

# COMPARISON BETWEEN QT-INTERVAL PARAMETERS IN TYPE 2 DIABETIC AND NON-DIABETIC PATIENTS IN NON-ST SEGMENT ELEVATION MI

*Original Research*

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## ABSTRACT

**Background:** Diabetes mellitus is known to increase cardiovascular risk and exacerbate complications during acute coronary events. QT interval abnormalities have been linked to adverse cardiac outcomes, particularly in diabetic individuals. Evaluating QT interval parameters in patients with non-ST segment elevation myocardial infarction (NSTEMI) may offer valuable insight into arrhythmic risk and guide clinical decision-making. This study aimed to compare QT interval characteristics between type 2 diabetic and non-diabetic patients presenting with NSTEMI to better understand this electrophysiological interplay.

**Objective:** To compare QT-interval parameters in type 2 diabetic and non-diabetic patients with NSTEMI.

**Methods:** This quasi-experimental study was conducted at the Department of Cardiology, Khyber Teaching Hospital, Peshawar, over a six-month period from July 11, 2024, to January 10, 2025. A total of 58 patients aged 35 to 80 years diagnosed with NSTEMI were enrolled and categorized into two groups: diabetic (n=29) and non-diabetic (n=29). Type 2 diabetes status was confirmed using HbA1c (>6.5% for diabetics, <5.5% for non-diabetics). A standard 12-lead ECG was obtained from all participants. QT max, QT min, QTc max, QTc min, QT dispersion (QTd), and corrected QT dispersion (QTcd) were measured. Mean values were compared between the groups using an independent samples t-test. SPSS version 26.0 was used for data analysis.

**Results:** QTd was significantly higher in diabetics ( $47.31 \pm 7.74$  ms) than in non-diabetics ( $38.83 \pm 5.78$  ms,  $p < 0.001$ ). QTc min was also significantly prolonged in diabetics ( $53.00 \pm 8.62$  ms) compared to non-diabetics ( $41.58 \pm 6.54$  ms,  $p < 0.001$ ). QTc max showed a non-significant elevation in diabetics ( $425.83 \pm 36.52$  ms) versus non-diabetics ( $414.83 \pm 19.21$  ms,  $p = 0.157$ ). QTcd values were higher in diabetics ( $372.82 \pm 37.49$  ms) than non-diabetics ( $373.24 \pm 22.90$  ms) but not statistically significant ( $p = 0.960$ ).

**Conclusion:** QT interval parameters, particularly QTd and QTc min, were significantly prolonged in diabetic patients with NSTEMI, suggesting a heightened risk of ventricular repolarization disturbances in this group. These findings support the use of ECG-based QT metrics for enhanced risk stratification in diabetic cardiac patients.

**Keywords:** Cardiac electrophysiology, Diabetic patients, Electrocardiography, Myocardial infarction, NSTEMI, QT dispersion, QT interval.

## INTRODUCTION

Myocardial infarction (MI) can trigger life-threatening arrhythmias due to increased electrical instability in the heart, particularly through disturbances in repolarization. The surface electrocardiogram (ECG), especially measurements associated with the QT interval, serves as a non-invasive and widely available tool to evaluate this electrical heterogeneity. Specifically, the QT dispersion (QTd)—the difference between the maximum and minimum QT intervals across ECG leads—offers a quantitative measure of repolarization dispersion and has emerged as a potential predictor of mortality in patients with myocardial ischemia (1,2). Individuals with diabetes mellitus are at an elevated risk of both fatal and non-fatal cardiac events compared to the general population (3,4). This increased cardiovascular vulnerability has prompted the exploration of various risk stratification tools in diabetic patients. Among these, QT interval parameters have gained attention, with several studies indicating that diabetic individuals tend to have higher QTd values. This suggests a possible link between altered ventricular repolarization and cardiovascular outcomes in this population (5,6). In a long-term cohort of patients with type 2 diabetes, a study demonstrated that QTd independently predicted cardiovascular mortality over a 15-year follow-up period (7). Supporting these findings, a study identified both QTc prolongation and the presence of bundle branch block as prognostic indicators in the PROactive trial involving type 2 diabetes mellitus (8). Furthermore, another study highlighted that QTc was associated with mortality in type 1 diabetes, while resting heart rate was a stronger predictor in those with type 2 diabetes (9,10).

