Original Article

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Relationship of Microvascular Obstruction with Global and Regional Myocardial Function Determined by Cardiac Magnetic Resonance after ST-Segment Elevation Myocardial Infarction

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ABSTRACT

Objective To investigate the impact of microvascular obstruction (MVO) on the global and regional myocardial function by cardiac magnetic resonance feature-tracking (CMR-FT) in ST-segment-elevation myocardial infarction (STEMI) patients after percutaneous coronary intervention.

Methods Consecutive acute STEMI patients who underwent cardiac magnetic resonance imaging 1 - 7 days after successful reperfusion by percutaneous coronary intervention treatment were included in this retrospective study. Based on the presence or absence of MVO on late gadolinium enhancement images, patients were divided into groups with MVO and without MVO. The infarct zone, adjacent zone, and remote zone were determined based on a myocardial 16-segment model. The radial strain (RS), circumferential strain (CS), and longitudinal strain (LS) of the global left ventricle (LV) and the infarct, adjacent, and remote zones were measured by CMR-FT from cine images and compared between patients with and without MVO using independent-samples *t*-test. Logistic regression analysis was used to assess the association of MVO with the impaired LV function.

Results A total of 157 STEMI patients (mean age 56.66 ± 11.38 years) were enrolled. MVO was detected in 37.58% (59/157) of STEMI patients, and the mean size of MVO was 3.00 ±3.76 mL. Compared with patients without MVO (n =98), the MVO group had significantly reduced LV global RS (t = −4.30, P < 0.001), global CS (t = 4.99, P < 0.001), and global LS (t = 3.51, P = 0.001). The RS and CS of the infarct zone in patients with MVO were significantly reduced (t = −3.38, P = 0.001; t = 2.64, P = 0.01; respectively) and the infarct size was significantly larger (t = 8.37, P < 0.001) than that of patients without MVO. The presence of LV MVO [*OR*= 4.10, 95%*CI*: 2.05 - 8.19, *P*<0.001) and its size [*OR*=1.38, 95%*CI*: 1.10-1.72, *P*=0.01], along with the heart rate and LV infarct size were significantly associated with impaired LV global CS in univariable Logistic regression analysis, while only heart rate (*OR*=1.08, 95%*CI*: 1.03 - 1.13, *P*=0.001) and LV infarct size (*OR*=1.10, 95%*CI*: 1.03 - 1.16, *P*=0.003) were independent influencing factors for the impaired LV global CS in multivariable Logistic regression analysis.

Conclusion The infarct size was larger in STEMI patients with MVO, and MVO deteriorates the global and regional LV myocardial function.

Key words: cardiac magnetic resonance feature tracking; ST-segment elevation myocardial infarction; microvascular obstruction; myocardial strain; Myocardial function

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INTRODUCTION

Percutaneous coronary intervention (PCI) combined with antithrombotic therapy is the recommended treatment for acute ST-segment elevation myocardial infarction (STEMI)[1]. However, perfusion of the ischemic myocardium is not restored in up to 30% of patients due to microvascular obstruction (MVO) even if the infarct-related artery was successfully recanalized[2]. MVO contributes to an important mechanism of reperfusion injury after STEMI and is associated with adverse ventricular remodeling, diminished recovery of left ventricular (LV) function, and major adverse cardiac events (MACE)[2,3].

LV ejection fraction (LVEF), a routine parameter describing LV function, has major limitations due to its inability to detect subtle changes in cardiac function. Strain, the myocardial deformation parameter, reflects not only the different spatial components of contractile function in radial strain (RS), circumferential strain (CS), or longitudinal strain (LS) but also global and regional conditions[4]. Speckle tracking echocardiography is the most widely used technique to quantify myocardial deformation, but it has some limitations such as poor spatial resolution and considerable inter-operator variability[4]. Cardiac magnetic resonance (CMR) imaging can evaluate subtle changes in myocardial deformation accurately and objectively[5,6]. However, CMR tissue tagging technique is challenging to be applied in patients with acute STEMI due to its long scan time, holding breath, and complex postprocessing[7]. Recently, CMR feature tracking (CMR-FT), a novel postprocessing method, allows to measure myocardial strain by tracking tissue voxel motion in Steady State Free Procession cine images without using additional sequences to acquire images and demonstrates greater robustness and reproducibility[8]. Previous reports on myocardial strain in patients with STEMI mainly focused on either global LV strain[9,10] or in small single-center studies with CMR tagging technique[11-13]. Although there were some reports on the impact of MVO on contractile function of the infarct zone, the results were conflicting[7,14]. In this study, we investigated the MVO impact on global and regional function in radial, circumferential and longitudinal directions by CMR-FT technique in a large group of patients with revascularized STEMI. Furthermore, we used regression analysis to explore the impact of MVO and other factors on LV myocardial function in STEMI patients.

