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 (≤6h) 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

T-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. 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, 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 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-
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) trial.22 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 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 method:24 Regional wall motion of the LV was calculated by the centerline method:25 that is, it was measured at 100 equidistant chords perpendicular to a centerline 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 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
ST Depression in the Lateral Chest Leads in AMI

Introduction

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 angiogram. 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-
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 (i.e., 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.

**References**