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Cardiac TB: A Diagnostic Challenge

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ABSTRACT

Cardiac tuberculosis is relatively rare, very difficult for doctors to diagnose, and is the leading cause of death from infectious diseases. It is one of the top 10 causes of death worldwide, with a disproportionate impact in low- and middle-income countries. Radiologists play an important role because CMR is a non-invasive radiological method that helps identify potential overlaps and differential diagnoses between tuberculosis, mass lesions, pericarditis, and myocarditis. fulfill. The combination of clinical and specific radiological features that are also discernible in comparison with his previous and subsequent CMR scans, regardless of similarities or overlaps in observations, aids in differential diagnosis. CMR offers significant advantages over echocardiography in the detection, characterization, and assessment of cardiovascular abnormalities. Knowledge of LGE, feature tracking, and parametric imaging in CMR, coupled with clinical manifestations, may aid early detection of tuberculous pericarditis, serve as a surrogate for endomyocardial biopsy, and lead to more rapid diagnosis and treatment. increase. The purpose of this article is to describe the current state of cardiac tuberculosis, the diagnostic utility of CMR in tuberculosis (TB) patients, and to provide an overview of the various imaging and laboratory tests used to detect cardiac tuberculosis.

INTRODUCTION:

Tuberculosis of the cardiovascular system is quite rare. Although TB is generally treatable, it continues to be the leading cause of morbidity and mortality worldwide, second only to HIV as the most common infectious disease [1]. Cardiovascular TB has experienced a remarkable resurgence in the context of the HIV pandemic, with an increase in extrapulmonary or diffuse lesions. Tuberculosis is the cause of virtually all pericardial effusions in HIV-positive patients in sub-Saharan Africa, compared with 50–70% in the general population and less than 5% in the industrialized world. [2]. Tuberculosis-associated heart damage occurred in 0.3% of autopsied patients [3]. Pericardial TB (TB) is found in approximately 1% of all cases of TB at autopsy and in 1-2% of cases of pulmonary tuberculosis [4]. Isolated TB myocarditis is extremely rare; however, it is more common in HIV-infected individuals and those with chronic immunosuppression, with a prevalence of 51% [2]. MRI has a significant advantage over echocardiography in the detection, characterization, and evaluation of cardiovascular abnormalities.

Heart Tuberculosis: Clinical Symptoms and Signs:

Myocardial TB, first detected by Maurocordat on postmortem in 1664, is un- typical and often remains undiagnosed while the patient is still alive [5]. Cardiopulmonary tuberculosis accounts for between 1% and 2% of all tuberculosis cases in immunocompetent persons [6]. In a study of 19 individuals with cardiovascular TB, one case was diagnosed antemortem, 11 had nodular lesions on autopsy, and 7 had miliary lesions; only one patient had acid-fast bacilli [7]. Most often retrogradely from mediastinal lymph nodes, hematogenous from a primary tuberculosis infection, and infrequently contiguously are the three main ways that TB infection of the cardiovascular system is spread [8,9]. Several studies have found that the right ventricle and right atrium are the most typically impacted, most likely due to the frequent involvement of right mediastinal lymph nodes, hematogenous from a primary tuberculosis infection, and rarely contiguously [8,9]. The right ventricle and right atrium are the most frequently affected organs, according to several studies. This is probably because the right mediastinal lymph nodes are frequently involved, which leads to myocardial involvement. [7,10–12]. Tuberculous pericarditis has four recognized pathological stages: (1) fibrinous exudation with initial polymorphonuclear leukocytosis with the loose organization of macrophages and T cells; (2) serosanguineous effusion with a predominantly lymphocytic exudate composed of monocytes and foam cells; (3) effusion absorption with the organization

of granulomatous caseation and pericardial thickening; and (4) constrictive scarring [8].

Clinical Signs and Symptoms of Heart Tuberculosis:

There are numerous ways in which TB of the myocardium can be present. Just 3% to 8% of individuals with tuberculous pericarditis exhibit acute pericarditis, which is an uncommon clinical manifestation of the condition. Severe pericarditis chest pain, pericardial friction rub, pervasive ST-segment and T wave abnormalities, and PR segment depression are its defining features [13,14].

On the other hand, the presentation is more subtle, and systemic signs and symptoms, such as chest pain, fever, dyspnea, pericardial rub, paradoxical pulse, and hepatomegaly are more prevalent [15].

Clinical symptoms are influenced by the rate of fluid accumulation, the hemodynamic effect on cardiac contraction, and the degree of infection-induced inflammation [16].

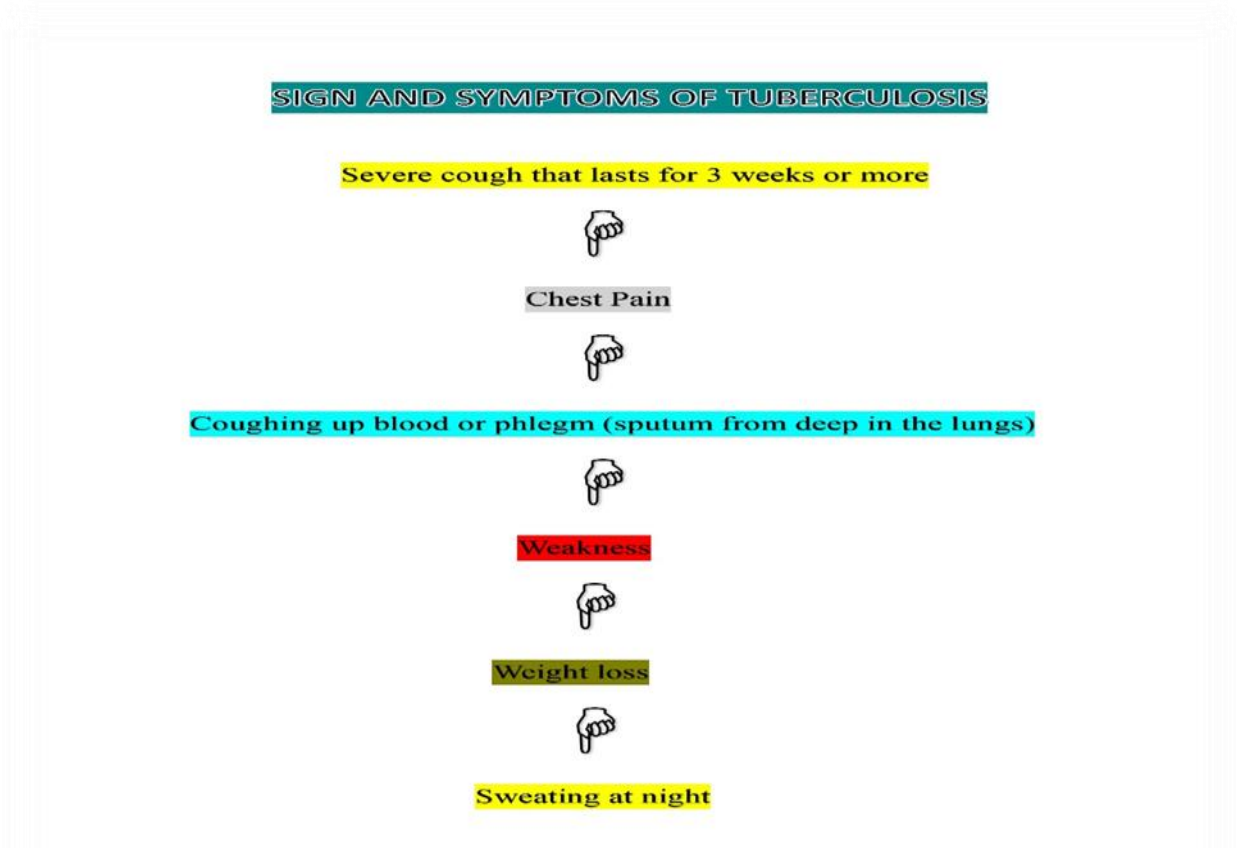
The most common symptoms of fluid accumulation are broad systemic symptoms or heart failure [17,18]. When fluid accumulates rapidly, and compensatory mechanisms are unavailable, the patient develops tachycardia and hypotension.

