



## Original Article



## Prevalence and Correlates of Hyperuricemia in Patients with Hypertension: A Cross-Sectional Study from a Tertiary Care Hospital in Pakistan

Abdul Ahad<sup>1</sup>, Saima Qadir<sup>1</sup>, Shumaila Khan<sup>1</sup>, Taimour Khan<sup>1</sup>, Shahab Khan<sup>1</sup> and Fawad Khalid<sup>1</sup><sup>1</sup>Department of Medicine, Saidu Medical College, Saidu Sharif, Pakistan

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Department of Medicine, Saidu Medical College,  
Saidu Sharif, Pakistan  
[dr.aahad@yahoo.com](mailto:dr.aahad@yahoo.com)Received date: 12<sup>th</sup> August, 2024Acceptance date: 6<sup>th</sup> February, 2025Published date: 28<sup>th</sup> February, 2025

## ABSTRACT

The link between Serum Uric Acid (SUA) and hypertension debated among researchers.

**Objective:** To determine the prevalence and correlates of hyperuricemia in patients with hypertension. **Methods:** This cross-sectional study was conducted at the General Medicine Department, Saidu Teaching Hospital, Swat (October 2023–April 2024), analyzing hypertensive patients aged 30–60 years. SUA was measured via an automated micro lab, with hyperuricemia defined as SUA >7.0 mg/dl (men) and >6.0 mg/dl (women). Chi-square tested categorical data, while Pearson correlation assessed SUA relationships with SBP, DBP, and BMI ( $p < 0.05$  significant). **Results:** In a total 266 patients, 136 (51.1%) were male. The mean age, BMI were  $45.41 \pm 25.92$  years, and  $27.28 \pm 3.25$  kg/m<sup>2</sup>, respectively. The obesity was noted in 72 (27.1%) patients. Dyslipidemia was found in 42 (15.8%) patients. There were 54 (20.3%) patients who were newly diagnosed cases of hypertension. The mean systolic blood pressure (SBP), diastolic blood pressure (DBP) were  $141.76 \pm 8.24$  mm Hg, and  $91.36 \pm 8.45$  mm Hg, respectively. The mean SUA level was  $6.03 \pm 1.13$  mg/dl, and the frequency of hyperuricemia was noted in 61 (22.9%) patients. Obesity ( $p = 0.033$ ), and known diagnosis of hypertension ( $p < 0.001$ ) were significantly linked with hyperuricemia, Scatter plot revealed positively linear and significant correlation of SBP ( $r = 0.261$ ,  $p < 0.001$ ), and DBP ( $r = 0.319$ ,  $p < 0.001$ ) with SUA levels. The BMI was also found to have positively linear and significant relationship with SUA levels ( $r = 0.122$ ,  $p = 0.047$ ). **Conclusion:** Hyperuricemia (22.9%) in hypertensive patients correlated with blood pressure and BMI, indicating its role in complications.

## INTRODUCTION

Hypertension (HTN) is a major global health issue. Data from the developed world depicts that one out of four adults are estimated to be affected by HTN [1]. Predicting the underlying causes of HTN is a challenging task due to the intricate interaction between genetic predispositions and environmental influences. Numerous factors contribute to the development of HTN, making it a multifaceted condition to analyze and understand [2]. Among these potential contributors, the association between elevated serum uric acid (SUA) levels and HTN has been a subject of ongoing debate and extensive research within the medical community [3–5]. While some studies suggested a possible link between hyperuricemia (HU) and HTN, it is crucial to account for various confounding factors that may influence this relationship. Metabolic syndrome,

diabetes mellitus, chronic kidney disease (CKD), along with lifestyle factors and high salt intake, are all significant contributors to the development of both hyperuricemia (HU) and HTN. Therefore, it is essential to account for these overlapping factors when studying the link between the two conditions. [6, 7]. Any attempt to establish a clear connection between HU and HTN must carefully consider these confounders to avoid oversimplification and to provide a more accurate understanding of the underlying mechanisms at play. In developing countries, very limited data exists about the burden of HU and its possible linkage with HTN. One study done in Nepal concluded a positive connection among HU and HTN, showed that almost 29% of HTN patients had HU [8]. The impact of SUA on arterial stiffness was studied in China, and it was concluded that a



