

Relationship Between Terminal QRS Distortion on the Admission Electrocardiogram and the Time Course of Left Ventricular Wall Motion in Anterior Wall Acute Myocardial Infarction

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In order to clarify the time course of left ventricular (LV) wall motion in patients with anterior acute myocardial infarction (AMI) showing terminal QRS distortion on the admission electrocardiogram (ECG), the present study examined 106 patients with their first anterior AMI (≤ 6 h) who underwent emergency coronary arteriography and cardiac catheterization at 1 and 6 months after the infarction. The patients were classified into 2 groups according to the presence (group A, $n=23$) or absence (group B, $n=83$) of terminal QRS distortion (emergence of the J point at $\geq 50\%$ of the R-wave amplitude in leads with QR configuration and/or absence of S waves in leads with RS configuration) on the admission ECG. Group A had a lower LV ejection fraction and more reduced regional wall motion (RWM) in the infarct region at both 1 and 6 months after AMI than group B. The degree of improvement in RWM between 1 and 6 months after AMI was less in group A than in group B (-0.1 ± 0.5 vs 0.4 ± 0.6 SD/chord, $p < 0.01$). This study indicates that patients with anterior AMI showing terminal QRS distortion on the admission ECG have more severely depressed LV wall motion and less improvement in RWM in the infarct region in the healing stage, suggesting that this sign is an indicator of severe myocardial damage. (*Jpn Circ J* 2001; 65: 63–66)

Key Words: Acute myocardial infarction; Electrocardiography; QRS

Many studies have tried to clarify the value of the admission electrocardiogram (ECG) for predicting infarct size or prognosis in patients with anterior acute myocardial infarction (AMI).^{1–6} In these studies, much attention has been paid to the magnitude of ST-segment elevation, such as the sum of ST-segment elevation or the number of leads with ST-segment elevation. In contrast, the QRS morphology on the admission ECG in patients with AMI has been paid little attention, but was recently investigated by Birnbaum et al^{7–10} who found that a specific QRS morphology (ie, distortion of the terminal portion of the QRS complex) is independently associated with increased in-hospital mortality in these patients. However, the relation of this ECG finding to the time course of left ventricular (LV) wall motion is unclear, so we sought to clarify this relationship in the healing stage of a first anterior AMI.

Methods

Patients

Between January 1991 and December 1998, we had 176 patients with anterior AMI undergo emergency coronary arteriography within 6 h of the onset of symptoms. The data on these patients had been prospectively entered into a computer database, out of which we selected a total of 106

patients (84 men, 22 women; mean age, 60 ± 9 years) who met the following criteria: (1) typical chest pain lasting for ≥ 30 min; (2) ST-segment elevation ≥ 0.2 mV and upright T waves in ≥ 2 adjacent precordial leads on the admission ECG; (3) an increase in serum creatine kinase (CK) more than twice the upper limit of normal; (4) no history of previous myocardial infarction; (5) lack of bundle branch block or intraventricular conduction disturbance, and no evidence of LV hypertrophy confirmed by echocardiography; (6) technically adequate ECG recordings; (7) no other heart or lung disease; (8) identification of the infarct-related lesion by emergency coronary arteriography; and (9) the performance of coronary arteriography and left ventriculography at 1 and 6 months after anterior AMI. Informed consent for revascularization therapy and follow-up catheterization had been obtained from all patients.

Twelve-Lead ECGs

Standard 12-lead ECGs were recorded at a paper speed of 25 mm/s and a standardization of 10 mm = 1 mV. We evaluated the presence or absence of terminal QRS distortion on the admission ECG using the definition of Birnbaum et al:^{7–10} (1) pattern A = emergence of the J point at $\geq 50\%$ of the R-wave amplitude in leads with qR configuration; or (2) pattern B = disappearance of the S waves in leads with an Rs configuration (leads V₁–V₃). Distortion of the terminal portion of the QRS complex was defined as pattern A and/or pattern B in at least 2 adjacent precordial leads. All ECGs were analyzed by consensus of 2 observers who were blinded to the patients' angiographic and clinical data. Representative ECGs obtained from patients with and without terminal QRS distortion are shown in Fig 1.

