



QT Dispersion: Does It Change after Percutaneous Coronary Intervention?

Mohammad Alasti, MD^{1*}, Mohammad Hassan Adel, MD¹, Ekhlas Torfi, MD¹, Mohammad Noorizadeh, MD¹, Sara Bahadoram, Mahsa Asadi Moghaddam, Mohammad Bahadoram, Bita Omidvar, MD², Mohammad Hossein Jadbabaei, MD²

¹Imam Khomeini Hospital, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.

²Golestan Hospital, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.

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Abstract

Background: Myocardial ischemia is one of several causes of prolonged QT dispersion. The aim of this study was to evaluate the effect that percutaneous coronary intervention has on the depolarization and repolarization parameters of surface electrocardiography in patients with chronic stable angina.

Methods: We assessed the effects of full revascularization in patients with chronic stable angina and single-vessel disease who underwent percutaneous coronary intervention. Twelve-lead electrocardiograms were recorded before intervention and 24 hours subsequently. We measured parameters including QRS duration, QT and corrected QT durations, and JT and corrected JT duration in both electrocardiograms and compared the values.

Results: There were significant differences between the mean QRS interval (0.086 ± 0.01 sec vs. 0.082 ± 0.01 second; p value = 0.01), mean corrected QT dispersion (0.080 ± 0.04 sec vs. 0.068 ± 0.04 sec; p value = 0.001), and mean corrected JT dispersion (0.074 ± 0.04 sec vs. 0.063 ± 0.04 sec; p value = 0.001) before and after percutaneous coronary intervention. No significant differences were found between the other ECG parameters.

Conclusion: Our data indicate that the shortening of corrected QT dispersion and corrected JT dispersion in patients undergoing percutaneous coronary intervention is prominent.

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Introduction

The inter-lead variation of QT interval provides an index of the heterogeneity of ventricular refractoriness. The most commonly used index to calculate QT dispersion has been the difference between the longest and shortest QT intervals on the twelve-lead electrocardiography (ECG), which is

often adjusted for heart rate. Abnormally prolonged QT dispersion has been correlated with the risk of arrhythmic death in some disorders, including coronary artery disease, although the results are not consistent.¹

Ischemia can increase QT dispersion.² Percutaneous coronary intervention (PCI) is widely used to manage ischemia in patients with coronary artery disease. However,

*Corresponding Author: Mohammad Alasti, Assistant Professor of Cardiology, Jondishpour University of Medical Sciences, Imam Khomeini Hospital, Azadegan, Avenue, Ahvaz, Iran. Tel: +98 611 4457205. Fax: +98 611 4457205. E-mail: alastip@gmail.com.

there is a dearth of information on the influence of elective PCI on ECG parameters, especially QT parameters.³

The aim of the study was to evaluate the effect that percutaneous coronary intervention may have on such ECG parameters as QRS duration, QT interval, corrected QT interval, JT interval, corrected JT interval, QT dispersion, JT dispersion, corrected QT interval, and corrected JT interval.

Methods

The patients included were selected from those admitted for elective PCI on the basis of the following inclusion criteria: (1) angiographic evidence of significant stenosis ($\geq 70\%$) in only one main coronary artery, (2) chronic stable angina pectoris, and (3) successful angiographic PCI.

The exclusion criteria were: (1) acute coronary syndrome, (2) electrolyte disturbances, (3) ventricular pacing, (4) QRS duration > 0.12 seconds, (5) not being in sinus rhythm, (6) need for emergent coronary artery bypass grafting (CABG) or repeat PCI during a 24-hour period after the procedure, (7) if QT interval could not be reliably measured in at least nine leads, (8) taking anti-arrhythmic, anti-psychotic, and anti-depressant drugs, which might affect QT interval and QT dispersion, and (9) twofold increase in cardiac enzymes or sustained monomorphic ventricular tachycardia or ventricular fibrillation during a 24-hour period after PCI.

