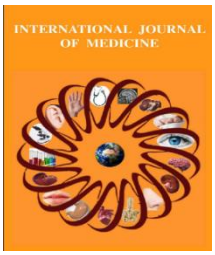

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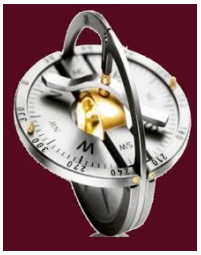

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Original Research

Association of Serum Uric Acid with Hypertension and Metabolic Risk Factors in Adults: A Hospital-Based Observational Study.

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Abstract

Background: Serum uric acid has been linked with hypertension and metabolic abnormalities, but its clinical relevance in hospital-attending Indian adults remains incompletely defined. *Objectives:* To assess the association of serum uric acid with hypertension and selected metabolic risk factors among adults attending a tertiary-care hospital. *Methods:* This hospital-based cross-sectional observational study included 80 adults recruited at Government Medical College Nizamabad, Telangana, India, from September 2024 to March 2025. Demographic information, anthropometry, blood pressure, fasting glucose, serum uric acid, and lipid parameters were recorded. Hyperuricaemia was defined as serum uric acid >7.0 mg/dL in males and >6.0 mg/dL in females. Group comparisons, correlation analysis, and multivariable logistic regression were performed. *Results:* The mean age was 49.2 ± 11.8 years; 46 (57.5%) participants were males. Hypertension was present in 36 (45.0%), metabolic syndrome in 28 (35.0%), and hyperuricaemia in 29 (36.3%). Mean serum uric acid was significantly higher in hypertensive than normotensive participants (7.0 ± 1.5 versus 5.5 ± 1.3 mg/dL; p<0.001). Hyperuricaemia occurred in 61.1% of hypertensive and 15.9% of normotensive participants and was associated with hypertension (odds ratio 8.31; 95% confidence interval 2.80-24.66). Serum uric acid correlated positively with waist circumference, systolic blood pressure, body mass index, diastolic blood pressure, and triglycerides, and inversely with high-density lipoprotein cholesterol. After adjustment, hyperuricaemia remained independently associated with hypertension (adjusted odds ratio 4.18; 95% confidence interval 1.29-13.54). *Conclusion:* Elevated serum uric acid was independently associated with hypertension and clustered metabolic risk factors. Serum uric acid assessment can support cardiometabolic risk stratification in hospital-attending adults.

Keywords: *Hyperuricaemia; hypertension; metabolic syndrome; obesity; cardiometabolic risk; serum uric acid.*

Introduction

Uric acid is the final product of purine metabolism in humans and is predominantly eliminated through the kidneys. At physiological concentrations, it contributes to extracellular antioxidant activity; however, persistent elevation is increasingly recognised as a marker of adverse cardiometabolic status. Hyperuricaemia commonly accompanies obesity, hypertension, insulin resistance, dyslipidaemia, diabetes mellitus, chronic kidney disease, and cardiovascular disease. Experimental and clinical evidence indicates that excess uric acid can influence vascular and renal homeostasis through oxidative stress, reduced endothelial nitric oxide availability, activation of the renin-angiotensin system, inflammation, and renal microvascular injury. These pathways provide biological plausibility for a relationship between serum uric acid and elevated blood pressure. [1,2]

The association between serum uric acid and hypertension has been examined in diverse populations. A systematic review of prospective cohorts reported a higher risk of incident hypertension among individuals with hyperuricaemia and demonstrated an incremental rise in risk with increasing uric acid concentration. [3] Longitudinal evidence from a Japanese cohort further identified serum uric acid as an independent risk marker for progression from prehypertension to hypertension. [4] A dose-response meta-analysis of prospective studies also supported a graded association between serum uric acid and new-onset hypertension. [5] Cross-sectional findings from South Asian adults showed positive relationships between uric acid, systolic blood pressure, diastolic blood pressure, and hypertension after adjustment for demographic and metabolic covariates. [6]

