REVIEW

Review of Multimodal Imaging in Cardiac Sarcoidosis

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Abstract : Sarcoidosis is a multisystemic disease that causes organ dysfunction through the formation of non-caseating granulomas. Cardiac sarcoidosis (CS) is a phenotype associated with a poor prognosis and benefits from timely diagnosis and management. A nonspecific clinical presentation and the lack of a reliable diagnostic gold standard make obtaining a definitive diagnosis challenging. Advanced cardiac imaging techniques, such as cardiac magnetic resonance imaging (CMR) and ^{18}F-labeled fluorodeoxyglucose (^{18}F-FDG)-positron emission tomography (PET), play an essential role in CS assessment and have been incorporated into several diagnostic guidelines. These modalities have significantly improved our knowledge and understanding of CS by contributing to risk stratification and the assessment of inflammatory and therapeutic response monitoring. The integration of hybrid imaging techniques, such as PET/CMR and PET/computed tomography (PET/CT), has also demonstrated potential in enhancing diagnostic accuracy and disease staging. Each modality offers complementary insights, and their integration through multimodal and hybrid imaging improves diagnostic confidence, disease staging, and therapy monitoring. This review synthesizes current evidence and illustrative cases to highlight the clinical utility of multimodal imaging in CS and discusses limitations and emerging tools guiding future directions. J. Med. Invest. 72 : 225-234, August, 2025

Keywords **:** *cardiac sarcoidosis, ^{18}F- fluorodeoxyglucose-positron emission tomography, cardiac magnetic resonance imaging, positron emission tomography/magnetic resonance imaging*

INTRODUCTION

Cardiac sarcoidosis (CS) is a potentially life-threatening manifestation of systemic sarcoidosis, an inflammatory granulomatous disease (1). While sarcoidosis commonly manifests in the lungs, clinically evident cardiac involvement is reported in fewer than 10% of cases, (2) with a higher prevalence observed among the Japanese, Scandinavian, and African American populations (2, 3). CS is often subclinical, and post-mortem studies have revealed cardiac involvement in 20–58% of patients with sarcoidosis, suggesting that CS is frequently underdiagnosed (4, 5).

Affected patients are typically between 20 and 60 years of age ; the peak incidence among women is between 20 and 40 years of age (2, 5). CS is associated with conduction disturbances, ventricular arrythmias, progressive heart failure and sudden cardiac death (1, 4). Its burden is amplified by diagnostic uncertainty, as patchy myocardial involvement and non-specific symptoms often delay recognition (6-9). Early detection and treatment are critical, as immunosuppressive therapy can improve cardiac function and reduce arrhythmic risk when initiated promptly (1, 10, 11).

While histologic confirmation via endomyocardial biopsy is considered the definitive diagnostic standard for CS, its clinical utility is limited. The sensitivity of EMB is estimated at 20-30%, primarily due to the multi-focal and non-uniform distribution of granulomatous inflammation in the myocardium and the challenge of sampling affected regions (9-11). This limitation has prompted a paradigm shift toward non-invasive imaging as the central diagnostic tool in CS.

In response, major clinical societies have introduced diagnostic frameworks that incorporate imaging findings alongside or in place of biopsy. The Japanese Circulation Society (JCS), the Heart Rhythm Society (HRS) , the World Association of Sarcoidosis and Other Granulomatous Disorders Sarcoidosis Organ criteria, and the American Heart Association (AHA) (9-12) all recognize cardiac magnetic resonance (CMR) and ^{18}F-fluorodeoxyglucose positron emission tomography (^{18}F-FDG-PET) as major diagnostic criteria for CS. Gallium- 67 citrate (^{67}Ga) scintigraphy and myocardial perfusion imaging with SPECT also remain in use in Japan and other resource- limited settings (13, 14). Despite these developments, no unified international gold standard exists, and diagnostic criteria vary across societies and regions. Given the complexity of CS and the limitations of individual modalities, there is a growing consensus that multimodal imaging, whether sequential or integrated, is essential to improve diagnostic certainty, monitor therapeutic response and guide prognosis (3, 9, 15).

This review aims to map the current practices and developments of multimodal approaches for cardiac sarcoidosis imaging, by contextualizing both literature and institutional case examples.

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PATHOLOGY RELEVANT TO CARDIAC IMAGING

CS is thought to result from a dysregulated immune response in genetically predisposed individuals leading to granulomatous inflammation driven by activated T-helper cells and macrophages (1). It is a dynamic disease that progresses through overlapping phases, from early granulomatous inflammation to mechanical dysfunction and conduction instability. This pathologic progression is rarely linear or spatially uniform (7, 16-18).

In the early phase, granulomas infiltrate the myocardium, particularly the basal septum and lateral wall which are areas critical for conduction and contractile function. These lesions may arise through hematogenous spread or direct extension from mediastinal lymph nodes and are associated with increased glucose metabolism and tissue edema (1, 17). ^{18}F- FDG PET is useful in detecting inflammation during this phase, owing to the affinity of mononuclear cells for ^{18}F-FDG as a glucose analog, especially when suppression protocols minimize background or physiological myocardial uptake. T2-weighted or mapping sequences on CMR can also reflect myocardial edema, although their specificity is limited (18-20).

