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# Can native T1 mapping sequence be used as a non-invasive alternative imaging tool to LGE sequence for evaluating DCM patients?

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## Abstract

**Background** Myocardial fibrosis is the potential outcome of dilated cardiomyopathy (DCM). Cardiac MRI is considered one of the most essential imaging methods in DCM evaluation by using the late gadolinium enhancement (LGE) sequence and native T1 mapping technique. The study aimed to assess the diagnostic accuracy and reliability of the native T1 mapping technique for detecting myocardial fibrosis in DCM patients and correlate the values with the LGE in such a patient population.

**Results** LGE was present in ten patients (33.33%) and 46 out of 480 myocardial segments (9.58%). T1 native values were significantly higher in the LGE group compared to the non-LGE group ( $P < 0.001$ ). Furthermore, the non-LGE group showed higher T1 native values than the control group. Based on receiver operating characteristics (ROC) curves analysis, the best cut-off value of T1 native for the discrimination between normal myocardium and DCM was 1060 ms, while 1125 ms was the optimal cut-off value for LGE prediction among DCM patients (AUC; 0.919 and 0.904), respectively.

**Conclusions** Native T1 mapping technique can be used as a simple, practical, and reproducible method for characterizing myocardial fibrosis in patients with DCM with high diagnostic accuracy and specificity.

**Keywords** Cardiomyopathy, Cardiac MRI, Contrast media, Myocardial disease, Prognosis

## Background

Dilated cardiomyopathy (DCM) is a non-ischæmic myocardial disease characterized by anatomical and functional cardiac alterations, in the absence of hypertension, congenital heart disease, coronary artery disease and valvular disease. The clinical picture of DCM is characterized by left or biventricular dilatation and systolic impairment [1]. This clinical condition accounts for many

newly reported heart failure (HF) patients and is the most common cause of heart transplantation in adolescents and adults [2].

A key factor in the progression of myocardial dysfunction in various cardiomyopathies is myocardial fibrosis which results in myocardial remodeling and poor outcomes [3, 4]. In the later phases of the disease, replacement fibrosis develops when cellular damage and cardiomyocyte necrosis/apoptosis occur [5].

Although endomyocardial biopsy is the gold standard for identifying heart muscle abnormalities, its broad use in clinical practice is constrained by its invasiveness, poor clinical utility, risk of sampling errors, and paucity of well-established therapeutic approaches [6]. Currently, the gold standard for the clinical diagnosis of

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focal myocardial fibrosis is cardiac magnetic resonance (MR) with late imaging following injection of a gadolinium-based contrast agent (late gadolinium enhancement (LGE)) [7–9].

About 30% of individuals with dilated cardiomyopathy exhibit mid-wall LGE in sites that do not match a coronary artery territory [8–10]. Myocardial replacement fibrosis detected by CMR-LGE in DCM provides prognostic information because it is associated with an elevated mortality risk, ventricular arrhythmia, decompensated heart failure, and sudden cardiac death [7, 11].

It is known that patients with HF and end-stage chronic kidney disease (CKD) cannot receive gadolinium-based contrast agents due to the possibility of developing nephrogenic systemic fibrosis. Thus, an alternative non-invasive method for evaluating myocardial fibrosis is needed, especially in such a patient population [12].

The T1 mapping technique is a non-invasive imaging tool for myocardial tissue characterization. Moreover, it can identify, and measure diffuse and focal abnormalities in myocardial structure [13–15]. Native T1 mapping can detect myocardial replacement fibrosis, consistent with LGE in DCM [16]. Furthermore, according to Puntmann et al. [17], native myocardial T1 values are the most effective method for differentiating between normal and diffusely diseased myocardial tissue, such as that affected by hypertrophic cardiomyopathy (HCM) and DCM.

This study aims to estimate the diagnostic accuracy and reliability of the native T1 values derived from pre-contrast T1 mapping for detecting myocardial fibrosis in DCM patients and correlate the values with the late gadolinium enhancement in such a patient population.

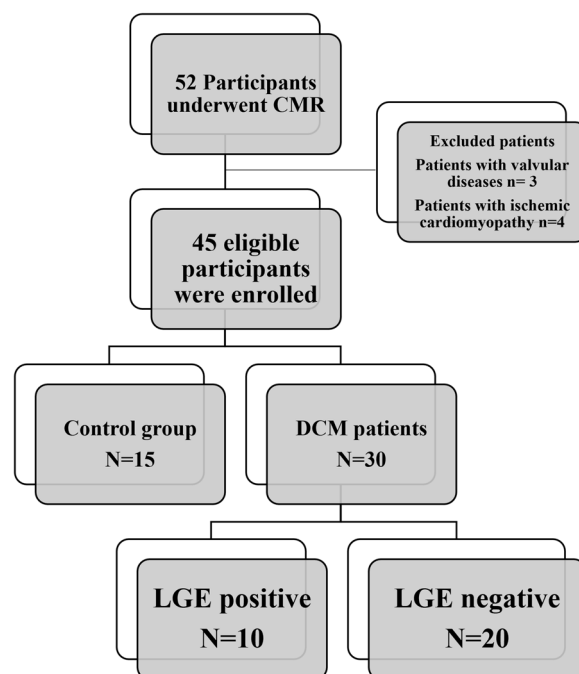
## Methods

### Study design and population

The current study followed the Standards for Reporting Diagnostic Accuracy (STARD) statement recommendations [18]. This research has been approved by our institutional review board (reference number: 5793), and informed written consent was obtained from each patient. We followed the Declaration of Helsinki's ethical concepts.

This was a prospective single-center study. A total of 30 consecutive patients (24 males and six females) with non-ischemic dilated cardiomyopathy (NIDCM) were recruited prospectively from the cardiology department between September 2020 and November 2022.

