Is there an association between hibernating myocardium and left ventricular mechanical dyssynchrony in patients with myocardial infarction?

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Abstract

Objective: Left ventricular mechanical dyssynchrony (LVMD) is an important factor in the prognosis of patients with myocardial infarction (MI). The aim of this study was to identify the correlation between LVMD and hibernating myocardium in MI patients by radionuclide myocardial imaging. **Subjects and Methods:** This study consisted of 91 patients who had a history of definite prior MI and underwent both technetium-99m methoxyisobutylisonitrile (^{99m}Tc-MIBI) gated single photon emission tomography (SPET) myocardial perfusion imaging (MPI) and fluorine-18 fluorodeoxyglucose (^{18}F-FDG) positron emission tomography (PET) myocardial metabolic imaging. Left ventricular (LV) functional and LVMD parameters were measured from gated SPET MPI, while myocardial viability was assessed by the integral quantitative analysis of SPET MPI and ^{18}F-FDG PET. Left ventricular MD was defined as >mean+2SD of phase bandwidth (PBW) in the control group. **Results:** Left ventricular MD was present in 37/91 (40.7%) MI patients. The extent of hibernating myocardium (SPET/PET mismatch) and scar (SPET/PET match) in patients with LVMD was significantly higher than in patients without LVMD (15.24±11.26% vs 4.89±5.41%, P<0.001; 11.11±9.42% vs 4.72±5.71%, P< 0.001; respectively). Phase bandwidth correlated with hibernating myocardium and scar (r=0.542, 0.469, P< 0.001; respectively). The multivariate logistic regression analysis showed that hibernating myocardium was an independent factor of LVMD in MI patients (OR=1.110, P=0.007), and >6.5%, hibernating myocardium as a threshold can be used to predict LVMD. In addition, the improvement of PBW (ΔPBW) after coronary artery bypass graft (CABG) at a median follow-up time of 6 months was related with the amount of hibernating myocardium.**Conclusion:** Myocardial infarction patients with left ventricular mechanical dyssynchrony showed significantly more segments of hibernating myocardium and scars as compared to those without LVMD. Hibernating myocardium is independently associated with LVMD in MI patients.

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Introduction

Left ventricular mechanical dyssynchrony (LVMD) refers to differences in the timing of contraction between the different myocardial segments [1]. Previous studies [2-4] have demonstrated that LVMD may serve as a predictive factor of both heart failure and all-cause mortality among patients with coronary artery disease. The amount of hibernating myocardium is related to the recovery of LV function and the degree of LV reverse remodeling, and has an instructive value to determine whether a patient would benefit from revascularization [5, 6]. Moreover, both LVMD and myocardial viability are associated with response to cardiac resynchronization therapy (CRT) [7, 8].

The phase analysis technique on gated single photon emission tomography (gSPET) myocardial perfusion imaging (MPI) is well established for the quantitative measurement of LVMD [9]. Furthermore, technetium-99m methoxyisobutylisonitrile (^{99m}Tc-MIBI) SPET MPI combined with fluorine-18-fluorodeoxyglucose (^{18}F-FDG) positron emission tomography (PET) myocardial metabolic imaging can accurately assess the mismatch (hibernating myocardium) and scar [10, 11]. Samad et al. (2011) [12] showed that LVMD was independently predicted by a reduced LV ejection fraction (LVEF), increasing QRS duration in the electrocardiogram (ECG) and also the extent of scars on SPET MPI in patients with heart failure. Nevertheless, the interactions between clinical data, QRS duration, LV volume, hibernating myocardium, scar and the presence of LVMD in MI patients are not well defined. Recently, it has been reported that LVMD parameters measured by phase analysis on gated SPET MPI may be spuriously increased by scar in patients with ischemic cardiomyopathy [13]. The objective of this study was to identify the correlation between

LVMD and hibernating myocardium in MI patients.

