Clinical Implications of Serial Changes in ST-Segment Elevation After Reperfusion in Patients With Anterior Acute Myocardial Infarction

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Background In patients with acute myocardial infarction (AMI), the relationship of serial changes in ST-segment elevation after reperfusion to left ventricular (LV) function remains unclear.

Methods and Results The study group comprised 164 patients with reperfused anterior AMI within 6 h of symptom onset. The sum of ST-segment deviation was calculated on admission (Σ ST-admission), and 1 h (Σ ST-1 h) and 24 h (Σ ST-24 h) after reperfusion. ST resolution was defined as a reduction in Σ ST-1 h of $\geq 50\%$ as compared with Σ ST-admission. Patients were classified into 3 groups: group A, 82 patients with ST resolution in whom Σ ST-1 h $\geq \Sigma$ ST-24 h; group B, 37 patients with ST resolution in whom Σ ST-1 h $< \Sigma$ ST-24 h; group C, 45 patients without ST resolution. Peak creatine kinase were higher in groups B and C than in group A ($4,578 \pm 2,176$, $4,236 \pm 2,638$, $2,222 \pm 1,926$ mU/ml, $p < 0.01$). At 6 months follow-up, the LV ejection fraction were lower in groups B and C than in group A (53 ± 8 , 54 ± 12 , $62 \pm 9\%$, $p < 0.01$).

Conclusions An increase in ST-segment elevation 1–24 h after reperfusion, despite ST resolution, is associated with a larger infarction and poorer LV function in patients with reperfused anterior AMI. (Circ J 2008; 72: 409–414)

Key Words: Electrocardiogram; Myocardial infarction; ST-segment

The main goals of reperfusion therapy for acute myocardial infarction (AMI) are early, sustained patency of the infarct-related artery (IRA) and optimal microvascular reperfusion.^{1–6} Recent studies have shown that resolution of ST-segment elevation (ST resolution) on the 12-lead electrocardiogram (ECG) after reperfusion is associated with adequate microvascular perfusion in AMI and may predict smaller infarct size, better left ventricular (LV) function, and better outcomes.^{7–10} However, we previously reported that some patients have a larger infarct size, LV dysfunction, and inadequate myocardial perfusion despite ST resolution, suggesting that ST resolution does not consistently predict myocardial salvage.¹¹ Moreover, Brodie et al reported a poorer correlation between ST resolution and late cardiac mortality in anterior myocardial infarction (MI) than in the non-anterior type.¹² To clarify the clinical significance of ST resolution in more detailed, we examined the relationship between ST-segment changes up to 24 h after reperfusion and LV function in patients with reperfused anterior wall AMI.

Methods

Study Population

We studied 164 consecutive patients (mean age 62 ± 11 years, range 30–86; 134 men, 30 women) with anterior AMI who were admitted to hospital and fulfilled the following criteria: (1) reperfusion (defined as restoration of Thrombolysis In Myocardial Infarction (TIMI) grade 3 flow) of the left anterior descending artery within 6 h of symptom onset and persistent patency of the IRA at a mean of 14 days and 6 months as confirmed by coronary angiography (CAG); (2) absence of conditions precluding the evaluation of ST-segment changes on ECG (eg, left or right bundle branch block, ventricular pacing); (3) ECG recorded on admission and at 1 h and 24 h after reperfusion; (4) absence of cardiogenic shock on admission. Patients with a re-elevation of serum creatine kinase (CK) (ie, re-infarction) during hospitalization were also excluded, because re-occlusion of the IRA could cause recurrent ST-segment elevation. The diagnosis of AMI was based on typical chest pain lasting ≥ 30 min, ST-segment elevation ≥ 2.0 mm in ≥ 2 contiguous precordial leads, and a typical increase in the serum CK level to more than twice the upper limit of normal. Pre-infarction angina was defined as the presence of typical chest pain occurring at rest or during exercise and persisting for less than 30 min within 24 h of the onset of AMI.¹³ The protocol was approved by the hospital's internal review board and all patients gave informed consent.

