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ORIGINAL PAPER

Cardiac magnetic resonance-tissue tracking for the early prediction of adverse left ventricular remodeling after ST-segment elevation myocardial infarction

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Abstract

Cardiac magnetic resonance-tissue tracking (CMR-TT)-derived myocardial strain after ST-elevation myocardial infarction (STEMI) is related to adverse cardiac events. We aimed to investigate the feasibility of CMR-TT for the early prediction of adverse left ventricular (LV) remodeling after STEMI. We retrospectively searched our institution's STEMI registry for patients who underwent reperfusion therapy, post-reperfusion CMR within 1 week after STEMI, and follow-up CMR. CMR-TT analysis was performed using cine imaging of post-reperfusion CMR. Adverse LV remodeling was defined as an increase in end-diastolic LV volume by 20% or more on follow-up CMR (median interval between serial CMR exams, 197 days; interquartile, 174–241 days). A total of 82 patients (age, 59.2 ± 11.1 years; male:female = 73:9) were included and divided into two groups: STEMI without (n = 62) and with (n = 20) adverse LV remodeling. Patients with LV remodeling showed significantly higher peak creatine kinase-MB and troponin I levels and a larger infarct size compared with those without LV remodeling ($p = 0.001$, $p = 0.001$, and $p = 0.010$, respectively). Global circumferential, radial, and longitudinal strain (GLS) also differed significantly between the groups ($p = 0.001$, $p = 0.004$, and $p < 0.001$, respectively). Logistic regression and receiver operating characteristic curve analyses demonstrated that GLS was an independent predictor of LV remodeling [odds ratio (OR) = 1.282, 95% confidence interval (CI) = 1.060–1.55 $p = 0.011$] with an optimal cut-off of − 12.84 (AUC = 0.756, 95% CI = 0.636–0.887, $p < 0.001$). CMR-TT-derived GLS may aid the early prediction of adverse LV remodeling after reperfusion, within 1 week after STEMI.

Keywords Cardiac magnetic resonance imaging · Ventricular remodeling · ST-segment elevation myocardial infarction

Introduction

Percutaneous coronary intervention has long been the cornerstone in the treatment of myocardial infarction (MI), and improvements in MI management have led to increased survival rates in the acute post-MI period. However, despite this progress, many patients are still exposed to a chronic consequence of MI known as ventricular remodeling. The importance of ventricular remodeling stems from the fact that the degree of adverse remodeling is associated with mortality [1]. The term ventricular remodeling refers to alterations in ventricular architecture, such as size and shape, which are driven by changes on a histologic level [2]. From a practical perspective, the pattern of remodeling can be categorized into two groups: that which achieves functional recovery and that which is adverse and irreversible [3]. A larger infarct size, greater extent of initial wall abnormalities, and greater initial

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reduction of systolic function are known to be independent predictors of adverse ventricular remodeling [1].

In an attempt to identify further predictors of long-term outcomes early in the post-reperfusion period, various clinical, echocardiographic, and cardiac magnetic resonance imaging (CMR) parameters have been evaluated [4–14]. Myocardial strain analysis, which was initiated using speckle tracking echocardiography (STE), has gained popularity as a prospective prognostic factor for the prediction of adverse left ventricle (LV) remodeling [4–6, 8, 10, 15]. Recently, a convenient method of CMR strain analysis called tissue tracking (CMR-TT) has been introduced. CMR-TT tracks features of interest along designated myocardial contours on routinely acquired cine images, thereby circumventing the need for additional sequences [16]. CMR-TT has been demonstrated as a feasible option in evaluating acute ST-elevation myocardial infarction (STEMI) patients and assessing their prognosis [17, 18]. Herein, we sought to investigate the feasibility of 1-week post-STEMI CMR-TT for the prediction of adverse LV remodeling, based on sequential CMR findings.

Methods

The institutional review board of Samsung medical center approved the study, and informed consent was waived for the use of patients' medical and imaging data.