Subjects and Methods

Subjects

We retrospectively studied 122 MI patients who had underwent both gSPET MPI and ^{18}F-FDG PET myocardial metabolism imaging at the Third Affiliated Hospital of Soochow University from October 2010 to November 2016. The enrollment criteria were patients who had a prior MI more than 3 months ago, which was confirmed by history, echocardiography, and ECG [14] and was accompanied with LV dysfunction, as defined by a LVEF <50% along with regional wall dyskinesia on echocardiography. Patients with acute MI, severe arrhythmia, bundle branch block, severe valvular disease, hypertrophic/dilated cardiomyopathy, prior coronary artery bypass graft (CABG), previous insertion of implantable CRT, cardioverter-defibrillators (ICD) or with pacemakers were excluded. Seventy-four patients with suspected chest tightness or chest pain but excluded cardiovascular disease from normal ECG, echocardiography, and stress/rest MPI were included as control group to obtain the cut-off value of LVMD parameter. The study was approved by the institutional ethics committee of the Third Affiliated Hospital of Soochow University.

Image acquisition

Resting gSPET MPI was performed 60-90 minutes after intravenous injection (i.v.) of ^{99m}Tc-MIBI 740-925MBq in all subjects, using a 2-detector 90° camera (Symbia T16, Siemens Medical Systems, Erlangen, Germany) equipped with a low-energy and high-resolution parallel hole collimator centered on the 140keV photopeak with a 20% symmetric energy window. An ECG R-wave detector provided gating to acquire 8 emission frames per cardiac cycle. Sixty-four images covering 180° were acquired with a 64×64 matrix and 1.45 magnification. The images were then reconstructed using the filtered back projection method (order, 5; cutoff frequency, 0.4) and then reoriented to obtain LV short-axis, horizontal long-axis, and vertical long-axis images.

After 6 hours of fasting on the following day, MI patients underwent ^{18}F-FDG cardiac PET/CT study. Depending on their blood glucose level, an oral glucose of 25-50g was given to each patient. For diabetics, acipimox was administrated (500mg oral dose) before glucose loading. Insulin was intravenously administrated if the blood glucose level was >9mmol/L at 45 minutes after oral glucose administration with close monitoring of blood glucose. When serum glucose level reached 5.55-7.77mmol/L, ^{18}F-FDG (3MBq/kg) was administered i.v. [15]. Metabolic images were acquired 1-2 hours after tracer injection using a PET/CT (Biograph mCT 64-s, Siemens Medical Systems, Erlangen, Germany). The cardiac PET scan was performed with the acquisition matrix of 128×128, a magnification of 2, and a photopeak of 511keV. The images were reconstructed with an iterative algorithm (OSEM, 4 iterations and 8 subsets) and then reoriented to acquire LV short-axis, horizontal long-axis, and vertical long-axis images.

Image processing and analysis

Imaging by SPET/PET images was evaluated for quality assurance by consensus of two experienced observers who were blinded to the patients' clinical data. Reconstructed SPET/PET image data sets were imported to a dedicated software package (QPS2009, Cedars-Sinai Medical Center, Los Angeles, CA, USA). An automated method of image analysis was applied to the SPET/PET image data to yield quantified measures of the extent and severity of the scar and the mismatch. Quantification of the viability scores (for mismatch and scar areas) was performed after direct image co-unt normalization between the perfusion and viability scans according to the previous studies [16, 17]. The scar and mismatch (hibernating myocardium) were computed as the sum of all segmental scar and mismatch scores respectively, and reported as a percentage in the total area of the LV myocardium [7]. An example of quantifying myocardial viability is shown in Figure 1.

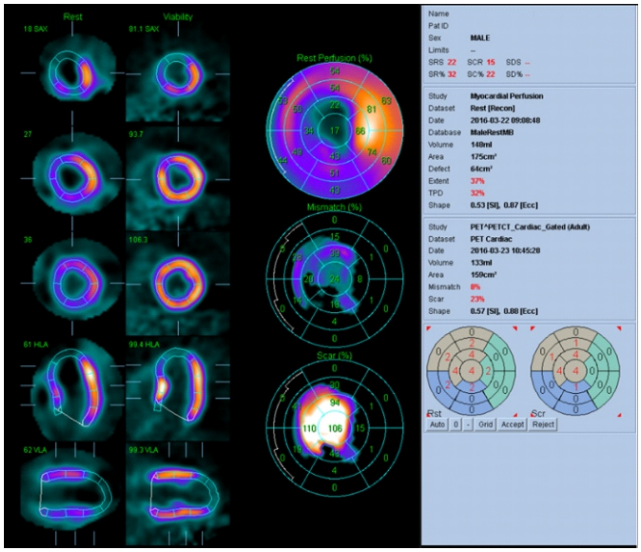

Figure 1. Quantification of rest perfusion deficit and mismatch. Left two columns: Rest perfusion and viability images. Middle column: Quantification shows resting perfusion defect with total perfusion deficit of 32% (polar map at the top), mismatch of 8% (polar map in the middle) and scar of 23% (polar map at the bottom).

