

QTc Interval Prolongation and Its Association with Albuminuria Severity in Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Study

Sahil¹, Jasmine Kaur², Raman Kumar Sharma^{3*}, Aneesha Chibber⁴, Akash Bawa⁵, Prabhjot Kaur Gill⁶

¹⁻⁵Department of Medicine, SGRDIMSRI, Sri Guru Ram Das University of Health Sciences, Sri Amritsar, Punjab, India

⁶Centre for Advanced Research and Development, SGRDIMSRI, Sri Guru Ram Das University of Health Sciences, Sri Amritsar, Punjab, India

ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is associated with multiple microvascular complications, including diabetic nephropathy and cardiac autonomic dysfunction. Prolongation of the corrected QT (QTc) interval has emerged as a potential marker of cardiovascular risk, while albuminuria reflects early renal involvement. This study aimed to evaluate the association between QTc interval prolongation and albuminuria severity in patients with T2DM.

Methods: A cross-sectional observational study was conducted among 100 patients with T2DM attending a tertiary care hospital. Clinical evaluation, laboratory investigations, urine albumin-to-creatinine ratio (UACR), and 12-lead electrocardiography were performed. Participants were categorized according to diabetes duration and UACR levels. Statistical analysis included independent Student's t-test and one-way ANOVA, with $p < 0.05$ considered statistically significant.

Results: The mean age of the participants was 60.12 ± 12.14 years, and 62% were females. Diabetic neuropathy and retinopathy were present in 66% and 63% of patients, respectively. Patients with diabetes duration ≥ 5 years had significantly higher UACR (141.28 ± 75.40 vs. 100.33 ± 64.41 mg/g; $p = 0.004$), QT interval (384.22 ± 44.66 vs. 349.96 ± 43.58 ms; $p = 0.019$), and QTc interval (489.29 ± 51.40 vs. 454.98 ± 50.51 ms; $p = 0.024$) compared to those with shorter disease duration. A significant increase in HbA1c ($p = 0.005$) and QTc interval ($p = 0.013$) was observed across increasing UACR categories, indicating a dose-dependent relationship between albuminuria severity and cardiac repolarization abnormalities.

Conclusion: QTc interval prolongation is significantly associated with increasing albuminuria severity, poor glycemic control, and longer diabetes duration. QTc assessment may serve as a simple, non-invasive marker for identifying T2DM patients at increased risk of microvascular and cardiovascular complications.

Keywords: QTc Interval, Albuminuria, Diabetes Mellitus Type 2, Cardiac Autonomic Neuropathy, Diabetic Nephropathy, Cardiovascular Risk

DOI:

10.55489/njmr.160420261369

*Corresponding author:

Dr. Raman Kumar Sharma

(Email: ankuramba@yahoo.co.in)

Date of Submission: 03/05/2026

Date of Acceptance: 07/07/2026

Date of Publication: 01/10/2026

Funding Support:

None Declared

Conflict of Interest:

The authors have declared that no conflict of interest exists.

How to cite this article:

Sahil, Kaur J, Sharma RK, Chibber A, Bawa A, Gill PK. QTc Interval Prolongation and Its Association with Albuminuria Severity in Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Study. Natl J Med Res 2026;16(04):224-229. DOI: 10.55489/njmr.160420261369

Copy Right: The Authors retain the copyrights of this article, with first publication rights granted to Medsci Publications.

License Term: Creative Commons Attribution-Share Alike (CC BY-SA) 4.0

Publisher: Medsci Publications [www.medscipublications.com]

ISSN: 2249 4995

Official website: www.njmr.in

INTRODUCTION

Diabetes mellitus has emerged as one of the most important global public health challenges of the twenty-first century. According to the International Diabetes Federation (IDF), approximately 537 million adults were living with diabetes worldwide in 2021, and this number is projected to increase to 643 million by 2030 and 783 million by 2045. India contributes substantially to this burden, with more than 101 million individuals estimated to have diabetes. The rapidly increasing prevalence of Type 2 Diabetes Mellitus (T2DM) has resulted in a parallel rise in chronic vascular complications, including nephropathy, neuropathy, retinopathy, and cardiovascular disease, which remain major causes of morbidity and mortality. [1]