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Serum uric acid is also closely linked to metabolic syndrome, a cluster of abdominal adiposity, elevated blood pressure, hyperglycaemia, hypertriglyceridaemia, and reduced high-density lipoprotein cholesterol. [7] Prospective evidence demonstrates that increasing serum uric acid is associated with a higher risk of developing metabolic syndrome. [8] Population studies have reported higher uric acid concentrations among adults with obesity, central adiposity, dyslipidaemia, and metabolic syndrome, while national datasets and systematic reviews indicate that this association persists after adjustment for major confounders. [9-11] Hyperinsulinaemia can reduce renal urate excretion, whereas adipose tissue dysfunction, dietary fructose exposure, and increased xanthine oxidase activity can raise uric acid production. Consequently, serum uric acid can reflect the combined influence of several interconnected metabolic processes.

Asian Indians develop cardiometabolic complications at comparatively lower levels of body mass index and waist circumference, making population-specific evaluation important. [12] Although hypertension and metabolic disorders are common in Indian hospital practice, serum uric acid is not routinely incorporated into cardiometabolic assessment outside gout and renal disease. Standardised blood-pressure classification and careful evaluation of metabolic risk factors can clarify whether hyperuricaemia identifies a clinically distinct high-risk group. [13] Recent evidence in older adults has also shown positive correlations of uric acid with body mass index, waist circumference, blood pressure, and triglycerides, together with an inverse relationship with high-density lipoprotein cholesterol. [14]

The present study was undertaken to determine the association between serum uric acid and hypertension among adults attending a tertiary-care hospital and to evaluate its relationship with obesity, central obesity, diabetes mellitus, dyslipidaemia, metabolic syndrome, fasting blood glucose, and lipid parameters. A secondary objective was to identify whether hyperuricaemia remained independently associated with hypertension after adjustment for major demographic and metabolic risk factors.

Materials and Methods

Study design and setting: This hospital-based cross-sectional observational study was conducted in the Department of General Medicine, Government Medical College Nizamabad, Telangana, India, from September 2024 to March 2025. Adult patients attending outpatient and inpatient services were screened consecutively.

Participants and eligibility: Adults aged 18 years or older who provided written informed consent and had complete clinical and fasting biochemical information were eligible. Patients with acute gout, urate-lowering treatment, pregnancy, acute inflammatory illness, active malignancy, advanced chronic kidney disease, decompensated liver disease, or incomplete records were excluded. Of 86 individuals assessed, six were excluded and 80 were analysed. The sample comprised all consecutively eligible participants recruited during the predefined period.

Data collection and clinical assessment: Demographic details, smoking status, previous diagnoses, and treatment history were recorded using a structured proforma. Height was measured without footwear and weight with light clothing. Body mass index was calculated as weight in kilograms divided by height in metres squared and classified as normal, overweight, or obese. Waist circumference was measured midway between the lower rib margin and iliac crest. Central obesity was defined as waist circumference ≥90 cm in males or ≥80 cm in females. 12 Blood pressure was recorded in the seated position after five minutes of rest using a calibrated sphygmomanometer. Two readings were obtained five minutes apart, and their average was used. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, a documented diagnosis, or antihypertensive treatment. 13

Laboratory measurements and operational definitions: After an overnight fast of 8-12 hours, venous blood was collected under aseptic precautions. Serum uric acid was measured by the uricase-peroxidase enzymatic method. Hyperuricaemia was defined as serum uric acid >7.0 mg/dL in males and >6.0 mg/dL in females. Fasting blood glucose, total cholesterol, triglycerides, and high-density lipoprotein cholesterol were measured using standard enzymatic procedures. Low-density lipoprotein cholesterol was calculated or directly measured according to laboratory protocol. Diabetes mellitus was identified by a previous diagnosis, glucose-lowering treatment, or fasting blood glucose ≥126 mg/dL. Dyslipidaemia was defined by abnormal lipid values or lipid-lowering treatment. Metabolic syndrome was diagnosed when at least three harmonised components were present. 7

Statistical analysis: Data were analysed using IBM SPSS Statistics version 26.0. Continuous variables were summarised as mean ± standard deviation and categorical variables as frequency and percentage. Normality was assessed before parametric testing. Independent-samples t-test and one-way analysis of variance were used for continuous comparisons; chi-square or Fisher's exact test was applied to categorical variables. Pearson correlation assessed relationships between serum uric acid and cardiometabolic variables. Multivariable logistic regression estimated adjusted odds ratios with 95% confidence intervals for hypertension. Clinically relevant variables and factors associated in univariable analysis were entered into the model. A two-sided p-value <0.05 was statistically significant. Complete-case analysis was performed.

