Cardiac Strain as a Predictor of Adverse Events and Ventricular Remodeling: A Cohort Study

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Abstract

Background: It remains controversial whether cardiac strain accurately predicts adverse events after acute ST-segment elevation myocardial infarction (STEMI). The aim of the present study was to evaluate the effects of cardiac strain revealed on cardiac magnetic resonance (CMR) imaging on cardiac events and adverse left ventricular (LV) remodeling.

Methods: Between February 2015 and September 2016, we conducted a prospective two-center cohort study of patients with STEMI treated with primary percutaneous coronary intervention comprising stent implantation. All included patients underwent CMR imaging before discharge. Major adverse cardiac events (MACE) and LV remodeling were assessed during 6 months of follow-up.

Results: Seventy-six patients were available for the final analysis. The MACE rate was 23.7%, using cardiac death, reinfarction, unplanned revascularization, and heart failure as combined events during 6 months of follow-up. The global longitudinal strain (GLS) was an independent predictor of MACE (OR=1.21 (1.07–1.36), P=0.002) and LV remodeling (OR=2.06 (1.14–3.73), P=0.017).

Conclusion: In patients with STEMI treated with primary percutaneous coronary intervention, the GLS determined on CMR imaging performed before discharge is a predictor of MACE and adverse LV remodeling during 6 months of follow-up.

Background

Patients with acute ST-segment elevation myocardial infarction (STEMI) have a high risk of adverse outcomes, even after timely revascularization (1,2). Hence, early risk stratification of such patients is clinically important. Patients with STEMI treated with stent implantation often exhibit remodeling of the left ventricle. As left ventricular (LV) remodeling is associated with future adverse outcomes, changes in cardiac parameters may be risk factors for adverse events after revascularization in patients with STEMI.

Substantial studies have reported the superior prognostic power of global longitudinal strain (GLS) for predicting LV remodeling and predicting adverse events using the speckle tracking echocardiography technique (3-5). However, cardiac magnetic resonance (CMR) imaging is considered the gold standard for the assessment of regional myocardial strain. CMR feature tracking (CMR-FT) acquires steady-state free-precession cine images and accurately predicts transmural infarction, myocardial deformation, and wall motion (6,7). Although CMRFT indices show independent prognostic implications in dilated and chronic ischemic cardiomyopathy as well as tetralogy of Fallot (8-10), the usefulness of CMR-FT in myocardial infarction is controversial (11,12). Eitel et al. suggested an incremental prognostic role of CMR-FT-derived GLS above classical CMR prognostic markers in patients with acute myocardial infarction (including STEMI and non-STEMI) (11). In contrast, a retrospective study failed to demonstrate a prognostic value of GLS over other established CMR parameters in 323 patients with STEMI (12).

The objective of the present study was to assess the effects of CMR-determined cardiac strain on cardiac events and adverse LV remodeling. We hypothesized that cardiac GLS measured before discharge is a good predictor of cardiac events and adverse LV remodeling after revascularization for STEMI. To test this hypothesis, the present prospective two-center study evaluated patients with STEMI treated with primary percutaneous coronary intervention (PCI) comprising stent implantation. These patients underwent CMR imaging before discharge. Follow-up data at 6 months after treatment were obtained from hospital records or face-to-face visits. The study findings will aid in the prediction of adverse events after revascularization in patients with STEMI.

Methods

Study Design: This prospective cohort study was performed in Peking University First Hospital and the Affiliated Hospital of Xuzhou Medical University. The study was approved by the Institutional Review Board of each participating center, and conformed to the Declaration of Helsinki and Good Clinical Practice Guidelines of the China Food and Drug Administration. All patients provided written informed consent.

Participants: The study cohort comprised 86 patients with STEMI treated with primary PCI comprising stent implantation in one of the two participating centers from February 2015 to September 2016. All patients underwent CMR imaging before discharge.

Exclusion criteria were: 1) atrial fibrillation, frequent premature contractions, persistent ventricular tachycardia, or other tachyarrhythmia; 2) previous cardiac surgery or myocardial infarction; 3) severe liver and/or kidney dysfunction; 4) malignancy; 5) life expectancy of less than 1 year; 6) pregnancy; and 7) contraindications to magnetic resonance imaging (e.g. contrast agent allergy, ferromagnetic objects in the body, claustrophobia).