Cardiac tamponade may occur in up to half of patients receiving insufficient care, and death rates can reach 85% at six months [19,20]. Symptoms of acute tuberculous myocarditis include abnormalities in the conduction system, such as prolonged QT syndrome, ventricular fibrillation, or cardiac arrest [6,8,21–25].

On the other hand, chronic myocardial involvement may become apparent due to worsening heart failure symptoms or a postmortem discovery of an asymptomatic patient [26].

Similar signs and symptoms to pericarditis characterize subacute endocarditis. When vegetation interferes with hemodynamics and drastically reduces valve performance, symptoms including dyspnea and heart failure manifest [27].

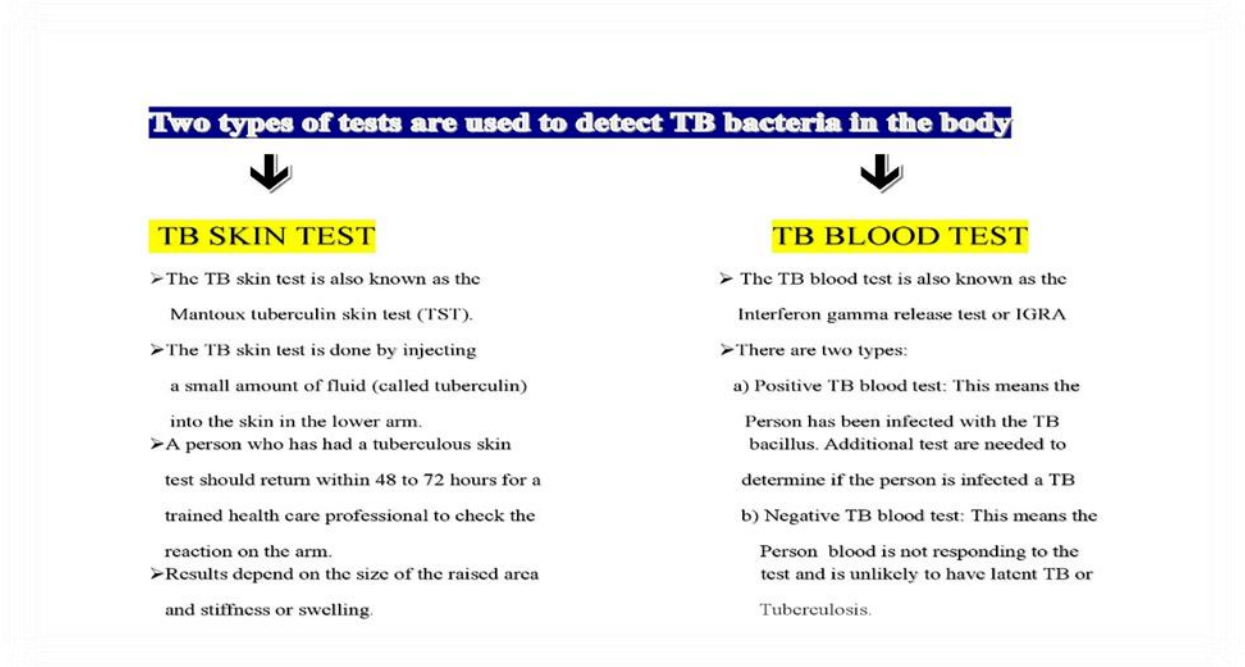
Ad- dictionary, the literature has identified the blockage of the right ventricle's outflow tract, ventricular aneurysm, ventricular pseudoaneurysm, congestive heart failure, valvular endocarditis, and myopericarditis [6,8,21–25,28].



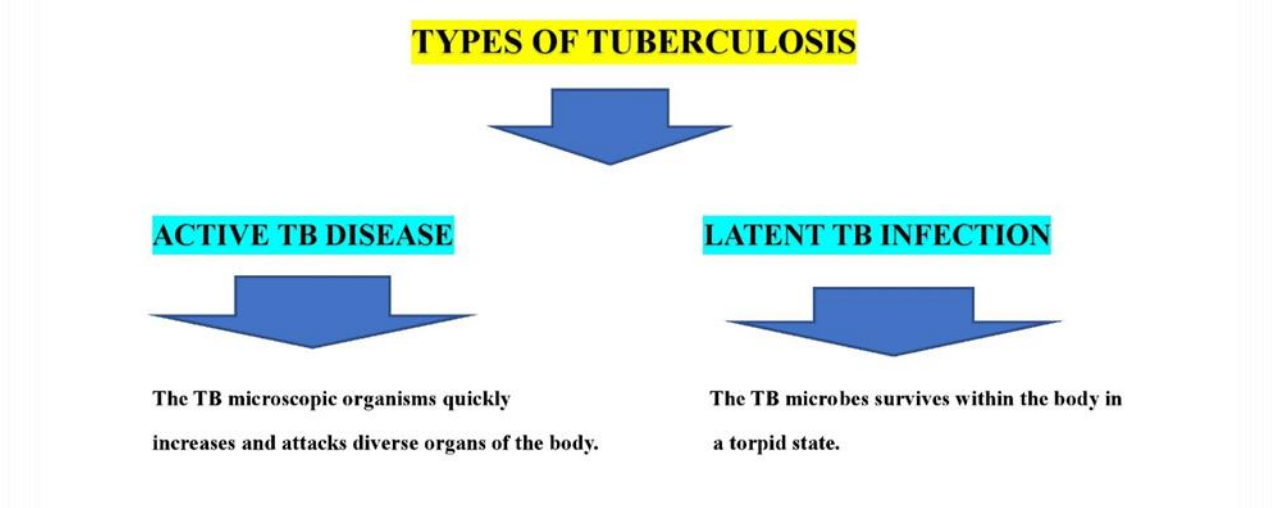
Test for Laboratory Diagnostics:

The tubercle bacillus must be directly or indirectly isolated by culture from pericardial fluid, the heart, or vascular tissue in order to provide a clear diagnosis of cardiac TB. In reality, however, isolation can be difficult. A method for detecting TB in the circulatory system was established by Lopez et al. [9] by integrating clinical, laboratory, and radiological tests, including cardiac CT and CMR. When pericardial effusion was evident and there were clinical signs of fever, weight loss, and night sweats, pericardiocentesis was advised [9]. To confirm an infection, many tests can be used, each with a different diagnostic performance. Depending on where the test is most likely to be administered, cardiovascular disease and the test's accessibility in the neighborhood. The diagnosis of tuberculosis is made through clinical examination, specific pericardial fluid tests for adenosine deaminase (ADA) levels, gamma interferon (IFN-y), and polymerase chain reaction testing for Mycobacterium tuberculosis [20]. Mutyaba et al. offer the following diagnostic approach for TB pericarditis in endemic settings in their proposal: (1) Obtain biomarkers such as IFN-y or ADA levels in pericardial fluid and confirm tuberculosis in other places to rule out other potentially fatal causes of an inflammatory exudative effusion (e.g., bacterial, neoplastic, or uremic) (e.g., lymph nodes, sputum, or pleural fluid) [8,29]. The conversion of adenosine to inosine requires an enzyme

called ADA. Lymphoid tissue has the highest concentration, but may be present in many tissues. Elevated ADA activity appears to be associated with an activated antigenic response of lymphocytes in tuberculosis [8]. In a systematic review and meta-analysis, the sensitivity and specificity of the ADA test were 88% and 83%, respectively, with a receiver operating characteristic area of 0.9539 [9]. Therefore, ADA activity is of significant value in the diagnosis of pericardial tuberculosis [8]. Moreover, unstimulated IFN-γ in pericardial fluid is a TB biomarker. Its value is based on the fact that T lymphocytes produce IFN-γ when exposed to certain antigens [30].



