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Original Research Article

ASSOCIATION OF QTC INTERVAL PROLONGATION WITH MICROALBUMINURIA IN TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is associated with wide-ranging microvascular and macrovascular complications. Microalbuminuria is an early marker of diabetic nephropathy and generalised endothelial dysfunction, while prolongation of the corrected QT (QTc) interval reflects cardiac autonomic neuropathy and predisposes to arrhythmic risk. The relationship between the two remains incompletely defined in Indian tertiary-care settings. **Objectives:** To evaluate the association between QTc interval prolongation and microalbuminuria in patients with T2DM, and to determine the relationship of QTc interval with duration of diabetes and other clinical variables.

Materials and Methods: This observational comparative study enrolled 62 patients with T2DM, divided equally into a microalbuminuria group (n=31) and a normoalbuminuria group (n=31). Clinical evaluation, HbA1c, urinary albumin-to-creatinine ratio, and electrocardiographic QTc (Bazett's formula) were assessed. Independent t-test, chi-square test, Pearson's correlation and multiple linear regression were used; $p<0.05$ was considered significant.

Results: Patients with microalbuminuria were significantly older (63.90±15.27 vs 52.35±10.78 years, p=0.001) and had longer diabetes duration (10.06±6.03 vs 7.19±2.85 years, p=0.019). Mean QTc was significantly prolonged in the microalbuminuria group (444.77±9.15 vs 419.42±7.86 ms, $p<0.001$), and QTc prolongation (>440 ms) occurred in 61.3% versus 0.0% of normoalbuminuric patients ($\chi^2=24.59$, $p<0.001$). QTc correlated significantly with diabetes duration, HbA1c, microalbumin-to-creatinine ratio, and serum creatinine, and on multivariate regression the microalbumin-to-creatinine ratio was the sole independent predictor of QTc interval (p=0.010).

Conclusion: Microalbuminuria is strongly and independently associated with QTc prolongation in T2DM. Given their simplicity and low cost, combined assessment of urinary microalbumin and QTc interval may aid early cardiovascular risk stratification in diabetic patients.

Keywords: QTc interval; Microalbuminuria; Type 2 diabetes mellitus; Cardiac autonomic neuropathy; Diabetic nephropathy.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is among the most rapidly growing global health challenges, driven by urbanisation, sedentary lifestyles and an ageing population, and is a major cause of chronic morbidity and mortality worldwide1. Diabetic nephropathy is one of its most consequential microvascular complications2, and microalbuminuria — a modest but abnormal increase in urinary albumin excretion — is recognised as the earliest clinical marker of renal involvement, often preceding overt proteinuria by years3. Beyond its renal implications,

microalbuminuria is closely linked with generalised endothelial dysfunction and increased cardiovascular risk, including autonomic neuropathy, arrhythmias and sudden cardiac death4. Cardiac autonomic neuropathy (CAN) is an underrecognised but serious complication of T2DM arising from damage to autonomic fibres innervating the heart and vasculature5. Among electrocardiographic markers of CAN, prolongation of the corrected QT interval (QTc) has attracted particular attention because it is easy to measure, reproducible, and a strong predictor of arrhythmogenic risk; QTc prolongation reflects delayed ventricular repolarisation and predisposes to malignant ventricular arrhythmias, including torsades de pointes, and is associated with increased cardiovascular mortality6. Several studies have reported a high prevalence of QTc prolongation in T2DM, particularly among patients with poor glycaemic control, longer disease duration and microvascular complications, suggesting a shared pathophysiological pathway linking nephropathy and cardiac autonomic dysfunction7.

Both microalbuminuria and QTc prolongation emerge early in the natural history of diabetes and share common mechanistic underpinnings — chronic hyperglycaemia, oxidative stress, advanced glycation end-product accumulation, and endothelial dysfunction — raising the possibility that the two abnormalities are interrelated manifestations of a common systemic microvascular process7,8. Microalbuminuria has also been associated with structural and functional myocardial alterations, including impaired diastolic function, which may further influence ventricular repolarisation9. If confirmed, this relationship would allow QTc assessment, using a simple and inexpensive electrocardiogram, to serve as a practical adjunct to urine albumin screening for early cardiovascular risk stratification in diabetic patients, well before overt cardiac disease develops10.

