Congenital malaria: Is it really rare? A case report

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ABSTRACT

A 7-day-old term male infant weighing 2.4 kg was admitted with fever, pallor, icterus, and splenomegaly for 3 days. The primi mother was treated for pyrexia in the last trimester. Investigation revealed anemia, hyperbilirubinemia, and thrombocytopenia and demonstrated *Plasmodium vivax* in peripheral blood smear and card test. C-reactive protein was raised and blood culture was sterile. The baby was responded well to intravenous (IV) artesunate. Recent studies suggest that congenital malaria (CM) is not as rare as previously thought. Cord blood shows greater parasitemia as compared to neonatal blood. Besides light microscopy, plasmodium antigen detection and polymerase chain reaction of blood may help in diagnosis. CM can be confused with toxoplasmosis, rubella, cytomegalovirus, herpes simplex syndrome, and neonatal sepsis.

Key words: *Congenital malaria, Neonatal malaria, Placental transmission, Plasmodium vivax*

Congenital malaria (CM) is believed to be rare due to belief that physical barrier of placenta to infected red cells, passive transfer of maternal antimalarial antibodies, and poor plasmodial replication in low free oxygen tension in fetal hemoglobin protect a neonate from acquiring malarial infection. Congenital malaria is defined as malaria acquired by the fetus or newborn from the mother, either in utero or at parturition. However there is no consensus on application of this definition. It can also be defined as the presence of asexual malaria parasites in the erythrocytes of newborns aged <7 days [1-3]. However, recent cross-sectional studies suggest the prevalence of 10.8-54.2% in the endemic areas [3,4]. The placental barrier does not seem to be very effective in preventing transplacental transmission of maternal malarial infection to the neonate in non-immune mothers. However, in 93% of such neonates, spontaneous clearing of infection takes place [2].

A study from India showed a lower incidence of CM 29/1000 live births [4,5]. CM is an important cause of abortions, miscarriage, stillbirths, premature births, intrauterine growth restriction, and neonatal deaths [6]. However, placental infection occurs in as many as one-third of women who acquire an infection during pregnancy. While CM due to *Plasmodium falciparum* is frequently reported from the African continent, *Plasmodium vivax* CM is reported from Southeast Asia and India sporadically. Here, we report one case of CM due to *P. vivax*.

CASE REPORT

Our case was a 7-day-old term appropriate for gestational age male baby, weighing 2.4 kg (birth weight 2.5 kg) admitted to neonatal intensive care unit with the history of refusal to feed, marked pallor, and yellowish discoloration of eyes for 3 days. He was delivered by normal vaginal delivery at a government hospital and was hospitalized there for 5 days for birth asphyxia (APGAR was not documented). His clinical examination revealed heart rate of 140/min, respiratory rate of 55/min, temperature 98.4°F (by rectal), and capillary refill time <2 s. He had severe pallor, and icterus was present till legs and there were no dysmorphic features. On systemic examination, splenomegaly 3 cm below costal margin was present and the rest of systemic examination findings were within normal limits.

His primigravida mother had a history of moderate grade pyrexia with chills and anemia for 2 weeks in the last trimester for which she received therapy from some practitioner. There was no history of multiple vaginal examinations, leaking or foul smelling discharge/abortions/still births, or antepartum hemorrhage in the past. Her peripheral blood examination did not show any evidence of parasitemia.

On laboratory investigations, his hemoglobin was 9.7 g/dl, total leukocyte counts were 9360/mm^3, platelets were 0.14 lacks/mm^3, and random blood sugar was 42 mg/dl. His serum bilirubin was 22.8 mg/dl with an indirect fraction of 7 mg/dl and direct fraction of 5.8 mg/dl. Peripheral smear examination revealed *P. vivax* parasite trophozoites. Card test was positive for *P. vivax* antigen. His C-reactive protein was elevated (88.4 mg/dl), but blood culture was sterile. Serology for toxoplasmosis, rubella, cytomegalovirus, herpes simplex, and HIV was negative. The baby was managed with IV artesunate 3 mg/kg/day for 6 days along with supportive therapy of IV antibiotics, IV fluids, platelet and packed cell transfusion, and phototherapy. His condition

improved with regression of spleen, liver, pallor, and icterus. Repeat peripheral smear examination after therapy showed no hemoparasite. He was discharged after 7 days of hospitalization.

DISCUSSION

Lack of consensus on definition of CM and variety of definitions of CM and neonatal malaria has resulted in researchers reporting the different prevalence of the illness. We considered the definition of CM as the presence of asexual stages of malarial parasite in cord blood smear at delivery or peripheral blood smear of the neonate in first 7 days of life, irrespective of clinical symptoms [2]. The heavily parasitized placenta can result in transplacental transmission of malarial parasite (MP) both in cases of falciparum and vivax malaria [7,8]. The pregnant women in the endemic areas may experience heavy parasitemia, fever, severe anemia, abortions, stillbirths, and heavy placental parasitization [9]. Higher parasitemia is noted in cord blood as compared to neonatal blood [10]. Therefore, one must keep this condition in mind in unimmunized or semi-immune mother traveling to endemic zones. CM does not seem to be a rare disease as previously thought.

The onset of CM usually occurs at 10-30 days after birth, coinciding with the estimated half-life of maternal immunoglobulin in the neonate. Predominant clinical features include fever, anemia, and splenomegaly in the majority of cases. Other clinical features include poor feeding, regurgitation, diarrhea, drowsiness, restlessness, cyanosis, jaundice, and hepatomegaly. Maternal history suggestive of residing in the endemic zone, present or past malarial infection, stillbirths, abortions, HIV infections, and low-birth-weight babies should raise suspicion about this condition. The neonate may be symptomatic even on the 1st day of life. The delayed presentation may be due to fetal hemoglobin, abnormal hemoglobin resistant to malarial infections, and partial antimalarial chemotherapy during pregnancy.

Diagnosis of CM is established by microscopic identification of organism on Giemsa-stained thin and thick smear of cord blood or neonatal peripheral smear. The diagnosis of CM is frequently missed due to the limited sensitivity of light microscopy and additional investigations such as plasmodial antigen detection and polymerase chain reaction of blood may be necessary to arrive at diagnosis [11].

In the USA, whether symptomatic or not babies born to non-immune mothers with malaria are treated with quinine. Chloroquine-resistant babies have shown a good response to sulfadoxine-pyrimethamine combinations. Another regime approved in the USA is quinine plus clindamycin in both *P. vivax* and *P. falciparum* malarial infections [1]. In India, CM babies generally do not respond to chloroquine but respond well to oral and IV artesunate. IV artesunate is recommended as the drug of choice in all cases of severe malaria including young children. Primaquine is not required in infants with CM with *P. vivax*, but infected mothers can be treated with it after excluding G6PD deficiency [3,12,13].

CONCLUSION

CM prevalence should not be underestimated in the endemic zones, and a high index of suspicion should be kept in mothers and neonates with relevant risk factors. As clinical features of CM resemble with neonatal sepsis, malarial screening of febrile neonates especially in the endemic zone is suggested.

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