

Clinical Research

Prognostic Value of ST-Segment Elevation in Posterior Precordial Leads (V_7 , V_8 , V_9) on the Initial ECG of Patients with Inferior Acute Myocardial Infarction who Received Thrombolysis

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Key words:
Acute inferior myocardial infarction, posterior leads, thrombolysis, prognosis.

Manuscript received:
January 15, 2003;
Accepted:
November 4, 2003.

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Introduction: Electrocardiographic diagnosis of a posterior wall infarction is difficult to accomplish through 12-lead ECG alone, especially in the acute setting. Furthermore, it is not possible to accurately estimate the size of infarction after thrombolytic therapy. The aim of this study was to assess the role of ST-segment elevation in posterior precordial leads (V_7 , V_8 , V_9) as an indicator for the initial diagnosis of a posterior wall acute myocardial infarction (MI) and as a prognostic factor after thrombolysis.

Methods: 160 patients (125 men – 35 women / mean age 64.5 ± 18.7 y) with an inferior acute MI, receiving thrombolytic therapy with rt-PA, were included in this study. They were divided into two groups according to the presence (Group A: 95 patients / 70 men – 25 women / mean age 61.5 ± 10.65 y) or absence (Group B: 65 patients / 55 men – 10 women / mean age 62.54 ± 12.65 y) of ST-segment elevation in leads V_7 , V_8 , V_9 on the initial ECG. Complete demographic and epidemiological data were recorded in all subjects, the infarct size was estimated (peak CPK/MB values), left ventricular function was assessed echocardiographically (through left ventricular ejection fraction [LVEF] values on the 6th day of hospitalization and 2 months later), the presence of complications was identified (reinfarction, cardiac failure, arrhythmias, post infarction angina, death) and the infarct-related artery patency was evaluated (catheterization was performed 15-30 days after MI) in all subjects.

Results: Group A patients had a much more extensive infarction (CPK values: 1651.6 ± 328.46 vs. 1372.71 ± 285.8 , $p=0.02$) resulting in much lower LVEF values (44.34 ± 8.5 vs. 52.6 ± 10.51 , $p<0.01$). They were more likely to present reinfarction, cardiac failure, post-infarction angina and/or death within the first 6 months of the follow up period ($p<0.05$). Post infarction coronary artery patency (TIMI 2,3) in group A was related to higher LVEF values (54.3 ± 12.2 vs. 44.6 ± 10.2 , $p<0.01$ on hospital discharge and 55.2 ± 8.4 vs. 49.2 ± 8.7 , $p<0.05$, two months later). In contrast, group B patients showed no significant differences regarding LVEF values during the follow up period (57.9 ± 8.8 vs. 55.6 ± 7.5 , $p=0.4$ on hospital discharge and 59.88 ± 9.6 vs. 58.5 ± 12.2 , $p=0.7$, two months later) independently of infarct-related artery patency.

Conclusion: ST-segment elevation in posterior precordial leads (V_7 , V_8 , V_9) on the initial ECG of patients with an inferior acute MI indicates an extensively affected area, possibly involving both inferior and posterobasal walls. Furthermore, there seems to be true medical benefit from the administration of early iv thrombolysis in those patients.

Electrocardiographic diagnosis of a posterior wall myocardial infarction (MI) is difficult to accomplish through the standard 12-lead ECG, especially during the acute phase.^{1,2} Although an infarction involving the posterior wall might occur as an isolated event, it is more often associated with an inferior myocardial infarction.³ Involvement of the posterior wall in a myocardial infarction is sometimes manifested with back pain, even though this is not a specific finding.

Unlike anterior wall MI, which is a fairly homogeneous entity, the extent of an inferior infarction depends on the infarct related artery (IRA) and its size. It can be defined as posterobasal, septal or posterolateral, but it can also involve 2-3 segments simultaneously.⁴ The IRA in almost 50% of cases is the right coronary artery (RCA), the occlusion of which is often accompanied by hemodynamic complications (hypotension and bradycardia).⁵ Treatment, and sometimes prevention, of these complications may be facilitated by the early identification of the IRA (right coronary or left circumflex artery).⁶

Diagnosis of a posterior wall infarction in the acute setting is typically based on the ECG detection of ST segment depression in leads V₁ to V₃.⁷ However, these changes are relatively insensitive and not specific for the diagnosis of an acute posterolateral infarction, since they may also represent inferoseptal infarction, anterior ischemia or non-Q wave MI.⁸

Taking into account the fact that the benefit of thrombolytic therapy is proportional to the amount of jeopardized myocardium,⁹ it becomes obvious that the early detection of posterior wall involvement in an inferior MI is of paramount importance for the therapeutic outcome.¹⁰ On the other hand, it is not possible to estimate either the extent of a posterior MI or the IRA using the standard 12-lead ECG, especially after thrombolysis.¹¹ Previous studies have shown that posterior ECG leads (V₇, V₈, V₉) can identify patients with posterior wall infarction.^{12,13} Thus, posterior leads might be useful during the acute phase of inferior MI.

The aim of our study was to assess the role of ST segment elevation in posterior wall leads on the admission ECG (V₇, V₈, V₉) for the diagnosis of posterior wall MI and the identification of IRA, as well as its prognostic value following thrombolysis.

