Increased Myocardial Native T$_1$ Relaxation Time in Patients With Nonischemic Dilated Cardiomyopathy With Complex Ventricular Arrhythmia

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Purpose: To study the relationship between diffuse myocardial fibrosis and complex ventricular arrhythmias (ComVA) in patients with nonischemic dilated cardiomyopathy (NICM). We hypothesized that NICM patients with ComVA would have a higher native myocardial T$_1$ time, suggesting more extensive myocardial diffuse fibrosis.

Materials and Methods: We prospectively enrolled NICM patients with a history of ComVA ($n = 50$) and age-matched NICM patients without ComVA ($n = 57$). Imaging was performed at 1.5T with a protocol that included cine magnetic resonance imaging (MRI) for left ventricular (LV) function, late gadolinium enhancement (LGE) for focal scar, and native T$_1$ mapping for diffuse fibrosis assessment.

Results: Global native T$_1$ time was significantly higher in patients with NICM with ComVA when compared to patients with NICM without ComVA (1131 ± 42 vs. 1107 ± 45 msec, $P = 0.006$), and this finding remained after excluding segments with scar on LGE (1124 ± 36 vs. 1102 ± 44 msec, $P = 0.006$). Native T$_1$ was similar in NICM patients with and without the presence of LGE (1121 ± 39 vs. 1117 ± 48 msec, $P = 0.68$) and mildly correlated with LV end-diastolic volume index ($r = 0.27$, $P = 0.005$), LV end-systolic volume index ($r = 0.24$, $P = 0.01$), and LV ejection fraction ($r = -0.28$, $P = 0.003$). Native T$_1$ value for each 10-msec increment was an independent predictor of ComVA (odds ratio 1.14, 95% confidence interval 1.03–1.25; $P = 0.008$) beyond LV function and LGE.

Conclusion: NICM patients with ComVA have higher native T$_1$ compared to NICM without any documented ComVA. Native myocardial T$_1$ is independently associated with ComVA, after adjusting for LV function and LGE.

Level of Evidence: 2

Technical Efficacy: Stage 2

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Nonischemic dilated cardiomyopathy (NICM) is associated with significant morbidity and mortality due to the associated risk for progressive heart failure and sudden cardiac death (SCD).[1,2] SCD due to ventricular arrhythmias accounts for ~30% of deaths in NICM.[3,4] Although focal fibrosis evident by the presence and extent of late gadolinium enhancement (LGE) is known to be associated with increased risk of ventricular arrhythmia and adverse arrhythmic outcomes,[5–7] the electrophysiologic substrate for ventricular arrhythmias in patients with NICM is not clearly defined. In contrast to ischemic heart disease, interstitial fibrosis, myocyte disarray, and membrane abnormalities likely form the substrate responsible for ventricular tachycardia (VT) in patients with NICM.[2] Prior studies have demonstrated that diffuse myocardial fibrosis

alters electrical propagation between myocytes with potential proarrhythmic consequences.[8,9]

LGE is present in 40% of patients with NICM, with a distinct mid-myocardial presence.[10,11] However, arrhythmia could occur in patients without any evidence of scar in LGE. There is little known about the substrate of arrhythmia in NICM patients with and without scar on LGE. Myocardial tissue characterization using T_1 or extracellular volume (ECV) mapping allows for assessment of diffuse myocardial fibrosis.[12–15] Recent studies have shown that native T_1 is elevated in patients with NICM compared with healthy volunteers.[12,16] Furthermore, native T_1 moderately correlated with cardiac structural makers including LV functional decline and volumetric dilatation.[16,17] In a study by Puntmann et al, increased native T_1 was shown to be predictive of heart failure events and all-cause mortality in NICM.[18] Chen et al recently demonstrated that native T_1 was useful for predicting appropriate implanted cardioverter defibrillator (ICD) therapy in a heterogeneous patient population including ischemic and nonischemic cardiomyopathy undergoing ICD implantation.[19] However, the relationship between T_1-map-derived diffuse myocardial fibrosis and ventricular arrhythmia in NICM patients remains to be clarified. We hypothesized that NICM patients with ventricular arrhythmias would have higher global native T_1 time, suggesting a more extensive myocardial diffuse fibrosis that may play an important role in the genesis of VT. Consequently, the aim of this study was to compare the degree of diffuse myocardial fibrosis using T_1 mapping in patients with NICM, with and without ventricular arrhythmias.

