Cardiovascular Magnetic Resonance Parametric Mapping Techniques: Clinical Applications and Limitations

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Abstract

Purpose of the review: Parametric mapping represents a significant innovation in cardiovascular magnetic resonance (CMR) tissue characterisation, allowing the quantification of myocardial changes based on changes on T1, T2 and T2* relaxation times and extracellular volume (ECV). Its clinical use is rapidly expanding but it requires availability of dedicated equipment as well as expertise in image acquisition and analysis. This review focuses on the principles of CMR parametric mapping, its current clinical applications, important limitations, as well as future directions of this technique in cardiovascular medicine.

Recent Findings: There is increasing evidence that CMR parametric mapping techniques provide accurate diagnostic and prognostic tools that can be applied to and support the clinical management of patients with a range of cardiovascular disease.

Summary: The unique capability of CMR myocardial tissue characterisation in cardiovascular diseases has further expanded by the introduction of parametric mapping. Its use in clinical practice presents opportunities but has also limitations.

Keywords: Cardiac Magnetic Resonance imaging, T1 mapping, T2 mapping, T2* mapping, ECV, cardiomyopathies, myocardial tissue characterization

Introduction

Cardiac Magnetic Resonance (CMR) imaging holds a unique position in providing non-invasive myocardial tissue characterization both with and without contrast. Changes in myocardial tissue properties utilising non-contrast T1 and T2-weighted imaging techniques are widely used for the assessment of myocardial inflammation and oedema. Coupled with the late gadolinium enhancement (LGE) technique, myocardial scar or fibrosis can be subsequently visualised following the administration of gadolinium-based contrast agents (GBCAs), which accumulate in the extra-cellular compartment of diseased tissue. However, these techniques can only identify florid focal myocardial abnormalities.

CMR parametric mapping offers a more contemporary technique which utilises relaxometry to permit both visualization and quantification of the disease process, independent of whether myocardial disease is focal or diffuse. The Heart Failure Association (HFA) of the European Society of Cardiology has recently recognized CMR parametric mapping as an important diagnostic tool in the evaluation of heart failure patients[1]. T1 and T2 mapping quantification has also been included in the updated guidelines for the diagnosis of myocarditis using CMR[2]. This review focused on the clinical indications and applications of CMR parametric mapping, outlining the basic principles and potential pitfalls of such techniques.

A. T1 Mapping

A1. General principles and technique

T1 is a parameter describing the longitudinal" or "spin-lattice" recovery of magnetization after the excitation of protons by a radiofrequency pulse. In this technique, multiple T1-weighted images are acquired during recovery time and T1 values are derived by fitting the acquired signals to an exponential recovery curve. Finally, T1 pixel maps are generated, and every pixel corresponds to a specific T1 value, thus quantifying the myocardial tissue T1 property[3].

The CMR sequences used for the acquisition of T1 mapping are based on inversion-recovery, saturation-recovery or hybrid approaches[3,4]. The most commonly used techniques are the Modified Look-Locker Inversion Recovery (MOLLI)[5], the shortened Modified Look-Locker Inversion recovery (ShMOLLI)[6], and a saturation recovery single-shot acquisition (SASHA)[7]. Combined saturation and inversion recovery sequences for improved visualization of myocardial characteristics in patients with arrhythmia showed promising results, but further investigations are required for their validation[8].

A2. Native T1 mapping

The native T1, or non-contrast myocardial T1, refers to T1 relaxation time of myocardium without the administration of GBCAs. Native T1 values are sensitive to the magnetic resonance field strength with longer T1 measurements being

measured using 3T field strength compared to 1.5T. T1 relaxation time is also influenced by the sequence design parameters such as flip angle, matrix size and slice profile[3,9,10]. Additionally, physiologic factors such as heart rate, sex and body temperature impact on the normal T1 values[3,11,12]. Female sex is associated with higher native T1 measurements compared to males[13,14], but the exact effect of age on T1 parameters remains inconclusive with several studies showing conflicting results[13,15-17].

A2.1 Clinical applications

The main advantage of native T1 mapping is that it reflects focal and diffuse myocardial disease involving both the intracellular and interstitial compartments without the need for administration of GBCAs. Native T1 values are increased with myocardial water content, enabling the detection of both acute pathologies, including myocardial oedema or inflammation, but also other processes such as fibrosis or myocardial infiltration by fat, amyloid, or iron.