Previous literature on this association has been inconsistent, with study-to-study variation in patient selection, sample size, disease duration and QTc measurement methodology11. Some cohorts have shown a clear, graded relationship between the degree of albuminuria and QTc prolongation, while others have found the association attenuated after adjustment for glycaemic control, blood pressure or disease duration, leaving the independent contribution of nephropathy itself uncertain. Data specific to Indian tertiary-care settings, where the burden of both diabetic nephropathy and cardiovascular disease is substantial and routine cardiac autonomic testing is rarely feasible, remain particularly limited.

A simple, reproducible marker capable of flagging diabetic patients at higher cardiovascular risk at the point of routine nephropathy screening would have considerable practical value, since it would require no additional infrastructure beyond an electrocardiogram machine already available in most outpatient diabetic clinics. Clarifying whether microalbuminuria independently predicts QTc prolongation, or whether the relationship is largely explained by shared risk factors such as age, glycaemic control or disease duration, is therefore of direct clinical relevance to routine diabetic care.

The present study was undertaken to evaluate the association between QTc interval prolongation and microalbuminuria in patients with T2DM attending a tertiary-care hospital in South India, and to determine the relationship of QTc interval with duration of diabetes and other clinical and biochemical variables, including its independent predictors on multivariate analysis.

MATERIALS AND METHODS

Study Design and Setting

This observational, comparative study was conducted in the Department of General Medicine and Endocrinology, Vydehi Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka, a tertiary-care teaching hospital, over one and a half years (January 2024 to June 2025). The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

Participants

Adult patients (≥18 years) with a confirmed diagnosis of T2DM as per American Diabetes Association criteria were screened from outpatient and inpatient departments; both newly and previously diagnosed diabetics, irrespective of treatment type, were eligible. Patients were excluded if they had ischaemic heart disease, congestive cardiac failure, left bundle branch block, atrial fibrillation or other significant arrhythmias, uncontrolled hypertension, hypocalcaemia, urinary tract infection or non-diabetic renal pathology, or were receiving drugs known to prolong the QT interval (anti-arrhythmics, selected antipsychotics, tricyclic antidepressants, digoxin, ketoconazole, fluoroquinolones) or ACE inhibitors/angiotensin receptor blockers.

Sample Size and Grouping

The sample size was calculated from previously reported proportions of QTc prolongation (67.76% in microalbuminuric and 24.13% in normoalbuminuric diabetics)8, with $\alpha=0.05$ and 90% power, yielding a minimum of 28 patients per group; this was rounded up to 31 per group (62 total) after allowing for a 10% non-response margin. Participants were assigned by simple random sampling into two groups based on urinary albumin excretion: Group A, T2DM with microalbuminuria (urinary albumin 30–300 mg/day, n=31), and Group B, T2DM without microalbuminuria (<30 mg/day, n=31). The two groups were matched as far as possible for age, sex and diabetes duration.

Clinical and Biochemical Evaluation

A structured proforma recorded demographic details, diabetes duration, blood pressure and relevant history. Biochemical assessment included HbA1c, spot urinary microalbumin-to-creatinine ratio, serum creatinine, serum calcium, and urine routine microscopy (to exclude urinary tract infection as a confounder). A resting 12-lead electrocardiogram was recorded for all participants, and the QT interval was corrected for heart rate using Bazett's formula ($QTc = QT/\sqrt{RR}$); QTc >440 ms was defined as prolonged.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation and compared using the independent-samples t-test; categorical variables were expressed as frequencies and percentages and compared using the chi-square test. Pearson's correlation coefficient was used to assess the relationship between QTc interval and continuous clinical/biochemical variables, and multiple linear regression was performed to identify independent predictors of QTc interval. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Sixty-two patients with T2DM were studied, 31 with microalbuminuria (Group A) and 31 without (Group B). Baseline and comparative clinical characteristics are summarised in Table 1.

Patients in the microalbuminuria group were significantly older (63.90±15.27 vs 52.35±10.78 years; p=0.001), and a greater proportion were aged ≥60 years (67.7% vs 25.8%; χ^2=11.01, p=0.004). Males predominated in the microalbuminuria group (71.0% vs 48.4%), although this difference did not reach significance (p=0.070). Duration of diabetes was significantly longer in the microalbuminuria group (10.06±6.03 vs 7.19±2.85 years; p=0.019), and a disease duration ≥10 years was more common in this group (58.1% vs 22.6%; p=0.005). Systolic and diastolic blood pressure did not differ significantly between groups (p=0.843 and p=0.087, respectively). Mean HbA1c was significantly higher in the microalbuminuria group (8.09±1.35% vs 7.33±0.54%; p=0.005). Serum creatinine (1.23±0.91 vs 0.84±0.31 mg/dL; p=0.026) and, as expected, the microalbumin-to-creatinine ratio (185.32±231.86 vs 18.08±6.03 mg/g; p<0.001) were significantly higher in the microalbuminuria group. Serum calcium and RR interval did not differ significantly between groups, and urinary leukocytes were absent in all participants, excluding urinary tract infection as a confounder.

