Angiographic and Clinical Significance of 'Transient' ST-Segment Depression in the Lateral Chest Leads in Anterior Wall Acute Myocardial Infarction

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This study aimed to clarify the significance of ST-segment depression in the lateral chest leads in anterior wall acute myocardial infarction (AMI) with ST-segment elevation. A total of 196 patients with their first anterior wall AMI (≤ 6 h) were divided into 2 groups according to the presence (group A, $n=39$) or absence (group B, $n=157$) of ST-segment depression ≥ 0.1 mV in V₅ and/or V₆ on the admission electrocardiogram. Patients with electrocardiographic confounding factors were excluded. No patients had persistent ST-segment depression in the lateral chest leads. Emergency coronary angiography revealed that group A had higher incidences of occlusion of the left anterior descending coronary artery (LAD) proximal to its first septal branch (77% vs 51%, $p<0.01$) and good collateral circulation than group B (46% vs 25%, $p<0.05$). Peak creatine kinase levels were significantly lower in group A than in group B (2060 ± 1099 vs 2873 ± 2077 IU/L, $p<0.01$). Left ventricular ejection fraction in the chronic phase was significantly greater in group A than in group B. Regional wall motion in the infarct region in the chronic phase was better in group A than in group B. These results indicate that patients with 'transient' ST-segment depression in the lateral chest leads in anterior wall AMI had a relatively smaller infarct size, despite their higher incidence of occlusion of the LAD proximal to its first septal branch, because of their higher incidence of good collateral circulation. (*Jpn Circ J* 1999; 63: 873–876)

Key Words: Acute myocardial infarction; Electrocardiography; ST-segment depression

ST-segment depression in any lead is frequently observed in anterior wall acute myocardial infarction (AMI) and previous reports have shown that patients with this sign have a poor prognosis.^{1–3} However, those studies did not differentiate patients with ST-segment depression in the inferior leads from those with ST-segment in the lateral chest leads. There may be differences in the significance between the 2 types of ST-segment depression and although there have been many reports with regard to ST-segment depression in the inferior leads in anterior wall AMI,^{4–21} the angiographic and clinical significance of ST-segment depression in the lateral chest leads in anterior wall AMI has not yet been fully investigated, especially in the case of AMI accompanied by ST-segment elevation in the precordial leads. Accordingly, the present study aimed to clarify the angiographic and clinical features of patients with ST-segment depression in the lateral chest leads on the admission electrocardiogram (ECG) in anterior wall AMI with ST-segment elevation.

Methods

Patients

Between January 1989 and December 1997, 252 patients with anterior wall AMI were admitted within 6 h of the onset of chest pain. The data regarding these patients was prospectively entered into a computer database. Patients

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meeting the following criteria were selected for this study: (1) typical chest pain lasting ≥ 30 min; (2) ST-segment elevation ≥ 0.2 mV in ≥ 2 contiguous precordial leads on the admission ECG; (3) increase in serum creatine kinase (CK) level more than twice the normal value; (4) no previous myocardial infarction (MI); (5) no confounding ECG findings, such as left ventricular (LV) hypertrophy (confirmed by echocardiography), left or right bundle branch block, intraventricular conduction disturbance, ventricular rhythm and Wolf-Parkinson-White syndrome, or poor-quality ECG recordings; (6) no other heart or lung disease; and (7) identification of the infarct-related lesion by emergency coronary arteriography. In total, 196 patients (141 men and 55 women, mean age 63 ± 10 years) met these criteria. The patients were classified into 2 groups according to the presence (group A, $n=39$) or absence (group B, $n=157$) of ST-segment depression ≥ 0.1 mV in V₅ and/or V₆ on the admission ECG. Of 37 group A patients still living, none had persistent ST-segment depression in the lateral chest leads during the in-hospital period.

Standard 12-Lead ECG

Standard 12-lead ECGs were recorded at a paper speed of 25 mm/s and a standardization of 10 mm = 1 mV. The magnitude of ST-segment elevation or depression relative to the TP-segment was measured to the nearest 0.5 mm at 80 ms after the J point. All ECGs were analyzed by the consensus of 2 observers who were blinded to all clinical and angiographic data.

Emergency Coronary Arteriograms

Emergency coronary arteriography was performed using either the Judkins or Amplatz techniques. Multiple projec-

Table 1 Clinical Characteristics

	Group A (n=39)	Group B (n=157)	p value
Age (years)	62±9	63±11	NS
Male	26 (67%)	115 (73%)	NS
Time to admission (min)	142±72	154±82	NS
Peak CK (IU/L)	2060±1099	2873±2077	<0.01
Postinfarction angina	2 (5%)	2 (1%)	NS
Re-infarction	1 (3%)	4 (3%)	NS
In-hospital death	2 (5%)	10 (6%)	NS

Data presented are means ± SD or number (%).

CK, creatine kinase; NS, not significant.

