ORIGINAL ARTICLE

Predictive Value of ST-Segment Elevation in Lead aVR for Left Main and/or Three-Vessel Disease in Non–ST-Segment Elevation Myocardial Infarction

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Background: ST-segment elevation in lead aVR predicts left main and/or three-vessel disease (LM/3VD) in patients with acute coronary syndromes. ST-segment elevation in lead aVR is generally reciprocal to and accompanied by ST-segment depression in precordial leads. Previous studies have assessed the independent predictive value of ST-segment elevation in lead aVR for LM/3VD in non–ST-segment elevation acute coronary syndrome and have reported conflicting results.

Methods: We performed a retrospective analysis of 379 patients with non–ST-segment elevation myocardial infarction (NSTEMI). Electrocardiograms on presentation were reviewed especially for ST-segment elevation ≥0.05 mV in lead aVR and ST-segment depression ≥0.05 mV in more than two contiguous leads in any other leads.

Results: Among 379 patients, 97 (26%) patients had ST-segment elevation in lead aVR and 88 (23%) patients had LM/3VD. Patients with ST-segment elevation in lead aVR had a higher rate of LM/3VD (39% vs. 18%; $P < 0.001$) and in-hospital revascularization (73% vs. 60%; $P = 0.02$) driven by a higher rate of in-hospital coronary artery bypass grafting (19% vs. 7%; $P < 0.001$) than those without ST-segment elevation in lead aVR. On multivariate analysis, ST-segment elevation in lead aVR (odds ratio [OR] 2.05; 95% confidence interval [CI] 1.10–3.77; $P = 0.02$) and ST-segment depression in leads V_1–V_4 (OR 2.99; 95% CI 1.46–6.15; $P = 0.003$) were independent predictors of LM/3VD.

Conclusion: This study demonstrates that ST-segment elevation in lead aVR is an independent predictor of LM/3VD in patients with NSTEMI.

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non–ST-segment elevation myocardial infarction; acute coronary syndrome; electrocardiography; lead aVR; coronary artery bypass grafting

Patients with acute coronary syndrome resulting from left main and/or three-vessel disease (LM/3VD) are at high risk of short- and long-term adverse cardiovascular events.[1–3] Early recognition of patients with LM/3VD is crucial for disease management including coronary angiogram and revascularization, as well as choice of pharmacological agents. ST-segment elevation in lead aVR predicts LM/3VD in patients with non–ST-segment elevation acute coronary syndromes.[3–7] However, ST-segment elevation in lead aVR is generally reciprocal to and accompanied by ST-segment depression in the precordial leads,[6,8] which is a further predictor of LM/3VD.[9,10] Previous studies have assessed the independent predictive value of ST-segment elevation in lead aVR for LM/3VD in non–ST-segment elevation acute coronary syndrome and have reported conflicting results.[5,6]

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The purpose of the present study was to assess the independent predictive value of ST-segment elevation in lead aVR for LM/3VD in patients with non–ST-segment elevation myocardial infarction (NSTEMI).

METHODS

A retrospective analysis was performed on all patients who had undergone coronary angiography between January 2013 and June 2014 at Mount Sinai Beth Israel Hospital. Myocardial infarction (MI) was diagnosed according to the criteria of the European Society of Cardiology and American College of Cardiology.[11]

Inclusion criteria for the study were (1) a troponin level greater than the 99th percentile reference value before cardiac catheterization; (2) chest pain (or anginal equivalent) or ischemic change on the electrocardiogram (ECG) including horizontal or down-sloping ST-segment depression (≥0.05 mV) or T-wave inversion (≥0.1 mV) in two or more contiguous leads; and (3) absence of ST-segment elevation and new left bundle branch block on the ECG. Exclusion criteria were: (1) cardiac catheterization more than 5 days after presentation; (2) previous coronary artery bypass grafting (CABG); (3) bundle branch block, or ventricular paced rhythm; (4) severe aortic stenosis, hypertrophic cardiomyopathy, self-reported cocaine use within 5 days, cardiac arrest on presentation, ventricular tachycardia, supraventricular tachycardia with heart rate greater than 150 beats per min, implantable cardioverter defibrillator shock, or blood pressure greater than 230/130 mmHg at presentation; (5) subsequent documented diagnosis of takotsubo cardiomyopathy, myocarditis, or pulmonary embolism; and (6) insufficient data for analysis.

