Evaluation of QT, QT Dispersion, and T-Wave Peak to End Time Changes After Primary Percutaneous Coronary Intervention in Patients Presenting With Acute ST-Elevation Myocardial Infarction

Vahid Eslami, MD1, Morteza Safi, MD1, Maryam Taherkhani, MD1, Ali Adibi, MD1, Mohammad Reza Movahed, MD, PhD2,3

ABSTRACT: Background. Acute ST-elevation myocardial infarction (STEMI) is associated with significant arrhythmia and cardiac arrest. QT prolongation can occur in the setting of ischemia or acute STEMI as a risk factor for arrhythmia. The goal of this study was to investigate corrected QT interval (QTc), QT dispersion (QTd), and T-wave peak to end (TPE) times in this patient population and evaluate the effect of primary percutaneous coronary intervention (PCI) in STEMI patients on these indices. Methods. This study was a clinical trial, whereby eligible patients presenting with acute STEMI who were appropriate candidates for primary PCI were enrolled. QTc, QTd, and TPE indices were calculated before and after the procedure. Results. Eighty patients (60 male, 20 female) with a mean age of 58.8 years were evaluated. We found significant reduction in QTd after PCI (mean, 5.8 ms before PCI vs 3.6 ms after PCI; P=0.001) and significant reduction in TPE after PCI (mean, 9.7 ms before PCI vs 7.8 ms after PCI; P=0.001). QTc did not show significant changes before or after PCI (44.9 vs 43.7; P=0.057). Conclusion. Our study showed that primary PCI was effective in reducing the degree of arrhythogenic indices such as QTd and TPE. Our findings suggest that ischemia-induced QTd and TPE are important arrhythogenic parameters responding to successful primary PCI and may be used as markers for successful reperfusion.


Key words: coronary artery disease, QT, QT prolongation, QT dispersion, arrhythmia, primary PCI, TPE percutaneous coronary intervention, PTCA, stenting, acute myocardial infarction, acute coronary syndrome

The QT interval reflects the duration of ventricular electrical activity determined by the phases of depolarization and repolarization.1 It was proposed that the different electrocardiographic (ECG) leads magnify the ECG signal of different myocardial regions; consequently, QT dispersion (QTd), the maximum variation in the QT interval in 12-lead ECG, reflects inhomogeneity of ventricular repolarization and spatial dispersion of ventricular recovery time.2 Clinical and experimental studies have shown a clear relationship between dispersion and other repolarization indices,3 suggesting that QTd can show spatial projection in various ECG leads.3

The significance of QTd partially persists even with the use of 6 precordial leads instead of 12 leads, as well as with other lead combinations.2 QTd has been shown to correlate with increased arrhythmic vulnerability in various types of cardiac diseases, such as coronary artery disease,6 long QT syndrome,7 and congestive heart failure.8 It is also considered a predictor of ischemic cardiac events and sudden cardiac death.9,10 In addition, corrected QTd before percutaneous coronary intervention (PCI) has been associated with an increased risk of major adverse cardiac event (MACE) and mortality after successful PCI in acute ST-elevation myocardial infarction (STEMI).

Some researchers have shown an increase of QTd in STEMI patients.11 Interestingly, QTd variability has been reported to be associated with myocardial viability in the setting of acute STEMI12 and successful thrombolytic therapy has resulted in the reduction of QTd.13 A T-wave on surface ECG is a representative of voltage gradient between subendocardial and subepicardial region. In addition to QTd, some studies used T-wave peak to end (TPE) to evaluate repolarization inhomogeneity, where the peak of the T-wave coincides with the end of epicardial repolarization while the end of the T-wave indicates the end of repolarization of the whole ventricular myocardium.14,15 The goal of this study was to investigate corrected QT interval (QTc), QTd, and TPE in this patient population and evaluate the effect of primary percutaneous coronary intervention (PCI) in STEMI patients on these indices.

Methods

The patients enrolled were initially selected from those with clinical history and symptoms suggestive of a first episode of acute STEMI who had presented within 12 hours after the onset of symptoms. In all cases, acute STEMI was documented based on ECG and later confirmed by invasive coronary angiography. Ultimately, only patients who underwent successful PCI with TIMI flow grade 3 post PCI with a door-to-balloon time of <90 minutes were included.

Patients with history of previous myocardial infarction, atrial fibrillation, left bundle branch block, QRS >12 ms, or difficult to determine end of T-wave were excluded from the study. Informed consent was obtained from each patient involved in this study. The study was approved by institutional review committee.
 QTd has an upper normal limit of 50 ms and is longer in patients with previous myocardial infarction than in normal subjects. Some studies have shown that even transient balloon inflation during angioplasty has an impact on action potential and repolarization duration.

