Integrated ^{99m}Tc-PYP Scintigraphy, ^{18}F-FDG PET/CT, and Cardiac Magnetic Resonance in Suspected Infiltrative and Inflammatory Cardiomyopathy: Initial Experience

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ABSTRACT

Background: Cardiac amyloidosis (CA) and cardiac sarcoidosis (CS) are underrecognized causes of cardiomyopathy and may phenotypically overlap with hypertrophic cardiomyopathy (HCM). Accurate differentiation among infiltrative, inflammatory, and phenotypically similar cardiomyopathies is clinically important because treatment and prognosis differ substantially.

Objective: To assess the role of a multimodality imaging approach using ^{99m}Tc-pyrophosphate (PYP) scintigraphy, ^{18}F-FDG PET/CT, and cardiac magnetic resonance (CMR) in patients with infiltrative or inflammatory cardiomyopathy.

Patients & methods: This prospective observational study enrolled 25 consecutive patients (aged 19–80 years; 48% male) who were referred for nuclear imaging evaluation of suspected infiltrative or inflammatory cardiomyopathy between August 2023 and December 2025. All patients underwent electrocardiography, echocardiography, nuclear imaging, and laboratory evaluation. CMR was performed in 15 patients, while 10 did not undergo CMR. ^{99m}Tc-PYP scintigraphy was used to assess transthyretin cardiac amyloidosis (ATTR-CM), and ^{18}F-FDG PET/CT with dietary suppression was performed to evaluate myocardial inflammation. No patient underwent endomyocardial biopsy.

Results: Non-invasive etiologic classification was achieved in 17 of 25 patients (68.0%): CA in 4 (16.0%), CS in 8 (32.0%), and HCM in 5 (20.0%); 8 patients (32.0%) remained indeterminate. All CA cases demonstrated grade 2–3 myocardial uptake on PYP scintigraphy in the absence of monoclonal protein on serum immunofixation electrophoresis. Among CS cases, FDG PET/CT showed focal, focal-on-diffuse or heterogenous myocardial uptake in 5 of 8 patients, with extracardiac pulmonary involvement in 2.

Conclusion: Multimodality imaging with PYP scintigraphy, FDG PET/CT, and CMR may improve etiologic classification and diagnostic confidence in patients with suspected infiltrative and inflammatory cardiomyopathy, particularly in settings where biopsy and genetic testing are not routinely available.

Keywords: Cardiac amyloidosis; Cardiac sarcoidosis; ^{99m}Tc-pyrophosphate scintigraphy; ^{18}F-FDG PET/CT; Cardiac magnetic resonance; Infiltrative cardiomyopathy.

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INTRODUCTION

Infiltrative and inflammatory cardiomyopathies represent a challenging spectrum of myocardial disorders that can cause progressive structural and functional impairment. Cardiac amyloidosis (CA) is characterized by extracellular deposition of misfolded protein fibrils, whereas cardiac sarcoidosis (CS) is an inflammatory granulomatous disease marked by non-caseating granuloma formation within the myocardium. Because these entities often mimic the clinical and structural features of hypertrophic or restrictive cardiomyopathy, they frequently remain unrecognized or are misdiagnosed in routine practice (1-4).

Establishing a definitive diagnosis is often hampered by limited access to advanced diagnostic tools. In many clinical settings, genetic testing infrastructure is scarce, and the utilization of endomyocardial biopsy remains low because of its invasive nature. Consequently, clinicians often rely on indirect imaging surrogates (1-4). Recent advances in nuclear cardiology have helped address this gap: 99mTc-pyrophosphate (PYP) scintigraphy has shown high utility in identifying transthyretin cardiac amyloidosis (ATTR-CM), while 18F-fluorodeoxyglucose (FDG) PET/CT enables visualization of active inflammatory foci in sarcoidosis (5-10). When combined with the tissue characterization capabilities of cardiac magnetic resonance (CMR), these modalities offer a comprehensive noninvasive diagnostic pathway (1, 2, 5, 11-14).

In this study, we evaluated the clinical role of a structured multimodality imaging strategy using 99mTc-PYP

scintigraphy, ^{18}F-FDG PET/CT, and cardiac magnetic resonance in patients with suspected infiltrative or inflammatory cardiomyopathy, with the aim of assessing how these modalities complement one another in noninvasive etiologic classification.

PATIENTS AND METHODS

This prospective observational study enrolled 25 consecutive patients (aged 19–80 years; 48% male) who were referred for nuclear imaging evaluation of suspected infiltrative or inflammatory cardiomyopathy between August 2023 and December 2025. Patients were identified on the basis of clinical presentation and transthoracic echocardiographic findings suggestive of infiltrative, inflammatory, or hypertrophic cardiomyopathy. All patients underwent comprehensive evaluation, including electrocardiography, transthoracic echocardiography, myocardial perfusion imaging, nuclear imaging, and laboratory assessment. Cardiac magnetic resonance (CMR) was performed in 15 of 25 patients, while 10 patients did not undergo CMR because it was not feasible or available at the time of evaluation. No patient underwent endomyocardial biopsy.

