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Diagnostic Role of Cardiac Magnetic Resonance Imaging in Cardiac Sarcoidosis: A Case Report

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Abstract **Case Report**

Sarcoidosis is a granulomatous, multisystemic disease which can affect the heart and is a challenging disease to diagnose and predict. This case report emphasized the importance of multimodality imaging in detecting active Cardiac Sarcoidosis, particularly in a patient who rejected any pharmacologic treatment. The case report involved a 50-year-old female who had a five-year history of systemic sarcoidosis with recurrent palpitations. Physical examination was unremarkable, and Holter monitoring showed premature ventricular contractions with short nonsustained ventricular tachycardia. Echocardiography revealed standard structure and functioning. Monitoring with Fluorine-18 Fluorodeoxyglucose Positron Emission Tomography scan illustrated focal hypermetabolic function in the interventricular septum (SUV max 5.14) and resolution of previous mediastinal lesions. The Magnetic Resonance Imaging of the heart revealed a normal biventricular function, no hypertrophy or late gadolinium enhancement and T1/T2 values at the upper normal limits, indicating mild inflammatory activity. Concordant Positron Emission Tomography and Magnetic Resonance Imaging supported active cardiac sarcoidosis. The case report highlighted the diagnostic utility of combined imaging and the need to follow up such patients who refuse corticosteroid therapy.
Keywords: Sarcoidosis, Cardiac; Positron-Emission Tomography; Magnetic Resonance Imaging; Ventricular Function.

INTRODUCTION

Sarcoidosis is a non-caseating granulomatous disease of unknown etiology characterized by the formation of non-caseating granulomas in the affected tissues[1]. The disease can affect any organ, such as the skin, eyes, liver, spleen, and heart[2]. Nevertheless, the lungs and intrathoracic lymph nodes are the most commonly affected organs [3]. Cardiac Sarcoidosis (CS) is one of the fatal illnesses. CS is a prolonged inflammatory condition marked by non-caseating granulomas that may involve the cardiac tissue[4]. The condition may lead to life-threatening complications such as atrioventricular blocks, ventricular arrhythmias, and sudden cardiac death (SCD)[5].

The prevalence of sarcoidosis is 60 per 100,000. According to the current literature, sarcoidosis is higher in the Midwest and Northeast than in the Southwest United States [6]. It has been estimated that higher rates of sarcoidosis have been reported within the groups of African Americans, Scandinavians, and Japanese. African Americans have been observed to be identified with Sarcoidosis 3 times more than Caucasians, irrespective of geographic area [7, 8]. Clinically, cardiac involvement is evident in approximately 5% of sarcoidosis cases. Moreover, cardiac involvement is subclinical in up to 25-30% of cases, remaining unidentified [9, 10].

Sarcoidosis has an unclear aetiology, but it may be due to an extreme host reaction to some unknown antigen in genetically vulnerable individuals. The condition may develop from environmental factors or Mycobacterium and Propionibacterium acnes infection. T-helper type 1 (Th1) cell activation, macrophage aggregation, and granuloma development afterwards characterize the immune response[11]. This immune-mediated inflammation may involve the myocardium and pericardium in cardiac tissue, leading to patchy fibrosis and scar tissue, which impairs cardiac functioning[12].

People with CS could possess a normal Electrocardiogram (ECG) and a routine echocardiography. Hence, cardiac magnetic resonance imaging (CMR) and fluorine-18 fluorodeoxyglucose positron emission tomography (18F-FDG -PET) are the imaging modalities that detect CS effectively. Combined use increases diagnostic accuracy and could inform management if the software and clinical expertise are present[13].

The diagnosis of CS is a challenge, as most diagnostic methods do not have enough sensitivity. Although endomyocardial biopsy is the gold standard, its sensitivity is below 30% due to the possibility of missing granulomatous lesions[14]. Thus, non-invasive imaging modalities have gained significant importance in diagnosis. Myocardial inflammation and fibrosis can be detected using cardiac magnetic resonance imaging (MRI) with late gadolinium enhancement (LGE), with characteristic patchy, non-ischemic patterns[15]. Therly, the 18F-fluorodeoxyglucose positron emission tomography (FDG-PET) can detect active inflammation and determine disease activity[16]. Integrating MRI with PET imaging increases the accuracy of the diagnosis and assists in monitoring the treatment[17].

