A new era of imaging for diagnosis and management of cardiac sarcoidosis: Hybrid cardiac magnetic resonance imaging and positron emission tomography

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CASE PRESENTATION

A 49-year-old African American female with lymph node biopsy-proven pulmonary sarcoidosis complicated by skin and eye involvement presented with dyspnea on exertion. The patient was in her usual state of health until 2 years prior to presentation at which time she reported decreasing exercise tolerance. She otherwise denied chest pain, palpitations, paroxysmal nocturnal dyspnea, orthopnea, lower extremity edema or syncope. She was found on transthoracic echocardiogram (TTE) to have severe mitral regurgitation due to bileaflet mitral valve prolapse and subsequently underwent mitral valve repair with relief of symptoms. She continued to do well until 1 year later, she noted recurrent symptoms with worsening edema. Repeat TTE revealed failure of the prior repair with recurrent severe mitral regurgitation and decline in left ventricular (LV) function. She underwent a mitral valve replacement with bioprosthesis (27 mm Edwards Lifesciences pericardial tissue valve, Magna type). Despite successful replacement and guideline-directed medical therapy (GDMT), patient had repeat hospitalizations for heart failure. Of note, cardiac tissue surrounding the excised mitral valve apparatus at time of MV replacement was notable for non-necrotizing granulomatous inflammation without clear identifiable pathogen. The patient was initiated on prednisone 40 mg daily given histological diagnosis consistent with cardiac sarcoidosis. GDMT was further optimized. She was discharged with a life vest as there was evidence of non-sustained ventricular tachycardia (VT) on telemetry monitoring. She presented for a routine follow-up visit.

The patient was on maximally tolerated doses of GDMT including beta-blocker, angiotensin receptor-neprilysin inhibitor, mineralocorticoid antagonist and diuretic. She was also taking prednisone 40 mg daily, Vitamin D/Calcium supplementation, proton-pump inhibitor and single-dose trimethoprim sulfa daily. She had no known drug allergies. Family history was unknown as the patient was adopted. She was a five pack-year former smoker. She denied alcohol or illicit drug use.

The patient's vital signs were in normal range with blood pressure 118/83, heart rate 64 beats per minute (bpm), and 98% oxygen saturation on room air. Her physical examination was notable for mildly elevated jugular venous pressure with prominent v-wave and II/VI holosystolic murmur at right sternal border.

On review of laboratory data, patient had mild leukocytosis with white blood cell count 13.8 $\times$ 10^9 cells per liter with a normocytic anemia with a hemoglobin of 10.3 g/dL and normal platelet count. Her creatinine was 1.0 mg/dL. NT-proBNP was elevated at 1300 pg/mL and ACE (angiotensin converting enzyme) level was elevated at 138 U/L (normal 9 to 67 U/L). Liver tests and pulmonary function tests were normal. She had normal inflammatory markers, calcium level and 1,25-OH vitamin D levels.

Electrocardiogram (ECG) showed normal sinus rhythm at a heart rate 75 bpm, first-degree

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atrioventricular block and left bundle branch block. Computed tomography (CT) chest imaging was notable for mediastinal adenopathy. TTE revealed normal LV end-diastolic diameter, paradoxical septal motion due to bundle branch block with severely reduced LV function 20% to 25% (Figure 1, Supplementary Video 1). The right ventricle was normal in size and function with moderate tricuspid regurgitation and severe pulmonary hypertension (pulmonary artery systolic pressure of 60 mm Hg). The bioprosthesis was visualized in the mitral valve position with a peak velocity 1.8 m/s and mean gradient of 5 mm Hg at a heart rate 91 bpm and trace mitral regurgitation consistent with normal bioprosthetic function. Coronary angiography prior to her MV operation revealed angiographically normal coronaries. There were no shock therapies delivered by the Lifevest and no further evidence of non-sustained ventricular tachycardia (VT) on medical therapy.

Prior to a 2-week follow-up visit, diuretics were augmented and GDMT was further uptitrated. Patient underwent a hybrid cardiac magnetic resonance imaging (CMR)—positron emission tomography (PET) protocol study to determine course of immunosuppressive regimen and help risk-stratify the patient. The CMR showed normal LV size with moderately decreased LV systolic function 35% with global hypokinesis with mild basal septal hypertrophy. On delayed-contrast enhanced images, there were no areas of late gadolinium enhancement to suggest myocardial infarction, inflammation, or fibrosis (Figure 2).

