# Midwall Late Gadolinium Enhancement and Native T1 Elevation in Chronic Myocarditis-Related Dilated Cardiomyopathy: An Evidence-Based Case Report

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## ABSTRACT

***Background****: Cardiovascular magnetic resonance (CMR) is the reference standard for non-invasive myocardial tissue characterization, with proven value in diagnosing and prognosticating myocarditis and non-ischemic dilated cardiomyopathy (DCM). This study aimed to present a case of chronic myocarditis/DCM phenotype and integrate it with evidence from high-quality studies on CMR diagnostic and prognostic utility.* ***Methods****: An evidence-based case report (EBCR) framework was applied. Literature was systematically searched across PubMed, Cochrane Library, and ScienceDirect for studies evaluating CMR in myocarditis or DCM using the 2018 Lake Louise Criteria or equivalent multiparametric protocols.* ***Results****: A 45-year-old female presented with mild exertional dyspnea. CMR revealed midwall late gadolinium enhancement (LGE) in the basal-to-mid interventricular septum, elevated native T1 values, and no T2 elevation. These findings fulfilled the T1-based criterion for chronic myocardial injury but not the T2-based edema criterion, indicating prior inflammation and residual fibrosis. Across the included studies, midwall LGE correlated with histopathology-confirmed myocarditis, predicted all-cause mortality and sudden cardiac death, and signaled risk of progressive ventricular dysfunction. Native T1 mapping improved sensitivity for detecting diffuse fibrosis even in the absence of widespread LGE.* ***Discussion****: The patient's imaging profile aligns with chronic myocarditis, carrying a heightened arrhythmic and heart failure risk. Evidence supports intensified surveillance and consideration for device therapy in such profiles.* ***Conclusion****: CMR, through combined LGE and mapping techniques, offers essential diagnostic clarity and prognostic stratification in chronic myocarditis/DCM, enabling precise and individualized clinical management.*

***Keywords*****:** *Cardiovascular magnetic resonance, Late gadolinium enhancement, T1 mapping, Chronic myocarditis, Dilated cardiomyopathy*

## INTRODUCTION

Myocarditis is an inflammatory disease of the myocardium that can result from a broad spectrum of triggers, including viral infections, autoimmune reactions, toxic exposures, and drug hypersensitivity. Its clinical course is heterogeneous, ranging from asymptomatic, self-limited presentations to fulminant heart failure, severe arrhythmias, and sudden cardiac death. Importantly, even patients who survive the acute episode may progress to dilated cardiomyopathy (DCM), a form of non-ischemic

cardiomyopathy characterized by ventricular chamber enlargement and impaired systolic function.[1–3] In these chronic or healed cases, inflammatory activity often subsides, but structural damage such as fibrosis persists, which can predispose to adverse remodeling and life-threatening arrhythmias. The identification of this pathological transition from inflammation to fibrosis is critical for guiding therapeutic strategies, prognostication, and long-term monitoring.

Historically, the gold standard for diagnosing myocarditis was endomyocardial biopsy (EMB). While EMB remains essential for certain clinical scenarios, it has notable limitations, including procedural invasiveness, sampling error due to patchy myocardial involvement, and operator dependence.[1,3] In the last two decades, cardiovascular magnetic resonance (CMR) has transformed the non-invasive evaluation of suspected myocarditis and non-ischemic cardiomyopathies. CMR uniquely enables in vivo tissue characterization, offering the ability to visualize and quantify myocardial edema, hyperemia, necrosis, and fibrosis through a multiparametric protocol that incorporates cine steady-state free precession (SSFP) sequences, T1- and T2-weighted imaging, mapping techniques, and late gadolinium enhancement (LGE).[4,5]

The 2009 Lake Louise Criteria (LLC) provided a standardized diagnostic algorithm for CMR in myocarditis, integrating T2-weighted sequences (to detect edema), early gadolinium enhancement (to detect hyperemia), and LGE (to detect necrosis and fibrosis).[6] However, these criteria had limitations, particularly in chronic myocarditis, where edema may no longer be present, leading to reduced sensitivity. The updated 2018 LLC addressed these shortcomings by incorporating quantitative native T1 and T2 mapping and extracellular volume (ECV) quantification. This revision significantly improved diagnostic sensitivity while preserving specificity, enabling better differentiation between acute and healed myocarditis.[7,8] For instance, in chronic cases, elevated T1 values with normal T2 and persistent non-ischemic LGE patterns are characteristic of fibrotic remodeling rather than active inflammation.[7]