The technique of phase analysis was used to measure LVMD from gSPET MPI using quantitative software (QGS 2009, Cedars-Sinai Medical Center, Los Angeles, CA, USA). The phase bandwidth (PBW) and phase standard deviation (PSD) from phase histogram were recorded to characterize the global LVMD. A higher PSD or PBW corresponds to increased LVMD, and vice versa. According to a previous report [18], LVMD was defined as >mean+2SD of PBW (60.6° was accordingly applied), which was derived from the control group of 74 subjects. Left ventricular end diastolic volume (LVEDV), LV end systolic volume (LVESV) and LVEF were measured from resting gSPET MPI using the above QGS software.

Statistical analysis

The SPSS software package (IBM SPSS Statistics 21.0, SPSS Inc, Chicago, IL) was used for statistical analysis. The metrological data with normal distribution were expressed as mean±SD and analyzed using t-test for independent samples. Metrological data that did not meet the normal distribution were expressed as median [Q1, Q3], and Mann-Whitney U rank-sum test was used for the comparison between groups. Count data was expressed as the composition ratio, and the comparison between groups was analyzed using the χ^2 test. The correlation test was performed using the Pearson linear correlation analysis. In addition, we built a univariate logistic regression model to identify predictors for the presence of LVMD. Then, to identify independent predictors of LVMD, significant univariate predictors including New York Heart Association (NYHA), QRS duration, LVEDV, hibernating myocardium and scar were incorporated in the multivariate regression model. The threshold for hibernating myocardium was estimated using a receiver operating characteristic (ROC) curve. The paired Student's t-test was used to compare differences of LVMD parameters between pre-CABG and post-CABG. P<0.05 was considered to be statistically significant.

Results

General information of subjects

Two patients with prior CABG, 5 patients with left bundle branch block, 5 patients with prior CRT and 19 patients with unqualified ^{18}F-FDG myocardial metabolism images were excluded. Eventually a total of 91 patients (82.4% male; 63±9 years old; LVEF 48.0±15.9%) were included in the MI group, and 74 individuals (74.3% male; 60±13 years old; LVEF 68.9±9.5%) were included in the control group. The MI patients' demographic data and clinical characteristics are listed in Table 1. Table 2 lists the parameters of LVMD and the LV functional parameters in the MI group and in the control group. In addition, there were 24 diabetics, 15 hyperlipidemia and 2 hypertensive patients in the control group. There were no significant differences in age and gender between the two groups (all P>0.05).

Comparison between MI patients with and without LVMD

According to the definition of LVMD mentioned above, 37 of the 91 (40.7%) MI patients had LVMD. Compared to the non-LVMD patients, LVMD patients had lower LVEF, higher NYHA

Table1. *Baseline characteristics of enrolled MI patients.*

Characteristics	Total (n=91)	Non-LVMD (n=54)	LVMD (n=37)	P value
Age (years old)	63±9	64±8	62±9	0.357
Male (%)	75 (82.4)	47 (87)	28 (75.7)	0.162
BMI(kg/m^2)	24.60 ±3.03	25.0 ±2.6	24.1 ±3.5	0.158
Hypertension (%)	68 (74.7)	40 (74.1)	28 (75.7)	0.863
Diabetes (%)	30 (33.0)	17 (31.5)	13 (35.1)	0.716
Hyperlipidemia (%)	18 (19.8)	10 (18.5)	8 (21.6)	0.715
Prior PCI	9 (9.9)	6 (11.1)	3 (8.1)	0.637
History of angina (%)	73 (80.2)	42 (77.8)	31 (83.8)	0.480
COPD (%)	2 (2.2)	1 (1.9%)	1 (2.7)	0.786
Stenosis vessel number	2.4 ±0.7	2.5 ±0.7	2.3 ±0.8	0.376
Two-vessels disease	35	19	16	0.288
Three-vessels disease	56	33	23	0.548
NYHA Class	1.9 ±1.1	1.6 ±1.0	2.2 ±1.1	0.007
QRS Duration (ms)	103 ±17	99.4 ±12.8	107.3 ±20.4	0.042
PBW(°)	68.9 ±42.4	39.6 ±10.0	111.7 ±34.2	<0.001
PSD(°)	20.6 ±12.4	13.4 ±5.9	31.0 ±12.0	<0.001
LVEDV(mL)	125 ±62	97.8 ±29.6	163.7 ±75.8	<0.001
LVESV(mL)	73 ±60	44.7 ±23.3	114.0 ±72.8	<0.001
LVEF (%)	48 ±16	56.6 ±10.3	35.5 ±14.4	<0.001
HM (%)	6 (2,12)	3 (2,6)	12 (7,24)	<0.001
Scar (%)	5 (2,10)	3.5 (1,6)	8 (4,16)	<0.001