Concomitant ^{18}F-FDG PET (Figure 3) showed prominent uptake in the basal anteroseptal, anterolateral and inferolateral walls. FDG uptake was also seen in bilateral hilar and mediastinal lymph nodes. These findings were most consistent with active sarcoidosis involving the ventricular myocardium and thoracic lymph nodes.

CARDIAC SARCOIDOSIS

Cardiac sarcoidosis is a multi-system disease characterized by non-necrotizing granulomatous inflammation most often associated with lymph node and pulmonary involvement. The annual incidence is reported as 10.9 per 100,000 in whites and 35.5 per 100,000 in African Americans and occurs generally between the ages of 25 and 60 years of age.[1] More than 25% of patients with sarcoidosis may have cardiac involvement, of whom ~ 5% become clinically relevant.[1] Clinical manifestations include atrial and ventricular arrhythmias, conduction disease, valvular dysfunction (most often mitral), LV dysfunction or heart failure, and/or sudden cardiac death, which can be seen in up to 25% as an initial clinical presentation. Diagnosis remains challenging as there is no clinical 'gold standard' and endomyocardial biopsy has low diagnostic yield (~ 25%).[2] Diagnosis of cardiac sarcoidosis is currently made based on the 2006 Japanese Ministry of Health and Welfare (JMHW) criteria and/or 2014 HRS Consensus

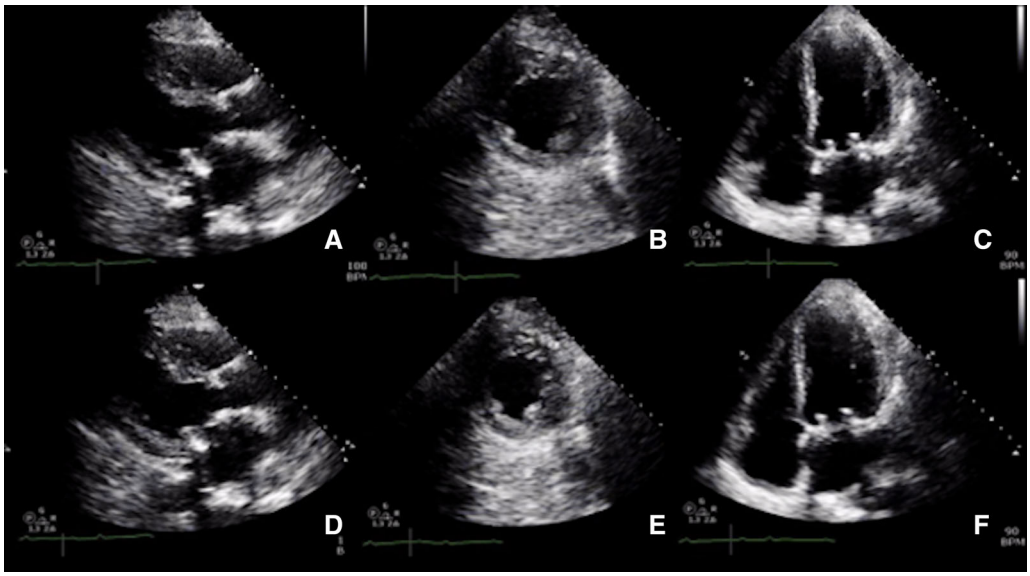


Figure 1. Transthoracic echocardiogram end-diastolic and end-systolic frames of parasternal long-axis (**A**, **D**), short-axis (**B**, **E**) and apical 4-chamber (**C**, **F**) views. The left ventricle was non-dilated with severely reduced left ventricular function. The right ventricle was normal in size and function. The mitral valve bioprosthesis was normal in function without mitral regurgitation.