Recent comparative studies have begun to explore QT interval differences between diabetic and non-diabetic individuals in the context of acute coronary syndromes. For instance, one study noted that while some QT interval parameters such as maximum QT and minimum QTc did not differ significantly between groups, measures like QTd, QTc max, and corrected QT dispersion (QTcd) were markedly higher in type 2 diabetic patients, with statistically significant p-values for these differences (11-13). These findings underscore the potential of QT-related parameters as risk markers in diabetic individuals, particularly during episodes of myocardial ischemia. Despite growing interest, there remains a paucity of data evaluating QT interval dynamics in the specific setting of non-ST segment elevation myocardial infarction (NSTEMI) among diabetics. Given the high prevalence of diabetes and the growing burden of ischemic heart disease in many regions, especially in developing countries, there is a pressing need to refine prognostic tools tailored to these high-risk subgroups. Therefore, this study was designed to compare QT-interval parameters between type 2 diabetic and non-diabetic patients presenting with NSTEMI, with the objective of contributing novel insights to the current body of cardiovascular research and supporting the development of locally relevant clinical guidelines.

## METHODS

This quasi-experimental study was conducted in the Department of Cardiology at MTI-Khyber Teaching Hospital, Peshawar, over a six-month period from July 11, 2024, to January 10, 2025. It aimed to compare QT-interval parameters between type 2 diabetic and non-diabetic patients presenting with non-ST segment elevation myocardial infarction (NSTEMI). The study population included male and female patients aged 35 to 80 years, diagnosed with NSTEMI based on clinical presentation, elevated cardiac biomarkers (cardiac troponin I > 0.04 ng/mL and CK-MB > 25 IU/L), and the presence of ST-segment depression on ECG. Patients with ST-elevation MI (STEMI), type 1 diabetes mellitus, non-sinus rhythm, or those taking medications known to influence cardiac electrophysiology were excluded to avoid confounding effects. Diabetes status was confirmed using HbA1c levels, with a threshold of >6.5% to categorize patients as diabetic and <5.5% as non-diabetic. Eligible participants were allocated into two groups: Group A (diabetic) and Group B (non-diabetic), using non-probability consecutive sampling. The sample size was calculated using the WHO sample size formula for comparison of means, based on previously reported QTcd values in diabetic ( $52.2 \pm 13.0$  ms) and non-diabetic ( $42.1 \pm 14.2$  ms) groups, with a confidence level of 95% and power of 80%, yielding a total of 58 patients (29 per group). After obtaining written informed consent, participants were enrolled following approval from the Institutional Review Board and the Ethical Committee of the hospital as well as the ER Department, CPSP Karachi. Patients meeting the inclusion criteria were recruited from the cardiology outpatient department, and relevant demographic information such as age, gender, residence, occupation, educational background, and social class was recorded.

All patients were managed according to standard care protocols aligned with ACC/AHA guidelines. A resting 12-lead ECG was used to measure QT interval parameters. These included maximum QT interval (longest QT interval measured from the beginning of the QRS complex to the end of the T wave), minimum QT interval (shortest interval measured similarly), QT dispersion (QTd, defined as the difference between maximum and minimum QT), and their corrected counterparts—QTc max, QTc min, and QT corrected dispersion (QTcd)—adjusted for heart rate using Bazett’s formula. Two blinded investigators independently analyzed all ECG tracings to ensure objectivity, with no access to the patients’ clinical profiles. Data was initially compiled in Microsoft Excel and analyzed using SPSS version 23.0. Mean and standard deviation were reported for continuous variables such as age, BMI, and QT parameters, while categorical data including gender, social status, lifestyle, smoking status, educational level, beta-blocker use, need for revascularization, coronary artery involvement, ventricular arrhythmias, and in-hospital mortality were summarized as frequencies and percentages. The Kolmogorov–Smirnov test was used to assess the normality of continuous variables. Intergroup comparisons of quantitative variables were performed using the independent-samples t-test, while qualitative variables were assessed using the chi-square test. Stratification of results was performed to evaluate potential effect modifiers. A p-value of <0.05 was considered statistically significant.

