Combined Assessment of Left Ventricular End-Diastolic Pressure and Ejection Fraction by Left Ventriculography Predicts Long-Term Outcomes of Patients with ST-Segment Elevation Myocardial Infarction

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ABSTRACT

Background: In patients with ST-segment elevation myocardial infarction (STEMI), it is unclear if combined assessment of left ventricular end-diastolic pressure (LVEDP) and left ventricular ejection fraction (LVEF) improves prediction of major adverse cardiac events (MACE).

Methods: We analyzed data from 266 STEMI patients who underwent successful percutaneous coronary intervention and subsequent left ventriculography (LVG). Patients were divided into 4 groups, as follows: Group 1, LVEDP <21 mmHg and LVEF ≥55%; Group 2, LVEDP <21 mmHg and LVEF <55%; Group 3, LVEDP ≥21 mmHg and LVEF ≥55%; and Group 4, LVEDP ≥21 mmHg and LVEF <55%. Multivariate Cox proportional hazards analysis was used to determine if LVEDP and LVEF were associated with MACE (including cardiac death, non-fatal myocardial infarction, and heart failure requiring hospitalization). Change in LV parameters was assessed in the subset of 183 patients who underwent serial LVG (mean interval 6.3±1.6 months).

Results: During a mean follow-up of 43±31 months, 29 patients (10.9%) had a MACE. As compared with Group 1, MACE risk was significantly higher in Group 3 (hazard ratio [HR] 3.26; 95% confidence interval [CI] 1.05-10.0) and Group 4 (HR 3.99; 95% CI 1.44-11.0) but not in Group 2 (HR 0.46, 95% CI 0.54-3.96). In sub-analyses, LV end-systolic volume index after PCI was significantly higher in Group 4 than in the other groups and remained higher during follow-up.

Conclusion: Combined LVEDP/LVEF assessment was useful in predicting MACE after successful PCI for STEMI patients and could facilitate risk stratification, as it predicts LV remodeling.

Keywords

Left ventricular end-diastolic pressure, Left ventricular ejection fraction, ST-segment elevation myocardial infarction, Left ventriculogram, Left ventricular remodeling

INTRODUCTION

Left ventricular (LV) systolic function and diastolic function are thought to be associated with future cardiac events, and 12.8% of patients with ST-segment elevation myocardial infarction (STEMI) had impairment of both systolic and diastolic LV function (1). Assessment of systolic and diastolic LV function is important in predicting future cardiac event risks among patients with STEMI (1). Therefore, comprehensive assessment of LV function might improve risk management of STEMI patients.

LV diastolic dysfunction can be identified by measuring LV end-diastolic pressure (LVEDP) on left ventriculography (LVG). LV diastolic function is measured by determining LV filling pressure (2). Although echocardiography is a noninvasive method of assessing LV diastolic dysfunction, echocardiography findings are sometimes inadequate for evaluating LV diastolic dysfunction (2). LVG is a straightforward method for assessing LV parameters; however, because LVEDP has not been routinely measured or reported, its prognostic potential is not well-known. This study examined whether combined assessment with LVEDP and LV ejection fraction (LVEF) measured by LVG could predict major adverse cardiac events (MACE) in patients with STEMI.

MATERIALS AND METHODS

Study population

This retrospective, single-center, observational study analyzed data from 537 consecutive patients with STEMI who transferred to our hospital (Toho University Omori Hospital, Tokyo, Japan) during the period from October 2006 through June 2014. We identified 266 STEMI patients who had undergone successful percutaneous coronary intervention (PCI) within 12 hours after chest symptoms and subsequent LVG to measure LVEDP and LVEF. STEMI was defined by electrocardiography findings indicating a new ST elevation at the J point in at least 2 contiguous leads of ≥2 mm (0.2 mV) in men or ≥1.5 mm (0.15 mV) in women in leads V2–V3 and/or of ≥1 mm (0.1 mV) in other contiguous chest leads or the limb leads, or a new left bundle branch block (3). The exclusion criteria were absence of LVG data (n=223), a thrombolysis in myocardial infarction (TIMI) flow grade less than III after PCI (n=6), and use of an assisted circulation device (n=42).

