

Congestive heart failure triggered by *Plasmodium falciparum* malaria in a patient with rheumatic heart disease: A case report

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Abstract

Cardiovascular complications of *Plasmodium falciparum* malaria are uncommon and may be difficult to distinguish from pre-existing cardiac disease or treatment-related adverse effects. We report a case of acute cardiac decompensation in a patient with rheumatic heart disease (RHD) during *falciparum* malaria. A 46-year-old woman with a history of RHD and previous balloon mitral valvotomy presented with a 5-day history of fever, chills, rigors, body ache, and headache. Malaria rapid diagnostic testing and peripheral blood smear confirmed *P. falciparum* infection. Approximately 16 hours after admission, she developed acute orthopnoea, cough with pink frothy sputum, chest pain, palpitations, syncope, hypoxaemia, tachycardia, and hypotension. Echocardiography demonstrated severe mitral stenosis, severe tricuspid regurgitation, severe pulmonary arterial hypertension, biatrial enlargement, and preserved left ventricular ejection fraction. Cardiac enzymes and N-terminal pro-B-type natriuretic peptide were within normal limits. She was treated with antimalarial therapy, inotropic and supportive measures, diuretics, pulmonary vasodilator therapy, and anticoagulation. Her clinical condition improved within 12 hours, and vital parameters normalized within 36 hours. She was discharged after 7 days and referred to a higher centre for definitive valvular intervention. This case highlights the potential for *P. falciparum* malaria to precipitate acute cardiac decompensation in patients with underlying severe valvular heart disease and emphasizes the importance of early cardiovascular assessment in patients with severe malaria.

Keywords: Malaria; *Plasmodium falciparum*; Heart failure; Rheumatic heart disease; Mitral stenosis; Tricuspid regurgitation; Pulmonary hypertension; Echocardiography.

Introduction

Malaria has a broad clinical spectrum, ranging from asymptomatic parasitaemia to severe, life-threatening disease. *Plasmodium falciparum* is particularly associated with severe multisystem complications, including cerebral malaria, acute kidney injury, severe anaemia, acute respiratory distress syndrome, metabolic disturbances, hepatic dysfunction, and circulatory shock [1-3].

Cardiovascular involvement in malaria is less frequently recognized than other complications. Reported manifestations include myocarditis, pericarditis, arrhythmias, conduction abnormalities, myocardial ischaemia, and heart failure [4,5]. Cardiovascular manifestations may result from systemic inflammation, endothelial dysfunction, sequestration of parasitized

erythrocytes, tissue hypoxia, and metabolic disturbances. In addition, some antimalarial agents may produce cardiovascular adverse effects, further complicating clinical assessment [5].

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Patients with pre-existing structural heart disease may be particularly vulnerable to haemodynamic deterioration during acute systemic infection. We report a case of acute cardiac decompensation in a patient with severe rheumatic mitral stenosis and tricuspid regurgitation during *P. falciparum* malaria in a resource-limited field hospital in South Sudan. The case highlights the diagnostic and therapeutic challenges associated with acute cardiovascular deterioration in a patient with significant underlying valvular heart disease and concomitant malaria.

Case presentation

A 46-year-old woman presented to our field hospital with a 5-day history of fever associated with chills, rigors, body ache, and headache. She had a history of rheumatic heart disease diagnosed in 2011 and had previously undergone balloon mitral valvotomy. On admission, she was haemodynamically stable, and her general examination did not reveal any significant abnormality. Written informed consent obtained from the patient for publication of this case report and the accompanying images.

A malaria rapid diagnostic test was positive for *P. falciparum* (Figure 1a). The diagnosis was confirmed by thin peripheral blood smear, which demonstrated *P. falciparum* schizonts within infected erythrocytes (Figure 1b).

