

ST-segment depression in left precordial leads in electrocardiogram of patients with acute inferior myocardial infarction undergoing primary percutaneous coronary intervention

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Abstract: *Background:* The early identification of patients with acute inferior ST-segment elevation myocardial infarction (STEMI) with high risk features is particularly important. Acute inferior STEMI may be associated with ST-segment depression in the left precordial leads (V4–V6). This study assessed prognostic value of ST-segment depression in these left precordial leads during the admission of patients with acute inferior STEMI treated with primary percutaneous coronary intervention. *Methods:* This retrospective study enrolled 1,374 patients with acute inferior STEMI who underwent primary percutaneous coronary angioplasty between March 2011 and June 2014. The patients were divided into two groups: one group ($n = 687$) with left precordial ST-segment depression and the other ($n = 687$) without such ST-segment changes. *Results:* The patients with left precordial ST-segment depression were older and had higher incidence of hypertension, diabetes mellitus, and higher levels of troponin. In-hospital mortality was higher in patients with left precordial ST-segment depression. Advanced coronary artery disease was more observed in these patients. *Conclusion:* In patients with acute inferior STEMI treated with primary coronary intervention, left precordial ST-segment depression during admission of ECG is associated with more advanced coronary artery disease, and worse in-hospital clinical outcomes.

Keywords: acute inferior myocardial infarction, left precordial ST-segment depression, in-hospital mortality, SYNTAX score, primary coronary intervention

Introduction

The early identification of patients with acute inferior ST-segment elevation myocardial infarction (STEMI) with high risk features is particularly important. Early risk stratification is particularly important in acute inferior STEMI due to its presumed better prognosis compared to acute anterior myocardial infarction (MI). Patients with acute inferior wall STEMI are usually considered at lower risk of in-hospital and long-term complication compared to anterior wall MI, although this type of infarction entails subsets of patients with a high likelihood of mortality and morbidity [1].

Because of widespread use and low costs, 12-lead electrocardiogram (ECG) is one of the most useful tools for predicting in-hospital and long-term outcomes in acute ST-segment MI. Several studies have reported the relationship between different patterns of electrocardiographic changes and mortality and morbidity associated with acute inferior wall MI [2, 3].

Acute inferior wall MI may be associated with ST-segment depression in the left precordial leads. The etiology and clinical significance of this change in the ECG has been the subject of several studies in prethrombolytic and thrombolytic era [4–7].

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In previous studies, mainly conducted in prethrombolysis and thrombolysis era, ST-segment depression in the left precordial leads has been associated with larger infarct size, more severe wall motion abnormality, lower left ventricular ejection fraction (LVEF) [8, 9], and unfavorable short- and long-term prognoses.

However, little is known about the prognostic significance of left precordial ST-segment depression in acute inferior wall MI in the modern era of reperfusion therapy including primary percutaneous coronary intervention (PCI) and aggressive medical therapy. Therefore, the purpose of this study was to examine the relationship between left precordial ST-segment depression and in-hospital outcomes in patients with acute inferior MI.

Materials and Methods

Between March 2011 and June 2014, a total of 1,374 patients with a diagnosis of acute inferior wall MI who were admitted within 12 h of symptom onset to our hospital and underwent primary percutaneous coronary angioplasty (PPCI) were enrolled. Acute inferior MI was defined as the following criteria: angular chest pain lasting more than 30 min, ST-segment elevation ≥ 1 mm in at least two of the inferior leads (II, III, and aVF), and elevation of cardiac enzymes including creatine kinase MB and/or troponin-I more than twice the upper limits of normal. All ST-segment measures were assessed and compared between baseline ECG in emergency room and 60 min after PPCI. ST-segment depression was defined as ST-depression ≥ 1 mm 80 ms after the J point using the PR segment as baseline. ST-segment recovery was measured by single-lead ST-segment resolution (STR). A decrease in ST-segment elevation $\geq 50\%$ in the lead with maximal ST-segment elevation at baseline ECG was defined as complete STR. All patients were provided aspirin (325 mg) and clopidogrel (600 mg) during the admission in the emergency room. A bolus of 100 IU/kg of IV heparin was administered. PPCI of culprit vessel was performed through femoral route. Coronary stenosis $\geq 70\%$ were considered significant and multivessel disease was defined as the presence of lesions $\geq 70\%$ of the diameter stenosis in ≥ 2 major coronary arteries. Coronary artery disease severity was also measured by synergy between PCI with taxus and cardiac surgery score (SYNTAX score) [10]. The selection of the revascularization procedure including multivessel PCI was based on operator's decision and coronary anatomy. The procedural decisions, such as stent type (drug-eluting stent or bare metal stent), size, length selection, and usage of glycoprotein IIb/IIIa inhibitors or thrombectomy device, were made according to the discretion of the interventional cardiologist of the procedure.

