CMR in Cardiomyopathies: The Added Value of T1 and T2 Mapping

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Introduction

Cardiomyopathies are a heterogeneous group of myocardial diseases. They are defined as conditions in which the myocardium is structurally and functionally abnormal in the absence of coronary artery disease, hypertension, valvular disease, or congenital heart disease sufficient on their own to explain the observed abnormality [1]. In 2023, the European Society of Cardiology published its first set of guidelines dedicated entirely to cardiomyopathies (with the exception of the hypertrophic cardiomyopathy section, already updated in 2014). The document proposes a phenotype-based approach: Classification starts from the morphological and functional pattern observed on imaging — hypertrophic (HCM), dilated (DCM), non-dilated left ventricular (NDLVC, a new nomenclature), arrhythmogenic (ARVC), and restrictive (RCM) cardiomyopathy — and then works back, through a multiparametric pathway (clinical assessment, genetics, multimodality imaging), to the specific etiology [1]. On this pathway, cardiac magnetic resonance (CMR) occupies a central position starting from the earliest stages of evaluation. Compared with echocardiography, which remains the first-line test, CMR offers myocardial tissue characterization that no other imaging modality can replicate with the same accuracy. CMR can distinguish, within the same morphological pattern (e.g., left ventricular hypertrophy [2]), genuinely genetic forms from phenocopies such as cardiac amyloidosis or Anderson-Fabry disease. It can recognize the non-ischemic fibrosis pattern characteristic of NDLVC, identify the fibrofatty infiltration typical of ARVC, and guide the etiologic diagnosis in DCM through the pattern and distribution of fibrosis [1]. The guidelines recommend that the initial CMR evaluation in a patient with suspected cardiomyopathy should systematically include, in addition to cine sequences and late gadolinium enhancement (LGE), T2-weighted sequences and pre- and post-contrast T1 mapping, reserving T2* for suspected iron overload/hemochromatosis [1]. This is because LGE, while often likened to a "non-invasive biopsy" for focal fibrosis, cannot detect diffuse myocardial disease. This is where parametric mapping adds a quantitative layer of information, capable of revealing abnormalities in the myocardial interstitium even when LGE is negative [3]. However, it should be highlighted that linking mapping parameters to a single pathologic abnormality of a tissue may be misleading. For example, changes such as myocardial edema can influence not only T2 mapping but also native T1 and extracellular volume (ECV) [4], making a comprehensive and complementary assessment of these parameters necessary. The following paragraphs briefly examine how T1 and T2 mapping contribute to differential diagnosis, prognostic stratification, and treatment monitoring in cardiomyopathies. Three practical examples are portrayed in the figures.

T1 and T2 mapping for early diagnosis

The clinical value of mapping lies, above all, in detecting myocardial disease before it becomes morphologically overt — a window in which LGE is frequently still negative. In sarcomeric HCM, native T1 and T2 are elevated even in non-hypertrophic, LGE-negative segments, suggesting that tissue remodeling precedes both wall thickening and contractile impairment [5]. Native T1 is also higher in genotype-positive sarcomeric mutation carriers without overt hypertrophy, supporting its use as an early, pre-phenotypic biomarker, and it discriminates HCM from hypertensive heart disease [6]. In cardiac amyloidosis, native T1 elevation can precede the development of left ventricular hypertrophy, LGE, or biomarker rise, and a clinical algorithm based on native T1 alone allows diagnosis without gadolinium in a substantial proportion of patients [7]. T2 mapping adds a further diagnostic dimension, specifically in amyloid light chain (AL) amyloidosis: Myocardial edema from direct light chain toxicity raises native T2 more markedly than in transthyretin amyloidosis (ATTR, from amyloid transthyretin). Therefore, T2 is both consistently elevated and disproportionately high in untreated AL disease, a pattern that can help point to the amyloid subtype alongside the more established free light chain and scintigraphy workup [8]. Anderson-Fabry disease shows the opposite pattern: Intracellular sphingolipid storage shortens native T1, so a low — rather than an elevated — T1 value in a hy-

