



Native T1 mapping to detect extent of acute and chronic myocardial infarction: comparison with late gadolinium enhancement technique

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Abstract

Investigate whether native-T1 mapping can assess the transmural extent of myocardial infarction (TEI) thereby differentiating viable from non-viable myocardium without the use of gadolinium-contrast in both acute and chronic myocardial infarction (aMI and cMI). Sixty patients (30 cMI > 1 year and 30 aMI day 2 STEMI) and 20 healthy-controls underwent 1.5 T CMR to assess left ventricular function (cine), native-T1 mapping (MOLLI sequence 5(3)3, motion-corrected) and the presence and TEI from late gadolinium enhancement (LGE) images. Segments with TEI > 75% was considered non-viable. Gold-standard LGE-TEI was compared with corresponding segmental native-T1. Segmental native-T1 correlated significantly with TEI ($R=0.74$, $p<0.001$ in cMI and $R=0.57$, $p<0.001$ in aMI). Native-T1 differentiated segments with no LGE (1031 ± 31 ms), LGE positive but viable (1103 ± 57 ms) and LGE positive but non-viable (1206 ± 118 ms) in cMI ($p<0.01$). It also differentiated segments with no LGE (1054 ± 65 ms), LGE positive but viable (1135 ± 73 ms) and LGE positive but non-viable (1168 ± 71 ms) in aMI ($p<0.01$). ROC analysis demonstrated excellent accuracy of native-T1 mapping compared to LGE-TEI (AUC = 0.88, $p<0.001$ in cMI, vs AUC = 0.83, $p<0.001$ in aMI). Native-T1 performed better in cMI than aMI ($p<0.01$). In cMI a segmental T1 threshold of 1085 ms differentiated viable from non-viable segments with a sensitivity 88% and specificity of 88% whereas a T1 of 1110 ms differentiated viable from nonviable with 79% sensitivity and 79% specificity in aMI. Native-T1 mapping correlates significantly with TEI thereby differentiating between normal, viable, and non-viable myocardium with distinctive T1 profiles in aMI and cMI. Native T1-mapping to detect MI performed better in cMI compared to aMI due to absence of myocardial oedema. Native-T1 mapping holds promise for viability assessment without the need for gadolinium-contrast agent.

Keywords Viability · T1 mapping · Cardiovascular magnetic resonance · Myocardial infarction

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Abbreviations

CMR	Cardiovascular magnetic resonance
LGE	Late gadolinium enhancement
TEI	Transmural extent of infarct
AHA	American Heart Association
LV	Left ventricular
MI	Myocardial infarction
STEMI	ST-elevation myocardial infarction
MVO	Microvascular obstruction
AUC	Area under curve
ROC	Receiver operating characteristic

Introduction

Acute myocardial infarction leads to reversible (viable/stunned) and irreversible(non-viable) myocardial injury, even after successful coronary reperfusion. Early recognition of viable myocardium after a myocardial infarction(MI) is of clinical relevance as the affected segments have the potential for recovery. Targeted revascularization of viable myocardium improves clinical outcomes. Hence viability assessment is a key aspect in the management of ischaemic heart disease (IHD). Late gadolinium enhancement (LGE) CMR is increasingly used in clinical practice as a viability assessment tool [1, 2]. LGE viability assessment is performed by analyzing the extent of scar thickness, with a myocardial segment $\geq 75\%$ transmural deemed mostly non viable. The transmural extent of infarct (TEI) assessed by LGE has been shown to be inversely related to functional recovery after reperfusion [3, 4]. LGE imaging requires administration of gadolinium-based contrast agents. The use of these contrast agents is limited in patients with moderate to severe renal dysfunction due to the potential risk of nephrogenic systemic fibrosis [5]. In addition there have been several reports of gadolinium-based deposits in the central nervous system structures following repeated use of gadolinium contrast, whose clinical meaning is still uncertain [6]. An alternative viability imaging method that might obviate the use of contrast-agents is desirable.

Myocardial T1 mapping is emerging as a novel non-contrast CMR imaging tool in the assessment of non-IHD and IHD [7]. Native T1 mapping technique is highly sensitive to changes in myocardial tissue, thereby providing excellent tissue characterization obviating for the need for contrast administration. Kali et al. looked at diagnostic accuracy of T1-mapping in the detection of overall transmural in chronic MI using a thresholding-based detection [8]. We tested the hypothesis that native segmental T1 mapping can assess the TEI thereby differentiating between viable and non-viable myocardium in IHD without the use of gadolinium contrast.

Aim

The aim of the study was to compare the diagnostic performance of native segmental T1 mapping in quantifying the TEI/myocardial scarring with LGE in chronic as well as acute MI patients using a 1.5 T MRI system.

Methods

Study population

Sixty patients with myocardial infarction were recruited: 30 successfully re-perfused STEMI patients (mean age 61 ± 10 years and 80% males) scanned at day 2 and 30 chronic MI patients (> 1 year after MI) (mean age 67 ± 10 years and 80% males). All patients were diagnosed with MI according to guidelines [9, 10]. Exclusion criteria were: general contraindications to CMR, chronic atrial fibrillation, renal impairment with eGFR < 30 , and cardiogenic shock. 20 age- and sex-matched healthy volunteers served as control (all free of cardiovascular disease). The study was approved by the local Institutional Research and Innovation department. All patients gave informed written consent.

Image acquisition

Patients underwent CMR scans on a 1.5-T scanner (Magnetom Avanto, Siemens Healthneers) with a standard 8-channel matrix coil configuration.