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Fig 1. (A) Electrocardiograms (ECGs) from a patient with ST resolution in whom the Σ ST at 1 h was greater than the value at 24 h (group A). The sum of ST-segment deviation on admission (Σ ST-admission), and at 1 h (Σ ST-1 h) and 24 h (Σ ST-24 h) after reperfusion were 32, 3, and 2.5 mm, respectively. The culprit lesion was segment 7. Time from symptom onset to reperfusion was 3.1 h. Left ventricular ejection fraction (LVEF) was 61% at 6 months later. (B) ECGs from a patient with ST resolution in whom Σ ST at 1 h was less than that at 24 h (group B). The Σ ST-admission, Σ ST-1 h and Σ ST-24 h were 32, 9, and 18 mm, respectively. The culprit lesion was segment 6. Time from symptom onset to reperfusion was 4.1 h. LVEF was 47% at 6 months later. (C) ECGs from a patient without ST resolution (group C). The Σ ST-admission, Σ ST-1 h and Σ ST-24 h were 28, 21, and 8 mm, respectively. The culprit lesion was segment 6. Time from symptom onset to reperfusion was 2.3 h. LVEF was 54% at 6 months later.

CAG

CAG was performed immediately after admission and reperfusion (defined as TIMI grade 3 flow) was established by thrombolysis in 52 patients and by primary or facilitated coronary angioplasty in 98. The grade of collateral filling in the left descending coronary artery was evaluated as de-

scribed by Rentrop et al¹⁴. A good collateral channel was defined as grade 2 or 3. The allocation of reperfusion therapy was left to the physician's discretion. Reperfusion time was defined as that from symptom onset to the time of confirming TIMI grade 3 flow angiographically. In patients with an improvement in symptoms and a decrease in ST-

Table 1 Baseline Characteristics

	Group A ST resolution $\Sigma ST-1\text{ h} \geq \Sigma ST-24\text{ h}$ (n=82)	Group B ST resolution $\Sigma ST-1\text{ h} < \Sigma ST-24\text{ h}$ (n=37)	Group C No ST resolution (n=45)	p value
Age (years)	60±11	64±11	63±12	0.07
Men	70 (85%)	28 (76%)	37 (82%)	0.44
Heart rate on admission (beats/min)	81±18	83±19	88±21	0.20
SBP on admission (mmHg)	149±29	148±28	149±30	0.97
Killip ≥ 2 on admission	2 (2%)	2 (5%)	6 (13%)	0.048
Previous angina within 24 h before AMI	30 (37%)	10 (27%)	22 (49%)	0.11
Good collateral circulation*	6/18 (33%)	9/22 (41%)	11/27 (41%)	0.22
Percutaneous coronary intervention	33 (41%)	30 (81%)	35 (77%)	<0.001
Time from symptom onset to reperfusion (min)	139±68	202±94	187±108	0.002
Current smoker	58 (71%)	24 (65%)	26 (58%)	0.32
Hypercholesterolemia	45 (55%)	19 (51%)	28 (62%)	0.61
Diabetes mellitus	23 (28%)	13 (35%)	17 (38%)	0.52
Hypertension	34 (41%)	21 (57%)	26 (58%)	0.16
Multivessel disease	19 (23%)	12 (32%)	12 (27%)	0.61
Culprit lesion (segment 6)	54 (66%)	21 (57%)	30 (67%)	0.53
TIMI flow grade 0 or 1 at initial angiography	18 (22%)	22 (59%)	27 (60%)	<0.001
Discharge medications				
Aspirin	82 (100%)	37 (100%)	45 (100%)	1.00
ACEI/ARB	66 (80%)	32 (86%)	37 (82%)	0.76
-blocker	39 (48%)	21 (57%)	31 (69%)	0.10
Statin	54 (66%)	23 (62%)	28 (62%)	0.83

*Grade 2 or 3 collateral flow to the left anterior descending coronary artery only in patients who had TIMI flow grade 0 or 1 on initial angiography.

Data are mean values±SD or numbers (%) of patients.

$\Sigma ST-1\text{ h}$, the sum of ST-segment deviation 1 h after reperfusion; $\Sigma ST-24\text{ h}$, the sum of ST-segment deviation 24 h after reperfusion;

SBP, systolic blood pressure; AMI, acute myocardial infarction; TIMI, Thrombolysis In Myocardial Infarction; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; SD, standard deviation.

segment deviation (ΣST) before cardiac catheterization in whom TIMI grade 3 flow was confirmed at initial angiography, reperfusion time was defined as that from symptom onset to the time when the improvement in symptoms and the decrease in ST-segment elevation were confirmed.