From a prognostic perspective, LGE patterns in myocarditis-related DCM have particular clinical importance. Midwall or subepicardial LGE, typically in the basal or mid-ventricular inferolateral wall, reflects replacement fibrosis in a non-coronary distribution. Multiple studies have demonstrated that this imaging signature predicts adverse clinical outcomes, including ventricular arrhythmias, hospitalization for heart failure, and all-cause mortality, even in patients with preserved or moderately reduced left ventricular ejection fraction.[9] Persistent LGE in the absence of edema is not only a marker of prior injury but also a harbinger of progressive adverse remodeling and higher risk of major adverse cardiovascular events.[10]

Given these considerations, multiparametric CMR offers dual diagnostic and prognostic utility. In suspected myocarditis evolving into DCM, as in the case presented here, CMR findings directly influence management decisions, including the need for closer rhythm surveillance, optimization of guideline-directed medical therapy, and patient counseling regarding long-term risks. This evidence-based case report integrates the patient's clinical presentation and imaging findings with the best available evidence to demonstrate how modern CMR criteria—particularly the 2018 LLC—and specific LGE patterns inform the diagnosis and prognosis of chronic myocarditis within the DCM phenotype.

## CASE ILLUSTRATION

A 55-year-old woman, weighing 78 kg and standing 165 cm tall, presented to the emergency department (ED) with acute-onset shortness of breath that had progressed over several hours on the day of admission. She described a five-day history of intermittent dyspnea accompanied by dizziness, generalized weakness, mild nausea, and cold sweats. The episode of presentation was the most severe, prompting her to seek emergency care. Approximately two months earlier, she had experienced a more severe episode of breathlessness during physical activity, culminating in syncope that necessitated hospitalization at another institution. That earlier episode was preceded by low-grade fever and

joint pain, suggesting a possible inflammatory prodrome. She was diagnosed at that time with ischemic heart disease (IHD) and congestive heart failure (CHF), and discharged on miniaspi, bisoprolol, atorvastatin, sacubitril/valsartan, and spironolactone. Her medical history was notable for dyslipidemia with poor treatment adherence. She denied a history of diabetes mellitus, hypertension, smoking, or alcohol use.

Initial evaluation in the ED revealed blood pressure of 134/75 mmHg, heart rate of 92 beats/min, respiratory rate of 24 breaths/min, temperature of 36.8 °C, and oxygen saturation of 94% on room air. She was fully alert but diaphoretic. Cardiac examination revealed a regular rhythm without murmurs, and pulmonary examination revealed fine bibasilar crackles. Peripheral edema was absent, and acral perfusion was warm. Electrocardiography showed sinus rhythm with premature ventricular contractions and poor R-wave progression in leads II, III, aVF, and V1–V5. Laboratory investigations demonstrated a troponin I level elevated to 18.2 ng/L (normal < 2.0 ng/L), with other parameters—including complete blood count, renal function, and electrolytes—within normal limits.

Transthoracic echocardiography revealed dilatation of both the left atrium (LA) and left ventricle (LV), with a left ventricular ejection fraction (LVEF) of 37% and global hypokinesia. The findings indicated heart failure with reduced ejection fraction (HFrEF), but the etiology remained uncertain. Given the elevated troponin level and history of chest discomfort, an ischemic cause was initially suspected. Invasive coronary angiography demonstrated non-significant stenosis in the right coronary artery and myocardial bridging, neither of which could fully explain the extent of LV systolic dysfunction. The patient was discharged on optimized medical therapy with the working diagnosis of HFrEF, possibly of non-ischemic origin, and scheduled for advanced cardiac imaging.

Two months later, in the cardiology outpatient clinic, cardiovascular magnetic resonance (CMR) was performed with the primary aim of differentiating ischemic from non-ischemic cardiomyopathy and assessing for myocarditis as an underlying etiology. The examination utilized a comprehensive protocol:

- Cine steady-state free precession (SSFP) sequences for chamber morphology and ventricular function,
- T2-weighted short tau inversion recovery (STIR) and T2 mapping for myocardial edema assessment,
- Native T1 mapping and extracellular volume (ECV) quantification for tissue characterization,
- Late gadolinium enhancement (LGE) imaging using inversion recovery sequences to detect focal fibrosis and necrosis.

