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Original Article

Native T1-mapping detects acute myocardial infarction without application of gadolinium contrast agents

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Abstract: Objective: This study aimed to demonstrate T1-mapping how to display the patterns of myocardial injury and to quantify myocardial involvement in acute myocardial infarction (AMI) without application of contrast agents. Methods: Total 50 patients with suspected AMI (median 3 days from presentation) enrolled as myocardial infarction group and 20 as normal controls group. Then, analysis of these patients were performed, including: lesion myocardial T2 signal intensity (SI) ratio compared to remote normal myocardium, non-contrast myocardial T1 times, left ventricular function including ejection fraction, myocardial mass and left ventricular volume, and areas of injury by T2W, T1-mapping and LGE. Results: Compared with normal controls, AMI patients showed more edema (myocardial T2 SI ratio 1.85 ± 0.30 vs 1.55 ± 0.14, $P < 0.05$), higher mean non-contrast myocardial T1 (1220 ± 50 ms vs 981 ± 23 ms) and more areas of injury as detected by T2W (median 15%), T1 values (median 28%), and LGE (median 12%). However, no significant difference was identified in LV mass and ventricular volume ($P > 0.05$). With T1 > 1300 ms (sensitivity 90%, specificity 75%), detected significantly larger areas of involvement were significantly detected compared with T2W and LGE imaging. T1-mapping significantly improved the diagnostic confidence which cannot detected using T2W or LGE). Using incremental thresholds, T1-mapping can display the patterns of injury typical of acute myocardial infarction. Conclusion: Native T1-mapping as a novel technique for detecting the patterns in acute myocardial infarction without application of contrast medium. Compared to T2W and LGE imaging, T1-mappng detects additional areas of myocardial tissues, and offers significant diagnostic value in AMI detection.

Keywords: Native T1-mapping, acute myocardial infarction, cardiovascular magnetic resonance, late gadolinium enhancement, T2-weighted imaging

Introduction

Acute myocardial infarction (AMI) is one of the most common cardiovascular disease with high morbidity and mortality worldwide [1]. Coronary artery occlusion is the main inducement of AMI and myocardial tissue edema at the ischemic area is the first signs responded to ischemia injury [2]. Despite the improvement of AMI treatment, the occurrence of AMI in China is significantly increased, but the clinic outcomes are not satisfied owing to the limitation of diagnostic and understanding of AMI [3]. Cardiovascular magnetic resonance (CMR) is the common imaging tool using in AMI detecting owing to its multiple-parameter tissue characterization. For examples, Dark-blood T2-weighted (T2W) used for edema imaging, T1-weight used for pre- and post-contrast hyperemia imaging, myocardium first perfusion used for ischemia and late gadolinium enhancement (LGE) used for myocardial necrosis and viability assessment, and all of these are conventional CMR methods applied to assess AMI with different pathogenesis [4]. Specially, myocardium first-pass perfusion combine with LGE is especially powerful in distinguishing necrosis tissues from the normal myocardium, and allowing visualization of LGE patterns and identification difference between ischemic and non-ischemic pathological changes during the pathogenesis of myocardial disease.

However, there are still some limitations of traditional diagnostic methods in detecting AMI. Especially, the very small areas of myocardial infarction or subendocardial myocardial infarction are frequently missing visualized. T2W-

CMR techniques, which are considered to have high sensitivity of AMI detection, also presented increase diagnostic sensitivity in the detection of AMI with or without water content. However, the further generalization of this technique in clinics is obviously restricted due to adjacent to the sub-endocardium ventricle slow-flow blood bright signal, image quality impairment in tachyarrhythmias, long breath-holds, and need for a "normal" reference region of interest (ROI) [4, 5]. In the early hours post ischemia, dynamic change of LGE is frequently occurred in the ischemic myocardium together with a significant regression of LGE over time and full function recovery, which resulting in negative effect on LGE imaging [6-9]. Additionally, despite the low incidence rate, gadolinium-based contrast agents can induce serious complication of nephrogenic systemic fibrosis in patients suffered with grievous renal function impairment. Therefore, it is of important to develop new method to visualize myocardium edema of AMI without exogenous inject contrast agents.

