CASE PRESENTATION

Congenital Malaria

Akbar Mohsin Mohammad, MD, FAAP*
Govind Swaminathan, MBBS, DCH**

Congenital malaria is extremely rare, due to the passive immunity acquired from mothers who live in endemic areas. This immunity exists in spite of the fact that the placenta is often infected. This is followed by a period of a few months when mild attacks occur as a result of waning immunity. Severe attacks develop between the ages of nine months and two years[1,2].

When an infant is born to an infected mother, malaria parasites may cross the placenta and congenital malaria will ensue. We present a case of malaria in a three weeks old infant born to a mother who suffered from malaria and was treated during pregnancy.

THE CASE

SMS, a 23-day old Sudani male infant was admitted to the paediatric ward of Bahrain Defence Force Hospital, Bahrain with history of intermittent high grade fever, loose stools and non-projectile, non-bilious vomiting. His fever was not associated with chills or rigour. There was no history of irritability or lethargy and the infant was actively sucking breast milk.

The mother contracted malaria when seven months pregnant and had a course of medication in Sudan. Although she was told that the treatment was for malaria, we could not obtain any details about the drugs used except that she received injections and pills. The serological evidence confirmed diagnosis of malaria in the mother.

On examination, the infant was afebrile (although the next day he did start running a fever). He looked generally well and was able to feed without difficulty. He was pale but not icteric. A soft GI/VI systolic heart murmur was audible on chest auscultation. Abdominal examination showed some distension and mild hepatosplenomegaly. The rest of the systemic examination was unremarkable.

A provisional diagnosis of sepsis along with gastroenteritis was made. He was started on a course of antibiotics consisting of ampicillin and gentamycin, pending blood culture results. His diarrhoeal losses were replaced by appropriate intravenous fluids.

Laboratory Data

Haemoglobin level was 10.1 g/dl, and Hct 30.0%, platelets count 85,000/cmm, WBC 9.0 x 10/L with normal differential count. Red cell indices were appropriate for age. Blood chemistry, which included serum

* Consultant Haematologist
Oncologist, BDF Hospital &
Associate Professor
College of Medicine & Medical Sciences
Arabian Gulf University
State of Bahrain

** Senior Resident
BDF Hospital
State of Bahrain

electrolytes, BUN and blood glucose, were within normal limits. Blood and urine cultures were sterile. Peripheral blood smear examination showed trophozoites and schizonts of Plasmodium vivax.

He was given an oral course of chloroquine, following which he became afebrile, his diarrhoea and vomiting subsided and parasitemia disappeared. Primaquine was not started because of the complications that can arise if this drug is given to children below the age of three[3].

Course of Illness

The infant remained afebrile and continued to feed well. However, his haemoglobin level dropped with concomitant significant reticulocytosis but without jaundice. His condition necessitated packed red blood cell transfusion. Prior to transfusion and after obtaining his mother's consent the following investigations were carried out:

1. Total immunoglobulins (Ig) and specific IgM antibody levels against P.Vivax from the infant and mother showed a moderate reaction for IgG against P.vivax and a weak reaction for IgM in the infant. In the mother the reaction for IgG was moderate and IgM against P. Vivax was negative.
2. Complete blood grouping panel including minor blood groups revealed: Duffy (Fy^a): positive; (fyb) negative in the mother and Duffy: (fy^a) and (fyb) positive in the infant.

DISCUSSION

Malaria remains a big killer, causing morbidity in millions of humans in endemic parts of Asia and Africa. Nearly 4% of the Arab population still live in areas where no organised antimalarial measures are being carried out[4]. Malaria is blamed for a significant proportion of deaths during the weaning period with strong susceptibility to the disease has been noted in hyperendemic areas of Sudan, Somalia and southern coastal areas of the Arabian Peninsula[5-7]. Non-immune children of all ages including neonates are at risk of succumbing to this disease in endemic areas. The latter group is likely to be infected if the mothers lack immunity themselves.

Congenital malaria however, is rare, and there have been only 200 reported cases up to 1991[8,9]. Our patient was diagnosed as having malaria at three weeks of age, which may point to the possibility of neonatal rather than intra-uterine infection. Presence of a specific IgM antibody to Plasmodium vivax favours the diagnosis of congenital malaria.

Plasmodium falciparum malaria is known to be associated with diarrhoea in small children[10]. Since the plasmodium parasite recovered in this patient is of the vivax species we presume the diarrhoea with which he presented was not related to his malaria.

Human red cells lacking Duffy blood group or fetal haemoglobin are naturally resistant to P. vivax and P.falciparum respectively[11]. Screening the minor blood groups revealed absence of Duffy in the mother but not the patient. The presence of this blood group in our patient is not consistent with the general trend of susceptibility to P.vivax where it is usually absent.

CONCLUSION

Congenital malaria needs to be considered in the differential diagnosis of febrile neonates coming from endemic regions. An inexpensive, simple peripheral blood smear test, as was done in the case of this patient, will clinch the diagnosis.

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