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Emergency Coronary Arteriography and Reperfusion Therapy

Emergency coronary arteriography was performed using either the Judkins or Amplatz technique. Proximal left anterior descending coronary artery (LAD) occlusion was defined as occlusion of the artery proximal to its first septal branch. The coronary flow in the infarct-related artery was graded according to the classification used in the Thrombolysis in Myocardial Infarction (TIMI) trial.¹¹ The grade of collateral filling in the LAD was determined according to the criteria of Rentrop et al.¹² A collateral circulation with a grade of 2 or 3 was defined as good. If total occlusion of

the LAD was confirmed by initial coronary arteriography, intracoronary isosorbide dinitrate was administered, followed by intracoronary thrombolysis with urokinase or tissue plasminogen activator (n=63), or primary coronary angioplasty (n=34). Rescue or immediate coronary angioplasty was performed in 27 patients. Successful reperfusion was defined as the establishment of TIMI grade 3 flow in the infarct-related artery.

Left Ventriculography

Coronary arteriography and left ventriculography in the chronic phase were performed at 1 and 6 months after anterior AMI. The left ventriculogram performed in the 30-degree right anterior oblique projection was analyzed for LV ejection fraction (LVEF) and regional wall motion by an experienced cardiologist who was unaware of the patients' data. LVEF was calculated by the area-length method.¹³ Regional wall motion (RWM) was calculated by the center-line method¹⁴ and expressed as the mean values of SD/chord in the anterolateral and apical region (chord number 21–60). The mean of absolute differences between the 1st and 2nd measurements of RWM in the same observer in 20 patients with anterior AMI was 0.3 ± 0.25 SD/chord.

Measurements of CK Levels

In order to determine the peak CK level, 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.

Statistical Analysis

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

Results

The patients were divided into 2 groups according to the presence (group A, n=23) or absence (group B, n=83) of distortion of the terminal QRS complex on the admission ECG.

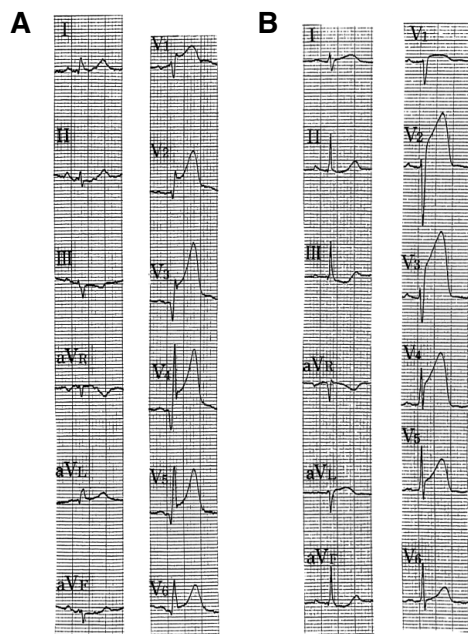


Fig 1. Representative electrocardiograms obtained from patients with (A) and without (B) terminal QRS distortion on the admission ECG.

Table 1 Clinical and Angiographic Characteristics

	Group A (n=23)	Group B (n=83)	p value
Age (years)	59 $\pm$ 10	60 $\pm$ 8	NS
Men	22 (96%)	62 (75%)	NS
Time to admission (min)	151 $\pm$ 72	153 $\pm$ 81	NS
Preinfarction angina	6 (26%)	43 (52%)	<0.01
Emergency coronary arteriography			
TIMI flow 3	1 (4%)	6 (7%)	NS
Good collaterals	7 (30%)	28 (34%)	NS
Multivessel disease	9 (39%)	19 (23%)	NS
Proximal LAD occlusion	15 (65%)	51 (61%)	NS
Reperfusion therapy			
Intracoronary thrombolysis	15 (65%)	48 (58%)	NS
PTCA	19 (83%)	42 (51%)	NS
Direct PTCA	8 (35%)	26 (31%)	NS
Unsuccessful reperfusion	2 (9%)	18 (22%)	NS
Peak CK (IU/L)	4617 $\pm$ 2619	2695 $\pm$ 1768	<0.01
TIMI flow 3 at 1 month after AMI	22 (96%)	77 (93%)	NS
Revascularization at 1 month after AMI	4 (17%)	20 (24%)	NS
TIMI flow 3 at 6 months after AMI	21 (91%)	77 (93%)	NS
Angiotensin-converting enzyme inhibitor	7 (30%)	26 (31%)	NS

Data presented are mean $\pm$ SD or number (%). TIMI, Thrombolysis in Myocardial Infarction; LAD, left anterior descending coronary artery; PTCA, percutaneous transluminal coronary angioplasty; CK, creatine kinase; AMI, acute myocardial infarction.