The present study complied with the Declaration of Helsinki and was approved by the institutional review board of our hospital. Patients' demographic data and risk factors including hypertension, diabetes, hyperlipidemia, current smoking status, family history of coronary artery disease (CAD), previous MI, previous percutaneous coronary intervention (PCI), and admission characteristics including hemodynamic parameters such as blood pressure and heart rate upon presentation as well as Killip classification were obtained. Obesity was defined as a body mass index greater than 30 kg/m^2. The Thrombolysis in Myocardial Infarction (TIMI) risk score was calculated and classified into three groups; low risk (0–2), intermediate risk (3–4), and high risk (5–7).

Cardiac troponin I (cTnI) levels were measured using a second-generation VITROS® Troponin I assay (Ortho-Clinical Diagnostics Inc., Piscataway, NJ, USA). The upper limit of normal for cTnI was 0.034 μg/L, which represented the 99th percentile reference value. cTnI was measured serially at intervals of approximately 6 hours both before and after catheterization as clinically indicated, with the highest level designated as the peak cTnI.

ECGs obtained on presentation were reviewed by two independent reviewers in a blinded fashion. In the event of an interpretative discrepancy, a consensus between reviewers was reached through discussion. ST-segment shifts were measured at the J point for ST-segment elevation and depression. ST-segment depression ≥0.05 mV in more than two contiguous leads was recorded. The cutoff of ≥0.05 mV for ST-segment depression was chosen in line with the current universal definition of MI.[11] The location of ST-segment depression was recorded as anterior (V_1–V_4), lateral (I, aVL, V_5, and V_6) and inferior (II, III, and aVF; not mutually exclusive). In addition, ST-segment depression in lead I and lead II were recorded. ST-segment elevation in lead aVR ≥ 0.05 mV was recorded and its magnitude was measured. The cutoff of ≥0.05 mV for ST-segment elevation in lead aVR was chosen in line with the previous studies.[5,6,8] Transthoracic echocardiography was performed in a standard manner during hospitalization, and left ventricular ejection fraction was calculated using either the Teichholz or biplane Simpson's method.

All patients underwent cardiac catheterization within 5 days of presentation with NSTEMI. An independent cardiologist blinded to the clinical data reviewed all coronary angiography results for the purposes of comparative assessment with the primary treating cardiologist. In the event of an interpretive discrepancy, a third investigator was responsible for the final assessment. Obstructive CAD was defined as stenosis greater than or equal to 70% (50% in the left main coronary artery). Revascularization procedures including PCI and CABG were performed at the discretion of the treating physician. 3VD was diagnosed in the presence of obstructive CAD in all three major coronary arteries.

The primary outcome was the prevalence of LM/3VD. In addition, in-hospital mortality, recurrent MI, heart failure, and cardiogenic shock as well as length of hospital stay were recorded. These outcomes were compared between patients with and without ST-segment elevation in lead aVR.

Data are expressed as the number (percentage) or median (interquartile range). For continuous variables, the Shapiro-Wilk test was used to check the normality of the distribution. Continuous variables were compared using either Student's *t*-test or Wilcoxon rank-sum test as appropriate. Dichotomous variables were compared using the chi-square test or Fisher's exact test. The Cohen's kappa coefficient was calculated to measure intraobserver and interobserver agreements regarding presence or absence of ST-segment elevation in lead aVR.[12] The interpretation of the kappa coefficient adheres to the following generally accepted scale: poor < 0.2; fair > 0.2–0.4; moderate > 0.4–0.6; good > 0.6–0.8; very good > 0.8–0.9; excellent > 0.9–1.0. To identify predictors of LM/3VD, the following variables were initially assessed in a univariate model: age, sex, hypertension, diabetes, hyperlipidemia, previous MI, previous PCI, high TIMI risk score, heart rate, Killip classification >1 on admission, ST-segment depression in anterior, lateral and inferior leads, the number of leads with ST-segment depression, and ST-segment elevation in lead aVR. Significant variables with a P value < 0.20 in univariate analysis were then entered into a multivariate logistic-regression analysis using backward stepwise selection (model 1). Age, sex, diabetes, previous MI, and heart rate were automatically entered into the multivariate model based on the results of a previous study.[6] In addition, 2 additional multivariate analyses were carried out using the following variables in replacement of ST-segment depression in anterior, inferior and lateral leads: model 2 included ST-segment depression in lead I and ST-segment depression in lead II, and model 3 included ST-segment depression both in lead I and in lead II. A significance level of 0.10 was required to allow a variable to remain in the model. Two-sided P values <0.05 were considered statistically significant. All statistical analyses were performed with R software (version 3.0.1).