higher baseline SUA is an independent risk factor for developing arterial stiffness but the predictive value of SUA on arterial stiffness is still unanswered in long-term longitudinal studies [9]. The ROVIGO study took into account the role of HU in resistant HTN in the general population, and concluded that in older females, SUA above 6.8 mg/dl increased the risk of resistant HTN as 3 folds [10]. Due to the increasing incidence of asymptomatic HU in HU patients [11], there is a need to conduct trials on the interrelationship of HU and HTN. It is hypothesized that the burden of HU might be high in HTN. In developing countries, limited data exists regarding the burden of HU and its relationship with HTN, particularly among different ethnic and regional populations. Studies conducted in other parts of the world have shown varying frequencies of HU among hypertensive patients, influenced by genetic, dietary, and environmental factors [8-10]. Not such studies have been conducted in Khyber Pakhtunkhwa Province, Pakistan, where cultural and lifestyle variations may influence this relationship. Given the lack of region-specific data, this study aims to determine the prevalence of HU and its clinical correlates in hypertensive patients presenting to a tertiary care hospital in Swat, Pakistan.

Understanding the burden of HU in this specific population may contribute to improved hypertension management strategies and help identify high-risk groups requiring early intervention.

## METHODS

This cross-sectional study was carried out at the Outpatient Department of the Department of Medicine, Saidu Teaching Hospital, Swat, Pakistan, from October 2023 to April 2024. Approval from Ethical Review Committee was obtained (letter number: 489/MMWA/023). A sample size of 266 was calculated, with a 95% confidence interval, 7% margin of error, and keeping the anticipated frequency of HU as 29% in HTN patients [8]. The inclusion criteria were patients of either gender, aged 30-60 years, and presenting with HTN. The exclusion criteria were patients with suspected secondary HU (e.g., drugs, malignancies, uremia, and other conditions with rapid cell turnovers like psoriasis (confirmed through SUA > 7.2 mg/dl). Sample selection was made using a non-probability consecutive sampling technique. Informed and written consents were obtained from all study participants. Necessary demographics like age, gender, and residential status were recorded. The HTN was defined as systolic blood pressure (SBP)  $\geq$  130 mm Hg and/or diastolic blood pressure (DBP)  $\geq$  80 mm Hg [12]. Standard protocols were followed for the diagnosis of newly diagnosed cases (high BP on two or more occasions at least one week apart). The blood pressure measurement was taken in the right arm with the help of a mercury sphygmomanometer with an

adequate sized cuff, with an individual seated quietly in a chair for 5 minutes with feet on the floor. A detailed physical examination was conducted for all patients. A blood sample was taken from the peripheral vein to measure SUA in the institutional laboratory through an automated micro lab. HU was labeled as a SUA level of > 7.0 mg/dl in men and > 6.0 mg/dl in women [13]. Patients with a BMI > 30.0 kg/m<sup>2</sup> were labeled obese. Dyslipidemia was defined as any one of these; total cholesterol  $\geq$  200 mg/dl, high-density lipoprotein  $\leq$  40 mg/dl for men or  $\leq$  50 in women, low-density lipoprotein  $\geq$  100 mg/dl, or triglycerides  $\geq$  150 mg/dl [14]. All the concerned data were recorded on a specifically pre-designed proforma. The statistical analysis was performed employing IBM-SPSS Statistics version 26.0. Mean and standard deviation were calculated for age, BMI, SBP, DBP, and SUA level. Frequency and percentages were calculated for gender, smoking status, obesity, dyslipidemia, and HU. HU was stratified by age, gender, obesity, smoking status, and dyslipidemia to see the effect modification. A post-stratification chi-square test was applied. Pearson correlation test was applied to explore the correlation of SUA levels with SBP, DBP, and BMI. For all statistical inferences,  $p < 0.05$  was taken as statistically significant.

## RESULTS

In a total 266 patients, 136 (51.1%) were male. The mean age was  $45.41 \pm 25.92$  years, ranging between 30-60 years. There were 106 (40.8%) patients who had age between 51-60 years. The mean BMI was  $27.28 \pm 3.25$  kg/m<sup>2</sup> while obesity was noted in 72 (27.1%) patients. Dyslipidemia was found in 42 (15.8%) patients. There were 54 (20.3%) patients who were newly diagnosed cases of HTN. The mean SBP, DBP were  $141.76 \pm 8.24$  mm Hg, and  $91.36 \pm 8.45$  mm Hg, respectively. Family history of hyperuricemia was documented in 39 (14.7%) patients. Table 1 shows demographic and clinical characteristics of hypertensive patients studied (Table 1).