Native T1-mapping provides a quantitative outcome in the detection of myocardial edema by directly measuring the T1 relaxation time of tissues on a pixel-by-pixel basis with high resolution. Because different tissue types present a different normal characteristic of T1 relaxation times in particular magnetic field strength, which has been validated in large and multicenter normal cohort study. T1-mapping has an advantage over T2-weighted imaging in detecting myocardium edema without reference ROI, which ensures a quantitative techniques T1-mapping and reducing subjectivity assessment and error during diagnosis. Histological assessment study has demonstrated that the area of increased water content promotes the area of infarction [10]. Other study suggests that the increased T1 values associate with prolonged ischemia due to larger concentrations of other water construction [11]. Recently study shows that T1-mapping is suitable for the diagnosis of patients suffered with AMI not only because the significantly shorter the breath-hold time and scan time, but also the more accurate estimation of long T1 values at higher rates. Furthermore, native T1-mapping also provides a prior sensitivity in T2W and LGE imaging without further post-processing. To provide a single numeric average myocardial T1 value, T1-maps should be fully applied for visual and spatial characterization of variation in the left ventricular myocardium [12].

This study aimed to explore the diagnostic ability of T1 mapping in detecting AMI compared with traditional CMR techniques (LGE, T2W), and reveal whether T1-mapping was suitable for the detection of ischemic patterns after injury.

Material and methods

Characteristics of the study population

This study was designed as a prospective study. From January 2013 to December 2014, 50 patients (age 55 ± 13 years; 36% female), which presenting with suspected AMI as inpatients in our hospital and received CMR scanning within 14 days (median 3 days) of presentation, were enrolled in this study. Patients were included if they were suffering acute chest pain and cardiac troponin I level > 0.04 μg/L (median 4.7 μg/L). Patients were excluded if they met any of the following terms: (a) not allowed to CMR (e.g., pacemaker), (b) unstable hemodynamic force (systolic blood pressure < 90 mm Hg and using inotropic drugs or intra-aortic balloon pump), (c) obvious arrhythmias, (d) markedly renal dysfunction (estimated glomerular filtration rate < 30 ml/min), and (e) previous myocarditis, myocardial infarction, or any other chronic cardiac diseases. Meanwhile, 20 age and gender distribution matched healthy volunteers (age 56 ± 8 years; 40% female) were enrolled as controls for this study. The inclusive criteria for healthy controls were: (a) without cardiac history, (b) without obvious cardiac risk factors, (c) not taking cardiovascular medications; and (d) with a normal electrocardiogram assessed using CMR. This study was authorized by local ethical committee and signed informed consents were obtained from all patients enrolled in this study.

Cardiovascular magnetic resonance

CMR were conducted on a single 1.5 Tesla clinical MR scanner (Avanto, Siemens Healthcare, Germany) using a 16-channel phased-array coil. Heart localization was carried out using a true fast imaging with steady-state precession (FISP) under electrocardiographic gating. Cine images were obtained in the short-axis plane covering the base to apex of the heart (10 to 12 slices) with the sequence of truth balanced

Table 1. Baseline characteristics of the study population

Characteristics	Controls (n = 20)	Patient (n = 50)	*P* value
Age (yrs)	56 ± 8	55 ± 13	0.74
Female, n (%)	8 (40)	18 (36)	0.65
Hypertension, n (%)	n/a	10 (20)	-
Dyslipidemia, n (%)	n/a	18 (36)	-
Current smoker, n (%)	4 (20)	10 (20)	0.58
Diabetes, n (%)	n/a	16 (32)	-
Family history, n (%)	n/a	7 (14)	-
Troponin I (μg/L)	n/a	9.6 (2.8 to 38)	-

Value are mean ± SD, n (%), or median (interquartile range).