BMI, body mass index; NYHA, New York Heart Association classification of heart failure; COPD, chronic obstructive pulmonary disease; LVEF, left ventricular ejection fraction; LVESV, left ventricular end systolic volume; LVEDV, left ventricular end diastolic volume; PBW, phase histogram bandwidth; PSD, phase standard deviation; PCI, percutaneous coronary intervention, HM, hibernating myocardium.

Table 2. *Comparison between MI patients and controls.*

Group	Age (years)	Male gender(%) (82.4%))	PBW (º)	PSD (º)	LVEDV (mL)	LVESV(mL)	LVEF (%)
MI (n=91)	62.8±8.6	75(82.4%)	68.9±42.4	20.6±12.4	124.6±62.2	72.8±60.1	48.0±15.9
Control (n=74)	60.1±12.7	55(74.3%)	37.2±11.7	11.8±5.4	76.8±22.8	25.5±13.1	68.9±9.5
P value	0.117	0.252	<0.001	<0.001	<0.001	<0.001	<0.001

LVEF, left ventricular ejection fraction; LVESV, left ventricular end systolic volume; LVEDV, left ventricular end diastolic volume; PBW, phase histogram bandwidth.

Table 3. *Regression models.*

Univariate regression model	OR	95% CI	P value
Age	0.977	0.930-1.026	0.354
Gender	0.463	0.155-1.382	0.168
BMI	0.901	0.778-1.402	0.159
Hypertension	1.089	0.414-2.863	0.863
Diabetes	1.179	0.486-2.860	0.716
Hyperlipidemia	1.214	0.429-3.438	0.715
COPD	1.472	0.089-24.306	0.787
Stenosis vessel number	0.770	0.434-1.376	0.373
History of angina	1.476	0.499-4.366	0.481
NYHA	1.716	1.137-2.588	0.010
QRS duration	1.030	1.002-1.059	0.034
LVEDV	1.029	1.015-1.043	0.000
Hibernating myocardium	1.171	1.085-1.264	0.000
Scar	1.129	1.049-1.214	0.001
Multivariate regression model			
NYHA	1.482	0.899-2.441	0.123
QRS duration	1.010	0.969-1.054	0.632
LVEDV	1.014	1.000-1.030	0.058
HM	1.110	1.029-1.197	0.007
Scar	1.042	0.955-1.137	0.351

BMI, body mass index; NYHA, New York Heart Association classification of heart failure; COPD, chronic obstructive pulmonary disease; LVEF, left ventricular ejection fraction; LVEDV, left ventricular end diastolic volume, HM, hibernating myocardium.

class, wider QRS duration, and greater PBW, PSD, LVEDV, LVESV, hibernating myocardium and scar (all P<0.05). However, there were no statistically significant differences in age, gender, body mass index (BMI), diabetes mellitus, hypertension, hyperlipidemia, angina pectoris history, chronic obstructive pulmonary disease (COPD) and stenosis vessel number (all P>0.05). Myocardial infraction patients with LVMD showed larger areas of hibernating myocardium and scar as compared to those without LVMD (Figure 2). In addition, the areas of hibernating myocardium was clearly larger than that of scar in MI patients with LVMD (Figure 3). Furthermore, in this MI population, PBW was shown to be positively correlated with hibernating myocardium (r=0.542, P<0.001), scar (r=0.469, P<0.001) respectively, whereas PBW and LVEF were negatively correlated (r=-0.689, P<0.001), as shown in Figure 4.

