Relationship between Infarction Location and Size to QT Dispersion in Patients with Chronic Myocardial Infarction

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SUMMARY

The relationship between the location and size of an infarction and QT dispersion was investigated in 84 Japanese patients with chronic myocardial infarction (54 with anteroseptal infarction and 30 with inferior infarction). The control group consisted of 23 subjects without ischemic heart disease (13 normal subjects and 10 hypertensive patients). Corrected QT dispersion (maximum corrected QT interval minus minimum corrected QT interval: QTc dispersion), was significantly larger in the anterior infarction group than in the control group (69.9±21.5 msec vs 53.0±17.6 msec), while the inferior infarction group showed no significant difference from control subjects. QTc dispersion was significantly greater in the patients with large anterior infarcts than in those with small anterior infarcts (80.5±20.5 msec vs 61.9±18.8 msec). In patients with chronic myocardial infarction, QT dispersion is influenced by the infarct location and size. Accordingly, interpretation of QT dispersion data should take these factors into consideration. (Jpn Heart J 2002; 43: 455-461)

Key words: QT dispersion, Myocardial infarction

AFTER a myocardial infarction occurs, an increase in QT dispersion reflects the dispersion of ventricular repolarization, and an association between increased QT dispersion and severe arrhythmias has been reported.1-3) However, the dispersion of ventricular repolarization may depend on the time after myocardial infarction, the location and size of the infarct, the presence or absence of residual ischemia, and the patient's drug therapy. The effects of time after infarction,4) residual ischemia,5-7) and various drugs8,9) have already been studied. Regarding the infarct location and size, Raev, et al10) and Mirvis, et al11) have reported on the QT inter-
val, while Moreno, et al\textsuperscript{12} studied QT dispersion after acute myocardial infarction. However, no assessments have been done in the chronic phase. This study was performed to clarify QT dispersion in relation to infarct location and size in patients with chronic myocardial infarction that had occurred at least one year previously.

**METHODS**

The QT interval on standard 12-lead resting electrocardiograms was measured in a total of 107 subjects (Table), comprising 84 patients with chronic myocardial infarction (54 with anteroseptal infarction and 30 with inferior infarction) that had occurred at least one year previously and who were not receiving oral antiarrhythmic agents, as well as 23 individuals without ischemic heart disease (13 normal subjects and 10 hypertensive patients). The QT interval was determined visually by one cardiologist using a digitizer (Picture Analyzer Σ-5/E, Nishimoto Sangyo, Japan).\textsuperscript{7} The QT interval of three heart beats in each of the 12 leads was measured and the mean values were used to calculate the minimum corrected QT (QTc) interval, maximum QTc interval, and QTc dispersion (maximum QTc-minimum QTc) by Bazett's equation (QTc=QT/√R-R[sec]).\textsuperscript{13} QTc dispersion was compared among the control group and the patients with anteroseptal infarction or inferior infarction. Patients with anteroseptal infarction were classified into two subgroups based on infarct size and QTc dispersion was compared between these subgroups as well as between subgroups with or without ventricular aneurysm. Patients with anterior infarction showing abnormal Q waves in up to three leads were classified into the small infarct group (\(n=29\)) and those with abnormal Q waves in four leads or more plus those with ventricular aneurysm were classified into the large infarct group (\(n=25\)). The results are presented as the mean ± SD. Statistical analysis was performed using analysis of variance, Fisher's protected least significant difference test, and the Mann-Whitney U test, as appropriate. A \(P\) value <0.05 was considered significant.

<table>
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<th>Table. Clinical Characteristics of the Subjects</th>
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<tr>
<td><strong>Anterior infarction group</strong></td>
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<tr>
<td>Number of patients</td>
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<td>Age (yr, mean ± SD)</td>
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<td>Gender (male/female)</td>
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<td>Post-infarction period (yr, mean±SD)</td>
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<td>Use of β-blockers</td>
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RESULTS

There was no significant difference in the minimum QTc interval among the three groups. The maximum QTc interval was 445±29 msec in the anterior infarction group, 439±38 msec in the inferior infarction group, and 424±37 msec in the control group, with the difference between the anterior infarction and control groups being significant (Figure 1). QTc dispersion was 69.9±21.5 msec in the anterior infarction group, 62.2±26.9 msec in the inferior infarction group, and 53.0±17.6 msec in the control group, with the difference between the anterior infarction and control groups again being significant (Figure 2). The size of the

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**Figure 1.** Corrected QT intervals.

**Figure 2.** Corrected QT dispersions.
anterior infarction and the presence or absence of a ventricular aneurysm had no significant influence on the minimum QTc interval (Figure 3). The maximum QTc interval was 455.3±31.0 msec in the large anterior infarct group and 436.9±25.2 msec in the small anterior infarct group, showing a significant difference. QTc dispersion was 80.5±20.5 msec in the large infarct group and 61.9±18.8 msec in the small infarct group, with the difference again being significant (Figure 4).