PATIENTS AND METHODS

Patient population

The study protocol was approved by a local ethics committee (S2021-126-02). The present retrospective study included consecutive acute STEMI patients who underwent CMR imaging 1 to 7 days after successful PCI treatment within 12 h after onset of symptoms from January 2013 to June 2020. STEMI was diagnosed according to the European Society of Cardiology/American College of Cardiology (ESC/ACC) committee criteria[15]. The exclusion criteria were: patients with previous myocardial infarction (MI), previous revascularization procedure (coronary artery bypass grafts or PCI), unsuccessful PCI, (supra-)ventricular arrhythmias, or poor image quality.

CMR image acquisition

CMR was performed on a clinical 1.5-Tesla (Multiva, Philips, Netherlands) scanner using a 8-element-body phased-array coil system. Balanced turbo field echo (BTFE) cine imaging sequences were acquired in standard views, including LV 2-, 3- and 4-chamber orientation and contiguous short-axis slices covering the whole left ventricle. Typical imaging parameters of cine imaging included: cardiac phases 25, echo time 1.87 ms, repetition time 3.7 ms, slice thickness 8 mm, flip angle 60°, and typical in-plane resolution 1.40 mm × 1.44 mm. Late gadolinium enhancement (LGE) images were acquired 10–15 minutes after intravenous injection of gadolinium contrast agent (Gadopentetate Dimeglumine, BeiLu, Beijing, China) with breath-hold phase-sensitive segmented inversion recovery (PSIR) fast field echo sequence. Typical imaging parameters of LGE imaging included: repetition time 6.2 ms, echo time 3 ms, slice thickness 8 mm, flip angle 25°, and typical in-plane resolution 1.60 mm × 1.65 mm.

CMR image analysis

A commercial software (cvi42 version 5.12.1, Circle Cardiovascular Imaging, Calgary, Alberta, Canada) was used to analyze CMR parameters. According to the American Heart Association (AHA) segmentation recommendation[16], the infarct, adjacent, and remote zones of LV myocardium were defined based on a myocardial 16-segment model (excluding apical segments) on the short-axis LGE images. The segments with hyperenhancement were identified as infarct zone.

Accordingly, adjacent zone was defined as the myocardial segment next to the infarct zone without hyperenhancement, and the rest of LV myocardium was defined as the remote zone (**Fig. 1A**). For example, if the infarct zone included the segments 1, 2, 7, 8, 13, 14, 15 and 16, the adjacent zone was the segments 3, 6, 9, 10, 11 and 12, and the rest of LV myocardium was defined as the remote zone (**Fig. 1B**).

On the infarct zone, infarct size was calculated with a semi-automated algorithm by signal intensity five or more standard deviations above the mean signal intensity of the normal myocardium and is expressed as percentage of LV myocardial volume, as previously reported[17]. MVO was identified as a hypointense area within infarct zone, the contour of which was manually traced to obtain the MVO size[18] (**Fig. 2A**).

For cine CMR, LV structural and functional parameters were obtained by automated feature-tracking post-processing method. The cardiac phases of end-systole and end-diastole were obtained by automatically detecting the smallest and the largest cavity size of left ventricule in contiguous short-axis slices and 2-, 3- and 4-chamber orientation cine images. Then, the LV endocardial and epicardial borders were respectively automatic delineated on the end-systolic and end-diastolic phases. The LV papillary muscles were included in the ventricular cavity. Finally, all borders of outlined myocardium were visually reviewed and manually adjusted by point-and-click approach by a radiologist with 3 years of experience in CMR and blinded with clinical data. Thus, the LVEF, end-diastolic volume index (EDVI), end-systolic volume index (ESVI), LV mass index (LVMI), wall thickening rate (WTR), and global peak strain were calculated according to the current guidelines[19,20].

The peak strain in three directions (radial, circumferential, and longitudinal) is the absolute maximum value of heart muscle deformation in shape and dimension over the entire cardiac cycle. The peak strains in three directions were obtained according to the LV 16-segment model (**Fig. 2**). Radial strain (RS) and circumferential strain (CS) values were calculated on the short-axis slices, while longitudinal strain (LS) values were obtained on the three long-axis slices. We investigated global strain difference in STEMI patients with MVO and without MVO. Furthermore, LV myocardium strains within the infarct, adjacent, and remote zones in patients with STEMI stratified by presence of MVO were also assessed. Finally, we explored whether MVO and other factors affected the LV function.