A cut-off value of 50 pg/mL resulted in 92% and 100% sensitivity and specificity compared to the reference standard [9]. Despite its high diagnostic accuracy, the test is expensive and not widely used, especially in low- and middle-income countries. A (PCR, polymerase chain reaction) test is a molecular biological technique for amplifying specific DNA sequences (deoxyribonucleic acids). The polymerase chain reaction has high specificity (96% to 100%) for detecting tuberculosis in pericardial fluid, but several studies have shown low sensitivity (15-20%). has been [20,31]. Comparing three pericardial tuberculosis tests, IFN-γ showed the highest sensitivity and specificity with a sensitivity of 95.7% and a specificity of 96.3%, demonstrating that IFN-γ is the most effective diagnostic test. Laboratory testing, including PCR, is currently the fastest and most sensitive molecular biological technique when applied to pericardial fluid. Nonetheless, liquid cultures for tuberculosis remain the gold standard for laboratory diagnosis.

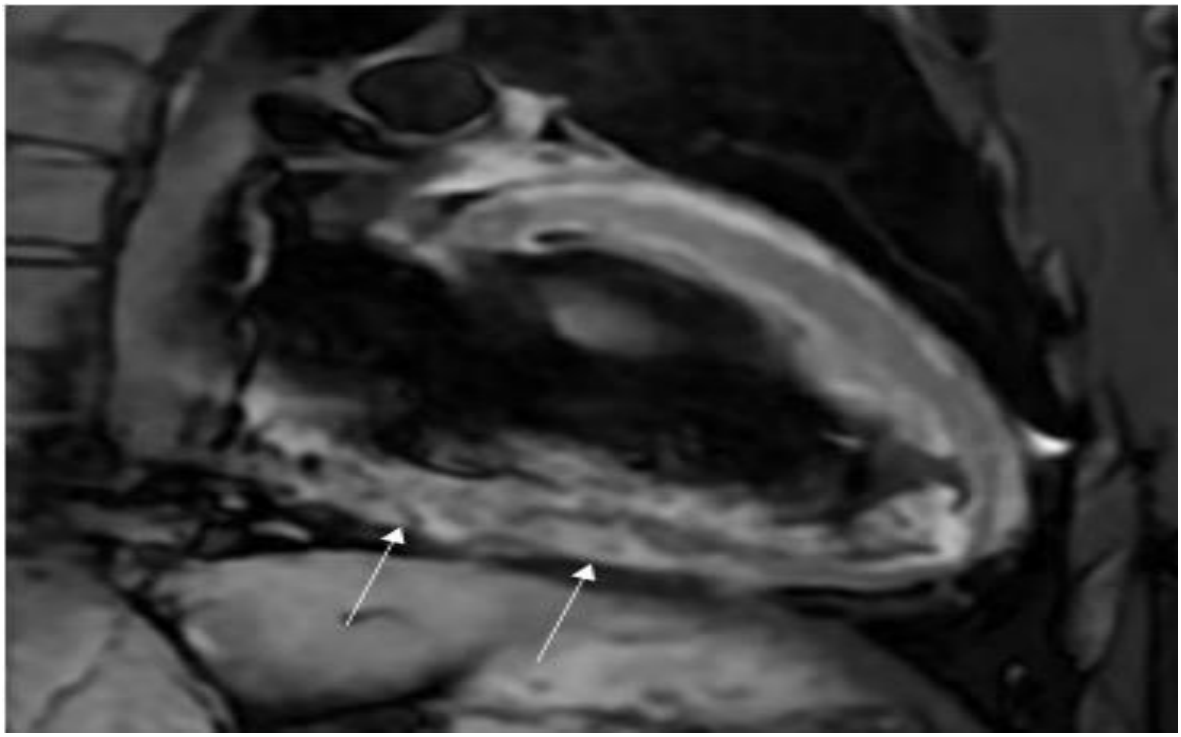


The role of cardiac MRI:

Pericarditis and myocarditis:

Myocarditis is a term used to describe a condition that is predominantly pericardial. Pericarditis is often associated with myocarditis of varying severity. Because of similar pathogens, cardiotropic viruses, pericarditis, and myocarditis may coexist in clinical practice [28]. The evaluation of pericardial disorders requires more than a morphological examination; The diagnostic challenge is to determine the effect of the abnormal pericardium on cardiac filling. The ability to combine anatomical and functional data in a single examination, the ability to characterize tissues and determine the presence and severity of inflammation and disease activity, and the value of CMR to accurately assess the rest of the heart, especially the myocardium, which is currently a diagnostic challenge, are all convincing arguments in favor of CMR. The detection of pericarditis has become easier with the development of new CMR techniques [32,33]. T1-weighted spin-echo MRI or cine MRI can detect pericardial thickness and pericardial effusion simultaneously, while T2-weighted spin-echo STIR MRI can detect pericardium edema. Pericardial enhancement during CMR with gadolinium injection is an appropriate method for detecting pericarditis (Figure 1-3). Both LGE CMR and spin echo CMR are beneficial. A fat-suppressing preparation may improve the visibility of pericarditis [32]. Drawing of the pericardium, which may become more irregular in chronic pericarditis, and striated enhancement of surrounding fat and adjacent myocardium, suggest pericarditis or concurrent myocarditis time and are additional attractive visual features [32]. Yelgec and colleagues recently found pericardial enhancement in 9 people and pericardial effusion in 6 of 20 patients tested by CMR because of the clinical

suspicion of acute myocarditis, confirming the rate of pericardial effusion. rates of myocarditis [34]. Also, Sayedetal. Thus, the two results demonstrated that myocarditis and pericarditis can be generated by the same etiological factors and coexist in the same patient.



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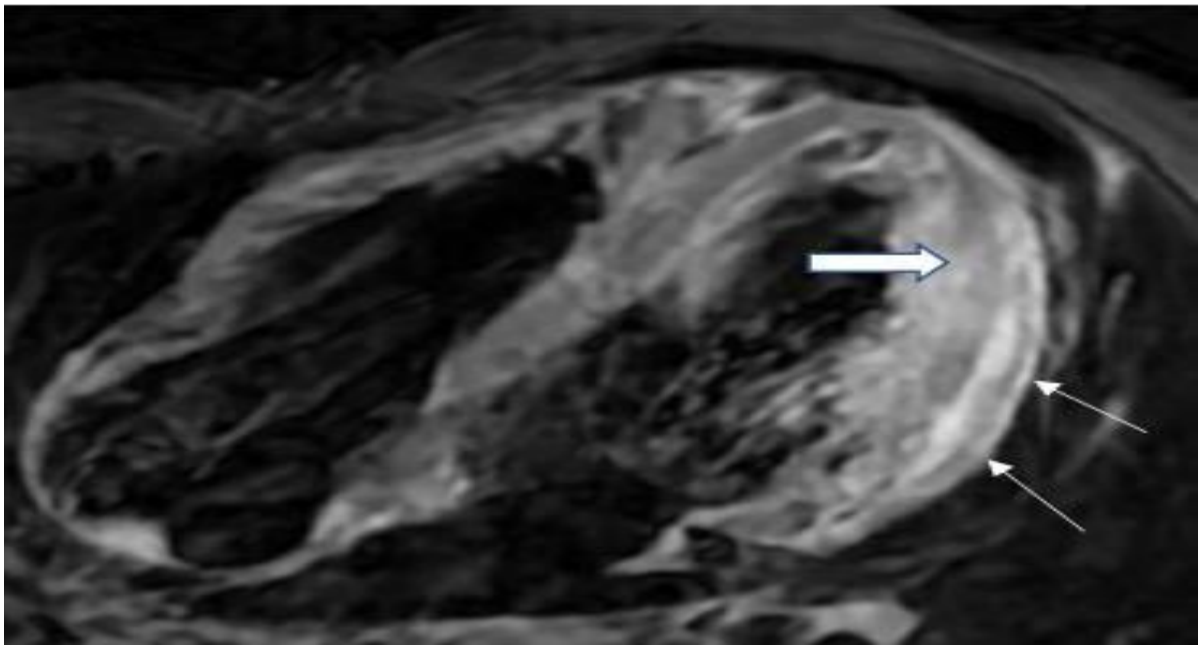

