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Prognostic Significance of ST Segment Depression in Lateral Leads I, aVL, V_5 and V_6 on the Admission Electrocardiogram in Patients With a First Acute Myocardial Infarction Without ST Segment Elevation

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OBJECTIVES We sought to investigate the short-term prognostic value of the admission electrocardiogram (ECG) in patients with a first acute myocardial infarction (MI) without ST segment elevation.

BACKGROUND ST segment depression on hospital admission predicts a worse outcome in patients with a first acute MI, but the prognostic information provided by the location of ST segment depression remains unclear.

METHODS In 432 patients with a first acute MI without Q waves or ≥0.1 mV of ST segment elevation, we evaluated the ability of the initial ECG to predict in-hospital death.

RESULTS The presence, magnitude and extent of ST segment depression were associated with an increased mortality, but the only electrocardiographic variable that was significant in predicting death after adjusting for baseline predictors was ST segment depression in two or more lateral (I, aVL, V_5, or V_6) leads (odds ratio 3.5, 95% confidence interval 1.2 to 10.6). Patients with lateral ST segment depression (n = 91, 21%) had higher rates of death (14.3% vs. 2.6%, $p < 0.001$), severe heart failure (14.3% vs. 4.1%, $p < 0.001$) and angina with electrocardiographic changes (20.0% vs. 11.6%, $p = 0.04$) than did the remaining patients, even though they had similar peak creatine kinase, MB fraction levels (129 ± 96 vs. 122 ± 92 IU/liter, p = NS). In contrast, ST segment depression not involving the lateral leads did not predict a poor outcome. Among patients who were catheterized, those with lateral ST segment depression had a lower left ventricular ejection fraction (57 ± 12% vs. 66 ± 13%, $p = 0.001$) and more frequent left main coronary artery or three-vessel disease than did the remaining patients (60% vs. 22%, $p < 0.001$).

CONCLUSIONS In patients with a first non–ST segment elevation acute MI, ST segment depression in the lateral leads on hospital admission predicts a poor in-hospital outcome. (J Am Coll Cardiol 2000;35:1813–9) © 2000 by the American College of Cardiology

In view of the wide range of in-hospital outcomes observed in patients with acute myocardial infarction (MI) who present without ST segment elevation (1), early risk stratification seems essential for appropriate management. Because of its simplicity, widespread availability and low cost, the electrocardiogram (ECG) has been extensively used for this purpose. Although a normal or nearly normal ECG predicts an uncomplicated course (2–6), the presence of ST segment depression alone or in combination with other repolarization changes has been consistently associated with a worse short- and long-term outcome (1,6–16).

However, because ST segment depression is the main electrocardiographic finding in a broad spectrum of patients, with regard to the size of the infarction, the severity of coronary artery disease (CAD) and the amount of jeopardized myocardium (10,16,17), there is a need for further criteria that would allow for a more accurate gradation of risk. The magnitude and extent of ST segment depression have predicted the clinical outcome in some studies (9,10,16), but not in others (8). In contrast, studies that have addressed the prognostic value of the location of ST segment depression have included patients with concomitant ST segment elevation (6,11,13,18). These studies,

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Abbreviations and Acronyms

CAD	= coronary artery disease
CI	= confidence interval
CK-MB	= creatine kinase, MB isoenzyme
ECG	= electrocardiogram
LVH	= left ventricular hypertrophy
MI	= myocardial infarction
OR	= odds ratio

moreover, have also included patients with a previous MI, which may interfere with the prognostic value of the initial ECG changes. Accordingly, we sought to assess, in a group of patients with a first acute MI without Q waves or significant ST segment elevation, the ability of the presenting ECG to predict in-hospital death and other adverse events. In patients who were catheterized before hospital discharge, the relation between the electrocardiographic patterns and left ventricular function and coronary anatomy was also examined.