Materials and Methods

Study Population

We prospectively recruited NICM patients with or without documented complex ventricular arrhythmia (ComVA) and 10 age-matched control subjects with normal LV systolic function, no symptoms of heart failure, and no other cardiovascular diseases. The study protocol was approved by our Institutional Review Board, and written informed consent was obtained from all study participants.

Patients were selected from general cardiology as well as cardiac electrophysiology clinics over 3 years who had been referred for a clinical cardiac MR exam. All patients were diagnosed with NICM by their treating cardiologists based on medical history, physical examination, electrocardiography (ECG), and echocardiography.[20] Exclusion criteria were: 1) diabetes mellitus, 2) history of myocardial infarction or a subendocardial based LGE pattern, 3) hypertrophic and infiltrative cardiomyopathies, or 4) inherited syndromes associated with VT (catecholaminergic, Brugada, or idiopathic QT syndromes, diagnosed on the basis of the family history, ECG, and, where available, genetic testing for known genes). ComVA was defined as Grade IVb or higher by the Lown and Wolf classification.[21] ComVA was documented on a Holter monitor, event monitor, or ECG. The absence of ComVA was determined by a negative Holter or event monitor, or no ECG record of ventricular arrhythmias while being regularly followed by local cardiologists. Patient characteristics were reviewed from the hospital electronic medical records.

Cardiac MR Protocol

Cardiac MR images were acquired using a Philips Achieva 1.5T scanner with a 32-channel cardiac receiver coil. We acquired steady-state free-precision cine MR (repetition time / echo time [TR/TE] = 3.3/1.6 msec; flip angle = 60°; field of view [FOV] = 320 × 320 mm², acquisition matrix = 188 × 188; slice thickness = 8 mm; gap = 2 mm), and native T_1 mapping by slice-interleaved T_1 mapping (STONE) sequence with the following parameters: diastolic acquisition, 5 slices, TR/TE = 2.8/1.4 msec, flip angle = 70°, FOV = 360 × 350 mm², voxel size = 2.1 × 2.1 mm², slice thickness = 8 mm, turbo field echo (TFE) factor = 86, parallel imaging SENSE factor = 2.[22] 3D LGE images were acquired 10–20 minutes after injection of 0.1–0.2 mmol/kg of Gd-DTPA (Magnevist; Bayer Schering, Berlin, Germany) or Gd-BOPTA (MultiHance; Bracco Imaging, Milan, Italy) with the following imaging parameters: TR/TE = 5.3/2.1 msec; flip angle = 70°; FOV = 320 × 320 × 125 mm³; acquisition matrix = 224 × 224 × 23; spatial resolution = 1.4 × 1.4 × 4 mm³ to 5 mm; reconstruction resolution = 0.6 × 0.6 × 2 to 2.5 mm³.[23,24] T_1 maps were generated after motion correction using the adaptive registration of varying contrast-weighted images for improved tissue characterization.[25] A two-parameter fit model was used to estimate voxelwise T_1 values. Native T_1 time of 16 myocardial segments from three slices (basal-, mid-, and apical-slice) was measured using custom software developed in MatLab (MathWorks, Natick, MA). Since the presence of scar can result in an increase in native T_1 values in the corresponding segments, an additional analysis of T_1 values was performed after excluding the LGE-positive myocardial segments identified by visual assessment. Left ventricular volumes, ejection fraction, and mass were measured using dedicated software (Extend MR WorkSpace, v. 2.3.6.3, Philips Healthcare, Best, Netherlands). Left ventricular volumes and mass were indexed to body surface area. The absence or presence and location of LGE were determined visually and if LGE was present, LGE volume was assessed using a custom software developed in MatLab, which enables manual segmentation of diseased myocardium and normal remote region and quantifies LGE region using thresholding techniques. For each short-axis cross section, after the endocardial and epicardial borders were traced, a region of interest was defined in the normal remote myocardium without any artifact. The software calculated mean and standard deviation (SD) of remote region signal intensity (SI) and thresholds of all pixels with SI greater than mean +4 SD of remote region, and reported a total volume and percentage of total LV mass. In 74 NICM patients (32 with ComVA, 42 without ComVA) with a postcontrast T_1 map and hematocrit assessment on the day of scanning, T_1 times of LV myocardium and blood pool were similarly determined, before and after contrast injection. Then extracellular volume fraction was calculated according to the formula as:

$$\text{Extracellular volume fraction} = (1 - \text{hematocrit})$$
$$\times \{[(1/T1\text{myocardium.post}) - (1/T1\text{myocardium.pre})]$$
$$/ [(1/T1\text{blood.post}) - (1/T1\text{blood.pre})]\} \times 100 \ (\%)$$

