E-ISSN: 3078-4514 | P-ISSN: 3105-1316
**Vol. 2, No. 2, 2025**
**DOI: 10.71295/JHDT.2025.02.2.05**

LITERATURE REVIEW

# Advanced Tissue and Functional Cardiac MRI Markers for Risk Stratification in Dilated Cardiomyopathy

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**Received:** 27/11/2025 **Revised:** 02/12/2025 **Accepted:** 06/12/2025

## Abstract

Dilated cardiomyopathy (DCM) is characterized by broad clinical heterogeneity, ranging from stable disease to rapid progression and arrhythmic events. Advanced cardiac magnetic resonance (CMR) techniques—including native T1 mapping, extracellular volume fraction (ECV), late gadolinium enhancement (LGE), and feature-tracking strain—provide unique insights into myocardial tissue composition and mechanical function.

Native T1 and ECV quantify diffuse interstitial fibrosis, while LGE detects replacement fibrosis; together, they refine risk assessment beyond conventional measures. Myocardial strain, including left ventricular (LV) global longitudinal strain (GLS) and left atrial (LA) reservoir strain, captures early mechanical dysfunction and offers independent prognostic value.

This review synthesizes current evidence on the prognostic utility of these biomarkers in DCM and highlights opportunities for multiparametric CMR integration into personalized risk stratification strategies.

**Keywords:** Dilated Cardiomyopathy, Cardiac Magnetic Resonance, Prognosis, T1 Mapping, Extracellular Volume, Myocardial Strain, Fibrosis.

## Introduction

Dilated cardiomyopathy (DCM) remains one of the leading causes of heart failure and a major indication for cardiac transplantation. Despite advances in therapy, its clinical course is highly variable: some patients undergo substantial reverse remodeling, while others deteriorate rapidly or experience malignant arrhythmias. This heterogeneity underscores the need for refined, individualized risk stratification.

Left ventricular ejection fraction (LVEF) traditionally serves as the cornerstone of prognostic assessment, yet it fails to capture the complexity of myocardial injury. Patients with similar LVEF values may follow vastly different trajectories, reflecting the inadequacy of functional measures alone.

Cardiac magnetic resonance (CMR) has revolutionized the evaluation of cardiomyopathies by combining precise volumetric assessment with tissue characterization. Late gadolinium enhancement (LGE) identifies focal replacement fibrosis—an important prognostic marker—but does not adequately detect diffuse interstitial fibrosis, which plays a central role in disease progression.

The development of T1 mapping and extracellular volume fraction (ECV) quantification permits detection of diffuse fibrosis, offering prognostic insights beyond LGE. Elevated native T1 and increased ECV correlate with adverse events, including arrhythmias, heart-failure progression, and mortality. Meanwhile, feature-tracking CMR enables measurement of myocardial strain, particularly LV GLS and LA reservoir strain, which reflect early mechanical dysfunction.

Together, T1 mapping, ECV, and strain form a multiparametric, biologically grounded framework for evaluating DCM. This review synthesizes current evidence on their prognostic significance and discusses how these complementary biomarkers may be integrated into future clinical decision-making.

### *Pathophysiology Of Dilated Cardiomyopathy*

Dilated cardiomyopathy (DCM) arises from a broad spectrum of genetic, inflammatory, metabolic, and toxic insults, yet these diverse triggers converge on a common sequence of structural and cellular alterations. Regardless of etiology, the disease is characterized by progressive ventricular

dilation, impaired systolic function, and increasing electrical instability. Understanding these biological processes provides essential context for interpreting advanced CMR-derived biomarkers.