METHODS

Patients. The study initially included 470 consecutive patients admitted to our coronary care unit during a 12-year period (1984 through 1995) with a first acute MI without ST segment elevation (≥0.1 mV) or left bundle branch block (QRS complex ≥0.12 s, predominantly negative in lead V_1 and with an upright notched or slurred R wave in leads I and V_6). Diagnosis of acute MI was based on the presence of anginal pain and elevation of serum creatine kinase, MB isoenzyme (CK-MB) activity levels. Medical histories were carefully reviewed, and patients with valvular or myocardial heart disease, previous coronary artery bypass graft surgery or recent (less than six months) coronary angioplasty were excluded. The first 12-lead ECG taken in the emergency room, or occasionally another more representative tracing obtained within the first 24 h after the onset of MI, was used for analysis. From the patients initially screened, 15 were excluded because the presenting ECG showed a Q wave or posterior infarction (Q waves ≥30 ms in two or more contiguous leads, R wave ≥40 ms in lead V_1 and R/S ratio >1 in lead V_2) (19), and 23 were excluded because their medical charts were incomplete or lacked an ECG tracing from the first 24 h after the onset of MI. Thus, 432 patients were finally included in the study.

Electrocardiographic evaluation. Admission ECGs were carefully examined by one investigator who had no knowledge of the patients' clinical outcome. Right bundle branch block was diagnosed using standard criteria. Left ventricular hypertrophy (LVH) was defined by a sum of R waves in lead V_5 or V_6, plus an S wave in lead V_1 or V_2 ≥3.5 mV (9). ST segment shifts were measured in all leads, with the exception of aVR, at 80 ms after the J point for ST segment depression and at 20 ms after the J point for ST segment elevation, using the preceding TP segment as a baseline value. The magnitude and location of the maximal ST segment depression, the number of leads with ST segment depression ≥0.1 mV and the sum of ST segment depression in all leads were measured. Anterior, inferior and lateral ST segment depressions were defined, respectively, as ST segment depression ≥0.1 mV in at least two leads oriented anteriorly (V_1 to V_4), inferiorly (II, III and aVF) or leftward, either laterally or apically (I, aVL, V_5, and V_6). The presence of minor (0.05 to 0.1 mV) ST segment elevation or T-wave inversion was also evaluated.

Clinical data collection. Demographic data, risk factors for coronary heart disease, ongoing treatment with antithrombotic or antianginal medications and data from physical examination on hospital admission were collected. Main in-hospital clinical events such as death, reinfarction, heart failure, postinfarction angina and coronary revascularization were also recorded in all patients, without knowledge of the ECG findings. The rates of death and reinfarction at 30 days could be determined in 306 (71%) and 292 (68%) patients, respectively. Total CK and CK-MB activities were measured during the initial 48 h of the hospital stay at 4- to 6-h intervals and after episodes of chest pain of ≥30 min to rule out reinfarction. Diagnosis of reinfarction was established when a re-elevation of serum CK-MB over a previously normal value in at least two consecutive samples occurred. Angiographic data were available in 215 patients (50%) who underwent cardiac catheterization before hospital discharge, 8 ± 4 days after MI. Stenosis ≥50% in the diameter of the left main coronary artery or stenosis ≥70% in one or more of the major epicardial vessels or their main branches was considered significant. In 153 patients (71% of those catheterized), the infarct-related vessel could be identified.

Statistical analysis. Statistical analysis was performed with the aid of SPSS software (version 6.0). Categoric variables are described as frequencies and percentages, and continuous variables as the mean value ± SD. Comparisons between two discrete variables were performed by using the chi-square test or the Fisher exact test, and comparisons between two continuous variables by using the Student *t* test. Multivariate logistic regression techniques (backward-elimination method, elimination criterion, $p > 0.05$) were used to identify baseline clinical predictors of in-hospital death among the following variables: age, gender, tobacco use, history of hypertension, diabetes mellitus, peripheral arteriopathy or previous angina, ongoing treatments and heart rate, systolic arterial pressure and Killip class on hospital admission (complete data were available in 409 patients). Subsequent analyses were performed to identify baseline electrocardiographic predictors of death after adjusting for the clinical predictors, among the following variables: LVH; number of leads with ST segment depression; sum of ST segment depression; anterior, inferior or lateral ST

Table 1. Baseline Clinical Characteristics Among Patients With and Without In-Hospital Death