Statistical Analysis

The characteristics of the participants are presented as mean ± SD for continuous variables, median (quartiles) if not normally distributed, and number (%) of participants for categorical variables. Continuous variables are compared using an unpaired Student's t-test, noncontinuous variables using a Mann–Whitney nonparametric test, and categorical variables using a chi-square test. Depending on data distribution, either a Pearson or Spearman correlation coefficient was calculated to investigate possible associations of continuous outcome measures. In addition, the cutoff value of native T_1 was determined by the area under the receiver operating characteristic (ROC) curve analysis in predicting ComVA. Multivariable logistic regression analyses with six potentially confounding factors (age, male gender, New York Heart Association [NYHA] functional class, LV end-diastolic volume index, LGE volume, and global native T_1) were performed with ComVA as the dependent variable, using the rule of thumb of having between 5 and 10 outcomes per predictor. We report the adjusted odds ratios (OR) and corresponding 95% confidence interval (CI). All tests were two-sided and $P < 0.05$ was considered statistically significant. Statistical analyses were performed using SPSS (IBM, Armonk, NY) and MedCalc for Windows (MedCalc Software, Ostend, Belgium).

Results

A total of 107 patients with NICM were recruited, including 50 patients with a history of ComVA (35 nonsustained VT, 15 sustained monomorphic VT) and 57 patients without a history of ComVA. The mean age of all patients was 52 years (range 19–89). There was no significant difference between the two groups in terms of body surface area, baseline blood pressure, heart rate, history of hypertension, dyslipidemia, and smoking. NICM patients with ComVA were frequently male, more likely to be on beta-blockers and angiotensin-converting enzyme inhibitors/angiotensin-receptor blockers, and have higher NYHA functional classification. Among the cohort with ComVA, 34% (17/50) of the patients underwent an electrophysiologic study (EPS). Eleven of the 17 had inducible monomorphic VT during EPS and subsequently underwent VT ablation (Table 1).

Table 2 summarizes the cardiac MR findings. There was a trend for higher LV end-diastolic, end-systolic volume and stroke volume, and lower LV ejection fraction in patients with Com VA. Nonischemic LGE was observed in 35 patients (33%); mid-wall LGE in 18 patients, focal LGE in 12 patients; and transmural LGE in five patients. Patients with ComVA had a similar prevalence of LGE (36% vs. 30%, $P = 0.50$); however, they were more likely to have higher scar volume compared with those without Com VA (6.7% vs. 3.5%, $P = 0.03$). Pertaining to T_1 map analysis, 49 segments (3%) were excluded for analysis due to motion artifacts or incomplete registration. NICM patients had significantly higher global native T_1 compared with age-matched controls (1118 ± 45 vs. 1073 ± 25 msec, $P < 0.001$). Global native T_1 time was significantly higher in patients with NICM with ComVA when compared with patients with NICM without ComVA

