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ORIGINAL ARTICLE

Serum Uric Acid and Its Correlation with Blood Pressure in Hypertensive Patients. A Comparative Study

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ABSTRACT

Background: Hypertension is a major global health problem and an important risk factor for cardiovascular morbidity and mortality. In recent years, serum uric acid (SUA) has emerged as a potential biomarker linked to vascular dysfunction and elevated blood pressure. However, the association between SUA and hypertension remains controversial, particularly in South Asian populations where the burden of both hypertension and metabolic disorders is rising.

Objective: This study aimed to compare serum uric acid levels between hypertensive patients and normotensive controls and to evaluate the correlation of SUA with systolic and diastolic blood pressure.

Methods: A comparative cross-sectional study was conducted at Multan Medical and Dental College (MMDC), Multan, in collaboration with Nishtar Medical University, Multan, from January 2022 to January 2023. A total of 100 participants were included, consisting of 50 hypertensive patients and 50 normotensive age- and sex-matched controls. Blood pressure was recorded according to WHO guidelines, and fasting blood samples were collected to measure serum uric acid using the Uricase-Peroxidase enzymatic colorimetric method. Data were analyzed using SPSS version 26, with t-tests for group comparisons and Pearson's correlation for association analysis.

Results: Mean SUA levels were significantly higher in hypertensive patients (6.8 ± 1.3 mg/dL) compared to controls (5.1 ± 1.1 mg/dL, $p < 0.001$). A significant positive correlation was observed between SUA and systolic blood pressure ($r = 0.42$, $p = 0.002$) and diastolic blood pressure ($r = 0.36$, $p = 0.009$) in hypertensive patients, whereas no significant correlation was noted in controls.

Conclusion: Serum uric acid is elevated in hypertensive patients and shows a positive correlation with both systolic and diastolic blood pressure. Monitoring SUA may provide an additional tool for risk assessment and management in hypertensive individuals.

Keywords: Hypertension, Serum uric acid, Blood pressure, Cardiovascular risk, Comparative study

INTRODUCTION

Hypertension is a major global public health challenge and is recognized as one of the leading contributors to morbidity and mortality worldwide. It affects approximately 1.3 billion people, with a steadily increasing prevalence in both developed and developing countries[1]. The condition is strongly associated with cardiovascular complications such as ischemic heart disease, stroke, and chronic kidney disease, making it a critical focus of preventive medicine. Despite the availability of numerous antihypertensive drugs, the burden of uncontrolled hypertension remains high, indicating the need for additional biomarkers to improve risk stratification, early detection, and therapeutic outcomes[2,3].

One such biomarker that has gained increasing attention is serum uric acid (SUA), the end product of purine metabolism in humans. Uric acid is produced in the liver and excreted primarily by the kidneys[4]. While traditionally considered relevant only in the context of gout and renal stones, emerging evidence over the past two decades has highlighted its potential role in metabolic and cardiovascular disorders. Epidemiological and clinical studies suggest a close link between elevated SUA and the development of hypertension, metabolic syndrome, insulin resistance, and cardiovascular disease[5].

The biological plausibility of uric acid contributing to hypertension is supported by several mechanisms. Elevated SUA has been shown to induce endothelial dysfunction by reducing nitric oxide availability, leading to impaired vasodilation[6]. It also promotes oxidative stress, systemic inflammation, and smooth muscle cell proliferation within the vasculature. Furthermore, uric acid has been implicated in the activation of the renin–angiotensin system and the development of renal microvascular disease, thereby increasing salt sensitivity and raising blood pressure. These mechanistic insights support the hypothesis that SUA is not merely a marker but may also act as a mediator in the pathogenesis of hypertension[7].

Several population-based studies have reported a positive association between SUA levels and both systolic and diastolic blood pressure. Notably, longitudinal studies indicate that hyperuricemia often precedes the development of hypertension, particularly in younger individuals, suggesting a causal relationship[8]. Clinical trials of uric acid–lowering therapies, such as allopurinol and febuxostat, have also demonstrated reductions in blood pressure among adolescents and young adults, further reinforcing this association. However, the relationship appears to be more complex in older individuals, where comorbidities, long-standing vascular changes, and renal dysfunction may confound the role of SUA[9].

Despite these observations, the precise role of SUA in hypertension remains a subject of ongoing debate. Some investigators consider it an independent risk factor, while others view it as an epiphenomenon associated with other metabolic disturbances[10]. Differences in study populations, methodologies, and adjustment for confounders such as renal function, obesity, and dietary intake may account for the variability in results. Importantly, data from South Asian populations, where the prevalence of hypertension and metabolic disorders is rising at alarming rates, remain limited. Given the genetic, dietary, and environmental differences across populations, it is crucial to explore the SUA–blood pressure relationship in diverse cohorts[11,12].

