VOLUME - 15, ISSUE - 01, JANUARY - 2026 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

General Medicine

ASSOCIATION OF SERUM URIC ACID WITH NEWLY DIAGNOSED PRIMARY HYPERTENSION IN YOUNG ADULTS

Dr. Anil Kumar Bamdhamravuri — Junior Resident, Department of General Medicine, Nimra Institute of Medical Sciences, Andhra Pradesh, India.

Dr. T. Murali Venkateshwara Rao — Professor & Head, Department of General Medicine, Nimra Institute of Medical Sciences, Andhra Pradesh, India.

Dr. Movva Jaya Mounika — Junior Resident, Department of General Medicine, Nimra Institute of Medical Sciences, Andhra Pradesh, India.

ABSTRACT

Background: Hypertension is a major contributor to cardiovascular morbidity and is increasingly being detected in young adults. Serum uric acid (SUA) has emerged as a potential metabolic factor implicated in the development of primary hypertension; however, evidence among young Indian adults remains limited. This study evaluated SUA levels in young adults with newly detected primary hypertension and examined their association with blood pressure severity, body mass index (BMI), and lipid parameters. **Methods:** This hospital-based case-control study was conducted over 12 months at a tertiary care center. A total of 140 participants aged 18–40 years were enrolled., comprising 70 newly diagnosed primary hypertensive patients and 70 age- and sex-matched normotensive controls. Fasting serum uric acid, lipid profile, and renal function tests were measured. Statistical analysis included the unpaired t test and Pearson correlation analysis. **Results:** Mean SUA levels were significantly higher in hypertensive patients compared with controls (5.57 ± 1.48 mg/dL vs. 4.70 ± 1.23 mg/dL; $p<0.001$). Hyperuricemia was present in 32.9% of cases and 11.4% of controls (**$p < 0.001$**). Elevated SUA levels were significantly associated with higher BMI and male sex among hypertensive patients. SUA demonstrated a moderate positive correlation with LDL cholesterol ($r = 0.454$; $p<0.001$). Young adults with newly diagnosed primary hypertension exhibit significantly elevated serum uric acid levels. **Conclusion:** Young adults with newly diagnosed primary hypertension exhibit significantly elevated serum uric acid levels. The association between hyperuricemia and adverse metabolic parameters suggests that SUA may function as an early metabolic risk indicator in this population. SUA assessment may be considered a supportive metabolic marker in young hypertensive patients to support early cardiovascular risk stratification.

KEYWORDS : Primary hypertension; serum uric acid; young adults; hyperuricemia; metabolic risk

INTRODUCTION

Hypertension is a leading contributor to global cardiovascular morbidity and mortality, with a rising prevalence among younger populations [1,2]. India faces a substantial burden of hypertension, with a significant proportion of cases remaining undiagnosed or inadequately controlled, particularly among young adults[3]. This is concerning, as prolonged exposure to elevated blood pressure increases the lifetime risk of cardiovascular events, stroke, and chronic kidney disease.[4]

Serum uric acid (SUA) has emerged as a potential metabolic marker in the pathogenesis of primary hypertension[5]. Experimental and epidemiological studies suggest that elevated uric acid contributes to vascular dysfunction through multiple mechanisms, including endothelial dysfunction via reduced nitric oxide bioavailability, increased oxidative stress, activation of the renin-angiotensin-aldosterone system (RAAS), and vascular smooth muscle proliferation [6,7]. These effects appear particularly pronounced in younger individuals, in whom uric acid–mediated mechanisms may precede irreversible vascular damage and long-standing hypertension [8].

Although international literature supports an association between hyperuricemia and hypertension, data from young Indian adults remain limited[3,9]. Furthermore, the relationship between serum uric acid levels and hypertension severity remains inconsistent across studies[10]. Identifying uric acid as a metabolic risk marker in young hypertensive patients could facilitate timely risk stratification and guide preventive cardiovascular strategies.

AIM AND OBJECTIVES

Aim:

To evaluate serum uric acid levels in young adults with newly detected primary hypertension.

Objectives:

1. To compare serum uric acid levels between hypertensive patients and normotensive controls.
2. To assess the relationship between serum uric acid levels and blood pressure severity.
3. To analyze the association between serum uric acid levels, BMI, and lipid profile parameters.