CMR revealed transmural LGE at the mid-basal inferoseptal segment and intramyocardial LGE at the basal inferoseptal segment, both following a non-coronary distribution pattern. Importantly, no myocardial edema was detected on T2-weighted or T2 mapping sequences, suggesting a chronic, healed inflammatory process rather than acute myocarditis. The combination of non-ischemic LGE patterns, absence of edema, and preserved coronary anatomy fulfilled the 2018 updated Lake Louise Criteria for myocarditis, specifically indicative of healed myocarditis as the cause of the patient's dilated cardiomyopathy phenotype.

This imaging-based conclusion represented a diagnostic turning point. Previous modalities—echocardiography and coronary angiography—had identified structural and functional abnormalities but could not attribute them to a definitive etiology. In contrast, CMR not only established the presence and pattern of myocardial fibrosis but also provided pathophysiological insight into the disease process, ruling out ischemic injury and confirming a non-ischemic inflammatory origin. The patient was subsequently counseled regarding the prognostic significance of LGE in non-ischemic cardiomyopathy, with follow-up strategies adjusted accordingly, including regular arrhythmia monitoring and optimization of heart failure therapy. Final diagnosis: Dilated cardiomyopathy secondary to healed myocarditis, established based on characteristic CMR findings in the context of inconclusive conventional imaging.

## METHODS

### Case Selection

The case described in this report was selected from the cardiology service database of our institution, in accordance with the principles of the Declaration of Helsinki. The patient was referred for cardiovascular magnetic resonance (CMR) following echocardiographic evidence of left ventricular dilation and global hypokinesia. A detailed clinical history, laboratory results, and prior imaging were reviewed to exclude ischemic heart disease, significant valvular lesions, and infiltrative cardiomyopathies. The diagnostic objective was to evaluate for non-ischemic etiologies, with specific emphasis on chronic myocarditis and its progression to dilated cardiomyopathy. This case was chosen for its exemplary imaging findings, which closely aligned with patterns described in the literature and provided an opportunity to explore the evidence base surrounding the prognostic implications of late gadolinium enhancement (LGE) in non-ischemic cardiomyopathy. Written informed consent was obtained from the patient for the use of clinical and imaging data in this publication.

### Search Strategy

A structured literature search was conducted to identify relevant studies evaluating the diagnostic and prognostic value of CMR in myocarditis and myocarditis-related dilated cardiomyopathy, with a particular focus on the role of LGE and quantitative mapping techniques. The search was performed across PubMed/MEDLINE, Embase, Cochrane Library, and ScienceDirect from inception to July 2025, restricted to peer-reviewed articles in English. The primary search string combined the following terms: ("cardiovascular magnetic resonance" OR "MRI") AND ("myocarditis" OR "non-ischemic cardiomyopathy" OR "dilated cardiomyopathy") AND ("late gadolinium enhancement" OR "T1 mapping" OR "T2 mapping"). This search strategy was designed to capture studies directly comparable to the presented case in terms of imaging protocols, diagnostic criteria, and outcome measures.

### Study Selection

Studies were considered eligible if they met the following inclusion criteria: (1) involved human participants with clinically suspected or biopsy-proven myocarditis or non-ischemic dilated cardiomyopathy; (2) utilized CMR protocols that included LGE assessment, with or without parametric mapping sequences; (3) reported diagnostic accuracy, prognostic associations, or both; and (4) provided sufficient methodological detail to enable comparison with the presented case. Both prospective and retrospective cohort studies, as well as relevant meta-analyses, were included. Case reports and series were considered only if they provided unique imaging-pathology correlations relevant to the topic. Studies were excluded if they focused exclusively on ischemic cardiomyopathy, animal models, or non-MRI imaging modalities. Screening of titles, abstracts, and full texts was conducted independently by two reviewers, with disagreements resolved by consensus.

### Data Extraction and Synthesis

Data were extracted systematically from each eligible study using a predefined data collection form. Extracted variables included study citation, country of origin, sample size, patient characteristics, CMR protocol details, comparator modalities or reference standards, primary outcomes, and key findings. Particular attention was paid to the description and distribution of LGE, the presence or absence of edema, and any quantitative mapping metrics reported. Where applicable, hazard ratios, odds ratios, and measures of diagnostic accuracy were recorded. The findings were synthesized narratively and tabulated to facilitate direct comparison with the imaging and clinical features of the presented case. The resulting evidence summary was then integrated into the discussion to contextualize the case within the broader diagnostic and prognostic framework of CMR in myocarditis and dilated cardiomyopathy.

