Neonatal Malaria : Report of two Cases

Lt Col C Vidyashankar*, Lt Col RK Choudhary†, Lt Col (Ms) Shobana Das#

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Introduction

There has been a resurgence of malaria in India despite ongoing malaria control programmes. The province of Jharkhand in Eastern India is one such area where malaria is rampant. Among the unusual presentations of malaria is the occurrence of malaria in neonates.

Neonates are considered relatively resistant to malaria because of the preponderance of fetal red blood corpuscles, which are relatively resistant to penetration by the malarial parasite. Neonatal malaria can be either due to vertical transmission from the mother following a breach in the placental barrier, or due to a fresh infection in the neonate either by transfusions or mosquito bites [1]. We report two cases of chloroquine resistant falciparum malaria in neonates, one of which was in a premature neonate.

Case Report

The first case, a 26 day old male neonate with a weight of 3.5 kg was admitted with history of fever of two days duration. The delivery was normal with a birth weight of 2.9 kg. The neonate was active and reflexes were good. Spleen and liver were palpable at 3 cm below the costal margin. There were no other positive findings on examination. Peripheral smear examination revealed a mixed infection with *Plasmodium vivax* and *P. falciparum*. 10% of red blood cells were infected with malarial parasites.

The neonate was initially treated with a course of chloroquine syrup (15 mg/kg on day -1, 5 mg/kg on days 2 and 3). Though the neonate became afebrile on day-2 of treatment, the *P. falciparum* parasitemia persisted at the same levels till day 4. R3 resistance was suspected and the neonate was then given a seven day course of oral quinine (10mg/kg 8 hourly) which resulted in clearance of parasitemia and clinical recovery. The neonate was discharged on completion of quinine treatment. Repeat peripheral smear examinations for malarial parasite done 7 days, 14 days and 28 days later were negative.

The second case was a female neonate, born premature at 32 weeks gestation with a birth weight of 1.7 kg and developed jaundice on the tenth day of life. Though the neonate was jaundiced, the reflexes, activity and appetite were normal. The neonate had a palpable hepatosplenomegaly of 3 cm.

The neonate was investigated and found negative for haemolysis and blood group incompatibility. The serum bilirubin increased to 15mg% (conjugated hyperbilirubinemia of 8mg%) with elevated hepatic enzymes. The glucose 6 phosphate dehydrogenase levels were normal. An ultrasound examination of abdomen for structural abnormalities of liver and biliary tract was negative. Sepsis screen was negative. Peripheral smear examination for malarial parasites carried out on day 10,11, and 12 were negative.

The neonate developed intermittent fever on the twenty second day of life, which was diagnosed as falciparum malaria with a parasitemia of 15% on repeat peripheral smear examination. The parasitemia persisted at the same levels despite a three day course of chloroquine given orally, following which she was started on intramuscular artesunate in a dose schedule of 3.2 mg/kg body weight on the first day, followed by 1.6 mg/kg for six days. The neonate responded to artesunate and remained free from relapse. Though the initial peripheral smear examinations for malaria were negative, in the absence of evidence of other causes of jaundice, the conjugated hyperbilirubinemia could have been due to malarial hepatitis. The neonate also had features of R3 resistance.

Both the neonates were given a single oral dose of pyrimethamine-sulfadoxine (125 mg pyrimethamine and 6.25 mg sulfadoxine) prior to discharge. Neither of the infants had received blood transfusions prior to the onset of fever. None of the mothers gave history of malaria in the antenatal period, while their peripheral smear examination following delivery were negative for malaria.

Discussion

Malarial infection in the neonate occurs predominantly through vertical transmission from the mother, with transfusion-mediated infections and acquired forms being less common. Unless the parasitemia is severe, malarial infection is unlikely in neonates because of the protection offered by the placenta, presence of maternal antibodies and preponderance of fetal red blood cells. Malaria in the neonate presents as fever, apnea and respiratory distress, diarrhoea, anaemia, jaundice, hepatosplenomegaly and thrombocytopenia [1]. The symptoms and signs closely mimic sepsis and hence a high index of suspicion is needed to diagnose this condition.

*Classified Specialist (Paediatrics), †Classified Specialist (Pathology), Military Hospital, Namkum, #Officer Commanding, SHO, Namkum.

While neonatal malaria has been reported in Africa [2] there is a paucity of similar reports from other parts of the world. Reports of malaria in the neonatal period were common in India in the 1970's and 1980's [3,4]. However, though there has been a resurgence of malaria in India in the last decade, reports of neonatal malaria are hard to come by now.

In our report, the first neonate presented with fever and had a mixed infection that was resistant to chloroquine. Infection in this case is likely to have been acquired from environment because of the time lag between birth and onset of symptoms and the negative history in mother. The second was a premature neonate that presented on the tenth day of life with jaundice. Though the mother showed no features of malaria, the short incubation period suggests that the infection in this case could be congenital. The absence of parasitemia in mothers of neonates with congenital malaria has also been reported in a study from Uganda. This could be because of low levels of parasitemia that may not be picked up on routine microscopy [5]. Malaria in premature neonates is extremely rare, with only a few case reports existing in English-medical literature [6,7]. To the best of our knowledge, this is the first instance of use of artesunate in a premature neonate.

Reports of neonatal malaria resistant to chloroquine have been on the rise in the last decade. 25% cases of congenital malaria from Nigeria were found resistant to chloroquine [1]. Reports of resistance to quinine have also been noted and are a cause for serious concern because of limited experience in use of alternates like halofantrine, mefloquine and artesunate in neonates

[1,8,9]. It is a matter of concern that both the neonates in our report were resistant to chloroquine, though they eventually responded to other anti-malarials.

To conclude we have reported two cases of chloroquine resistant falciparum malaria from India, one of which was in a premature neonate. In endemic areas, malaria has to be considered in the differential diagnosis of neonatal sepsis.

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Answer to Radiological Quiz :

Chest radiograph and CT scan chest show miliary shadows. CT scan abdomen shows multiple hypoechoic lesions in liver and spleen. This individual was a case of HIV infection. Liver biopsy showed the lesions to be tubercular granulomas with abundance of acid-fast bacilli in necrotising lesions.

HIV infection has a significant impact on the demographics of tuberculosis including miliary tuberculosis. Extra pulmonary disease occurs in more than 70% patients with tuberculosis and pre-existing AIDS [1]. Lesions can be seen in spleen, liver, lungs, kidneys, adrenals and eyes [2]. In majority of cases with splenic involvement, the lesions are in the form of

multiple abscesses. Most often, the finding of multiple hypodense, small splenic foci is the result of granulomas or abscesses caused by *Mycobacteria*, *Pneumocystis carinii* or fungi. Similar small abscesses are frequently reported with Gram-negative infection [3].

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