Inclusion criteria were: (i) patients with proven DCM based on medical history, clinical examination, ECG, echocardiography, and coronary angiography (i.e., NIDCM was defined as an increase of left ventricular volume (LV end diastolic volume (EDV) > 117%) coupled with reduction of the systolic function



**Fig. 1** Flow chart of the research process

globally with ejection fraction (EF) < 45%) [19], and (ii) patients  $\geq 18$  years old. Exclusion criteria were: (i) ischemic cardiomyopathy based on coronary artery disease on coronary angiography (n=4), (ii) myocarditis, (iii) severe valvular diseases (n=3), (iv) renal dysfunction with estimated glomerular filtration rate (eGFR) (< 30 ml/min/m<sup>2</sup>), (v) allergy to MRI contrast media, and (vi) general contraindications to MRI scan, such as cochlear implants, claustrophobia, and non-MRI conditional pacemaker.

Fifteen healthy participants (14 males and one female) who had a normal clinical examination, normal ECG, and did not have a history of cardiac diseases, cardiac risk factors, or systemic diseases were enrolled as a control group. Once recruited, both groups underwent CMR with a non-contrast T1 mapping sequence. However, the DCM group only underwent a post-contrast LGE sequence to evaluate myocardial fibrosis. The study process flow chart is illustrated in (Fig. 1).

### Protocol of cardiac MRI

#### Patient preparation

A full explanation of the CMR exam and breathing instructions were provided for each subject. All subjects were instructed to fast for 4–6 h without discontinuing the medications. Participants were asked to void before the procedure and to remove any metal objects. Then, an IV line was inserted at the antecubital vein. ECG triggering was used for cardiac gating, the skin should be

clean for better ECG electrode contact, and the participant should be positioned supine. The ECG signal was checked; if it was inaccurate, the position of the electrode was changed. A digital Body coil was placed over the chest and tightened securely with straps to prevent respiratory motion artifacts. Cushions under the knee and the head were provided for better comfort. The laser beam was centered over the mid-chest at the level of the nipples.

#### **Scan protocol and parameters**

A 1.5 T scanner (Achieva 1.5 T, Philips Medical Systems, Best, the Netherlands), a 8-channel coil and ECG gating were used for the CMR examination.

After cardiac planning, cine images were obtained utilizing breath-hold steady-state free precession (SSFP) sequence in 2, 3, and 4 chamber views. Short axis stack was obtained to cover the left ventricle (LV) from the base to its apex with a slice thickness of 8 mm and a 2 mm interslice gap. The parameters: TR=2.8 ms, TE=1.41 ms, flip angle=60°, field of view (FOV)=300×300 mm, and imaging matrix=176×170 were used to obtain cine sequences.

In a single breath hold, native T1 mapping was carried out at three short-axis levels (basal, mid, and apical left ventricular level) utilizing the modified look-locker inversion-recovery (MOLLI) sequence with an inversion-recovery single-shot (SSFP) using the following parameters: TE and TR were 1.07 and 2.4 ms, respectively; slice thickness was 10 mm; flip angle was 35°; FOV was 300×300 mm; and imaging matrix was 152×150.

LGE sequence (inversion-recovery technique) in a short axis covering the whole left ventricle was acquired 10 min after injecting a dose of 0.15 mmol/kg gadopentetate dimeglumine (Magnevist) and 30 ml isotonic saline flush. The used parameters were slice thickness of 10 mm; flip angle of 15°; FOV of 300×300 mm; imaging matrix of 124×121; TR of 5.4 ms; and TE of 2.7 ms. Inversion time (TI) nullifying the myocardium was determined using T1 scout imaging (Look-locker). Cine, T1 mapping, and LGE imaging planes were matched.

#### **Image analysis and interpretation**

All images were individually evaluated and analyzed using Philips software (IntelliSpace Portal [ISP]) by two experienced radiologists (4- and 5-year of experience in CMR imaging interpretation), blinded to patients' clinical data.

The following MRI-derived parameters and features were assessed for each patient. (i) LV function and volumes were evaluated on short-axis cine MR images. The

LV epicardial and endocardial borders were semi-automatically delineated at end-diastolic (ED) and end-systolic (ES) images. The volume of the LV cavity included the papillary muscles. Using Simpson's rule, the LVED and LVES volumes were automatically calculated and standardized to the body surface area. (ii) Focal scar tissue: myocardial scar was detected using the myocardial LGE sequence, and it can be distinguished from nulled myocardium subjectively by the myocardial post-contrast signal intensity, which is considerably brighter. When LGE was established, the pattern was evaluated. (iii) Myocardial native T1 values for detection of fibrosis/scar: according to the AHA recommendation for the regional study of LV myocardium, omitting the apical segment, pre-contrast T1 mapping images generated in three short-axis sections (basal, mid, and apical LV level) were split into 16 segments [20]. To quantify the myocardial native T1 value, the region of interest (ROI > 20 pixels) was drawn, and the partial volume averaging artifact was avoided. The ROIs excluded the epicardial fat and LV cavity. Blindly drawn ROIs were placed on the relevant LGE images. The ROI was set up to incorporate the LGE once it was established.

#### **Statistical analysis**

Data analysis was conducted using the SPSS (Statistical Package for the Social Sciences) software version 26. Categorical data were described using numbers and frequencies and were compared by Chi-square or Fisher exact tests when appropriate. The Shapiro–Wilk test was utilized to identify the normality of the continuous data. Continuous data were described using means and standard deviations. Furthermore, continuous data between groups were compared using a one-way ANOVA test in normally distributed data. ROC curve was utilized to calculate the best cut-off of T1 native value for detecting myocardial fibrosis of DCM and for predicting LGE in DCM patients. The inter-reader agreement regarding T1 native values was assessed using Intra class correlation test with ICC of less than 0.5, 0.5–0.75, 0.75–0.9 and more than 0.9 indicates poor, fair, good and excellent agreement, respectively. A  $P < 0.05$  was considered as statistically significant, while  $P \leq 0.001$  was defined as highly statistically significant.

