Section: Radiodiagnosis

Original Research Article

PATTERNS OF LATE GADOLINIUM ENHANCEMENT IN CARDIAC MAGNETIC RESONANCE IMAGING IN PATIENTS OF NON ISCHEMIC CARDIOMYOPATHY

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ABSTRACT

Background: Delayed contrast enhancement in cardiac magnetic resonance (CMR) imaging plays a pivotal role in assessing myocardial tissue characteristics in non-ischemic cardiomyopathy (NICM). This study aims to explore the various patterns of late gadolinium enhancement (LGE) observed in NICM patients and their correlation with clinical outcomes. The study identifies distinct morphological and distributional variations, including diffuse, patchy, subepicardial, mid-wall, and subendocardial enhancement. Recognizing these patterns enhances diagnostic accuracy and improves prognostic evaluations in NICM, ultimately guiding therapeutic strategies for better patient management. **Objective:** The primary aim of this prospective study is to evaluate the patterns of delayed contrast enhancement in NICM patients using CMR imaging.

Material & Methods: A total of 40 NICM cases were included in this study, conducted in the Department of Radiodiagnosis at G.S.V.M. Medical College, Kanpur, from September 2022 to July 2024. Patients were selected in collaboration with LPS Institute of Cardiology based on clinical and echocardiographic criteria. Cardiac MRI was performed on a 3T machine and the images were qualitatively analyzed for abnormal myocardium.

Results: The mean age of patients in this study was 58 years. Of the total, 30 (75%) had dilated cardiomyopathy (DCM), 5 (12.5%) had myocarditis, 3 (7.5%) had restrictive cardiomyopathy (RCM), and 2 (5%) had sarcoidosis. Among the DCM patients, mid-wall enhancement was seen in 10 (33.33%). In the myocarditis group, 2 (40%) patients showed a subepicardial pattern of enhancement. Both sarcoidosis patients exhibited involvement of the subendocardium, mid-wall, and subepicardium. Additionally, one (33.3%) patient with RCM demonstrated subendocardial enhancement. A significant association was found between enhancement patterns and the underlying pathologies (p=0.0001).

Conclusions: LGE CMR is becoming an indispensable tool for detecting, characterizing, and differentiating various cardiomyopathies. By classifying delayed myocardial enhancement based on its precise anatomical location, it is possible to distinguish between non-ischemic and ischemic (infarct-related) cardiomyopathies. When clinical suspicion is high, delayed-enhanced cardiac MRI can facilitate early detection and prompt management of cardiomyopathies, improving patient outcomes.

Keywords: LGE - late gadolinium enhancement, NICM - non-ischemic cardiomyopathy, CMR - cardiac magnetic resonance, DCM - dilated cardiomyopathy, RCM - restrictive cardiomyopathy.

INTRODUCTION

Traditionally, delayed contrast-enhanced cardiac magnetic resonance (CMR) imaging has been extensively utilized for the evaluation of ischemic heart disease, particularly for assessing myocardial infarction and viability. However, there is an increasing trend toward using CMR for assessing non-ischemic cardiomyopathies (NICM).[1] Cardiomyopathies can result from various factors such as infections, inflammatory processes, connective tissue disorders, infiltrative diseases like sarcoidosis and amyloidosis, and ischemic heart disease.[2] These conditions often present with similar clinical symptoms, making differential diagnosis challenging.

Delayed contrast-enhanced CMR can assist in evaluating functional decline, distinguishing between ischemic and non-ischemic causes of cardiomyopathy, and narrowing the differential diagnosis for NICM based on characteristic enhancement patterns.[4] Abnormal areas of increased enhancement can also help improve the diagnostic yield of endomyocardial biopsy.[5] In pediatric patients or those with significant comorbidities, certain enhancement patterns observed on CMR may even obviate the need for biopsy if they align with a clinically suspected diagnosis.[6] This article explores the distinct patterns of delayed contrast enhancement in the left ventricular myocardium seen in NICM and provides a systematic approach to evaluating these patterns based on their distribution and morphology.