CMR Imaging Measurements: CMR imaging was performed before discharge (generally 5–7 days after the index event). All patients were examined with a 1.5 Tesla GE magnetic resonance imaging scanner. Three long-axis views (four-, three-, and two-chamber orientation) and short-axis stacks were acquired using a balanced steady-state free-precession imaging technique for functional cardiac analyses. Native T2, T2-weighted, and post-contrast T1-weighted image sequences were used for the assessment of edema, infarction size, microvascular obstruction (MVO), and intramyocardial hemorrhage. T1-weighted images were obtained 15 minutes after the administration of gadolinium-based contrast agent.

CMR Imaging Analysis: The analysis was performed offline by two experienced radiologists. Infarct size, edema, MVO, and intramyocardial hemorrhage were quantified using CVI 42 software (Circle Cardiovascular Imaging Calgary, Canada) (13). CMR-FT strains (GLS, global circumferential strain (GCS), and global radial strain (GRS), LV end-diastolic volume (LVEDV), LV end-systolic volume, and LV ejection fraction (LVEF) were determined using the TomTec Imaging System (2D CPA MR, Cardiac Performance Analysis, version 1.1.2, TomTec Imaging Systems, Germany) (14,15). Briefly, LV contours were drawn semi-automatically at the end of diastole and systole. Subsequently, image features throughout an entire

cardiac cycle were determined by the automatic border tracking algorithm of the software. Accurate tracking was confirmed by visual review of all borders and manual adjustments with consequent reapplication of the algorithm if necessary.

Follow-up Examination: The incidence of major adverse cardiac events (MACE), including cardiac death, reinfarction, unplanned revascularization, and heart failure within 6 months after STEMI was obtained from hospital records or face-to-face visits. Heart failure manifestations were defined as the exacerbation of exertional dyspnea or pulmonary edema requiring hospital admission, initiation of diuretics, or an increase in an existing diuretic regimen. Follow-up CMR imaging was performed at 6 months after STEMI. Adverse LV remodeling was defined as an LVEDV of > 15% greater than the LVEDV before discharge from the hospital.

Statistical Analysis: Variables are denoted as mean ± standard deviation, and the independent t test or Fisher exact test was used to compare differences between groups. Variables that were not normally distributed (as determined by Kolmogorov–Smirnov tests) were expressed as medians with 25th and 75th percentiles, and were compared using the Mann-Whitney U test. Based on the ratio of the infarcted myocardial mass to the LV mass (IM%LV), patients were divided into group A (IM%LV < 10%), group B (10% ≤ IM%LV < 20%), and group C (IM%LV ≥ 20%).

A comparison of multiple variables was performed between patients with LV remodeling and patients without LV remodeling, and between patients who did and did not develop MACE during follow-up. Uni- and multivariate logistic backward stepwise regression analyses were performed to evaluate the potential correlations between clinical parameters and CMR imaging parameters to MACE and LV remodeling. Because of the small sample size, the parameters with a p value < 0.05 in comparisons between the MACE and no MACE groups (and between the LV remodeling and no LV remodeling groups), sex, and age were selected for the logistic regression analysis. If there were multiple parameters with high correlations, only the most clinically significant parameter was selected for the analysis. For example, as LVEDV, LV end-systolic volume, and LVEF were highly correlated, only LVEF was used in the analysis. Receiver operating-characteristic (ROC) curve analysis was used to determine the cutoff values of the GLS for predicting MACE. All statistical analyses were performed with a test significance level of 0.05 using SAS version 9.4 (SAS Institute, Inc., Cary, NC).

Results

The follow-up analysis was carried out in 76 patients (age 55.5 ± 10.7 years; 88% male) who were treated with primary PCI for STEMI and underwent CMR imaging examination before discharge, as shown in Fig. 1. Baseline patient characteristics are presented in Table 1. The most commonly accessed vessel was the left anterior descending artery, followed by the right coronary artery and left circumflex artery. The baseline characteristics that differed between patients with different degrees of myocardial infarction were the peak brain natriuretic peptide (BNP) level, peak cardiac troponin I level, and symptom-to-balloon time.

Table 2 lists the cardiac characteristics obtained from CMR imaging at baseline. Group C had the lowest absolute GLS, GCS, and GRS values and the lowest LVEF, while group A had the highest values. Group C also had the worst features regarding the other variables. In 6 months of follow-up, MACE occurred in 18 patients (23.7%), including one patient with cardiac death, one with non-fatal reinfarction, four with unplanned revascularization, and 12 with heart failure. Patients with a higher IM%LV had a higher incidence of MACE than those with a lower IM%LV. Table 3 summarizes the baseline clinical and CMR imaging characteristics of the patients with MACE compared with those without MACE. Patients with MACE had a higher peak BNP level, higher peak cardiac troponin I level, longer hospital stay, longer symptom-to-balloon time, and worse CMR parameters than those with no MACE.

