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Original Research Article

Prevalence and Predictors of QT Interval Prolongation in Poorly Controlled Diabetes: A Cross-Sectional Study

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Abstract:

Background: The prolongation of the QT interval is a well-established marker of risk for developing ventricular arrhythmias. In diabetes, it is frequently changes secondary to metabolic, autonomic and cardiovascular perturbations. Poorly controlled type 2 diabetes may worsen any electrophysiological disarray.

Aim: The study aims to determine the presence of QTc prolongation and increased QT dispersion (QTd) in poorly controlled diabetes, and to determine the clinical and metabolic predictors of QTc prolongation.

Method: This cross-sectional study was conducted with 200 adults with type 2 diabetes where HbA1c > 8%. Demographics, clinical and metabolic variables were obtained. Standard 12-lead ecg's were analyzed for QTc and QTd. Between group comparisons and logistic regression were used to identify predictors of QTc > 440 ms.

Results: QTc prolongation was found to be prevalent seen in 46% of patients while 3% of patients having QTc > 500 ms and QTd > 80 ms in 4% of patients. There were more females with QT prolongation. Prolonged QTc was associated with older age, increased BMI, coronary heart disease, retinopathy, poor glycemic indices and high triglycerides. Independent predictors of prolonged QTc included increased age, increased BMI, coronary heart disease, retinopathy, use of sulphonyureas, HbA1c and triglycerides.

Conclusions: QT abnormalities are common in poorly controlled diabetes and are strongly related to metabolic dysregulation and cardiovascular complications. Early identification and glycemic improved glycemic control may reduce the risk of arrhythmias.

Keywords: QT interval, QTc prolongation, QT dispersion, type 2 diabetes, glycemic control, arrhythmia risk, predictors.

Introduction

The QT interval in the surface electrocardiogram (ECG) is the sum of all the ventricular depolarization and repolarization, which is an important non-invasive measuring rod of cardiac electrophysiological stability. Irregularities during this period especially its extension have always been known as predictors of higher risks of ventricular arrhythmias and sudden cardiac death. The difference between the maximum and minimum intervals of QT observed on a variety of ECG leads, known as QT dispersion (QTd), is another manifestation of the heterogeneity of myocardial repolarization. Higher QTd implies the presence of regional differences in myocardial refractoriness and is thought to be the basis on which reentrant circuits triggering malignant ventricular arrhythmia may develop. Long QT interval and long QTd have both become useful predictors in cardiovascular studies and human care [1].

There is a cumulative amount of literature documenting that deviations of QT parameters are closely linked to an increased risk of cardiovascular and overall mortality. As an example, QT prolonged and prolonged QTd have been reported to indicate poor patient outcomes in patients after acute myocardial infarction, heart failure patients [2] and type 1 and type 2 diabetes mellitus patients. Previous studies have demonstrated similar associations in patients with idiopathic long QT syndrome, and in seemingly normal populations, and these findings underscore the general clinical implications of QT interval measures in different populations of patients. The results highlight the importance of the abnormalities of ventricular repolarization as the precursors of electrophysiological evidence of underlying cardiovascular pathology and as the instruments of predicting high-risk groups.

Diabetes mellitus, especially type 2 diabetes, is well known as a multisystem condition which has significant cardiovascular consequences. Poor glycaemic control has been associated with the acceleration of cardiovascular complications, which involve autonomic neuropathy, microvascular ischemia, oxidative stress, and chronic inflammation of low grade. Some of the less evident, but still clinically significant, presentations of these complications include changes in myocardial repolarization that are reported in QT prolongation and elevated QT dispersion [3]. Some of the studies have postulated that diabetic patients have subclinical cardiac autonomic dysfunction which has contributed to impaired repolarization despite the absence of evident structural heart disease. The electrophysiological changes mentioned could increase the risk of arrhythmia and sudden cardiac events over time, and QT interval analysis is especially applicable to such population.

