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Research Article

Association of Serum Uric Acid Levels with Cardiovascular Risk Factors among Patients with Hypertension

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ABSTRACT

Background: Hypertension is a major contributor to global cardiovascular morbidity and mortality. Emerging evidence suggests that elevated serum uric acid (SUA) levels may play an important role in the development of cardiovascular complications among hypertensive individuals through mechanisms involving oxidative stress, endothelial dysfunction, and inflammation.
Objectives: To evaluate serum uric acid levels and determines their association with cardiovascular risk factors among patients with hypertension.
Methods
This hospital-based cross-sectional study was conducted among 320 hypertensive patients attending department of general medicine, government general hospital, Nalgonda, from June 2025 to march 2026. Clinical, anthropometric, and biochemical parameters including blood pressure, body mass index (BMI), fasting blood glucose, lipid profile, and serum uric acid levels were assessed. Statistical analysis was performed using IBM SPSS Statistics. Independent t-test, Chi-square test, Pearson correlation, and multivariate logistic regression analysis were applied. A p-value <0.05 was considered statistically significant.
Results: The mean age of participants was 54.8 ± 11.6 years, and 56.9% were males. Hyperuricemia was observed in 36.9% of patients. Elevated SUA levels were significantly associated with higher systolic and diastolic blood pressure, BMI, fasting blood glucose, total cholesterol, LDL-C, and triglyceride levels ($p<0.001$). Significant associations were also observed between hyperuricemia and obesity, diabetes mellitus, dyslipidemia, smoking, sedentary lifestyle, and prolonged duration of hypertension. SUA showed positive correlations with systolic blood pressure ($r=0.412$), BMI ($r=0.468$), fasting blood glucose ($r=0.382$), and triglycerides ($r=0.401$).
Conclusion: Elevated serum uric acid levels were significantly associated with major cardiovascular risk factors among hypertensive patients, suggesting that SUA may serve as a useful biomarker for cardiovascular risk stratification.

Keywords*: Hyperuricemia; Hypertension; Serum Uric Acid; Cardiovascular Risk Factors; Dyslipidemia; Obesity; Metabolic Syndrome.*

INTRODUCTION

Hypertension remains one of the most prevalent non-communicable diseases and constitutes a major contributor to global cardiovascular morbidity and mortality. According to the World Health Organization, approximately 1.28 billion adults worldwide are affected by hypertension, with a substantial proportion remaining undiagnosed or inadequately controlled [1]. Persistent elevation of blood pressure is strongly associated with adverse cardiovascular outcomes, including coronary artery disease, stroke, heart failure, peripheral vascular disease, and chronic kidney disease [2]. Despite considerable advances in antihypertensive therapy and preventive cardiology, cardiovascular complications secondary to hypertension continue to represent a significant public health burden, particularly in low- and middle-income countries. Consequently, identification of additional biomarkers associated with cardiovascular risk in hypertensive individuals has gained increasing clinical importance.

Serum uric acid (SUA), the final metabolic product of purine degradation catalyzed by xanthine oxidase, has emerged as a potentially important biomarker in cardiovascular medicine. Traditionally, hyperuricemia has been primarily associated with gout and renal dysfunction; however, growing epidemiological and experimental evidence suggests that elevated SUA levels may also play a significant role in the pathogenesis of hypertension and cardiovascular disease [3]. Physiologically, uric acid possesses antioxidant properties in extracellular environments. Nevertheless, intracellular accumulation of uric acid may induce oxidative stress, endothelial dysfunction, vascular smooth muscle proliferation, systemic inflammation, and activation of the renin–angiotensin–aldosterone system (RAAS), thereby contributing to vascular injury and hypertension progression [4].