Methods

The study group included 160 consecutive patients (125 male – 35 female, mean age 64.5±18.7 years)

with a first inferior MI. All patients received thrombolytic therapy with accelerated rt-PA, within 8 hours from symptom onset. The diagnosis of inferior MI was based on a history of chest pain lasting >30 min along with ST segment elevation ≥1 mm in at least two of the leads II, III and aVF on the admission ECG. The diagnosis was confirmed by our laboratory in all patients, with elevation of creatine kinase (CPK) levels above the normal value (<180 IU/l) and CK-MB fraction (10% of the total CK). CK and CK-MB levels were determined at hospital admission, every 4 hours during the first 24h and twice daily thereafter. Ten patients were excluded from the study: two patients with complete LBBB, two patients with contraindications for thrombolysis (one with previous hemorrhagic stroke and another with active upper gastrointestinal bleeding), three patients with previous by-pass surgery and three patients with an ECG pattern of left ventricular hypertrophy.

A 15-lead ECG was recorded at hospital admission before initiation of rt-PA and included, in addition to the standard 12 leads, three posterior chest leads (V₇, V₈, V₉). Leads V₇ to V₉ were recorded on the same horizontal plane as lead V₆—more specifically, lead V₇ on the posterior axillary line, lead V₈ on the posterior scapular line and lead V₉ on the left border of the spine. The ECG was performed using a three-channel ECG recorder so that the ECG from leads V₇ to V₉ was recorded simultaneously using standard electrodes for leads V₄₋₆ with the patient in the supine position. ST segment elevation ≥0.5 mm in the posterior chest leads was considered to be significant because of the greater distance separating the posterior chest wall from the heart.¹⁴ A decrease of ≥2 mm in the summed ST segment elevations within 2 hours from thrombolysis suggested successful reperfusion therapy, as previously proposed by other investigators.^{15,16}

Patients were divided into two groups according to the presence of ST segment elevation ≥0.5 mm on posterior leads V₇, V₈ and V₉ on the admission ECG (Group A: 95 patients, 70 male – 25 female, mean age 61.5±10.65 years) or not (Group B: 65 patients, 55 male – 10 female, mean age 62.44±12.65 years). Figure 1 shows a typical ECG from a patient in Group A.

A complete evaluation of the study population was made on admission, including clinical, epidemiological and biochemical data, as well as of the diagnostic and therapeutic interventions that follow-

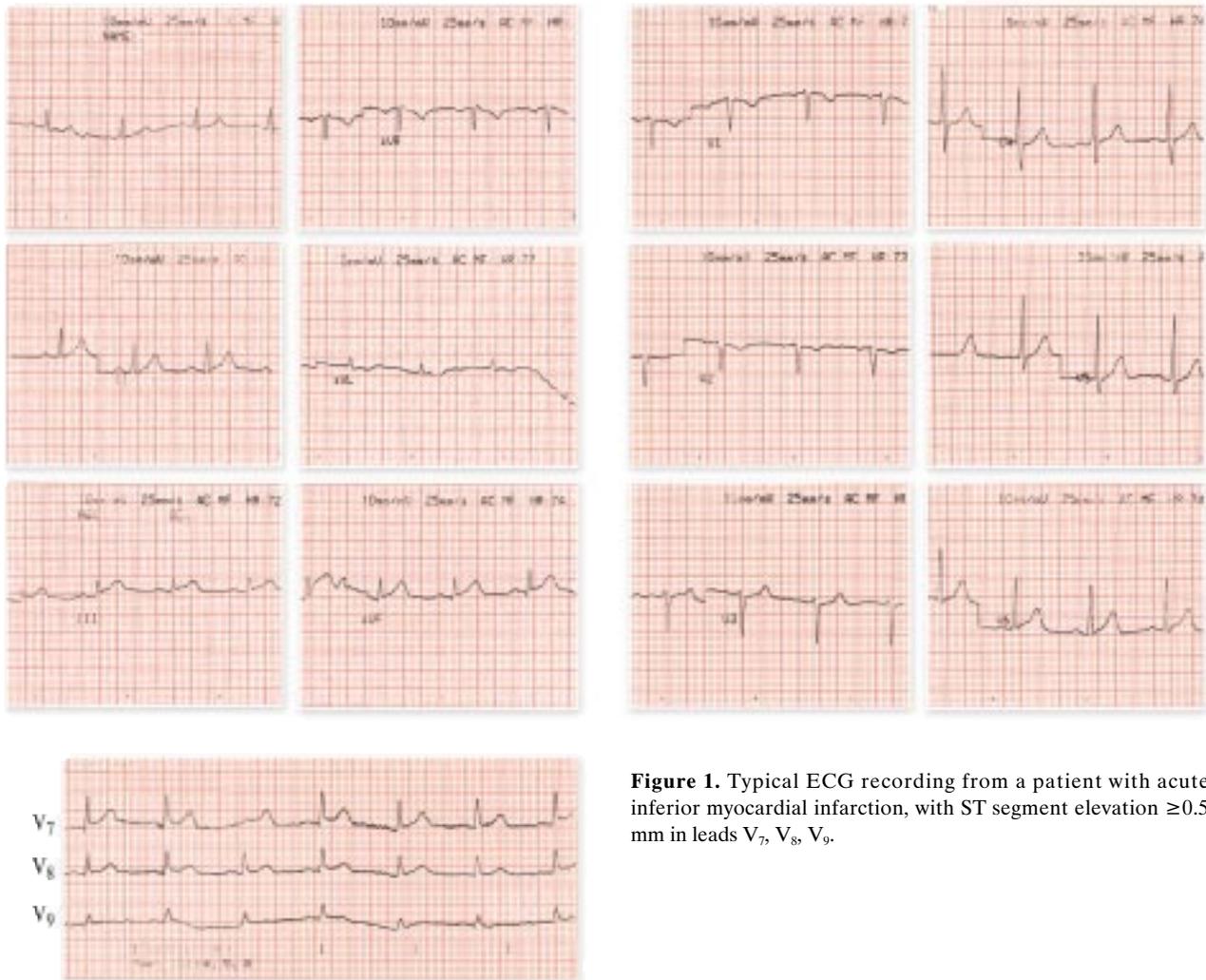


Figure 1. Typical ECG recording from a patient with acute inferior myocardial infarction, with ST segment elevation ≥ 0.5 mm in leads V₇, V₈, V₉.

ed (thrombolysis, catheterization). In addition, we analyzed the extent of MI (highest peak CPK/MB values), left ventricular function (echocardiographic measurement of left ventricular ejection fraction – LVEF, on the 6th day of hospitalization), the incidence of complications (reinfarction, postinfarction angina, congestive heart failure, arrhythmias) and mortality (in hospital and at 6 months follow-up).

