts as a barrier to the malarial parasites, the role of passive immunity by maternal antibodies, and due to the protective role of fetal Hb [5,6].

The symptoms of congenital malaria are usually delayed and literature describes the clinical symptom onset to be between 3 and 6 weeks after birth [1]. Placental malaria does not always end in malarial infection of newborn but can lead to low birth weight of newborns, intraterine growth restriction, preterm labor, and intraterine fetal death.

In this case, mother was smear positive for *P. vivax* in her first trimester for which she was adequately treated. However, it failed to limit transmission to the newborn emphasizing the fact that congenital malaria should be kept in mind as differential diagnosis in newborn presenting with clinical features of sepsis even if mother has been adequately treated or if the infection is acquired in the first trimester.

**CONCLUSION**

Antimalarial therapy to pregnant mother might not always prevent transmission of malaria to newborn. Congenital malaria should be considered as a differential diagnosis in newborn presenting with clinical features of sepsis even if the infection is acquired in the first trimester and the mother has been adequately treated.

**REFERENCES**