Myocardial T1 values are increased in acute myocardial infarction (MI)[18,19], acute myocarditis[20,21] or stress (Takotsubo) cardiomyopathy and provides an essential tool in the evaluation of chest pain syndromes in clinical practice[22]. Native T1 mapping can determine the myocardial areas most at risk following an acute MI[19], with one study demonstrating good correlation of T1 mapping with Tc99m-

sestamibi single photon emission tomography (SPECT) in the assessment of at-risk myocardial territories[23].

Although endomyocardial biopsy (EMB) remains the gold standard for the diagnosis of acute myocarditis, its limited use in routine clinical practice arises from its low diagnostic accuracy and the periprocedural risks associated with this invasive approach[24]. CMR is a validated, non-invasive diagnostic technique which has decreased the use of EMB in a proportion of patients. The Lake Louise Criteria (LCC) were initially established in 2009 utilising specific CMR techniques including T2-weighted sequences, early and late gadolinium enhancement sequences (EGE and LGE respectively) and thresholds aiming at standardising image analysis and interpretation[25]. Based on recent advances of T1 and T2 mapping [26] a recent iteration of the LCC support the use of CMR relaxometry among the diagnostic criteria[2]. Considering that persistent myocardial oedema following the acute phase of myocarditis is associated with progression to dilated cardiomyopathy and subsequent poorer prognosis, the improved diagnostic accuracy offered by T1 mapping may also be of prognostic value in this context[27].

T1 mapping also plays an important role in detecting chronic ischemic processes. T1 values are higher in chronic ischemic scar compared to remote myocardium and higher in acute vs chronic infarction due to expansion of the interstitial

space due to myocardial oedema[28,29]. However, in cases of lipomatous metaplasia of myocardial infarction, native T1 values might be low depending on the percentage of fat disposition within the scarred/infarcted area[30]. Native T1 mapping could help differentiate between viable and non-viable myocardium in chronic myocardial infarction (sensitivity 88% and specificity 88% with a T1 threshold of 1085 ms) and acute myocardial infarction (79% sensitivity and 79% specificity with a T1 threshold of 1110 ms)[31]. The presence of microvascular obstruction (MVO) in the acute phase creates some challenges in the use of native T1 mapping in the discriminating viable vs non-viable myocardium, as areas of MVO display low native T1 mapping leading to a pseudonormal range of native T1 values (average of low values in MVO area and high values in infarcted area)[31].

T1 quantification can detect diffuse myocardial fibrosis, but also subtle myocardial abnormalities that can be detected in the early phase of cardiomyopathic processes such as dilated (DCM) and hypertrophic cardiomyopathies (HCM) [11,32]. Diffuse myocardial fibrosis detected by increased native T1 values in patients with aortic stenosis has been correlated with the collagen volume fraction by EMB[33]. Native T1 values can be also used for the detection of subclinical myocardial disease in patients with rheumatoid arthritis and systemic lupus erythematosus[34,35]. Native T1 imaging also provides

improved risk stratification and prognostic value, being shown to predict overall mortality in non-ischemic DCM, hospitalization due to heart failure and heart transplantation[36,37].

T1 mapping plays a critical role in the diagnosis of infiltrative diseases, where the accumulation of pathological proteins within the interstitial or intracellular compartment changes the myocardial T1 properties. Native T1 values are typically shortened by fat and iron deposition. Anderson-Fabry disease is a rare X-linked lysosomal storage disorder, where glycosphingolipids are accumulating within myocardial cells due to genetic deficiencies of the enzyme alpha-galactosidase A, resulting in left ventricular hypertrophy (LVH) and ultimately fibrosis[38]. Here, native T1 is low in response to sphingolipid accumulation. Such accumulation can be seen in 40% - 50% of patients without the presence of LVH, highlighting T1 mapping as a robust diagnostic technique at an early stage in its pathogenesis, before the development of the LVH phenotype[39]. Early diagnosis has important clinical implications as prompt initiation of enzyme replacement therapy is associated with better clinical outcomes[40]. It should be noted that T1 values may increase during the disease course, becoming pseudonormalized or elevated depending on the development of fibrosis. Sado et al demonstrated normal or elevated T1 values in the inferolateral wall, which corresponded with

the presence or absence of LGE, suggesting that native T1 quantification should be performed in remote myocardium[41].

