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Relationship Between Serum Uric Acid Levels and QTc Interval Prolongation in Hypertensive Patients: A Cross-Sectional Analysis

Saifullah Khan[1], Amna Nasir Yousif Alawasia Alzaabi[2], Christiana Yeboah[3], Emmanuel Asamoah[4], Emmanuel Mensa Bonsu[5], Alwaleed Alfadhli[6], Asma maryam[7], Joshua Odidison[6], Muhammad Ahmed Ashfaque[6], Muhammad Ali Niaz[6]

Abstract

Background: Hyperuricemia has recently emerged as a potential cardiovascular risk factor, particularly among patients with hypertension. Prolongation of the corrected QT (QTc) interval is associated with an increased risk of ventricular arrhythmias and sudden cardiac death. However, the relationship between serum uric acid levels and QTc interval prolongation in hypertensive patients has not been fully explored.

Objective: To evaluate the association between serum uric acid levels and QTc interval prolongation in patients with hypertension.

Methods: This cross-sectional study was conducted on 200 patients with hypertension. Serum uric acid levels were measured, and participants were categorized into normouricemic and hyperuricemic groups. The QTc interval was calculated using standard 12-lead electrocardiograms. Statistical analysis, including correlation analysis and multivariable logistic regression, was performed to assess the association between serum uric acid levels and QTc interval prolongation after adjusting for potential confounding variables.

Results: Patients with hyperuricemia had longer QTc intervals than normouricemic patients. A positive association was observed between serum uric acid levels and QTc interval duration. Multivariable logistic regression analysis showed that elevated serum uric acid levels were independently associated with an increased risk of QTc interval prolongation after adjustment for potential confounders.

Conclusion: Elevated serum uric acid levels are associated with QTc interval prolongation in hypertensive patients. These findings suggest that serum uric acid may serve as a useful marker for identifying individuals at increased risk of cardiac electrical abnormalities.

Keywords: Hyperuricemia; QTc interval; Hypertension; Arrhythmia; Cardiovascular risk.

Introduction

Hypertension is one of the most prevalent chronic diseases worldwide and a major contributor to cardiovascular morbidity and mortality. It affects more than one billion people globally and is strongly associated with an increased risk of coronary artery disease, stroke, heart failure, and sudden cardiac death [1, 2]. Despite advances in pharmacological treatment, patients with hypertension remain vulnerable to adverse cardiovascular outcomes, highlighting the need to identify additional risk factors and early markers of cardiac dysfunction [3]. Serum uric acid, the final product of purine metabolism, has attracted increasing attention as a potential contributor to cardiovascular disease. Elevated serum uric acid levels, commonly referred to as hyperuricemia, have been associated with endothelial dysfunction, oxidative

Affiliation:

[1]MedSTAR Washington Hospital

[2]MOI

[3]Hopewell Community Clinic, Accra, Ghana

[4]University of Cape Coast School of Medical Sciences

[5]Kwame Nkrumah University of Science and Technology, School of Medicine and Dentistry

[6]Royal College of Surgeons, Ireland

[7]Watim medical and dental college Rawalpindi

***Corresponding author:**

Saifullah Khan, MedSTAR Washington Hospital.

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stress, systemic inflammation, and activation of the renin-angiotensin-aldosterone system [4]. These pathophysiological mechanisms may not only contribute to the development and progression of hypertension but may also affect cardiac electrophysiology. Growing evidence suggests that hyperuricemia may also be associated with an increased risk of arrhythmias and adverse cardiovascular events [5].

The QT interval on electrocardiography reflects the time required for ventricular depolarization and repolarization, while the corrected QT (QTc) interval adjusts for heart rate [6]. Prolonged QTc is a clinically important marker of delayed ventricular repolarization and is associated with a higher risk of life-threatening arrhythmias, including torsades de pointes and sudden cardiac death [7]. Although electrolyte disturbances, medications, and underlying cardiovascular conditions are well-recognized determinants of QTc duration, recent studies suggest that metabolic abnormalities may also play an important role [4, 8]. Although the association between serum uric acid and cardiovascular outcomes has been explored previously, the relationship between serum uric acid levels and QTc interval prolongation, particularly in hypertensive patients, has not been adequately investigated [9]. Because hypertension and hyperuricemia frequently coexist and may exert a synergistic effect on cardiac function, understanding this relationship is of considerable clinical importance. In addition, limited regional data, particularly from developing countries, highlight the need for further research in diverse populations [10].