Electrocardiography was assessed for low voltage, left ventricular hypertrophy, atrial fibrillation, and conduction abnormalities. Transthoracic echocardiography evaluated myocardial wall thickness, including interventricular septal thickness and patterns of symmetric or asymmetric hypertrophy, myocardial texture, left ventricular systolic function, diastolic function, atrial size, and global longitudinal strain, with particular attention to features suggestive of infiltrative cardiomyopathy such as relative apical sparing where applicable. CMR was used as a complementary modality for tissue characterization and assessment of late gadolinium enhancement and other supportive structural findings when available.

For evaluation of suspected cardiac amyloidosis, ^{99m}Tc-pyrophosphate (PYP) scintigraphy was performed according to contemporary ASNC/EANM recommendations (5, 6). Patients received a weight-based intravenous dose of 555–740 MBq (15–20 mCi) of ^{99m}Tc-PYP, followed by planar and SPECT/CT acquisition after a 1–3-hour uptake period. Myocardial uptake was assessed visually using the Perugini grading scale (0–3) and semi-quantitatively using the heart-to-contralateral lung (H/CL) ratio; an H/CL ratio ≥ 1.5 at 1 hour or ≥1.3 at 3 hours was considered supportive of ATTR cardiac amyloidosis (6). SPECT/CT was used to distinguish true myocardial tracer uptake from residual blood-pool activity (5, 6).

^{18}F-FDG PET/CT was performed after high-fat, low-carbohydrate dietary preparation for 48 hours and overnight fasting to suppress physiologic myocardial glucose uptake. A dose of ^{18}5 MBq (5 mCi) of ^{18}F-FDG was administered intravenously (7, 10, 15). Active myocardial inflammation was defined as focal, focal-on-diffuse, or heterogeneous myocardial tracer uptake. Extracardiac uptake was also assessed, and myocardial perfusion imaging was integrated where relevant to identify perfusion–metabolism mismatch patterns suggestive of cardiac sarcoidosis.

Laboratory evaluation included NT-proBNP, serum free light chain assay, and serum immunofixation electrophoresis to assess for monoclonal gammopathy (2, 5, 6, 8, 9). Angiotensin-converting enzyme levels were measured in selected patients with clinical suspicion of sarcoidosis. All nuclear imaging studies were interpreted independently by two experienced nuclear medicine physicians blinded to the patients' clinical and laboratory data.

Final etiologic classification was based on integrated interpretation of clinical findings, echocardiography, nuclear imaging, laboratory data, and CMR where available. A non-invasive diagnosis of probable transthyretin cardiac amyloidosis was assigned in patients with grade 2–3 myocardial uptake on ^{99m}Tc-PYP scintigraphy, with SPECT/CT confirmation of true myocardial tracer localization, in the absence of monoclonal gammopathy on serum immunofixation electrophoresis and serum free light chain assessment (2, 6, 8, 9). Cardiac sarcoidosis was classified as probable on the basis of FDG PET/CT findings consistent with active myocardial inflammation, supported by cardiac magnetic resonance findings, extracardiac involvement, and compatible clinical features, in accordance with contemporary cardiac sarcoidosis guidance documents. (3, 7, 10, 15). Hypertrophic cardiomyopathy was assigned in patients with a hypertrophic phenotype

without imaging or laboratory evidence supporting infiltrative or inflammatory cardiomyopathy. Cases with discordant, incomplete, or non-diagnostic findings were categorized as indeterminate.

RESULTS

The study cohort comprised 25 patients aged 19–80 years, of whom 48% were male. Using an integrated multimodality imaging approach, non-invasive etiologic classification was achieved in 17 of 25 patients (68.0%), including cardiac amyloidosis in 4 patients (16.0%), cardiac sarcoidosis in 8 patients (32.0%), and hypertrophic cardiomyopathy in 5 patients (20.0%). The remaining 8 patients (32.0%) were classified as indeterminate. The demographic characteristics of the study population are summarized in Table 1 and the clinical, electrocardiographic, and echocardiographic findings of the study cohort are summarized in Table 2.

Table 1. Demographic characteristics of patients in the study cohort (n = 25)

Group	n	Age range (years)	Male/Female
Cardiac amyloidosis (CA)	4	40–68	2/2
Cardiac sarcoidosis (CS)	8	38–80	4/4
Hypertrophic cardiomyopathy (HCM)	5	19–48	3/2
Indeterminate	8	25–65	3/5

Abbreviations: CA = cardiac amyloidosis; CS = cardiac sarcoidosis; HCM = hypertrophic cardiomyopathy.