The typical diffuse infiltration of the interstitial space by pathologic amyloid proteins in both light-chain (AL) and transthyretin (ATTR) amyloidosis results in markedly elevated native T1 measurements with AL demonstrating higher values[42,43]. Importantly, T1 mapping may have a higher sensitivity in detecting early disease compared to conventional T1-weighted sequences[42].

Furthermore, native T1 mapping can be applied in the identification of iron overload cardiomyopathy, with up to 32% of cases having a low native T1 but normal T2* values[44]. The identification of iron overload cardiomyopathy has major clinical implications as it triggers the initiation of chelation therapy, a therapy that improves survival[45]. However, the different diagnostic accuracy of T1 mapping over the conventional T2* quantifications in these patients require further evaluation.

A3. Post-contrast T1 mapping and extra-cellular volume mapping (ECV)

Post-contrast T1 mapping refers to T1 quantification after the administration of GBCAs, which generally shortens T1 relaxation time. Post-contrast T1 mapping depends on various factors such as GBCA dose, glomerular filtration rate, acquisition time post-GBCA, body composition and haematocrit[3]. Notably, post-contrast T1 values in isolation were found not to correlate with histological

collagen volume fraction and as a consequence, post-contrast T1 values are used in association with the native T1 values to derive extracellular volume (ECV)[46]. The expansion of the ECV which is comprised of interstitial and intravascular spaces is a common pathophysiological mechanism of many myocardial pathologies, which plays an important role in LV remodelling and has been a therapeutic target in many trials[47-50]. ECV can be calculated using the myocardial T1 and blood T1 values pre- and post-GBCA administration alongside the haematocrit[3,11]. It represents the contrast uptake in the extracellular space relative to the blood assuming equilibration of GBCA between extravascular interstitial fluid and intravascular plasma:

$ECV = [(1/T1_{myopostGd} - 1/T1_{myopreGd}) : (1/T1_{bloodpostGd} - 1/T1_{bloodpreGd})] * (1 - Haematocrit)$

The dynamic equilibrium of the contrast distribution between blood and tissue can be achieved by imaging 15 minutes after administration of an intravenous bolus of contrast, although for recently infarcted myocardium a more prolonged acquisition time may be required[3,46,51]. Haematocrit should be measured at the time of the CMR or within 24 hours. Alternatively, synthetic haematocrit can be derived from the longitudinal relaxation rate of blood ($R1 = 1/T_{1blood}$) without the need of blood sampling; this should be calibrated for the specific T1 mapping technique[52]. Contrary to the post-contrast T1 mapping values, ECV values are reproducible when performed using the same techniques and platforms[53,54] and

there is good correlation between ECV quantification with histological collagen volume fraction[46].

Much like native T1 mapping, studies have highlighted women having higher ECV values compared to males[13,14] and likewise, the data on the effect of age on ECV measurements remain inconclusive[13,15,16].

A3.1. Clinical applications

ECV values are increased in all cardiac conditions accompanied by expansion of extracellular space, whether this is the interstitial or intravascular space or both. As a result, elevated ECV measurements have been reported in myocardial oedema (increased interstitial water content), infiltrative diseases (amyloidosis), fibrosis (focal or diffuse) and in vasodilator stress perfusion imaging as a result of expansion of the intravascular compartment due to subsequent coronary vasodilation[55].

It is established that cardiac extracellular matrix (ECM) can lead to ventricular remodelling subsequently resulting in heart failure[56]. Indeed, ECV values have been strongly associated with diastolic dysfunction in patients with HF with preserved EF (HFpEF)[57]. Notably, ECV measurements were a stronger predictor of mortality and heart failure-related hospitalizations when compared to LGE in diabetic patients, suggesting that ECV could be a potential therapeutic target in this or similar cohorts[58].

Furthermore, ECV values are significantly higher in patients with HCM compared with patients with hypertensive heart disease; however, there is evidence that native T1 mapping is a stronger discriminator between the HCM and hypertensive heart disease than ECV, LV wall thickness and indexed LV mass[59]. Differentiating physiological LVH in athletes from pathological HCM is a frequent clinical dilemma with obvious clinical implications. ECV is a promising imaging biomarker in differentiating athletic remodelling from pathologic hypertrophy. In an athlete's heart, lower ECV values are observed secondary to relatively higher cardiomyocyte volumes. Comparably, patients with HCM exhibit increased ECV values due to myocardial disarray and extracellular matrix expansion[60].

