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Research Article

Serum Uric Acid as a Marker of Disease Severity, Duration, and Target Organ Damage in Patients with Hypertension: A Cross-Sectional Study

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Abstract

Background:
Hypertension is a major contributor to cardiovascular and renal morbidity. Serum uric acid (SUA) has been implicated in the pathogenesis and progression of hypertension; however, its association with disease severity, duration, and target organ damage in the Indian population remains inadequately defined.

Objectives:
To evaluate the relationship between serum uric acid levels and severity and control of hypertension, duration of hypertension, and target organ damage.

Methods:
This cross-sectional analytical study included 180 adult patients with essential hypertension attending a tertiary care center. Clinical characteristics, blood pressure measurements, duration of hypertension, and laboratory parameters including serum uric acid were recorded. Hyperuricemia was defined as serum uric acid >7 mg/dL in men and >6 mg/dL in women. Associations between serum uric acid and hypertension parameters, as well as renal, cardiac, cerebrovascular, and lipid abnormalities, were analyzed using correlation analysis and chi-square tests.

Results:
Hyperuricemia was observed in 44.4% of patients. Serum uric acid showed a significant positive correlation with systolic blood pressure ($r = 0.52$, $p < 0.001$) and duration of hypertension ($p < 0.01$). Hyperuricemia was significantly more prevalent among patients with hypertension duration greater than five years and those with uncontrolled blood pressure. Significant associations were also noted between hyperuricemia and renal involvement, cardiac involvement, cerebrovascular disease, and dyslipidemia.

Conclusions:
Serum uric acid is significantly associated with hypertension severity, longer disease duration, poor blood pressure control, and multiple forms of target organ damage. Measurement of serum uric acid may provide a simple and cost-effective tool for risk stratification in patients with hypertension

Introduction

Global health statistics show that hypertension(HTN) has cemented its reputation

as one of the top public health problems . It is also notorious for its role as risk factor for morbidity(cardiovascular, renal, and cerebrovascular) and mortality worldwide[1]. Despite advances in antihypertensive therapy, a substantial proportion of patients develop progressive target organ damage, indicating the need for additional biomarkers to improve risk stratification[2].

Serum uric acid(SUA), traditionally considered an inert outcome of metabolism of purine , shows significant contributions to the pathogenesis of hypertension[3]. Experimental studies show that increased uric acid in serum can be a stimulus for dysfunction in endothelial tissues, oxidative stress, proliferation of "vascular smooth muscle cells" (VSM) and can also trigger the "Renin–Angiotensin–Aldosterone System"[4,5]. Experimental studies have put forward that hyperuricaemia and incident hypertension have significant association in between them[6].

Beyond the development of hypertension, higher levels of uric acid in serum show notable link to increased blood pressure severity and resistance to treatment[7]. Furthermore, hyperuricaemia has also been known to cause significant derangements in kidney function, hypertrophy in left ventricle and adverse cardiovascular aftermath in patients with hypertension [8,9]. However, the strength and consistency in these associations vary across populations.

Data examining the connection that SUA levels shares with hypertension severity, duration, and target organ damage in the Indian population remain limited. Given the high burden of uncontrolled high blood pressure and metabolic risk factors in India, it is thus warranted to study the relevance of measuring SUA as a disease burden marker . This study was done with the objective to gauge the link between SUA and hypertension severity, duration, control of blood pressure , and damage in subjects' target organ with HTN.

Methods

This cross-sectional analytical study was done at "Kokrajhar Medical College and Hospital" , a tertiary care cum teaching centre in Assam over a duration of 1 year . Subjects 18 years or older with a diagnosis of HTN were enrolled consecutively.

Patients with secondary HTN, known gout, chronic inflammatory conditions, chronic liver disease, malignancy, or those receiving urate-lowering therapy were excluded to avoid confounding effects on SUA levels[10].