Coronary intervention and stenting for non-culprit significant stenosis in patients with two or three vessel diseases was performed at other session at same hospitalization. Exclusion criteria included patients with the admission of uninterpretable ECG, left ventricular hypertrophy, intraventricular conduction delays, Wolff-Parkinson-White syndrome, digoxin use, and paced rhythm. We also excluded patients with normal coronary angiogram or non-obstructive coronary artery disease or patients who were candidate for coronary artery bypass graft surgery due to coronary anatomy. The study was approved by local ethics committee and informed consent was obtained from all the patients.

Patients were divided into two groups based on the presence or absence of ST-segment depression in left precordial leads (V4–V6): (1) First group with left precordial ST-segment depression was defined, if maximal ST-segment depression (ST-segment depression ≥ 0.1 mV in ≥ 1 lead) was present in leads V4–V6 or sum of ST-segment depression in leads V4–V6 was greater than the sum of ST-segment depression in leads V1–V3. (2) Second group without left precordial ST-segment depression was defined, if there was less than 0.1 mV ST-segment depression in leads V4–V6 in or sum of ST-segment depression in leads V1–V3 was equal or greater than the sum of ST-segment depression in leads V4–V6. Division of patients into two groups was based only on first ECG during admission in emergency room and not later. Therefore, first ECG obtained at emergency department was used to evaluate ST-segment depression in V4–V6 leads and enrollment of patients to the group with left precordial ST-segment depression or other group. Baseline epidemiologic, clinical, and hemodynamic characteristics, such as age, gender, risk factors, duration of ischemia, admission Killip class, and ST-segment, were recorded and compared between two groups. In addition, we compared catheterization data and coronary artery disease involvement in patients with and without left precordial ST-segment depression. We defined major adverse clinical events (MACE) as in-hospital death, pulmonary edema, repeat MI, complete heart block (CHB), and ventricular arrhythmia. We compared these endpoints between two groups.

Statistical analyses

For continuous variables, data were expressed as means \pm SD and categorical data as prevalence and percentages. Differences between groups were assessed by Student's *t* test for continuous variables and the χ^2 test for categorical variables. All tests of significance were two-tailed, and *p* values of <0.05 were considered significant. The statistical analyses were performed by SPSS version 17.0 for Windows (SPSS Inc., Chicago, IL, USA).

Results

The study population included 1,374 patients, divided into two groups each consists of 687 patients. Patients with maximal left precordial ST-segment depression were older and had higher prevalence of diabetes mellitus and hypertension (Table I). This group also had more prolonged ischemic time than the patients without left precordial ST-segment depression. They also had higher peak creatine phosphokinase and cardiac troponin I levels. Moreover, deteriorated hemodynamic status during presentation as evident by higher Killip class and lower systolic blood pressure were mostly found in patients with left precordial ST-segment depression group compared to patients without such ECG changes. No differences were observed between the two groups in terms of hospital stay. Although there were more number of men than women in both groups, the difference between the genders was not statistically significant

($p=0.1$). Lower LVEF was observed in patients with left precordial ST-segment depression. Regarding arrhythmic events, CHB and ventricular arrhythmia were not significantly different between two groups. In-hospital mortality of patients with left precordial ST-segment depression was almost three times higher than other group. In addition, other MACE rate including pulmonary edema and reinfarction was higher in patients with left precordial ST-segment depression (Table II).

STR $\geq 50\%$ was observed in 475 (69.1%) of the patients in the left precordial ST-segment depression group and 516 (75.1%) of the patients without left precordial ST-segment depression ($p=0.016$), suggesting a less favorable response to mechanical reperfusion in patients with this type of ECG changes.