The myocardium can be divided into three primary layers: the subendocardium, subepicardium, and mesocardium (or midmyocardium). Each of these layers can exhibit specific patterns of delayed contrast enhancement in NICM, which help in differentiating ischemic from non-ischemic etiologies.[7] For instance, ischemic cardiomyopathy, typically related to myocardial infarction, shows delayed enhancement predominantly in the subendocardium or across the entire myocardial thickness in a vascular distribution (8). Conversely, non-ischemic cardiomyopathies often display delayed enhancement in non-coronary vascular territories, sparing the subendocardium or showing isolated involvement of the mid-wall or subepicardial regions.[9] Recognizing these patterns is crucial for accurate diagnosis and treatment planning, as they can point toward specific underlying conditions, such as myocarditis, sarcoidosis, or amyloidosis.

Evaluating delayed enhancement patterns can also provide prognostic information in NICM. Certain enhancement patterns, like mid-wall enhancement in dilated cardiomyopathy or patchy enhancement in sarcoidosis, have been associated with increased risk of adverse outcomes, including arrhythmias and sudden cardiac death.[10] This underscores the utility of CMR not only in diagnosing but also in risk stratifying patients with NICM. As a non-invasive imaging modality, CMR with late gadolinium enhancement has become an essential tool for guiding clinical management by providing insights into myocardial tissue characterization, fibrosis extent, and disease progression.[11]

MATERIALS AND METHODS

This prospective study was conducted from September 2022 to July 2024 at GSVM Medical College, Kanpur, within the Department of Radiodiagnosis, in collaboration with the LPS Institute of Cardiology. Patients meeting the inclusion and exclusion criteria were selected for the study after obtaining informed consent. The inclusion criteria required patients to be aged between 18 and 75 years, have a left ventricular ejection fraction (LVEF) of less than 50%, be suspected of having non-ischemic cardiomyopathy, and have a normal coronary angiogram. The exclusion criteria included patients below 18 or above 75 years, those with coronary artery disease, hypertrophic cardiomyopathy, chronic kidney disease, cardiac prosthetic devices, joint replacements, metallic implants, chronic obstructive pulmonary disease, claustrophobia, or those who refused to provide consent.

Cardiac MRI was performed using a 3T MRI (MAGNETOM Vida system, syngo MRXA31) with a cardiac phased-array surface coil. Imaging was conducted during breath-holding for 12 to 15 cardiac beats (10 to 15 seconds on average) at the end of expiration, synchronized with ECG. Two pulse sequences were utilized: cine-CMR with the steady-state free precession technique (SSFP) and late gadolinium enhancement (LGE). Initial images were acquired to confirm proper coil positioning and to define both short- and long-axis views of the left ventricle (LV). To cover the LV from apex to base, 8 to 12 short-axis slices of 8 mm thickness at 2 mm intervals were obtained. Cine-CMR with SSFP was performed to assess ventricular morphology, volumes, ejection fraction, and mass. Key parameters included a field of view of 34 to 38 cm, a matrix of 256×160, receiver bandwidth of 125 kHz, k-space segmented into 8 to 12 lines, TR of 3.9 ms, TE of 1.4 ms, flip angle of 45°, a 3/4 field of view, and 20 phases per cardiac cycle. (Figure 1,2,3,4)

For LGE imaging, patients were administered an intravenous bolus of 0.2 mmol/kg of gadolinium-based contrast agent. Ten minutes post-contrast administration, images were acquired using the LGE technique, employing a gradient-echo pulse sequence with an inversion-recovery preparatory pulse. The imaging parameters included a TR of 7.2 ms, TE of 3.2 ms, a matrix of 256×192, a flip angle of 20°, bandwidth of 31.2 kHz, inversion time ranging from 150 to 300 ms, two excitations, and acquisition during every heart beat (1 RR). The inversion time was carefully adjusted to null the

signal from normal myocardium, ensuring that abnormal regions appeared as intensely bright on delayed images. Statistical analysis was performed using SPSS version 27.0 and GraphPad Prism version 5, with significance set at a p-value of <0.05. The chi-squared test or Fischer's exact test, along with independent and paired t-tests, were used to assess statistical relationships.

