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Plasmodium vivax myocarditis in a child

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Abstract

We report the first documented case of myocarditis associated with *Plasmodium vivax* malaria in a child who presented with features of cerebral malaria. Clinical features of shock developing in a patient of severe *Plasmodium vivax* malaria, especially with stable haematocrit, should be investigated by electrocardiogram and/ or cardiac enzymes and echocardiogarphy to rule out myocarditis.

Keywords: Myocarditis, *Plasmodium vivax*, cerebral malaria

Abbreviations Used: NVBDCP: National Vector Borne Disease Control Programme, SGOT: Serum glutamate oxaloacetate transaminase, ECG: Electrocardigraphy, *P. Vivax*: *Plasmodium Vivax*, *P. faliciparum*: *Plasmodium faliciparum*

INTRODUCTION

Malaria is one of the major public health problems in India. Around 1.5 million confirmed cases are reported annually by the National Vector Borne Disease Control Programme (NVBDCP), of which 40–50% is due to *Plasmodium falciparum*. [1] Complications in malaria are frequently associated with *P. falciparum* such as cerebral malaria, severe anemia, hypoglycemia, bleeding tendencies and shock. Cardiac complications (cardiac tamponade, myocarditis and conduction abnormalities) with *P. falciparum* are rarely seen. [1-3] Rarely the above mentioned complications have been reported with *P. vivax* malaria as well. [4-5] To the best of our knowledge there has been no reported case of myocarditis in vivax malaria in children. We report an interesting and rare case of cerebral malaria with myocarditis in vivax malaria.

CASE REPORT

A 12 year male child reported to our emergency section in an unconscious state with the history of high grade fever with chills and rigors for 4 days and generalized tonic clonic seizures 2 hours prior to admission. There was no history of seizure or cardiac disorder in the past.

On physical examination he was mildly pale and his blood pressure was 110/70 mm Hg, pulse rate 134/ min, respiration rate 32/min, and body temperature 40°C. Central nervous system examination revealed EMV of 8. There was no focal deficit and meningeal signs were absent. Fundus examination was normal. He had mild splenomegaly with normal liver span. His total leucocyte count was 2,750/mm^3, hemoglobin 10.7 g/dL, and platelet count 84,000/mm^3. The capillary blood sugar level was normal at the time of presentation. Peripheral blood smear revealed schizonts and trophozoites of *P. vivax*. Antigen test (OptiMAL) for *P. vivax* was positive while that for *P. falciparum* was negative. The CSF examination was within normal limits except for mild elevation in the protein level (65 mg/dL). His Liver functions and renal function tests were unremarkable. Serum sodium was 137 meq/L and calcium 8.2 mg/dL. Typhidot IgM was negative for *S. typhi* and blood culture was sterile. Artesunate was started in recommended dosage. The patient gained consciousness after 14 hours and became afebrile on second day. Repeat smear after 48 hours showed clearance of asexual stages of *P. vivax*.

However on day 3 his peripheral pulse became feeble with a drop in pulse rate from the baseline. Blood pressure was 84/66 mm Hg and cardiovascular examination revealed muffling of heart sounds with no gallop or murmur. Electrocardiogram was suggestive of myocarditis (low voltage QRS complexes, prolongation of PR interval and non specific ST-T changes in all unipolar leads). The haematocrit and serum electrolytes were normal but the level of SGOT and troponin I was elevated. Transthoracic 2D Echocardiography showed global hypokinesia with an ejection fraction of 55%. Supportive management was started along with ionotrope (dobutamine) infusion. He showed clinical improvement and normalization of ECG after 48 hours. He was discharged without any complications and repeat ECHO and serum SGOT levels on follow-up were normal.

DISCUSSION

P. vivax malaria in children is generally considered to be benign in nature but there are few case reports suggesting fatal complications like cerebral malaria, pancytopenia, hydrocephalus, renal failure, shock, acute respiratory distress syndrome and pulmonary edema. Cardiac dysfunctions though rare in malaria, published cases have been reported mainly in infection with P. falciparum. A study of cardiac involvement in 22 adult cases of *P.*

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faciparum malaria by Franzen et al showed ECG abnormalities in 23% cases followed by Pericardial effusion and Global hypokinesia in 9% and 4.5% of cases respectively. [3] Recently Kumar PP has reported cardiac involvement (Left ventricular myocardial dysfunction) associated with severe falciparum malaria in two pediatric cases from India. [6] However, cardiac complications due to vivax malaria are extremely rare in the literature. In 1960, a case of fatal ischemic myocarditis associated with vivax malaria in an 8 year old boy was reported by Herrera JM. [7] Recently, a case of myocarditis associated with *P. vivax* has been reported in a 27 year old woman by Soon et al, who had no sequalae. [4] She had spontaneous recovery within 3 days. The present case had cerebral and cardiac involvement with *P. vivax* malaria and recovered without any sequalae. To the best of our knowledge, it is the first pediatric case in the literature to have myocarditis with cerebral malaria owing to *P. vivax*.

The pathophysiology of cardiac involvement in malaria is still unclear. There are few hypotheses regarding the disease. Acidosis and hypoglycemia seen in severe malaria may impair the myocardial integrity and function leading to raised levels of cardiac enzymes. [8] Another possible mechanism may be the mechanical blockage of capillaries by malarial parasites and parasitized red blood cells (PRBC). Post mortem microscopic examination of heart tissue shows congestion of the myocardial capillaries with PRBC, pigment-laden macrophages, lymphocytes, and plasma cells. [9] Clinical data provided by Kochar et al. indicates that *P. vivax* can cause both sequestration- related and non-sequestration related complications of severe malaria, all of which are commonly associated with *P. falciparum* infections. [10]

Thus we conclude that a possibility of myocarditis should be considered in children with vivax malaria who develop hypotension during the course of treatment.

Conflict of interest: Nil

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