Case Report

Congenital Malaria due to Chloroquine-Resistant *Plasmodium Vivax*: A Case Report

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Summary

The clinical manifestation of malaria in neonates and young infants is non-specific and differs from that of adults and older children. So a high index of suspicion is needed to diagnose malaria in early infancy. Chloroquine is the first-line treatment for *Plasmodium vivax* malaria in most parts of the world. This case report details a case of chloroquine-resistant malaria due to *P. vivax* by transplacental transmission from mother with mixed infection of *P. falciparum* and *P. vivax* in a 26-day-old young infant who presented with moderate grade fever and reviews the literature of malaria in infantile and neonatal age groups. And we concluded that high suspicion of malaria is needed to diagnose congenital malaria. Primigravida women with placental malaria pose high risk for congenital infection in baby and emerging chloroquine-resistant *P. vivax* in congenital malaria.

Key words: *Plasmodium vivax*, congenital, chloroquine resistant, malaria.

Introduction

Geographically most widespread and the second prevalent cause of malaria is *Plasmodium vivax* [1]. The prevalence of *P. vivax* cases range from 130 to 435 million worldwide [2]. Malaria in the new born and infantile age groups is rare and the clinical manifestation of neonatal malaria is quite different and merits separate attention. Most cases of malaria reported in this age group are either due to *P. falciparum* or *P. vivax* [3].

We report a case of chloroquine-resistant malaria due to *P. vivax* by transplacental transmission from mother with mixed infection of *P. falciparum* and *P. vivax* in a 26-day-old young infant who presented with moderate grade fever, and review the literature dealing with malaria in infantile and neonatal age groups. The incidence of placental malaria in endemic areas may be as high as 30% while incidence of congenital malaria in infants of immune mothers is estimated as low as 0.3% (up to 10% in non-immune mothers) [4].

Case Report

A 26-day-old male baby was admitted with 10-day history of fever. Baby was full term weighing 4 kg and born via Caesarean section. The antenatal and post-natal period was uneventful. At the time of the onset of child’s illness, mother was having non-specific fatigue and pallor but no obvious clinical illness. But in the last trimester of pregnancy, she had history of fever with chills and rigors which was partially relieved by taking some medications.

On examination, the baby had moderate grade pallor and temperature. Abdominal examination revealed hepatomegaly (6 cm) and splenomegaly (4 cm). Based on these clinical features, possibility of sepsis or congenital infection was considered and the patient was put on intravenous (IV) antibiotics (ceftriaxone and amikacin) along with IV fluids and antipyretics. Investigations revealed moderate grade haemolytic anaemia and thrombocytopenia. General blood picture (GBP) revealed ring stages of *P. vivax*. Chloroquine salt (25 mg kg$^{-1}$) was given orally but even after 2 days of completion of therapy fever persisted without any appreciable regression of hepatosplenomegaly. Peripheral blood smear (PBS) was done and it came out to be positive for high-density *P. vivax* ring stage. Then quinine sulphate was given 20 mg kg$^{-1}$ stat. in 5% dextrose infusion then 10 mg kg$^{-1}$day$^{-1}$ IV 8 hourly for 7 days in 5% dextrose infusion with good clinical response and negative blood smear after 2 days of completion of therapy.

The PBS of mother was positive for mixed infection with *P. vivax* and *P. falciparum* and she was successfully treated with IV artesunate.
**Discussion**

Chloroquine-resistant congenital malaria due to *P. vivax* has been rarely reported in the literature [5–7]. Baird *et al.* [6] concluded that a parasitaemia by *P. vivax* recurring in 28 days after full compliance to standard chloroquine therapy demonstrates resistance which is consistent with antimalarial drug resistance report of WHO [8]. Khichi *et al.* [9] in their study treated cases of congenital malaria with the standard dose of chloroquine sulphate and labelled poor clinical response with persistence of the parasites in the blood or recurrence of disease with parasitaemia as chloroquine resistant. Our case is an established case of chloroquine-resistant congenital malaria due to *P. vivax*. Mother was primigravida and suffering from fever fatigue and pallor since the third trimester of pregnancy. The PBS of mother showed mixed infection with *P. vivax* and *P. falciparum* but Singh [4] has documented that the blood film of the mother is often negative for malaria parasites. At the same time, GBP of the baby showed ring stage of *P. vivax*. Wiwanitkit [10] reported *P. vivax* as the commonest cause for congenital malaria.

In a case of congenital malaria, symptoms and signs most commonly occur between 10 and 30 days of age (range 14 h to several months of age) [11]. Thapa *et al.* [12] in their study showed that anaemia (100%), pyrexia (90%), splenomegaly (84%), hepatomegaly (64%) and jaundice (22%) were the presenting features in neonatal malaria. Our case at the age of 26 days showed fever, hepatosplenomegaly, anaemia and thrombocytopenia.

**Conclusion**

Our case highlights the importance of being highly suspicious and considering malaria in all neonates and infants living in malaria-endemic areas presenting with fever and emerging chloroquine resistance in *P. vivax*. Primigravida women with placental malaria pose high risk for congenital infection in baby.

**References**