This comparative study was therefore designed to evaluate serum uric acid levels in hypertensive patients and compare them with normotensive controls, while also examining the correlation of SUA with systolic and diastolic blood pressure. By conducting this study in a Pakistani clinical setting, we aim to provide region-specific insights into the potential role of SUA as a biomarker for

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hypertension. A better understanding of this relationship could not only aid in early identification of individuals at risk but may also open new avenues for therapeutic interventions targeting uric acid metabolism in the management of hypertension[13].

MATERIALS AND METHODS

Study Design and Setting: This was a hospital-based, comparative cross-sectional study conducted at Multan Medical and Dental College (MMDC), Multan, in collaboration with Nishtar Medical University, Multan. The study was carried out over a period of twelve months, from January 2022 to January 2023.

Study Population: The study population comprised both hypertensive patients and normotensive individuals for comparison. Hypertensive patients were recruited from the outpatient and inpatient services of both institutions, while normotensive controls were selected from hospital staff and community volunteers. Individuals of both genders between the ages of 30 and 70 years were considered eligible for participation.

Sample Size: A total of 100 individuals were enrolled in the study. Among these, 50 were confirmed hypertensive patients, while 50 were normotensive individuals matched for age and sex. The sample size was chosen to ensure adequate comparison between groups within the available study period.

Inclusion and Exclusion Criteria: Participants with hypertension were defined as those with a systolic blood pressure of 140 mmHg or greater and/or a diastolic blood pressure of 90 mmHg or greater, or those already on antihypertensive therapy. Controls were considered normotensive when systolic blood pressure was less than 130 mmHg and diastolic blood pressure was less than 80 mmHg. Patients with a history of gout, chronic kidney disease, liver dysfunction, malignancy, or systemic inflammatory disorders were excluded. Individuals taking uric acid–lowering drugs or long-term diuretic therapy, as well as pregnant women, were also not included in the study.

Data Collection Procedure: After obtaining written informed consent, demographic details and clinical information were recorded in a structured proforma. Blood pressure was measured in all participants using a calibrated mercury sphygmomanometer according to the World Health Organization guidelines. Two readings were taken in the sitting position, separated by five minutes, and the average value was considered for analysis. Venous blood samples of 5 mL were drawn from each participant following an overnight fast. Serum was separated by centrifugation and stored at –20°C until tested. Serum uric acid levels were analyzed using the Uricase-Peroxidase enzymatic colorimetric method on an automated biochemistry analyzer. Internal quality control measures were applied to maintain accuracy and reliability throughout laboratory procedures.

Statistical Analysis: Data analysis was performed using SPSS version 26. Continuous variables, including age, blood pressure, and serum uric acid, were presented as mean ± standard deviation. Categorical variables such as gender distribution were expressed as frequencies and percentages. The independent sample t-test was used to compare mean serum uric acid levels between hypertensive and normotensive groups. Pearson's correlation coefficient was applied to determine the association between serum uric acid and both systolic and diastolic blood pressure. A p-value of less than 0.05 was taken as statistically significant.

Ethical Considerations: The study was approved by the Institutional Review Boards (IRBs) of Multan Medical and Dental College, Multan, and Nishtar Medical University, Multan. Written informed consent was obtained from all participants prior to enrollment, and confidentiality of personal data was strictly ensured.

RESULTS

Baseline Characteristics: A total of 100 participants were enrolled in the study, including 50 hypertensive patients and 50 normotensive controls. The mean age of hypertensive patients was 51.6 ± 8.4 years, while that of normotensive controls was 49.2 ± 9.1 years. The distribution of gender was similar in both groups, with 54% males and 46% females among hypertensive patients, and 52% males and 48% females in the control group. No significant differences were observed between the two groups in terms of age and gender distribution, indicating adequate matching (Table 1).

Table 1. Baseline Characteristics of Study Participants

Variable	Hypertensive Patients (n=50)	Normotensive Controls (n=50)	p-value
Mean Age (years)	51.6 ± 8.4	49.2 ± 9.1	0.218
Gender (Male/Female)	27 / 23	26 / 24	0.841

Table 1 shows that the two groups were comparable in terms of age and gender distribution.

Serum Uric Acid Levels: The mean serum uric acid (SUA) level in hypertensive patients was 6.8 ± 1.3 mg/dL, whereas in normotensive controls it was 5.1 ± 1.1 mg/dL. The difference was statistically significant ($p < 0.001$). These findings demonstrate that hypertensive patients had significantly higher SUA levels compared to normotensive individuals (Table 2).

