

Comparison between QT Interval Parameters in Type 2 Diabetic and Nondiabetic Patients with Non-ST Elevation Myocardial Infarction

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Received 28 July 2013; Accepted 19 July 2014

Abstract

Background: QT interval parameters have been suggested as a predictor of lethal arrhythmia and mortality in patients with myocardial infarction. The aim of the present study was to compare the value of QT interval indices in patients presenting with non-ST-segment elevation myocardial infarction (NSTEMI) between a group of patients with type 2 diabetes mellitus and a nondiabetic group of patients.

Methods: This case-control study evaluated QT interval parameters in 115 patients (47 diabetic and 68 nondiabetic patients) diagnosed with NSTEMI between September 2011 and July 2012. The following QT interval indices were analyzed: maximum (max) and minimum (min) QT interval; max and min corrected QT interval (QTc); QT dispersion (QTd); and corrected QT dispersion (QTcd). All the patients were observed for ventricular arrhythmia during their hospital course and underwent coronary angiography. They were selected to undergo coronary artery bypass surgery (CABG) or percutaneous coronary angioplasty (PCI) based on their coronary anatomy.

Results: The mean age of the patients was 60.8 ± 11.4 years. The patients were 40.0% female and 60.0% male. There were no significant differences in clinical characters between type 2 diabetic and nondiabetic patients with NSTEMI. Compared with post-myocardial infarction patients without diabetes, those with type 2 diabetes had higher QTc max, QTd and QTcd (p value < 0.05). There was a significant difference in QTd and QTcd in the patients needing coronary revascularization with diabetes as opposed to the nondiabetics (p value = 0.035 and p value = 0.025, respectively) as well as those who had ventricular arrhythmia with diabetes (p value = 0.018 and p value = 0.003, respectively). QTcd was higher in the patients who had higher in-hospital mortality (p value = 0.047). The QTc max, QTd and QTcd were significantly (all p values < 0.05) associated with ventricular arrhythmia, QTcd with need for revascularization and QTc max with in-hospital mortality in the diabetic patients.

Conclusion: Based on the findings of this study, it seems that type 2 diabetics with NSTEMI have greater QTc max, QTd, and QTcd and these QT parameters may have a relationship with worse cardiac outcomes and poorer prognoses.

J Teh Univ Heart Ctr 2014;9(4):166-173

This paper should be cited as: Bonakdar HR, Aslanpour M, Moladoust H, Sadeghipour P, Mohamadi F, Assadian Rad M, Kheirkhah J. Comparison between QT Interval Parameters in Type 2 Diabetic and Nondiabetic Patients with Non-ST Elevation Myocardial Infarction. *J Teh Univ Heart Ctr 2014;9(4):166-173.*

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Keywords: Myocardial infarction • Diabetes mellitus, type 2 • Electrocardiography

Introduction

Increased electrical inhomogeneity in myocardial infarction is considered a substrate of lethal arrhythmia.¹ The dispersion of repolarization can be measured on the surface electrocardiogram (ECG) using QT interval parameters.³ The method is simple and accessible and has shown promising results in predicting mortality in patients with myocardial ischemia.¹⁻⁴

Myocardial events (fatal and nonfatal) happen with a greater incidence in patients with diabetes mellitus (DM).⁵⁻⁸ Different methods of risk stratification have been proposed. Several studies have shown a greater QT dispersion (QTd) in DM patients and have suggested the measure as a predicting tool for cardiovascular mortality in this population.⁹⁻¹² The predictive value of QT interval parameters in the DM population has been greatly debated.⁹⁻¹² Giunti et al.¹² showed that QTd predicted 15-year cardiovascular mortality among a large type 2 DM population. Similarly, Pfister et al.¹¹ evaluated different ECG parameters in the PROactive trial population and found that bundle branch block and QT corrected (QTc) were predictive measures in patients with type 2 DM. Stettler et al.¹⁰ in their long-term follow-up study reported that QTc was correlated with mortality in type 1 DM, whereas resting heart rate was associated with mortality in type 2 DM.¹⁰

In the present study, we evaluated the value of QT interval parameters in patients presenting with non-ST-segment elevation myocardial infarction (NSTEMI) in two groups with and without DM.