Patients were divided into 4 groups, as follows: Group 1, LVEDP <21 and LVEF ≥55%; Group 2, LVEDP <21 and LVEF <55%; Group 3, LVEDP ≥21 and LVEF ≥55%; and Group 4, LVEDP ≥21 and LVEF <55%. The overall median LVEDP (21 mmHg) was defined as the optimal cut-off for elevated LVEDP. Based on the prior study demonstrating that patients with an LVEF of ≤55% had more cardiac events than did those with an LVEF >55% in this study (4). Therefore, we used an LVEF cut-off of 55% in this study (4). The study protocol was approved by the ethics committee of Toho University Omori Hospital (no. M16259).

Clinical data and endpoint

Baseline clinical information was obtained from medical records.

Hypertension was defined as a systolic blood pressure ≥140 mmHg or a diastolic blood pressure ≥90 mmHg at the time of the visit, a physician diagnosis of hypertension, or use of antihypertensives. Diabetes mellitus was defined as a fasting blood glucose value ≥126 mg/dl, a physician diagnosis of diabetes, or use of diabetes medication. Dyslipidemia was defined as a total cholesterol concentration >240 mg/dl, a physician diagnosis of dyslipidemia, or use of lipid-lowering medications. Among the 20 patients who had used statins before MI, 5 patients had a prior history of stroke, and were judged to have dyslipidemia. Patients were considered current smokers if they had smoked at least 100 cigarettes in their life and had smoked during the past 30 days. Baseline laboratory data and information on blood pressure and heart rate were collected at admission. Troponin-I and creatinine kinase-MB (CK-MB) were measured at least twice a day, until peak values were recorded.

The primary endpoint was any MACE (including cardiac death, non-fatal myocardial infarction, and heart failure requiring hospitalization) during the observation period.

Invasive coronary angiography and percutaneous coronary intervention strategy

LVEF and LVEDP were measured by LVG after PCI (5). LVG was performed with a 5- or 4-French pigtail catheter inserted into the left ventricle from a femoral, brachial, or radial approach. LVEDP was recorded just before contrast injection and was measured at the Z-point, which is identified on an LV pressure trace as the point at which the slope of the ventricular pressure upstroke changes. LV end-diastolic and end-systolic volumes were measured by using the method of Sandler and Dodge (6). LVEF was calculated by subtracting LV end-systolic volume (LVESV) from LV

end-diastolic volume (LVEDV) and dividing the difference by the LVEDV. LV stroke volume (LVSV) was calculated as the difference between LVEDV and LVESV. LVESV, LVEDV, and LVSV were indexed to body surface area (i.e., LVESVI, LVEDVI, and LVSVI).

Statistical analysis

Data were analyzed with R (R Development Core Team, Vienna, Austria). Continuous variables are expressed as mean ± SD. Categorical variables are expressed as frequency and percentage. We used one-way analysis of variance to compare the average of continuous variables and the Fisher exact test to compare proportions of categorical variables between Groups 1, 2, 3, and 4. Significant differences were analyzed with the Tukey test for multiple comparisons. Kaplan–Meier curves were used to estimate event rates at follow-up and to plot time-to-event curves. Comparisons between the four groups were done with the log-rank test.

Cox proportional hazards analysis was used to identify independent predictors of MACE during the observation period. A multivariate Cox proportional hazards model was built by backward stepwise variable selection, with entry and exit criteria set at P <0.10.

In a subset of 183 patients (68% of the study population) who had undergone serial LVG during follow-up (mean interval 6.3 ± 1.6 months), changes in LVEDP, LVEF, LVSVI, LVEDVI, and LVESVI were assessed between the four groups. The patients who had undergone serial LVG were divided into 4 groups, as follows: Group 1', follow-up LVEDP <21 and follow-up LVEF ≥55% (n=109); Group 2', follow-up LVEDP <21 mmHg and follow-up LVEF <55% (n=49); Group 3', follow-up LVEDP ≥21 mmHg and follow-up LVEF ≥55% (n=18); and Group 4', follow-up LVEDP ≥21 mmHg

and follow-up LVEF <55% (n=10).

RESULTS

Of the 537 patients, we analyzed clinical data from the 266 STEMI patients who had undergone PCI and had data on hemodynamics; 271 patients were excluded. The baseline characteristics of the 266 patients included in the analysis and the 271 patients excluded from the analysis were well-matched, including the proportion of males (78.8% vs 77.2%, *P*=0.60), history of hypertension (61.5% vs 56.3%, p=0.16), diabetes (39.8% vs 30.5%, *P*=0.05), MI (4.4% vs 4.6%,P=0.99), baseline heart rate (76.1 ± 23.8 vs 76.0 ± 19.6 beats/minutes, p=0.96), and peak CK-MB level (323.8 ± 294.4 vs 266.0 ± 243.5 unites, *P*=0.28). However, the excluded patients were older (68.7 ± 13.0 years vs 64.1 ± 12.2 years, *P*<0.0001), more likely to have a history of dyslipidemia (36.1% vs. 24.6%, *P*=0.04), and had lower baseline systolic blood pressures (126.8 ± 26.2 mmHg vs. 144.4 ± 65.5 mmHg, *P*=0.0008) and higher peak troponin-I levels (126.4 ± 197.1 U/L vs. 92.3 ± 99.1 U /L, *P*=0.02).