The imaging protocol consisted of three long-axis (four-, three-, and two-chamber view) and a full stack of short-axis steady-state free precession cine images as previously described [11]. This was followed by the acquisition of three short-axis slices (basal, mid-cavity, and apical) by using T1 mapping sequence (details of the sequence provided below). Subsequently, acquisition of the same three short-axis slices (basal, mid-cavity, and apical) was repeated by using a segmented inversion-recovery gradient-echo sequence (IR-GRE) 1–3 min after the intravenous administration of 0.1 mmol/kg of gadobutrol (Gadovist; Bayer Schering Pharma, Berlin-Wedding, Germany) (early gadolinium enhancement EGE). Late gadolinium enhancement (LGE) images were acquired 15–20 min after contrast agent injection in the three long-axis and the full stack of short-axis views [12]. In the acute MI cohort T2weighted STIR(short tau inversion recovery) images were acquired in the short-axis planes covering the entire

left ventricle prior to gadolinium administration, in the corresponding short-axis cine image planes.

Late gadolinium enhancement imaging

Images were acquired at least 15 min following administration of 0.1 mmol/kg gadobutrol. LGE images were obtained using an inversion recovery prepared breath-hold gradient-echo technique. Typical image parameters were TR 700 ms, TE 4.33 ms; matrix 256×256 ; flip angle 30° ; slice thickness 8.0 mm, no interslice gap, and voxel size $1.7 \times 1.4 \times 8$ mm. The inversion time was progressively optimized to null normal myocardium (typical values, 250–350 ms). Images were acquired on both the long- and short-axis planes covering the entire left ventricle. Each slice was obtained during a breath-hold of 10–15 s depending on the patient's heart rate.

T1 mapping sequence

Myocardial T1-mapping was performed using the modified Look-Locker inversion recovery (MOLLI) sequence 5(3)3 (Siemens Healthineers, Germany). The 5(3)3 MOLLI protocol was used to ensure more-complete recovery of the inversion pulse at higher heart rates by acquiring a set of images for at least 5 s after the first inversion pulse, followed by a 3-s pause and then acquiring a set of images after the second inversion pulse for at least 3 s. The acquisition parameters were: pixel bandwidth, 977 Hz/pixel; echo time = 1.12 ms; flip angle = 35° ; matrix = 256×144 ; and 8 mm slice thickness. A nonlinear least-square curve fitting and motion correction were performed at different inversion times with the set of images acquired to generate a pixel-wise colored T1 map by the scanner [13].

T2w STIR (edema imaging)

A breath-hold black-blood segmented turbo spin echo sequence was adopted for T2w STIR imaging, using a triple inversion recovery preparation module in order to suppress signal from flowing blood as well as from fat, with surface coil normalization [14]. Typical imaging parameters were TR 2 R-to-R intervals, TE 75 ms, flip angle 90° , TI 170 ms, slice thickness 8 mm, no interslice gap, field of view 340–400 mm, matrix 208×256 , and a voxel size of $2.3 \times 1.4 \times 8$ mm. Each slice was obtained during a breath-hold of 10–15 s depending on the patient's heart rate. To accommodate poor breath-holders, turbo factor was increased as necessary.

Image analysis

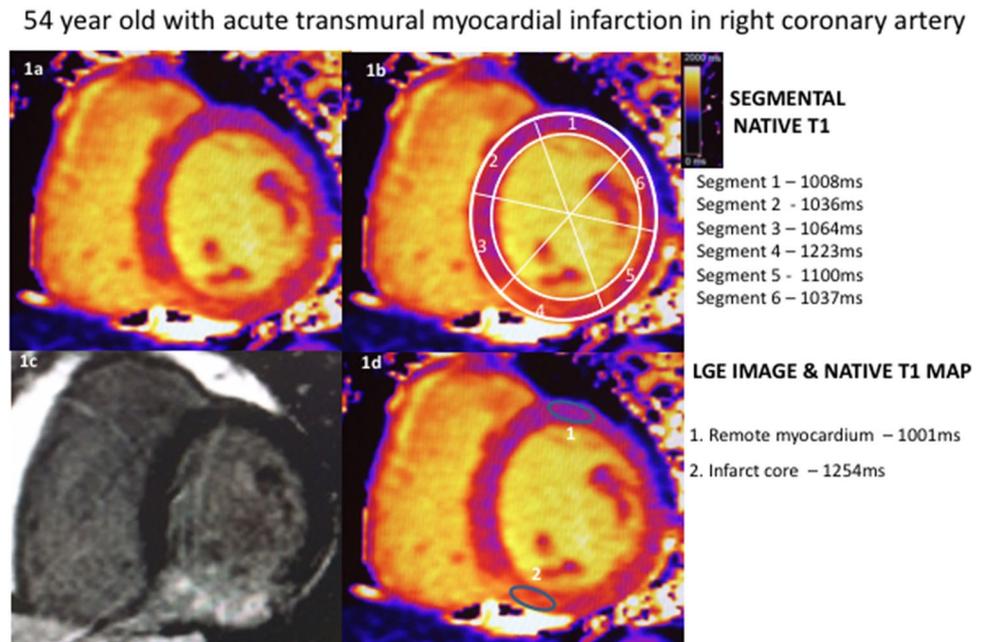
Argus software (Siemens Healthineers, Germany) was used for the quantification of LV volumes and ejection fraction (EF) [15]. Segmental TEI was assessed by contouring the area of LGE and dividing it by the area of the whole segment using full width at half maximum (FWHM) technique using CVI42 software (Circle Cardiovascular Imaging, Calgary, Alberta, Canada).