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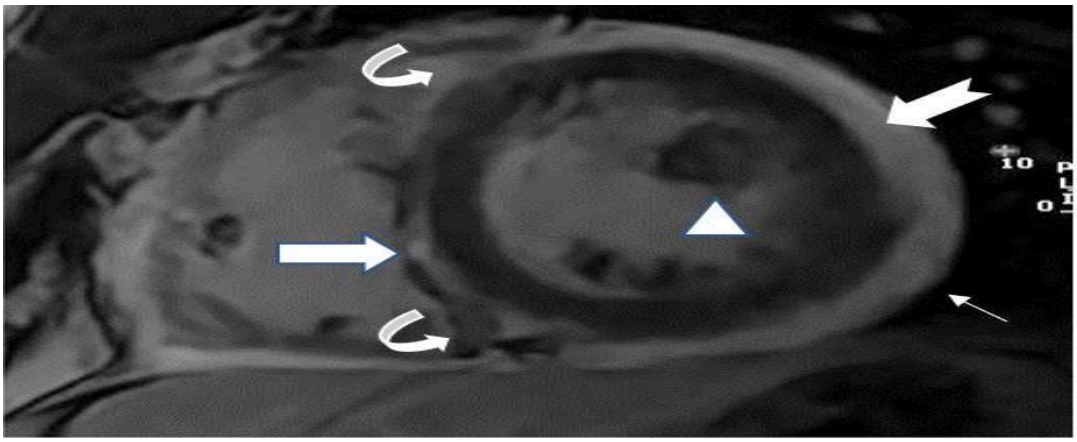
Figure 1. (A) A 35-year-old man with a recent history of TB pericarditis treated for 6 months had CMR due to ventricular tachycardia. The 2CH T2w STIR image shows increased epicardial and transmural signals in the inferior wall (arrow).

(B) A 35-year-old man with a recent history of tuberculosis pericarditis treated for 6 months has CMR due to ventricular tachycardia. The 4CH T2w STIR image shows irregular thickening of the pericardium (small arrows) with increased transmural signal from the medial-lateral wall (thick arrows).

Pericardial spasm is usually idiopathic, although it can also be caused by inflammation of the pericardium, such as tuberculosis. Tuberculosis decreased compared with other cardiac surgery-associated diseases, while radiation-induced pericarditis increased [35]. Increased systemic venous pressure and inadequate cardiac output are common signs of pericardial spasm [36]. Chronic sclerosing pericarditis is characterized by a thickened, fibrous, and calcified pericardium that constricts the heart and affects filling [37]. Therefore, the diagnosis of heart failure should always be checked when the patient is symptomatic [38]. Equalizing end-diastolic pressure in all four chambers of the heart and improving ventricular connectivity are pathophysiological features of pericardial constriction caused by confinement of the cardiac chambers by pericardial volume. the heart is stiff and unchanged [35,39].



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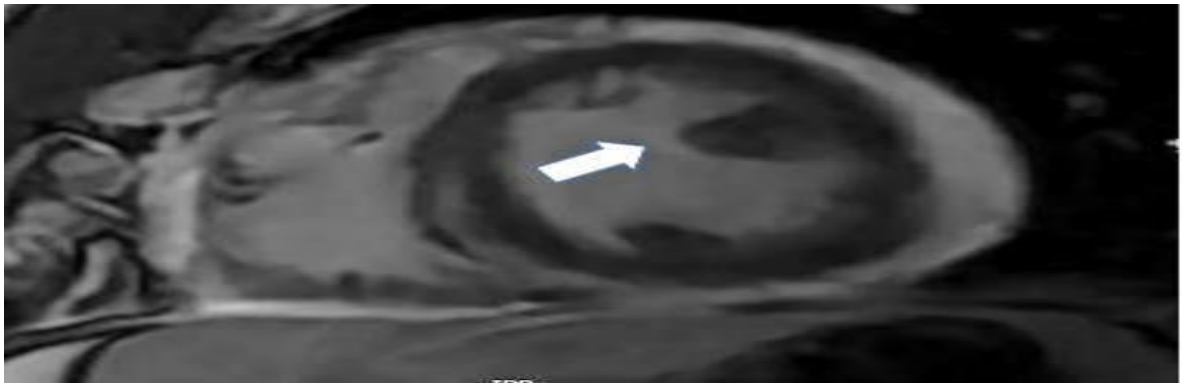

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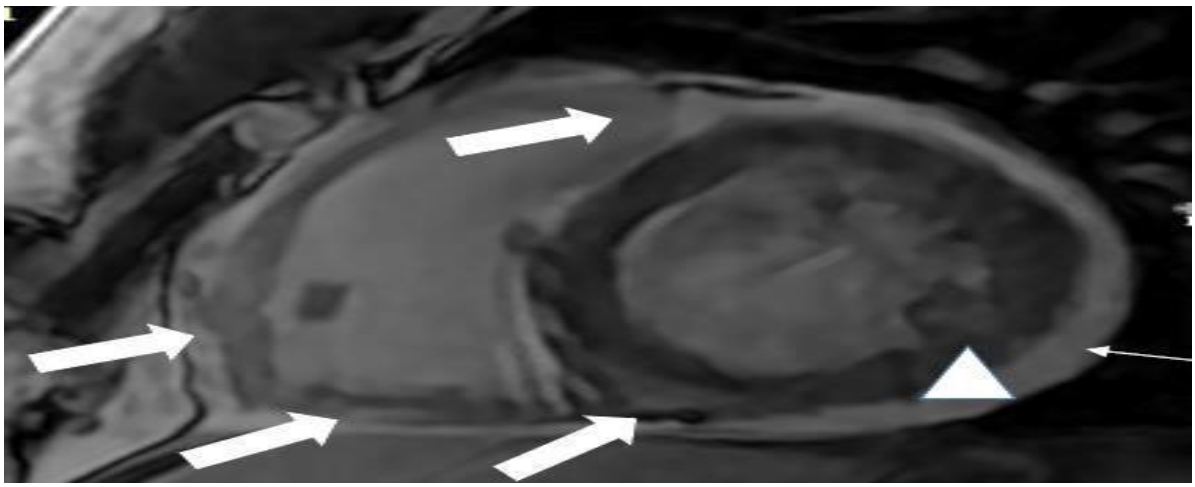

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Figure 2. (A) A 35-year-old man with a recent history of tuberculous pericarditis treated for 6 months underwent CMR for ventricular tachycardia. Short axial mid cardiogram, late enhanced image, showing anterior and posterior RV subpericardial space (knotted arrows), medial and inferior septal fascial centers (white arrows), and anterior and posterior RV insertion points (curved arrow) with pericardial thickening and improved pericardial fat (thin arrow). Expanded AL papillary muscle (arrowhead).

(B) A 35-year-old man with a recent history of tuberculosis pericarditis treated for 6 months has CMR due to ventricular tachycardia. Mid-slice of the short-axis myocardium shows all features of (A) and asymmetric enlargement of the AL papillary muscle (arrow).

(C) A 35-year-old man with a recent history of tuberculosis pericarditis treated for 6 months had CMR due to ventricular tachycardia. Late enhancement in the short-axis midsection shows the lateral and inferior RV walls (arrows), and superior and inferior compression

(white arrows) with enhancement of epicardial ointment (thin arrows). The PM papillary muscle has central enhancement (arrowheads).