RESULTS

The study included a total of 58 participants, evenly distributed between diabetic (n=29) and non-diabetic (n=29) groups. The mean age of participants with diabetes was  $59.31 \pm 7.20$  years, slightly higher than the non-diabetic group with a mean age of  $57.97 \pm 6.43$  years. Mean BMI was also marginally higher in the diabetic group at  $25.08 \pm 1.12$  kg/m<sup>2</sup> compared to  $24.91 \pm 1.02$  kg/m<sup>2</sup> in non-diabetics. Among participants aged over 55 years, 53.8% were diabetic, indicating a slightly higher prevalence of diabetes in older age. Gender distribution was equal between groups, with males and females accounting for 50% each in the total sample. Smoking was more frequent in the non-diabetic group (56.3%) compared to diabetics (43.8%), while beta-blocker use was slightly higher among non-diabetics (51.3%) versus diabetics (48.7%). In-hospital mortality occurred in 15 patients, with a comparable distribution between diabetics (53.3%) and non-diabetics (46.7%). Notably, coronary artery lesions were more frequently observed among diabetics (61.8%) than non-diabetics (38.2%). In terms of electrocardiographic parameters, the mean maximum QT interval was  $417.62 \pm 40.26$  ms in diabetics and  $411.65 \pm 23.80$  ms in non-diabetics, a difference that was not statistically significant (p=0.495). However, a statistically significant difference was found in minimum QT interval values, with diabetics showing a higher mean of  $47.31 \pm 7.74$  ms compared to  $38.83 \pm 5.78$  ms in non-diabetics (p<0.001). Similarly, the minimum corrected QT interval (QTc min) was significantly prolonged in diabetics ( $53.00 \pm 8.62$  ms) compared to non-diabetics ( $41.58 \pm 6.54$  ms), with a p-value <0.001. Although QTc max was elevated in the diabetic group ( $425.83 \pm 36.52$  ms) versus the non-diabetic group ( $414.83 \pm 19.21$  ms), the difference was not statistically significant (p=0.157). No statistically significant differences were observed in QT dispersion ( $370.31 \pm 41.38$  ms in diabetics vs.  $372.82 \pm 25.14$  ms in non-diabetics, p=0.781) or in corrected QT dispersion (QTcd) values ( $372.82 \pm 37.49$  ms vs.  $373.24 \pm 22.90$  ms, p=0.960), indicating comparable variability in ventricular repolarization across both groups despite some significant interval differences.

Table 1: Descriptive statistics of study participants (n = 58)

Group		Mean	Std. Deviation
Diabetic (n = 29)	Age (years)	59.31	7.202
	BMI (kg/m <sup>2</sup> )	25.076	1.1182
	QT Maximum (ms)	417.6207	40.26733
	QT Minimum (ms)	47.3103	7.74183
	QTc Maximum (ms)	425.8276	36.52012
	QTc Minimum (ms)	53.0000	8.62306
Non diabetic (n = 29)	Age (years)	57.97	6.428
	BMI (kg/m <sup>2</sup> )	24.914	1.0246
	QT Maximum (ms)	411.6552	23.79867
	QT Minimum (ms)	38.8276	5.77599
	QTc Maximum (ms)	414.8276	19.21322
	QTc Minimum (ms)	41.5862	6.54390

**Table 2: Baseline demographics and clinical characteristics of study participants (n = 58)**

		Group		Total
		Diabetic (n = 29)	Non diabetic (n = 29)	
Age (years)	55 or below	8	11	19
		42.1%	57.9%	100.0%
	More than 55	21	18	39
		53.8%	46.2%	100.0%
Gender	Male	14	15	29
		48.3%	51.7%	100.0%
	Female	15	14	29
		51.7%	48.3%	100.0%
BMI (kg/m <sup>2</sup> )	25.0 or below	18	19	37
		48.6%	51.4%	100.0%
	More than 25.0	11	10	21
		52.4%	47.6%	100.0%
Residence	Rural	16	19	35
		45.7%	54.3%	100.0%
	Urban	13	10	23
		56.5%	43.5%	100.0%
Education	No formal schooling	10	4	14
		71.4%	28.6%	100.0%
	Matric or below	10	12	22
		45.5%	54.5%	100.0%
Profession	Above matric	9	13	22
		40.9%	59.1%	100.0%
	Salaried	14	14	28
		50.0%	50.0%	100.0%
Smoking	Business	15	15	30
		50.0%	50.0%	100.0%
	Yes	14	18	32
		43.8%	56.3%	100.0%
Beta Blocker	No	15	11	26
		57.7%	42.3%	100.0%
	Yes	19	20	39
		48.7%	51.3%	100.0%
Revascularization	No	10	9	19
		52.6%	47.4%	100.0%
	Yes	12	15	27
		44.4%	55.6%	100.0%
In hospital Mortality	No	17	14	31
		54.8%	45.2%	100.0%
	Yes	8	7	15
		53.3%	46.7%	100.0%
Coronary Artery Lesion	No	21	22	43
		48.8%	51.2%	100.0%
	Yes	21	13	34
		61.8%	38.2%	100.0%
	No	8	16	24
		33.3%	66.7%	100.0%

