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Original Article

Correlation Between Serum Uric Acid Levels and Cardiometabolic Risk Factors in Hypertensive Patients

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ABSTRACT

Background: Hyperuricemia is frequently observed in individuals with hypertension and has been linked to adverse metabolic profiles.
Objectives: To evaluate the correlation between serum uric acid (SUA) levels and major cardiometabolic risk factors among hypertensive patients.
Methods: A hospital-based observational study was conducted among 100 adults with hypertension over six months. Demographic variables, anthropometry, blood pressure, glycemic indices, and lipid parameters were recorded. SUA was measured using a standard enzymatic method, and participants were stratified into SUA tertiles. Correlation analyses and multivariable logistic regression were performed to identify independent associations. **Results:** The mean age was 54.2 ± 9.8 years and 58% were men. Mean SUA was 6.18 ± 1.52 mg/dL; hyperuricemia was present in 38% of participants. Higher SUA levels were associated with greater adiposity and higher blood pressure. SUA correlated positively with systolic and diastolic blood pressure, fasting blood glucose, HbA1c, total cholesterol, LDL-C, and triglycerides, and negatively with HDL-C. Metabolic syndrome was identified in 46% overall and was more frequent among participants with hyperuricemia. On adjusted analysis, elevated SUA remained independently associated with higher BMI, hypertriglyceridemia, and metabolic syndrome.
Conclusion: In hypertensive adults, higher SUA levels were associated with an adverse cardiometabolic phenotype, particularly central obesity, dysglycemia, hypertriglyceridemia, and metabolic syndrome.

Keywords: Serum uric acid; hyperuricemia; hypertension; metabolic syndrome; dyslipidemia; cardiometabolic risk.

INTRODUCTION

Hypertension remains one of the most prevalent non-communicable disorders worldwide and is a leading contributor to cardiovascular morbidity and mortality. Beyond elevated blood pressure alone, hypertensive individuals commonly exhibit coexisting metabolic abnormalities such as central obesity, insulin resistance, and atherogenic dyslipidemia, which together amplify long-term cardiometabolic risk[1,2]. Identifying low-cost biochemical markers that track with these clustered risks is clinically valuable in routine care, particularly in resource-limited settings where comprehensive cardiometabolic profiling is not always feasible.

Serum uric acid (SUA), the final product of purine metabolism, has gained attention as a potential marker and mediator of cardiometabolic dysfunction. Epidemiologic studies consistently report higher SUA levels in people with obesity, dysglycemia, and hypertension. Several biological mechanisms have been proposed, including endothelial dysfunction through reduced nitric oxide bioavailability, oxidative stress, vascular inflammation, and activation of the renin–angiotensin system, all of which can contribute to blood pressure elevation and vascular injury [3,4]. Experimental and

clinical observations also support bidirectional links between SUA and insulin resistance, suggesting that SUA elevation can coexist with impaired glucose handling and dyslipidemia [5-7].

Hyperuricemia has been associated with incident hypertension in longitudinal cohorts, including the Normative Aging Study and the Multiple Risk Factor Intervention Trial, indicating that SUA elevation can precede the development of sustained high blood pressure [3,4]. Meta-analyses further demonstrate a dose–response relationship between SUA and incident hypertension, although the causal role remains debated [5,6]. Importantly, SUA has been linked to metabolic syndrome and its components, particularly elevated triglycerides and reduced HDL-C, suggesting that SUA may capture broader cardiometabolic burden rather than reflecting hypertension alone [8-10]. Recent work has also highlighted composite indices such as SUA-to-HDL ratios as markers of inflammatory-metabolic risk, reinforcing the close connection between urate biology and dysmetabolism [11].

In Indian clinical practice, hypertensive patients frequently present with variable combinations of obesity, impaired fasting glucose, diabetes, and dyslipidemia. However, local data describing how SUA relates to these cardiometabolic risk factors in hospital-based hypertensive cohorts are still limited. Understanding these correlations can support pragmatic screening approaches and inform preventive counseling within hypertension clinics.

The objectives of this study were to estimate the prevalence of hyperuricemia among hypertensive patients attending a tertiary care hospital and to evaluate the relationship between SUA levels and cardiometabolic risk factors including anthropometry, blood pressure, glycemic parameters, lipid profile, and metabolic syndrome.