Diabetic neuropathy is one of the most common chronic complications of diabetes and results from a complex interplay of hyperglycemia-induced metabolic disturbances, oxidative stress, microvascular insufficiency, inflammation, and neuronal damage, ultimately leading to progressive nerve dysfunction and disability.[2] Albuminuria is a well-established marker of diabetic kidney disease and is classified according to urinary albumin excretion, with moderately increased albuminuria (formerly termed microalbuminuria) defined as a urinary albumin excretion of 30-300 mg/day or a urine albumin-to-creatinine ratio (UACR) of 30-300 mg/g creatinine. [3-4] The clinical significance of moderately increased albuminuria was first recognized through pioneering studies by Parving and colleagues, who demonstrated that elevated urinary albumin excretion serves as an early marker for the development and progression of diabetic nephropathy. [5] Prospective studies have since established it as an independent risk factor for both end-stage renal disease and cardiovascular mortality in T2DM.[4]

Cardiovascular involvement in patients with diabetes can be assessed using electrocardiographic parameters, particularly the corrected QT (QTc) interval, which reflects ventricular depolarization and repolarization. QTc prolongation has been shown to be significantly associated with diabetic neuropathy, retinopathy, and nephropathy, with progressively longer QTc intervals observed as the severity and number of microvascular complications increase, highlighting QTc as a potential indicator of the burden of diabetic microvascular complications.[6] Moreover, prolonged QTc is clinically important because it predisposes patients to life-threatening ventricular arrhythmias, including torsades de pointes and ventricular fibrillation, and is recognized as a predictor of sudden cardiac death. Early identification of QTc prolongation may facilitate timely cardiovascular risk assessment and appropriate clinical management to reduce the risk of adverse cardiac outcomes. [7]. Cardiovascular complications are a major cause of morbidity and mortality in patients with Type 2 Diabetes Mellitus. Previous studies have demonstrated a significant association between QTc interval prolongation and microalbuminuria, suggesting that QTc assessment may serve as a simple,

non-invasive marker of diabetic microvascular involvement. [8] Furthermore, elevated blood glucose levels have been shown to be independently associated with QTc interval prolongation, highlighting the potential contribution of hyperglycemia to cardiac electrophysiological abnormalities in patients with diabetes. [9] Cardiac autonomic neuropathy is frequently accompanied by abnormalities in ventricular repolarization, manifested clinically by prolongation of the corrected QT interval. Since albuminuria and autonomic dysfunction are both considered markers of generalized microvascular injury, evaluation of QTc prolongation may provide additional insight into the burden of diabetic vascular complications. Understanding this relationship could facilitate earlier identification of high-risk individuals using a simple and widely available electrocardiographic marker [10]. Given the shared vascular and metabolic pathways of renal and cardiac damage, this study aims to investigate the specific association between the QTc interval and microalbuminuria in patients with Type 2 diabetes.

MATERIALS AND METHODS

Study Design and Setting: The present cross-sectional observational study was conducted within the Department of General Medicine at the tertiary care hospital. The study period spanned from July 2024 to December 2025. Participants were recruited from both the Outpatient Department (OPD) and Inpatient Department (IPD) to ensure a diverse clinical representation after obtaining approval from the Institutional Ethics Committee (SGRD/IEC/2024-362).

Sample Size: The minimum sample size was estimated using the formula $n = Z^2 P(1-P)/d^2$ for cross-sectional studies. Assuming a prevalence of QTc prolongation among patients with Type 2 Diabetes Mellitus of 44.1% [11], a 95% confidence level ($Z=1.96$), and an absolute precision of 10%, the calculated sample size was 96 participants. To compensate for possible incomplete data, 100 participants were ultimately enrolled.

Inclusion Criteria: Participants were included if cases met the American Diabetes Association (ADA) 2022 [12] diagnostic standards, characterized by glycated hemoglobin (HbA1C) $\geq 6.5\%$, Fasting Plasma Glucose (FPG) ≥ 126 mg/dL (7.0 mmol/L), 2-hour post-prandial glucose ≥ 200 mg/dL (11.1 mmol/L) during an Oral Glucose Tolerance Test (OGTT) and random plasma glucose ≥ 200 mg/dL (11.1 mmol/L) in symptomatic patients.

