Case Report

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Internal Medicine Section

A Case of Tuberculous Myocarditis Presenting as Monomorphic Ventricular Tachycardia: Multimodality Imaging-guided Diagnosis and Management

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ABSTRACT

Myocardial involvement in tuberculosis is extremely uncommon and is often diagnosed only at autopsy. Ventricular arrhythmias as the presenting manifestation of tuberculous myocarditis are rare and pose important diagnostic and therapeutic challenges. A 36-year-old female presented with palpitations and sustained monomorphic ventricular tachycardia requiring electrical cardioversion. Baseline electrocardiography showed sinus rhythm with right bundle branch block, and transthoracic echocardiography demonstrated a structurally normal heart. Electrophysiological study revealed three distinct ventricular tachycardia morphologies, suggesting a diffuse arrhythmogenic substrate. Cardiac magnetic resonance imaging demonstrated myocardial oedema and late gadolinium enhancement involving the interventricular septum and lateral wall. Fluorine-18 Fluorodeoxyglucose Positron Emission Tomography-Computed Tomography (FDG PET-CT) showed patchy myocardial uptake and multiple hypermetabolic cervical and mediastinal lymph nodes. Computed Tomography (CT)-guided lymph node biopsy revealed necrotising granulomatous lymphadenitis consistent with tuberculosis, and the tuberculin skin test was strongly positive. Given the high risk of recurrent malignant arrhythmias during the inflammatory phase, an implantable cardioverter-defibrillator was implanted prior to initiation of antitubercular therapy. The patient received antitubercular therapy with adjunctive corticosteroids and completed nine months of treatment. She remains asymptomatic on follow-up with no recurrence of ventricular tachycardia. The present case highlights the importance of considering tuberculosis in patients presenting with inflammatory ventricular arrhythmias in endemic regions and demonstrates the complementary role of cardiac magnetic resonance imaging and fluorine-18 FDG PET-CT in establishing the diagnosis and guiding management.

Keywords: Arrhythmias, Cardioversion, Diagnostic imaging, Electrocardiogram, Granuloma, Lymphadenitis

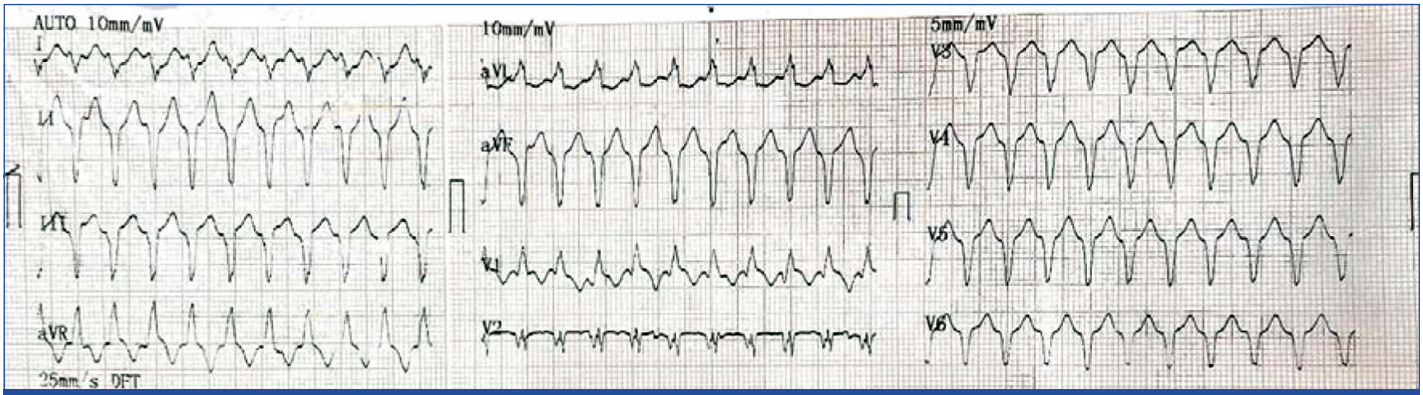
CASE REPORT

A 36-year-old woman with no prior history of cardiovascular disease, diabetes mellitus, hypertension, autoimmune disease, or known tuberculosis presented to an outside hospital with a one-day history of acute-onset palpitations associated with presyncope. She denied fever, cough, weight loss, night sweats, recent travel, or known close contact with individuals diagnosed with tuberculosis. There was no family history of sudden cardiac death or inherited cardiomyopathy. The presenting electrocardiogram demonstrated sustained monomorphic ventricular tachycardia, and she underwent successful direct-current cardioversion.