METHODOLOGY

Study design and setting:

This hospital-based observational study was conducted at Mamata General Hospital, Khammam, Telangana, India, over a six-month period from March 2025 to August 2025.

Study population and sampling:

Adult patients diagnosed with hypertension who attended the outpatient department or were admitted to the medicine wards during the study period were screened. A total sample size of 100 hypertensive patients was enrolled using a consecutive sampling approach.

Eligibility criteria:

Patients aged ≥18 years with a clinical diagnosis of hypertension (documented history of hypertension or blood pressure ≥140/90 mmHg on examination) were included. Patients with known gout on urate-lowering therapy, chronic inflammatory disorders, malignancy, pregnancy, or severe renal impairment requiring dialysis were excluded to reduce confounding from secondary causes of hyperuricemia and altered urate handling.

Data collection and measurements:

After obtaining informed consent, demographic details and clinical history, including duration of hypertension and current antihypertensive therapy, were recorded. Anthropometric measurements were obtained using standardized procedures. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Waist circumference was measured at the midpoint between the lower margin of the last rib and the iliac crest. Blood pressure was recorded with an appropriate cuff after at least five minutes of rest; two readings were taken and the average was used.

Venous blood samples were collected after an overnight fast to estimate fasting blood glucose (FBG), serum uric acid (SUA), and lipid profile (total cholesterol, triglycerides, HDL-C, and LDL-C). HbA1c was measured using a standardized laboratory method. Hyperuricemia was defined as SUA >7.0 mg/dL in males and >6.0 mg/dL in females [6]. Participants were also categorized into SUA tertiles: <5.5 mg/dL, 5.5–7.0 mg/dL, and >7.0 mg/dL.

Outcome definitions:

Glycemic abnormality was defined as impaired fasting glucose or diabetes based on fasting glucose and/or HbA1c thresholds used in routine clinical practice. Metabolic syndrome was determined using standard diagnostic criteria based on central obesity, elevated blood pressure, dysglycemia, hypertriglyceridemia, and low HDL-C [9,10].

Statistical analysis:

Data were analyzed using descriptive and inferential statistics. Continuous variables were summarized as mean ± standard deviation and categorical variables as frequency (%). Between-tertile comparisons were assessed with appropriate tests for trend. Pearson's correlation coefficient quantified relationships between SUA and continuous cardiometabolic variables. Multivariable logistic regression was performed to identify independent associations of elevated SUA after adjusting for age and sex, reported as adjusted odds ratios (OR) with 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant.

Ethics:

The study protocol was approved by the Institutional Ethics Committee of Mamata Medical College. Confidentiality was maintained and participation was voluntary.

RESULTS

A total of 100 hypertensive patients were included. The mean age of the cohort was 54.2 ± 9.8 years (range 32–72 years), with males comprising 58% of participants. The mean duration of hypertension was 6.4 ± 3.1 years. Mean SUA was 6.18 ± 1.52 mg/dL, and hyperuricemia was observed in 38% of participants, with a higher proportion among males than females. Baseline demographic and clinical characteristics are summarized in Table 1.

Table 1. Baseline Demographic and Clinical Characteristics of Hypertensive Patients (N = 100)

Variable	Value
Age (years), mean ± SD	54.2 ± 9.8
Age range (years)	32–72
Male, n (%)	58 (58%)
Female, n (%)	42 (42%)
Duration of hypertension (years), mean ± SD	6.4 ± 3.1
Serum uric acid (mg/dL), mean ± SD	6.18 ± 1.52
Hyperuricemia, n (%)	38 (38%)
Males with hyperuricemia, n (%)	24 (24%)
Females with hyperuricemia, n (%)	14 (14%)

Participants were stratified into SUA tertiles: Group I (<5.5 mg/dL; n=30), Group II (5.5–7.0 mg/dL; n=32), and Group III (>7.0 mg/dL; n=38). Anthropometric indices increased progressively across tertiles. Patients in the highest SUA tertile had significantly higher BMI and waist circumference than those in the lowest tertile. Similarly, systolic and diastolic blood pressures were significantly higher among patients with SUA >7.0 mg/dL, indicating an adverse hemodynamic profile with rising SUA (Table 2).