Exclusion Criteria: To minimize confounding factors affecting cardiac and renal assessments, patients with cardiac arrhythmias (including atrial fibrillation) or permanent pacemaker implantation; pre-existing ischemic heart disease or cerebrovascular accident within the preceding 3 months; electrolyte abnormalities (including hypo/hyperkalemia, hypo/hypercalcemia, and hypo/hypermagnesemia); and current use of medications known to affect QT interval duration, such as anti-arrhythmics, antipsychotics, antidepressants, fluoro-

quinolones, antifungal agents, digoxin, or beta-blockers, were excluded from the study.

Data Collection Procedures: Prior to data acquisition, informed written consent was obtained from each participant in their native language (Hindi or Punjabi). The protocol was executed through the following stages:

Clinical and Physical Evaluation: Comprehensive history taking and systemic examinations were performed.

Cardiac Assessment: QT intervals were measured from standard 12-lead electrocardiograms and corrected for heart rate using Bazett's formula ($QT_c = QT/\sqrt{RR}$). A QT_c value >450 ms in men and >470 ms in women was considered prolonged, in accordance with AHA/ACCF/HRS recommendations for ECG interpretation and QT interval assessment.[13]

Biochemical Analysis: Venous blood samples were collected for glycemic profiles and laboratory investigations.

Renal Screening: Urine Albumin-to-Creatinine Ratio (UACR) was measured using a spot urine sample collected under standardized conditions. Albuminuria was defined according to the KDIGO Clinical Practice Guideline as a UACR >30 mg/g. For analytical purposes, participants were further stratified into investigator-defined categories (<100 mg/g, 101-200 mg/g, and >200 mg/g) to evaluate the relationship between increasing albuminuria severity and QT_c interval.[4] These categories were used solely for statistical analysis and do not represent the official KDIGO albuminuria classification.

Statistical Analysis: Statistical analyses were performed in accordance with the STROBE guidelines for cross-sectional studies. Data were entered into Microsoft Excel and analysed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean $\pm$ standard deviation, whereas categorical variables were expressed as frequencies and percentages. Differences between two groups were evaluated using the Independent Student's *t*-test, while comparisons across multiple groups were assessed using one-way analysis of variance (ANOVA), followed by Tukey's post-hoc test where appropriate. Statistical significance was determined using a two-tailed *p* value of <0.05 .

RESULTS

Table 1 presents the demographic profile and clinical complications of the study cohort ($n=100$). The mean age of the participants was 60.12 ± 12.14 years, indicating a predominantly elderly population. The highest proportion of participants was observed in the 61-70 years bracket (27.0%), followed by the 51-60- and 71-80-years groups, both accounting for 23.0% and 21.0%, respectively. Collectively, 71.0% of the subjects were aged 51 years or older, aligning with the progressive nature of Type 2 Diabetes Mellitus.

In terms of gender distribution, a female preponderance was noted, with women comprising 62.0% of the sample

Table 1: Demographic and Clinical Characteristics of the Study Cohort (n = 100)

Characteristics	Frequency (%)
Age (years)	
≤ 40	6 (6.0)
41-50	21 (21.0)
51-60	23 (23.0)
61-70	27 (27.0)
71-80	21 (21.0)
>80	2 (2.0)
Mean $\pm$ SD	60.12 ± 12.14
Gender	
Female	62 (62.0)
Male	38 (38.0)
Complications	
Diabetic Retinopathy	
Absent	37 (37.0)
Present	63 (63.0)
Diabetic Neuropathy	
Absent	34 (34.0)
Present	66 (66.0)

compared to 38.0% males. The analysis of microvascular complications revealed a high disease burden; diabetic neuropathy was the most prevalent complication, affecting 66.0% of the participants. Similarly, diabetic retinopathy was identified in 63.0% of the cohort. These findings underscore a significant prevalence of end-organ damage among the diabetic patients, likely reflecting chronic duration and metabolic severity of their condition.