The patient was subsequently referred to the study centre for further evaluation. On admission, she was haemodynamically stable. Physical examination was unremarkable, with no clinical features suggestive of heart failure, systemic infection, or extracardiac tuberculosis.

Baseline electrocardiography demonstrated sinus rhythm with right bundle branch block [Table/Fig-1a]. The presence of right bundle branch block raised the possibility of conduction system involvement and suggested an underlying myocardial disease process rather than idiopathic ventricular tachycardia. Transthoracic echocardiography revealed normal left and right ventricular size and systolic function, with no regional wall motion abnormalities or significant valvular pathology.

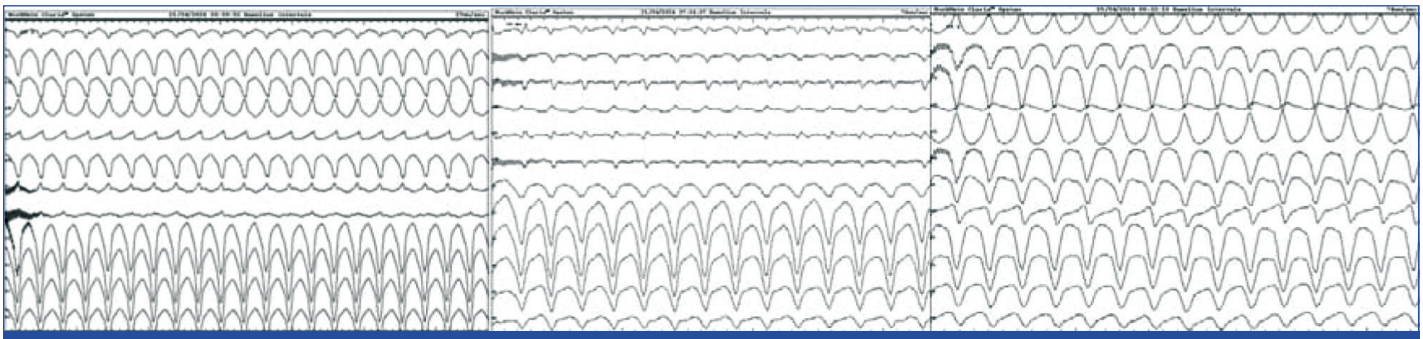
Given the occurrence of sustained ventricular tachycardia in an apparently structurally normal heart, an electrophysiological study was performed. Programmed ventricular stimulation induced three distinct ventricular tachycardia morphologies. The presence of multiple inducible ventricular tachycardia morphologies suggested a diffuse and heterogeneous arrhythmogenic substrate involving different myocardial regions, favouring an inflammatory or infiltrative



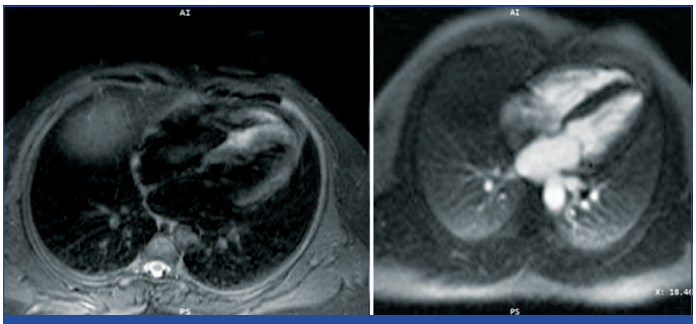
[Table/Fig-1a]: Baseline electrocardiogram demonstrating sinus rhythm with the right bundle branch block.

myocardial process rather than a focal idiopathic ventricular tachycardia. These findings prompted further evaluation for occult inflammatory cardiomyopathy [Table/Fig-1b].

Cardiac magnetic resonance imaging demonstrated increased myocardial T2 values consistent with myocardial oedema, and patchy mid-myocardial and subepicardial late gadolinium enhancement involving the interventricular septum and lateral wall, in keeping with active myocardial inflammation [Table/Fig-1c].



[Table/Fig-1b]: Electrophysiology study demonstrating multiple ventricular tachycardia morphologies.



[Table/Fig-1c]: Cardiac magnetic resonance imaging demonstrating myocardial inflammation.