stead-state free-precession (TRUFI, SEMIENS), and the typical parameters were: repetition time (TR) = 55 ms, echo time (TE) = 1.1 ms, flip angle = 67°, slice thickness = 8 mm, slice gap = 2 mm, acquisition matrix = 192 × 109, and field of view 320 × 400 mm. Left ventricle tissue characterization from base to apex was conducted using T2W, T1-mapping and LGE imaging with matched short-axis slices. STIR sequence was used for dark-blood T2-weighted imaging of edema, and parameters were set as follows: TR = 430 ms, TE = 52 ms, TI = 170 ms, flip angle = 180°, Slice thickness = 8 mm, acquisition matrix = 256 × 156 mm, and view field = 320 × 400 mm. Before the application of contrast agent, T1-mapping using the Modified Look-Locker Inversion recovery (MOLLI) sequence [13], and the parameters were utilized as follows: slice thickness = 8 mm, TR = 740 ms, TE = 1.06 ms, flip angle = 35°, parallel imaging factor 2, acquisition matrix = 192 × 124, and view field = 320 × 400 mm. Acquisition in late diastole on every other heartbeat; minimal inversion time = 190 ms; increment = 80 ms. T1 mapping scheme included 3 acquisitions after the first inversion pulse, followed by a 3-heartbeat pause, a second 3-heartbeat pause, and a third inversion for the last 5 acquisitions. LGE imaging was acquired in the long and short-axis planes using a T1-weighted phase-sensitive inversion recovery prepared fast gradient echo (PSIR) sequence 10 minutes after intravenous administration of contrast agent (Gadodiamide, Omniscan, GE Healthcare, total 0.10 mmol/kg). The LGE imaging parameters were: TR = 600 ms, TE = 3.4 ms, flip angle = 25°, slice thickness = 8 mm, acquisition matrix = 256 × 156 mm, and field of view = 320 × 400 mm. The T1 mapping, T2W, and LGE images were obtained in the same axis and slice thickness as for cine images. A contrast media bolus was intravenously administered at 1 ml/s, followed by 20 ml of normal saline at the same rate for flush using a power injector.

Image analysis

To obtain a full understand of tissues, results of matching short-axis slices were analyzed along with cine, dark-blood T2W, T1-mapping, and LGE imaging. Quantifications of left ventricular ejection fraction and LV volumes were analyzed using Argus software (Siemens Medical Solutions) on cine images. Analysis of all matching short axis images from T1-mapping, T2W and LGE imaging was carried out using in-house software MC-ROI. Briefly, based on the images provided by dark-blood T2W, edema was confirmed when myocardial T2 SI ratio was > 2:1 compared to that of remote unaffected myocardium in the same slice. Normal myocardium represents a region of myocardium with no obvious visual T2 SI elevation and no LGE. Meanwhile, abnormal low signal should also be attended during assessment. All T1 images were quantitative analyzed to obtain the final T1-mapping result. To minimize the partial volume effect on myocardial T1 values, contaminations caused by blood-pool and extra-myocardial structures should be restricted during the placement of the endo- and epicardium contours. Focal areas of LGE were defined as those with 2 standard deviations (SD) based on the mean intensity of reference ROI located in remote unaffected myocardium. For all of those analyses, the abnormal myocardial regions were definition as contiguous area of > 40 mm^2 [14]. To calculate the extent of myocardial injury provided by the tissue characterization techniques, percentage of each abnormal myocardium segment was determined, and t mean value of each subject was computed. Additionally, to acquire accuracy quantitative image analytical results, qualitative visual analysis for T2W and LGE images were conducted by at least 2 expert CMR cardiologists. Disagreement was solved by introducing another additional expert CMR cardiologist.

CMR image quality evaluation

Images quality of each LV myocardial segment was rigorously estimated. According to pervious criteria, segments with no or minimal arti-

Table 2. Tissue characteristics in controls and patient group

CMR finding	Control (n = 20)	All patients (n = 50)	Group I (n = 37)	Group II (n = 5)	Group III (n = 8)
Ejection fraction (%)	71 ± 4	64 ± 13*	61 ± 11†	69 ± 8	71 ± 5
LV Mass, g/m^2	52 ± 11	51 ± 8	51 ± 10	51 ± 14	52 ± 4
LVEDV, ml/m^2	77 ± 16	75 ± 19	73 ± 10	75 ± 19	76 ± 11
LVESV, ml/m^2	19 ± 5	21 ± 5	22 ± 19*	20 ± 8	19 ± 11
T2 SI ratio	1.55 ± 0.14	1.85 ± 0.30*	1.79 ± 0.24†	1.59 ± 0.19	1.56 ± 0.11
Mean myocardial T1 (ms)	981 ± 23	1220 ± 50*	1334 ± 62†	1207 ± 76†	994 ± 55
T1 ≥ 1200 ms (%)	0 (0 to 1)	30 (15 to 60)*	47 (25 to 69)†	15 (12 to 29)†	5 (2 to 9)
Myocardium LGE (%)	0 (0 to 1)	11 (4 to 20)*	19 (8 to 23)†	3 (1 to 5)†	1 (0 to 2)