**Table 1:** Frequency Distribution of the Study Characteristics of Patients with Hypertension (n=266)

Characteristics	Category	Frequency (%)
Age (Years)	30-40	51 (19.2%)
	41-50	109 (41.0%)
	51-60	106 (39.8%)
Gender	Male	136 (51.1%)
	Female	130 (48.9%)
Residence	Urban	103 (38.7%)
	Rural	163 (61.3%)
Smoking status	Yes	88 (33.1%)
	No	178 (66.9%)
Obesity	Yes	106 (39.8%)
	No	160 (60.2%)
Dyslipidemia	Yes	59 (22.2%)

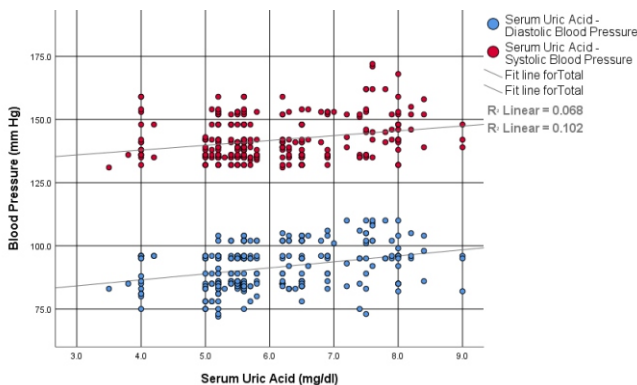
	No	207 (77.8%)
Newly diagnosed hypertension	Yes	54 (20.3%)
	No	212 (79.7%)
Family history of hyperuricemia	Yes	39 (14.7%)
	No	227 (85.3%)

The mean SUA level was  $6.03 \pm 1.13$  mg/dl, and the frequency of HU was noted in 61 (22.9%) patients. Stratification of HU showed that no statistically significant associations were found with respect to gender ( $p=0.956$ ), age groups ( $p=0.992$ ), smoking status ( $p=0.955$ ), dyslipidemia ( $p=0.178$ ), or family history of hyperuricemia ( $p=0.094$ ). Obesity ( $p=0.033$ ), and known diagnosis of HTN ( $p<0.001$ ) were significantly linked with HU, and the details are shown in table 2.

**Table 2:** Stratification of Hyperuricemia with Respect to Study Characteristics of the Patients with Hypertension (N=266)

Characteristics	Category	Hyperuricemia Frequency (%)		p-Value
		Present (n=61)	Absent (n=205)	
Gender	30-40	31 (51.7%)	105 (51.2%)	0.956
	41-50	30 (48.3%)	100 (48.8%)	
Age (Years)	51-60	12 (19.7%)	39 (19.0%)	0.992
	Male	25 (41.0%)	84 (41.0%)	
	Female	24 (39.3%)	82 (40.0%)	
Smoking Status	Urban	20 (32.8%)	68 (33.2%)	0.955
	Rural	41 (67.2%)	137 (66.8%)	
Obesity	Yes	23 (37.7%)	49 (23.9%)	0.033
	No	38 (62.3%)	156 (76.1%)	
Dyslipidemia	Yes	13 (21.3%)	29 (14.1%)	0.178
	No	48 (78.7%)	176 (85.9%)	
Newly Diagnosed Hypertension	Yes	6 (9.8%)	48 (23.4%)	<0.001
	No	55 (90.2%)	157 (76.6%)	
Family History of Hyperuricemia	Yes	13 (21.3%)	26 (12.7%)	0.094
	No	48 (78.7%)	179 (87.3%)	

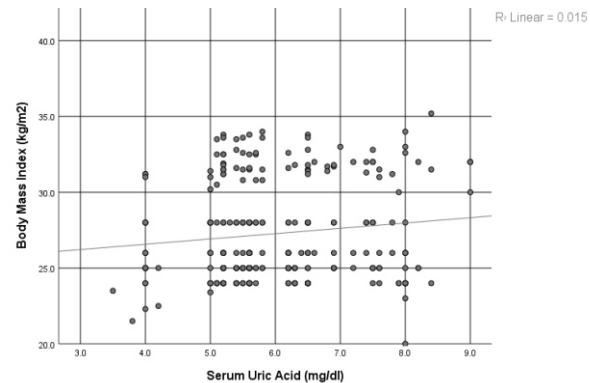
Scatter plot revealed positively linear and significant correlation of SBP ( $r=0.261$ ,  $p<0.001$ ), and DBP ( $r=0.319$ ,  $p<0.001$ ) with SUA levels as exhibited in figure 1.