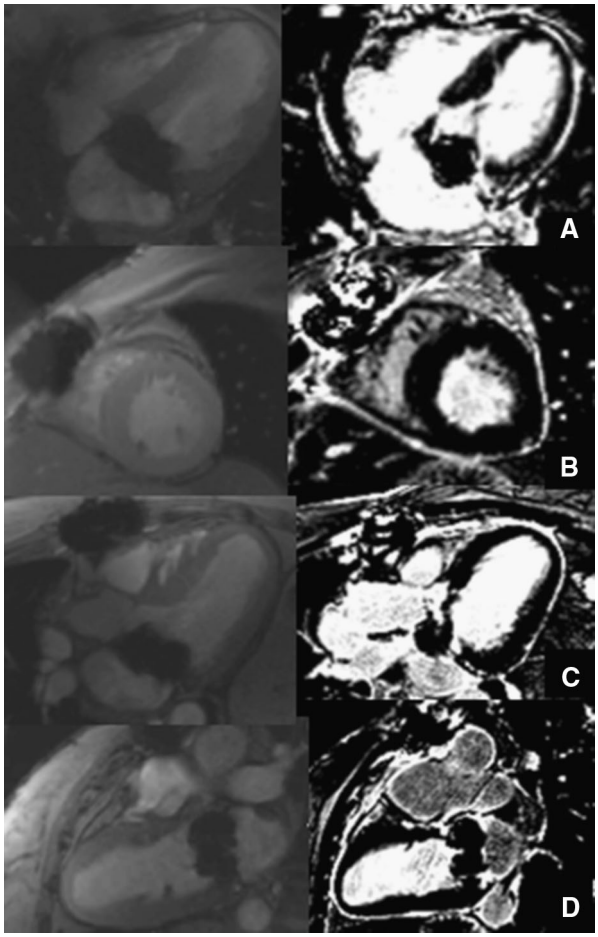


Figure 2. Cardiac magnetic resonance imaging diastolic still cine and delayed-contrast images 4-chamber (**A**), short-axis (**B**), long-axis frames (**C**, **D**). The left ventricle was normal in size with moderate-severely reduced systolic function. Paradoxical septal motion was seen with LBBB and post cardiac surgery, otherwise the wall motion was globally hypokinetic. There was significant local susceptibility artifact due to bioprosthetic mitral valve. There was no significant mitral regurgitation. The right ventricle was normal size and systolic function. On delayed-contrast images, there were no areas of late gadolinium enhancement to suggest myocardial inflammation or fibrosis.

Guidelines[3] and requires histological diagnosis of either myocardial tissue or extra-cardiac tissue along with other clinical diagnostic criteria (Table 1). Among patients with extra-cardiac sarcoidosis, screening for cardiac sarcoidosis involves thorough history and physical examination, ECG and transthoracic echocardiogram followed by imaging with CMR and/or PET if suspicion for cardiac sarcoidosis remains high. Holter monitor may be helpful in patients for further risk stratification; however, the HRS consensus writing committee did not make a formal recommendation for screening for cardiac involvement with a Holter monitor.

WHAT IS THE ROLE OF IMMUNOSUPPRESSION IN TREATMENT OF CARDIAC SARCOIDOSIS?

Corticosteroid therapy has been the mainstay therapy for treatment of active sarcoidosis. Observational data has shown that early initiation of steroids in heart failure may be associated with improved LV systolic function and may decrease the overall burden of ventricular arrhythmias and atrioventricular block.[4] There is additional observational data that suggests that long-term maintenance therapy with steroids may preserve LV function and prevent cardiac death.[5] Use of steroid-sparing agents has also been proposed,[6] methotrexate (MTX) being the most studied. Alternative agents such as mycophenolate, azathioprine, cyclophosphamide, infliximab, adalimumab, and rituximab have also been used in small case studies. On the contrary, a recent retrospective study showed no difference between treatment groups, including those not receiving any immunosuppression, in incidence of a composite endpoint of ventricular assist device placement, transplant or death during a follow-up period of 44 months,[7] though this is biased by its retrospective nature. Regardless, overall event rates for ventricular arrhythmias and heart failure were high, and patients who presented with cardiomyopathy had a worse survival. One promising effort to address the lingering question of optimal immunomodulatory regimen in these patients is the Cardiac Sarcoidosis Multi-Center Prospective Cohort (CHASM-CS NCT01477359), a registry of sarcoidosis patients with and without cardiac involvement to better understand current diagnostic approaches, management and treatment of cardiac sarcoidosis.

In addition, guideline-directed medical therapy should be implemented in patients with LV dysfunction and recommendations on implantable cardiac defibrillator (ICD) is referenced in the 2014 HRS Expert Consensus Statement on ICD implant in cardiac sarcoidosis.[3]

In this case, the patient continued prednisone therapy and was initiated on methotrexate as a steroid-sparing agent. Figure 6 outlines a proposed clinical protocol for treatment for cardiac sarcoidosis that has been implemented at our institution.

WHAT IS THE ROLE OF CARDIAC MAGNETIC RESONANCE IMAGING?

