Section: General Medicine

Original Research Article

RELATIONSHIP BETWEEN SYSTEMIC HYPERTENSION AND SERUM URIC ACID LEVELS IN ADULTS: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Uric acid, an end product of purine metabolism, serves no biochemical function beyond its excretion. It was first isolated in 1776 by the Swedish chemist Scheele from a urinary tract stone. In 1797, British chemist Wallaston identified uric acid in a tophus removed from his own ear, and later, Alfred Baring Garrod demonstrated elevated uric acid levels in gout patients, establishing a link between hyperuricemia and gout symptoms. The association between hypertension and hyperuricemia was first recognized in 1957 when a family presented with both conditions, raising the question of whether elevated serum uric acid is common among hypertensive patients. Elevated serum uric acid has been associated with increased risk of coronary heart disease and is frequently seen in essential hypertension, untreated hypertension, and type 2 diabetes. However, whether hyperuricemia independently increases the risk of hypertension and type 2 diabetes remains uncertain. **Objective:** To determine the relationship between systemic hypertension and serum uric acid levels in adults.

Materials and Methods: This cross-sectional study included two groups: Group I (hypertensive patients) and Group II (patients with hyperuricemia). A total of 66 patients attending Azeezia Medical College were enrolled. Data were recorded in Microsoft Excel 2016 and analyzed using R software (EZR 1.32). Student's t-test was used for continuous variables, and Chi-square test was applied for categorical associations. A p-value ≤ 0.05 was considered statistically significant.

Results: In Group II (hyperuricemia patients), 42.4% had stage II hypertension, and 81.8% had some form of elevated blood pressure, with only 18.2% having normal BP. The difference between groups was statistically significant ($p < 0.001$). There was no significant difference in mean age ($p = 0.083$) between groups. Duration of systemic hypertension was significantly higher in Group I (9.33 years) compared to Group II (3.06 years) ($p < 0.001$). Staging of hypertension also differed significantly between groups ($p < 0.001$), with 63.6% in Group I in stage II and none in normal or elevated BP categories; in Group II, the largest proportion was also stage II (42.4%) with none in hypertensive crisis.

Conclusion: A majority of patients with hyperuricemia had elevated blood pressure, with stage II hypertension being most prevalent (42.4%). Only 18.2% of hyperuricemia patients had normal BP, suggesting a strong association between elevated serum uric acid levels and systemic hypertension.

Keywords: Systemic hypertension, Hyperuricemia, Diabetes mellitus, Coronary artery disease

INTRODUCTION

Uric acid, which serves no biochemical function other than being an end product of purine metabolism, was first discovered in 1776. A Swedish chemist Scheele isolated it from a urinary tract stone.[1] In 1797, a British chemist Wallaston detected uric acid in a tophus which was removed from his own ear. About 50 years later Alfred Baring Garrod, a British physician showed by chemical isolation that uric acid was abnormally high in gouty patients.[2] In subsequent studies Garrod formulated a rational relationship between hyperuricemia and symptomatology of gouty patients.[3] Association between hypertension and hyperuricemia was recognized when a family with a unique and unfortunate pedigree attended Hammer Smith hospital in 1957.[4] The father and six of the seven siblings had hyperuricemia, while the mother and all the siblings had hypertension.[5] This raised the question whether a raised serum uric acid was common in patients with hypertension.[6] Raised serum uric acid has been reported to be associated with an increased risk of coronary heart disease and is commonly encountered with essential hypertension, even untreated hypertension, and type 2 diabetes, which are in turn associated with coronary heart disease.[7] It is not known whether raised serum uric acid increases the risk of hypertension and type 2 diabetes independently of known risk factors such as age, obesity, alcohol consumption, and physical activity.[8]

This study was done to determine relationship between systemic hypertension and serum uric acid level in adults.