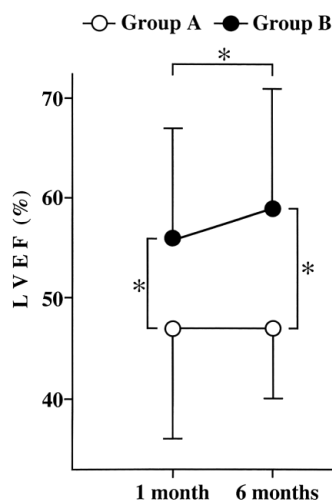


Fig2. Left ventricular ejection fraction (LVEF) at 1 and 6 months after anterior acute myocardial infarction. * $p < 0.01$.

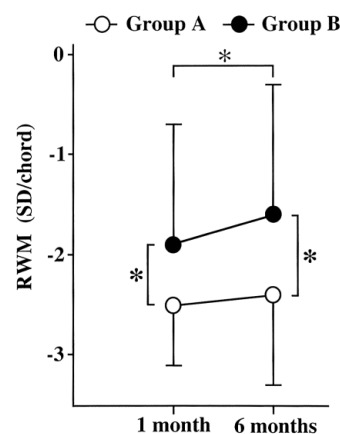


Fig3. Regional wall motion (RWM) in the infarct region at 1 and 6 months after anterior acute myocardial infarction. * $p < 0.01$.

Table 2 Degree of Improvement in LVEF and RWM Between 1 and 6 Months After Anterior AMI

	Group A (n=23)	Group B (n=83)	p value
LVEF (%)	-0.4 ± 6.7	2.9 ± 5.6	< 0.05
RWM (SD/chord)	-0.1 ± 0.5	0.4 ± 0.6	< 0.01

Data presented are mean $\pm$ SD. LVEF, left ventricular ejection fraction; RWM, regional wall motion.

ECG. Of 23 patients with terminal QRS distortion, patterns A, B, and both patterns together were observed in 19, 2, and 2 patients, respectively. Table 1 shows the clinical and angiographic characteristics; there was no difference between the 2 groups in age, gender, or the time elapsed from the onset of anterior AMI to admission. The frequency of preinfarction angina was lower in group A than in group B (26% vs 52%, $p < 0.01$). Emergency coronary arteriograms showed no difference between the 2 groups in the incidence of TIMI grade 3 flow on the initial angiogram, good collateral circulation to the LAD, multivessel disease, or proximal LAD occlusion. Methods of reperfusion therapy or the rate of unsuccessful reperfusion therapy did not differ between the 2 groups. Group A had greater peak CK levels than group B (4617 ± 2619 vs 2695 ± 1768 IU/L, $p < 0.01$). The incidence of TIMI grade 3 on coronary arteriography at 1 and 6 months after anterior AMI did not differ between the 2 groups. There were no differences between the 2 groups in the frequency of administration of angiotensin-converting enzyme inhibitor or the number of patients who underwent coronary angioplasty at 1 month after anterior AMI.

LVEF at both 1 and 6 months after anterior AMI was lower in group A than in group B (1 month: $47 \pm 11\%$ vs $56 \pm 11\%$, $p < 0.01$; 6 months: $47 \pm 7\%$ vs $59 \pm 12\%$, $p < 0.01$) (Fig 2). Regional wall motion in the infarct region at both 1 and 6 months after anterior AMI was significantly reduced in group A than in group B (1 month: -2.5 ± 0.6 vs -1.9 ± 1.2 SD/chord, $p < 0.01$; 6 months: -2.6 ± 0.7 vs -1.6 ± 1.3 SD/chord, $p < 0.01$) (Fig 3). Group B had significant improvement in LVEF and RWM in the infarct region between 1 and 6 months after anterior AMI, whereas group A did not (Figs 2, 3). The degree of improvement in LVEF and RWM between 1 and 6 months after anterior AMI was less in group A than

in group B (LVEF: $-0.4 \pm 6.7\%$ vs $2.9 \pm 5.6\%$, $p < 0.01$; RWM: -0.1 ± 0.5 vs 0.4 ± 0.6 SD/chord, $p < 0.01$) (Table 2).