Table 2. Distribution of Cardiometabolic Parameters According to Serum Uric Acid Tertiles

Parameter	Group I (<5.5 mg/dL) (n=30)	Group II (5.5–7.0 mg/dL) (n=32)	Group III (>7.0 mg/dL) (n=38)	p-value
BMI (kg/m²), mean ± SD	25.8 ± 2.9	27.4 ± 3.1	29.1 ± 3.2	<0.01
Waist circumference (cm), mean ± SD	90.6 ± 7.9	94.3 ± 8.1	98.4 ± 8.7	<0.001
SBP (mmHg), mean ± SD	142.3 ± 11.6	149.4 ± 12.2	154.2 ± 10.8	<0.01
DBP (mmHg), mean ± SD	89.6 ± 7.8	92.4 ± 8.1	95.3 ± 8.4	0.02

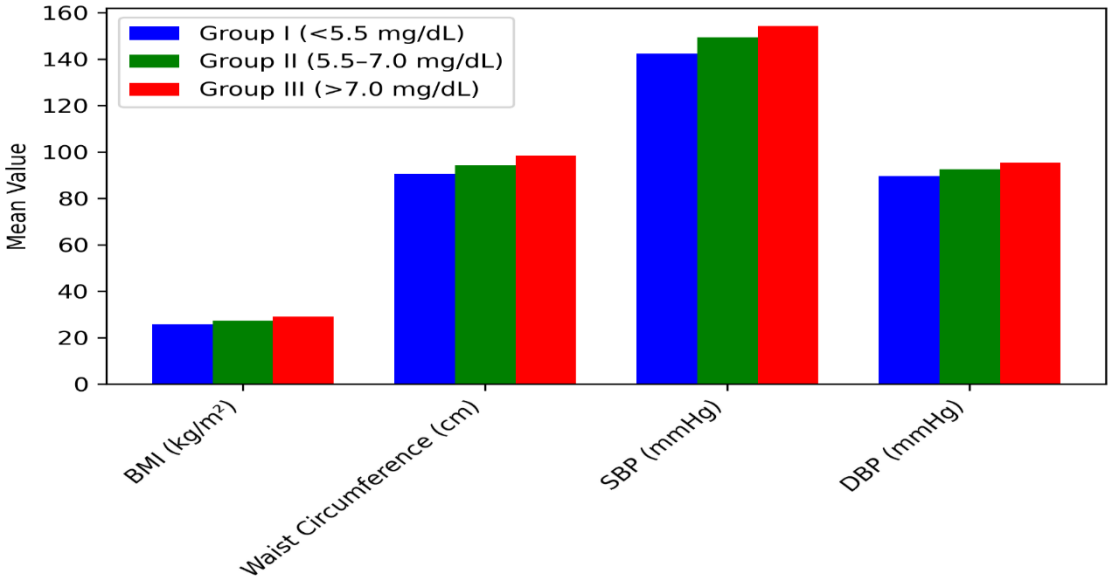

Figure 1: Distribution of Cardiometabolic Parameters According to Serum Uric Acid Tertiles

Correlation analyses demonstrated significant positive associations between SUA and multiple cardiometabolic risk factors. SUA correlated moderately with systolic blood pressure (r=0.41; p<0.001) and weakly with diastolic blood pressure (r=0.29; p=0.004). Glycemic indices also showed positive correlations with SUA, including fasting blood glucose and HbA1c. Among lipid parameters, triglycerides had the strongest positive correlation, whereas HDL-C showed a significant inverse correlation (Table 3).

Table 3. Correlation Between Serum Uric Acid and Cardiometabolic Risk Factors

Variable	Correlation coefficient (r)	p-value
Systolic blood pressure	0.41	<0.001
Diastolic blood pressure	0.29	0.004
Fasting blood glucose	0.36	<0.001
HbA1c	0.31	0.002
Triglycerides	0.45	<0.001
HDL-C	–0.33	0.001
Total cholesterol	0.21	<0.05
LDL-C	0.19	<0.05

Clinically, impaired fasting glucose or diabetes was more prevalent in the hyperuricemic group than in participants with normal SUA. Metabolic syndrome was present in 46% of the cohort and was significantly more frequent among hyperuricemic patients (65.8% vs 33.9%). On multivariable analysis adjusted for age and sex, elevated SUA remained independently associated with increased BMI, hypertriglyceridemia, and metabolic syndrome (Table 4).