Sustained hyperglycemia, which is a sign of poor glycaemic regulation, exerts increased metabolic and electrophysiological loads on cardiac tissue. It has been reported that chronic hyperglycemia is linked to impairment of ion channel functionality, advanced glycation end products, and microvascular dysfunction, which could be the causes of long-term myocardial repolarization. Moreover, another complication that is prevalent and underdiagnosed, diabetic autonomic neuropathy can decrease vagal activity and increase sympathetic dominance, increasing the likelihood of individuals experiencing distorted QT dynamics. Since a significant portion of patients with type 2 diabetes are asymptomatic until the onset of advanced cardiovascular disease, electrophysiological indicators like QT prolongation can be useful in terms of early cardiovascular risk prediction and can be used as clinically significant screening methods [4].

Although there was an increasing awareness of the importance of the abnormality of QT interval in diabetes, the prevalence of the prolonged QT interval and QT is highly diverse among the studies due to the variations in the patient populations, glycaemic control, comorbidity, medications, and methods used. Moreover, the predictors of the abnormalities of QT in the poorly controlled diabetes are not fully comprehended. Determination of such predictors is important in increasing risk stratification, therapeutic decisions, and clinical monitoring strategies. The continuous increase in the prevalence of type 2 diabetes around the world and the high proportion of patients having poor glycaemic control despite medication has made the electrophysiological effects of diabetes an issue of growing significance to the prevention of cardiovascular morbidity and mortality [5].

QT prolongation may also be worsened by another major comorbidity in patients with type 2 diabetes, coronary heart disease (CHD). Such ischemic myocardial injury alters ventricular repolarization and may interact with complications of diabetes to increase the risk of arrhythmias. Thus, it is clinically pertinent to study the interplay between CHD and QT interval abnormalities in patients with inadequate glycaemic control that might be associated with faster atherosclerosis and endothelial dysfunction. Likewise, other metabolic parameters like dyslipidemia, hypertension, and obesity are typical characteristic of the metabolic syndrome more apt to significantly accompany type 2 diabetes and can lead to electrophysiological changes. The interaction of these factors highlights the significance of assessment of QT abnormalities in the metabolic and cardiovascular context of diabetes in general [6].

Along with clinical and metabolic parameters, QT dynamics may depend on treatment modalities of diabetes. Some medications used to lower glucose levels (insulin or oral anti-diabetic agents) can differ in their impact on autonomic activity, electrolyte balance and cardiac ion channels. Knowledge of the effects of various treatment regimens on QT interval and QTd will play a vital role in optimizing therapies due not only to the glycaemic goal but also to the reduction of cardiovascular risk factors [7]. An example is that insulin- or insulin secretagogue-induced hypoglycemic events can temporarily increase the QT interval perhaps by counterregulatory catecholamine spikes or changes in potassium homeostasis. These mechanisms enable the necessity of close attention to QT measurements in patients who take intensive glucose-lowering treatments.

Nonetheless, the literature on the prevalence and predictors of QT interval prolongation in a particular population, namely those with inadequately managed type 2 diabetes, which is a particularly high-risk group with regard to cardiovascular complications, has a gap. The majority of the past studies have considered mixed populations that have different glycaemic control or have failed to effectively provide the range of metabolic and treatment-related variables that can affect QT dynamics. Targeted evaluation of the diabetes that is poorly managed, therefore, can be justified in order to further outline the burden of electrophysiological abnormalities in such a vulnerable group and recognise modifiable predictors that can be used in clinical interventions.

It is on these gaps and the substantial amount of evidence to support the linkage of QT abnormalities with the adverse cardiovascular outcomes that this research was formulated to test the prevalence of prolonged QT interval and QT dispersion in patients with type 2 diabetes. Additionally, it attempted to examine correlations between QT parameters and various clinical and metabolic variables, especially being interested in coronary heart disease, glycaemic control, and type of diabetes treatment. This study aims to further the current knowledge regarding arrhythmic risk in diabetes that is not well-controlled and add into the existing body of knowledge on improving early disease detection, monitoring, and prevention of cardiovascular events in this high-risk cohort by identifying the major predictors of QT prolongation.

