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Severity of Residual Stenosis of Infarct-Related Lesion and Left Ventricular Function after Single-Vessel Anterior Wall Myocardial Infarction: Implication of ST-Segment Elevation in Lead aVL of the Admission Electrocardiograms

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Summary

Background: The relationship between the severity of chronic-phase stenosis of infarct-related lesions (IRLs) and chronic left ventricular function in anterior acute myocardial infarctions (AMI) has not been adequately investigated.

Hypothesis: This study investigated whether ST elevation in lead aVL of admission electrocardiogram (ECG) would be a determinant factor of the relationship between the severity of stenosis of the IRL and chronic left ventricular function after anterior wall AMI.

Methods: One month after AMI, the IRL was evaluated by coronary angiography in 98 patients with anterior AMI, and left ventricular ejection fraction (LVEF) was determined using multigated radionuclide angiocardiography. Patients were classified according to the severity of the IRL: patients with 100% occlusion (Group O), patients with 90 to 99% stenosis (Group H), and patients with ≤75% stenosis (Group L). Patients with ST elevation ≥0.1 mV in the aVL lead on their admission ECG were included in the ST-elevation group, and those with ST elevation <0.1 mV were included in the non-ST-elevation group.

Results: The LVEF was greater in the non-ST-elevation group than in the ST-elevation group ($p<0.0001$), and the LVEF in a whole group as follows: Group L LVEF > Group H LVEF > Group O LVEF ($p = 0.0160$). In the ST-elevation group, LVEF was higher in Group L than in the other groups ($p = 0.0251$). There were three independent predictors of a reduced LVEF: ST-elevation in aVL [odds ratio (OR): 3.38, $p = 0.0044$], IRL stenosis ≥90% (OR: 2.90, $p = 0.0044$), and the IRL occurring in the left anterior descending artery proximal to the first diagonal branch (OR: 6.31, $p = 0.0024$).

Conclusion: Left ventricular function was preserved, regardless of the severity of residual stenosis, in patients without ST elevation in aVL if the IRL was not totally occluded. In patients with ST elevation in aVL, LVEF was lower in patients with more severe stenosis, even if the IRL was patent.

Key words: myocardial infarction, electrocardiography, left ventricular function

Introduction

Reperfusion therapy has been established as an acute treatment for acute myocardial infarction (AMI)[1–8] and preserves left ventricular (LV) function if patency of the infarct-related coronary artery is maintained.[9–12] However, the relationship between the severity of chronic-phase stenosis of infarct-related lesions (IRLs) and chronic LV function has not been adequately investigated.[13]

In most of the multicenter trials[1–3] and meta-analyses[4] that were performed to determine the efficacy of reperfusion therapy for AMI, and in the recent studies[14–20] comparing the relative merits of acute reperfusion therapy using coronary thrombolysis versus primary percutaneous transluminal coronary

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angioplasty, patients with anterior wall AMI have been treated as a single group. Therefore, these studies have failed to take into account the size of the risk area. We have previously reported that in the setting of an anterior wall AMI, the presence of the IRL proximal to the bifurcation of the first diagonal branch of the left anterior descending coronary artery is an independent predictor of left ventricular dilation.[21] The location of the IRL determines the area at risk for AMI and is an important factor affecting chronic LV function. However, there have been no studies of the relationship between the residual stenosis of the IRL and LV function in patients with anterior wall AMI based on the size of the risk area.

In many patients with anterior wall AMI and ST elevation in lead aVL of electrocardiograms (ECGs) obtained during the acute stage of infarction, the IRL is located in the left anterior descending coronary artery proximal to the first diagonal branch.[22] The area of ischemia is more extensive in these patients[23] and the severity of myocardial damage is significantly greater than that seen in patients without ST elevation in aVL. Standard 12-lead electrocardiography is a simple and reproducible noninvasive examination, and it can estimate the site and extent of infarction with a certain degree of accuracy. No studies, however, have been done using admission 12-lead ECGs obtained immediately after the onset of AMI to characterize the risk area. The objective of the present study was to determine the relationship between the severity of stenosis of the IRL 1 month after AMI and LV function by classifying patients with single-vessel anterior wall AMI into two groups based on the presence or absence of ST elevation in aVL of ECGs obtained during the acute stage of infarction.