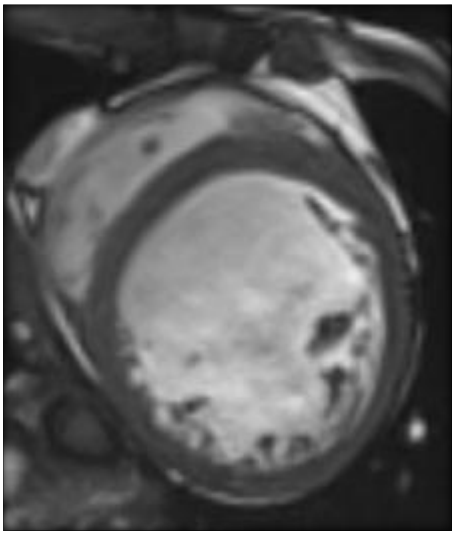
RESULTS

The study included a total of 40 patients. The mean age of the cohort was 50.8 ± 9.6 years, with the majority of patients falling within the 51-60 years age group (32.5%), followed by the 41-50 years age group (30%), as outlined in Table 1. Males were predominant, constituting 72.5% of the population, while females made up 27.5%.

Among the diagnoses, 30 patients (75%) were diagnosed with dilated cardiomyopathy (DCMP), 5 (12.5%) with myocarditis, 3 (7.5%) with restrictive cardiomyopathy (RCMP), and 2 (5%) with sarcoidosis, as shown in Table 2. Late gadolinium enhancement (LGE) images revealed that 25 (62.5%) patients showed no enhancement, while 15 (37.5%) demonstrated varying patterns of enhancement (Table 3). The enhancement patterns were further detailed, with 8 (20%) patients exhibiting mid-wall enhancement, and smaller groups showing combinations of subendocardial, subepicardial, and mid-wall enhancements.

The analysis of enhancement morphology indicated that 6 (15%) patients displayed a striated appearance, while 4 (10%) had patchy enhancement (Table 4). For DCMP patients, 10 (33.3%) showed significant LGE, with 8 (26.7%) displaying a mid-wall striated pattern and 2 (6.7%) showing combined mid-wall and subendocardial enhancement. Among myocarditis patients, 2 (40%) displayed LGE with subepicardial lesions. Both sarcoidosis patients exhibited patchy and patchy-transmural LGE involving the subendocardium, mid-wall, and subepicardium, while 1 RCMP patient (33.3%) showed diffuse subendocardial enhancement (Table 5).

Statistical analysis using the chi-square test revealed a significant association between enhancement patterns and the underlying diagnosis ($p<0.0001$), highlighting the diagnostic relevance of LGE in differentiating between various forms of non-ischemic cardiomyopathies (Table 5).



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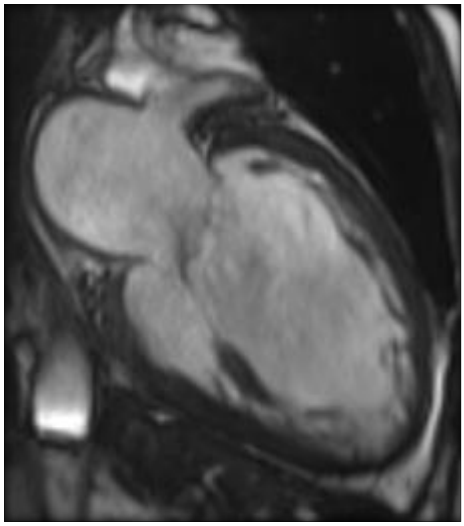