## **Results**

#### **Basic and clinical characteristics of the studied cohort**

A total of 30 DCM patients (24 males and six females) and 15 healthy participants (14 males and one female) were enrolled in the current study. The mean age of the DCM and control groups was  $38.6 \pm 9.89$  and  $33.72 \pm 2.72$ ,

respectively. Ten patients showed LGE, and 20 patients showed non-LGE in the post-contrast LGE sequence. NYHA classification showed a statistically significantly higher class in the LGE group than in the non-LGE group ( $P=0.045$ ). Moreover, there was a statistically significant difference between both groups concerning the functional parameters of the left ventricle (LVEF, LVESV, Index LVESV, LVEDV, Index LVEDV, ED LV mass, and Index ED LV mass) ( $P<0.001$ ). LGE was detected in ten patients (33.33%) and 46 out of 480 myocardial segments (9.58%). LGE was present in 32 out of 150 (basal, midventricular, or apical) septal segments (21.33%). However, of the 330 non-septal segments, 14 segments (4.42%) showed LGE. In 22 segments (68.75%), the septal LGE showed a mid-wall stria pattern, while ten segments (31.25%) displayed a patchy pattern in the right ventricular insertion sites. The basic and demographic

characteristics of the participants are demonstrated in (Table 1).

**Diagnostic validity of T1 native mapping values for diagnosing DCM**

Based on a segment-by-segment analysis, T1 native values showed high diagnostic accuracy in diagnosing DCM. T1 native values showed a sensitivity, specificity, and accuracy of 70.6, 96.3, and 79.2%, respectively. The diagnostic accuracy of the T1 native values is described in (Table 2).

**Diagnostic validity of T1 native mapping values for predicting LGE in DCM patients**

As shown in (Table 3), T1 native values showed a sensitivity of 78.3%, specificity of 85.7%, and accuracy of 85% in predicting LGE among DCM patients.

**Table 1** Basic and clinical characteristics of the patients

	DCM LGE group (n = 10)	DCM Non-LGE group (n = 20)	Control group (n = 15)	P value
Age (mean ± SD)	39 ± 8.12	38.4 ± 10.86	33.72 ± 2.72	0.473 <sup>‡</sup>
Sex (n; %)				
Male	7 (70%)	17 (85%)	14 (93.3%)	0.422 <sup>■</sup>
Female	3 (30%)	3 (15%)	1 (6.7%)	
Body surface area (BSA) (mean ± SD)	1.92 ± 0.14	1.88 ± 0.17	1.92 ± 0.1	0.609 <sup>‡</sup>
Smokers (n; %)	Zero (0%)	5 (25%)	1 (6.7%)	0.156 <sup>■</sup>
Vital data (mean ± SD)				
Pulse	72.1 ± 4.48	69.45 ± 6.56	68.87 ± 5.37	0.368 <sup>‡</sup>
Systolic blood pressure	110.5 ± 4.97	113.75 ± 7.05	114.67 ± 4.81	0.223 <sup>‡</sup>
Diastolic blood pressure	71 ± 5.16	73.5 ± 6.3	74.67 ± 4.42	0.27 <sup>‡</sup>
Co-morbidities (n; %)				
Diabetes Mellitus (DM)	2 (20%)	2 (10%)		0.584*
Atrial fibrillation (AF)	4 (40%)	4 (20%)		0.384*
Drugs (n; %)				
Beta blockers	8 (80%)	15 (75%)		0.99*
ACEI/ARBS	10 (100%)	17 (85%)		0.532*
Diuretic	5 (50%)	6 (30%)		0.284*
New York Heart Association (NYHA) (n; %)				
Class 2	4 (40%)	16 (80%)		<b>0.045*</b>
Class 3	6 (60%)	4 (20%)		
Functional parameters of the left ventricle (LV) (mean ± SD)				
LVEF (%)	22.5 ± 5.28	29.05 ± 3.94	62.07 ± 1.67	<b>&lt; 0.001<sup>‡</sup></b>
LVESV (ml)	228.09 ± 57.03	167.47 ± 33.1	63.94 ± 5.75	
LVESV index (ml/m <sup>2</sup> )	118.84 ± 28.51	89.55 ± 18.41	33.22 ± 2.63	
LVEDV (ml)	294.91 ± 62.1	235.78 ± 38.32	170.65 ± 10.87	
LVEDV index (ml/m <sup>2</sup> )	153.54 ± 29.55	125.96 ± 20.74	88.78 ± 5.28	
ED LV mass (gm)	169.51 ± 27.38	147.91 ± 22.53	86.91 ± 9.07	
ED LV mass index (gm/m <sup>2</sup> )	88.24 ± 11.15	78.67 ± 9.25	45.12 ± 3.69	

Bold values denote that the p-value is significant in that variable

LGE late gadolinium enhancement, EF ejection fraction, LV left ventricle, ESV end systolic volume, EDV end diastolic volume

<sup>‡</sup> One way ANOVA test; <sup>■</sup> Chi square test; \*Fisher Exact test; P < 0.05 is statistically significant, P < 0.01 is highly statistically significant

**Table 2** Diagnostic accuracy of T1 native values in detecting myocardial fibrosis in DCM

	T1 native value
AUC	0.919
Cut off	≥ 1060 ms
Sensitivity	70.6%
Specificity	96.3%
PPV	97.4%
NPV	62.1%
Accuracy	79.2%

