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ORIGINAL RESEARCH

Serum Uric Acid Levels in Patients with Essential Hypertension: A Clinical Study

Pradeep Kurmi[1], Lokendra Rekwal[2], Ronak Bhandari[3], Sudarshan Patidar[4]

[1-4]MGMMC Superspeciality Hospital, Indore, Madhya Pradesh, India

Corresponding author
Sudarshan Patidar
MGMMC Superspeciality Hospital, Indore, Madhya Pradesh, India
Email: drsudarshanpatidar@gmail.com

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ABSTRACT

Background: Elevated serum uric acid (SUA) has been linked to hypertension (HTN) through various mechanisms. This study aimed to assess SUA levels in patients with essential HTN and to explore their association with age, sex, body mass index (BMI), smoking, diabetes, and target organ damage. **Methods**: A hospital-based case-control study was conducted at Ramkrishna Care Hospital, Raipur, from May 2021 to March 2022. The patients aged 25 years and older diagnosed with essential HTN of both sexes were included with the written informed consent. Data collection involved medical history, physical examination, fasting serum uric acid levels, a blood haemogram,blood urea and serum creatininetests, blood pressure measurement, and BMI. **Results**: A total of 100 patients were included, with 32 cases and 68 controls. The mean (SD) age of cases was 56.25 (10.9) years, while control group had 52.4 (11.53) years. Significant differences in blood pressure (BP) were observed, with mean systolic BP of 165.13 mmHg in cases vs. 122.29 mmHg in controls (P<0.001) and mean diastolic BP of 103.94 mmHg in cases vs. 77.74 mmHg in controls (P<0.001). A significant difference was observed in mean urea levels, with cases having 31.38 (6.37) mg/dL and controls with 28.07 (5.43) mg/dL.The mean (SD) serum creatinine levels were elevated in cases [1.01 (0.28 mg/dL)] compared to controls [0.88 (0.16 mg/dL), P<0.001].The SUA levels were significantly higher in cases (6.33 mg/dL) compared to controls (5.01 mg/dL, P<0.001). **Conclusion**: Elevated SUA levels are significantly associated with essential HTN, with higher SUA levels observed in patients with HTN compared to controls.

Keywords: Diastolic blood pressure, systolic blood pressure, serum creatinine, urea.

INTRODUCTION

Hypertension (HTN) is defined asa systolic blood pressure (SBP) of 130 mmHg or higher, and/or a diastolic blood pressure (DBP) greater than 80 mmHg. The majority of HTN cases are classified as idiopathic, commonly knownas essential HTN [1].According to WHO, around 1.28 billion adults aged 30-79 years worldwide are affected by HTN, with roughly two-thirds residing in low-and middle-income countries [2]. Obesity, insulin resistance, excessive alcohol and salt consumption, age, a sedentary lifestyle, stress, and inadequate potassium and calcium intake are some of the factors that can cause blood pressure to rise [3].Serum uric acid (SUA) abnormalities area possible factor inthe onset and progression of essential HTN [4].In older hypertensive individuals,hyperuricemia is considered a key marker of cardiovascular risk for developing HTN [5].

Uric acid, the end product of purine nucleoside degradation, possesses antioxidant properties that enable it to scavenge oxygen radicals, thusprotecting the erythrocyte membrane from lipid oxidation [6]. However, elevated uric acid levels can arise from increased purine breakdown in conditions with high cell turnover, such as rhabdomyolysis, hemolysis, and tumor lysis, or from reduced excretion due to metabolic acidosis and renal insufficiency [5].

Elevated uric acid has been linked to HTN through various mechanisms. It promotes oxidative stress, reduces endothelial nitric oxide, and activates renin and angiotensin pathways, which leads to kidney vasoconstriction, ischemia, and further oxidative damage. This triggers immune system activation, resulting in sustained kidney vasoconstriction and contributing to salt-sensitive HTN [7]. This suggests that hyperuricemia is associated with an increased risk of cardiorenal disease and the development of new-onset HTN. In younger population, urate-reducing therapy is linkedto the normalization of both serum uric acid and blood pressure and SUA [5].