Table 2 Emergency Coronary Angiographic Findings

	Group A (n=39)	Group B (n=157)	p value
TIMI 3	4 (10%)	11 (7%)	NS
Good collaterals	18 (46%)	40 (25%)	<0.05
Multivessel disease	15 (38%)	45 (29%)	NS
LAD occlusion proximal to its first SB	30 (77%)	80 (51%)	<0.01
LAD occlusion proximal to its first DB	21 (54%)	71 (45%)	NS
Reperfusion therapy			
PTCR	31 (79%)	107 (68%)	NS
PTCA	15 (38%)	82 (52%)	NS
TIMI 3 after reperfusion therapy	27 (69%)	113 (72%)	NS

LAD, left anterior descending coronary artery; SB, septal branch; DB, diagonal branch; PTCR, percutaneous transluminal coronary recanalization; PTCA, percutaneous transluminal coronary angioplasty.

tions were recorded to ensure optimal visualization of the coronary vessels. The coronary flow in the infarct-related artery was graded according to the classification used in the Thrombolysis In Myocardial Infarction (TIMI) trial²². The grade of collateral filling in the left anterior descending coronary artery (LAD) was determined according to the criteria of Rentrop et al²³; collateral circulation of grade of 2 or 3 was defined as 'good'. After angiographic confirmation of total or subtotal occlusion of the LAD, intracoronary isosorbide dinitrate was administered, followed by intracoronary urokinase or tissue plasminogen activator. If reperfusion was not successful, coronary angioplasty was performed. Forty-two patients underwent direct angioplasty. Successful reperfusion was defined as the establishment of TIMI grade 3 flow in the infarct-related artery.

Cardiac Catheterization in the Chronic Phase

Of 196 patients studied, 173 (88%) underwent coronary arteriography and left ventriculography about 1 month after AMI. Left ventriculograms performed in the 30° right anterior oblique projection were analyzed for the LV ejection fraction (LVEF) and regional wall motion by an experienced cardiologist who was unaware of the patient's data. The LVEF was calculated by the area-length method²⁴. Regional wall motion of the LV was calculated by the centerline method²⁵; that is, it was measured at 100 equidistant chords perpendicular to a center line drawn midway between the end-diastolic and end-systolic LV silhouettes and numbered clockwise from the anterior aortic valve. To normalize for heart size, the motion at each chord was divided by the length of the end-diastolic perimeter to yield a dimensionless shortening fraction. Because comparison of different regions requires that motion values be comparable, the normalized or shortening fraction of each chord was converted into units of standard

Fig 1. Comparison of left ventricular ejection fraction (LVEF) in the chronic phase.

Fig 2. Comparison of regional wall motion (RWM) in the anterolateral and apical region in the chronic phase.

deviation (SD) from the normal group mean. Regional wall motion was expressed as the mean values of SD/chords in the anterolateral and apical region (chords 21–60).

Cardiac Enzyme Measurement

Blood samples were obtained every 3 h during the first 24 h and once daily from the second day until a normal value was obtained for determination of the peak serum CK activity.

Statistical Analysis

Continuous variables are expressed as mean values ± SD. Categorical data were analyzed by the Fisher's exact test or chi-square test. Continuous variables were analyzed by the unpaired t test. A p value < 0.05 was considered statistically significant.

Results

Clinical characteristics are shown in Table 1. There were no differences between the 2 groups in age, gender, and the time elapsed from the onset of chest pain to admission. Peak CK levels were significantly lower in group A than in group B (2060±1099 vs 2873±2077 IU/L, p<0.01). The incidences of postinfarction angina and re-MI during the in-hospital period were similar between the 2 groups (5% vs 1% and 3% vs 3%, respectively). In-hospital mortality was similar in the 2 groups (5% vs 6%). The causes of

death in group A were ventricular fibrillation (n=1) and congestive heart failure (n=1), whereas in group B they were congestive heart failure (n=5), cardiac rupture (n=4), and cerebrovascular disorder (n=1).

Emergency coronary angiography (Table 2) revealed that group A had a higher incidence of occlusion of the LAD proximal to its first septal branch (77% vs 51%, $p<0.01$) and better collateral circulation than group B (46% vs 25%, $p<0.05$). There were no differences in the incidences of multivessel disease and occlusion of the LAD proximal to its first diagonal branch between the 2 groups (38% vs 29% and 54% vs 45%, respectively). The rate of establishment of TIMI grade 3 flow after reperfusion therapy was similar in the 2 groups (69% vs 72%).

There were no differences in age, gender, time elapsed from the onset of chest pain to admission, and the rate of unsuccessful reperfusion therapy between the 2 groups undergoing coronary arteriography and left ventriculography about 1 month after AMI. The incidence of TIMI grade 3 flow on coronary arteriography in the chronic phase was similar between the 2 groups (84% vs 85%). The LVEF in the chronic phase was significantly greater in group A than in group B ($58\pm 11\%$ vs $52\pm 13\%$, $p<0.05$) (Fig 1). Regional wall motion in the anterolateral and apical region was significantly less reduced in group A than in group B (-1.6 ± 1.2 vs -2.1 ± 1.3 , $p<0.05$) (Fig 2).