As the disease advances, inflammation may resolve or evolve into fibrosis. Granulomas become compact and fibrotic, eventually forming dense scar tissue (1). CMR with late gadolinium enhancement (LGE) is highly sensitive for detecting fibrotic changes, typically in mid-myocardial or subepicardial layers, and frequently involving the basal septum and inferolateral wall (20). Concomitant hypoperfusion can occur as microvascular inflammation and edema progress. Myocardial perfusion imaging (MPI) using SPECT or PET tracers such as rubidium-82 or N-13-ammonia can identify microvascular dysfunction (16, 21). Dual tracer SPECT may show mismatch patterns in early dysfunction and matched reductions in scarred myocardium (21, 22).

Granulomatous lesions may involve all myocardial layers but are most seen in the mid-and/or epicardial layers in a patchy distribution, which contributes to the low sensitivity of EMB (1). Inflammation and fibrosis often coexist, and new inflammatory activity may emerge near fibrotic areas (3, 23). These complex transitions limit the reliability of any single imaging modality and support the need for a multimodal approach to improve diagnostic precision, guide therapy and monitor disease evolution (16). A visual representation of these overlapping stages and their imaging correlations is presented in Figure 1.

OVERVIEW OF MULTIMODAL CARDIAC IMAGING

CS requires a combination of imaging techniques to assess its distinct inflammatory and fibrotic phases. The JCS 2016 guidelines formally incorporate FDG-PET, CMR, and perfusion imaging as diagnostic criteria (Table 1), reflecting their distinct and complementary roles. The following sections examine each core modality in detail, supported by representative clinical cases.

^{18}F-FDG PET

^{18}F-FDG PET has become an essential for detecting active myocardial inflammation in CS (18). It is particularly valuable in patients with implantable cardiac devices who cannot undergo CMR and offers whole-body imaging to assess extracardiac involvement (24). The tracer is taken up by metabolically active immune cells, especially macrophages, with areas of increase uptake indicating inflammation. Uptake patterns in CS are typically focal or focal-on-diffuse. The "focal" pattern is more commonly associated with CS than physiologic uptake, especially in isolated cases, whereas "focal-on-diffuse" patterns may overlap with normal variants and require cautious interpretation and correlation with perfusion imaging (25, 26). Perfusion-metabolism mismatch using combined rest MPI further supports diagnosis.

Quantitative parameters such as SUVmax provide additional insight. Higher SUVmax values are associated with regions of LGE on CMR, indicating inflammatory activity overlying

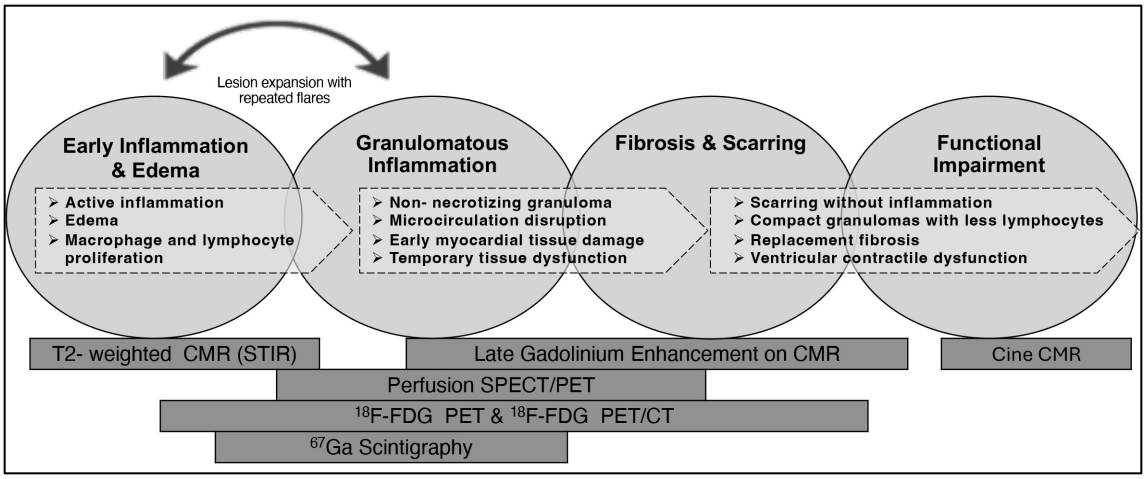

Figure 1. Temporal and Pathophysiologic Correlation of Imaging Modalities in CS
This figure illustrates the pathological progression of CS, from early inflammation and edema to fibrosis and functional impairment, along with their corresponding features and applicable imaging modalities. Different imaging modalities target specific disease processes : ^{18}F FDG-PET and PET/CT detect active inflammation (3, 56), CMR with T2-weighted STIR identifies myocardial edema (50) ; LGE on CMR reflects fibrosis (20) ; Cine CMR, by assessing ventricular contractility and wall motion, supports the evaluation of functional impairment (46). Perfusion PET/SPECT shows perfusion defects linked to inflammation and scarring (27, 53) ; and ^{67}Ga-scintigraphy visualizes granulomatous inflammation (10, 13). The overlapping imaging targets reflect the dynamic and non-linear progression of CS.
Abbreviations : CMR cardiac magnetic resonance imaging, STIR short tau inversion recovery, LGE late gadolinium enhancement, ^{18}F- FDG, ^{18}F fluorodeoxyglucose, PET positron emission tomography, CT computed tomography, SPECT single positron emission computed tomography

Table 1. Diagnostic Criteria for Cardiac Sarcoidosis According to the 2016 Japanese Circulation Society Guidelines

