Malarial Fever: A Rare Complication of *Plasmodium vivax*. Case Report

Sandeep Kumar*, Srikant Behera and Adil Ahmad Khan
Department of Medicine, Jawahar Lal Nehru Medical college, AMU, India

Abstract

Malaria is one of the commonest parasitic diseases in the tropical countries like India. Complications of *P. falciparum* malaria are well recognised, but the complications of *P. vivax* malaria still continues to give us surprises. Myocardial involvement is a very rare complication of *P. vivax* infection. In this context, we report a case of *P. vivax* induced myocarditis and sinus exit block in our patient with review of the literature related to this rare entity.

Keywords: Complication; Malaria; Myocarditis; *Plasmodium vivax*

Case Report

A 22 year male presented with complains of intermittent fever with rigor since 8 days and jaundice since last 4 days, fever was high grade and associated with shortness of breath and palpitations. There was no history of previous cardiac disease, skin rash, swelling over body, decreased urine output, or bleeding from any site. There was no previous similar complains in past. Family history was unremarkable. Patient was student having no history of addiction of tobacco or alcohol, blood transfusion or sexual contact. On examination, he was conscious and oriented, his vitals were pulse rate of 120/min regular low volume, blood pressure was 100/60 mmHg, respiratory rate of 26/min and temperature of 101°F, his peripheral oxygen saturation was 93% at room air. He was ectic and mild pallor was present with tender hepatomegaly of total liver span of 18 cm and nontender, firm spleen was palpable 4 cm below the left costal margin. chest was clear with normal breath sound. Rest of the examination was unremarkable. His investigations showed haemoglobin -8 gm/dl, TLC-9.2 × 109/L, platelet count 80 × 109/L. Liver function tests documented indirect hyperbilirubinaemia, with normal blood glucose level, PT/INR values and renal function test and cardiac enzymes was normal. Peripheral smear showed ring and schizont stage of *P. vivax*, with no toxic granules. Optimal test was positive for *P. vivax* only. Widal test, Leptospira slide macro agglutination test was negative. Viral serology for Hepatitis A, Hepatitis B, Hepatitis C and Hepatitis E, HIV were negative. ECG showed sinus exit block (Figure 1). Chest X-ray suggested cardiomegaly (Figure 2). Echocardiography was carried out, which was suggestive of dilated left ventricle and left atrium with moderate mitral regurgitation, severe LV systolic dysfunction, ejection fraction of 25% (Figure 3). Cardiac enzymes were negative. A diagnosis of *P. vivax* induced myocarditis was made and the patient was treated with intravenous Artesunate, IV fluids and antipyretics, after which, he was conscious and oriented, his vitals were pulse rate of 120/min regular low volume, blood pressure was 100/60 mmHg, respiratory rate of 26/min and temperature of 101°F, his peripheral oxygen saturation was 93% at room air. He was ectic and mild pallor was present with tender hepatomegaly of total liver span of 18 cm and nontender, firm spleen was palpable 4 cm below the left costal margin. chest was clear with normal breath sound. Rest of the examination was unremarkable. His investigations showed haemoglobin -8 gm/dl, TLC-9.2 × 109/L, platelet count 80 × 109/L. Liver function tests documented indirect hyperbilirubinaemia, with normal blood glucose level, PT/INR values and renal function test and cardiac enzymes was normal. Peripheral smear showed ring and schizont stage of *P. vivax*, with no toxic granules. Optimal test was positive for *P. vivax* only. Widal test, Leptospira slide macro agglutination test was negative. Viral serology for Hepatitis A, Hepatitis B, Hepatitis C and Hepatitis E, HIV were negative. ECG showed sinus exit block (Figure 1). Chest X-ray suggested cardiomegaly (Figure 2). Echocardiography was carried out, which was suggestive of dilated left ventricle and left atrium with moderate mitral regurgitation, severe LV systolic dysfunction, ejection fraction of 25% (Figure 3). Cardiac enzymes were negative. A diagnosis of *P. vivax* induced myocarditis was made and the patient was treated with intravenous Artesunate, IV fluids and antipyretics, after which, the patient improved clinically. A repeat echocardiography carried out after 8 days showed reduction in LV cavity dimension and improvement in cardiac function with Ejection fraction of 50% to 55%, with no MR. Also, the ECG changes reverted to normal after 8 days.

Discussion

Malarial fever is an important public health problem in India. More than two-thirds of the Indian population lives in malaria endemic zone. Though *P. vivax* accounts for nearly 50% of total malaria cases, it is rarely associated with serious complications like impaired consciousness with unarousable coma, jaundice, progressive renal impairment, metabolic acidosis, hyperlactataemia and hypoglycaemia, respiratory distress, pulmonary oedema, severe anaemia, retinal haemorrhage and splenic infarction [1]. Various cardiac complications of malaria include: myocarditis, bundle branch block, pericardial effusion and cardiomyopathy [2]. Nearly all reported cases of cardiac complications of malaria have been limited to *P. falciparum*. Myocarditis as a complication of *P. vivax* is a very rare complication. The exact mechanism of cardiac complication associated malaria is not clear. The *P. vivax* has been demonstrated to cause both sequestration related as well as non-sequestration related complications of severe malaria [3]. The various possible pathogenesis are Plasmodium induced myocarditis includes (1) mechanical blockage of capillaries by malaria parasite and parasitized red blood cells, (2) myocardial damage by pigment laden macrophages, (3) toxic effects of tumour necrosis factor (TNF) on myocardium, (4) fatty change in the myocardium and capillary fibrin thrombii (5) hypoglycaemia and acidosis caused by severe malaria may impair the myocardial integrity and function (6) increase thrombospondin secretion which enhances the sequestration of knob-bearing parasitized red cells [4].

ECG and Echocardiography are usually helpful in diagnosing cardiac complications and myocardial dysfunction. ECG may show conduction block and/or T-ST changes. This suggests that the electrophysiology of cardiac myocytes altered before myocytolysis occurs [4]. A regional wall abnormality would suggest myocarditis or coronary artery disease.

Treatment of myocarditis due to *P. vivax* is rest and to avoid exertional activities and similar as that of myocarditis due to other causes.

In our case patient was presented with complains of fever, jaundice and shortness of breath, on work up there was anaemia, thrombocytopenia, hyperbilirrubinemia, cardiomegaly in chest X-ray and sinus exit block in ECG.

In view of complain of shortness of breath, sinus exit block in ECG and cardiomegaly in chest X-ray echocardiography was done which shows dilated left ventricle with moderate MR and enlarged left atrium. In our case, cardiac enzymes were not elevated. In a study, it was seen that the serum concentration of cardiac troponin T was found to be elevated in only 0.6% of patients [5]. Cardiac complications due to vivax malaria are extremely rare. Gupta et al. reported a case of *P. vivax* induced myocarditis with pericardial effusion 20-years young boy [6]. Soon et al. reported a case of myocarditis associated with *P. vivax* in a 27 years young woman [7] and Mustafa et al. [8] also reported a case of *P. vivax* myocarditis in a child.

*Corresponding author: Sandeep Kumar, Department of Medicine, Jawahar Lal Nehru Medical college, AMU, India, Tel: 05712700920; E-mail: sandeep.jnmc@gmail.com

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Conclusion

Now-a-days, *P. vivax* is manifesting with uncommon complications, which may be lethal. We report on a case of *P. vivax* infection complicated by myocarditis and first degree AV block. It is important to consider a possible cardiac complication when patients have shortness of breath and retrosternal discomfort. Thus, *P. vivax* should not be considered a benign infection anymore.

References