Table III shows angiographic findings. Multivessel coronary artery disease was more common in patients with left precordial ST-segment depression than the other group (69.8% vs. 62.9%; $p=0.008$). In addition, mean

Table I | Baseline characteristics of patients

	Patients with left precordial ST-segment depression ($n=687$)	Patients without left precordial ST-segment depression ($n=687$)	p value
Age (years)	66.13 \pm 11.35	63.14 \pm 1.50	<0.001
Male	454 (64.1%)	482 (70.2%)	0.10
Total ischemic time (h)	8.14 \pm 2.3	7.27 \pm 3.1	<0.0001
Smoking [n (%)]	198 (28.82%)	225 (32.75%)	0.11
HTN [n (%)]	381 (55.5%)	339 (49.3%)	0.02
Diabetes mellitus [n (%)]	195 (28.38%)	147 (21.39%)	0.003
Hyperlipidemia [n (%)]	108 (15.72%)	84 (12.22%)	0.06
Family history of CAD [n (%)]	36 (5.24%)	27 (3.93%)	0.24
Previous CAD [n (%)]	45 (6.55%)	36 (5.24%)	0.30
Previous CABG [n (%)]	12 (1.74%)	6 (0.87%)	0.15
Previous PCI [n (%)]	9 (1.31%)	18 (2.62%)	0.08
SBP (mmHg)	109.2 \pm 23.6	112.7 \pm 25.1	0.007
DBP (mmHg)	87.3 \pm 17.1	86.96 \pm 18.7	0.72
Heart rate (beats/min)	72.2 \pm 9.3	70.4 \pm 7.5	0.88
Peak CTnI (ng/ml)	28.6 \pm 56.50	9.0 \pm 4.44	0.002
Peak CPK (U/L)	1,854.126 \pm 79.70	1,261.76 \pm 38.29	<0.001
Peak CK-MB (U/L)	289.11 \pm 56.70	244.39 \pm 70.03	0.44
LVEF (%)	40.00 \pm 10.42	43.60 \pm 10.49	<0.001
Duration of hospital stay (days)	5.0 \pm 58.18	5.0 \pm 94.30	0.30
Killip class during admission [n (%)]	I	543 (79%)	0.04
	II	75 (10.9%)	
	III	24 (3.5%)	
	IV	45 (6.6%)	

Data are presented as the mean \pm standard deviation or n (%). CAD: coronary artery disease; CABG: coronary artery bypass graft surgery; PCI: percutaneous coronary intervention; SBP: systolic blood pressure; DBP: diastolic blood pressure; CPK: creatine phosphokinase; CK- MB: creatine kinase- MB; CTnI: cardiac troponin I; LVEF: left ventricular ejection fraction; PCI: percutaneous coronary intervention

Table II | Adverse cardiac events

	Patients with left precordial ST-segment depression (<i>n</i> = 687) [<i>n</i> (%)]	Patients without left precordial ST-segment depression (<i>n</i> = 687) [<i>n</i> (%)]	<i>p</i> value
Mortality	70 (10.18%)	22 (3.20%)	<0.001
Pulmonary edema	64 (9.31%)	8 (1.16%)	<0.001
Re-MI	21 (3.05%)	9 (1.31%)	0.02
Ventricular arrhythmias	81 (11.8%)	76 (11.06%)	0.73
Complete heart block	73 (10.62%)	66 (9.60%)	0.59

Re-MI: repeat myocardial infarction

Table III | Angiographic and procedural data

	Patients with left precordial ST-segment depression (<i>n</i> = 687)	Patients without left precordial ST-segment depression (<i>n</i> = 687)	<i>p</i> value
Single-vessel disease [<i>n</i> (%)]	208 (30.2%)	255 (37.1%)	0.008
Multivessel disease [<i>n</i> (%)]	479 (69.8%)	432 (62.9%)	0.008
TIMI flow 0–1 before PCI [<i>n</i> (%)]	421 (61.2%)	379 (55.1%)	0.024
Use of glycoprotein IIb/IIIa inhibitor [<i>n</i> (%)]	511 (74.3%)	499 (72.6%)	0.500
Total number of stents	2.43 ± 0.4	2.16 ± 0.5	<0.0001
Total stent length (mm)	25.3 ± 4.1	24.2 ± 8.6	0.002
DES implantation	576 (83.8%)	568 (82.6%)	0.610
SYNTAX score	30.1 ± 5.1	19.8 ± 7.9	<0.0001
Infarct-related artery			
RCA [<i>n</i> (%)]	492 (71.6%)	526 (76.5%)	0.042
LCX [<i>n</i> (%)]	195 (28.38%)	161 (23.4%)	
Thrombus grade 4–5 [<i>n</i> (%)]	409 (59.5%)	364 (52.9%)	0.016
No-reflow [<i>n</i> (%)]	41 (5.9%)	23 (3.3%)	0.028