The mean QTc interval was markedly and significantly prolonged in the microalbuminuria group compared with the normoalbuminuria group (444.77±9.15 ms vs 419.42±7.86 ms; p<0.001). On categorical analysis (Table 2), QTc prolongation (>440 ms) was present in 61.3% of patients with microalbuminuria compared with none (0.0%) of normoalbuminuric patients, a highly significant association (χ^2=24.59, p<0.001).

On Pearson correlation analysis (Table 3), QTc interval correlated significantly and positively with duration of diabetes (r=0.349, p=0.005), HbA1c (r=0.321, p=0.011), microalbumin-to-creatinine ratio (r=0.432, p<0.001), and serum creatinine (r=0.386, p=0.002); the strongest correlation was with the microalbumin-to-creatinine ratio. No significant correlation was found between QTc and age, systolic or diastolic blood pressure, or RR interval.

On multiple linear regression analysis (Table 4), incorporating age, diabetes duration, systolic and diastolic blood pressure, HbA1c, RR interval, serum creatinine and microalbumin-to-creatinine ratio, only the microalbumin-to-creatinine ratio remained an independent, statistically significant predictor of QTc interval (β=0.035, p=0.010); diabetes duration showed borderline significance (β=0.818, p=0.054), while the remaining variables were not independently predictive.

Table 1: Comparative Clinical and Biochemical Characteristics of Study Groups

Parameter	Microalbuminuria (n=31)	Normoalbuminuria (n=31)	t / χ^2	p value
Age (years), mean±SD	63.90±15.27	52.35±10.78	t=3.44	0.001*
Age ≥60 years, n (%)	21 (67.7)	8 (25.8)	χ^2=11.01	0.004*
Male gender, n (%)	22 (71.0)	15 (48.4)	χ^2=3.28	0.070
Duration of diabetes (years), mean±SD	10.06±6.03	7.19±2.85	t=2.40	0.019*
Duration ≥10 years, n (%)	18 (58.1)	7 (22.6)	χ^2=7.87	0.005*
Systolic BP (mmHg), mean±SD	126.19±17.34	126.90±9.57	t=-0.20	0.843
Diastolic BP (mmHg), mean±SD	74.61±10.14	78.48±7.08	t=-1.74	0.087
HbA1c (%), mean±SD	8.09±1.35	7.33±0.54	t=2.94	0.005*
Serum creatinine (mg/dL), mean±SD	1.23±0.91	0.84±0.31	t=2.29	0.026*
Serum creatinine >1.2 mg/dL, n (%)	14 (45.2)	4 (12.9)	χ^2=8.21	0.004*
Serum calcium (mg/dL), mean±SD	8.52±0.54	8.70±0.50	t=-1.06	0.292
Microalbumin-creatinine ratio (mg/g), mean±SD	185.32±231.86	18.08±6.03	t=4.01	<0.001*
RR interval (sec), mean±SD	0.734±0.064	0.733±0.059	t=0.08	0.934
QTc interval (ms), mean±SD	**444.77±9.15**	**419.42±7.86**	**t=11.70**	**<0.001***

***Statistically significant (p<0.05).**

Table 2: Association between QTc Prolongation (>440 ms) and Study Groups

QTc Category	Microalbuminuria n (%)	Normoalbuminuria n (%)	Statistic
Normal (≤440 ms)	12 (38.7)	31 (100.0)	χ^2=24.59
Prolonged (>440 ms)	19 (61.3)	0 (0.0)	p<0.001*

*Statistically significant (p<0.05).

Table 3: Correlation of Clinical Variables with QTc Interval

Variable	r value	p value	Interpretation
Age (years)	0.205	0.110	Not significant
Duration of diabetes (years)	0.349	0.005*	Significant, moderate positive
Systolic BP (mmHg)	-0.067	0.605	Not significant
Diastolic BP (mmHg)	-0.201	0.117	Not significant
HbA1c (%)	0.321	0.011*	Significant, moderate positive
Microalbumin-creatinine ratio (mg/g)	**0.432**	**<0.001***	**Significant, strongest positive**
RR interval (sec)	-0.004	0.978	No correlation
Serum creatinine (mg/dL)	0.386	0.002*	Significant, moderate positive

*Statistically significant (p<0.05).