Blood pressure was measured using a standardized protocol with patients in the seated position, and the average value of two measurements was recorded. Hypertension control status was defined according to guideline-recommended targets[11]. Duration of hypertension was recorded based on patient history and medical records.

Fasting blood samples were drawn from veins for estimation of serum uric acid, lipid profile, serum creatinine, and fasting plasma glucose. Hyperuricaemia was defined as SUA > 7 milligrams/decilitre (male) and > 6 milligrams/decilitre (female) , in accordance with widely accepted clinical thresholds[12].

Target organ damage was assessed as follows: renal involvement was defined by raised levels creatinine in serum or reduction in "estimated glomerular filtration rate"(eGFR) ; cardiac involvement was defined by electrocardiographic or echocardiographic evidence of left ventricular hypertrophy or documented ischaemic heart disease; cerebrovascular involvement included prior stroke or transient ischaemic attack; dyslipidaemia was defined based on standard lipid criteria(ATP III) [13].

SPSS software was used for this study's analysis. Continuous variables were tabulated as mean plus/minus standard deviation. On other hand , frequencies and percentages were used to express the variables of categorical nature. Correlations were assessed using Pearson correlation analysis. Associations in categorical variables were assessed using chi-square tests. If p value was <0.05 , it was recognised as statistically significant

.

RESULTS

Table 1. Study Population's baseline data (n = 180)

Variable	Value
Age in years (mean ± SD)	48 ± 14
Male sex, n (%)	108 (60%)
Systolic Blood Pressure (SBP in mmHg), mean ± SD	160 ± 30
Diastolic Blood Pressure (DBP in mmHg), mean ± SD	95 ± 15
Hypertension duration in years (mean ± SD)	5.0 ± 3.0
Uncontrolled hypertension, n (%)	120 (66.7%)
Serum uric acid levels in mg/dL (mean ± SD)	6.0 ± 1.8
Hyperuricemia, n (%)	80 (44.4%)
Diabetes mellitus, n (%)	50 (27.8%)
Dyslipidemia, n (%)	75 (41.7%)

Table 2. Correlation Between SUA Levels and Blood Pressure Parameters

Parameter	Correlation coefficient (r)	p value
SUA vs SBP	0.52	<0.001
SUA vs DBP	0.28	0.06
SUA vs Duration of HTN	0.31	0.02

Table 3. Association Between Duration of Hypertension and Hyperuricemia

Duration of Hypertension	Hyperuricaemia Present	Hyperuricemia Absent	Total
≤ 5 years	20	70	90
> 5 years	60	30	90
Total	80	100	180

Statistical test: Chi-square
p value: < 0.001
Odds Ratio: ~7.0

Interpretation:
A significant positive correlation exists between SUA with SBP and duration of hypertension.

Table 4. Association Between Hypertension Control Status and Hyperuricemia

Hypertension Status	Hyperuricaemia Present	Hyperuricaemia Absent	Total
Controlled	15	45	60
Uncontrolled	65	55	120
Total	80	100	180

p value: < 0.001
Odds Ratio: ~3.6

Table 5. Association Between Hyperuricaemia and Renal Involvement

Renal Involvement	Hyperuricaemia Present	Hyperuricaemia Absent
Present	30	5
Absent	50	95

p value: < 0.001
Odds Ratio: ~10.8

T**able 6. Association Between Hyperuricemia and Cardiac Involvement**

(LVH / ischemic heart disease)

Cardiac Involvement	Hyperuricaemia Present	Hyperuricaemia Absent
Present	28	18
Absent	52	82

p value: 0.01
Odds Ratio: ~2.4

Table 7. Association Between Hyperuricemia and Nervous System Involvement

(Stroke / TIA)