Table 1 shows the baseline characteristics of the 266 patients. As compared with the other three groups, Group 4 had a longer symptom-to-balloon time and a higher heart rate, Killip class, and serum BNP level. Peak troponin I and CK-MB levels increased progressively from Group 1 to Group 4; thus, Group 4 had the highest levels. **Table 2** shows LVG hemodynamic measurements after successful PCI. As was the case for LVEF, the LVESVI values for Groups 2 and 4 were significantly higher than those for Groups 1 and 4 (Group1, 27.7 ± 8.1 ml/m^2 vs. Group2, 45.6 ± 9.5 ml/m^2 vs. Group3, 29.5 ± 8.6 ml/m^2 vs. Group4, 51.8 ± 25.2 ml/m^2, *P*<0.0001). LVEDVI was highest in group 4 (Group1, 77.1 ± 16.7 ml/m^2 vs. Group2, 88.4 ± 17.0 ml/m^2 vs. Group3, 81.2 ± 15.8 ml/m^2 vs. Group4, 91.8 ± 31.0 ml/m^2, *P*=0.003)

During a mean follow-up of 43 ± 31 months, 29 patients (10.9%) had a MACE.

As shown in **Table 3**, MACE incidence differed among the patient groups (*P*=0.01): 11 patients (4.1%) experienced cardiac death caused by heart failure (Group 1, n=1; Group 2, n=0; Group 3, n=1; Group 4, n=2), myocardial infarction (Group 1, n=0; Group 2, n=0; Group 3, n=3; Group 4, n=2), and arrhythmia (Group 1, n=0; Group 2, n=1; Group 3, n=0; Group 4, n=1). In Kaplan–Meier estimates for MACE (**Fig. 1**), Group 4 had the highest MACE risk, followed by Group 3. In age-adjusted multivariate analysis, as compared with Group 1, MACE risk was significantly higher for Group 3 and higher still for Group 4 (**Table 4**).

Table 5 shows data for LV parameters (including LVEDP, LVEF, LVSVI, LVEDVI, and LVESVI at baseline and follow-up) and changes in LV parameters in a subset of 183 patients. At follow-up, LVEDP, LVEDVI, and LVESVI were highest in Group 4, which indicated less improvement in these LV parameters (**Table 5**). In patients with an LVEF <55% (Groups 2 and 4), ΔLVEF was significantly lower in Group 4 than in Group 2 (5.3 ± 11.0 ml/m^2 vs. 11.1 ± 10.1 ml/m^2, respectively; *P*=0.04); thus, LVEF at follow-up was consistently lower in Group 4 than in the other groups. The results were similar for LVESVI: baseline LVESVI was higher in Group 4 than in the other groups and remained higher during follow-up. In Groups 2 and 4 (patients with an LVEDP <22 mmHg), ΔLVESVI was lower in Group 4 than in Group 2, but the difference was not significant (*P*=0.06).

Patients who underwent serial LVG were classified into 4 groups. There was no significant difference in MACE incidence between the 4 groups (**Table 6**).

DISCUSSION

We found that combined assessment with LVEDP and LVEF accurately estimated MACE risk after successful PCI for STEMI. Among four patient groups

stratified by LVEF and LVEDP, MACE risk was highest for patients with impaired LVEF and LVEDP. Additionally, MACE risk was higher for patients with elevated LVEDP, regardless of LVEF, and when LVEF was <55%. In an analysis of patients who had undergone serial LVG, less improvement in LVEF was seen among patients with higher LVEDP than among those with lower LVEDP, when baseline LVEF was <55%. In addition, LVESVI after PCI was highest in Group 4 and remained so throughout follow-up.