Discussion

Our results indicate that patients with terminal QRS distortion on the admission ECG have a lower LVEF and more reduced RWM in the infarct region at both 1 and 6 months after anterior AMI. Furthermore, patients with terminal QRS distortion have less improvement in RWM in the infarct region between 1 and 6 months after anterior AMI than those without this ECG finding. These results show that the presence of terminal QRS distortion on the admission ECG reflects more severe and irreversibly ischemic myocardial damage in the setting of a first anterior AMI. Alternatively, the rate of developing myocardial necrosis may be more rapid in these patients with than in those without terminal QRS distortion. In the present study, patients without terminal QRS distortion on the admission ECG had a higher incidence of preinfarction angina than those with it. Preinfarction angina may prevent the development of terminal QRS distortion by its ischemic preconditioning effect, independent of collateral flow.¹⁵⁻²¹

Mechanism of Terminal QRS Distortion

The 5 major insertion sites of the proximal Purkinje conduction system into the ventricular subendocardial myocardium are at the distal extremity of the coronary circulation and are therefore affected by ischemia. The electrical excitation of both ventricles from these insertion sites is a combination of rapid spread throughout the endocardial peripheral Purkinje network and the slower spread from the endocardium through the mural myocardium. Severe re-

gional ischemia with electrical conduction delay through any of the endocardial sites of Purkinje insertion would affect initial and terminal QRS morphology.^{22,23} In particular, right septal conduction delay, combined right and left septal conduction delay, and anterosuperior, right septal and left septal conduction delay would be expected to produce a decrease in the amplitude of the S wave.^{22,23} Thus, terminal QRS distortion is thought to reflect the conduction delay caused by severe regional ischemia.

Study Limitations

First, the study sample was relatively small, and relatively few patients had terminal QRS distortion. Further studies with a larger population are needed to confirm our results. Second, 70 (40%) of the 176 patients with anterior AMI who were admitted within 6 h of the onset of anterior AMI and underwent emergency coronary arteriography were excluded from data analysis. Therefore, our results cannot be generalized to all patients with anterior AMI. In addition, further studies are needed to clarify whether our results can apply to patients with inferior or posterior AMI. Third, we have clarified the relationship between terminal QRS distortion and the time course of LV wall motion between 1 and 6 months after anterior AMI, but this relationship within 1 month of anterior AMI is still unclear and also must be clarified in future studies.

Conclusions

This study indicates that patients experiencing their first anterior AMI who have terminal QRS distortion on the admission ECG have more severely depressed LV wall motion and less improvement in RWM in the infarct region during the healing stage than those without this finding, suggesting that it may be an indicator of severe and irreversible ischemic damage.

