Case Report

Plasmodium Vivax Malaria Complicated by Pericardial Effusion

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Abstract

We describe herein an unusual and sole cardiac complication of pericardial effusion in a case of plasmodium vivax malaria.

Keywords

Malaria, Plasmodium vivax, pericardial effusion

Introduction

The incidence of malaria has been increasing in recent years despite of worldwide attempts at control. Common manifestations include fever, anemia, thrombocytopenia and splenomegaly. Cardiac involvement is very rare and seems to be limited to acute infection with Plasmodium falciparum. However, we recently encountered cardiac complication of pericardial effusion in a case of Plasmodium vivax malaria. Therefore treating physicians should consider cardiac involvement in patients residing in malaria endemic areas and presenting with hypotension and fever.

Case Report:

A 27-year-old woman presented in emergency department with a 2 weeks history of high grade fever associated with chills and rigor relapsing every 48 hours. Upon admission, he complained additionally of nausea, vomiting, breathlessness and chest pain. There was no past history of ischaemic/valvular heart disease. On physical examination her blood pressure was 80/64 mmHg, pulse rate 112/min (low voluminous, regular), respiration rate 28/min, and body temperature 101.2°F. Jugular venous pressure was raised 5cm above sternal angle but there was no pulsus paradoxus. Splenomegaly was observed 3 cm below subcostal margin. On auscultation bilateral lung fields were clear with no adventitious sounds. Precordium was silent with muffled S_1/S_2 . On laboratory investigations her white blood cell count was 14,330/µL (reference value, 4,000–10,000/µL) with polymorphonuclear cytosis. Peripheral blood smear revealed P vivax ring forms, schizonts, and gametocytes . Parasitemia density was 6,990/µL. Blood biochemistry including liver and renal function test and complete urine examination were normal. Chest skiagram showed cardiomegaly (**Fig. 1**). Unique alteration observed in the electrocardiogram was a low amplitude in QRS complexes with sinus tachycardia. On transthoracic echocardiography, we found mild to moderate pericardial effusion with thick pericardium and right atrial collapse (**Figs. 2, 3**). There was no regional wall motion abnormality with preserved left ventricular functions . So the patient was treated with 10 mg/kg of quinidine gluconate 8 hourly, plus ceftriaxone 2 g daily intravenously and oral doxycycline 100 mg BD. Given the severity of clinical setting, intramuscular artemisin (300 mg loading dose, followed by 100 mg/daily) was initiated at 40 hours after admission. Since the pericardial effusion was localized predominantly behind right atrium it was not safe for

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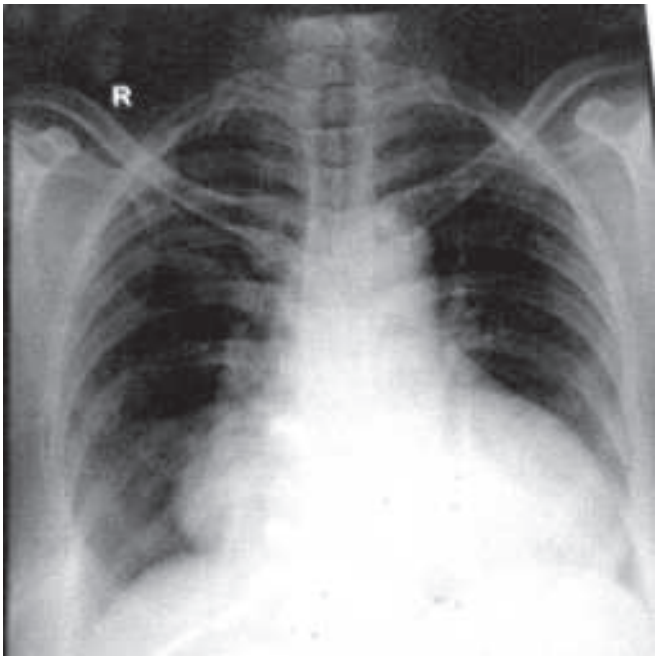

Fig. 1
Chest skiagram showing cardiomegaly

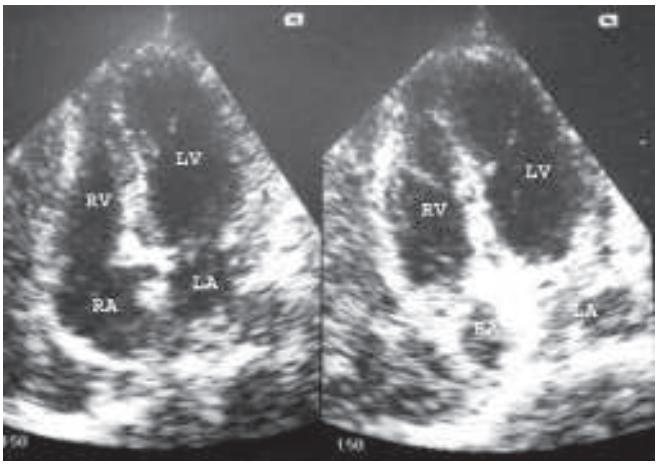

Fig. 2
Echocardiogram showing pericardial effusion with thick pericardium and right atrial collapse

pericardiocentesis, so the patient was managed medically with diurtics. After 7 days of conservative management she was afebrile with marked relief in her symptoms and was discharged. Repeat echocardiogram during follow up after 1 month showed complete resolution of pericardial effusion (**Fig. 4**).

