Case Report

Chloroquine resistant vivax malaria in an infant: a report from India

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Plasmodium vivax is seen in tropical areas predominantly in southeast Asia and India. Infact, it forms 60–70% of cases of malaria in India¹. Chloroquine resistant *P. falciparum* has been increasingly found, however, it still remains an effective drug in the treatment of *P. vivax*. WHO still recommends chloroquine as the first line treatment for benign vivax malaria². The first chloroquine resistant isolates of *P. vivax* were reported from Indonesia and Papua New Guinea³. Chloroquine resistant *P. vivax* malaria was first reported in India in 1995^{4,5}. We report chloroquine resistant vivax malaria in an infant for the first time from India.

Case report: A 4-month old girl born of non-consanguineous marriage presented with fever for four days. There was no vomiting, cough, diarrhoea, urinary complaints or altered sensorium. Her elder brother also had fever and was diagnosed to be suffering from vivax malaria. Her birth history was uneventful. On examination, she was well-nourished (weight = 6.5kg, 50th centile) and had hepatosplenomegaly. Other examination findings were normal. Her investigations showed haemoglobin of 9.3 g/dL with white cell count of 6,500/mm³ and platelet count of 17,000/ mm^3 . Her peripheral smear showed ring forms of P. vivax. Renal and liver function tests were found normal. She was treated with chloroquine but even after three days, her fever persisted and peripheral smear showed persistence of malarial parasites. At that time,

her haemoglobin dropped to 6.7 g/dL and platelet count was still low (36,000/mm³). She was then treated with oral quinine for seven days. Within 48 h of quinine therapy, her malarial parasites disappeared on peripheral smear, fever defervescenced and platelet count increased to 2,00,000/mm³ with decrease in the liver span and splenic size. She was advised regular follow-up.

Discussion: Chloroquine resistance with P. vivax is rare as compared to P. falciparum. Recurrence with total dose of chloroquine of 25 mg/kg is 1-2%⁵. Because P. vivax does not sequester in the microvasculature, drug studies in P. vivax malaria provide a direct measure of antimalarial activity⁶. Dua et al⁴ have reported chloroquine resistance in spite of blood chloroquine levels more than 8–15 times concentration considered lethal to P. vivax. In child we could not do chloroquine blood levels due to unavailability but the parasites still persisted in the blood with clinical deterioration in spite of 25 mg/kg of chloroquine over three days suggestive of treatment failure. Even though chloroquine remains the drug of choice for treatment of vivax malaria, it is necessary to look for relapse or recrudescence by smear examination to prevent treatment failure and if necessary treatment with artemisinin compounds or quinine or antifolates may be instituted. Also rampant misuse of antimalarials should be avoided to prevent emergence of resistant strains.

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