	In-Hospital Death (n = 22)	Discharged Alive (n = 410)	p Value
Age (years)	67 ± 7	59 ± 10	<0.001
Female gender	11 (50%)	82 (20%)	0.002
Active smoking	3 (14%)	196 (48%)	0.002
Diabetes mellitus	15 (68%)	74 (18%)	<0.001
Hypertension	12 (54%)	187 (46%)	NS
Hyperlipidemia	8 (36%)	151 (37%)	NS
Peripheral vascular disease	9 (41%)	44 (11%)	<0.001
Cerebrovascular disease	1 (4%)	16 (4%)	NS
Previous angina	21 (96%)	275 (67%)	0.005
Angina since >6 months	14 (64%)	98 (24%)	<0.001
Current use of nitrates	5 (23%)	46 (11%)	NS
Current use of beta-blockers	2 (9%)	34 (8%)	NS
Current use of calcium antagonists	8 (36%)	66 (16%)	0.03
Current use of aspirin	5 (23%)	22 (5%)	0.008
Heart rate (beats/min)	99 ± 28	77 ± 18	0.001
Systolic blood pressure (mm Hg)	129 ± 44	146 ± 29	NS
Killip class			<0.001
I	6 (27%)	376 (92%)	
II	6 (27%)	23 (6%)	
III	5 (23%)	8 (2%)	
IV	5 (23%)	0 (0%)	

Data are presented as the mean value ± SD or number (%) of patients.
NS = not significant.

segment depression; minor ST segment elevation; and negative T waves (415 patients had complete data). Wald chi-square values are reported, as well as multivariable odds ratios (ORs) and 95% confidence intervals (CIs) for the electrocardiographic variables. A two-tailed p value <0.05 was considered significant.

RESULTS

Baseline clinical variables associated with in-hospital death. Twenty-two patients (5.1%) died 12 ± 10 days after hospital admission—15 due to cardiogenic shock (in three, reinfarction was confirmed before death), two due to electromechanical dissociation and five after coronary artery bypass graft surgery (one with reinfarction before bypass surgery). Baseline clinical characteristics are summarized in Table 1. As compared with survivors, those who died in the hospital were older and more frequently female and had a lower prevalence of active smoking and a higher prevalence of diabetes mellitus, peripheral vascular disease and previous angina. Heart failure at presentation was also associated with in-hospital death. Multivariant analysis retained a higher Killip class (chi-square = 32.50, $p < 0.001$), a lower systolic arterial pressure (chi-square = 5.43, p = 0.020), peripheral vascular disease (chi-square = 4.97, p = 0.026) and absence of active smoking (chi-square = 4.89, p = 0.027) as predictors of in-hospital mortality.

Baseline electrocardiographic predictors of in-hospital death. The baseline ECG was normal in 54 patients (13%); it showed minor ST segment shifts or isolated T-wave changes in 149 patients (34%) and ≥0.1 mV of ST segment depression in 229 patients (53%). Table 2 summarizes the main electrocardiographic findings in patients who died during their hospital stay and in those who survived. The presence of ST segment depression on the admission ECG, as well as the magnitude and extent of ST segment depression, was associated with in-hospital death. The location of ST segment depression provided additional prognostic information, because only ST segment depression in the lateral or inferior leads was related to mortality. Other repolarization changes such as minor ST segment elevation or T-wave inversion were not associated with in-hospital death.

After adjusting for the significant baseline clinical predictors and for the electrocardiographic covariates, the presence of ST segment depression in two or more lateral leads remained the only variable from the admission ECG that was significant (chi-square = 5.15, p = 0.023) in predicting the risk of in-hospital death (OR 3.5, 95% CI 1.2 to 10.6).

Clinical significance of ST segment depression in lateral leads. Ninety-one patients (21% of the total group and 40% of those with ST segment depression) had lateral ST segment depression on the admitting ECG, which involved

Table 2. Baseline Electrocardiographic Findings in Patients With and Without In-Hospital Death

	In-Hospital Death (n = 22)	Discharged Alive (n = 410)	p Value
Sinus rhythm	19 (86%)	388 (95%)	NS
Right bundle branch block	2 (9%)	9 (2%)	NS
Left ventricular hypertrophy	3 (14%)	14 (3%)	0.05
ST segment depression ≥0.1 mV	17 (77%)	212 (52%)	0.02
Maximal ST segment depression (mV)	0.21 ± 0.14	0.12 ± 0.13	0.003
Sum of ST segment depression (mV)	0.81 ± 0.56	0.44 ± 0.50	0.001
No. of leads with ST segment depression ≥0.1 mV	3.7 ± 2.4	1.9 ± 2.3	<0.001
Anterior ST segment depression*	8 (36%)	115 (28%)	NS
Inferior ST segment depression*	7 (32%)	45 (11%)	0.01
Lateral ST segment depression*	13 (59%)	78 (19%)	<0.001
Minor (0.05 to 0.1 mV) ST segment elevation	6 (27%)	108 (26%)	NS
Negative T waves	3 (14%)	89 (22%)	NS