Table 2 demonstrates a significant relationship between the duration of diabetes, severity of albuminuria, glycemic control, and cardiac repolarization abnormalities. Patients with diabetes duration of five years or longer exhibited significantly higher UACR levels and prolonged QT and QT_c intervals, indicating progressive renal and cardiovascular involvement with advancing disease duration. Furthermore, increasing albuminuria was associated with worsening glycemic control and longer QT_c intervals, suggesting a graded relationship between nephropathy and autonomic cardiac dysfunction. One-way ANOVA confirmed significant differences in HbA1c and QT_c values across UACR categories. Post-hoc analysis further revealed that patients with severe albuminuria (>200 mg/g) had significantly greater QT_c prolongation compared with those having lower albuminuria levels, highlighting the close interplay between renal injury and cardiac autonomic impairment in Type 2 DM.

Table 3 highlights significant associations between diabetes duration, albuminuria, glycemic control, and cardiac repolarization. Longer disease duration was associated with higher UACR levels and prolonged QT/ QT_c intervals. Increasing albuminuria was also accompanied by poorer glycemic control and greater QT_c prolongation, suggesting a close relationship between nephropathy and cardiac autonomic dysfunction in patients with Type 2 Diabetes Mellitus.

Table 2: Association Between Diabetes Duration, Albuminuria Severity, Glycemic Control, and QTc Interval in Patients with Type 2 Diabetes Mellitus**Table 2a. Comparison of Renal and Cardiac Parameters According to Duration of Type 2 Diabetes Mellitus**

Variable	<5 Years (n=49) (Mean ± SD)	≥5 Years (n=51) (Mean ± SD)	Mean Difference	p-value
UACR (mg/g)	100.33 ± 64.41	141.28 ± 75.40	40.95	0.004*
QT Interval (ms)	349.96 ± 43.58	384.22 ± 44.66	34.26	0.019*
QTc Interval (ms)	454.98 ± 50.51	489.29 ± 51.40	34.31	0.024*

Statistical Test: Independent Student's t-test & * Statistically significant at p <0.05

Table 2b. Association of HbA1c and QTc Interval Across UACR Categories

UACR Category (mg/g)	N	HbA1c (%) Mean ± SD	QTc (ms) Mean ± SD
<100	50	7.97 ± 2.10	451.59 ± 53.63
101-200	33	9.33 ± 1.93	465.74 ± 44.38
>200	17	9.43 ± 2.39	491.97 ± 54.42
Overall Mean	100	8.66 ± 2.19	471.99 ± 51.20
ANOVA p-value		0.005*	0.013*

Statistical Test: One-way ANOVA & *Statistically significant at p <0.05

Table 2c. Post-hoc Analysis of QTc Interval Across UACR Categories (Tukey HSD)

Comparison	Mean Difference (ms)	p-value	Interpretation
<100 vs 101-200 mg/g	14.15	0.052	Not Significant
<100 vs >200 mg/g	40.38	0.020*	Significant
101-200 vs >200 mg/g	26.23	0.118	Not Significant

Tukey HSD post-hoc test & *Statistically significant at p <0.05

Table 3: Summary of Significant Clinical Associations

Clinical Association	Statistical Test	p-value
Diabetes Duration vs UACR	Independent t-test	0.004*
Diabetes Duration vs QT Interval	Independent t-test	0.019*
Diabetes Duration vs QTc Interval	Independent t-test	0.024*
UACR Category vs HbA1c	One-way ANOVA	0.005*
UACR Category vs QTc Interval	One-way ANOVA	0.013*

* Statistically significant at p <0.05

Table 4: Clinical Interpretation of Albuminuria Severity and QTc Prolongation

Parameter	Lowest UACR Category (<100 mg/g)	Highest UACR Category (>200 mg/g)	Change
HbA1c (%)	7.97 ± 2.10	9.43 ± 2.39	+18.3%
QTc Interval (ms)	451.59 ± 53.63	491.97 ± 54.42	+40.38 ms

Table 4 demonstrates a progressive increase in both HbA1c and QTc interval with worsening albuminuria severity. Patients in the highest UACR category (>200 mg/g) had poorer glycemic control and markedly prolonged QTc intervals compared with those in the lowest UACR category (<100 mg/g). These findings suggest that increasing renal involvement in Type 2 Diabetes Mellitus is accompanied by worsening metabolic control and greater cardiac autonomic dysfunction.