Group I: Edema+ and LGE+; Group II: Edema+ and LGE-; Group III: Edema- and L GE-. Values are presented as mean ± SD or median (interquartile). *P < 0.05 when compare to controls. In subgroup analyses: †Significantly different from controls; areas of injury by edema were significantly larger in Group I than Groups II and III; areas of injury by both T1 and LGE were significantly larger in Group I > II > III patients.

Table 3. Diagnostic performance of CMR tissue characterization methods in the detection of acute myocardial infarction

Tissue criteria	Sensitivity (%)	Specificity (%)	Accuracy (%)	PPV (%)	NPV (%)
T1-mapping*	90	75	84	93	88
Dark-blood T2W*	70	87	71	80	60
LGE	72	96	86	97	67
T1-mapping and T2	80	96	85	95	51
T1-mapping and LGE*	69	97	89	98	64
T1-mapping T2 and LGE*	50	97	70	98	58

*Statistically different (P < 0.05); T1-mapping: myocardial injury is detected when T1 is ≥ 1200 ms; Dark-blood T2-weighted imaging: edema is diagnosed when T2 SI ratio is ≥ 2; LGE = Late gadolinium enhancement; PPV = positive predictive value; NPV = negative predictive value.

facts were considered to meet the requirements. As a result, for cline imagine, 13% of segments were rejected caused by left ventricular outflow tract or motion artifacts. For dark-blood T2W imaging, 25% were rejected due to breathing motion artifacts, signal dropout or off-resonance artifacts. On T1-maps imaging, 19% of the segments were excluded due to off-resonance artifacts, partial volume effect, patient movement artifacts, respiratory motion artifact, and image with poor SNR. Moreover, for LGE imaging, 7% were rejected owing to artificial errors, such as patient movement, or poor image quality. To minimize negative effects on any single technique, analyses were repeated with all artifacts by two experts of CMR cardiologists.

Statistical analysis

Statistical analysis was performed using SPSS 17.0 (IBM) was utilized for statistical analysis involved in this study. Continuous variables are presented as mean ± standard deviation (SD), and non-parametric data are showed as median with interquartile range (IQR). Comparisons between unpaired samples groups were evaluated using unpaired 2-tailed Student's t-test, and comparisons between non-parametric data were carried out using the Mann-Whitney U test. All statistical tests were performed with two-tailed, and *p*-value < 0.05 was set as the threshold of statistically significant. To determine the differences of multiple CMR methodologies used in groups, comparisons of parametric data were performed using ANOVA analysis with Bonferroni corrected, and e Kruskl-Wallis one-way analysis of variance was performed to compare the difference between non-parametric data. Receiver operator characteristic (ROC) analysis was utilized to compare the diagnostic abilities of the CMR methods in detecting myocardial variations between AMI patients and healthy controls.

Results

Primary characteristics of patients

Primary characteristics of enrolled patients are presented in **Table 1**. Of the 55 patients enrolled, 5 patients did not complete the CMR protocol towing to claustrophobia or incompatibility to the end of examination. Thus, a total of 50 patients (age 51 ± 3 years; 36% female) were scanned with 3 days post symptoms onset.

