Left Ventricular Tubercular Myocarditis with Unique Imaging Features on Cardiac MRI: A Case Report

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Received 22 February 2024; Accepted 7 November 2024

Abstract

Myocarditis is a rare manifestation of tuberculosis, often associated with high morbidity and mortality. Cardiac magnetic resonance imaging (MRI) is a critical imaging tool for assessing infiltrative myocardial conditions. We describe the case of an adult patient from a tuberculosis-endemic region who presented with sudden-onset symptomatic arrhythmias. Cardiac MRI findings included heterogeneous signal intensity in the left ventricular myocardium, altered myocardial nulling time, and a patchy "zebroid-like" pattern of late gadolinium enhancement. Additionally, necrotic supraclavicular and retroperitoneal lymphadenopathy were observed. Fine-needle aspiration cytology of the affected lymph node revealed epithelioid inflammatory granulomas. The patient was diagnosed with disseminated tuberculosis and tubercular myocarditis. Following the initiation of a standard anti-tubercular regimen, significant clinical improvement was noted at the 2-month follow-up.

J Teh Univ Heart Ctr 2024;19(S1):60-62

This paper should be cited as: Rana P, Sheth M, Dikshit A, Deshpande S, Garachh M, Patel S, et al. Left Ventricular Tubercular Myocarditis with Unique Imaging Features on Cardiac MRI: A Case Report. J Teh Univ Heart Ctr 2024;19(S1):60-62.
DOI: 10.18502/jthc.v19is1.18479

Keywords: Myocarditis; Cardiac MRI; Case report

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DOI: 10.18502/jthc.v19is1.18479

Case Presentation

A 43-year-old man with no prior known comorbidities presented with sudden-onset palpitations accompanied by nausea and sweating. He reported significant weight loss over the preceding 2 to 3 months but denied any history of chest pain, fever, or similar symptoms. Notably, his wife had been diagnosed with pulmonary tuberculosis 10 years earlier, for which she had completed a full course of anti-tubercular treatment and remained asymptomatic at the time of his presentation.

On examination, the patient's pulse rate was 100 bpm. Blood pressure and oxygen saturation (SpO_2) were within normal limits, measuring 125/85 mm Hg and 100%, respectively. An ECG revealed changes consistent with pleomorphic ventricular tachycardia. Laboratory results showed a hemoglobin level of 10.4 g/dL, a total leukocyte count of 15,420/µL, and a platelet count of 357,000/µL. Elevated inflammatory markers were noted, with a C-reactive protein level of 12.5 mg/L and an erythrocyte sedimentation rate of 32 mm/h.

Transthoracic echocardiography revealed mild systolic dysfunction, with normal left ventricular dimensions and no evidence of pericardial effusion. A Holter monitor recorded episodes of ventricular tachycardia, with heart rates ranging between 76 and 200 bpm, including several instances of non-sustained ventricular tachycardia.

A cardiac magnetic resonance imaging (MRI) scan was performed to investigate potential infiltrative myocardial conditions. The results revealed patchy areas of increased signal intensity in the myocardium of the septum, anterior wall, and lateral wall of the left ventricle on cine-true fast imaging with steady-state free precession (TRUFI) and short tau inversion recovery (STIR) sequences. Mildly depressed systolic function was observed, with an ejection fraction of 46% (Figure 1). TI scout images (7 minutes post-gadolinium injection) showed an altered myocardial nulling pattern, with inhomogeneous myocardial nulling occurring before that of the blood pool in certain myocardial regions (Figure 2). Patchy, linear, subendocardial to mid-myocardial late gadolinium enhancement (LGE) was noted in the anterior, septal, and lateral walls of the left ventricle at the mid-cavity level, while transmural LGE was observed in the anterior wall at the apical level (Figure 1). The non-ischemic distribution of LGE exhibited a "zebroid-like" pattern in the 4-chamber view (Figure 1, E). Moreover, elevated T1, T2, and extracellular volume values (66.8%) were detected in the affected areas (Figure 3). Extracardiac findings included right supraclavicular lymphadenopathy (Figure 4, A).

Given the findings, an inflammatory etiology, particularly tuberculosis, was considered a likely diagnosis due to the highly suggestive clinical background and the endemic prevalence of tuberculosis in our region.