Patient population

This retrospective study was performed in a single tertiary center. We retrospectively searched our institution's acute MI registry between July 2010 and September 2015 for patients who underwent reperfusion therapy, post-reperfusion CMR, and follow-up CMR. This yielded a total of 127 patients. Of these, 84 were diagnosed as STEMI patients. Two patients who had undergone post-reperfusion CMR over 7 days after reperfusion therapy were excluded. Finally, 82 STEMI patients who underwent reperfusion therapy, post-reperfusion CMR within 1 week after STEMI, and follow-up CMR were enrolled in the study (Fig. 1). The median time interval between serial CMR exams was 197 days (interquartile, 174–241 days). Patients were divided into two groups based on the presence of adverse LV remodeling at follow-up CMR. Adverse LV remodeling was defined as an increase of LV end-diastolic volume (LVEDV) by 20% or more on follow-up CMR, as compared to the initial CMR [19]. Patients clinical information and laboratory results were obtained from chart review.

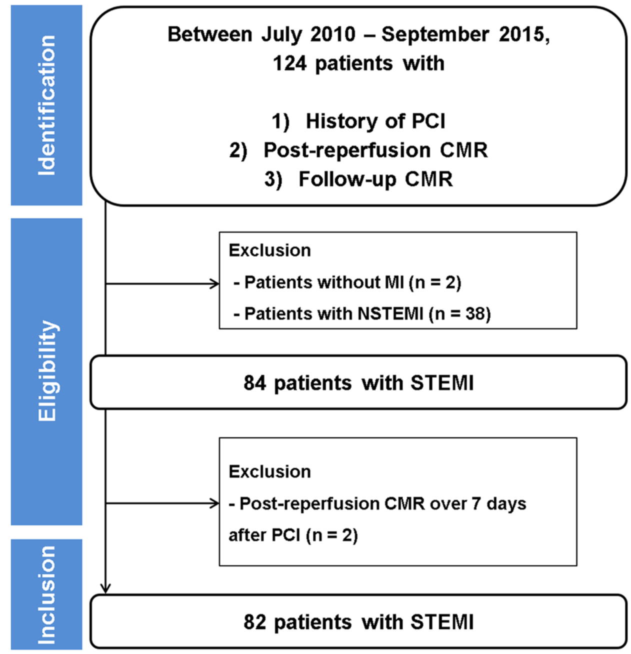

Fig. 1 Flow chart of patient enrollment

CMR imaging protocol

All MR images were obtained using a 1.5–T MR system (Magnetom Avanto; Syngo MR B17 version; Siemens Medical Solutions, Erlangen, Germany) equipped with a maximum strength gradient of 45 mT/m, a 200 mT/m/msec slew rate, and a 32–channel body array coil. Nitrates and beta–blockers were not used before examinations. After the acquisition of localizing images (axial, coronal, and sagittal), cine images of the LV were acquired through a steady-state free-precession sequence in the short-axis, 4-chamber, 3-chamber, and 2-chamber views. Short-axis views of T2-weighted black blood images, rest perfusion scans (with an intravenous infusion of 0.15 mmol/kg gadobutrol [Bayer Healthcare, Berlin, Germany] at an injection rate of 3 mL/s, followed by a 30 mL saline flush), and late gadolinium enhancement (LGE) scans were also acquired.

CMR analysis including functional parameters

LV functional analyses were done by one experienced investigator (Y.S.K, with 6 years of CMR experience) who was blinded to the clinical results. Quantitative evaluation of LV volume and mass at end-diastole and end-systole was performed. Frames with the largest and smallest ventricular volumes were chosen as the end-diastole and end-systole, respectively. LVEDV, LV end-systolic volume (LVESV), LV ejection fraction (LVEF), stroke volume (SV), cardiac output (CO), and LV mass (LVM) were calculated from the short-axis cine images using the ARGUS™ software (Siemens Healthcare, Erlangen, Germany) with semiautomatic contour detection. Manual correction of the automatically rendered endo- and epicardial contours was performed in all datasets, and papillary muscles and myocardial trabeculations were included in the ventricular cavity. Index values of LVEDV and LVESV were calculated as LVEDV and LVESV divided by the body surface area, respectively. LVM was measured by multiplying the sum of the total LV myocardial volumes from the cine images by the specific gravity of the myocardium.