pertrophied or even a pre-hypertrophied heart should raise suspicion for Fabry disease. Low T1 can be present in up to 90% of Fabry patients with left ventricular hypertrophy and may be detectable years before hypertrophy develops, reflecting a distinct "accumulation phase" of disease that precedes inflammation, hypertrophy, and eventual fibrosis, at which point T1 pseudonormalizes [9]. DCM offers a further example of diagnostic gain in the absence of LGE: In patients with DCM and no LGE, who account for approximately half of the DCM population, native T1 and ECV are still significantly elevated compared with controls, indicating diffuse interstitial fibrosis that focal LGE imaging misses entirely [10]. Together, T1 and T2 mapping also help separate early, non-ischemic DCM from physiological ventricular remodeling in trained athletes: Native T1, ECV, and T2 are all higher in DCM than in exercisers with comparable ejection fraction [11]. In routine practice, adding T1 and T2 mapping to a core CMR protocol for suspected cardiomyopathy measurably improves diagnostic sensitivity for myocarditis, Fabry disease, and amyloidosis [12].

T1 and T2 mapping for prognosis

Beyond diagnosis, mapping parameters carry independent prognostic weight, often outperforming or complementing LGE. In non-ischemic dilated cardiomyopathy, Puntmann et al. [13] showed that abnormal native T1 predicts all-cause mortality and heart failure events at least as well as LGE presence or severely reduced ejection fraction, with a clear dose-response relationship between the degree of T1 elevation and survival. Subsequent meta-analyses have confirmed this signal: Both native T1 and ECV are independently associated with major adverse cardiac events in DCM [14]. In HCM, ECV has recently emerged as an incremental prognostic marker beyond LGE. In a cohort of 990 HCM patients, ECV ≥ 35% remained an independent predictor of sudden cardiac death, heart failure events, and HCM-related death after adjustment for ejection fraction and LGE burden, and was able to identify high-risk patients even among those with low LGE burden, who would otherwise be classified as lower risk [15]. T2 mapping adds a complementary signal in HCM, where elevated T2 reflects active myocardial injury, ischemia, and microvascular dysfunction, and has been linked to worse prognosis [16]. In cardiac amyloidosis, ECV stands out as the single best CMR marker of amyloid infiltration. Because amyloidosis is the archetypal interstitial disease, ECV acts as a direct surrogate of amyloid burden. It rises earlier than LGE or overt structural and functional changes, and it correlates with sequential functional thresholds: Longitudinal strain becomes abnormal at relatively low ECV, while biventricular ejection fraction declines only once ECV is markedly elevated. Unlike native T1, which reflects a composite intracellular and extracellular signal and does not by itself distinguish the pathological process, ECV isolates the extracellular compartment and has been shown to be a more robust prognostic marker than either native T1 or LGE in cardiac amyloidosis. This has recently been formalized in ATTR amyloidosis: A large single-center cohort study defined calibrated ECV categories spanning the full disease spectrum (from genotype-positive carriers to overt cardiomyopathy), each carrying stepwise, independent prognostic significance, with patients below 30% having survival comparable to those without cardiac involvement, and those above 40% facing substantially worse outcomes regardless of conventional biomarkers or echocardiographic staging [17].

T1 and T2 mapping for treatment monitoring

Because mapping quantifies myocardial tissue composition rather than providing a single anatomic snapshot, it is naturally suited to tracking therapy response — a role that is also increasingly recognized outside the cardiomyopathies proper. In acute takotsubo syndrome, a propensity-matched study of SGLT2 inhibitors initiated within six hours of admission showed significantly lower septal and global T2 mapping values and higher left ventricular ejection fraction at CMR performed within a week of presentation compared with untreated controls, suggesting that T2 mapping can capture a faster resolution of myocardial edema with treatment [18]. In aortic stenosis — again not a cardiomyopathy, but a useful illustration of the same principle — T1 mapping and ECV track the cellular and extracellular components of hypertrophy regression after aortic valve replacement (AVR): Cellular hypertrophy regresses substantially within weeks to months of AVR, while the extracellular (fibrotic) compartment regresses much less, so ECV can paradoxically rise even as left ventricular mass falls, a dissociation that would be entirely invisible on LGE or echocardiography alone [19]. In cardiac amyloidosis, ECV has become the reference biomarker for monitoring disease-modifying therapy [20]. With patisiran, ECV fell significantly relative to untreated controls over 12 months, accompanied by falling NT-proBNP and improved 6-minute walk distance, indicating amyloid regression rather than mere stabilization [21]. Longer-term natural history data confirm that untreated ATTR cardiomyopathy shows progressive ECV rise (36% of patients progressing by 1 year, 62% by 2 years), whereas ECV remains stable in the great majority of patisiran-treated patients, and a rise in ECV at 1 year is independently associated with mortality — establishing ECV change as both a treatment-response and a prognostic marker [22]. In sarcomeric HCM, cardiac myosin inhibitors provide an emerging example of mapping-based monitoring. In the SEQUOIA-HCM CMR substudy, aficamten reduced left ventricular mass index, maximal wall thickness, left atrial volume index, and native

