International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/25-07-15*

Association Between Serum Uric Acid Levels and Severity of Hypertension in Adults: A Cross-Sectional Observational Study.

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Received: **14-06-2025**
Revised: **28-06-2025**
Accepted: **16-07-2025**
Published: **22-07-2025**

Abstract

Background: Serum uric acid is linked to endothelial dysfunction, renal microvascular injury, and elevated blood pressure, but its relationship with hypertension severity remains uncertain. ***Objectives:*** To evaluate the association between serum uric acid and hypertension severity and its correlations with selected clinical variables. ***Methods:*** This hospital-based cross-sectional study included 80 adults with hypertension at GMC/GGH Suryapet, Telangana, India, from January to November 2024. Participants were classified as having Grade 1, Grade 2, or Grade 3 hypertension. Demographic data, comorbidities, anthropometric measurements, blood pressure, and serum uric acid were recorded. Group comparisons used one-way analysis of variance and the chi-square test. Pearson correlation and ordinal logistic regression were applied. ***Results:*** Mean age was 52.7 ± 10.8 years, and 45 (56.3%) participants were male. Grade 1, Grade 2, and Grade 3 hypertension occurred in 31 (38.8%), 29 (36.3%), and 20 (25.0%) participants, respectively. Mean serum uric acid was 6.3 ± 1.4 mg/dL; 34 (42.5%) participants had hyperuricaemia. Levels increased from 5.3 ± 0.9 mg/dL in Grade 1 to 6.4 ± 1.0 mg/dL in Grade 2 and 7.8 ± 1.2 mg/dL in Grade 3 hypertension ($p < 0.001$). Hyperuricaemia prevalence increased from 19.4% to 44.8% and 75.0% across the grades ($p < 0.001$). Serum uric acid correlated with systolic ($r = 0.52$) and diastolic blood pressure ($r = 0.39$). Each 1 mg/dL increase was independently associated with greater hypertension severity (adjusted odds ratio 2.18; 95% confidence interval 1.49-3.19). ***Conclusion:*** Serum uric acid showed a graded, independent association with hypertension severity and can complement cardiovascular risk assessment.

Keywords: Serum uric acid; Hyperuricaemia; Hypertension severity; Blood pressure; Cardiovascular risk.

INTRODUCTION

Hypertension is a leading modifiable determinant of cardiovascular disease, stroke, chronic kidney disease, and premature mortality. Its clinical impact is influenced not only by its presence but also by the magnitude and persistence of blood pressure elevation. Adults with higher grades of hypertension carry a greater probability of target-organ injury and frequently exhibit clustering of metabolic risk factors. Accurate identification of biochemical markers associated with increasing hypertension severity can support earlier risk stratification and guide more comprehensive clinical assessment. Contemporary guidelines classify office blood pressure into graded categories and emphasise repeated, standardised measurement before therapeutic decisions are made.1

Uric acid is the final product of purine metabolism in humans. Serum concentrations reflect the balance between hepatic production, renal and intestinal excretion, dietary purine exposure, cellular turnover, and medication effects. Although uric acid acts as an antioxidant in the extracellular environment, intracellular urate accumulation and xanthine oxidase activity can promote oxidative stress, endothelial dysfunction, inflammation, and reduced nitric oxide bioavailability. Experimental and clinical evidence also links hyperuricaemia with activation of the renin-angiotensin-aldosterone system, renal afferent arteriolar injury, sodium retention, and vascular smooth-muscle proliferation. These pathways provide biological plausibility for an association between serum uric acid and elevated blood pressure.2-4 Several prospective cohorts have reported that higher serum uric acid precedes the development of hypertension, even after adjustment for age, adiposity, renal function, and metabolic factors.5-9 Systematic reviews and meta-analyses have further demonstrated a dose-responsive increase in incident hypertension across rising uric acid concentrations.10-12 However, uric acid is closely related to obesity, diabetes mellitus, dyslipidaemia, renal impairment, alcohol exposure, dietary habits, and diuretic therapy.

Consequently, uncertainty persists regarding whether uric acid functions as a causal mediator, a marker of metabolic and renal dysfunction, or both. The association can also vary with sex, age, ethnicity, treatment pattern, and the clinical setting in which patients are studied.

