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Adjacent QT dispersion: A good predictor of ventricular arrhythmias after myocardial infarction

Research Article

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Abstract: The 12-lead surface electrocardiogram adjacent QTc dispersion, which is the maximum difference of corrected QT interval between two adjacent leads, is a simple method to determine regional variation in repolarization and refractoriness. The aim of this study is to evaluate adjacent QTc dispersion as a marker of susceptibility to ventricular arrhythmias after myocardial infarction. A total of 135 consecutive patients with acute myocardial infarction were enrolled in the study. Adjacent QTc, measured by lens magnifier, was calculated on the first, second and third days after acute myocardial infarction. On the second day after acute myocardial infarction, adjacent QTc dispersion was significantly greater in patients with ventricular arrhythmias ($P < 0.001$). Adjacent QTc dispersion on the first and fifth day after acute myocardial infarction was not associated with development of ventricular arrhythmias. On the second day after acute myocardial infarction, adjacent QTc dispersion is a simple and feasible method for prediction of ventricular arrhythmias.

Keywords: *Myocardial Infarction • QT Dispersion • Ventricular Arrhythmia*

1. Introduction

Acute myocardial infarction is one of the most common cause of mortality in middle and older age populations [1,2]. Malignant ventricular arrhythmias are the most common cause of mortality in the pre-hospital and early-hospital periods of these patients [1,2]. QTc dispersion, which is the difference between the maximum and minimum of corrected QT interval in different leads, may be a marker of repolarization inhomogenecity in the cardiac electrical system. Adjacent QTc dispersion, which is the maximum difference of QTc interval between two adjacent leads of a standard electrocardiogram, is a simple method to determine regional variation in repolarization and refractoriness. Regional electrical inhomogenecity is the cornerstone for development of reentrant ventricular tachyarrhythmia [3]. The aim of this study is to evaluate adjacent QTc dispersion as a marker of susceptibility to ventricular arrhythmias after myocardial infarction.

2. Material and Methods

2.1. Patients

Between September 2004 and April 2005, 135 consecutive patients who were admitted in CCU because of acute myocardial infarction (AMI) were enrolled in the study.

2.2. Definition of AMI

Myocardial infarction was defined by the presence of ECG changes associated with typical chest pain and elevated cardiac enzymes. These enzymes were measured at 6- to 8-hour intervals during the first 24 hours. The cardiac enzymes were considered

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Table 1. Age, sex and different medications received by two study groups.

	Patients with ventricular arrhythmias	Patients without ventricular arrhythmias	P value
Age	59.3 ±10.7	58.9±12.8	NS*
Male sex	37(54.4%)	37 (55.2%)	NS*
b-blocker	62 *91.2%)	63 (94%)	NS*
Ca-blocker	5 (7.4%)	10 (14.9%)	NS*
ACE inhibitor	32 (47.1%)	27 (40.3%)	NS*
Nitrate	67 (98.5%)	65 (97%)	NS*
ASA	68 (100%)	67 (100%)	NS*
Streptokinase	25 (36.8%)	19 (28.4%)	NS*

** Non-significant P value (>0.05).*

elevated if Troponin I or both creatine kinase (CK) and its MB isoenzyme (CK-MB) were greater than two times the upper normal limit.

2.3. Definition of Ventricular Arrhythmia

Ventricular arrhythmia was defined as one of the following: 1) ventricular tachycardia; 2) ventricular fibrillation; 3) malignant premature ventricular complexes (PVCs) (Bigemini, couplet, Salvos, multiform PVCs, and PVCs with R on T); or 4) more than 5 PVCs/min.

2.4. Detection of Ventricular Arrhythmia

Siemen's CCU monitoring system had the capability to record any abnormal rhythm. Therefore, we checked the system memory at the end of the third day of CCU admission. All abnormal recorded beats were evaluated by two cardiologists.

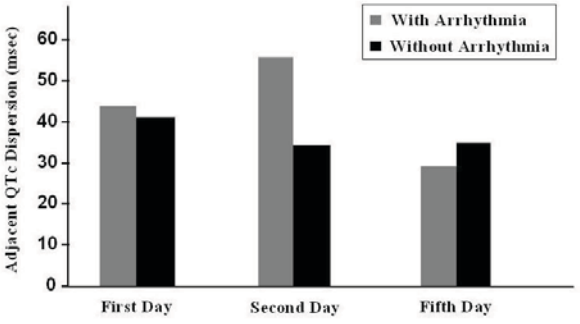
2.5. Measurement of adjacent QTc dispersion

QT intervals were measured in all leads on the first, second and third days after AMI. Lens magnifier (10, millisecond units) was used to calculate QT intervals. Measured QT intervals were corrected by Bazett's formula (QTc = QT interval (sec) / √RR (sec)).