All patients underwent coronary angiography 19.1 \pm 3.8 days after MI. The patency of the coronary arteries and in particular of the IRA was tested and documented by the following criteria:¹⁷ a) total or subtotal occlusion of a vessel that perfuses a hypokinetic or asynergic myocardial area, b) angiographic presence of thrombus or ruptured plaque in the vessel. It should be noticed here that four patients with significant stenosis in both right and left circumflex coronary arteries were excluded from the study, while the

presence of significant stenosis in major diagonal or marginal branches was considered left anterior descending or left circumflex artery disease, respectively. IRA patency was classified according to TIMI flow grading system criteria.¹⁷ More specifically, a patent vessel was defined as TIMI 2 or 3 and an occluded vessel as TIMI 0 or 1. Significant coronary artery disease was defined as a decrease of more than 50% of the lumen diameter on any angiographic projection.¹⁸ In this way the number of coronary arteries with significant stenosis and the extent of coronary disease were assessed in each patient.

Statistical analysis

Continuous clinical and epidemiological variables are presented as mean \pm SD and were analyzed using the Student t-test, whereas discrete variables were com-

pared using either chi-square analysis or Fischer exact tests. The independent prognostic value of ST segment elevation in posterior leads V₇, V₈, V₉ at admission time was also analyzed. A model of multivariate analysis was used (forward stepwise multiple regression model), in order to examine the impact of several parameters, such as gender, age (>65 years), risk factors (smoking, hypertension, dyslipidemia, diabetes), history of coronary artery disease, ST elevation in posterior leads V₇, V₈, V₉ and ST depression in leads V₁-V₃ on admission ECG (independent variables), on six-month mortality and on the incidence of complications (dependent variables).

In addition, in-hospital and 6-month clinical course (reinfarction, post infarction angina, arrhythmias, congestive heart failure and death) were analyzed using the same statistical model, in order to determine its correlation with the above mentioned independent variables.

All tests of significance were 2-tailed and p values of <0.05 were considered statistically significant. The statistical analysis was performed using the SPSS, version 9.0 for Windows, statistical package.

Results

Baseline and epidemiological characteristics of both groups of patients are listed in table 1. There were no significant differences, apart from the presence of preinfarction angina, which was more frequently encountered in group A patients (n= 27 [28.42%] vs. n=8 [12.3%], p=0.02).

Group A patients had significantly larger infarcts (higher peak CPK levels) compared with group B (CPK: 1651.6±328.46 IU/lit vs. 1372±285.82 IU/lit, p=0.02), associated with greater left ventricular systolic dysfunction (echocardiographic measurement

Table 2. Clinical, biochemical, electrocardiographic and echocardiographic characteristics.

	Group A n=95	Group B n=65	p
Interval from onset of symptoms to thrombolysis (min)	123.3±22.2	120.4±18.3	0.2
CPK (IU)	1651.6±328.46	1372±285.82	0.02
CPK-MB (IU)	185.6±83.75	95.72±19.28	0.02
LVEF (%)	44.34±8.5	52.6±10.51	0.01
ST depression (V₁-V₃) n (%)	23 (24.2)	10 (15.4)	0.12
Hypokinetic - Dyskinetic wall segments, n (%)	80 (84.2)	27 (41.54)	0.03

LVEF-left ventricular ejection fraction.

of LVEF=44.34±8.5% vs. 52.6±10.51%, p=0.01). The difference in left ventricular function was attributed to the greater hypokinesia of posterolateral wall that was found in group A (n=80 [84.2%] vs. n=27 [41.54%], p=0.03). Table 2 summarizes the clinical, biochemical, electrocardiographic and echocardiographic characteristics of patients.

Group A patients had a more complicated in-hospital course and greater incidence of adverse effects at 6 months of follow up, as shown in table 3.

The IRA was identified through coronary angiography in 157 patients (98.12%). In three patients (one in group A and two in group B) no significant stenosis was found. The left circumflex coronary artery was the IRA in a significantly higher proportion of patients in group A (n=41, 43.16%) than in group B (n=8, 12.3%, p=0.001), whereas the right coronary artery was the IRA more frequently in group B (n=54, 83.08%) than in group A (n=52, 54.74%,

Table 1. Baseline and epidemiological characteristics.

	Group A n=95	Group B n=65	p
Age (yr)	61.5±10.65	62.54±12.65	0.1
Gender M/F, n (%)	70/25 (73.68/26.32)	55/10 (84.6/15.4)	0.2
Smoking, n (%)	68 (71.58)	46 (70.77)	0.6
Dyslipidemia, n (%)	29 (30.53)	19 (29.23)	0.7
Hypertension, n (%)	17 (17.89)	14 (21.54)	0.3
Diabetes mellitus, n (%)	10 (10.52)	8 (12.3)	0.4
Preinfarction angina, n (%)	27 (28.4)	8 (12.3)	0.02

Table 3. Complications - Mortality.

