AMERICAN JOURNAL OF DIAGNOSTIC IMAGING, 2022
VOL 8, NO. 1, PAGE 13–20
10.5455/ajdi.20210107023143

ORIGINAL ARTICLE

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Cardiac MRI in the evaluation of pericardial and myocardial involvement in tuberculosis with 2 case reports

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ABSTRACT

Tuberculosis (TB) caused by Mycobacterium TB is a major health care problem in India. While pulmonary manifestations are well characterized, cardiac TB is rare. It is estimated that 1% of all cases of TB have cardiac involvement. We present a case series of two cases of Pericardial TB presenting as pericardial thickening with nodular masses mimicking Lymphoma and another case of TB presenting as myocarditis with concomitant lymphadenopathies. Cardiac magnetic resonance imaging is a problem-solving tool in differentiating the unusual cardiac masses. It offers several advantages over echocardiogram in that detailed anatomical extent as well as simultaneous detection of concomitant chest abnormalities can be done. Parametric mapping and delayed contrast imaging enable detection of myocardial edema and fibrosis. A high index of suspicion needed to detect Pericardial and Myocardial TB. In view of patient morbidity and mortality, prompt recognition and early diagnosis of potentially treatable cardiac TB is essential.

ARTICLE HISTORY

Received January 07, 2021
Accepted December 31, 2021
Published January 10, 2022

KEYWORDS

Cardiac tuberculosis; cardiac MRI; Pericardial TB; Myocardial TB; constrictive pericarditis; post contrast imaging; native T1 mapping

Introduction

Tuberculosis (TB) caused by Mycobacterium TB is a major health care problem in India. While pulmonary manifestations are well characterized, cardiac TB is rare. It is estimated that 1% of all cases of TB have cardiac involvement [1]. We present two cases of cardiac TB with one presenting as pericardial thickening with nodular masses mimicking Lymphoma and another diagnosed as myocarditis with concomitant lymphadenopathies.

Case–1

A 20-year-old male patient presented with hemoptysis, cough with expectoration for 10 days. The echocardiogram showed well defined hypoechoic lesion measuring 15 × 16 × 32 mm within the right atrial cavity attached to the atrial free wall (Fig. 1) with ventricular impedance and paradoxical septal bounce. Contrast enhanced computed tomography chest showed pericardial mass lesions with central necrotic regions extending into right atrium involving right atrial free wall, atrioventricular groove, left ventricular lateral wall, and encasing Superior Vena Cava (SVC) and nodular pericardial thickness of maximum 2.2 cm (Fig. 2). Inferior Vena Cava (IVC) was dilated. Multiple mediastinal lymph nodes with central necrosis were noted. Bilateral moderate pleural effusion and left lung volume loss noted.

Cardiac magnetic resonance imaging (MRI) was done in a 3-Tesla MRI (Siemens Healthineers, Skyra, Erlangen, Germany). Morphology was evaluated with Haste axial, Trufi coronal, and axial sequences with Cine Axial stack and standard cardiac cine images. myocardial tagging and contrast imaging with 3D vibe sequence was done. Diffuse pericardial thickening was noted abutting the left ventricle lateral wall, atrioventricular groove, and right atrial free wall (Fig. 3) with multiple necrotic masses observed (Fig. 4). SVC was encased by the lesion but still permeable. Minimal right pleural effusion was noted. IVC appears prominent and dilated. Multiple enlarged pretracheal, paratracheal, and aortopulmonary lymph nodes with rim enhancement noted.