The existing literature has focused on the diagnostic value of cardiac MRI and FDG-PET in CS; however, the clinical correlation in patients with limited or no cardiac symptoms and routine echocardiography has not been explored[18]. The concordance of multimodal imaging has also not been well studied to provide direction in diagnosis where there is any ambiguity in conventional criteria.

The current case report intends to emphasize the diagnostic value of concordant CMR/FDG-PET results in identifying early CS, testing with a normal ECG and echocardiography. This report fills this gap in the literature on subclinical manifestations. It highlights the vital role of multimodality imaging in detecting diseases early and characterizing them accurately by revealing how some minor metabolic and structural imaging deviations can prove active disease.

Case Presentation

A 50-year-old female with a known history of sarcoidosis with systemic articular involvement presented with isolated palpitations, prompting a detailed cardiac examination. The patient had no history of diabetes, hypertension, dyslipidemia, or other cardiovascular risk factors, and no significant family history of cardiac disease or autoimmune disorders. Five years after the initial diagnosis, she presented to the cardiology department, Hospital of Marrakech, Morocco with recurrent palpitations throughout the day.

Medical History

The patient had a five-year history of joint inflammation of both shoulders, typically the left shoulder, including the hip and cervical spine. Mediastinal lesions were diagnosed during the initial assessment through PET/CT of 18F-FDG and followed up by the internal medicine department cardiology department, Hospital of Marrakech, Morocco. Although systemic corticosteroid therapy was indicated, the patient declined any pharmacologic therapy. The patient preferred a conservative treatment that entails lifestyle changes, stress level reduction, exercise, and a balanced diet with anti-inflammatory properties. She adheres to her planned annual PET scan follow-ups, demonstrating intermittent inflammatory activity without disease progression.

Clinical and Radiological Examination

After five years had passed since the patient's initial diagnosis, the patient presented to the cardiology department with repeated palpitations that did not appear to be connected to physical activity or emotional stress. The patient described the palpitations as irregular and disturbing. She denied any chest pains, syncope or dyspnea. The physical examination was unremarkable, and the vital signs were stable. A 24-hour Holter ECG revealed an isolated ventricular ectopic beating and short runs of non-sustained ventricular tachycardia (NSVT). Additional examination was performed using multimodality cardiac imaging (MCI), such as echocardiography and CMR, to determine potential myocardial involvement.

Echocardiography and PET Findings

Echocardiography revealed a normal heart structure and functioning. Afterwards, a follow-up positron emission tomography scan (CPET) revealed a focal hypermetabolic region in the interventricular septum, with the highest standardized uptake value (SUV max) of 5.14. Cervical and mediastinal lesions of 2022 had disappeared previously. As hypermetabolic foci were observed in repeated [18 F] FDG-PET, they were proposed to be active CS, associated with premature ventricular contractions (PVCs) and tachycardia episodes.

Cardiac MRI Findings

The cardiac MRI showed normal ventricular volumes and functioning with a left ventricular ejection fraction (LVEF) of 64%, right ventricular ejection fraction of 62%, and a 42 g/m2 myocardial mass. Atria were regular in size with no valvular disease, abnormal wall motion, or left ventricular hypertrophy. By parametric mapping, T1 and T2 were found to be on the higher end of the normal range, and the extracellular volume was in the normal range. There was no myocardial edema or LGE. These MRI results supported active CS with systemic involvement and palpitations, which aligned with PET findings. Please see Figure 1, Figure 2 and Figure 3.

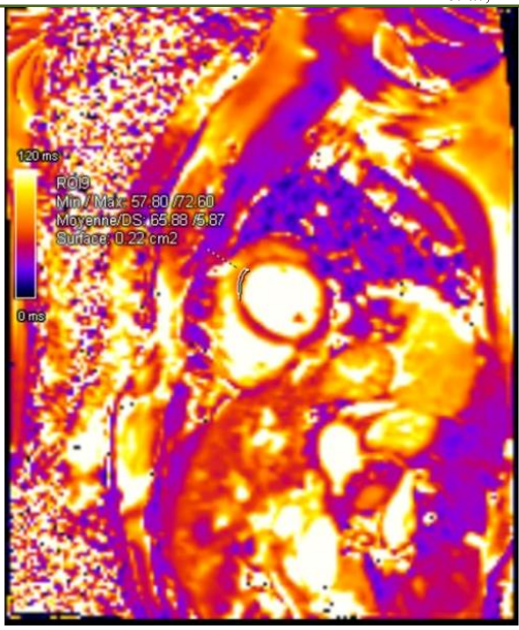

Figure 1: Cardiac MRI – T2 mapping short-axis view showing increased T2 relaxation time (≈65 ms) in the basal-to-mid inferolateral segment, consistent with myocardial edema indicating active inflammation