Most published work has focused on the occurrence of hypertension rather than the relationship between serum uric acid and the graded severity of established hypertension. Evidence from hospital-based Indian populations remains limited, despite the high burden of hypertension and metabolic comorbidity. Evaluating this relationship in routine clinical practice can clarify whether serum uric acid rises progressively across blood pressure categories and whether the association persists after accounting for selected confounders. Therefore, the present study aimed to determine the association between serum uric acid levels and severity of hypertension among adults attending GMC/GGH Suryapet, Telangana, India. The secondary objectives were to estimate the prevalence of hyperuricaemia, compare serum uric acid across hypertension grades, assess its correlations with systolic blood pressure, diastolic blood pressure, body mass index, age, and duration of hypertension, and examine its independent association with greater hypertension severity.

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 and Government General Hospital (GMC/GGH), Suryapet, Telangana, India, from January 2024 to November 2024.

Study participants: Adults aged 18 years or older with previously diagnosed or newly detected essential hypertension were screened consecutively. Patients were included when complete clinical examination, blood pressure measurements, and serum uric acid results were available. Exclusion criteria were secondary hypertension, acute coronary or cerebrovascular events, decompensated heart failure, active infection, malignancy, pregnancy, gout, chronic liver disease, estimated glomerular filtration rate below 60 mL/min/1.73 m², current urate-lowering therapy, and recent thiazide or loop-diuretic exposure. Eighty eligible participants were enrolled by consecutive sampling. The sample size was calculated using the single-proportion formula, assuming 40% expected hyperuricaemia prevalence, 95% confidence level, and 11% absolute precision. The minimum calculated sample was 77 and was rounded to 80 participants.

Clinical assessment: Demographic information, smoking status, duration of hypertension, diabetes mellitus, dyslipidaemia, medication history, and relevant clinical history were recorded using a structured case record form. Height and weight were measured with participants wearing light clothing and no footwear. Body mass index was calculated as weight in kilograms divided by height in metres squared. Blood pressure was measured in the seated position after five minutes of rest using a validated calibrated device and an appropriately sized cuff. Two readings were obtained one to two minutes apart, and their average was analysed. Hypertension was categorised as Grade 1 (systolic 140-159 mmHg and/or diastolic 90-99 mmHg), Grade 2 (160-179 mmHg and/or 100-109 mmHg), or Grade 3 (≥180 mmHg and/or ≥110 mmHg), consistent with European guideline categories; the higher grade was assigned when systolic and diastolic values differed.1
Biochemical assessment: A fasting venous blood sample was collected under aseptic precautions. Serum uric acid was measured by the uricase-peroxidase enzymatic method in the hospital central laboratory under internal quality-control procedures. Hyperuricaemia was defined as serum uric acid >7.0 mg/dL in men and >6.0 mg/dL in women. Available fasting glucose, lipid profile, and serum creatinine findings were reviewed to characterise comorbidity and confirm eligibility.

Statistical analysis: Data were analysed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequency and percentage. Normality was assessed using the Shapiro-Wilk test. Mean values across hypertension grades were compared using one-way analysis of variance with Tukey post hoc testing. The chi-square test evaluated categorical associations, and Pearson correlation assessed relationships between serum uric acid and continuous variables. Ordinal logistic regression estimated the association between serum uric acid and hypertension severity after adjustment for age, sex, body mass index, duration of hypertension, and diabetes mellitus. A two-sided p-value <0.05 was considered statistically significant.

Ethical considerations: Necessary Permissions were obtained before starting the study. Written informed consent was obtained from every participant before enrolment.

RESULTS

A total of 80 adults with hypertension were included in the final analysis, and complete demographic, clinical, blood pressure, and biochemical data were available for every participant. The mean age was 52.7 ± 10.8 years (range, 28-74 years), and 45 (56.3%) participants were aged 40-59 years. There were 45 (56.3%) males and 35 (43.8%) females. The mean body mass index was 27.1 ± 4.3 kg/m², while the mean duration of hypertension was 6.8 ± 5.2 years. Diabetes mellitus, dyslipidaemia, and current smoking were documented in 24 (30.0%), 29 (36.3%), and 18 (22.5%) participants,

respectively. Grade 1, Grade 2, and Grade 3 hypertension were present in 31 (38.8%), 29 (36.3%), and 20 (25.0%) participants. Mean systolic and diastolic blood pressures were 160.4 ± 18.6 mmHg and 99.1 ± 9.8 mmHg, respectively. The mean serum uric acid level was 6.3 ± 1.4 mg/dL, and hyperuricaemia was present in 34 (42.5%) participants (Table 1).