Prior multicenter studies reported an association between cardiac risk and elevated LVEDP (1) (7). In 1909 patients with primary PCI, elevated LVEDP (≥22 mmHg) was independently associated with mortality risk at 90 days (7). Similarly, among 2797 STEMI patients after PCI, an LVEDP ≥18 mmHg was positively associated with death or reinfarction at 30 days and 2 years (1). The findings of that long-term observational are consistent with those of previous studies, which showed that LVEDP had short- and medium-term prognostic value for STEMI patients (1) (7). In addition, the LVEDP cutoff used in the current study is similar to values observed in those studies (1) (7). Several mechanisms explain these findings. Elevated LVEDP results from an acute change in LV parameters caused by increased LV stiffness after myocardial ischemia (8). Increased myocardial wall stiffness is thought to occur before development of cardiac systolic dysfunction, at an earlier stage after MI (9). Elevated LVEDP likely increases wall stress and the risk of pulmonary congestion (10). Furthermore, increased LVEDP leads to decreased perfusion pressure, microvascular dysfunction, subendocardial oxygen delivery, and sympathetic and neurohormonal activation (11, 12). In patients with increased LVEDP, incomplete reperfusion after revascularization may result in larger infarcts and poor outcomes. In the current study, peak troponin I and CK-MB levels were significantly higher in the

groups with higher LVEDP values, which suggests that LVEDP is positively associated with infarct size after STEMI and mortality (1).

In the current study, combined LVEDP/LVEF assessment predicted MACE among patients with MI, which was not reported in prior studies (1) (7). Elevated LVEDP was associated with increased MACE risk, irrespective of LVEF. When baseline LVEF was <55%, MACE risk was higher in patients with higher LVEDP than in those with lower LVEDP. Multiple studies reported that LVEF was associated with MACE risk among patients with MI (13, 14) (15). In contrast, LVEF at the time of admission was not a strong long-term predictor of MACE (16). The present findings are likely attributable to LV remodeling. In 271 STEMI patients undergoing cardiac magnetic resonance imaging, progression of LV remodeling was associated with infarct size, as indicated by peak troponin T and peak CK-MB at administration (17). Low LVEF at admission was reported to be associated with increased LV remodeling and outcome in STEMI patients (18). In the present study, peak troponin I and CK-MB levels were highest in patients with impaired LVEDP and LVEF. These findings are consistent with the hypothesis that combined LVEDP/LVEF assessment is better able to predict MACE, as LVEF is not always strongly correlated with LVEDP (1).

To confirm associations between impairment of LV parameters, LV remodeling, and MACE risk, we assessed the natural course of LV parameters in 68% of the study subjects. Higher LVEDP at baseline was associated with decreased LVEF and LVESVI recovery during follow-up; thus, higher LVEDP after successful PCI may be a good predictor of subsequent LV remodeling. A previous study showed that ΔLVEDVI and ΔLVESVI were lower among patients with LV diastolic dysfunction after MI than among those without LV diastolic dysfunction (19). Duration of LVEDVI improvement was longer among MI patients with larger infarcts than among those

with smaller infarcts (20). Our findings are consistent with studies (19) (20) showing that patents with impaired LVEDP and LVEF had higher baseline LVESVI values and less improvement as compared with patients in other groups. To reduce the risk of cardiovascular events, angiotensin-converting-enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARB) should be started at low doses and titrated to higher doses (21). In the current study, 71 (78.7%) patients in Group 4 had used an ACE inhibitor or ARB, but only 8 patients had received the maximum dose. This finding supports the hypothesis that patients with impairment in both LVEDP and LVEF have greater LV remodeling, which increases MACE risk after STEMI. To prevent MACE, such patients should be carefully managed with cardioprotective medications such as ACE inhibitors and β-blockers, and with anticoagulant therapy and should be received higher doses.

Limitations

This was a retrospective study performed at a single center. "Sicker" patients with lower baseline systolic blood pressures and higher peak troponin I levels were excluded from the present study because operators were likely to avoid LVG, even after successful PCI. Thus, the rate of MACE in patients who did not undergo LVG may be higher than our reported value. In addition, as only 30 patients (11%) had the information regarding LV function by echocardiography prior to AMI, we could not assess the relationship of LV functions between before and after AMI. There may have been differences in LV function at admission and during follow-up, and in the endpoint, between patients with previously normal LV function and those with LV dysfunction. Serial LVG values may be associated with long-term MACE. However, because the numbers of patients with serial LVG data were small (18 in Group 3’ and

10 in Group 4'), we were unable to perform multivariate analysis.

Echocardiography is noninvasive but was not routinely used to measure LV diastolic function. However, it may be more straightforward and easier for risk stratification in patients with STEMI.