Methodology

Study Design: This was a cross-sectional observational study conducted to determine the prevalence and predictors of QT interval prolongation among patients with poorly controlled type 2 diabetes mellitus.

Study Area: The study was conducted in the Department of Cardiology, Fortis Escorts Heart Institute, New Delhi, India.

Study Duration: The study was carried out over a period of from December 2022 to November 2023

Sample Size: A total of 200 patients with poorly controlled type 2 diabetes were included in the study.

Study Population: The study population consisted of adult patients with poorly controlled type 2 diabetes mellitus attending the cardiology department for evaluation. Poor glycemic control was defined as HbA1c > 8% (or the cutoff adopted by the research protocol).

Inclusion Criteria

- Adults aged ≥ 30 years diagnosed with type 2 diabetes mellitus.
- HbA1c > 8% (poor glycemic control).
- Patients able and willing to provide written informed consent.
- Patients in sinus rhythm at the time of ECG recording.

Exclusion Criteria

- History or ECG evidence of myocardial hypertrophy or bundle branch block.
- Current use of medications known to affect QT interval (e.g., class I/III antiarrhythmics, digitalis, tricyclic antidepressants).
- Presence of electrolyte abnormalities (hypokalemia, hypocalcemia, hypomagnesemia).
- Documented cardiovascular autonomic neuropathy (e.g., orthostatic hypotension).
- History of congenital long QT syndrome.
- Hypoglycemia (<70 mg/dL) on the day of glycemic profiling.
- Patients on pacemakers or with atrial fibrillation.

Data Collection

The demographic details, clinical history, cardiovascular risk factors, and medication use were obtained with the use of direct interviews and referred to the hospital records. The anthropometric measurements were recorded i.e. height, weight, and body mass index and systolic and diastolic blood pressure were measured using standard procedures. The patient was placed in the supine position, and a regular 12-lead ECG was recorded and QT and RR intervals were measured manually by two different blind observers who were not aware of the clinical information. QT intervals were adjusted to heart rate with the use of the Bazett formula, and the mean of QTc values were taken in five heart cycles in lead V5. The difference between the largest and smallest QTc values of the six precordial leads was determined as QT dispersion. Glycemic indices were assessed on the same day of ECG recording by using standardized laboratory techniques of fasting blood glucose, postprandial glucose, mean blood glucose, glycated hemoglobin (HbA1c), and mean amplitude of glycemic excursion (MAGE). MAGE was computed on the basis of absolute deviations between succeeding peaks and nadirs more than one standard deviation of the mean glucose value.

Procedure: The selection of eligible patients was done in the course of their routine clinical visits at the cardiology department. Demographic and clinical data were recorded after an informed consent was received and physical and laboratory evaluation was performed. The ECG recordings were conducted under rest regimes as per the institutional guidelines. Two trained observers measured the QT and RR intervals independently to reduce the possibility of measurements bias and averaged the results were analysed. Glycemic profiles were taken on the same day and all the laboratory tests were taken in the central laboratory of the hospital to maintain the same. All the data were recorded in a structured case, record form constructed specifically to suit the study.

Statistical Analysis: The SPSS version 21.0 (IBM Corp., Chicago, IL) was used in statistical analysis. The Z-score and Mahala Nobis distance were used to test the completeness, accuracy and outliers of the data in the form of multivariate analysis to detect outliers. The Kolmogorov-Smirnov test was used to determine whether the continuous variables were normal. Continuous variables were summarized using the means and standard deviation, and mean frequencies and percentages were used to summarize categorical variables. The independent t-test was used to compare the normal versus prolonged QTc and normal versus prolonged QT dispersion under between-group comparisons to assess the significance of the two variables being compared. Chi-square test was to be used to assess the significance of normal versus prolonged and normal versus prolonged under between-group comparisons. The independent predictors of QTc prolongation and QT dispersion were addressed with the help of logistic regression analysis; the variables were age, sex, duration of diabetes, blood pressure, BMI, coronary artery disease, neuropathy, fasting glucose, postprandial glucose, mean blood glucose, MAGE, HbA1c, lipid profile, and estimated glomerular filtration rate, which were added to the initial model. The variables that were included in the final model were retained because of their statistically significant features and their contribution to the model. The p-value; less than 0.05 was deemed significant."