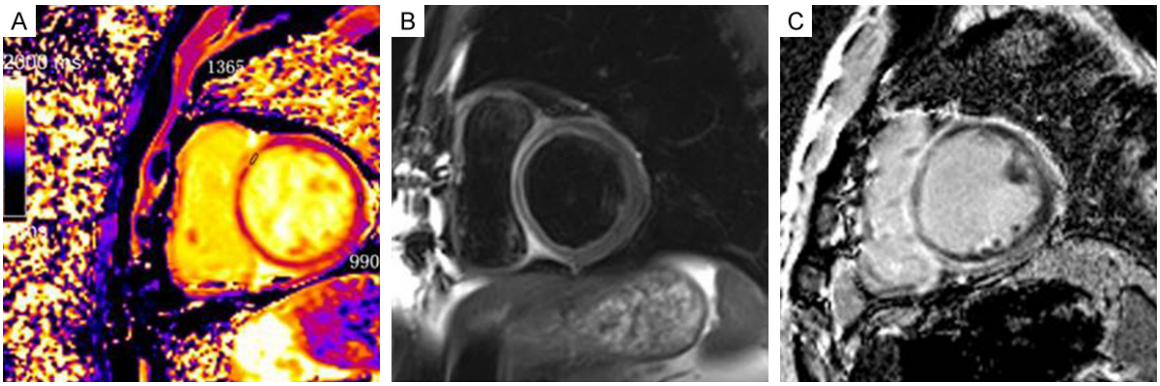

Figure 1. Acute myocardial infarction in a 58-year-old male with troponin I rise up to 30 μg/L. T1 map (A), obtained 5 days after the acute chest pain onset, shows strip schistose of high T1 values in the anterior septum wall (1365 ms, vs 990 ms the remote myocardium in lateral wall). T2-STIR image (B) shows a similar high signal area in the septum wall, however, with obscure boundary and smaller area. Corresponding LGE image (C) depicts the more clearly patchy hype-enhanced almost in the same location as T1 map.

CMR findings

Identifications of CMR methods are summarized in **Table 2**. Compared to healthy controls, significantly lower mean LV ejection fraction (EF), higher mean myocardial T1 values, and larger areas of myocardial injury were measured using T2W and LGE in AMI patients.

T1 values in AMI area

Incremental values of T1-mapping were identified in the detection of AMI areas in **Table 3**. Increased T1 values were identified in the area of myocardium along with the areas of LGE and increased signal on T2W. However, the area of increase T1 values is larger than LGE and T2W image [15]. In order to reveal the diagnostic accuracy of T1 and T2W, the variability of T1 values in remote non-affected myocardial segments were assessed and compared it to the signal intensities in T2W imaging of corresponding segments [4, 16]. The average T1 value in non-affected myocardium were closer to the normal volunteers (990 ± 27 ms, 981 ± 23 ms, P = 0.16).

Receiver operator characteristics (ROC) analysis comparing Diagnostic performance of the techniques were assessed using ROC analysis and the results showed that the diagnostic values of T1-mapping and LGE (with area under the curve (AUC) = 0.92 and 0.94, respectively; P = ns) were significantly higher than that of dark-blood T2W imaging in detecting AMI (AUC 0.72, all P < 0.05 h). These results suggest that T1-mapping and LGE images have the same diagnostic performance in detect injury of AMI, and better than dark-blood T2W imaging in the estimation of myocardial edema in both ischemic and non-ischemic heart disease. Recently multicenter studies support these similar results as mentioned [4, 7, 15, 17].

In order to assess whether T1-mapping offers any incremental value compare to conventional CMR techniques (T2W, LGE) in detecting AMI. Patients were further categorized in subgroups according to edema diagnostic results provided by T2W and LGE imaging. Both edema and LGE patterns were identified in group I patients (edema+, LGE+) along with AMI (**Figure 1**). Meanwhile, compared with normal controls, significantly higher expression level of Troponin I level (P = 0.025) and lower EF t (P = 0.020) were uncovered in patient with AMI. Moreover, Group I patients also presented significantly edema (P = 0.012) and LGE levels (P = 0.034) compared to controls and other subgroups.

Group II patients (edema+, LGE-) did not completely comply criteria of LGE, but showed typical ischemic heart disease patterns. Meanwhile, compared with group I, group II presented less severe myocardial injury, a significantly lower median Troponin I level, but smaller area of edema (P = 0.045). However, no significant difference was identified in mean EF compared with normal controls.

Group III patients (edema-, LGE-) did not presented edema or LGE on CMR, suggesting this group perhaps suffered softly ischemic myocardial injury, however, when taken CMR scans

ischemic myocardium almost recovery from injury. And there's another perspective to explain this new technology detects injury of myocardium is sensitively. These patients had a very lower median Troponin I levels compared with normal controls, but no significant difference was identified in the mean EF.