ECV mapping also plays important role in identifying occult myocardial involvement in autoimmune diseases and can play a crucial role in disease monitoring over time[61]. There is evidence that native T1 and ECV values are elevated in ANCA-associated vasculitis independent of LGE, implying the role of T1 and ECV mapping as a diagnostic tool in the assessment of these patients[62]. In patients with systemic sclerosis, both native T1 and ECV values are significantly elevated and associated with disease activity and severity, highlighting its potential contributions for emerging therapeutics[63].

Among infiltrative disorders, cardiac amyloid is associated with significantly higher ECV than any other cardiomyopathy due to the marked extra-cellular amyloid infiltration[64]. ECV in cardiac amyloidosis can be used in diagnosis, to guide and monitor therapies and predict prognosis[65-67]. ECV values in Anderson-Fabry disease have been reported to be similar to healthy controls[68] given that accumulation of glycosphingolipids is intracellular and that the extracellular spaces are relatively spared prior to the progression to the fibrotic stage.

Figure 1 compares native T1 mapping and corresponding LGE imaging in a range of cardiomyopathic processes including infarction, inflammatory and infiltrative diseases.

A5. Challenges and Pitfalls

Although the CMR relaxometry techniques have developed into new clinical tools ready for primetime, several factors have restricted the widespread implementation of T1 quantification in clinical practice. These include standardization of acquisition protocols, needs of local normal range values, departmental-specific T1 sequences, patients' heart rate, room temperatures and contrast protocol used[3,11]. Regular testing of technique stability over time is highly recommended in order to maintain consistency in the data acquired over time. Parameters that can affect the precision of ECV quantification are faster

contrast clearance and the incomplete equilibrium of GBCA between interstitial fluid and intravascular plasma[46,53, 55].

Despite the presence of motion correction (MOCO) algorithms, parametric error maps should be recorded in all patients as a quality control measure prior to interpretating the T1 data[3]. In order to avoid misinterpretation of ECV data[3], the pre- and post-contrast T1 maps should match in slice parameters, cardiac phase and slice position. Slice position can certainly affect T1 quantification with higher values noted towards the apex compared with the basal and mid-cavity ventricle due to the pronounced partial volume effect at the apical level. Similarly, partial voluming of blood or fat can result in major imprecisions in T1 and ECV values, especially in thin-walled structures such as the right ventricular (RV) free wall or areas of thinned chronic myocardial scar. Additionally, the lateral wall can present lower ECV values reflecting off-resonance effects and reduced signal-to-noise ratio in this wall, rather than reflecting myocardial pathology[69]. The use of very small targeted ROIs (e.g. < 40 mm^2) should be avoided during the image analysis as it will affect the precision of the quantification.

B. T2 mapping

B1. General principles and technique

T2 or spin-spin relaxation time is the MR constant governing the diphase of transverse magnetization following MR excitation[55]. Many different T2 mapping

techniques have been described including single-shot balanced steady-state free precession (bSSFP) sequences with different T2 preparation times, gradient[70] and spin echo (GraSE) or fast spin echo (FSE)-based pulse sequences[71,72]. Recent consensus statements recommend the use of T2-prepared bSSFP or gradient echo pulse sequences with a minimum of three T2-weighted images[11]. Comparably to T1 relaxation times, native T2 times vary according to multiple factors, including field strength and the specific sequence used. T2 mapping techniques based on SSFP read-out, result in higher T2 values compared with fast low-angle shot (FLASH) read-out whilst offering more signal-to-noise and less image artifact[71,73,74]. T2 measurements are higher at 3T compared to 1.5T[70,75]. While there is some evidence that females have higher T2 values compared to men[76,77], there remains conflicting data on the impact of age on T2 measurements[17,76,77].

B2. Clinical Applications

Similar to T1 values, myocardial T2 values represent a composite signal of both the intracellular and extracellular spaces. T2 values are typically higher in the presence of increased water content such as myocardial oedema, inflammation and acute infarction. T2 mapping overcomes some of the known limitations of conventional T2-weighted sequences including incomplete blood suppression and variations in myocardial signal intensity caused by phased array coils. This

allows T2 mapping to accurately detect oedema in acute myocardial infarction[78], both in acutely damaged myocardial tissue and in the myocardium at risk, both of which display greater T2 values compared to remote myocardium[79]. Likewise, another study highlighted the superior diagnostic accuracy of T2 mapping when compared to standard LCC for the diagnosis of EMB-proven acute myocarditis[80]. T2 mapping holds an important role in establishing a CMR diagnosis of myocarditis[80-82] such that T2 quantification has since been included in the revised LCC for the diagnosis of myocarditis[2,26]. T2 mapping is highly sensitive and specific in identifying areas of myocarditis (94% and 97% respectively) beyond the detection of wall motion abnormalities or late gadolinium enhancement[81]. In patients with active myocarditis presenting with recent-onset heart failure and reduced LV function, T2 mapping exhibits superiority to native T1 and ECV mapping in confirming myocardial changes consistent with myocarditis[82].