Figure 3. Corrected QT interval in the large and small anterior myocardial infarction groups.

Figure 4. Corrected QT dispersion in the large and small anterior myocardial infarction groups.
The QTc dispersion of the anterior infarction group with β-blocker therapy was 75.6±21.1 msec, and without β-blocker therapy was 67.1±21.6 msec. The QTc dispersions of the inferior infarction group and control group with and without β-blocker therapy were 59.8±23.2 msec, 65.7±32.4 msec, 55.1±14.4 msec, and 52.5±18.7 msec, respectively. There were no significant differences between with and without β-blocker therapy in any group.

**DISCUSSION**

In patients with myocardial infarction, QT dispersion is of special interest as a predictor of fatal arrhythmias, of wall motion abnormalities, and of the prognosis. Above all, an association between QT dispersion and fatal arrhythmias has frequently been reported. QT dispersion is increased in patients with ventricular tachycardia or those who develop ventricular tachycardia and ventricular fibrillation during the acute phase of myocardial infarction, and it has been suggested that increased QT dispersion is the substrate for ventricular arrhythmias. In a study investigating the association between reentrant ventricular tachyarrhythmias and QT dispersion after myocardial infarction, Perkiomaki, et al found that the maximum QT interval was significantly longer in patients with ventricular tachycardia than in patients without arrhythmia or normal subjects, and that QT dispersion was an independent predictor of prognosis for the patient group according to multivariate analysis. Zareba, et al reviewed the outcome of patients with coronary artery disease, and concluded that a prolonged QRS interval and increased QT dispersion were risk factors for arrhythmia leading to sudden death.

Mirvis, et al first reported the relationship between the site of myocardial infarction and the QT interval. They found that the maximum QT interval was significantly prolonged in patients who had acute anterior or inferior infarction when compared with normal subjects, although no significant difference was observed between the two groups of patients. They also reported that the leads with the maximum QT interval differed between the patients and the normal subjects. Raev, et al investigated the relationship between QT dispersion and infarct size at four days after onset, and noted that QT dispersion was smaller and wall motion abnormalities were milder in patients with inferior infarction than in those with anterior infarction. In a study assessing the relationship between coronary lesions and QT dispersion at nine days after infarction, Moreno, et al found that QT dispersion was significantly larger in patients with lesions of the left anterior descending coronary artery than in those with lesions of the left circumflex coronary artery or right coronary artery. They also found that a reduction in infarct size after reperfusion caused a decrease in QT dispersion, indicating
that dispersion was dependent on both the location and size of the infarct. Since there was no significant difference between the anterior infarction and inferior infarction groups, the effect of infarct location on QTc dispersion was unsatisfactory in the present study.

Since QT dispersion varies greatly in the acute phase of myocardial infarction, Glancy, et al have pointed out that the time of measurement should be taken into consideration when interpreting QT dispersion data. Gabrielli, et al reported that QT dispersion was increased soon after infarction and then decreased again after ten days, although not all patients showed improvement. Schneider, et al studied patients in the chronic phase of infarction and found that QT dispersion was decreased in patients who showed improvement in wall motion after four months, while it showed little change in those without improvement in wall motion, suggesting that QT dispersion could be used as an index of cardiac function in chronic myocardial infarction.

Since the above studies were performed with an emphasis on the acute phase of myocardial infarction, the present study was aimed at clarifying the relationship between infarct location and size and QT dispersion in patients with chronic infarction. We found that QT dispersion in the chronic phase was also larger in patients with anterior infarction, as was the case in the acute phase. This suggests that QT dispersion should be assessed while taking into consideration the location of the infarct as well as the time of measurement, as pointed out by Glancy, et al. The present study also showed that QT dispersion increased as the infarct became larger. It is not clear whether a larger infarct can directly cause an increase in QT dispersion or is associated with wall motion abnormalities that lead to a secondary increase in QT dispersion, but QT dispersion was suggested to be increased in patients with extensive myocardial infarction. This is in agreement with the report of Stajer, et al who suggested that the presence of ventricular aneurysm and other extensive wall motion abnormalities might have a marked influence on QT dispersion in the acute phase. Thus, QT dispersion after myocardial infarction is influenced by many factors, including drug therapy, time after infarction, the infarct location and size, and the presence/absence of ventricular aneurysm, suggesting that interpretation of QT dispersion data should involve the consideration of these factors.

**Limitations:** The subjects in the present study were outpatients who remained on their usual treatment. Thus, the patients were treated orally with various drugs, including β-blockers, although those receiving antiarrhythmic agents were excluded. As a result, differences may have been minimized between the anterior and inferior infarction groups as well as between the inferior infarction and control groups. The inclusion of hypertensive patients in the control group may have also been responsible for reducing the differences between the groups. In addi-
tion, the present study only provided one point data in each patient with chronic myocardial infarction. Accordingly, longitudinal studies are needed to establish the changes in QTc dispersion over time.

REFERENCES