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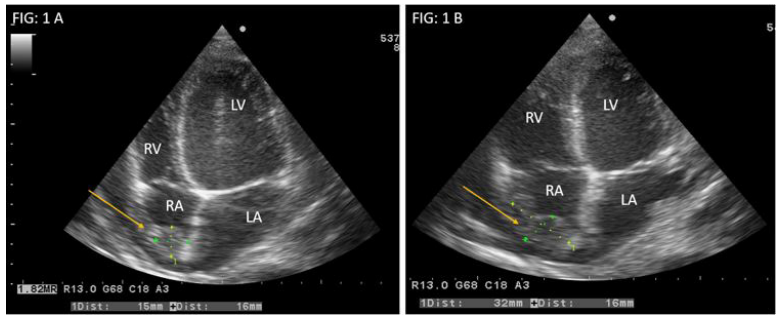


Figure 1. A and B. Four chamber cardiac ultrasound images show a lobulated hypoechoic mass in the right atrial free wall measuring 32 × 16 mm (yellow arrow) with extension within the right atrial cavity and fixed to its free wall.

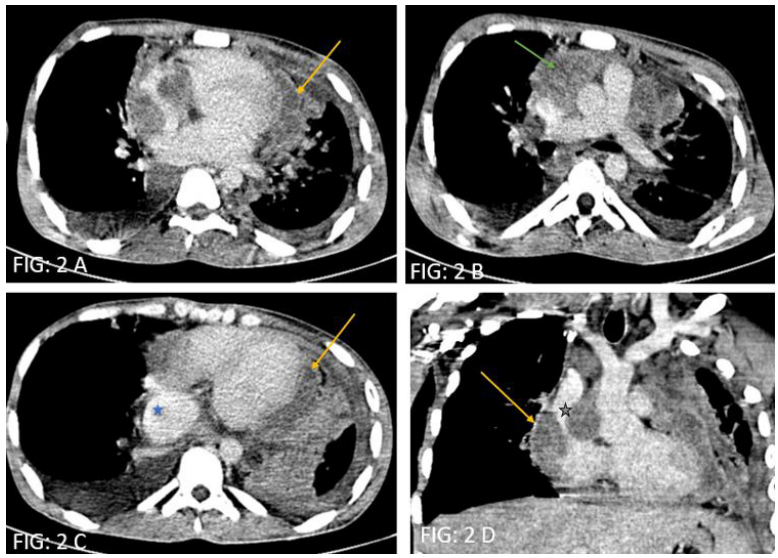


Figure 2. A, B C, and D. Axial cect-chest images show diffuse pericardial thickening with non enhancing necrotic areas (yellow arrow) involving predominantly right atrial and left ventricular walls. Inferior vena cava is dilated (blue asterix) and conglomerated mediastinal lymph nodes are noted (green arrow). Note: bilateral moderate pleural effusion and left lung volume loss. Coronal multiplanar reconstruction showing superior vena cava compressed by the mass lesion (black asterix).

Myocardial tagging showed non-breakage of grid lines abutting the thickened pericardium reflecting pericardial constriction (Fig. 5). A provisional diagnosis of Pericardial TB was made. Histopathology of the mediastinal lymphadenopathy showed caseating granulomatous inflammation due to Mycobacterium TB. The patient was started on anti-TB therapy and symptomatically improved on follow-up.

Case–2

A 41-year-old male patient presented with shortness of breath, chest pain, and on and off palpitations. Electrocardiogram (ECG) showed sinus rhythm with occasional premature ventricular complex. Echocardiogram showed mild left ventricular dysfunction and mild mitral regurgitation with an Ejection Fraction (EF) of 45%. Cardiac troponin levels were found normal. Coronary angiogram was normal, favoring underlying non-ischemic pathology.