AUC area under curve, CI confidence interval, PPV positive predictive value, NPV negative predictive value

**Table 3** Diagnostic accuracy of T1 native values in predicting LGE among DCM patients

	T1 native value
AUC	0.904
Cut off	≥ 1125 ms
Sensitivity	78.3%
Specificity	85.7%
PPV	36.7%
NPV	97.4%
Accuracy	85%

AUC area under curve, CI confidence interval, PPV positive predictive value, NPV negative predictive value

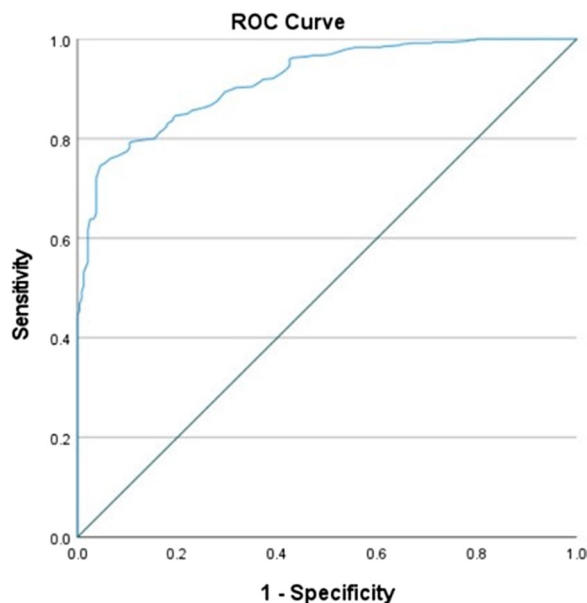
**Analysis of ROC curves**

**ROC curve of T1 native values in detecting myocardial fibrosis in DCM**

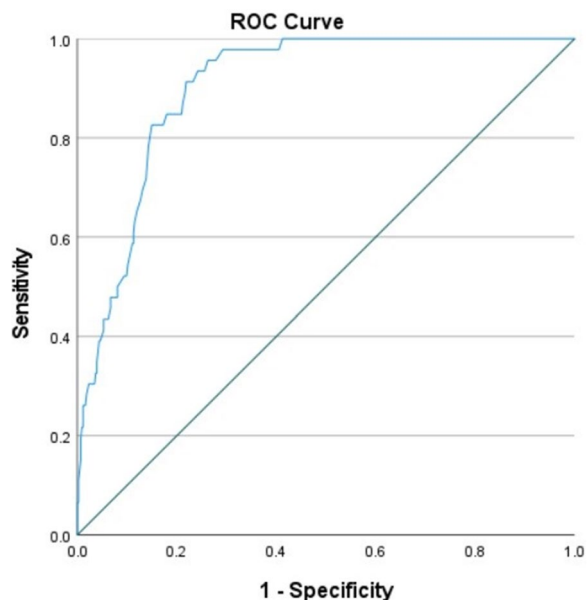
We utilized the ROC curve to calculate the optimal cut-off value for detecting myocardial fibrosis in DCM based on the diagnostic validity of T1 native values (Fig. 2). T1 native value of ≥ 1060 ms was the optimal cut-off value for distinguishing between myocardial fibrosis in DCM and normal myocardium in healthy controls, with an area under the curve (AUC) of 0.919 (Table 2).

**ROC curve of T1 native values in predicting LGE among DCM patients**

The ROC curve was utilized to estimate the best cut-off value for LGE prediction among DCM patients based on the diagnostic validity of T1 native values (Fig. 3). T1 native value of ≥ 1125 ms was the optimal cut-off value for predicting LGE in DCM cases, with an area under the curve (AUC) of 0.904 (Table 3).



**Fig. 2** ROC curve analysis of T1 native values in detecting myocardial fibrosis in DCM



**Fig. 3** ROC curve analysis of T1 native values in predicting LGE among DCM patients

**Comparison between the control group and the DCM group (non-LGE and LGE) concerning T1 native values**

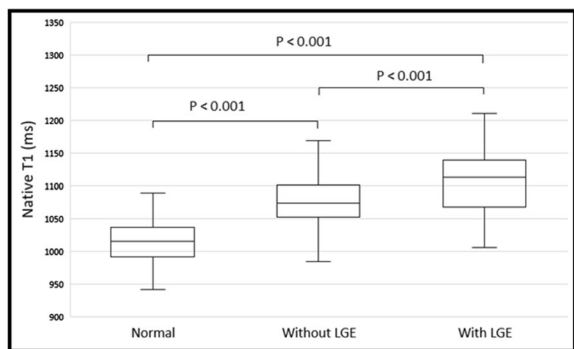
The comparison between the studied groups concerning T1 native values is summarized in (Table 4). LGE segments showed significantly higher T1 native values

**Table 4** Comparison between the control and DCM groups (LGE and non-LGE segments) concerning T1 native values

Group	LGE positive group N = 46 segments	LGE negative group N = 434 segments	Control group N = 240 segments	P value
T1 native value (mean ± SD)	1147.43 ± 30.49 ms	1080.40 ± 40.04 ms	1014.61 ± 29.46 ms	<0.001*

\* One way ANOVA test; P < 0.05 is statistically significant and P < 0.001 is highly significant