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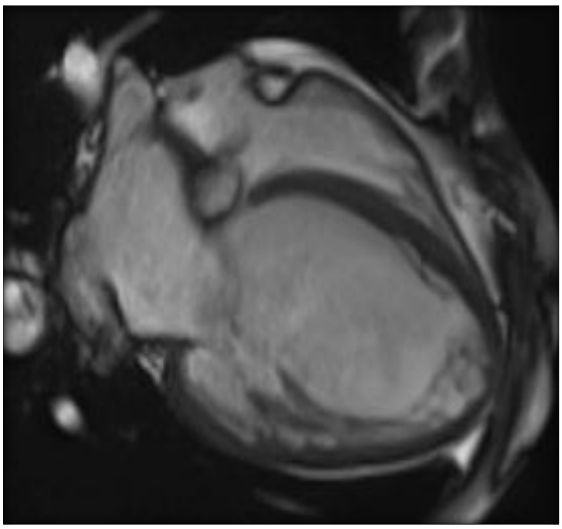

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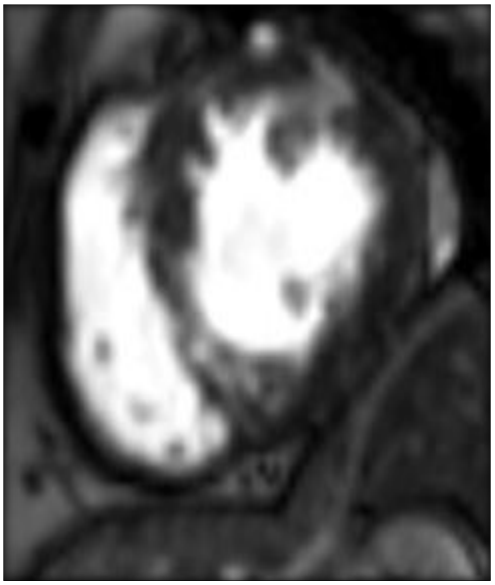
Figure 1: Cine cardiac magnetic resonance images of dilated cardiomyopathy patient show A. short axis at mid ventricle B. 2 chamber (vertical long axis) view C. 4 chamber (horizontal long axis) view



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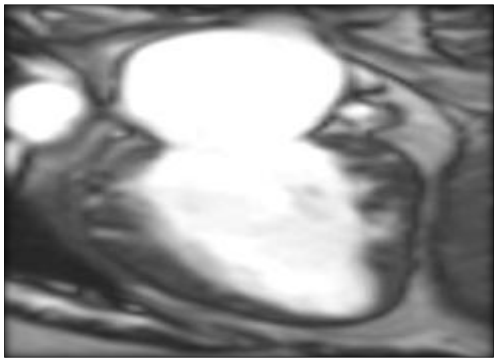

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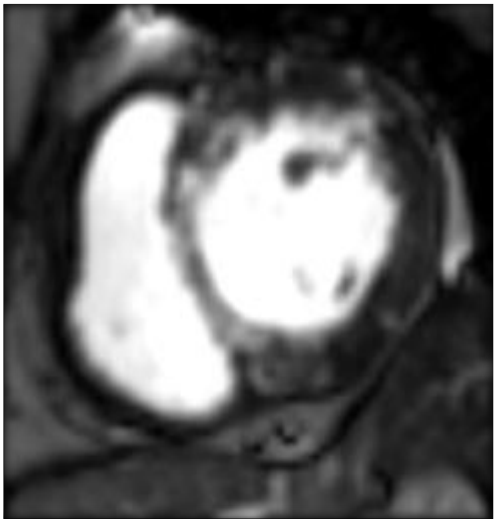
Figure 2: 50 year old female presented with chest pain on examination patient had arrhythmia and mild left ventricular dysfunction, a clinically suspected case of sarcoidosis, cardiac MR cine images show thinning of the basal inferior septum and delayed enhancement images show predominantly patchy and some transmural pattern involving sunendocardium, mid wall and subepicardium