Several biological mechanisms have been proposed to explain the association between hyperuricemia and cardiovascular risk. Elevated uric acid levels can impair nitric oxide bioavailability, resulting in endothelial dysfunction and reduced vasodilatation [5]. Furthermore, uric acid-mediated oxidative stress promotes inflammatory cytokine production and enhances proliferation of vascular smooth muscle cells, leading to arterial stiffness and atherosclerotic changes [6]. Experimental studies have also demonstrated that hyperuricemia may stimulate RAAS activation and renal microvascular damage, which further exacerbates blood pressure elevation and cardiovascular remodeling [7]. These pathophysiological processes suggest that SUA may not merely represent a metabolic byproduct but rather an active participant in cardiovascular disease development.

Numerous observational studies have reported a positive association between elevated SUA levels and hypertension, metabolic syndrome, diabetes mellitus, obesity, dyslipidemia, and adverse cardiovascular outcomes [8,9]. Large cohort studies, including the Framingham Heart Study and the URRAH project, demonstrated that higher SUA concentrations were associated with increased cardiovascular mortality and morbidity [10,11]. Recent Mendelian randomization analyses have further supported the hypothesis of a causal relationship between genetically elevated SUA levels and cardiovascular diseases, including hypertension, myocardial infarction, and heart failure [12]. Additionally, meta-analyses have shown that hyperuricemia significantly increases the risk of coronary artery disease and cardiovascular mortality independent of conventional risk factors [13].

Despite substantial evidence supporting this association, the role of SUA as an independent cardiovascular risk factor remains controversial. Some investigators argue that hyperuricemia may simply reflect the coexistence of established cardiovascular risk factors such as obesity, insulin resistance, renal dysfunction, and metabolic syndrome rather than acting as an independent pathogenic factor [14]. Certain epidemiological studies have failed to demonstrate a consistent independent association between SUA and cardiovascular events after adjustment for confounding variables [15]. Moreover, uncertainty persists regarding whether urate-lowering therapy directly reduces cardiovascular risk or merely improves associated metabolic abnormalities. Consequently, the precise clinical significance of SUA in hypertensive patients continues to be debated.

In developing countries, including India, limited hospital-based studies have comprehensively evaluated the association between SUA levels and multiple cardiovascular risk factors among hypertensive patients. Most available studies involve relatively small sample sizes or focus on isolated metabolic parameters without extensive cardiovascular risk stratification. Furthermore, variations in demographic characteristics, dietary habits, genetic predisposition, and healthcare accessibility necessitate region-specific investigations to better understand the clinical implications of hyperuricemia in hypertensive populations.

The present study was therefore undertaken to evaluate serum uric acid levels and their association with cardiovascular risk factors among patients with hypertension attending a tertiary care hospital. The study aims to determine whether elevated SUA levels are associated with obesity, dyslipidemia, diabetes mellitus, smoking status, and duration of hypertension. It is hypothesized that hypertensive patients with elevated SUA levels exhibit a significantly higher prevalence of cardiovascular risk factors compared with those having normal SUA concentrations. Identification of such associations may facilitate early cardiovascular risk stratification and contribute to improved preventive and therapeutic strategies in hypertensive individuals.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based observational cross-sectional study was conducted in the department of general medicine, government general hospital, Nalgonda, India between June 2025 to march 2026. The study aimed to evaluate serum uric acid (SUA) levels and their association with cardiovascular risk factors among hypertensive patients.

Study Population and Sample Size

A total of 320 adult patients diagnosed with primary hypertension were included in the study. The sample size was calculated using the standard prevalence formula:

$$n=Z^2P(1-P)/d^2$$

assuming a prevalence of hyperuricemia among hypertensive individuals of approximately 28%, 95% confidence

interval, and 5% margin of error. The calculated minimum sample size was 288; however, 320 participants were enrolled to improve statistical power and compensate for incomplete data.[8]

Inclusion and Exclusion Criteria

Patients aged ≥18 years with established or newly diagnosed primary hypertension who provided written informed consent were included. Patients with secondary hypertension, chronic kidney disease, gout, chronic liver disease, malignancy, acute inflammatory disorders, pregnancy, or those receiving urate-lowering drugs or medications affecting uric acid metabolism were excluded.