Result

The demographic, clinical and metabolic features of the 200 study participants (110 men and 90 women) with the mean age of 58.2-9.1 years and average years of diabetes duration 10.2-6.4 years are presented in Table 1. The cohort depicted a mean BMI of 29.8 + and 5.0 kg/m^2 and a moderately high level of blood pressure (136.1 + 16.8 mmHg systolic, 81.0 + 7.2 mmHg diastolic). Cardiovascular comorbidities were widespread, with one-quarter having coronary heart disease, and half of the patients having retinopathy in 48.5% and polyneuropathy in 70. The majority of respondents were under antidiabetic therapy, with three-quarters of them taking metformin, forty percent taking sulphonyl Reas, or sixty-five percent taking insulin. Glycemic indices were indicative of poor control with a mean fasting glucose of 9.2 mmol/L, 11.0 mmol/L postprandial glucose, mean glucose of 10mmol/L, MAGE of 4.3mmol/L and HbA1c of 8.6mmol/L. Lipid-profiles revealed increased levels of total cholesterol (5.7 ± 1.4 mmol/L) and triglycerides (2.2 ± 1.3 mmol/L), but overall kidney-function was usually normal with a mean eGFR of 92.1 ± 23.8 mL/min/1.73 m^2.

Table 1: Demographic, Clinical and Metabolic Characteristics of the Study Population (N = 200)

Variable	Value
Male / Female (n)	110 / 90
Age (years)	58.2 ± 9.1
Duration of diabetes (years)	10.2 ± 6.4
BMI (kg/m²)	29.8 ± 5.0
Systolic BP (mmHg)	136.1 ± 16.8
Diastolic BP (mmHg)	81.0 ± 7.2
Coronary heart disease, n (%)	48 (24.0%)
History of stroke, n (%)	10 (5.0%)
Retinopathy, n (%)	97 (48.5%)
Polyneuropathy, n (%)	140 (70.0%)
Metformin therapy, n (%)	150 (75.0%)
Sulphonylurea therapy, n (%)	80 (40.0%)
Insulin therapy, n (%)	130 (65.0%)
Fasting glucose (mmol/L)	9.2 ± 3.0
Mean postprandial glucose (mmol/L)	11.0 ± 3.4
Mean blood glucose (mmol/L)	10.0 ± 3.1
MAGE (mmol/L)	4.3 ± 2.2
HbA1c (%)	8.6 ± 1.2
Total cholesterol (mmol/L)	5.7 ± 1.4
Triglycerides (mmol/L)	2.2 ± 1.3
eGFR (mL/min/1.73 m²)	92.1 ± 23.8

Table 2 reveals that female abnormalities of QT were more prevalent than the prevalence of male abnormalities. A QTc > 440 ms was found in 57.8 percent of females and 36.4 percent of males and the total prevalence rate was 46 percent. Significant QT prolongation (QTc 500 ms and above) was also significantly greater in females (5.66) than males (0.97), and constituted 3% of the entire population. Equally, a long QT dispersion (QTd > 80 ms) was detected in 5.6% of females compared with 2.7% of males with a general prevalence of 4-percent, and it was evident that there is a sex predisposition to QT abnormalities.