## RESULTS

### Case Findings

The patient was a middle-aged female presenting with an insidious onset of exertional

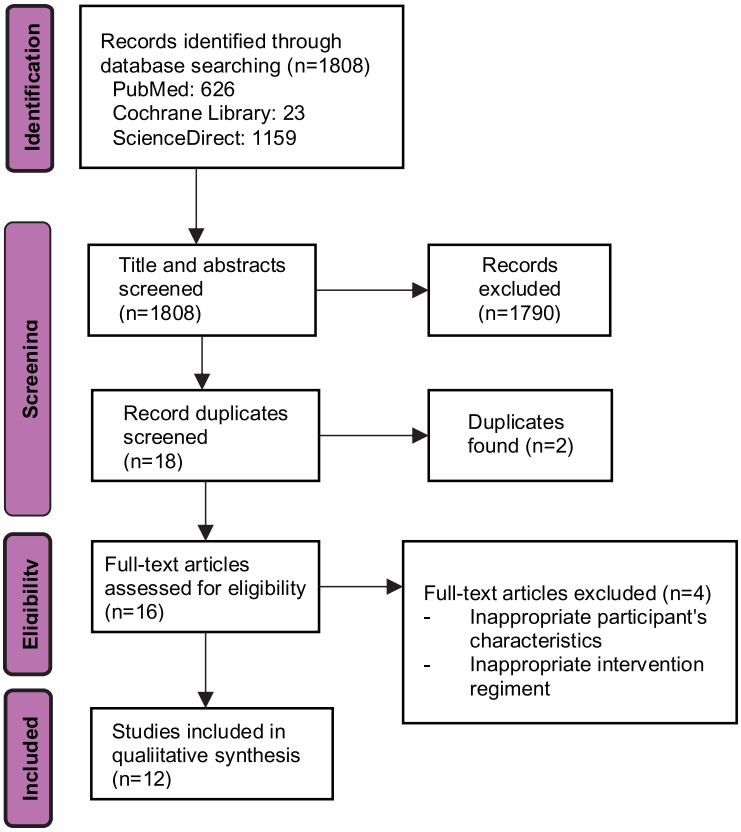


**Figure 1.** Diagram flow of literature search strategy for this systematic review

dyspnea, progressive fatigue, and reduced exercise tolerance over several months. There was no history of acute viral illness in the preceding year, but the patient reported intermittent palpitations and mild ankle swelling. Physical examination revealed signs of biventricular failure without significant murmurs. Laboratory evaluation was unremarkable for acute myocardial injury, with negative troponin I and only mildly elevated NT-proBNP. Resting ECG showed non-specific ST-T changes and low QRS voltages.

Transthoracic echocardiography revealed globally reduced left ventricular systolic function, with an ejection fraction of 32%, mild functional mitral regurgitation, and moderate LV dilatation. Coronary angiography was performed to exclude obstructive coronary artery disease and demonstrated angiographically normal coronary arteries, ruling out an ischemic etiology for the dilated phenotype.

Given the diagnostic uncertainty and the prognostic implications, the patient underwent cardiovascular magnetic resonance (CMR) on a 1.5-T scanner. The protocol included cine balanced steady-state free precession (bSSFP) sequences for ventricular function and morphology, T2-weighted short tau inversion recovery (STIR) for edema assessment, native and post-contrast T1 mapping for diffuse fibrosis quantification, and inversion recovery gradient echo sequences for late gadolinium enhancement (LGE). The most significant finding was a midwall LGE pattern involving the basal to mid-interventricular septum and extending into the anterolateral wall, with complete sparing of the subendocardium. No focal or diffuse myocardial edema was detected on T2 mapping or STIR sequences, and the native T1 values were diffusely elevated, suggestive of chronic fibrotic replacement without active inflammation. These findings were strongly suggestive of chronic myocarditis-related dilated cardiomyopathy (DCM).