LGE late gadolinium enhancement

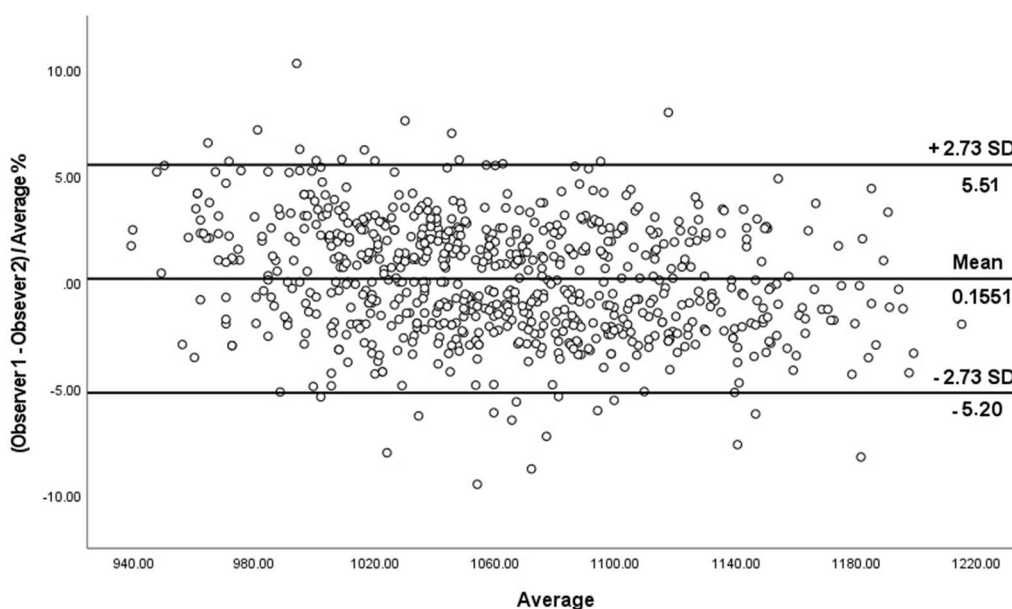


**Fig. 4** Comparison between the studied groups regarding T1 native values

than non-LGE segments and those of the control group ( $P < 0.001$ ; Fig. 4).

**Inter-reader agreement regarding T1 native values measurement**

As illustrated in (Fig. 5), there was an excellent agreement between both readers regarding T1 native values measurement with ICC = 0.928.



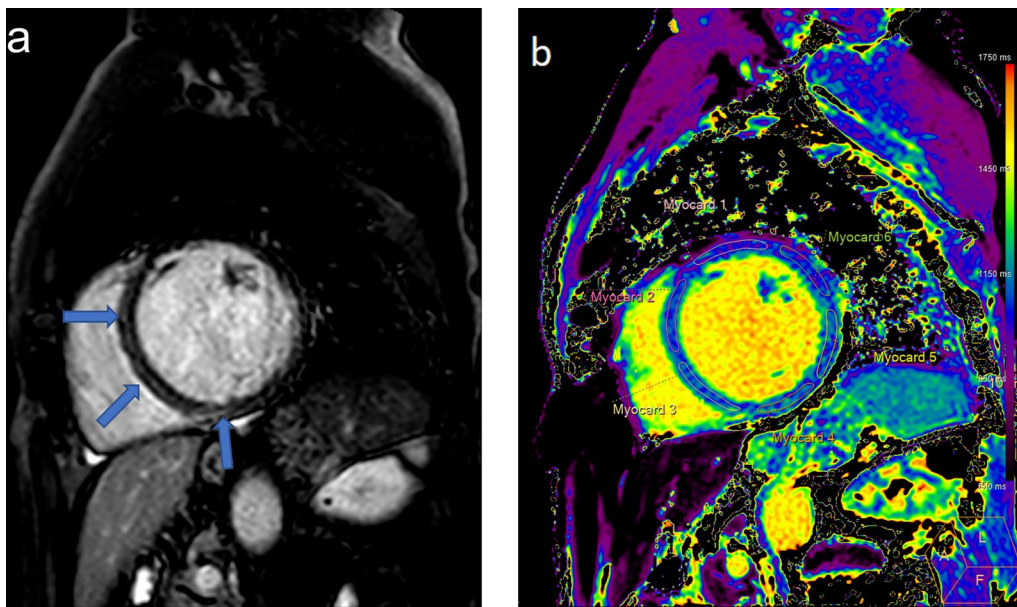
**Fig. 5** Intra class correlation between the 2 readers concerning T1 native values

Our cases are illustrated in (Figs. 6 and 7).