### *Structural Remodeling and Chamber Dilation*

The fundamental morphological feature of DCM is left ventricular dilation accompanied by declining systolic performance. Myocyte injury, cytoskeletal disruption, and extracellular matrix changes collectively weaken the structural integrity of the myocardium. As wall stress increases, neurohormonal pathways—including the renin–angiotensin–aldosterone and sympathetic systems—become chronically activated, driving further dilation and promoting a more spherical ventricular configuration [1,2]. These changes impair mechanical efficiency and perpetuate a cycle of adverse remodeling. In advanced disease, the right ventricle often becomes involved, contributing significantly to symptoms and poorer outcomes.

### *Myocyte Injury and Cellular Remodeling*

Myocyte dysfunction results from impaired mitochondrial energetics, oxidative stress, abnormal calcium handling, and downregulated β-adrenergic signaling [3]. These processes weaken contractile performance while disrupting electrical coordination. Progressive myocyte loss through apoptosis and necrosis disrupts myocardial architecture, accelerates mechanical decline, and establishes the substrate for replacement fibrosis. Cellular remodeling of this kind not only reduces contractile reserve but also heightens arrhythmic susceptibility.

### *Interstitial and Replacement Fibrosis*

Fibrosis is a hallmark of DCM, manifesting in both diffuse interstitial and focal replacement forms, each with distinct implications for mechanical and electrical function.

### *Interstitial (Diffuse) Fibrosis*

Diffuse fibrosis reflects chronic activation of fibroblasts and expansion of the extracellular matrix. This process increases ventricular stiffness, impairs diastolic filling, and reduces myocardial compliance [4]. It often evolves early in the disease course and may precede overt systolic dysfunction.

### *Replacement Fibrosis*

Replacement fibrosis forms in regions of irreversible myocyte loss. Its characteristic mid-wall distribution in nonischemic cardiomyopathy is strongly associated with ventricular arrhythmias, heart failure progression, and mortality [5]. The coexistence of diffuse and replacement fibrosis shapes both the mechanical and electrophysiological properties of the failing heart.

### *Ventricular–Atrial Interaction and Atrial Remodeling*

Sustained elevation of left ventricular filling pressures promotes left atrial (LA) dilation, increased wall stress, and progressive structural remodeling. Atrial fibrosis, often subtle and diffuse, leads to impaired reservoir and conduit function and contributes to the development of atrial fibrillation [6]. Changes in LA function closely reflect chronic hemodynamic burden and provide prognostic information independent of ventricular indices.

### *Electrical Instability and Arrhythmic Risk*

Structural remodeling, myocyte loss, and fibrosis disrupt normal conduction pathways, creating regions of slow conduction and electrical heterogeneity. These conditions favor re-entry and increase susceptibility to ventricular tachyarrhythmias and sudden cardiac death [7]. Abnormal calcium handling, impaired repolarization, and heightened sympathetic tone amplify this risk. The combination of structural and electrophysiological changes contributes significantly to the morbidity and mortality burden of DCM.

### *T1 Mapping In Dilated Cardiomyopathy*

T1 mapping is a core component of contemporary cardiac magnetic resonance (CMR) assessment in dilated cardiomyopathy (DCM). Native T1 values reflect alterations in tissue composition, including diffuse interstitial fibrosis—the dominant pathological substrate in DCM. Multiple validation studies have demonstrated strong correlations between native T1 and histological collagen volume fraction, establishing T1 as a reliable non-invasive marker of diffuse fibrosis [10,14].

In nonischemic DCM cohorts, elevated native T1 has consistently been associated with adverse ventricular remodeling. Higher T1 values correlate with increased LV volumes, reduced systolic function, impaired global longitudinal strain, and elevated natriuretic peptide levels [8,10]. These associations underscore the pathophysiological link between diffuse fibrotic expansion and mechanical dysfunction.

The prognostic significance of T1 mapping is well documented. Puntmann et al. demonstrated that elevated native T1 independently predicts all-cause mortality and heart failure hospitalization in nonischemic DCM, even after adjusting for LGE and LVEF [8]. Similarly, Cadour et al. showed that both T1 and ECV independently predict arrhythmic events and heart-failure–related outcomes, with significantly higher event rates in patients with elevated T1 values [9]. These findings indicate that native T1 detects high-risk phenotypes that may not be captured by traditional markers such as LVEF or LGE.