A scar transmural scale of 0–4 was used for the 16 AHA segment (0 = no scar, 1 = 1–24%, 2 = 25–49%, 3 = 50–74% and 4 = $\geq 75\%$ scar thickness). A scar transmural grade of 3 or less ($< 75\%$ TEI) was deemed viable [3, 16]. Observer blinded to the LGE data (AGD) drew regions of interest within the native T1 map in each of the American Heart Association (AHA) 16 segments in the short axis motion corrected maps using Argus software, adjusting for partial-volume and/or artefact, as previously described [17] Fig. 1. The LGE data was blinded for the T1 mapping analysis in order to test the hypothesis that T1 mapping can assess viability without the need of LGE. Images were randomized for analysis. In patients with acute MI, the hypointense areas in the LGE images (representing MVO), when present, were included in the contoured areas for the segment wise calculation of TEI. The infarct size was calculated from the LGE images by semi-automated software based analysis (FWHM) and expressed in % of the total LV volume. Another observer drew ROIs (GP) in the infarct core and remote myocardium in the T1 maps corresponding to the LGE image (unblinded). Remote myocardium was defined as myocardial tissue 180° to the infarct with no evidence of hyperenhancement or myocardial oedema, as previously described [18]. Analysis was also done as per the coronary artery territory. Myocardial segments were assigned to coronary arteries as described in the AHA 16 segment model (excluding the apical cap), with 6 segments for the left anterior descending artery, 5 for the right coronary artery, and 5 for the left circumflex artery [19]. In each patient 'global native T1' was derived by taking the mean T1 of all 16 AHA segments. Images were suboptimal/non diagnostic if there was artefact or signal loss that interfered with the ability of the observers to interpret the image. This was observed in three patients but to overcome the problem the sequences were repeated. In the final analysis no images were considered non-diagnostic.

Statistical analysis

Statistical analysis was performed using SPSS V.23 (Armonk, New York, USA: IBM Corp.) and R version 3.1.2 (R Core Team 2014). Categorical variables were analyzed using Fisher exact tests. Normally distributed continuous variables were expressed as mean \pm standard deviation and

Fig. 1 CMR images from a 54 year old with acute transmural myocardial infarction in right coronary artery, **a** T1 map of the short axis, **b** region of interest drawn in each of the segment with corresponding native T1 value (blinded to LGE image), **c** LGE image in the short axis, **d** T1 map of the corresponding short axis with region of interest in the remote myocardium and infarct core (unblinded to LGE image)



compared using unpaired Student *t* tests or one-way analysis of variance with Bonferroni post hoc correction for between-groups comparisons, as appropriate. Continuous variables that were not normally distributed were compared by Kruskal–Wallis tests. *R*-values quoted are for Pearson's correlation coefficient. The potential of quantitative T1-mapping to assess the TEI or viability on a segmental basis against the current gold standards of LGE were explored. The analysis was also performed as per each coronary artery territory. Interobserver (AGD and IH) and intraobserver (AGD) variability for segmental native T1 was assessed in 20 patients (10 acute MI and 10 chronic MI patients), and expressed as intraclass correlation coefficient (ICC) and 95% confidence interval.

Segmental analysis run a risk of class effect due to the potential of multiple samples from the same patient. To nullify the class-effect a series of generalized linear mixed effect models, with the random intercept for each person and for each segment, were performed to assess the association between T1 mapping and viability/LGE TEI. The models were used to account for the fact that different segments from the same patient or same segment location from different patients may not behave independently. These models were conducted for both acute and chronic MI assessments. Receiver operating characteristic (ROC) analysis was performed to identify area under curve (diagnostic accuracy) of native T1 as a marker of TEI by using corresponding LGE scoring (LGE score > 3) as the gold standard. Cut-off values of T1 relaxation times as a marker of viability were also calculated from the ROC curve. Statistical significance of the differences between ROC curves was assessed using the online calculator (<http://www.vassarstats.net>). To take

into consideration potential within-subject interaction of segments, clustered ROC analysis was also conducted to assess the performance of native T1 to predict viability. Significance was defined as two-tailed $p < 0.05$.

Results

The demographic and the CMR characteristics of the healthy control and the patient cohort (60 MI patients—acute and chronic MI subgroups) are presented in Table 1. The mean age of the healthy volunteers was 57 ± 13 years and 75% were male. The mean age was significantly lower in the acute MI group (61 ± 10 years versus 67 ± 10 years, $p = 0.017$) compared to the chronic MI group. In 5% ($n = 3$) of patients the T1 maps had artefacts or signal. To overcome this problem, the mapping images were repeated. Out of the 960 segments analysed from patients with previous MI (acute and chronic), 286 segments had evidence of LGE. Sixteen of the 30 acute MI patients had microvascular obstruction (MVO) (53%).

Interobserver and intraobserver variability for segmental T1 measurement

There was excellent inter and intra-observer agreement for native segmental T1 measurement. The intraclass correlation coefficient (ICC) and 95% confidence interval were 0.968 (0.958–0.976) for interobserver and 0.992 (0.967–0.998) for intraobserver agreement respectively.