Methods

Study Patients

Consecutive patients with anterior wall AMI were selected according to the following criteria: the presence of persistent ST-segment elevation ≥ 0.2 mV in two or more contiguous anterior chest leads (V_1 to V_4) during the acute phase of infarction, and a compatible clinical syndrome (severe chest pain lasting >30 min unresponsive to nitroglycerin or isosorbide dinitrate) and a serum creatine phosphokinase activity that was more than two times of upper limit of normal. Of the 286 patients who were admitted to our coronary care unit between January 1988 and May 1995 within 24 h of the onset of symptoms with the diagnosis of a first anterior wall AMI, 240 patients were discharged alive. Of these, 122 patients were found by coronary angiography to have single-vessel disease involving the left anterior descending coronary artery. Of these, 107 patients underwent multigated radionuclide angiocardiography to evaluate LV systolic function 1 month after AMI. We excluded nine patients with either complete right or left bundle-branch block. Therefore, 98 patients [80 men, 59.3 ± 8.9 years of age (mean ± standard deviation (SD), range: 38–81 years] were included in this retrospective study.

Reperfusion Therapy

From 1988 to 1990, we mainly performed intravenous coronary thrombolysis. From 1991 to 1993, we mainly performed intracoronary thrombolysis. After May 1993, we mainly performed coronary angioplasty for reperfusion therapy. In 38 of the 98 patients, acute reperfusion therapy by either intravenous or intracoronary thrombolysis was performed within 6 h of the onset of chest pain. In 36 patients, coronary angioplasty was performed to achieve acute reperfusion. The other 24 patients received conventional conservative treatment without reperfusion therapy. In 66 of the 98 patients (67%), emergency coronary angiography was performed to initiate reperfusion therapy or to confirm restoration of coronary blood flow. If coronary blood flow was Thrombolysis in Myocardial Infarction (TIMI)[24] grade 2 or better, coronary angiography was terminated without the institution of reperfusion therapy.

Admission Electrocardiography

Patients were classified into two groups, those with and those without ST elevation, based on the presence or absence of ST-segment elevation ≥ 0.1 mV in lead aVL.

Multigated Radionuclide Angiocardiography

All patients underwent multigated radionuclide angiocardiography 1 month (24–51 days, mean: 29 ± 6 days) after the onset of myocardial infarction. Following intravenous injection of 740 MBq of technetium-99m (^{99m}Tc)-labeled human serum albumin, data acquisition was performed using a single-crystal gamma camera (model GCA-90B, Toshiba, Tokyo, Japan) fitted with a low-energy, all-purpose, parallel-hole collimator and interfaced to a dedicated minicomputer (model GMS-55U, Toshiba, Tokyo, Japan) in list mode. The left anterior oblique projection that best displayed the interventricular septum was used for imaging (approximately 45° with 10° of caudal angulation). Left ventricular ejection fraction (LVEF) was computed from the global time-activity curve using the LV region of interest semiautomatically drawn on the functional phase image.

Exercise Testing and Thallium-201 Single-Photon Emission Computed Tomographic Imaging

Patients underwent thallium-201 (^{201}Tl) single-photon emission computed tomographic imaging with exercise stress test in the fasting state 1 month after the onset of myocardial infarction or before cardiac catheterization. Imaging began within 5 min of the injection of the thallium and was repeated 3 h later to assess the presence or absence of tracer redistribution. Perfusion defect severity in the initial and delayed images was graded visually in nine segments of the left ventricle using a 4-point grading system (0 = normal perfusion, 1 = mildly diminished perfusion, 2 = moderately diminished perfusion, and 3 = severely diminished to absent perfusion). The initial and 3-h delayed images were interpreted independently by two

experienced observers blinded to the clinical data or the results of coronary angiography. Differences in interpretation between observers were resolved by consensus.

Cardiac Catheterization and Analysis of Coronary Angiograms

Cardiac catheterization was performed using the Seldinger technique 1 month after AMI or before discharge. Biplane left ventriculography and coronary angiography with multiple views were performed in the routine manner using the Judkins technique. Informed consent was obtained from all patients. Cineangiograms were assessed visually by two independent experienced observers blinded to the clinical data or ECG findings. The culprit lesion was determined based on its angiographic characteristics (the presence of residual thrombus, ulcerated plaque, or the severity and irregularity of the lesion) or by detection of a complete obstruction of the left anterior descending coronary artery. The spatial relationship between the site of the left anterior descending coronary artery occlusion or residual stenosis and the origins of the first septal perforator and the first diagonal branch was determined.[25,26]

The degree of coronary stenosis was determined using the American Heart Association classification,[26] and any stenosis ≥75% stenosis affecting noninfarcted arteries was defined as significant for the purpose of detecting multivessel disease. Patients with multivessel disease were excluded from this study. Patients were classified into three groups: mild stenosis group (patients with ≤75% of the culprit lesion), severe stenosis group (patients with 90 to 99% stenosis), occlusion group (patients with total occlusion).