1. Histological Diagnosis	
Biopsy (endomyocardial or surgical) shows non-caseating epithelioid granulomas	
2. Clinical Diagnosis (when endomyocardial biopsy is negative or not performed)	
▪ Epithelioid granulomas are found in extra-cardiac organs, and clinical signs strongly suggest cardiac involvement in cardiac sarcoidosis ; or ▪ An evidence of pulmonary or ophthalmic sarcoidosis with ≥2 characteristic laboratory and imaging findings and clinical signs strongly suggestive of cardiac involvement (≥2 major criteria or ≥1 major and ≥2 minor criteria).	
Clinical Signs of Cardiac Involvement	
1 Major Criteria	**2 Minor Criteria**
a) High-grade atrioventricular block or fatal ventricular arrhythmias (VF, sustained VT) b) Basal ventricular septum thinning or abnormal ventricular wall anatomy (e.g., aneurysm, thinning, regional thickening) c) Left ventricular ejection fraction <50% or focal ventricular wall asynergy d) Abnormal tracer accumulation in the heart on ^{67}Ga citrate scintigraphy or ^{18}F-^{18}F-FDG PET images e) Late gadolinium enhancement on cardiac magnetic resonance images	a) Abnormal electrocardiographic findings : ventricular arrhythmias (NSVT, frequent PVCs), BBB, axis deviation, or abnormal Q waves b) Perfusion defects on myocardial perfusion scintigraphy (SPECT) c) Endomyocardial biopsy showing monocyte infiltration and moderate-to-severe interstitial fibrosis
Characteristic Laboratory and Imaging Findings for Cardiac Sarcoidosis	
▪ High serum ACE[6] activity or elevated serum lysozyme levels ▪ High serum soluble interleukin-2 receptor levels ▪ Increased tracer uptake on ^{67}Ga citrate scintigraphy or ^{18}F-FDG- PET images ▪ A high percentage of lymphocytes in BAL[7] with a CD4/CD8 ratio of >3.5 ▪ Bilateral hilar lymphadenopathy	
Isolated Cardiac Sarcoidosis Diagnostic Guidelines	**Isolated Cardiac Sarcoidosis Diagnostic Pathway**
▪ No clinical findings of organ involvement other than the heart ▪ No increased uptake in organs other than the heart on ^{67}Ga or ^{18}F-FDG PET images ▪ Chest computed tomography image showing no lymphatic shadowing, hilar, or mediastinal lymphadenopathy	▪ **Histological diagnosis** : endomyocardial biopsy or surgical biopsy showing non-caseating epithelioid granulomas ▪ **Clinical diagnosis** : isolated cardiac sarcoidosis diagnosis is made when criteria for cardiac involvement 1(d) and ≥3 of the 1(a), (b), (c), (e) are satisfied
VF = ventricular fibrillation VT = ventricular tachycardia NSVT = non-sustained ventricular tachycardia	PVC = premature ventricular contractions BBB = bundle branch block ACE = angiotensin converting enzyme

This table presents the 2016 JCS diagnostic criteria for cardiac sarcoidosis (10), including histological confirmation and clinical diagnosis based on a combination of major and minor criteria. It highlights the role of core imaging modalities, ^{18}F-FDG PET, ^{67}Ga scintigraphy, CMR with LGE, and myocardial perfusion SPECT, which serve as key components in evaluating myocardial inflammation, fibrosis, and perfusion abnormalities. These modalities contribute to both diagnostic stratification and disease staging, particularly when endomyocardial biopsy is not feasible.

fibrosis (27). Elevated right ventricular uptake is linked to worse outcomes (7, 27). PET metrics such as cardiac metabolic volume (CMV) and total lesion glycolysis (TLG) have also been associated with prognosis and treatment response (28-31). Physiologic myocardial glucose uptake can obscure findings, particularly in the basal septum. Dietary preparation, including high-fat, low-carb meals and prolonged fasting, is necessary to suppress background uptake (10, 24). Despite compliance, up to 30% of patients may still exhibit incomplete suppression (21, 32). Serum beta-hydroxybutyrate (BHB) has emerged as a potential marker for suppression adequacy, with levels below 0.35 mmol/L predicting poor suppression (32, 33). Recent studies have evaluated suppression quality and FDG quantification (28, 29, 31-33) giving promise to the utility of SUVmax, CMV, and serum BHB as tools for inflation assessment and scan optimization.

^{18}F-FDG PET is also widely used to monitor treatment response. Serial scans can reveal reductions in SUVmax and CMV, supporting therapy effectiveness, or detect persistent inflammation requiring adjustment (34, 35). Figure 2 demonstrates the utility of ^{18}F-FDG PET in detecting post-treatment inflammatory activity in a representative case.

Case-based evaluations, such as those by Norikane *et al*. (25), further emphasize the practical applications of ^{18}F-FDG-PET in differentiating uptake patterns, correlating findings with perfusion imaging, and guiding clinical management. These findings reinforce the modality's central role in both diagnostic workflows and therapeutic monitoring.

GALLIUM SCINTIGRAPHY

Gallium-67 (^{67}Ga) citrate scintigraphy was one of the earliest nuclear imaging techniques used to detect inflammation in sarcoidosis. The tracer accumulates in areas of active granulomatous inflammation, reflecting macrophage activity and granuloma density (14). Because ^{67}Ga does not physiologically accumulate in the myocardium, cardiac uptake is considered abnormal and suggestive of CS once malignancy and other inflammatory diseases are excluded (10, 36).

Historically, ^{67}Ga scintigraphy has been used to localize active disease and monitor steroid response since the 1970s (14). It is included in the JMHW criteria and referenced in both the HRS and JCS (10, 11) guidelines as a supportive or alternative imaging tool when PET is unavailable. It is reported to have a

specificity between 80–100%, however, its overall sensitivity is low (<50%) due to poor resolution, high background activity, and interference from adjacent mediastinal uptake (37, 38).