Methods

The study population was recruited from patients hospitalized in Heshmat Heart Center between September 2011 and July 2012. All the patients were diagnosed to have NSTEMI by the following criteria: clinical presentation (acute onset chest pain or other chest pain equivalent); elevated cardiac biomarkers (cardiac troponin I and creatine kinase MB isoenzyme [CK-MB]); ECG changes; and imaging evidence of ischemia (echocardiography in the present study).¹³ In accordance with the American College of Cardiology/American Heart Association (ACC/AHA) 2007 guidelines on the management of NSTEMI/unstable patients regarding cardiac enzyme rise, the whole study population underwent coronary angiography. Significant coronary artery stenosis was defined as a luminal narrowing $\geq 50\%$.¹³ The patients were divided into type 2 DM and non-

DM groups, and the diagnosis of type 2 DM was based on the plasma glucose criteria (fasting blood glucose ≥ 126 mg/dl) or hemoglobin A1C $\geq 6.5\%$.¹⁴ The exclusion criteria of the present study were: 1) patients presenting with ST elevation myocardial infarction (STEMI) and unstable angina; 2) Type I DM and patients hospitalized with ketoacidosis; 3) non-sinus rhythm at the baseline rhythm; 4) history of using antiarrhythmic, antipsychotic, and antidepressant medications; 5) conduction abnormality (atrioventricular block and intraventricular conduction defect); 6) electrolyte disturbances; 7) history of chronic renal disease (CrCl < 60 mL/min/1.73 m²); and 8) history of hospitalization for acute coronary syndrome during the last 3 months (due to the possible influence of previous myocardial ischemia on repolarization). Need for revascularization was defined as any patient requiring percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery (CABG) during the hospital course. Ventricular arrhythmias were considered frequent premature ventricular contractions (PVCs), defined as 5 or more PVCs per minute, and/or ventricular tachycardia, defined as 3 or more consecutive PVCs during cardiac monitoring. Written informed consent was signed by all the patients prior to participation in the study.

First, a 12-lead ECG was obtained from each participant in the emergency department using Innomed Heart Mirror IKO-3 (Innomed Medical Co., Budapest, Hungary). The ECGs were taken upon admission in each patient admitted with acute coronary syndrome. The collected ECGs were analyzed with Innobase ECG software (Innomed Medical Co., Budapest, Hungary) by 2 investigators, who were not aware of the other clinical data. QT interval was measured from the beginning of the QRS complex to the termination of the T-wave and measured in milliseconds. In the presence of the U-wave, QT interval was terminated at the nadir of the T-U curve. Thereafter, QTc was measured using the Bazett formula.¹⁵ For each patient, maximum and minimum QT and QTc were determined. Finally, QTd and QTc dispersion (QTcd) were defined as the difference between the maximum and minimum of QT and QTc intervals, respectively.¹⁶

For the statistical analyses, the statistical software SPSS version 18.0 for Windows (SPSS Inc., Chicago, IL) was used. Distribution normality was tested via the Kolmogorov-Smirnov test or the Shapiro-Wilk test. Comparisons between the clinical continuous variables were performed using the independent-samples t-test or the Mann-Whitney U-test. The categorical variables were compared using the chi-squared test. A p value < 0.05 was considered significant. Logistic regression analysis was performed to test whether QT prolongation could predict worse clinical outcomes.

Results

The study population comprised 115 patients with NSTEMI. Their clinical characteristics are summarized in Table 1. Of this population, 47 (40%) patients had DM; therefore, fasting blood glucose and hemoglobin A1C were significantly different between the two groups (p value < 0.05). The two groups, at a mean age of 60.8 ± 11.2 years, were not significantly different with respect to their clinical features except for smoking. Need for revascularization was significantly higher in the DM group than in the non-DM

group (24 [51.0%] vs.18 [26.4%]; p value = 0.007). The characteristics of coronary arteries with significant lesions are summarized in Table 2, which reveals no statistically significant difference between different subgroups. Fourteen DM and 10 non-DM patients underwent PCI, and the rest of the patients candidate for revascularization benefited from CABG. Also, ventricular arrhythmia was remarkably higher in the DM patients (9 [19.1%] vs. 4 [5.4%]; p value = 0.027). Moreover, in-hospital mortality occurred in 5 DM patients, 4 of whom had refractory ventricular arrhythmia: of these 4 patients, 2 individuals had three-vessel disease (with the