Univariate logistic regression analysis revealed that the variables predicting MACE were peak BNP level, LVEF, IM%LV, MVO, GLS, and GCS. Backward stepwise multivariate analysis confirmed that GLS was an independent predictor of MACE (OR=1.21 (1.07–1.36); P=0.001; Table 4). Figure 2 shows the ROC curve of the GLS. The area under the ROC curve was 0.763. The best cutoff value of GLS for predicting MACE was -14.6%, with a diagnostic sensitivity of 72.2% and a diagnostic specificity of 74.2%.

CMR imaging was performed at 6 months after STEMI in 24 patients (age 54 ± 11 years; 88% male). CMR imaging parameters showed improved cardiac function at 6 months after STEMI treatment compared with baseline (Table 5). Table 6 shows a comparison of the clinical and CMR imaging characteristics at baseline in patients with LV remodeling versus patients without LV remodeling on follow-up examination. Univariate logistic regression analysis revealed that the variables predicting LV remodeling were symptom-to-balloon time and GLS. Backward stepwise multivariate analysis confirmed that GLS was an independent predictor of LV remodeling (OR=2.06 (1.14–3.73); P=0.017; Table 7).

Discussion

The present study showed the usefulness of GLS in the prediction of MACE and LV remodeling after PCI in patients with STEMI. After adjustments for clinical and morphometric parameters, the CMR-determined GLS before discharge was independently associated with adverse remodeling and outcomes at 6 months after STEMI treatment.

Relationship between GLS and MACE

The present study reported a MACE rate of 23.7%, using cardiac death, reinfarction, unplanned revascularization, and heart failure as combined events. Previous studies have reported similar MACE rates of 22% using cardiac death and heart failure as combined events (16), and 21% using cardiac death, acute myocardial infarction, and heart failure as combined events (17).

When measured soon after revascularization, the LVEF is a proven predictor of poor outcome in patients with myocardial infarction (18-20). However, the LVEF is a global parameter that represents the entire LV function and is thus a weak predictor of late myocardial dysfunction (21,22). Assessments of the myocardial strain in the circumferential, longitudinal, and radial directions (i.e., GLS, GCS, and GRS,

respectively) are sensitive markers of intrinsic myocardial function, providing an improved analysis of cardiac dysfunction early after myocardial infarction on local and global levels (7,23). Myocardial strain assessed using the speckle tracking echocardiography technique accurately predicts adverse events (24-26). Based on the gold standard CMR imaging measurements (27-29), the present study showed that GLS was an independent predictor of MACE, with an optimal cutoff value of -14.6%. Similarly, previous studies have reported that GLS is a strong and independent predictor of adverse events (30,31), and a study of 659 patients with acute myocardial infarction demonstrated that a GLS value of greater than -15.1% was an independent predictor of cardiovascular events, either by combining all events or separating these events into mortality, reinfarction, revascularization, and hospitalization for heart failure (30).

Relationship between GLS and LV remodeling

Even after PCI, adverse LV remodeling occurs in 30–35% of patients with STEMI (32), and is an important predictor of arrhythmias, heart failure, and mortality (33,34). In the present study, the rate of adverse LV remodeling at 6 months after STEMI treatment was 29%, which is similar to the rate reported in a previous study (35). Although the LVEF is routinely used to assess LV systolic function, it cannot predict the development of LV remodeling. GLS is reportedly an independent predictor of adverse LV remodeling (16,30,35). The present study confirmed that GLS was an independent predictor of adverse LV remodeling. Similarly, a previous prospective study identified GLS as a strong predictor of clinical outcomes and an independent predictor of MACE and LV remodeling in multivariable logistic regression analysis after adjustment for other established prognostic risk factors, including the LVEF and infarct size (11,12).

Study Limitations

The present study has several limitations. First, the sample size was relatively small and was limited to patients with STEMI treated with primary PCI comprising stent implantation in two centers. Hence, selection bias and low statistical power should be considered when interpreting the findings. Second, patients with cardiogenic shock and those requiring mechanical ventilation or intra-aortic balloon counter-pulsation therapy were not included. Third, the echocardiographic GLS was not measured, and so could not be compared with the CMR-derived GLS. Finally, heart rate and blood pressure, which influence strain computation, were not available for all patients when underwent CMR imaging.

Conclusions

In patients with STEMI treated with primary PCI, the CMR-determined GLS before discharge was a predictor of MACE and adverse LV remodeling at 6 months after STEMI treatment. Hence, GLS has potential as a risk factor to quantify ventricular dysfunction.