Table 2. Clinical, Electrocardiographic, and Echocardiographic Analysis of the Study Cohort (N=25)

Parameter Category	Specific Finding	Frequency (n)	Percentage (%)
Clinical Presentation	- Dyspnoea	13	52%
	- Palpitation	9	36%
	- Chest pain	8	32%
	- Swelling / Edema	4	16%
ECG Findings	- Left Ventricular Hypertrophy (LVH)	8	32%
	- Atrial Fibrillation / Atrial Flutter	7	28%
	- Ischemic changes / Old MI	5	20%
	- Low voltage complexes	4	16%
	- Left Bundle Branch Block (LBBB)	2	8%
	- Bradycardia	2	8%
Echo Morphological Patterns	- Restrictive Cardiomyopathy (RCM)	8	32%
LV Systolic Function	- Preserved LVEF (≥50%)	13	52%
	- Reduced LVEF (<50%)	12	48%

Among the 4 patients classified as cardiac amyloidosis, echocardiography demonstrated increased interventricular septal thickness (13–16 mm), a restrictive filling pattern, characteristic granular ("sparkling") myocardial texture, and reduced global longitudinal strain in most cases. All 4 patients showed grade 2–3 myocardial uptake on ^{99m}Tc-PYP scintigraphy, supporting transthyretin cardiac amyloidosis, and had no evidence of monoclonal gammopathy on serum immunofixation electrophoresis; these patients were not preselected on the basis of monoclonal protein status but emerged from the consecutively referred cohort. FDG PET/CT showed no myocardial uptake in any CA case. CMR findings were supportive in 3 patients, while 1 patient did not undergo CMR but had concordant echocardiographic, scintigraphic, and biochemical findings. **Figures 1 and 2** illustrate a representative case of cardiac amyloidosis.