	Group A n=95	Group B n=65	p
Reinfarction, n (%)	12 (12.63)	4 (6.15)	0.03
Post infarction Angina, n (%)	4 (4.21)	2 (3.08)	0.1
Congestive heart failure, n (%)	6 (6.32)	2 (3.08)	0.05
Arrhythmias, n (%)	2 (2.1)	1 (1.54)	0.1
Complicated clinical course, n (%)	20 (21.05)	4 (6.15)	0.04
Mortality 6 months, n (%)	4 (4.21)	1 (1.54)	0.05
Invasive therapy 60 days, n (%)	10 (10.53)	2 (3.08)	0.03

Table 4. Angiographic findings.

	Group A n=95	Group B n=65	p
Time to catheterization(days)	15.3±10.7	14.5±11.3	0.8
Patent IRA (TIMI 2,3), n (%)	62 (65.26)	44 (67.69)	0.7
LCx, n (%)	41 (43.16)	8 (12.3)	0.001
RCA, n (%)	52 (54.74)	54 (83.08)	0.02
LAD, n (%)	1 (1.05)	1 (1.59)	0.8
Multivessel CAD, n (%)	42 (44.2)	28 (43.08)	0.8
Undetermined vessel, n (%)	1 (1.05)	2 (3.08)	0.8

IRA- infarct related artery, LCx-left circumflex artery, RCA-right coronary artery, LAD-left anterior descending coronary artery, CAD-coronary artery disease.

p=0.02). No differences were found between the two groups of patients concerning TIMI flow score 2,3 of the IRA and multivessel disease. The angiographic findings are shown in table 4.

In patients presenting with concomitant ST segment elevation in the posterior leads (V₇, V₈, V₉) IRA patency was associated with higher LVEF values on hospital discharge (54.3%±12.2% with TIMI 2,3 vs. 44.6%±10.2% with TIMI 0,1: p<0.01) and at 30 days of follow up (55.2%±8.4% with TIMI 2,3 vs. 49.2±10.2% with TIMI 0,1: p=0.05). In

contrast, in group A, LVEF was similar regardless of IRA patency on hospital discharge (57.9±8.8% with TIMI 2,3 vs. 55.6±7.9% with TIMI 0,1: p=0.4) and on 30 days' follow-up visit (59.8±9.6% with TIMI 2,3 vs. 58.9±12.2% with TIMI 0,1: p=0.6). The relationship between successful thrombolysis, left ventricular function and ST segment elevation in posterior leads (V₇ to V₉) is shown in figure 2.

Multivariate analysis (forward stepwise multiple regression) showed that arrhythmias (25.5%: F=54.1, p<0.0001), congestive heart failure (13.9%: F=29.8, p<0.0001) and ST segment elevation in posterior leads on admission ECG (11.3%: F=5.85%, p=0.017) were independent prognostic factors for mortality at 6 months of follow-up. In contrast, ST segment depression in leads V₁-V₃ did not have any impact on mortality. Thus, the higher mortality rates seen at 6 months of follow-up correlated significantly and independently with the presence of arrhythmias, congestive heart failure and ST segment elevation in posterior leads (V₇ to V₉) on the admission ECG (Table 5).

Using the same method we found that ST segment elevation in the posterior leads on admission ECG (19.1%: F=11.7, p= 0.001), a history of coronary artery disease (5.2%: F=8.6, p=0.004) and diabetes mellitus (2.9%: F=3.9, p=0.049) were independent prognostic factors for complicated in-hospital course (reinfarction, congestive heart failure, postinfarction angina, arrhythmias, death).

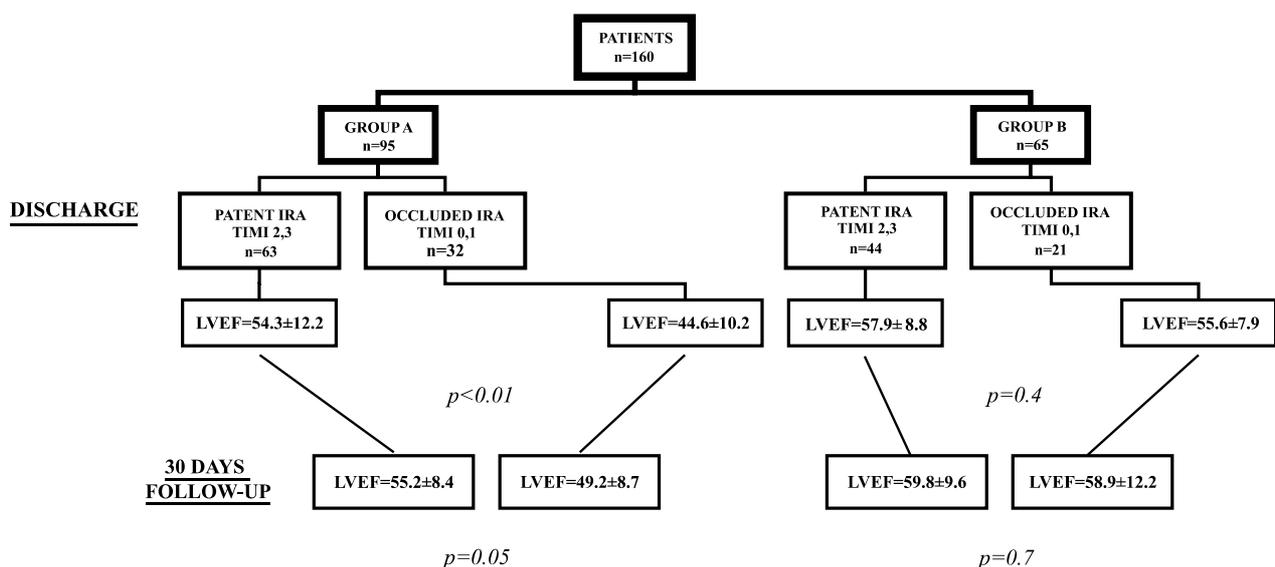


Figure 2. Flowchart illustrating distribution of patients with and without ST segment elevation in leads V₇, V₈, V₉ at hospital admission according to infarct related artery (IRA) patency and left ventricular ejection fraction (LVEF) (pre-discharge and at 30 days of follow-up).

