

CONGENITAL MALARIA*

A Case Report

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Congenital malaria is an uncommon disease even in endemic areas. A 19-day-old female infant with congenital malaria is presented. The mother of the patient was diagnosed to have malaria at the seventh month of gestation and was treated with chloroquine orally for three days. No malarial prophylaxis was given. The infant developed fever, hyperbilirubinemia, anemia and hepatosplenomegaly postnatally. Thin blood smears revealed many *Plasmodium vivax* parasites. She was treated with oral chloroquine for three days. We emphasize the importance of adequate antenatal medical therapy and prophylaxis during pregnancy. *Key words:* congenital malaria, newborn, pregnancy.

Congenital clinical malaria is rare, even in endemic areas, although the presence of parasites in cord blood is not infrequently reported. It occurs in the offsprings of 0.3 percent of immune and 10 percent of nonimmune mothers with malaria; placental infection occurs in more than 30 percent in endemic areas^{1,2}.

Vertical transmission of malaria from the symptomatic or asymptomatic mother to the fetus, attributable to the failure of the barrier action of the placenta, is generally believed to be uncommon^{1,3}.

We report a case of congenital *Plasmodium vivax* malaria to emphasize the importance of antenatal antimalarial therapy.

Case Report

A 19-day-old female neonate was admitted to our hospital with a history of fever and poor feeding for two days. She was born to a 19-year-old mother. At the seventh month of gestation the mother had a febrile illness that was diagnosed as malaria by blood smear and was treated with chloroquine orally for three days. No malaria prophylaxis was given. She had fever and chills one week before delivery. Unfortunately, she did not seek medical care at that time and thus was not given any treatment. Pregnancy and delivery were otherwise uncomplicated.

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Physical examination revealed an icteric, but otherwise well-appearing neonate with mild dehydration, heart rate 176/minute, respiratory rate 60/minute, axillary temperature 38.5 °C, length 52 cm, weight 2670 g and head circumference 36 cm. Significant pathologic findings included a palpable liver four cm below the right costal margin and a palpable spleen four cm below the left costal margin.

Laboratory findings revealed: hemoglobin 8.5 g/dl; mean corpuscular volume 95 fl; white blood cell count $7.6 \times 10^9/L$ with 72 percent lymphocytes, 28 percent neutrophils; platelets $50,000 \times 10^9/L$; and unconjugated bilirubin 9.22 mg/dl. Other biochemical tests and urinalysis were normal. Peripheral blood smear revealed many *Plasmodium vivax* parasites.

She was treated with oral chloroquine phosphate, 10 mg/kg of base as an initial dose, followed in six hours by 5 mg/kg. Subsequent doses of 5 mg/kg of chloroquine were given at 24 and 48 hours after the first dose. She was afebrile on the first day, and on the third day of treatment no parasite was noted on peripheral blood smear. Her mother was treated with chloroquine and primaquine.

Discussion

Congenital malaria can develop with any of the species of *Plasmodium* but most commonly is due to *falciparum* and *vivax*³.

In pregnancy, immunity acquired in the past against malaria is often lost and susceptibility to malaria increases. Besides, pregnancy is associated with heightened risk for relapse or for an increase in parasitemia^{4, 5}, possibly due to a reduction in the rate of gamma-globulin synthesis⁶. The exact mechanism and time of transmission of the malarial parasites from the mother to the fetus are not known. The placenta normally forms an effective barrier against the parasite. However, when the placenta is damaged, either during delivery or owing to placental abnormalities, infected red blood cells are transferred into the fetal circulation^{1, 3, 7, 8}. Nonetheless, infants of mothers who have had malaria in the past are probably less affected because of maternal IgG transfer, but they are not completely protected⁹. Congenital malaria is more common among infants of women who had clinical attacks of malaria during pregnancy than of those with chronic subclinical infections. However, congenital malaria may occur in infants of mothers who are asymptomatic throughout their pregnancy^{5, 10}.

In addition to congenital transmission, the newborn may acquire malaria by transfusion of blood products, as a simple transfusion or exchange transfusion, postnatally^{3, 5}.

Symptomatic congenital malaria should be distinguished from cordblood parasitemia and transfusion-induced malaria. In cord blood parasitemia, parasites are cleared without involving the peripheral circulation and it is not associated with clinical disease^{3, 5}.

The diagnosis of congenital malaria can be easily missed if it is not considered, especially in infants of asymptomatic women¹⁰. The examination of a single peripheral blood smear may not yield the diagnosis.

Clinical signs and symptoms similar to other congenital infections, such as fever, anorexia, hepatosplenomegaly, hemolytic anemia and hyperbilirubinemia present days or weeks after birth. Fever pattern is generally not synchronized^{1, 3, 11}. Our patient had several features of congenital malaria such as fever, poor feeding, hepatosplenomegaly, jaundice and thrombocytopenia.

Treatment of congenital malaria, in a patient who is not very sick consists of oral administration of chloroquine (10 mg/kg of base followed by 5 mg/kg of base at 6, 24 and 48 hours). Primaquine is not needed for the newborn infant, because no persistent liver phase exists in congenitally acquired infections^{3, 8, 9}. In severely ill infants parenteral chloroquine is effective and safe¹². If chloroquine-resistant *P. falciparum* is suspected, mefloquine and quinine are the drugs of choice. In addition to quinine, pyrimethamine in combination with a sulfonamide (Fansidar) should be used^{3, 5, 8}.

As malaria often results in abortion, prematurity, low birth weight, still birth and neonatal death, adequate antenatal treatment is essential. Chloroquine is known to be safe in pregnancy, including use as a prophylactic agent in endemic areas¹³. Primaquine should not be given during pregnancy because of unknown effects on the fetus. In *P. vivax* and *P. ovale* infections, it is recommended to give chloroquine therapy and then to continue with the prophylactic treatment once weekly until after delivery. Afterwards, primaquine can be given for prophylaxis^{10, 13}. The mother of our patient was prescribed chloroquine therapy at seven months of gestational age. As chloroquine prophylaxis was not given, relapse and subsequent congenital malaria occurred.

We recommend that once malaria is diagnosed in pregnancy, especially in nonimmune women, chloroquine chemoprophylaxis should be continued until delivery.

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