To improve spatial resolution, ^{67}Ga SPECT and integrated SPECT/CT have been developed, improving sensitivity to 64–77% and specificity to 54–100% (37). These techniques may better localize cardiac uptake and distinguish overlapping thoracic activity. Figure 3 demonstrates a representative case where ^{67}Ga SPECT/CT and CMR together illustrated both resolution of inflammation and persistent myocardial abnormalities after therapy.

Several studies have explored the combined use of ^{67}Ga with perfusion tracers such as ^{99m}Tc-sestamibi or ^{201}Tl (13, 36, 38). A lack of ^{67}Ga uptake with a matched perfusion defect may indicate fibrosis, suggesting reduced response to immunosuppressive therapy (24, 38). However, mismatches in tracer distribution that do not follow coronary territories can complicate interpretation (36-38).

Despite advancements, the clinical utility of ^{67}Ga scintigraphy has declined with the widespread availability of ^{18}F-FDG-PET, which offers superior sensitivity, spatial resolution, and preparation logistics. Nonetheless, ^{67}Ga remains a feasible alternative in centers without PET access (36).

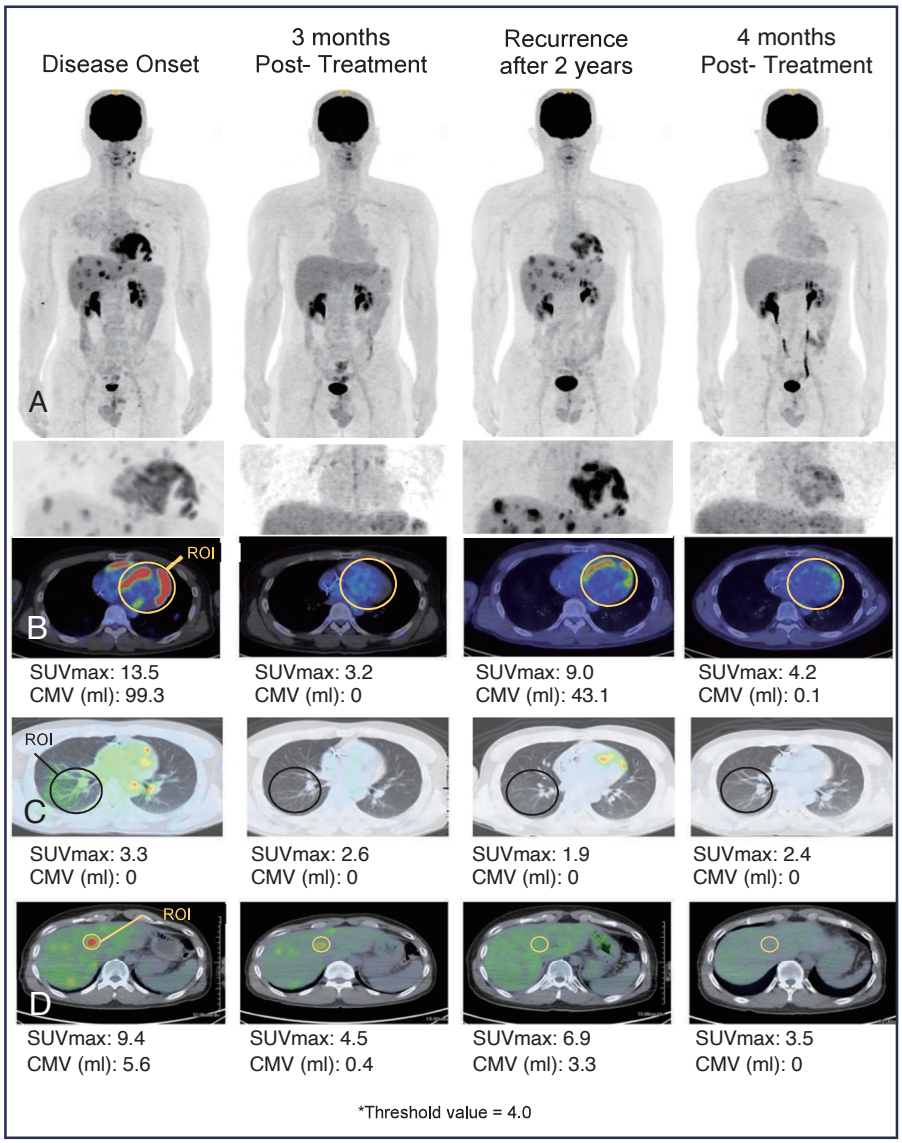

Figure 2. Case 1 : A male patient in his 30s with clinically diagnosed cardiac and extracardiac sarcoidosis developed a recurrence of disease 2 years after an initial treatment. (A) Serial MIP of the whole body and chest, along with ^{18}F-FDG PET/CT images featuring SUVmax and CMV analysis of the (B) heart, (C) lung, and (D) liver were performed to document the statuses at disease onset, post-treatment, on follow up, and after a second round of therapy due to recurrence. Corticosteroids are the first line of treatment for sarcoidosis and have been recognized to improve overall disease control, especially with pulmonary sarcoidosis. Indices such as SUVmax and CMV provide objective markers for inflammatory and metabolic activity. (B, D) The observed reduction in SUVmax and CMV post-treatment correlates with response to treatment and control of active inflammation. At the same time, persistently low SUVmax and CMV (D) suggest no active inflammation or a chronic fibrotic stage.

