SIGNIFICANCE OF Q WAVE DISAPPEARANCE IN THE CHRONIC PHASE FOLLOWING TRANSMURAL ACUTE MYOCARDIAL INFARCTION

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The mechanism and prognostic implications of Q wave regression following transmural acute myocardial infarction (AMI) were assessed in 54 patients. Of these subjects, 14 lost their Q waves. Exercise myocardial thallium-201 (^{201}Tl) scintigraphy and two-dimensional echocardiography were performed before the patients were discharged from hospital. Two-dimensional echocardiography and electrocardiography were simultaneously repeated about 18 months after AMI. Both the relative ^{201}Tl activity in the infarcted area and the improvement of echocardiographic wall motion index were higher in patients who had lost their Q waves than in those with retained Q waves ($70 \pm 14\%$ vs $58 \pm 13\%$, $p < 0.01$; 5.2 ± 3.0 vs 2.0 ± 3.4 , $p < 0.01$, respectively). The prevalence of post-infarction angina pectoris was significantly higher in the former (29% vs 0% , $p < 0.01$).

We concluded that remnants of viable myocardial muscle might be responsible for Q wave regression following transmural acute myocardial infarction, and the prevalence of post-infarction angina pectoris was high among these patients.

It has recently been recognized that loss of Q waves following transmural acute myocardial infarction (AMI) is more common than previously suspected!¹⁻³ However, published reports have been conflicting with regard not only to prevalence but also with prognostic implications. Discrepancies among the various series can be expected because the available studies deal with selected groups of patients and, in addition, lack

angiographic and myocardial ^{201}Tl scintigraphic data. Furthermore, the mechanisms of Q wave regression are not fully understood. It is possible that viable myocardial muscle in the infarcted area may play an important role in this respect.

We investigated this possibility in patients with documented Q wave regression, measuring relative ^{201}Tl activity (RA) and the improvement of chronic echocardiographic wall motion (ΔWMI) in the infarct area.

Key words:

Myocardial infarction
Q wave loss
Relative thallium-201 activity
Wall motion index
Post-infarction angina pectoris

MATERIALS AND METHODS

Between December 1985 and April 1988,

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TABLE I SUBJECTS

		No of cases	Age (yrs)
Anterior MI	Q (-)	10	60±7 (46-70)
	Q (+)	26	54±11 (28-75)
Inferior MI	Q (-)	4	57±15 (38-75)
	Q (+)	14	59±12 (38-73)

Q (-) = patients with lost waves;

Q (+) = patients with retained Q waves.

90 patients with AMI were admitted to our coronary care unit. AMI was defined by at least two of the following: 1) angina lasting longer than 30 min and unrelieved by rest, 2) serum CK-MB fraction greater than 5%, 3) inversion or elevation of the ST-T waves, 4) a Q wave with a duration of 0.04 sec. or more, or with a depth of 25% or more of the height of the R wave in the same complex in the leads, and 5) positive results from either the ^{99m}Tc pyrophosphate or ^{201}Tl scan⁴⁻⁷

Of the 75 survivors, 21 were excluded from study due either to noncardiac death (3 patients), a previous myocardial infarction (5 patients), non-Q wave AMI (7 patients), and 6 with an infarcted left circumflex artery (LCX) because it was difficult to obtain the precise relative ^{201}Tl activity (RA) values in the infarcted area. Thus a total of 54 patients with acute transmural myocardial infarction were included in this study. Transmural myocardial infarction was coded according to location as anterior, inferior or combined. Criteria for infarction were based on major Minnesota Code (1,1), moderate (1,2) or minor (3) electrocardiographic find-

ings, as determined by the duration and depth of Q waves and the leads in which they appear⁸. There were 36 patients with anterior myocardial infarction and 18 with inferior myocardial infarction. Of these patients, 10 had Q wave regression associated with anterior infarction, while 4 had Q wave regression associated with inferior infarction (Table I). All the patients were treated by conventional therapy, and none of the patients were treated by percutaneous transluminal coronary recanalization or percutaneous transluminal coronary angioplasty. Twelve-lead ECGs were recorded before hospital discharge and about 18 months after the onset of AMI (6-42 months). These two ECGs were used to determine the Q wave regression after transmural AMI. Regression of Q waves was defined as follows: Q wave loss and the r wave more than 0.1 mV had to appear in at least one of the Leads, V₁, V₂, V₃, V₄, V₅, V₆ in anterior MI, and one of the Leads, II, III, aV_F in inferior MI.

As well as recording the ECGs, two-dimensional echocardiograms were obtained using an electrical scan echocardiographic unit (Models SSH-60 A or SSH-65 A, Toshiba). We recorded them at the following views: long-axis view, short-axis view at the level of chordae tendineae or papillary muscle or apex, apical right anterior oblique view, apical left anterior oblique view and apical four chamber view.