T2 quantification plays an important role in the diagnosis of sarcoidosis with higher specificity compared to Fluorine-18 fluorodeoxyglucose positron emission tomography computed tomography ([18F] FDG-PET CT) (76.9% versus 38.5 %)[83,84]. In particular, a retrospective study of 50 consecutive subjects with histologically proven sarcoidosis found 41% of patients without LGE exhibited T2 abnormalities suggesting an ‘occult hot phase’ of sarcoidosis preceding

myocardial fibrosis[85]. This has important clinical implications, such that T2 mapping could guide early initialisation of steroids or immunosuppressive therapy, potentially preventing the deterioration of ventricular function and scar formation[86].

T2 mapping can facilitate the diagnosis of Takotsubo cardiomyopathy[87] in which increased T2 values are in keeping with the presence of myocardial oedema in corresponding areas of regional wall motion abnormalities.[87,88]

Another significant clinical application of T2 mapping is the detection of acute cardiac allograft rejection, which is a predictor of survival during the highest risk period of the immediate 12 months following heart transplantation[89,90]. Acute transplant rejection is characterized by complex processes precipitating an inflammatory response with concomitant myocardial oedema. Increased T2 values corelated with biopsy-determined grades of acute transplant rejection[90]. Moreover, elevated T2 values normalize after response to immunosuppression therapy highlighting the role of T2 mapping to measure treatment response following resolution of myocardial oedema[91]. However, further evidence is warranted to confirm that T2 mapping can effectively guide the selective use of endomyocardial biopsy in the setting of transplant rejection in clinical practice.

Finally, an emerging clinical application of T2 mapping is the early diagnosis of chemotherapy-induced cardiotoxicity preceding the deterioration of LV

function. Myocardial oedema detected by T2 mapping in women treated with anthracyclines ± trastuzumab for breath cancer correlated with the deterioration of LV systolic function at follow-up[92]. Experimental animal studies demonstrated that increases in T2 relaxation time were the earliest marker of cardiotoxicity before alterations of LV function were detected following anthracyclines use[93]. Crucially, this study noted that the early discontinuation of doxorubicin following the identification of prolonged T2 relaxation time, prevented the progress to myocardial dysfunction and resulted in T2 normalization[93].

Figure 2 compares T2 mapping and corresponding LGE imaging in a range of cardiomyopathic processes to normal myocardium.

B3. Challenges and pitfalls

The limitations of the T1 mapping technique are also applicable to T2 mapping. Indeed, defining normal ranges to differential health and disease is pivotal for the interpretation on T2 data[11]. Furthermore, T2 mapping techniques are highly dependent on heart rate. To avoid imprecision in T2 quantification associated with higher heart rates, an increased sampling interval is required to allow complete T1 recovery[55,94]. More clinical diagnostic and prognostic data is required to consolidate the role of T2 mapping in clinical practice.

C. T2* mapping

C1. General principles and technique

T2* relaxation time represents the inherent decay of transverse magnetisation caused by a combination of spin-spin relaxation (T2) and magnetic field inhomogeneity (T2)[55,94]. This technique uses series of T2*-weighted images with different echo times (TEs) to assess signal decay and produce a single T2* value, creating a T2* map. Gradient echo (GRE) sequences which do not include a refocusing pulse to correct dephasing due to magnetic field inhomogeneity are used in T2* mapping technique[55,94].

C2. Clinical applications

Iron and oxygen are typical molecules that induce local magnetic field inhomogeneity which shorten T2*, affording its use for the detection of iron loading. A normal mean T2* value > 40ms has been widely reported in healthy volunteers, whilst T2* values < 20ms are suggestive of iron overload cardiomyopathy[94-96]. Cardiac siderosis is the most common mechanism of death in patients with thalassaemia major but liver iron loading does not correlate with cardiac iron overload[96]. Furthermore, the onset of ventricular dysfunction is usually late in the natural history of the disease such that the early detection of the 'occult' stage of disease provided by T2* imaging is crucial to guide early initiation of chelation therapy and monitor treatment response [97,98].