Conclusions

When evaluating LV parameters by LVG, combined LVEDP/LVEF assessment better predicted MACE after successful PCI for STEMI patients. Combined LVEDP/LVEF assessment could facilitate risk stratification, as it predicts LV remodeling.

Conflicts of Interest

T.I. received grant support from Bristol-Myers Squibb and Daiichi Sankyo and lecture fees from Bayer Healthcare, Bristol-Myers Squibb, Pfizer, Daiichi Sankyo, Tanabe-Mitsubishi, and Ono Pharmaceutical. The other authors report no conflicts of interest.

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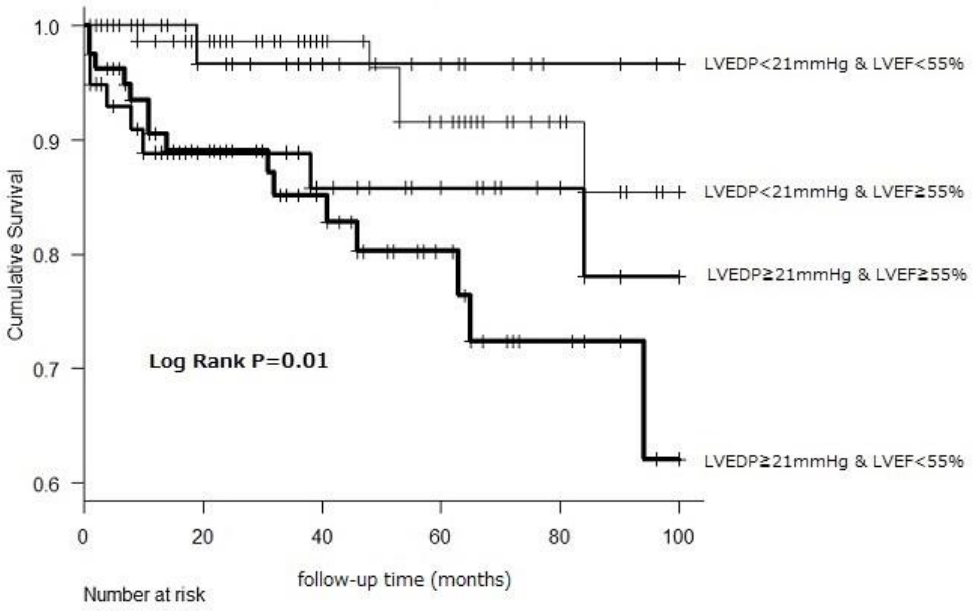
Figure legends

Figure 1. Kaplan–Meier curves for MACE among patients in Groups 1 through 4. LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction; MACE, major adverse cardiac events



Number at risk

LVEDP<21mmHg & LVEF≥55%	85	64	46	37	19	3
LVEDP<21mmHg & LVEF<55%	41	28	20	13	6	1
LVEDP≥21mmHg & LVEF≥55%	59	33	26	20	12	4
LVEDP≥21mmHg & LVEF<55%	80	56	37	24	12	3

Table 1. Baseline characteristics and in-hospital management of patients

	Group 1	**Group 2**	**Group 3**	**Group 4**	
	LVEDP <21 mmHg LVEF ≥55%	LVEDP <21 mmHg LVEF <55%	LVEDP ≥21 mmHg LVEF ≥55%	LVEDP ≥21 mmHg LVEF <55%	*P* value
Number of patients	85	42	59	80	
Age (years)	65.1±11.7	64.1±14.0	63.8±12.6	64.4±11.7	0.92
Male (%)	68/85 (80.0)	35/42 (83.3)	49/59 (83.0)	60/80 (75.0)	0.62
Body mass index (kg/m^2)	23.3±4.2	24.6±10.2	23.6±3.1	23.5±4.8	0.63
Diabetes (%)	25/85 (29.4)	13/42 (30.9)	18/59 (30.5)	26/80 (32.5)	0.98
Hypertension (%)	50/85 (58.8)	28/42 (66.6)	34/59 (57.6)	41/80 (51.2)	0.43
Dyslipidemia (%)	31/85 (36.4)	15/42 (35.7)	22/59 (37.2)	33/80 (41.2)	0.92
Current smoking (%)	49/85 (57.6)	28/42 (66.6)	35/59 (59.3)	52/80 65.0)	0.83
Previous MI (%)	4/85 (4.7)	4/42 (9.5)	2/59 (3.3)	3/80 (3.7)	0.51
Previous PCI (%)	5/85 (5.8)	4/42 (9.5)	3/59 (5.0)	2/80 (2.5)	0.40
Previous CABG (%)	0	1/42 (2.3)	0	0	0.15
Previous Heart Failure (%)	0	1/42 (2.3)	0	1/80 (1.2)	0.32
Symptom-to-balloon time (hours)	4.1±2.7	5.4±2.9***	3.2±2.0**	4.5±2.9***	0.0005
Heart rate (beat/min)	73.3±17.2	80.1±22.7	70.5±19.0	78.8±20.3	0.02
Systolic blood pressure (mmHg)	156.0±111.7	144.3±29.0	131.0±27.7	143.3±28.7	0.18
Killip class I (%)	81/85 (95.3)	37/42 (88.0)	51/59 (86.4)	70/80 (87.5)	0.009
Killip class II or III (%)	3/85 (3.5)	4/42 (9.5)	6/59 (10.2)	10/80 (12.5)	
Killip class IV (%)	1/85 (1.2)	1/42 (9.5)	2/59 (3.4)	0	
Laboratory results					