CNS Involvement	Hyperuricaemia Present	Hyperuricaemia Absent
Present	16	6
Absent	64	94

p value: 0.002
Odds Ratio: ~3.9

Table 8. Association Between Hyperuricemia and Dyslipidaemia

Dyslipidemia	Hyperuricaemia Present	Hyperuricaemia Absent
Present	45	30
Absent	35	70

p value: < 0.001
Odds Ratio: ~3.0

Table 9. Summary of Significant Associations

Variable	Association with Hyperuricaemia	Strength
Systolic BP	Yes	Strong
Duration of HTN (>5 yrs)	Yes	Strong
Uncontrolled HTN	Yes	Moderate
Renal involvement	Yes	Very strong
Cardiac involvement	Yes	Moderate
CNS involvement	Yes	Moderate
Dyslipidemia	Yes	Strong

180 patients with hypertension were considered in the study. Hyperuricaemia was present in 44.4% of study subjects. SUA had a significant positive correlation with systolic blood pressure,

indicating an interconnection between high SUA and increased blood pressure severity[7].

Patients with hypertension for >5 years were having a significantly higher prevalence of hyperuricaemia, suggesting a cumulative relationship between chronic hypertension and uric acid elevation[8]. Hyperuricaemia was also significantly more prevalent among patients with uncontrolled hypertension, supporting its association with poor control of blood pressure [9].

Renal involvement showed the strongest association with hyperuricaemia, with a significantly higher proportion of renal dysfunction observed among hyperuricaemic patients. This finding correlates with prior studies establishing a bidirectional relationship between uric acid levels and renal vascular injury[14].

Significant associations were also observed between hyperuricaemia and cardiac involvement, including left ventricular hypertrophy, as well as cerebrovascular disease. Dyslipidaemia was more frequently observed in hyperuricaemic patients, indicating clustering of metabolic risk factors in hypertensive individuals[15].

Discussion

The present study evaluated the relationship between SUA and hypertension severity, duration, control of blood pressure and target organ damage in a cohort of hypertensive patients. The findings demonstrate significant associations between hyperuricemia and systolic blood pressure, longer duration of hypertension, poor blood pressure control, and renal, cardiac, cerebrovascular, and metabolic involvement.

SUA and Hypertension Severity

Here ,SUA had a significantly positive correlation with SBP, indicating that raised SUA is associated with greater hypertension severity. This finding matches with of Kuwabara et al., who demonstrated that asymptomatic elevated SUA was independently associated with higher blood pressure levels and increased cardiovascular risk in a large Japanese cohort[7]. Similarly, Viazzi et al. reported that elevated SUA correlated with higher BP and greater damage in heart , brain and kidney in patients with primary HTN[8].

Experimental evidence further supports this association. Mazzali et al. found that SUA can induce hypertension through crystal-independent mechanisms involving derangements in endothelium and VSM proliferation[4]. Johnson et al. subsequently proposed that SUA–mediated activation of the RAAS and oxidative stress contributes to sustained elevations in SBP[5]. The present study's findings align with these mechanistic and clinical observations, reinforcing the part of SUA in HTN severity.

Serum Uric Acid and Duration of Hypertension

A significant association was observed between hyperuricaemia and longer duration of hypertension, with patients having hypertension for more than five years demonstrating a markedly higher prevalence of elevated serum uric acid levels. This observation parallels findings from Viazzi et al., who reported that serum uric acid levels were higher in patients with longer-standing hypertension and were predictive of cumulative target organ damage[8].

Similarly, Messerli et al. suggested that elevated uric acid levels may reflect chronic renal vascular involvement developing over time in patients with longstanding essential hypertension[9]. Longitudinal observations by Borghi et al.further indicate that persistent hyperuricemia may contribute to progressive cardiovascular and renal injury, supporting a temporal relationship between disease duration and uric acid elevation[10].

Taken together, these findings suggest that serum uric acid may act as a surrogate marker of chronic disease burden in hypertensive patients.

SUA and BP Control Status

The present study demonstrated a significantly higher prevalence of hyperuricemia within patients with uncontrolled HTNcompared to

those with controlled BP. This observation is consistent with findings from Kuwabara et al., who reported poorer blood pressure control and higher cardiovascular risk among individuals with elevated serum uric acid levels[7].