Table 5. Stepwise multivariate regression coefficients for predicting mortality at 6 months follow up (only significant variables included).

Variable	R	B	Standard Error b	Beta	t	p
Arrhythmias	0.50	0.57	0.09	0.45	6.97	<0.0001
CHF	0.64	0.26	0.05	0.35	5.46	<0.0001
ST segment elevation (V ₇ -V ₈ -V ₉)	0.69	0.17	0.04	0.16	2.42	0.017

CHF-congestive heart failure

These findings demonstrate that ST segment elevation on admission ECG in the posterior leads (V₇ to V₉), a history of coronary artery disease and, finally, diabetes mellitus were independent prognostic factors for the combined clinical end point at 6 months of follow-up (Table 6).

Discussion

The results of this study indicate that ST segment elevation in posterior leads (V₇, V₈, V₉) on the admission ECG in patients with an acute inferior MI suggests the presence of concomitant posterolateral MI. We studied consecutive patients whose admission ECGs were obtained about 2 hours after symptom onset, just before the beginning of thrombolysis, by which time the ST segment elevation recorded was more likely to be the peak one.

The clinical importance of ST segment elevation in posterior leads has not yet been clarified, even though ECG findings can identify a subset of high risk patients with left circumflex occlusion responsible for a larger MI and a more complicated clinical

Table 6. Stepwise multivariate regression coefficients for predicting combined complicated clinical course at 6 months follow up (only significant variables included).

Variable	R	B	Standard Error b	Beta	t	p
ST segment elevation (V ₇ -V ₈ -V ₉)	0.26	0.17	0.07	0.19	2.5	0.001
History of CAD	0.34	0.26	0.09	0.23	3.05	0.004
Diabetes mellitus	0.37	0.11	0.06	0.15	1.98	0.049

CAD-coronary artery disease.

course. In general, its prognostic implications are not widely known, since Matesky et al¹⁹ found that ST segment elevation in posterior leads is combined with a larger MI and a more complicated clinical course (reinfarction, heart failure, mortality), whereas Zalenski et al²⁰ failed to show any difference in the incidence of ST segment elevation in posterior leads, in acute MI patients with or without a complicated clinical course.

These ECG findings provide greater accuracy for the diagnosis of posterolateral MI than the associated ST depression in precordial leads.^{21,22} Such precordial ST segment depression is not specific and may also reflect anterior wall ischemia or non-Q wave MI.²³ Besides, precordial ST segment depression may sometimes be masked by the development of complete RBBB or may be diminished due to right ventricular infarction.²⁴ Consequently, this ECG sign is of low sensitivity and specificity for the diagnosis of posterior wall infarction.²⁵

In our study, ST segment elevation in the posterior leads (V₇, V₈, V₉) was associated with a larger inferior MI, as estimated by higher CPK/MB levels and poorer left ventricular function. In addition, these patients had a more complicated clinical course, with a higher incidence of reinfarction, postinfarction angina, heart failure and/or death at 6 months of follow-up. Moreover, according to multivariate analysis, ECG changes involving the posterior leads are independent prognostic factors for a more complicated clinical course during the 6 months' follow up.

In addition to the above, we have demonstrated that left circumflex artery occlusion was more frequently implicated in cases of inferior infarcts presenting with ST segment elevation in the posterior leads. This finding is consistent with the results of previous studies,^{26,27} and explains the absence of high degree atrioventricular block (almost entirely restricted to right coronary-artery related infarction),²⁸ a complication seen more often in group B than in group A patients.

In general, the 12-lead ECG is less sensitive in identifying left circumflex occlusion. Huey et al found that 52% of patients with acute MI from left circumflex disease did not show any ST segment elevation,²⁶ while other investigators recently reported that acute left circumflex occlusion either does not bring about any changes at all in the standard 12-lead ECG²⁹ or generates only ST depression in the precordial leads.³⁰ Scintigraphic studies showed that

thallium myocardial perfusion defects in posterolateral segments are relatively specific for left circumflex occlusion.³¹ Thus, posterior leads may contribute to the regional diagnosis of an acute inferior MI.

A recent study showed that posterior lead recordings may lead to an increase in diagnoses of acute MI (30,000 patients/year in the USA with ST segment elevation in the posterior leads) when the IRA is the left circumflex artery, resulting in a survival benefit of 500 patients/year through thrombolysis administration.^{32,33} The same study claims that an increase in posterior lead sensitivity from 57.7% to 59.7% could lead to a beneficial clinical outcome. Another study showed that the criterion of ST segment ≥ 0.5 mm in the 15 lead ECG (12 classic and V₇, V₈, V₉) can improve the sensitivity of the diagnosis of acute coronary syndromes attributed to left circumflex occlusion by at least 94%.³⁴

An equally interesting result of this study is that extensive MIs presenting with ST segment elevation in the posterior leads were associated with more favorable effects from thrombolytic therapy administration. In particular, these patients showed significant improvement of LVEF due to a patent IRA (TMI flow 2,3). Both recent and previous studies^{35,36} showed that the percentage of myocardial salvage through thrombolysis is proportional to the size of the initial jeopardized myocardium. Thus, the concomitant posterolateral involvement in patients with ST segment elevation in leads V₇, V₈, V₉ on hospital admission ECG, indicative of a larger infarct, presumably accounts for the greater beneficial effect seen in these patients. Therefore, thrombolytic therapy is strongly indicated in such patients, since an occluded artery leads to a low LVEF, whereas in the case where the MI is limited to the inferior wall, thrombolysis might be elective (relative contraindications for thrombolysis) since even an occluded vessel is associated with a good LVEF. Moreover, in patients with an inferior infarction, posterolateral involvement is associated with the development of significant mitral regurgitation. Successful thrombolysis reduces the prevalence and severity of mitral regurgitation in conjunction with a reduction in the prevalence of posterior wall motion abnormalities, leading to an improvement of total wall motion.³⁷ This beneficial effect is preserved for at least 2 months after the event.

