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ORIGINAL RESEARCH

SERUM URIC ACID LEVELS IN ESSENTIAL HYPERTENSION AND THEIR ASSOCIATION WITH THE SEVERITY AND DURATION OF DISEASE

Rahul Mahajan[1], **Navpreet Kaur**[2*]

[1] *Senior Consultant, Department of Neurosciences, Max Hospital, Mohali, Punjab, India.*

[2] *Assistant Professor, Department of Pathology, Dr. B. R. Ambedkar State Institute of Medical Sciences, Mohali, Punjab, India.*

*Correspondence: Navpreet Kaur <dr.navpreet3489@gmail.com>

ABSTRACT

Hypertension is a leading cause of cardiovascular and renal morbidity and mortality. Emerging evidence suggests that serum uric acid (SUA) may contribute to the pathogenesis of essential hypertension through endothelial dysfunction and oxidative stress, yet population-based data from North India, particularly Punjab, remain limited. To measure serum uric acid levels in patients with essential hypertension and to examine their association with the severity and duration of disease. This hospital-based, cross-sectional, observational study enrolled 100 patients older than 18 years with essential hypertension diagnosed according to JNC VII criteria at the Department of Medicine, Rajindra Hospital, Patiala. Patients were categorised by severity (Stage I and Stage II) and by disease duration (fewer than 5 years and more than 5 years). Those with secondary hypertension, diabetes mellitus, obesity, renal disease, or conditions and drugs known to affect SUA were excluded. Serum uric acid was measured by the uricase–PAP end-point method. The mean age was 50.88 ± 13.65 years and 72% of participants were male. The mean SUA was 6.04 ± 1.55 mg/dl (range 3.20–9.50 mg/dl), and hyperuricaemia (SUA > 6.8 mg/dl) was present in 35%. SUA increased significantly with advancing age ($p < 0.001$). Patients with Stage II hypertension had significantly higher SUA than those with Stage I disease (6.45 ± 1.65 vs 5.21 ± 0.86 mg/dl; $p < 0.001$), and patients with a disease duration exceeding 5 years had higher levels than those with shorter-duration disease (6.96 ± 1.21 vs 5.22 ± 1.36 mg/dl; $p < 0.001$). No significant sex difference was observed. Serum uric acid is significantly associated with both the severity and the duration of essential hypertension. Routine SUA estimation may serve as a useful adjunct for cardiovascular and renal risk stratification in hypertensive patients.

Keywords: Essential hypertension; Serum uric acid; Hyperuricaemia; Cardiovascular risk; Severity of hypertension; Duration of hypertension.

INTRODUCTION

Hypertension ranks among the foremost contributors to global cardiovascular morbidity and mortality and remains one of the most pressing public health challenges worldwide. The World Health Organization estimates that approximately 40% of adults aged 25 years or older are hypertensive. Of the roughly 17 million deaths from cardiovascular disease each year, around 9.4 million are directly attributable to raised blood pressure, which underlies an estimated 45% of deaths from ischaemic heart disease and 51% of deaths from stroke [1]. In developing countries such as India, prevalence continues to rise, driven by rapid urbanisation, sedentary lifestyles, obesity, dietary change, psychological stress, smoking, alcohol consumption, and increasing life expectancy.

The relationship between blood pressure and cardiovascular risk is continuous and graded. In a meta-analysis of individual data from more than one million adults enrolled in 61 prospective studies, mortality from ischaemic heart disease and stroke increased progressively from systolic pressures above 115 mmHg and diastolic pressures above 75 mmHg; each 20 mmHg increment in systolic or 10 mmHg increment in diastolic pressure was associated with approximately a doubling of cardiovascular mortality [2]. Hypertension is thus a well-established, independent risk factor for cardiovascular disease, with no discernible threshold below which risk plateaus [2].