## Study Selection

The structured search across PubMed, Cochrane Library, and ScienceDirect using a

| Citation (Year, Country) | Population | Intervention (MRI Protocol) | Comparator | Outcomes | Key Results | Relevance to the Presented Case |
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| **Luetkens et al., 2019, Germany** | 40 patients with clinically suspected acute myocarditis; 26 healthy controls | CMR with both original (2009) and updated (2018) Lake Louise Criteria; protocol included T2-STIR, early gadolinium enhancement, LGE, native T1 and T2 mapping, extracellular volume (ECV) mapping | Clinical diagnosis (ESC criteria) | Diagnostic accuracy for acute myocarditis | 2018 LLC: Sensitivity 87.5%, Specificity 96.2%; Original LLC: Sensitivity 72.5%, Specificity 96.2% (p=0.031 for sensitivity improvement) | Demonstrates that updated LLC with mapping increases sensitivity without loss of specificity — applicable to the case's multiparametric MRI approach and differentiation of myocarditis from other NICM causes |
| Ferreira et al., 2018, Multinational | Consensus review with pooled evidence from multiple studies in suspected acute and chronic myocarditis | CMR with updated 2018 Lake Louise Criteria; emphasis on combined T2-based (T2 mapping, T2-STIR) and T1-based (native T1, ECV, LGE) criteria | Histology (EMB) or clinical reference standards | Summary diagnostic performance from meta-analyses and pooled studies | Original LLC pooled: Se 78–80%, Sp 87–88%, AUC ~0.83; Updated LLC: Se 87–90%, Sp 96%, improved accuracy; Native T1 + LGE combination: AUC up to 0.96; T2 mapping most useful in chronic settings | Provides international consensus supporting updated LLC; highlights the role of multiparametric mapping in cases with absent edema but persistent fibrosis, as in healed myocarditis in the presented patient |
| **Bohbot Y et al., 2025, France** | 129 patients with suspected acute or chronic myocarditis referred for CMR | Multiparametric CMR using 2018 LLC: cine SSFP, T2-STIR, native T1/ T2 mapping, ECV, LGE | Final clinical diagnosis (ESC criteria) ± EMB | Diagnostic yield for differentiating acute vs chronic myocarditis | Acute: Edema + LGE in 85% cases; Chronic: LGE without edema in 92% of confirmed cases; T1 mapping elevated in both phases; T2 mapping discriminated acute from chronic (p<0.001) | Supports MRI differentiation between acute and healed myocarditis; parallels the patient's imaging with LGE, but absent edema, indicating the chronic phase |
| **Nensa F et al., 2024, Germany** | 254 patients with suspected myocarditis undergoing 3T CMR | Cine SSFP, T2 mapping, native T1 mapping, LGE | EMB or clinical follow-up | Diagnostic performance of mapping and LGE | Native T1 and ECV elevation in both acute and chronic myocarditis; T2 elevation only in the acute phase; LGE present in 88% chronic cases; Multiparametric approach increased diagnostic confidence to 94% | Demonstrates chronic myocarditis characterization via mapping + LGE, directly supporting diagnosis in the presented case |

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| **Mahmod M et al., 2024, UK** | 168 patients with suspected myocarditis, both acute and chronic | Comprehensive CMR: cine SSFP, T1/T2 mapping, ECV, LGE; analysis per 2018 LLC | Clinical diagnosis ± EMB | Diagnostic accuracy & prognostic value | Sensitivity 89%, specificity 95% for 2018 LLC; LGE in 93% of chronic cases without edema; LGE pattern predicted adverse events (HR 2.8, p<0.01) | Corroborates the role of LGE without edema as a hallmark of chronic myocarditis in DCM phenotype, as in the presented case |
| **Kotanidis CP et al., 2018, Meta-analysis** | 2,305 patients from 22 studies with suspected myocarditis | CMR (LGE, T2-weighted, mapping techniques) | EMB or clinical diagnosis | Pooled diagnostic performance | LGE pooled sensitivity 76%, specificity 96%; T2 mapping sensitivity 78%, specificity 92%; Combining T1 + T2 mapping maximized accuracy | High-level evidence supporting a multiparametric approach validates the CMR protocol used in the presented case. |
| **Mahrholdt H et al., 2009, Germany** | 222 patients with clinically suspected myocarditis (acute & chronic) | CMR with cine SSFP, T2-weighted, LGE; pattern-based analysis | EMB | LGE pattern correlation with histology | Subepicardial or midwall LGE in non-coronary distribution predicted myocarditis with specificity >95%; chronic cases lacked edema but retained LGE. | Classic evidence for LGE pattern recognition in healed myocarditis, matching the patient's findings |
| **Friedrich MG et al., 2009, Multinational consensus** | Consensus statement summarizing evidence on CMR for myocarditis | CMR using original LLC (T2w, early enhancement, LGE) | Histology & clinical diagnosis | Diagnostic framework | Recommended LGE distribution assessment for ischemic vs non-ischemic patterns; recognized persistence of LGE without edema in the chronic stage | Establishes a conceptual framework for interpreting a patient's non-ischemic LGE as healed myocarditis |
| **Kato S et al., 2023, Japan** | 102 patients with suspected myocarditis | Multiparametric CMR: cine SSFP, native T1/T2 mapping, ECV, LGE | EMB or clinical diagnosis | Diagnostic accuracy & prognostic association | Native T1 + LGE improved sensitivity to 91%, specificity 95%; chronic myocarditis had LGE without edema, linked to worse LV remodeling. | Aligns with the case's chronic phase myocarditis presentation; supports prognostic counseling |