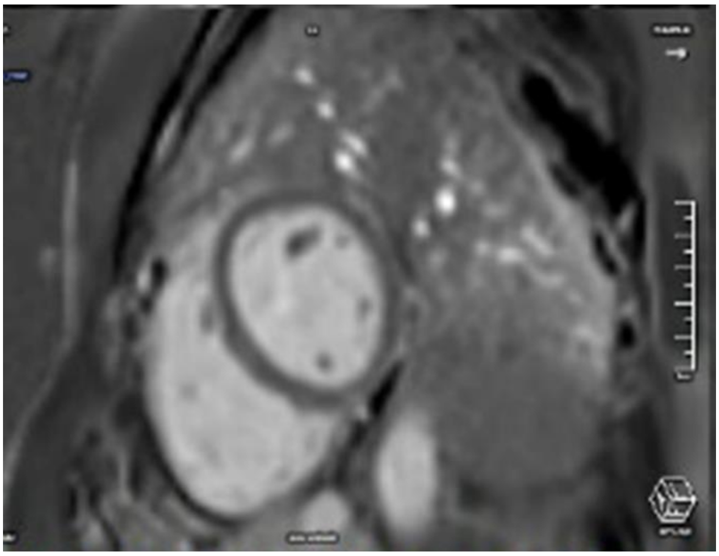

Figure 2: Late gadolinium enhancement (LGE) short axis view showing no LGE à 10 min post contrast

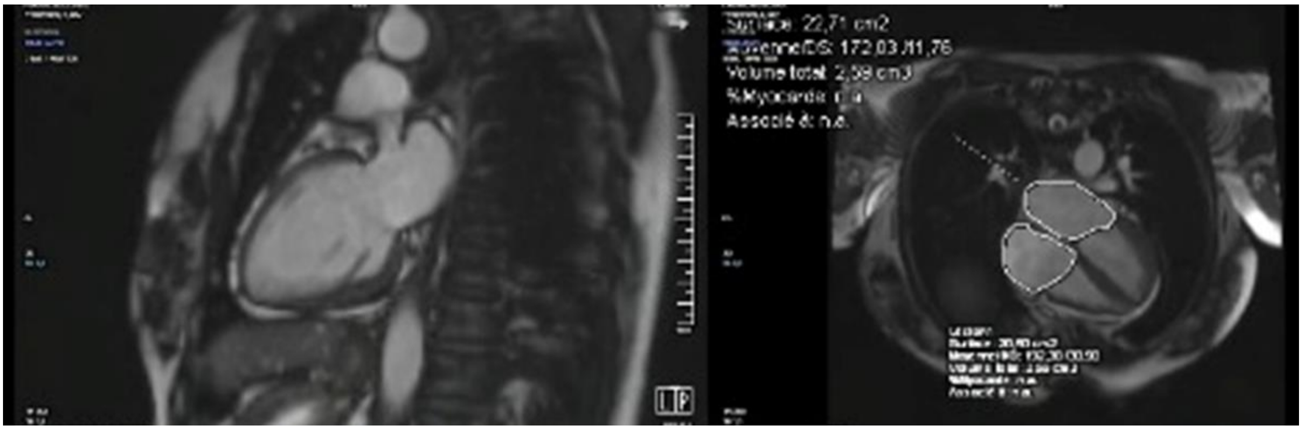

Figure 3: Cine SSFP 4-chamber view et 2 chamber view confirming the absence any regional wall motion abnormality and preserved global systolic function