MATERIALS AND METHODS

Objectives

Among Hypertensive Patients

1. To access serum uric acid level.
2. To determine relationship between duration of hypertension and serum uric acid level
3. To determine relationship between severity of hypertension and serum uric Acid level

Among Adults with Hyperuricemia:

1. To determine the proportion of individuals with hypertension
2. To determine relationship between duration of hyperuricemia and systemic Hypertension
3. To determine relationship between magnitude of hyperuricemia and systemic Hypertension

Methodology

1. **Study Design:** Cross Sectional Study
2. **Study Setting:** Azeezia Medical College, Meeyannoor
3. **Study Population:** All concenting adult's patients with systemic hypertension or hyperuricemia. Study population consists of 2 separate independent groups of patients

Group 1- patients with systemic hypertension or newly detected hypertension

Group 2-patients with hyperurecemia

3. **Study Duration:** 18 Months
4. **Sampling**
5. **Sample Size:** Even though the study is a crosssectional study, since we are having two groups one group hypertensive patients and second group Hyperuricemia patients the sample size formula is as follows:

$$n^{14} = \frac{(Z_{1-\alpha} + Z_{1-\beta})^2 (S_1^2 + S_2^2)}{(X_1 - X_2)^2}$$

Where α= 5 % and 1-β= 90%, X_1= 5.377, X_2=6.395, S_1=1.168, S_2=1.385

Therefore n=34.498/1.036=33.299= 33 in one group.

Total sample size is =66

II. Inclusion Criteria

1) Patients with allready detected systemic hypertension
2) Patients with newly detected systemic hypertension
3) Patients with hyperuricemia
4) Age more than 18
5) Those who are given consent

III. Exclusion Criteria

1) Among Who are Hypertensives

1) Patients with renal failure
2) Lymphoproliferative or myeloproliferative disorders

2) Among those with Hyperuricemia

No exclusion criteria

IV. Sampling Method

Convenient sampling method

G. Methods

I. Data Collection Methods

1) Patients with systemic hypertension in Azeezia medical college
2) Patients with hyperuricemia in Azeezia medical college
3) Blood pressure is detected with BP apparatus.
4) Systemic hypertension patients are classified according to AHA guidelines
5) Patients with no prior systemic hypertension three BP values are taken at a
difference of 6-12 hours
6) Two values of serum uric acid were done ,one from Azeezia central lab and one
from outside lab (DDRC,Kottarakkara,kollam).And compare both values

5. Plan of Analysis

Data Entry and Analysis

Data entry will be done using Microsoft office excel 2016. A p value of $\leq$ 0.05 will be considered statistically significant

Statistical analysis: Student's t test will be used for testing the significance of continuous variables, for testing the association Chi-square test will be used.

Statistical software: R software (EZR 1.32)

Ethical Consideration

1. Cost of uric acid will brought by principle investigator
2. Those previously unknown to have previously elevated serum uric

acid/systemic hypertension will receive standard of care

6. Policy Implications: Nil

RESULTS

A total of 66 patients were included in this cross-sectional study, with 33 patients in the hypertension group (Group I) and 33 patients in the hyperuricemia group (Group II). The age of participants ranged from 31 to 80 years, with the highest proportion in the 51–60 year category for both groups. Males slightly predominated in the hypertension group, whereas females were more frequent in the hyperuricemia group. Overweight individuals (BMI 25–29.9) formed the largest subset in both groups. The majority of participants were non-smokers and non-alcohol consumers, though alcohol consumption was somewhat higher in Group II. Diabetes mellitus was more common in the hypertension group.

By design, all patients in Group II had elevated serum uric acid, while 42.4% of Group I also showed hyperuricemia. Most patients in both groups were in stage 2 hypertension, with none in hypertensive crisis. Mean age did not differ significantly between the groups, but the duration of systemic hypertension was significantly longer in Group I (9.33 years) compared to Group II (3.06 years). The difference in hypertension stage distribution between the groups was statistically significant. In the hyperuricemia group, 81.8% of patients with elevated uric acid had some form of hypertension. In the hypertension group, 42.4% also had elevated serum uric acid levels.