Therefore, the present study was conducted to evaluate the relationship between serum uric acid levels and QTc interval prolongation in patients with hypertension using a cross-sectional design. By examining the potential link between a metabolic marker and an electrophysiological abnormality, this study aims to contribute to improved cardiovascular risk stratification and the early identification of hypertensive patients who may be at increased risk of cardiac complications.

Rationale and Objectives

Hypertension is commonly associated with a range of metabolic disturbances, among which elevated serum uric acid is considered a potential contributor to cardiovascular dysfunction. Hyperuricemia has been linked to endothelial injury, increased oxidative stress, and impaired regulation of vascular tone, all of which may influence cardiac electrophysiology. At the same time, prolongation of the corrected QT (QTc) interval is a recognized marker of myocardial electrical instability and is associated with a higher risk of potentially life-threatening arrhythmias and sudden cardiac death. The coexistence of these factors in hypertensive patients raises an important clinical question regarding their possible interrelationship.

Although the role of serum uric acid in cardiovascular disease has received increasing attention, its association with QTc interval prolongation has not been adequately investigated, particularly among patients with hypertension. This gap is especially relevant in developing countries such as Pakistan, where the burden of hypertension is high and access to advanced risk stratification tools may be limited. Exploring this relationship may provide valuable insight into the early identification of high-risk patients and support improved clinical management. Therefore, the present study was conducted to assess the relationship between serum uric acid levels and QTc interval prolongation in hypertensive patients using a cross-sectional design. It also aimed to compare QTc interval values between normouricemic and hyperuricemic patients, determine the frequency of QTc prolongation across uric acid groups, and evaluate this association after accounting for potential confounding factors, including age, gender, and body mass index.

Materials and Methods

The present cross-sectional study was conducted to determine the relationship between serum uric acid levels and QTc interval prolongation in patients with hypertension. The study was carried out at multiple healthcare facilities, including tertiary care hospitals and private cardiology clinics in Pakistan, in order to obtain a diverse and representative sample. A total of 200 participants (N = 200) with a confirmed diagnosis of hypertension were included in the study. The sample comprised 100 male and 100 female patients aged between 30 and 75 years. A non-probability consecutive sampling technique was used to recruit participants according to predefined inclusion and exclusion criteria. Eligible participants were required to have a documented history of hypertension for at least six months, along with a recent electrocardiographic (ECG) record and serum uric acid measurement. Patients with a history of cardiac arrhythmias, congenital long QT syndrome, electrolyte imbalance, chronic kidney disease, or use of medications known to affect the QT interval, including antiarrhythmic drugs, were excluded from the study.

Data were collected through both clinical and laboratory assessment. Venous blood samples were obtained from all participants for measurement of serum uric acid levels using standard biochemical analysis. The electrocardiographic analysis was performed using a standard 12-lead ECG, and the QT interval was corrected for heart rate using Bazett's formula: $QTc = QT / \sqrt{RR}$. Demographic and clinical information, including age, sex, body mass index (BMI), duration of hypertension, and smoking status, was collected using a structured data collection form. Participants were categorized into normouricemic and hyperuricemic groups according to standard reference values. Hyperuricemia was defined as a serum uric acid level >7 mg/dL in males and

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>6 mg/dL in females. QTc interval prolongation was defined as a QTc value >440 ms in males and >460 ms in females. These classifications were used for group comparisons and risk assessment [6]. Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 26. Descriptive statistics were used to summarize the data. Continuous variables were presented as mean ± standard deviation, whereas categorical variables were expressed as frequencies and percentages. Pearson correlation analysis was used to assess the relationship between serum uric acid levels and QTc interval duration. Independent-samples t tests were used to compare mean QTc values between normouricemic and hyperuricemic groups, while chi-square tests were applied to compare the frequency of QTc prolongation between the two groups [9]. In addition, multivariable logistic regression analysis was performed to determine the independent association between serum uric acid levels and QTc prolongation after adjustment for potential confounding variables, including age, sex, body mass index, smoking status, duration of hypertension, and blood pressure.