CMR aids in the diagnosis of cardiac sarcoidosis and provides prognostic information in determining adverse cardiac events. The sensitivity in diagnosing

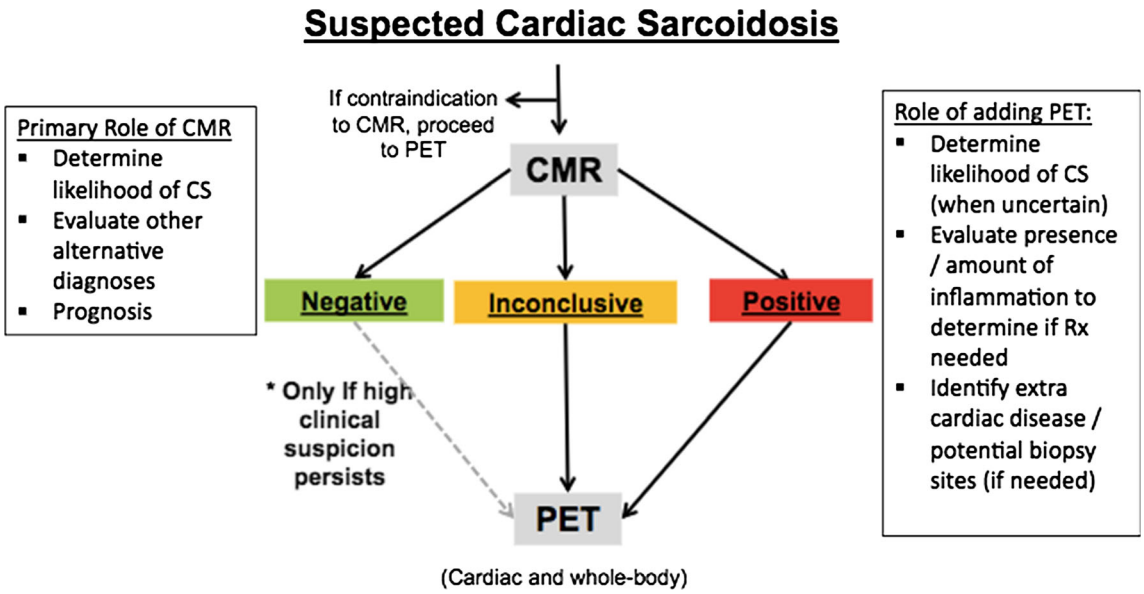


Figure 3. Current diagnostic algorithm with multi-modality imaging in work-up for suspected cardiac sarcoidosis. Reproduced with permission from Bravo et al. JNC 2018[13].

cardiac sarcoidosis ranges from 76 to 100% and specificity 78% to 92%.[1] The pattern of late gadolinium enhancement (LGE) seen in cardiac sarcoidosis can be non-specific, representative of myocardial fibrosis or inflammation. The most common segments involved include the LV basal septum and basal lateral walls in the mid-myocardium and epicardium. T2-weighted imaging can be helpful in detecting edema and active inflammation; however, the absence of T2-weighted signal does not rule out cardiac sarcoidosis. LGE is associated with cardiovascular death and ventricular arrhythmia.[8] Large extent LGE defined by LGE mass $>20\%$ LV mass compared to small-extent LGE has been associated with worse combined adverse outcomes including cardiac death, hospitalization for heart failure, and life-threatening arrhythmias.[9] The extent of LGE may also help predict corticosteroid responsiveness.[9] In this case, CMR was reassuring as a prognostic marker for absence of LGE.

WHAT IS THE ROLE OF CARDIAC POSITRON EMISSION TOMOGRAPHY?

^{18}F-FDG is a glucose analog that helps detect active inflammation due to high metabolic glucose utilization by activated macrophages that can suggest active cardiac sarcoidosis. The sensitivity of PET to detect active sarcoidosis is reported as 89% with a specificity of 78%,[10] though the diagnostic accuracy of the test relies on adherence to a strict pre-test diet of high fat/very low to absent carbohydrate intake.[11] Cardiac FDG uptake pattern is generally characterized as: 1. No FDG uptake, 2. Diffuse FDG uptake, 3. Focal FDG uptake, 4. Focal on diffuse FDG uptake.[12] 'Focal' or 'focal on diffuse' FDG pattern is abnormal and may be consistent with cardiac inflammation from sarcoidosis. Diffuse uptake may be more non-specific, and can represent either inadequate suppression of normal myocardial glucose uptake or possibly multiple sarcoid granulomas with heterogeneous FDG uptake in a diffuse distribution.[12]

^{18}F-FDG-PET is routinely recommended if CMR is abnormal. If CMR is negative yet high clinical suspicion remains for cardiac sarcoidosis, PET should be obtained.[13] (Figure 3). Serial PET imaging has been used to help assess response to and guide titration of immunosuppressive therapy, particularly advantageous in patients who have an ICD implant that may limit the use of CMR.[12] In this patient, ^{18}F-FDG-PET noted focal FDG uptake despite the absence of LGE on CMR imaging. Prominent FDG uptake was observed in the basal anteroseptum and entire lateral wall, along with uptake in hilar and mediastinal lymph nodes consistent with the diagnosis of active sarcoidosis (Figure 5A).

