Implications of precordial ST segment depression during acute inferior myocardial infarction

Arteriographic and ventriculographic correlations during the acute phase

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SUMMARY Thirty two patients presenting with acute transmural inferior wall myocardial infarction underwent cardiac catheterisation and angiography within 12 hours of the onset of symptoms. Twelve lead electrocardiograms performed within one hour of catheterisation showed ST segment depression in the anterior precordial leads in addition to inferior wall changes in 17 patients and no ST segment changes in the anterior leads in 15. When the clinical, arteriographic, and ventriculographic variables were compared between the two groups no significant differences were noted with regard to age, sex, risk factors for coronary disease, duration of symptoms before angiography, Killip class, number of inferior leads with ST segment elevation, or initial serum creatine kinase activity. The extent of coronary artery disease as well as the prevalence of severe disease in the left anterior descending artery were similar for both groups. Biplane left ventriculography showed no significant differences between the two groups with regard to global ejection fraction or to the prevalence of posterolateral or anterior segmental wall motion abnormalities.

Patients presenting with acute transmural inferior infarction often have ST segment depression in the anterior precordial leads. Investigators have suggested that, in contrast to patients with inferior infarction without additional precordial ST depression, patients with anterior changes have larger infarctions, more extensive coronary artery disease, and a poorer long term prognosis.¹⁻⁵ Other investigators, however, found no significant differences between these two groups of patients.⁶⁻¹⁰ These conflicting results are probably due to differences in patient selection and the timing and method of evaluating the electrocardiographic and angiographic changes. In order to minimise the biases introduced by (a) evaluating coronary anatomy and ventricular function days or months after the acute phase of infarction and (b) non-randomised patient selection we studied patients

with inferior wall myocardial infarction extracted from a larger pool of consecutive patients who underwent cardiac catheterisation with 12 hours of the onset of symptoms of infarction. In addition, the electrocardiographic and angiographic data were obtained within one hour of each other. Lastly, biplane ventriculography, the most sensitive method currently available for evaluating segmental wall motion abnormalities and ejection fraction, was used in all patients. We compared the differences between patients with and without precordial ST segment depression during acute inferior wall infarction with regard to their clinical, arteriographic, and ventriculographic characteristics.

Patients and methods

STUDY POPULATION

Patients presenting with complaints of acute chest pain suggestive of myocardial ischaemia were screened for possible immediate cardiac catheterisation and coronary arteriography. Inpatients requiring

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emergency cardiac consultation for acute chest pain were screened in a similar fashion. No patients with (a) valvar or congenital heart disease, (b) active psychiatric disorder or alcohol addiction, or (c) contraindication to thrombolytic treatment underwent immediate catheterisation. No patients were excluded from immediate catheterisation because of hypotension or cardiogenic shock. Patients with persistent ischaemic chest pain lasting longer than 30 minutes suggestive of acute myocardial infarction underwent immediate cardiac catheterisation after informed consent was obtained. In 52 patients who underwent immediate catheterisation the diagnosis of acute myocardial infarction was established retrospectively based on increased creatine phosphokinase and creatine phosphokinase MB activity to at least twice the upper limit of normal. Thirty five of these patients had electrocardiographic evidence of transmural inferior wall infarction. Three of these 35 patients, however, had previous anterior wall infarction and were excluded from this study. The remaining 32 patients constitute the study population. All patients received the same medical regimen—that is, prophylactic intravenous lignocaine and morphine sulphate for analgesia—until after baseline electrocardiography and angiography had been performed.

ELECTROCARDIOGRAM

All patients had a standard 12 lead electrocardiogram recorded on admission and within one hour of cardiac catheterisation. All electrocardiograms were recorded on either three or six channel recorders using a paper speed of 50 mm/s and including 1 mV standardisation. Q waves of longer than 0.04 s or 0.03 s in association with a Q : R ratio 1 : 3 were considered to be abnormal. In lead V1, R > S and R > 5 mm was considered to be pathological. ST segment elevation of ≥ 1 mm above the baseline (as defined by the preceding TP segment) occurring 0.02 s after the end of the QRS complex in any lead was considered to be abnormal. The sum of the number of inferior leads in which abnormal ST segment elevation was present was calculated for each patient. ST segment depression ≥ 1 mm occurring 0.08 s after the J point in any lead was considered to be significant. The electrocardiogram recorded within one hour before cardiac catheterisation was used for the analysis of all the electrocardiographic variables in this study.