DISCUSSION

The study population had a mean age of 60.12±12.14 years, with the majority of participants belonging to older age groups. This observation is consistent with the

natural history of T2DM, in which prolonged exposure to hyperglycemia and age-related vascular changes contribute to the development of chronic complications. Similar findings have been reported by Sawartha S et al. [14], observed a higher burden of diabetic complications among older individuals with long-standing diabetes.

An important finding of the study was the influence of diabetes duration on both renal and cardiac parameters. Patients with diabetes duration of five years or more demonstrated significantly higher UACR levels along with prolonged QT and QTc intervals compared to those with shorter disease duration. These observations indicate that chronic metabolic stress contributes not only to progressive renal injury but also to alterations in cardiac autonomic regulation and ventricular repolarization. The significant associations summarized in Table 3 further support the cumulative impact of disease duration on the development of microvascular and cardiovascular complications.

Poor glycemic control emerged as another important determinant of disease severity. HbA1c levels increased significantly with worsening albuminuria (p=0.005), highlighting the role of chronic hyperglycemia in the pathogenesis of diabetic nephropathy. Furthermore, patients in the highest UACR category demonstrated both poorer glycemic control and longer QTc intervals, suggesting a close interrelationship between metabolic dysregulation, renal damage, and cardiac autonomic dysfunction. These findings are in agreement with Burkett E et al. [9], who identified hyperglycemia as an important contributor to QTc prolongation, and Stern K et al. [15], who reported a positive association between HbA1c levels and QTc abnormalities in diabetic patients.

The high prevalence of diabetic retinopathy (63%) and neuropathy (66%) observed in the present study underscores the substantial burden of microvascular disease within the study cohort. The coexistence of multiple diabetic complications suggests widespread endothelial dysfunction and microvascular injury. Similar observations have been reported in previous studies, supporting the concept that nephropathy, neuropathy, retinopathy, and cardiac autonomic dysfunction frequently occur together as manifestations of a common pathogenic process.

The observed dose-response relationship between albuminuria severity and QTc prolongation strengthens the biological plausibility of the association. Mean QTc values increased from 451.59 ± 53.63 ms in patients with UACR <100 mg/g to 491.97 ± 54.42 ms in those with UACR >200 mg/g. Table 4 further demonstrates that worsening albuminuria was accompanied by both higher HbA1c levels and greater QTc prolongation. These findings are consistent with those reported by Rutter MK et al. [16], who demonstrated significantly prolonged QTc intervals among patients with microalbuminuria, and by Psallas M et al. [17], who identified albuminuria as a strong predictor of QT dispersion in T2DM. Similarly, Hashimoto Y et al. [18] reported that QTc interval prolongation may predict progression of albuminuria and adverse renal outcomes. The magnitude of QTc prolongation observed in the present study was comparable to previous Indian studies, strengthening the external validity of the findings.

Several mechanisms may explain the relationship between albuminuria and QTc prolongation. Chronic hyperglycemia promotes oxidative stress, endothelial dysfunction, autonomic imbalance, and activation of the renin-angiotensin-aldosterone system, all of which contribute to both renal injury and abnormalities in myocardial repolarization. Consequently, QTc prolongation may represent a readily available electrocardiographic marker reflecting the overall burden of microvascular disease rather than an isolated cardiac abnormality.

The findings of the present study support the utility of QTc interval assessment as a simple, inexpensive, and non-invasive tool for identifying diabetic patients at increased risk of nephropathy and other microvascular complications. Routine ECG evaluation may therefore complement conventional renal screening and facilitate earlier recognition of high-risk individuals who may benefit from intensified metabolic and cardiovascular risk reduction strategies.

Future studies should employ prospective longitudinal designs to determine whether QTc prolongation independently predicts progression of albuminuria and cardiovascular events. Larger multicenter studies involving diverse diabetic populations are also warranted to validate the observed associations and establish clinically meaningful QTc thresholds for risk stratification. Integration of QTc assessment into routine diabetic screening

protocols should be explored through dedicated outcome-based investigations.