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Clinically, hypertension is defined as the level of blood pressure above which treatment reduces blood-pressure–related morbidity and mortality, conventionally taken as a sustained systolic pressure ≥ 140 mmHg and/or diastolic pressure ≥ 90 mmHg [3]. The Seventh Report of the Joint National Committee (JNC VII) classifies blood pressure into prehypertension, Stage I, and Stage II disease [4]. Essential (primary) hypertension accounts for 90–95% of all cases; the remainder are secondary to renal, endocrine, vascular, or neurological causes [5,6]. The pathogenesis of essential hypertension is multifactorial, encompassing increased peripheral vascular resistance, endothelial dysfunction, sympathetic overactivity, abnormal renal sodium handling, insulin resistance, oxidative stress, and genetic predisposition [6,7].

Often characterised as a “silent killer”, hypertension may remain asymptomatic for years while progressive target-organ damage accrues [8]. Sustained pressure elevation affects the heart, kidneys, retina, and brain, and is a major risk factor for coronary artery disease, myocardial infarction, congestive heart failure, stroke, chronic kidney disease, and hypertensive retinopathy [9]. The modifiable contributors are well characterised: obesity confers a substantial independent risk, as demonstrated in the Framingham Heart Study [9], while physical inactivity, high dietary sodium intake, smoking, and alcohol use each raise blood pressure through distinct but partially overlapping mechanisms [10].

Serum uric acid (SUA) has emerged as a clinically relevant biochemical correlate of hypertension and cardiovascular disease. Uric acid is the terminal metabolite of purine catabolism in humans and is excreted principally by the kidneys; its production involves the enzyme xanthine oxidase, which is also a source of reactive oxygen species [11]. Hyperuricaemia is conventionally defined as a serum uric acid concentration exceeding 6.8 mg/dl, the limit of urate solubility at physiological temperature and pH. Although classically associated with gout, elevated SUA is now increasingly recognised in the context of hypertension, metabolic syndrome, chronic kidney disease, and cardiovascular disorders.

The association between hypertension and hyperuricaemia has been recognised for several decades. As early as 1966, Breckenridge documented elevated SUA in hypertensive patients and attributed the finding to impaired renal urate excretion [12]. In animal models, induced hyperuricaemia consistently raises blood pressure, and this effect can be prevented by uricosuric agents or xanthine oxidase inhibitors [13]. The proposed mechanisms linking uric acid to hypertension include endothelial dysfunction, activation of the renin–angiotensin system, oxidative stress and free-radical generation, vascular smooth-muscle proliferation, reduced nitric oxide bioavailability, and hypertensive microvascular disease in the renal cortex [14,15]. A comprehensive appraisal by Feig, Kang, and Johnson synthesised the experimental and clinical evidence for these pathways and proposed that hyperuricaemia may represent a genuine causal contributor to, rather than merely a marker of, hypertension and cardiovascular disease [16].

Numerous clinical investigations have reported a high prevalence of hyperuricaemia among hypertensive patients, though estimates vary considerably with the case mix and treatment exposure. Early series by Kinsey et al. and Kolbel et al. described rates of 46% and 56%, respectively [17,18]. Cannon et al. found hyperuricaemia in 25% of untreated hypertensives, rising to 50% in patients receiving diuretics and exceeding 75% in malignant hypertension [19]. Bulpitt et al. reported an overall prevalence of 43.8%, particularly among men and patients on thiazide diuretics [20], while Kim et al. observed significantly higher SUA in essential hypertension than in normotensive controls and noted an association with target-organ damage [21]. Dzielak and Kivlighn found rates of 25–50% in untreated patients and linked hyperuricaemia to increased cardiovascular mortality [22].

Long-term follow-up studies have reinforced these observations. In a 20-year prospective cohort of 7978 treated hypertensive patients, Alderman et al. demonstrated that elevated SUA was independently associated with cardiovascular events after adjustment for major risk factors [23]. Verdecchia et al. similarly reported that raised SUA in untreated essential hypertension independently predicted cardiovascular events, fatal cardiovascular outcomes, and all-cause mortality [24]. A direct relationship between SUA and hypertension severity has also been described: Campo et al. documented rising SUA with increasing severity [25], Ouppatham et al. identified positive correlations with both systolic and diastolic blood pressure in a large occupational cohort [26], and both Kumar et al. and Meti et al. reported significantly higher SUA in Stage II disease and with longer disease duration [27,28].