Infarct size was measured by an investigator (Y.S.K, with 6 years of CMR experience) on LGE and then confirmed by another investigator (S.M.K., with 7 years of CMR experience). The volume of MI was calculated as the sum of the area of LGE within each segment of the short-axis images multiplied by the slice thickness and gap to cover the entire LV. The infarct size was defined as the proportion of LGE to LV myocardial volume. The presence of microvascular obstruction (MVO) and intramyocardial hemorrhage (IMH) was also assessed by two investigators (C.M.J. with 4 years and S.M.K. with 7 years of CMR experience) on post-reperfusion CMR, and decisions were reached in consensus. MVO was defined as a region of hypoenhancement within the core of the infarction on LGE images. IMH was assessed on T2-weighted black blood sequences and appeared as a hypointense region within the infarcted area [20].

CMR tissue-tracking analysis

CMR-TT analysis was performed using a commercially available software (cvi42 version 5, Circle Cardiovascular Imaging Inc., Calgary, Alberta, Canada) on post-reperfusion CMR. Three short-axis sections were selected, one each from the apical, mid-ventricular, and basal levels. Three long-axis sections (2-chamber, 3-chamber, and 4-chamber views) were used. The preferred images were loaded into the analysis/viewer frame of the software and analyzed in a random order by two investigators (H.N.J. with 3 years and J.H.L with 2 years of CMR experience) who were independently blinded to the clinical findings. Semi-automatic delineation of the LV endocardial and epicardial borders was performed in each section at end-diastole. The mitral valve plane and apex were drawn on all long-axis sections at end-diastole and automatically propagated to the other phases. Manual correction was performed when the automatic propagation obviously deviated from the mitral valve plane and apex. The RV upper septal insertion point was manually defined on the basal short-axis section. In each subsequent frame, the displacements of the myocardial tissues, including the borders, were automatically determined using a gradient-based optical flow method [21]. More precisely, the displacement of myocardial segments can be measured, based on defining small square windows, centered around a feature, on a first image and searching the "as-much-as-possible similar" greyscale pattern on the following image [22, 23]. Global and segmental strain values from 2D strain analysis were obtained for all studies and exported for statistical analysis.

Statistical analysis

Statistical analysis was executed using SAS version 9.4 (SAS Institute, Cary, NC) and R 3.3.1 (Vienna, Austria; https://www.R-project.org/). Baseline clinical and laboratory data were compared between the two groups. CMR parameters (LVESV, LVESV index, LVEDV, LVEDV index, LVEF, LVM, infarct size, and the presence of MVO and IMH) and strain values (peak global circumferential, radial, and longitudinal strain) acquired from the initial CMR were compared using the two-sample *t* test, Wilcoxon rank sum test, or chi-square test. The correlations between the strain values and LVEF on follow up CMR were evaluated using Spearman correlation analysis. Multivariate logistic regression with the backward stepwise method was performed to identify the predictors of adverse LV remodeling. The variables with $p < 0.05$ on univariate analysis were used as the input variables for the multivariate analysis. Firth's penalized maximum likelihood estimation method was applied when rare events were encountered on logistic regression. Receiver operating curve (ROC) analysis with area under the curve (AUC) was also performed. The intraclass correlation coefficients (ICCs) were obtained for inter- and intraobserver variability.

Results

Baseline clinical and CMR characteristics

The baseline characteristics of the patients with and without adverse LV remodeling are summarized in Table 1. Of the 82 patients (age, 59.2 ± 11.1 years; M:F = 73:9), there were 62 patients without adverse remodeling (change of LVEDV = 1.05% ± 10.60%) and 20 patients with adverse remodeling (change of LVEDV = 33.56% ± 11.17%). Female sex was more frequent in those with LV remodeling ($p = 0.035$). In addition, peak CK-MB and troponin I levels were significantly higher in patients with LV remodeling compared with those without ($p = 0.001$ and $p = 0.001$, respectively). On the contrary, the time intervals of symptom