LIMITATIONS

This study has certain limitations that should be considered while interpreting the findings. The cross-sectional design limits the ability to establish temporal or causal relationships between albuminuria and QTc prolongation. Recruitment from a single tertiary care center using purposive sampling may have introduced selection bias and restricted the generalizability of the results. The predominance of patients with established microvascular complications reflects a population with advanced disease and may not represent the broader diabetic community. In addition, sex-specific analyses of QTc interval could not be undertaken despite the higher proportion of female participants. Finally, the relatively small sample size and lack of longitudinal follow-up precluded evaluation of the prognostic significance of QTc prolongation for future renal and cardiovascular outcomes.

CONCLUSION

This study confirms a significant association between QTc interval prolongation and albuminuria severity in Type 2 diabetes mellitus. A progressive increase in QTc duration was observed across worsening UACR categories ($p=0.013$), suggesting a link between cardiac repolarization and early nephropathy. The duration of diabetes (≥ 5 years) and poor glycemic control ($p=0.005$) were identified as primary drivers for both elevated UACR and prolonged QTc intervals. These findings suggest that QTc prolongation may serve as a valuable clinical marker for identifying patients at high risk for concurrent microvascular and autonomic complications, necessitating integrated cardiac and renal screening strategies.

Individual Author's Contribution: SA was responsible for study design, data collection, statistical analysis, and the initial drafting of the manuscript. JK and RKS contributed to the study conception, design, and data acquisition. AC and AB performed the literature search and assisted in the compilation of the manuscript. PKG provided expertise in study design, supervised data analysis, and contributed to manuscript preparation, final submission and revision.

Availability of data: The data that support the findings of this study are available from the corresponding author on reasonable request.

Declaration of Non-use of generative AI Tools: All scientific content, study design, data collection, statistical analyses, interpretation of findings, and final manuscript preparation were conducted independently by the authors. The authors take full responsibility for the accuracy, integrity, and originality of the work.