Results

A total of 200 hypertensive patients were included in the study, comprising 100 males (50.0%) and 100 females (50.0%). The mean age was 52.3 ± 10.6 years, and most participants were aged 41–60 years (n = 84, 42.0%). Regarding body mass index, 72 participants (36.0%) were overweight and 56 (28.0%) were obese. The mean duration of hypertension was 6.8 ± 3.5 years. Table 1 summarizes the demographic and clinical profile of the study participants. The sample showed a balanced sex distribution, with most participants falling in the middle-age category and a substantial proportion being overweight or obese.

Table 1: Demographic and Clinical Characteristics of Participants (N = 200)

Variable	Category	Frequency (n)	Percentage (%)	Mean ± SD
Age (years)	30–40	36	18	52.3 ± 10.6
	41–60	84	42	
	61–75	80	40	
Gender	Male	100	50	–
	Female	100	50	–
Body Mass Index	Underweight (<18.5)	20	10	27.8 ± 4.6
	Normal (18.5–24.9)	52	26	
	Overweight (25–29.9)	72	36	
	Obese (≥30)	56	28	
Duration of Hypertension (years)	<5 years	78	39	6.8 ± 3.5
	5–10 years	82	41	
	>10 years	40	20	
Serum Uric Acid (mg/dL)	Normouricemic	118	59	6.4 ± 1.2
	Hyperuricemic	82	41	8.2 ± 1.5
QTc Interval (ms)	Normal QTc	116	58	441.3 ± 21.5
	Prolonged QTc	84	42	457.8 ± 24.2

BMI: body mass index; QTc: corrected QT interval.

Values are presented as frequency (n), percentage (%), and mean ± standard deviation (SD). QTc was calculated using Bazett's formula. Hyperuricemia was defined as serum uric acid levels >7 mg/dL in males and >6 mg/dL in females. QTc prolongation was defined as QTc >440 ms in males and >460 ms in females. Table 2 presents the correlations among the study variables. Serum uric acid showed a positive relationship with QTc interval as well as with age, body mass index, and duration of hypertension.

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Table 2: Correlation Matrix of Study Variables (N = 200)

Variable	1	2	3	4	5
Serum Uric Acid (mg/dL)	—				
QTc Interval (ms)	0.312*	—			
Age (years)	0.218*	0.265*	—		
BMI (kg/m²)	0.174*	0.149*	0.201*	—	
Duration of Hypertension (years)	0.196*	0.238*	0.310*	0.172*	—

QTc: corrected QT interval; BMI: body mass index.

Values are expressed as Pearson correlation coefficients (r). A positive value indicates a direct relationship between variables. The symbol (*) denotes statistical significance at $p < 0.05$. QTc was calculated using Bazett's formula. Table 3 compares the demographic and clinical variables between normouricemic and hyperuricemic participants. The hyperuricemic group showed higher QTc interval values and a greater frequency of QTc prolongation.

Values are presented as mean ± standard deviation (SD) for continuous variables and frequency (percentage) for categorical variables. Independent sample t-test was used for continuous variables, while chi-square (χ^2) test was applied for categorical variables. A p-value of <0.05 was considered statistically significant. QTc was calculated using Bazett's formula. Effect size is expressed as Cohen's d for continuous variables and Cramér's V for categorical variables. Table 4 presents the association between serum uric acid levels and QTc interval prolongation. A higher proportion of prolonged QTc was observed in the hyperuricemic group than in the normouricemic group.

Values are presented as frequency (n) and percentage (%). Chi-square (χ^2) test was used to compare categorical variables between normouricemic and hyperuricemic groups. A p-value of <0.05 was considered statistically significant. QTc was calculated using Bazett's formula. QTc prolongation was defined as QTc >440 ms in males and >460 ms in females. Cramér's V was used as a measure of effect size for categorical comparisons. Table 5 presents the results of multivariable logistic regression analysis for predictors of QTc interval prolongation. Hyperuricemia remained significantly associated with QTc prolongation after adjustment for relevant confounding variables.