Electrocardiographic Analysis

The 12-lead ECG was recorded at a paper speed of 25 mm/s and an amplification of 10 mm/mV. All ECGs were reviewed by an observer who was unaware of the patients' clinical data. The isoelectric line was defined as the level of the preceding TP segment. The ST-segment was measured 20 ms after the J point, and the sum of ΣST on admission, and at 1 h and 24 h after reperfusion was calculated by previously described methods.¹⁵ The negative value of the ST-segment elevation in the II, III, and aVF leads were added to the positive value of the ST-segment of anterior leads. ST resolution was defined as a reduction of at least 50% in ΣST on ECGs obtained 1 h after reperfusion as compared with the value on admission. The numbers of leads with ST-segment elevation ≥ 1 mm were measured on the admission ECG.¹⁶ The 32-point Selvester QRS score was also calculated,¹⁷ because since because it has been validated in patients with AMI and strongly correlates with infarct size.¹⁸ The difference in the QRS scores before and after reperfusion reflects the amount of myocardial damage related to reperfusion injury.⁷ Additional ST-segment elevation of at least 5 mm in leads V1–6 immediately after reperfusion was defined as additional ST-segment elevation.¹⁹

Blood Samples

Samples for measurement of serum C-reactive protein (CRP) levels were taken on admission and every 24 h for at least 7 days. Patients with infectious disease, collagen dis-

ease, malignant disease, or severe hepatic or renal failure were excluded from the analysis of CRP levels. Samples for measurement of serum CK were taken on admission and at 3-h intervals during the first 24 h, at 12-h intervals for the next 2 days, and then daily until discharge. Peak levels of CK and CK-MB and the areas under the curve (AUC) for CK and CK-MB as calculated by the linear-trapezoidal method^{20,21} were derived. Samples for measurement of plasma brain natriuretic peptide (BNP) levels were taken a mean of 14 days and 6 months after AMI.

Analysis of LV Function

LV function was evaluated on the right anterior oblique views of left ventriculograms obtained a mean of 14 days and 6 months after AMI. The LV ejection fraction (LVEF) was determined by the area–length method as described by Sandler and Dodge.²² Regional wall motion in the territory of the IRA was assessed by the centerline method and expressed as standard deviation (SD)/chord.²³ Infarct size was assessed by calculating the percentage of chords in the infarct zone >2 SDs below normal wall motion (percent abnormally contracting segment).

Statistical Analysis

Data are expressed as mean values±SD for continuous variables and as percentages for categorical variables. Continuous variables were compared by nonparametric tests (Mann-Whitney U test between groups). Chi-square analysis or Fisher's exact test was used to compare categorical variables. Differences were considered significant at $p < 0.05$. Statistical analysis was performed with StatView, version 5.0 (SAS Institute, Cary, NC, USA). The serial changes of the ST segment among groups were analyzed by mixed effect model. Data were analyzed using SAS (version 8.2,

Fig 2. Course of the sum of ST-segment elevation from admission to 24h after reperfusion. * $p < 0.01$ vs group A, # $p < 0.01$ vs group C.

SAS Institute).

Multivariate logistic regression analysis was used to identify clinical predictors of larger infarct size defined as the upper tertile of the AUC of CK-MB among the variables associated ($p < 0.05$) with this index on univariate analysis. Odds ratios and 95% confidence intervals were calculated. Data were analyzed using SPSS (Release 10, SPSS Inc, Chicago, IL, USA).

Results

Of the 164 patients, 119 had ST resolution, and 45 did not. Patients with ST resolution were classified according to the change in the Σ ST value between 1 and 24h after reperfusion: group A, 82 patients with ST resolution in whom Σ ST at 1h was equivalent to or greater than Σ ST at 24h (Σ ST-1h $\geq$ Σ ST-24h); and group B, 37 patients in whom Σ ST at 1h was less than Σ ST at 24h (Σ ST-1h $<$ Σ ST-24h). The other patients without ST resolution comprised group C. Representative cases are shown in Fig 1.

Patients' Characteristics

There were no significant differences among the 3 groups with regard to age, sex, systolic blood pressure and heart rate on admission, previous angina, coronary risk factors, culprit lesion, collateral circulation, or multivessel disease. Medications at discharge were similar among the 3 groups. Patients who were in Killip ≥ 2 were more frequent in groups B and C. Percutaneous coronary intervention was more frequently performed in groups B and C. The time from symptom onset to reperfusion was longest in group B, and the

TIMI flow grade 0 or 1 at initial angiography was less frequent in group A (Table 1).

Electrocardiographic Findings

Both the number of leads with ST elevation ≥ 1 mm and the Σ ST value on admission were highest in group B. The Σ ST value at 1h after reperfusion was higher in group C than in the other 2 groups, but was similar in groups A and B (Fig 2). Additional ST-segment elevation immediately after reperfusion occurred more frequently in groups B and C. The QRS score on admission and at 1h and 24h after reperfusion was also higher in groups B and C than in group A (Table 2).