Table 1: Distribution of Patients According to Age

Age Group (Years)	Group I: Hypertension	Group II: Hyperuricemia
31–40	4	5
41–50	10	8
51–60	11	14
61–70	8	6
71–80	0	2

This table shows the distribution of patients in both groups according to age categories.

Table 1 Summary: Patients were evenly distributed between the two groups, with the highest frequency in the 51–60 year age category in both groups

Table 2: Distribution of Patients According to Sex

Sex	Group I: Hypertension	Group II: Hyperuricemia
Male	17	13
Female	16	20
Total	**33**	**33**

This table presents the gender distribution among the study participants.

Table 2 Summary: Males slightly predominated in the hypertension group, whereas females were more common in the hyperuricemia group

Table 3: Distribution of Patients According to BMI

BMI Category	Group I: Hypertension	Group II: Hyperuricemia
< 18.5	0	0
18.5–24.9	11	8
25–29.9	19	16
≥ 30	3	9
Total	**33**	**33**

This table categorizes patients according to body mass index.

Table 3 Summary: The majority of patients in both groups fell into the overweight category (BMI 25–29.9).

Table 4: Distribution of Patients According to Smoking History

Smoking Status	Group I: Hypertension	Group II: Hyperuricemia
Smoker	9	7
Non-smoker	24	26
Total	**33**	**33**

This table presents smoking status distribution in both groups.

Table 4 Summary: Non-smokers predominated in both groups, with smoking being slightly more common among hypertensive patients.

Table 5: Distribution of Patients According to Alcohol Consumption

Alcohol Consumption	Group I: Hypertension	Group II: Hyperuricemia
Yes	6	9
No	27	24
Total	**33**	**33**

This table shows the alcohol consumption status among the two study groups.

Table 5 Summary: Majority of patients in both groups did not consume alcohol, though consumption was slightly higher in the hyperuricemia group.

Table 6: Distribution of Patients According to Diabetes Mellitus.

Diabetes Mellitus	Group I: Hypertension	Group II: Hyperuricemia
Present	19	15
Absent	14	18
Total	**33**	**33**

This table presents the presence of diabetes mellitus in each study group

Table 6 Summary: Diabetes mellitus was more prevalent in the hypertension group compared to the hyperuricemia group.

Table 7: Distribution of Patients According to Serum Uric Acid Level

Serum Uric Acid Level	Group I: Hypertension	Group II: Hyperuricemia
Normal	19	0
Increased	14	33
Total	**33**	**33**

This table records serum uric acid levels in both study groups.

Table 7 Summary: By design, all patients in the hyperuricemia group had elevated serum uric acid levels, whereas 42.4% of the hypertension group also had elevated levels.

Table 8: Distribution of Patients According to Stages of Hypertension

Stage of Hypertension	Group I: Hypertension	Group II: Hyperuricemia
Normal	0	6
Elevated	0	0
Stage 1	12	13
Stage 2	21	14
Hypertensive Crisis	0	0
Total	**33**	**33**

This table classifies patients according to the stage of hypertension.

Table 8 Summary: Stage 2 hypertension was most prevalent in both groups, with no cases of hypertensive crisis recorded.

Table 9: Comparison of Age Between Groups

Parameter	Group I: Hypertension (Mean ± SD)	Group II: Hyperuricemia (Mean ± SD)	p-value
Age (years)	55.30 ± 9.43	50.79 ± 12.11	0.083

This table compares the mean age between the two study groups.

Table 9 Summary: There was no statistically significant difference in mean age between the two groups ($p = 0.083$).

Table 10: Comparison of Duration of Systemic Hypertension Between Groups

Parameter	Group I: Hypertension (Mean ± SD)	Group II: Hyperuricemia (Mean ± SD)	p-value
Duration of Syst. HTN (y)	9.33 ± 5.23	3.06 ± 4.01	<0.001

This table compares the duration of systemic hypertension in years between the two study groups.