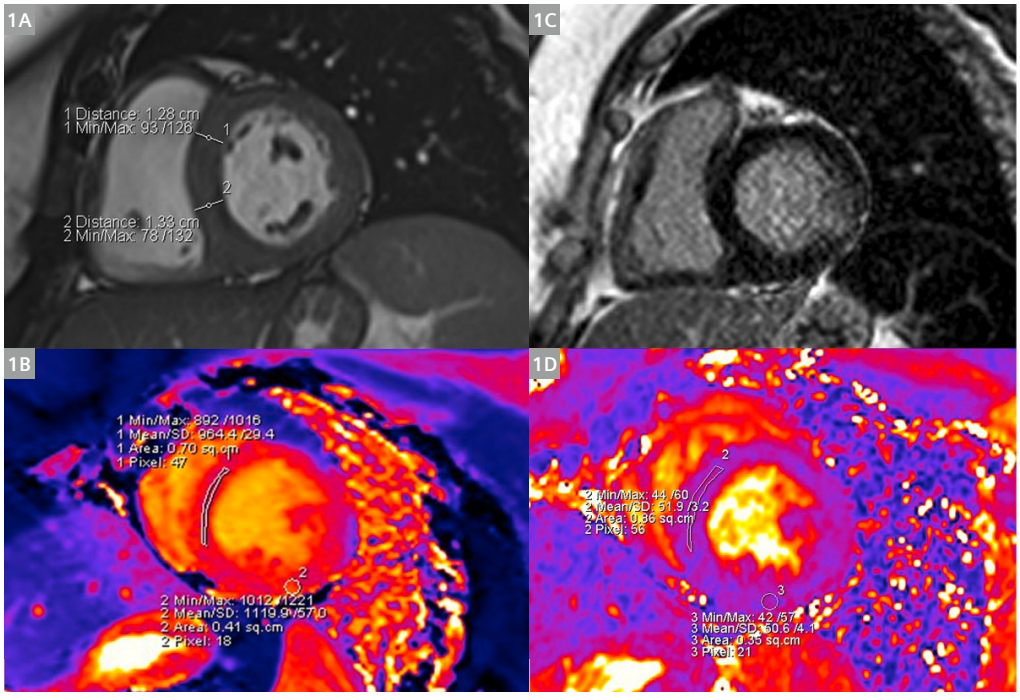
T1 relaxation time relative to placebo at 24 weeks, while LGE remained stable, suggesting favorable remodeling without progression of fibrosis [23]. Finally, T2* mapping remains the paradigm example of mapping-guided treatment titration, long predating its cardiomyopathy applications: In transfusion-dependent thalassemia and other iron-overload states, serial cardiac T2* is used to grade myocardial iron burden and to guide the intensity of iron chelation (single-agent versus combination deferoxamine/deferiprone/deferasirox regimens), with regular T2*-guided adjustment shown to reduce the risk of iron-overload heart failure, historically the leading cause of death in this population [24].