INTEGRATING IMAGING MODALITIES: HYBRID CMR AND PET

CMR and PET evaluate different stages of the pathological disease process. Fibrosis detected by LGE on CMR likely represents a later stage of disease involvement with or without ongoing inflammation,

Table 1. Diagnostic criteria based on 2006 JMHW and HRS[7]. Reproduced with permission from Chareonthaitawee et al. JNC 2017[18]

JMHW	HRS
Histologic diagnosis group	Histologic diagnosis from myocardial tissue
CS confirmed by EMB, and histologic or clinical diagnosis of extraCS	Noncaseating granuloma on EMB with no alternative cause identified
Clinical diagnosis group	Clinical diagnosis
Histologic or clinical diagnosis of extraCS and	Probable diagnosis of CS exists if
Two or more major criteria or	There is histologic diagnosis of extraCS* and
One major criterion and two or more minor criteria	One or more of the following is present:
Major criteria	Cardiomyopathy or atrioventricular block responsive to immunosuppressive treatment*
Advanced atrioventricular block	Unexplained reduced LVEF (< 40%)
Basal thinning of intraventricular septum	Unexplained ventricular tachycardia
^{67}Ga uptake in heart	Mobitz II second- or third-degree heart block
Depressed LVEF (< 50%)	Patchy ^{18}F-FDG uptake on cardiac PET consistent with CS*
Minor criteria	Late gadolinium enhancement on cardiac MRI consistent with CS
Electrocardiography: ventricular tachycardia, PVCs, RBBB, abnormal axis, abnormal Q wave	Cardiac ^{67}Ga uptake and
Echocardiography: structural or wall motion abnormality	Exclusion of other causes of cardiac manifestations
Nuclear medicine: perfusion defect, ^{201}Tl, ^{99m}Tc*	
Cardiac MRI: late gadolinium enhancement	
EMB: moderate fibrosis or monocyte infiltration	

CS, cardiac sarcoidosis; *EMB*, endomyocardial biopsy; *LVEF*, left ventricular ejection fraction; *PVC*, premature ventricular contraction; *RBBB*, right bundle branch block, *MRI*, magnetic resonance imaging
*Notes a difference between JMHW and HRS guidelines

whereas uptake on ^{18}F-FDG-PET represents active inflammation and potential for ongoing damage to the myocardium. Thus, the complementary value of hybrid PET/MRI has been proposed to give insight on both disease involvement in the past and prognosis, as well as active disease that necessitates therapy. More recent studies have incorporated the use of hybrid imaging with PET/MRI into clinical practice to diagnose active sarcoidosis and guide patient management. In addition, the concurrent use of myocardial perfusion imaging (such as with N-13) can provide additional supplemental information in adjudicating active inflammation vs. scar.[12] FDG uptake and rest perfusion imaging patterns and staging system have been described for interpretation of normal variants, early and later stages of the disease.[12] The presence of perfusion defects and abnormal FDG uptake has been associated with increased risk of death or VT.[14]

In one retrospective study, patients were classified based on likelihood of CS based on predefined criteria and a final diagnosis was made after using clinical, imaging and pathological data. Among the patients who received CMR along with PET, 45% of patients were re-classified as having a higher or lower likelihood of CS which also led to changes in immunosuppressive regimen.[15] Another study aimed to assess the diagnostic and prognostic utility of simultaneous hybrid cardiac PET/MR that included 51 consecutive patients with suspected CS based on the JMHW guidelines. Abnormal findings on both PET and CMR was the strongest predictor of major adverse cardiac events including composite of death, aborted sudden cardiac death, sustained ventricular arrhythmia, complete heart block, and hospital admission with decompensated heart failure.[16] The overall sensitivity of the hybrid CMR and PET was 94% in diagnosis of active sarcoidosis, which is the highest reported in the literature.

In this case, the patient had no evidence of LGE on CMR, yet had a positive PET scan that was very convincing for active disease. In the study mentioned above, the overall survival in patients with PET positive CMR negative was lower than those with normal combined CMR/PET,[14] suggesting that absence of LGE on MRI alone may not completely exclude active sarcoid involvement in the myocardium, especially earlier in disease course.