**Table 3: Comparison of QT interval parameters between diabetic and non-diabetics (n = 58)**

QT parameters (ms)	Group	N	Mean	S. D	Mean difference	P value
QT Max	Diabetic	29	417.62	40.26	5.96	0.495
	Non diabetic	29	411.65	23.79		
QT Min	Diabetic	29	47.31	7.74	8.48	0.000
	Non diabetic	29	38.82	5.77		
QTc Max	Diabetic	29	425.82	36.52	11.00	0.157
	Non diabetic	29	414.82	19.21		
QTc Min	Diabetic	29	53.00	8.62	11.41	0.000
	Non diabetic	29	41.58	6.54		
dQT	Diabetic	29	370.31	41.38	2.51	0.781
	Non diabetic	29	372.82	25.14		
dQTc	Diabetic	29	372.82	37.49	0.413	0.960
	Non diabetic	29	373.24	22.90		

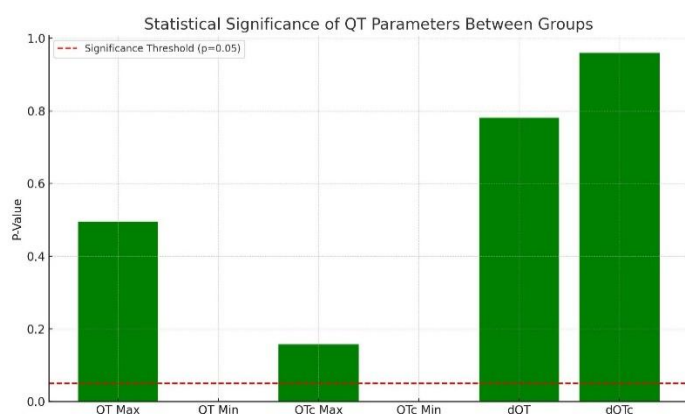


Figure 1 Statistical Significance of QT Parameters Between Groups

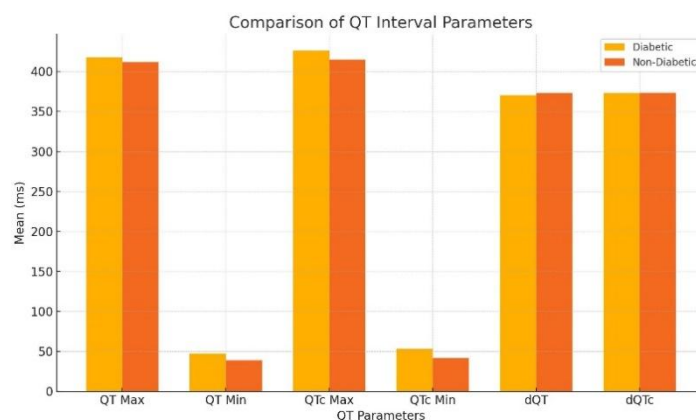


Figure 2 Comparison of QT Interval Parameters

## DISCUSSION

The present study demonstrated that patients with type 2 diabetes mellitus (DM) presenting with non-ST segment elevation myocardial infarction (NSTEMI) exhibited prolonged QT interval characteristics compared to their non-diabetic counterparts. All measured QT parameters, including QT max, QT min, QTc max, QTc min, QTd, and QTcd, remained elevated in the diabetic group. These findings align with previous evidence reporting that individuals with DM are more likely to have altered cardiac electrophysiology and repolarization abnormalities, particularly under ischemic conditions (14–16). The prolonged QTc interval in diabetics has been widely attributed to autonomic imbalance—characterized by increased sympathetic and diminished parasympathetic activity—which contributes to myocardial electrical instability and vulnerability to arrhythmias (17,18). Hyperglycemia may further exacerbate this vulnerability through elevated intracellular calcium levels and heightened sympathetic outflow, culminating in ventricular repolarization abnormalities (19,20). QTd and QTcd were notably more pronounced in diabetic individuals who required coronary revascularization, suggesting a greater burden of electrical instability and myocardial ischemia in this subgroup. This observation supports the premise that diabetes mellitus amplifies the electrophysiological disturbances triggered by acute ischemic events such as NSTEMI. While myocardial ischemia alone is known to induce QT prolongation and predispose patients to malignant ventricular arrhythmias, the concurrent presence of diabetes may act synergistically, leading to more pronounced electrophysiological derangements and potentially worse clinical outcomes (1,2). Repolarization abnormalities in acute ischemia are further compounded by autonomic dysfunction, characterized by sympathetic hyperactivity and vagal withdrawal, both of which are frequently observed in the diabetic population.