Previous studies that evaluated the results of thrombolytic therapy in the treatment of inferior MIs demonstrated only an insignificant trend towards

mortality reduction and yielded conflicting conclusions with respect to the resulting improvement in left ventricular function.^{38,39} Nevertheless, only a few studies have evaluated the beneficial effect of thrombolysis in high risk subsets of patients with an inferior MI.⁴⁰ Bar et al⁴¹ showed that in patients with inferior MI and summed ST segment elevation >6 mm infarct size limitation was greater after thrombolysis than in patients with a lower summed ST segment elevation. However, they did not extrapolate their results in terms of ventricular function.

Until now, more than 150,000 patients have been included in randomized multicenter studies that compared thrombolytic therapy with control groups in terms of mortality and thrombolytic regimens. According to the FTT trial, in cases where patients presented within 6 hours from symptom onset with ST segment elevation or newly diagnosed bundle branch block there were 30 lives saved per 1000 treated, whereas for those presenting between 7 to 12 hours from symptom onset the benefit was 20 lives saved per 1000 treated. Late thrombolysis, beyond 12 hours, showed no benefit from thrombolytic therapy. Moreover, the greatest total benefit was seen in high risk patients (as reported from prognostic parameters – admission ECG, echocardiogram, peak CPK/MB etc.).⁴² Finally, a recent meta analysis of FTT showed that thrombolysis reduced mortality even in patients older than 75 years (from 29.4% to 26%, $p=0.03$).⁴³

Berland et al⁴⁴ suggested that post reperfusion improvement of segmental contraction occurs only in patients who had concomitant precordial lead ST segment depression on the admission ECG, but failed to show similar results regarding LVEF. Finally, Bates et al,⁴⁵ along with more recent studies,⁴⁶ postulated an insignificant improvement in either regional or global left ventricular function in patients with an inferior MI after thrombolytic therapy, regardless of ST segment depression in leads V₁ to V₄.

Patients in our study underwent cardiac catheterization by the 19th day from admission, on average. Thus, only patency and not early reperfusion could be estimated. However, the similar timing of catheterization along with the similar incidence of complications (mainly reinfarction) in both groups, as well as the presence of clinical signs of reperfusion in patients with patent IRA (TIMI flow 2,3), suggest that the inter-group differences in left ventricular function response to patency reflect differences in the beneficial effects of thrombolysis.

The present study is limited by its rather small sample size and larger studies are needed to confirm the beneficial effects of thrombolysis in clinical (complications) and mechanical (functionality, LVEF) terms, in patients with acute inferior MI and concomitant involvement of posterolateral wall as defined from posterior leads.

Clinical applications

Based on the results of the present study we recommend routine recording of posterior leads (V_7 , V_8 , V_9) in all patients admitted to the coronary unit with an acute inferior MI. In this way posterolateral wall involvement may be estimated, as right precordial leads help in the diagnosis of right ventricular involvement during an acute inferior MI. Moreover, the European Society of Cardiology suggests recording of additional leads in selected cases (diagnosis of true posterior infarction).⁴⁷ ST segment elevation in posterior leads is associated with a large area of jeopardized myocardium and strongly implies a need for thrombolytic therapy (especially in district hospitals without facilities for catheter-based reperfusion).⁴⁸

Conclusion

ST segment elevation in posterior ECG leads (V_7 , V_8 , V_9) in patients with acute inferior MI is associated with a greater extent of jeopardized myocardium and possible involvement of posterolateral wall. These patients seem to benefit more from thrombolytic therapy.