Declarations

Ethics approval and consent to participate: The study was approved by the Institutional Review Board of each participating center, and conformed to the Declaration of Helsinki and Good Clinical Practice Guidelines of the China Food and Drug Administration. All patients provided written informed consent for study participation.

Consent for publication: Not applicable.

Availability of data and materials: The datasets used and/or analyzed in the current study are available from the corresponding author on reasonable request.

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Authors' contributions: Yanjun Gong and Yuan Lu assessed the patients for study eligibility, performed data analysis, and wrote and edited the manuscript. Jessica C. Huo polished the language of the manuscript. Zhi Wang, Fan Yang, and Lin Qiu helped perform the patient follow-up and data collection. Shu Fang edited the manuscript. Jianxing Qiu performed cardiac magnetic resonance imaging. Yong Huo helped in designing the study, performing data analysis, and editing the manuscript.

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Abbreviations

STEMI: ST-segment elevation myocardial infarction; LV: Left ventricular; MVO : Microvascular obstruction; GLS: Global longitudinal strain; CMR:Cardiac magnetic resonance; CMR-FT : CMR feature tracking; PCI: Percutaneous coronary intervention; GCS: Global circumferential strain; GRS: Global radial strain; LVEDV: LV end-diastolic volume; LVEF: LV ejection fraction; MACE: Major adverse cardiac events; IM%LV: The infarcted myocardial mass to the LV mass ROC: Receiver operating-characteristic; BNP: brain natriuretic peptide.

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Tables

Table 1. Baseline characteristics of the study population

Variables	Total (n=76)	Group A (n=29) (IM%LV<10%)	Group B (n=33) (10%≤IM%LV<20%)	Group C (n=14) (IM%LV≥20%)	P
Age (years)	55.5±10.7	53.7±11.3	56.7±9.6	56.3±12.1	0.519
Male (n, %)	67 (88.2)	26 (89.7)	29 (87.9)	12 (85.7)	0.930
BMI (kg/m^2)	24.9±3.1	25.0±3.3	24.4±3.0	25.7±2.7	0.422
Smoking (n, %)	50 (65.8)	17 (58.6)	25 (75.7)	8 (57.1)	0.275
Diabetes mellitus (n, %)	12 (15.8)	3 (10.3)	6 (18.2)	3 (21.4)	0.570
Hypertension (n, %)	38 (50)	16 (55.2)	17 (51.5)	5 (35.7)	0.476
Systolic blood pressure (mmHg)	135±9	140±19	130±18	136±16	0.164
Heart rate (beats/min)	73.5±10.5	75.4±10.2	73.1±11.1	74.4±9.4	0.670
LDL-C (mmol/L)	2.8±0.8	2.8±0.7	2.7±0.7	2.8±1.2	0.787
HDL-C (mmol/L)	1.1±0.2	1.1±0.2	1.0±0.2	1.1±0.4	0.123
Peak BNP (pg/ml)	1452±1219	626±698	1500±1019	3051±1648	<0.001
Peak cTNI (ng/ml)	8.53 (3.39, 14.29)	2.6 (1.4, 4.25)	11.4 (8.02, 14.9)	26.4 (13.1, 69.8)	<0.001
Multivessel coronary disease (n, %)	21 (27.6)	6 (20.7)	10 (30.3)	5 (35.7)	0.529
Culprit lesion on CAG LAD (n, %)	43 (56.6)	16 (55.2)	18 (54.5)	9 (64.3)	0.812
Hospital stay (days)	10 (8, 12)	8 (7, 10)	10 (8, 12)	11 (10, 13)	0.091
TIMI flow frame count (frames)	28 (20, 37)	28 (21.2, 38.9)	26 (18.82, 36.0)	33.0 (25.9, 38.0)	0.476
TIMI Myocardial Perfusion Classification ≤ level 2 (n, %)	4 (5.26)	2 (6.8)	1 (3.03)	1 (7.1)	0.362
Symptom to balloon time (minutes)	346±156	219±115	369±110	535±130	0.002
With Collateral circulation (n, %)	11 (14.5)	6 (20.9)	4 (12.1)	1 (7.1)	0.436

IM%LV: infarcted myocardial mass/left ventricular mass; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; BNP: brain natriuretic peptide; cTNI: cardiac troponin I.