In another study of 25 patients diagnosed with probable cardiac sarcoidosis by JMHW criteria who underwent hybrid CMR/PET, a total of eight patients had a negative CMR and positive PET with either diffuse or focal on diffuse FDG uptake pattern.[17] However, all eight patients were deemed to have likely ''false-positive'' results for active sarcoidosis, although early active CS not yet visible on CMR could not be ruled out.

CASE FOLLOW-UP

The patient had a 6-month follow-up hybrid CMR and PET protocol that revealed marked interval decrease in intensity and extent of uptake in the basal septum, mid and distal inferior, and basal anterior wall (Figure 5B). There was no evidence of any new foci of increased myocardial uptake and the LVEF was unchanged. N-13 ammonia perfusion images demonstrated a large, moderate perfusion defect involving the apex, anteroseptal and anterior wall (Figure 6). Mediastinal adenopathy and lung changes consistent with her diagnosis of sarcoid were visualized with somewhat decreased intensity and extent of uptake compared to prior imaging.

Figure 5. A Baseline CMR and cardiac PET with prominent ▶ FDG uptake in the basal anteroseptal wall (red arrow), anterolateral (blue arrow), and inferolateral walls representative of active myocardial sarcoidosis before treatment. **B** On treatment, repeat study demonstrated marked interval decrease in intensity and extent of uptake in the basal septum, mid and basal inferior wall (yellow arrow), and basal anterior wall (red arrow) without evidence of any new foci of increased myocardial uptake.

CONCLUSION

This case highlights a patient with mitral valve disease and new onset heart failure due to non-ischemic cardiomyopathy who was diagnosed with active cardiac sarcoidosis confirmed by biopsy and PET despite negative CMR. The diagnosis of cardiac sarcoidosis can be particularly challenging given the lack of clinical gold standard due to low diagnostic yield with endomyocardial biopsy. Initial evaluation with CMR is standard

