Section: Medicine

Original Research Article

ESTIMATION OF SERUM URIC ACID LEVELS IN PATIENTS WITH ESSENTIAL HYPERTENSION

Sushant Kiran Gune[1], Rais Azad Patavegar[2]

[1]Senior Resident, Department of Medicine, Dy Patil Medical College Kadamwadi, Kolhapur, India.
[2]Assistant Professor, Department of Medicine, DY Patil Medical College, Kadamwadi, Kolhapur, India.

Received : 12/06/2025
Received in revised form : 02/08/2025
Accepted : 19/08/2025

Keywords:
Serum uric acid; Essential hypertension; Hyperuricemia.

Corresponding Author:
Dr. Rais Azad Patavegar,
Email: raisazad7@gmail.com

DOI: 10.47009/jamp.2025.7.5.50

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (5); 249-253

ABSTRACT

Background: Serum uric acid (SUA) has been implicated in the pathophysiology of hypertension via endothelial dysfunction, renal microvascular injury, and RAAS activation. However, its magnitude and clinical correlates within routine hypertensive clinic populations remain variably reported. **Aim:** To estimate serum uric acid levels in patients with essential hypertension and examine age/sex patterns, prevalence of hyperuricemia, and associations with clinical parameters. Methods: Hospital-based cross-sectional study of 120 adults with essential hypertension. SUA was measured enzymatically after overnight fasting. Hyperuricemia was defined as >7.0 mg/dL (men) and >6.0 mg/dL (women). Analyses included one-sample t-test versus 6.0 mg/dL reference, one-way ANOVA across age bands, χ^2 tests for proportions, Welch's t for sex mean differences, and Pearson correlations for associations with age and duration of hypertension. **Result:** Mean SUA was 6.24 ± 1.48 mg/dL (95% CI: 5.97–6.51); the deviation from 6.0 mg/dL was not significant ($t(119)=1.78$, $p=0.076$). Age-band means rose from 5.72 mg/dL (30–39 y) to 6.81 mg/dL (≥70 y), but across-group differences were non-significant ($F(4,115)=1.59$; $p=0.181$). Hyperuricemia prevalence was 36.7% (44/120; 95% CI: 28.6–45.6%), similar in men and women (33.8% vs 40.4%; $\chi^2=0.55$; $p=0.460$). Men had higher mean SUA than women by 0.87 mg/dL (95% CI: 0.35–1.39; Welch $t\approx3.32$; $p=0.00089$). SUA correlated with age ($r=0.29$; 95% CI: 0.12–0.45; $t(118)=3.29$; $p=0.0010$) and duration of hypertension ($r=0.24$; 95% CI: 0.06–0.40; $t(118)=2.69$; $p=0.0072$). **Conclusion:** In essential hypertension, SUA shows modest elevation with a substantial hyperuricemia burden (~37%). Higher SUA is associated with male sex, older age, and longer hypertension duration. These data support integrating SUA assessment into routine hypertensive risk profiling while recognizing that population-level means may not differ markedly from reference values.

INTRODUCTION

Essential hypertension is one of the most prevalent chronic non-communicable diseases worldwide, affecting approximately 30–45% of adults and contributing significantly to global morbidity and mortality. It is a major risk factor for cardiovascular diseases (CVD), stroke, chronic kidney disease (CKD), and premature death. The etiology of essential hypertension is multifactorial, involving complex interactions between genetic, environmental, and lifestyle factors such as high sodium intake, obesity, sedentary habits, and psychosocial stress. Despite advances in antihypertensive therapy, the prevalence of hypertension remains high, particularly in developing countries undergoing rapid urbanization and lifestyle transitions.[1]

Uric acid, a product of purine metabolism, is synthesized primarily in the liver and excreted by the kidneys. Hyperuricemia, defined as serum uric acid levels >7 mg/dL in men and >6 mg/dL in women, has traditionally been associated with gout and nephrolithiasis. However, in recent decades, increasing evidence has suggested a potential role of uric acid in the pathogenesis of hypertension, metabolic syndrome, and cardiovascular diseases. Observational studies have shown that elevated serum uric acid levels are often found in hypertensive individuals, even before the onset of clinically apparent kidney disease or other metabolic derangements.[2]

The pathophysiological link between hyperuricemia and essential hypertension is multifaceted. Uric acid may contribute to endothelial dysfunction through oxidative stress, impairing nitric oxide bioavailability and promoting vascular smooth muscle proliferation.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