(1131 ± 42 vs. 1107 ± 45 msec, $P = 0.006$). In 74 of 107 NICM patients with extracellular volume fraction data, the global extracellular volume fraction appeared significantly higher in patients with ComVA compared with those without ComVA (0.31 ± 0.03 vs. 0.29 ± 0.04, $P = 0.03$). Even after excluding 131 segments with LGE (80 segments in ComVA group and 51 segments in non-ComVA group), native myocardial T_1 remained significantly higher in NICM patients with ComVA (1124 ± 36 vs. 1102 ± 44 msec, $P = 0.006$) (Figure 1). Figure 2 shows representative NICM cases with and without ComVA. Global native T_1 time over the 16 segments was increased to 1156 msec in a case with ComVA despite the absence of LGE (Figure 2A), while a case without ComVA showed mid-wall LGE in the basal septum, resulting in relatively high native T_1 of 1117 msec in the corresponding segment. However, global native T_1 of 1066 msec was in the normal range (Figure 2B). No significant difference in native T_1 was observed between patients with and without the presence of LGE (1121 ± 39 vs. 1117 ± 48 msec, $P = 0.68$). In the subgroup analysis of patients with ComVA, global native T_1 was more likely to be increased in NICM patients with sustained VT vs. nonsustained VT (1147 ± 57 vs. 1125 ± 34 msec, $P = 0.11$). Figure 3 shows the relationship between native T_1 and LV volumes and ejection fraction. There was a mild positive correlation with LV end-diastolic volume index ($r = 0.27$, $P = 0.005$), LV end-systolic volume index ($r = 0.24$, $P = 0.01$), and negative correlation with LV ejection fraction ($r = -0.28$, $P = 0.003$). The ROC curves showed native T_1 in the whole myocardium of >1110 msec to be the optimal cutoff points for ComVA, with 64% sensitivity and 56% specificity and area under the ROC curve of 0.64 (95% CI: 0.54–0.74). In multivariable analysis, the native T_1 value for each 10 msec increment was an independent predictor of ComVA (OR 1.14, CI 1.03–1.25; $P = 0.008$) beyond LV function and LGE (Table 3).

Discussion

In this study of NICM patients with and without ComVA, we found that patients with ComVA had significantly higher global native T_1 time compared to NICM patients without ComVA. Native T_1 was independently associated with ComVA beyond LV function and LGE, although there was a univariate correlation between native T_1 and LV function and a trend for higher native T_1 in patients with large extent but not the presence of LGE.

The pathophysiology of postinfarct VT has been well described, and relates to the propagation of a wave front around ventricular scar sustained by a reentrant mechanism involving a "protected isthmus."[26–29] However, the mechanism for ventricular arrhythmia in NICM patients is less well understood. Indeed, scar and fibrosis in patients with NICM differ from that in patients with ischemic cardiomyopathy and tend to be smaller, less confluent, less transmural involvement with a

TABLE 1. Baseline Patient Characteristics

Characteristics	Control (*n* = 10)	+Com VA (*n* = 50)	–Com VA (*n* = 57)	*P* value
Age (years)	54 ± 6	54 ± 16	52 ± 14	0.54
Male (%)	7 (70)	39 (78)	38 (67)	0.19
BSA (m^2)	1.71 ± 0.17	2.04 ± 0.21	1.99 ± 0.21	0.20
Hypertension (%)	1 (10)	9 (18)	14 (25)	0.41
Systolic blood pressure (mmHg)	124 ± 21	119 ± 17	116 ± 18	0.37
Diastolic blood pressure (mmHg)	76 ± 13	73 ± 11	72 ± 16	0.90
Diabetes mellitus (%)	0 (0)	0 (0)	0 (0)	—
Dyslipidemia (%)	2 (20)	9 (18)	13 (23)	0.54
Current smoking (%)	0 (0)	7 (14)	13 (23)	0.24
Medication				
Beta-blocker	—	46 (92)	39 (68)	0.003
ACEi/ARB	—	42 (84)	37 (65)	0.03
Antiarrhythmic	—	10 (20)	6 (11)	0.17
NYHA functional class				0.01
I-II	—	29 (58)	43 (75)	
III-IV	—	21 (42)	14 (25)	
Etiology				0.09
Idiopathic	—	40 (80)	53 (93)	
Arrhythmia-induced	—	4 (8)	1 (2)	
LV noncompaction	—	4 (8)	0 (0)	
Chronic myocarditis	—	2 (4)	2 (3)	
Peripartum	—	0 (0)	1 (2)	
Electrophysiologic study	—	17 (34)	—	—
Ablation for inducible VA	—	11 (22)	—	—