Table 1 Demographic/CMR characteristics table

Characteristics	Control (n=20)	Acute MI (n=30)	Chronic MI (n=30)	p-value (acute versus chronic)
Age (years)	57(±13)	61(±10)	67(±10)	0.017
Female sex (%)	25%	20%	20%	1.0
Smoking (%)	–	27	17	0.356
Hypertension (%)	–	43	53	0.447
Diabetes (%)	–	10	23	0.171
CMR				
LV EF (%)	66(±7)	51(±9)	51(±13)	0.833
EDV (ml)	146(±35)	152(±29)	167(±56)	0.218
ESV (ml)	51(±18)	77(±26)	85(±48)	0.406
SV (ml)	95(±22)	75(±13)	83(±23)	0.144
Infarct size (%)	–	21(±10)	16(±12)	0.135

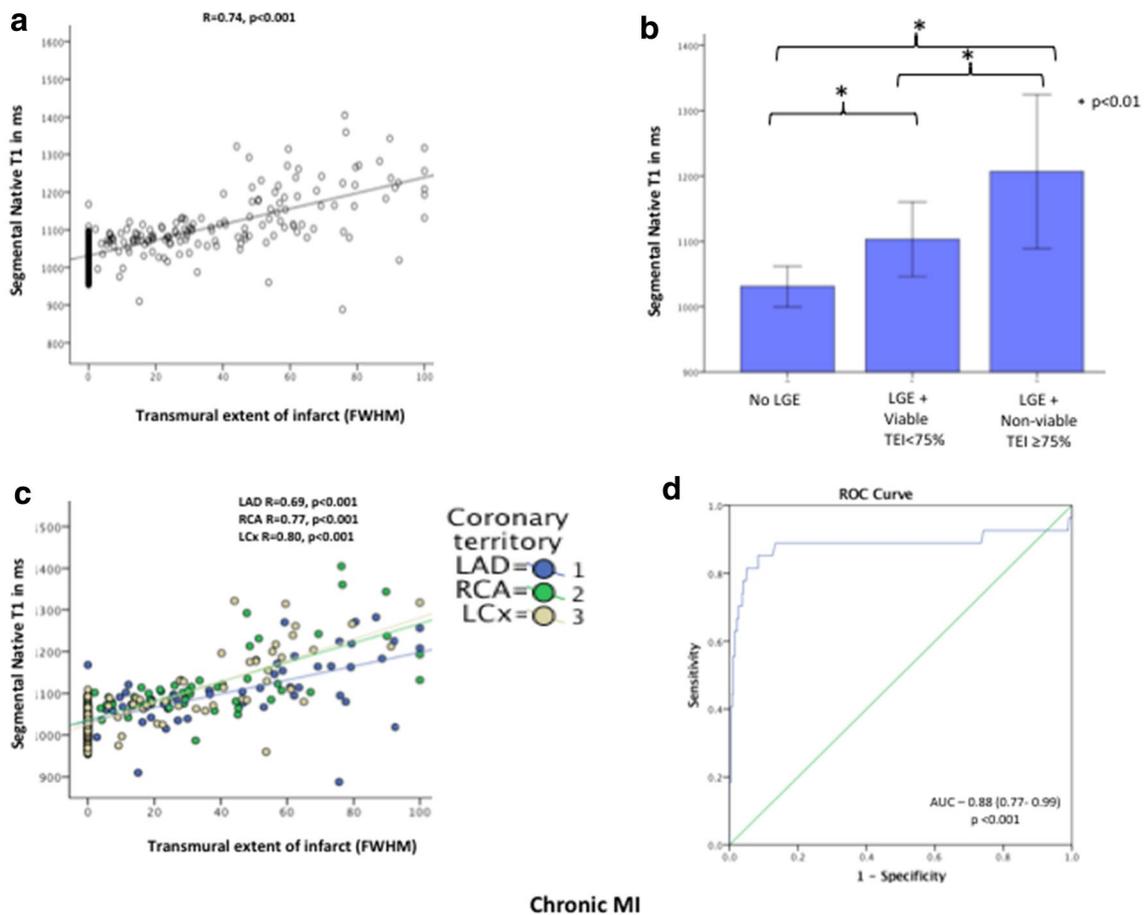


Fig. 2 a Scatter plot: chronic MI cohort showing native T1 value versus the TEI, **b** bar diagram with error bar(SD): Chronic MI cohort showing segmental native T1 value in LGE negative, LGE positive with viability and LGE segments with non-viability **c** scatter plot:

Correlation between segmental T1 and Transmural extent of Infarct—as per coronary artery territory in chronic MI **d** ROC curve: Chronic MI cohort comparing segmental native T1 versus LGE viability

Chronic MI

T1 mapping versus scar transmural (TEI)

In patients with chronic MI, the segmental T1 correlated significantly with LGE TEI ($R=0.74$, $p<0.001$) Fig. 2a. The mean segmental T1 value for LGE negative segments (myocardial segments with no infarction) was 1031 ± 31 ms, LGE positive but viable (scar grade 1–3): 1103 ± 57 ms and LGE positive but non viable (scar grade 4): 1206 ± 118 ms. The mean segmental T1 in each of the 3 categories (no LGE, LGE positive but viable and LGE positive but non-viable) were significantly different on a pairwise comparison with Bonferroni correction ($p<0.01$) Fig. 2b. The mean native T1 in the infarct core and remote myocardium were 1171 ± 76 ms and 1005 ± 19 ms respectively Table 2.

When grouping the segments into coronary artery territories, the correlation between native T1 and LGE TEI for RCA, LCx and LAD territories were $R=0.77$, 0.80 , 0.69 respectively ($p<0.001$) Fig. 2c.