Baseline hemoglobin level (g/L)	14.0±1.6	14.0±2.0	13.8±2.1	14.5±2.1	0.22
Baseline creatinine (μmol/L)	0.8±0.2	0.84±0.2	0.8±0.1	0.9±0.2	0.39
Baseline BNP (pg/ml)	67.6±90.7	102.3±109.6	76.4±100.9	143.5±199.5*,***	0.003
Peak troponin I (U/L)	53.0±50.4***	62.7±84.8	102.3±108.2*	137.3±108.7*,**	<0.0001
Peak CK-MB (U/L)	204.5±316.8	224.9±165.7	239.3±156.4	366.7±225.1*,**,***	0.0001
Medications before PCI					
Aspirin (%)	6/85 (7.0)	8/42 (19.0)	7/59 (11.9)	5/80 (6.3)	0.11
Ticlopidine (%)	1/85 (1.2)	1/42 (9.5)	0	0	0.53
Clopidogrel (%)	0	1/42 (9.5)	1/59 (1.7)	0	0.14
ARB (%)	10/85 (11.7)	6/42 (14.2)	7/59(11.8)	11/80 (13.7)	0.96
ACE inhibitor (%)	2/85 (2.3)	3/42 (7.1)	0	2/80 (2.5)	0.19
β-blocker (%)	1/85 (1.2)	1/42 (9.5)	2/59 (3.4)	1/80 (1.2)	0.80
Statin (%)	8/85 (9.4)	2/42 (4.8)	4/59 (6.7)	6/80 (7.5)	0.86
In-hospital medications (%)					
Aspirin (%)	85/85 (100)	42/42 (100)	59/59 (100)	80/80 (100)	1.0
Ticlopidine (%)	20/85 (23.5)	11/42 (26.2)	18/59 (30.5)	11/80 (13.8)	0.09
Clopidogrel (%)	65/85 (76.5)	31/42 (73.8)	41/59 (69.5)	69/80 (86.2)	0.09
Heparin (%)	85/85 (100)	42/42 (100)	59/59 (100)	80/80 (100)	1.0
ARB (%)	28/85(32.9)	14/42(33.3)	26/59(29.2)	21/80(26.2)	0.17
ACE inhibitor (%)	46/85 (54.1)	25/42 (59.5)	28/59 (47.5)	50/80 (62.5)	0.33
β-blocker (%)	56/85 (65.8)	35/42 (83.3)	37/59 (62.7)	67/80 (83.8) ***	0.005
Statin (%)	61/85 (71.8)	28/42 (66.7)	43/59 (72.8)	58/80 (72.5)	0.86

*P<0.05 vs Group 1

** P<0.05 vs Group 2

*** P<0.05 vs Group 3

Abbreviations: LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction; MI, myocardial infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; BNP, brain natriuretic peptide; CK-MB, creatinine kinase-myocardial band; ARB, angiotensin II–receptor blocker; ACE inhibitor, angiotensin-converting–enzyme inhibitor