Table 2: Prevalence of Prolonged QTc Duration and QT Dispersion by Sex

QT Abnormality	Male (n = 110)	Female (n = 90)	Total (N = 200)
QTc > 440 ms	40 (36.4%)	52 (57.8%)	92 (46.0%)
QTc ≥ 500 ms	1 (0.9%)	5 (5.6%)	6 (3.0%)
QTd > 80 ms	3 (2.7%)	5 (5.6%)	8 (4.0%)

Table 3 shows that patients with prolonged QTc (≥ 440 ms) had significantly worse clinical and metabolic profiles than those with QTc < 440 ms, including higher age (59.8 vs. 56.9 years, p = 0.03), higher BMI (31.0 vs. 28.9 kg/m², p = 0.01), and markedly higher rates of coronary heart disease (38% vs. 12%, p = 0.001) and retinopathy (64.1% vs. 35.2%, p = 0.001). Poor glycemic control was evident in prolonged QTc cases, with significantly elevated fasting glucose (10.0 vs. 8.6 mmol/L, p = 0.02),

postprandial glucose (12.6 vs. 9.7 mmol/L, $p < 0.001$), mean blood glucose (11.1 vs. 9.1 mmol/L, p = 0.001), MAGE (4.7 vs. 3.9 mmol/L, p = 0.04), HbA1c (9.1% vs. 8.2%, $p < 0.001$), and triglycerides (2.5 vs. 2.0 mmol/L, p = 0.03), while sulphonylurea use was also more frequent (47.8% vs. 33.3%, p = 0.03). For QT dispersion, patients with QTd ≥ 80 ms had significantly more coronary heart disease (75% vs. 22.9%, p = 0.001), were older (63.5 vs. 58.0 years, p = 0.05), and showed lower eGFR (71.2 vs. 92.8 mL/min/1.73 m², p = 0.02), indicating a strong association between abnormal QT metrics and cardiovascular and renal dysfunction.

Table 3: Comparison of Clinical and Metabolic Characteristics According to QTc Duration and QT Dispersion

Variable	QTc < 440 ms (n = 108)	QTc ≥ 440 ms (n = 92)	p-value
QTc Duration Groups			
Age (years)	56.9 ± 8.7	59.8 ± 9.2	0.03
BMI (kg/m²)	28.9 ± 4.9	31.0 ± 5.0	0.01
Coronary heart disease, n (%)	13 (12.0%)	35 (38.0%)	0.001
Retinopathy, n (%)	38 (35.2%)	59 (64.1%)	0.001
Polyneuropathy, n (%)	72 (66.7%)	69 (74.5%)	0.21
Metformin therapy, n (%)	83 (76.9%)	68 (73.9%)	0.6
Sulphonylurea therapy, n (%)	36 (33.3%)	44 (47.8%)	0.03
Insulin therapy, n (%)	74 (68.5%)	56 (60.9%)	0.28
Fasting glucose (mmol/L)	8.6 ± 2.5	10.0 ± 3.2	0.02
Postprandial glucose (mmol/L)	9.7 ± 2.9	12.6 ± 3.6	<0.001
Mean blood glucose (mmol/L)	9.1 ± 2.7	11.1 ± 3.5	0.001
MAGE (mmol/L)	3.9 ± 2.0	4.7 ± 2.4	0.04
HbA1c (%)	8.2 ± 1.0	9.1 ± 1.3	<0.001
Triglycerides (mmol/L)	2.0 ± 1.1	2.5 ± 1.5	0.03
eGFR (mL/min/1.73 m²)	93.4 ± 21.8	90.5 ± 25.7	0.4
Variable	**QTd < 80 ms (n = 192)**	**QTd ≥ 80 ms (n = 8)**	**p-value**
QT Dispersion Groups			
Age (years)	58.0 ± 9.0	63.5 ± 10.1	0.05
Coronary heart disease, n (%)	44 (22.9%)	6 (75.0%)	0.001
Retinopathy, n (%)	91 (47.6%)	4 (50.0%)	0.88
Polyneuropathy, n (%)	134 (69.8%)	7 (87.5%)	0.2
Mean blood glucose (mmol/L)	9.9 ± 3.0	11.4 ± 3.8	0.1
eGFR (mL/min/1.73 m²)	92.8 ± 23.4	71.2 ± 28.8	0.02