Analysis of QT interval. All standard 12-lead ECGs were recorded at 25 mm/s speed and 10 mm/mv gain. The QT data obtained at admission and 24 hours after primary PCI were manually measured with a ruler. QT interval was measured from the beginning of QRS to the end of the T-wave. The end of the T-wave was defined as the nadir between the T- and U-waves. In instances where the T-wave could not be reliably determined due to extremely low voltage (<1 mv), measurement of QT interval was not established and consequently these leads were excluded from analysis. In order to exclude the effects of heart rate (HR) on the QT interval, the QT interval was corrected according to the Bazett formula (QTc = QT/square root of RR interval). QTd was defined as the difference between the maximum and minimum QT intervals. TPE was measured with a ruler from the peak of the T-wave to its end. The criteria to determine the endpoint of the T-wave were similar to the aforementioned criteria considered for QT measurement. Statistical analysis of data was performed with SPSS 16 software. Differences in mean values before and after PCI were compared using paired T-tests and P-values <.05 were considered statistically significant.

Results

The study population consisted of 80 patients (60 male, 20 female) diagnosed with acute STEMI who underwent successful primary PCI. Baseline characteristics of our patients are summarized in Table 1. After coronary angiography, the left anterior descending (LAD) coronary artery was found to be the most common culprit vessel (42 patients; 60%), whereas the left circumflex (LCX) coronary artery and right coronary artery (RCA) were equally affected (18 patients each; 20%). Coronary artery stenosis distribution is summarized in Table 1. After PCI, ejection fraction (EF) was >50% in 12 patients, between 35%-50% in 38 patients, and <35% in 30 patients. We found significant reduction in QTd after PCI (mean, 5.8 ms before PCI vs 3.6 ms after PCI; P<.001) and significant reduction in TPE after PCI (mean, 9.7 ms before PCI vs 7 ms after PCI; P<.001). QTc did not show any significant changes before or after PCI (44.9 ms vs 43.7 ms, respectively; P=.057). Table 2 shows the mean ± standard deviation (SD) of these indices. There was no significant correlation between QTd and TPE before PCI (r=-0.1) or after PCI (r=-.2). Among our total population of 80 patients, fifty-two patients (65%) demonstrated a decrease in QTd post PCI, while 2 cases (2.5%) showed an increase with no difference observed in the 26 remaining patients (32.5%).

Discussion

QTd as Markers of Successful Reperfusion

Table 1. Coronary artery stenosis distribution.

<table>
<thead>
<tr>
<th>Vessel Type</th>
<th>MI Culprit</th>
<th>&lt;50% Stenosis</th>
<th>50%-70% Stenosis</th>
<th>70%-99% Stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>LCX</td>
<td>16 (20%)</td>
<td>34 (42.5%)</td>
<td>14 (17.5%)</td>
<td>16 (20%)</td>
</tr>
<tr>
<td>RCA</td>
<td>16 (20%)</td>
<td>18 (22.5%)</td>
<td>18 (22.5%)</td>
<td>26 (32.5%)</td>
</tr>
<tr>
<td>LAD</td>
<td>48 (60%)</td>
<td>18 (22.5%)</td>
<td>6 (7.5%)</td>
<td>8 (10%)</td>
</tr>
</tbody>
</table>

Table 2. Electrocardiographic parameters.

<table>
<thead>
<tr>
<th>Electrocardiographic Parameter</th>
<th>Mean ± SD</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QTc (before MI)</td>
<td>44.9 ± 4.2</td>
<td>.057</td>
</tr>
<tr>
<td>QTc (after MI)</td>
<td>43.7 ± 3.2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>QT dispersion (before)</td>
<td>5.8 ± 2.3</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>QT dispersion (after)</td>
<td>3.6 ± 1.5</td>
<td>.057</td>
</tr>
<tr>
<td>T-wave peak to end (before)</td>
<td>9.7 ± 2.7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>T-wave peak to end (after)</td>
<td>7 ± 2.6</td>
<td>.057</td>
</tr>
</tbody>
</table>

Figure 1. Baseline characteristics of the patients. HTN = hypertension; DM = type 2 diabetes mellitus; HLP = hyperlipidemia; SM = smoking; FH = family history of coronary artery disease.
changes can also occur in the setting of elective PCI, including successful PCI of chronic total occlusions suggesting that chronic ischemia also affects QTd.\textsuperscript{27,28} QTd measured before PCI also has been found to have direct prognostic value on outcome.\textsuperscript{29} Even very late successful PCI in the setting of myocardial infarction has shown to improve QTd.\textsuperscript{30} Our study did not show any significant differences in regard to QTc before or after PCI. The reason for this finding is most likely related to the timing of ECG obtained after the reperfusion. It is known that QT is prolonged during ischemia.\textsuperscript{31} However, the timing of normalization of QT after reperfusion is not immediately and usually occurs 5 days post reperfusion.\textsuperscript{31} The reason for this time delay is not known, but it explains why our study did not show any difference in the QTc before or after PCI since our ECGs were done in the first 24 hours after successful PCI and not after 5 days. Based on our findings, QTd and TPE appear to be markers of successful reperfusion. However, our study is limited by the small number of patients enrolled and larger trials are needed to correlate our findings with long-term outcomes.

Conclusion

Our study showed that primary PCI was effective in reducing the degree of arrhythmogenic indices such as QTd and TPE. Our findings suggest that ischemia-induced QTd and TPE are important arrhythmogenic parameters responding to successful primary PCI and may be used as markers for successful reperfusion.

References