Table 1 Baseline characteristics of patients with and without adverse left ventricular remodeling

Characteristics	Patients without LV remodeling (n = 62)	Patients with LV remodeling (n = 20)	*P* value
Age, y	58.8 ± 11.6	60.2 ± 9.7	0.615
Sex (male:female)	58:4	15:5	**0.035**
Height (m)	1.68 ± 0.06	1.66 ± 0.09	0.480
Weight (kg)	71.9 ± 11.8	67.4 ± 11.3	0.138
Body mass index, kg/m2	25.3 ± 3.3	24.1 ± 3.0	0.181
Body surface area (m^2)	1.81 ± 0.16	1.75 ± 0.17	0.175
Hypertension (%)	25 (40.3%)	10 (50.0%)	0.604
Culprit coronary artery (%)*			0.243
Left main coronary artery	0 (0%)	1 (5.0%)	
One-vessel [RCA/LAD/LCx]	54 (87.1%) [21/28/5]	18 (90.0%) [2/16/0]	
Multi-vessel	8 (12.9%)	1 (5.0%)	
Serologic tests			
Peak CK-MB	185.3 ± 124.8	332.9 ± 277.2	**0.001**
Peak troponin I	98.1 ± 90.3	198.5 ± 162.8	**0.001**
Symptom to PCI (min)	566.6 ± 1843.8	322.3 ± 335.9	0.323
PCI to post-reperfusion CMR (days)	2.9 ± 0.9	3.7 ± 1.3	0.356

Values are presented as mean ± standard deviation or n (%). *Percentages refer to each affected coronary artery, with the total of 100% per group

Boldface indicates p values less than 0.05

LV left ventricular, *RCA* right coronary artery, *LAD* left anterior descending artery, *LCx* Left circumflex artery, *CK-MB* creatine kinase-MB, *PCI* percutaneous coronary intervention, *CMR* cardiac magnetic resonance

onset to PCI and PCI to CMR acquisition were not significantly different between the two groups ($p = 0.323$ and $p = 0.356$, respectively).

In terms of CMR functional analysis, there was no significant difference in the LV functional parameters on post-reperfusion CMR between the two groups (Table 2).

Table 2 Comparison of CMR parameters and strain characteristics between patients with and without adverse left ventricular remodeling

Characteristics	Patients without LV remodeling (n = 62)	Patients with LV remodeling (n = 20)	*P* value
CMR parameters			
LVEF (%)	55.3 ± 9.3	50.9 ± 12.0	0.148
LVEDV (mL)	141.6 ± 29.9	125.8 ± 35.0	0.084
LVEDV index (mL/m^2)	77.7 ± 13.0	71.5 ± 17.9	0.164
LVESV (mL)	66.8 ± 27.0	63.7 ± 28.3	0.670
LVESV index (mL/m^2)	36.5 ± 13.1	36.3 ± 15.5	0.963
End-diastolic wall mass (mL)	130.1 ± 36.9	129.6 ± 36.9	0.960
Infarct size (%)	19.2 ± 10.6	27.3 ± 15.1	**0.010**
Microvascular obstruction (%)	49 (79.0%)	15 (75%)	0.759
Intramyocardial hemorrhage (%)	37 (59.7%)	14 (70.0%)	0.442
CMR strain			
Peak global circumferential	− 15.6 ± 4.2	− 12.1 ± 3.7	**0.001**
Peak global radial	33.8 ± 10.2	26.8 ± 8.2	**0.004**
Peak global longitudinal	− 13.6 ± 3.5	− 10.2 ± 3.0	**< 0.001**

Values are presented as mean ± standard deviation or n (%)

Boldface indicates *p* values less than 0.05

LVEF left ventricular ejection fraction, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume