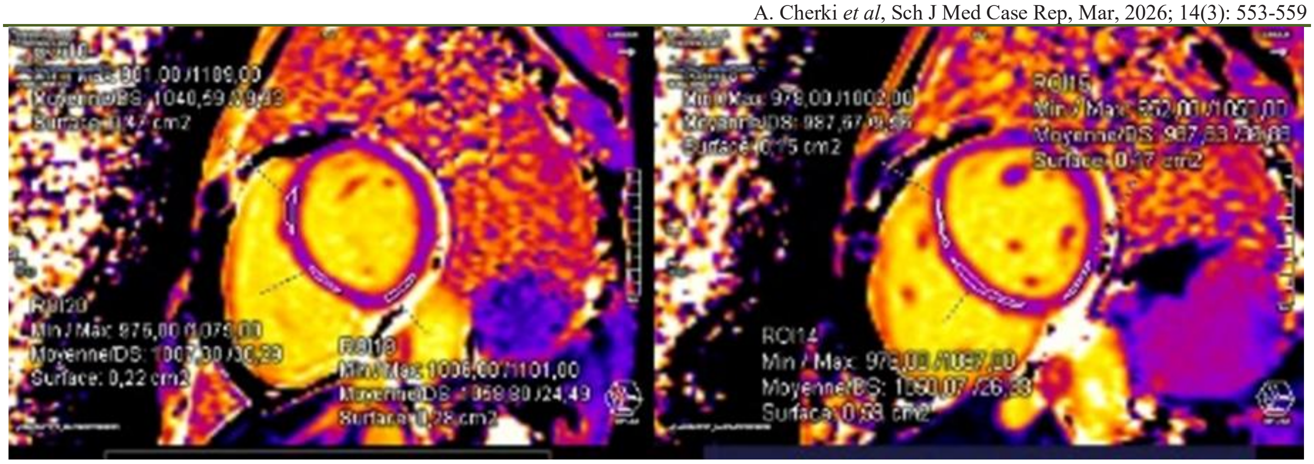

Figure 4: Native T1 mapping demonstrating elevated myocardial T1 values in the basal lateral wall (≈1,090 ms), supporting tissue inflammation and interstitial expansion

Management and Follow-up

The patient refused to start anti-inflammatory pharmacologic therapy, despite the doctor's recommendation. It was stressed that the progression of the disease should be monitored with the help of regular follow-up to minimize the chances of potentially serious cardiac complications.

DISCUSSION

A rare CS with 5% symptomatic involvement remains a major predictor of death[19]. Early detection and proper treatment are essential to reduce adverse cardiac events[20]. Imaging studies are critical in the appropriate diagnosis and effective treatment of CS as they offer a comprehensive and detailed account of the myocardial involvement. Such advanced imaging techniques play a significant role in informing the therapeutic choices and proper patient care, which all translate into improved health outcomes for people with such a condition[21].

ECG is significant in initially evaluating and treating suspected CS patients[22]. It is highly accessible and may reveal gross abnormalities, such as regional wall-motion defects, aneurysms, ventricular dilation, a decreased LVEF or regional strain abnormalities[23]. However, ECG has low sensitivity (≈10–47%) for early CS, and is typically used to assess ventricular function. Strain imaging (speckle-tracking) may detect subtle involvement, but a negative echo cannot rule out CS[24]. For an apprehensive tissue characterization, cardiac MRI (CMR) and ^18F-FDG PET/CT have become the non-invasive gold standards [24]. The standard CS protocols include steady-state cine sequences for function, T2-weighted "black-blood" images for edema, and inversion-recovery LGE imaging ≈10–15 minutes post-contrast. Newer protocols add parametric mapping: native T1 and T2 mapping and extracellular volume (ECV) quantification, which can detect diffuse fibrosis or edema even when LGE is absent[25].

The hallmark CMR finding in CS is patchy LGE in a non-ischemic distribution[26]. LGE in CS is typically subepicardial or mid-myocardial, often in the basal interventricular septum and LV free wall and frequently involves the right ventricular insertion points. In contrast, ischemic scars are subendocardial and follow coronary territories[27]. Basal septal enhancement or thinning "inverted septum" is a classic sign[28]. CMR may also reveal ventricular dilation or aneurysms. According to a meta-analysis, CMR-LGE has ~93–95% sensitivity and ~84–85% specificity for CS[29]. Importantly, the presence and extent of LGE are strongly prognostic and extensive LGE correlates with a higher risk of ventricular arrhythmias, heart failure, and death. However, LGE cannot distinguish active granulomas from chronic scar[30]. T2-weighted imaging can show myocardial edema during active inflammation, but has limited sensitivity. Quantitative T2 mapping is more reliable, and values >50–51 ms at 1.5T, including basal septum, have been associated with active CS[25]. PET is highly sensitive (~84%) and specific (~83%) for CS[31]. FDG-PET is especially valuable for detecting active disease (inflammation) and extracardiac sarcoid (mediastinal nodes, lung lesions), and can guide biopsy targets[32].

