INTRODUCTION

A 12-year-old boy was referred for evaluation of cardiac mass. He had progressive exertional fatigue for 1 year and palpitations for the past 6 months. There was no history of prolonged fever, joint pains, weight loss, or chest trauma. There was no known heart disease in the past. On examination, heart rate of 100/min, blood pressure of 110/70 mmHg, and raised jugular venous pressure of 7 cm above the sternal angle were found. The cardiac examination was apparently normal. Other systems examination was also normal, except a 5 cm hepatomegaly. The chest X-ray showed mild cardiomegaly and clear lung fields [Figure 1]. Routine hematological and biochemical investigations were unrevealing. The electrocardiogram (ECG) showed incomplete right bundle branch block and prominent, diffuse ST-T changes, including saddle-shaped elevation in V1 [Figure 2]. An echocardiogram showed an infiltrating mass lesion involving both the right and the left ventricles. The mass appeared more prominent around the right ventricular outflow tract. The contractility was mildly reduced in both the ventricles. The diagnosis was not clear.

A cardiac magnetic resonance (CMR) imaging (1.5 T, Magnetom Avanto, Siemens, Germany) was performed. Spin-echo T1-weighted axial images [Figure 3a and b] showed isointense, multiple, discrete or contiguous, nodular infiltrative masses along the anterior wall of the right atrium (which was dilated), right ventricular outflow tract, and along both the right and the left ventricles. The lesions appeared to predominantly involve the outer myocardium, with the pericardium being indistinct in several places. There was no mediastinal lymphadenopathy and no pleural or pericardial effusions. On the spin-echo T2-weighted images [Figure 3c], the lesions were isointense to mildly hyperintense. The steady state free precession (SSFP) sequence [Figure 3d] showed the lesions to be homogenously hyperintense with reduced systolic ventricular function. On the delayed enhanced sequence [Figure 3e], the lesions enhanced with small areas of heterogeneity. There was no thrombus. A non-contrast computed tomography of the chest was also performed and was normal. Differential diagnosis included metastasis, sarcoma, and lymphoma. In view of the clinical history, absence of imaging characteristics typical of these lesions, and our own experience with imaging of cardiac tuberculosis, the latter was strongly considered and antitubercular therapy was initiated. Tissue diagnosis was not performed.

Six months after the antitubercular treatment, the patient had clinical signs of mild constrictive pericarditis. A repeat CMR showed significant reduction in size of all the lesions, with only mild residual thickening at the previous sites of involvement [Figure 4].

DISCUSSION

Cardiac tuberculosis usually involves the pericardium. Myocardial involvement is rare, and is described in up to 0.3% of the patients dying from tuberculosis. Myocardial tuberculosis can present with rhythm disturbances, congestive heart failure, ventricular aneurysms, right ventricular outflow obstruction,
coronary involvement, caval obstruction, and aortic insufficiency.\(^1\) The modes of spread to the myocardium include through lymphatics from mediastinal nodes, directly from the pericardium, or via a hematogenous route. While mass-like lesions are reported,\(^1\) a diffuse infiltrative lesion is extremely rare.

CMR has certain advantages over echocardiography for the detection, characterization, and evaluation of the complete extent of cardiac masses. These include a high contrast resolution, unrestricted field of view, and multiplanar imaging capability.\(^5\) Our case showed iso- to mildly hyperintense lesions on T1- and T2-weighted spin echo images, indistinct pericardium, and contrast enhancement with mild heterogeneity. These imaging features help to differentiate the lesion from metastasis (usually quite hyperintense on T2-weighted images with pericardial effusion and prominent enhancement), sarcoma (similar to metastasis with prominent heterogeneity due to hemorrhage and necrosis, and intramyocardial involvement), and lymphoma (which usually enhance homogenously).

The treatment of myocardial tuberculosis primarily involves antitubercular therapy, and complete clinical and imaging resolution has been reported.\(^4,6\)

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