Case Report

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Imported *Plasmodium vivax* malaria complicated by reversible myocarditis

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Abstract:

Myocarditis as a result of malaria infection is uncommon. However, we report a case of a Pakistani emigrant who presented with respiratory distress, fever, chills, and nausea at the emergency room. The patient had traveled to visit his relatives in Pakistan without receiving antimalarial chemoprophylaxis. (Travel clinic advises travelers to Pakistan to take antimalarial drugs as prophylaxis.) Cardiorespiratory examination revealed bilateral crepitation in all lung fields, as well as triple heart sounds. Examination of blood smear showed trophozoites and schizonts of *Plasmodium vivax* with low parasite density. Echocardiography showed diffuse hypokinesia with an ejection fraction of 22% consistent with acute myocarditis. The patient was given chloroquine phosphate, digoxin, perindopril arginine, furosemide, and spironolactone and gradually improved.

Keywords:

Cardiomyopathy, chemoprophylaxis, malaria, *Plasmodium vivax*

Introduction

Myocarditis has several symptoms ranging from reversible mild dyspnea, tachypnea, and chest pain to cardiogenic shock and sudden death. The development of myocarditis has been linked with a wide variety of infections, systemic diseases, drugs, and toxins. The most common cause of infectious myocarditis is a viral infection.[1] Myocarditis secondary to malarial infection is a rare clinical entity; the majority of published cases are attributed to *Plasmodium falciparum* infection. Herein, we describe a rare case of imported *Plasmodium vivax* malaria complicated with reversible myocarditis.

Case Report

A 56-year-old Pakistani expatriate presented to the emergency department with shortness of breath, fever, chills, and nausea. These symptoms began 2 weeks after he had returned from Pakistan. His fever continued even after taking paracetamol. Two days prior to admission, he developed shortness of breath. The patient had no relevant medical history. He had traveled to visit his relatives in Pakistan where he had spent 2 months. He had not received any antimalarial chemoprophylaxis.

On examination, the patient looked ill and orthopneac but was conscious and oriented. His temperature was 38.8°C, blood pressure 95/65 mmHg, pulse rate 120/min, and respiratory rate 31/min. Cardiorespiratory examination revealed bilateral crepitation in all lung fields; there were triple heart sounds but no murmurs. Oxygen saturation (pulse oximetry) was 94% on 6 l oxygen via the nasal cannula. The remainder of the examination was normal.

White blood cell count, hemoglobin level, and platelet count were within the normal limits. Arterial blood gas analysis with the patient on 6-liter oxygen via the nasal cannula was as follows: pH: 7.5, PaO_2: 65 mmHg, $PaCO_2$: 29 mmHg, and bicarbonate: 22 mmol/L. Blood urea

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nitrogen was 5.2 mmol/L, serum creatinine was 275 μmol/L, serum aspartate aminotransferase was 58 U/L, serum alanine transaminase was 87 U/L, and total bilirubin was 103 μmol/L (direct bilirubin: 83 μmol/L). Lactate dehydrogenase was 589 U/L and haptoglobin was 112 mg/dl.

G6PD deficiency was not found and reticulocyte was 0.9%. A chest radiograph showed cardiomegaly with bilateral fluffy airspace shadows denoting the development of pulmonary edema. Throat swab for respiratory viruses was negative. Examination of blood smear showed trophozoites and schizonts of *P. vivax* with low parasite density. Samples of blood, urine, and sputum were sent to the laboratory for culture and susceptibility testing. On electrocardiography trace, there were sinus tachycardia and nonspecific ST and T-wave changes, whereas echocardiography revealed diffuse hypokinesia with an ejection fraction of 22%. These findings were consistent with acute myocarditis.

The patient was admitted to the ward as a case of myocarditis due to malarial infection. He was given chloroquine phosphate, digoxin, perindopril arginine, furosemide, and spironolactone and gradually improved. On the days following, sepsis workups were negative. The fever subsided while the patient remained hemodynamically stable and was discharged home after 12 days of treatment. At home, he was advised to continue taking perindopril arginine and furosemide. Three months later, the patient attended the clinic for follow-up and had no symptoms. Echocardiography showed normal wall motion with an ejection fraction of 55%. The final diagnosis of malaria-related myocarditis was made, based utterly on the clinical laboratory, electrocardiogram, and echocardiographic findings.

Discussion

Malaria is no longer endemic in Qatar; however, it is found to be one of the most commonly imported infectious diseases in this country, with the majority of reported cases being attributed to *P. vivax* infection.[2] Classic symptoms of *P. vivax* in adults include tertian fever, chills, headache, weakness, vomiting, and diarrhea. Severe manifestations, such as acute renal failure, disseminated intravascular coagulation, acute respiratory distress syndrome, hypoglycemia, coma, or seizures, may occur with this infection, as recent studies have shown that the term "benign tertiary malaria" is no longer valid for *P vivax* monoinfection. Although rare, unusual presentations such as nystagmus, psychosis, splenic infarction, splenic rupture, splenic torsion, acalculous cholecystitis, and hepatitis, cardiac diseases and acute pancreatitis may occur, especially with *P. falciparum* infection. The majority of cardiac complications such as pericardial effusion, bundle branch block, cardiomyopathy, and myocarditis have been attributed to *P. falciparum* infections.[3-5] Acute myocarditis secondary to *P vivax* monoinfection is very rare. Very few cases were found after reviewing the literature.[5-9]

High levels of parasitemia and patients' nonimmune status may constitute predisposing factors for severe and complicated malaria. However, severe *P. vivax* infection is usually associated with relatively low parasite density.[5] In our patient, parasite density was low and he was supposed to be immune or at least semi-immune as he came from an endemic country. The pathogenesis of acute myocarditis, although unclear, probably does not differ from that of other organ involvements in malaria and includes mechanical blockage of capillaries resulting from cyto-adherence of infected red blood cells (RBCs) to the vascular endothelium, sequestration of RBCs, and rosetting, in addition to the tumor necrosis factor mediated toxic effects on the myocardium.[4,8]

All reported cases of *P. vivax*-induced myocarditis, including ours, except one, were reversible and showed complete recovery after treatment of malaria infection.

The diagnosis of myocarditis is difficult as there is no established noninvasive gold standard. Transthoracic echocardiography, however, is the most common noninvasive technique used to evaluate patients with suspected myocarditis. It commonly reveals localized wall motion abnormalities including hypokinesia, akinesia, and dyskinesia.[10] In our patient, the diagnosis was based on the echocardiographic findings of global hypokinesia and low ejection that was completely resolved after the treatment.

In conclusion, as this infection is no longer benign, doctors should be familiar with the various complications of vivax malaria including malaria-related myocarditis. Thus, maintenance of a high index of suspicion for this clinical entity is crucial for the prevention of further complications.

Declaration of patient consent

The author certifies that he obtained all appropriate patient consent forms to publish the case report. On the forms, the patient/parent gave his consent for his images and other clinical information to be reported in the journal. The patient understood that his name and initials will not be published and whatever is necessary will be done to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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