Table 2. Comparison of Serum Uric Acid Levels Between Groups

Group	Serum Uric Acid (mg/dL) Mean ± SD	p-value
Hypertensive Patients	6.8 ± 1.3	<0.001
Normotensive Controls	5.1 ± 1.1	

As shown in Table 2, serum uric acid levels were significantly higher among hypertensive patients compared to controls.

Correlation of Serum Uric Acid with Blood Pressure: Pearson's correlation analysis revealed a positive association between SUA and both systolic and diastolic blood pressure among hypertensive patients. The correlation coefficient between SUA and systolic blood pressure was $r = 0.42$ ($p = 0.002$), while the correlation with diastolic blood pressure was $r = 0.36$ ($p = 0.009$). In contrast, no significant correlation was observed in the normotensive control group (Table 3).

Table 3. Correlation of Serum Uric Acid with Systolic and Diastolic Blood Pressure

Group	Variable	Correlation Coefficient (r)	p-value
Hypertensive Patients	SUA vs SBP	0.42	0.002
Hypertensive Patients	SUA vs DBP	0.36	0.009
Normotensive Controls	SUA vs SBP	0.11	0.421
Normotensive Controls	SUA vs DBP	0.08	0.537

Table 3 indicates a significant positive correlation of serum uric acid with both systolic and diastolic blood pressure in hypertensive patients, while no meaningful correlation was found in normotensive individuals.

In summary, the present study demonstrated that serum uric acid levels were significantly elevated in hypertensive patients compared to normotensive controls. Furthermore, a strong positive correlation was observed between SUA and both systolic and diastolic blood pressure among hypertensive patients, while no such relationship was noted in the control group. These results suggest that elevated serum uric acid may play a contributory role in the pathogenesis of hypertension.

Discussion

The present study demonstrated that serum uric acid (SUA) levels were significantly higher in hypertensive patients compared to normotensive controls, and that SUA correlated positively with both systolic and diastolic blood pressure among hypertensive

individuals[13]. These findings support the growing body of evidence that hyperuricemia is closely linked with the pathophysiology of hypertension[14].

Several mechanisms have been proposed to explain this relationship. Uric acid is known to induce endothelial dysfunction by impairing nitric oxide production, thereby reducing vasodilation and increasing vascular resistance[15]. It has also been implicated in the generation of oxidative stress and inflammatory mediators that promote vascular remodeling. Additionally, SUA may activate the renin–angiotensin–aldosterone system, increase sodium retention, and contribute to renal microvascular damage, all of which predispose individuals to elevated blood pressure. These mechanistic pathways provide a biological basis for the association observed in this study[16,17].

The positive correlation between SUA and blood pressure was more pronounced in hypertensive patients, while no significant association was observed among normotensive controls. This finding suggests that uric acid may play a more direct role in the maintenance and progression of hypertension rather than in normotensive states[18]. Similar results have been reported in previous clinical and epidemiological studies, where SUA was not only elevated in hypertensive patients but was also shown to predict the onset of hypertension, particularly in younger populations. Clinical trials have further demonstrated that uric acid–lowering agents such as allopurinol can reduce blood pressure in adolescents and young adults, reinforcing the potential causal relationship[19].

The results of this study also align with previous observations in South Asian cohorts, where dietary patterns, genetic predisposition, and metabolic risk factors contribute to both elevated SUA and hypertension. Given the rising prevalence of hypertension in Pakistan, coupled with increasing rates of metabolic syndrome, monitoring SUA levels could provide an additional marker for early detection and risk stratification in clinical practice[17,20].

Nevertheless, certain limitations of this study should be acknowledged. First, the sample size was modest, which may limit the generalizability of findings. Second, the cross-sectional design prevents the establishment of a causal relationship between uric acid and hypertension[21]. Third, factors such as diet, renal function, and body mass index, which can influence uric acid levels, were not controlled in detail. Despite these limitations, the study adds valuable data to the regional literature by highlighting the association between SUA and hypertension in a Pakistani population[22].

Future research with larger, longitudinal cohorts and interventional trials is warranted to further clarify the causal role of SUA in hypertension and to determine whether uric acid–lowering therapies can serve as effective adjuncts in the management of hypertensive patients[23-25].

CONCLUSION

This study concludes that serum uric acid levels are significantly higher in hypertensive patients compared to normotensive individuals. A strong positive correlation was observed between SUA and both systolic and diastolic blood pressure among hypertensive patients, whereas no such relationship was noted in controls. These findings suggest that serum uric acid may act as a potential biomarker and contributory factor in the development and progression of hypertension. Regular monitoring of SUA in hypertensive patients may be useful for risk stratification, and further interventional research is required to explore the therapeutic implications of targeting uric acid metabolism in the prevention and management of hypertension.

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