Case study #1

A woman in her 40s with genetically confirmed Fabry disease, on enzyme replacement therapy, was referred for follow-up CMR. Left ventricular hypertrophy was present (Fig. 1A). Septal native T1 was 964 ms (Fig. 1B) — within the local normal range (approximately 930–1010 ms) — despite the hypertrophy. Non-ischemic-pattern LGE was seen in the infero-lateral wall (Fig. 1C), and T2 mapping showed mild elevation (52 ms; normal < 50 ms) consistent with active myocardial inflammation (Fig. 1D). Isolated low native T1 from glycosphingolipid deposition is the classic teaching point in Fabry disease, but this only holds in earlier disease stages. As the disease progresses, superimposed diffuse interstitial fibrosis raises native T1 back toward — or even above — normal. A "normal" native T1 therefore does not exclude Fabry disease and can in fact reflect an advanced, fibrotic stage rather than the absence of storage. This pseudo-normalization is well documented in the natural history CMR literature on Fabry disease [9]. The clinical pearl is contextual: In a hypertrophied ventricle, the expected pattern is elevated native T1 from diffuse fibrosis, as in hypertensive heart disease [2]. A hypertrophied heart with native T1 in the normal (or low-normal) range is therefore paradoxical and should prompt consideration of a storage disorder such as Fabry disease, particularly when accompanied by classic infero-lateral LGE. The concurrent T2 elevation in this case further supports an active inflammatory component superimposed on the storage/fibrosis process, which has been described in Fabry cardiomyopathy and may have implications for monitoring disease activity on enzyme replacement therapy. This case illustrates why isolated native T1 values should always be interpreted against ventricular phenotype (hypertrophied vs. non-hypertrophied) and alongside LGE and T2 mapping, rather than as a stand-alone rule-in/rule-out threshold for Fabry disease.

Case study #2

A patient in her 70s was referred to serial CMR scans for the assessment of known hereditary ATTR cardiac amyloidosis. Of note, the patient carried the Phe64Leu variant — one of the well-recognized ATTR mutations associated with false-negative bone scintigraphy despite unequivocal echocardiographic and CMR evidence of cardiac involvement



1 CMR images in a patient with Fabry disease. Left ventricular hypertrophy was present **(1A)**. Septal native T1 was normal with 964 ms **(1B)**, despite the hypertrophy. Nonischemic-pattern LGE was seen in the inferolateral wall **(1C)**, and T2 mapping showed mild elevation (52 ms; normal < 50 ms) consistent with active myocardial inflammation **(1D)**.

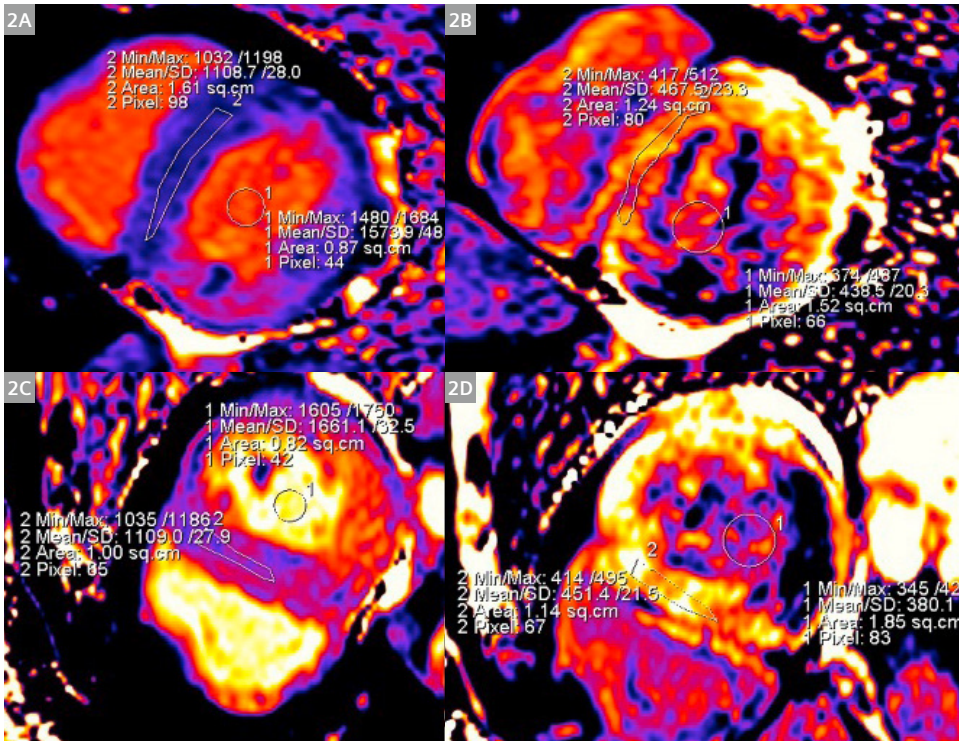
[25]. Short-axis, mid-ventricular native and post-contrast T1 maps are shown at baseline (treatment-naïve, Fig. 2 top panels) and at 18-month follow-up after initiation of vutrisiran (Fig. 2 bottom panels). At 18 months, ECV had regressed compared with baseline, while native T1 was essentially unchanged between the two timepoints. This dissociation is a useful teaching point rather than a discordant or confusing finding: At more advanced stages of cardiac amyloidosis, ECV can track disease progression better than native T1 [26].