A standard institutional cardiac MRI protocol was performed along with post-contrast and delayed phase-sensitive inversion recovery (PSIR) imaging. Pre-contrast native T_1 maps were obtained from three short-axis images (basal, mid cavity, and apical) of the left ventricle using a single short True Fast imaging with steady state free precession (FISP) based on Modified Look-Locker Inversion recovery sequence. A gadolinium-based contrast agent was administered intravenously at 0.1 mmol/kg body weight. Patchy hyperintensities noted in the mid-septum and left ventricular apex in Short tau inversion recovery (STIR) images (Fig. 6). Cine

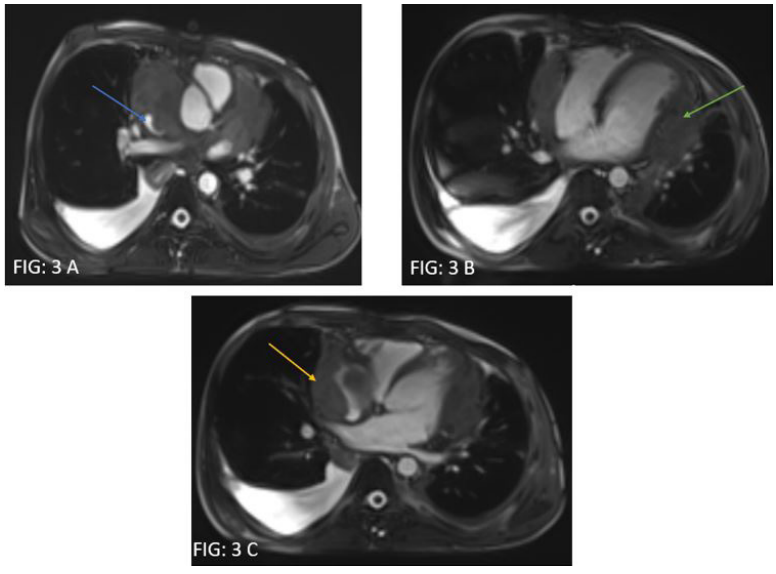


Figure 3. A, B and C. Cine trufi axial images show diffuse pericardial thickening with predominant involvement of right atrial (yellow arrow) and left ventricular walls (green arrow). Svc is encased by the mass (blue arrow). Note the moderate right pleural effusion and no pericardial effusion.

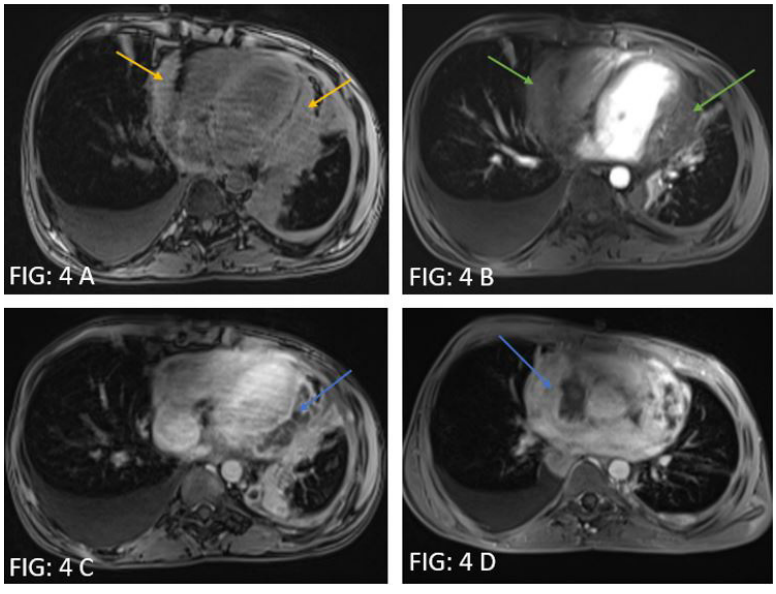


Figure 4. A, B, C and D. Native axial T_1 sequence shows pericardial thickening isointense to myocardium (yellow arrow). Post contrast axial T_1 in early phase shows heterogenous enhancement of thickened pericardium (green arrow). Post contrast axial T_1 in delayed phase shows peripherally enhancing pericardium with central non enhancing necrotic areas (blue arrow). Note: all images shows moderate pleural effusion with left lung volume loss.

imaging revealed relative global hypokinesia of the left ventricle. Delayed post-contrast images showed hyperenhancement in the interventricular septum (patchy) and the subepicardial myocardium involving the anterior, anteroseptal, inferior, and inferolateral walls (Fig. 7). Native T_1 mapping revealed significantly increased native T_1 values (1,350 ms relative to Normative T_1 values: 1,186.47 +/− 45 ms) in the above corresponding affected regions of the myocardium (Fig. 8).