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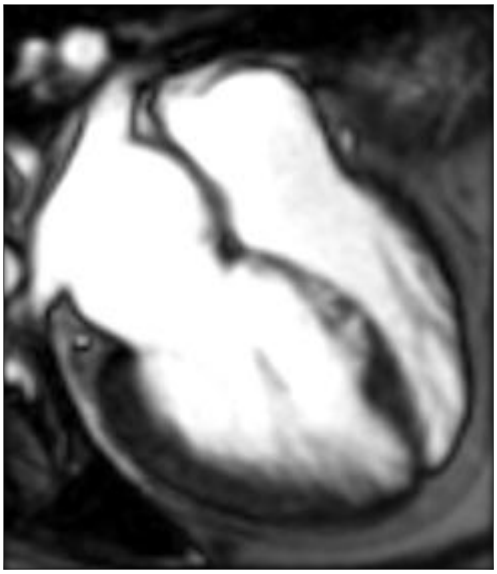

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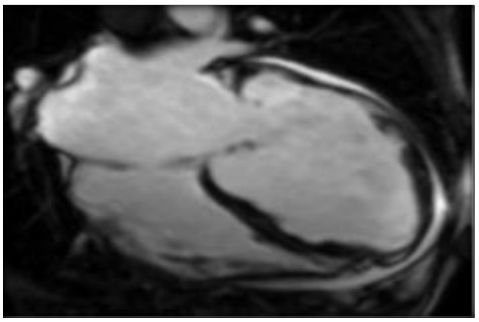
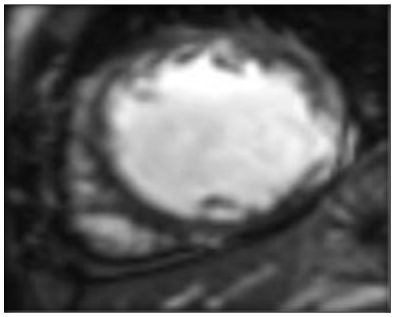




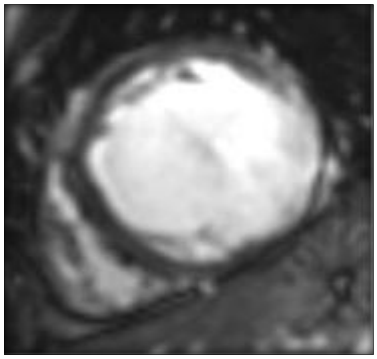
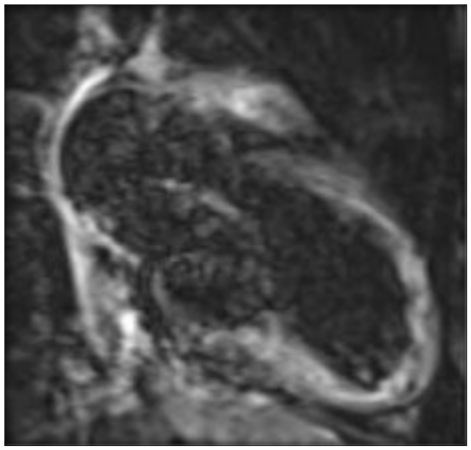


Figure 3: In a patient of dilated cardiomyopathy (DCMP) A. horizontal long axis (4 chamber) delayed phase sensitive inversion recovery (PSIR) sequence image give patchy subendocardial with mid wall enhancement involving lateral wall of the left ventricle and B. short axis view describing the same



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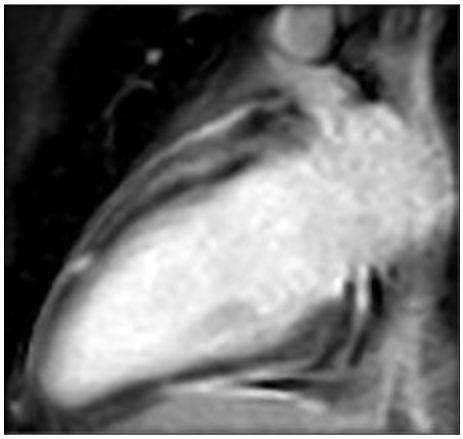