CS is typically diagnosed through histology and imaging, according to the Heart Rhythm Society (HRS) 2014 criteria. This requires either histologic evidence of noncaseating granulomas in cardiac tissue or a compatible clinical picture in a patient with proven extracardiac sarcoidosis. The HRS approach requires biopsy-proven sarcoidosis outside the heart for clinical diagnosis[33]. On the contrary, the Japanese Circulation Society (JCS) 2016 guidelines permit a diagnosis of CS without any extracardiac biopsy, using multiple imaging/clinical criteria[34].

While comparing CMR and FDG PET, CMR provides superior spatial resolution and scar visualization with no radiation. In contrast, FDG-PET uniquely images active inflammation and extracardiac involvement. Still, the congruence between the hypermetabolic activity on PET and the T1/T2 hyperintensities on CMR signifies active inflammation and edema, respectively, establishing a strong diagnostic link for CS[35].

The key finding was the concordance between focal FDG uptake and borderline mapping abnormalities in the septum. Although LGE is the reference for detecting myocardial fibrosis, it cannot distinguish active inflammation from chronic scarring, limiting its value in follow-up[36]. T1 and T2 mapping with extracellular volume (ECV) quantification overcome this limitation by providing quantitative biomarkers of myocardial edema and extracellular expansion, enabling detection of both acute and chronic inflammatory changes[37].

Managing CS requires a multidisciplinary approach combining immunosuppression, arrhythmia prevention, and heart failure therapy. Corticosteroids such as prednisone, 30-40 mg/day, tapered to a maintenance dose, would be used as the first-line therapy. Even though relapse is typical, likely, early steroid-sparing treatment with methotrexate, azathioprine, mycophenolate, or TNF-α inhibitors is required. Data is primarily anecdotal, and there is no randomization of dosage. The prognosis is different; a poor prognosis is associated with large-scale myocardial LGE, sustained PET uptake, and high T2 mapping values in CMR.

Prolonged follow-up and surveillance are encouraged, such as periodic clinical assessment, ECG/Holter, and echocolor cardiography after 6-12 months. Recurrent CMR every 1-2 years or in case of clinical change is also recommended, and serial FDG-PET at 3-12 months is useful in response monitoring. The patient in the present case report did not experience NSVT or poor performance. The arrhythmic risk may exist and should be monitored. Her denial of therapy reflects a clinical dilemma and highlights the significance of systematic follow-up with ECG/Holter, repeat PET/CMR and clinical examination.

Strengths and Limitations of the Case Report

This case report underscored the diagnostic efficacy of multimodality imaging, concordant between [18F]FDG-PET and cardiac MRI, to verify an active cardiac sarcoidosis. It has highlighted the significance of longitudinal observation in a patient with a treatment-averse nature, and how the disease activity may vary without pharmacologic support. One of the significant limitations is the single-patient design, which restricts generalizability. Further, the absence of histopathological confirmation and the patient's denial of corticosteroid therapy limited the evaluation of the responsiveness to treatment and prognosis.

Conclusion

Concordant Positron Emission Tomography and subtle mapping abnormalities on Magnetic Resonance Imaging suggested active disease. Follow-up is obligatory in patients who are not willing to start therapy, and the disease may progress to life-threatening arrhythmias or heart failure.

Statements and Declarations

Authorship

M.Z: Conceptualizaton
A.C: Writing-original draft
M.E.H: Validation

Patient Informed Consent Statement

I hereby certify that written informed consent was obtained from the patient for the publication of the medical case and any accompanying images. The patient was informed that all personal identifiers would remain fully anonymous, and that the information would be used strictly for scientific and educational purposes. The patient has given full permission for this material to be published.

Conflict of Interest: None disclosed

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