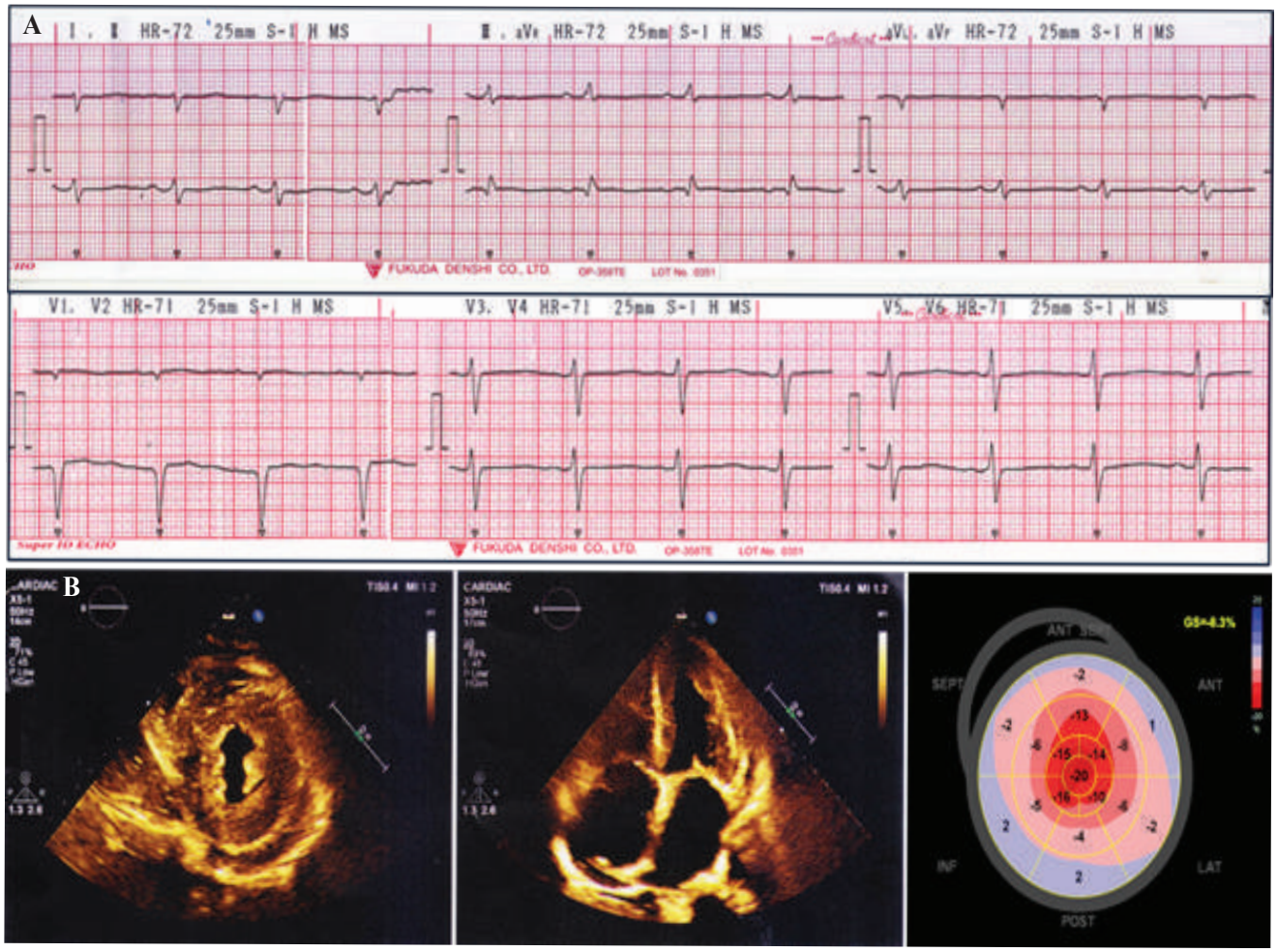

Figure 1: A 45-year-old woman presented with occasional chest pain, palpitations, and fatigue. Electrocardiogram shows sinus rhythm with low QRS voltage in the limb leads and poor anterior R-wave progression with a pseudo-infarct pattern. B Transthoracic echocardiography (parasternal short-axis and apical four-chamber views) showing concentric left ventricular hypertrophy (interventricular septal thickness 14 mm), bi-atrial enlargement, and a granular sparkling myocardial appearance, with moderately reduced left ventricular systolic function (LVEF ~40%), and mild pericardial effusion. Speckle-tracking echocardiography demonstrates reduced global longitudinal strain (GLS −8.3%) with relative apical sparing, a pattern classically associated with cardiac amyloidosis.