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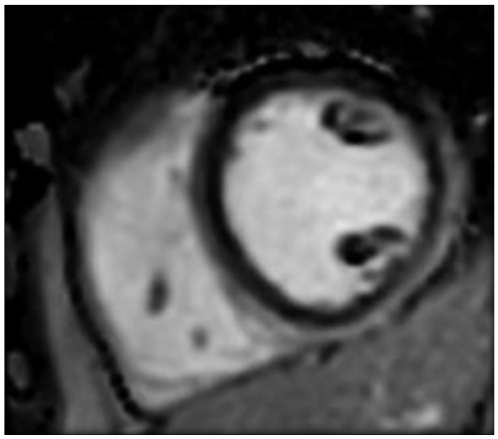

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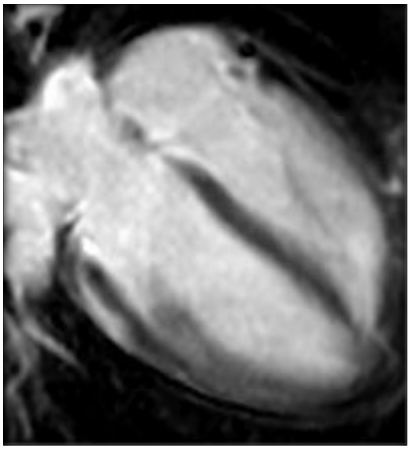

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Figure 4: Patient of myocarditis with A. STIR sequence shows myocardial edema, B. short axis PSIR sequence with subepicardial delayed contrast enhancement consistent on C. vertical long axis (2 chamber) and D. horizontal long axis (4 chamber) view

Table 1: Distribution of Age in Group

Age Group	Frequency	Percent
<30	1	2.5%
31-40	6	15.0%
41-50	12	30.0%
51-60	13	32.5%
>70	8	20.0%
Total	**40**	**100.0%**

Table 2: Distribution of Diagnosis

Diagnosis	Frequency	Percent
DCMP	30	75.0%
Myocarditis	5	12.5%
RCMP	3	7.5%
Sarcoidosis	2	5.0%
Total	40	100.0%

Table 3: Distribution of Late Gadolinium Enhancement

LGE (Late Gadolinium Enhancement)	Frequency	Percent
No Enhancement	25	62.5%
Present	15	37.5%
Total	40	100.0%

Table 4: Distribution of Enhancement Morphology

Enhancement Morphology	Frequency	Percent
Diffuse	1	2.5%

No Enhancement	26	65.0%
Patchy	4	10.0%
Patchy, Striated	2	5.0%
Patchy, Transmural	1	2.5%
Striated	6	15.0%
Total	40	100.0%

Table 5: Association between Enhancement Pattern and Diagnosis

Enhancement Pattern	DCMP	Myocarditis	RCMP	Sarcoidosis	Total
Mid-wall	8	0	0	0	8
Mid-wall, Subendocardial	2	0	0	0	2
No Enhancement	20	3	2	0	25
Subendocardial	0	0	1	0	1
Subendocardial, Mid-wall, Subepicardial	0	0	0	2	2
Subepicardial, Mid-wall	0	1	0	0	1
Subepicardial	0	1	0	0	1
Total	30	5	3	2	40

Chi-square value: 69.0133, df: 18, p-value: <0.0001

DISCUSSION

Cardiac magnetic resonance (CMR) is currently the most accurate non-invasive technique for evaluating myocardial necrosis and fibrosis. It plays a critical role in identifying the root causes of left ventricular (LV) dysfunction and can provide essential prognostic data. Late gadolinium enhancement (LGE) has been shown to be a valuable tool for excluding severe coronary artery disease in patients recently diagnosed with left ventricular failure, particularly in cases where there is no evidence of ischemic disease.[1]

The examination of LGE patterns is advantageous in distinguishing non-ischemic causes of LV dysfunction, such as dilated cardiomyopathy (DCMP), cardiac sarcoidosis, myocarditis, hypertrophic cardiomyopathy, cardiac amyloidosis, and Anderson-Fabry disease.[2,3] However, it is important to recognize that LGE patterns are not always disease-specific, making the inclusion of clinical context essential for accurate diagnosis.[4]

The observed patterns of LGE in our study align with previous research on non-ischemic cardiomyopathy (NICM), indicating that LGE distribution in NICM does not follow specific vascular territories and can affect any region of the myocardium.[5] In this study, normal coronary angiography excluded the presence of coronary artery disease (CAD), helping to solidify a diagnosis of NICM.