Table 2. Cardiac characteristics obtained from CMR imaging at baseline

Variables	Group A (n=29) (IM%LV<10%)	Group B (n=33) (10%≤IM%LV<20%)	Group C (n=14) (IM%LV≥20%)	P
LVEDV (ml)	123.9±14.8	124.2±12.3	144.6±16.2	0.037
LVESV (ml)	57.1±16.1	66.14±11.6	80.5±12.5	0.021
LVEF (%)	57.5±7.5	47.7±7.6	44.2±4.6	<0.001
IMH	2 (6.9%)	7 (21.2%)	6 (42.9%)	0.020
MVO	7 (24.1%)	18 (54.5%)	11 (78.5%)	0.002
IMH+MVO	2 (6.9%)	7 (21.2%)	6 (42.9%)	0.020
Edema/LV (%)	23.3 (20.5, 30.2)	32.3 (27.5, 38.0)	36.12 (34.2, 39.1)	0.002
Salvage/LV (%)	18.8 (14.9, 24.6)	17.8 (12.9, 21.9)	12.1 (6.0, 14.4)	0.002
IM%LV (%)	6.2±5.2	14.5±5.8	26.0±7.3	0.002
GLS (%)	-18.8±3.8	-15.6±3.5	-7.3±2.2	P<0.001
GCS (%)	-28.2±5.16	-22.5±5.6	-18.4±3.25	P<0.001
GRS (%)	36.87 (33.5, 42.4)	30.24 (23.6, 37.5)	24.58 (20.6, 32.6)	P=0.005

LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; LVEF: left ventricular ejection fraction; IMH: intramyocardial hemorrhage; MVO: microvascular obstruction; GLS: global longitudinal strain; GCS: global circumferential strain; GRS: global radial strain.

Table 3. Baseline clinical and CMR imaging characteristics by presence or absence of MACE

Variables	MACE (n=18)	no MACE (n=58)	P
Age (years)	55.6±8.7	55.2±11.3	0.627
Male (n, %)	17（94.4）	50（86.2）	0.598
BMI (kg/m^2)	25.6±2.9	24.66±3.09	0.244
Smoking (n, %)	11（61.1）	39（67.2）	0.229
Diabetes mellitus (n, %)	3（16.7）	9（15.5）	0.999
Hypertension (n, %)	11（61.1）	27（46.6）	0.281
Systolic blood pressure (mmHg)	140±24	134±17	0.254
Heart rate (beats/min)	77.5±10.3	73.2±10.3	0.125
LDL-C (mmol/L)	2.91±0.75	2.69±0.83	0.331
HDL-C (mmol/L)	1.03±0.26	1.06±0.24	0.561
Peak BNP (pg/ml)	2309.5±1610.9	1185.7±1157.6	0.002
Peak cTNI (ng/ml)	12.7 (4.81, 24.51)	7.45 (2.82,13.35)	<0.001
Multivessel coronary disease (n, %)	5（27.8）	16（27.6）	0.987
Culprit lesion on CAG LAD (n, %)	12（66.7）	31（53.4）	0.323
Hospital stay time (days)	12±4	9±3	0.011
TIMI flow frame count (frames)	34（24-46）	27（20-36）	0.113
TIMI Myocardial Perfusion Classification ≤ level 2 (n, %)	3（16.7）	2（3.4）	0.152
Symptom to balloon time (minutes)	442.7±255.7	315.7±298.6	0.022
With Collateral circulation (n, %)	2(11.1%)	9(15.5%)	0.936
LVEDV (ml)	141.2±22.5	128.7±26.4	0.034
LVESV (ml)	76.1 ±15.6	61.9±18.2	0.002
LVEF (%)	46.1±6.9	52.3±9.0	0.009
IMH	6（33.3%）	9（15.5%）	0.020
MVO	13（72.2%）	23（39.7%）	0.002
IMH+ MVO	6（33.3%）	9（15.5%）	0.020

Edema/LV (%)	34.50±8.76	30.40±8.79	0.094
Salvage/LV (%)	16.52±11.41	18.36±8.61	0.532
IM%LV (%)	18.0±9.9	12.0±6.6	0.027
GLS (%)	-11.44±5.07	-16.48±4.8	<0.001
GCS (%)	-20.6±5.1	-24.9±6.2	0.008
GRS (%)	25.7 (20.2-32.6)	34.7 (25.8-39.4)	0.015

IM%LV: infarcted myocardial mass/left ventricular mass; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; BNP: brain natriuretic peptide; cTNI: cardiac troponin I; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; LVEF: left ventricular ejection fraction; IMH: intramyocardial hemorrhage; MVO: microvascular obstruction; GLS: global longitudinal strain; GCS: global circumferential strain; GRS: global radial strain.