Case study #3

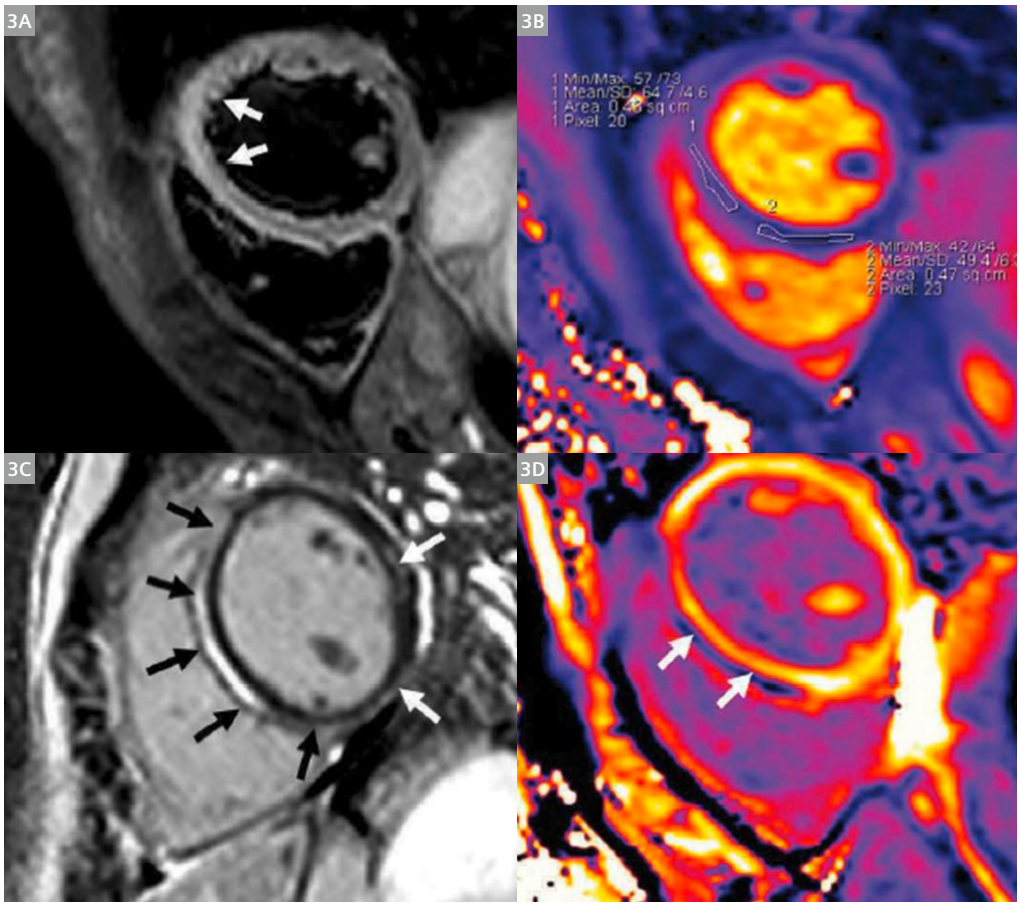
A woman in her 20s presented with chest pain, elevated troponin, and a history of two prior acute myocarditis (AM) episodes. CMR revealed myocardial edema alongside a typical ring-like pattern of LGE extending beyond the edematous area, indicating both acute and chronic myocardial injury. Specifically, CMR images of mid-ventricular short-axis are reported in Fig. 3, adapted from [27]: (Fig. 3A) STIR sequence demonstrating focal myocardial edema in the anterior septum; (Fig. 3B) T2 mapping confirming the regional edema, with normal T2 values preserved in the posterior interventricular septum; (Fig. 3C) LGE imaging revealing a ring-like enhancement pattern that extends beyond the edematous area; (Fig. 3D) post-contrast T1 mapping confirming the spatial distribution of the LGE. Prompted by this specific scar morphology and the recurrent clinical presentation, genetic testing was performed and identified a pathogenic desmoplakin mutation. The patient was diagnosed with desmoplakin cardiomyopathy presenting with recurrent inflammatory "hot phases." Because AM predominantly affects males and rarely recurs, this case demonstrates that female patients experiencing recurrent AM — particularly those exhibiting a ring-like LGE pattern on CMR — should be strongly considered for targeted genetic screening to uncover underlying inherited cardiomyopathies [28]. Due to cases like this one, the European Society of Cardiology strengthened the role of CMR in the 2025 guidelines for the management of myocarditis and pericarditis [29]. The particular strength of CMR (multiparametric examination including function, mapping, LGE, STIR) helped to stratify the patient workup toward a differential diagnosis.