To assess the degree to which the left anterior descending coronary artery extends over the apex of the heart, the following grading system was used:[21] grade 1, the left anterior descending coronary artery does not reach the apex; grade 2, the left anterior descending coronary artery supplies the anterior wall of the left ventricle as far as the apex, but does not supply the inferior wall; grade 3, the left anterior descending coronary artery continues beyond the apex along the diaphragmatic surface of the inferior wall. Collateral blood supply to the territory at risk was assessed by visual analysis of cineangiograms using the system described by Rentrop.[27]

Measurement of Peak Serum Creatine Phosphokinase Activity

Blood samples for the measurement of creatine phosphokinase activity were collected every 3 h until the peak value was obtained.

Multivariate Analysis

The effects of various factors on LVEF were examined by multivariate analysis using the stepwise increment method. Covariates in the analysis of an LVEF ≤50% included the following eight items: age (≥65 years), male gender, use of reperfusion therapy, coronary angioplasty during acute infarction, ST-segment elevation in aVL, IRL residual stenosis ≥90% 1 month after infarction, and the presence of the IRL along the left anterior descending coronary artery prior to the origins of the first septal perforator or the first diagonal branch, and the presence of the IRL along the left anterior descending coronary artery prior to the first diagonal branch.

Statistical Analysis

Statistical analysis was performed using StatView 4.11. Logistic regression was performed by the "Logistic Regression" procedure of SPSS for Macintosh (Version 6.1J) using the forward-stepping selection method with maximum-likelihood estimates and default criteria. Data are expressed as the mean ± SD. Comparisons between two groups were performed using the unpaired Student's *t*-test. Differences among the three groups were determined by one-way analysis of variance, followed by the Bonferroni method for comparisons. The chi-square test was used for comparisons of categorical data. Results were considered statistically significant if $p < 0.05$. The odds ratios (OR), 95% confidence intervals (CI), and p values reported are those for the final model (i.e., adjusted for all other significant covariates).

Results

In all 98 patients, LVEF was highest in patients with mild stenosis of the IRL, followed by those with severe stenosis, and then by those with occlusion (54.0 ± 9.4% vs. 50.0 ± 12.4% vs. 42.5 ± 11.4 %, $p = 0.0160$). The difference in LVEF between the mild stenosis and occlusion groups was significant, but there were no other statistically significant differences.

ST elevation in the aVL lead obtained at the time of admission was ≥0.1 mV for 43 patients (the ST-elevation group) and <0.1 mV for 55 patients (the non-ST-elevation group). There were no significant differences between the ST-elevation and non-ST-elevation groups with respect to age, gender, coronary risk factors at the time of admission, reperfusion therapy during hospitalization, oral drug therapy during hospitalization, the length of the left anterior descending coronary artery, or the amount of collateral blood flow determined during coronary angiography in the acute stage of infarction (Table I), nor was there any significant difference in the severity of residual stenosis 1 month after AMI between the two groups. However, the peak serum creatine phosphokinase activity was significantly greater in the ST-elevation group than in the non-ST-elevation group.

Left ventricular ejection fraction was significantly lower in the ST-elevation group than in the non-ST-elevation group (45.2 ± 9.1% vs. 56.3 ± 10.4%, $p < 0.0001$). In patients with ST elevation in lead aVL, IRL is more frequently located proximal to the diagonal branch of the left anterior descending coronary artery than in those without ST elevation in lead aVL ($p < 0.0001$, Table I). In addition, the total defect score for the delayed thallium myocardial scintigraphic images was higher in the ST-elevation group than in the non-ST-elevation group (11.9 ± 4.9 vs. 8.3 ± 5.3, $p = 0.0010$).