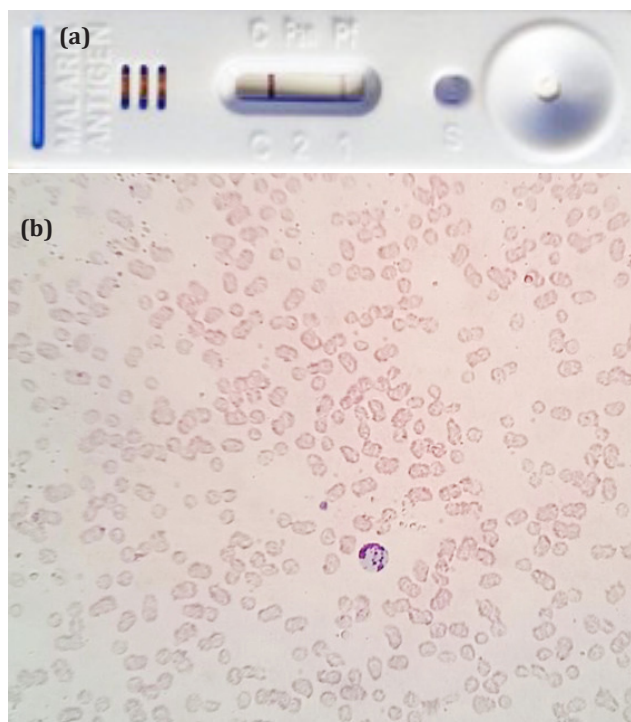


Figure 1: Diagnosis of *Plasmodium falciparum* malaria. (A) Malaria rapid diagnostic test positive for *P. falciparum*. (B) Thin peripheral blood smear demonstrating *P. falciparum* schizonts within infected erythrocytes.

Clinical deterioration

The patient was initiated on artemisinin-based antimalarial therapy and antipyretics. Approximately 16 hours after admission, she developed acute-onset orthopnoea, corresponding to New York Heart Association functional class IV symptoms, together with cough producing pink frothy sputum. She also reported chest pain, palpitations, and syncope. Her heart rate was 110 beats/min, blood pressure was 90/40 mmHg, oxygen saturation was 80% on room air and improved to 90% with oxygen at 6 L/min through a face mask. Her respiratory rate was 30 breaths/min.

Cardiovascular examination revealed a grade III mid-diastolic rumbling murmur with presystolic accentuation, best heard at the cardiac apex in the mitral area. Bilateral coarse crackles were present throughout both lung fields.

Investigations

Laboratory investigations showed leucocytosis ($11,800/\text{mm}^3$), markedly elevated C-reactive protein (CRP), prolonged prothrombin time (16.7 seconds; INR, 1.26), elevated D-dimer ($3740 \mu\text{g/L}$), and hyponatraemia (serum sodium, 124 mEq/L). Platelet count and serum potassium were within normal limits. Cardiac enzyme levels were normal, and NT-proBNP was 71.6 pg/mL .

Posteroanterior chest radiography demonstrated cardiomegaly with a cardiothoracic ratio of approximately 0.51 and radiographic features of left atrial enlargement, including an increased carinal angle of 106° and right retrocardiac double density (Figure 2). No definite radiographic evidence of pulmonary oedema was identified.

Electrocardiography demonstrated sinus tachycardia with P pulmonale (Figure 3). Urgent two-dimensional echocardiography demonstrated severe rheumatic mitral stenosis, severe tricuspid regurgitation, and severe pulmonary arterial hypertension, with a peak systolic tricuspid regurgitation pressure gradient of 71 mmHg. The left atrium was moderately enlarged, measuring 5 cm, while the right atrium and right ventricle were dilated. Left ventricular systolic function was preserved, with an ejection fraction of 70%.

Treatment and outcome

The patient was managed with antimalarial therapy, intramuscular benzathine penicillin (1.2 million units monthly), intravenous dobutamine infusion (10 mg/h, titrated according to haemodynamic response), oral digoxin, furosemide, sildenafil, and rivaroxaban. She was maintained in a propped-up position and received supplemental oxygen.