In 32 patients acute inferior myocardial infarction was diagnosed on the basis of abnormal Q waves or ST segment elevation in at least two of the following leads: II, III, or aVF. In five patients true posterior wall infarction was diagnosed on the basis of R > S and R > 5 mm in lead V1 in the presence of an upright T wave.¹¹

No patient in the study group was taking digoxin or had left ventricular hypertrophy, intraventricular conduction abnormality, right bundle branch block, or left bundle branch block. Seventeen of the 32 patients (group 1) had precordial ST segment depression in at least two of the following leads: V1-V4 and 15 did not (group 2). Four patients in group 1 had electrocardiographic evidence of a true posterior myocardial infarction in contrast to only one patient in group 2.

ANGIOGRAPHY AND VENTRICULOGRAPHY

The technique of angiography during the acute phase of myocardial infarction has been described previously.¹² The degree of luminal narrowing in the coronary arteries was assessed by the method of Gensini.¹³ Stenosis in the coronary artery was considered to be significant if the luminal diameter was reduced by >70%. Wall motion abnormalities were described according to the method of Herman *et al.*¹⁴ The specific myocardial segments were identified from the right anterior oblique and left anterior oblique ventriculograms in a manner similar to that in the coronary artery surgery study.¹⁵

Determination of infarct vessel

The infarct vessel was determined by the following angiographic criteria: coronary artery disease resulting in a total or subtotal occlusion of one vessel (29 patients) and >90% stenosis in two or three vessels (three patients). In these three patients, there were arteriographic features suggesting acute thrombus in one of the vessels. The arteriographic findings of persistent staining of intraluminal material with dye, or intraluminal filling defects completely surrounded by dye, were considered to suggest a thrombus.^{16,17} Wall motion abnormalities were present in the areas subtended by the infarct related vessel in all 32 patients.

STATISTICS

Continuous variables are expressed as the mean (standard deviation). Differences between the means of independent continuous variables were evaluated by a *t* test. Differences between discrete variables were evaluated by a χ^2 test. Fisher's exact test was used for small samples in fourfold tables. A probability value of <0.05 was considered to be significant. Statistical comparisons were performed between groups 1 and 2 before and after those patients with electrocardiographic evidence of true posterior infarction had been excluded from both groups. Inferences, based on the data presented, are made with some reservation because of the relatively small sample size.

Table Clinical and angiographic characteristics

	Group 1 (n=17)	Group 2 (n=15)
Mean (SD) age (yr)	56.7(8.9)	53.4(11.1)
Sex (M/F)	16/1	13/2
% of patients with diabetes or hypertension	8%	6%
Duration from onset of symptoms to angiography (h, mean (SD))	4.7(4.1)	6.5(6.0)
No of patients in Killip class: I or II	15	15
III or IV	2	0
Number of inferior leads with ST segment elevation	2.7(0.8)	2.1(1.1)
Initial creatine kinase activity (IU/l, mean (SD))	168(155)	157(264)
Infarct related vessel (No of patients):		
Right coronary	13	13
Circumflex	4	2
Extent of coronary artery disease:		
One vessel	7	6
Two vessel	6	7
Three vessel	4	2
Left ventricular ejection fraction (%), mean (SD)	53.4(9.8)	56.1(12.0)

Results

CLINICAL AND ANGIOGRAPHIC CHARACTERISTICS

There were no significant differences detected between the two groups of patients with respect to age, sex, or the prevalence of diabetes mellitus or hypertension (Table). The duration from the onset of symptoms of acute infarction to cardiac angiography was 4.7(4.1) h for group 1 and 6.5(6.0) h for group 2 (NS). Clinical evaluation of the patients just before angiography showed no significant differences with respect to Killip class or initial serum creatine kinase activity. Although patients in group 1 had a higher number of leads with ST segment elevation than those in group

2, this difference did not reach statistical significance.