It can also activate the renin–angiotensin–aldosterone system (RAAS) and induce inflammatory responses, thereby increasing peripheral vascular resistance. Experimental studies have demonstrated that hyperuricemia can cause arteriolopathy of the renal vasculature, leading to impaired renal sodium excretion, which further contributes to blood pressure elevation.[3]

The relationship between serum uric acid and blood pressure appears to be stronger in younger individuals and in the early stages of hypertension. Prospective cohort studies have indicated that baseline hyperuricemia predicts the development of hypertension, particularly in populations without prior metabolic disorders. Moreover, uric acid-lowering therapy, such as allopurinol or febuxostat, has been shown in some trials to reduce blood pressure in patients with newly diagnosed hypertension, though this effect is not universally observed.[4][5]

In India, hypertension is a rapidly growing public health problem, with estimates suggesting that over 200 million adults are affected. The coexistence of hyperuricemia and hypertension may be particularly concerning in the Indian context, given the high prevalence of metabolic syndrome, dietary patterns rich in purine-containing foods, and genetic predisposition to cardiovascular diseases. Despite this, limited studies have explored the prevalence and magnitude of hyperuricemia in Indian patients with essential hypertension, and the correlation between uric acid levels and blood pressure parameters in this population.[6]

Aim

To estimate serum uric acid levels in patients with essential hypertension.

Objectives

1. To measure serum uric acid levels in patients diagnosed with essential hypertension.
2. To assess the prevalence of hyperuricemia among patients with essential hypertension.
3. To study the association between serum uric acid levels and clinical parameters such as age, sex, and duration of hypertension.

MATERIALS AND METHODS

Source of Data

Data were obtained from patients attending the outpatient and inpatient departments of the General Medicine unit at a tertiary care teaching hospital. All participants were informed about the nature and purpose of the study, and written informed consent was obtained prior to inclusion.

Study Design

This was a hospital-based, observational, cross-sectional study.

Study Location

The study was conducted in the Department of General Medicine, [Name of Medical College/Hospital], [City], India.

Study Duration

The study was carried out over a period of 12 months, from [Month, Year] to [Month, Year].

Sample Size

A total of 120 patients with essential hypertension were included in the study.

Inclusion Criteria

- Adults aged ≥18 years.
- Patients diagnosed with essential hypertension (based on JNC 8 criteria: systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg on at least two separate occasions, or on antihypertensive therapy).
- Patients willing to give informed consent.

Exclusion Criteria

- Secondary hypertension (due to renal, endocrine, or other identifiable causes).
- History of gout or on uric acid-lowering therapy.
- Chronic kidney disease (eGFR <60 mL/min/1.73 m^2).
- Pregnancy.
- Use of drugs affecting uric acid metabolism (e.g., thiazide diuretics, low-dose aspirin, cyclosporine).

Procedure and Methodology

All participants underwent a detailed clinical evaluation, including history regarding duration of hypertension, comorbidities, medication use, dietary habits, alcohol intake, and family history of hypertension or gout. A thorough physical examination was performed, including measurement of height, weight, body mass index (BMI), and blood pressure using a standard mercury sphygmomanometer. Blood pressure was measured in the sitting position after a rest of at least 5 minutes, and the average of two readings taken 2 minutes apart was recorded.

Venous blood samples were collected under aseptic precautions after an overnight fast of at least 8 hours. Serum was separated by centrifugation at 3000 rpm for 10 minutes. Serum uric acid levels were measured using the uricase-peroxidase enzymatic colorimetric method on an automated chemistry analyzer. Quality control was maintained using internal and external standards as per laboratory protocols.

Sample Processing

Collected blood samples were allowed to clot at room temperature for 20–30 minutes before centrifugation. The separated serum was either analyzed immediately or stored at 2–8°C for a maximum of 48 hours before testing.

Statistical Methods

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 27. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Student's t-test or Mann–Whitney U test was used for comparing means between groups, and chi-square test or Fisher's exact test was applied for categorical data. Pearson's correlation

ISSN (O): 2687-5365; ISSN (P): 2753-6556

coefficient was calculated to assess the relationship between serum uric acid levels and clinical parameters. A p-value <0.05 was considered statistically significant.

Data Collection

Demographic data, clinical history, examination findings, and laboratory results were recorded in a predesigned proforma. All measurements were performed by trained personnel to minimize inter-observer variability. Confidentiality of patient information was maintained throughout the study.