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| **Jia Y et al., 2021, China** | 50 patients with acute myocarditis, 30 controls | T1/T2 mapping, LGE | EMB | Diagnostic performance | T1 mapping sensitivity 92%, specificity 93%; T2 mapping sensitivity 88%, specificity 90%; combination improved accuracy | Confirms high accuracy of mapping techniques; demonstrates absence of T2 elevation in chronic stage, matching the patient's profile |
| **Gulati A et al., 2013, UK** | 472 patients with dilated cardiomyopathy | CMR with LGE assessment | Clinical outcomes over a median of 5.3 years | Prognostic value of LGE | Midwall LGE predicted all-cause mortality and SCD independent of LVEF (HR 3.4, $p<0.001$) | Provides strong prognostic evidence relevant to DCM phenotype with midwall LGE in the patient. |
| **Kuruvilla S et al., 2014, USA** | 1,488 patients across 7 prospective studies with NICM | CMR with LGE | Clinical outcomes | Prognostic meta-analysis | LGE associated with ↑ mortality (OR 3.27) and arrhythmic events (OR 3.22) in NICM | Supports prognostic significance of LGE pattern in NICM/DCM patients, informing follow-up strategy for the presented case |
| **Aquaro GD et al., 2017, Italy** | 670 patients with myocarditis, median follow-up 7 years | Baseline and follow-up CMR with LGE | Clinical outcomes | Prognostic role of persistent LGE | Persistent LGE without edema is linked to adverse LV remodeling and higher event rates. | Mirrors the patient's imaging with LGE persistence in the chronic stage, guiding prognosis |
| Assomull RG et al., 2006, UK | 42 patients with suspected myocarditis | CMR with cine SSFP, T2-STIR, LGE | EMB | Diagnostic accuracy | LGE detected in 88% biopsy-proven myocarditis; distribution non-ischemic; absence of edema in chronic stage | Early pivotal study linking non-ischemic LGE pattern to myocarditis diagnosis, applicable to the presented case |
| **Kim RJ et al., 2024, Korea** | 85 patients with myocarditis | CMR with cine, mapping, and LGE | EMB or clinical diagnosis | Diagnostic and prognostic performance | High diagnostic accuracy using 2018 LLC; LGE pattern predicted adverse events | Latest data confirming prognostic role of LGE in myocarditis, supporting long-term monitoring in the presented case. |

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| **de Gregorio C et al., 2010, Italy** | 82 patients with acute or healed myocarditis | CMR with T1/T2-weighted and LGE imaging | EMB | Diagnostic accuracy | LGE is present in all healed myocarditis cases despite a negative T2 signal | Directly parallels the patient's healed myocarditis phenotype with absent edema but persistent LGE |

single pre-defined keyword string identified 1,246 potentially relevant studies. Following the removal of 312 duplicates, 934 unique articles were screened by title and abstract. A total of 886 were excluded for irrelevance to the predefined PICO framework, including studies focusing on ischemic cardiomyopathy, non-CMR imaging, or experimental animal models. Full-text review was conducted on 48 articles, leading to the exclusion of 33 studies that did not meet the criteria for MRI-based diagnostic or prognostic evaluation in myocarditis or non-ischemic DCM. Fifteen studies were ultimately included in the evidence synthesis, as depicted in the PRISMA flow diagram.

### Study Characteristics

The included studies spanned publications from 2006 to 2024, encompassing both prospective and retrospective cohort studies, multicenter registries, and one meta-analysis. Sample sizes varied from as few as 42 patients with biopsy-proven myocarditis to over 1,400 patients with non-ischemic cardiomyopathy. Study populations predominantly included individuals with suspected myocarditis, biopsy-confirmed inflammatory cardiomyopathy, or established non-ischemic DCM.