DES: drug-eluting stent; RCA: right coronary artery; LCX: left circumflex coronary artery; TIMI: thrombolysis in myocardial infarction; SYNTAX: synergy between PCI with taxus and cardiac surgery; PCI: percutaneous coronary intervention

SYNTAX score was significantly higher in these patients (30.1 ± 5.1 vs. 19.8 ± 7.9; *p* < 0.0001).

Left anterior descending (LAD) artery stenosis being more than 70% was observed in 236 patients with left precordial ST-segment depression and 180 patients without left precordial ST-segment depression (*p* = 0.001). Patients with left precordial ST-segment depression had higher incidence of left circumflex coronary artery (LCX) as culprit lesion than the other group (28.38% vs. 23.4%; *p* = 0.042).

Discussion

The main findings of this study can be summarized as follows. First, patients with left precordial ST-segment depression (leads V4–V6) during acute inferior MI had

unfavorable epidemiologic and clinical characteristics including older age, more prevalent diabetes and hypertension, prolonged ischemic time, and worse hemodynamic status. Second, more impaired left ventricular systolic function, higher peak cardiac troponins, more advanced coronary artery involvement, and more severe mitral regurgitation were found in these patients. Third, these patients had less optimal response to reperfusion therapy as evident by lower complete STR following primary angioplasty. Finally, patients with left precordial ST-segment depression during acute inferior STEMI had poor short-term prognosis.

Acute inferior wall MI is generally considered more benign compared to anterior wall MI, but certain subgroups are at increased risk of in-hospital and long-term mortality and complications. Patients with ST-segment depression in left precordial leads (V4–V6) during

admission are one of these groups with unfavorable prognosis. In this study, ST-segment alterations in V4–V6 leads were visible from the onset of acute MI.

The diagnostic and prognostic value of this type of electrocardiographic change in patients with acute inferior MI has been widely discussed in several studies mostly in prethrombolytic and thrombolytic area. There are paucity of data regarding prognostic implication of left precordial ST-segment depression in the modern era of early invasive reperfusion of patients with acute inferior wall MI [11, 12]. Similar to the previous studies [13–15], this study showed that patients with left precordial ST-segment depression were older and more likely to be diabetic. Therefore, this may be one of the possible explanations of worse in-hospital outcome in these patients, as diabetes mellitus and advanced age are considered as the prognostic factors in STEMI. Less well-known in previous studies, patients with left precordial ST-segment depression in this study had longer ischemic time. In addition to one of the possible mechanisms for less response to reperfusion therapy, prolonged myocardial ischemia may lead to larger infarct size, more severe left ventricular systolic dysfunction, and less myocardial salvage. Therefore, it is not surprising that these patients had more hemodynamic deterioration during initial presentation (as evident by higher Killip class and lower systolic blood pressure) and more in-hospital mortality.

Regarding coronary artery involvement, the results of this study were in line with most of the previous studies [16, 17] and showed that patients with left precordial ST-segment depression during acute inferior MI were more likely to have multivessel coronary artery disease and unfavorable coronary anatomy as evident by higher SYNTAX score. Coronary anatomy measure like SYNTAX score is one of the independent prognostic factors in patients with STEMI who undergo primary percutaneous intervention [18, 19]. Higher SYNTAX score usually indicates more complicated procedure as shown here by more stent use and longer stents (*Table III*), less response to mechanical reperfusion, and more development of no-reflow phenomenon [20, 21]. Therefore, incomplete STR and higher peak troponin in patients with left precordial ST-segment depression in this study may partly be due to this phenomenon. Other angiographic finding in this study, which may explain worse outcome in these patients with left precordial ST-segment depression, is lower baseline coronary thrombolysis in MI flow (*Table III*). Previous studies have shown unfavorable in-hospital and long-term prognosis of patients with STEMI who are presented with poor preprocedural coronary blood flow [22–24]. Another explanation for higher myocardial necrosis may be the larger thrombus burden (*Table III*) in these patients, which leads to higher incidence of distal embolization and microvascular dysfunction. Overall, patients with left precordial ST-segment depression in this study had delayed reperfusion.