Dilated cardiomyopathy (DCMP) is the most common form of NICM, characterized by impaired LV contraction leading to a reduced ejection fraction of less than 40%.[6] In most cases, LGE CMR findings in DCMP reveal a mid-wall striated pattern of enhancement, with occasional localized patchy enhancement.[7] Restrictive cardiomyopathy (RCMP) is another form of NICM, commonly caused by the abnormal accumulation of proteins, glycogen, or iron within the myocardium. This leads to ventricular stiffening and impaired relaxation during diastole.[8] Sarcoidosis, amyloidosis, hemochromatosis, endomyocardial fibrosis, scleroderma, and Löffler endocarditis are all frequently observed forms of RCMP.[9,10]

Sarcoidosis, a systemic condition characterized by the formation of noncaseating granulomas in multiple organs, affects the heart in at least 25% of sarcoidosis patients.[11] While endomyocardial biopsy can confirm cardiac involvement, its sensitivity is limited due to the sporadic nature of granuloma distribution.[12] CMR with LGE imaging is highly sensitive in detecting cardiac involvement in sarcoidosis, showing non-ischemic enhancement patterns that can affect the subendocardium, mid-wall, subepicardium, and transmural regions in a patchy distribution.[13] A meta-analysis comparing CMR with FDG-PET for detecting cardiac sarcoidosis found that CMR had higher sensitivity while maintaining comparable specificity to FDG-PET.[14] This increased sensitivity makes CMR particularly valuable in excluding cardiac sarcoidosis when the results are negative.[15]

Cardiac amyloidosis is another significant cause of NICM, involving the extracellular deposition of misfolded proteins, such as light chains (AL) or transthyretin amyloid (ATTR).[16] These deposits enlarge the interstitium and delay gadolinium clearance, resulting in a characteristic subendocardial LGE pattern.[17] Maceira et al. (2013) and Vogelsberg et al. (2015) identified a distinct global subendocardial enhancement pattern termed "amyloid LGE".[18,19] Perugini et al. also described a range of LGE patterns in amyloidosis, including transmural, subendocardial, and localized patchy patterns.[20]

In myocarditis, we observed a typical LGE distribution affecting the subepicardial layer of the myocardial wall, with normal LV function in affected patients. The subepicardial location is often associated with the inflammation's proximity to the pericardium. In our study, we found qualitative LGE CMR assessment to be particularly useful in distinguishing different causes of NICM. This non-invasive method can help avoid the need for more invasive procedures like endomyocardial biopsy, particularly in cases where LGE patterns are highly

suggestive of a specific diagnosis.[23,24] However, we encountered challenges such as unsteady breath-holding and cardiac arrhythmias, which increased noise in the images. Additionally, the longer scan times reduced patient compliance in some cases.

In summary, while LGE CMR is invaluable for diagnosing and differentiating NICM, technical and patient-related issues can affect image quality and patient cooperation during scans. Nonetheless, the technique remains an essential tool in the non-invasive evaluation of myocardial diseases.

CONCLUSION

Diagnosing nonischemic cardiomyopathies can be particularly challenging due to their often subtle and overlapping symptoms, as well as the nonspecific results from many diagnostic tests. However, late gadolinium-enhanced cardiac MRI has emerged as a valuable tool for detecting, characterizing, and differentiating these conditions. By classifying abnormal myocardial enhancement based on its precise location—whether subendocardial, transmural, subepicardial, or mesocardial—it becomes possible to distinguish between ischemic cardiomyopathy (due to heart attack) and nonischemic forms. This approach helps narrow the differential diagnosis for nonischemic cardiomyopathies. When used in conjunction with strong clinical suspicion, delayed contrast-enhanced cardiac MRI can facilitate timely identification and appropriate management of nonischemic cardiomyopathies.

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