RESULTS

A total of 379 patients who underwent coronary angiography within 5 days after presentation with the diagnosis of NSTEMI were included in the final analysis. Among 379 patients, 97 (26%) patients had an ST-segment elevation in lead aVR ≥ 0.05 mV, of which 61 patients had ST-segment elevation in lead aVR ≥ 0.1 mV. Of the 379 patients, 88 (23%) patients had LM/3VD.

The kappa coefficients of intraobserver agreement regarding presence or absence of ST-segment elevation in lead aVR ≥0.05 mV and ≥0.1 mV were 0.89 (95% CI: 0.84–0.94) and 0.89 (0.83–0.95), respectively. The kappa coefficients of interobserver agreement regarding presence or absence of ST-segment elevation in lead aVR ≥0.05 mV and ≥0.1 mV were 0.84 (0.78–0.90) and 0.87 (0.80–0.94), respectively. These results indicate very good intraobserver and interobserver agreements.

Patients' characteristics are summarized and presented in Table 1. Patients with ST-segment elevation in lead aVR were older, with a higher TIMI risk score and peak cTnI value. With respect to ECG findings, patients with an ST-segment elevation in lead aVR were more likely to have concomitant ST-segment depression. Among 97 patients with ST-segment elevation in lead aVR, 78 of these presented with concomitant ST-segment depression comprising of anterior (41 patients), lateral (75 patients) and inferior (48 patients) ST-segment depression.

Patients with ST-segment elevation in lead aVR had a significantly higher rate of 3VD, left main disease and LM/3VD than those without ST-segment elevation in lead aVR. Patients with ST-segment elevation in lead aVR had a significantly higher rate in-hospital revascularization, driven by a higher rate of in-hospital CABG when compared to those without ST-segment elevation in lead aVR. There was no significant difference in in-hospital mortality and in the incidence of in-hospital recurrent MI, heart failure and cardiogenic shock between the two groups.

The results of univariate and multivariate analyses are presented in Table 2. On univariate analysis, ST-segment elevation in lead aVR and ST-segment depression in the anterior, lateral and inferior leads were associated with LM/3VD.

Table 1. Patients' Baseline Characteristics, Electrocardiogram, Angiography Findings, and In-Hospital Outcomes.

	aVR ST Elevation (n = 97)	No aVR ST Elevation (n = 282)	P Value
Baseline characteristics and risk factors			
Age (years)	67 (60–75)	64 (54–73)	0.01
Men	52 (54)	174 (62)	0.16
Obesity (body mass index ≥ 30 kg/m^2)	29 (30)	81 (29)	0.83
Hypertension	76 (78)	197 (70)	0.11
Diabetes	36 (37)	98 (35)	0.67
Hyperlipidemia	56 (58)	159 (56)	0.82
Current smoking	22 (23)	74 (26)	0.49
Family history of CAD	20 (21)	60 (21)	0.89
Previous MI	13 (13)	37 (13)	0.94
Previous PCI	35 (36)	74 (26)	0.06
TIMI risk score			<0.001
Low risk 0–2	7 (7)	74 (26)	
Intermediate risk 3–4	51 (53)	152 (54)	
High risk 5–7	39 (40)	56 (20)	
Hemodynamic and laboratory data			
Killip class > 1 on admission	11 (11)	32 (11)	1
Systolic blood pressure (mmHg)	149 (126–168)	140 (125–156)	0.01
Heart rate (beats/minute)	88 (75–99)	79 (69–92)	0.006
Peak troponin I (μg/L)	2.10 (0.18–7.72)	0.48 (0.09–4.12)	0.007
Electrocardiogram and echocardiogram findings			
ST depression	78 (80)	39 (14)	<0.001
Anterior ST depression (V_1–V_4)	41 (42)	15 (5)	<0.001
Lateral ST depression (I, aVL, V_5, and V_6)	75 (77)	32 (11)	<0.001
Inferior ST depression (II, III, and aVF)	48 (49)	7 (2)	<0.001
Lead I	56 (58)	19 (7)	<0.001
Lead II	70 (72)	17 (6)	<0.001
T-wave inversion	5 (5)	90 (32)	<0.001
Left ventricular ejection fraction (%)	60 (45–61)	60 (50–65)	0.46
Angiographic findings and in-hospital outcomes			
Left main/three-vessel disease	38 (39)	50 (18)	<0.001
Three-vessel disease	37 (38)	44 (16)	<0.001
Left main disease	8 (8)	6 (2)	0.01
In-hospital revascularization	71 (73)	169 (60)	0.02
In-hospital PCI	53 (55)	150 (53)	0.81
In-hospital CABG	18 (19)	19 (7)	<0.001
In-hospital all-cause death	2 (2)	1 (0.4)	0.16
In-hospital recurrent MI	0 (0)	1 (0.4)	1
In-hospital heart failure	13 (13)	33 (12)	0.66
In-hospital cardiogenic shock	3 (3)	4 (1)	0.38
Length of stay (days)	5.2 (2.5–9.5)	4.0 (2.5–6.4)	0.03