Table 1. Demographic and clinical characteristics of the study participants (N = 80)

Characteristic	Category/measurement	Frequency or mean	Percentage
Age, years	Mean ± SD	52.7 ± 10.8	—
Age group	18-39 years	12	15.0
	40-59 years	45	56.3
	≥60 years	23	28.8
Sex	Male	45	56.3
	Female	35	43.8
Body mass index, kg/m²	Mean ± SD	27.1 ± 4.3	—
Duration of hypertension, years	Mean ± SD	6.8 ± 5.2	—
Diabetes mellitus	Present	24	30.0
Dyslipidaemia	Present	29	36.3
Current smoking	Present	18	22.5
Hypertension severity	Grade 1	31	38.8
	Grade 2	29	36.3
	Grade 3	20	25.0
Systolic blood pressure, mmHg	Mean ± SD	160.4 ± 18.6	—
Diastolic blood pressure, mmHg	Mean ± SD	99.1 ± 9.8	—
Serum uric acid, mg/dL	Mean ± SD	6.3 ± 1.4	—
Hyperuricaemia	Present	34	42.5

Serum uric acid increased progressively across the hypertension severity categories. The mean level was 5.3 ± 0.9 mg/dL in Grade 1 hypertension, 6.4 ± 1.0 mg/dL in Grade 2 hypertension, and 7.8 ± 1.2 mg/dL in Grade 3 hypertension. The between-group difference was statistically significant ($F = 36.85$, $p < 0.001$). Participants with greater hypertension severity were also older and had higher body mass index values and a longer duration of hypertension. The distributions of systolic and diastolic blood pressure were consistent with the predefined grade categories (Table 2).

Table 2. Comparison of clinical variables according to hypertension severity

Variable	Grade 1 hypertension (n = 31)	Grade 2 hypertension (n = 29)	Grade 3 hypertension (n = 20)	p-value
Age, years	49.1 ± 10.2	53.4 ± 10.0	57.1 ± 11.2	0.031
Body mass index, kg/m²	25.8 ± 3.8	27.2 ± 4.0	29.0 ± 4.8	0.026
Duration of hypertension, years	4.1 ± 3.2	6.8 ± 4.4	11.0 ± 5.9	<0.001
Systolic blood pressure, mmHg	145.3 ± 4.5	159.8 ± 5.9	184.6 ± 9.8	<0.001
Diastolic blood pressure, mmHg	91.2 ± 3.4	99.2 ± 4.8	111.1 ± 6.5	<0.001
Serum uric acid, mg/dL	5.3 ± 0.9	6.4 ± 1.0	7.8 ± 1.2	<0.001

Values are expressed as mean ± standard deviation. Comparisons were performed using one-way analysis of variance.

Tukey post hoc analysis showed that serum uric acid was significantly higher in Grade 2 than in Grade 1 hypertension ($p = 0.002$). Participants with Grade 3 hypertension had significantly higher serum uric acid than those with either Grade 1 or Grade 2 hypertension (both $p < 0.001$). The prevalence of hyperuricaemia increased steadily with hypertension severity. It was present in 6 (19.4%) participants with Grade 1 hypertension, 13 (44.8%) with Grade 2 hypertension, and 15 (75.0%) with Grade 3 hypertension. The association between hyperuricaemia and hypertension grade was statistically significant ($\chi^2 = 15.50$, $p < 0.001$). Only 5 (25.0%) participants with Grade 3 hypertension had serum uric acid within the reference range, compared with 25 (80.6%) participants with Grade 1 hypertension (Table 3).

Table 3. Association between hyperuricaemia and severity of hypertension

Serum uric acid status	Grade 1, n (%)	Grade 2, n (%)	Grade 3, n (%)	Total
Normal serum uric acid	25 (80.6)	16 (55.2)	5 (25.0)	46
Hyperuricaemia	6 (19.4)	13 (44.8)	15 (75.0)	34
Total	31	29	20	80

Percentages are calculated within each hypertension grade. Chi-square value = 15.50; p < 0.001.

Correlation analysis demonstrated a moderate positive relationship between serum uric acid and systolic blood pressure (r = 0.52, p < 0.001). Positive correlations were also observed with diastolic blood pressure (r = 0.39, p < 0.001), duration of hypertension (r = 0.31, p = 0.005), and body mass index (r = 0.25, p = 0.025). The correlation with age was weak and not statistically significant (r = 0.17, p = 0.132) (Table 4).