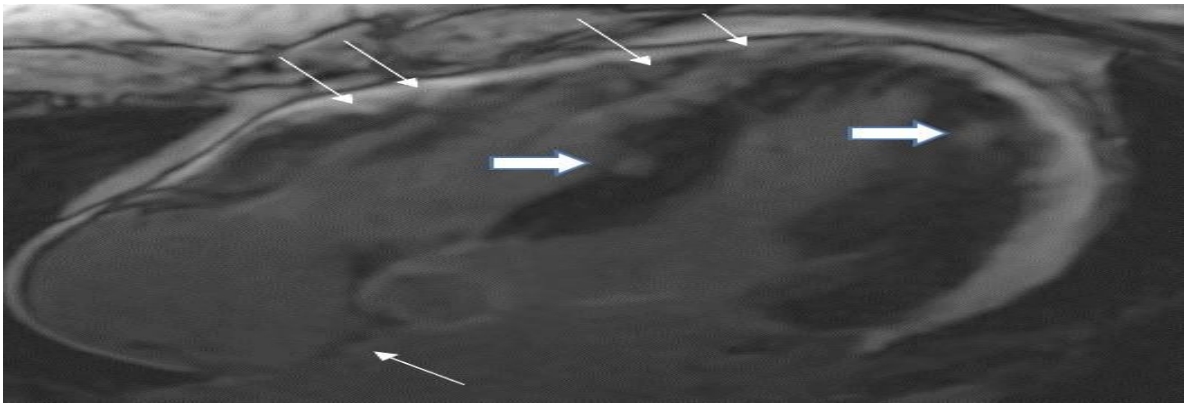


Figure 3. A 35-year-old man with a recent history of TB pericarditis treated for 6 months underwent a CMR scan for ventricular tachycardia. 4 CH, late enhancement, shows enhancement of the RV free wall (thin arrows in the epicardium and transmural), inferior septum (thick transmural arrow), lateral LV wall (thick transmural arrow)) and the atrial septum (thin arrow).

Pericardial thickness has long been recognized as a strong predictor of constrictive pericarditis [40,41]. The thickness of the pericardium varied from 1.5 to 3.9 mm in some sites of tuberculous myocarditis (Figure 1B). On pathologic examination, maximal pericardial thickness varied significantly (1–17 mm; mean, 4 mm) in patients with pericardial constriction (96%), up to 20% of patients with a normal thickness (2 mm) [40, 41]. According to Feng et al., the pericardial thickness in subjects with chronic constrictive pericarditis was significantly lower than in those with reversible constrictive pericarditis (2 mm1 vs 4 mm1; p). > 0.001) [42]. Thus, patients with end-stage constrictive pericarditis have a narrower pericardium than those with chronic inflammation who survive. In addition, the CMR labeling technique can be advantageous in accurately displaying pericardial adhesions [32].

TB Myocarditis:

Myocarditis is thought to involve diffusely both ventricles or, less commonly, the right ventricle alone. In one of the studies with endocardial biopsies, biventricular damage was noted in about 70% of patients and only RV myocarditis in 8% [43]. Myocardial involvement can be identified by abnormal electrocardiographic changes, transient global and local

movement abnormalities of the cardiac wall, and elevated cardiac enzyme levels. The CMR image of the patient included here (Figure 1-4) shows elevated VT and cardiac enzymes with the clinical diagnosis of myocarditis. MRI verified all three Lake Louise consensus criteria and decreased bilateral ventricular function in the present case. There was asymmetric LGE in the RV free lateral wall, the inferior wall, the superior and inferior RV/LV junctions, and the atrial septum (Figure 2). It supports the pathogenesis of cardiac tuberculosis by showing that the right side is affected more frequently, possibly due to the proximity of the right mediastinal lymph node [7,10–12]. Transmural LGE, eccentric center (septum), and epicardium are also seen in the left ventricle (Figure 2A). The motion abnormalities of the subregional septum (Figure 2A) correlate well with mesenchymal enhancement and may represent an area of active inflammation progressing to chronic scarring due to tuberculous infiltrates [44]. The diagnostic accuracy of myocarditis supported by the original T1 map was 1132,178 (Figure 4) and the ECV was 258%. Those with acute chest discomfort can be differentiated from those with acute coronary syndrome or myocarditis by using native T1 and ECV [45]. To identify and evaluate diffuse myocardial fibrosis and edema, native T1 and ECV mapping is more sensitive than LGE [45].

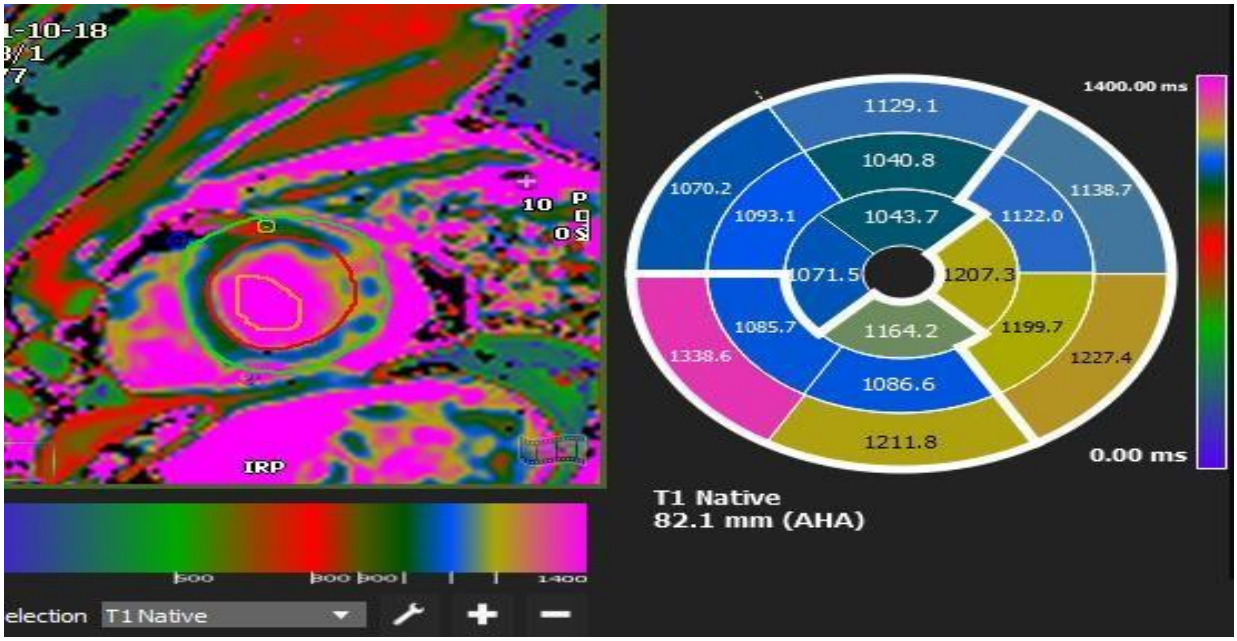
Expanding papillary muscle:

Papillary muscle enlargement has not been previously described in tuberculous myocarditis (Figure 2B). Hypertrophy or infiltrative diseases (eg, amyloidosis, sarcoidosis, iron deposition) can thicken the papillary muscle, affecting the papillary muscle [44]. Although coronary atherosclerosis is the most common cause of unruptured papillary muscle LGE, the disorder can also result from shock, infective endocarditis, acute regurgitation, anemia, LVOT blockade, and hyperemia. systemic pressure, cardiomyopathy, endocardial fibrosis, endocardial fibrosis. and cardiomyopathy [44,46].

Endocardial TB:

Tuberculosis is a mass lesion of cardiac TB [5,47,48]. Endocardial TB is extremely rare, occurring in only 19 of 13,658 autopsies (0.14%) [7]. Myocardial tuberculosis is most commonly found in the right heart, especially in the wall of the right atrium. They are often different from the surrounding parenchyma and may be single or multiple [10, 49]. Cardiovascular tuberculous tumors may be asymptomatic or present with pulmonary venous obstruction due to large left atrium lesions, left ventricular aneurysm, right ventricular

outflow tract obstruction, vena cava obstruction, coronary artery occlusion, or cardiac dysfunction. ventricular function, ventricular rupture, aortic failure, or regurgitation. arrhythmia, complete heart block or sudden cardiac death [50,51]. Nodular TB is associated with the development of ventricular aneurysms [52]. The actual development of an aneurysm due to myocarditis is very rare.