References

1. Trzeciak S, Erickson T, Bunney EB, Sloan EP: Variation in patient management based on ECG interpretation by emergency medicine and internal medicine residents. *Am J Emerg Med* 2002; 20: 188-195.
2. Hathaway WR, Peterson ED, Wagner GS, Granger CB, Zabel KM, Pieper KS, et al: Prognostic significance of the initial electrocardiogram in patients with acute myocardial infarction. GUSTO-I Investigators. *Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries*. *JAMA* 1998; 279: 387-391.
3. Menown IB, Allen J, Anderson JM, Adgey AA: Early diagnosis of right ventricular or posterior infarction associated with inferior wall left ventricular acute myocardial infarction. *Am J Cardiol* 2000; 85: 934-938.
4. Hudson MP, Cohen MG, Maynard C, Patterson J, Campbell PT, Kruse K, et al: Electrocardiographic comparison of myocardial salvage with primary revascularization versus thrombolysis in inferior myocardial infarction. *J Electrocardiol* 2002; 35: 11-18.
5. Webb JG, Sleeper LA, Buller CE, Boland J, Palazzo A, Buller E, et al: Implications of the timing of onset of cardiogenic shock after acute myocardial infarction: a report from the SHOCK Trial Registry. *SHould we emergently revascularize Occluded Coronaries for cardiogenic shock?* *J Am Coll Cardiol* 2000; 36: 1084-1090.
6. Berger PB, Ryan TJ: Inferior myocardial infarction: high risk subgroups. *Circulation* 1990; 81: 401-411.
7. Kornreich F, Montague TJ, Rautaharju PM. Body surface potential mapping of ST segment changes in acute myocardial infarction. Implications for ECG enrollment criteria for thrombolytic therapy. *Circulation* 1993; 87: 773-782, Comment in: *Circulation*. 1993; 87: 1040-1042.
8. Putini RL, Natale E, Ricci R, Minardi G, Tubaro M, Lioy E, et al: Dipyridamole echocardiography evaluation of acute inferior myocardial infarction with concomitant anterior ST segment depression. *Eur Heart J* 1993; 14: 1328-1333.
9. Hackworthy RA, Vogel MB, Harris PJ: Influence of infarct artery patency on the relation between initial ST segment elevation and final infarct size. *Br Heart J* 1986; 56: 222-225.
10. Brady WJ, Erling B, Pollack M, Chan TC: Electrocardiographic manifestations: acute posterior wall myocardial infarction. *J Emerg Med* 2001; 20: 391-401.
11. Brady WJ: Acute posterior wall myocardial infarction: electrocardiographic manifestations. *Am J Emerg Med* 1998; 16: 409-413.
12. Schmitt C, Lehmann G, Schmieder S, Karch M, Neumann FJ, Schomig A: Diagnosis of acute myocardial infarction in angiographically documented occluded infarct vessel: limitations of ST-segment elevation in standard and extended ECG leads. *Chest* 2001; 120: 1540-1546.
13. Wung SF, Drew BJ: New electrocardiographic criteria for posterior wall acute myocardial ischemia validated by a percutaneous transluminal coronary angioplasty model of acute myocardial infarction. *Am J Cardiol* 2001; 87: 970-974.
14. Tamura A, Kataoka H, Nagase K, Mikuriya Y, Nasu M: Clinical significance of inferior ST elevation during acute anterior myocardial infarction. *Br Heart J* 1995; 74: 611-614.
15. Anderson RD, White HD, Ohman EM, Wagner GS, Krucoff MW, Armstrong PW, et al: Predicting outcome after thrombolysis in acute myocardial infarction according to ST-segment resolution at 90 minutes: a substudy of the GUSTO-III trial. *Global Use of Strategies To Open occluded coronary arteries*. *Am Heart J* 2002; 144: 81-88.
16. Dong J, Ndrepepa G, Schmitt C, Mehilli J, Schmieder S, Schwaiger M, et al: Early resolution of ST-segment elevation correlates with myocardial salvage assessed by Tc-99m sestamibi scintigraphy in patients with acute myocardial infarction after mechanical or thrombolytic reperfusion therapy. *Circulation* 2002; 105: 2946-2949.
17. de Lemos JA, Antman EM, Giugliano RP, McCabe CH, Murphy SA, Van de Werf F, et al: ST-segment resolution and infarct-related artery patency and flow after thrombolytic therapy. *Thrombolysis in Myocardial Infarction (TIMI) 14 investigators*. *Am J Cardiol* 2000; 85: 299-304.
18. Matetzky S, Novikov M, Gruberg L, Freimark D, Feinberg M, Elian D, et al: The significance of persistent ST elevation