Data Collection

Demographic and clinical information including age, sex, duration of hypertension, smoking status, alcohol consumption, and physical activity were collected using a structured questionnaire. Anthropometric measurements and biochemical investigations were performed for all participants.

Blood pressure was measured using a calibrated mercury sphygmomanometer according to standard guidelines. Two readings were obtained after 5 minutes of rest, and the average value was recorded. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared.

Fasting venous blood samples were collected after overnight fasting.[11] Serum uric acid was measured using the enzymatic uricase-peroxidase method. Fasting blood glucose and lipid profile including total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were analyzed using automated biochemical analyzers.

Hyperuricemia was defined as SUA >7.0 mg/dL in males and >6.0 mg/dL in females. Cardiovascular risk factors assessed included obesity, diabetes mellitus, dyslipidemia, smoking, alcohol intake, sedentary lifestyle, and prolonged duration of hypertension.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics Version 26. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Independent t-test and Chi-square test were used for comparison between groups. Pearson correlation analysis was performed to evaluate the association between SUA and cardiovascular risk parameters. Multivariate logistic regression analysis was conducted to identify independent predictors of hyperuricemia. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 320 hypertensive patients were included in the study. The mean age of the study population was 54.8 ± 11.6 years, with males constituting 56.9% of participants. Hyperuricemia was identified in 118 participants (36.9%). Patients with elevated serum uric acid (SUA) levels demonstrated significantly higher prevalence of obesity, diabetes mellitus, dyslipidemia, smoking, and prolonged duration of hypertension compared with participants having normal SUA levels.

Table 1. Demographic Characteristics of the Study Population (n = 320)

Variable	Total (n=320)
Age (years), mean ± SD	54.8 ± 11.6
Male, n (%)	182 (56.9)
Female, n (%)	138 (43.1)
Duration of hypertension >5 years, n (%)	176 (55.0)
Current smokers, n (%)	92 (28.8)
Alcohol consumers, n (%)	84 (26.3)
Sedentary lifestyle, n (%)	188 (58.8)
Diabetes mellitus, n (%)	116 (36.3)
Dyslipidemia, n (%)	148 (46.3)
Obesity (BMI ≥30 kg/m²), n (%)	102 (31.9)
Hyperuricemia, n (%)	118 (36.9)

Table 2. Clinical and Biochemical Characteristics According to SUA Status

Variable	Normal SUA (n=202)	Elevated SUA (n=118)	p-value
SUA (mg/dL)	5.2 ± 0.9	7.8 ± 1.2	<0.001
Systolic BP (mmHg)	142.6 ± 13.4	151.8 ± 15.7	<0.001
Diastolic BP (mmHg)	88.4 ± 9.1	94.7 ± 10.3	<0.001
BMI (kg/m²)	25.9 ± 3.6	29.1 ± 4.2	<0.001
Fasting blood glucose	108.2 ± 24.7	128.5 ± 31.8	<0.001

(mg/dL)			
Total cholesterol (mg/dL)	184.5 ± 32.4	214.2 ± 36.8	<0.001
LDL-C (mg/dL)	111.7 ± 24.3	134.9 ± 28.6	<0.001
HDL-C (mg/dL)	45.2 ± 8.1	39.8 ± 7.4	<0.001
Triglycerides (mg/dL)	148.6 ± 40.2	192.7 ± 51.5	<0.001

Table 3. Association BetweenHyperuricemia and Cardiovascular Risk Factors

Risk Factor	Hyperuricemia Present n (%)	Hyperuricemia Absent n (%)	p-value
Diabetes mellitus	64 (54.2)	52 (25.7)	<0.001
Dyslipidemia	78 (66.1)	70 (34.7)	<0.001
Obesity	58 (49.2)	44 (21.8)	<0.001
Smoking	46 (39.0)	46 (22.8)	0.003
Alcohol intake	38 (32.2)	46 (22.8)	0.071
Sedentary lifestyle	82 (69.5)	106 (52.5)	0.004
Hypertension duration >5 years	82 (69.5)	94 (46.5)	<0.001