Intra- and inter-observer variability

Intra- and inter-observer variabilities for strains of LV global and infarct zone in three directions (radial, circumferential, and longitudinal) were assessed in 20 randomly selected patients with STEMI by using intraclass correlation coefficient (ICC). For intra-observer variability analysis, one radiologist performed peak strains for the second time after a 3-month interval. Inter-observer variability was analyzed by another radiologist who was blinded to the results of the first radiologist.

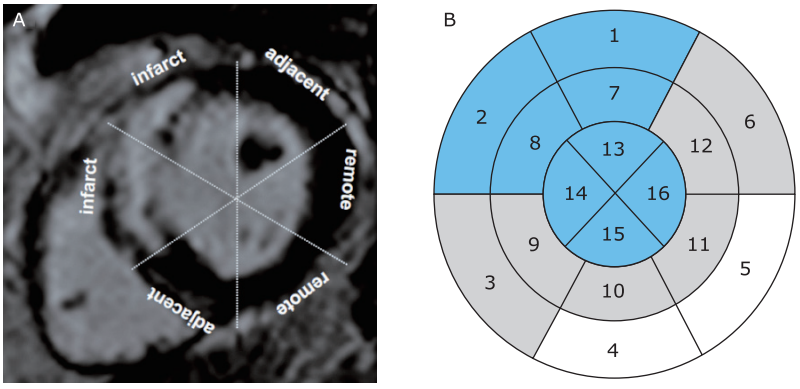

Figure 1. The division of the infarct, adjacent and remote segments of left ventricle.

(A) Definition of the infarct, adjacent and remote zone of myocardium on a representative short-axis late gadolinium enhancement image of a patient with anterior myocardial infarction. (B) Schematic diagram of the infarct, adjacent and remote zones based on a myocardial 16-segment model recommended by the American Heart Association (AHA). If the infarct zone includes the segments 1, 2, 7, 8, 13, 14, 15 and 16, the adjacent zone includes the segments 3, 6, 9, 10, 11 and 12, and the rest of myocardium is defined as the remote zone.

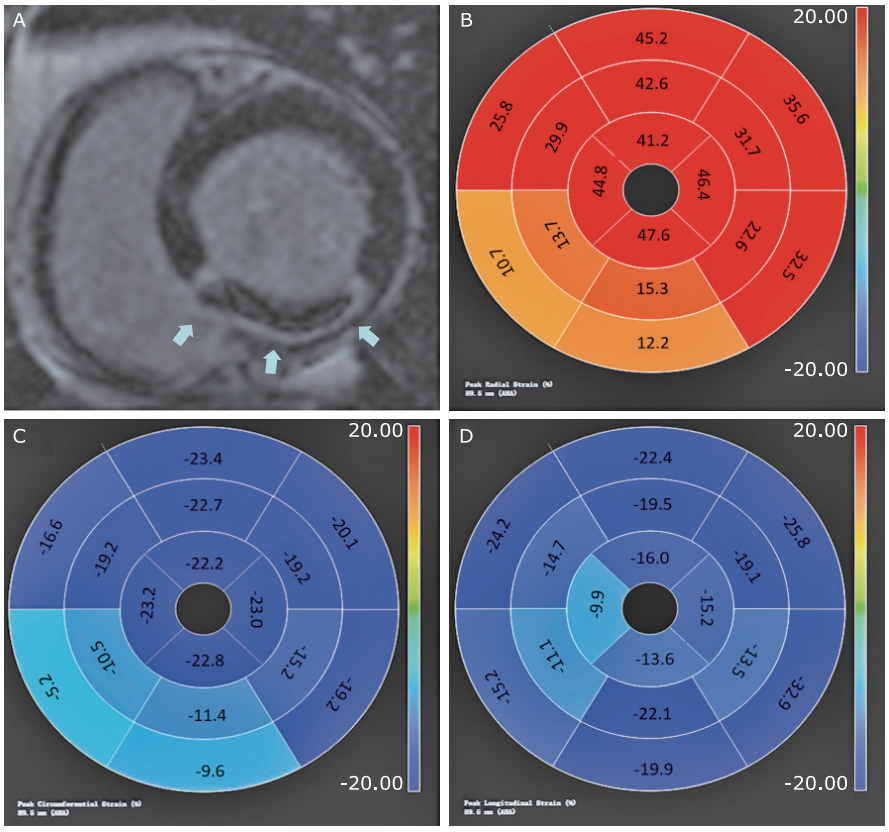

Figure 2. Schematic diagram of myocardial strain assessment.

(A) A typical example of short axis image of inferior myocardial infarction with MVO scanned by LGE imaging (arrows). (B) Representative bull's eye map for radial strain assessment. (C) Representative bull's eye map for circumferential strain assessment. (D) Representative bull's eye map for longitudinal strain assessment.