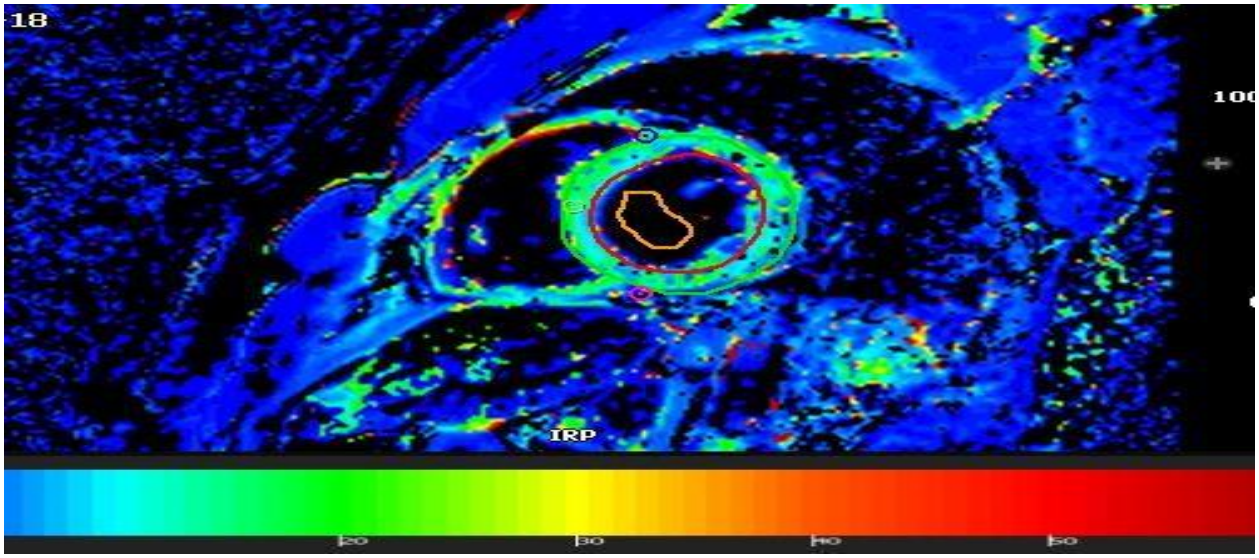
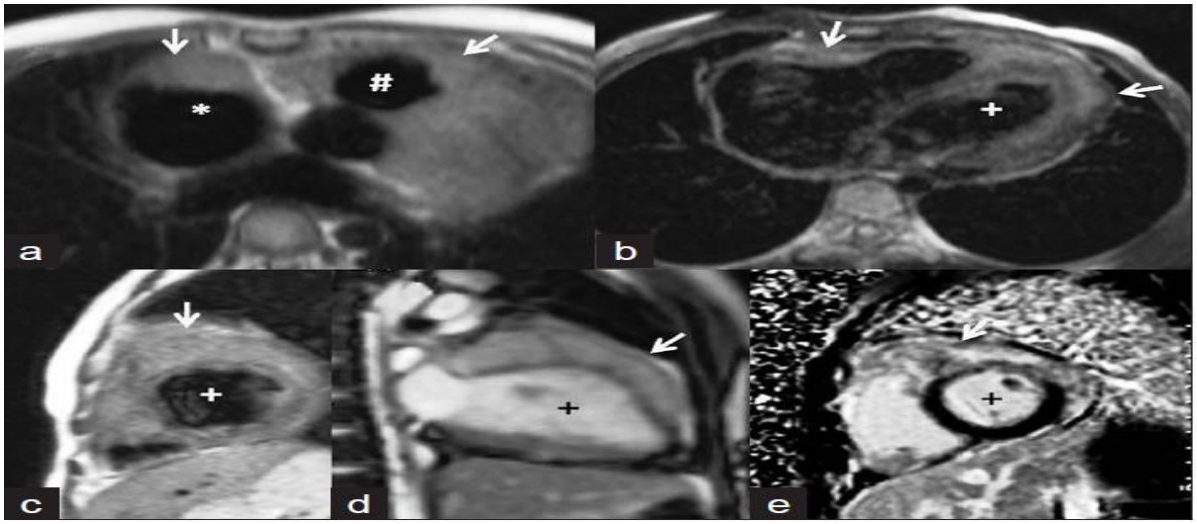


Figure 4. A 35-year-old man with a recent history of TB pericarditis treated for 6 months underwent CMR for ventricular tachycardia. The original T1 map was 1132 ± 178 (Figure 4) and the ECV was 25 ± 8%.

Cardiovascular magnetic resonance imaging is a relatively new method showing mild to moderate T2 shortening (Figure 5, This image is adapted from Dixit et al. [53] and 6, This image is modified and adjusted from Gulati et al [47]) is comparable to that seen in TB-associated brain tumors [53]. A central co-signaling nucleus within the central capsule, a hypointense rim relative to the capsule, and a thin hyperintense line around inflammatory cell infiltration can be observed on T2-weighted images [53]. Ring enhancement with agglomeration can be seen on post-gadolinium MRI (Figure 6, this image is adapted from Gulati et al. [47]). Aggregation develops in the presence of granulomatous lesions, which is rare in tumors [53].



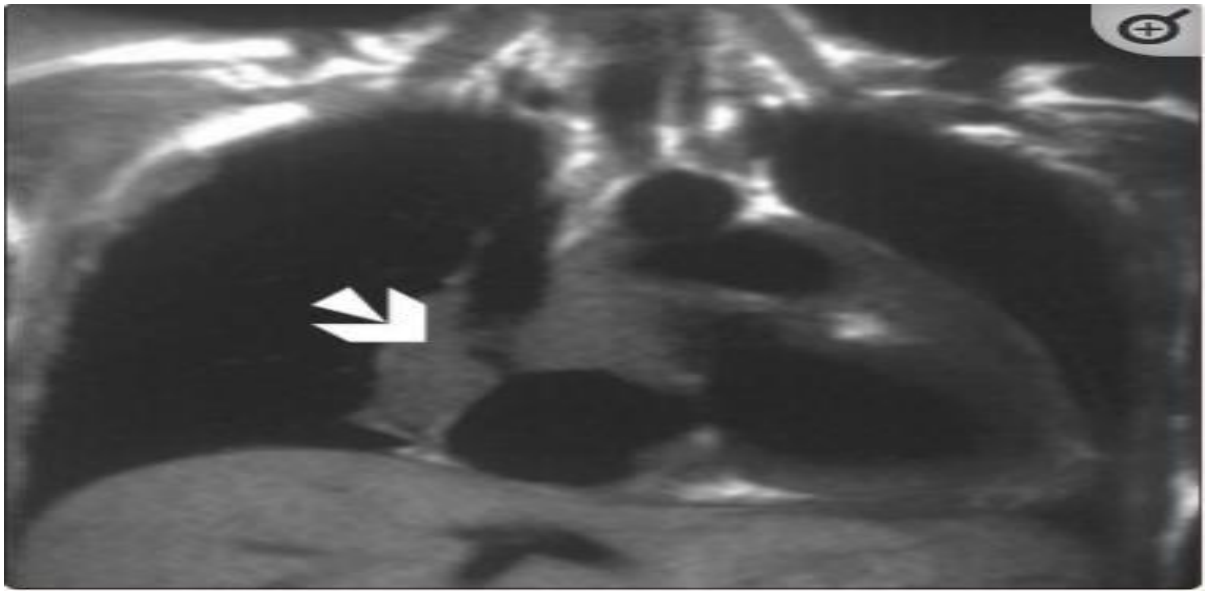


Figure 5. Coronary dark blood T2-weighted MRI image shows diffuse pericardial thickening. The thickening is hypointense on T2W images and causes deterioration of the proximal SVC (arrow). The image is adapted from [53].

Figure 6. Magnetic resonance imaging of the heart. (a,b) Axial T1-weighted image shows co-intensity blocks (arrows) along the right anterior atrium (*), right ventricular outflow tract (#) and along both ventricles (+ indicates heart left ventricle). (c) Short-axis T2-weighted image shows mild hyperintensity. (d) Steady-state free precession image showing the infiltrative nature of the lesion along the left ventricle. The delayed short-axis enhancement (e) shows a heterogeneous enhancement of the mass. The image is adapted from [47].