Crude OR represents unadjusted analysis, while adjusted OR accounts for potential confounding variables, including age, gender, body mass index, smoking status, duration of hypertension, and blood pressure. Logistic regression analysis was used to identify independent predictors of QTc interval prolongation. A p-value of <0.05 was considered statistically significant. QTc was calculated using Bazett's formula.

Table 3: Comparison of Clinical Variables Between Normouricemic and Hyperuricemic Groups (N = 200)

Variable	Normouricemic (n = 118)	Hyperuricemic (n = 82)	Test Statistic	p-value	95% CI / χ^2	Effect Size
Age (years)	49.8 ± 9.8	55.6 ± 10.9	t = -3.84	<0.001	-8.7 to -2.9	0.56
BMI (kg/m²)	27.5 ± 4.3	28.2 ± 4.9	t = -1.02	0.309	-2.0 to 0.6	0.15
Duration of Hypertension (years)	6.1 ± 3.2	7.8 ± 3.8	t = -3.29	0.001	-2.7 to -0.7	0.49
Systolic BP (mmHg)	138.4 ± 12.6	145.7 ± 14.2	t = -3.64	<0.001	-11.1 to -3.5	0.55
Diastolic BP (mmHg)	86.2 ± 8.4	89.5 ± 9.1	t = -2.52	0.013	-5.9 to -0.7	0.38
QTc Interval (ms)	432.1 ± 18.7	458.6 ± 22.4	t = -8.72	<0.001	-32.5 to -20.3	1.29
Serum Uric Acid (mg/dL)	6.4 ± 1.2	8.2 ± 1.5	t = -10.5	<0.001	-2.2 to -1.4	1.40
Smoking Status (Yes)	34 (28.8%)	38 (46.3%)	χ^2 = 6.12	0.013	–	0.18
QTc Prolongation (Yes)	34 (28.8%)	50 (61.0%)	χ^2 = 18.6	<0.001	–	0.31

QTc, corrected QT interval; BMI, body mass index; BP, blood pressure.

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Table 4: Association Between Serum Uric Acid Levels and QTc Interval Prolongation (N = 200)

Variable	Normouricemic (n = 118)	Hyperuricemic (n = 82)	Total (N = 200)	χ^2 value	p-value	Cramér's V
Male participants, n (%)	60 (50.8%)	40 (48.8%)	100 (50.0%)	0.08	0.776	–
Female participants, n (%)	58 (49.2%)	42 (51.2%)	100 (50.0%)	0.08	0.776	–
Normal QTc in males	46 (76.7%)	19 (47.5%)	65 (65.0%)	9.21	0.002	0.3
Prolonged QTc in males	14 (23.3%)	21 (52.5%)	35 (35.0%)	9.21	0.002	0.3
Normal QTc in females	38 (65.5%)	13 (31.0%)	51 (51.0%)	11.84	<0.001	0.34
Prolonged QTc in females	20 (34.5%)	29 (69.0%)	49 (49.0%)	11.84	<0.001	0.34
Overall normal QTc	84 (71.2%)	32 (39.0%)	116 (58.0%)	18.6	<0.001	0.31
Overall prolonged QTc	34 (28.8%)	50 (61.0%)	84 (42.0%)	18.6	<0.001	0.31

QTc, corrected QT interval.