MVO: microvascular obstruction; LGE: late gadolinium enhancement.

Statistical analysis

All variables were assessed for normal distribution using the Kolmogorov–Smirnoff test. Continuous normally distributed variables are expressed as mean ± standard deviation and comparisons between two groups were performed with independent-samples *t*-test. Categorical variables were displayed as numbers or percentages and compared either by the Chi-square or Fisher exact test.

Moreover, using univariate and multivariate Logistic regression analysis, we studied the possible factors associated with the reduced LV function. All statistical analyses were performed using IBM SPSS Statistics software (version 22.0, Chicago, IL, USA) and two tailed *P*-value < 0.05 was considered statistically significant.

RESULTS

Characteristics of the study population

A total of 216 patients with STEMI were recruited, while 59 were excluded due to the following reasons: previous MI and revascularization procedure (n = 35), unsuccessful PCI (n = 4), (supra-)ventricular arrhythmias (n = 1), and poor image quality (n = 19). Finally, LV strain analysis was performed in 157 STEMI patients. The flowchart of patient enrollment is provided in **Fig. 3**.

MVO was present in 37.58% (59/157) of patients. MVO mean size was 3.00 ± 3.76 mL in patients with MVO. Demographic and clinical characteristics of patients are listed in **Table 1**. No significant differences were found in family history of coronary artery disease, hypertension, hypercholesterolemia, diabetes mellitus, and smoking between STEMI patients with and without MVO. STEMI patients with MVO were younger than those without MVO (t = -2.19, P = 0.03). The maximum cardiac creatine kinase MB (CK-MB) (t = 5.54, P < 0.001) and peak troponin (t = 5.93, P < 0.001) were significantly greater in STEMI patients with MVO than those in STEMI patients without MVO.

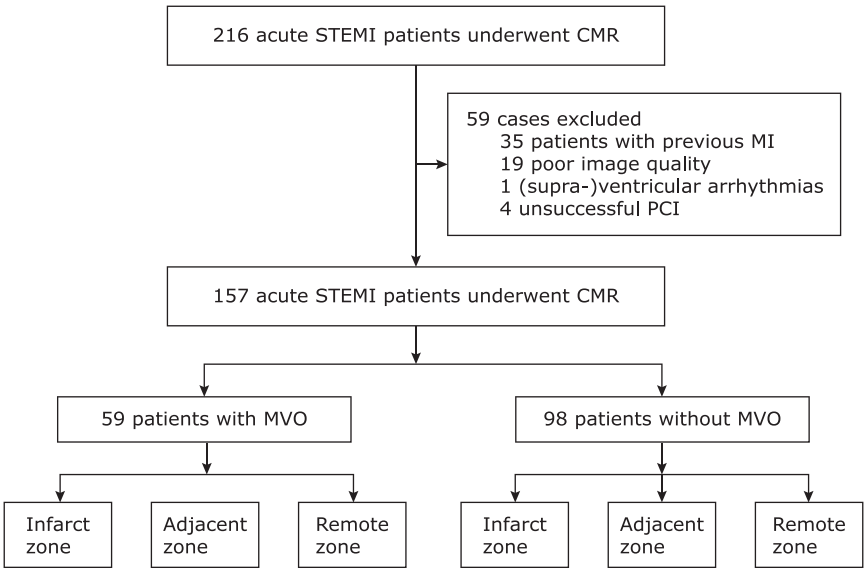

Figure 3. Flowchart of the patient enrollment.

STEMI: ST-segment-elevation myocardial infarction; CMR: cardiac magnetic resonance; MI: myocardial infarction; PCI: percutaneous coronary intervention.

Comparisons of LV global and regional functions between STEMI patients with and without MVO

As shown in **Table 2**, the patients with MVO exhibited higher values of LV ESVI (t = 2.94, P = 0.004), myocardial mass index (t = 2.04, P = 0.04), and LV infarct size (t = 8.37, P < 0.001) than those without MVO. Compared to the patients without MVO, LVEF (t = -3.41, P = 0.001) and LV global WTR (t = -2.95, P = 0.004) were significantly decreased in the patients with MVO.

Patients with MVO had reduced global RS (GRS) (t=−4.30, P < 0.001), global CS (GCS) (t=4.99, P <0.001), and global LS (GLS) (t=3.51, P =0.001) compared with patients without MVO. In addition, the infarct zone of STEMI patients with MVO had lower RS (t=−3.38, P = 0.001) and CS (t=2.64, P = 0.01) compared with those patients without MVO. Although there was no statistical difference, the LS of infarct zone in patients with MVO was also lower (−7.13% ± 3.65% *vs*. −7.47% ± 4.93%; P = 0.65). For adjacent and remote zones, no significant differences were found on peak strain between the patients with MVO and without MVO in three directions (all P > 0.05).