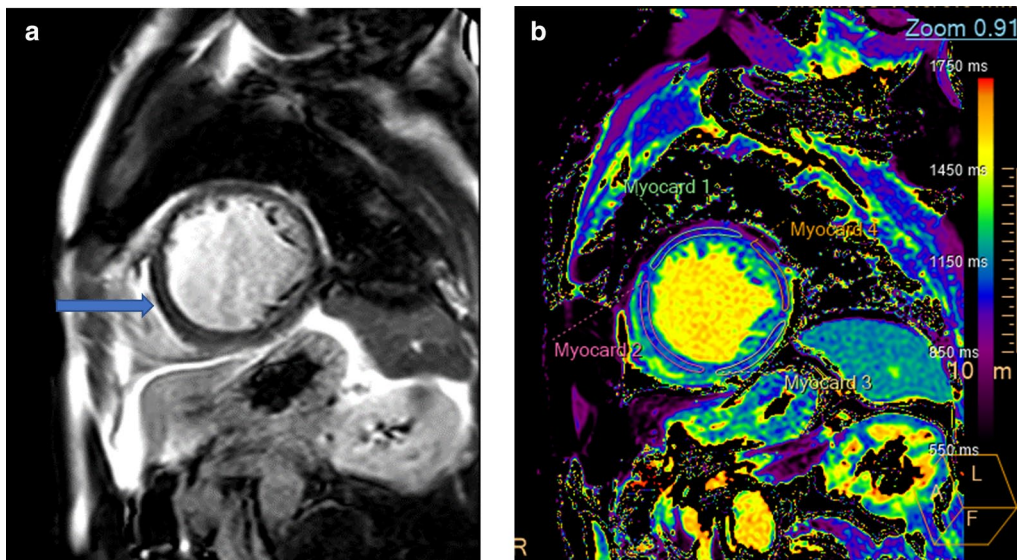
**Discussion**

The current study attempts to enrich the literature with the clinical significance and the diagnostic performance of native T1 mapping in detecting myocardial fibrosis in DCM patients without needing a post-contrast LGE sequence. Previous studies reported that native T1 techniques could be considered a valid and reliable imaging tool for evaluating myocardial fibrosis with high diagnostic accuracy [15, 17, 21]. Moreover, Puntmann et al. [22] and Li et al. [23] reported that the native T1 mapping technique was used to assess diffuse myocardial fibrosis, which predicts heart failure and high mortality rates.

The current study demonstrates robust evidence about the high diagnostic accuracy and specificity of T1 native values in detecting and quantifying myocardial fibrosis in DCM. We included 30 DCM patients and 15 healthy participants. Ten patients showed LGE, while 20 patients showed non-LGE in the post-contrast sequence. Furthermore, there was a statistically significant difference between LGE and non-LGE groups concerning NYHA



**Fig. 6** Thirty-nine-year-old male with dilated cardiomyopathy (DCM), presented with dyspnea and easy fatiguability. **a** Late gadolinium enhancement (LGE) is seen in the mid-wall of the antero-septal, infero-septal and inferior segments (arrow). **b** Native T1 mapping shows that the native T1 value of the antero-septal segment (Myocard 2) is 1139 ms, native T1 value of the infero-septal segment (Myocard 3) is 1148 ms, and native T1 value of the inferior segment (Myocard 4) is 1165 ms, which are more than 1125 ms. The native T1 value of the anterior (Myocard 1), infero-lateral (Myocard 5) and antero-lateral (Myocard 6) segments was 1068, 1087 and 1079 ms respectively



**Fig. 7** Forty-five-year-old male with dilated cardiomyopathy (DCM), presented with symptoms and signs of heart failure. **a** Late gadolinium enhancement (LGE) is seen in the mid-wall of the septal segment (arrow). **b** Native T1 mapping shows elevated native T1 value of the septal segment (Myocard 2) (1145 ms). Native T1 values of the anterior segment (Myocard 1), inferior segment (Myocard 3), and lateral segment (Myocard 4) were 1075 ms, 1092 ms and 1084 ms respectively

class and MRI-derived functional parameters, such as EF, LVEDV, LVESV and LV mass. On ROC curve analyses, the T1 native value of 1060 ms was recorded as the optimal cut-off value for the discrimination between

myocardial fibrosis in DCM and normal myocardium with a sensitivity of 70.6% and a specificity of 96.3%. Moreover, the T1 native value of 1125 ms was reported as the best cut-off for predicting LGE in DCM patients, with

a sensitivity of 78.5% and specificity of 85.7%. In comparing the studied groups concerning T1 native values, myocardial segments with LGE showed higher T1 native values than non-LGE segments. Consequently, non-LGE segments showed higher T1 native values than myocardial segments of the control group.

Regarding MRI-derived functional parameters and NYHA classes between the studied groups, Alba et al. [11] recorded that the LGE group had a lower EF and higher LVESV, LVEDV, LV mass index and NYHA score than the non-LGE group. These findings are in agreement with our results. Another study [10] in the UK reported a significant difference between LGE and non-LGE groups regarding EF, LVESV, and LVEDV; however, LV mass and NYHA score did not differ. These results partly agree with our report concerning EF, LVESV, and LVEDV but are not in line with the current study findings regarding LV mass and NYHA score. This could be attributed to the small number of included DCM patients in our study.

Concerning the diagnostic accuracy of T1 native values in detecting myocardial fibrosis in DCM, Puntmann et al. [17] recorded a T1 native value cut-off of 1184 ms for diagnosing myocardial fibrosis with a sensitivity and a specificity of 100% and 97%, respectively. We recorded a lower sensitivity than Puntmann et al. (70.6% V 100%). This difference could be explained by the fact that their study included mixed cohorts of hypertrophic cardiomyopathy and DCM and a relatively larger sample size. Moreover, CMR examination was performed using a 3 T machine versus 1.5 T in our study. Another team [24] in Egypt conducted a study to investigate the diagnostic performance of T1 native mapping in assessing myocardial fibrosis in cardiomyopathy and reported a T1 native cut-off value of 1070 ms for diagnosing myocardial fibrosis with a sensitivity of 66% and a specificity of 68.5%. These findings align with our report regarding the cut-off value, but our study showed a higher specificity than the El-safty et al. study [24] (96.3% V 68.5%). This difference might be attributed to the higher prevalence of diseased subjects in their study and the enrollment of a mixed cohort with different myocardial pathologies, such as HCM, DCM, and infiltrative diseases.

When investigating the best T1 native cut-off value in predicting LGE, Yanagisawa et al. [25] recorded a T1 native value of 1349.4 ms with a sensitivity of 75% and a specificity of 92%. This cut-off value is higher than that recorded in the current report (1349.4 V 1125 ms) and with similar diagnostic accuracy. To the best of our knowledge, T1 native values are usually affected by field strength, the cardiac phase, the pulse sequence used, and the ROIs performed for T1 native values measurement. Therefore, there are differences in the T1 native optimal

cut-off value for assessing myocardial fibrosis between MRI scanners.

