Vivax Malaria Complicated by Myocarditis
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Abstract
Plasmodium infection (mainly P. falciparum) is usually complicated by cerebral malaria, haemolysis, acute kidney injury and respiratory distress. Myocardial involvement is a rare complication of plasmodium infection. We have reported a case of plasmodium infection (P. vivax) complicated by myocarditis.

Introduction
Malaria in man is caused by four distinct species of the malaria parasite - P. vivax, P. falciparum, P. malariae and P. ovale. Plasmodium vivax has the widest geographic distribution throughout the world. In India, about 50% of the infections are reported to be due to P. falciparum and 4-8% due to mixed infection and rest due to P. vivax. P. malariae has a restricted distribution (less than 1%) in India, mainly in Tumkur and Hassan districts in Karnataka. P. ovale is a very rare parasite of man, mostly confined to tropical Africa.

Malaria continues to pose a major public health threat in India, particularly due to Plasmodium falciparum which is prone to complications. In India about 27% population lives in malaria high transmission (> 1 case/1000 population) areas and about 58% in low transmission (0-1 case/1000 population) areas. About 88% of malaria cases and 97% of deaths due to malaria is reported from North-eastern States.

Case Presentation
A 17 year old boy was admitted with the history of high grade intermittent fever (104°F - 105°F) with chills and rigor that occurred every 48 hrs for 8 days. He was on oral Ofloxacin and injectable Ceftriaxone at Rural Hospital for the preceding 5 days without relief. He developed dyspnoea that required his transfer to Midnapur Medical College and Hospital. On admission the patient was febrile (oral temperature of 104°F) with mild dehydration. He had no history of cardiac problems. He had tachycardia (PR 120/min) with supine BP of 100/60 mm of Hg. Spleen was enlarged 2.5 cm below the left costal margin, tender and soft in consistency. Examination of the systems was non-contributory. Complete blood count and routine biochemistry were insignificant other than a raised sedimentation rate (50 mm in 1st hour) but peripheral picture showed ring forms, schizonts and gametocytes of plasmodium vivax. Malarial antigen test of plasmodium vivax was positive (Malaria antigen kit test of falciparum was done twice and showed negative) and dengue serology was nonreactive. Widal test was negative. G6PD value was normal. Cardiac enzymes study was normal initially. Blood and urine were sent for culture and he was started on chloroquine. USG abdomen documented splenomegaly. On the 3rd post admission day the patient developed acute onset shortness of breath with retrosternal discomfort. On examination the patient was febrile, tachypnoeic (RR: 34/mn) with a pulse rate of 130/min and BP of 86/64 mm of Hg (supine). Examination of the Cardio-respiratory system was unremarkable. ECG showed tachycardia, 1° AV block (PR: 0.24 sec) (Figure 1). The cardiac enzymes were elevated [CK:1978 U/L (normal 55-170) and CKMB: 95 U/L (normal < 25)]. Echocardiogram documented generalised hypokinesia of myocardium.
Fig. 1: Showing tachycardia, with 1° AV block

with compromised LV systolic function, LVEF-44%). Blood and urine C/S showed no growth. Diagnosis was made as Vivax myocarditis, he was put on high dose dexamethasone (3 mg/kg followed by 1 mg/kg QDS for 8 such). The patient improved symptomatically, became afebrile on 7th day with complete resolution AV conduction disturbance. A repeat peripheral smear of malarial parasites and rapid malarial antigen test were negative. The cardiac enzymes came down within the reference range and a repeat 2D Echo documented improved cardiac contractility and systolic function (LVEF 68%). He was discharged in a stable condition on 7th post admission day.

Discussion

Plasmodium vivax infections are rarely associated with serious complications, including acute respiratory distress syndrome, retinal haemorrhage and splenic infarction.3-6 In our country, Kaur et al7 at Delhi and Kocher et al8 at Bikaner showed vivax malaria associated with acute renal failure, thrombocytopenia, acute respiratory distress syndrome and hepatic dysfunction. However, cardiac complications due to vivax malaria are extremely rare. There were reports by Herrera9 on fatal ischaemic myocarditis and by Kim10 on reversible myocarditis both due to P. vivax in an 8 year old boy and 27 year old woman respectively. Nearly all reported cases regarding malaria with cardiac complications have been limited to P. falciparum infections like pericardial effusion, bundle branch block, cardiomyopathy, and myocarditis. A prospective study of cardiac involvement for 22 malaria patients without a history of cardiac disease also showed electrocardiogram abnormalities (23%), pericardial effusion (9%), and global hypokinesia (5%) during the acute phase.11 However, there was no persistent cardiac damage after malaria in any patient. Though the mechanism of cardiac complication associated malaria is not clear, possible pathogenesis are the mechanical blockage of capillaries by malarial parasites and parasitised red blood cells (PRBC) and toxic effects on myocardium mediated by tumour necrosis factor (TNF).12 Regional wall motion abnormalities would be suggestive of myocarditis or coronary artery disease, whereas restrictive pattern and biventricular hypertrophy would occur in hydroxychloroquine induced cardiac toxicity.13 In our case, it was thought that myocarditis developed due to vivax malaria and echocardiography showed mild global hypokinesia.

In conclusion, we report on a case of P. vivax infection complicated by myocarditis. It is important to consider a possible cardiac complication when patients have shortness of breath and retrosternal discomfort in vivax malaria.

References