Regression analysis

In the univariate regression model (Table 3), NYHA, QRS duration, LVEDV, hibernating myocardium and scar were identified as predictors of LVMD in MI cohort, whereas age, gender, BMI, diabetes, hypertension, hyperlipidemia, COPD, stenosis vessel number or the history of angina were not. In the multivariate binary regression model including the predictive variables of the univariate model, only hibernating myocardium was identified as the independent risk factor of

the presence of LVMD (OR=1.110, 95% CI:1.029-1.197, P=0.007). The ROC curve indicated that the maximum sensitivity-specificity product was found at a threshold of 6.5% hibernating myocardium. The sensitivity, specificity, and accuracy under the curve area of 0.807 were 78.4%, 77.8%, and 78.0%, respectively (95% CI: 0.710-0.904). In addition, it was shown that MI patients with ≥6.5% hibernating myocardium had a significant higher percentage of LVMD than that in the patients without (70.73% vs. 16.7%, χ^2=27.97, P<0.001). Figure 5 illustrates a typical case of a MI patient who had hibernating myocardium of 29% and showed significant LVMD.

Follow-up

During our follow-up, 50 MI patients whose coronary angiography showed ≥50% stenosis of left main or of three-vessels coronary arteries, severe angina and intermediate/high-risk findings on noninvasive testing underwent CABG surgery. However, there were only 38 patients (41.8%) who underwent gSPET MPI again to assess LV function and LVMD at a median follow-up time of 6 months after CABG. Of these patients, the mean percentage of mismatch was 5.5±6.5%. As a consequence, the values of PBW (41.21±15.54 vs 55.26±30.24; P<0.05) and of PSD (13.83±6.69 vs 17.18±8.88; P<0.05) were significantly reduced after CABG compared to those prior to CABG, while the LVEF was significantly improved after CABG (48.13±12.06% vs 51.21±13.44%; P<0.05). In addition, the improvement of PBW (ΔPBW) after CABG was related with the amount of hibernating myocardium. (r=-0.485, P=0.002), while the correlation between ΔPBW and the amount of scar was not significant (r=-0.324, P>0.05).

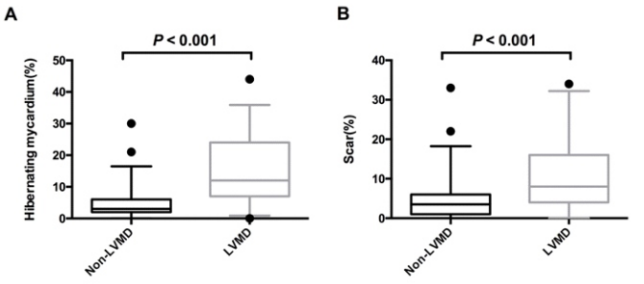

Figure 2. Comparison of hibernating myocardium and scar between MI patients without LVMD and with LVMD.

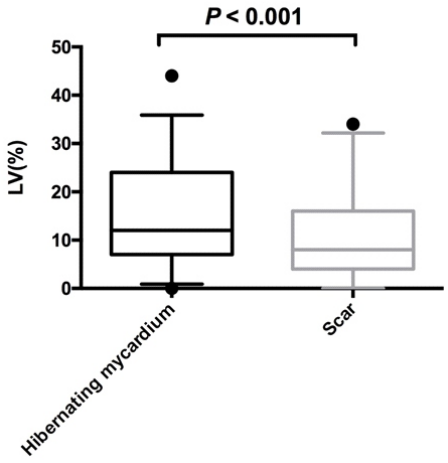

Figure 3. Hibernating myocardium and scar of MI patients with LVMD.

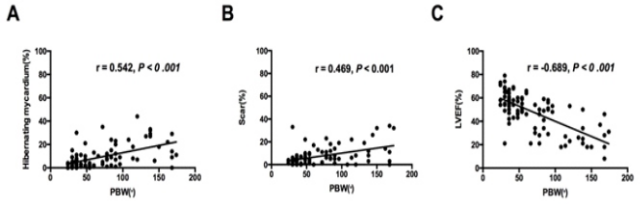

Figure 4. Correlation between hibernating myocardium, scar, LVEF, and PBW in MI patients (n=91).