T1-mapping, this study we using T1 value 1200 ms as threshold, which can detect significantly larger or the identical extent of myocardial injury compared to T2 and LGE imaging in all patient subgroups and normal controls [18]. These findings suggested that T1-mapping are more accurately assess myocardial injury than conventional methods (T2W, LGE). In Group II (edema+, LGE-) patients, T1 mapping was able to detect areas of AMI in group II, representing, while LGE imaging did not provide a diagnosis of injury. Further, in Group III (edema-, LGE-) patients, T1-mapping was able to detect small focal areas of injury, and not due to artifacts or poor T1 fit. Further information of myocardium characterization may be acquired from native T1-maps using incremental thresholds. These indicated that the underlying pattern of myocardial injury, similar to that typically seen on LGE imaging [19]. With the criterion of T1 > 1200 ms, larger area injury was revealed in AMI using incremental thresholds. Meanwhile, the areas with the highest T1 values were close to the areas of enhancing LGE images. This finding was consistent with the findings that patients segments with LGE had significantly higher T1 values were identified in pathogenic segments compared with normal controls (1334 ± 62 ms vs 981 ± 23 ms; $P < 0.001$), indicating that areas without apparent LGE were also involved in this procedure. However, among patients with low troponin levels, variations were not detected using a higher T1 threshold (e.g. T1 = 1200), and the threshold of T1 > 1200 ms allowed immediate visualization of the area of abnormality in a predominantly subepicardial or midwall pattern. Thus, a range of T1 thresholds might allow the simultaneous quantification of myocardial involvement as well as visualization of the topography of injury within the affected myocardium.

Discussion

In our study, T1-mapping has shown that in acute myocardium infarction for the first time as follows: (1) can detect extra small areas of focal injury which will missed by T2W and LGE imaging; (2) have a higher diagnostic ability in detecting larger extent of myocardial involvement than T2W and LGE methods; (3) have a comparable diagnostic ability LGE imaging in detecting injury tissues without application of exogenous contrast agents.

Consistent with precious report, this study suggested that increased myocardium T2 SI and T1 values might contribute to the notion that edema and inflammation are important components in the acute pathophysiologic process. While multiple researches have reported that acute edema is known to increase native T1 relaxation, some additional changes may be found, such as the distribution of free water fractions in the intra-and-extra-cellular compartments, which are more easily detected by T1-mapping than by T2W methods. Compared with T2W and LGE imaging, MOLLI T1-mapping presented more sensitive and specificity with the stated thresholds, which may be attributed to its tight normal range and with low variability. The results of this study suggested that the higher sensitivity of T1-mapping, as well as directly quantification of myocardial variations without utilization of reference ROIs, may be explained that T1-mapping was able to detect larger areas of AMI areas than T2W and LGE imaging [4, 20, 21].

T1-mapping may replace dark-blood T2W as a new method for detecting edema of acute myocardium infarction in earlier stage. Because of T1-mapping was significantly superior in detecting changes of as a single criterion (T1-mapping vs Dark-blood T2; $P < 0.05$), MOLLI T1-mapping can be particularly applied when patients with the problem of tachyarrhythmia, or inability to perform long breath-hold, or muscle inflammation, which conventional dark-blood T2W imaging fails to detect edema or fails to meet the requirement due to the influence of artifacts.

Native T1-mapping shows that the area of necrosis is relate to prolong T1 values, however, T1 values prolong was also be detected in the area of surrounding the pure myocardial necrosis. The reason for T1 value change in the peripheral zones of the infarction is still under discussion, perhaps increased water content of the tissue from infarct-related edema might be responsible for the alteration. It is still unclear

whether or not these changes in magnetic properties reflect transient structural change associated with myocardial stunning. The increased water content in suffered tissue leads to the change of T1 values, as a result, T1-mapping is sensitive to detect edema than convent blood-dark T2W, and detect a larger extent of myocardial involvement than T2W.

Our study confirmed the hypothesis that threshold T1 values can be used to differentiate infarcted myocardium from normal myocardium. With the T1-threshold approach, infarcted myocardium segments could be identified with high sensitivity and specificity. Native T1 > 1200 ms as threshold detected segmental defects caused by acute myocardium infarction with 94% sensitivity and 89% specificity, respectively. In our study, we also found that the native T1 values was higher in infarcted than in remote area, and highest increase T1 values (> 1400) in the acute stage.