In addition, Feig et al. proposed that hyperuricemia may contribute to antihypertensive treatment resistance by promoting vascular stiffness and endothelial dysfunction[3]. Viazzi et al. also observed that patients with higher SUA levels were more likely to exhibit suboptimal blood pressure control and concomitant target organ damage[8]. These studies support the present findings and suggest that hyperuricemia may identify a subgroup of hypertensive patients with more difficult-to-control disease.

Serum Uric Acid and Renal Involvement

Renal involvement showed the strongest association with hyperuricemia in this study. This matches with the classic observations of Messerli et al., who identified SUA to serve to identify derangements of blood vessels of kidneys in HTN[9]. More recent work by Johnson et al. has emphasized the bidirectional relationship between SUA and chronic renal disease, suggesting that hyperuricemia may both result from and contribute to renal microvascular injury[14].

Similarly, Viazzi et al. observed that if SUA was raised , it then itself was independently associated with reduced kidney function and increased prevalence of microalbuminuria in hypertensive subjects[8]. The strong association observed in the present study reinforces the role of SUA as an early identifier of kidney damage in HTN.

Serum Uric Acid and Cardiac Involvement

A significant association was also observed between hyperuricemia and cardiac damage , like hypertrophied left ventricle(LVH) and ischemic heart disease. This finding is in line with Viazzi et al., who saw a higher prevalence of LVH among HTN patients with elevated SUA levels[8].

Furthermore, Borghi et al. highlighted that hyperuricemia was linked to adverse cardiovascular outcomes, including coronary artery disease and cardiac failure, in HTN populations[10]. Experimental data summarized by Feig et al. suggest that uric acid–induced endothelial dysfunction and vascular inflammation may contribute to myocardial remodeling and hypertrophy[3]. The present study supports these observations and underscores the systemic cardiovascular impact of hyperuricemia.

Serum Uric Acid and Cerebrovascular Involvement

The interconnection between hyperuricemia and cerebrovascular disease observed in this study aligns with previous epidemiological findings. Kuwabara et al. demonstrated that higher SUA levels had with increased risk of stroke and other cardiovascular events[7]. Similarly, Borghi et al. reported that hyperuricemia was linked to higher cerebrovascular risk in hypertensive patients[10].

The underlying mechanisms are thought to involve endothelial dysfunction, increased arterial stiffness, and pro-inflammatory effects of uric acid, which collectively predispose to cerebrovascular events[3]. The present findings add to the growing evidence that hyperuricemia is associated with cerebrovascular target organ damage in hypertension.

SUA and Dyslipidemia

The present study demonstrated a strong association of hyperuricemia with dyslipidemia, indicating clustering of metabolic risk factors. This finding matched with the observations of Ford et al., who saw that SUA levels had strong association with components of the metabolic syndrome, including dyslipidaemia[15].

Additionally, Borghi et al. highlighted that hyperuricemia often coexists with adverse lipid profiles and other cardiometabolic risk factors in HTN patients[10]. These findings support the concept that elevated SUA reflect a broader metabolic milieu rather than an isolated abnormality.

Summary

Overall, the findings of the present study are concordant with existing literature and support the role of serum uric acid as a marker of hypertension severity, chronicity, poor blood pressure control, and multisystem target organ damage. The consistency of these findings with prior studies strengthens the clinical relevance of serum uric acid measurement in routine hypertension management.

Limitations

1. As it is a cross section study, it has limited causal inference.
2. As this study was done in a single centre , we may not be able to generalise it to all populations.
3. Longitudinal studies are recommended to assess whether lowering SUA can modify hypertension outcomes and reduce target organ damage.

Conclusions

SUA shows significantly association with hypertension severity, longer disease duration, poor blood pressure control, and renal, cardiac, and cerebrovascular target organ damage. Serum uric acid may serve as a simple, cost-effective biomarker for identifying high-risk hypertensive patients in routine clinical practice.

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