The patient was also found to have extrapulmonary TB (cervical and axillary lymphadenopathy). A diagnosis of myocarditis was made, based on relative hypokinesia of the left ventricle with significantly increased native T_1 relaxation values and patchy subepicardial oedema and late enhancement.

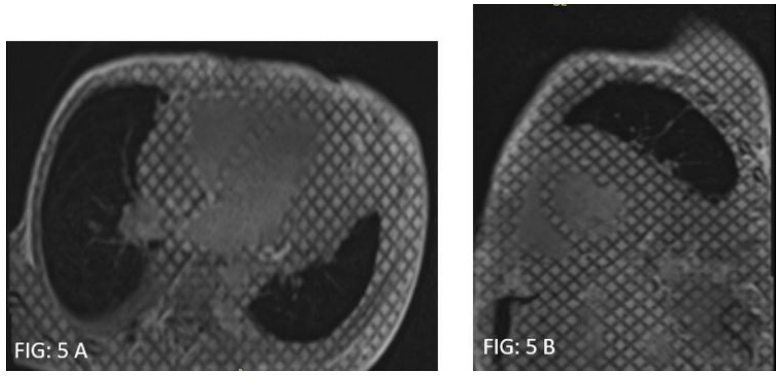


Figure 5. A and B. Axial and sagittal myocardial tagging shows non – breakage of grid lines abutting the thickened pericardium reflecting pericardial constriction.

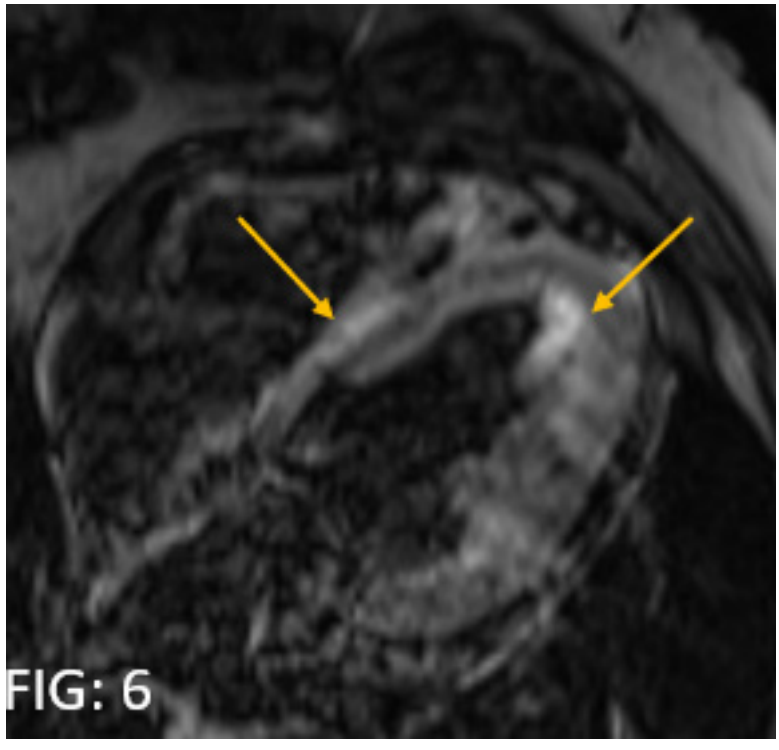


Figure 6. Stir axial image shows patchy hyperintensities in the mid-septum and left ventricular apex (orange arrow).