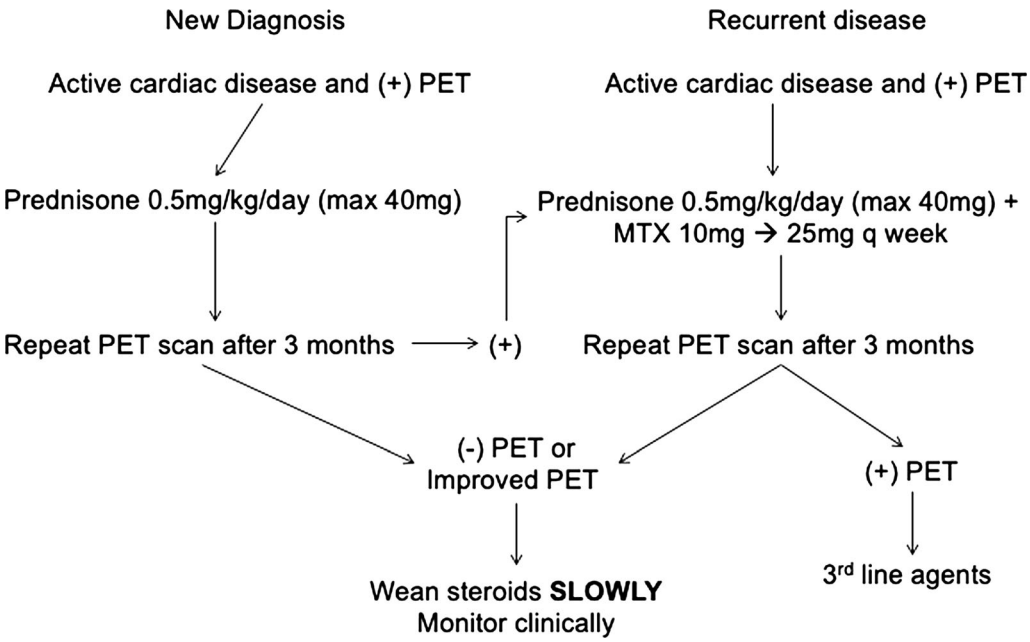
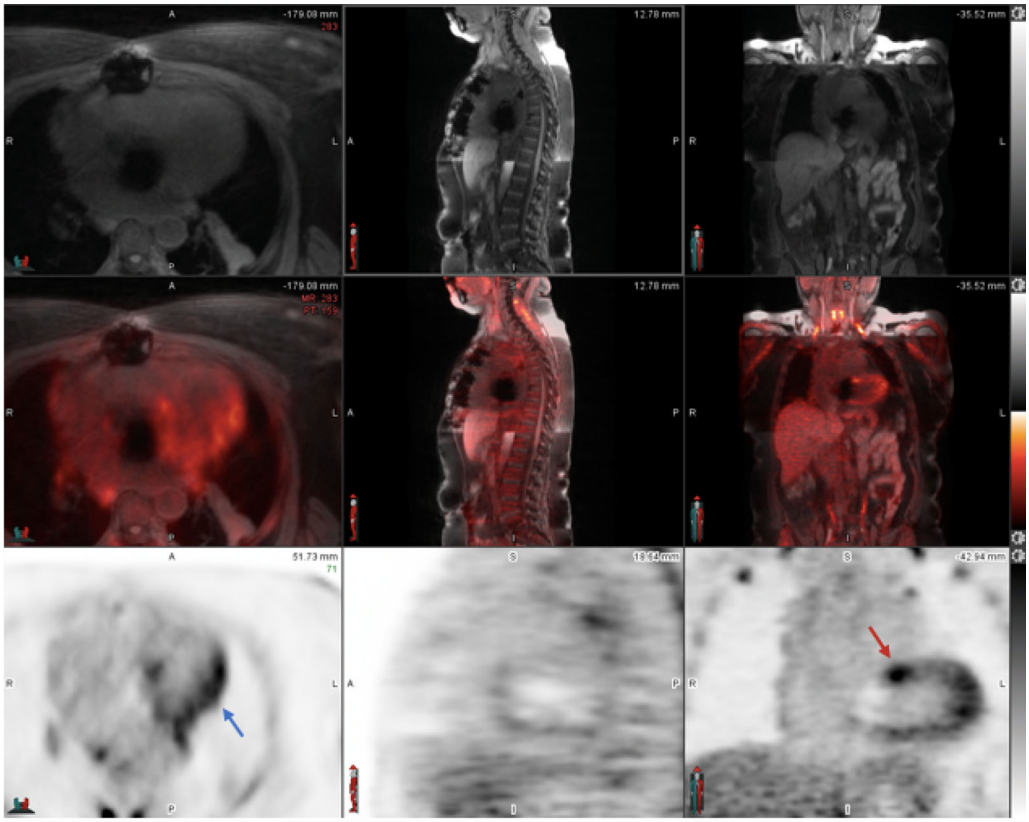
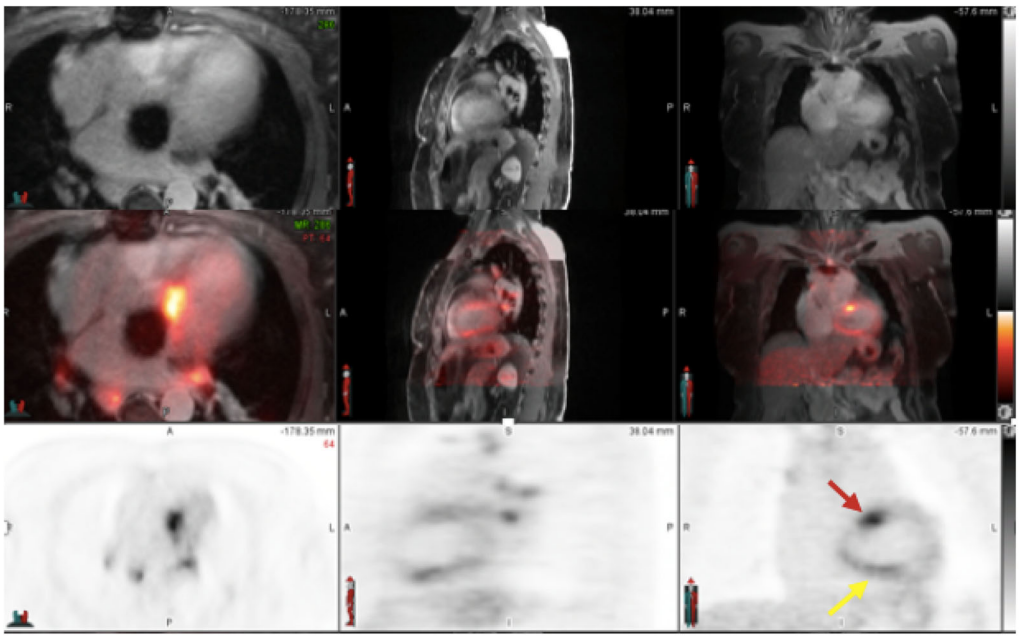


Figure 4. Proposed Clinical Protocol for Treatment of Cardiac Sarcoidosis. *MTX*, methotrexate; *PET*, positron emission tomography.