Conclusion

T1 and T2 mapping have moved CMR imaging beyond morphology and LGE alone, adding tissue-level information that sharpens phenotyping, uncovers disease before structural changes appear, and refines risk stratification across the cardiomyopathies. As the three case studies illustrate, interpreting these parameters requires context: Native T1 can be misleadingly normal in Fabry disease, while ECV can outperform native T1 in tracking amyloid burden and treatment response. With the growing availability of phar-



2 CMR images in a patient with cardiac amyloidosis. Short-axis, midventricular native and post-contrast T1 maps are shown at baseline (treatment-naïve, native T1 1109 ms, ECV 42%; **2A, 2B**) and at 18-month follow-up after initiation of vutrisiran (native T1 1109 ms, ECV 36 %; **2C, 2D**). *Figure adapted with permission from [20].*



3 CMR images in a patient with recurrent acute myocarditis. **(3A)** STIR sequence demonstrating focal myocardial edema in the anterior septum. **(3B)** T2 mapping confirming the regional edema, with normal T2 values preserved in the posterior interventricular septum. **(3C)** LGE imaging revealing a ring-like enhancement pattern that extends beyond the edematous area. **(3D)** Post-contrast T1 mapping confirming the spatial distribution of the LGE. *Figure adapted with permission from [27].*

macological therapies in sarcomeric HCM, amyloidosis, and other cardiomyopathies, tissue mapping is increasingly positioned not just as a diagnostic tool but as a quantitative biomarker for monitoring treatment efficacy. Wider standardization of acquisition and normal reference ranges across centers remains essential to fully realize this potential in routine practice.

References

1 Arbelo E, Protonotarios A, Gimeno JR, Arbustini E, Barriales-Villa R, Basso C, et al. 2023 ESC Guidelines for the management of cardiomyopathies. Eur Heart J [Internet]. 2023;44(37):3503–626. Available from: http://dx.doi.org/10.1093/eurheartj/ehad194

2 Kolentinis M, Maestrini V, Vidalakis E, Cimino S, Arcari L. CMR in Hypertrophic Cardiac Conditions—an Update. Curr Cardiovasc Imaging Rep [Internet]. 2020;13:13. Available from: http://dx.doi.org/10.1007/s12410-020-9533-1

3 Puntmann VO, Peker E, Chandrashekhar Y, Nagel E. T1 mapping in characterizing myocardial disease: A comprehensive review. Circ Res [Internet]. 2016;119(2):277–99. Available from: http://dx.doi.org/10.1161/CIRCRESAHA.116.307974

4 Arcari L, Camastra G, Ciolina F, Limite LR, Danti M, Sclafani M, et al. Myocardial oedema contributes to interstitial expansion and associates with mechanical and electrocardiographic changes in takotsubo syndrome: a CMR T1 and T2 mapping study. Eur Heart J Cardiovasc Imaging [Internet]. 2023;24(8):1082–91. Available from: http://dx.doi.org/10.1093/ehjci/jead035

5 Huang L, Ran L, Zhao P, Tang D, Han R, Ai T, et al. MRI native T1 and T2 mapping of myocardial segments in hypertrophic cardiomyopathy: Tissue remodeling manifested prior to structure changes. Br J Radiol [Internet]. 2019;92(1104):20190634. Available from: http://dx.doi.org/10.1259/bjr.20190634

6 Hinojar R, Varma N, Child N, Goodman B, Jabbour A, Yu C-Y, et al. T1 mapping in discrimination of hypertrophic phenotypes: Hypertensive heart disease and hypertrophic cardiomyopathy: Findings from the international T1 multicenter cardiovascular magnetic resonance study. Circ Cardiovasc Imaging [Internet]. 2015;8(12):e003285. Available from: http://dx.doi.org/10.1161/CIRCIMAGING.115.003285