The association of MVO and other factors with the reduced LV function

By using univariate Logistic regression analysis on baseline clinical characteristics and CMR parameters, we found that heart rate [*OR* = 1.06, 95%*CI*: 1.02-1.09, *P*=0.001], the culprit lesion in the left anterior descending artery [*OR* = 3.25, 95%*CI*: 1.69-6.27, *P*<0.001], N-terminal pro b-type natriuretic peptide [*OR* = 1.00, 95%*CI*: 1.00-1.00, *P*=0.01], CK-MB [*OR* = 1.01, 95%*CI*: 1.00-1.01, *P*<0.001], peak troponin [*OR* = 1.12, 95%*CI*: 1.06-1.20], LV ESVI [*OR* = 1.08, 95%*CI*: 1.05-1.12, *P*=0.001], LV myocardial mass index [*OR* = 1.08, 95%*CI*: 1.04-1.12,*P*=0.001], LV infarct size [*OR* = 1.11, 95%*CI*: 1.07-1.15, *P*=0.001], the presence of LV MVO [*OR* = 4.10, 95%*CI*: 2.05-8.19, *P*<0.001], and LV MVO size [*OR* = 1.38, 95%*CI*: 1.10-1.72, *P*=0.01] were associated with impaired LV GCS (**Table 3**).

Based on the univariate analysis, the covariates (P < 0.1) were selected for further multivariate analysis. In a multivariate model (**Table 3**) including male gender, smoking, drinking, heart rate, the culprit lesion in the left anterior descending artery, N-terminal pro b-type natriuretic peptide, CK-MB, peak troponin, LV ESVI, LV myocardial mass index, LV infarct size, the presence of LV MVO, and LV MVO size, heart rate (*OR* = 1.08, 95%*CI*: 1.03-1.13, *P*=0.001) and LV infarct size (*OR* = 1.10, 95%*CI*: 1.03-1.16, *P*=0.003) were independently significantly associated with impaired LV GCS.

Reproducibility

Both intra- and inter-observer reproducibility of GRS (*ICC*: 0.98 and 0.83), GCS (*ICC*: 0.97 and 0.87), and

Table 1. Baseline characteristics of the STEMI patients with and without microvascular obstruction (*n* = 157)

Items	All patients (*n* = 157)	Patient with MVO (*n* = 59)	Patient without MVO (*n* = 98)	*P* value
Demographic characteristics				
Age (yrs, mean ± SD)	56.66 ± 11.38	54.12 ± 11.63	58.18 ± 11.01	0.03
Gender [male *n* (%)]	136 (86.6)	53 (89.8)	83 (84.7)	0.36
Body mass index [kg/m^2, mean ± SD]	25.68 ± 3.43	25.76 ± 3.20	25.63 ± 3.57	0.49
Clinical parameters				
Family history of CAD [*n* (%)]	15 (9.6)	5 (8.5)	10 (10.2)	0.72
Hypertension [*n* (%)]	79 (50.3)	28 (47.5)	51 (52.0)	0.58
Hypercholesterolemia [*n* (%)]	33 (21.0)	17 (28.8)	16 (16.3)	0.63
Diabetes mellitus [*n* (%)]	23 (14.6)	11 (18.6)	12 (12.2)	0.27
Smoking [*n* (%)]	103 (65.6)	42 (71.2)	61 (62.2)	0.25
Drinking [*n* (%)]	37 (23.6)	13 (22.0)	24 (24.5)	0.73
Heart rate (beats/min, mean ± SD)	71.29 ± 11.61	73.59 ± 11.97	69.91 ± 11.22	0.054
Killip class > Ⅰ [*n* (%)]	16 (10.2)	7 (11.9)	9 (9.2)	0.59
Multivessel disease [*n* (%)]	101 (64.3)	36 (60.1)	65 (66.3)	0.501
Infarct related artery [*n* (%)]				0.52
Left anterior descending artery	69 (43.94)	28 (47.46)	41 (41.84)	
Right coronary artery	70 (44.59)	22 (37.29)	48 (48.98)	
Left circumflex artery	18 (11.46)	6 (10.17)	12 (12.24)	
TIMI flow pre PCI < 3 [*n* (%)]	148 (94.3)	58 (98.3)	90 (91.8)	0.06
TIMI flow post PCI < 3 [*n* (%)]	2 (1.3)	1 (1.7)	1 (1.0)	0.72
Blood test results				
NT-proBNP (pg/mL, mean ± SD)	1,616.01 ± 1,552.90	1,838.72 ± 2,118.44	1,481.92 ± 1,070.77	0.23
Maximum CK-MB (U/L, mean ± SD)	233.28 ± 163.89	317.34 ± 173.51	182.67 ± 135.31	<0.001
Peak troponin (mg/L, mean ± SD)	7.85 ± 7.84	12.65 ± 9.13	4.95 ± 5.15	<0.001
Medication [*n* (%)]				
Aspirin	156 (99.4)	58 (98.3)	98 (100)	0.16
Statin	156 (99.4)	59 (100)	97 (99.0)	0.33
ACEI/ARB	70 (44.6)	21 (35.6)	49 (50.0)	0.08
Beta-blockers	135 (86.0)	50 (84.7)	85 (86.7)	0.73
Diuretic	76 (48.4)	31 (52.5)	45 (45.9)	0.42