Abbreviations : MIP maximum intensity projection, SUVmax maximum standard uptake volume, CMV cardiac metabolic volume, ^{18}F-FDG PET ^{18}F fluorodeoxyglucose positron emission tomography, CT computed tomography

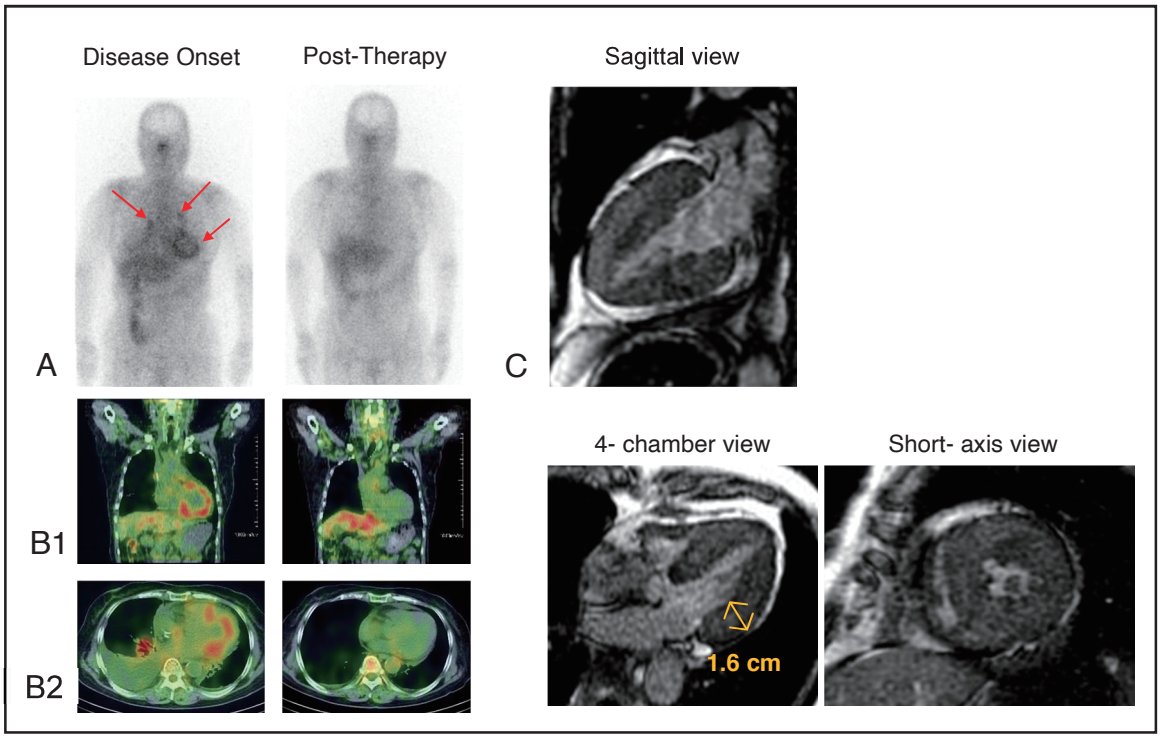

Figure 3. Case 2 : A female patient in her 70s clinically was diagnosed with cardiac sarcoidosis ; imaging was conducted at the disease onset and again 8 months after initiating steroid therapy. Whole body MIP with ^{67}Ga scintigraphy (A) shows diffuse ^{67}Ga uptake in the myocardium and hilar region at the disease onset (red arrows), which resolved post-therapy. ^{67}Ga SPECT/CT images (B) reveal tracer uptake in the left ventricular myocardium on coronal (B1) and axial (B2) views. Significant pleural perfusion on the right side of the lung is also seen, and partial resolution was noted post-therapy. (C) CMR images reveal slightly diffuse late gadolinium enhancement in the myocardium, with myocardial thickness of 1.6 cm. Abnormal ventricular wall anatomy such as regional ventricular thickening is included as a major criterion for cardiac involvement in sarcoidosis.
Abbreviations : MIP maximum intensity projection, SPECT single positron emission computed tomography, CT computed tomography, CMR cardiac magnetic resonance imaging

CARDIAC MAGNETIC RESONANCE IMAGING

CMR is often the initial imaging modality used in suspected cardiac sarcoidosis, offering high spatial resolution without ionizing radiation (1, 20, 39). Its capacity to characterize myocardial structure, function, and tissue composition makes it especially valuable in detecting scarring and fibrosis (40). Sensitivity for CMR ranges from 76–100%, with specificity from 78–92% (41). Compared with transthoracic echocardiography and the JMHW criteria, CMR demonstrates superior performance with reported sensitivity of 96.9% and specificity of 100% (42).

Late gadolinium enhancement (LGE) is a hallmark finding in CS and a major diagnostic criterion in the JCS, and HRS guidelines (10, 11). LGE occurs in approximately 80% of CS cases (43) and reflects areas of myocardial fibrosis or scarring. Gadolinium, an extracellular contrast agent, accumulates in areas of damaged myocardium due to altered washout kinetics, resulting in hyperenhancement on T1-weighted images (16, 18, 44). LGE in CS typically appears in non-coronary distributions and shows patchy or multifocal enhancement in the mid-myocardial or subepicardial layers of the basal septum, inferoseptal, or inferolateral walls (39, 45). Other reported patterns include involvement of the RV free wall, the RV side of the septum, and the inferior wall of the LV (44). The "hook sign," in which enhancement extends from the septum into the RV insertion point, is considered highly suggestive of CS in the absence of alternative pathology (46). Despite its diagnostic utility, CMR has limitations. Image quality can be degraded in patients with implanted cardiac devices, and contrast administration is contraindicated in severe renal dysfunction (16). Moreover, LGE is less effective in detecting early or active inflammation without established fibrosis.