Table 4. Correlation of clinical and CMR variables with MACE according to the logistic regression model using uni- and multivariate analyses

Variables	Univariate Analysis	Multivariate Stepwise Analysis
	OR (95%CI) P value	OR (95%CI) P value
Age, per 1 year	1.01 (0.96, 1.06) 0.6217	-
Male vs Female	2.72 (0.32, 23.36) 0.3618	8.30 (0.74, 93.43) 0.0868
Peak BNP, per 100 pg/ml	1.04 (1.00, 1.08) 0.0450	-
Peak cTNI, per 100 ng/ml	1.02 (0.99, 1.04) 0.1723	-
Symptom to balloon time, per 30 minutes	1.04 (0.99, 1.09) 0.1246	-
LVEF, per 1%	0.91 (0.85, 0.98) 0.0137	-
MVO, yes vs no	3.68 (1.16, 11.71) 0.0271	-
IM%LV, per 1%	1.10 (1.02, 1.18) 0.0102	-
GLS, per 1%	1.16 (1.05, 1.28) 0.0040	1.21 (1.07, 1.36) 0.0016
GCS, per 1%	1.14 (1.03, 1.27) 0.0122	-
GRS, per 1%	0.93 (0.85, 1.03) 0.1554	-

BNP: brain natriuretic peptide; cTNI: cardiac troponin I; LVEF: left ventricular ejection fraction; MVO: microvascular obstruction; IM%LV: infarcted myocardial mass/left ventricular mass; GLS: global longitudinal strain; GCS: global circumferential strain; GRS: global radial strain.

Table 5. Cardiac characteristics at baseline and after 6 months of follow-up

Variables	baseline (n=24)	6 months (n=24)	P
LVEDV (ml)	145.2±21.23	152.6±26.07	<0.001
LVESV (ml)	77.22±16.59	74.41±24.52	<0.001
LVEF (%)	46.85±7.74	52.03±9.90	0.002
IM%LV (%)	17.2 (10.6, 26.7)	12.6 (6.4, 21.7)	<0.001
GLS (%)	-13.22±4.89	-16.16±5.84	0.002
GCS (%)	-22.88±5.74	-23.21±6.92	0.007
GRS (%)	29.91±9.69	33.40±9.14	0.071

LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; LVEF: left ventricular ejection fraction; IM%LV: infarcted myocardial mass/left ventricular mass; GLS: global longitudinal strain; GCS: global circumferential strain; GRS: global radial strain.

Table 6. Baseline clinical and CMR imaging characteristics by presence or absence of LV remodeling

CMR parameters	LV remodeling (n=7)	no LV remodeling (n=17)	P
Age (years)	58±8	52±9	0.152
Male (n, %)	6(85.7)	15（88.2）	0.999
BMI (kg/m^2)	24.4±1.9	25.8±2.6	0.124
Smoking (n, %)	4（57.1）	11(64.7)	0.999
Diabetes mellitus (n, %)	1(14.3)	2(11.8)	0.999
Hypertension (n, %)	4(57.1)	11(64.7)	0.999
Systolic blood pressure (mmHg)	141.0±21	135.0±24	0.614
Heart rate (beats/min)	74±8	77±12	0.738
LDL-C (mmol/L)	2.90±0.9	2.83±0.9	0.844
HDL-C (mmol/L)	1.2±0.1	1.0±0.2	0.113
Peak BNP (pg/ml)	1862.0±1359.4	772.5±530.4	0.080
Peak cTNI (ng/ml)	1934 (564.1, 3666.0)	480 (49.93, 2559.5)	<0.001
Multivessel coronary disease (n, %)	6(85.7)	7(41.2)	0.124
Culprit lesion on CAG LAD (n, %)	6(85.7)	7(41.2)	0.124
Hospital stay time (days)	10.0±2.2	10.2±2.5	0.833
TIMI flow frame count (frames)	35.9±6.5	30.6±12.5	0.251
TIMI Myocardial Perfusion Classification ≤ level 2 (n, %)	2(28.5)	0(0.0)	0.076
Symptom to balloon time (minutes)	412.9±156.1	185.1±44.9	0.029
With Collateral circulation (n, %)	0 (0%)	2 (11.8%)	0.892
LVEDV (ml)	141.1±13.7	146.8±23.8	0.564
LVESV (ml)	82.7±11.3	75.0±18.1	0.308
LVEF (%)	41.6±3.4	49.0±8.0	0.004
IMH	0 (0%)	4 (23.5%)	0.283
MVO	7 (100%)	11 (64.7%)	0.130
IMH+MVO	0 (0%)	4 (23.5%)	0.283
Edema/LV (%)	33.42±9.86	33.87±8.90	0.916