Table 4. Pearson Correlation Analysis Between SUA and Cardiovascular Risk Parameters

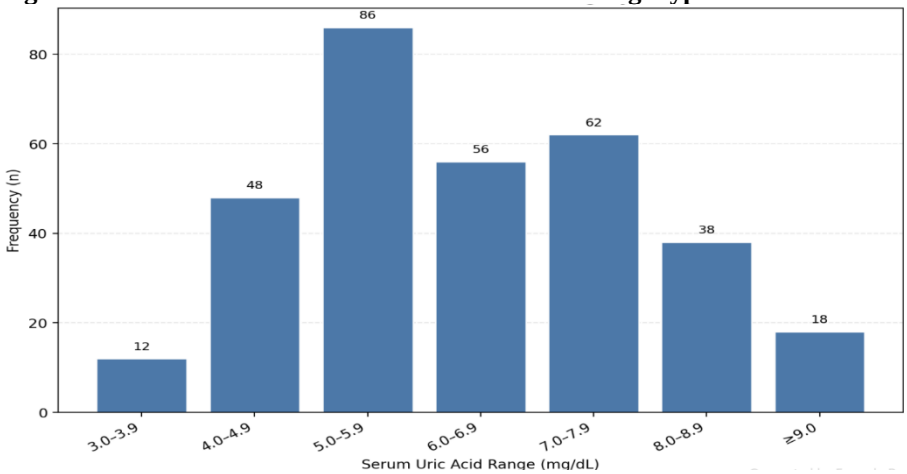
Variable	Correlation Coefficient (r)	p-value
Systolic blood pressure	0.412	<0.001
Diastolic blood pressure	0.336	<0.001
BMI	0.468	<0.001
Fasting blood glucose	0.382	<0.001
Total cholesterol	0.429	<0.001
LDL-C	0.401	<0.001
Triglycerides	0.401	<0.001
HDL-C	-0.298	<0.001

Table 5. Multivariate Logistic Regression Analysis for Predictors of Hyperuricemia

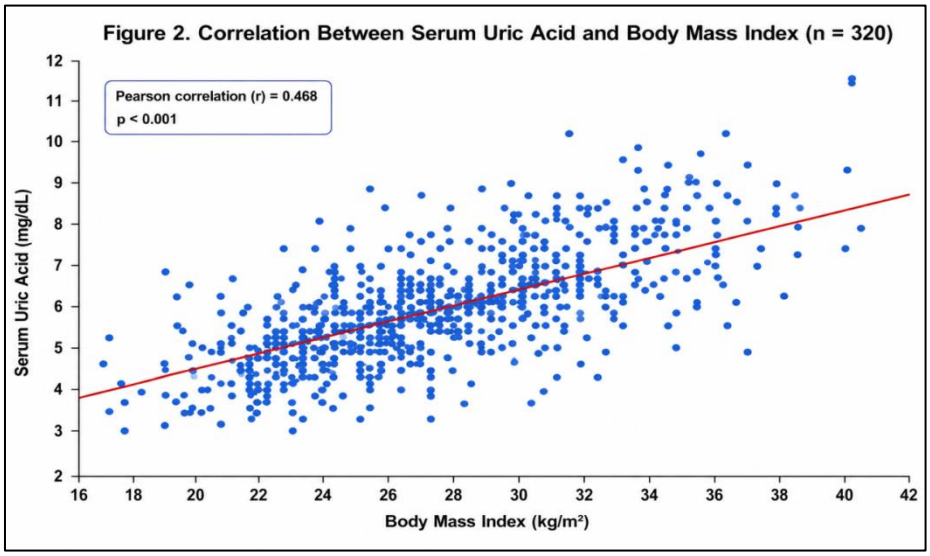
Variable	Adjusted OR	95% CI	p-value
Obesity	2.41	1.42–4.09	0.001
Diabetes mellitus	2.18	1.29–3.67	0.003
Dyslipidemia	2.45	1.48–4.08	<0.001
Smoking	1.88	1.04–3.38	0.036
Hypertension duration >5 years	2.09	1.23–3.54	0.006
Sedentary lifestyle	1.72	1.01–2.94	0.045