Follow-up CMR characteristics:

Myocarditis is a difficult diagnosis to make and there is no clear gold standard in vivo because a negative endomyocardial biopsy result does not rule out the diagnosis. In recent years, abnormal myocardial mechanics, mainly the total longitudinal tension (GLS) of the left ventricle, has been extensively studied for the diagnosis of cardiac dysfunction in various cardiovascular diseases. [54]. In addition, abnormalities in myocardial stress, ejection fraction, and other traditional risk factors are strong predictors of poor outcomes in cardiovascular disease. Initially, most of this work was done using custom CMR pulse sequences that required extensive post-processing. More recently, the development of CMR "feature tracking" technology has enabled the quantification of GLS using standard films, thus eliminating the need for specific pulse sequences. A single-center retrospective investigation tested the predictive power of feature-track-derived GLS in 455 patients with clinical presentation consistent with myocarditis [55]. The results of our CMR feature follow-up corroborate the above-mentioned experience, with a significant reduction in GLS in the LV and RV, with additional reductions in baseline and mean RVCS (right ventricular circumference stress).

Advantages of CMR with echocardiography:

Although echocardiography is the preferred cardiac imaging modality, CT and MRI are probably underutilized due to the potential for increased visibility. and describe the characteristics of the lesion. In addition, imaging results vary widely depending on the substrate of the disease. CMR is a promising technique to identify pericarditis, diagnose, and monitor myocarditis [23,29–31]. MRI is superior to echocardiography in detecting

tuberculous myocarditis, similar to what was reported in the Western European series on myocarditis [28, 56]. MRI also has several advantages over echocardiography in identifying, characterizing, and evaluating purely cardiac abnormalities. These include high contrast resolution, unlimited field of view, the ability to perform multi-plane imaging and characterization, multi-parameter imaging, anamorphic images, and high levels of contrast variation. MRI is probably a more powerful imaging modality in this setting, as disseminated intracardiac TB has been demonstrated using gadolinium enhancers [12, 28, 47, 57, 58].

Role of Tc-99m Sestamibi and SPECT:

Myocardial scintigraphy with Tc-99m pyrophosphate is also useful in myocarditis, especially TB myocarditis when combined with Tc-99m sestamibi. In one case study, SPECT was more effective than CMR in locating biological prosthetic valve abscesses and it may be useful in tuberculosis research [8,59]. Gallium scintigraphy may be a more effective test for the diagnosis of tuberculous myocarditis than indium scintigraphy because it has lower sensitivity for acute inflammation but higher sensitivity for chronic inflammation [59]. Despite the widespread use of Tc-99m sestamibi in various cardiac diseases, its application in the assessment of myocardial viability remains controversial [60].

Role of 18F-FDG PET in the Heart:

Cardiac TB was recently diagnosed using 18F-FDG PET. In their case report, Sundaraiya et al. presented a case of sarcoidosis-like cardiac tuberculosis [61]. PET images show patchy areas of increased FDG uptake in the apex to mid-anterolateral region, anterior septal to basal/right ventricular region, and moderate increases in FDG uptake in CMR findings of myocardium below the LV apex (Figure 7, this image is adapted from Sundaraiya et al [61]). On whole-body PET-CT, multiple hypermetabolic adenomas above and below the diaphragm were detected, but no lung injury was detected. Necrotizing granulomatous inflammation of the left para-aortic lymph node is consistent with tuberculosis [61]. Although tuberculosis closely resembles sarcoidosis, FDG-PET is very useful. FDG-PET has several disadvantages compared with CMR, including non-inflammatory FDG uptake by the myocardium despite good FDG-PET preparation, false-positive results due to atrial fibrillation or bundle branch block, which may affect local glucose utilization and radiation exposure [62].

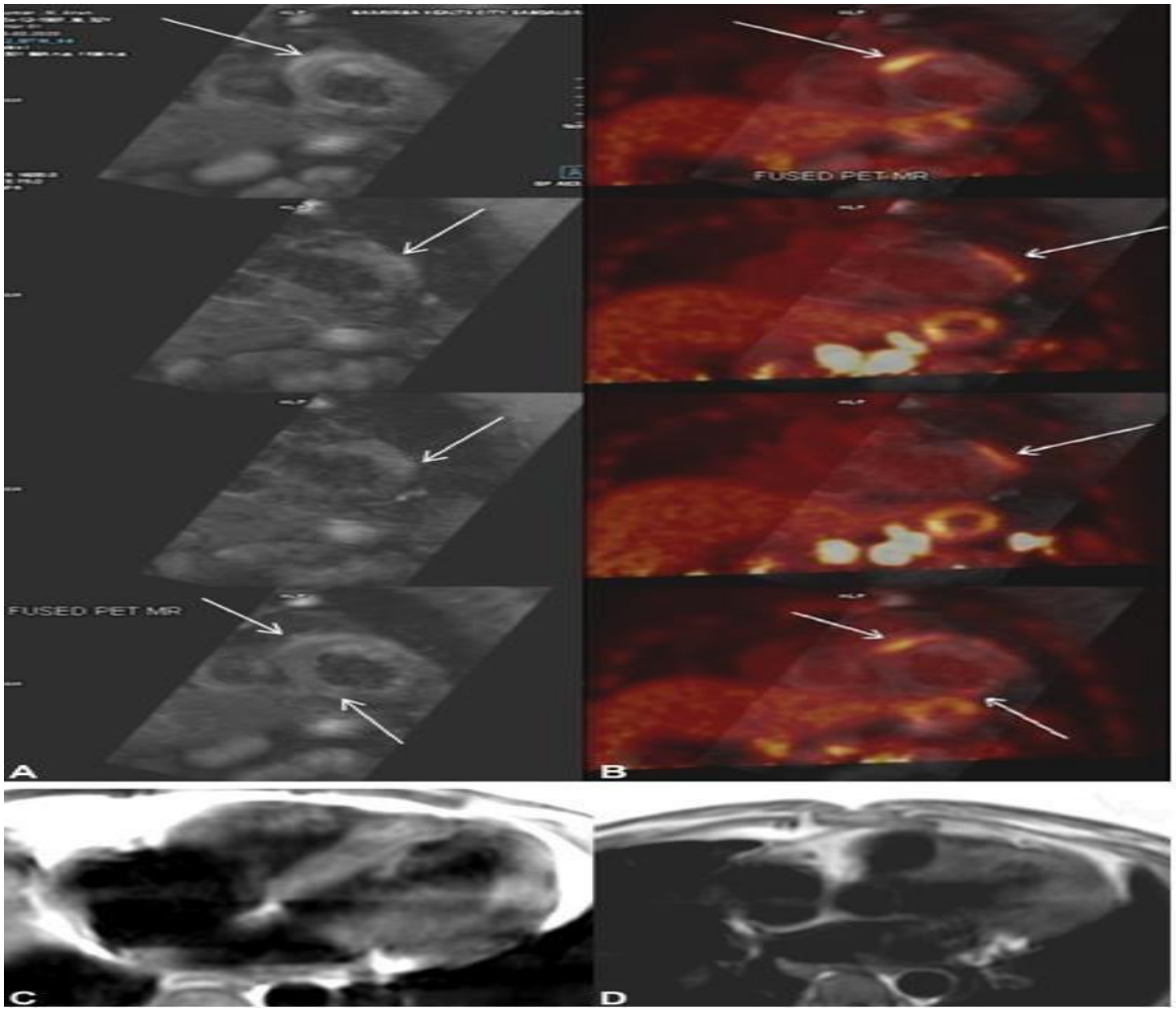


Figure 7. (A) Cardiac Magnetic Resonance (CMR) shows multifocal linear angiography from subepicardial to mid myocardium along the site of right ventricular insertion, anterior anterolateral and inferior medial segments (arrows) with corresponding focal myocardial edema.