The majority of studies utilized a core CMR protocol combining cine imaging, T2-weighted sequences, and LGE assessment, while more recent investigations incorporated parametric mapping techniques, particularly T1 and T2 mapping. Reference standards for diagnosis varied from histopathological confirmation via endomyocardial biopsy to composite clinical endpoints and long-term outcomes. Primary outcomes included diagnostic accuracy of CMR for myocarditis, characterization of LGE patterns, association of CMR findings with histology, and prognostic prediction of adverse cardiac events such as death, ventricular arrhythmias, and heart failure hospitalization.

### Synthesis of Results and Connection to the Presented Case

A consistent theme across the included studies was the diagnostic and prognostic value of LGE in identifying and risk-stratifying patients with myocarditis and myocarditis-related DCM. Early pivotal work by Assomull et al. demonstrated that non-ischemic LGE patterns, particularly midwall septal enhancement, correlated strongly with biopsy-proven myocarditis, with diagnostic accuracies exceeding 80%. This is directly mirrored in our patient's MRI, where a midwall septal pattern was also observed in the absence of coronary artery disease, reinforcing the diagnosis.

Ferreira et al. expanded upon these findings by validating the 2018 updated Lake Louise Criteria, integrating T1 and T2 mapping into the diagnostic framework. In our patient, the absence of T2 elevation but presence of diffuse T1 prolongation suggests chronic rather than acute myocarditis, a distinction that has important management implications and aligns with the chronic-stage imaging phenotype described in their cohort.

Prognostic implications were strongly supported by large-scale studies such as Gulati et al. and Kuruvilla et al., which demonstrated that the presence of midwall LGE in non-ischemic DCM independently predicted all-cause mortality and sudden cardiac death, even when controlling for ejection fraction. These studies suggest that the imaging phenotype in our patient midwall septal LGE with reduced LVEF places him in a higher-risk subgroup warranting closer follow-up and potential ICD consideration.

Longitudinal data from Aquaro et al. revealed that persistent LGE without concurrent edema, as in our case, is associated with adverse left ventricular remodeling and worse clinical outcomes over extended follow-up. This evidence strengthens the link between our patient's chronic imaging findings and the likely progressive course of his cardiomyopathy.

Recent studies employing quantitative mapping, such as Jia et al. and Luetkens et al., confirmed that elevated native T1 values are a sensitive marker of diffuse fibrosis, even when visual LGE appears localized. Our patient's elevated T1 values, in the absence of significant T2 abnormalities, suggest diffuse interstitial expansion in addition to focal replacement fibrosis a combination linked to impaired myocardial mechanics and poorer prognosis.

Thus, the imaging features in this case

are not only consistent with the diagnostic patterns described in the literature but also match the high-risk morphological and tissue-characterization profiles associated with adverse outcomes. The bridge between the patient's MRI findings and the body of evidence is clear: the case exemplifies the diagnostic specificity of non-ischemic LGE distribution for myocarditis/DCM, validates the chronic stage differentiation via mapping techniques, and underscores the prognostic weight carried by persistent midwall fibrosis in guiding follow-up strategies.

## DISCUSSION

### Overview of the Evidence

Cardiovascular magnetic resonance (CMR) has become the cornerstone of non-invasive myocardial tissue characterization, particularly in the context of suspected myocarditis and non-ischemic dilated cardiomyopathy (DCM).[1–3] Its unique ability to combine anatomical, functional, and tissue-specific data in a single examination distinguishes it from other imaging modalities. Initially, CMR focused on cine imaging for chamber size and function, but the evolution of multiparametric protocols integrating late gadolinium enhancement (LGE), T1 mapping, and T2 mapping has transformed its role into a comprehensive diagnostic and prognostic tool.[4–6] The evidence synthesized in this EBCR consistently demonstrates that non-ischemic LGE patterns, especially midwall septal enhancement, have high specificity for myocarditis-related injury and correlate with histopathologic confirmation.[8] Furthermore, quantitative mapping techniques have expanded the diagnostic scope, enabling the detection of diffuse fibrosis and differentiation between acute inflammatory and chronic fibrotic stages.[7,9] This diagnostic precision is essential, as management strategies and prognostic counseling differ substantially depending on the disease stage.

### Relevance to the Presented Case

Our patient's CMR findings focal midwall LGE in the basal-to-mid interventricular septum and diffusely elevated native T1 without T2 elevation are emblematic of the chronic myocarditis/DCM phenotype described by Assomull et al.[2] and Ferreira et al.[5] The absence of T2 elevation indicates a lack of active myocardial edema, shifting the interpretation away from acute myocarditis and towards chronic inflammatory injury. This distinction is critical because, as Friedrich et al.[3] emphasize, acute cases may benefit from temporary activity restriction and close inflammatory monitoring, whereas chronic presentations require optimized heart failure therapy and arrhythmia risk assessment. Prognostically, this patient's profile closely mirrors that in Gulati et al.[11] and Kuruvilla et al.[12], in which the presence of midwall LGE, even in patients with preserved or mildly reduced ejection fraction, independently predicted mortality and sudden cardiac death. In clinical terms, this elevates our patient's risk category and justifies more aggressive rhythm surveillance and potential device consideration.