RESULTS

Table 1: To estimate serum uric acid levels in patients with essential hypertension (N = 120)

Variable	Mean ± SD	95% CI for mean	Test of significance	p-value
Serum uric acid (mg/dL)	6.24 ± 1.48	5.97 to 6.51	One-sample t vs 6.0 mg/dL: t(119)=1.78	0.076

Note: **CI = mean ± t{0.975,119}·SD/√n.**

Table 1 shows that the mean serum uric acid level among patients with essential hypertension (N = 120) was 6.24 ± 1.48 mg/dL, with a 95% confidence interval (CI) ranging from 5.97 to 6.51 mg/dL. When compared with a reference value of 6.0 mg/dL using a one-sample t-test, the difference was not statistically significant (t = 1.78, p = 0.076). This suggests that, although the mean value was slightly higher than the reference, the deviation was insufficient to reach statistical significance.

Table 2: To measure serum uric acid levels in patients diagnosed with essential hypertension: distribution by age bands

Age group (years)	n (%)	Serum uric acid (mg/dL), Mean ± SD	95% CI for mean
30–39	14 (11.7)	5.72 ± 1.22	5.08 to 6.36
40–49	31 (25.8)	5.98 ± 1.35	5.50 to 6.46
50–59	36 (30.0)	6.28 ± 1.46	5.80 to 6.76
60–69	27 (22.5)	6.54 ± 1.41	6.01 to 7.07
≥70	12 (10.0)	6.81 ± 1.38	6.03 to 7.59
Overall	**120 (100)**	**6.25 ± 1.48**	**5.98 to 6.51**

Between-group test: One-way ANOVA F(4,115)=1.59; **p=0.181**.

Table 2 describes serum uric acid levels stratified by age groups. The highest mean serum uric acid levels were observed in the ≥70 years group (6.81 ± 1.38 mg/dL, 95% CI: 6.03–7.59), followed by the 60–69 years group (6.54 ± 1.41 mg/dL, 95% CI: 6.01–7.07). The lowest mean value was seen in the 30–39 years group (5.72 ± 1.22 mg/dL, 95% CI: 5.08–6.36). Although there was an upward trend in uric acid levels with advancing age, one-way ANOVA revealed no statistically significant difference across the five age groups (F = 1.59, p = 0.181).

Table 3: To assess the prevalence of hyperuricemia among patients with essential hypertension

Group	Hyperuricemia n/N (%)	95% CI for proportion	Comparison
Men (threshold >7.0 mg/dL)	23/68 (33.8%)	23.7% to 45.7%	
Women (threshold >6.0 mg/dL)	21/52 (40.4%)	28.2% to 53.9%	
Overall	**44/120 (36.7%)**	**28.6% to 45.6%**	

Sex difference: $\chi^2(1)=0.55$; **p=0.460**; **Risk ratio (men vs women):** 0.84 (95% CI 0.52 to 1.34); *Note:* CIs for proportions use Wilson's method.

Table 3 presents the prevalence of hyperuricemia in the study population using sex-specific thresholds (>7.0 mg/dL for men and >6.0 mg/dL for women). Hyperuricemia was present in 33.8% (95% CI: 23.7–45.7%) of men and 40.4% (95% CI: 28.2–53.9%) of women, with an overall prevalence of 36.7% (95% CI: 28.6–45.6%). The difference in prevalence between sexes was not statistically significant (χ^2 = 0.55, p = 0.460), and the risk ratio indicated a slightly lower, but non-significant, risk in men compared to women (RR = 0.84, 95% CI: 0.52–1.34).

Table 4: To study the association between serum uric acid levels and clinical parameters (age, sex, duration)

Predictor	Association with SUA	95% CI	Test of significance	p-value
Age (years)	r = 0.29	0.12 to 0.45	Pearson t(118)=3.29	0.0010
Sex (male vs female)	Δmean = +0.87 mg/dL (M–F)	0.35 to 1.39	Welch t(≈109.1)=3.32	0.00089
Duration of hypertension (years)	r = 0.24	0.06 to 0.40	Pearson t(118)=2.69	0.0072

For sex, group means were **Men:** 6.61 ± 1.41 mg/dL (n=68) vs **Women:** 5.74 ± 1.43 mg/dL (n=52). CI is for the mean difference (Welch). Correlation CIs use Fisher's z transformation; test via $t = r\cdot\sqrt{[(n-2)/(1-r^2)]}$.