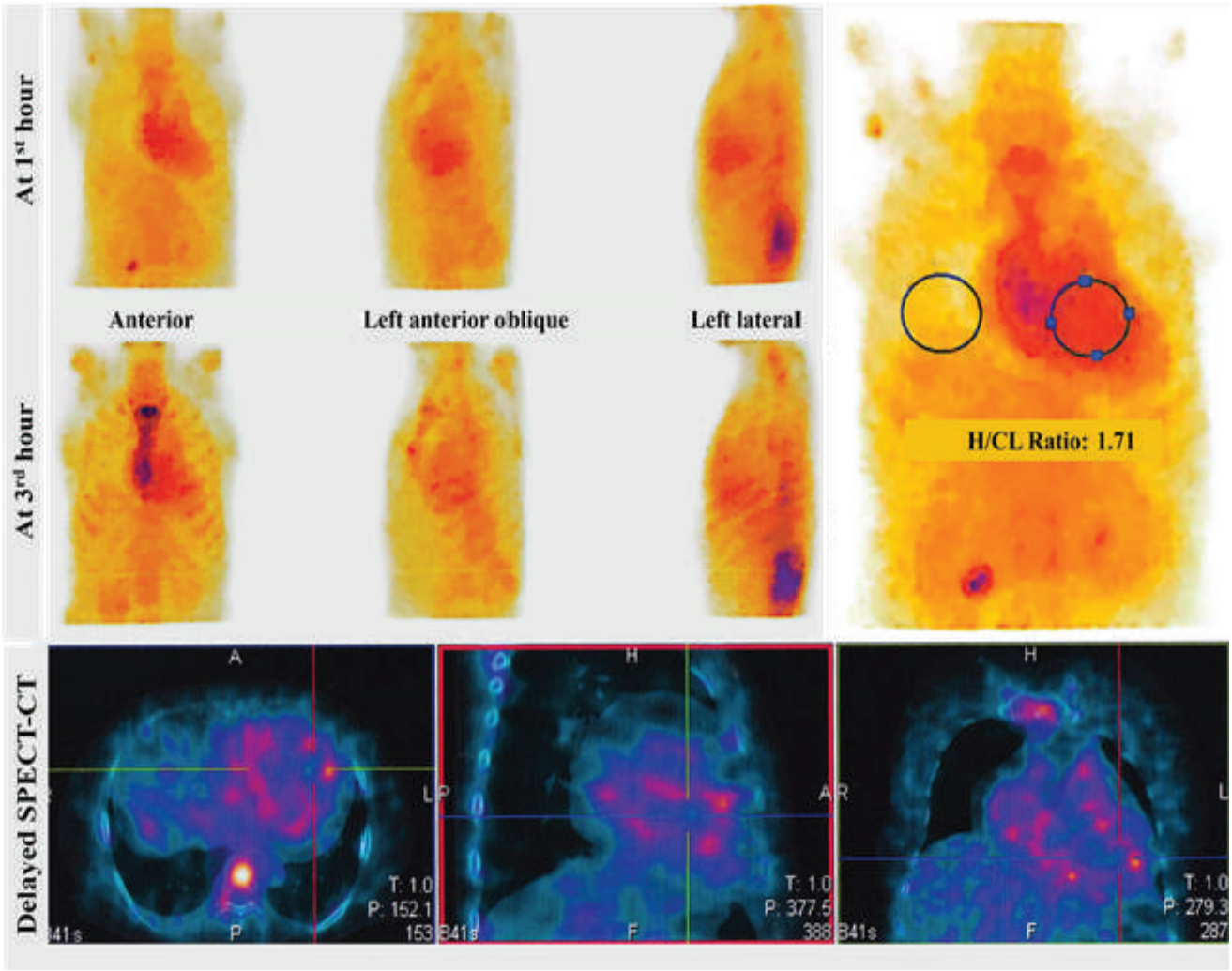

Figure 2: ^{99m}Tc-pyrophosphate (PYP) scintigraphy findings in the same patient. Planar ^{99m}Tc-PYP scintigraphy shows increased myocardial uptake (Perugini grade 3 at 1 hour and grade 2 at 3 hours) with an H/CL ratio of 1.71. SPECT/CT confirms true myocardial tracer localization. These findings are consistent with transthyretin cardiac amyloidosis (ATTR-CA) in the absence of monoclonal protein on serum immunofixation electrophoresis. ^{18}F-FDG PET/CT shows no focal or diffuse myocardial FDG uptake, effectively excluding active cardiac sarcoidosis. Cardiac magnetic resonance findings are also supportive.

Among the 8 patients classified as cardiac sarcoidosis, FDG PET/CT demonstrated focal or focal-on-diffuse or heterogenous myocardial uptake consistent with active inflammation in 5 cases (62.5%). Two patients also showed extracardiac pulmonary involvement. CMR findings were suggestive of CS in 7 patients, while 1 patient was classified as probable CS based on FDG PET/CT and echocardiographic findings in the absence of CMR. No patient in the CS group demonstrated myocardial uptake on ^{99m}Tc-PYP scintigraphy. Figures 3-5 illustrate two representative cases of cardiac sarcoidosis.