Advanced CMR techniques such as T1 and T2 mapping provide quantitative markers of myocardial tissue composition. T2 mapping highlights edema, while T1 mapping can reflect both inflammation and fibrosis (47, 48). Although promising, these techniques face limitations such as overlapping values, image artifacts, and lack of standardized cutoffs (18, 49). Some studies have proposed combining T1/T2 mapping with ^{18}F-FDG-PET to improve sensitivity for detecting active inflammation and monitoring therapy response (47, 50). Overall, CMR remains a central imaging modality for cardiac sarcoidosis, particularly for identifying myocardial fibrosis and stratifying risk of adverse cardiac events (40, 42).

MYOCARDIAL PERFUSION IMAGING

MPI detects perfusion abnormalities associated with cardiac sarcoidosis, particularly when fibrosis or microvascular dysfunction is present. The JCS guidelines list resting MPI findings, especially non-coronary distribution defects, as a minor diagnostic criterion for CS (10). These perfusion defects often correspond to granulomatous inflammation or fibrosis and may be associated with regional wall motion abnormalities (51).

While MPI alone is not fully validated as a primary diagnostic test for CS, it plays a key role when used in combination with ^{18}F-FDG PET. A perfusion-metabolism mismatch, where FDG uptake is present but perfusion is preserved, suggests active

inflammation. In contrast, a matched pattern, with both FDG uptake and perfusion defects, may indicate scar-related inflammation or chronic disease (52). Because perfusion abnormalities can also occur in coronary artery disease, it is essential to exclude ischemic causes.

SPECT-based MPI using tracers like ^{201}Tl or ^{99m}Tc-sestamibi has long been used in settings without PET. Fixed perfusion defects that do not follow coronary territories may support a diagnosis of CS (53, 54), although sensitivity and specificity for sarcoidosis are generally low. Notably, increased tracer washout, particularly of ^{99m}Tc-MIBI or tetrofosmin, has been linked to mitochondrial dysfunction and may correlate with FDG uptake, suggesting active inflammation (52). Additionally, ^{201}Tl SPECT may show reverse redistribution, a pattern thought to reflect transient microvascular constriction near granulomas (53). A comprehensive case integrating MPI-SPECT, ^{67}Ga, and CMR is shown in Figure 4, illustrating perfusion-metabolism mismatch, tracer washout, and spatial correlation with LGE, fulfilling both major and minor JCS diagnostic criteria. Despite technical limitations, MPI continues to serve as a valuable adjunct to metabolic imaging and remains a practical alternative in PET-limited environments (27, 52).

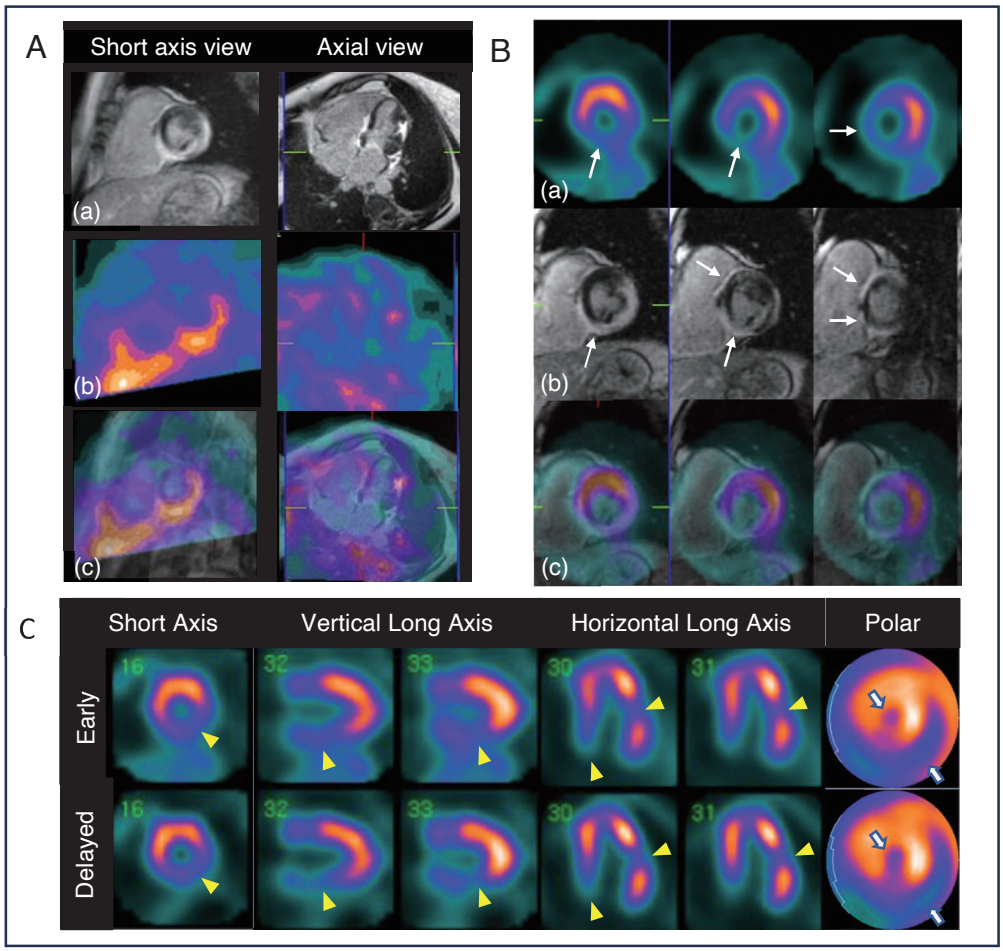

Figure 4. Case 3. A female patient in her 50s was diagnosed with pathologically confirmed extracardiac sarcoidosis for 2 years before developing cardiac symptoms.