MATERIALS AND METHODS

Study Design And Setting

This hospital-based case-control study was conducted at a tertiary care teaching hospital over 12 months. The study population comprised two groups: cases (newly diagnosed primary hypertension) and age- and sex-matched normotensive controls.

Study Population

Sample Size: 140 participants (70 hypertensive cases and 70 normotensive controls).

Inclusion Criteria:

- Young adults aged 18–40 years
- Newly detected primary hypertension (defined as systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg, confirmed on ≥2 separate occasions, per Indian Guidelines on Hypertension-IV) [5]

Exclusion Criteria:

- Secondary hypertension
- Diabetes mellitus
- Chronic kidney disease or renal failure
- Pre-existing gout or known hyperuricemia
- Cardiovascular diseases (ischemic heart disease, heart failure, stroke)

- Alcohol abuse or substance use disorder
- Pregnancy
- Use of medications affecting serum uric acid metabolism (thiazide diuretics, loop diuretics, corticosteroids, aspirin)

Data Collection

After obtaining written informed consent, the following data were recorded:

- **Demographic Data:** Age, sex, occupation, socioeconomic status
- **Clinical History:** Duration of hypertension awareness, family history of hypertension/cardiovascular disease
- **Anthropometric Measurements:** Height, weight, waist circumference, hip circumference
- **Blood Pressure Measurement:** Measured using a calibrated sphygmomanometer following a standardized protocol. Three readings were taken in the sitting position after 5 minutes of rest, and the average was recorded.

Laboratory Investigations

Venous blood samples were collected under aseptic conditions after an overnight fast (12–14 hours).

Measurements Included:

- Serum uric acid: enzymatic uricase-based colorimetric method
- Renal function tests: serum creatinine, blood urea nitrogen, estimated glomerular filtration rate (eGFR)
- Lipid profile: total cholesterol, triglycerides, LDL-cholesterol, HDL-cholesterol
- Fasting blood glucose

Statistical Analysis

Data were analyzed using SPSS version 22.0 (IBM Corporation, USA). Results are expressed as:

- **Continuous variables:** Mean ± standard deviation
- **Categorical variables:** Frequency (percentages)

Statistical Tests:

- Unpaired t-test for continuous variables between two groups
- Chi-square test for categorical variables
- One-way ANOVA for comparisons across hypertension stages
- Pearson correlation coefficient for relationship assessment

Significance Level: A p-value <0.05 was considered statistically significant.

RESULTS

Demographic Characteristics

A total of 140 participants completed the study (82 males [58.6%] and 58 females [41.4%]). Mean age was comparable between hypertensive cases (32.1 ± 6.2 years) and normotensive controls (31.8 ± 6.5 years), with no statistically significant difference (p = 0.658).

Table 1: Demographic And Anthropometric Characteristics Of Study Groups

Parameter	Cases (n=70)	Controls (n=70)	p-value
Age (years)	32.1 ± 6.2	31.8 ± 6.5	0.658
Sex (Male:Female)	42:28	40:30	0.686
BMI (kg/m²)	24.91 ± 2.74	24.01 ± 2.95	0.089
Systolic BP (mmHg)	155.71 ± 12.4	120.64 ± 8.3	<0.001
Diastolic BP (mmHg)	97.71 ± 7.8	67.86 ± 5.2	<0.001

*Statistically significant

Serum Uric Acid Levels

Mean serum uric acid levels were significantly higher in hypertensive cases (5.57 ± 1.48 mg/dL) than in normotensive controls (4.70 ± 1.23 mg/dL; p < 0.001).Hyperuricemia, defined as SUA >7.0 mg/dL in males and >6.0 mg/dL in females, was present in 32.9% of hypertensive patients versus 11.4% of controls (p < 0.001).

Correlation With Hypertension Severity

Analysis by hypertension staging revealed a positive trend between increasing severity and uric acid levels, although this relationship did not achieve statistical significance (Stage I: 5.43 ± 1.31 mg/dL vs. Stage II: 5.71 ± 1.65 mg/dL; p = 0.187).