Salvage/LV (%)	12.51±7.00	20.13±10.84	0.102
IM(%LV) (%)	26.8 (22.5, 30.0)	11.9 (9.7, 20.2)	0.004
GLS (%)	-7.9±4.1	-15.4±3.26	<0.001
GRS (%)	28.7±10.7	30.4±9.6	0.707
GCS (%)	-19.6±3.2	-24.2±6.1	0.071

IM%LV: infarcted myocardial mass/left ventricular mass; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; BNP: brain natriuretic peptide; cTNI: cardiac troponin I; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; LVEF: left ventricular ejection fraction; IMH: intramyocardial hemorrhage; MVO: microvascular obstruction; GLS: global longitudinal strain; GCS: global circumferential strain; GRS: global radial strain.

Table 7. Correlation of clinical and CMR variables with LV remodeling according to the logistic regression model using uni- and multivariate analyses

Variables	Univariate Analysis	Multivariate Stepwise Analysis
	OR (95%CI) P value	OR (95%CI) P value
Age, per 1 year	1.08 (0.97, 1.19) 0.1566	-
Male vs Female	0.16 (0.01, 2.11) 0.1620	-
Peak cTNI, per 100 ng/ml	1.02 (0.97, 1.07) 0.4175	1.09 (1.00, 1.20) 0.0630
Symptom to balloon time, per 30 minutes	1.18 (1.02, 1.37) 0.0220	-
LVEF, per 1%	0.92 (0.81, 1.04) 0.1983	-
IM%LV, per 1%	1.09 (0.98, 1.21) 0.1159	-
GLS, per 1%	1.65 (1.11, 2.45) 0.0127	2.06 (1.14, 3.73) 0.0171

IM%LV: infarcted myocardial mass/left ventricular mass; cTNI: cardiac troponin I; LVEF: left ventricular ejection fraction; GLS: global longitudinal strain.

