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Myocarditis Complicating *Plasmodium vivax* Malaria

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ABSTRACT

Myocarditis complicating *Plasmodium vivax* malaria is an extremely rare complication. We report this development in a young girl who was diagnosed to have *P. vivax* malaria on the basis of peripheral smear. While undergoing antimalarial treatment, she developed respiratory distress requiring invasive mechanical ventilation and inotropic support due to cardiogenic shock secondary to myocarditis. Cardiovascular complications are well recognized with *Plasmodium falciparum* malaria. Nevertheless, a high index of suspicion should be maintained for the same in *Plasmodium vivax* infection especially if symptoms of heart failure develop in a young patient.

Key Words: *Plasmodium vivax malaria. Myocarditis. Acute respiratory distress syndrome.*

INTRODUCTION

Malaria continues to be a major public health problem in the tropical developing world. According to the World Health Organization, in 2010, there were an estimated 216 million cases of malaria in 106 endemic countries and territories and an estimated 655,000 deaths because of the disease worldwide. It is endemic throughout South and South-East Asia, South America and Africa. Furthermore, approximately half the world's population is at risk of developing malaria particularly those that reside in low to middle income countries.¹

Plasmodium (P.) vivax is the most widespread of the four *Plasmodium* species (*P. falciparum*, *P. vivax*, *P. malariae* and *P. ovale*) that infect human with up to 2.5 billion people at risk and an estimated 80 million to 300 million clinical cases every year.² It is manifested by sudden onset of fever with chills, headaches and vomiting but the illness is self-limiting in most cases. Also known as benign tertian malaria, it is not always benign by course.³ Serious life threatening complications have been reported from endemic regions. On the other hand, *Plasmodium falciparum* is well-known to be significantly associated with severe clinical course and complications such as cerebral malaria, multiorgan failure, shock and fatal outcome.⁴

Cardiac complications have rarely been reported in patients affected by this type of infection. We report here a case of *Plasmodium vivax* malaria with an unusual complication of myocarditis and cardiogenic shock.

CASE REPORT

A 20 years old girl with no prior co-morbidities presented to emergency room (ER) with complaints of fever for 8 days, lower abdominal pain and nausea and vomiting for 2 days. The fever was high grade without chills, rigors, or cough associated with it. The abdominal pain was dull, constant and non-radiating in character. On examination, she appeared toxic and dehydrated with collapsed neck veins. Initial vital signs included a heart rate of 107 beats/minute, blood pressure of 82/59 mm Hg and afebrile at that time. Systemic examination revealed generalized tenderness of the upper abdomen with no apparent visceromegaly and positive gut sounds, with coarse crackles and decreased breath sounds at the right base of lung. Rest of the examination was unremarkable.

The results of the initial investigations revealed hemoglobin level of 12.3 g/dl, hematocrit of 36, white cell count of 10,200/ul and platelets of 25,000/cubic millimeter. Her peripheral film showed trophozoites and gametocytes of *Plasmodium vivax* malaria. Her blood urea nitrogen was 29 mg/dl, serum creatinine was 0.9 mg/dl; serum concentration of sodium was 132 meq/l, of potassium was 3.3 meq/l, of chloride was 105 meq/l and of bicarbonate was 23.2 meq/l. Her coagulation profile showed prothrombin time of 11 seconds, activated partial thrombin time of 30.4 seconds. Her blood cultures reported no growth whereas her immunohistochemistry for *Plasmodium vivax* was positive. A chest radiograph showed infiltrates in the right lower zone. Hence, she was diagnosed as a case of *P. vivax* malaria with right lower lobe pneumonia and hypovolemic shock. She was treated with Chloroquine phosphate, Levofloxacin 750 mg intravenous (IV) daily and resuscitated with intravenous (IV) fluids and admitted in the main medical ward.

In the ward, she became tachypneic with a respiratory rate of 40 breaths per minute. Her oxygen saturation dropped to 90% on room air and on examination, she

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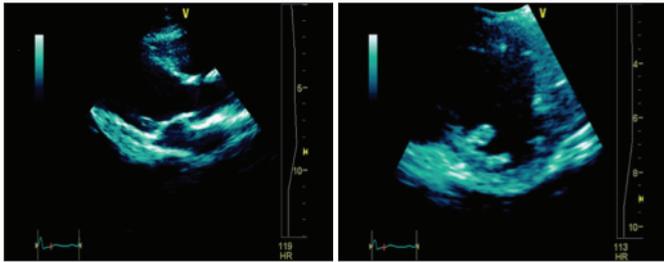


Figure 1: Echocardiogram.

Figure 2: Echocardiogram.

was noted to have jugular venous distention and bilateral fine inspiratory crackles. Her electrocardiogram showed sinus tachycardia with a heart rate of 140 beats per minute. Arterial blood gas showed respiratory alkalosis. At this point, it was thought that she had developed complicated malaria with pneumonia with possible acute respiratory distress syndrome (ARDS). Her medications were modified and she was switched to Piperacillin/Tazobactam 4.5 g IV TID, Quinine dihydrochloride 20 mg/kg IV stat followed by 10 mg/kg IV TID and she was transferred to the intensive care unit.