Panel A : (a) CMR images exhibit LGE in the basal and mid-septal regions and in the basal and mid-lateral areas of the left ventricle on the axial view (arrows). In the short axis view, the accumulation extends from the anterolateral to the inferoseptal region of the left ventricle. (b) ^{67}Ga-SPECT findings exhibit accumulation in areas consistent with CMR findings, and (c) SPECT/CMR fusion image[1] confirms that uptake in both images correspond to the same areas, which may suggest coexisting myocardial inflammation and fibrosis.

Panel B : (a) MPI-SPECT (^{99m}Tc-MIBI) reveals a moderate perfusion defect in the inferolateral to inferoseptal segments (arrows). (b) CMR shows LGE in the same segments, suggesting a perfusion-metabolic mismatch. (c) MPI-SPECT/CMR fusion image[1] visualizes the mismatch completely as both (a) and (b) are combined. Perfusion defect on myocardial scintigraphy fulfills a minor criterion while LGE on CMR fulfills a major criterion based on JCS 2016 guidelines.

Panel C : ^{99m}Tc-MIBI myocardial perfusion study panel sections showing sustained perfusion defects and further tracer washout of the affected areas (yellow arrowheads) during the delayed phase, suggesting microvascular impairment of the myocardium at the affected areas. Polar mapping also regionally shows the same effect along the apex and basal inferior and basal antero-inferolateral regions of the left ventricle (white arrows).

Abbreviations : CMR cardiac magnetic resonance imaging, LGE late gadolinium enhancement, SPECT single positron emission computed tomography, MPI-SPECT myocardial perfusion imaging- single positron emission computed tomography, ^{99m}Tc-MIBI 99m technecium methoxy isobutyl isonitrile, JCS Japan Circulation Society

[1] SYNAPSE VINCENT software (Fujifilm Medical Co., Ltd., Tokyo, Japan)

HYBRID AND FUSION IMAGING (PET/CT and PET/MRI)

Hybrid and fusion imaging approaches seek to improve diagnostic precision in cardiac sarcoidosis by combining the strengths of metabolic and structural modalities. These include the co-registration of ^{18}F-FDG PET with CMR or CT, either through image fusion or simultaneous acquisition using hybrid scanners.

PET/CT fusion imaging, often combined with MPI, enables anatomical localization of FDG uptake and has demonstrated a sensitivity of 89% and specificity of 78% for CS diagnosis (25, 28). In one multicenter study, PET/CT findings reclassified 40% of patients' diagnostic likelihood when integrated with CMR, with 80% concordance with final clinical diagnosis (47). This fusion also supports staging by distinguishing metabolically active inflammation from fibrotic scarring (3, 15).

PET/MRI combines the metabolic assessment of ^{18}F-FDG-PET with the detailed tissue characterization of MRI including LGE, T2-weighted edema imaging, and T1 mapping. This integration improves the distinction between active inflammation and irreversible fibrosis and informs prognosis. For instance, the coexistence of FDG uptake and LGE has been associated with adverse outcomes and therapeutic escalation (47, 51).

Unlike PET/CT, PET/MRI avoids ionizing radiation from CT and allows truly simultaneous image acquisition, reducing spatial misalignment due to cardiac motion (46). However, PET/MRI adoption remains limited by high cost, technical complexity, and challenges in standardizing glucose suppression protocols (54).

Preliminary studies suggest PET/MRI can improve risk stratification and may aid in reclassifying ambiguous cases. Novel metrics such as SUVmax combined with extracellular volume (55) and fibrosis burden are under investigation (47, 56). The incorporation of machine learning could further enhance diagnostic workflows, but broader multicenter validation is needed (16, 47, 50). A representative case of multimodal fusion, including co-registered CMR, ^{67}Ga-SPECT, and MPI-SPECT, is also illustrated in Figure 4, highlighting the value of spatially correlated imaging in assessing both inflammation and fibrosis.

As a reference, Table 2 summarizes the diagnostic targets, key advantages, limitations, and guideline classifications of the imaging modalities discussed, with reference to the JCS 2016 criteria.

DISCUSSION

The value of multimodality imaging in CS lies in its ability to address diverse manifestations of the disease and overcome limitations inherent in any single modality (15, 18, 40). This review highlights how the strategic integration of functional, structural, and metabolic imaging modalities enhances diagnostic precision across different pathological stages of CS. As demonstrated in Figure 1, each imaging technique maps onto specific phases of disease activity, ranging from active granulomatous inflammation to chronic fibrosis and functional decline.

Among the modalities, ^{18}F-FDG-PET has emerged as a robust tool for identifying inflammation and guiding treatment response (23, 24). Its utility is reinforced by follow-up imaging over time, such as the case shown in Figure 2, where reductions in SUVmax and CMV correlated with therapeutic response (56). Supporting this, Norikane *et al*. reported that semiquantitative indices such as Target-to-Background Ratio and CMV in cardiac and extracardiac lesions offer added diagnostic and prognostic value in ^{18}F-FDG-PET-based sarcoidosis evaluation (25). CMR remains central for fibrosis detection through LGE (20), and when combined with functional markers such as T1/T2 mapping, can offer early inflammatory insights (47, 51, 57). Figures 3 and 4 emphasize how CMR, when fused with nuclear imaging, improves localization and diagnostic confidence, particularly in patchy or atypical presentations (5, 20).