Figures

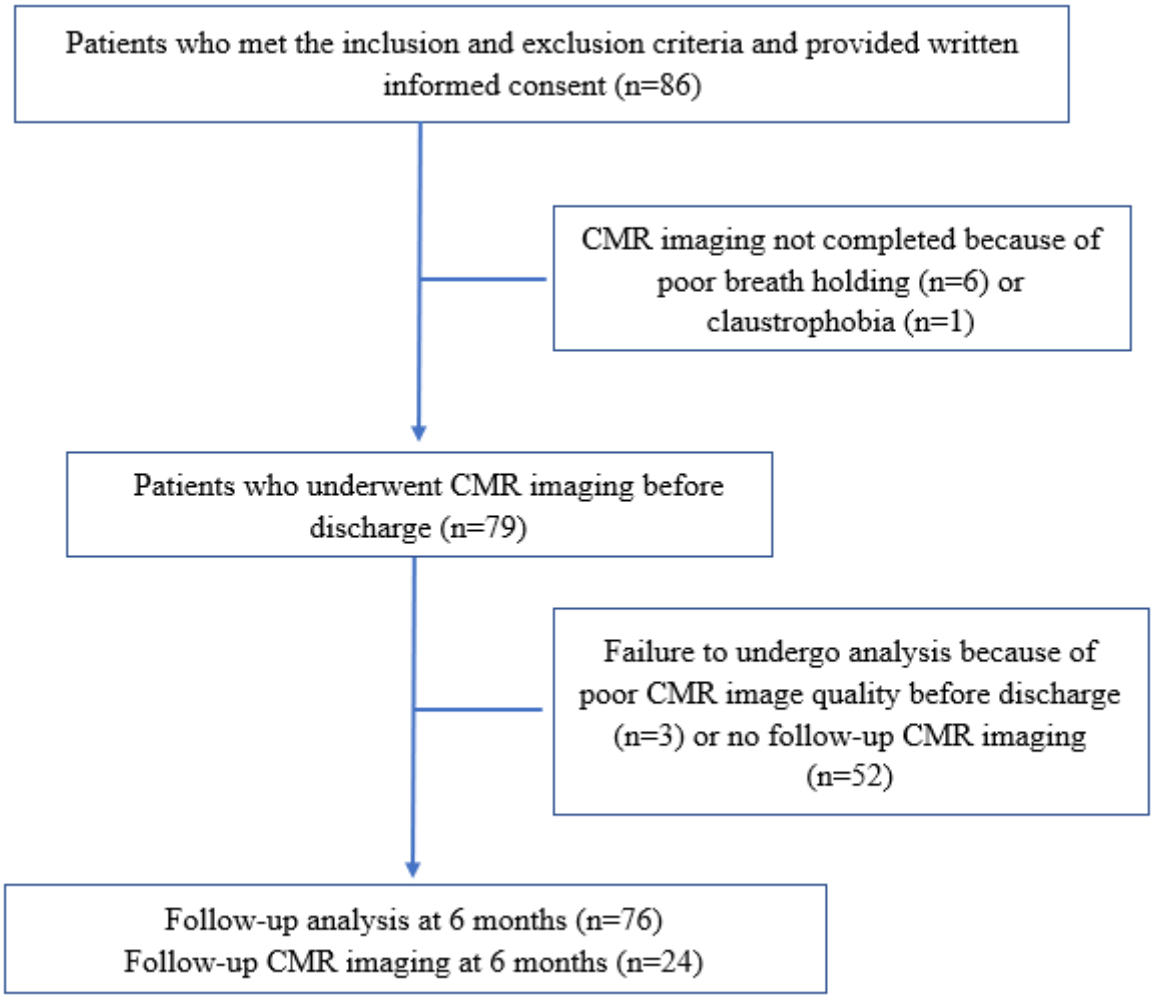

Figure 1

Flowchart of the study. CMR: cardiac magnetic resonance

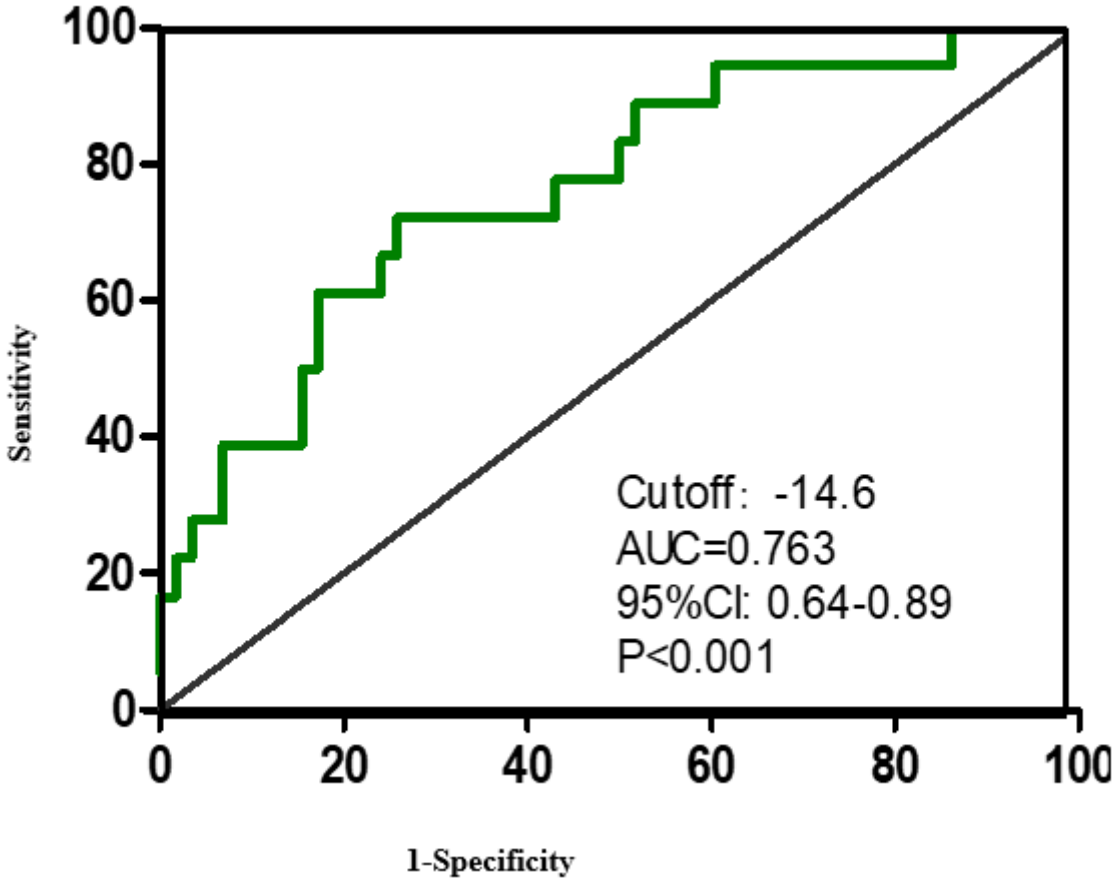

Figure 2

Receiver operating-characteristic curve for the prediction of major adverse cardiac events within 6 months after acute ST-segment elevation myocardial infarction, using the global longitudinal strain as the independent variable.