Table 1. Clinical characteristics of the study population in the two groups of diabetic and nondiabetic patients*

	Type 2 diabetes	Nondiabetes	P value
Number of patients	47 (40.9)	68 (59.1)	0.009
Age (y)	62.7±12.3	59.5±10.5	0.143
Male	24 (51.1)	45 (66.3)	0.149
BMI (kg/m ²)	26.2±4.3	27.1±4.5	0.264
SBP (mmHg)	143.1±24.2	135.2±21.3	0.060
DBP (mmHg)	82.0±14.1	79.3±12.2	0.236
Tobacco usage	15 (31.9)	37 (54.4)	0.017
FBS (mg/dl)	174.4±22.6	93.1±16.4	< 0.001
HbA1C (%)	7.1±1.3	5.9±0.8	< 0.001
LDL (mg/dl)	125.0±42.1	25.9±40.1	0.917
TG (mg/dl)	173.4±86.1	161.1±25.3	0.467
LVEF (%)	41.4±9.9	44.1±10.3	0.158
ACEI use	10 (21.2)	9 (13.2)	0.254
Beta-blocker use	10 (21.2)	15 (22.3)	0.921
Need for revascularization	24 (51.0)	18 (26.4)	0.007
Ventricular arrhythmia	9 (19.1)	4 (5.8)	0.027
In-hospital mortality	5 (10.6)	1 (1.4)	0.029

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

BMI, Body mass index; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; FBS, Fasting blood sugar; HbA1C, Hemoglobin A1C; LDL, Low-density lipoprotein; TG, Triglyceride, LVEF, Left ventricular ejection fraction, ACEI, Angiotensin-converting enzyme inhibitors

Table 2. Characteristics of percentages of coronary arteries with significant lesions in the patients

Subgroup of patients	No of patients	LM (%)*	P value	LAD (%)*	P value	LCX (%)*	P value	RCA (%)*	P value
Diabetics	47	0	NC	80.08	0.544	21.27	0.655	25.53	0.3421
Nondiabetics	68	0		75.00		27.94		33.82	
Diabetics with Ventricular Arrhythmia	9	0	NC	77.77	0.912	44.44	0.852	33.33	0.763
Nondiabetics with Ventricular Arrhythmia	4	0		75.00		50.00		25.00	
Diabetics needed revascularization	24	0	NC	83.33	0.650	41.66	0.856	54.16	0.789
Nondiabetics needed revascularization	18	0		77.77		38.88		50.00	
Diabetics with mortality	5	20.00	NC	80.00	0.624	80.00	0.624	60.00	0.438
Nondiabetics with mortality	1	0		100		100		100	

*Percentage in each territory shows the percentage of patients' subgroups with significant stenosis in the same territory

LM, Left main; LAD, Left anterior descending coronary artery; LCX, Left circumflex coronary artery; RCA, Right coronary artery; NC, Not calculable



involvement of the proximal left anterior descending artery) and moderate left ventricular systolic dysfunction and the other 2 had three-vessel disease with mid left anterior descending artery lesion and mild left ventricular systolic dysfunction. These patients had refractory ventricular tachycardia and/or ventricular fibrillation during the 48-hour period before angiography, which could be related to underlying ischemia. In addition, one patient with left main, right coronary artery disease, and mild left ventricular dysfunction died of pump failure. There was one death among the non-DM patients due to profound decompensated heart failure in an old man with severe three-vessel disease and left ventricular dysfunction. Accordingly, in-hospital mortality was significantly lower in the non-DM group (1.4% vs. 10.6%; p value = 0.029).