**Figure 1:** Relationship of Blood Pressure with SUA Levels

Figure 2 illustrated the correlation between Body Mass Index (BMI) and Serum Uric Acid (SUA) levels among

hypertensive patients. The scatter plot demonstrated BMI was also found to have positively linear and significant relationship with SUA levels ( $r=0.122$ ,  $p=0.047$ ).



**Figure 2:** Relationship of BMI with SUA Levels

## DISCUSSION

The literature proposes linkage between elevated SUA and an increased risk for HTN and cardiovascular diseases, but these associations have been uncertain [15, 16]. In this study, the frequency of HU was found to be 22.9% among patients with HTN. Local data described the frequency of HU in adult patients with HTN to be 41.0% which is much more than what we documented [17]. Another study by Raja et al., analyzing risk factors for HU in adult population showed that mean SUA levels were  $6.64 \pm 1.63$  mg/dl versus  $5.35 \pm 1.60$  mg/dl in hypertensive and non-hypertensive patients ( $p<0.001$ ), respectively [18]. Long ago, Breckenridge demonstrated that 58% of patients receiving anti-hypertensive medication had elevated SUA levels, while 27% of patients presenting to the clinic at that time had HU [19]. Messerli et al., analyzed established hypertensive cases showed that 72% of them had a raised SUA [20]. The hypothesis argued that the burden of HU in HTN may be explained to either diminished renal perfusion or underlying renal dysfunction. A study by Khaliq et al analyzing patients of HU described that 84% patients with HU had HTN versus 41% controls (odds ratio: 7.55;  $p<0.001$ ) [21]. It was found that among hypertensive patients, presence of HU had significant association with obesity. Some researchers have shown that the correlation between raised SUA and HTN decreased after adjusting for BMI, which implies that the association may be linked to obesity [22]. This association linked to obesity is also demonstrated by Qiu et al, in which attenuation was observed after adjustment of waist circumference [23]. Obesity is associated with hyperinsulinemia, which in turn causes decreased excretion of SUA. This implies increased chances of progression to HTN in metabolic syndromes (central obesity, insulin resistance, and HTN) [24]. These insights can further emphasize the linkage between HU, with hypertriglyceridemia which should be considered confounders while studying the linkage between HU and

HTN. Some researchers have proposed that toxins in the blood cause damage to the kidney and heart's vasculature, leading to HTN, while a potential agent could be uric acid as a key HTN mediator [25]. Experts also connect high blood pressure to uric acid and recommend dietary modifications to reduce uric acid and regulate blood pressure in the general public [26]. Apart from being a distinct risk factor for incident HTN in the general population, HU could have significant differences in consequences depending on age, sex, and race. With appropriate intervention in the early phases of HTN, this model implies that there can be a period of reversible HTN. Although no significant association was found between hyperuricemia and gender or age groups, prior studies have suggested that hormonal factors in premenopausal women may contribute to lower SUA levels [27], while aging-related metabolic changes may influence uric acid metabolism [28]. The non-significance in this study could be due to a relatively uniform age range (30-60 years) and a lack of differentiation between premenopausal and postmenopausal women. Present findings suggested that HU is highly prevalent in patients with HTN has significant clinical implications. HU in its coexistence with HTN can further elevate the likelihood of adverse cardiovascular events, necessitating careful monitoring and management of uric acid levels in these patients. HU can further contribute to the development and worsening of HTN, potentially complicating blood pressure control. This underscores the importance of integrating strategies to lower uric acid levels, such as lifestyle modifications and appropriate pharmacotherapy, into the management plan for hypertensive patients. The combination of HTN and HU may also heighten the risk of CKD, as both conditions independently contribute to renal damage. Therefore, regular assessment of renal function is crucial in hypertensive patients with elevated uric acid levels to prevent the progression of CKD and ensure comprehensive care. Being a single center study, conducted on a relatively modest sample were some of the inherent limitations of this study. We were unable to record the impact of various anti-hypertensive treatments and its relationship to existing uric acid levels. Cross-sectional design of this research may not explore causal inferences, so a longitudinal design could provide better insights in the future.

## CONCLUSIONS

The frequency of hyperuricemia was high (22.9%) in patients with hypertension. Further prospective studies should be planned to record the clinical impact of hyperuricemia among hypertensive patients.

## Authors Contribution

Conceptualization: AA, SQ, TK, SK

Methodology: AA, SQ,

Formal analysis: SK

Writing, review and editing: AA, SQ, SK, TK, SK, FK

All authors have read and agreed to the published version of the manuscript

## Conflicts of Interest

The authors declare no conflict of interest.

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