Table 2. Lesion characteristics and LVG data after PCI

	Group 1	**Group 2**	**Group 3**	**Group4**	
	LVEDP <21 mmHg LVEF ≥55%	LVEDP <21 mmHg LVEF <55%	LVEDP ≥21 mmHg LVEF ≥55%	LVEDP ≥21 mmHg LVEF <55%	*P* value
Coronary angiography findings					
1-vessel disease (%)	53/85 (62.4)	25/42 (59.5)	39/59 (66.1)	44/80 (55.0)	0.92
2-vessel disease (%)	24/85 (28.2)	12/42 (28.5)	17/59 (28.8)	27/80 (33.7)	
3-vessel disease (%)	8/85 (9.4)	5/42 (12.0)	3/59 (5.1)	9/80 (11.3)	
Culprit lesion					0.92
RCA (%)	38/85 (44.7)	17/42 (40.5)	27/59 (45.8)	14/80 (17.5)	
LAD (%)	36/85 (42.4)	21/42 (50.0)	24/59 (40.7)	58/80 (72.5)	
LCx (%)	11/85 (12.9)	4/42 (9.5)	8/59 (13.5)	8/80 (10.0)	
TIMI flow before PCI, 0/1 (%)	59/85 (69.4)	28/42 (66.7)	45/59 (76.3)	63/80 (78.8)	0.37
LVG					
LVEDP (mmHg)	16.0±3.2***	15.7±3.5***	26.0±4.2*,**	27.9±5.1*,**	<0.0001
LVEF (%)	64.1±5.9**	46.8±6.3*,***	63.0±6.0**	43.7±8.2*,***	<0.0001
LVSVI (ml/m^2)	49±10.5**	41.8±8.5*,***	51.6±10.0**	40.2±10.0*,***,	<0.0001
LVEDVI (ml/m^2)	77.1±16.7	88.4±17.0	81.2±15.8	91.8±31.0*,	0.003
LVESVI (ml/m^2)	27.7±8.1**	45.6±9.5*,***	29.5±8.6**	51.8±25.2*,***,	<0.0001

*P<0.05 vs Group 1; ** P<0.05 vs Group 2; ***P<0.05 vs Group 3

Abbreviations: LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction; RCA, right coronary artery; LAD, left anterior descending artery; LCx, left circumflex artery; TIMI, thrombolysis myocardial infarction; LVG, left ventriculography; LVSVI, left ventricular stroke volume index; LVEDVI, left ventricular

end-diastolic volume index; LVESVI, left ventricular end-systolic volume index

Table 3. Incidences of MACE components during follow-up (43 ± 31 months)

	Group 1	**Group 2**	**Group 3**	**Group 4**	
	LVEDP <21 mmHg LVEF ≥55%	LVEDP <21 mmHg LVEF <55%	LVEDP ≥21 mmHg LVEF ≥55%	LVEDP ≥21 mmHg LVEF <55%	*P* value
Number of patients	85	42	59	80	
Cardiac death (n, %)	1 (1.2)	1 (2.3)	4 (6.8)	5 (6.3)	0.22
Non-fatal MI (n, %)	3 (3.5)	0	2 (3.4)	4 (5.0)	0.65
Hospitalization due to heart failure (n, %)	1 (1.2)	0	2 (3.4)	6 (7.5)	0.09
MACE (n, %)	5 (5.9)	1 (2.3)	8 (13.6)	15 (18.8)	0.01

Abbreviations: LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction; MI, myocardial infarction; MACE, major adverse cardiac event

Table 4. Results of univariate and multivariate Cox proportional hazards analysis of MACE risk

	Univariate Analysis		**Multivariate Analysis**	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Age, per 1 year increase	1.05 (1.011.09)	0.003	1.06 (1.02-1.10)	0.001
Male sex	0.64 (0.28-1.45)	0.29		
Diabetes	1.02 (0.46-2.24)	0.95		
Smoking	0.83 (0.40-1.73)	0.63		
Killip class II–VI (vs I)	1.18 (0.35-3.93)	0.77		
Baseline TIMI flow 0/1 (vs 2/3)	1.05 (0.45-2.48)	0.89		
Creatinine level, per 1 μmol/L increase	0.86 (0.14-5.10)	0.87		
Hemoglobin level, per 1 g/L decrease	0.92 (0.77-1.10)	0.37		
Peak CK-MB level, per 1 U/L increase	1.00 (0.99-1.00)	0.39		
Peak Troponin-I level, per 1 U/L increase	1.00 (0.99-.100)	0.22		
Symptom-to-balloon time, per 1 hour increase	0.92 (0.92-1.18)	0.47		
LAD infarct	1.71 (0.80-3.62)	0.15		
LVEDP, per 1 mmHg increase	1.11 (1.05-1.17)	0.0001		
LVEF, per 1 % decrease	0.96 (0.93-0.99)	0.01		
Group 1 (LVEDP <21 mmHg and	1 (ref)	-	1 (ref)	-