Ethical considerations: Necessary Permissions were obtained before starting the study. Written informed consent was obtained from each participant, and confidentiality was maintained.

Results

A total of 86 adults were assessed for eligibility. Six individuals were excluded because they did not satisfy the eligibility criteria or had incomplete biochemical data, leaving 80 participants for analysis. The mean age was 49.2 ± 11.8 years (range, 25-72 years), and 46 (57.5%) participants were males. The mean body mass index and waist circumference were 26.8 ± 4.3 kg/m² and 92.1 ± 11.6 cm, respectively. Hypertension was present in 36 (45.0%), hyperuricaemia in 29 (36.3%), dyslipidaemia in 35 (43.8%), and metabolic syndrome in 28 (35.0%). The complete baseline profile is presented in Table 1.

Table 1. Baseline demographic, clinical, and biochemical characteristics (n=80)

Characteristic	Frequency/Mean	Percentage
Age, years, mean ± SD	49.2 ± 11.8	–
Age ≥50 years	41	51.3
Male sex	46	57.5
Female sex	34	42.5
Body mass index, kg/m², mean ± SD	26.8 ± 4.3	–
Normal body mass index	25	31.3
Overweight	31	38.8
Obesity	24	30.0
Waist circumference, cm, mean ± SD	92.1 ± 11.6	–
Central obesity	38	47.5
Systolic blood pressure, mmHg, mean ± SD	134.8 ± 18.6	–
Diastolic blood pressure, mmHg, mean ± SD	84.1 ± 10.7	–
Hypertension	36	45.0
Diabetes mellitus	20	25.0
Dyslipidaemia	35	43.8
Metabolic syndrome	28	35.0

Current smoking	18	22.5
Serum uric acid, mg/dL, mean ± SD	6.2 ± 1.6	–
Hyperuricaemia	29	36.3
Fasting blood glucose, mg/dL, mean ± SD	111.6 ± 32.4	–
Total cholesterol, mg/dL, mean ± SD	191.8 ± 39.7	–
Triglycerides, mg/dL, mean ± SD	157.2 ± 68.5	–
High-density lipoprotein cholesterol, mg/dL, mean ± SD	43.6 ± 10.2	–
Low-density lipoprotein cholesterol, mg/dL, mean ± SD	117.3 ± 34.8	–

SD: standard deviation. Values are presented as mean ± SD or number and percentage.

Participants with hypertension had significantly higher serum uric acid concentrations than normotensive participants (7.0 ± 1.5 versus 5.5 ± 1.3 mg/dL; p<0.001). Hyperuricaemia was observed in 22 of 36 hypertensive participants (61.1%) and 7 of 44 normotensive participants (15.9%). Accordingly, the prevalence of hypertension was 75.9% among participants with hyperuricaemia and 27.5% among those with normal serum uric acid. Hyperuricaemia was associated with an eightfold increase in the unadjusted odds of hypertension (odds ratio 8.31; 95% confidence interval 2.80-24.66; p<0.001), as detailed in Table 2.

Table 2. Association between serum uric acid and hypertension

Variable	Normotensive (n=44)	Hypertensive (n=36)	p-value
Serum uric acid, mg/dL, mean ± SD	5.5 ± 1.3	7.0 ± 1.5	<0.001
Hyperuricaemia	7 (15.9%)	22 (61.1%)	<0.001
Normal serum uric acid	37 (84.1%)	14 (38.9%)	–
Systolic blood pressure, mmHg	121.6 ± 8.9	150.9 ± 13.4	<0.001
Diastolic blood pressure, mmHg	76.9 ± 6.8	92.9 ± 7.4	<0.001

SD: standard deviation. Values are mean ± SD or n (%).