T1 mapping as a marker of transmural of infarction

Native T1 mapping showed excellent diagnostic accuracy in predicting transmural LGE $\geq 75\%$ - AUC-0.88(CI 0.77–0.99, $p<0.001$) Fig. 2d. A cut-off of native T1 of 1085 ms differentiated viable from non-viable segments with 88% sensitivity and 88% specificity. Clustered ROC curve analysis showed that segmental T1 mapping had an AUC of 0.8338 (95% CI 0.7002–1.00) to predict viability.

Mixed effects model with random intercept

Generalized linear mixed effect models, with the random intercept to assess the association between native T1 and viability/TEI correcting for the class effect due to multiple segments from same person or same segment location. Presence of non viability was significantly associated with native T1 (Chi square 307.02, $p<0.01$), increasing it by 131.9 ± 6.3 ms (standard errors). TEI significantly affected T1 (Chi square 388.9, $p<0.01$), increasing it by 2.07 ± 0.1 ms (standard errors) for every percent increase in TEI.

Acute MI

T1 mapping versus scar transmural (TEI)

In patients with acute MI, the segmental T1 correlated significantly with LGE TEI with $R=0.57$, $p<0.001$ Fig. 3a. The mean segmental T1 value for scar transmural grade 0 (no LGE) was 1054 ± 65 ms, LGE positive but viable (scar grade 1–3): 1135 ± 73 ms and LGE positive but non viable (scar grade 4): 1168 ± 71 ms. The mean segmental T1 in the three categories (no LGE, LGE positive but viable and LGE positive but non-viable) were significantly different on a pairwise comparison with Bonferroni correction ($p<0.01$) Fig. 3b. The mean native T1 in the infarct core and remote myocardium were 1308 ± 71 ms and 1054 ± 65 ms respectively Table 2.

When grouping the segments into coronary artery territories, the correlation between native T1 and LGE TEI for RCA, LCx and LAD territories were $R=0.66$, 0.57 , 0.57 respectively ($p<0.001$) Fig. 3c.

T1 mapping as a marker of transmural of infarction

ROC analysis of segmental native T1 in acute MI against gold standard LGE viability ($>75\%$ TEI) showed good diagnostic accuracy AUC-0.83(CI 0.78–0.88, $p<0.001$) Fig. 3d. A T1 threshold of 1110 ms most optimally differentiated viable from non-viable segments with 79% sensitivity and 79% specificity. Clustered ROC curve analysis showed that segmental T1 mapping had an AUC of 0.8338 (95% CI 0.7587–0.9088) to predict viability.

Mixed effects model with random intercept

Presence of non viability was significantly associated with native T1 (Chi square 145.4, $p<0.01$), increasing it by 100.1 ± 7.7 ms (standard errors). TEI significantly affected native T1 (Chi square 206.1, $p<0.01$), increasing it by 1.5 ± 0.1 ms (standard errors) for every percent increase in TEI.

Table 2 Mean native T1(in ms) in the LGE negative segments, LGE positive but viable segments, LGE positive non-viable segments, Infarct core and Remote myocardium

Category	LGE negative segments	LGE positive viable segments	LGE positive non-viable segments	Infarct core	Remote myocardium
Chronic MI Mean T1 (ms) \pm SD	1031 ± 31	1103 ± 57	1206 ± 118	1171 ± 76	1005 ± 19
Acute MI Mean T1(ms) \pm SD	1054 ± 65	1135 ± 73	1168 ± 71	1308 ± 71	1054 ± 65