Atherosclerosis risk factors were defined as follows: Hypertension was defined as taking antihypertensive drugs or baseline blood pressure $\geq 140/90$ mmHg. Diabetes mellitus was defined as taking hypoglycemic agents or a fasting plasma glucose level ≥ 126 mg/dl, a two-hour value in an oral glucose tolerance test ≥ 200 mg/dl, or a random plasma glucose concentration ≥ 200 mg/dl in the presence of symptoms. Hyperlipidemia was defined as a total cholesterol level ≥ 220 mg/dl or a triglyceride level ≥ 150 mg/dl. Active smoking was considered a risk factor.

The patients were imaged in the left lateral decubitus position in the parasternal and apical views using a commercially available system (Vingmed Three, General Electric-Vingmed, Milwaukee, WI, USA) during the first day of admission.

All the coronary angioplasties were performed at Imam Khomeini General Hospital. PCI was performed according to standard techniques.

Blood samples were obtained immediately after admission and 24 hours after PCI. Serum creatinine, sodium, potassium, calcium, and cardiac enzymes including troponin and CK concentrations were measured.

Standard twelve-lead ECGs were recorded before PCI and 24 hours after that at 7:30 am with an electrocardiograph simultaneously acquiring six standard leads at a paper speed of 25 mm/s and a gain of 10 mm/mV. QT interval analysis was performed after magnification by two observers, who

were not given the clinical data, and the mean of the two measurements was used. QT interval was measured from the beginning of QRS complex to the end of T wave using a ruler. The end of T wave was defined as the point of return to the isoelectric line. When a T wave was interrupted by a U wave, the end of the T wave was defined as the nadir between the T and the U waves. If the end of the T wave could not be reliably determined due to extremely low voltage (< 0.1 mV), measurements of QT interval were not made and these leads were excluded from the analysis. In order to exclude the effect of heart rates on QT intervals, QT intervals were corrected according to the Bazett formula: $QTc = QT / \text{square root of RR interval}$. QT dispersion was defined as the difference between the maximum and minimum QT intervals.

QT dispersion was corrected according to the Bazett formula. QRS interval was measured from the beginning of QRS complex to its end. JT interval was calculated in each lead as the difference between QT and QRS interval. Corrected JT dispersion was defined as the difference between maximum and minimum JT intervals corrected according to the heart rate: $JTc = JT / \text{square root of RR interval}$. Each measurement was performed in three successive beats because averaging QT interval reduces the bias.⁴

The study protocol was approved by the Ethics Committee of Jundishapur University of Medical Sciences. All the patients provided written informed consent.

The continuous data were expressed as mean \pm standard deviation. Differences in the mean values before and after the procedure were compared using the paired T-test. A p value < 0.05 was considered statistically significant.

Results

The study population consisted of 96 patients: 67 (69.8%) men and 29 (30.2%) women. The mean age was 53.2 ± 9.5 (range: 33-76) years. The baseline characteristics of the study population are summarized in Table 1.

Table 1. Patients' baseline characteristics (n=96)*

Age (y)	53.24 \pm 9.57
Male	67 (69.8)
Hypertension	52 (55.2)
Diabetes mellitus	25 (26)
Dyslipidemia	43 (45)
Smoking	4 (4.2)
History of MI	9 (9.4)
LVEF (%)	49.94 \pm 4.26

*Data are presented as mean \pm SD or n (%)

MI, Myocardial infarction; LVEF, Left ventricular ejection fraction

The patients were on aspirin (n = 96, 100%), beta blockers (n = 51, 73%), angiotensin-converting enzyme inhibitors (n = 16, 17%), and lipid-lowering medications (n = 31, 32%).



Interobserver variability was evaluated in all the ECG measurements. The mean of the absolute difference between the first and second measurements of QT dispersion was 0.06 ± 0.02 second (range: 0.01-0.10 seconds) with a correlation coefficient of 0.93.

The ECG parameters before and after PCI are presented in Table 2. There were significant differences between the mean QRS interval, mean QT_c dispersion, and mean JT_c dispersion before and after PCI. No significant differences were found between the other ECG parameters.