Discussion

In a cohort of 91 MI patients assessed by gSPET MPI and ^{18}F-FDG PET imaging, only hibernating myocardium was independently associated with the occurrence of LVMD. Besides, the amount of hibernating myocardium was related to the improvement of LVMD after CABG. In addition, the univariate regression analysis indicated that NYHA, QRS duration LVEDV and scar were identified as predictors of LVMD in this cohort. This is to the best of our knowledge the first study using the integral analysis of myocardial perfusion/metabolism imaging to investigate the relationship between hibernating myocardium and LVMD in patients with MI.

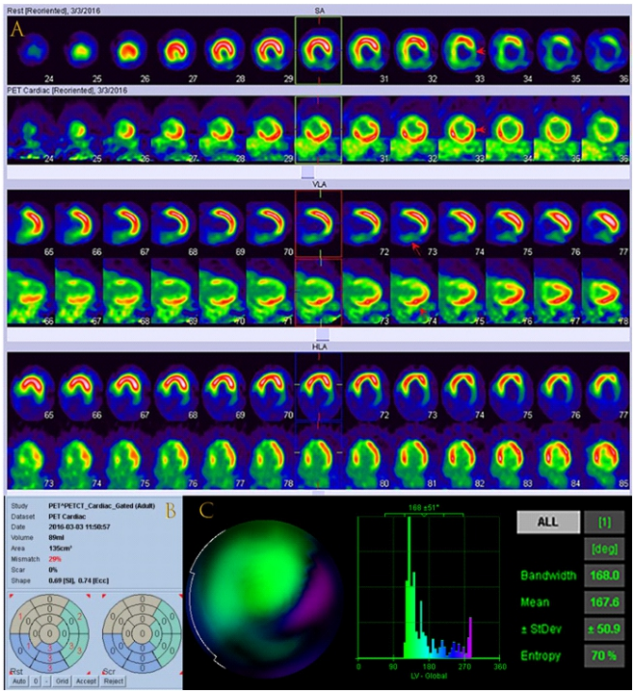

Figure 5. Images of a 70 years old male MI patient with tri-vessels disease (LVEF: 18%). A: Results of ^{99m}Tc-MIBI SPET/^{18}F-FDG PET. Rows 1, 3 and 5 are gSPET MPI images, and rows 2, 4 and 6 are ^{18}F-FDG PET myocardial metabolic images. Technitium-99m-MIBI SPET images show perfusion defects of the inferior wall, lateral wall and posteroseptal wall. All above segments have ^{18}F-FDG uptake, suggesting that they are hibernating myocardium. B: QPS shows mismatch of 29% and scar of 0%. C: Phase analysis shows non-uniform left ventricular phase polar map, asymmetric, multi-peak and wide phase histogram which has phase bandwidth of 168° and phase standard deviation of 50.9°, suggesting that LVMD is present.

It is well known that the wall motion in infarcted areas of MI patients can be weakened, disappearing or contradictory, while that in non-infarcted areas can be temporarily weakened, normal or compensatorily increased. Disparities in wall contraction timing after MI can appear as LVMD and thus affect the LV systolic function [19]. In this study, a reduced LVEF correlated with increasing PBW (r=-0.689, P< 0.001), which confirmed that LVMD is an important factor influencing LV function in MI patients. Moreover, the result revealed that both the hibernating myocardium and scars were more extensive in MI patients with LVMD than in those without LVMD, and the hibernating myocardium and scar were moderately associated with PBW (r=0.542 and 0.469, respectively; P<0.001). This is plausible since scarred and hibernating segments being hypokinetic, dyskinetic or akinetic lead to abnormal contraction patterns and dyssynchrony [20].

Samad et al. (2011) [12] reported that not only the scar burden (as assessed by summed resting perfusion score, SRS), but also QRS duration and LVEF were independent predictors of LVMD measured from SPET MPI in 260 patients with LV dysfunction. The univariate regression analysis of our study also indicated significant correlation between LVMD and QRS duration. Furthermore, because LVEF was significantly associated with PBW in our study, it was not included in the multivariate analysis in order to ensure the discriminatory power of the model. Ludwig et al. (2014) [13] found that the PSD measured from SPET MPI in 50 patients with ischemic cardiomyopathy was significantly reduced after excluding scar tissues using image processing techniques (P< 0.001), which indicates the important influence of scar to LVMD in patients with ischemic cardiomyopathy. However, different from the above studies, our study found the closest relationship between hibernating myocardium and LVMD in MI patients. Several reasons may contribute to the difference of results between the above two studies and ours. Firstly, we used gSPET MPI and ^{18}F-FDG PET imaging to assess myocardial viability for a more accurate evaluation of both hibernating myocardium and scar [10, 11], while the above two studies assessed myocardial viability by SPET MPI alone, which has well-recognized limitations to accurately detect viability. The "scar" by their definition (<50% maximum uptake or SRS) may consist of both a portion of hibernating myocardium and scar, leading to a higher correlation between scar and PSD (r=0.74 or 0.87, P<0.001). Secondly, the cohort in our study had higher LVEF and less extensive scar. Another reason might be that the amount of hibernating myocardium was significantly higher than that of scar in our study. Therefore, although scar was also an influencing factor of LVMD in the univariate model, it was not as significant as hibernating myocardium in the multivariate binary regression model.