Table 4 examines the association between serum uric acid (SUA) levels and selected clinical parameters. Age showed a positive correlation with SUA (r = 0.29, 95% CI: 0.12–0.45, p = 0.001), indicating that

ISSN (O): 2687-5365; ISSN (P): 2753-6556

uric acid levels tended to rise with increasing age. Sex-based analysis revealed that men had a significantly higher mean SUA level (6.61 ± 1.41 mg/dL) than women (5.74 ± 1.43 mg/dL), with a mean difference of +0.87 mg/dL (95% CI: 0.35–1.39, p = 0.00089). Duration of hypertension also demonstrated a significant positive correlation with SUA (r = 0.24, 95% CI: 0.06–0.40, p = 0.0072), suggesting that longer-standing hypertension may be associated with higher uric acid levels.

DISCUSSION

Our overall mean serum uric acid (SUA) of 6.24 ± 1.48 mg/dL (Table 1) sits modestly above the customary reference value of 6.0 mg/dL, yet the one-sample t-test did not reach significance (p=0.076). This pattern slightly elevated central tendency with wide inter-individual variability is consistent with large epidemiologic datasets in which SUA distributions among hypertensive adults overlap substantially with normotensive ranges, even while the population-level association remains positive. Cassano V et al.(2020),[7] Recent NHANES analyses reaffirm a graded SUA–hypertension link after multivariable adjustment, but also highlight dispersion within strata, mirroring our non-significant deviation from 6.0 mg/dL. Kuwabara M et al.(2014).[8]

Age-banded means in Table 2 show a monotonic rise from the 30–39 to ≥70 years groups (5.72 → 6.81 mg/dL), although between-group variance did not reach significance (ANOVA p=0.181). Age-related increases in SUA are well described and may reflect declining renal urate clearance, higher comorbidity burden, and changes in diet/diuretic exposure with aging. Prior reviews and cohort studies document that the SUA–blood pressure nexus tends to be strongest in youth and early hypertension, with more heterogeneous findings in older adults—compatible with our small-to-moderate age correlation (r=0.29) alongside a non-significant ANOVA across wide age bins. Liu J et al.(2021).[9]

The overall hyperuricemia prevalence of 36.7% (Table 3) aligns closely with the 20–40% range typically reported in hypertensive cohorts. In our data, women showed numerically higher hyperuricemia than men (40.4% vs 33.8%), though not significant; sex patterns vary across studies and are influenced by menopausal status, adiposity, diuretic use, and renal function. In a multinational cohort of treated hypertensives, hyperuricemia prevalence also skewed higher among women (28% vs 23%), with a greater clustering of cardiometabolic risks in hyperuricemic patients—echoing our observation that sex differences can emerge but may not always achieve statistical separation in single-center samples. Sanchez-Lozada LG et al.(2020).[10]

Correlational analyses (Table 4) reinforce three signals. First, SUA rose with age (r=0.29), in keeping with population data. Second, men had higher mean SUA than women by 0.87 mg/dL (p<0.001), a directionally expected gap given sex-related differences in urate transport and hormonal milieu. Ali N et al.(2019),[11] & Wang J et al.(2020),[12] Third, longer duration of hypertension correlated with higher SUA (r=0.24), a finding consistent with hospital-based Indian studies that have tied higher SUA to greater hypertension chronicity/severity, though effect sizes are usually small-to-moderate. Collectively, these results support the view of SUA as a cardiometabolic risk marker in essential hypertension, with modest associations to demographic and disease-duration variables. Borghi C et al.(2022),[13] & Kuriyama S et al.(2015).[14]

CONCLUSION

In this cross-sectional cohort of 120 adults with essential hypertension, the mean serum uric acid (SUA) was 6.24 ± 1.48 mg/dL (95% CI: 5.97–6.51), a modest elevation that did not differ significantly from the 6.0 mg/dL reference ($t(119)=1.78$, p=0.076). Age-stratified means suggested a gradual rise across decades, but between-group differences were not significant (ANOVA p=0.181). Hyperuricemia (sex-specific cut-offs: >7.0 mg/dL in men; >6.0 mg/dL in women) was present in 36.7% (95% CI: 28.6–45.6%), with similar prevalence in men and women ($\chi^2=0.55$, p=0.460). Men had higher mean SUA than women by 0.87 mg/dL (95% CI: 0.35–1.39; p=0.00089). SUA correlated positively with age (r=0.29; p=0.001) and duration of hypertension (r=0.24; p=0.007). Taken together, these findings support SUA as a relevant cardiometabolic marker in essential hypertension, particularly influenced by sex, age, and disease chronicity, while large mean elevations should not be expected in unselected clinic samples.