Infarct Size and LV Function

Peak CK, CK-MB activity, AUC for CK, CK-MB and CRP level 3 days after AMI were highest in group B, followed by group C, and lowest in group A. LVEF and regional wall motion were lowest and the percent abnormally contracting segment and LV end-diastolic volume index was largest in group B 6 months later. The plasma BNP level 6 months later was highest in group B, followed by group C, and lowest in group A (Table 3).

Predictors of Larger Infarct Size

In the multivariate models, ST resolution with an increase in the Σ ST between 1 and 24h, no ST resolution, Killip class ≥ 2 on admission, culprit lesion (segment 6), and TIMI grade 0 or 1 on initial CAG were independent predictors of larger infarct size defined as the upper tertile of the AUC of CK-MB (Table 4). The other variables, including pre-infarction angina ($p = 0.06$) and percutaneous coronary intervention ($p = 0.59$), which were associated with larger infarct size on univariate analysis, were not significant predictors of larger infarct size.

Discussion

Most previous studies have found that ST resolution after reperfusion is associated with a smaller infarct size and better LV function in patients with AMI. In our study, however, patients in whom the Σ ST increased between 1 and 24h after reperfusion had a larger infarct size and poorer LV function, despite the presence of ST resolution according to the widely acceptable criteria.¹⁵ These poorer results were similar to those in patients without ST resolu-

Table 2 Electrocardiographic Findings

	Group A ST resolution Σ ST-1h $\geq$ Σ ST-24h (n=82)	Group B ST resolution Σ ST-1h $<$ Σ ST-24h (n=37)	Group C No ST resolution (n=45)	p value
No. of leads with ST elevation ≥ 1 mm	4.9 $\pm$ 1.5	5.6 $\pm$ 1.3	4.9 $\pm$ 0.9	0.025
Sum of ST-segment elevation: Σ ST (mm)				
On admission	19 $\pm$ 10	25 $\pm$ 10	18 $\pm$ 6	<0.001
1 h after reperfusion	5 $\pm$ 4*	7 $\pm$ 3*	12 $\pm$ 4*	<0.001
24 h after reperfusion	4 $\pm$ 3*	9 $\pm$ 3*†	9 $\pm$ 4*†	<0.001
Additional ST-segment elevation	29 (35%)	23 (62%)	28 (62%)	<0.001
QRS score				
On admission	2.3 $\pm$ 2.4	3.6 $\pm$ 2.2	3.9 $\pm$ 2.7	0.002
1 h after reperfusion	3.8 $\pm$ 3.3	6.6 $\pm$ 2.7	6.5 $\pm$ 2.8	<0.001
24 h after reperfusion	4.1 $\pm$ 3.4	7.4 $\pm$ 2.5	7.3 $\pm$ 3.1	<0.001

Data are mean values $\pm$ SD or numbers (%) of patients.

* $p < 0.01$ vs value on admission, † $p < 0.05$ vs value at 1 h after reperfusion.

Abbreviations see in Table 1.

Table 3 Infarct Size and LV Function at Discharge and 6 Months Later

	Group A ST resolution $\Sigma ST-1\text{ h} \geq \Sigma ST-24\text{ h}$ (n=82)	Group B ST resolution $\Sigma ST-1\text{ h} < \Sigma ST-24\text{ h}$ (n=37)	Group C No ST resolution (n=45)	p value
Peak CK (mU/ml)	2,222±1,926	4,578±2,176	4,236±2,638	<0.001
Peak CK-MB (mU/ml)	205±170	427±197	393±276	<0.001
AUC for CK (mU/ml×h)	65,992±49,714	128,895±78,543	118,956±72,242	<0.001
AUC for CK-MB (mU/ml×h)	4,788±3,372	8,588±3,137	8,465±5,331	<0.001
CRP levels at 3 days after AMI (mg/dl)	6.1±4.0	8.6±4.6	8.1±4.2	<0.02
<i>At discharge</i>				
LV ejection fraction (%)	59±11	51±10	49±13	<0.001
Regional wall motion (SD/chords)	-2.3±1.1	-3.2±0.8	-3.2±0.9	<0.001
Percent abnormally contracting segment	22±17	38±14	38±14	<0.001
LV end-diastolic volume index (ml/m ²)	69±15	77±15	75±17	0.08
Plasma BNP level (pg/ml)	152±195	288±276	410±318	<0.01
<i>6 months later</i>				
LV ejection fraction (%)	62±9	53±8	54±12	<0.001
Regional wall motion (SD/chords)	-2.1±1.0	-3.1±0.5	-2.9±0.9	<0.001
Percent abnormally contracting segment	20±16	40±9	33±16	<0.001
LV end-diastolic volume index (ml/m ²)	65±15	81±16	77±20	<0.001
Plasma BNP level (pg/ml)	50±39	140±127	126±147	<0.03

Data are mean values ± SD.