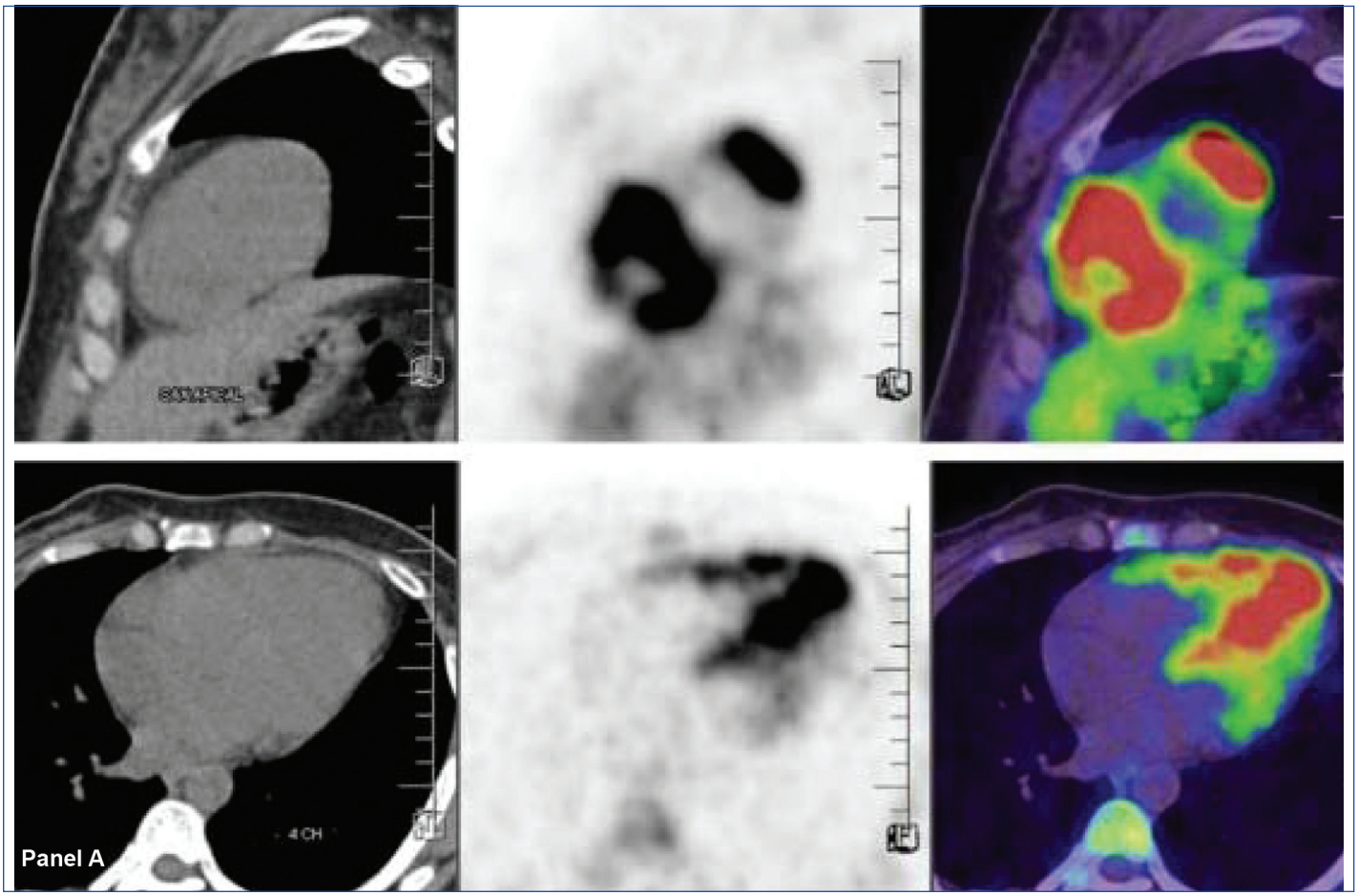
To further evaluate for an underlying inflammatory or infiltrative process, 18F-FDG PET-CT was performed. The study showed heterogeneous patchy myocardial FDG uptake and multiple FDG-avid supraclavicular, cervical, and paratracheal lymph nodes [Table/Fig-2].