Infarct size, MVO, IMH, and CMR-TT-derived myocardial strains

A significant difference was observed in the myocardial infarct size between those with and without LV remodeling (27.3 ± 15.1% vs. 19.2 ± 10.6%; $p = 0.035$). However, the presence of MVO and IMH did not differ significantly between the two groups ($p = 0.759$ and $p = 0.442$, respectively). All the measurements on CMR-TT analyses demonstrated significant differences, including the global circumferential strain (GCS) (− 12.1 ± 3.7 vs. − 15.6 ± 4.2; $p = 0.001$), global radial strain (GRS) (26.8 ± 8.1 vs. 33.8 ± 10.1; $p = 0.004$), and global longitudinal strain (GLS) (− 10.2 ± 3.0 vs. − 13.6 ± 3.5; $p < 0.001$), between the two groups (Table 2 and Fig. 2) (Supplemental materials). Spearman correlation analyses demonstrated moderate degree of correlations between GCS (rho = − 0.542; $p < 0.001$), GRS (rho = 0.481; $p < 0.001$), and GLS (rho = − 0.441; $p < 0.001$) and LVEF on approximately 6-month-follow-up CMR.

Predictors of adverse left ventricular remodeling within one week after STEMI

Backward stepwise logistic regression analyses for predicting adverse LV remodeling were performed. Parameters including peak CK-MB and troponin I levels, infarct size on CMR, and CMR-TT strain values were considered as input variables for the multivariate analysis. Multivariate logistic regression analysis demonstrated that GLS was an independent predictor of adverse LV remodeling [odds ratio (OR) = 1.282, 95% confidence interval (CI) = 1.060–1.550, $p = 0.011$] (Table 3). In the ROC analyses, GLS was the most accurate diagnostic parameter for adverse LV remodeling with an optimal cut-off of − 12.84 (AUC = 0.756, 95% CI = 0.636–0.876, sensitivity = 85.0%, specificity = 61.3%, $p < 0.001$), as compared with peak CK-MB level (AUC = 0.665; 95% CI = 0.517–0.813, $p = 0.027$), peak troponin I level (AUC = 0.688; 95% CI = 0.541–0.813, $p = 0.012$), GCS (AUC = 0.735; 95% CI = 0.612–0.859, $p = 0.002$), GRS (AUC = 0.304; 95% CI = 0.177–0.431, $p = 0.009$), and infarct size (AUC = 0.675; 95% CI = 0.526–0.824, $p = 0.019$) (Fig. 3).

Intra- and interobserver variability of the CMR strains

The ICCs for interobserver variability were 0.691 (95% CI = 0.220–0.878) for GCS, 0.845 (95% CI = 0.608–0.939) for GRS, and 0.772 (95% CI = 0.425–0.910) for GLS, respectively. For intraobserver variability, the ICCs were 0.724 (95% CI = 0.302–0.890) for GCS, 0.811 (95% CI = 0.523–0.925) GRS, and 0.760 (95% CI = 0.396–0.905) for GLS, respectively.

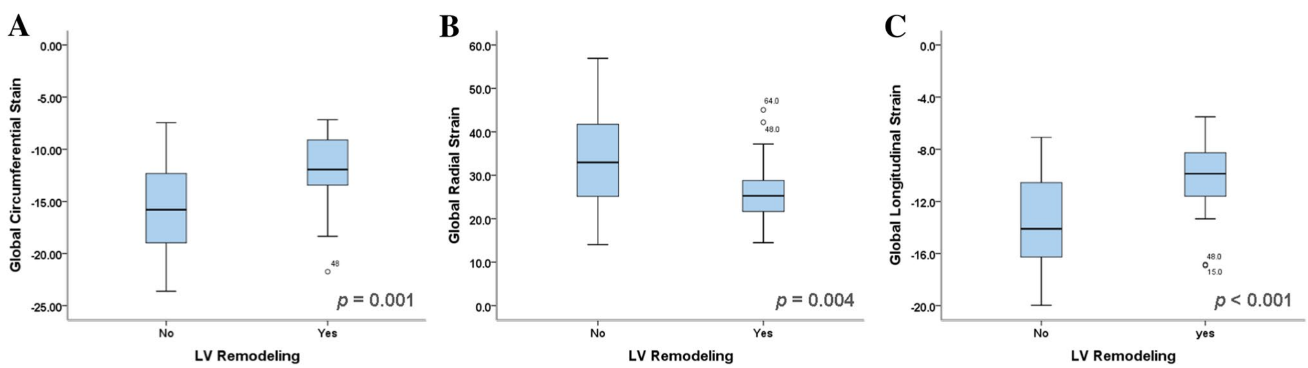

Fig. 2 Comparison of peak global circumferential (**a**), radial (**b**), and longitudinal (**c**) strains between patients with and without left ventricular remodeling

