

Abstract

Sickle Cell Cardiopathy and Rheumatic Valvular Disease: A Double Burden

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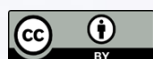
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Introduction: Sickle cell disease (SCD) and rheumatic heart disease (RHD) are important causes of cardiovascular morbidity and mortality, particularly in low- and middle-income countries. SCD is an autosomal recessive hemoglobinopathy that causes chronic vaso-occlusion, inflammation, and multiorgan dysfunction, including cardiac involvement. Rheumatic heart disease, in turn, remains an important public health problem that predisposes patients to cardiac disease. Although their underlying mechanisms differ, the coexistence of these conditions may impose a double cardiovascular burden. **Case Report:** We present the case of a 20-year-old female patient with sickle cell disease who presented to the emergency department with progressive fatigue, dyspnea on moderate and minimal exertion, weight loss, and lower-limb edema. One week before admission, she had experienced feverishness, chest pain, and osteoarticular pain. On admission, she was in poor general condition, tachypneic, and showing signs of respiratory distress. Blood pressure was 116/76 mmHg, heart rate 71 bpm, respiratory rate 23 breaths/min, and oxygen saturation 94%. On cardiopulmonary examination, there were decreased breath sounds at the right lung base, prominent heart sounds with an S3 gallop, and a grade III holosystolic murmur with an opening snap over the mitral area. Laboratory tests revealed leukocytosis (21,005 cells/mm³), severe anemia (hemoglobin, 5.4 g/dL; hematocrit, 15.1%; mean corpuscular volume, 84.8 fL; mean corpuscular hemoglobin, 30 pg; mean corpuscular hemoglobin concentration, 35.4 g/dL), platelet count of 404,000/mm³, positive malaria testing with 1,000 parasites/mm³, creatinine of 1.2 mg/dL, and total bilirubin of 4.1 mg/dL. Abdominal ultrasonography revealed hepatomegaly with regular contours. Chest radiography showed cardiomegaly and an inflammatory infiltrate at the right lung base. Electrocardiography revealed signs of left ventricular overload. Echocardiography demonstrated a thickened mitral valve with rheumatic morphology, estimated pulmonary artery systolic pressure of 51 mmHg, a dilated left ventricle with a left ventricular ejection fraction of 36%, and a mild circumferential pericardial effusion measuring approximately 9 mm. Decompensated heart failure with reduced ejection fraction was diagnosed, precipitated by malaria and pneumonia. During hospitalization, the patient received ceftriaxone 1 g every 12 hours, clarithromycin 500 mg every 12 hours, antimalarial therapy, oral bisoprolol 5 mg daily, intravenous furosemide 40 mg daily, oral spironolactone 25 mg daily, and oral enalapril 10 mg daily. After 10 days of hospitalization, the patient was discharged with clinical improvement, outpatient follow-up, heart failure therapy, and intramuscular benzathine penicillin

1,200,000 IU every 21 days. **Conclusion:** Although traditionally considered distinct conditions, the coexistence of sickle cell disease and rheumatic valvular disease imposes a double cardiovascular burden. This case report highlights this uncommon coexistence and discusses its clinical implications in an African setting, where both diseases continue to represent important public health challenges.



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