Regarding comparing the T1 native values between the studied groups in our cohort, our findings agree with Dass et al. [26], who conducted similar research and reported that LGE segments showed higher T1 native values than non-LGE segments of DCM patients. Furthermore, non-LGE segments showed higher T1 native values than segments of healthy participants. These results suggest that LGE and T1 mapping assess overlapping pathologies. Consequently, T1 mapping evaluates the interstitium and the myocytes, thus investigating the conditions that affect the myocardium diffusely, while LGE denotes extracellular myocardial disease. In contrast, Iles et al. [27] found no significant difference in myocardial T1 native values between the patients with heart failure and the healthy subjects. This could be explained by the fact that they included heart failure patients and used a different T1 mapping sequence.

In terms of T1 native values reliability, Puntmann et al. [17] and Yanagisawa et al. [25] recorded an excellent inter-observer agreement regarding T1 native values estimation with ICC of 0.98 and 0.88, respectively. These results are in line with our report. Therefore, T1 mapping technique is considered a reliable and reproducible imaging tool.

Based on the current study findings, the native T1 mapping sequence can be used as a non-invasive and alternative imaging tool to LGE sequence for evaluating DCM patients and detecting focal and diffuse myocardial fibrosis without the need for contrast media which is crucial in patients with renal dysfunction to reduce the incidence of nephrogenic systemic fibrosis in such a patient population. Moreover, Nakamori et al. [28] found that T1 native values correlate well with the histological collagen fraction in DCM patients. Therefore, native T1 mapping is a promising imaging method for the follow-up to assess the serial changes of the myocardium with high diagnostic accuracy for better assessment of patient outcomes.

The current study has some strengths. First, the T1 mapping technique was performed using the MOLLI sequence, considered a reliable and accurate technique in T1 relaxation time assessment. Second, we enrolled DCM patients only to evaluate myocardial fibrosis in such a population. However, there are several limitations to our study. First, the sample size was small and the LGE patients are low in number; however, 720 ROIs were evaluated in DCM and control groups. Thus, we recommend further prospective multi-centeric studies with a large sample size to investigate the validity of native T1 in diagnosing DCM. Second, it was a single-center study. Third, myocardial T1 native values are usually affected by the MRI field strength and the used protocol. Therefore,



the recorded T1 native cut-off values cannot necessarily apply to other institutions. However, we conducted such a study trying to set a cutoff value for our department device. Fourth, post-contrast T1 mapping was not performed. Fifth, there was no correlation between MRI imaging findings and the histological evaluation in DCM patients because the endomyocardial biopsy has a significant risk, although it is the reference standard. Instead, we depended on clinical examination, functional echocardiography parameters, and ECG, excluding ischemic cardiomyopathy by normal coronary angiogram. Finally, the prognosis of the DCM patients was not evaluated.

Regarding the overall results of the current study, we recommend conducting future longitudinal prospective studies to validate these findings. Furthermore, we assessed the inter-rater variability to provide evidence for the stability of the technique. However, we recommend using this technique by low experienced readers or early trainers to prove the reproducibility of it.

## Conclusions

Native T1 mapping technique can be used as a simple, practical, and reproducible method for characterizing myocardial disease in patients with DCM with high diagnostic accuracy and specificity. It can evaluate focal and diffuse myocardial fibrosis using native T1 optimal value without contrast media administration. Therefore, the native T1 mapping sequence can be used as an alternative imaging technique to the LGE sequence for patients with contrast media contraindications, such as patients with renal insufficiency and contrast allergy. Moreover, this may be also of value in low resource countries due to shortage of the contrast agents.

## Abbreviations

DCM	Dilated cardiomyopathy
LGE	Late gadolinium enhancement
HF	Heart failure
CMR	Cardiac magnetic resonance
CKD	Chronic kidney disease
HCM	Hypertrophic cardiomyopathy
NIDCM	Non-ischemic dilated cardiomyopathy
LV	Left ventricle
EDV	End diastolic volume
ESV	End systolic volume
EF	Ejection fraction
SSFP	Steady state free precession
FOV	Field of view
TE	Time echo
TR	Time repetition
MOLLI	Modified look-locker inversion recovery

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Not applicable.

## Author contributions

S.E. and N.Y. carried out the study concept and design, participated in the sequence alignment and drafted the manuscript. E.Z. and S.S. carried out

the process of literature search. G.K., H.S. and R.M. participated also in the sequence alignment and participated in the design of the study. S.E. and N.Y. performed the statistical analysis. All authors read and approved the final manuscript.

## Funding

Not applicable.

## Availability of data and materials

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

## Declarations

### Ethics approval and consent to participate

This study was approved by the Institutional Review Board (IRB) of Zagazig University. Written informed consents from all patients before the study were filled and signed, which are also approved by the Institutional Review Board (IRB) of Zagazig University with Reference Number: 5793.

### Consent for publication

All patients included in this research gave written informed consent to publish the data contained within this study.

### Competing interests

The authors declare that they have no competing interests.