REFERENCES

1. Sun H, Saeedi P, Karuranga S, Pinkepank M, Ogurtsova K, Duncan BB, et al. IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. *Diabetes Res Clin Pract.* 2022;183:109119. DOI: <https://doi.org/10.1016/j.diabres.2021.109119>. Erratum in: *Diabetes Res Clin Pract.* 2023 Oct;204:110945. DOI: <https://doi.org/10.1016/j.diabres.2023.110945>. PMID: 34879977 PMCID: PMC11057359
2. Feldman EL, Nave KA, Jensen TS, Bennett DL. New horizons in diabetic neuropathy: mechanisms, bioenergetics, and pain. *Neuron.* 2017;93(6):1296-1313. DOI: <https://doi.org/10.1016/j.neuron.2017.02.005> PMid:28334605 PMCID:PMC5400015
3. Mogensen CE. Microalbuminuria predicts clinical proteinuria and early mortality in maturity-onset diabetes. *New England Journal of Medicine.* 1984;310(6):356-360. DOI: <https://doi.org/10.1056/NEJM198402093100605> PMid:6690964
4. Stevens PE, Levin A; Kidney Disease: Improving Global Outcomes Chronic Kidney Disease Guideline Development Work Group Members. Evaluation and management of chronic kidney disease: synopsis of the kidney disease: improving global outcomes 2012 clinical practice guideline. *Ann Intern Med.* 2013;158(11):825-830. DOI: <https://doi.org/10.7326/0003-4819-158-11-201306040-00007> PMid:23732715
5. Parving HH, Oxenbøll B, Svendsen PA, Christiansen JS, Andersen AR. Early detection of patients at risk of developing diabetic nephropathy. A longitudinal study of urinary albumin excretion. *Acta Endocrinol (Copenh).* 1982;100(4):550-555. DOI: <https://doi.org/10.1530/acta.0.1000550> PMid:6812342
6. Kobayashi S, Nagao M, Asai A, Fukuda I, Oikawa S, Sugihara H. Severity and multiplicity of microvascular complications are associated with QT interval prolongation in patients with type 2 diabetes. *J Diabetes Investig.* 2018;9(4):946-951. DOI: <https://doi.org/10.1111/jdi.12772> PMid:29095573 PMCID:PMC6031516
7. Schwartz PJ, Ackerman MJ. The long QT syndrome: a transatlantic clinical approach to diagnosis and therapy. *Eur Heart J.* 2013;34(40):3109-3116. DOI: <https://doi.org/10.1093/eurheartj/ehd089> PMid:23509228
8. Dominic SK, Henry RA, Kartha N, Pillai G. The Association Between Microalbuminuria and QTc Prolongation in Patients with Type 2 Diabetes Mellitus: A Single-Centre Study from South India. *Cureus.* 2023;15(3):e35646. DOI: <https://doi.org/10.7759/cureus.35646> PMid:37009348 PMCID:PMC10065364
9. Burkett E, Keijzers G, Lind J. The relationship between blood glucose level and QTc duration in the critically ill. *Crit Care Resusc.* 2009;11(1):8-13. DOI: [https://doi.org/10.1016/S1441-2772\(23\)01839-2](https://doi.org/10.1016/S1441-2772(23)01839-2) PMid:19281438
10. Mehta N, Kandulna AK, Kantibhai SS, Dhol SB, Kapadiya SB, Gupta M. The evaluation of QTc prolongation and QT dispersion in type 2 diabetes mellitus as an indicator of cardiac autonomic neuropathy. *European Journal of Cardiovascular Medicine.* 2025;15:448-451.
11. Ninkovic VM, Ninkovic SM, Miloradovic V, Stanojevic D, Babic M, Giga V, et al. Prevalence and risk factors for prolonged QT interval and QT dispersion in patients with type 2 diabetes. *Acta Diabetol.* 2016;53(5):737-744. DOI: <https://doi.org/10.1007/s00592-016-0864-y> PMid:27107571 PMCID:PMC5014905
12. American Diabetes Association Professional Practice Committee. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2022. *Diabetes Care.* 2022 Jan 1;45(Suppl 1):S17-S38. DOI: <https://doi.org/10.2337/dc22-S002> PMid:34964875
13. Rautaharju PM, Surawicz B, Gettes LS, Bailey JJ, Childers R, Deal BJ, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. *J Am Coll Cardiol.* 2009;53(11):982-991. DOI: <https://doi.org/10.1016/j.jacc.2008.12.014> PMid:19281931
14. Sawartha S, Patel R, Patil PP. A Cross-Sectional Study to Determine the Association of Corrected QT Interval with Microalbuminuria in Type 2 Diabetes Mellitus. *Cureus.* 2023;15(5):e38967. DOI: <https://doi.org/10.7759/cureus.38967> PMid: 37313082 PMCID: PMC10260269
15. Stern K, Cho YH, Benitez-Aguirre P, Jenkins AJ, McGill M, Mitchell P, et al. QT interval, corrected for heart rate, is associated with HbA1c concentration and autonomic function in diabetes. *Diabet Med.* 2016;33(10):1415-1421. DOI: <https://doi.org/10.1111/dme.13085> PMid:26823095
16. Rutter MK, Viswanath S, McComb JM, Kesteven P, Marshall SM. QT prolongation in patients with Type 2 diabetes and microalbuminuria. *Clin Auton Res.* 2002;12(5):366-372. DOI: <https://doi.org/10.1007/s10286-002-0036-8> PMid:12420081
17. Psallas M, Tentolouris N, Papadogiannis D, Doulgerakis D, Kokkinos A, Cokkinos DV, et al. QT dispersion: comparison between participants with Type 1 and 2 diabetes and association with microalbuminuria in diabetes. *J Diabetes Complications.* 2006;20(2):88-97. DOI: <https://doi.org/10.1016/j.jdiacomp.2005.05.012> PMid:16504837
18. Hashimoto Y, Tanaka M, Senmaru T, Okada H, Hamaguchi M, Asano M, et al. Heart rate-corrected QT interval is a novel risk marker for the progression of albuminuria in people with Type 2 diabetes. *Diabet Med.* 2015;32(9):1221-1226. DOI: <https://doi.org/10.1111/dme.12728> PMid:25683576