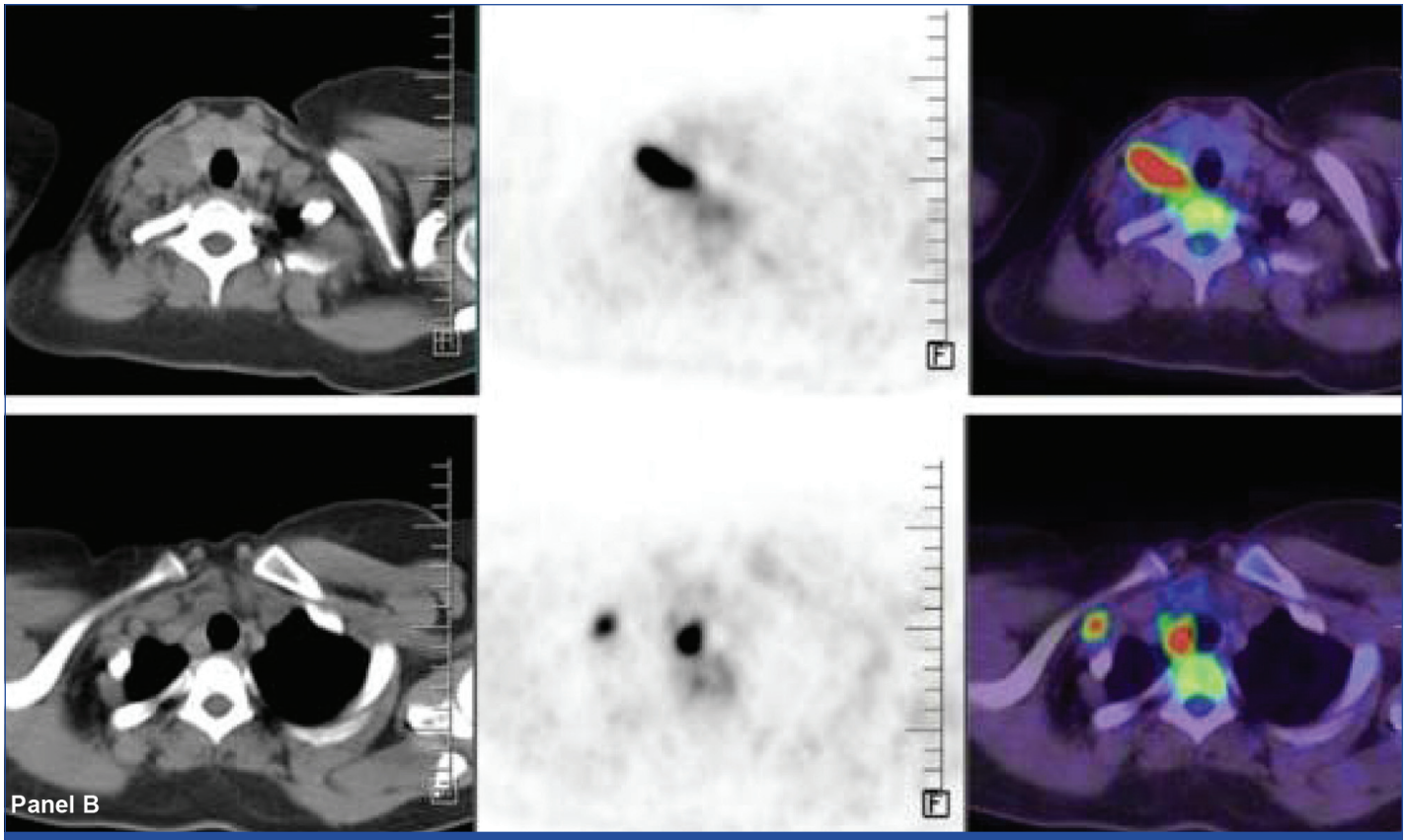
The CT-guided biopsy of a supraclavicular lymph node demonstrated necrotising granulomatous lymphadenitis. No alternative infectious or malignant aetiology was identified, and the findings were consistent with tuberculosis [Table/Fig-3]. The tuberculin skin test was strongly positive.

Based on the presence of active myocardial inflammation, sustained ventricular tachycardia and histological confirmation of tuberculosis from extracardiac tissue, a diagnosis of tuberculous myocarditis presenting with malignant ventricular arrhythmias was established.