Prospective data further support a role for SUA as an independent predictor of incident hypertension. Nagahama et al. found that baseline hyperuricaemia predicted future hypertension in both sexes in a screened Japanese cohort [29].

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Sundström et al., drawing on Framingham data, demonstrated that raised SUA predicted both the incidence of hypertension and the longitudinal progression of blood pressure [30]. Krishnan et al. confirmed that normotensive men with hyperuricaemia at baseline carried a higher risk of incident hypertension even in the absence of metabolic syndrome [31], and Soletsky and Feig showed that urate-lowering therapy with allopurinol significantly reduced systolic and diastolic pressure in obese prehypertensive adolescents [32]. A systematic review and meta-analysis by Grayson et al., pooling prospective data from more than 15,000 participants across 18 cohort studies, confirmed that baseline hyperuricaemia was independently associated with incident hypertension (pooled relative risk 1.41; 95% CI 1.23–1.58), providing the most robust summary estimate of this relationship to date [33].

Despite this accumulating body of evidence, the precise nature of the relationship between hyperuricaemia and essential hypertension remains incompletely characterised, and population-based data from North India, particularly Punjab, remain limited. The present study was therefore undertaken to measure serum uric acid in patients with essential hypertension and to examine its association with the severity and duration of disease.

MATERIALS AND METHODS

This was a hospital-based, cross-sectional, observational study conducted in the Department of Medicine, Rajindra Hospital, Patiala. One hundred patients with essential hypertension diagnosed according to JNC VII criteria [4] were enrolled consecutively over the study period.

Inclusion criteria

Patients of either sex aged more than 18 years were eligible. Hypertension was defined as a systolic pressure ≥ 140 mmHg and/or a diastolic pressure ≥ 90 mmHg [3,4]. Patients were classified by severity into Stage I (systolic 140–159 mmHg and/or diastolic 90–99 mmHg) and Stage II (≥ 160 mmHg systolic and/or ≥ 100 mmHg diastolic) hypertension, and by duration of diagnosed hypertension into fewer than 5 years and more than 5 years.

Exclusion criteria

Patients were excluded if they had any of the following: secondary hypertension; diabetes mellitus; obesity (body mass index ≥ 30 kg/m²); coronary artery disease or congestive cardiac failure; renal disease; hypothyroidism; or hyperparathyroidism. Patients with alcohol misuse disorder were also excluded, as were those receiving drugs known to influence serum uric acid, including allopurinol, febuxostat, ethambutol, pyrazinamide, levodopa, nicotinic acid, cytotoxic agents, low-dose aspirin, or thiazide diuretics.

Clinical assessment

A standardised history was taken in every patient, documenting the duration and treatment history of hypertension, as well as relevant comorbidities, family history, dietary and lifestyle factors, and current medications. A thorough general physical and systemic examination was performed. Blood pressure was measured in the seated position after at least five minutes of rest using a calibrated mercury sphygmomanometer and an appropriate cuff size [3]. Participants were instructed to abstain from smoking and caffeine for at least 30 minutes beforehand. A minimum of two readings were recorded on each occasion, and systolic and diastolic pressures were identified at the appearance (Korotkoff phase I) and disappearance (phase V) of the Korotkoff sounds, respectively. Standard investigations were performed, including a complete blood count, renal function tests, fasting blood glucose, electrocardiogram, and serum uric acid.

Biochemical analysis

Serum uric acid was estimated from a fasting venous blood sample using the uricase–PAP end-point method [34,35]. In this assay, uricase oxidises uric acid to allantoin and hydrogen peroxide; the hydrogen peroxide reacts with 4-aminoantipyrine and a chromogen in the presence of peroxidase to form a red quinoneimine dye whose absorbance, measured colorimetrically at 520 nm, is proportional to the uric acid concentration. Hyperuricaemia was defined as SUA > 6.8 mg/dl.