Variables given are mean ± SD or *N* (%) or median (interquartile range). *P*-value is for +ComVA versus –ComVA.
ACEi = angiotensin-converting enzyme inhibitors; ARB = angiotensin-receptor blockers; BSA = body surface area, ComVA = complex ventricular arrhythmia NYHA = New York Heart Association; PCI = percutaneous coronary intervention.
ComVA (complex ventricular arrhythmia); BSA (body surface area); SBP (systolic blood pressure); DBP (diastolic blood pressure); ACEI (angiotensin-converting enzyme inhibitor)/ARB (angiotensin-receptor blocker); NYHA (New York Heart Association)

predilection for the mid-myocardium and epicardium, and near the base and perivalvular regions.[30,31] In addition, nonischemic LGE are patchy, with fewer fixed boundaries and protected isthmuses, which can change the extent of local conduction slowing.[32] Although the presence and extent of LGE are associated with an increased risk of future cardiac events in NICM patients,[5–7] LGE cannot provide absolute quantification of diffuse myocardial fibrosis and many patients with NICM have no focal scar on LGE. The current result is consistent with the recent study by Chen et al[19] that T_1 mapping is useful for the prediction of ventricular arrhythmia in ischemic and nonischemic patients with implantable cardioverter-defibrillators. However, in their study, native T_1 was measured on a single mid-septal myocardium and a heterogeneous patient population was included. Diffuse myocardial fibrosis rather than focal fibrosis/scar is an essential process in pathologic remodeling in NICM, and the role of diffuse myocardial fibrosis in the pathophysiology of VT in NICM might be more important. The current study demonstrated that global native T_1 values are significantly higher in patients with NICM with ComVA when compared to those without ComVA. Importantly, this highlights that native T_1 mapping can be applicable for assessing arrhythmic risk in patients with NICM.

While the mechanism of VT in NICM is still unclear, the combination of focal myocardial fibrosis creating regions of conduction block, with nonuniform anisotropy and slow

TABLE 2. Cardiac MR Measurements

Characteristics	Control (n = 10)	+Com VA (n = 50)	–Com VA (n = 57)	P value
LV EDV (ml)	112.3 ± 21.3	282.6 ± 102.2	254.1 ± 94.1	0.14
LV EDV index (ml/m^2)	65.5 ± 8.3	138.0 ± 44.0	127.2 ± 41.4	0.19
LV ESV (ml)	44.6 ± 9.0	212.5 ± 107.0	179.1 ± 92.2	0.09
LV ESV index (ml/m^2)	26.1 ± 4.1	104.1 ± 48.7	89.3 ± 42.2	0.10
LV stroke volume (ml)	67.7 ± 13.3	84.9 ± 22.5	75.0 ± 20.8	0.02
LV stroke volume index (ml/m^2)	39.4 ± 4.9	41.6 ± 10.0	37.8 ± 9.9	0.05
LV ejection fraction (%)	60.3 ± 2.7	33.1 ± 12.3	32.6 ± 12.6	0.85
LV mass (g)	66.5 ± 16.9	142.4 ± 60.1	138.0 ± 53.8	0.69
LV mass index (g/m^2)	38.5 ± 6.7	69.5 ± 26.4	68.8 ± 23.9	0.90
LGE, n (%)	0 (0)	18 (36%)	17 (30%)	0.50
LGE volume (g)	—	8.1 (2.8-13.3)	4.4 (2.8-7.4)	0.09
LGE volume / LV mass (%)	—	6.7 (2.8-7.9)	3.5 (1.8-5.4)	0.03
Heart rate (beats/min)	68 ± 10	74 ± 18	75 ± 14	0.71
Native T1 (msec)	1073 ± 25	1131 ± 42	1107 ± 45	0.006
T1 in basal slice (msec)	1088 ± 24	1124 ± 42	1104 ± 50	0.03
T1 in mid slice (msec)	1061 ± 35	1130 ± 58	1104 ± 50	0.02
T1 in apical slice (msec)	1078 ± 35	1144 ± 61	1115 ± 49	0.009
Native T1 without LGE segments (msec)	—	1124 ± 36	1102 ± 44	0.006

Variables given are mean ± SD, N (%), or median (interquartile range) P-value is for +ComVA versus –ComVA.
LV = left ventricular; EDV = end-diastolic volume; ESV = end-systolic volume; LGE = late gadolinium enhancement.
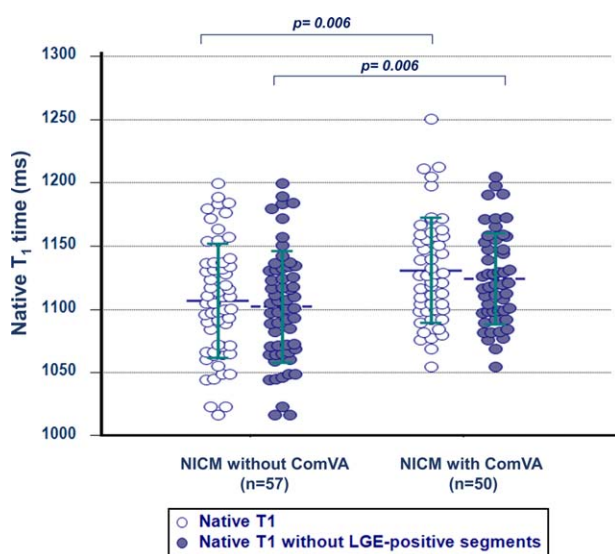