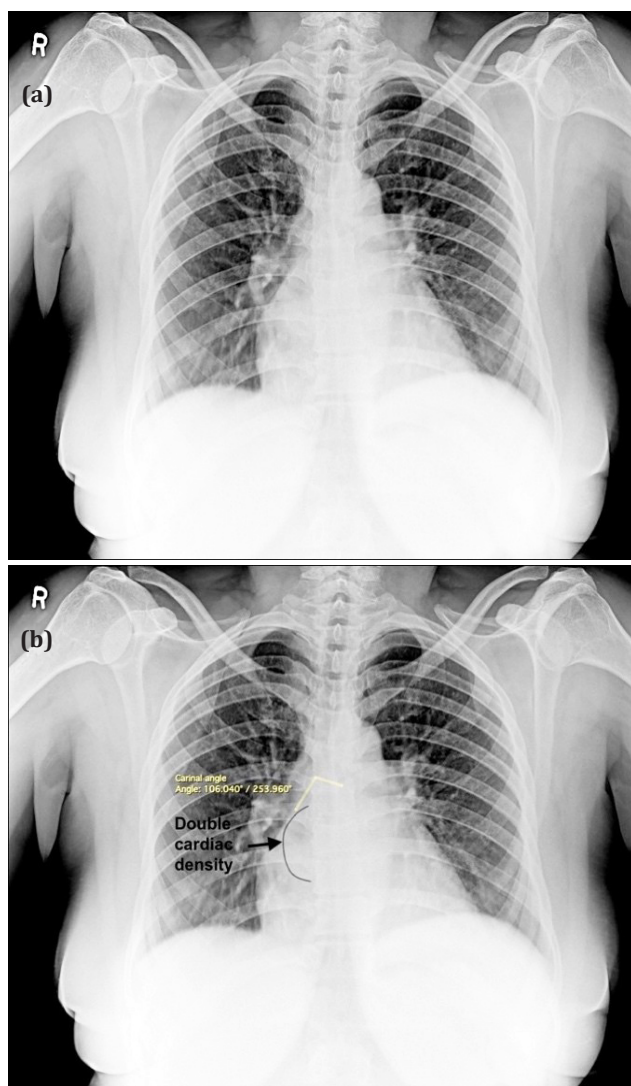


Figure 2: Posteroanterior chest radiograph (a) Unannotated image, (b) Annotated image demonstrating cardiomegaly and features of left atrial enlargement. No definite pulmonary oedema is seen.

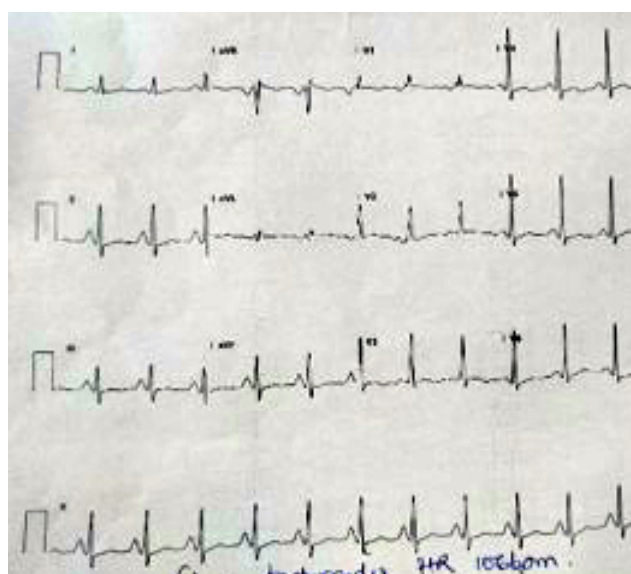


Figure 3: Electrocardiogram demonstrating sinus tachycardia and P pulmonale.

Clinical improvement was observed within 12 hours of initiating treatment, and her vital parameters normalized within 36 hours. After 7 days of hospitalization, she was discharged in a clinically stable condition and referred to a higher centre for definitive management of her mitral and tricuspid valve disease.

Discussion

Malaria is a multisystem infectious disease with manifestations ranging from uncomplicated parasitaemia to severe, life-threatening illness. *P. falciparum* is particularly associated with severe disease because of its capacity for erythrocyte sequestration, endothelial interaction, and systemic inflammatory activation [1,2]. Cardiovascular involvement is less frequently recognized but encompasses a broad spectrum of abnormalities, including myocarditis, pericarditis, arrhythmias, conduction disturbances, myocardial ischaemia, and heart failure [4,5].