CAD: coronary artery disease; TIMI: thrombolysis in myocardial infarction; NT-proBNP: N-terminal pro b-type natriuretic peptide; CK-MB: creatine kinease MB; ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor blocker.

GLS (*ICC*: 0.94 and 0.93) were excellent. Similarly, intra- and inter-observer agreement of RS (*ICC*: 0.91 and 0.81), CS (*ICC*: 0.89 and 0.84), and LS (*ICC*: 0.85 and 0.77) of infarct zone were good.

DISCUSSION

This study comprehensively assessed clinical features, the LV global and regional function, and tissue characteristics by CMR in a large population of patients with STEMI. Our main findings can be summarized as follows: first, compared with patients without MVO, CK-MB and peak troponin were significantly elevated in the STEMI patients with MVO; second, LV ESVI, LV myocardial mass index, and LV infarct size were larger in the STEMI patients with MVO than those without MVO; third, LV global function such as LVEF, WTR, and global strain in three directions were significantly reduced in STEMI patients with MVO; fourth, the peak strains of infarct zone were significantly reduced in STEMI patients with MVO compared with patients without MVO, although the peak strains of adjacent and remote zone were not significantly reduced; and fifth, heart rate and LV infarct size demonstrated obvious associations with the reduced LV function.

Previous study showed that MVO would confer

Table 2. Comparisons of LV global and regional functions between STEMI patients with and without MVO (mean ± SD)

Items	All Patients (n = 157)	Patient with MVO (n = 59)	Patient without MVO (n = 98)	t value	P value*
LV EDVI (mL/m^2)	80.81 ± 18.94	83.31 ± 16.07	79.30 ± 20.40	1.29	0.20
LV ESVI (mL/m^2)	43.44 ± 13.40	47.39 ± 13.04	41.06 ± 13.11	2.94	0.004
LV myocardial mass index (g/m^2)	59.83 ± 11.61	62.24 ± 9.42	58.37 ± 12.58	2.04	0.04
LV infarct size (mL)	24.96 ± 16.18	36.55 ± 16.55	17.97 ± 11.23	8.37	<0.001
LVEF (%)	46.14 ± 9.23	43.01 ± 9.45	48.02 ± 8.61	-3.41	0.001
LV global WTR (%)	44.25 ± 13.64	40.21 ± 11.62	46.68 ± 14.23	-2.95	0.004
LV GRS (%)	22.70 ± 5.84	20.25 ± 4.75	24.17 ± 5.96	-4.30	<0.001
LV GCS (%)	-14.19 ± 2.80	-12.85 ± 2.52	-14.99 ± 2.66	4.99	<0.001
LV GLS (%)	-11.95 ± 3.28	-10.80 ± 3.39	-12.64 ± 3.03	3.51	0.001
Infarct zone RS (%)	14.46 ± 7.77	12.05 ± 5.83	15.92 ± 8.42	-3.38	0.001
Infarct zone CS (%)	-9.99 ± 5.49	-8.53 ± 4.83	-10.87 ± 5.70	2.64	0.01
Infarct zone LS (%)	-7.34 ± 4.48	-7.13 ± 3.65	-7.47 ± 4.93	0.46	0.65
Adjacent zone RS (%)	22.52 ± 7.59	22.90 ± 7.07	22.29 ± 7.92	0.49	0.63
Adjacent zone CS (%)	-14.71 ± 3.93	-14.42 ± 3.48	-14.89 ± 4.18	0.73	0.47
Adjacent zone LS (%)	-7.25 ± 4.18	-6.50 ± 3.46	-7.69 ± 4.51	1.75	0.08
Remote zone RS (%)	30.31 ± 9.85	31.94 ± 9.84	29.33 ± 9.78	1.62	0.11
Remote zone CS (%)	-17.30 ± 4.39	-17.20 ± 3.99	-17.36 ± 4.63	0.22	0.83
Remote zone LS (%)	-5.15 ± 9.67	-3.60 ± 8.98	-6.09 ± 9.99	1.57	0.12

LV: left ventricular; EDVI: end-diastolic volume index; ESVI: end-systolic volume index; LVEF: left ventricular ejection fraction; WTR: wall thickening rate; GRS: global radial strain; GCS: global circumferential strain; GLS: global longitudinal strain; RS: radial strain; CS: circumferential strain; LS: longitudinal strain.