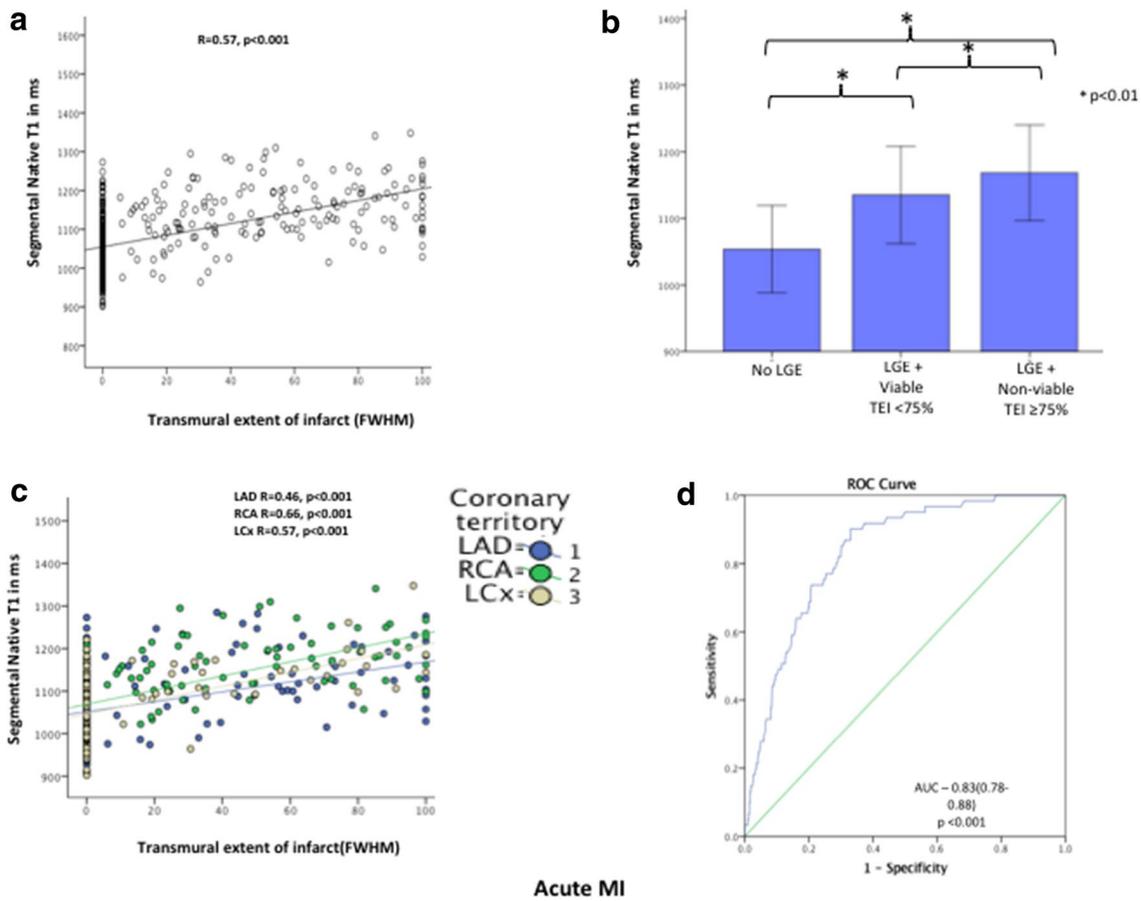


Fig. 3 **a** Scatter plot: Acute MI cohort showing native T1 value versus the LGE TEI, **b** bar diagram with error bar(SD): Acute MI cohort showing segmental native T1 value in LGE negative, LGE positive with viability and LGE segments with non-viability **c** scatter plot:

Correlation between segmental T1 and Transmural extent of Infarct—as per coronary artery territory in acute MI **d** ROC curve: Acute MI cohort comparing segmental native T1 versus LGE viability

T2w STIR area at risk (oedematous area) in acute MI

All the acute MI scans showed evidence of myocardial oedema in the infarct related artery territory. The area at risk (total area of oedema) in the acute MI was $31 \pm 12\%$ of the total LV.

Global native T1

Global native T1 in normal versus affected

Global native T1 in normal healthy volunteers was compared with all patients with previous MI (acute and chronic). The mean global T1 in normal volunteers was 1028 ± 28 ms, which was significantly lower than patients with previous MI 1070 ± 36 ms, $p < 0.0001$. The global native T1 in patients with acute MI was 1082 ± 34 ms ($p < 0.0001$ vs normal volunteer), whereas for chronic MI it was 1058 ± 34 ms ($p = 0.002$ vs normal volunteer) Fig. 4.

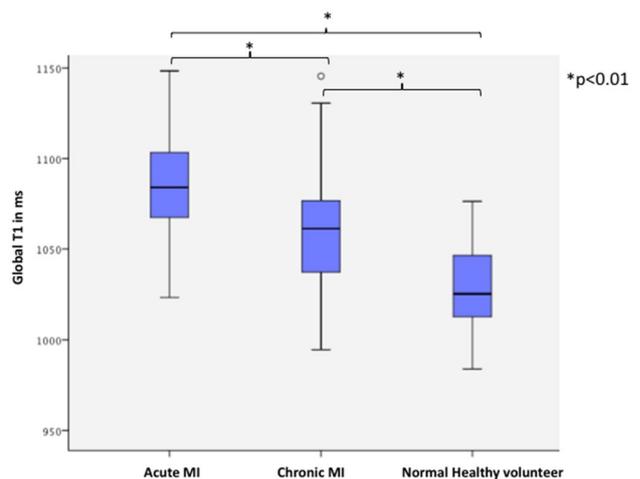


Fig. 4 Boxplot showing the global native T1 in acute MI cohort, chronic MI cohort and the normal healthy volunteer