The study aimed to assess uric acid levels in patients with essential HTN, and to explore the associations between age, sex, body mass index (BMI), smoking,

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diabetes,and target organ damage and the elevated level of serum uric acid.

MATERIALS AND METHODS

Study design

This hospital-based case control study was conducted at the Department of Medicine, Ramkrishna Care Hospital, Raipur, from May 2021 to March 2022. The study was approved by the ethical committee of the institute, and written informed consent was obtained from the patients before conducting the study.

Inclusion and exclusion criteria

The study included patients with essential HTN of both sexes, aged 25 years or older. Patients with renal failure or secondary HTN were excluded. Additionally, individuals who consumed alcohol regularly, those undergoing chemotherapy or anti-metabolite treatments, or those with clinical signs of gout or extra-articular manifestations of hyperuricemia were excluded. Furthermore, patients with a history of using medications known to cause hyperuricemia, such as thiazide diuretics, as well as pregnant women, were also excluded from the study.

Control group

The control group consisted of patients without HTN or any other medical condition that could cause hyperuricemia and was matched to the case group for age and sex.

Data collection

The clinical examination involved a detailed medical history, a complete physical examination (including blood pressure measurement and anthropometric measurements like height and body weight), as well as laboratory tests which included fasting serum uric acid levels, a blood haemogram, renal function tests (such as blood urea and serum creatinine), and an electrocardiogram, all of which was recorded for every patients. The SBP/DBP was measured, followed by the calculation of BMI.

RESULTS

Analysis of cases and control according to age

This study included 100 patients, consisting of 32 cases and 68 controls. The age of the study population ranged from 26 to 76 years. The mean (standard deviation) [SD] age was 56.25 (10.9) years for the cases and 52.4 (11.53) years for the controls.There was no significant difference (P=0.12) in the age between those with and without HTN in this study. Therefore,the elevated SUV in both cases and controls was independent of age.

BMI distribution among study group

The mean (SD) BMI among the cases was 23.95 (2.49) kg/m², while for the controls, it was 22.96 (2.5) kg/m². There was no significant difference (P=0.07) in BMI between individuals with and without HTN.

Distribution of SBP/DBP amongstudy group

The mean (SD) of SBP was significantly higher in the cases(165.13 [17.41] mmHg) compared tothecontrols(122.29 [8.1] mmHg). Similarly, DBP(103.94 [9.52] mmHg) was also higher in the cases than in the controls (77.74 [3.88] mmHg).

Distribution of random blood sugar levels (RBS) among study group

The majority of both cases and controls had RBS levels <140 mg/dL, with proportions of 93.75% and 98.53%, respectively. The mean (SD) RBS levels were 102.03 (21.9) mg/dL in the cases and 96.99 (16.7) mg/dL in the controls, with no significant difference (P=0.21) between the case and control groups with respect to RBS levels (Table 1).

Distribution of study group in relation to renal parameters

The majority of cases and controls had significantly higher urea levels (>24 mg/dL), with proportions of 81.25% and 70.59%, respectively. The mean (SD) urea levels were 31.38 (6.37) mg/dLin cases and 28.07 (5.43) mg/dL in controls. A significant difference (P=0.01) was observed between the groups with and without HTN.The majority of cases (59.38%) and controls (83.82%) had normal serum creatinine levels (0.8-1.2 mg/dL) with P<0.001. However,the mean (SD) serum creatinine levels were elevated in cases [1.01 (0.28 mg/dL)] compared to controls [0.88 (0.16 mg/dL), P<0.001]. The mean (SD) SUA level in cases was [6.33 (1.16 mg/dL)], significantly higher than in controls [5.01 (1.03 mg/dL), P<0.001] (Table 2). Thus, hyperuricemia was statistically significant in hypertensives compared to normotensives.Among cases and controls, the mean (SD) SUA levels in males were 6.29 (1.22) mg/dL and 5.1 (1.06) mg/dL, respectively, while in females, they were 6.43 (1.06) mg/dL and 4.7 (0.87) mg/dL,respectively (P<0.001) (Figure 1). This shows that SUV levels were significantly influenced by systemic HTN among patients.