*Compared between the patients with MVO and without MVO.

Table 3. Univariate and multivariate Logistic regression analysis of the factors associated with impaired LV GCS

Items	Univariate Logistic regression		Multivariate Logistic regression	
	P value	OR [95%CI]	P value	OR [95%CI]
Age (yrs)	0.81	1.00 (0.97 - 1.03)		
Gender (Male)	0.06	2.65 (0.97 - 7.24)		
Body mass index (kg/m^2)	0.27	1.05 (0.96 - 1.16)		
Family history of CAD	0.15	2.30 (0.75 - 7.08)		
Hypertension	0.23	1.47 (0.78 - 2.76)		
Hypercholesterolemia	0.25	0.63 (0.29 - 1.38)		
Diabetes mellitus	0.70	1.19 (0.49 - 2.89)		
Smoking	0.09	1.80 (0.92 - 3.52)		
Drinking	0.06	2.08 (0.98 - 4.42)		
Killip class > I	0.24	1.89 (0.65 - 5.49)		
Heart rate	0.001	1.06 (1.02 - 1.09)	0.001	1.08 (1.03 - 1.13)
Culprit lesion in the LAD (%)	<0.001	3.25 (1.69 - 6.27)		
NT-proBNP (pg/mL)	0.01	1.00 (1.00 - 1.00)		
Maximum CK-MB (U/L)	<0.001	1.01 (1.00 - 1.01)		
Peak troponin (mg/L)	<0.001	1.12 (1.06 - 1.20)		
LV EDVI	0.35	1.01 (0.99 - 1.03)		
LV ESVI	<0.001	1.08 (1.05 - 1.12)		
LV myocardial mass index (g/m^2)	<0.001	1.08 (1.04 - 1.12)		
LV infarct size (mL)	<0.001	1.11 (1.07 - 1.15)	0.003	1.10 (1.03 - 1.16)
Presence of LV MVO (%)	<0.001	4.10 (2.05 - 8.19)		
LV MVO size (mL)	0.01	1.38 (1.10 - 1.72)		

OR: odds ratio; 95%CI: 95% confidence interval.

harmful effect on the infarct myocardium directly after PCI[21]. In our study, the values of both CK-MB and peak troponin were higher in STEMI patients with MVO. Huang *et al*.[22] also found that peak troponin is a predictor for the presence of MVO. In addition, like previous research[23], our results showed that patients with MVO had larger LV ESVI and LV myocardial mass index and more impaired LVEF, WTR, and global peak strains. However, limited data reported the relationship between MVO and regional dysfunction in patients with STEMI in previous studies[24,25]. In the present study, we compared the strain characteristics of infarct zone, adjacent zone, and remote zone in patients with or without MVO. Our study found that patients with MVO had lower RS and CS in infarct zone than those without MVO, although, the two strains in adjacent and remote zones had no significant differences. Similarly, Podlesnikar *et al*.[23] also found that the infarct zone in patients with MVO had lower CS compared to those without MVO, and CS in the non-infarct zone had no significant differences between MVO and non-MVO groups. Therefore, we believe that although MVO does not affect the function of the adjacent and remote zones, the function of infarct zone is extremely reduced by MVO, which will worsen the global LV function of patients with MVO. In addition, as concluded by Pankaj *et al*.[26] found that MVO was associated with LV contraction, we also found that the presence of LV MVO [*OR* = 4.10, 95%*CI*: 2.05 - 8.19] and LV MVO size [*OR* = 1.38, 95%*CI*: 1.10 - 1.72] were associated with decreased LV function. In multivariate regression analysis, only heart rate and LV infarct size were associated with LV CS. As the faster heart rate is, the shorter ventricular diastolic period will be. This can lead to the decrease of ventricular filling volume and coronary blood flow, resulting in insufficient myocardial blood supply, and therefore affect LV function. In addition, our study suggested that the infarct size was an independent influencing factor of decreased LV CS. The STEMI patients with MVO exhibited larger LV infarct size, so we believe that the impact of MVO on LV function cannot be ignored.