The cardiovascular manifestations of malaria are likely multifactorial. Proposed mechanisms include systemic inflammatory responses, endothelial dysfunction, sequestration of parasitized erythrocytes in the microcirculation, impaired tissue oxygenation, metabolic disturbances, and direct or indirect myocardial injury [5-9]. Cytokine-mediated myocardial dysfunction has also been proposed, with severe malaria associated with increased circulating concentrations of pro- and anti-inflammatory cytokines [8]. Anaemia, renal dysfunction, altered intravascular volume, and other systemic complications may further increase cardiovascular stress [10-12].

Our patient had significant pre-existing structural heart disease, including severe rheumatic mitral stenosis, severe tricuspid regurgitation, and severe pulmonary arterial hypertension. These abnormalities provide a plausible substrate for cardiac decompensation during an acute systemic infection. The temporal relationship between *P. falciparum* infection and the sudden development of orthopnoea, hypoxaemia, hypotension, and pulmonary congestion-like symptoms suggests that the acute infection may have acted as a precipitating stressor. However, the available findings do not establish direct myocardial injury due to malaria. Therefore, the presentation is more appropriately interpreted as acute cardiac decompensation occurring in the setting of falciparum malaria and severe underlying valvular heart disease.

The absence of elevated cardiac enzymes and the preserved left ventricular ejection fraction argue against an overt myocardial infarction or severe global left ventricular systolic dysfunction. The normal NT-

proBNP concentration also makes interpretation of the acute heart failure syndrome less straightforward. Nevertheless, natriuretic peptide concentrations should be interpreted in the context of the clinical presentation and underlying valvular pathology rather than in isolation. The ECG showed sinus tachycardia and P pulmonale, findings compatible with the patient's haemodynamic burden and pulmonary hypertension but not specific for malaria-related myocardial involvement.

Cardiovascular abnormalities in malaria may be under-recognized because clinical manifestations can overlap with severe systemic infection and pre-existing cardiovascular disease. Gupta et al. emphasized the range of cardiovascular manifestations associated with malaria and the potential role of systematic cardiovascular assessment [7]. A nationwide Danish cohort study also reported an association between previous imported malaria, particularly *P. falciparum* infection, and subsequent risk of heart failure, although the study addressed long-term cardiovascular outcomes rather than acute cardiac decompensation [4].

Echocardiography was particularly valuable in this case because it demonstrated the severity of the underlying valvular disease and pulmonary hypertension. In a patient with malaria who develops unexplained dyspnoea, hypotension, hypoxaemia, chest discomfort, palpitations, or other cardiovascular symptoms, careful clinical assessment supplemented by ECG, cardiac biomarkers, and echocardiography may help identify clinically important cardiac involvement and distinguish it from other complications of severe malaria.

The management of this patient was challenging because definitive valvular intervention was not immediately available in the resource-limited field-hospital setting. Initial treatment therefore focused on antimalarial therapy, haemodynamic support, diuresis, oxygen supplementation, and management of the underlying cardiovascular condition. The patient's rapid clinical improvement following treatment supports the importance of early recognition and stabilization while arranging definitive cardiac evaluation.

This case has important limitations. It is a single-patient observation, and the temporal association between malaria and cardiac decompensation does not establish causality. The patient had severe pre-existing rheumatic valvular disease, which itself provides a strong explanation for susceptibility to acute haemodynamic deterioration. In addition, advanced cardiac investigations such as cardiac magnetic resonance imaging and invasive haemodynamic assessment were

not available in the field setting. These limitations should be acknowledged when interpreting the case.

Overall, this case illustrates that *P. falciparum* malaria may precipitate clinically significant cardiovascular deterioration in patients with severe underlying structural heart disease. A high index of suspicion and early cardiovascular evaluation are warranted when patients with malaria develop acute cardiorespiratory symptoms.

Conclusion

P. falciparum malaria may precipitate acute cardiac decompensation in patients with severe underlying rheumatic valvular heart disease. In this case, severe mitral stenosis, tricuspid regurgitation, and pulmonary hypertension provided a substantial substrate for haemodynamic deterioration, with malaria acting as a potential precipitating stressor. Early recognition of cardiovascular deterioration, prompt echocardiographic assessment, appropriate supportive treatment, and referral for definitive valvular management are important, particularly in resource-limited settings.

Conflict of Interest

The authors declare no conflict of interest.

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