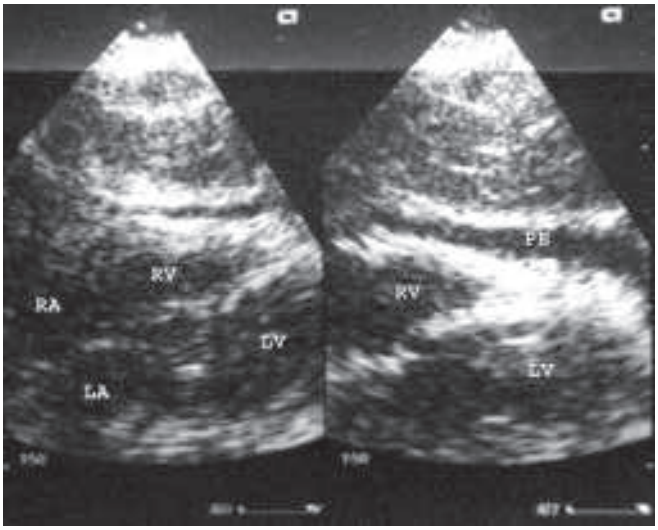

Fig. 3
Echocardiogram showing pericardial effusion with thick pericardium and right atrial collapse

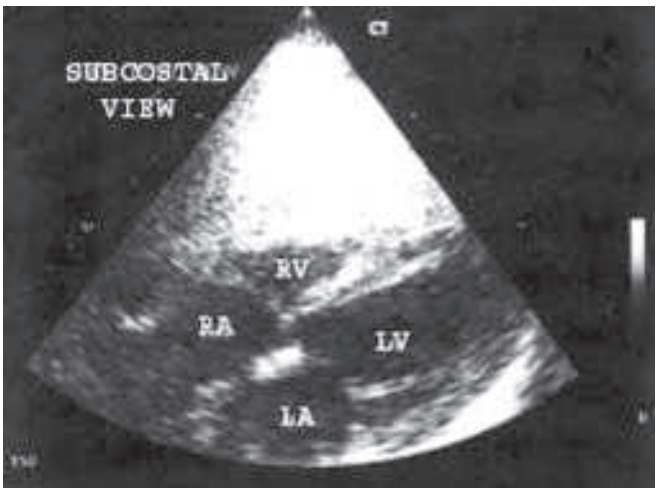

Fig. 4
Repeat echocardiogram showing complete resolution of pericardial effusion

Discussion

Upon exploring literature Plasmodium vivax infections have been found to be usually associated with complications such as adult respiratory distress syndrome, retinal hemorrhage, spontaneous rupture of spleen, and splenic infarction[1-4]. Cardiac complications due to vivax malaria are extremely rare. There was only a single report by Herrera[5] on fatal ischemic myocarditis due to P vivax in an 8-year-old boy. Nearly all published cases regarding malaria with

cardiac complications have been limited to P falciparum infections[6]. A prospective study of cardiac involvement for 22 malaria patients without a history of cardiac disease showed electrocardiogram abnormalities (23%), pericardial effusion (9%), and global hypokinesia (5%) during the acute phase[7]. However, there was no persistent cardiac damage after malaria in any patient.

The pathogenesis of cardiac complication associated malaria is not clear. One possible mechanism is the mechanical blockage of capillaries by malarial parasites and parasitized red blood cells (PRBC). On autopsy of such patients there are striking microscopic findings with congestion of the myocardial capillaries and of those of pericardial fat with PRBC, pigment-laden macrophages, lymphocytes, and plasma cells[8]. In a few cases, fatty changes in the myocardium and capillary fibrin thrombi with well-demarcated myocardial necrosis have been seen. More fulminant clinical picture in P falciparum malaria than malaria caused by other Plasmodium species is related to the high burden of parasitemia, more PRBC, and increased ability to sequester in the deep microvasculature.

There is also evidence suggesting that toxic effects on myocardium mediated by monokines such as tumor necrosis factor (TNF) may play a significant role[9]. Hydroxychloroquine-induced cardiac toxicity such as conduction disturbance and congestive heart failure has also been reported[10].

We reported herein a unique and unusual case of P vivax malaria complicated soley by pericardial effusion . It is important to consider this rare possibility when patients of vivax malaria have chest pain associated with cardiomegaly and hypotension.

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