Although LGE remains a robust predictor of malignant arrhythmic events, it primarily identifies focal replacement fibrosis. T1 mapping complements LGE by revealing diffuse fibrosis that often precedes the appearance of mid-wall scar. Studies comparing the two techniques show that combining native T1 with LGE provides superior prognostic stratification compared with LGE alone [8,13,25].

Despite its strengths, interpretation of T1 mapping requires careful attention to methodological variability. Native T1 values differ across vendors, field strengths, and sequences such as MOLLI or ShMOLLI; thus, absolute cut-offs cannot be universally applied [14,15]. Nevertheless, relative elevations within center-specific reference ranges remain highly reproducible and clinically meaningful.

In summary, native T1 mapping offers a quantitative assessment of diffuse interstitial fibrosis and provides strong, independent prognostic information in DCM. When integrated with ECV, strain, and LGE, T1 mapping significantly enhances risk stratification across a broad range of disease severity [8–11,13].

### *Extracellular Volume (Ecv) In Dilated Cardiomyopathy*

Extracellular volume (ECV) quantification provides a direct measure of diffuse interstitial fibrosis and is one of the most robust CMR biomarkers in dilated cardiomyopathy (DCM). Numerous validation studies have shown that ECV strongly correlates with histological collagen volume fraction and is less affected by sequence or vendor variability compared with native T1 [14,15]. In healthy individuals, ECV typically ranges from 23–27%, whereas DCM patients frequently exhibit substantially higher values, reflecting progressive extracellular matrix expansion [9,11].

Elevated ECV is closely linked to structural and functional abnormalities in DCM. Higher ECV values correlate with increased LV volumes, reduced ejection fraction, impaired global longitudinal and circumferential strain, and elevated natriuretic peptides [9,10]. Because ECV quantifies diffuse fibrosis rather than focal scar, it captures myocardial involvement that often extends beyond areas visible on LGE imaging, providing complementary information on disease severity [13,25].

The prognostic significance of ECV is well established. Cadour et al. demonstrated that ECV independently predicts heart-failure–related events and arrhythmic outcomes, with values above approximately 32% identifying high-risk phenotypes [9]. Similarly, Vita et al. showed that ECV improves prediction of heart failure events beyond LGE and conventional CMR parameters [13]. Barison et al. also reported that increased ECV was independently associated with adverse outcomes despite optimal medical therapy [11]. Notably, several studies show that ECV predicts events even in the absence of LGE, underscoring its value in identifying diffuse disease not detectable by conventional scar imaging [9,11,13].

Although more standardized than native T1, ECV measurement is not free from methodological limitations. Accurate hematocrit measurement is essential, and variability in contrast agent type or timing of post-contrast imaging can influence results [14]. Acute inflammation or edema may transiently elevate ECV, requiring clinical context for proper interpretation.

Overall, ECV is a powerful prognostic marker in DCM. By quantifying diffuse interstitial fibrosis, it provides information that complements both native T1 and LGE and improves risk stratification across a wide spectrum of disease severity [8–11,13,25].

### *Myocardial Strain Imaging In Dilated Cardiomyopathy*

Myocardial strain analysis has emerged as a highly sensitive tool for detecting early mechanical dysfunction in dilated cardiomyopathy (DCM). Feature-tracking cardiac magnetic resonance (CMR-FT) allows deformation analysis using standard cine images without the need for dedicated tagging sequences. This technique is robust and easily applicable across a range of clinical settings [19].

In DCM, global longitudinal strain (GLS) is consistently impaired even when left ventricular ejection fraction (LVEF) remains relatively preserved. Several studies have shown that reduced GLS reflects early disruption of subendocardial fiber mechanics and correlates with diffuse interstitial fibrosis measured by T1 and extracellular volume (ECV) mapping [16,20]. GLS also correlates with biomarkers such as NT-proBNP and is closely linked to NYHA functional class, underscoring its sensitivity to early hemodynamic stress [16].