*Anterior, inferior and lateral ST segment depressions are defined as ≥0.1 mV of ST segment depression in at least two leads with their positive poles oriented anteriorly (V_1 to V_4), inferiorly (II, III and aVF) or leftward, respectively, either laterally or apically (I, aVL, V_5 and V_6).
Data are presented as the number (%) of patients or the mean value ± SD.
NS = not significant.

leads I and aVL in 7 patients, leads V_5 and V_6 in 65, all four of these leads in 12 and lead I or aVL and lead V_5 or V_6 in 7. Concomitant ST segment depression in other territories was absent in 36 patients (four died) but present in the anterior leads in 30 patients (four died), in the inferior leads in 10 (four died) and in the anterior plus inferior leads in 15 (one died). Maximal ST segment depression was located in the lateral leads in only 32 of these 91 patients, whereas it was located in the anterior leads in 53 patients and in the inferior leads in 6.

Besides an increased mortality rate, patients with lateral ST segment depression had higher rates of heart failure and recurrent ischemic events than did those without it (Table 3), although peak CK-MB levels were similar (129 ± 96 vs. 122 ± 92 IU/liter, p = NS). Thirty-day mortality was also higher in patients with lateral ST segment depression (14.3% vs. 4.2% in the remaining patients, p = 0.007), whereas the rates of 30-day reinfarction were similar (4.9% vs. 4.3%, p = NS). Categorization of patients with ST segment depression into lateral and nonlateral ST segment depression groups revealed distinct patterns of adverse events. Patients with lateral ST segment depression were at highest risk for most in-hospital complications, whereas those with ST segment depression not involving the lateral leads had a similar risk to those without significant ST segment shifts (Fig. 1). Among patients with lateral ST segment depression, the rates of death, reinfarction, severe heart failure and angina with electrocardiographic changes were, respectively, 0%, 14.3%, 0% and 28.6% in patients with ST segment depression in leads I and aVL; 13.8%, 1.5%, 15.4% and 16.9% in those with ST segment depression in leads V_5 and V_6; 16.7%, 0%, 16.7% and 8.3% in those with ST segment depression in all four leads; and 28.6%, 14.3%, 14.3% and 66.7% in those with ST segment depression in lead I or aVL and lead V_5 or V_6.

Cardiac catheterization was performed in 40 patients with ST segment depression in the lateral leads (44%) and in 175 patients without this electrocardiographic change

Table 3. In-Hospital Events in Patients With and Without Lateral ST Segment Depression on the Baseline ECG

	Lateral ST Segment Depression (n = 91)	No Lateral ST Segment Depression (n = 341)	p Value
Death	13 (14.3%)	9 (2.6%)	<0.001
Reinfarction, total	3 (3.3%)	9 (2.6%)	NS
Reinfarction, nonfatal	2 (2.2%)	5 (1.5%)	NS
Angina, total	26 (28.6%)	75 (22.0%)	NS
Angina with ECG changes	18 (20.0%)	39 (11.6%)	0.04
Pulmonary edema/cardiogenic shock	13 (14.3%)	14 (4.1%)	<0.001
Coronary revascularization	16 (17.6%)	35 (10.3%)	NS

Data are presented as the number (%) of patients.
NS = not significant.