References

1. Aldrich HR, Wagner NB, Boswick J, Corsa AT, Jones MG, Grande P, et al: Use of initial ST-segment deviation for prediction of final electrocardiographic size of acute myocardial infarcts. *Am J Cardiol* 1988; **61**: 749–753
2. Willems JL, Willems RJ, Willems GM, Arnold AER, van de Werf F, Verstraete M: Significance of initial ST segment elevation and depression for the management of thrombolytic therapy in acute myocardial infarction. *Circulation* 1990; **82**: 1147–1158
3. Clements IP, Kaufmann UP, Bailey KR, Pellikka PA, Behrenbeck T, Gibbons RJ: Electrocardiographic prediction of myocardial area at risk. *Mayo Clin Proc* 1991; **66**: 985–990
4. Clemmensen P, Grande P, Aldrich HR, Wagner GS: Evaluation of formulas for estimating the final size of acute myocardial infarcts from quantitative ST-segment elevation on the initial standard 12-lead ECG. *J Electrocardiol* 1991; **24**: 77–83
5. Christian TF, Gibbons RJ, Clements IP, Berger PB, Selvester RH, Wagner GS: Estimates of myocardium at risk and collateral flow in acute myocardial infarction using electrocardiographic indexes with comparison to radionuclide and angiographic measures. *J Am Coll Cardiol* 1995; **26**: 388–393
6. Arnold AER, Simoons ML: 'Expected infarct size without thrombolysis', a concept that predicts immediate and long-term benefit from thrombolysis for evolving myocardial infarction. *Eur Heart J* 1997; **18**: 1736–1748
7. Birnbaum Y, Sclarovsky S, Blum A, Mager A, Gabbay U: Prognostic significance of the initial electrocardiographic pattern in a first acute anterior wall myocardial infarction. *Chest* 1993; **103**: 1681–1687
8. Birnbaum Y, Herz I, Sclarovsky S, Zlotikamien B, Chetrit A, Olmer L, et al: Prognostic significance of the admission electrocardiogram in acute myocardial infarction. *J Am Coll Cardiol* 1996; **27**: 1128–1132
9. Birnbaum Y, Kloner RA, Sclarovsky S, Cannon CP, McCabe CH, Davis VG, et al, for the TIMI 4 investigators: Distortion of the terminal portion of the QRS on the admission electrocardiogram in anterior myocardial infarction and correlation with infarct size and long-term prognosis (Thrombolysis In Myocardial Infarction 4 Trial). *Am J Cardiol* 1996; **78**: 396–403
10. Birnbaum Y, Maynard C, Wolfe S, Mager A, Strasberg B, Rechavia E, et al: Terminal QRS distortion on admission is better than ST-segment measurements in predicting final infarct size and assessing the potential effect of thrombolytic therapy in anterior wall acute myocardial infarction. *Am J Cardiol* 1999; **84**: 530–534
11. The TIMI study group: The Thrombolysis in Myocardial Infarction (TIMI) trial: Phase I findings. *N Engl J Med* 1985; **312**: 932–936
12. Rentrop KP, Cohen M, Blanke H, Phillips RA: Changes in collateral channel filling immediately after controlled coronary artery occlusion by an angioplasty balloon in human subjects. *J Am Coll Cardiol* 1985; **5**: 587–592
13. Kennedy JW, Trenholme SE, Kasser IS: Left ventricular volume and mass from single-plane cineangiocardiograms: A comparison of antero-posterior and right anterior oblique methods. *Am Heart J* 1970; **80**: 343–352
14. Sheehan FH, Bolson EL, Dodge HT, Mathey DG, Schofer J, Woo HW: Advantages and applications of the centerline method for characterizing regional ventricular function. *Circulation* 1986; **74**: 293–305
15. Ottani F, Galvani M, Ferrini D, Sorbello F, Limonetti P, Pantoli D, et al: Prodromal angina limits infarct size: A role for ischemic preconditioning. *Circulation* 1995; **91**: 291–297
16. Nakagawa Y, Ito H, Kitakaze M, Kusuoka H, Hori M, Kuzuya T, et al: Effect of angina pectoris on myocardial protection in patients with reperfusion anterior wall myocardial infarction: Retrospective clinical evidence of 'preconditioning'. *J Am Coll Cardiol* 1995; **25**: 1076–1083
17. Anzai T, Yoshikawa T, Asakura Y, Abe S, Akaishi M, Mitamura H, et al: Preinfarction angina as a major predictor of left ventricular function and long-term prognosis after a first Q-wave myocardial infarction. *J Am Coll Cardiol* 1995; **26**: 319–327
18. Nagao K, Satou K, Arima K, Watanabe I, Yamashita M, Kanmatsuse K: Relationship between preinfarction angina and time interval to reperfusion with thrombolytic therapy in acute myocardial infarction. *Jpn Circ J* 1997; **61**: 843–849
19. Ishihara M, Sato M, Tateishi H, Kawagoe T, Shimatani Y, Kurisu S, et al: Implications of prodromal angina pectoris in anterior wall acute myocardial infarction: Acute angiographic findings and long-term prognosis. *J Am Coll Cardiol* 1997; **30**: 970–975
20. Andreotti F, Pasceri V, Hackett DR, Davies GJ, Haider AW, Maseri A: Preinfarction angina as a predictor of more rapid coronary thrombolysis in patients with acute myocardial infarction. *N Engl J Med* 1996; **334**: 7–12
21. Noda T, Minatoguchi S, Fujii K, Hori M, Ito T, Kanmatsuse K, et al: Evidence for the delayed effect in human ischemic preconditioning: Prospective multicenter study for preconditioning in acute myocardial infarction. *J Am Coll Cardiol* 1999; **34**: 1966–1974
22. Wagner NB, Sevilla DC, Krucoff MW, Lee KL, Pieper KS, Kent KK, et al: Transient alterations of the QRS complex and ST segment during percutaneous transluminal balloon angioplasty of the left anterior descending coronary artery. *Am J Cardiol* 1988; **62**: 1038–1042
23. Selvester RH, Wagner NB, Wagner GS: Ventricular excitation during percutaneous transluminal angioplasty of the left anterior descending coronary artery. *Am J Cardiol* 1988; **62**: 1116–1121