Table 4: Multiple Linear Regression Analysis for QTc Interval

Variable	β Coefficient	p value	Interpretation
Age (years)	-0.077	0.585	Not significant
Duration of diabetes (years)	0.818	0.054	Borderline, not independent
Systolic BP (mmHg)	-0.077	0.689	Not significant
Diastolic BP (mmHg)	-0.137	0.634	Not significant
HbA1c (%)	1.337	0.492	Not significant after adjustment
Microalbumin-creatinine ratio (mg/g)	**0.035**	**0.010***	**Independent significant predictor**
RR interval (sec)	-32.49	0.282	Not significant
Serum creatinine (mg/dL)	0.848	0.805	Not significant

*Statistically significant (p<0.05).

DISCUSSION

This study demonstrates a strong and statistically robust association between microalbuminuria and QTc interval prolongation in patients with T2DM, with the microalbumin-to-creatinine ratio emerging as the sole independent predictor of QTc interval on multivariate analysis. These findings align closely with, and extend, existing literature on the nephropathy–cardiac repolarisation axis in diabetes.

The mean QTc interval in the microalbuminuria group (444.77±9.15 ms) was comparable to values reported by Bhise et al. (448.2±23.6 ms),[12] Dominic et al. (457±21 ms),[13] Sawarthia et al. (451.2±22.5 ms),[14] and Chander et al. (462±18 ms),[15] all of whom similarly documented significantly higher QTc in microalbuminuric compared with normoalbuminuric diabetics. Likewise, the prevalence of QTc prolongation in the present cohort (61.3%) closely parallels the 66.6–68% range reported by Bhise, Dixit and Chander et al.[12,16,15] reinforcing the consistency of this association across different populations.

Age was significantly associated with microalbuminuria in the present study, consistent with Shi and Jiang, who identified older age as a risk factor for prolonged QTc in T2DM,[17] although this contrasts with Sawarthia et al., who found no significant age difference between groups despite comparable QTc differences.[14] Gender showed a numerical male predominance in the microalbuminuria group without reaching statistical significance, in keeping with Sawarthia et al,[14] and broadly consistent with Subbalakshmi et al., who reported sex-specific patterns of QTc prolongation related to specific comorbidities rather than sex alone.[18]

Duration of diabetes was significantly longer in the microalbuminuria group, corroborating findings by Dominic et al,[13] and Chander et al., the latter reporting a similar positive correlation between QTc and diabetes duration (r=0.52).[15] Poor glycaemic control, reflected by higher HbA1c, was also significantly associated with microalbuminuria, mirroring the findings of Dominic et al,[13] and Chander et al,[15] although the association weakened when HbA1c was dichotomised, similar to the pattern noted by Subbalakshmi et al., who found QTc more closely linked to microvascular and autonomic complications than glycaemic levels per se18. Blood pressure parameters did not differ significantly between groups in the present study, suggesting that the QTc–albuminuria relationship observed here was not primarily hypertension-driven, in partial contrast to Janakiraman et al. and Subbalakshmi et al., who reported an accentuating effect of hypertension on QTc prolongation in diabetics.[19,18]

Renal parameters clearly differentiated the two groups, with significantly higher serum creatinine and microalbumin-to-creatinine ratio in the microalbuminuria group, in agreement with Duran, who reported a significantly elevated median microalbumin/creatinine ratio alongside prolonged QTc indices in microalbuminuric diabetics.[20] On correlation analysis, QTc showed its strongest relationship with the microalbumin-to-creatinine ratio, echoing the pattern seen by Chander et al. and Dixit et al., both of whom documented significant positive correlations between QTc and markers of

nephropathy or disease duration.[15,16] Notably, on multivariate regression, the microalbumin-to-creatinine ratio remained the only independent predictor of QTc interval after adjustment for age, blood pressure, HbA1c and serum creatinine — suggesting that the effects of these other variables on QTc are largely mediated through albuminuria itself. This finding is broadly consistent with the multivariable approach of Shi and Jiang, who identified obstructive sleep apnoea (rather than glycaemic parameters) as an independent QTc predictor in a comparable regression framework, underscoring the general utility of multivariate analysis in isolating the dominant driver of repolarisation abnormality in diabetic populations.[17]

