A RECENT CASE OF CONGENITAL MALARIA IN SINGAPORE

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ABSTRACT
We report a case of congenital malaria in a 2-month-old baby girl, born in Singapore of a Singaporean mother, who presented with fever for one week, gross hepatosplenomegaly and anaemia (haemoglobin 5.6 g/dL) and thrombocytopenia. Peripheral blood films showed Plasmodium vivax. There was no local transmission at that time, but the mother had spent the first 6 months of her pregnancy in Pakistan, where she had been treated for prolonged fever at 4 months amenorrhoea with ibuprofen alone. The mother and 2 siblings were asymptomatic and repeatedly tested negative on blood films for malaria parasites, but the mother tested positive for antibodies to Plasmodium on the Fluorescent Antibody Test. The child was treated with oral chloroquine and made an uneventful recovery. We postulate that this is believed to be the first recent case of congenital malaria in Singapore since it was declared malaria-free in 1982. The diagnosis should be considered in babies of mothers who have travelled to places where malaria is endemic, as maternal infection may be unrecognised, and the child may be asymptomatic at birth.

Keywords: congenital malaria, hepatosplenomegaly, neonatal infection, Singapore

INTRODUCTION
We often think of malaria in the context of a febrile illness when the patient gives a recent history of travel to a region where malaria is endemic, but we should also consider congenital malaria in the differential diagnosis of a pale febrile infant when the mother has been to an endemic region. We report a case of congenital Plasmodium vivax malaria in a 2-month-old baby girl, born in Singapore of a Singaporean mother, who presented with fever, hepatosplenomegaly, anaemia and thrombocytopenia and who had never been out of Singapore. We postulate that this is the first recent case of congenital malaria in Singapore since it was declared malaria-free in 1982.

CASE HISTORY
MMK, born in Singapore to a Singaporean mother of Pakistani ethnic origin, presented at 2 months of age to the Department of Paediatrics, Singapore General Hospital with fever for 6 days and cough. She had been born at term by normal vaginal delivery, birth weight 2775 g, at the Kandang Kerbau Maternity Hospital. She had been discharged well at Day 3 of life, with a normal glucose-6-phosphate dehydrogenase (G6PD) level, and had been well till her presentation to us. She had never travelled out of Singapore. Physical examination revealed a fever of 39.3°C, pallor, an enlarged liver 6 cm below the costal margin and a spleen of 5 cm. There was a soft systolic murmur but the lungs were clear and there were no other signs suggestive of haematological malignancy or intra-uterine infection. Her initial full blood count showed a haemoglobin (Hb) of 7.4 g/dL, haematocrit (Hct) at 21.5%, and mean corpuscular volume 81.4 fl, total white cell count of 11 500 per mm³, and a platelet count of 91 000 per mm³. The haemoglobin dropped to 5.3 g/dL, the haematocrit to 14.6%, the total white cell count to 6 060 per mm³ and platelet count to 69 000/mm³ over the next few hours. Blood films for malaria parasites (BF for MP) showed Plasmodium vivax with a parasite load of 2+. A bone marrow aspirate was done for gross hepatosplenomegaly and showed a regenerating marrow with evidence of peripheral platelet destruction. The child responded well to oral chloroquine 50 mg stat followed by 25 mg at 6, 12, 24 and 48 hours and a blood transfusion, but primaquine was omitted in view of the child’s age. Chloroquine 25 mg weekly was given for a further 6 weeks on an outpatient basis. She was discharged six days after admission, with a haemoglobin of 8.7 g/dL, a reticulocyte count of 10%, a total white cell count of 14 820 per mm³, a platelet count of 239 000 per mm³, and blood films negative for malaria parasites for 3 consecutive days. At that time, there were no local cases of malaria reported in Singapore, the only cases having been imported. The child had never been out of the country, but her mother had spent the first 6 months of her pregnancy in Pakistan, where she had been treated for prolonged 'typhoid fever' in her fourth month of pregnancy with ibuprofen alone.

The mother also gave a history of having had 3-4 episodes of what was thought to be 'typhoid fever' between 1985-1989, and recalled having had jaundice in 1987 in Pakistan. None of these illnesses was identified or treated as malaria. The mother and the patient’s two brothers were asymptomatic and repeatedly tested negative for malaria parasites on peripheral blood films, but were subsequently found to have antibodies to Plasmodium by Indirect Fluorescent Antibody (IFA) testing done at the Parasitology Laboratory of the NUS Department of Microbiology. The mother’s IFA titre was positive at 1:16. The mother and siblings were treated with chloroquine and primaquine. The child’s hepatosplenomegaly regressed completely by 1 year of age and her haemoglobin level rose to 11.0 g/dL and her mean corpuscular volume to 77.3 fl (normal range 76-96 fl) 10 months later. She has continued to thrive, with a weight maintained at the 25-50th centile and height at about the 50-75th centile. There has been no reoccurrence of malaria over 2 years and 6 months of follow-up.

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Our patient did not receive any primaquine, though we gave primaquine to the patient's mother and two brothers. There is no exo-erythrocytic phase in the course of congenital or transfusion-acquired malaria, so only the erythrocytic phase requires treatment. This circumvents the concern of adverse reactions, most notably intravascular haemolysis, that is sometimes seen in patients who are given primaquine. It is also useful to note that the diagnosis of transfusion-acquired malaria should be considered in the appropriate setting. Symptoms of this disease generally occur sooner than those of congenital malaria and include fever, anaemia and hepatosplenomegaly, but intervals of 7-10 weeks between transfusion and onset of symptoms have been reported. The use of stored blood does not prevent the transmission of transfusion-acquired malaria, as plasmodia have been shown to survive in stored blood for up to 2 weeks.

CONCLUSION
This case illustrates the need to keep an open mind for the causes of hepatosplenomegaly and pallor in an infant, if treatment for malaria is not to be delayed. Our working diagnosis was that of a haematological malignancy or intra-uterine infection and the blood film for malaria parasites was done because of the mother's travel history.

The following points may be reiterated:

1. Congenital malaria can occur in babies of asymptomatic mothers, who have had no evidence of malaria in pregnancy.
2. The history of mother's foreign travel or residence in an endemic region may be remote in time, but cannot be ignored in a febrile mother or child.
3. The diagnosis is not difficult once the possibility is considered.

REFERENCES