TABLE I Baseline characteristics of patients with or without ST elevation in lead aVL

	With ST elevation n = 43	Without ST elevation n = 55	p Value
Age (years)	57.4 ± 9.0	60.9 ± 8.7	NS
Sex (M/F)	37/6	43/12	NS
Risk factors			
Hypertension (%)	36	42	NS
Diabetes (%)	24	17	NS
Hyperlipidemia (%)	26	43	NS
Smoking (%)	74	72	NS
Medications			
ACE-I (%)	33	19	NS
ß-blocker (%)	3	7	NS
Ca-antagonist (%)	57	63	NS
Nitrate (%)	75	80	NS
Digitalis (%)	7	7	NS
Diuretics (%)	17	4	NS
Reperfusion therapy			NS
Angioplasty (%)	37	36	
Thrombolysis (%)	44	35	
Conservative (%)	19	29	
Stenosis 1 month after AMI			NS
≤ ± 75% (%)	53	49	
75% to 99% (%)	37	44	
100% (%)	9	7	
Infarct-related lesion			
Pre-D group (%)	70	25	< 0.0001
Pre-S group (%)	74	36	0.0002
Length of LAD	2.8 ± 0.7	2.8 ± 0.7	NS
Collateral flow	0.5 ± 0.9	0.7 ± 1.0	NS
Peak CPK (IU/l)	6496 ± 3054	2903 ± 2458	< 0.0001
LVEF total (%)	45.2 ± 9.1	56.3 ± 10.4	< 0.0001
≤ 75% (%)	48.7 ± 8.0	58.5 ± 8.2	< 0.0001
75% to 99% (%)	41.6 ± 8.5	55.8 ± 11.3	0.0001
100% (%)	40.0 ± 11.6	45.0 ± 12.3	NS
Total defect score	11.9 ± 4.9	8.3 ± 5.3	0.0010

Patients with a culprit lesion proximal to the origin of the first septal perforator were included in the pre-S group. Patients with a culprit lesion proximal to the origin of the first diagonal branch were included in the pre-D group.

Abbreviations: ACE-I = angiotensin-converting enzyme inhibitor, LAD = left anterior descending coronary artery, LVEF = left ventricular ejection fraction, AMI = acute myocardial infarction, NS = not significant.

Severity of Chronic Residual Stenosis and Left Ventricular Ejection Fraction in Patients with or without ST-Elevation (Fig. 1)

In the 55 patients in the non-ST-elevation group, there was a significant difference in LVEF between the mild stenosis (58.5 ± 8.2 %) and occlusion groups (45.0 ± 12.3%, p = 0.0471), but not between the mild and severe stenosis groups (55.8 ± 11.3%). For the 43 patients in the ST-elevation group, LVEF was highest in the mild stenosis group (mild stenosis group vs. severe stenosis group vs. occlusion group: 48.7 ± 8.0% vs. 41.6 ± 8.5% vs. 40.0 ± 11.6%, p = 0.0251). There was no significant difference in LVEF between the severe stenosis group and the occlusion group, but the difference between the mild and severe stenosis groups was statistically significant. Comparison of the ST-elevation and non-ST-elevation groups based on the severity of stenosis demonstrated that LVEF was significantly lower in the ST-elevation group than in the non-ST-elevation group in patients with mild or severe stenosis. There was no significant difference in LVEF between the occlusion groups.

Multivariate Analysis of Predictors of Chronic Left Ventricular Dysfunction

Multivariate analysis was performed to identify predictors of an LVEF ≤ 50%, using the eight variables enumerated in the Methods section as covariates. The following variables were

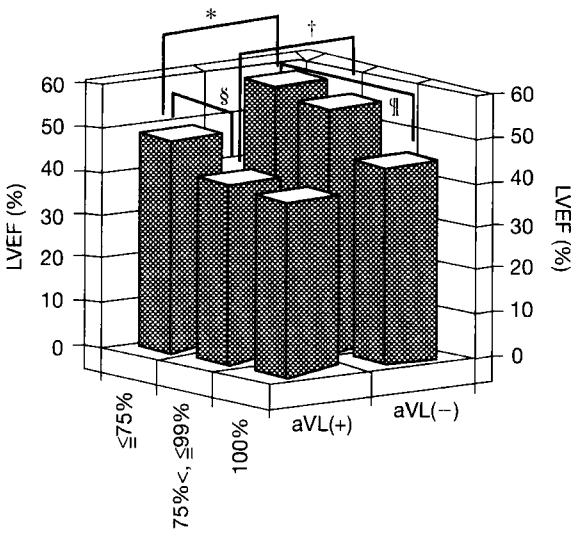

FIG. 1 The relationship between severity of stenosis of the infarct-related lesion (IRL) and left ventricular ejection fraction (LVEF). For patients without aVL ST elevation, LVEF differed significantly between the mild stenosis and occlusion groups (¶: $p = 0.0471$), but there was no significant difference in LVEF between the mild and severe stenosis groups, or between the severe stenosis and occlusion groups. For the patients with aVL ST elevation, LVEF was highest in the mild stenosis group (§: $p = 0.0251$). There was no difference in LVEF between the severe stenosis and occlusion groups, but the difference between the mild and severe stenosis groups was significant. LVEF was significantly lower in the patients with mild (*: $p<0.0001$) or severe stenosis (†: $p = 0.0001$) in the ST-elevation group than in the patients in the non-ST-elevation group, but the LVEFs for the occlusion groups were not significantly different.