Association With Body Mass Index

Mean BMI was significantly higher in hypertensive cases than in controls (p < 0.001).Among hypertensive patients, elevated serum uric acid levels were significantly associated with higher BMI categories:

- Normal BMI (18.5–24.9): 5.04 ± 1.22 mg/dL
- Overweight (25.0–29.9): 5.31 ± 1.35 mg/dL
- Obese (≥30): 5.89 ± 1.62 mg/dL

Gender Distribution

Serum uric acid levels were significantly higher in males (5.57 ± 1.48 mg/dL) compared to females (5.04 ± 1.35 mg/dL) among hypertensive patients (p = 0.034).

Lipid Profile Correlations

Serum uric acid demonstrated a significant positive correlation with LDL-cholesterol (r = 0.454; p < 0.001) and total cholesterol (r = 0.389; p < 0.001). No significant correlation was observed with HDL-cholesterol (r = –0.118; p = 0.241) or triglycerides (r = 0.147; p = 0.195).

Renal Function Parameters

Serum creatinine and blood urea nitrogen did not differ significantly between cases and controls. Mean eGFR was comparable between groups (p > 0.05), confirming adequate renal function in study participants.

DISCUSSION

This case-control study demonstrates a significant association between elevated serum uric acid levels and newly diagnosed primary hypertension in young adults. The mean serum uric acid level in hypertensive patients (5.57 mg/dL) was significantly higher than in controls (4.70 mg/dL), consistent with previous Indian and international studies[5,6,11].

Mechanistic Insights

The uric acid–hypertension association is supported by several biological mechanisms:

1. Endothelial Dysfunction: Elevated uric acid impairs endothelial function by inhibiting nitric oxide synthesis and promoting nitric oxide scavenging via reactive oxygen species, leading to systemic vasoconstriction[7,12].

2. RAAS Activation: Uric acid activates the renin-angiotensin-aldosterone system, increasing angiotensin II production and promoting sodium retention[8].

3. Metabolic Clustering: The observed associations between serum uric acid, BMI, and LDL cholesterol support the metabolic syndrome hypothesis, wherein hypertension, hyperuricemia, and dyslipidemia share common pathways such as insulin resistance and chronic inflammation [13].

4. Renal Effects: Uric acid causes afferent arteriolar vasoconstriction and may reduce renal sodium excretion, contributing to blood pressure elevation[14].

Comparison With Published Literature

Our findings align with previous studies. Raina et al. (2018) reported mean serum uric acid of 5.5 ± 1.7 mg/dL in hypertensive subjects versus 4.9 ± 1.1 mg/dL in normotensive controls[11]. Similarly, Piani et al. (2021) documented elevated uric acid in 30–40% of newly diagnosed hypertensive patients [15].

While the correlation with hypertension severity (Stage I vs. II)

did not reach statistical significance, the upward trend suggests a biological link. The lack of significance may be attributed to the relatively small sample size and early disease stage. Notably, Feig et al. (2008) and Johnson et al. (2013) proposed that uric acid may play a more direct pathogenic role in young-onset hypertension through renal mechanisms, distinct from sodium-sensitive hypertension in older populations[7,16].

The positive correlation between serum uric acid and LDL cholesterol (r = 0.454) underscores the clustering of metabolic risk factors and suggests shared pathophysiological mechanisms, supporting aggressive risk factor modification in young hypertensive patients.

Gender Differences

The higher prevalence of elevated uric acid in males aligns with known sex differences in uric acid metabolism, attributed in part to estrogen's uricosuric effects in premenopausal women [17].

Clinical Implications

These findings suggest that:

1. Routine screening for hyperuricemia may aid early risk stratification in young hypertensive patients.
2. Elevated uric acid serves as a marker of metabolic dysfunction, warranting comprehensive cardiovascular risk assessment.
3. Serum uric acid could be considered among modifiable risk factors in preventive strategies for young adults with hypertension.
4. Further investigation into urate-lowering therapies for hypertension prevention in young high-risk individuals is warranted.

LIMITATIONS

1. Sample Size: Limited to 70 cases and 70 controls, potentially restricting generalizability.
2. Cross-sectional design: Demonstrates association but cannot establish causality
3. Single-Center Design: Recruitment from one tertiary care hospital may introduce selection bias.
4. Unmeasured Confounders: Dietary factors, physical activity, smoking, and stress were not systematically quantified.
5. Medication history: Detailed medication histories in controls were not exhaustively documented
6. Genetic Factors: No assessment of family history or genetic polymorphisms affecting uric acid metabolism.