Serum uric acid increased progressively across body mass index categories: 5.3 ± 1.2 mg/dL in participants with normal body mass index, 6.1 ± 1.4 mg/dL in overweight participants, and 7.2 ± 1.5 mg/dL in participants with obesity (p<0.001). Participants with central obesity had higher uric acid levels than those without central obesity (7.0 ± 1.5 versus 5.5 ± 1.3 mg/dL; p<0.001). Higher concentrations were also observed with diabetes mellitus, dyslipidaemia, hypertriglyceridaemia, low high-density lipoprotein cholesterol, and metabolic syndrome (Table 3). Hyperuricaemia was present in 18 of 38 participants with central obesity, 16 of 24 with obesity, 12 of 20 with diabetes mellitus, and 19 of 28 with metabolic syndrome.

Table 3. Serum uric acid levels according to metabolic risk factors

Metabolic risk factor	Present, mean ± SD (mg/dL)	Absent, mean ± SD (mg/dL)	p-value
Obesity	7.2 ± 1.5	5.8 ± 1.4	<0.001
Central obesity	7.0 ± 1.5	5.5 ± 1.3	<0.001
Diabetes mellitus	6.9 ± 1.6	5.9 ± 1.5	0.011
Dyslipidaemia	6.8 ± 1.6	5.7 ± 1.4	0.002
Hypertriglyceridaemia	6.9 ± 1.5	5.8 ± 1.5	0.003
Low HDL cholesterol	6.7 ± 1.6	5.8 ± 1.4	0.012
Metabolic syndrome	7.2 ± 1.4	5.6 ± 1.4	<0.001

HDL: high-density lipoprotein; SD: standard deviation.

Serum uric acid demonstrated moderate positive correlations with waist circumference (r=0.51), systolic blood pressure (r=0.48), body mass index (r=0.44), and diastolic blood pressure (r=0.41), with all p-values <0.001. Positive correlations were also observed with triglycerides, fasting blood glucose, and age. An inverse correlation was present with high-density lipoprotein cholesterol (r=−0.29; p=0.009), whereas total cholesterol and low-density lipoprotein cholesterol were not significantly correlated with serum uric acid (Table 4).

Table 4. Correlation of serum uric acid with clinical and biochemical variables

Variable	Correlation coefficient (r)	p-value
Age	0.23	0.041
Body mass index	0.44	<0.001
Waist circumference	0.51	<0.001
Systolic blood pressure	0.48	<0.001
Diastolic blood pressure	0.41	<0.001
Fasting blood glucose	0.28	0.012
Total cholesterol	0.18	0.112
Triglycerides	0.36	0.001
HDL cholesterol	−0.29	0.009
LDL cholesterol	0.20	0.078

HDL: high-density lipoprotein; LDL: low-density lipoprotein. Pearson correlation coefficients are shown.

In multivariable logistic regression, hyperuricaemia remained independently associated with hypertension after adjustment for age, sex, obesity, central obesity, diabetes mellitus, and dyslipidaemia (adjusted odds ratio 4.18; 95% confidence interval 1.29-13.54; p=0.017). Age ≥50 years and central obesity were also independent correlates of hypertension. Male sex, obesity, diabetes mellitus, and dyslipidaemia did not retain statistical significance in the adjusted model (Table 5).

Table 5. Multivariable logistic regression analysis of factors associated with hypertension

Variable	Adjusted odds ratio	95% confidence interval	p-value
Hyperuricaemia	4.18	1.29-13.54	0.017
Age ≥50 years	3.26	1.12-9.48	0.030
Male sex	1.31	0.46-3.72	0.611
Obesity	2.08	0.65-6.64	0.216
Central obesity	3.07	1.01-9.35	0.048
Diabetes mellitus	1.86	0.57-6.08	0.304
Dyslipidaemia	1.64	0.56-4.80	0.365

The dependent variable was hypertension. Odds ratios were mutually adjusted for all variables displayed in the table.