The prognostic significance of CMR-derived strain is well established. In a large multicenter cohort, Romano et al. demonstrated that GLS was a powerful independent predictor of all-cause mortality in patients with ischemic and non-ischemic DCM, incremental to LVEF and late gadolinium enhancement (LGE) [17]. Buss et al. similarly showed that GLS provides superior risk discrimination compared with conventional volumetric indices, with impaired GLS associated with a significantly higher risk of major adverse cardiac events [16]. More recent findings from Tang et al. confirmed that GLS independently predicts heart-failure hospitalization and mortality in DCM, emphasizing its utility in contemporary risk stratification [18].

Beyond left ventricular deformation, atrial strain parameters have gained increasing attention. Left atrial (LA) reservoir and conduit strain reflect chronic elevation of filling pressures and atrial myopathy. Xiang et al. demonstrated that combining LA strain with LV and RV strain improves prediction of clinical events and offers incremental value beyond LGE and LVEF [21]. Raafs et al. further reported that LA strain may outperform LV GLS and even LGE in predicting adverse outcomes, suggesting that atrial mechanical impairment captures disease severity earlier in the remodeling cascade [22]. In addition, Chen et al. showed that LA strain rate provides independent prognostic information and may identify patients at risk for rapid progression [23].

Strain imaging therefore complements mapping and LGE by quantifying functional consequences of structural disease. When integrated with T1, ECV, and LGE, GLS and LA

strain enhance risk stratification, helping identify patients at high risk for arrhythmic events, worsening heart failure, or poor response to therapy [16–23].

### *Multiparametric CMR Integration In Dilated Cardiomyopathy*

The integration of multiple cardiac magnetic resonance (CMR) biomarkers—native T1 mapping, extracellular volume (ECV), late gadolinium enhancement (LGE), and myocardial strain—has significantly improved the characterization and prognostication of dilated cardiomyopathy (DCM). Each parameter reflects a distinct aspect of myocardial pathology, and their combined use provides a more comprehensive assessment than any single marker alone.

LGE remains a powerful predictor of sudden cardiac death and all-cause mortality in non-ischemic DCM, particularly when mid-wall fibrosis is present [25]. However, LGE primarily detects focal replacement fibrosis and often underestimates diffuse interstitial fibrosis, which is common in DCM. Native T1 and ECV mapping overcome this limitation by quantifying diffuse matrix expansion across the entire myocardium, capturing early pathological changes that precede LGE manifestation [8,9,14,15].

Multiple studies have shown that T1 and ECV provide prognostic information independent of and additive to LGE. Puntmann et al. demonstrated that native T1 predicts adverse outcomes even when adjusted for LGE and LVEF, reflecting the added value of detecting diffuse fibrosis [8]. Similarly, Vita et al. and Cadour et al. reported that incorporating ECV significantly improves risk prediction beyond LGE alone, particularly for heart-failure events and arrhythmic outcomes [9,13]. These findings highlight the complementary nature of mapping techniques and LGE in assessing myocardial damage.

The integration of myocardial strain further enhances risk stratification. Feature-tracking CMR-derived global longitudinal strain (GLS) has shown independent prognostic value across multiple DCM cohorts, often outperforming LVEF [16–18]. Because strain reflects the functional consequences of fibrosis and cytoskeletal disruption, it provides insight into mechanical impairment that is not fully captured by structural imaging. Xiang et al. demonstrated that combining LV, RV, and LA strain identifies high-risk patients more accurately than any single metric [21]. Raafs et al. extended these findings by showing that LA strain may even surpass LGE in prognostic strength, particularly in early disease [22].