Table 4. Association of Hyperuricemia with Glycemic Status, Lipid Abnormalities, and Metabolic Syndrome

Variable	Hyperuricemia (n=38)	Normal SUA (n=62)	p-value
Impaired fasting glucose/Diabetes, n (%)	17 (44.7%)	14 (23.4%)	0.01
Metabolic syndrome, n (%)	25 (65.8%)	21 (33.9%)	<0.001

Table 4A. Multivariate Logistic Regression Analysis (Adjusted for Age and Sex)

Variable	Adjusted OR	95% CI	p-value
Increased BMI	1.72	1.18–2.51	0.004
Hypertriglyceridemia	2.14	1.39–3.28	0.001
Metabolic syndrome	2.36	1.47–3.81	<0.001

DISCUSSION

In this hospital-based cohort of hypertensive adults, SUA demonstrated consistent relationships with several cardiometabolic risk factors. The prevalence of hyperuricemia (38%) aligns with the concept that urate elevation frequently accompanies hypertension and metabolic disturbances [6]. Across SUA tertiles, progressive increases in BMI and waist circumference were observed, supporting prior evidence that urate concentrations rise with adiposity and central fat distribution, both of which are linked to insulin resistance and low-grade inflammation [8-10].

Blood pressure indices also increased with rising SUA, and correlation analysis showed a moderate association between SUA and systolic blood pressure. Longitudinal studies have reported that higher urate levels predict incident hypertension in diverse populations, including the Normative Aging Study and the Multiple Risk Factor Intervention Trial, reinforcing the temporal link between urate elevation and blood pressure dysregulation [3,4]. Mechanistically, urate-related endothelial dysfunction, oxidative stress, and activation of neurohormonal pathways have been proposed as contributors to vascular tone abnormalities and microvascular remodeling [11,12].

A key finding of the present study is the positive correlation between SUA and glycemic indices (FBG and HbA1c), along with a higher burden of impaired fasting glucose/diabetes in the hyperuricemic group. These results are consistent with the reported interplay between urate biology and insulin sensitivity, where hyperinsulinemia can reduce renal urate excretion and elevated urate can impair nitric oxide-dependent insulin signaling [4,7]. Furthermore, triglycerides showed the strongest positive correlation with SUA, while HDL-C correlated inversely—an atherogenic pattern frequently described in metabolic syndrome cohorts [8-10]. The independent association between elevated SUA and hypertriglyceridemia in adjusted analysis strengthens the clinical relevance of urate as a marker of dyslipidemic risk clustering.

Metabolic syndrome was common in this hypertensive cohort (46%) and substantially more prevalent among hyperuricemic individuals. Prior studies and meta-analyses have similarly shown higher SUA levels among individuals with metabolic syndrome and a graded relationship with the number of syndrome components [13,14]. Recent evidence

also suggests that urate-to-HDL derived indices correlate with metabolic syndrome severity, emphasizing that urate is intertwined with lipid and inflammatory pathways [11]. Collectively, the current findings indicate that among hypertensive patients, SUA elevation signals a broader cardiometabolic risk phenotype that extends beyond blood pressure control alone.

Limitations

This single-center study used a modest sample size and a cross-sectional analytical approach, limiting inference on temporality and causation. Potential confounders such as detailed dietary purine intake, alcohol use, renal function staging, and medication effects (including diuretics) were not quantified in the dataset. Single-point biochemical measurements were used, and variability over time was not assessed. These factors restrict precision in estimating independent urate-related effects.

CONCLUSION

In this six-month observational study of 100 hypertensive adults, serum uric acid levels were strongly aligned with key cardiometabolic risk markers. Hyperuricemia was frequent and was linked to higher body mass index, greater waist circumference, and higher systolic and diastolic blood pressures. Serum uric acid also correlated with dysglycemia and an atherogenic lipid profile, particularly elevated triglycerides and reduced HDL-C. Metabolic syndrome was substantially more common among hyperuricemic patients, and elevated uric acid remained independently associated with increased BMI, hypertriglyceridemia, and metabolic syndrome after adjustment. These findings support incorporating uric acid assessment into routine evaluation of hypertensive patients to aid early detection of clustered cardiometabolic risk**.**

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