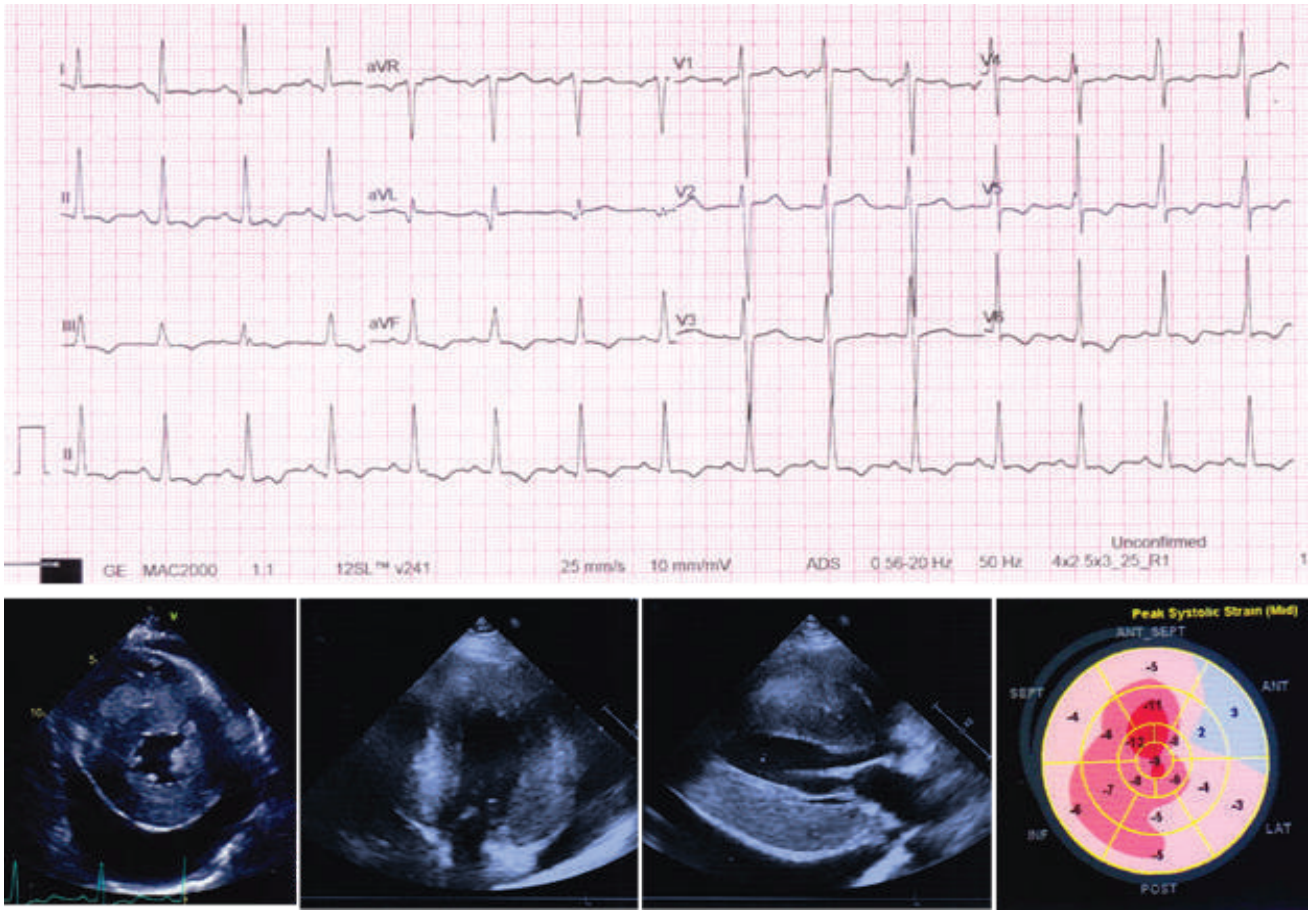

Figure 3: A 50-year-old man with exertional dyspnea. Electrocardiogram shows sinus rhythm with left ventricular hypertrophy and probable left atrial enlargement. Transthoracic echocardiography demonstrates marked concentric left ventricular hypertrophy with a granular myocardial appearance and moderate pericardial effusion. Global longitudinal strain is reduced (−9.3%) with relative apical sparing, consistent with an infiltrative cardiomyopathy. Negative serum free light chains and ^{99m}Tc-PYP scan, with elevated angiotensin-converting enzyme levels, raise suspicion for cardiac sarcoidosis.

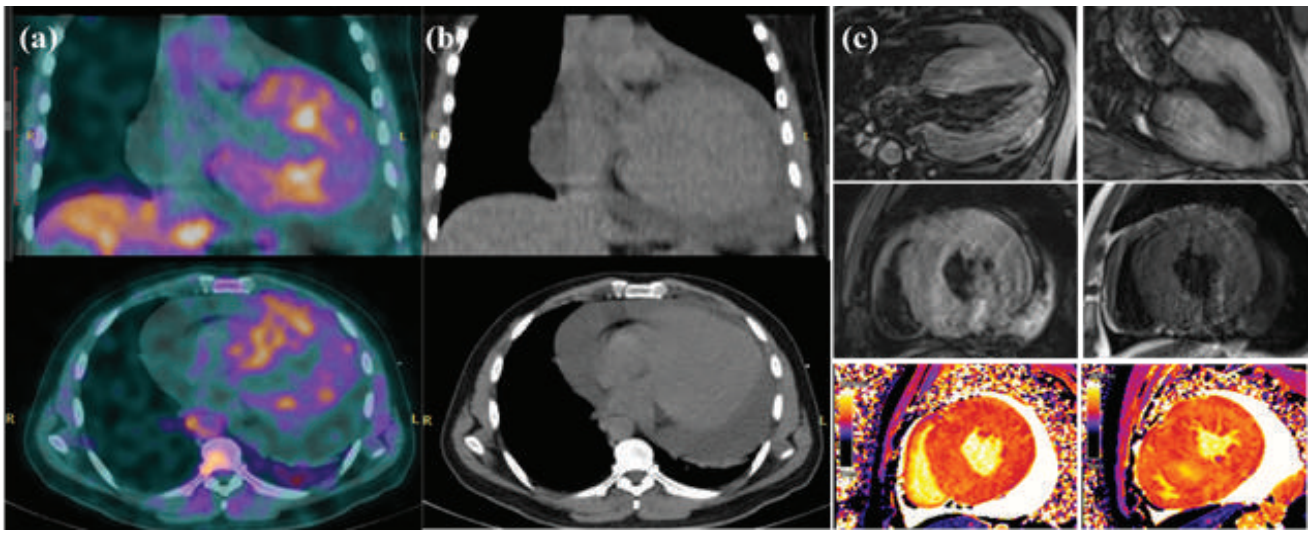

Figure 4: ^{18}F-FDG PET-CT and Cardiac MRI findings in the same patient. (a) Fused PET-CT images demonstrate diffuse, heterogeneous FDG uptake in the left ventricular myocardium, consistent with active myocardial inflammation. (b) CT images show marked pericardial effusion.

(c) CMR images (cine, late gadolinium enhancement [LGE], and T1 mapping) demonstrate preserved ventricular systolic function with non-dilated ventricles, diffuse left ventricular wall thickening, and bi-atrial enlargement, suggestive of restrictive physiology. LGE shows patchy intramural (mid-wall) enhancement involving the anteroseptal and inferoseptal segments at the basal and mid-ventricular levels in a nonischemic distribution with elevated native T1 mapping time (≈1320 ms), consistent with active inflammatory myocardial infiltration. Overall findings support active cardiac sarcoidosis.