The genesis of LVMD is complex and is related to several pathophysiologic mechanisms. Left ventricular mechanical dyssynchrony could be the result of combined abnormal electrical activation and abnormal wall contraction of the LV. Abdelmoniem et al. (2012) [21] reported that LVMD is the result of delayed electrical and mechanical activation in the dysfunctional myocardium. It is commonly accepted that viable myocardium includes both stunning myocardium and hibernating myocardium. In our study, since the patients with acute MI were excluded, viable myocardium was regarded as hibernating myocardium [22]. Others [23] conducted a study on porcine chronic myocardial hibernation models and found a decrease in the expression of calmodulin, leading to the decrease in the maximum contractility of the myocardium during its hibernation. Hibernating myocardium has reduced myocardial contractility at rest due to persistently decreased coronary blood flow for self-protection by reserving the complete structures of cardiomyocytes [24]. In addition, Nina et al. (2008) [25] supported their finding that hibernating myocardium is frequently associated with electrical dysfunction. Besides, others [26] confirmed that severe mechanical dyssynchrony induced by 21 days of sustained, high-frequency pacing the LV free wall causes regional hibernation-like changes in pigs with non-ischemic heart failure. Furthermore, our study found that LVMD in MI patients was significantly improved by CABG, and the degree of the improvement was also closely associated with the amount of hibernating myocardium instead of scar, which further validated the fact that hibernating myocardium is independently associated with LVMD. As a result, LVMD is partly decreased on the basis of effective revascularization by saving regionally hibernating myocardium rather than irreversibly scarred myocardium.

Assessment of hibernating myocardium is clinically relevant in patients with MI, as the contractile function can be partially or completely restored by improving coronary blood flow [27]. Scar mainly refers to permanent death of myocytes after continuous hours of myocardial ischemia. Scar can lead to LVMD due to both damages to cardiomyocyte structure and the loss of myocardial contractility [28, 29], and is generally thought of as being electrically isolated [30], which can never be restored since the cell structure is irreversibly destroyed. Since we cannot do anything to improve the contractile function or activate the electric conduction in scar myocardium, the clinical significance of hibernating myocardium for LVMD is more important than that of scar. Accordingly, we should focus on hibernating myocardium and make it a therapeutic target for the sake of the improvement of LVMD.

Study limitations

Several limitations should be acknowledged. First, the majority of the study cohort had a mild or moderate LV dysfunction and small sizes of hibernating myocardium and scar, and the amount of hibernating myocardium was significantly higher than that of scar, which may lower the effect of scar in LVMD in this population. Future studies should examine patients especially with the large perfusion defect and a severely reduced LVEF. Second, this is a small cross-sectional single-center study, and a large-multicenter prospective study is needed to ascertain the generalizability and accuracy of our findings. Third, gSPET MPI was chosen for analyzing LV function and dyssynchrony rather than ^{18}F-FDG-PET which may exhibit higher image resolution but has not been extensively validated [31]. Fourth, considering the discriminatory power of the multivariate model, LVEF and LVESV were not involved in, which may lead to some bias. Lastly, due to the

retrospective design of the present study there is only a small number of MI patients who received CABG and underwent gSPET MPI after CABG during our follow-up, so that not enough detailed data about functional recovery can be presented after CABG, which needs to be further investigated.

In conclusion, our study showed that myocardial infarction patients with left ventricular mechanical dyssynchrony had significantly more segments of hibernating myocardium and more scars as compared to those without LVMD. Hibernating myocardium is independently associated with LVMD in myocardial infarction patients.

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The authors declare that they have no conflicts of interest.

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