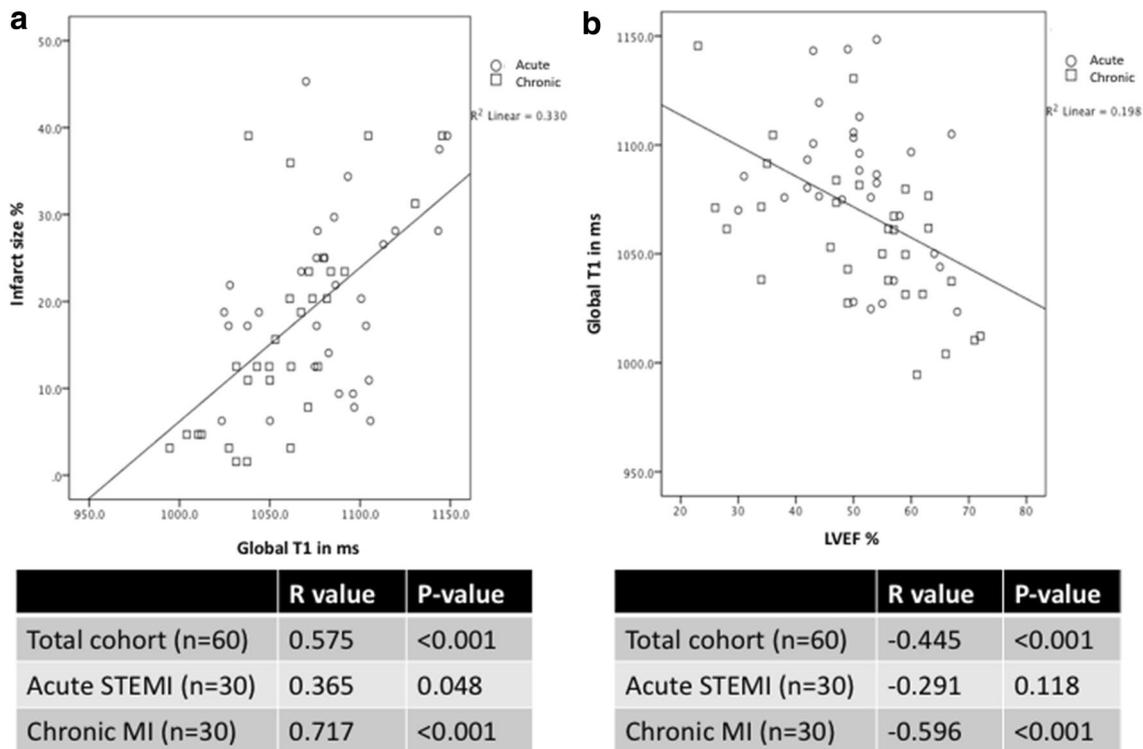


Fig. 5 **a** Scatter plot showing the correlation between global T1 and infarct size, **b** Scatter plot showing the correlation between global T1 and LV ejection fraction

Global native T1 versus Infarct size

Global native T1 was compared with the infarct size. For the total cohort (n = 60) it showed a good positive correlation $r = 0.575$, $p < 0.001$. Subgroup analysis: the correlation was found to be excellent in chronic MI $r = 0.717$, $p < 0.001$ whereas for acute MI it was weakly significant $r = 0.365$, $p = 0.048$ (Fig. 5a).

Global native T1 versus LV EF

Global native T1 was compared with the left ventricular ejection fraction. For the total cohort (n = 60) it showed a significant inverse correlation $r = -0.445$, $p < 0.001$. Subgroup analysis: the correlation was found to be statistically significant in chronic MI $r = -0.596$, $p < 0.001$ but not in acute MI $r = -0.291$, $p = 0.118$ Fig. 5b.

T1 mapping versus wall motion score versus wall thickness for the assessment of viability

Segmental native T1 was compared with segmental wall thickness and segmental wall motion score against gold standard LGE viability. ROC analysis of the 960 segments for viability assessment showed the highest diagnostic

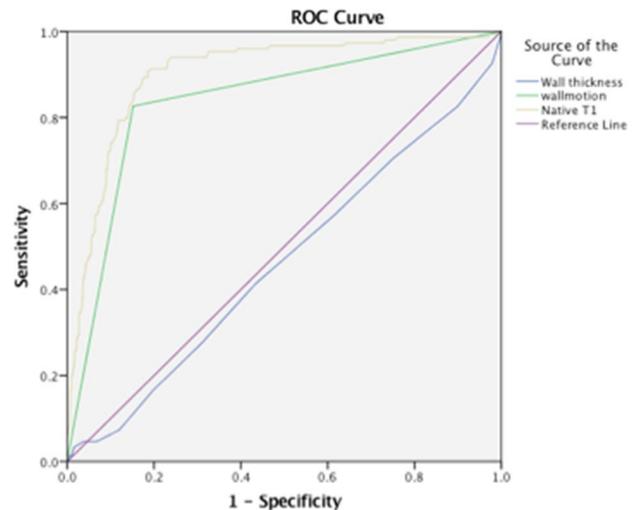


Fig. 6 ROC curve: comparing segmental native T1, segmental wall motion score, and segmental wall thickness versus LGE viability

accuracy of T1 mapping (AUC = 0.9, 95%CI 0.87–0.93, $p < 0.0001$) when compared to wall motion score (AUC 0.837, $p < 0.0001$, 95%CI 0.8–0.875), and segmental wall thickness (AUC 0.453, $p = 0.07$, 95%CI 0.401–0.505) Fig. 6.

Discussion

The study looked at the diagnostic performance of segmental T1 mapping as a marker of LGE TEI and myocardial viability in patients with previous MI (acute and chronic). Overall native segmental T1 mapping had an excellent diagnostic accuracy, in distinguishing viable from non-viable myocardium when using LGE $\geq 75\%$ transmural as a reference standard to define infarcted non-viable myocardium. Diagnostic accuracy was better when used in chronic MI (AUC – 0.88) rather than acute MI (AUC – 0.83). The clinical value of T1 mapping lies in the evaluation of myocardial scar extent, on a voxel-wise basis, without the need to use a contrast agent.

Myocardial infarction is a regional disease affecting parts of the myocardium supplied by the occluded/stenosed coronary artery. Segmental analysis as per the AHA nomenclature is the most widely used cardiac imaging technique in current clinical practice. The study was designed to look at the T1 profile in different LGE TEI. In a segment wise analysis for T1 there was a statistically significant positive correlation between LGE TEI and the native T1 values. The association of native T1 and viability/TEI remained significant even adjusting for segments from same patient bias as well as same segment locations from different patient bias using the mixed effect model with random intercepts.

The opportunity to correlate presence and potential extent of infarction/scarring without the use of a contrast agent is clinically attractive. The T1-mapping as a single criterion demonstrated around 88% sensitivity, specificity in differentiating viable from nonviable myocardium in chronic MI. Our results were similar to the study by Ferreira et al looking at the performance of T1-mapping in delineating the extent and patterns of acute myocarditis [20]. It may be possible in future to perform a contrast-free CMR protocol using cine and T1-mapping for the assessment of TEI.