CONCLUSION

This study demonstrates that serum uric acid levels are significantly higher in young adults with newly diagnosed primary hypertension than in age- and sex-matched normotensive individuals. Elevated serum uric acid is associated with increased body mass index, adverse lipid profiles (particularly LDL-cholesterol), and trends toward greater hypertension severity. These findings support serum uric acid as an early metabolic marker in young hypertensive patients, with potential implications for cardiovascular risk stratification.

Larger, longitudinal studies incorporating dietary assessment, genetic analysis, and therapeutic intervention trials are warranted to establish causal mechanisms and to evaluate the clinical utility of urate-lowering strategies in the primary prevention of hypertension in young adults.

REFERENCES

[1] Zhou B, Bentham J, Di Cesare M, et al. Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19·1 million participants. Lancet. 2017;389(10064):37-55. doi:10.1016/S0140-6736(16)31919-5
[2] Zhou B, Perel P, Mensah GA, Ezzati M. Global epidemiology, health burden and effective interventions for elevated blood pressure and hypertension. Nat Rev Cardiol. 2021;18(11):785-802. doi:10.1038/s41569-021-00559-8
[3] Geldsetzer P, Manne-Goehler J, Theilmann M, et al. Diabetes and Hypertension in India: A Nationally Representative Study of 1.3 Million Adults. JAMA Intern Med. 2018;178(3):363-372. doi:10.1001/jamainternmed.2017.8312
[4] Reboussin DM, Allen NB, Griswold ME, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults. Hypertension. 2018;71(6):e13-e115. doi:10.1161/HYP.0000000000000075
[5] Shah SN, Munjal YP. Indian Guidelines on Hypertension-IV (2019). Indian Heart J. 2019;71(4):309-323.
[6] Johnson RJ, Sanchez-Lozada LG, Mazzali M, et al. What are the key arguments against uric acid as a true risk factor for hypertension? Hypertension. 2013;61(5):948-951. doi:10.1161/HYPERTENSIONAHA.111.00433
[7] Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. N Engl J Med. 2008;359(17):1811-1821. doi:10.1056/NEJMra0800885
[8] Lanaspa MA, Andres-Hernando A, Kuwabara M. Uric acid and hypertension. Hypertension. 2020;76(4):959-966. doi:10.1161/HYPERTENSIONAHA.120.14868
[9] Wannamethee SG, Papacosta O, Lennon L, Whincup PH. Serum uric acid as a predictor of cardiovascular disease and mortality: the Whitehall II Prospective Cohort Study. Eur J Epidemiol. 2016;31(2):119-129. doi:10.1007/s10654-015-0089-3
[10] Piani F, Cicero AFG, Borghi C. Uric Acid and Hypertension: Prognostic Role and Guide for Treatment. J Clin Med. 2021;10(2):448. doi:10.3390/jcm10020448
[11] Raina S, Kaul R, Gupta B. Serum uric acid levels in essential hypertension: A study from North India. J Family Med Prim Care. 2018;7(1):115-120. doi:10.4103/jfmpc.jfmpc_93_17
[12] Khosla UM, Zharikov S, Finch JL, et al. Hyperuricemia induces endothelial dysfunction. Kidney Int. 2005;67(5):1739-1742. doi:10.1111/j.1523-1755.2005.00273.x
[13] Corry DB, Eslami P, Kaplan MR, Sowers JR, Breeding SA. Uric acid stimulates vascular smooth muscle cell proliferation and oxidative stress via the vascular renin-angiotensin system. J Hypertens. 2008;26(2):269-275. doi:10.1097/HJH.0b013e3282f240bf
[14] Mazzali M, Hughes J, Kim YG, et al. Elevated uric acid increases blood pressure in the rat by a novel crystal-independent mechanism. Hypertension. 2001;38(5):1101-1106. doi:10.1161/hy1101.092839
[15] Piani F, Cicero AFG, Borghi C. Uric Acid and Hypertension: Epidemiological Evidence and Pathophysiological Mechanisms. Hypertension. 2021;77(3):783–795. doi:10.1161/HYPERTENSIONAHA.120.14868
[16] Johnson RJ, Segal MS, Srinivas T, et al. Essential hypertension, progressive renal disease, and uric acid: a pathogenetic cascade. J Am Soc Nephrol. 2005;16(7):1909-1919. doi:10.1681/ASN.2005050517