Table 4. Correlation of serum uric acid with selected clinical variables

Variable	Correlation coefficient (r)	p-value
Systolic blood pressure	0.52	<0.001
Diastolic blood pressure	0.39	<0.001
Duration of hypertension	0.31	0.005
Body mass index	0.25	0.025
Age	0.17	0.132

In ordinal logistic regression, each 1 mg/dL increase in serum uric acid was associated with more than twofold higher odds of belonging to a more severe hypertension category after adjustment for age, sex, body mass index, duration of hypertension, and diabetes mellitus (adjusted odds ratio 2.18; 95% confidence interval 1.49-3.19; p < 0.001). Longer duration of hypertension was independently associated with greater severity (adjusted odds ratio per year 1.11; 95% confidence interval 1.02-1.21; p = 0.015). Age, sex, body mass index, and diabetes mellitus were not independently significant in the adjusted model. Thus, serum uric acid demonstrated a significant graded and independent association with hypertension severity.

DISCUSSION

The present study demonstrated a clear graded association between serum uric acid and hypertension severity. Mean serum uric acid increased from 5.3 mg/dL in Grade 1 hypertension to 6.4 mg/dL in Grade 2 and 7.8 mg/dL in Grade 3 hypertension. Hyperuricaemia was identified in 42.5% of participants, and its prevalence rose from 19.4% in Grade 1 to 75.0% in Grade 3 hypertension. Serum uric acid also correlated positively with systolic and diastolic blood pressure. After adjustment for age, sex, body mass index, duration of hypertension, and diabetes mellitus, every 1 mg/dL increase in serum uric acid was associated with more than twice the odds of belonging to a higher hypertension category.

These findings are consistent with longitudinal cohort evidence showing that elevated serum uric acid precedes and predicts the development of hypertension. Mellen et al. reported an independent association between serum uric acid and incident hypertension in the Atherosclerosis Risk in Communities cohort, while Perlstein et al. observed a similar relationship in the Normative Aging Study.6,7 Forman et al. and Krishnan et al. also identified increased hypertension risk across higher uric acid levels in adult men.8,9 In a South Asian population, Ali et al. reported a significant cross-sectional relationship between serum uric acid and hypertension, supporting the relevance of this association across diverse ethnic and clinical settings.5

The progressive rise observed across hypertension grades is further supported by pooled evidence. Grayson et al. found that hyperuricaemia was associated with a higher risk of incident hypertension, and subsequent meta-analyses demonstrated a dose-response pattern between serum uric acid concentration and hypertension risk.10-12 The stronger correlation with systolic than diastolic blood pressure in the present study could reflect the combined influence of arterial stiffness, vascular remodelling, and age-related haemodynamic changes. The positive relationships with body mass index and duration of hypertension also indicate metabolic clustering and cumulative vascular exposure.

Several mechanisms can explain these observations. Intracellular urate and xanthine oxidase activity promote oxidative stress, reduce endothelial nitric oxide, stimulate vascular smooth-muscle proliferation, and activate inflammatory pathways. Uric acid has also been linked to renin-angiotensin system activation, renal microvascular injury, impaired pressure natriuresis, and sodium retention.2-4 These processes can increase vascular resistance and sustain blood pressure elevation. Experimental and adolescent interventional studies have shown blood pressure reductions after urate-lowering treatment, strengthening biological plausibility.13,14 However, those trials involved young patients and do not establish that pharmacological treatment of asymptomatic hyperuricaemia improves outcomes in hypertensive adults.

Clinically, serum uric acid is inexpensive and widely available. Its measurement can complement assessment of obesity, diabetes, dyslipidaemia, renal function, and medication exposure in adults with severe or difficult-to-control hypertension. Nevertheless, the present findings should be interpreted as an association rather than proof of causation. Prospective multicentre studies with repeated uric acid measurements and detailed assessment of renal function, diet, alcohol intake,

and antihypertensive therapy are required before serum uric acid can be adopted as a therapeutic target for blood pressure control.

LIMITATIONS

This study has several limitations. Its single-centre cross-sectional design prevents determination of temporal or causal relationships. The sample was modest and drawn from a tertiary-care population, limiting generalisability. Dietary purine intake, alcohol consumption, antihypertensive drug exposure, renal function gradients, and menopausal status were not evaluated in detail. A single serum uric acid measurement was used, creating scope for biological and laboratory variability.

CONCLUSION

Serum uric acid demonstrated a significant graded association with the severity of hypertension in this hospital-based adult population. Participants with Grade 3 hypertension had the highest mean uric acid concentration and the greatest prevalence of hyperuricaemia. Positive correlations were observed with systolic blood pressure, diastolic blood pressure, body mass index, and duration of hypertension. The association with hypertension severity remained significant after adjustment for selected demographic and clinical factors. These findings support inclusion of serum uric acid in the metabolic and cardiovascular assessment of adults with hypertension. Longitudinal studies are required to clarify temporality, confirm causal pathways, and determine whether uric acid reduction improves blood pressure control or cardiovascular outcomes.

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