Distribution of cases and controls in relation to target organ damage

Left ventricular hypertrophy (LVH) was significantly more prevalent in cases (31.25%)compared tocontrols (1.47%), with a P<0.001. Similarly, coronary artery disease (CAD) was observed more frequently in cases (46.88%)than in controls(16.18%), with a P=0.001.No significant difference was noted in other risk factors such as family history, smoking, diabetes mellitus, and cerebrovascular accidents (Table 3).

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Table 1: Baseline characteristics

Parameters	Case (N=32)	Control (N=68)	P value
Age (years)			
<=30	1 (3.13)	4 (5.88)	
31-40	2 (6.25)	10 (14.71)	
41-50	6 (18.75)	15 (22.06)	0.69
51-60	12 (37.5)	19 (27.94)	
61-70	9 (28.13)	18 (26.47)	
>70	2 (6.25)	2 (2.94)	
Age (years), mean (SD)	56.25 (10.9)	52.4 (11.53)	0.12
BMI (Kg/m^2)			
<18.5	-	1 (1.47)	0.38
18.5-24.9	17 (53.13)	44 (64.71)	
>=25	15 (46.88)	23 (33.82)	
BMI (Kg/m^2), mean (SD)	23.95 (2.49)	22.96 (2.5)	0.07
SBP (mmHg)	165.13 (17.41)	122.29 (8.1)	-
DBP (mmHg)	103.94 (9.52)	77.74 (3.88)	-
RBS (mg/dL)			
<140	30 (93.75)	67 (98.53)	0.19
>=140	2 (6.25)	1 (1.47)	
RBS (mg/dL),mean (SD)	102.03 (21.9)	96.99 (16.7)	0.21
Data presented as n (%), unless otherwise specified. BMI, body mass index; DBP, diastolic blood pressure; RBS, random blood sugar; SBP, systolic blood pressure; SD, standard deviation.			

Table 2: Renal parameters

Parameters	Case (N=32)	Control (N=68)	P value
Urea (mg/dL), n (%)			
<6	-	-	0.25
Normal (6-24)	6 (18.75)	20 (29.41)	
Raised >24	26 (81.25)	48 (70.59)	
Urea	31.38 (6.37)	28.07 (5.43)	0.01
Creatinine (mg/dL), n (%)			
Low(<0.8)	7 (21.88)	11 (16.18)	<0.001
Normal (0.8-1.2)	19 (59.38)	57 (83.82)	
Raised(>1.2)	6 (18.75)	0 (0)	
Creatinine	1.01 (0.28)	0.88 (0.16)	<0.001
Uric acid level (mg/dL)	6.33 (1.16)	5.01 (1.03)	<0.001
Data presented as mean (SD), unless otherwise specified. SD, standard deviation.			

Table 3: Distribution of cases and controls in relation to target organ damage

Parameters	Case (N=32)	Control (N=68)	P value
Risk factors			
FH	3 (9.38)	11 (16.18)	0.36
SMK	13 (40.63)	23 (33.82)	0.50
DM	-	-	-
LVH	10 (31.25)	1 (1.47)	<0.001
CAD	15 (46.88)	11 (16.18)	0.001
CVA	4 (12.5)	10 (14.71)	0.76
Data presented as n (%). CAD, coronary artery disease; CVA, cerebrovascular accident; DM, diabetes mellitus; FH, family history; LVH, left ventricular hypertrophy; SMK, smoking.			