Table 3 Logistic regression analysis for predicting adverse left ventricular remodeling

Characteristics	Odds ratio	95% Confidence interval	*P* value
Peak CK-MB	1.003	0.999–1.006	0.117
Peak troponin I	1.002	0.995–1.009	0.556
Infarct size (%)	0.965	0.892–1.043	0.369
Global radial strain	1.013	0.876–1.171	0.862
Global circumferential strain	1.040	0.790–1.370	0.779
Global longitudinal strain	1.282	1.060–1.550	**0.001**

Boldface indicates *p* values less than 0.05

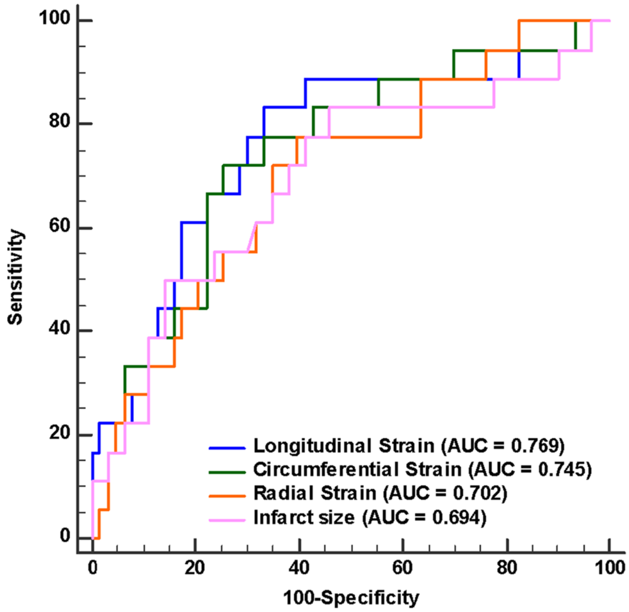

Fig. 3 Receiver operating curve analysis for predicting adverse left ventricular remodeling

Discussion

The main finding of our study is that GLS using CMR-TT is a valid predictor of adverse LV remodeling within 1 week after STEMI. Longitudinal strain is always a negative value by definition; therefore, a patient showing less longitudinal deformity is at higher risk of adverse remodeling. This is in line with previous comparable studies based on STE, which demonstrated that GLS by STE was independently associated with LV dilatation and subsequent cardiac events [24–26]. In addition to longitudinal strain, we also found significant differences in serum cardiac markers, infarct size on CMR, and other CMR-TT strain values including GCS and GRS between those with and without adverse LV dilatation. However, GLS was the most accurate diagnostic parameter for adverse LV remodeling with an optimal cut-off of − 12.84. An interesting finding was that known CMR predictors of adverse remodeling such as MVO and IMH did not show a significant correlation with adverse remodeling. We speculate that this may be due to the lack of quantification or further classification of these parameters.

One of the strengths of CMR-TT is that it is a contrast agent-free technique. The measurements of infarct size and MVO on LGE are known to have an additive value as a predictor of adverse outcomes after acute MI [27, 28]. However, it is not always possible to use a gadolinium contrast agent in post-reperfusion STEMI patients, especially in those with severe renal impairment [29]. Moreover, we must take into consideration the relatively long time it takes to perform CMR including LGE, usually over 30 min, which makes it difficult to perform CMR in every acute MI patient. Our study demonstrated that GLS using the CMR-TT of cine images might provide a better prediction of adverse LV remodeling over infarct size on LGE. Indeed, CMR-TT may play a potential role as an independent predictor of adverse LV dilatation, especially in those for whom LGE acquisition is not possible.