Emerging multiparametric models combining T1, ECV, LGE, and strain have shown promising results, providing superior predictive accuracy for heart-failure progression, arrhythmic risk, and mortality in DCM [9,13,16–22,25]. By characterizing both the structural and functional components of the disease, multiparametric imaging allows clinicians to more precisely identify reversible phenotypes, stratify arrhythmic risk, and tailor follow-up strategies.

Overall, integrating mapping, strain, and LGE into a unified CMR assessment reflects the biological complexity of DCM and supports a more personalized approach to risk assessment and therapeutic planning. As evidence continues to accumulate, multiparametric CMR is poised to become a central component of contemporary DCM evaluation.

### *Treatment Response and Clinical Application*

Predicting treatment response remains an essential yet challenging component of managing dilated cardiomyopathy (DCM). Advanced cardiac magnetic resonance (CMR) biomarkers—particularly native T1, extracellular volume (ECV), and myocardial strain—offer valuable insights into myocardial health that help identify patients most likely to benefit from therapy and those at risk for clinical deterioration.

Diffuse interstitial fibrosis plays a central role in determining reversibility. Elevated native T1 and ECV values reflect matrix expansion and increased collagen deposition, which limit contractile reserve and reduce the likelihood of meaningful reverse remodeling. Several studies have demonstrated that increased diffuse fibrosis is associated with poorer recovery of left ventricular function, a higher risk of progression, and reduced responsiveness to guideline-directed medical therapy (GDMT) [8–11]. In particular, Cadour et al. showed that patients with elevated ECV had significantly higher rates of heart-failure–related events despite optimal therapy [9], suggesting that fibrosis burden may help identify individuals who require closer monitoring or early intensified treatment.

Myocardial strain provides additional prognostic information relevant to therapeutic response. Reduced global longitudinal strain (GLS) reflects impaired subendocardial fiber mechanics and predicts limited improvement in systolic function following medical therapy. Romano et al. demonstrated that impaired GLS remains a strong predictor of mortality, even after accounting for LVEF and LGE, underscoring its prognostic sensitivity [17]. Similarly, Buss et al. showed that GLS identifies patients with a reduced probability of reverse remodeling, enabling more accurate stratification of expected treatment benefit [16]. More recent work by Tang et al. confirmed that strain abnormalities predict adverse outcomes independently of traditional CMR markers [18].

Left atrial (LA) strain has also emerged as a valuable tool for assessing chronic diastolic burden and treatment response. Xiang et al. found that impaired LA strain was strongly associated with clinical progression and provided incremental value beyond LV strain and LGE [21]. Raafs et al. further showed that LA strain may outperform conventional ventricular metrics in identifying patients who fail to respond

favorably to therapy [22]. These findings suggest that atrial mechanical impairment may reflect early hemodynamic stress and help predict which patients may require more aggressive management.

Beyond pharmacologic therapy, CMR biomarkers may also guide decisions regarding device therapy. Patients with extensive diffuse fibrosis, as reflected by elevated T1 or ECV, may experience limited benefit from cardiac resynchronization therapy (CRT), while those with preserved myocardial integrity demonstrate more robust reverse remodeling [8–11,13]. Similarly, diffuse fibrosis and severe strain impairment have been linked to increased arrhythmic risk, providing additional context when considering implantable cardioverter-defibrillator (ICD) implantation beyond LVEF thresholds [17,21,25].

Overall, multiparametric CMR—combining native T1, ECV, LGE, and strain—enhances the ability to predict therapeutic response and guide individualized treatment strategies in DCM. By integrating both structural and functional biomarkers, clinicians can identify reversible disease phenotypes, anticipate clinical outcomes, and optimize decision-making across pharmacologic and device-based therapies [8–11,13,16–23,25].

### *Limitation of Evidence and Methodological Considerations*

Although advanced CMR techniques have significantly improved the assessment of dilated cardiomyopathy (DCM), several methodological and evidence-related limitations should be considered when interpreting T1, ECV, and strain metrics.