identified as significant independent factors: ST elevation in aVL (OR: 3.38, 95% CI: 1.14–10.0, $p = 0.0044$); chronic IRL stenosis ≥90% (OR: 2.90, 95% CI: 1.02–8.24, $p = 0.0044$); and presence of the IRL proximal to the first diagonal branch (OR: 6.31, 95% CI: 2.15–18.6, $p = 0.0024$).

Discussion

Residual Stenosis and Left Ventricular Function

No previous studies have examined the effect of reperfusion on LV function in patients with anterior wall AMI based on differences in the size of the risk area. In the present study, LVEF was significantly lower in the complete occlusion group than in either the severe or mild stenosis groups, but there was no significant difference between the severe and mild stenosis groups when patients with anterior wall AMI were treated as a single group. These findings are consistent with those of a previous study[9] which found no significant difference in LV function during the first month after AMI when patients were stratified according to the severity of stenosis of the IRL. However, when the relationship between the severity of residual stenosis and LV function was studied based on the presence or absence of ST elevation in lead aVL, LV function in the group with ST elevation was found to be significantly lower when the stenosis was more severe. In the non-ST-elevation group, LV function was preserved and similar in the severe and mild stenosis groups. These results indicate that the presence or absence of ST elevation in aVL can be used to stratify patients following anterior wall AMI into two groups. Changes in the ST segment in the aVL lead provide information concerning the anterior free wall and indicate changes in the region of the diagonal branch of the left anterior descending coronary artery.[28] In patients with anterior wall AMI and aVL ST elevation, the IRL is generally proximal to the diagonal branch of the left anterior descending coronary artery. Conversely, in patients without ST elevation, the lesion is often distal to the diagonal branch.[22] The risk area is therefore believed to be greater in patients with than in those without ST elevation. In the present study, patients with aVL ST elevation had greater peak serum creatine phosphokinase activity and total defect scores for thallium myocardial scintigraphy than did patients without ST elevation. In addition, the myocardial damage was more extensive in patients with aVL ST elevation.

Clinical Implications

The information necessary to select the method of reperfusion therapy for AMI must be obtained as soon as possible following presentation of the patient. Important prognostic information can be obtained from the presence or absence of ST elevation in the aVL lead. The relative merits of coronary thrombolysis and primary coronary angioplasty as methods of acute reperfusion therapy for AMI have been debated.[14–20] However, no conclusion has yet been reached as to which is superior. However, the two methods differ appreciably in terms of the severity of the residual stenosis and success of reperfusion. Some studies have reported an acceptable outcome from reperfusion therapy, regardless of the severity of residual stenosis, provided that TIMI-3 blood flow is obtained.[29, 30] However, the results of the present study suggest that patients with aVL ST elevation require aggressive reperfusion therapy to reduce the residual stenosis. In contrast, patients without ST elevation may not necessarily require aggressive revascularization, provided that reperfusion is obtained. We would like to call for a prospective study to test our hypothesis and for analysis of a large database, such as Global Utilization of Streptokinase and t-PA Activator for Occluded Coronary Arteries (GUSTO) II, with respect to overall prognostic features and outcomes in patients with anterior wall infarction, by analyzing the aVL ST-segment status.

Limitations of the Study

The present study was retrospective and, therefore, did not prospectively examine the effects of acute therapy choice on chronic residual stenosis and LV function. In some patients, the IRL will reopen during the acute stage of infarction, and in others reduction in the degree of stenosis will occur in the chronic stage of infarction. This may explain why the specific

acute therapy did not affect LV function in the multivariate analysis performed in the present study.

Conclusion

In patients with an anterior wall AMI, the LVEF was well preserved regardless of the severity of residual stenosis in patients without ST elevation in aVL if the patency of the IRL could be confirmed. In contrast, in patients with aVL ST elevation, the LVEF was significantly lower when the stenosis was severe, even if the IRL was patent.

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