LVEF ≥55%)				
Group 2 (LVEDP <21 mmHg and LVEF <55%)	0.47 (0.05-4.05)	0.49	0.46 (0.54-3.96)	0.48
Group 3 (LVEDP ≥21 mmHg and LVEF ≥55%)	2.66 (0.87-8.14)	0.08	3.26 (1.05-10.0)	0.03
Group 4 (LVEDP ≥21 mmHg and LVEF <55%)	3.65 (1.32-10.08)	0.01	3.99 (1.44-11.0)	0.007

Abbreviations: TMI, thrombolysis myocardial infarction; CK-MB, creatinine kinase-myocardial band; LAD, left anterior descending artery; LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction

Table 5. LVG data during follow-up and change in LV function in a subgroup of 183 patients

	Group 1	Group 2	Group 3	Group 4	*P* value
LV parameters after PCI					
LVEDP (mmHg)	16.3±3.1[***]	15.6±3.7[***]	26.5±4.4[*,**]	27.3±4.7[*,**]	<0.0001
LVEF (%)	63.9±5.9[**]	46.8±6.2[*,***]	62.9±5.8[**]	44.7±7.6[*,***]	<0.0001
LVSVI (ml/m^2)	52.1±13.0[**]	41.2±11.4[*,***]	52.8±10.4[**]	40.3±10.1[*,***]	<0.0001
LVEDVI (ml/m^2)	81.6±19.3	87.2±19.7	84.2±16.9	90.5±31.5	0.22
LVESVI (ml/m^2)	29.3±8.8[**]	45.7±10.8[*,***]	31.8±9.0[**]	50.5±25.9[*,***]	<0.0001
LV parameters at follow-up					
LVEDP (mmHg)	15.3±5.7	15.5±5.5	16.0±6.1	14.5±5.4	0.66
LVEF (%)	62.9±7.8	57.9±11.0	60.4±8.3	50.0±11.0[*,**,***]	<0.0001
LVSVI (ml/m^2)	51.7±15.6	52.1±18.7	51.8±11.8	47.6±11.9	0.45
LVEDVI (ml/m^2)	82.7±25.5	90.4±27.2	86.0±18.7	99.3±35.7[*]	0.02
LVESVI (ml/m^2)	31.0±13.5	38.0±16.3	34.1±12.4	51.5±29.5[*,**,**]	<0.0001
Changes in LV parameters					
∆LVEDP (mmHg)	-1.0±5.8[***]	-0.0±6.1[***]	-10.4±5.1[*,**]	-12.8±7.5[*,**]	<0.0001
∆LVEF (%)	-0.9±8.4[**]	11.1±10.1[*,***]	-2.4±9.1[**]	5.3±11.0[*,**,***]	<0.0001
∆LVSVI (ml/m^2)	-0.3±15.9[**]	10.8±15.7[*,***]	-1.0±13.9[**]	8.2±15.5[*,***]	0.0003
∆LVEDVI (ml/m^2)	1.1±23.6	3.2±24.8	1.7±21.4	10.9±36.3	0.27
∆LVESVI (ml/m^2)	1.6±12.4	-7.6±15.7	2.8±13.3	2.2±26.5	0.03

[*] P<0.05 vs Group 1

[**] P<0.05 vs Group 2

[***] P<0.05 vs Group 3

Abbreviations: PCI, percutaneous coronary intervention; LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction; LVSVI, left ventricular stroke volume index; LVEDVI, left ventricular end-diastolic volume index; LVESVI, left ventricular end-systolic volume index

Table 6. Incidences of MACE components in patients who underwent serial LVG

	Group 1' follow-up LVEDP <21 follow-up LVEF≥55%	Group 2' follow-up LVEDP <21 mmHg follow-up LVEF<55%	Group 3' follow-up LVEDP ≥21 mmHg follow-up LVEF≥55%	Group 4' follow-up LVEDP ≥21 mmHg follow-up LVEF<55%	*P* value
Number of patients	109	46	18	10	
Cardiac death (n, %)	4 (3.7)	1 (2.2)	2 (11.1)	0	0.41
Non-fatal MI (n, %)	2 (1.8)	2 (4.3)	2 (11.1)	0	0.12
Hospitalization due to heart failure (n, %)	2 (1.8)	1 (2.2)	0	1 (10.0)	0.33
MACE (n, %)	8 (7.3)	4 (8.7)	4 (22.2)	1 (10.0)	0.20

Abbreviations: LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction; MI, myocardial infarction; MACE, major adverse cardiac event