As shown in Fig. 1, the left ventricle was divided into 13 segments. Considering all of the recorded echocardiograms, the degree of wall motion in each segment was classified on a 6-point scale scored as follows: 4: nor-

Fig.1. Schematic diagram of the left ventricle segment.

Fig.2. Quantitative method for analyzing the defect on myocardial scintigram.

A = ^{201}Tl activity of normal area; B = ^{201}Tl activity of infarcted area; Bcg = ^{201}Tl activity of upper mediastinal area as background

mal, 3: mild hypokinesis, 2: hypokinesis, 1: severe hypokinesis, 0: akinesis, -1: dyskinesis. The total score for each segment was defined as the wall motion index (WMI)⁹. Analysis of the echocardiograms was performed independently and blindly to the electrocardiographic and myocardial ^{201}Tl scintigraphic findings by different observers.

Exercise myocardial ^{201}Tl scintigraphy was performed about 4 weeks after the AMI. Symptom-limited exercise testing using the upright bicycle ergometer, was begun at the level of 25 watts and was increased by 25 watts every 3 min to achieve at least 85% of the maximal heart rate, chest pain, development of 1 mm of horizontal or downsloping ST depression on the ECG, or patient fatigue. Near peak exercise, ^{201}Tl , 2.0 mCi (74MBq) was injected intravenously and the patient continued to exercise for at least 1 min. Myocardial imaging was begun within 10 min after the injection and repeated 4 hr later. Images were obtained in the anterior and 45° and 70° left anterior oblique (LAO) projections using a gamma scintillation

TABLE II CLINICAL AND ANGIOGRAPHIC DATA

	Q (-)	Q (+)	p Value
No of patients	14	40	
Hypertension	5 (36)	19 (48)	NS
Diabetes mellitus	3 (21)	13 (33)	NS
Hyperlipidemia	4 (29)	5 (13)	NS
Smoking	10 (71)	34 (85)	NS
Hyperuricemia	3 (21)	6 (15)	NS
Family history	0 (0)	6 (15)	NS
Obesity	7 (50)	13 (33)	NS
Peak CPK (IU)	3231 ± 1421	4021 ± 2325	NS
Ejection fraction (%)	64 ± 11	50 ± 11	0.01
Coronary arteries narrowed >75% in diameter			
0	2 (15)	1 (2)	NS
1	9 (64)	28 (70)	NS
2	2 (14)	11 (28)	NS
3	1 (7)	0 (0)	NS
Total occlusion	5 (36)	21 (53)	NS
Coronary collaterals	6 (43)	15 (38)	NS

Parentheses mean %.

Q (-) = patients with lost Q waves; Q (+) = patients with retained Q waves

TABLE III ELECTROCARDIOGRAMS OF PATIENTS WITH Q WAVE LOSS

Patient No.	Age (yrs)	Sex	Infarcted site	Region of Q wave (acute)	Region of Q wave (chronic)	Interval until Q wave loss (month)	Post MI-AP
1.	63	M	Ant	V ₁ , V ₂ , V ₃	Absent	20	+
2.	63	M	Ant	III, aV _F , V ₁ , V ₂ , V ₃	V ₁	23	—
3.	68	M	Ant	aV _L , V ₁ , V ₂ , V ₃ , V ₄	V ₁	18	+
4.	57	M	Ant	aV _L , V ₁ , V ₂ , V ₃ , V ₄	aV _L	21	—
5.	59	M	Ant	V ₁ , V ₂ , V ₃	Absent	7	—
6.	59	F	Ant	V ₁ , V ₂	Absent	23	+
7.	56	M	Ant	V ₁ , V ₂ , V ₃ , V ₄	V ₂	33	—
8.	60	F	Ant	V ₁ , V ₂	V ₁	12	—
9.	70	M	Ant	I, aV _L , V ₁ , V ₂ , V ₃ , V ₄	V ₁	18	—
10.	46	M	Ant	aV _L , V ₁ , V ₂ , V ₃ , V ₄	V ₂ , V ₃	14	+
11.	57	M	Inf	II, III, aV _F	III	29	—
12.	57	M	Inf	III, aV _F	III	17	—
13.	38	M	Inf	II, III, aV _F	Absent	15	—
14.	75	M	Inf	II, III, aV _F	Absent	22	—

M = male; F = female; Ant = anterior; Inf = inferior; Post MI-AP = post-infarction angina pectoris.

Fig.3. Relative ²⁰¹Tl activity of infarcted area RA is higher in patients with lost Q waves than their counterparts. RA = relative ²⁰¹Tl activity; Q(-) = patients with lost Q waves; Q(+) = patients with retained Q waves

camera (Technicare sigma 438) equipped with a high resolution collimator: delayed images were acquired during the same period of time required to record the initial images. The initial images contained 300,000 counts in the entire field of view. Image analysis was done using the Sofer Medical

Fig.4. Wall motion index both in the acute phase and chronic phase.