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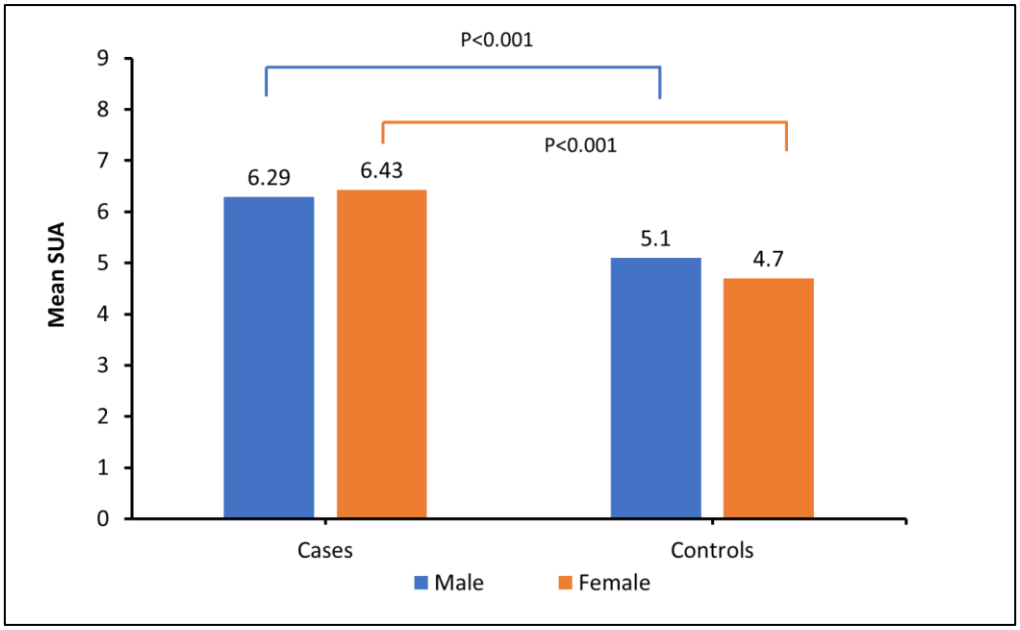

Figure 1: Comparison of SUA levels between cases and controls by gender SUA, serum uric acid.

DISCUSSION

Hyperuricemia is frequently observed in patients with HTN. Elevated SUA levels are strongly associated with the development of HTN and may act as an intermediary factor in its onset [8]. The present study aimed to evaluate SUA levels in patients with essential HTN and to investigate their association with factors such as ages, sex, BMI, smoking, diabetes, target organ damage and elevated uric acid.

In the current study, the mean age of the population among the cases and controls was 56.25 years and 52.4 years, respectively. These findings are consistent with those reported in a previous cross-sectional study, which found a mean age of 55.02 years among cases with HTN. Additionally, 28.9% of hypertensive cases were aged over 60 years [6]. Another case-control study, reported a mean age of 51.9 years among cases, while the mean age among controls was found to be 52.42 years [9].

An observational study reported a mean (SD) BMI of 26.04 (2.96) kg/m² in hypertensive cases,while the BMI in controls was 24.41 (2.71) kg/m² [10]. In another facility-based case control study, it was found that 8.5% of hypertensive cases had a BMI <18.5 kg/m², 57% had a normal BMI, 28% had a BMI between 25 and 29.9 kg/m², and 6.5% had a BMI >30 kg/m². Among controls, 3.5% had a BMI <18.5 kg/m² and 10% had a BMI between 25 and 29.9 kg/m² [5]. In the present study, the mean (SD) BMI among cases was 23.95 (2.49) kg/m², while for controls, it was 22.96 (2.5) kg/m².These findings suggest that BMI may still play a role in the development of HTN.

In a facility-based case-control study, the mean SBP among hypertensive case was 148.46 mmHg, while among controls, it was 121.44 mmHg. The mean DBP among cases was 94.46 mmHg, compared to 81.51 mmHg in the controls [5].A cross-sectional study found that the mean (SD) SBPwas 158.8 (15.9)mmHg in the cases and 114.8 (10.6) mmHgin the control group, while the mean (SD) DBP was 95.9 (11.1)mmHg in the cases and 74.0 (7.4) mmHgin the controls [11].Additionally, in a study by Kim YH, et al. the patient group had a mean SBP of 168.4 mmHg (range 130–220 mmHg) and a mean DBP of 114.5 mmHg (range 90–145 mmHg)[12]. In line with this study, in current study, the mean SBP was significantly higher in the cases (165.13 mmHg) compared tothe controls (122.29 mmHg). Similarly, DBP (103.94 mmHg) was also higher in the cases than in the controls (77.74 mmHg).The current studydemonstrates significantly higher blood pressure in hypertensive patients compared to controls.