The analysis of the patients was conducted according to their genders. The male group consisted of 67 patients. There were significant differences between pre- and post-PCI JT_c dispersion (74.46 vs. 65.78, p value = 0.001) and QT_c dispersion (78.25 vs. 69.04, p value = 0.001). The female group consisted of 29 patients. There were significant differences between pre- and post-PCI JT_c dispersion (73.85 vs. 59.78, p value = 0.031) and QT_c dispersion (83.98 vs. 66.32, p value = 0.038). There was no significant difference between the other ECG parameters.

The patients were also analyzed with respect to their involved arteries. The left anterior descending artery (LAD) group comprised 62 patients; there were significant differences between pre- and post-PCI JT_c dispersion (79.29 vs. 67.95, p value = 0.001) and QT_c dispersion (84.79 vs. 70.79, p value = 0.001). The left circumflex artery (LCX) group was comprised of 20 patients, and there were significant differences between pre- and post-PCI JT_c dispersion (74.93 vs. 67.04, p value = 0.01) and QT_c dispersion (76.94 vs. 74.16, p value = 0.041). The right coronary artery (RCA) group consisted of 14 patients, there being significant differences between pre- and post-PCI JT_c dispersion (95.76 vs. 85.63, p value = 0.001) and QT_c dispersion (106.09 vs. 89.22, p value = 0.001). The differences between the other parameters were not significant.

Discussion

QT dispersion is an index of the heterogeneity of ventricular refractoriness and is sensitive to age, time of day, season of the year, and even body position.¹ It has a highly specific upper normal limit of 50 ms and is longer in patients with a previous myocardial infarction than in normal subjects.⁵

Although many QT experts recommend correcting QT and JT interval according to the heart rate, Vassilikos et al. demonstrated that the heart rate did not influence QT dispersion and correction for rates faster than 100 beat/min could result in abnormally prolonged values.⁶ Theoretically, an accurate measurement of QT and JT dispersion requires all the twelve leads of the ECG to be recorded simultaneously. Therefore, simultaneous twelve-lead recordings have been proposed as a gold standard for a QT dispersion measurement, but simultaneous six-lead recordings can also be acceptable.⁷

The values of QT interval depend on the shape of the descending part of T wave. Q wave dispersion can also influence QT dispersion, although its effect is significantly smaller than T wave offset. Some experts believe that JT dispersion reflects better the dispersion of action potential duration and, consequently, suggest QT and JT dispersions be used as separate entities.⁷

Increased QT dispersion reflects inhomogeneous ventricular repolarization, which may provide a background for significant ventricular arrhythmias.⁸ Prolonged QT dispersion is associated with a higher risk of malignant ventricular arrhythmias in patients with the long QT syndrome, hypertrophic cardiomyopathy, and myocardial infarction.¹ Effective management of acute myocardial infarction or ventricular arrhythmias may reduce QT dispersion; i.e. successful reperfusion after thrombolysis as well as revascularization with angioplasty and CABG grafting, especially concomitant with aneurysmectomy.⁸⁻¹⁰

The effect of myocardial ischemia on QT dispersion has been described in different clinical circumstances.

Table 2. The electrocardiographic parameters before and 24 hours after percutaneous coronary intervention*

Variable	Before PCI	After PCI	P value
Cycle length (sec)	0.96±0.17	0.94±0.18	0.269
Mean QRS interval (sec)	0.08±0.01	0.08±0.01	0.010
Mean Max QT interval (sec)	0.43±0.04	0.45±0.04	0.274
Mean Min QT interval (sec)	0.36±0.04	0.36±0.04	0.543
Mean Max QT _c interval (sec)	0.44±0.03	0.43±0.04	0.321
Mean Min QT _c interval (sec)	0.38±0.04	0.35±0.04	0.123
Mean Max JT interval (sec)	0.35±0.04	0.36±0.04	0.134
Mean Min JT interval (sec)	0.27±0.04	0.28±0.04	0.012
Mean Max JT _c interval (sec)	0.35±0.04	0.36±0.04	0.322
Mean Min JT _c interval (sec)	0.27±0.04	0.28±0.04	0.345
Mean QT Dispersion (sec)	0.07±0.04	0.07±0.04	0.516
Mean QT _c Dispersion (sec)	0.08±0.04	0.06±0.04	0.001
Mean JT Dispersion (sec)	0.07±0.04	0.07±0.04	0.312
Mean JT _c Dispersion (sec)	0.07±0.04	0.06±0.04	0.001