WMI was higher in patients with lost Q waves than in their counterparts with retained Q waves both in the acute phase and chronic phase. In addition, WMI of both groups increased in the chronic phase as compared in the acute phase.

WMI = wall motion index; Q(-) = patients with lost Q waves; Q(+) = patients with retained Q waves

Fig.5. Improved rate of WMI in the chronic phase.

ΔWMI is significantly higher in the patients with lost Q waves than in their counterparts with retained Q waves.

Q (-) = patients with lost Q waves; Q (+) = patients with retained Q waves;

ΔWMI = improved rate of WMI

Fig.6. Correlation of RA with WMI of the chronic phase.

RA is related to WMI of the chronic phase.

RA = relative ^{201}Tl activity; WMI = wall motion index

Smis 4minicomputer.

RA is the value obtained from quantitative analysis of myocardial ^{201}Tl distribution

Occurrence of post-infarction angina pectoris

Fig.7. Occurrence rate of post-infarction angina pectoris.

The occurrence rate of post-infarction angina pectoris is significantly higher in the patients with lost Q waves than in their counterparts with retained Q waves.

Q (-) = patients with lost Q waves; Q (+) = patients with retained Q waves

on delayed image¹⁰⁻¹² That is, a region of interest (ROI) of 3×3 matrix was established in the infarcted area, the normal area, and the upper mediastinal area as the background (Fig. 2). RA is calculated as follows

$$\text{RA} (\%) = (B - \text{Bcg} / A - \text{Bcg}) \times 100.$$

A = ^{201}Tl activity of normal area

B = ^{201}Tl activity of infarcted area

Bcg = ^{201}Tl activity of upper mediastinal area as background

RA was obtained from 45° LAO view or 70° LAO view in anterior AMI, and was measured from the anterior view or 70° LAO view in inferior AMI.

Cardiac catheterization was performed 4 weeks after the acute event. Left ventriculography was carried out in the 30° right anterior oblique projection. Coronary arteriograms were obtained in multiple projections. Ejection fraction was determined using the area-length method¹³ Lesions of $<50\%$ reduction in diameter were considered to be nonobstructive. Total coronary occlusion was defined as the absence of forward flow of contrast material in the involved artery. Each patient was classified as

TABLE IV PATIENTS WITH POST-INFARCTION ANGINA PECTORIS

Patient No.	Age	Infarcted site	CAG	RA	²⁰¹ Tl scintigraphy (chronic phase)	Chronic WMI	ΔWMI
1.	63	Anterior	Seg.6: 90%	66%	Anterior:RD (+)	49	11
2.	68	Anterior	Seg.6: 99%	70%	Anterior:RD (+)	48	4
3.	59	Anterior	Seg.7: 99%	77%	Anterior:RD (+)	46	1
4.	46	Anterior	Seg.6: 99%	96%	Anterior:RD (+)	49	4

CAG = coronary angiogram; RA = relative ²⁰¹Tl activity; WMI = wall motion index; RD = reversible defect

having 1-, 2- or 3-vessel disease. Collaterals were judged to be well developed when the diameter of the receiving artery was measured >1 mm and the sending vessel showed no obstructive lesions. The data obtained from the above examinations were compared between patients with and without Q wave regression.

Lastly, we investigated the prevalence of post-infarction angina pectoris in each group. In order to observe the relationship between the occurrence of angina pectoris and the loss of Q wave, post-infarction angina pectoris in the present study was defined as one in which the ischemic area was coincident with the previous infarcted region on exercise myocardial ²⁰¹Tl scintigraphy. After all patients were told the objective of this study, they gave informed consent.

STATISTICS

Results are expressed as mean ± standard deviation. Statistical analysis was done by student's t test and χ^2 test. If variables were not continuous, statistical analysis was done by the Wilcoxon test. A p value less than 0.05 was considered to indicate statistical significance. Correlation coefficients were calculated using Spearman's rank test.

RESULTS

Baseline clinical data and catheterization findings are shown in Table II. Patients with lost Q waves tended to have a lower peak serum creatine kinase level (3231 ± 1421 vs 4021 ± 2325 IU) than their counterparts. There were no significant differences between the 2 groups with respect to age,

hypertension, hyperlipidemia, cigarette smoking, diabetes mellitus, hyperuricemia, family history and obesity. The ejection fraction was higher ($64 \pm 11\%$ vs $50 \pm 11\%$, $p < 0.01$) in those patients with lost Q waves than in those who retained them. The number of diseased arteries was comparable in both groups. Similarly, no differences were found between the 2 groups with regard to the presence of well developed collaterals or total occlusion of the involved coronary artery.

The electrocardiographic regions of Q wave loss and the duration between Q wave appearance and loss showed in Table III.