Discussion

The present study identified a clear association between serum uric acid, hypertension, and clustered metabolic risk factors. Hypertensive participants had substantially higher uric acid concentrations than normotensive participants, and hyperuricaemia was almost four times as common in the hypertensive group. Serum uric acid also increased across body mass index categories and was higher among participants with central obesity, diabetes mellitus, dyslipidaemia, hypertriglyceridaemia, low high-density lipoprotein cholesterol, and metabolic syndrome. Correlation analysis demonstrated the strongest relationship with waist circumference, followed by systolic blood pressure, body mass index, and diastolic blood pressure. Importantly, hyperuricaemia remained independently associated with hypertension after multivariable adjustment. These observations agree with the systematic review by Grayson et al., which demonstrated an increased risk of incident hypertension among individuals with hyperuricaemia. 3 Kuwabara et al. similarly reported that elevated uric acid predicted progression from prehypertension to hypertension over five years, independent of conventional risk factors. 4 The graded relationship described in a dose-response meta-analysis further supports the biological consistency of this association. 5 In a cross-sectional study of Bangladeshi adults, Ali et al. found higher serum uric acid among hypertensive participants and positive correlations with both systolic and diastolic blood pressure. 6 The higher unadjusted odds of hypertension in our hyperuricaemic participants, followed by attenuation after covariate adjustment, indicates that uric acid is related to blood pressure both directly and through shared metabolic pathways.

The metabolic findings are also consistent with previous literature. Yuan et al. documented a dose-dependent increase in metabolic-syndrome risk with rising serum uric acid. 8 Studies from Bangladesh and the United States found significant associations of uric acid with abdominal obesity, triglycerides, impaired glucose regulation, and metabolic syndrome. 9,10 A systematic review and meta-analysis confirmed a positive relationship between metabolic syndrome and uric acid across populations. 11 Recent data from elderly Chinese adults likewise demonstrated positive correlations with body mass index, waist circumference, blood pressure, fasting glucose, and triglycerides, alongside an inverse correlation with high-density lipoprotein cholesterol. 14 This pattern closely resembles the correlation profile observed in the present study. Several mechanisms can explain these findings. Intracellular uric acid promotes oxidative stress, endothelial dysfunction, reduced nitric oxide bioavailability, renin-angiotensin activation, and renal arteriolar injury, all of which favour increased vascular resistance and sodium sensitivity. 1,2 Insulin resistance and compensatory hyperinsulinaemia reduce renal urate clearance, while visceral adiposity increases uric acid production and inflammatory activity. Thus, hyperuricaemia can function as both a metabolic marker and a contributor to cardiometabolic dysfunction.

From a clinical perspective, serum uric acid is inexpensive and widely available. Its elevation, particularly when accompanied by central obesity or dyslipidaemia, should prompt comprehensive blood-pressure and metabolic-risk assessment. Nevertheless, the present results establish association rather than causation, and they do not support urate-lowering treatment solely for cardiovascular prevention. Prospective multicentre research is required to clarify temporal pathways and therapeutic implications.

LIMITATIONS

This study has several limitations. Its cross-sectional design prevents determination of temporal or causal relationships. Recruitment from a single tertiary-care hospital limits population-level generalisability and introduces referral bias. The modest sample size produced wide confidence intervals in multivariable analysis. Dietary purine intake, alcohol consumption, renal function, antihypertensive drug classes, and insulin resistance were not quantified in detail. Residual confounding therefore remains possible.

Conclusion

Among adults attending Government Medical College Nizamabad, higher serum uric acid was strongly associated with hypertension and an adverse metabolic profile. Hypertensive participants had substantially greater uric acid concentrations and a higher prevalence of hyperuricaemia than normotensive participants. Serum uric acid also tracked with obesity, central adiposity, dyslipidaemia, triglycerides, fasting glucose, and metabolic syndrome, while showing an inverse relationship with high-density lipoprotein cholesterol. Hyperuricaemia remained independently associated with hypertension after adjustment for major covariates. These findings support serum uric acid as a practical adjunctive marker during cardiometabolic assessment. Larger multicentre prospective studies should determine temporal relationships, population-specific thresholds, and whether urate-directed strategies improve blood-pressure or metabolic outcomes.

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