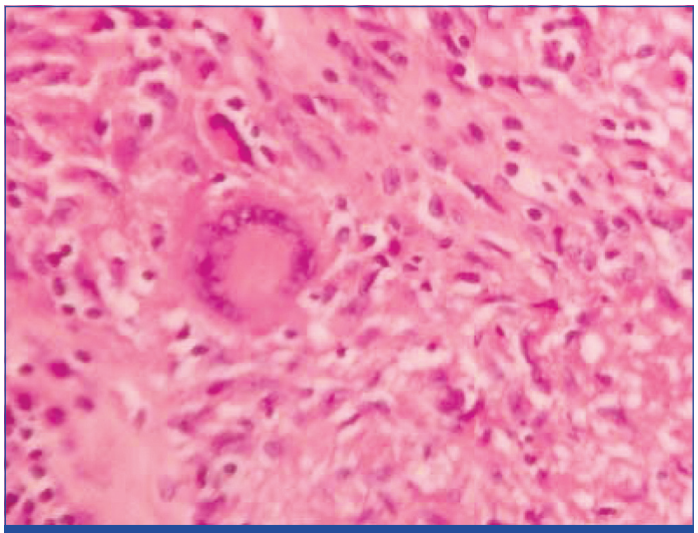
Given the recognised risk of recurrent ventricular arrhythmias during the inflammatory phase and early initiation of antitubercular therapy, a transvenous implantable cardioverter-defibrillator was implanted prior to commencing treatment [Table/Fig-4].

Standard antitubercular therapy consisting of isoniazid, rifampicin, pyrazinamide, and ethambutol was initiated. Adjunctive prednisolone (1 mg/kg/day) was administered during the initial inflammatory phase and gradually tapered over 6-8 weeks. The patient completed nine months of antitubercular therapy without complications. At

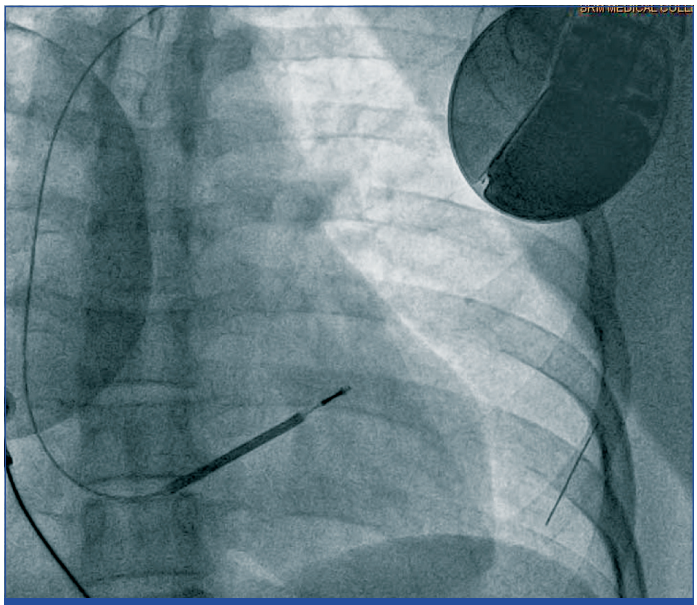




[Table/Fig-2]: Fluorine-18 Fluorodeoxyglucose Positron Emission Tomography-Computed Tomography (FDG PET-CT); Panel A: Axial computed tomography, PET, and fused PET-CT images demonstrating heterogeneous patchy myocardial FDG uptake involving the interventricular septum and lateral wall, suggestive of active myocardial inflammation; Panel B: Axial computed tomography, PET, and fused PET-CT images demonstrating intensely FDG-avid supraclavicular and mediastinal lymph nodes, facilitating extracardiac tissue diagnosis of tuberculosis.



[Table/Fig-3]: Histopathological examination of a CT-guided biopsy specimen obtained from a supraclavicular lymph node. (Haematoxylin and eosin staining ×200 magnification).



[Table/Fig-4]: Fluoroscopic image following implantable cardioverter-defibrillator implantation.

12-month follow-up, she remained asymptomatic, and device interrogation revealed no ventricular arrhythmia episodes or implantable cardioverter-defibrillator therapies.

DISCUSSION

Myocardial tuberculosis is an exceptionally rare manifestation of extrapulmonary tuberculosis. Historical autopsy studies have reported myocardial involvement in a very small proportion of patients with tuberculosis, and the condition is often diagnosed only post-mortem [1,2]. The myocardium may be involved through haematogenous dissemination, lymphatic spread from infected lymph nodes, or direct extension from adjacent mediastinal structures [3,4].

The pathological patterns of myocardial tuberculosis include diffuse infiltrative disease, nodular tuberculoma, and miliary involvement [2,4]. The diffuse infiltrative form is most often associated with conduction abnormalities and ventricular arrhythmias. Granulomatous inflammation, myocardial oedema, myocyte injury, and subsequent fibrosis may create areas of slow conduction and electrical heterogeneity, thereby promoting re-entry and ventricular tachycardia [5,6]. If unrecognised, tuberculous myocarditis may progress to ventricular dysfunction, refractory ventricular arrhythmias, complete heart block, cardiogenic shock, or sudden cardiac death [7,8].