A major challenge is the heterogeneity of T1 mapping across scanners, vendors, and pulse sequences. Native T1 values differ substantially between MOLLI, ShMOLLI, and SASHA protocols and vary by field strength, contrast timing, and post-processing methods. As a result, absolute cut-offs cannot be universally applied, and most studies rely on site-specific reference ranges rather than standardized thresholds [14,15]. ECV is more reproducible but remains affected by contrast type, hematocrit measurement, and imaging timing.

Feature-tracking strain analysis also suffers from inter-vendor and inter-software variability, with differences in tracking algorithms and image resolution contributing to inconsistent GLS or LA strain values [19]. This variability limits cross-study comparisons and complicates the development of standardized prognostic thresholds.

The current evidence base, while growing, is limited by small sample sizes, single-center designs, and heterogeneity in patient populations and imaging protocols. Even the best-designed prognostic studies—such as those on T1, ECV, or strain—often lack uniform outcome definitions or long-term follow-up [8–11,16–18]. The genetic and phenotypic diversity of DCM is rarely addressed, reducing the ability to generalize findings across etiologic subgroups.

Finally, practical barriers—including availability of mapping sequences, scan time, post-processing requirements, and operator expertise—affect the real-world feasibility of incorporating multiparametric CMR into routine care. These factors contribute to variation between centers and may limit clinical implementation in resource-constrained environments.

Overall, while T1, ECV, and strain provide valuable prognostic information, their integration into routine practice requires greater standardization, multicenter validation, and harmonized acquisition protocols to ensure broad applicability and reproducibility.

### *Future Directions and Research Opportunities*

Future developments in advanced cardiac magnetic resonance (CMR) have the potential to refine both prognostication and clinical management in dilated cardiomyopathy (DCM). A central priority is the standardization of T1 and ECV acquisition protocols across scanners and vendors. Significant variability in sequence design, field strength, and post-processing remains a major barrier to establishing universal thresholds for diffuse fibrosis quantification [14,15]. Multicenter harmonization efforts and standardized phantoms will be essential for improving reproducibility and expanding clinical adoption.

Another key direction is the incorporation of T1, ECV, and myocardial strain into prospective multicenter cohorts. Most existing studies—such as the prognostic investigations by Puntmann, Cadour, Li, and Buss—are single-center or modestly sized [8–11,16]. Larger datasets with standardized imaging and outcome definitions are needed to validate cut-off values, strengthen risk models, and determine optimal clinical integration.

Future research may also clarify how advanced biomarkers guide individualized management. For example, elevated ECV or severely impaired strain could identify patients less likely to experience reverse remodeling despite optimal medical therapy, potentially supporting earlier referral for intensified therapy or closer follow-up [9,16–18]. Left atrial strain, which has shown strong predictive value for clinical deterioration and arrhythmic risk, may further refine patient selection for device therapy or early rhythm management strategies [21–23].

Technological progress is also expected to improve clinical accessibility. Faster mapping sequences, better motion correction, and streamlined post-processing tools may enable more widespread use of multiparametric CMR within routine heart failure pathways. Reviews such as those by Haaf and Eichhorn highlight that as CMR becomes more efficient and standardized, its role in DCM management will continue to expand [14,25].

Overall, future work should focus on harmonization, multicenter validation, and treatment-guided imaging strategies to fully define how T1, ECV, and strain can support personalized risk stratification and management decision-making in DCM.

## Conclusion

Advanced CMR techniques provide a deeper understanding of myocardial injury in dilated cardiomyopathy. T1 mapping, ECV, and strain imaging reveal diffuse fibrosis and early mechanical dysfunction that are not captured by conventional measures. When used together, these biomarkers offer a more accurate assessment of disease severity and help identify patients at higher risk of adverse outcomes. As standardization improves and evidence grows, multiparametric CMR is likely to become a routine part of personalized evaluation and long-term management in DCM.

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