Although QTd is a relatively simple parameter to calculate, it offers meaningful insight into myocardial electrical heterogeneity and has been proposed as a surrogate marker for regional repolarization variability (16,21).

The precise pathophysiological basis for elevated QTd in diabetic patients remains incompletely understood. One proposed mechanism centers on diminished myocardial viability. Diabetes has been associated with larger infarct sizes, leading to greater myocardial damage, reduced viability, and increased electrical inhomogeneity, all of which can prolong QTd (22–24). Findings from earlier research have linked prolonged QT interval characteristics with higher risk of cardiac events and mortality, particularly in diabetic individuals with acute coronary syndromes (23–25). Comparative studies in STEMI patients also revealed significant differences in QT max and QTcd between diabetic and non-diabetic patients, which are consistent with the trends reported in this study. However, contrasting results from some investigations suggest that the influence of diabetes on QT parameters during acute ischemic events may not be uniformly observed across all populations and clinical scenarios (25). In-hospital mortality in this study occurred in a similar proportion between the diabetic and non-diabetic groups, with eight and seven deaths respectively, and this difference was not statistically significant. Despite this, higher QTcd and QTc max values were observed in individuals who died during hospitalization, indicating a potential association between these ECG parameters and mortality. Although causality could not be established due to the study’s sample size and observational nature, these findings are suggestive of a clinically meaningful relationship. Existing literature continues to debate the mechanisms by which QT prolongation contributes to mortality risk in diabetic patients. It is plausible that QTd and QTc reflect distinct dimensions of arrhythmic risk, with each parameter capturing different aspects of myocardial electrical vulnerability.

This study had several strengths, including a well-defined patient population, standardized QT interval measurement, and blinded ECG analysis to minimize interpretation bias. Furthermore, by focusing on NSTEMI, it addressed a clinical subset less frequently explored in QT interval research compared to STEMI. However, certain limitations must be acknowledged. The relatively small sample size limited the power to detect associations between QT parameters and clinical outcomes such as mortality. Additionally, the exclusion of patients with borderline glycemic profiles (pre-diabetes) may have restricted insights into the full spectrum of QT alterations across glycemic statuses. The cross-sectional design precluded longitudinal assessment of arrhythmic events or long-term mortality, and confounding variables such as electrolyte imbalances, autonomic testing, or concomitant medication use were not comprehensively controlled. Future research should include larger, multicenter cohorts with longitudinal follow-up to better understand the prognostic utility of QT interval characteristics in diabetic patients with NSTEMI. It would be beneficial to integrate autonomic function testing, myocardial viability assessment, and serial QT measurements to clarify the mechanistic underpinnings of repolarization abnormalities in this high-risk population. Incorporating such parameters could lead to refined risk stratification tools and tailored therapeutic strategies aimed at reducing cardiovascular morbidity and mortality in diabetic individuals following acute coronary events.

CONCLUSION

This study highlighted the significant prolongation of QTc max, QTd, and QTcd in patients with type 2 diabetes mellitus presenting with non-ST segment elevation myocardial infarction, underscoring the added electrophysiological burden posed by diabetes during acute coronary events. The associations observed between elevated QT dispersion indices and adverse outcomes—including ventricular arrhythmias, the need for coronary revascularization, and in-hospital mortality—suggest that these simple, non-invasive ECG parameters could serve as valuable adjuncts in early risk stratification. Incorporating QT interval characteristics into standard clinical assessment may enhance the identification of high-risk diabetic patients, ultimately supporting more tailored and proactive management strategies to improve cardiac outcomes in this vulnerable population.

AUTHOR CONTRIBUTION

Author	Contribution
Kamran Khan*	Substantial Contribution to study design, analysis, acquisition of Data
	Manuscript Writing
	Has given Final Approval of the version to be published
Saima Humayun	Substantial Contribution to study design, acquisition and interpretation of Data
	Critical Review and Manuscript Writing
	Has given Final Approval of the version to be published



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