There were no significant differences between the two groups with respect to the extent of coronary artery disease or to the left ventricular ejection fraction. Two patients in both groups had a stenosis $\geq 90\%$ in their left anterior descending artery. All patients in both groups, except for one in group 1, had a right dominant coronary circulation.

WALL MOTION ABNORMALITIES

The segmental wall motion abnormalities seen during biplane ventriculography are compared for both groups of patients in the Figure. Most patients, in both groups, had inferior and posterolateral wall motion abnormalities. Nevertheless, no significant differences were observed with regard to the frequency of posterolateral involvement. In addition, both groups of patients had comparably low frequencies of anterior and or septal wall motion abnormalities.

Discussion

Several reports have suggested that patients with precordial ST segment depression in acute inferior myocardial infarction were at higher risk than those with inferior wall infarction but without precordial ST segment depression.¹⁻⁵ The investigations of Salcedo *et al* and of Jennings *et al* suggested that patients with precordial ST segment depression were more likely to have additional coronary disease in the left anterior descending artery and anterior wall ischaemia.^{2,18} These studies, however, were based on patients who underwent cardiac catheterisation several weeks or months after their inferior infarction for progressive or recurrent symptoms. Since patients with postinfarction symptoms are more likely to have multivessel disease this selection bias may have affected their results.

Figure Prevalence of wall motion abnormalities in myocardial segments in patients with precordial ST segment depression (group 1) compared with those without (group 2).

Our study was based on arteriographic and ventriculographic data obtained during the acute phase of inferior infarction in consecutive patients. In this random population, in contrast to the studies mentioned above, we did not observe any significant differences in the prevalence of left anterior descending disease. In addition, both groups of patients had a comparably low prevalence of anterior or septal wall motion abnormalities.

Other studies have suggested that precordial ST segment depression in inferior infarction is a marker for a more extensive infarction.^{3,4,19} Gibson *et al*, using radionuclide techniques in the late hospital phase after myocardial infarction, noted a lower left ventricular ejection fraction in patients with precordial ST segment depression.⁴ They attributed this finding to larger inferior and inferoapical infarctions.

In our study calculation of left ventricular ejection fraction based on biplane ventriculography obtained within one hour of the electrocardiogram showed no significant differences between our two groups. Our findings agree with the studies of Croft *et al* and O'Neill *et al*, who also found no significant difference in ejection fraction between patients with and without precordial ST segment depression.^{6,20}

Myers *et al* correlated precordial ST segment depression in acute inferior infarction with pathological evidence of posterolateral extension of inferior wall infarction.²¹ More recently, Goldberg *et al* studied 25 patients with acute inferior infarction using radionuclide techniques during the acute phase of infarction.²² Their observations suggested that patients with precordial ST segment depression had a much higher prevalence of posterolateral wall motion abnormalities than those with inferior infarction without precordial ST segment depression. Eleven of the 14 patients in their group with precordial ST segment depression, however, met classical electrocardiographic criteria for additional posterior infarction in contrast to only four of 11 patients in their group without precordial ST segment depression. The uneven distribution of patients with additional electrocardiographic evidence of posterior infarction between their two groups of patients may explain the greater prevalence of posterolateral wall motion abnormalities in their patients with precordial ST segment depression.

Our study, in which patients with electrocardiographic evidence of posterior infarction were more evenly distributed between the two study groups, did not show a significantly higher prevalence of posterior wall motion abnormalities in the patients with precordial ST segment depression. In this regard our observations agree with the earlier work of Croft *et al*.⁶

Since several of our patients underwent interventions during the acute phase of their infarction this study cannot definitely evaluate differences in out-

come or prognosis between the two groups of patients. There is, however, a close relation between ejection fraction in the acute phase of myocardial infarction and long term prognosis.²³ The data on ejection fraction in the early phase may therefore have some prognostic significance.

Based on the correlations that were obtained during the early hours of infarction this study suggests that patients with anterior ST segment depression do not constitute a subset at increased risk. Clinical trials with larger patient populations, using angiography during the acute phase of infarction, should provide an even more definitive evaluation.

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