T2* mapping techniques can also be used to assess the presence and extent of intramyocardial haemorrhage which can cause magnetic field inhomogeneities secondary to by-products of haemoglobin degradation. T2* imaging was found to be superior to T2 imaging in the detection of acute reperfusion myocardial hemorrhage[99]. The identification of intramyocardial haemorrhage following acutely reperfused ST-Elevation Myocardial Infarction (STEMI) has important clinical implications, as it is associated with increased major adverse cardiovascular events at 6 months after the acute event[100].

Table 1 summarizes the changes in T1, ECV, T2 and T2* relaxation times in different cardiac pathologies, emphasizing the clinical utility of parametric mapping.

C3. Challenges and pitfalls

In iron overload cardiomyopathy, T2* measurements should be performed in the intraventricular septum to avoid susceptibility artifacts in the lateral walls. Similarly, signal contamination from the epicardial vessels at the LV and RV insertion points could also lead to errors in T2* measurements. In line with the other parametric techniques, cardiac and respiratory motion artifacts during image acquisition can affect image quality and subsequent T2* quantification.

Conclusion

Parametric mapping of myocardium is an emerging technique with the unique property of quantifying myocardial abnormalities at the early stage of disease compared to conventional CMR imaging. There is increasing evidence for the clinical utility of CMR mapping in diagnosis and prognostication for a range of cardiac conditions. Although clinicians need to also be aware of their limitations and potential pitfalls, CMR relaxometry techniques are here to stay and to be increasingly used in routine clinical practice.

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Figure Legends

Figure 1. Native T1 mapping (top row) and late gadolinium enhancement (LGE) imaging (bottom row) in a range of cardiomyopathic processes. **A1-A2**: Increased native T1 values in the mid inferior (1418 msec, normal range 1000 ± 50 msec) and inferoseptum (1214 msec, normal range 1000 ± 50 msec) with analogous LGE in a patient with inferior wall myocardial infarction. **B1-B2**: Reduced native T1 measurements measured in the mid septum (812 msec, normal range 1000 ± 50 msec) in a patient with Anderson-Fabry disease, increased values (1277 msec, normal range 1000 ± 50 msec) in the mid inferolateral wall, matching the area of LGE (myocardial fibrosis). **C1-C2**: Native T1 mapping and LGE sequence in a patient with acute lymphoblastic leukaemia. Increased native T1 measurements in the inferoseptum (1256 - 1320 msec, normal range 1000 ± 50 msec) and mid anterolateral (1227 msec, normal range 1000 ± 50 msec) walls and large nodular areas with lack of contrast penetration, representing myocardial infiltration without vascularity (cancerous tissue). **D1-D2**: Diffuse increased native T1 values (1180 - 1340 msec, normal range 1000 ± 50 msec) and extensive diffuse LGE in a patient with cardiac amyloidosis.

MI: myocardial infarction

Figure 2. T2 mapping (top row) and late gadolinium enhancement (LGE) imaging (bottom row) in a range of acute cardiac pathologies. **A1-A2**: Normal T2 values (48 - 50 msec, normal range 52 ± 4 msec) and LGE imaging in a normal patient. **B1-B2**: Elevated T2 values in the mid inferoseptum (66 msec, normal range 52 ± 4 msec) and mid anterolateral (67 msec, normal range 52 ± 4 msec) segments extending to apical septum (78 msec, normal range 52 ± 4 msec) and apical lateral (62 msec, normal range 52 ± 4 msec) segments in a patient with acute myocarditis. LGE imaging showing diffuse patchy epicardial and mid-wall late myocardial enhancement of the anterolateral wall from base to apex and of the mid to apical septum. **C1-C2**: Increased T2 values in the basal inferoseptum (89 msec, normal range 52 ± 4 msec) and inferior (96 msec, normal range 52 ± 4 msec) walls with matching transmural LGE in a patient with acute inferior myocardial infarction. **D1-D2**: Extensive increased T2 values in the mid-cavity septum and anterior walls (66 -75 msec, normal range 52 ± 4 msec) with an area of low T2 values (45-48 msec, normal range 52 ± 4 msec) in these segments in a patient with large acute anterior myocardial infarction. Microvascular obstruction (MVO) can be appreciated in the area of low T2 values. LGE imaging confirmed the evidence of MVO.

MI: myocardial infarction