The comparisons of QT interval parameters between the DM and non-DM patients are summarized in Table 3. Although all the QT interval measurements were longer in the diabetics, only QTc max (464.4 ± 51.1 vs. 440.4 ± 48.3

ms; p value = 0.011), QTd (49.1 ± 13.1 vs. 40.2 ± 23.0 ms; p value = 0.016), and QTcd (52.2 ± 13.0 vs. 42.1 ± 14.2 ms; p value < 0.001) were significantly longer in the DM group. Furthermore, QTd (51.1 ± 17.0 vs. 40.2 ± 15.1 ms; p value = 0.045) and QTcd (50.1 ± 18.0 vs. 37.1 ± 18.2 ms; p value = 0.025) were significantly higher in the DM patients who needed revascularization (Table 4). Also, QTd and QTcd were significantly higher in the patients who experienced ventricular arrhythmia and had DM versus the nondiabetics (57.0 ± 12.1 vs. 40.2 ± 0 ms; p value = 0.018; 56.2 ± 9.1 vs. 35.3 ± 10.0 ms; p value = 0.003, respectively) (Table 5). No significant difference in QT interval parameters was seen with the use of betablockers and angiotensin-converting enzyme inhibitors. Finally, QTcd was significantly higher in the patients who had higher in-hospital mortality (51.0 ± 15.2 vs. 40.0 ± 13.1 ms; p value = 0.047) (Table 6). When we performed simple binary regression analysis with ventricular arrhythmia as a dependent and QT interval

Table 3. Comparison of QT interval parameters between diabetic and nondiabetic patients*

	Type 2 diabetics	Non-diabetics	P value
Maximum QT (ms)	413.1±49.0	402.2±51.2	0.250
Minimum QT (ms)	364.2±42.1	362.3±45.0	0.810
QTd (ms)	49.1±13.1	40.2±23.0	0.016
Maximum QTc (ms)	464.4±51.1	440.4±48.3	0.011
Minimum QTc (ms)	411.3±44.4	399.2±39.1	0.126
QTcd (ms)	52.2±13.0	42.1±14.2	< 0.001

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

QTd, QT interval dispersion; QTc, Corrected QT interval

Table 4. Comparison of QT intervals in the patients needing revascularization

	Number of patients	Mean±SD	P value
QTmax (ms)			0.718
Diabetics	24	423.1±48.2	
Non-diabetics	18	429.3±59.1	
QTmin (ms)			0.439
Diabetics	24	372.2±52.0	
Non-diabetics	18	385.3±60.3	
QTd (ms)			0.035
Diabetics	24	51.1±17.0	
Non-diabetics	18	40.2±15.1	
QTc max (ms)			0.770
Diabetics	24	419.3±41.0	
Non-diabetics	18	423.0±47.2	
QTc min (ms)			0.189
Diabetics	24	369.4±40.1	
Non-diabetics	18	386.1±42.2	
QTcd (ms)			0.025
Diabetics	24	50.1±18.0	
Non-diabetics	18	37.1±18.2	

QTmax, Maximum QT interval; QTmin, Minimum QT interval; QTd, QT interval dispersion; QTc max, Maximum corrected QT interval; QTc min, Minimum corrected QT interval; QTcd, Corrected QT dispersion

Table 5. Comparison of QT intervals in the patients experiencing ventricular arrhythmia

	Number of patients	Mean±SD	P value
QTmax (ms)			0.245
Diabetics	9	429.3±32.1	
Non-diabetics	4	405.0±34.1	
QTmin (ms)			0.783
Diabetics	9	371.2±36.1	
Non-diabetics	4	365.1±34.2	
QTd (ms)			0.018
Diabetics	9	57.0±12.1	
Non-diabetics	4	40.2±0	
QTc max (ms)			0.068
Diabetics	9	440.4±33.3	
Non-diabetics	4	400.3±33.2	
QTc min (ms)			0.345
Diabetics	9	384.5±36.4	
Non-diabetics	4	365.4±34.3	
QTcd (ms)			0.003
Diabetics	9	56.2±9.1	
Non-diabetics	4	35.3±10.0	

QTmax, Maximum QT interval; QTmin, Minimum QT interval; QTd, QT interval dispersion; QTc max, Maximum corrected QT interval; QTc min, Minimum corrected QT interval; QTcd, Corrected QT dispersion

Table 6. Comparison of QT intervals in the patients regarding in-hospital mortality

	Number of patients	Mean±SD	P value
QTmax (ms)			0.258
Diabetics	6	442.2±38.1	
Non-diabetics	109	431.1±40.3	
QTmin (ms)			0.903
Diabetics	6	362.2±41.4	
Non-diabetics	109	364.4±39.3	
QTd (ms)			0.054
Diabetics	6	48.1±12.0	
Non-diabetics	109	39.3±11.1	
QTc max (ms)			0.397
Diabetics	6	466.1±45.2	
Non-diabetics	109	451.3±42.4	
QTc min (ms)			0.732
Diabetics	6	408.3±41.4	
Non-diabetics	109	415.2±49.3	
QTcd (ms)			0.047
Diabetics	6	51.0±15.2	
Non-diabetics	109	40.0±13.1	