A contrast-enhanced computed tomography scan of the chest and abdomen was performed to identify additional foci of pulmonary or extrapulmonary disease. The results revealed necrotic right supraclavicular lymphadenopathy and an enlarged retroperitoneal lymph node (Figure 4, B & C). No enlarged or calcified mediastinal or hilar lymph nodes were observed. An ultrasound-guided fine-needle aspiration biopsy of the right supraclavicular lymph node was conducted, which demonstrated epithelioid cells and lymphocytes forming granulomas (Figure 4, E & F). Nonetheless, a Ziehl-Neelsen stain for mycobacterium yielded negative results.

Considering the clinical presentation, imaging findings, the endemic prevalence of tuberculosis in our region, and the supportive cytological evidence from fine-needle aspiration, the patient was initiated on a standard anti-tubercular treatment regimen. At the 2-month follow-up, the patient showed significant clinical improvement and reported no ongoing symptoms. The positive response to anti-tubercular therapy further corroborates our diagnosis of tuberculosis, including tubercular myocarditis, in this case.

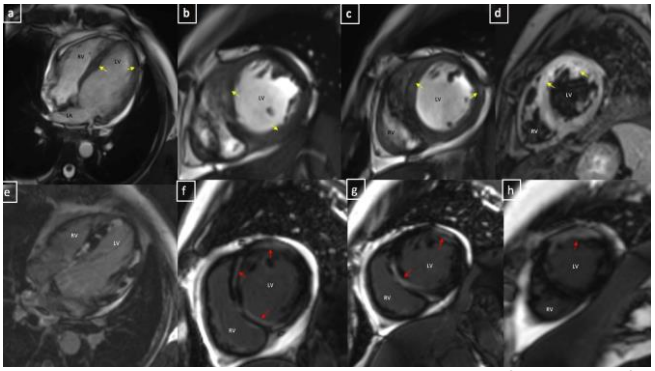

Figure 1. Cardiac magnetic resonance imaging static images of the cine-true fast imaging with steady-state free precession (TRUFI) sequence show the 4-chamber view (A), short-axis views (B & C), and short tau inversion recovery (STIR) sequence in the short-axis view (D). These images reveal patchy areas of increased signal intensity within the myocardium, specifically in the septum, anterior wall, and lateral wall of the left ventricle (arrows). The late gadolinium enhancement in the 4-chamber view (e) demonstrates patchy enhancement of the left ventricular myocardium in a non-ischemic distribution, resembling a "zebroid-like" pattern. The late gadolinium enhancement short-axis views of the mid-cavity (F & G) illustrate subendocardial to mid-myocardial enhancement (arrow), while the short-axis view of the apex (H) shows transmural myocardial enhancement in the anterior wall of the left ventricle (arrow).

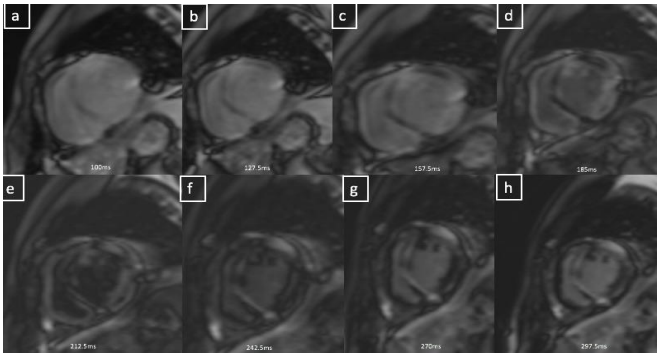

Figure 2. The image presents the cardiac magnetic resonance imaging short-axis view of the inversion-time scout assessment (TI scout), taken 7 minutes after the administration of gadolinium-based contrast material at increasing inversion times (A–F). The images reveal inhomogeneous nulling of the left ventricular myocardium, with certain areas of the myocardium demonstrating nulling (C) occurring before that of the blood pool (E).

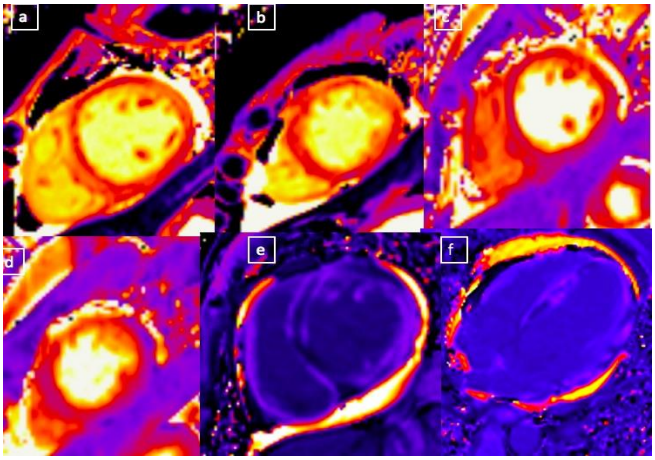

Figure 3. Native T1 (A & B) and native T2 (C & D) maps illustrate the elevated native T1 values (1430; reference age- and sex-matched normal for our system [3T Siemens] - 1079) and T2 values (58; reference age- and sex-matched normal for our system [3T Siemens] - 32) in regions of the myocardium with altered signal enhancement. Post-contrast T1 (E & F) maps reveal low T1 values in areas exhibiting gadolinium enhancement.