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## References

- McKenna WJ, Maron BJ, Thiene G (2017) Classification, epidemiology, and global burden of cardiomyopathies. *Circ Res* 121:722–730
- Towbin JA, Lowe AM, Colan SD et al (2006) Incidence, causes, and outcomes of dilated cardiomyopathy in children. *JAMA* 296:1867–1876. <https://doi.org/10.1001/jama.296.15.1867>
- Hill JA, Olson EN (2008) Cardiac plasticity. *N Engl J Med* 358:1370–1380
- Ho CY, Lopez B, Coelho-Filho OR et al (2010) Myocardial fibrosis as an early manifestation of hypertrophic cardiomyopathy. *N Engl J Med* 363:552–563
- Weber KT, Brilla CG (1991) Pathological hypertrophy and cardiac interstitium. Fibrosis and renin-angiotensin-aldosterone system. *Circulation* 83:1849–1865
- Yilmaz A, Kindermann I, Kindermann M et al (2010) Comparative evaluation of left and right ventricular endomyocardial biopsy: differences in complication rate and diagnostic performance. *Circulation* 122(9):900–909. <https://doi.org/10.1161/CIRCULATIONAHA.109.924167>
- Sibley CT, Noureldin RA, Gai N et al (2012) T1 Mapping in cardiomyopathy at cardiac MR: comparison with endomyocardial biopsy. *Radiology* 265(3):724–732. <https://doi.org/10.1148/radiol.12112721>
- Pennell DJ (2010) Cardiovascular magnetic resonance. *Circulation* 121(5):692–70520142462
- Vogel-Claussen J, Rochitte CE, Wu KC et al (2006) Delayed enhancement MR imaging: utility in myocardial assessment. *Radiographics* 26:795–810
- Assomull RG, Prasad SK, Lyne J et al (2006) Cardiovascular magnetic resonance, fibrosis, and prognosis in dilated cardiomyopathy. *J Am Coll Cardiol* 48(10):1977–1985
- Alba AC, Gaztañaga J, Foroutan F et al (2020) Prognostic value of late gadolinium enhancement for the prediction of cardiovascular outcomes in dilated cardiomyopathy. *Circ Cardiovasc Imaging* 13(4):e010105. <https://doi.org/10.1161/circimaging.119.010105>
- Thomsen HS, Morcos SK, Almén T et al (2013) Nephrogenic systemic fibrosis and gadolinium-based contrast media: updated ESUR Contrast Medium Safety Committee guidelines. *Eur Radiol* 23:307–318
- Haaf P, Garg P, Messroghli DR et al (2017) Cardiac T1 mapping and extracellular volume (ECV) in clinical practice: a comprehensive review. *J Cardiovasc Magn Reson* 18:89. <https://doi.org/10.1186/s12968-016-0308-4>

14. Diao KY, Yang ZG, Xu HY et al (2017) Histologic validation of myocardial fibrosis measured by T1 mapping: a systematic review and meta-analysis. *J Cardiovasc Magn Reson* 18:92. <https://doi.org/10.1186/s12968-016-0313-7>
15. Mewton N, Liu CY, Croisille P et al (2011) Assessment of myocardial fibrosis with cardiovascular magnetic resonance. *J Am Coll Cardiol* 57:891–903
16. Małek ŁA, Werys K, Kłopotowski M et al (2015) Native T1-mapping for non-contrast assessment of myocardial fibrosis in patients with hypertrophic cardiomyopathy—comparison with late enhancement quantification. *Magn Reson Imaging* 33:718–724
17. Puntmann VO, Voigt T, Chen Z et al (2013) Native T1 mapping in differentiation of normal myocardium from diffuse disease in hypertrophic and dilated cardiomyopathy. *J Am Coll Cardiol Img* 6:475–484
18. Bossuyt PM, Reitsma JB, Bruns DE et al (2015) STARD 2015: an updated list of essential items for reporting diagnostic accuracy studies. *BMJ* 351:h5527
19. Elliott P, Andersson B, Arbustini E et al (2008) Classification of the cardiomyopathies: a position statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J* 29:270–276
20. Cerqueira MD, Weissman NJ, Dilsizian V et al (2002) Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation* 105(4):539–542. <https://doi.org/10.1161/hc0402.102975>
21. Shah RV, Kato S, Roujol S et al (2016) Native myocardial T1 as a biomarker of cardiac structure in non-ischemic cardiomyopathy. *Am J Cardiol* 117(2):282–288. <https://doi.org/10.1016/j.amjcard.2015.10.046>
22. Puntmann OV, Carr-White G, Jabbour A et al (2018) T1-mapping and outcome in nonischemic cardiomyopathy: all-cause mortality and heart failure. *JACC Cardiovasc Imaging* 9(1):40–50. <https://doi.org/10.1016/j.jcmg.2015.12.001>
23. Li S, Zhou D, Sirajuddin A et al (2022) T1 mapping and extracellular volume fraction in dilated cardiomyopathy: a prognosis study. *JACC Cardiovasc Imaging* 15(4):578–590. <https://doi.org/10.1016/j.jcmg.2021.07.023>
24. Elsafty HG, El Shafey M, El Arabawy R et al (2021) Could native T1 mapping replace late gadolinium enhancement in the assessment of myocardial fibrosis in patients with cardiomyopathy? *Egypt J Radiol Nucl Med* 52:222. <https://doi.org/10.1186/s43055-021-00520-2>
25. Yanagisawa F, Amano Y, Tachi M et al (2019) Non-contrast-enhanced T1 mapping of dilated cardiomyopathy: comparison between native T1 values and late gadolinium enhancement. *Magn Reson Med Sci* 18(1):12–18. <https://doi.org/10.2463/mrms.mp.2017-0136>
26. Dass S, Suttie JJ, Piechnik SK et al (2012) Myocardial tissue characterization using magnetic resonance noncontrast T1 mapping in hypertrophic and dilated cardiomyopathy. *Circ Cardiovasc Imaging* 5(6):726–733. <https://doi.org/10.1161/CIRCIMAGING.112.976738>
27. Illes L, Pfluger H, Phrommintikul A et al (2008) Evaluation of diffuse myocardial fibrosis in heart failure with cardiac magnetic resonance contrast-enhanced T1 mapping. *J Am Coll Cardiol* 52(19):1574–1580
28. Nakamori S, Dohi K, Ishida M et al (2018) Native T1 mapping and extracellular volume mapping for the assessment of diffuse myocardial fibrosis in dilated cardiomyopathy. *JACC Cardiovasc Imaging* 11(1):48–59. <https://doi.org/10.1016/j.jcmg.2017.04.006>

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