Native T_2 values were also significantly increased ranging from 56 to 60 ms. Histopathology of axillary lymphadenopathy was consistent with TB. A diagnosis of TB myocarditis was made in correlation with the histopathology findings (Fig. 9). Since the patient did not consent to myocardial biopsy, he was started on anti-TB drug therapy and showed progressive improvement in his symptoms at follow-up.

Discussion

Cardiac TB is essentially a disease of the pericardium. Endocardial, myocardial, valvular, or coronary arterial involvement is exceedingly rare [1]. Myocardial TB presents as rhythm disturbances, heart failure, ventricular aneurysm, coronary involvement, and aortic insufficiency [2]. Cardiac involvement occurs by retrograde spread from the adjacent lymph nodes or hematogenous spread from primary parenchymal focus [3].

Histopathologically Pericardial TB is classified into:

Stage 1: Fibrinous exudation with polymorphonuclear leucocytosis / early granuloma formation (macrophages / T-cells)

Stage 2: Serosanguinous effusion with monocytes, lymphocytes, and foam cells.

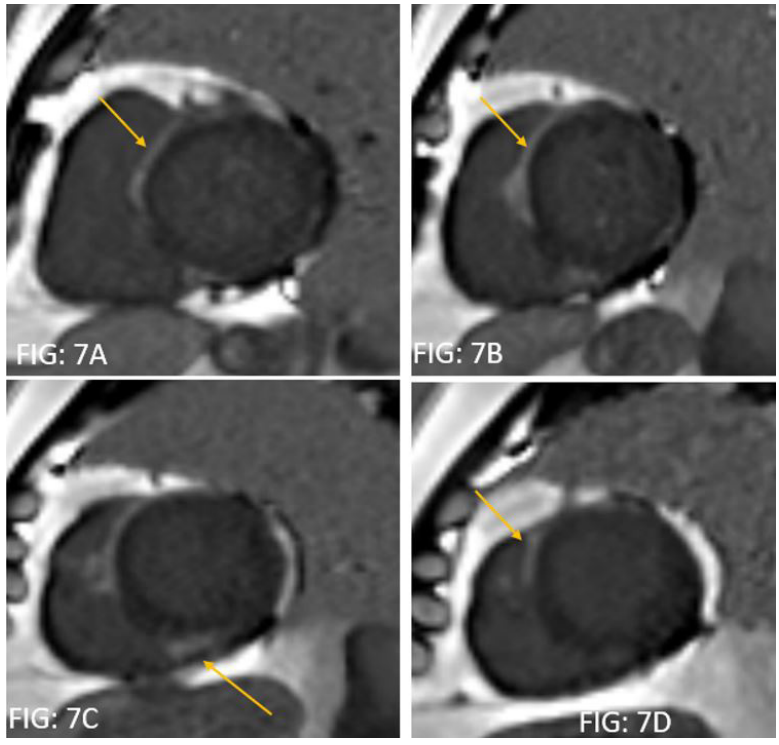


Figure 7. A–D. Delayed post contrast (gadolinium) PSIR images serial short axis views show delayed subepicardial myocardium hyperenhancement involving the anterior wall, antero-septal wall, inferior wall and inferolateral wall (orange arrow). No subendocardial delayed hyperenhancement was noticed.