Correlation analysis in the present study additionally showed that QTc interval was more closely related to renal and metabolic variables than to haemodynamic parameters: significant positive correlations were observed with duration of diabetes, HbA1c, microalbumin-to-creatinine ratio, and serum creatinine, whereas age, systolic and diastolic blood pressure, and RR interval showed no significant correlation. This pattern is consistent with Chander et al., who reported comparable positive correlations between QTc and both HbA1c and diabetes duration, and with Dixit et al., who similarly linked QTc prolongation to disease duration and other microvascular complications rather than to haemodynamic status alone. On multivariate regression, after simultaneous adjustment for all of these variables, only the microalbumin-to-creatinine ratio retained independent significance, while duration of diabetes fell to borderline significance and HbA1c, serum creatinine, blood pressure and RR interval lost significance entirely. This pattern strongly suggests that the univariate associations observed for diabetes duration, glycaemic control and serum creatinine were substantially mediated through their shared relationship with albuminuria, rather than representing independent pathways to QTc prolongation.

Taken together, these findings support a shared pathophysiological substrate — chronic hyperglycaemia-driven endothelial dysfunction, oxidative stress and microvascular injury — that concurrently affects the renal microvasculature (manifesting as albuminuria) and cardiac autonomic/repolarisation pathways (manifesting as QTc prolongation). Because both urine microalbumin estimation and electrocardiography are inexpensive, non-invasive and already integrated into routine diabetic care, this relationship carries direct clinical relevance: it raises the possibility that patients identified with microalbuminuria could be flagged for targeted ECG-based cardiovascular risk stratification, complementing rather than replacing structured cardiovascular assessment.

Recommendations

Based on these findings, routine electrocardiographic assessment of QTc interval may be considered in patients with T2DM who have microalbuminuria, particularly those who are older, have longer disease duration, or poorer glycaemic control. Since the microalbumin-to-creatinine ratio was identified as the strongest independent predictor of QTc interval, continued emphasis on urinary albumin screening is warranted, not only for nephropathy surveillance but also as a practical, low-cost tool for cardiovascular risk stratification. An integrated follow-up approach combining urine microalbumin estimation, glycaemic assessment, renal function testing and periodic ECG monitoring may help identify high-risk patients earlier, and clinicians should remain cautious when prescribing QT-prolonging drugs in this subgroup.

Strengths and Limitations

The study's principal strengths include its balanced comparative design with two clearly defined, equally sized groups, which allowed direct and interpretable comparison; the combined use of continuous and categorical analyses for major variables, which provided a broader understanding of the relationships observed; and the use of correlation together with multivariate regression, which moved the analysis beyond simple group comparison to identify the single most important independent predictor of QTc interval. The exclusion of urinary tract infection as a confounder, confirmed by absent urinary leukocytes in all participants, further strengthens confidence that the albuminuria measured reflected genuine diabetic renal involvement rather than a transient inflammatory process.

Several limitations must nonetheless be acknowledged. The sample size (n=62) was modest and drawn from a single centre, limiting statistical power to detect smaller but potentially clinically relevant associations and constraining generalisability to other populations with different iodine, dietary, or ethnic profiles. The cross-sectional, observational design permits demonstration of association but not causal inference, and QTc was assessed at a single time point, precluding evaluation of its dynamic changes over the course of diabetes or in response to treatment. Important potential confounders — including formal autonomic function testing, body mass index, lipid profile, smoking status, concomitant medications, and subclinical ischaemic heart disease — were not comprehensively assessed and could have influenced the observed relationships. Long-term cardiovascular outcomes such as arrhythmic events, syncope, or mortality were not evaluated, so the clinical consequences of the QTc prolongation observed here remain inferential. Larger, prospective, multicentric studies incorporating these variables, repeated measurements, and longitudinal follow-up are needed to confirm and extend these findings, and to

determine whether interventions that reduce albuminuria also normalise QTc interval.

CONCLUSION

This study demonstrates a strong and independent association between microalbuminuria and QTc interval prolongation in patients with T2DM, with the microalbumin-to-creatinine ratio emerging as the single most important predictor of QTc interval on multivariate analysis. These findings support the concept that microalbuminuria is not merely a renal marker but also a signal of systemic microvascular and cardiac electrical vulnerability. Given that both investigations are simple, inexpensive and already part of routine diabetic care, incorporating electrocardiographic QTc assessment into the evaluation of diabetic patients with microalbuminuria — particularly those who are older, have longer disease duration, or poorer glycaemic control — may help identify individuals at higher cardiovascular risk before overt disease manifests, enabling earlier preventive intervention.

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