Significance of Q-wave regression after anterior wall acute myocardial infarction

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Aims This study was conducted to clarify the significance of abnormal Q-wave regression in anterior wall acute myocardial infarction.

Methods A total of 74 patients who presented with a first anterior wall acute myocardial infarction within 6 h of onset were divided into two groups according to the presence (group A, n=29) or absence (group B, n=45) of regression of abnormal Q waves. Regression of abnormal Q waves was defined as the disappearance of the Q wave and the reappearance of the r wave $\geq 0.1$ mV in at least one of leads I, aVL, and V1 to V6.

Results Emergency coronary arteriography revealed that group A had a higher incidence of spontaneous recanalization or good collateral circulation than group B (55% vs 31%, $P <0.05$). Peak creatine kinase activity tended to be lower in group A than in group B ($2358 \pm 1796$ vs $3092 \pm 1946$ IU L$^{-1}$, $P =0.09$). Group A had a greater left ventricular ejection fraction and better regional wall motion at 1 and 6 months after acute myocardial infarction than group B. The degree of improvement of left ventricular ejection fraction and regional wall motion between 1 and 6 months after acute myocardial infarction was significantly greater in group A than in group B.

Conclusion Patients with anterior wall acute myocardial infarction showing Q-wave regression had a trend towards a smaller amount of necrotic myocardium and a significantly larger amount of stunned myocardium. (Eur Heart J 1998; 19: 742–746)

Key Words: Anterior wall acute myocardial infarction, abnormal Q wave, left ventricular wall motion.
myocardial infarction, coronary angioplasty, or bypass surgery during the 6-month follow-up period. In total, 74 patients (58 men and 16 women, mean age of 59 ± 9 years) met these criteria.

Standard 12-lead electrocardiograms

Standard 12-lead electrocardiograms were recorded on the admission, the 2nd-5th days, and at 1, 2, and 3 weeks, and at 1 and 6 months after anterior wall acute myocardial infarction in all patients. An abnormal Q wave was defined as the wave ≥0.04 s in duration. Regression of abnormal Q waves was defined as the disappearance of the Q wave and r-wave reappearance ≥0.1 mV in at least one of leads I, aVL, and V1 to V6.

Emergency coronary arteriograms and reperfusion therapy

Emergency coronary arteriography was performed using the Judkins or Amplatz technique. Multiple projections were recorded to ensure optimal visualization of the coronary vessels. The coronary flow in the infarct-related artery was graded according to the system used in the Thrombolysis in Myocardial Infarction (TIMI) trial[12]. The infarct-related artery was considered patent if the TIMI grade was 2 or 3. The grade of collateral filling in the left anterior descending coronary artery was determined according to the criteria of Rentrop et al.[13]. A collateral circulation with a grade of 2 or 3 was defined as good. After angiographic confirmation of the left anterior descending coronary artery, intracoronary isosorbide dinitrate was administered followed by intracoronary urokinase or tissue plasminogen activator. If successful reperfusion was not obtained, coronary angioplasty was performed. Thirteen (18%) patients underwent primary coronary angioplasty. Successful reperfusion was defined as the establishment of TIMI grade 2 or 3 flow in the infarct-related artery.

Follow-up cardiac catheterization

All patients underwent coronary arteriography and left ventriculography at 1 and 6 months after the onset of acute myocardial infarction. Left ventriculograms were recorded in the 30° right anterior oblique and 60° left anterior oblique views. End-diastolic and end-systolic endocardial contours were carefully traced by an experienced cardiologist who was unaware of the patient’s data. Global left ventricular ejection fraction and regional wall motion (standard deviation/chord) was calculated by the centreline method[14]. Regional wall motion was expressed as the mean values of standard deviation/chord in the anterolateral and apical regions. The degree of improvement of global left ventricular ejection fraction and regional wall motion was determined by subtracting their respective values at 1 month after the onset of acute myocardial infarction from their respective values at 6 months after the onset of acute myocardial infarction.

Cardiac enzyme measurements

Blood samples were obtained every 3 h during the first 24 h and once daily from the 2nd day until a normal value was obtained for determination of the peak serum creatine kinase activity.
Statistical analysis

Data are expressed as mean ± standard deviation. Categorical data were analysed by the Fisher’s exact test or chi-square test. Continuous variables were analysed by the paired or unpaired t-test. A P value <0·05 was considered statistically significant.

Results

Of the 74 patients studied, 29 (39%) had abnormal Q-wave regression (group A), and 45 (61%) did not have abnormal Q-wave regression (group B). Of 29 patients with Q-wave regression, seven had complete Q-wave regression, and 22 had partial Q-wave regression. Ten had Q-wave regression within 1 month after the onset of acute myocardial infarction, and 19 patients had it between 1 and 6 months after the onset of acute myocardial infarction.

There were no differences between the two groups in age, gender, the time elapsed from the onset of acute myocardial infarction to electrocardiogram recordings, the rate of unsuccessful reperfusion therapy, the re-occlusion rate, or the use of ACE inhibitor. All six patients whose infarct-related vessel was re-occluded on coronary arteriography performed at 1 or 6 months after the onset of acute myocardial infarction had no evidence of myocardial infarction. Group A tended to have lower peak serum creatine kinase levels than group B (2358 ± 1796 vs 3092 ± 1946 IU l⁻¹, P = 0·09) (Table 1).