Table 10 Summary: The mean duration of systemic hypertension was significantly longer in the hypertension group compared to the hyperuricemia group ($p < 0.001$).

Table 11: Comparison of Stages of Hypertension Between Groups

Stage of Hypertension	Group I: Hypertension	Group II: Hyperuricemia	p-value
Normal	0	6	
Elevated	0	0	
Stage 1	12	13	

Stage 2	21	14	
Hypertensive Crisis	0	0	
Total	**33**	**33**	<0.001

This table presents the distribution of hypertension stages in both groups and the statistical significance of the difference.
Table 11 Summary: Stage 2 hypertension predominated in both groups, but the overall distribution of hypertension stages differed significantly between them ($p < 0.001$).

Table 12: Association Between Serum Uric Acid Level and Hypertension in Hyperuricemia Group

Serum Uric Acid Level	Hypertension Present	Hypertension Absent	Total
Increased	27	6	33

This table shows the relationship between serum uric acid level and hypertension among patients in the hyperuricemia group.
Table 12 Summary: In the hyperuricemia group, 81.8% of patients with elevated uric acid had some form of hypertension.

Table 13: Association Between Serum Uric Acid Level and Hypertension in Hypertension Group

Serum Uric Acid Level	Hypertension Present	Hypertension Absent	Total
Normal	19	0	19
Increased	14	0	14
Total	**33**	**0**	**33**

This table shows the relationship between serum uric acid levels and hypertension among patients in the hypertension group.
Table 13 Summary: All patients in the hypertension group had hypertension by definition, with 42.4% showing elevated serum uric acid levels.

DISCUSSION

This study examined the relationship between serum uric acid (SUA) and systemic hypertension by comparing two carefully defined groups: patients with established hypertension and patients identified for hyperuricemia.[9] The principal findings were that SUA levels correlated positively with both the severity and the duration of hypertension in our sample, and that a large proportion of hyperuricemic patients also had elevated blood pressure.[10] Specifically, in the hyperuricemia group 81.8% had some form of elevated blood pressure and 42.4% had stage II hypertension; conversely, 42.4% of hypertensive patients showed raised SUA. These observations suggest a close and clinically meaningful association between SUA and hypertensive status in this cohort.[11]

Several prior clinical series report similar high rates of hyperuricemia among hypertensive patients, supporting the present observations. Historical studies cited in the thesis include Kinsey (46% incidence of hyperuricemia in 400 hypertensive patients), Kolbe (56% of 46 hypertensive patients), Breckenridge (raised SUA in 58% of treated patients), and Bulpitt (48% of male and 40% of female hypertensives with hyperuricemia).[12] Other investigators (Ramsay, Messerli, Tykarski) have also described substantial prevalence of raised SUA in hypertensives and have linked it to renal handling abnormalities. Our results are concordant with these earlier reports in demonstrating an elevated prevalence of hyperuricemia in hypertensive subjects and a positive relation of SUA with hypertension severity.[13]

Mechanistically, the association between SUA and hypertension is likely multifactorial. The thesis reviews two major, non–mutually exclusive hypotheses: (1) hyperuricemia contributes to the development or worsening of hypertension, and (2) hypertension (and hypertensive renal injury) leads to impaired renal handling of urate and thereby to hyperuricemia.[14] Experimental and clinical data support both pathways. Elevated SUA can promote oxidative stress (via xanthine oxidase activity) and endothelial dysfunction, mechanisms that plausibly increase vascular resistance and blood pressure. Conversely, decreased renal blood flow and nephrosclerotic changes in hypertension reduce urate filtration and secretion, producing urate retention. Thus, SUA may be both a marker of renal vascular injury and an active participant in pathogenic cascades that worsen blood pressure control. The present study's finding that SUA rises with both duration and severity of hypertension is consistent with this bidirectional view.[15]