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Statistical analysis

Data were entered and managed in Microsoft Excel and analysed using SPSS. Categorical variables are presented as frequencies and percentages; continuous variables are expressed as mean ± standard deviation. p value of less than 0.05 was considered statistically significant.

RESULTS

One hundred patients with essential hypertension were studied. The data were analysed with respect to age, sex, stage and duration of hypertension, and serum uric acid level.

The majority of patients (49%) were in the 41–60-year age group; the mean age was 50.88 ± 13.65 years (range 23–82 years). Seventy-two per cent were male and 28% were female. Sixty-seven per cent had Stage II hypertension and 33% had Stage I (Table 1). Fifty-three per cent had a disease duration of fewer than 5 years and 47% had been hypertensive for more than 5 years.

Table 1. Distribution of patients by stage of hypertension

Stage of hypertension	Number of cases	Percentage (%)
Stage I	33	33.0
Stage II	67	67.0
Total	100	100.0

The mean serum uric acid level in the study population was 6.04 ± 1.55 mg/dl (range 3.20–9.50 mg/dl). Hyperuricaemia (SUA > 6.8 mg/dl) was present in 35 of the 100 patients, giving a prevalence of 35% (Table 2).

Table 2. Prevalence of hyperuricaemia and mean serum uric acid in the study population

SUA category	Number of cases	Percentage (%)
Normal (≤ 6.8 mg/dl)	65	65.0
Hyperuricaemia (> 6.8 mg/dl)	35	35.0
Total	100	100.0
Range	3.20-9.50 mg/dl	
Mean ± SD	6.04 ± 1.55 mg/dl	

Serum uric acid rose progressively with age. Mean levels were 4.94 ± 1.10 mg/dl in patients aged ≤ 40 years, 5.92 ± 1.50 mg/dl in those aged 41–60 years, and 7.22 ± 1.16 mg/dl in those older than 60 years; the corresponding prevalence of hyperuricaemia was 12.5%, 24.5%, and 74%, respectively. Both the trend in mean SUA and the trend in hyperuricaemia prevalence across age groups were highly significant ($p < 0.001$). By contrast, no significant sex-based difference was observed: mean SUA was 6.01 ± 1.60 mg/dl in men and 6.11 ± 1.43 mg/dl in women, with hyperuricaemia in 32% and 42%, respectively (p = not significant).

Serum uric acid was strongly associated with the severity of hypertension. Patients with Stage I disease had a mean SUA of 5.21 ± 0.86 mg/dl, compared with 6.45 ± 1.65 mg/dl in those with Stage II disease ($p < 0.001$; Table 3). Hyperuricaemia was present in only 9% of Stage I patients, rising to 48% of Stage II patients ($p < 0.001$).

Table 3. Mean serum uric acid level by stage of hypertension

Stage of hypertension	No. of patients	Mean SUA (mg/dl) ± SD
Stage I	33	5.21 ± 0.86
Stage II	67	6.45 ± 1.65
Total	100	

SUA, serum uric acid; SD, standard deviation. Difference between Stage I and Stage II: $p < 0.001$

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Disease duration showed a comparable pattern. Patients hypertensive for fewer than 5 years had a mean SUA of 5.22 ± 1.36 mg/dl, whereas those with a duration exceeding 5 years had a mean of 6.96 ± 1.21 mg/dl ($p < 0.001$). The prevalence of hyperuricaemia rose from 9.4% in the shorter-duration group to 63.8% in the longer-duration group ($p < 0.001$; Table 4).

Table 4. Serum uric acid and prevalence of hyperuricaemia by duration of hypertension

Duration of hypertension	Mean SUA (mg/dl) ± SD	Hyperuricaemia prevalence	p-value
< 5 years	5.22 ± 1.36	9.4%	< 0.001
> 5 years	6.96 ± 1.21	63.8%	

SUA: Serum uric acid.