Emergency coronary arteriographic features

Emergency coronary arteriograms showed that there were no differences between the two groups in the incidence of spontaneous recanalization, the presence of good collateral circulation, multivessel disease, or proximal left anterior descending coronary artery occlusion. The incidence of spontaneous recanalization or good collateral circulation was significantly higher in group A than in group B (55% vs 31%, P < 0·05).

Left ventricular wall motion

Global left ventricular ejection fraction at both 1 and 6 months after the onset of acute myocardial infarction was significantly greater in group A than in group B (1 month; 59 ± 12% vs 53 ± 11%, P < 0·05, 6 months; 64 ± 11% vs 52 ± 12%, P < 0·01) (Fig. 1). Regional wall motion at 1 and 6 months after the onset of acute myocardial infarction was significantly less reduced in group A than in group B (55% vs 31%, P < 0·05).

The degree of improvement of global left ventricular ejection fraction and regional wall motion was significantly greater in group A than in group B (left ventricular ejection fraction; 5·5 ± 7·7% vs 0·3 ± 7·2%, P < 0·01, regional wall motion (standard deviation/chord); 0·9 ± 0·9 vs 0·2 ± 0·7, P < 0·01) (Fig.3).

Discussion

Several investigators[7-10] have reported that patients with Q-wave regression after acute myocardial infarction...
have a smaller infarct size and better left ventricular wall motion than those without it. However, Iwasaki et al. recently reported that Q-wave regression after anterior wall acute myocardial infarction did not correlate with infarct size and left ventricular wall motion abnormality. Patient selection may contribute to this discrepancy: the earlier studies included patients with both anterior, and inferior wall acute myocardial infarction whereas Iwasaki et al. included only patients with anterior wall acute myocardial infarction. Thus, we selected only patients with anterior wall acute myocardial infarction. In the present study, patients with Q-wave regression tended to have smaller peak creatine kinase activity than those without it. In addition, patients with Q-wave regression had significantly greater left ventricular ejection fraction and better regional wall motion at both 1 and 6 months after the onset of acute myocardial infarction. These results suggest that patients with Q-wave regression had a relatively smaller infarct size than those without it. Our results disagree with those in Iwasaki’s work. Although the definition of Q-wave regression and the patient selection were similar to the two studies, there are two apparent differences between the two studies. First, in their study, the incidence of Q-wave regression and the patient selection were similar to the two studies, there are two apparent differences between the two studies. First, in their study, the incidence of Q-wave regression was extremely high (77%) compared with that in our patients (39%). Second, their study included nine patients who had Q-wave regression between 6 months and 5 years after acute myocardial infarction: there may be differences in clinical features according to the timing of Q-wave regression. Further studies with large populations are desirable to determine the relationship between the timing of Q-wave regression and infarct size or left ventricular wall motion abnormality.

To date, the relationship between Q-wave regression after acute myocardial infarction and the time course of left ventricular wall motion has not been clarified. We investigated this relationship and found that patients with anterior wall acute myocardial infarction showing Q-wave regression had a significantly greater improvement of left ventricular ejection fraction and regional wall motion between 1 and 6 months after the onset of acute myocardial infarction. This result indicates that patients with anterior wall acute myocardial infarction showing Q-wave regression had a greater amount of stunned myocardium. This has an important clinical implication. Since, as this study indicated, left ventricular wall motion improves between 1 and 6 months after the onset of acute myocardial infarction in some patients, evaluation of left ventricular wall motion abnormality made at 1 month after acute myocardial infarction may yield erroneously unfavourable results, especially in cases with anterior wall acute myocardial infarction showing Q-wave regression.

In acute myocardial infarction, post-ischaemic ventricular dysfunction such as cytoplasmic vacuoles, intramyofibrillar oedema, glycogen depletion, and reduced adenosine triphosphate concentrations last for several days following the ischaemic insult. These pathophysiological changes that interfere with mechanical function can prevent the energy-dependent process of normal electrical depolarization of the affected myocardium, and interference with depolarization can result in transient Q waves. After reperfusion, electrical function is recovered, and abnormal Q waves may disappear. Other mechanisms for Q-wave regression after acute myocardial infarction have been proposed: (1) a reinfarction involving the contralateral ventricular wall causes an apparent regrowth of Q waves over the area of previous infarct or (2) a new left bundle branch block alters activation pathways and masks prior Q waves. However, these two hypotheses do not apply to the patients in our study because those with previous myocardial infarction, subsequent myocardial infarction during the 6-month follow-up period or a new left bundle branch block were excluded.

Figure 3  The degree of improvement of left ventricular ejection fraction (LVEF) and regional wall motion between 1 and 6 months after anterior wall acute myocardial infarction. RWM = regional wall motion; † = group A; ■ = group B. †P < 0.01.
In the present study, the incidence of spontaneous recanalization or good collateral circulation was significantly higher in the patients with Q-wave regression. The higher incidence of spontaneous recanalization or good collateral circulation is thought to contribute to a relatively smaller amount of necrotic myocardium and a significantly larger amount of stunned myocardium in the patients with Q-wave regression. In addition, of interest is that patients with complete Q-wave regression had a higher incidence of spontaneous recanalization than those with partial Q-wave regression (71% vs 28%, P < 0.01).

In conclusion, patients with anterior wall acute myocardial infarction showing Q-wave regression had a trend towards a smaller amount of necrotic myocardium and a significantly larger amount of stunned myocardium.

References