Subsequently, she developed hypoxemic respiratory failure necessitating intubation and mechanical ventilation. She required high ventilator support and a positive end expiratory pressure (PEEP) of 25 cm H₂O. Vasopressors were started for shock. Her central venous pressure (CVP) at this time was persistently more than 16 mmH₂O, leading to a suspicion of pulmonary edema due to a cardiac cause. A 2-dimensional transthoracic echocardiogram was performed at this point which revealed an ejection fraction of 30% with severe global hypokinesia. Cardiac chambers, however, were normal in size and mild to moderate mitral regurgitation as shown in Figures 1 and 2. The high CVP and low ejection fraction ruled out the development of ARDS. Hence, this patient had developed myocarditis secondary to *P. vivax* malaria.

Her IV fluids were stopped and she was started on inotropes, dopamine, dobutamine and diuresed with furosemide. Her repeat CXR confirmed parenchymal fluid in lungs and bilateral pleural effusions. Blood picture also revealed a platelet count of 55 and positive malarial parasite on peripheral film with scanty gametocytes of *Plasmodium vivax* of 1-10/100 power field.

Over the next 48 hours, the patient's ventilatory requirements gradually improved. Repeat complete blood count (CBC) showed negative malarial parasite on peripheral film. On 8th day a repeat echocardiogram revealed normal left ventricular systolic function and ejection fraction of 60%. The patient was extubated on the 10th day of admission. Her CBC significantly had improved with platelets of 558,000, hemoglobin of 10.9 g/dl and a negative peripheral smear for malarial parasite. Subsequently, the patient was shifted out of ICU to medical ward again and sent home.

DISCUSSION

We report a case of *P. vivax* malaria complicated by secondary myocarditis. *Plasmodium vivax* infection can rarely cause various complications which include ARDS/ALI, acute kidney injury, severe anemia and thrombocytopenia, hemophagocytic syndrome, as well as cerebral malaria.^{3,4} Although there are many cases reporting cardiac complications associated with *Plasmodium falciparum* infection, they have rarely been reported to be caused by *P. vivax* infection.

There are four case reports in literature about *Plasmodium vivax* infection complicated by myocarditis. Two of these have been described in children with only one case reported in an adult patient. The first case was reported by Herrera *et al.* about an 8 years old boy who died due to fatal myocarditis following *Plasmodium vivax* malaria.⁵ The second case was reported by Kim *et al.* which links *P. vivax* infection with myocarditis.⁶ The patient presented with fever and chills after travel to an endemic region in South Korea and was diagnosed with *P. vivax* malaria. Subsequently, she developed chest pain and non-specific ST-T wave changes in lead II, III and AVF on ECG and was found to have elevated troponin levels. Her ejection fraction was 61% with suspicious hypokinetic motion on inferior cardiac wall and normal global left ventricular systolic function. She was conservatively managed and her chest pain resolved followed by cardiac markers which returned to baseline over a few days. ECG in this patient showed sinus tachycardia and ST-T flattening in anterior leads and she developed cardiogenic shock due to severe myocarditis. A 2-dimensional echocardiogram showed severely reduced left ventricular systolic function, an ejection fraction of 30% and global hypokinesia. She subsequently required assisted ventilation and invasive hemodynamic monitoring. Hence, this is the first case to report development of cardiogenic shock secondary to myocarditis in a patient with *P. vivax* malaria. In a third reported case, *Plasmodium vivax* malaria presented with an unexpected combination of cerebral malaria complicated by myocarditis in a 12 years old child.⁷ A fourth case report from Northern India is about an adolescent girl who presented with fever, jaundice and right upper quadrant abdominal pain. Her clinical course, similar to our patient was complicated with the development of respiratory distress secondary to pulmonary edema, culminating in mechanical ventilation. An echocardiogram subsequently revealed generalized hypokinesia and an ejection fraction of 40%.⁸

Myocarditis complicating *P. vivax* malaria is extremely rare. The exact incidence and pathophysiology of this entity remains obscure. In *Plasmodium falciparum* associated myocarditis, the mechanism of myocardial injury is a result of a number of parasitic toxins released in the course of infection that activate the immune

response.⁹ Inflammatory cytokines like TNF α are known to exert toxic effects on the heart causing myocardial injury. Another mechanism of cardiac injury in malaria is occlusion of capillaries by parasites and parasitized RBCs causing ischemic cardiomyopathy.¹⁰ Also severe metabolic acidosis and hypoglycemia can compromise myocardial integrity and function with high concentrations of circulating cardiac biomarkers.

Acute myocarditis has a variable clinical course, ranging from asymptomatic disease to congestive heart failure and sudden death. Early recognition of this complication is important as it may lead to cardiogenic shock which carries a high mortality. These patients require intensive care with invasive hemodynamic monitoring and inotropic support. Despite global efforts aimed at eradication and control of malaria, this disease remains one of the most important public health challenges of these time.

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