Viability assessment is a common indication for CMR not only in chronic IHD but also in acute MI. The latest ESC guidelines on management of STEMI recommend viability assessment in an acute setting in selected cases such as in the setting of multivessel disease [21]. In addition to a chronic MI group our study included an acute MI group thereby giving an opportunity to delineate the native T1 findings in acute MI patients.

The study delineated a higher accuracy of native T1 in chronic MI compared to acute for the assessment of viability. The reason behind the lower performance of native T1 in acute MI is most likely due to its pathophysiologic difference with chronic MI. Acute MI is characterised by oedema, loss of cell membrane integrity, an inflammatory

response, necrosis, micro-vascular obstruction (MVO) and haemorrhage whereas chronic MI by scar tissue/fibrosis in the extracellular space. Our study showed that all the acute MI patients had evidence of oedema with a mean area at risk (oedematous area) of 31% of LV which was significantly higher than the mean infarct size (21% of LV). The myocardial oedema delineated may have impacted on the diagnostic performance of T1 mapping in acute MI. T1 mapping is influenced by all of these characteristics however LGE is not (especially oedema). In acute phase even LGE based TEI have been shown to be inaccurate [22] and hence may pose a limitation as a marker of viability/functional recovery. Beek et al demonstrated that 25% of segments with 75–100% TEI in acute MI had functional improvement at 13 weeks [23].

The assessment of TEI in patients post myocardial infarction is aimed both at guiding further revascularisation or to predict arrhythmic risk by determining the peri-infarct zone (gray zone) [24].

Our study was performed on 1.5 T scanner but the results were comparable to the study by Kali et al [8] in which they reported the diagnostic accuracy of T1-mapping in the detection of transmural in chronic MI. Our study included both acute and chronic MI, thereby helping to compare the native T1 mapping findings in the two groups. Our main aim was to assess the significance of segmental native T1 as a discriminator between viable and non-viable myocardium unlike the study by Kali et al where they compared the overall transmural between LGE and T1 mapping using thresholding technique. The native T1 value trends in the different infarct characteristics in acute and chronic MI in our study were comparable to the case series published by Dall'Armellina et al, although the actual values were different as her study was performed on 3 T and ours at 1.5 T [25]. A further study on native T1 in acute MI by Dall'Armellina et al. showed a strongly positive correlation between T1 value and the LGE TEI, but they excluded segments with MVO in the analysis [26]. The aim of our study was to assess viability without the use of contrast. It is very difficult to accurately identify MVO based on the T1 mapping alone without LGE imaging. In the study by Bulluck et al the diagnostic accuracy of T1 maps to identify MVO was 79–81% [27]. Hence in our study design the segmental native T1 analysis was done blinded to LGE data without excluding MVO.

Overall global native T1 showed a significant positive correlation with infarct size and LV ejection fraction. However, when the analysis was done for the acute and chronic groups the correlation was stronger in chronic compared to acute MI. The global native T1 did not correlate with LVEF in acute MI, again this is likely due to the effect of MVO or intramyocardial haemorrhage which reduces the T1 relaxation time.

Our study was performed on a 1.5 T scanner, which is the most widely used machine. We have also shown a significant correlation at an individual coronary territory level. The T1 analysis was done from the T1 maps which were available immediately on the scanner console with no need for further post-processing. Apical slices were not excluded from analysis unlike other similar studies on T1 mapping due to the potential for partial volume artefact [28]. Similar studies have often eliminated lateral wall infarcts due to off-resonance artefact, but this is less relevant at 1.5 T compared to 3 T [28]. Moreover, our study sample had infarcts in all the different coronary territories including the lateral wall (which we included) to reduce the selection bias and reflect real world practice, whilst most of previous studies mainly included LAD infarctions.

Study limitations

This is a single centre, single vendor study with a limited sample size. However, our findings were highly statistically significant. Larger multicenter, multivendor studies are warranted to confirm this proof of concept study. TEI on LGE imaging was used as the reference standard for viability rather than functional recovery which was used in other studies. TEI < 75% was used as the marker of viability in keeping with contemporary clinical practice. The acute and chronic MI cohorts were completely separate in our study unlike other studies. However, the study design was specifically constructed to remove any repeat measure from the same patient bias. Peri-infarct grey-zone on LGE, which has been shown to be associated with arrhythmic events, was not assessed. This may have explained the difference between acute and chronic MI. Lastly ischemic versus non ischemic cannot be differentiated based on the elevated segmental native T1 values. Nevertheless non ischemic cause was excluded in all our patients based on the LGE images.

Conclusions

Native T1 mapping correlates significantly with TEI thereby differentiating between normal, infarcted viable, and infarcted non-viable myocardium with distinctive T1 profiles in both chronic and acute MI. Native T1 mapping performed better in chronic MI compared to acute due to the absence of myocardial oedema and microvascular obstruction. T1 mapping holds promise for viability assessment without the need for gadolinium contrast agents.

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Authors' contributions AGD and CBD conceived the study design. EM & CBD coordinated and performed the MOIST trial. AGD, GP, VDB, IH, EDG, CM and IE participated in the analysis of CMR. AGD, IH, GP and CBD helped to interpret the data and draft the manuscript. All authors read and approved the final manuscript.

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Compliance with ethical standards

Conflict of interest Dr Chiara Bucciarelli-Ducci is a consultant for CircleCVI. The other authors of the manuscript declare no conflict of interest.

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