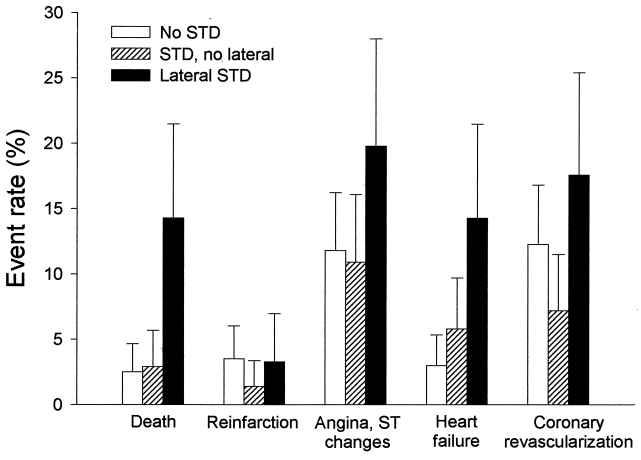

Figure 1. Rates of in-hospital events in patients without ST segment depression (STD), in those with ST segment depression not involving the lateral leads (I, aVL, V_5 and V_6) and in those with ST segment depression involving two or more lateral leads. Lateral ST segment depression identified a high risk subgroup, whereas patients with ST segment depression not involving the lateral leads had low complication rates, similar to those without ST segment depression. The 95% confidence limits are represented by error bars.

(51%, p = NS). Compared with the remaining patients, those with lateral ST segment depression had a lower left ventricular ejection fraction, more extensive CAD and a lower frequency of left circumflex coronary artery–related infarctions (Table 4).

DISCUSSION

This study suggests that in patients with a first acute MI without ST segment elevation, the presenting ECG contains important prognostic information based on the presence of ST segment depression in the lateral leads. In fact, this was the only variable from the initial ECG that was significant in predicting death after adjusting for the baseline predictors, whereas ST segment depression not involving the lateral leads did not carry an adverse prognosis. The relative value of lateral ST segment depression in predicting death was much lower than that provided by the presence of heart failure and was similar to the risk associated with arterial pressure, the presence of peripheral artery disease or the absence of active smoking. Patients with lateral ST segment depression also had increased rates of recurrent ischemia and heart failure, and their worse clinical outcome was not dependent on a larger infarct size, as they had similar peak CK-MB levels than did the remaining patients. In contrast, among patients who had left heart catheterization, those with lateral ST segment depression had more extensive CAD and a lower ejection fraction than did those without it.

Table 4. Cardiac Catheterization Data in Patients With and Without Lateral ST Segment Depression

	Lateral ST Segment Depression (n = 40)	No Lateral ST Segment Depression (n = 175)	p Value
Left ventricular ejection fraction*	57 ± 12%	66 ± 13%	0.001
Wall motion abnormalities*			
No	8 (23%)	75 (47%)	0.009
Anterior	8 (23%)	27 (17%)	NS
Posteroinferior	13 (37%)	53 (33%)	NS
Multiple segments	6 (17%)	5 (3%)	0.005
Number of diseased vessels			<0.001
0	2 (5%)	9 (5%)	
1	4 (10%)	78 (45%)	
2	11 (28%)	57 (33%)	
3	24 (60%)	37 (21%)	
LMCA or three-vessel disease	24 (60%)	39 (22%)	<0.001
Infarct-related coronary artery			
LMCA	1 (2%)	0 (0%)	NS
LAD	6 (15%)	34 (19%)	NS
LCx	4 (10%)	78 (45%)	<0.001
RCA	8 (20%)	21 (12%)	NS
Not identified	21 (52%)	41 (23%)	<0.001
Occluded infarct-related artery†	10 (46%)	49 (31%)	NS

*Values from the 195 patients who had a left ventriculogram. †Percentages correspond to the patients in whom the patency status of the infarct-related artery was determined.

Data are presented as the mean value ± SD or number (%) of patients.

LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; LMCA = left main coronary artery; NS = not significant; RCA = right coronary artery.

Prognostic significance of the location of ST segment depression. Although it has been established that ST segment depression on the baseline ECG in patients with non–ST segment elevation acute MI portends a worse outcome (1,6–16), little is known about the possible additional prognostic significance of the localization of ST segment depression, because the studies addressing this issue have had disparate results. Among 135 patients with non–Q wave MI, Kao et al. (18) reported that those with ST-T segment changes located in the anterior leads had a worse evolution after hospital discharge. Krone et al. (11) followed 1,234 patients who had a Q wave or non–Q wave MI and observed that anterior, lateral or inferior ST segment depression was associated with an increased one-year mortality rate, whereas ST segment depression in leads V_2 and V_3 with upright T waves was not. In 911 men with suspected unstable CAD, Nyman et al. (6) reported that the one-year risk of MI or death was comparable in patients with anterior, inferior or combined localization of ST-T segment changes, but was lower in those with isolated lateral localization. Finally, Cannon et al. (13) observed that in 1,289 patients with unstable angina or non–Q wave MI, those with anterior ST segment deviation had a worse one-year outcome than did the remaining patients.