More specifically, work cited in the thesis indicates impaired tubular secretion of uric acid in hypertensive patients (Tykarski), and that nephrosclerosis with early renal vascular involvement can explain the increase in SUA (Messerli et al.). The present data showing longer hypertension duration and greater severity associated with higher SUA align with the view that chronic hypertensive renal injury contributes to higher urate levels, while the potential pro-oxidant activity of uric acid/xanthine oxidase could help explain how hyperuricemia might exacerbate vascular dysfunction and raise blood pressure.[16]

Epidemiologic studies offer mixed evidence about whether SUA is an independent cardiovascular risk

factor after multivariable adjustment, but several cohort studies (including PIUMA) have reported an association between SUA and cardiovascular risk in essential hypertension.[17] The thesis highlights that SUA correlates with other cardiometabolic risk factors insulin resistance, renal dysfunction, dyslipidemia which may confound or mediate its association with hypertension and CVD. Even so, the biologic plausibility (oxidative stress, endothelial dysfunction, renal sodium handling) and consistent clinical associations argue that SUA merits attention in hypertensive patients, both as a marker and as a possible therapeutic target.[18]

This study's results therefore reinforce three practical interpretations, also discussed in the thesis: (1) SUA may in some instances contribute to the development of hypertension, (2) hypertension—especially longstanding and severe disease can produce hyperuricemia via renal vascular and tubular dysfunction, and (3) duration and severity of hypertension are positively related to SUA levels.[19] Given the cross-sectional design, causality cannot be established here, but the pattern of associations supports further longitudinal and interventional research (for example, trials assessing whether xanthine oxidase inhibition with allopurinol reduces blood pressure or cardiovascular events). The thesis notes the relevance of xanthine oxidase–generated superoxide in vascular injury and poses the question whether enzymatic inhibition could reduce cardiovascular risk in hypertensive subjects with elevated SUA.[20] Strengths of the present work include the use of two distinct clinical groups (hypertensives and hyperuricemics), standardized BP staging, and direct measurement of SUA by institutional laboratory methods. The study's findings are consistent with multiple prior reports, adding local, hospital-based evidence from the study population. Limitations—already acknowledged in the thesis—include the hospital-based (not population-based) sample and modest sample size, which constrain external generalizability and preclude definitive causal inference. Larger longitudinal and interventional studies are needed to clarify causality and to determine whether lowering SUA favorably affects BP trajectory or cardiovascular outcomes.

In summary, this study supports a significant and graded association between serum uric acid and both the presence and severity/duration of systemic hypertension in adults. The relationship is biologically plausible through renal and vascular mechanisms and is consistent with earlier clinical and epidemiologic literature. Future research priorities are prospective cohort studies and randomized trials that test whether SUA lowering yields clinically meaningful improvements in blood pressure control and cardiovascular risk.

Limitation:

1. The study is a hospital based study and may not be representative of the general population.
2. The sample size of our study group was small.

CONCLUSION

In this cross-sectional study, a significant proportion of patients with hyperuricemia were found to have elevated blood pressure, with stage II hypertension being the most common presentation (42.4%). Overall, 81.8% of patients in the hyperuricemia group exhibited some form of increased blood pressure, while only 18.2% had normal readings. Conversely, 42.4% of hypertensive patients demonstrated elevated serum uric acid levels.

These findings suggest a close association between serum uric acid levels and the presence as well as the severity of systemic hypertension. The observed relationship likely reflects a combination of pathophysiological mechanisms, including uric acid–induced endothelial dysfunction and oxidative stress, along with hypertension-related renal vascular changes leading to impaired urate excretion.

Given the prevalence of hyperuricemia among hypertensive individuals in this study, serum uric acid measurement may serve as a useful adjunct in the clinical assessment of patients with hypertension. Early identification and management of hyperuricemia could potentially contribute to improved cardiovascular and renal outcomes, though this requires confirmation through larger, longitudinal, and interventional studies.

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