The diagnosis is challenging because clinical manifestations are non-specific and may mimic cardiac sarcoidosis, viral myocarditis, or other inflammatory cardiomyopathies. In the present case, cardiac Magnetic Resonance Imaging (MRI) demonstrated myocardial

oedema and non ischaemic late gadolinium enhancement, fulfilling imaging criteria for active myocarditis [9]. The septal involvement also provided a possible explanation for the baseline right bundle branch block, suggesting inflammatory involvement of the conduction system.

Fluorine-18 FDG PET-CT provided complementary information by demonstrating active myocardial inflammation and identifying metabolically active cervical and mediastinal lymph nodes. This was clinically important because it enabled tissue diagnosis from an accessible extracardiac site, avoiding the limited diagnostic yield and procedural risks associated with endomyocardial biopsy [3,10].

Several similar cases have been reported in the literature. Khurana R et al., reported tubercular myocarditis presenting with ventricular tachycardia and highlighted the role of cardiac magnetic resonance imaging, lymph node biopsy, antitubercular therapy, corticosteroids, and cardioverter-defibrillator implantation [11]. Gautam MP et al., described tuberculous myocarditis presenting as refractory ventricular tachycardia of biventricular origin, supporting the concept of a diffuse arrhythmogenic substrate [12]. Clivillé DB et al., recently reported myocarditis with concomitant tuberculosis infection presenting with ventricular tachycardia, in which cardiac magnetic resonance imaging and fluorine-18 FDG PET-CT were central to diagnosis [13]. Vennamaneni V et al., also emphasised the diagnostic difficulty and variable presentation of tuberculous myocarditis in a recent case report and review [14]. Zhang L et al., reported sudden unexpected death due to tuberculous myocarditis involving the sinus node at autopsy, demonstrating the potentially fatal nature of unrecognised myocardial involvement [8].

The electrophysiological finding of three inducible ventricular tachycardia morphologies in the present patient strongly suggested a diffuse arrhythmogenic substrate rather than a focal idiopathic ventricular tachycardia. This finding was concordant with the imaging evidence of multifocal myocardial inflammation and supported the diagnosis of inflammatory cardiomyopathy.

There are no randomised data guiding management of ventricular arrhythmias in tuberculous myocarditis. Antiarrhythmic drugs may provide temporary suppression of arrhythmias, but they do not treat the underlying inflammatory and infective substrate. Catheter ablation is generally less attractive during active inflammation because the arrhythmogenic substrate may be evolving and recurrence risk may be high; it is usually reserved for recurrent drug-refractory ventricular tachycardia after disease stabilisation [6,15].

In the present patient, implantable cardioverter-defibrillator implantation was undertaken before initiation of antitubercular therapy because of sustained monomorphic ventricular tachycardia, multiple inducible ventricular tachycardia morphologies, and imaging evidence of active myocardial inflammation. In addition, paradoxical clinical worsening after antitubercular therapy has been described in tuberculous myocarditis, supporting close monitoring and arrhythmic protection during the early treatment phase [16].

Adjunctive corticosteroids were administered along with standard antitubercular therapy to suppress myocardial inflammation. Although robust evidence for corticosteroid therapy in myocardial tuberculosis is limited, recent case-series data and extrapolation from tuberculous pericardial disease support its use in selected patients with severe inflammatory cardiac involvement [3,17].

The patient remained asymptomatic during follow-up, and serial implantable cardioverter-defibrillator interrogation revealed no recurrent ventricular arrhythmias or device therapies. Long-term management should include clinical assessment, device interrogation, monitoring for drug-related adverse effects, and repeat imaging when clinically indicated to assess resolution of inflammation and detect residual scar. The absence of ventricular tachycardia recurrence after completion of therapy supports the concept that active myocardial inflammation played a central role in arrhythmogenesis.

CONCLUSION(S)

Tuberculous myocarditis is a rare but potentially life-threatening cause of ventricular tachycardia and should be considered in patients presenting with inflammatory ventricular arrhythmias, particularly in tuberculosis-endemic regions. Multimodality imaging with cardiac magnetic resonance imaging and FDG PET-CT can facilitate diagnosis by identifying myocardial inflammation and guiding tissue biopsy from extracardiac sites. Early initiation of antitubercular therapy together with appropriate arrhythmic protection, including implantable cardioverter-defibrillator implantation in selected high risk patients, may result in favourable clinical outcomes.

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