FIGURE 1: Comparison of global native T$_1$ time. Global native T$_1$ time between patients with NICM with and without ComVA (white). Native myocardial T$_1$ time after excluding segments with late gadolinium enhancement between patients with NICM with and without ComVA (blue). Global native T$_1$ is significantly higher in NICM patients with ComVA (P = 0.006 and 0.006, respectively). ComVA: complex ventricular arrhythmias; NICM: nonischemic cardiomyopathy.

conduction through interstitial fibrosis, may promote reentry and result in sustained ventricular arrhythmias. Furthermore, abnormal myocytes within areas of interstitial fibrosis might develop spontaneous diastolic depolarization, resulting in abnormal automaticity and arrhythmia. In addition, chronic volume overloading and LV dilatation associated with NICM could promote enhanced sympathetic tone and contribute to cyclic adenosine monophosphate (AMP)-mediated triggered sustained ventricular arrhythmias.[33] Diffuse myocardial fibrosis assessed by native T$_1$ mapping can provide incremental value in addition to LGE and LV functional assessments for identifying NICM patients with an underlying substrate for ventricular arrhythmia. LGE enables identification of myocardial fibrosis including myocardial infarction and focal myocardial fibrosis and its patterns can help differentiate between ischemic and nonischemic cardiomyopathy. The