In some cases of AMI, while a normal area detected using LGE imaging, T1-maps revealed that the myocardium was involved, but only the LGE-negative area had less elevated T1 values. This results was consistent with the findings that LGE showed a significantly ability in detecting pathogenic segment with higher T1 values and patient segments without LGE (from Groups II and III) still performed significantly higher T1 values compared with normal controls (1108 ± 68 vs 981 ± 31 ms; $P < 0.05$), which suggesting that these areas also likely contributed to AMI, but perhaps played roles in a milder degree without significant necrosis. Further, very small foci of necrosis may be beyond the resolution of LGE, and T1-maps may provide potential advantage. For instance, the PSIR sequence for LGE in this study offers a resolution of 1.5 × 1.5 × 8.0 mm, while T1-map has a voxel resolution of 0.9 × 0.9 × 8.0 mm. Thus, T1-maps with incremental thresholds not only presented the predominant function during AMI, but may also provide additional insights of the understanding of AMI pathogenesis.

There were also some clinical implications provided in this study. Results of this study presented that T1 thresholds can be utilized to reveal the infarcted area in T1-maps of patients with AMI without application of exogenous contrast agents. This might be a new way to extract information similar to LGE imaging without contrast agents. T1-mapping technique access to quantitative and more detail information on myocardial signal and add valuable insights. T1-mapping technique is suitable for measurements of infarct size in clinical setting.

T1-mapping has effectively ability in detecting edema and LGE pattern, and potential to play critical role in a gadolinium-free CMR protocol using T1-mapping. The patient fulfilled for T1-mapping and dark-blood T2-weighted imaging, which has a PPV of 95% for detecting AMI. However, if T1-mapping and Dark-blood T2 were negative, gadolinium may be administered in attempt to obtain a possible diagnosis based on convention criterion with clinical suspicious. Quantitative T1 measurement has the ability to distinguish normal tissue from abnormal myocardium. Dark-blood T2W imaging SI also plays an important diagnostic potential for myocardial diseases. However, inflammatory processes frequently are diffuse rather than discrete processes, conventional dark-blood T2WI SI rely on the comparison of remote unaffected areas of the myocardium or skeletal muscle. Viral myocarditis affects whole myocardial, as a result, there is no normal myocardium as contrast [22, 23]. T1-mapping could be suitable tool to directly identify inflamed myocardium and display involvement area. For T1-mapping to successfully translate into clinical practice, its ability to provide information on the entire myocardium in all territories would be important, especially for acute myocardium infarction. Immediate visual color T1-maps and those based on validated thresholds to detect disease with minimal extra post-processing steps may facilitate the adoption of T1-mapping into clinical practice [19, 24].

There still some limitations should be strengthened in this study. First, not all of the patients underwent X-ray coronary angiography. Second, part of the patients showed clinical signs of reperfusion, such as pain relief or resolution of ST segment on ECG. Endomyocardial biopsy is not routine clinical practice in our hospital. As a result, lack of the result of endomyocardial tissue biopsy, direct histopathologic correlation to the imaging findings is not available or inflammatory illness. Third, T1-mapping is not able to detect the small areas of injury in patients with negative identification of edema and LGE, which will result in an uncertain etiology of those findings. T1-thresholds T1 > 1200 ms for

distinguishing disease from normal may be platform-specific [25]. MOLLI based T1-mapping techniques exhibit some regional variation of T1 values across the myocardium in the detection of subtle, localized change, comparison to regional T1 norms may need multi center studies and clinical trials for further improve the ability of disease detection [26, 27].

In conclusion, native T1-mapping can be used to visualize myocardial T1 changes that occur in AMI, which diagnostic ability was comparable to LGE imaging without application of contrast agents. T1-mapping also improves the diagnostic confidence when conventional methods failed to identify abnormalities, and provides a potential ability for the measurement of infarct size. In future, it may be possible to perform gadolinium-free CMR, using cine and T1-mapping for tissue characterization, and may be particularly useful for patients in whom gadolinium contrast is contraindicated.

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Disclosure of conflict of interest

None.

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