The present finding of a worse outcome associated with lateral ST segment depression in non–ST segment elevation acute MI is also at variance with the results of these other studies. Some factors may help explain this discrepancy. First, our group was more homogeneous because we excluded patients with a previous MI or ST segment elevation, whereas in these other studies, the prevalence of previous MI ranged from 20% to 41%, and all of them included a variable proportion of patients with ST segment elevation. Second, some studies (6,18) considered the significance of pooled ST segment and T-wave changes, but not that of ST segment depression alone. Finally, in most of these studies (6,13,18), patients were categorized according to the predominant location of ST segment depression, which makes it difficult to assess the significance of the involvement of the lateral leads when other leads also had ST segment depression.

The present results would be in agreement, however, with recent observations in patients with Q wave inferior MI. Hasdai et al. (20) described that among 213 patients with an inferior acute MI, those with maximal ST segment depression in leads V_4 to V_6 were at higher risk for in-hospital death than were those with maximal ST segment depression in leads V_1 to V_3, and this finding was confirmed in a larger series of patients (21). Moreover, in these other studies, as in ours, the increased mortality in patients with lateral ST segment depression appeared to be independent of infarct size. This is consistent, in part, with the observation from the Global Utilization of Streptokinase and TPA for Occluded arteries (GUSTO-I) study, where patients with an inferior MI and ST segment depression in leads V_4 to V_6 had a similar 30-day and one-year mortality to those with ST segment depression in leads V_1 to V_3, despite the fact that they had smaller infarcts (22).

In our study, patients with lateral ST segment depression had a poorer left ventricular ejection fraction and more extensive CAD than did the remaining patients. Because patients with a previous MI were not included and peak CK-MB levels in those with and without lateral ST segment depression were similar, a likely explanation for the worse left ventricular function in patients with lateral ST segment depression could be that they had a larger area of ischemic but viable myocardium. The higher prevalence of multivessel disease in these patients is consistent with previous observations in patients with Q wave inferior acute MI and ST segment depression in the lateral leads (23,24). Birnbaum et al. (24) recently suggested that the left ventricular dysfunction in patients with maximal ST segment depression in leads V_1 to V_3 could be dependent on a larger infarct size, whereas in those with maximal ST segment depression in leads V_4 to V_6, it could reflect a contractile failure in noninfarcted segments related to extensive CAD.

Because lateral ST segment depression is a frequent finding in patients with LVH, and LVH is associated with a worse outcome after a non–Q wave acute MI (25), it might be argued that the adverse prognosis observed in our study of patients with lateral ST segment depression could be mainly related to an increased frequency of LVH. However, this explanation seems unlikely, because lateral ST segment depression was closely associated with more extensive CAD and remained significant in predicting death after adjusting for the diagnosis of LVH by QRS voltage criteria.

Study limitations. This is a retrospective study based on the experience of a single hospital, but it has been performed on a rather large number of consecutive patients who appear to be representative of patients with a first acute MI without Q waves and without ST segment elevation on hospital admission. The retrospective nature of the study prevented the documentation of left ventricular function and coronary anatomy in nearly 50% of our patients. This is a potential drawback, but in view of the fact that this exploration was carried out in similar proportions of patients with and without lateral ST segment depression, it is unlikely that the poorer left ventricular ejection fraction and the more severe CAD associated with lateral ST segment depression was due to a bias in the selection of patients for cardiac catheterization.

Clinical implications. This study describes an easily interpretable criterion that could be useful for early risk assessment in patients with non–ST segment elevation acute MI, if the present results are confirmed in the large data bases now available. Although ST segment depression on the admission ECG is a sensitive prognostic marker, its specificity is too low to identify patients at higher risk. In contrast, location of ST segment depression in the lateral leads allows a more efficient stratification, because it selects

a smaller high risk subgroup of patients and classifies a large number of those with ST segment depression as low risk. An additional advantage of this electrocardiographic criterion is that the worse outcome of patients with lateral ST segment depression does not appear to be consequent to a larger infarction but to a greater mass of jeopardized myocardium due to extensive CAD. Therefore, an aggressive antithrombotic or interventional approach could be potentially more beneficial in these patients than in patients without significant ST segment changes or in those with ST segment depression not involving the lateral leads.

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