2.6. Exclusion Criteria

Patients were excluded from the study for any of the following reasons: 1) electrolyte abnormalities; 2) renal failure; 3) bundle branch block, 4) AV blocks; 5) pre-excitation syndrome; 6) patient receiving digoxin or antiarrhythmic drugs except Lidocaine, beta-blockers and Ca-blockers; or 7) when T waves were flat or the end of T waves were indefinite.

Figure 1. Shows the distribution of adjacent QTc on the first, second and fifth days after acute myocardial infarction.



2.7. Statistical analysis

Continuous variables are presented as means ± 1 standard deviation. Categorical variables are displayed as percentages (%). Differences between groups were evaluated with t-tests for continuous variables or chi-square analyses for categorical variables, as appropriate. To determine cutoff values, analysis of receiver-operating characteristic (ROC) curve was made.

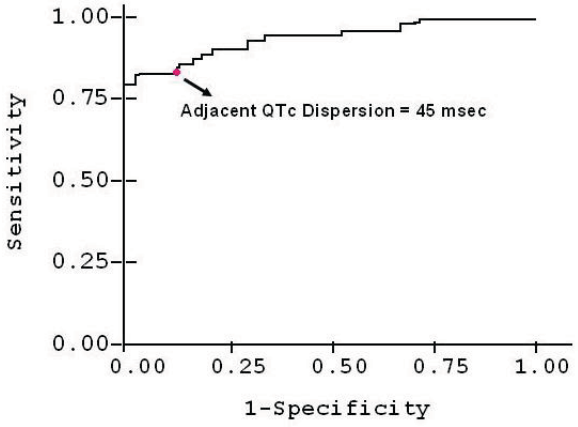
3. Results

During the screening period, 135 consecutive patients were enrolled in the study. Clinical data are listed in Table 1. Age, sex and medications were not significantly different between the two study groups (Table 1). Adjacent QTc values were not significantly different on the first and fifth day after AMI in patients who developed ventricular arrhythmias compared to those without ventricular arrhythmias (P= 0.28 and 0.30 respectively). Adjacent QTc on the second day after AMI was significantly greater in patients who developed ventricular arrhythmias compared to those without ventricular arrhythmias (56 ± 12 msec vs. 32 ± 14 msec; P < 0.001) (Table 2). Figure 1 shows the distribution of adjacent QTc on the first, second and fifth days after AMI. The area under the ROC curves for adjacent QTc dispersion for the prediction of ventricular arrhythmias on the second day after AMI was 0.85 (cut point ≥ 45 msec; Sensitivity = 80%, Specificity = 87.5%) (Figure 2). These data indicate that adjacent QTc dispersion ≥ 45 msec on the second day after AMI is a good discriminator of patients likely to develop ventricular arrhythmia after AMI.

Table 2. Adjacent QTc dispersion in patients with and without ventricular arrhythmias.

	With arrhythmias (n=68)	Without arrhythmias (n=67)	P value
1st day adj. QTc (msec)	45 ± 22	41 ± 18	0.28
2nd day adj. QTC (msec)	46 ± 20	33 ± 18	<0.001
5th day adj. QTC (msec)	30 ± 19	35 ± 19	0.3

Figure 2. Shows the area under the ROC curves for adjacent QTc dispersion for the prediction of ventricular arrhythmias on the second day after acute myocardial infarction. The cut point that better optimizes the values of sensitivity and specificity are for values ł 45 msec.



4. Discussion

During acute myocardial infarction, localized ischemia, increased sympathetic activity and regional ionic alteration can result in regional variation of repolarization and subsequently reentrant circuits. This can be a basis for starting malignant ventricular arrhythmias [1]. It seems reasonable that prolonged QT interval, particularly its regional dispersion, may be associated with increased chance of ventricular arrhythmias. Many studies have shown that higher QTc dispersion is associated with increased susceptibility to ventricular arrhythmias [4-10] but fewer studies rejected such a relationship like the study done by Leitch et al. [11]. There are fewer studies on adjacent QTc dispersion. These studies are emphasized on chronic ischemic heart disease patients [12,13] and the relation between ventricular arrhythmias. Adjacent QTc dispersion in the setting of acute myocardial infarction has not been reported previously. In the present study, we evaluated the relationship between adjacent QTc and susceptibility to ventricular arrhythmias in the setting of AMI. We found that adjacent QTc dispersion on the second day after AMI is associated with development of ventricular arrhythmias. Increased adjacent QTc dispersion on the second day of admission in patients with AMI may be related to reperfusion in the adjacent area that resulted in more regional variation of refractoriness. Theoretically, adjacent QTc dispersion is really a better precursor for dispersion of refractoriness in side-by-side areas and should be a better predictor of reentrant arrhythmias than QTc dispersion.

In conclusion adjacent QTc dispersion on the second day of CCU admission is a predictor of susceptibility to early ventricular arrhythmias in myocardial infarction patients.

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