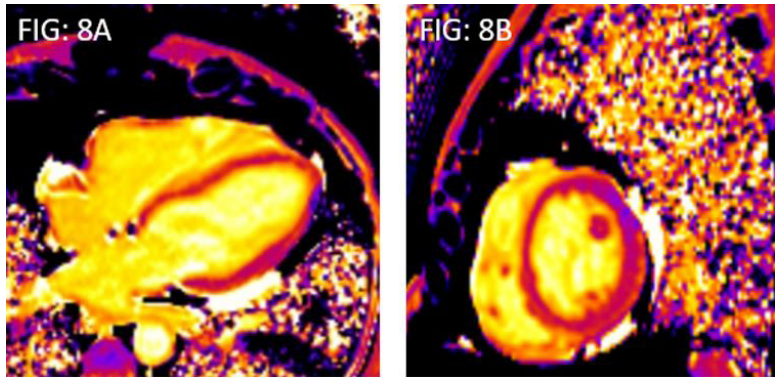


Figure 8. A and B. Native T_1 mapping four chamber view shows increased T_1 values in the ventricular septum. Mean T_1 value was 1,348 +/– 24 ms (for normal T_1 values: 1,186.47 +/– 45 ms). Native T_1 mapping short axis view shows increased T_1 values in the anterior, anteroseptal, inferior, and inferolateral walls. Mean T_1 value was 1,348 +/– 24 ms.

Stage 3: Resorption of effusion with the organization of granulomatous inflammation, pericardial thickening due to deposition of collagen, and fibrosis.

Stage 4: Fibro calcification of thickened pericardium impeding diastolic filling of ventricles (constrictive pericarditis) [4,5]. Our case demonstrates the stage between (three and four) - constrictive pericarditis with no calcification.

On post-contrast imaging, there is an enhancement of the thickened pericardium with non-enhancing areas suggestive of granulomatous caseating necrosis [6]. Our case showed a similar isointense mass with heterogeneous contrast enhancement. Identification of thickened pericardium and adhesion has shown to be an important differentiating feature of constrictive pericarditis of TB etiology from other causes. Cardiac MRI plays a vital role in evaluation of this condition since results from endomyocardial biopsy have been found to be not specific. The pericardial thickening can be assessed using standard cine images and the

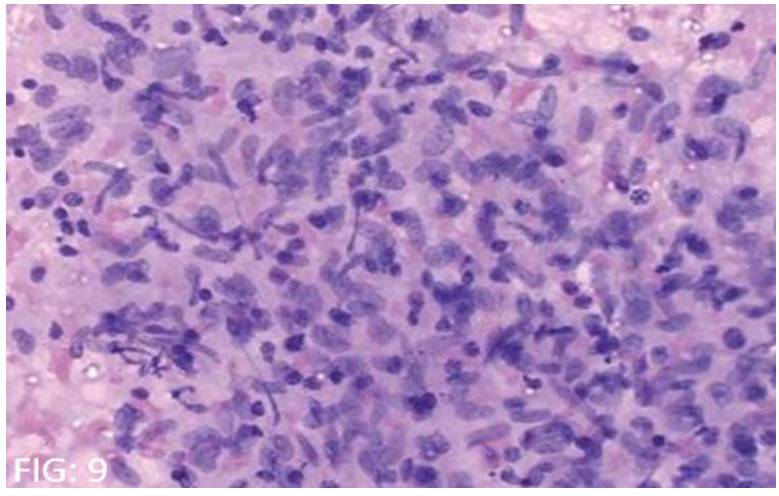


Figure 9. Histopathology from axillary node showing epitheloid granuloma with multinucleated cells and macrophages (yellow arrow) with caseous necrosis suggestive of TB etiology.

post contrast 3D VIBE sequences while the adhesions and fixation can be delineated using the myocardial tagging sequences, which shows non breakage of grid lines [7]. Cardiac MRI has been shown to demonstrate the pericardial thickening in earlier reports of TB pericarditis as well [8].

Cardiac Lymphoma presents as part of disseminated disease or rarely a primary malignancy [9,11] and presents as infiltrating epicardial or myocardial mass extending along epicardial surface involving coronary artery and aortic root [10] and most commonly infiltrates right atrium, IVC and SVC. Cardiac valves, ventricles, aortic root are usually spared. Pericardial effusion with pericardial thickening may be the usual common or the only finding in some cases [11, 12]. Morphologically Lymphoma will appear isointense and lobulated mass with homogenous enhancement with no necrosis. There may be other contiguous and non- contiguous lymph nodal involvement and it can also be present with multisystem involvement. In our case in addition to diffuse pericardial thickening with enhancement, other findings including multiple necrotic mediastinal and peritracheal nodes, bilateral pleural effusion and left lung volume loss and considering the age of the patient a provisional diagnosis of constrictive Pericardial TB was made rather than a primary cardiac Lymphoma.