QTmax, Maximum QT interval; QTmin, Minimum QT interval; QTd, QT interval dispersion; QTc max, Maximum corrected QT interval; QTc min, Minimum corrected QT interval; QTcd, Corrected QT dispersion



parameters (QT max, QTc max, QT min, QTc min, QTd, and QTcd) as independent variables, we found that QTc max (OR = 0.97, 95%CI: 0.95-0.99; p value = 0.018), QTd (OR = 0.94, 95%CI: 0.90-0.99; p value = 0.018), and QTcd (OR = 0.95, 95%CI: 0.91-0.99; p value = 0.034) were significantly associated with ventricular arrhythmia, QTcd (OR = 0.95, 95%CI: 0.91-0.99; p value = 0.030) with need for revascularization, and QTc max (OR = 0.97, 95%CI: 0.91-1.00; p value = 0.053) within-hospital mortality in the DM patients. There was no significant association between ventricular arrhythmia, need for revascularization, or in-hospital mortality as dependent and QT interval parameters as independent variables in the non-DM patients.

Multiple binary regression analysis with ventricular arrhythmia, need for revascularization, or in-hospital mortality as dependent and QT interval parameters as independent variables also revealed that QTd (OR = 0.94, 95%CI: 0.90-0.99; p value = 0.018) and QTc max (OR = 0.97, 95%CI: 0.95-0.99; p value = 0.018) were significantly and independently associated with ventricular arrhythmia in the DM patients. Also, a similar association was observed between QTcd (OR = 0.95, 95%CI: 0.91-0.99; p value = 0.030) and need for revascularization and QTc max (OR = 0.97, 95%CI: 0.91-1.00; p value = 0.053) and in-hospital mortality.

Discussion

Our study evaluated different QT interval parameters in patients presenting with NSTEMI between those with DM and those without it. Although all the parameters were longer in the DM group, the results illustrated a clear distinction between the two groups in that QTc max, QTd, and QTcd were significantly different. These findings were nearly compatible with the results of some previous studies.¹⁷⁻¹⁹ Takebayashi et al.²⁰ and Takhashiet al.²¹ stated that increased QTc reflected the imbalance of sympathetic and parasympathetic activity in DM patients. It has been suggested that some non-quantifiable sympathetic imbalance is responsible for QTc prolongation. Hyperglycemia may produce ventricular instability by increased sympathetic activity, increased cytosolic calcium content in myocytes, or both.²² Insulin stimulates sympathetic activity, and DM is known to be associated with impaired parasympathetic cardiac control. This is reflected in a reduced ability to regulate the heart rate as well as a reduction in the heart rate variability.²³

We found significantly longer QT parameters in the DM patients who needed coronary revascularization (QTd and QTcd) similar to the DM patients who manifested ventricular arrhythmia (QTd and QTcd). Our main objective was to find out whether NSTEMI could prolong QT more in DM patients than in non-DM ones. Ischemia by itself can prolong

QT and predispose patients to ventricular arrhythmia, while the additive effect of underlying DM could lead to more QT prolongation, which might result in a poorer prognosis. It is well proven that ventricular repolarization changes during the acute phase of ischemia, thus increasing the risk of lethal arrhythmia.^{1,2} Apart from the influence of ischemia on electrical disturbance, enhanced sympathetic, and reduced vagal activity also affect the repolarization properties of the myocardium. Several parameters have been proposed to quantify the local myocardial repolarization. Although QTd is easily measured, it provides valuable information on the electrical status of the myocardium.¹⁶ The exact cause of increased QTd in the DM population with ischemia and infarction has yet to be fully determined.^{20, 21, 24, 25} Myocardial viability has been suggested as one possible mechanism. The larger infarct in DM patients results in more reduction in myocardial viability and consequently more repolarization inhomogeneity and thus increased QTd.²⁴⁻²⁶ Chauhan et al.²⁷ showed that larger non-Q-wave myocardial infarction was allied to greater QTc and QTd, and longer QT interval parameters were associated with greater risk of mortality. Sakabe et al.²⁶ analyzed QT interval parameters in DM and non-DM patients presenting with STEMI. Rather similar to our study, they found significant differences in QT max and QTcd between their two groups of patients. In the Tentolouriset al.²⁸ study, QTd was not influenced by the presence of DM in the acute coronary syndrome patients. Need for revascularization could be representative of active ischemia, in which QT parameters have been influenced; this may even prove more valuable in the DM population. Moreover, an association between prolonged QTc interval and increased risk of ventricular arrhythmias and sudden death has been shown in DM patients.²⁷ Nevertheless, no study has so far demonstrated that DM itself may impact QT prolongation more prominently during acute ischemia and infarction.