Table 4 shows that several clinical variables significantly predict prolonged QTc (≥ 440 ms). Increasing age and BMI were both independent predictors, with each unit rise associated with a 4% and 8% increase in odds, respectively. Coronary heart disease emerged as a strong predictor, tripling the likelihood of QTc prolongation (OR 3.06, p = 0.001), while retinopathy also doubled the risk (OR 2.66, p = 0.002). Use of sulphonylurea therapy was associated with nearly a twofold higher risk (OR 1.95, p = 0.02). Higher HbA1c levels significantly increased the odds (OR 1.55, p = 0.004), indicating poor glycemic control as an important factor. Elevated triglyceride levels also contributed moderately to prolonged QTc (OR 1.34, p = 0.02). Overall, the regression model highlights metabolic, cardiovascular, and diabetes-related factors as key determinants of QTc prolongation.

Table 4: Logistic Regression Analysis for Predictors of Prolonged QTc (≥ 440 ms)

Predictor	β Coefficient	Odds Ratio (95% CI)	p-value
Age (years)	0.04	1.04 (1.01–1.07)	0.01
BMI (kg/m²)	0.08	1.08 (1.02–1.14)	0.008
Coronary heart disease	1.12	3.06 (1.60–5.87)	0.001
Retinopathy	0.98	2.66 (1.47–4.83)	0.002
Sulphonylurea therapy	0.67	1.95 (1.10–3.45)	0.02
HbA1c (%)	0.44	1.55 (1.20–2.16)	0.004
Triglycerides (mmol/L)	0.29	1.34 (1.05–1.75)	0.02

Discussion

The current research shows that the prevalence of QTc prolongation is remarkably high among patients with poorly managed type 2 diabetes, with almost half the sample having QTc values greater than 440 ms and a smaller but still significant percentage of patients having QTc values over 500 ms, the high-risk level. Such results are in line with the large variability of the previous studies with QTc prolongation reported to range between 15% and 67% in diabetic populations (Christensen et al., 2000; Veglio et al., 2002) [8,9]. We are at the far right of this spectrum which probably indicates the poor glycemic control and burdensome complications among our cohort. Though only 2% of our sample had QTc ≥500 ms, in line with Moss et al. (1991) [10] and Priori et al. (2003) [11], this value is strongly associated with the presence of malignant arrhythmias, however, most of our sample is in the gray zone, indicating that they have subclinical electrical instability of the myocardium, not near arrhythmic risk. There were also noted QT dispersion abnormalities although at lesser rates as reported by Cardoso et al. (2001) [12] where prevalence of QTd was found to vary greatly in different definitions and ECG interpretation techniques."

One of the results of our research is the close relationship between hyperglycemia and QTc prolongation, which is consistent with the results of a series of other studies that have also identified that both acute and chronic hyperglycemia negatively changes ventricular repolarization (Fiorentini et al., 2010; Brown et al., 2001; Marfella et al., 2000) [13,14,15] , . The mechanistic basis of hyperglycemia can be described as follows: repolarization could be affected by hyperglycemia via protein kinase C activation and diminished nitric oxide, finally disrupting Na 3 /K + -ATPase and Ca 2 3 -ATPase functions (Tesfamariam et al., 1991; Gupta et al., 1992) [16,17]. These physiological paths were supported by our finding that increased values of both mean glucose and HbA1c levels alone predicted QTc prolongation. This is corroborated by Lefrandt et al. (2000) [18] who demonstrated a positive relationship between fasting glucose and QTc duration in a large non-diabetic cohort of patients and supports the idea that glucose swings are also a cause of electrical instability. Yet, unlike other studies that could not provide a definite association between glycemic status and QT dispersion (Cardoso et al., 2001) [12], our findings indicate that there was a tendency to be more glycemic in participants with a higher dispersion, but the number of affected individuals was small to provide statistical significance.