7 Baggiano A, Boldrini M, Martinez-Naharro A, Kotecha T, Petrie A, Rezk T, et al. Noncontrast magnetic resonance for the diagnosis of cardiac amyloidosis. JACC Cardiovasc Imaging [Internet]. 2020;13 (1 Pt 1):69–80. Available from: http://dx.doi.org/10.1016/j.jcmg.2019.03.026

8 Kotecha T, Martinez-Naharro A, Treibel TA, Francis R, Nordin S, Abdel-Gadir A, et al. Myocardial Edema and Prognosis in Amyloidosis. J Am Coll Cardiol [Internet]. 2018;71(25):2919–31. Available from: http://dx.doi.org/10.1016/j.jacc.2018.03.536

9 Nordin S, Kozor R, Medina-Menacho K, Abdel-Gadir A, Baig S, Sado DM, et al. Proposed Stages of Myocardial Phenotype Development in Fabry Disease. JACC Cardiovasc Imaging [Internet]. 2019;12(8 Pt 2):1673–83. Available from: http://dx.doi.org/10.1016/j.jcmg.2018.03.020

10 Puntmann VO, Voigt T, Chen Z, Mayr M, Karim R, Rhode K, et al. Native T1 mapping in differentiation of normal myocardium from diffuse disease in hypertrophic and dilated cardiomyopathy. JACC Cardiovasc Imaging [Internet]. 2013;6(4):475–84. Available from: http://dx.doi.org/10.1016/j.jcmg.2012.08.019

11 Maestrini V, Torlasco C, Hughes R, Moon JC. Cardiovascular Magnetic Resonance and Sport Cardiology: a Growing Role in Clinical Dilemmas. J Cardiovasc Transl Res [Internet]. 2020;13(3):296–305. Available from: http://dx.doi.org/10.1007/s12265-020-10022-7

12 Warnica W, Al-Arnawoot A, Stanimirovic A, Thavendiranathan P, Wald RM, Pakkal M, et al. Clinical impact of cardiac MRI T1 and T2 parametric mapping in patients with suspected cardiomyopathy. Radiology [Internet]. 2022;305(2):319–26. Available from: http://dx.doi.org/10.1148/radiol.220067

13 Puntmann VO, Carr-White G, Jabbour A, Yu C-Y, Gebker R, Kelle S, et al. T1-Mapping and Outcome in Nonischemic Cardiomyopathy: All-Cause Mortality and Heart Failure. JACC Cardiovasc Imaging [Internet]. 2016;9(1):40–50. Available from: http://dx.doi.org/10.1016/j.jcmg.2015.12.001

14 Kiaos A, Antonakaki D, Bazmpani MA, Karvounis C, Rimoldi O, Karamitsos TD. Prognostic value of cardiovascular magnetic resonance T1 mapping techniques in non-ischemic dilated cardiomyopathy: A systematic review and meta-analysis. Int J Cardiol [Internet]. 2020;312:110–6. Available from: http://dx.doi.org/10.1016/j.ijcard.2020.04.052

15 Lee H-J, Gwak S-Y, Lee S, Kim K, Cho I, Shim CY, et al. Enhanced risk stratification in hypertrophic cardiomyopathy through the integration of extracellular volume fraction on cardiovascular magnetic resonance. Eur Heart J Cardiovasc Imaging [Internet]. 2026;27(9):1615–27. Available from: http://dx.doi.org/10.1093/ehjci/jeag146

16 Xu Z, Wang J, Cheng W, Wan K, Li W, Pu L, et al. Incremental significance of myocardial oedema for prognosis in hypertrophic cardiomyopathy. Eur Heart J Cardiovasc Imaging [Internet]. 2023;24(7):876–84. Available from: http://dx.doi.org/10.1093/ehjci/jead065

17 Sheikh A, Achten A, Aimo A, Razvi Y, Mansell J, Rauf MU, et al. Myocardial Amyloid Burden in Transthyretin Amyloidosis. J Am Coll Cardiol [Internet]. 2026;87(5):505–18. Available from: http://dx.doi.org/10.1016/j.jacc.2025.10.054