Table 5: Multivariable Logistic Regression Analysis for Predictors of QTc Interval Prolongation (N = 200)

Variable	Category	Crude OR	95% CI	p-value	Adjusted OR	95% CI	p-value
Serum Uric Acid	Normouricemic	1.00 (Ref)	–	–	1.00 (Ref)	–	–
	Hyperuricemic	3.86	2.12–7.03	<0.001	2.84	1.62–4.98	<0.001
Age (years)	Continuous	1.05	1.02–1.08	0.001	1.03	1.01–1.06	0.02
Gender	Female	1.00 (Ref)	–	–	1.00 (Ref)	–	–
	Male	1.32	0.80–2.17	0.28	1.21	0.72–2.03	0.46
BMI (kg/m²)	Continuous	1.07	1.00–1.14	0.048	1.05	0.98–1.12	0.14
Smoking Status	No	1.00 (Ref)	–	–	1.00 (Ref)	–	–
	Yes	1.94	1.10–3.42	0.021	1.58	0.88–2.85	0.12
Duration of Hypertension (years)	Continuous	1.06	1.01–1.11	0.014	1.04	0.99–1.09	0.09
Systolic BP (mmHg)	Continuous	1.02	1.01–1.04	0.005	1.01	0.99–1.03	0.18
Diastolic BP (mmHg)	Continuous	1.03	1.00–1.06	0.028	1.02	0.99–1.05	0.21

OR, odds ratio; CI, confidence interval; QTc, corrected QT interval; BMI, body mass index; BP, blood pressure; Ref, reference category.

Discussion

The present cross-sectional study evaluated the relationship between serum uric acid levels and QTc interval prolongation in patients with hypertension. The findings demonstrated a significant positive association between hyperuricemia and abnormalities of ventricular repolarization. Serum uric acid levels were positively correlated with QTc interval duration, and patients with hyperuricemia had higher mean QTc values as well as a greater frequency of QTc prolongation than normouricemic participants. In addition, multivariable logistic regression analysis showed that elevated serum uric acid remained independently associated with QTc prolongation after adjustment for age, gender, body mass index, smoking status, duration of hypertension, and blood pressure. Taken together, these findings suggest that hyperuricemia may have important clinical relevance in hypertensive patients with regard to myocardial electrical instability [11, 12].

A notable observation in this study was the clear difference in QTc interval characteristics between normouricemic and hyperuricemic patients. The hyperuricemic group demonstrated higher QTc values and a greater burden of QTc prolongation, supporting the view that serum uric acid may have prognostic value beyond its conventional metabolic role. Since prolonged QTc is associated with malignant arrhythmias and sudden cardiac death, hypertensive patients with elevated serum uric acid may represent a subgroup at increased cardiovascular risk [13, 14]. Several biological mechanisms may explain the observed association between serum uric acid and QTc interval prolongation. Uric acid is no longer regarded merely as a metabolic byproduct; it is increasingly recognized as a mediator of vascular and myocardial dysfunction [9, 15]. Elevated serum uric acid has been associated with oxidative stress, systemic inflammation, endothelial dysfunction, and activation of the renin-angiotensin-aldosterone system. These processes may

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impair myocardial perfusion and alter ion channel activity, thereby affecting ventricular repolarization. Because the QTc interval reflects ventricular depolarization and repolarization timing, any disturbance in myocardial electrical recovery may contribute to QTc prolongation [16]. Therefore, the association observed in this study is biologically plausible and consistent with the broader cardiovascular effects of hyperuricemia.

Another possible explanation relates to the interaction between hyperuricemia and hypertension itself. Hypertension is known to produce structural and functional cardiac changes, including left ventricular hypertrophy, arterial stiffness, and autonomic imbalance. When elevated serum uric acid coexists with hypertension, these pathological processes may be amplified, increasing the likelihood of electrophysiological abnormalities. In this context, serum uric acid may serve not only as a laboratory parameter but also as an indicator of cumulative metabolic, hemodynamic, and inflammatory burden. This may justify closer cardiovascular monitoring in hypertensive patients with hyperuricemia [17]. The sex-stratified findings also support the overall interpretation of the results. A higher frequency of prolonged QTc was observed in hyperuricemic participants of both sexes, suggesting that the association was not limited to one subgroup. This is clinically relevant because ventricular repolarization differs between males and females, and sex-specific QTc thresholds are already recognized in clinical practice [7, 18]. Although gender was not an independent predictor in the adjusted regression model, the distribution of QTc prolongation across both sexes strengthens the consistency of the observed relationship.