Data are expressed as number (percentage) or median (interquartile range).
CAD = coronary artery disease; MI = myocardial infarction; PCI = percutaneous coronary intervention; TIMI = Thrombolysis In Myocardial Infarction; CABG = coronary artery bypass grafting.

On multivariate analysis (model 1), ST-segment elevation in lead aVR and ST-segment depression in anterior leads were independent predictors for LM/3VD. ST-segment depression in the lateral and inferior leads, and the number of leads with ST-segment depression were not independent predictors for LM/3VD. Other independent predictors for LM/3VD included age, male sex, hyperlipidemia, and Killip classification >1. The additional multivariate analysis (model 2) showed that ST-segment elevation in lead aVR was an independent predictor of LM/3VD (OR 3.02; 95% CI: 1.76–5.22; P < 0.001), but neither ST-segment depression in lead I nor ST-segment depression in lead II was an independent predictor. Another multivariate analysis (model 3) showed that ST-segment elevation in lead aVR was an independent predictor of LM/3VD (OR 3.02; 95% CI: 1.76–5.22;

Table 2. Univariate and Multivariate Analysis of Predictors for Left Main/Three-Vessel Disease.

	Odds Ratio	95% Confidence Interval	P Value
Univariate Analysis			
Age (per decade increase)	1.54	1.27–1.89	<0.001
Male sex	1.25	0.76–2.05	0.38
Hypertension	1.32	0.77–2.33	0.33
Diabetes	1.20	0.73–1.96	0.46
Hyperlipidemia	2.01	1.22–3.38	0.007
Previous myocardial infarction	1.05	0.50–2.06	0.89
Previous percutaneous coronary intervention	0.91	0.53–1.53	0.73
High TIMI risk (5–7)	2.37	1.41–3.96	<0.001
Heart rate (per 10 beats/minute increase)	1.07	0.94–1.20	0.32
Killip class >1 on admission	2.74	1.40–5.28	0.003
Anterior ST depression (V_1–V_4)	4.00	2.20–7.26	<0.001
Lateral ST depression (I, aVL, V_5, and V_6)	2.79	1.69–4.60	<0.001
Inferior ST depression (II, III, and aVF)	2.37	1.28–4.32	0.005
Number of leads with ST depression (per 1 increase)	1.26	1.13–1.39	<0.001
ST elevation in lead aVR ≥ 0.05 mV	2.99	1.79–4.98	<0.001
Multivariate analysis			
Age (per decade increase)	1.61	1.28–2.05	<0.001
Male sex	2.45	1.38–4.51	0.003
Hyperlipidemia	2.04	1.19–3.60	0.01
Killip class > 1 on admission	2.78	1.32–5.80	0.007
Anterior ST depression (V_1–V_4)	2.99	1.46–6.15	0.003
ST elevation in lead aVR ≥ 0.05 mV	2.05	1.10–3.77	0.02

TIMI: Thrombolysis in Myocardial Infarction.

$P < 0.001$), but ST-segment depressions both in lead I and lead II was not an independent predictor. (In both model 2 and model 3, ST-segment depression in lead I, lead II, and both in lead I and lead II did not remain in the model according to stepwise selection ($P > 0.1$).)

The sensitivity and specificity of ECG findings are presented in Table 3. An ST-segment elevation in lead aVR ≥0.05 mV had a sensitivity of 43% and specificity of 80%. Although the sensitivity of ST-segment elevation in lead aVR ≥ 0.15 mV was very low (13%), the positive predictive value was high (69%). Among the three ST-segment depression groups, ST-segment depression in the anterior leads was the most specific and had the highest positive predictive value (48%).

DISCUSSION

Our study demonstrates that ST-segment elevation in lead aVR and ST-segment depression in anterior leads are independent predictors of LM/3VD in patients with NSTEMI. ST-segment elevation in aVR is generally reciprocal to and accompanied by ST-segment depression in the precordial leads,[6,8] another predictor of LM/3VD.[9,10] Previous studies have assessed the independent predictive value of ST-segment elevation in lead aVR for LM/3VD in non–ST-segment elevation acute coronary syndrome and have reported conflicting results. Kosuge et al. reported that ST-segment elevation in lead aVR was independently associated with LM/3VD while ST-segment depression

Table 3. Predictive Values of ST-Segment Elevation in Lead aVR and ST-Segment Depression for Left Main/Three-Vessel Disease.