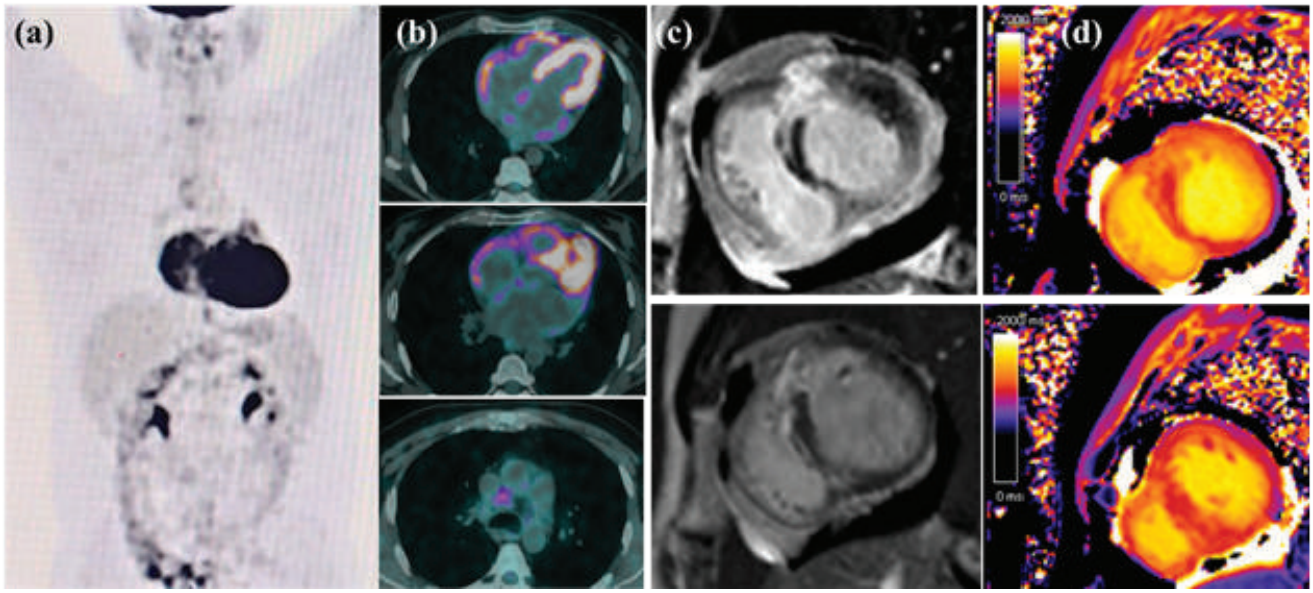

Figure 5: ^{18}F-FDG PET-CT and cardiac MRI findings in a 45-year-old man with cardiac sarcoidosis. (a) Whole-body maximum intensity projection and (b) fused PET-CT images demonstrate diffusely increased FDG uptake involving the left ventricular myocardium with additional scattered focal uptake in both atria, consistent with active myocardial inflammation. FDG-avid mediastinal lymph nodes are also noted. (c, d) CMR images (late gadolinium enhancement [LGE] and T1 mapping) show patchy mid-wall and subepicardial enhancement involving the anterior and anteroseptal segments at the basal and mid-ventricular levels in a nonischemic distribution, with elevated native T1 values, consistent with inflammatory myocardial infiltration. Findings are in keeping with active cardiac sarcoidosis.

In the hypertrophic cardiomyopathy group (n = 5), echocardiography demonstrated asymmetric or concentric hypertrophy with increased interventricular septal thickness. CMR confirmed a hypertrophic phenotype in 4 patients without features suggestive of infiltrative cardiomyopathy. FDG PET/CT showed no myocardial uptake in 3 patients, while 2 patients demonstrated nonspecific focal or mild diffuse uptake. None of the patients in this group showed myocardial uptake on 99mTc-PYP scintigraphy. One patient did not undergo CMR.

The 8 indeterminate cases had negative 99mTc-PYP scintigraphy and discordant, incomplete or inconclusive multimodality findings, precluding etiologic classification. CMR was performed in 15 of 25 patients; the limited availability of CMR, particularly among indeterminate cases, reduced diagnostic confidence. NT-proBNP levels generally paralleled disease severity and were highest in patients with more advanced clinical presentation.

DISCUSSION

This study demonstrates the clinical utility of an integrated multimodality imaging approach in patients with suspected infiltrative and inflammatory cardiomyopathy. In a cohort with overlapping clinical and echocardiographic features, the combined use of 99mTc-PYP scintigraphy, ^{18}F-FDG PET/CT, and CMR enabled noninvasive etiologic classification in 17 of 25 patients (68.0%), while 8 patients (32.0%) remained indeterminate. These findings highlight both the value

and the practical limitations of a structured imaging-based diagnostic pathway.