Recently, several studies analyzed myocardial strain using CMR-TT in patients with acute STEMI [18, 30]. The studies demonstrated the value of GLS as a significant predictor of adverse cardiac events and poor outcomes; however, its relationship with adverse LV dilatation, per se, was limited. Our study demonstrated the significance of GLS as a predictor of adverse LV remodeling, which may explain one of the mechanisms leading to poor prognosis [19]. The precise identification of adverse LV remodeling was achieved with follow-up CMR in each patient in the current study. We were able to predict long-term changes in LV myocardial geometry on the basis of the strain value early after STEMI, which may enhance our understanding of the disease course and the onset of heart failure from a pathophysiological standpoint.

In contrast, Shetye et al. reported that global myocardial strain assessment did not predict the development of adverse LV remodeling at 4-month follow-up [31]. However, they determined adverse remodeling based on the development of either an end-systolic volume index ≥ 15% or end-diastolic volume ≥ 20% at follow-up as compared with baseline. In addition, they acquired strain parameters based on CMR tagging and feature-tracking, which was obtained by defining endocardial border only. Another study by Garg et al. has reported that baseline GLS of CMR-TT, obtained based on both endocardial and epicardial borders, was strongly associated with adverse LV remodeling on 3-month follow-up after STEMI [32]. In this study, they performed CMR-TT analysis by use of the same software and methodology with our study. In terms of timescale for LV remodeling, it is known that LV remodeling may continue for weeks or months until the distending forces are counterbalanced by the tensile strength of the myocardial fibrosis [33]. Bolognese et al. has reported that adverse LV remodeling (end-diastolic volume ≥ 20%) was detected in 15% of patients within 1 month from MI, and in 14% of patients between 1-month and 6-month follow-up [19]. Indeed, the proportion of patients with adverse LV remodeling in our study population (24.4%) was larger than other studies based on 3-month (17%) or 4-month (16.9%) follow up [31, 32]. We believe that our study contributes to the growing body of literature on cardiac strain analysis and is unique in that it shows the relationship between CMR-TT measurements and adverse LV remodeling based directly on serial CMR.

To date, many predictors of adverse LV remodeling after MI have been identified. Well-known factors include infarct size, reduction of systolic function, serum cardiac biomarkers, and extent of initial wall motion abnormalities [1, 4, 5, 13, 14, 19, 25, 30, 34]. Myocardial deformation imaging has emerged as a novel tool for quantifying wall motion abnormalities. There are several representative methods of obtaining myocardial strain values, such as STE, CMR tagging, and CMR-TT. CMR tagging has been validated for myocardial strain quantification, however, it requires additional sequence acquisition and post-processing which is time-consuming [35]. STE has also been widely used in clinical practice with higher accessibility and rapidity, however, it has the inherent limitations of operator dependency and poor image quality. On the other hand, CMR-TT provides deformation parameters from routinely available cine images without additional sequences and time for post-processing is relatively short. There have been several studies on the comparison of different methods of strain computation, and showed reasonable agreements among STE and CMR-derived parameters [17, 31, 36, 37]. Our results showed that CMR-TT offers valuable information for identifying high-risk patients who may benefit from agents that prevent or treat adverse ventricular remodeling [1].

Limitations of our study include the retrospective design, small sample size, and imbalance of baseline characteristics such as the gender ratio and follow-up interval. In addition, quantitative evaluation of IMH and MVO was not performed, which may have led to the non-significance of these parameters in the two groups. Lastly, the present study lacks long-term follow-up data for an evaluation of cardiovascular outcomes.

In conclusion, strain analysis using CMR-TT obtained after reperfusion therapy for STEMI may facilitate the prediction of adverse LV remodeling within 1 week after PCI. In particular, GLS is a significant independent predictor of adverse LV dilatation. This knowledge may assist in the identification of high-risk patients who could benefit from close monitoring and early medical treatment.

Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

Informed consent The institutional review board of our institution approved the study, and informed consent was waived.

Research involving human and animal participants The ethical standards of the responsible committee on human experimentation (institutional and national) and Declaration of Helsinki of 1964 (revised in 2008) were followed.

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