*Data are presented as mean±SD

PCI, Percutaneous coronary intervention; Max, Maximum; Min, Minimum; QT_c, Corrected QT interval; JT_c, Corrected JT interval

Transient ischemia can prolong QT dispersion. A significant increase in QT dispersion during acute ischemia caused by balloon inflation has been demonstrated; it is, however, reversible and decreases on reperfusion.¹¹⁻¹² Michelucci et al. showed that both ischemia and reperfusion could influence ventricular repolarization and thus lead to a less homogeneous ventricular recovery pattern.¹³

A significant decrease in QT dispersion may help us as an electrocardiographic index for successful reperfusion.¹⁴ In one study, a comparison of QT dispersion after primary PCI with QT dispersion after thrombolysis showed a significant decrease in the first group;¹⁵ this difference may be related to more salvaged myocardium in the primary PCI group because QT dispersion is mainly determined by the amount of viable myocardium in the infarct region. Hence, QT dispersion may be utilized as a marker of viability in patients with chronic Q-wave myocardial infarction.¹⁶ Patients with chronic stable angina may have abnormal QT dispersion due to ongoing ischemia, previous myocardial damage, or both.

There are some studies about the effects of revascularization on QT dispersion in patients with chronic ischemia, but the results are controversial. Choi et al.¹⁷ demonstrated that QT dispersion decreased in coronary artery disease patients with no history of myocardial infarction at one month following a successful PCI. Another study performed by Aydinlar et al.³ revealed a reduction in QT dispersion immediately after percutaneous transluminal coronary angioplasty. According to our data, a successful full revascularization of patients with chronic stable angina is associated with a significant reduction in QTc and JTc dispersion and even QRS duration (not QT and JT dispersion) 24 hours after the procedure. Our data suggest that chronic ischemia alters ventricular repolarization in patients with chronic stable angina and prolongs QT and JT dispersion. These findings support the hypothesis that we can detect ischemia-induced inhomogeneity in ventricular refractoriness by a careful examination of QTc or JTc dispersion derived from the surface ECG.

The high values of QT dispersion in our patients show that they were a high-risk group and almost all of them had considerable ischemia. Hence, revascularization ameliorated this ischemia and led to a shortening of QTc and JTc dispersion.

In our patients, there was no relationship between the ECG parameters and the involved vessel, which is in contrast with the results of a previous study performed by Kilic et al.² It may also be related to our high-risk patient selection and the presence of considerable ischemia in the involved vessel territory. The QTc and JTc dispersion values were higher in the RCA group (a small group with only 14 patients). Furthermore, there was no significant difference between the baseline characteristics of these groups, and unfortunately we have no idea about the real reason. Therefore, we would

recommend that future studies be designed with larger sample sizes in each group so as for this issue to be investigated more comprehensively.

Our study has some important limitations. The major limitation of the present study may be represented by the small number of the patients. As a result, designing a larger study with more cases could be more informative. The other limitation is the relatively high interobserver differences in the measurement, which may have affected our results and reduced their reliability. Another limitation is that patients with unsuccessful PCI were not evaluated, which precluded an evaluation of the probable effects of the contrast media on the ECG parameters.

Conclusion

In conclusion, our data indicate that the shortening of QTc dispersion and JTc dispersion in patients undergoing PCI was prominent and coronary revascularization may prevent the frequency of repolarization abnormalities.

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