Among patients classified as having cardiac amyloidosis, all demonstrated grade 2–3 myocardial uptake on 99mTc-PYP scintigraphy, supporting the established role of PYP imaging in the non-biopsy diagnosis of ATTR-CM after exclusion of monoclonal gammopathy (2,5,6,8,9). However, PYP findings should be interpreted cautiously and integrated with laboratory and tomographic evaluation, as blood-pool activity and, less commonly, recent myocardial infarction may lead to false-positive interpretation if SPECT/CT is not carefully assessed (5, 6). In addition, although 99mTc-PYP imaging is highly sensitive for ATTR-CM, it does not exclude AL amyloidosis; therefore, a serum free light chain assay together with serum and urine immunofixation electrophoresis is recommended in all patients undergoing PYP imaging for suspected cardiac amyloidosis (2,5,6,8,9). No myocardial PYP uptake was observed in the cardiac sarcoidosis or hypertrophic cardiomyopathy groups, underscoring its discriminatory value in patients with increased wall thickness or a restrictive phenotype (2, 5, 6, 11-14).

^{18}F-FDG PET/CT was useful for identifying active myocardial inflammation in patients classified as having cardiac sarcoidosis, with focal or focal-on-diffuse uptake seen in 5 of 8 cases and extracardiac pulmonary involvement in 2. These findings support the complementary role of FDG PET/CT in detecting inflammatory activity and extracardiac disease burden. However, the absence of positive FDG uptake in some patients also underscores that PET/CT should be interpreted in conjunction with clinical, echocardiographic, and CMR findings rather than in isolation (3, 7, 10, 15).

CMR provided important additional information for tissue characterization and phenotype refinement. It supported the diagnosis in most patients with cardiac sarcoidosis and cardiac amyloidosis and helped confirm hypertrophic cardiomyopathy in patients without imaging evidence of infiltrative disease. However, CMR was performed in only 15 of 25 patients, and its limited availability contributed substantially to diagnostic uncertainty, particularly among indeterminate cases. This finding reflects real-world practice in resource-constrained settings, where complete multimodality evaluation is not always feasible (1, 2, 5, 11-15).

The HCM group is noteworthy because multimodality imaging was particularly useful in excluding cardiac amyloidosis and cardiac sarcoidosis in patients presenting with a hypertrophic phenotype. Negative 99mTc-PYP scintigraphy, absence of supportive inflammatory findings on ^{18}F-FDG PET/CT, and CMR features not suggestive of infiltrative cardiomyopathy supported classification as probable HCM. However, in the absence of genetic testing, a definite sarcomeric HCM diagnosis could not be confirmed, and other hereditary or storage-related causes of left ventricular hypertrophy, including Fabry disease, Danon disease, PRKAG2 syndrome, and Pompe disease, cannot be excluded. In selected patients, further evaluation with genetic testing for sarcomeric genes and relevant enzyme assays such as alpha-galactosidase A for Fabry disease and acid alpha-glucosidase for Pompe disease may help refine the diagnosis (1, 4).

The presence of an indeterminate group remains clinically important. These patients had negative PYP scintigraphy and discordant or inconclusive multimodality findings, precluding definitive etiologic classification. This emphasizes that no single imaging modality is sufficient in all cases and that incomplete evaluation may leave residual uncertainty. This study has several limitations, including its single-center design, small sample size, incomplete CMR availability, absence of systematic histopathologic confirmation, and lack of genetic testing in patients with a hypertrophic phenotype. Despite these limitations, the present study supports the practical role of multimodality imaging in improving diagnostic confidence and facilitating noninvasive etiologic classification in suspected infiltrative and inflammatory cardiomyopathy, particularly in real-world and resource-limited settings where biopsy and genetic testing are not routinely available.

CONCLUSION

In patients with suspected infiltrative or inflammatory cardiomyopathy, a structured multimodality imaging approach combining 99mTc-PYP scintigraphy, 18F-FDG PET/CT, and cardiac magnetic resonance can improve noninvasive etiologic classification and diagnostic confidence in differentiating cardiac amyloidosis, cardiac sarcoidosis, and phenotypic mimics. This approach appears particularly valuable in real-world settings where endomyocardial biopsy and genetic testing are not routinely available. However, the findings should be interpreted in the context of the study's small sample size, incomplete CMR availability, and lack of systematic histopathologic confirmation, and larger prospective studies are needed to validate this strategy.

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Conflict of Interest: The authors declare no conflict of interest.

Ethical Approval: The study was approved by the Institutional Review Board, INMAS, Dhaka, BAEC, Bangladesh.

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