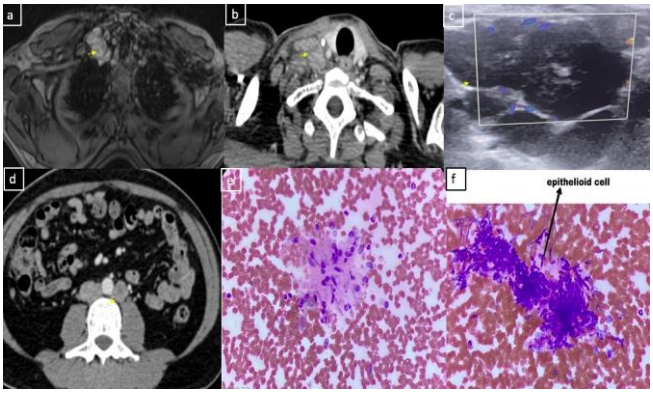

Figure 4. Post-contrast T1 DIXON axial magnetic resonance image (A), contrast-enhanced computed tomography axial images (B), and ultrasound axial images (C) of the lower neck reveal enlarged necrotic right supraclavicular lymph nodes (arrow). The contrast-enhanced computed tomography image of the abdomen (D) displays an enlarged retroperitoneal lymph node (arrow). Histological images (E & F) at low magnification show the presence of a few epithelioid cells and lymphocytes forming granulomas (H&E × 30 µm).

Discussion

Tubercular myocarditis is a rare yet severe manifestation of extrapulmonary tuberculosis associated with considerable morbidity and mortality.[1] The condition can arise through hematogenous spread, lymphatic dissemination, or direct extension from adjacent pericardial involvement. Clinical presentation is often nonspecific, overlapping with other cardiac disorders, and can range from asymptomatic cases to sudden cardiac death. Rhythm disturbances, such as heart block, refractory ventricular arrhythmias, and long QT syndrome, have been reported in patients with tubercular myocarditis. Diagnosis can be challenging, necessitating a high index of clinical suspicion and supportive imaging findings. Timely identification is critical to prevent life-threatening complications.

Pathologically, myocardial tuberculosis can be classified into 3 types based on the pattern of involvement: miliary, nodular (tuberculomas), and diffuse infiltrating types. The miliary type is the most common, typically resulting from hematogenous dissemination of tuberculosis.[2] Lesions may affect the atria, ventricles, or interventricular septum. There is a predilection for right-sided cardiac involvement, likely due to the anatomical connection with right-sided mediastinal lymph nodes.[3]

Transthoracic echocardiography is often the initial diagnostic tool, though its findings are typically nonspecific. Cardiac MRI has emerged as a critical imaging modality for assessing infiltrative myocardial conditions. In cases of tubercular myocarditis, cardiac MRI findings may include thickened myocardium with or without edema, T2 hypointense nodular foci within the myocardium representing tubercular granulomas, or a heterogeneous pattern of LGE.[3, 4]

To our knowledge, altered myocardial nulling and the distinctive patchy “zebroid-like” pattern of LGE on cardiac MRI have not been previously reported in cases of myocardial tuberculosis. Furthermore, our case demonstrated the rare involvement of the left ventricle. The altered nulling may result from the expansion of the extracellular space due to abnormal protein deposition, allowing increased gadolinium uptake, while the patchy “zebroid-like” pattern suggests a diffuse infiltrative process. Extracardiac findings of tuberculosis provide critical diagnostic clues in such cases.

The definitive diagnosis of tubercular myocarditis relies on identifying characteristic histological features, such as granulomas with caseating necrosis and the presence of myocardial bacilli. While endomyocardial biopsy remains the gold standard for diagnosis, it is an invasive procedure with associated periprocedural risks. Therefore, histological sampling from an easily accessible extracardiac site, such as supraclavicular lymph nodes—as demonstrated in our case—combined with a supportive clinical presentation, can be sufficient to confirm the diagnosis. This approach is further validated by an excellent response to anti-tubercular treatment.

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