LV, left ventricular; CK, creatine kinase; CK-MB, myocardial bound fraction of CK; AUC, area under the curve; CRP, C-reactive protein; BNP, brain natriuretic peptide. Other abbreviations see in Table 1.

Table 4 Multivariate Predictors of Larger Infarct Size Defined as the Upper Tertile of the AUC of CK-MB

	OR	95% CI	p value
Group A	Referent		
Group B	5.52	1.43–22.8	0.013
Group C	5.10	1.40–27.8	0.022
Killip ≥2 on admission	9.04	1.02–80.8	0.049
Culprit lesion (segment 6)	4.01	1.85–13.8	0.040
TIMI grade 0 or 1 at initial angiography	9.56	2.32–39.3	0.002

OR, odds ratio; CI, confidence interval. Other abbreviations see in Tables 1, 3.

tion. It is unclear why patients who had ST resolution with an increase in the ΣST between 1 and 24 h after reperfusion had poorer LV function. In this study, there were no significant differences in previous angina or collateral circulation among the 3 groups. However, patients who had ST resolution with an increase in ΣST between 1 and 24 h after reperfusion had a greater number of leads with ST-segment elevation ≥ 1 mm, and higher ΣST and QRS score on the admission ECG, suggesting more severe myocardial damage in a larger area at risk before reperfusion.^{24,25} We speculate that the mechanism underlying ST resolution differs between patients who had ST resolution with and without an increase in the ΣST between 1 and 24 h after reperfusion. Both patient groups had little residual ongoing ischemic myocardial injury 1 h after reperfusion, as reflected by ST resolution.^{6,26} However, the myocardium has been already irreversibly damaged in the former, whereas the latter patients had a relatively large amount of viable myocardium. It is plausible that more severe, rapidly progressing myocardial damage before and during reperfusion may no longer cause ST-segment elevation after reperfusion. Furthermore, we speculate that the cause of ST-segment elevation 24 h later might be different from that at 1 h later in patients with ST resolution and an increase in the ΣST between 1 and 24 h after reperfusion. We found that such patients had higher CRP levels 3 days after AMI, suggesting a more intense inflammatory response after reperfusion. Higher CRP levels may simply reflect a larger infarct size; however,

Anzai et al suggested that the CRP level early after AMI reflects the healing of the infarct rather than the extent of myocardial necrosis.^{27,28} The excessive inflammatory response to the healing process of myocardial necrosis in these patients may be 1 of the factors affecting ST-segment elevation 24 h later. Pericarditis²⁹ or infarct expansion³⁰ has also been associated with ST-segment elevation. Shimizu et al reported that expansion of the infarcted portion might contribute to ST-segment re-elevation.³⁰ In our study, the LV end-diastolic volume index at discharge was largest in patients who had ST resolution with an increase in the ΣST between 1 and 24 h after reperfusion.

Recently, Brodie et al reported a poorer correlation between ST resolution and late cardiac mortality in anterior MI than in the non-anterior type¹² and our results partly explain that finding because in the present study patients with ST resolution in whom ΣST increased between 1 and 24 h after reperfusion had a larger infarct size and poorer LV function.

Study Limitations

First, it was a small retrospective study of only patients with reperfusion anterior wall AMI within 6 h of symptom onset. Second, we defined ST resolution according to the criteria proposed by Lemos et al,¹⁵ which may be less objective and accurate than continuous ST-segment tracking with an automated analysis system. Continuous ST-segment tracking is accurate, but requires greater input from medi-

cal and nursing staff and can lead to technical difficulties. We therefore evaluated ST resolution statically from 12-lead ECGs. Third, the presence of ischemia in the non-infarcted area might influence the degree of ST-segment deviation in the infarcted area on the admission ECG.³¹ However, the rates of multivessel disease were not significantly different among the 3 groups.

Conclusions

Our findings suggest that ST-segment analysis 24h after reperfusion can provide further information that complements the results of ST-segment analysis 1h after reperfusion. Such information may be useful for identifying patients who are less likely to benefit from early reperfusion, even if they have ST resolution.

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