- versus early resolution of ST segment elevation after primary PTCA. *J Am Coll Cardiol* 1999; 34: 1932-1938.
19. Matetzky S, Freimark D, Chouraqui P, Rabinowitz B, Rath S, Kaplinsky E, et al: Significance of ST segment elevations in posterior chest leads (V_7 to V_9) in patients with acute inferior myocardial infarction: Application for thrombolytic therapy. *J Am Coll Cardiol* 1998; 31: 506-511.
 20. Zalenski RJ, Rydman RJ, Sloan EP, Hahn K, Cooke D, Tucker J, et al: ST segment elevation and the prediction of hospital life-threatening complications: the role of right ventricular and posterior leads. *J Electrocardiol* 1998; 31 (Suppl): 164-171.
 21. Gibson CM, Chen M, Angeja BG, Murphy SA, Marble SJ, Barron HV, et al: Precordial ST-Segment Depression in Inferior Myocardial Infarction is Associated with Slow Flow in the Non-Culprit Left Anterior Descending Artery. *J Thromb Thrombolysis* 2002; 13: 9-12.
 22. Birnbaum Y, Wagner GS, Barbash GI, Gates K, Criger DA, Sclarovsky S, et al: Correlation of angiographic findings and right (V_1 to V_3) versus left (V_4 to V_6) precordial ST-segment depression in inferior wall acute myocardial infarction. *Am J Cardiol* 1999; 83: 143-148.
 23. Porter A, Vaturi M, Adler Y, Sclarovsky S, Strasberg B, Herz I, et al: Are there differences among patients with inferior acute myocardial infarction with ST depression in leads V_2 and V_3 and positive versus negative T waves in these leads on admission? *Cardiology* 1998; 90: 295-298.
 24. Drew BJ, Adams MG, Pelter MM, Wung SF, Caldwell MA: Comparison of standard and derived 12-lead electrocardiograms for diagnosis of coronary angioplasty-induced myocardial ischemia. *Am J Cardiol* 1997; 79: 639-644.
 25. Assali AR, Hertz I, Vaturi M, Adler Y, Solodky A, Birnbaum Y, et al: Electrocardiographic criteria for predicting the culprit artery in inferior wall acute myocardial infarction. *Am J Cardiol* 1999; 84: 87-89.
 26. Huey BL, Beller GA, Kaiser DL, Gibson RS: A comprehensive analysis of myocardial infarction due to left circumflex artery occlusion: comparison with infarction due to right coronary artery and left anterior descending artery occlusion. *J Am Coll Cardiol* 1988; 12: 1156-1166.
 27. Bough EW, Korr KS: Prevalence and severity of circumflex coronary artery disease in electrocardiographic posterior myocardial infarction. *J Am Coll Cardiol* 1986; 7: 990-996.
 28. Kosuge M, Kimura K, Ishikawa T, Nakatogawa T, Saito T, Okuda J, et al: Clinical features of patients with reperfused inferior wall acute myocardial infarction complicated by early complete atrioventricular block. *Am J Cardiol* 2001; 88: 1187-1191.
 29. Agarwal JB, Khaw K, Aurignac F, LoCurto A: Importance of posterior chest leads in patients with suspected myocardial infarction, but nondiagnostic, routine 12-lead electrocardiogram. *Am J Cardiol* 1999; 83: 323-326.
 30. Jacobs AK, French JK, Col J, Sleeper LA, Slater JN, Carnendran L, et al: Cardiogenic shock with non-ST-segment elevation myocardial infarction: a report from the SHOCK Trial Registry. Should we emergently revascularize Occluded coronaries for Cardiogenic shock? *J Am Coll Cardiol* 2000; 36(3 Suppl A): 1091-1096.
 31. Newman NH, Dunn RF, Harris PJ, Bantovich GJ, McLanghlin AF, Kelly DT: Differentiation between right and circumflex coronary artery disease in thallium myocardial scanning. *Am J Cardiol* 1983; 51: 1052-1056.
 32. Matetzky S, Freimark D, Feinberg MS, Novikov I, Rath S, Rabinowitz B, et al: Acute myocardial infarction with isolated ST-segment elevation in posterior chest leads V7-9: "hidden" ST-segment elevations revealing acute posterior infarction. *J Am Coll Cardiol* 1999; 34: 748-753. Comment in: *J Am Coll Cardiol* 2000; 36: 658-659.
 33. Zalenski RJ, Rydman RJ, Sloan EP, Hahn K, Cooke D, Fagan J, et al: Value of posterior and right ventricular leads in comparison to the standard 12-lead Electrocardiogram in evaluation of ST-Segment elevation in suspected acute myocardial infarction. *Am J Cardiol* 1997; 79: 1579-1585.
 34. Wung SF, Drew BJ: Comparison of 18-lead ECG and selected body surface potential mapping leads in determining maximally deviated ST lead and efficacy in detecting acute myocardial ischemia during coronary occlusion. *J Electrocardiol* 1999; 32 Suppl: 30-37.
 35. Mauri F, Franzosi MG, Maggioni AP, Santoro E, Santoro L: Clinical value of 12-lead electrocardiography to predict the long-term prognosis of GISSI-1 patients. *J Am Coll Cardiol* 2002; 39: 1594-1600.
 36. Velury VS, Ma Y, Hurley T, Hebert JR, Becker CF, Becker RC: Clinical Utility of Electrocardiographic ST-Segment Area for Predicting Unsatisfactory Outcomes Following Thrombolytic Therapy. *J Thromb Thrombolysis* 1995; 2: 51-56.
 37. Tenenbaum A, Leor J, Motro M, Hod H, Kaplinsky E, Rabinowitz B, et al: Improved posterobasal segment function after thrombolysis is associated with decreased incidence of significant mitral regurgitation in a first inferior myocardial infarction. *J Am Coll Cardiol* 1995; 25: 1558-1563.
 38. O'Rourke M, Baron D, Keogh A, et al: Limitations of myocardial infarction by early infusion of recombinant tissue-type plasminogen activator. *Circulation* 1988; 77: 311-315.
 39. Zehender M, Kasper W, Kauder E, Geibel A, Schonthaler M, Olschewski M, et al: Eligibility for and benefit of thrombolytic therapy in inferior myocardial infarction: focus on the prognostic importance of right ventricular infarction. *J Am Coll Cardiol* 1994; 24: 362-369.
 40. Arnold AE, Simoons ML: "Expected infarct size without thrombolysis", a concept that predicts immediate and long-term benefit from thrombolysis for evolving myocardial infarction. *Eur Heart J* 1997; 18: 1736-1748.
 41. Bar FW, Vermeer F, de Zwaan C, et al: Value of admission electrocardiogram in predicting outcome of thrombolytic therapy in acute myocardial infarction: a randomized trial conducted by The Netherlands InterUniversity Cardiology Institute. *Am J Cardiology* 1987; 59: 6-13.
 42. Fibrinolytic Therapy Trialists (FTT) Collaborative Group: Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomized trials of more than 1000 patients. *Lancet* 1994; 343: 311-322.
 43. White H: Thrombolytic therapy in the elderly. *Lancet* 2000; 356: 2028-2030.
 44. Berland J, Cribier A, Behar P, Letac B: Anterior ST depression in inferior myocardial infarction: correlation with results of intracoronary thrombolysis. *Am Heart J* 1986; 111: 481-488.
 45. Bates ER, Clemmensen PM, Califf RM, Gorman LE, Aronson LG, George BS, et al: Precordial ST segment depression predicts a worse prognosis in inferior infarction despite re-

- perfusion therapy. The Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) Study Group. *J Am Coll Cardiol* 1990; 16: 1538-1544.
46. Bellotti G, Rochitte CE, de Albuquerque CP, Lima JA, Lopes N, Kalil-Filho R, et al: Usefulness of ST-segment depression in non-infarct-related electrocardiographic leads in predicting prognosis after thrombolytic therapy for acute myocardial infarction. *Am J Cardiol* 1997; 79: 1323-1328.
47. Van de Werf F, Ardissino D, Betriu A, Cokkinos D, Falk E, et al for the Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. *Eur Heart J* 2003; 24: 28-66.
48. Casas RE, Marriot HJ, Glancy DL: Value of leads V sub 7-V sub 9 in diagnosing posterior wall acute myocardial infarction and other causes of tall R waves in V sub 1- V sub 2. *Am J Cardiol* 1997; 80: 508-510.