In the current study, there was one death in the non-DM group. An evaluation of QT parameters influencing mortality in the two groups was not possible in terms of statistical analysis. Nonetheless, our results revealed that QTcd was significantly higher in 6 patients who had in-hospital mortality and that QTc max was significantly associated with in-hospital mortality. The mechanism whereby prolonged QT interval and increased QTd predict increased cardiac mortality and morbidity in DM has been much debated. It appears that these two parameters provide different information. QTd is believed to correlate to a greater extent with the risk of ventricular arrhythmias than QT prolongation, as we observed in 4 out of our 5 DM patients who died of ventricular arrhythmia in the current study. Some conditions are associated with a prolonged QT interval: for instance, therapy with Sotalol may actually reduce the risk of sudden death in association with a reduction in QTd but an increase in QTc.²⁹ It was initially thought that this is

because QTd reflects electrical heterogeneity, but this view has now been challenged and QTd has been proposed as a noninvasive marker of potentially lethal underlying cardiac abnormalities, the most important of them being ischemia.³⁰ It is further supported by the observation that QTd is prolonged immediately after a myocardial infarction and tends to reduce after successful thrombolysis.¹⁷ Cardoso et al.³¹ reported that arterial hypertension, diabetic cardiovascular complications, left ventricular hypertrophic changes, and conduction disturbances in the surface ECG significantly influenced QTd in the DM population. Some authors have proposed cardiac autonomic neuropathy as the main mechanism for different QTd in the DM population.^{20, 21}

Overall, increased QTd and QTcd seem to represent the sum of several adverse cardiac abnormalities such as fibrosis, hypertrophy, dilatation, ischemia and probably, autonomic dysfunction.^{30, 32} All these factors individually confer increased cardiovascular risk and QTd and/or QTcd, as a summation, could be a global prognostic marker for cardiac mortality in patients with DM. The debate on what causes sudden death (arrhythmia or ischemia), however, continues.

Although the resting heart rate sometimes is higher in DM patients, we found no significant difference between the two groups in terms of beta-blocker consumption. Additionally, QT intervals were not significantly different between the two study groups regarding the use of betablockers. Previous studies have shown that beta blockers increase the QT interval at slower heart rates and decrease the QT interval at faster heart rates, which may explain their protective effect in the long QT syndrome.³³ In our study, QT parameters were not significantly different with respect to beta-blocker consumption, which may be due to the small number of the patients who received betablockers.

One of the limitations of this study is that we evaluated the extension of ischemia and infarction only via echocardiography, and we were not able to provide the viability assessment with other imaging modalities. Also, there was one death in the non-DM patients, precluding the statistical analysis of the QT parameters between the two groups of diabetics and nondiabetics. Although the use of beta blockers was not different between our two study groups, we could not exclude the possible interaction between the drug and QT-interval measurement.

Conclusion

QTd is considered a measure of lethal arrhythmia and mortality in patients presenting with myocardial infarction. In the current study, conducted on patients presenting with NSTEMI, a significant increase in QTc max, QTd, and QTcd was shown in the DM patients as opposed to the non-DM patients. The results suggest that our DM patients who had ventricular arrhythmia and coronary revascularization had

higher QTd and QTcd and that there was a relationship between QTd and QTc max and ventricular arrhythmia, QTcd and need for revascularization, and QTcmax and in-hospital mortality. In addition, QTcd was increased in the DM patients with NSTEMI who had higher mortality. The inclusion of simple measures should enable us to achieve a better risk stratification.

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