Multivariate logistic regression analysis was adjusted for age, sex, BMI, diabetes mellitus, smoking status, dyslipidemia, and duration of hypertension. Elevated serum uric acid levels were significantly associated with adverse cardiovascular risk profiles among hypertensive patients.

Figure 1. Distribution of Serum Uric Acid Levels Among Hypertensive Patients



A histogram demonstrated a right-skewed distribution of SUA concentrations, with higher values observed predominantly among patients with obesity, diabetes mellitus, and dyslipidemia.



A scatter plot demonstrated a significant positive correlation between SUA levels and BMI ($r = 0.468$, $p < 0.001$), indicating progressively increasing SUA concentrations with increasing BMI.

DISCUSSION

The present study demonstrated a significant association between elevated serum uric acid (SUA) levels and multiple cardiovascular risk factors among hypertensive patients. Hyperuricemia was identified in 36.9% of the study population and was significantly associated with obesity, diabetes mellitus, dyslipidemia, smoking, sedentary lifestyle, and prolonged duration of hypertension. Furthermore, SUA levels showed significant positive correlations with systolic blood pressure, body mass index (BMI), fasting blood glucose, total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglyceride levels. Multivariate logistic regression analysis further confirmed obesity, dyslipidemia, diabetes mellitus, smoking, and prolonged hypertension duration as independent predictors of hyperuricemia. These findings suggest that elevated SUA may represent an important metabolic and cardiovascular risk marker among hypertensive individuals.

The prevalence of hyperuricemia observed in the present study is comparable to previous international reports demonstrating increased SUA levels among hypertensive populations. Several epidemiological studies have shown that elevated SUA is strongly associated with incident hypertension and adverse cardiovascular outcomes [8,9]. Kuwabara reported that hyperuricemia was independently associated with hypertension, metabolic syndrome, and cardiovascular disease, supporting the hypothesis that uric acid contributes directly to cardiometabolic dysfunction rather than merely reflecting associated metabolic abnormalities [6]. Similarly, studies from Asian and European populations demonstrated that hypertensive patients with elevated SUA levels exhibited significantly higher rates of obesity, dyslipidemia, and diabetes mellitus [9].

A major finding of the present study was the significant association between SUA and obesity. Participants with elevated BMI demonstrated significantly higher SUA concentrations compared with non-obese individuals. Obesity-related insulin resistance may reduce renal uric acid excretion, thereby contributing to hyperuricemia [5]. Adipose tissue inflammation and increased oxidative stress associated with obesity further promote endothelial dysfunction and vascular injury [5]. Choi and Ford also reported a strong relationship between hyperuricemia and metabolic syndrome components, particularly obesity and dyslipidemia [19].

The present study additionally demonstrated a strong association between hyperuricemia and dyslipidemia. Elevated triglycerides and LDL-C levels among hyperuricemic individuals may result from chronic oxidative stress and impaired endothelial nitric oxide production. Experimental studies have demonstrated that uric acid stimulates vascular smooth muscle proliferation and promotes lipid oxidation, thereby accelerating atherosclerotic changes [14]. The inverse relationship between SUA and HDL-C identified in the present study is also consistent with previous evidence linking hyperuricemia to adverse lipid metabolism and increased cardiovascular risk [20].