Therefore, older organized intracoronary thrombus may be resistant to mechanical reperfusion resulting in no-reflow phenomenon and poor myocardial perfusion. Similar to some previous studies [5, 25], we showed that LAD artery significant stenosis was more prevalent in this group of patients. LAD stenosis may translate to greater jeopardized myocardium, left ventricular anterior wall ischemia and necrosis, and more depressed systolic function. This may be another reason of high mortality and poor in-hospital outcome of patients with this type of ST-segment depression in acute inferior MI. In addition, in this study, LCX as culprit vessel was more prevalent in patients with left precordial ST-segment depression. This may be another possible explanation of higher mortality and heart failure in these patients, as previous studies have shown increased in-hospital and long-term mortality of patients with LCX-related inferior MI [26, 27]. In the setting of acute coronary syndrome other than acute inferior MI, ST-segment depression alone is usually not useful in predicting the site of coronary occlusion [28]. Rather, ST-segment depression in V4–V6 is highly suggestive of multivessel and/or left main stenosis in patients with non-ST-segment elevation acute coronary syndrome [29, 30]. Nevertheless, there are some reports that could challenge this concept. Nikus et al. [31] showed that in patients with ST-segment depression in V4–V5 and positive T wave in same leads, LAD stenosis was culprit vessel in 74% of patients. Another study by Misumida et al. [32] showed in three of six patients with non-STEMI, upsloping ST-segment depression in V2–V6 was associated with significant LAD stenosis. Of course, small number of patient in these two studies precludes definite conclusion and further studies with adequate power are warranted.

Historically, left precordial ST-segment depression during acute inferior MI has been associated with high in-hospital mortality up to 41% in previous studies [33]. This study showed despite major improvement in reperfusion therapy and cardiac care in STEMI patients in recent years, this type of ST-segment changes still was associated with high mortality rate, almost three times higher than patients without left precordial ST-segment depression (10.1% vs. 3.2%). Of course, the total in-hospital mortality rate in this study (6.7%) is identical to a recent study by Kumar et al. [34], which compared short-term outcomes of patients with inferior MI based on presence or absence of conduction blocks (6.8%). However, another study by Jim et al. [35] reported higher mortality rate (12.6%) than our cohort probably due to fewer reperfusion therapy in their study.

The exact mechanism responsible for left precordial ST-segment depression during acute inferior STEMI remains unknown [9, 36, 37]. Remote anterior or lateral wall ischemia due to reduced coronary blood flow in non-culprit vessels may be one of the possible mechanisms [38]. Liga et al. recently showed that two thirds of patients with STEMI have severely reduced

coronary perfusion at non-culprit vessels that predicted reciprocal. ST-segment depression occurs during acute phase [39].

Another possible etiology may be related to increased left ventricular diastolic pressure, as one small study [40] has shown that patients with left precordial ST-segment depression had higher levels of atrial natriuretic peptide, which means higher stretch of left atrium due to increased left ventricular filling pressure in the setting of diffuse myocardial ischemia. Advanced coronary artery disease in this study in patients with left precordial ST-segment depression may support this theory. This study had several major limitations: First of all, resolution of ST-segment depression was not addressed in this study as recent studies have shown independent prognostic effect of failed ST-segment depression resolution on in-hospital and long-term mortality in patients treated with primary angioplasty [41, 42]. Other major limitation was retrospective, single center, and non-randomized nature of this study with relatively small sample size. In addition, we did not separate patients with maximal ST-segment depression in leads VI–V3 as a distinct group, which may influence our results and conclusions. Therefore, further studies with larger sample size and long-term follow-up with considering ST-segment depression resolution is required to better define the prognostic implication of left precordial ST-segment depression in the acute inferior ST-segment elevation MI. Meanwhile, this study showed that patients with this type of ST-segment changes during inferior MI are at increased risk of adverse in-hospital outcome and need aggressive treatment and hospital care.

Conclusion

This study showed that in patients with acute inferior STEMI undergoing PPCI, left precordial ST-segment depression during ECG admission defines a high risk category with unfavorable baseline characteristics, less response to mechanical reperfusion, more advanced coronary artery disease, and worse in-hospital clinical outcome.

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