MPI, while technically limited, continues to serve as a practical adjunct to metabolic imaging and remains viable in PET-limited settings (29). Its role is enhanced when combined with ^{18}F-FDG-PET, particularly in detecting perfusion–metabolism mismatches (21, 52). ^{67}Ga scintigraphy, despite its declining use, still holds value in PET-inaccessible regions, especially when

Table 2. CS Imaging Modalities : Summary of Utility and JCS 2016 Criteria Alignment

Imaging Modality	Pathological Target/ Diagnostic Focus	Strengths	Limitations	JCS 2016 Criterion (10)	Reference
Cardiac MRI (LGE)	Myocardial fibrosis (scar)	High spatial resolution ; detects scar ; non-invasive	Cannot directly assess active inflammation ; device contraindications	Major criterion (LGE or wall motion abnormality)	24, 25, 45
T2-weighted STIR (CMR)	Myocardial edema/ inflammation	Sensitive to edema ; detects inflammation	Susceptible to motion artifacts ; nonspecific	Minor criterion (increased T2 signal)	16, 20, 39
^{18}F-FDG PET/CT	Active granulomatous inflammation	High sensitivity for inflammation ; quantitative	Requires dietary prep ; limited availability	Major criterion (focal FDG uptake in noncoronary distribution)	48-50
^{67}Ga Scintigraphy	Active inflammation/ granuloma	Widely available ; low cost ; Whole-body imaging	Low resolution ; nonspecific uptake	Minor criterion (abnormal myocardial uptake)	36-38
Myocardial Perfusion SPECT	Perfusion defects not in coronary distribution	Assesses perfusion ; detect fixed defects	Limited specificity and sensitivity	Minor criterion (noncoronary perfusion defects)	21, 22, 52, 53

This table summarizes key imaging modalities used in the evaluation of cardiac sarcoidosis, highlighting their pathological targets, diagnostic strengths and limitations, and corresponding criteria from the Japanese Circulation Society (JCS) 2016 guidelines (10). Major and minor diagnostic roles are indicated based on imaging findings relevant to myocardial inflammation, edema, fibrosis, and perfusion abnormalities. Reference numbers correspond to supporting literature cited in the manuscript.
Abbreviations : CMR Cardiac Magnetic Resonance ; LGE Late Gadolinium Enhancement ; STIR Short Tau Inversion Recovery ; FDG Fluorodeoxyglucose ; PET/CT Positron Emission Tomography / Computed Tomography ; SPECT Single Photon Emission Computed Tomography ; JCS Japanese Circulation Society

SPECT/CT is available to improve resolution (9, 12, 38).

Hybrid and fusion imaging, including PET/MRI and co-registered SPECT/CMR, illustrate the future direction of CS imaging by enabling simultaneous visualization of inflammation and fibrosis (51, 55). Figure 4 showcases how this multimodal integration can fulfill multiple diagnostic criteria in a single examination, improving both staging and clinical decision-making.

Nevertheless, limitations remain. Availability, cost, and variability in scan protocols challenge the widespread implementation of some advanced modalities (24). Furthermore, distinguishing active inflammation from chronic scar remains an interpretive challenge despite improved imaging resolution (16). These clinical complexities support the growing trend toward combining imaging modalities not just for initial diagnosis but also for longitudinal monitoring, as evidenced by our case-based illustrations. The integration of emerging approaches, including metabolic quantification, radiomics, and machine learning, suggests a paradigm shift from single-modality interpretation to dynamic, data-rich diagnostic frameworks (16, 47, 50, 58). Future efforts should prioritize standardization, validation, and accessible deployment of these tools to maximize clinical impact in CS.

FUTURE DIRECTIONS

Despite the evolving role of imaging in CS, several gaps and opportunities remain. Efforts to standardize imaging protocols across centers, including ^{18}F-FDG-PET myocardial suppression techniques (21, 46), CMR mapping thresholds, and PET/MRI fusion interpretation, are critical to ensuring consistency in diagnosis and response monitoring (18, 48-50).

Research into imaging biomarkers such as SUVmax, CMV, extracellular volume, and T1/T2 mapping parameters may enhance disease staging and prognostication (25, 47, 55, 56). Quantitative approaches combining metabolic and structural data hold promise for identifying high-risk patients and personalizing therapy (28, 31).

The potential of hybrid imaging platforms, particularly PET/MRI, is increasingly recognized (18). While technical and cost limitations remain, PET/MRI may offer comprehensive insights when evaluating patients with overlapping inflammatory and fibrotic findings. Studies integrating machine learning for pattern recognition and outcome prediction are also emerging (58). Continued multicenter studies, validation of new tracers beyond FDG, and integration of imaging into treatment algorithms are necessary to strengthen the role of multimodal imaging in cardiac sarcoidosis.

CONCLUSION

This review aimed to clarify the complementary roles of multimodal imaging in cardiac sarcoidosis by synthesizing current evidence and representative clinical examples. By aligning each modality with its pathophysiologic target, we outlined how integrated imaging improves diagnostic accuracy and informs disease monitoring. Continued research toward standardization, validation of hybrid modalities, and novel imaging biomarkers will be key to refining clinical pathways for CS.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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