Several biological mechanisms may explain the observed association between SUA and cardiovascular risk factors. Intracellular uric acid accumulation induces oxidative stress through activation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, resulting in increased reactive oxygen species production [17]. Oxidative stress subsequently reduces nitric oxide bioavailability and impairs endothelial-dependent vasodilatation, thereby contributing

to endothelial dysfunction and arterial stiffness [18]. In addition, uric acid has been shown to activate the renin–angiotensin–aldosterone system (RAAS), leading to vascular remodeling, sodium retention, and sustained elevation of blood pressure [7]. Hyperuricemia also stimulates inflammatory mediators including monocyte chemoattractant protein-1, tumor necrosis factor-alpha, and interleukin-6, which contribute to chronic vascular inflammation and progression of atherosclerosis [16].

The positive correlation observed between SUA and duration of hypertension in the present study may indicate progressive renal microvascular dysfunction associated with prolonged blood pressure elevation. Chronic hypertension may impair renal urate excretion, while hyperuricemia itself may worsen hypertension through renal vasoconstriction and endothelial dysfunction [7]. This bidirectional relationship between uric acid and hypertension has been supported by both experimental and clinical studies [3].

Despite growing evidence supporting the pathogenic role of SUA in cardiovascular disease, controversy persists regarding whether uric acid acts as an independent causal factor or simply reflects underlying metabolic abnormalities. Some large epidemiological investigations have shown attenuation of the association between SUA and cardiovascular outcomes after adjustment for obesity, renal dysfunction, and insulin resistance [21]. Moreover, certain Mendelian randomization studies have reported inconsistent evidence regarding a direct causal role of uric acid in hypertension and cardiovascular disease [12]. Nevertheless, the present study demonstrated significant independent associations even after adjustment for major confounding variables, suggesting that SUA may possess additional prognostic value beyond traditional cardiovascular risk factors.

The findings of this study have important clinical implications. Measurement of SUA levels in hypertensive patients may facilitate early identification of individuals at increased cardiovascular risk, particularly those with obesity, diabetes mellitus, and dyslipidemia. Routine evaluation of SUA could therefore improve cardiovascular risk stratification and guide implementation of preventive interventions. Lifestyle modifications including dietary control, weight reduction, smoking cessation, and increased physical activity may reduce both uric acid levels and cardiovascular burden. In addition, emerging evidence suggests that urate-lowering therapy may provide cardiovascular benefits in selected high-risk populations, although further large-scale randomized clinical trials are required [22].

From a public health perspective, hypertension and hyperuricemia frequently coexist in developing countries due to rapid urbanization, sedentary lifestyle patterns, unhealthy dietary habits, and increasing prevalence of obesity and metabolic syndrome. Early identification of hyperuricemia among hypertensive individuals may therefore help reduce long-term cardiovascular morbidity and mortality through targeted screening and preventive strategies.

The present study possesses several strengths, including a relatively adequate sample size, comprehensive assessment of multiple cardiovascular risk factors, and use of multivariate logistic regression analysis to adjust for confounding variables. Standardized biochemical and clinical measurements further enhanced methodological reliability. However, certain limitations should be acknowledged. The cross-sectional design precludes establishment of causal relationships between SUA and cardiovascular risk factors. The study was conducted at a single tertiary care center, which may limit generalizability to broader populations. Dietary factors, medication adherence, and long-term cardiovascular outcomes were not evaluated. Prospective longitudinal studies are therefore required to further clarify the causal relationship between hyperuricemia and cardiovascular disease progression in hypertensive patients.

CONCLUSION

The present study demonstrated a significant association between elevated serum uric acid (SUA) levels and major cardiovascular risk factors among hypertensive patients. Hyperuricemia was significantly associated with obesity, diabetes mellitus, dyslipidemia, smoking, and prolonged duration of hypertension. SUA levels also showed positive correlations with blood pressure, BMI, fasting blood glucose, and lipid abnormalities.

These findings suggest that serum uric acid may serve as an important biomarker for cardiovascular risk stratification in patients with hypertension. Early identification and management of hyperuricemia, along with modification of associated metabolic risk factors, may help reduce cardiovascular complications in hypertensive individuals. Further prospective studies are required to establish the causal role of SUA in cardiovascular disease progression.

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