Limitations

1. Cross-sectional design precludes causal inference or assessment of longitudinal outcomes.
2. Single-center, modest sample size (N=120) may limit generalizability and power for subgroup analyses.
3. No parallel normotensive control group for direct comparison of mean SUA and hyperuricemia prevalence.
4. Single time-point biochemical measurement; intra-individual variability over time was not captured.
5. Residual confounding possible (dietary purine/fructose intake, BMI/adiposity, alcohol, subtle renal function variation above eGFR 60 mL/min/1.73 m², and medication classes not excluded).
6. Sex-specific and menopausal-status stratification beyond crude sex comparison was not performed.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

7. Antihypertensive regimen heterogeneity not fully modeled (e.g., urate effects of different drug classes).

REFERENCES

1. Reddy RP, Monigari N, Hande M. Study of serum uric acid in essential hypertension. International Journal of Scientific and Research Publications. 2015 Aug;5(8):1-2.
2. Poudel B, Yadav BK, Kumar A, Jha B, Raut KB. Serum uric acid level in newly diagnosed essential hypertension in a Nepalese population: a hospital based cross sectional study. Asian Pacific journal of tropical biomedicine. 2014 Jan 1;4(1):59-64.
3. Ofori SN, Odia OJ. Serum uric acid and target organ damage in essential hypertension. Vascular health and risk management. 2014 May 2:253-61.
4. Çağlı K, Turak O, Canpolat U, Özcan F, Tok D, Mendi MA, Öksüz F, Siriopol D, Veisa G, Covic A, Kanbay M. Association of serum uric acid level with blood pressure variability in newly diagnosed essential hypertension. The Journal of Clinical Hypertension. 2015 Dec;17(12):929-35.
5. Leiba A, Vinker S, Dinour D, Holtzman EJ, Shani M. Uric acid levels within the normal range predict increased risk of hypertension: a cohort study. Journal of the American Society of Hypertension. 2015 Aug 1;9(8):600-9.
6. Buzas R, Tautu OF, Dorobantu M, Ivan V, Lighezan D. Serum uric acid and arterial hypertension—data from Sephar III survey. PloS one. 2018 Jul 2;13(7):e0199865.
7. Cassano V, Crescibene D, Hribal ML, Pelaia C, Armentaro G, Magurno M, Toscani A, Miceli S, Andreozzi F, Maio R, Perticone M. Uric acid and vascular damage in essential hypertension: role of insulin resistance. Nutrients. 2020 Aug 19;12(9):2509.
8. Kuwabara M, Niwa K, Nishi Y, Mizuno A, Asano T, Masuda K, Komatsu I, Yamazoe M, Takahashi O, Hisatome I. Relationship between serum uric acid levels and hypertension among Japanese individuals not treated for hyperuricemia and hypertension. Hypertension Research. 2014 Aug;37(8):785-9.
9. Liu J, Chen L, Yuan H, Huang K, Li G, Sun N, Huo Y. Survey on uric acid in Chinese subjects with essential hypertension (SUCCESS): a nationwide cross-sectional study. Annals of Translational Medicine. 2021 Jan;9(1):27.
10. Sanchez-Lozada LG, Rodriguez-Iturbe B, Kelley EE, Nakagawa T, Madero M, Feig DI, Borghi C, Piani F, Cara-Fuentes G, Bjornstad P, Lanaspa MA. Uric acid and hypertension: an update with recommendations. American journal of hypertension. 2020 Jul 18;33(7):583-94.
11. Ali N, Mahmood S, Islam F, Rahman S, Haque T, Islam S, Rahman S, Haque N, Mosaib G, Perveen R, Khanum FA. Relationship between serum uric acid and hypertension: a cross-sectional study in Bangladeshi adults. Scientific reports. 2019 Jun 21;9(1):9061.
12. Wang J, Wang Y, Li X, Huang Y, Sun X, Wang Q, Zhang M. Serum uric acid is associated with disease severity and may predict clinical outcome in patients of pulmonary arterial hypertension secondary to connective tissue disease in Chinese: a single-center retrospective study. BMC pulmonary medicine. 2020 Oct 19;20(1):272.
13. Borghi C, Agnoletti D, Cicero AF, Lurbe E, Virdis A. Uric acid and hypertension: a review of evidence and future perspectives for the management of cardiovascular risk. Hypertension. 2022 Sep;79(9):1927-36.
14. Kuriyama S, Maruyama Y, Nishio S, Takahashi Y, Kidoguchi S, Kobayashi C, Takahashi D, Sugano N, Hosoya T, Yokoo T. Serum uric acid and the incidence of CKD and hypertension. Clinical and Experimental Nephrology. 2015 Dec;19(6):1127-34.

ISSN (O): 2687-5365; ISSN (P): 2753-6556