TB myocarditis occurs as confluent masses, diffuse miliary, or infiltrative pattern [1]. MRI is particularly useful in detecting acute myocardial oedema, characterization of myocardial tuberculomas, and diffuse infiltrative involvement [13–15]. Serial MRI is helpful in follow up by demonstrating partial or complete resolution with or without the persistence of fibrosis.

T_1 mapping has shown consistency in determining the location, extent, and patterns of myocarditis, with significantly greater sensitivity than the Lake Louise criteria (LLC) (85% *vs.* 74%; p 5.025). Native T_1 and T_2 mapping with late gadolinium enhancement (LGE) significantly improved the diagnostic accuracy of CMR imaging to 96%. [16]

The LLC are currently the recommended diagnostic cardiac magnetic resonance imaging criteria for patients with suspected myocarditis. Original LLC use three major tissue based CMR markers, namely, T2 weighted (T2W) ratio, early gadolinium enhancement (EGE) and nonischaemic distribution pattern on LGE images. These parameters assess for myocardial oedema, hyperemia / capillary leak, and fibrosis/ necrosis, respectively.

The 2018 modified LLC consist of two main criteria, namely, increase of native T1 relaxation time, increase of extracellular volume (ECV), or positive non ischemic pattern of LGE and increased T2 relaxation times.

Since EGE and T2W images may prone to artifacts and misinterpretation, generalizing the application of the LLC criteria in routine clinical practice is quite challenging.

Native T1 mapping can detect myocarditis at various stages since both oedema and extracellular expansion contribute to T1 prolongation time. Native T1 is dependent on both intracellular and extracellular/ interstitial factors. This is an advantage compared to LGE since gadolinium contrast agents can only assess the extracellular space, and hence cannot detect intracellular oedema which is thought to occur in early stages of myocarditis.

Hence Native T1 offers both excellent sensitivity and specificity when the optimal cut off is chosen during the acute phases of myocarditis in which oedema is most prevalent.

It is also important to note that as the fibrosis occurs after early inflammation subsides, native T1 prolongation becomes less specific to myocarditis.

T2 relaxation times are most elevated during the acute phase of myocarditis and gradually normalize over months since they primarily detect free water content which could be useful for staging and monitoring recovery.

The main advantage of ECV when compared with LGE is that it can assess diffuse fibrosis and inflammation beyond focal areas of fibrosis. ECV measurement is relatively insensitive to field strength, when compared with native T1. Global ECV can identify diffuse myocardial injury in patients with negative LGE.

Endomyocardial biopsy is not routine clinical practice in many institutions since it is reported to be prone to a sampling error leading to false—negative results, especially in patients with focal myocardial inflammation and also due to patient non-compliance as in our case, no systematic endomyocardial biopsy as a reference standard was made.

Thus, in diagnosing patients with acute myocarditis, Native T1, T2 and ECV mapping were shown to be comparable to LLC. Native T1 had significantly better sensitivity than LLC and by only assessing a single parameter like native T1 could simplify the diagnosis of myocarditis when compared with using the LLC [17].

Conclusion

Parametric mapping and late gadolinium imaging enable detection of myocardial oedema and fibrosis. A high index of suspicion is needed to detect Myocardial TB than Pericardial TB. Native T_1 mapping is now part of the protocol for suspected myocarditis and depicts elegantly the fibrosis as shown in our second case. Cardiac TB is rare nowadays because of the control of the disease in the last decades, but it is still a diagnosis that we must make because of its severity, patient morbidity and mortality, prompt recognition, and early diagnosis of potentially treatable cardiac TB is essential.

Consent for publication

We have got the informed written patient consent regarding the publication of case images and clinical history without disclosing the details as per the journal protocol.

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