18 Arcari L, Camastra G, Sclafani M, Ciolina F, Belmonte E, Castiello T, et al. SGLT2 inhibitors therapy in patients with acute Takotsubo syndrome. Int J Cardiol [Internet]. 2026;452:134262. Available from: http://dx.doi.org/10.1016/j.ijcard.2026.134262

19 Treibel TA, Kozor R, Schofield R, Benedetti G, Fontana M, Bhuva AN, et al. Reverse Myocardial Remodeling Following Valve Replacement in Patients With Aortic Stenosis. J Am Coll Cardiol [Internet]. 2018;71(8):860–71. Available from: http://dx.doi.org/10.1016/j.jacc.2017.12.035

20 Sclafani M, Russo D, Oikonomou G, Camastra G, Belmonte E, Tini G, et al. Imaging Cardiac Amyloidosis: From Early Diagnosis to Risk Stratification and Evaluation of Treatment Efficacy. J Cardiovasc Dev Dis [Internet]. 2026;13(8):401. Available from: http://dx.doi.org/10.3390/jcdd13080401

21 Fontana M, Martinez-Naharro A, Chacko L, Rowczenio D, Gilbertson JA, Whelan CJ, et al. Reduction in CMR Derived Extracellular Volume With Patisiran Indicates Cardiac Amyloid Regression. JACC Cardiovasc Imaging [Internet]. 2021;14(1):189–99. Available from: http://dx.doi.org/10.1016/j.jcmg.2020.07.043

22 Patel RK, Ioannou A, Sheikh A, Razvi Y, Mansell J, Martinez-Naharro A, et al. Transthyretin amyloid cardiomyopathy: natural history and treatment response assessed by cardiovascular magnetic resonance. Eur Heart J [Internet]. 2025;46(46):5049–58. Available from: http://dx.doi.org/10.1093/eurheartj/ehaf412

23 Masri A, Cardoso RN, Abraham TP, Claggett BL, Coats CJ, Hegde SM, et al. Effect of Aficamten on Cardiac Structure and Function in Obstructive Hypertrophic Cardiomyopathy: SEQUOIA-HCM CMR Substudy. J Am Coll Cardiol [Internet]. 2024;84(19):1806–17. Available from: http://dx.doi.org/10.1016/j.jacc.2024.08.015

24 Pereira NL, Grogan M, Dec GW. Spectrum of Restrictive and Infiltrative Cardiomyopathies: Part 2 of a 2-Part Series. J Am Coll Cardiol [Internet]. 2018;71(10):1149–66. Available from: http://dx.doi.org/10.1016/j.jacc.2018.01.017

25 Musumeci MB, Cappelli F, Russo D, Tini G, Canepa M, Milandri A, et al. Low sensitivity of bone scintigraphy in detecting Phe64Leu mutation-related transthyretin cardiac amyloidosis. JACC Cardiovasc Imaging [Internet]. 2020;13(6):1314–21. Available from: http://dx.doi.org/10.1016/j.jcmg.2019.10.015

26 Martinez-Naharro A, Kotecha T, Norrington K, Boldrini M, Rezk T, Quarta C, et al. Native T1 and Extracellular Volume in Transthyretin Amyloidosis. JACC Cardiovasc Imaging [Internet]. 2019;12(5):810–9. Available from: http://dx.doi.org/10.1016/j.jcmg.2018.02.006.

27 Camastra G, Arcari L, Ciolina F, Danti M, Sbarbati S, Cacciotti L. Desmoplakin cardiomyopathy in a female patient with recurrent acute myocarditis. J Cardiovasc Med (Hagerstown) [Internet]. 2025;26(10):604–5. Available from: http://dx.doi.org/10.2459/JCM.0000000000001783

28 Lota AS, Hazebroek MR, Theotokis P, Wassall R, Salmi S, Halliday BP, et al. Genetic Architecture of Acute Myocarditis and the Overlap With Inherited Cardiomyopathy. Circulation [Internet]. 2022;146(15):1123–34. Available from: http://dx.doi.org/10.1161/CIRCULATIONAHA.121.058457

29 Schulz-Menger J, Collini V, Gröschel JW, Adler Y, Brucato A, Christian V, et al. 2025 ESC Guidelines for the management of myocarditis and pericarditis. Eur Heart J [Internet]. 2025;46(40):3952–4041. Available from: http://dx.doi.org/10.1093/eurheartj/ehaf192



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