presence of LGE has been shown to have important prognostic information[5–7] and cannot be replaced with native T$_1$ mapping at this point. Native T$_1$ mapping may provide additional information and may be used in NICM patients in whom administration of contrast agent is not desired. Further studies are needed to elucidate the
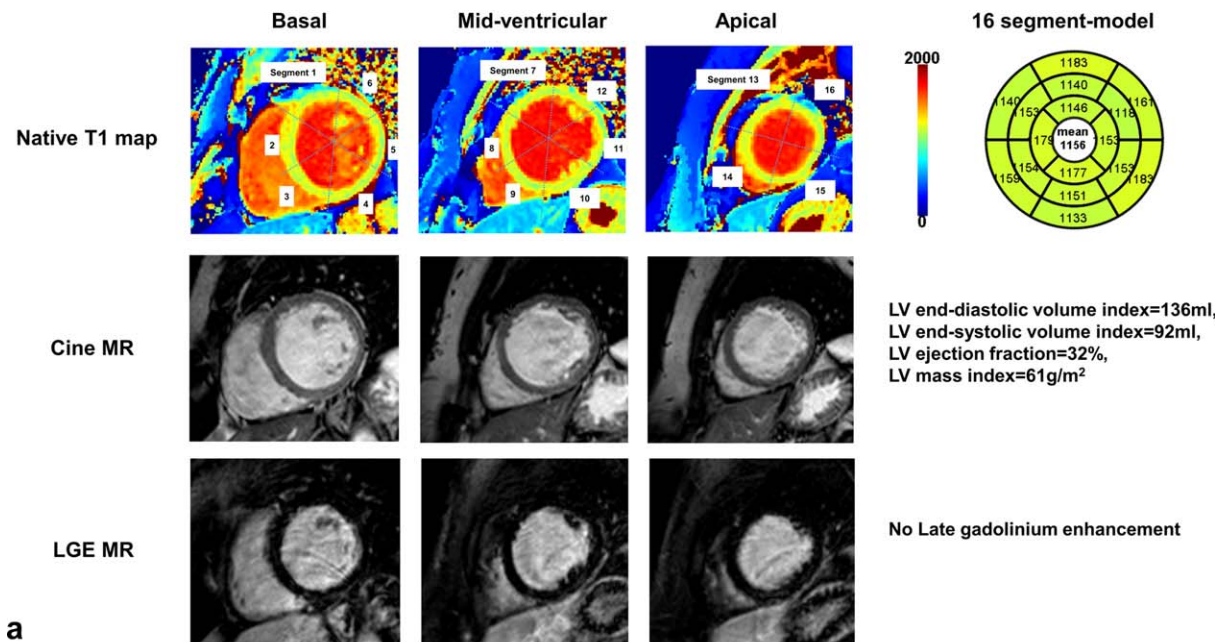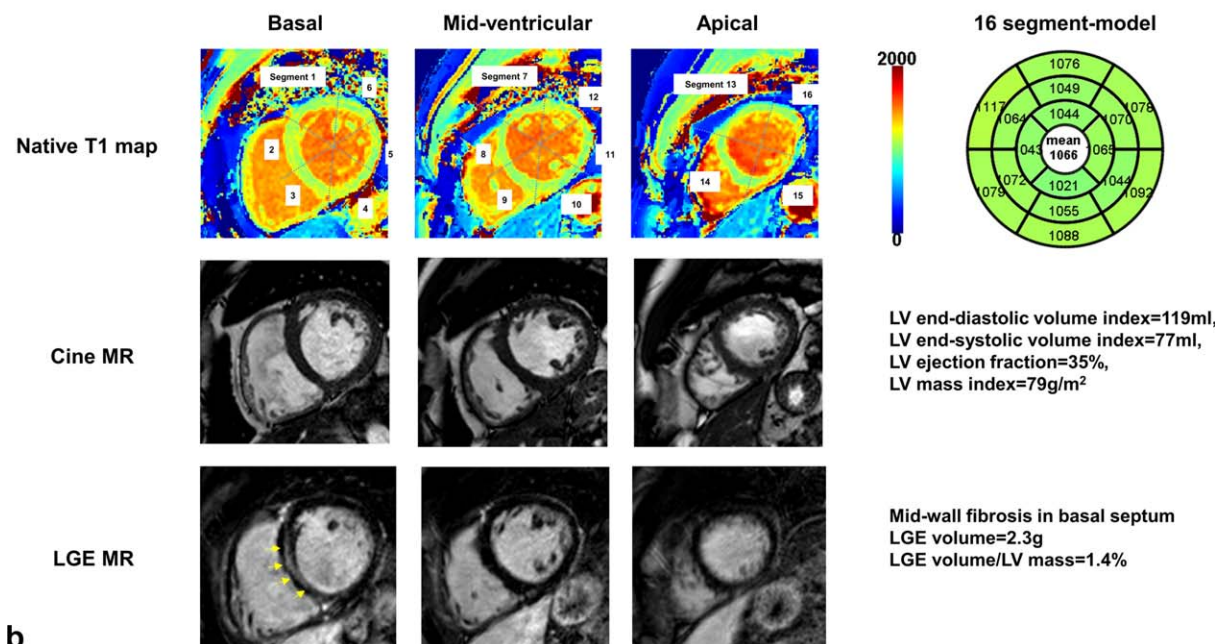
FIGURE 2: Representative cases with and without ComVA. A: A 57-year-old, nonischemic dilated cardiomyopathy patient with ComVA and high global native T_1 of 1156 msec. Severe LV systolic dysfunction was documented with severely dilated LV volume and global wall thinning. LGE image by cardiac MR showed no late gadolinium enhancement. **B:** A 62-year-old, nonischemic dilated cardiomyopathy patient with mid-wall LGE and no ComVA. Similar to case A, there was severe LV systolic dysfunction with dilated LV cavity size. Although mid-wall fibrosis in LGE images was observed in basal septum (arrows) and native T_1 in the corresponding segment was relatively high, global native T_1 was in the normal range. ComVA: complex ventricular arrhythmias, LGE: late gadolinium enhancement.

incremental value of native T_1 mapping in identifying patients with NICM who are at risk for arrhythmic events and who might benefit from implantable cardioverter defibrillator (ICD) implantation and/or VT ablation.