Before treatment



On treatment
(prednisone + MTX)

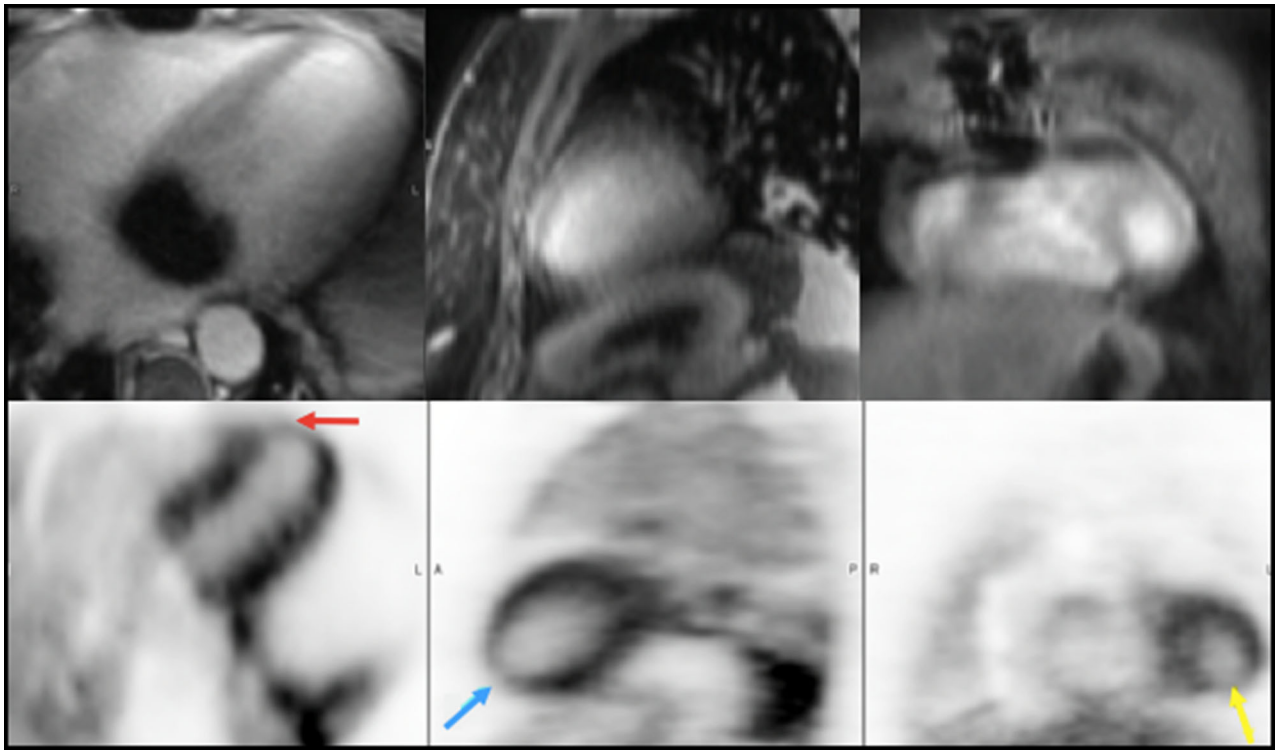


Figure 6. N-13 ammonia perfusion images demonstrated a large, moderate perfusion defect involving the apical septum (red arrow), apex (blue arrow) and apical inferior (yellow arrow) walls.

and has high diagnostic accuracy. Evidence confirms that CMR also provides prognostic value for patients with suspected cardiac sarcoidosis. Although absence of myocardial LGE on CMR may reduce the likelihood of cardiac sarcoidosis, there are instances in which this may not be accurate (such us early, active disease). Thus, if clinical suspicion for cardiac sarcoidosis remains high despite normal CMR findings, a patient should be recommended to undergo FDG-PET (Figure 3) as illustrated by this patient who had a positive endomyocardial biopsy, negative CMR, and positive PET result. There is new evidence to support of hybrid CMR/FDG PET with perfusion imaging in patients with high clinical suspicion for cardiac sarcoidosis with increased sensitivity for diagnosis up to 94%, which is the highest reported in the literature. FDG-PET and CMR combined offers complementary information on disease pathophysiology, diagnosis and prognosis, further advancing the field of cardiac sarcoidosis.

Disclosure

Monica Ahluwalia, Stephen Pan, Munir Ghesani and Lawrence Phillips declare that they have no conflict of interest.

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