CMR-FT is a relatively new imaging technique that can be used to measure myocardial deformation without additional acquisition and complex post-processing[27,28]. CMR-FT can accurately study cardiac mechanics by assessing global and regional myocardial deformation. Podlesnikar *et al*.[23] found that the infarct zone in patients with MVO had lower CS compared to those without MVO, however, the parameters of RS and LS were not mentioned. In the present study, we compared the strain characteristics between patients with MVO and patients without MVO in multiple directions, including LS, CS, and RS. Our study revealed that the patients with MVO had lower RS and CS in infarct zone than those without MVO; however, the parameter of LS in infarct zone showed no significant decrease. As is known that myocardial fibers of LV are divided into longitudinal fibers of endocardium, annular fibers of middle myocardium, and longitudinal fibers of epicardium, and there is a complex interaction of torsion along the three layers of myocardium[29]. CS mainly represents the contraction of midlayer fibers, while LS reflects that of the subendocardial layer. The mechanism of RS is a result of complex shearing between layers of myocytes[14].

There were several limitations in our study. Firstly, we did not assess myocardium strain in the infarct zone stratified by transmural extent, because the infarct segments of all patients were transmural in this study. Secondly, some patients in our study had two or three vessels diseased, so the impact of chronic, more mild ischemia in other coronary territories on myocardial strain of adjacent and remote segments was not fully considered.

In conclusion, strain parameters derived from CMR-FT may be new noninvasive imaging markers allowing comprehensive evaluation of global and regional myocardium deformation. STEMI patients with MVO have impaired LV global function and larger infarct size. In addition, MVO deteriorates the function of the infarct zone, although adjacent and remote zones will not be affected.

Conflict of interest

Yue XZ is a Philips employee and obtains personal fees from Philips Healthcare, which is outside the submitted work. Other authors have no competing interests.

Authors' contributions

Zhao YN: literature research and manuscript preparation; Cui JN: literature research and data analysis; XH Zhang: manuscript editing; Li JF: CMR images acquisition and data collection; Chen SM: statistical analysis; Yue XZ: study design and manuscript editing; Li T: guarantor of the entire study's integrity, study con-

cepts and design, and manuscript editing.

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论著

ST 段抬高型心肌梗死患者微血管阻塞与心脏磁共振测定的整体和局部心肌功能的关系

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摘要

目的 应用心脏磁共振特追踪技术（cardiac magnetic resonance feature-tracking，CMR-FT）探讨微血管阻塞（microvascular obstruction，MVO）对 ST 段抬高型心肌梗死（ST segment-elevation myocardial infarction，STEMI）患者整体和局部心功能的影响。

方法 回顾性连续纳入经皮冠状动脉介入术治疗成功后且 1~7 天进行心脏磁共振（cardiac magnetic resonance，CMR）检查的急性 STEMI 患者。依据延迟钆增强成像（late gadolinium enhancement，LGE）将患者分为 MVO 阳性组和 MVO 阴性组。参照左心室 16 节段模型，将左心室心肌分为梗死区、邻近区和遥远区。利用 CMR-FT，在电影图像上分别测量左心室整体以及梗死区、邻近区和遥远区的径向应变（radial strain，RS)、周向应变（circumferential strain，CS）及纵向应变（longitudinal strain，LS)，并且采用独立样本 t 检验比较 MVO 阳性和 MVO 阴性患者之间的差异。Logistic 回归分析用于探究 MVO 与 LV 功能受损的关联。

结果 在 157 名 STEMI 患者（平均年龄 56.66 ± 11.38 岁）中，发现 59 例 MVO 阳性患者（37.58%），且 MVO 阳性组平均 MVO 面积为 3.00±3.76mL。与 MVO 阴性患者（n=98）相比，MVO 阳性患者 LV 整体 RS（t = −4.30，P<0.001）、整体 CS（t = 4.99，P<0.001）、整体 LS（t=3.51，P = 0.001）显著降低。MVO 阳性患者梗死区 RS (t = −3.38, P=0.001) 和 CS (t = 2.64, P=0.01) 相较于 MVO 阴性的患者显著降低，梗死面积显著增大 (t = 8.37, P<0.001）。在单变量 Logistic 回归分析中，发生 LV MVO [OR= 4.10, 95%CI：2.05~8.19，P<0.001)、MVO 面积 [OR=1.38, 95%CI：1.10~1.72，P=0.01]、心率以及 LV 梗死面积与 CS 受损显著相关。然而，在多变量 Logistic 回归分析中只有心率（OR=1.08，95%CI：1.03~1.13，P=0.001）和 LV 梗死面积（OR=1.10，95% CI：1.03~1.16，P=0.003）是 LV 整体 CS 受损的独立影响因素。

结论 MVO 阳性的 STEMI 患者心肌梗死面积较大，并且 MVO 可使左心室整体和局部心功能恶化。

关键词：心脏磁共振特征追踪技术；ST 段型抬高型心肌梗死；微血管阻塞；心肌应变

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