Discussion

In the present study, patients with ST-segment depression in the lateral chest leads on the admission ECG had a higher incidence of occlusion of the LAD proximal to its first septal branch than those without it. However, despite this angiographic feature, patients with this type of ST-segment depression had a smaller infarct size and less regional wall motion abnormality in the chronic phase than those without it. This is thought to be partly because 56% of the former had TIMI grade 3 or good collateral circulation on the initial coronary arteriogram. As the present study did not include patients with persistent ST-segment depression in the lateral chest leads, our results represent the angiographic and clinical features of patients with anterior wall AMI showing 'transient' ST-segment depression in the lateral chest leads. The lack of patients with 'persistent' ST-segment depression in the lateral chest leads is probably because we excluded patients with LV hypertrophy or previous MI. The previous damage to the myocardium caused by ischemia or hypertension could be a major cause of 'persistent' ST-segment depression in the lateral chest leads after AMI. The effect of myocardial salvage by successful revascularization could be an alternative important cause of the lack of 'persistent' ST-segment depression in the lateral chest leads in the present study.

Mechanisms of ST-Segment Depression in the Lateral Chest Leads in Anterior Wall AMI With ST-Segment Elevation

Several factors could contribute to ST-segment depression in the lateral chest leads in anterior wall AMI with ST-segment elevation. First, as the lateral chest leads face the lower anterolateral and apical region, transmural ischemia in this region is expected to cause ST-segment elevation in the lateral chest leads. On the other hand, non-transmural ischemia in this region is expected to cause ST-segment depression in the lateral chest leads. Therefore, it is reason-

able that patients with ST-segment depression in the lateral chest leads have less regional wall motion abnormality in the anterolateral and apical region. Second, additional remote ischemia caused by multivessel disease may produce ST-segment depression in the lateral chest leads. However, as the incidence of multivessel disease was similar between patients with and without this type of ST-segment depression, this phenomenon is unlikely to strongly influence ST-segment deviation in the lateral chest leads. Third, ST-segment elevation in the right chest leads produced by transmural ischemia in the anterior septum perfused by septal branches can produce reciprocal ST-segment depression in the lateral chest leads.²⁶ Indeed, in the present study, the majority of patients with ST-segment depression in the lateral chest leads had an occlusion of the LAD proximal to its first septal branch. In group B patients with an occlusion of the LAD proximal to its first septal branch, the force of the current of injury produced by transmural ischemia in the lower anterolateral and apical region, which would produce ST-segment elevation in the lateral chest leads, might have exceeded the force of the current of injury produced by transmural ischemia in the anterior septum, resulting in absence of ST-segment depression in the lateral chest leads.

In-Hospital Prognosis

Despite their higher incidence of proximal LAD occlusion, patients with 'transient' ST-segment depression in the lateral chest leads had a relatively smaller infarct size because of their higher incidence of good collateral circulation, and are expected to be at increased risk of postinfarction angina and re-MI if a high-grade residual stenosis persists in the infarct-related vessel after the acute phase. Furthermore, because the majority of these patients appear to have an occlusion of the LAD proximal to its first septal branch, their prognosis is expected to be poor. However, contrary to our expectations, in the present study the in-hospital mortality and the incidences of postinfarction angina and re-MI during the in-hospital period did not differ significantly between patients with and without ST-segment depression in the lateral chest leads on the admission ECG, which might be because about 50% of the patients underwent emergency coronary angioplasty. Successful emergency coronary angioplasty might have decreased the incidence of postinfarction angina and re-MI, resulting in improvement in the prognosis of those patients.

ST-Segment Depression in the Inferior Leads Versus in the Lateral Leads

Patients with ST-segment depression in the inferior leads in anterior wall AMI have been shown to have a higher incidence of proximal LAD occlusion,^{15,18,20,21} a larger infarct size^{4,8,12,13,19} and a poorer prognosis.^{6,8,12,19} Therefore, the frequency of patients with ST-segment depression in the inferior leads could affect our results. However, in the present study, there was no difference in the frequency of patients with ST-segment depression in the inferior leads between patients with and without ST-segment depression in the lateral chest leads. Considering the results of the present study and previous studies with regard to ST-segment depression in the inferior leads in anterior wall AMI,^{4,6,8,12,13,19} there may be some differences in infarct size and prognosis between patients with ST-segment depression in the inferior leads and those with ST-segment depression in the lateral chest leads. Further investigation is

needed to precisely clarify any differences in clinical features according to the site of ST-segment depression in anterior wall AMI.

Study Limitations

First, group B included both patients without ST-segment deviation in the lateral chest leads and those with ST-segment elevation in these leads. Therefore, further studies are needed to clarify whether there is any angiographic and clinical difference according to the type of ST-segment deviation in the lateral chest leads (ie, ST-segment elevation, no ST-segment deviation, and ST-segment depression). Second, this study included only patients with anterior wall AMI showing ST-segment elevation on the admission ECG. Therefore, our results cannot be applied to anterior wall AMI patients without ST-segment elevation on the admission ECG.

Conclusions

The present study clarified the angiographic and clinical features of patients with 'transient' ST-segment depression in the lateral chest leads in anterior wall AMI with ST-segment elevation; that is, they had a relatively smaller infarct size, despite their higher incidence of occlusion of the LAD proximal to its first septal branch, because of their higher incidence of good collateral circulation.

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