In current study, the majority of cases (81.25%) and controls (70.59%)had significantly higher urea levels (>24mg/dL). The meanurea levels were 31.38mg/dL in the cases and 28.07mg/dL in the controls. A significant difference (P=0.01) was observed between the groups with and without HTN. Similarly, another study by Akpotaire PA, et al. reported the mean (SD) serum urea levels of 12.7 (6.23)mg/dLin the hypertensive group and 7.18 (5.06)mg/dL in the control group [13].This highlights the presence of higher urea levels among hypertensive patients compared to controls.

A study by Lee et al. [14] reported that elevated serum creatinine levels were closely correlated with higher SUA levels. Similarly, in a study by Kim YH, et al., hyperuricemia was found in approximately 51% of individuals with normal BUN and creatinine levels, compared to 88.9% in elevated creatinine group[12]. Additionally, in a study by Akpotaire PA, et al. the mean (SD) serum creatinine was 1.34 (0.96) mg/dLin the hypertensive group and 0.70 (0.14)mg/dLin the control group [15].In contrast, in present study, the majority of cases and controls had normal creatinine levels (0.8-1.2), with 59.38% of cases and 83.82% of

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controls. However, serum creatinine was significantly higher in the cases [1.01 (0.28 mg/dL)] compared to the controls [0.88 (0.16 mg/dL)], with a P-value of <0.001. Our findings show significantly higher serum creatinine levels in hypertensive cases compared to controls, despite most participants having normal creatinine levels.

In the present study the mean (SD) SUA level in cases was 6.33 (1.16) mg/dL, significantly higher than in the controls, where it was 5.01 (1.03) mg/dL (P<0.001). In consistent with this finding, a study conducted by Ansari RN et al. found that 65% of the 100 patients in their study had high SUA levels [16] . Similarly, in anobservational study, the mean SUA levels in cases was reported as 7.14 mg/dL, while in the control group it was 4.31 mg/dL (P<0.001) [8]. In a study by Kim YH, et al. the mean SUA was 6.60 (1.9) mg%(2.34–22.0 mg%) in the HTN group, whichwas significantly higher than that of the control group, where it was 3.87 (1.30) mg% (P<0.001) [12]. These findings collectively demonstrate significantly higher SUA levels among patients withHTNcompared to controls.

The Progetto Ipertensione Umbria Monitoraggio Ambulatoriale (PIUMA) study reported a significant increase in the onset of cardiovascular disease when SUA levels exceeded 6.2 mg/dL in males and 4.6 mg/dL in females [17].Similarly, an observational study reported significantly higher SUA levels in hypertensive cases compared to controls.Among the cases, the SUA level range in males was 1.80-11.50 mg/dL, with a mean value of 7.22 mg/dL, while in females it range was 1.29-10.80 mg/dL,with a mean value of 7.06mg/dL [8].In the present study, the mean SUA levels among hypertensive males were 6.29 (1.22) mg/dL and 6.43 (1.06) mg/dL among. In contrast, controls had mean levels of 5.1 (1.06) mg/dL for males and 4.7 (0.87) mg/dL for females (P<0.001). This indicates a significant difference in uric acid levels between hypertensive individuals and controls.

In the current study, LVH was significantly more prevalent in cases (31.25%)compared tocontrols (1.47%), with a P<0.001, followed by CAD. Similarly, LVH was more prevalent in hypertensive patients with hyperuricemia (70.5%) compared to those without hyperuricemia (42.0%), with a P=0.001 [11].

The study has its strengths and limitations. Strength of the study is that it identifies a clear relationship between serum uric acid levels and HTN, suggesting that SUA may contribute to oxidative stress and renal dysfunction through nephrosclerosis. However, a limitation of the study is the small sample size, whichmay restrict the generalizability of the findings to a larger population. Several studies support a positive correlation between SUA levels and HTN. However, due to ongoing debates about SUA is an independent cardiovascular risk factor, further prospective studies in newly diagnosed hypertensive populations are recommended.

CONCLUSION

Elevated SUA levelsare strongly associated withHTN compared and play a role in development of target organ damage, including LVH and CAD. However, factors such asfamily history, age, sex, BMI, diabetes and smoking do not correlate with SUA in patients with HTN. Findings highlight the potential importance of monitoring uric acid levels in patients with HTN, particularly among those with target organ involvement.

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