(B) CMR fused cardiac positron emission tomography showing patchy areas of F-fluorodeoxyglucose (FDG) hyperabsorption at the apex to mid-anterolateral side, mid-base of the anterior septum at the site of right ventricular intubation, and The slight increase in FDG uptake in the inferior wall of the apical segments of the left ventricular myocardium corresponds to areas of myocardial enhancement seen on CMR. (C,D) Dark blood T2 image shows T2 intensity changes in the left ventricular myocardium. The image is adjusted from [61].

Endomyocardial Biopsy:

Over time, the importance of endocardial biopsy (EMB) in the diagnosis of pathogenic

entities has evolved. In addition to myocarditis, EMB has a low susceptibility rate of 25% for lymphocytic myocarditis and 35% for cardiac sarcoidosis [63]. The application of EMB to tuberculosis has not been discussed previously. Because of its low sensitivity but high specificity, EMB is the diagnostic method of choice for many disorders. Therefore, a high pre-test probability is required. In patients with low pretest probability, it is best to use other tests to rule out pathology. These may include scans (indium-111, gallium-67) [64,65]. The combination of CMR and EMB has a synergistic effect on the diagnosis of myocarditis [66].

Advantages and disadvantages of imaging modalities (MRI, CT, PET):

Transthoracic echocardiography, which combines structural and physiological assessment, is the first-line technique to examine suspected patients. or known to have cardiomyopathy. However, diagnostic accuracy is reduced in those with poor acoustic windows, and transthoracic echocardiography is limited in its ability to help identify localized effusion, assess pericardial thickness, and characterize tissue scores [67,68]. However, cardiac computed tomography (CT), magnetic resonance (MR) and positron emission tomography (PET) are becoming increasingly popular to study this part of the heart to better define the anatomy. and heart function. In recent years, advances in magnetic resonance imaging technology and sequence design have enhanced the diagnostic value of this modality for the assessment of cardiomyopathy in recent years. For example, rapid MRI sequences allow real-time analysis of cardiac movements and inflow patterns during free breathing, helping to reveal the physiology of contraction [32, 69, 70]. In addition, slow (or slow) magnetic resonance imaging with gadolinium injection is useful for diagnosing myocarditis and monitoring the effects of anti-inflammatory drugs [33,71]. In constrictive pericarditis, MRI labeling methods are useful to detect both adhesions of the pericardial layers and cardiac involvement [72]. Intrapericardial content can often be better assessed by dynamic cine-MRI imaging of the internal blood, such as the visibility of fibrin fibers or the presence of clots [73]. The standard imaging method for detecting pericardial effusion is echocardiography. However, small effusions inadvertently located in the posterior or inferior wall cannot be detected on echocardiography [74]. On MRI images, the distinction between pericardial thickening and effusion is often straightforward.

The pericardium appears as a thin fibrous line on CT, but the visceral layer cannot be described independently. Pericardial layers can be accurately represented by CT images, with thickness and content assessed. Accordingly, CT imaging can help physicians distinguish

between primary pericardial effusion and effusion pericarditis or malignant pericardial disease [75]. In addition, pericardial fluid can be characterized to some extent by assessing attenuation values on CT images. Using CT imaging, the increased X-ray absorption of the pericardium helps distinguish the pericardium from surrounding fatty tissues, such as mediastinal fat or the pericardium/pericardium. Notably, a distinct advantage of CT imaging is its ability to accurately and completely identify pericardial calcifications, which can be crucial in differentiating between constrictive pericarditis and pericarditis. limited ventricular filling [75–77]. Currently, computed tomography is the best method to characterize even very small levels of calcium in the pericardium and to see problems such as enlargement in the myocardium of fibrosis, which may impede effectiveness of pericardial resection [75–77]. Preoperative computed tomography can be effective in providing a complete picture of both the degree of thickness as well as the presence and location of calcifications, thus facilitating the planning of calcifications. surgical planning and better risk classification. However, pericardial calcifications are less common today than in the past, which may be due to the decreasing incidence of tuberculosis and the increasing causes of treatment-induced vasospasm. Two recent surveys (74, 90) reported that 27% and 28% of patients with histologically verified constrictive pericarditis had pericardial calcification. Recently there have been reports of the use of CT to identify myocarditis; however, due to intrinsic radiation, it cannot be routinely used for functional studies [78].

Unlike CT and MR images, which are based on anatomical structure and function, PET images reflect the metabolism of 18-fluorodeoxyglucose (FDG) in vivo. PET imaging was used in both strategies to correlate metabolic alterations.

FDG-PET's ability to identify inflammation and different stages of myocardial injury enables early detection of infiltrative cardiac diseases such as such as sarcoidosis and tuberculosis and theoretically improves test sensitivity at the expense of specificity [61,79]. As a diagnostic tool, the increased sensitivity of FDG-PET is crucial for detecting disease at an earlier stage. Inflammatory processes are associated with mild to moderate pericardial FDG uptake and its use in the detection of myocardial tuberculosis is limited [61].

Remaining Challenges:

Regardless of their financial situation, people with cardiovascular disease or at high risk for cardiovascular disease need to be identified early. The application of cardiac imaging

technologies in poor countries faces a number of obstacles [80]. Some of the most powerful medical tools available to identify cardiovascular disease include diagnostic ultrasound, angiography, computed tomography, and nuclear cardiology [81]. However, these services are still lacking in the world. While CMR is commonly used in rich countries, its application in poor countries is inconsistent or non-existent. According to the World Health Organization, although X-rays and ultrasound can solve 70-80% of diagnostic problems, about two-thirds of the world's population does not have access to imaging facilities. image. The importance of ultrasound as a diagnostic modality for the heart is unmatched in many respects. Portable ultrasound has been shown to be useful in the assessment of many different diseases in poor countries and it can also be used to check cardiac function and hemodynamic status in patients like us [82].

Conclusions:

Heart damage is rare in tuberculosis, but can lead to heart failure, constrictive pericarditis, or death; therefore, early detection and management of these complications should be a primary concern. Cardiac TB is a rare disease that can be fatal if left untreated. Expanding access to diagnostic tools, such as laboratory tests (such as ADA or IFN-γ) and imaging, should be a priority. Although echocardiography remains the standard for the diagnosis of myocardial disorders, it remains a useful method in underdeveloped countries due to the inability to access other tests such as CT or CMR. However, techniques that provide better tissue characterization are often needed to aid in diagnosis and treatment. MRI can be used to obtain cardiac function and tissue morphology using a variety of acquisition protocols. CT scans, unlike MRIs, involve the use of radiation. In addition to the function and shape of the heart, a CT scan can also show pericardial calcifications.

Abbreviations:

VT-ventricular tachycardia, CMR-cardiac magnetic resonance imaging, T2w STIR-T2-weighted short tau reversibility, QFT-QuantiFERON-TB Gold, ECV-extracellular volume, tuberculosis-tuberculosis, HIV-deficiency virus human immunodeficiency, AL-frontal, LGE-late gadolinium enhancement, LVOT-left ventricular outflow tract, SPECT-single photon emission computed tomography and T1-spin lattice relaxation time, 18F-FDG PET -18F-fluorodeoxyglucose positron, FDG-fluorodeoxyglucose, PET CT-Positron emission computed tomography-computed tomography, Tc-99m-Technetium-99m, GLS-global

longitudinal stress, RVCS-peripheral stress right ventricle, RV-right ventricle, DNA-deoxyribonucleic acid, PCR-polymerase chain amplification, ADA-adenosine deaminase, IFN-interferon gamma, HIV-human immunodeficiency virus, TB-tuberculosis.

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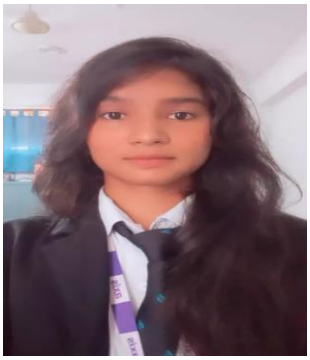	Author Name- Sapna Kumari Author Address- Rajendra Nagar Sikandara Kanpur Dehat