Age also emerged as an important predictor of QTc prolongation in the adjusted model, which is consistent with previous cardiovascular literature. Advancing age is associated with changes in autonomic tone, myocardial compliance, vascular stiffness, and comorbidity burden, all of which may influence ventricular repolarization [11]. However, the persistence of serum uric acid as a significant factor after adjustment suggests that the association between serum uric acid and QTc prolongation cannot be explained by age alone. This finding supports the possibility that hyperuricemia may contribute independently to repolarization abnormalities. In contrast, body mass index was not independently associated with QTc prolongation in the adjusted analysis, although a mild relationship was observed in unadjusted analysis. A similar pattern was seen for smoking status, duration of hypertension, and blood pressure measurements, which lost significance after adjustment. These findings suggest that the relationship between hyperuricemia and QTc prolongation is not merely a reflection of conventional cardiovascular risk factors. Rather, serum uric acid appears to retain potential predictive value even when other clinically relevant variables are considered simultaneously [18, 19].

The findings of this study may have practical clinical implications. Serum uric acid testing is inexpensive, widely available, and routinely performed in many healthcare settings. Electrocardiography is also a simple and accessible diagnostic tool. Together, these measures may help identify hypertensive patients who are at increased risk of arrhythmogenic complications [20]. This may be particularly valuable in low-resource settings, where access to more advanced cardiovascular risk stratification tools may be limited. In such contexts, patients with both hypertension and hyperuricemia may benefit from closer ECG surveillance, medication review, and more comprehensive cardiovascular assessment. This study also contributes to the growing literature on the cardiovascular significance of hyperuricemia. Although serum uric acid has traditionally been discussed in relation to gout and renal disease, increasing evidence suggests its association with hypertension, endothelial dysfunction, vascular injury, heart failure, and arrhythmic risk [21]. By specifically examining QTc interval prolongation in hypertensive patients, the present study adds an important electrophysiological dimension to this body of literature. Since QTc prolongation is a clinically established marker of adverse cardiac outcomes, the identification of serum uric acid as a possible independent correlate may have relevance for both preventive cardiology and routine hypertension care.

The study has several strengths. The relatively balanced representation of male and female participants improved the interpretability of sex-based comparisons. In addition, the use of both correlation analysis and multivariable regression strengthened the analytical framework. Adjustment for clinically relevant covariates, including age, body mass index, smoking status, duration of hypertension, and blood pressure, also added rigor to the analysis. However, certain limitations should be acknowledged. First, the cross-sectional design does not allow causal inference. Although an association between serum uric acid and QTc prolongation was observed, it cannot be concluded that hyperuricemia directly causes repolarization abnormalities [15, 22]. Second, the study was conducted in selected healthcare settings in Pakistan, which may limit the generalizability of the findings to other populations. Third, some potentially important confounders, including renal function, antihypertensive medication subtype, serum electrolytes, diabetes status, and left ventricular hypertrophy, were not assessed in detail. Finally, QTc was calculated using Bazett's formula, which is widely used in clinical research but may overcorrect or undercorrect under certain heart rate conditions.

Despite these limitations, the consistency of the findings across correlation analysis, group comparisons, categorical distribution, and multivariable regression supports the overall robustness of the observed association. The repeated pattern of higher QTc values and greater QTc prolongation

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among hyperuricemic patients suggests that this relationship is unlikely to be incidental [23]. Overall, the present study indicates that elevated serum uric acid may be associated with QTc interval prolongation in hypertensive patients and may have potential value in cardiovascular risk stratification. Further longitudinal and multicenter studies are needed to clarify causality, explore underlying mechanisms, and determine whether reducing serum uric acid levels can lower QTc-related cardiovascular risk.

Conclusion

Elevated serum uric acid may be associated with QTc interval prolongation in hypertensive patients and may help identify individuals at increased risk of ventricular repolarization abnormalities. These findings highlight the potential clinical relevance of serum uric acid as an accessible marker in cardiovascular risk stratification. However, prospective longitudinal studies are needed to clarify the causal relationship and determine whether lowering serum uric acid can reduce QTc-related cardiovascular risk.

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