Although there were significant differences between NICM patients with and without ComVA, we found that there is substantial overlap in T_1 values between them. This observation is similar to other studies in cardiac T_1 mapping to discriminate between different patient cohorts based solely on myocardial T_1 values.[12,13] This is an important observation that warrants further evaluation. There are many sources in variability that could contribute to this
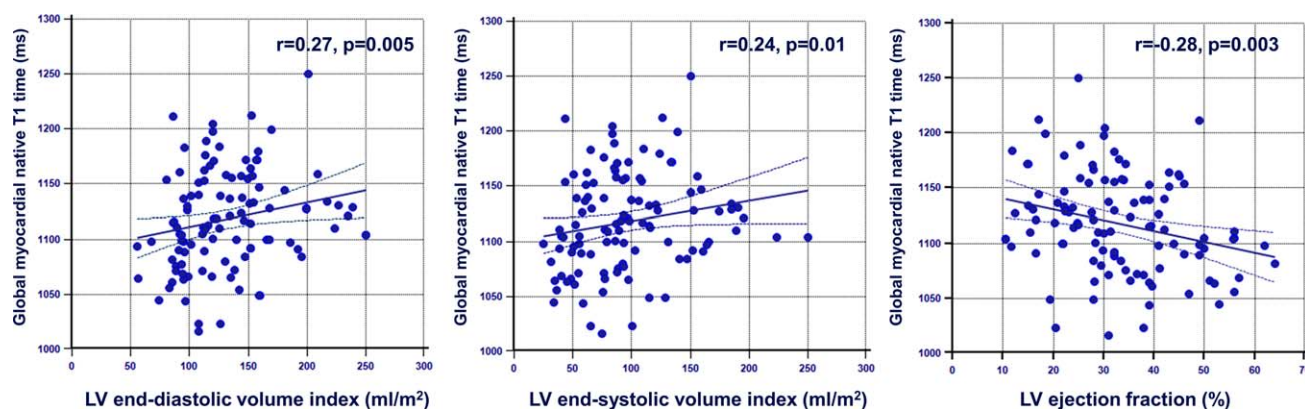
FIGURE 3: Correlation between native T$_1$ time and LV volumes and ejection fraction. Global native T$_1$ time was mildly correlated with LV (A) end-diastolic volume index, (B) end-systolic volume index, (C) ejection fraction.

overlap, including pulse sequence[34,35] and natural differences among different patients even within the same group. The latter variability is an important one that has clinical implications. While we categorize all these patients into one single large group, ie, NICM, there may be differences between them. Currently, more precise phenotyping of NICM patients is a major unmet clinical need that is being investigated using different approaches including genetic, proteomics, and blood biomarkers.[36–38] Myocardial T$_1$ mapping could also potentially have a role in phenotyping of these patients and warrants further studies. However, this requires a very large sample size, which was beyond the scope of this study.

Our study has several limitations. This is a single-center study with a small sample size. Our cohort included patients with nonsustained VT as well as sustained VT. The primary mechanism of these arrhythmias may be different, and a high proportion of NICM have nonsustained VT of unclear clinical significance. However, nonsustained VT and a higher burden of ventricular ectopy have been shown to be associated with increased risk for VT/VF and ICD therapies in patients with NICM.[39,40] Myocarditis was excluded based on medical, history, physical examination, ECG, echocardiography, and cardiac MR findings. None of patients had any ongoing symptoms suggesting presence of myocardial edema or inflammation. However, we cannot rule out edema in our patients. There are other mechanisms besides fibrosis that can alter T$_1$. Intramyocardial lipid accumulation and iron overload will change native T$_1$, resulting in the "pseudo normalization" of myocardial native T$_1$ even if diffuse myocardial fibrosis exists. Our T$_1$ mapping sequence cannot separate contribution of these imaging confounders in T$_1$ values.

In conclusion, we found that NICM patients with ComVA have significantly increased native T$_1$-derived diffuse myocardial fibrosis, compared to those without ComVA. Global native T$_1$ was independently associated with ComVA, after adjusting for LV function and LGE. Further studies are needed to confirm whether these results provide incremental value to LGE and LV function for risk stratification in patients with NICM at risk for ventricular arrhythmia and SCD.

TABLE 3. Multivariable Logistic Regression Analysis of NICM Patients With ComVA as the Dependent Variables

	Odds ratio	95% CI	P value
Native T$_1$ (each 10-msec increment)	1.14	1.03-1.25	0.008
NYHA functional class	2.70	1.28-5.71	0.009

The clinical characteristics that were univariate predictors of ComVA that were also included in the multivariable model were age ($P = 0.51$), male gender ($P = 0.28$), LV EDV index ($P = 0.87$), and LGE volume (%) ($P = 0.82$).

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