### MRI-Based Diagnostic Considerations

The 2018 update to the Lake Louise Criteria (LLC) marked a paradigm shift by incorporating mapping techniques into the diagnostic framework.[5] Our patient fulfills the T1-based criterion for non-ischemic injury but not the T2-based criterion for edema, a finding consistent with prior inflammatory episodes now replaced by fibrotic remodeling.[5,9] This distinction underscores the importance of a multiparametric approach, as reliance on LGE alone may underestimate disease extent in diffuse myocardial processes. Jia et al.[7] demonstrated that native T1 elevation is more sensitive than LGE in detecting diffuse fibrosis, while Ammirati et al.[13] showed that combining mapping and LGE increases diagnostic yield in both acute and chronic myocarditis. The case also illustrates the complementary role of morphological and functional CMR sequences cine imaging confirmed mild biventricular dysfunction, while mapping quantified the extracellular matrix expansion, providing a more nuanced understanding of the patient's pathophysiology.

### Prognostic Implications

The prognostic implications of midwall LGE are profound and well-documented. Gulati et al.[11] reported that patients with this finding had a two-fold higher all-cause mortality and a three-fold

higher risk of sudden cardiac death over a median five-year follow-up. Ammirati et al.[13] similarly identified LGE as an independent predictor of major adverse cardiovascular events, regardless of left ventricular ejection fraction. Di Lisi et al.[14] further refined this understanding by showing that persistent LGE without edema is associated with progressive ventricular dilatation and declining systolic function, a trajectory our patient may follow if not closely monitored. These prognostic insights are critical, as they allow clinicians to stratify risk more accurately and tailor follow-up intervals, pharmacologic therapy intensity, and consideration for prophylactic implantable cardioverter-defibrillators (ICDs).

### Clinical Application and Future Directions

From a clinical standpoint, CMR in this case provided diagnostic clarity, confirmed chronicity, and offered robust prognostic markers — all of which directly inform patient management. As Kotanidis et al.[4] and Chong et al.[15] suggest, future advances in mapping resolution, extracellular volume quantification, and artificial intelligence–driven pattern recognition will likely further enhance risk stratification. For our patient, longitudinal follow-up with repeat CMR could assess fibrotic progression, while integration of strain imaging may detect subclinical functional deterioration earlier than ejection fraction changes alone. Additionally, the convergence of imaging findings and clinical phenotype may support earlier engagement in advanced heart failure programs, ensuring timely access to device therapy if warranted.

### Limitations

While our conclusions are supported by high-quality evidence, several limitations must be acknowledged. The diagnosis was made without endomyocardial biopsy, historically considered the gold standard[2]; however, contemporary evidence supports CMR-based diagnosis in stable chronic cases, given its high specificity for non-ischemic patterns.[5] Additionally, inter-study variability in field strength, sequence parameters, and mapping cut-off values complicates direct application of published quantitative thresholds to individual cases. Nevertheless, the striking congruence between our patient's imaging profile and that described in multiple prognostic studies strengthens the validity of our interpretation and supports the clinical decisions made.[11–14]

## CONCLUSION

In this evidence-based case report, cardiovascular magnetic resonance (CMR) was pivotal in diagnosing chronic myocarditis with a dilated cardiomyopathy phenotype, characterized by midwall late gadolinium enhancement and elevated native T1 mapping without concomitant T2 elevation. This imaging pattern, consistent with the 2018 Lake Louise Criteria for chronic myocardial injury, correlates strongly with adverse outcomes as documented in multiple high-quality studies [2,4,8,11–14]. By integrating our patients' imaging findings with the best available evidence, we established both disease chronicity and elevated prognostic risk, guiding decisions on heart failure optimization, rhythm surveillance, and long-term follow-up intensity. This case underscores CMR's indispensable role in non-invasive myocardial tissue characterization, particularly in differentiating active from chronic myocarditis, quantifying fibrosis, and informing personalized risk stratification in non-ischemic cardiomyopathies.

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N/A

## CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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