The RA in the infarcted area was significantly higher ($70 \pm 14\%$, $58 \pm 13\%$, $p < 0.01$) in the patients showing Q wave regression (Fig. 3).

WMI was higher in patients who lost Q waves than in those who did not, both in the acute phase and chronic phase (acute: 40.1 ± 6.4 vs 33.9 ± 9.6 $p < 0.05$, chronic: 45.4 ± 6.2 vs 35.9 ± 10.6 $p < 0.01$). In addition, WMI of both groups increased in the chronic phase as compared with the acute phase (Fig. 4). The rate of improvement of WMI was also higher in the patients with lost Q waves (5.2 ± 3.0 vs 2.0 ± 3.4 $p < 0.01$) (Fig. 5). RA was related to WMI of chronic phase ($r_s = 0.73$) (Fig. 6).

Post-infarction angina pectoris was significantly more frequent in the patients who had lost their Q waves than in their counterparts (29% vs 0%, $p < 0.01$) (Fig. 7). The RA of those with post-infarction angina pectoris tended to be high (66% - 96%) and the reversible defect was observed in the previously infarcted area. Coronary arteriography revealed severe stenosis (>90%) in the involved coronary arteries of 4 patients with

post-infarction angina pectoris (Table IV).

DISCUSSION

There have been many reports on the prevalence of Q wave regression following transmural AMI^{2,14-19}. Master et al¹⁴ showed that 21.3% of 202 patients lost their Q waves during a post-MI follow-up period ranging from 1 month to 4 years. Kaplan et al² reported that the Q wave regressed in 30% of 208 patients. In the present study, the disappearance rate of Q waves was similar to these previous reports.

Few reports of Q wave regression present angiographic data²⁰. We have shown that the number of diseased arteries was comparable in patients who lost their Q waves and in those who retained them. However, the former had better left ventricular function, as substantiated by a higher ejection fraction. In addition, the peak serum creatine kinase tended to be lower in those patients with lost Q waves. These data suggest a smaller infarct size in such patients. Karnegis et al²¹ showed a negative correlation between the size of the infarct assessed by an ECG score and the left ventricular ejection fraction in patients with healed myocardial infarction. Our results tend to concur.

The duration between Q wave appearance and loss is very important in analyzing mechanism of Q wave regression. However, the duration in the present study is not as important since it was difficult to determine the true time of Q wave loss by recording the ECG once in the chronic phase. Serial recordings of ECGs are necessary to elucidate the mechanism of Q wave regression.

Saito et al¹² showed that the RA was inversely related to left ventricular wall motion asynergy. A few other reports have shown viable myocardial muscle in the infarcted area with slight wall motion asynergy on the left ventriculogram^{22,23}. These data suggest that it is reasonable to consider RA as an index of myocardial viability. The high RA value in AMI patients with lost Q waves suggests that viable myocardial muscle may still remain in the infarcted area. The relationship between RA and chronic WMI shows that improved wall motion may be due to the compensated hypertrophy of remaining vi-

able myocardial muscle in the infarcted region²⁴ or the recovery of stunned myocardium.

The mechanism for Q wave regression has not been fully described previously, but several recent speculations exist. First, a new ventricular conduction disturbance may alter activation pathways and mask prior Q waves. Second, a reinfarction involving the contralateral ventricular wall may cause an apparent regrowth of r waves over the area of previous infarct⁷. Third, the regression of Q waves may result from the progressive reduction of scar tissue²⁵. Fourth, remnants of viable myocardial muscle in the infarcted area may be related to the disappearance of Q waves. In our study, the former 2 mechanisms are unlikely as demonstrated by ECG readings in individual patients and in the case of reinfarction, by the lack of clinical manifestations. Considering the high value of RA in the infarcted area in patients with lost Q waves, remaining viable myocardial muscle may have caused Q wave regression in the present study. Further, the greater improvement of wall motion in the infarcted area in patients with lost Q waves may be related not only to viable myocardial muscle, but also to a progressive reduction of scar tissue. Consequently, the latter 2 mechanisms may account for the Q wave loss observed in our series.

The prevalence of post-infarction angina pectoris was significantly higher among patients with lost Q waves. In 4 patients with post-infarction angina pectoris, there was high RA, that is to say, considerable viable myocardial muscle and greater improved wall motion in the chronic phase. Both the increased oxygen consumption of myocardial muscle accompanied by improved wall motion and severe stenosis of the coronary artery seem to account for the occurrence of angina pectoris. From these results, we believe it necessary to monitor for the occurrence of post-infarction angina pectoris in patients with lost Q waves and a severe stenotic infarcted coronary artery.

We conclude that remaining viable myocardial muscle might lead to Q wave regression following transmural AMI, and that the prevalence of post-infarction angina pectoris is high among such patients with lost Q

waves.

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