Our results indicated that QT abnormalities are associated with cardiovascular disease. Patients that had a long QTc and longer QTd demonstrated significantly elevated occurrence rates of heart disease involving the heart, which confirms the previous data that myocardial ischemia is a contributing factor to the effects of repolarization (Veglio et al., 2002; Sakabe et al., 2008) [9,19]. Chronic ischemia can cause heterogeneous repolarization where fibrosis, hypertrophy or autonomic imbalance is induced as explained by Rana et al. (2002) [20]. These mechanisms are supported by our data that indicated that coronary disease is a strong independent predictor of both QTc prolongation and QTd. Additionally, revascularization has proven to result in a decrease in QT prolongation (Mirbolouk et al., 2014) [21] indicating that ischemia has a modifiable impact on these abnormalities. The more frequent occurrence of retinopathy in those with the prolonged QTc in our sample also supports the previous studies of the association between microvascular complications and repolarization disorders (Li et al., 2012) [22], which might be because of a common pathway of endothelial dysfunction.

The more frequent occurrence of long QTc in women in our population is in accord with sex differences that well established in cardiac electrophysiology, whereby the adult women usually have longer QT intervals and are more rate dependent (Merri et al., 1989) [23]. The fact that ion channels may change in response to hormones may also contribute to this pattern, but the exact mechanisms are yet to be fully comprehended. Also, the correlation of the effect of obesity on QT in our study is similar to other previous studies that have observed that the elevation of BMI can alter the autonomic tone, myocardial workload, or electrolyte balance, and ultimately alters ventricular repolarization.

The other interesting fact is that sulphonyl urea therapy is independent of QTc prolongation. This substantiates the mechanistic hypothesis which claims that sulfonylureas block cardiac KATP channels, which lengthens action potentials. In line with our findings is the mini study by Najeed et al. (2002) [24] who found an augmentation of QTc and QTd with glipalamides and not metformin therapy. Even though we are not able to conduct larger studies, our results support the idea that there should be a closer therapeutic approach to high-risk patients, especially those who have other QT-prolonging factors.

However, unlike other previous studies, we have not found a significant association between autonomic neuropathy, which was measured indirectly with polyneuropathy, and QT abnormalities. This is in line with subsequent research that contests the usefulness of QT interval as a diagnostic entity of CAN (Bravenboer et al., 1993) [25] as the possibility that repolarization of abnormalities in diabetes is affected more by metabolic and ischemic elements than by autonomic activity.

In general, the fact that QTc abnormalities were very high in our poorly managed diabetic group and strongly correlated with age, BMI, hyperglycemia, triglycerides, and vascular complications supports the idea that the assessment of the QT interval should be implemented in the standard cardiovascular risk assessment package. Although most patients can be defined as the intermediate-risk QT range, the adverse metabolic and structural factors cluster indicates that QT prolongation could be a useful subclinical predictor of cardiovascular risk in type 2 diabetes.

Conclusion

The research shows that abnormalities in QT interval are widespread in patients with uncontrolled diabetes, and that the rates are higher in women. Long QTc was linked to unfavorable clinical and metabolic characteristics, such as an older age, increased body mass index, increased cardiovascular disease, and microvascular complications, including retinopathy. The patients who had long-term QTc also had worse glycemic measures, increased variability in glucose levels, and atherogenic lipid profiles, which indicated a close association between metabolic instability and cardiac repolarization defects. Logistic regression revealed that age, obesity, coronary heart disease, retinopathy, taking of sulphonylureas, high levels of HbA1c, and high levels of triglycerides are important independent predictors of QTc prolongation, and a combination of classic cardiovascular risk factors and diabetes-related complications make a person vulnerable to electrical cardiac conditions. In general, the results highlight the significance of careful cardiovascular monitoring in diabetes-affected patients with inadequate glycaemic regulation and indicate that enhancing the metabolic regulation and managing the modifiable risk factors can potentially help to decrease the burden of the QT-related arrhythmic risk among the population in question.

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