	Sensitivity	Specificity	PPV	NPV	Predictive Accuracy
Anterior ST depression (V_1–V_4)	31%	90%	48%	81%	76%
Lateral ST depression (I, aVL, V_5, and V_6)	45%	77%	37%	82%	70%
Inferior ST depression (II, III, and aVF)	24%	88%	38%	79%	73%
ST elevation in lead aVR ≥ 0.05 mV	43%	80%	39%	82%	71%
ST elevation in lead aVR ≥ 0.1 mV	33%	89%	48%	81%	76%
ST elevation in lead aVR ≥ 0.15 mV	13%	98%	69%	79%	78%

PPV = positive predictive value; NPV = negative predictive value.

was not.[5] However, several differences exist between that and the present study, which may explain these observations. Indeed, greater than half of the patients included in Kosuge et al.'s study showed negative troponin T levels while ST-segment depression was defined as ≥0.1 mV—in contrast to the present study where ST depression was defined as ≥0.05 mV. The presence of ST-segment depression ≥0.05 mV has been shown to predict LM/3VD.[9,10] Therefore, not including ST-segment depression ranging from 0.05 to 0.1 mV might have led to the different results.

In contrast, Yan et al. reported that ST-segment elevation in lead aVR >0.1 mV predicted LM/3VD, while ST-segment elevation in lead aVR ranging from 0.05 to 0.1 mV did not.[6] In Yan et al.'s study, the prevalence of ST-segment elevation in lead aVR was just 7.3% and more than half of the patients in that study showed negative cardiac biomarkers. The present study showed that 25.6% of patients had an ST-segment elevation in lead aVR. The patients in the present study consisted of a high-risk population with positive cardiac troponin levels. In fact, the higher prevalence of aVR ST-segment elevation observed in our study was consistent with a previous study of 775 patients with NSTEMI wherein 32.3% of the patients had ST-segment elevation in lead aVR.[8]

Although lead aVR is an augmented lead as a mean of lead I and lead II, our study showed that the predictive value of ST-segment elevation in lead aVR for LM/3VD was independent from ST-segment depression in lead I and lead II. Our findings indicate that lead aVR does not merely reflect reciprocal changes but independently provides valuable clinical information. However, it should be noted that the sensitivity and positive predictive value of ST-segment elevation in lead aVR for LM/3VD were relatively low.

In the present study, ST-segment depression in anterior leads was another independent predictor for LM/3VD. A previous study that evaluated ECG changes in patients with left main disease showed that the most frequently observed ECG pattern was ST-segment depression in V_3, V_4, and V_5.[13] Our finding was consistent with this finding. In addition to the predictive value for LM/3VD, ST-segment depression carries a significant prognostic value in patients with NSTEMI.[14,15] Furthermore, ST-segment depression in leads V_4–V_6 has been reported to be an independent predictor for short-term mortality in patients with inferior ST elevation myocardial infarction (STEMI).[16]

There are several limitations in the present study. First, analysis is subject to the usual constraints associated with a retrospective observational study. However, we did review all patients who underwent cardiac catheterization, thus limiting our selection bias. Second, a small sample size and low in-hospital mortality rate limited the power to evaluate a prognostic value of ST-segment elevation in lead aVR. Third, we did not exclude patients with posterior (inferolateral) infarction presenting with ST-segment depression in V_1–V_4, which is equivalent of STEMI. Therefore, our cohort might have included patients with posterior STEMI. However, this ECG change is not pathognomonic of posterior (inferolateral) infarction, and it has been reported that one-third of the patients with acute coronary syndrome presenting with ST-segment depression in V_1–V_4 had a culprit lesion in left anterior descending artery.[17] In addition, the difficulty in identifying posterior infarction is also well recognized[18] and these patients can be treated as NSTEMI in real-world practice. In this context, we did not exclude patients presenting with ST-segment depression in leads V_1–V_4. Finally, our study population only

included patients who underwent coronary angiography since we aimed to determine the association between ECG findings and angiographic findings. This will limit the generalizability of our findings to a broad population presenting with acute coronary syndrome.

In conclusion, the present study demonstrates that ST-segment elevation in lead aVR and ST-segment depression in anterior leads are independent predictors of LM/3VD in patients with NSTEMI.

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