DISCUSSION

Elevated serum uric acid has been linked in numerous studies to increased cardiovascular morbidity and mortality through mechanisms including endothelial dysfunction, oxidative stress, inflammatory activation, and enhanced platelet aggregation [36]. The evidence is not entirely uniform: the original Framingham Heart Study analysis, for instance, did not retain SUA as an independent cardiovascular risk factor after full multivariate adjustment. However, the weight of more recent, better-powered evidence including the comprehensive review by Feig et al. [16] supports a genuine association between hyperuricaemia and both hypertension and cardiovascular disease, and has led to renewed debate about whether uric acid plays a pathogenic rather than merely a biomarker role.

Because SUA is linked to several established cardiovascular risk factors hypertension, renal dysfunction, obesity, insulin resistance, and dyslipidaemia it remains contentious whether it constitutes an independent risk factor or is principally a marker of associated metabolic derangement. Hypertension is itself among the most important of these factors, and the relationship between hypertension and raised SUA is increasingly well documented in the literature. The present study examined that relationship specifically in terms of hypertension severity and disease duration.

The mean age of our patients (50.88 ± 13.65 years) is comparable to that reported by Dasti et al. in a similar hospital-based study [37], and the male preponderance (72%) mirrors the sex distribution described by Ouppatham et al. [26]. Earlier work has produced variable findings regarding sex predilection in hypertension-associated hyperuricaemia; in the present cohort, SUA did not differ significantly between men and women. The mean SUA of 6.04 ± 1.55 mg/dl is consistent with that reported by Kim et al. [21], and the modest inter-study variation in mean SUA across the literature most likely reflects differences in demographic profile, ethnicity, dietary practices, inclusion criteria, and antihypertensive drug use.

The 35% prevalence of hyperuricaemia in this study is consistent with the figures reported by Dzielak and Kivlighn [22], Kashem et al. [38], and Meti et al. [28]. The considerably higher prevalence described in some older series, including those of Kinsey et al. [17] and Kolbel et al. [18], probably reflects the inclusion of patients with secondary hypertension and those receiving thiazide diuretics, both of which substantially raise SUA. Notwithstanding these methodological differences, the consistent message across studies is that hyperuricaemia is substantially more common in hypertensive patients than in the general population.

The directionality of the hypertension–hyperuricaemia relationship remains an area of active debate. Two mechanistic explanations have been advanced. The first holds that hyperuricaemia contributes causally to hypertension: Puig and Ruilope proposed that uric acid and the superoxide radicals generated during purine catabolism by xanthine oxidase cause vascular endothelial injury and impair nitric oxide synthesis [15]; experimental work by Johnson et al. [14] demonstrated that induced hyperuricaemia raises blood pressure in the rat through a crystal-independent mechanism; and the clinical evidence assembled by Feig, Kang, and Johnson provided a coherent mechanistic basis for a causal role [16]. Intervention studies by Soletsky and Feig further showed that lowering SUA with allopurinol reduced systolic and diastolic pressure in obese prehypertensive adolescents [32], and the meta-analysis by Grayson et al. confirmed that hyperuricaemia at baseline independently predicted incident hypertension in prospective cohorts [33]. The second, non-mutually exclusive explanation is that hypertension causes hyperuricaemia through haemodynamic and structural effects on the kidney.

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Breckenridge attributed the raised SUA in hypertensive patients to reduced renal urate clearance [12], and Messerli et al. proposed that hypertensive nephrosclerosis impairs tubular urate secretion [39]. Tykarski provided direct evidence for this mechanism, demonstrating reduced tubular urate secretion as the principal determinant of raised SUA in essential hypertension [40]. On this interpretation, hyperuricaemia is an early biochemical marker of hypertensive renal microvascular involvement.

Serum uric acid rose significantly with age in our cohort, from 4.94 ± 1.10 mg/dl in patients aged 40 years or below to 7.22 ± 1.16 mg/dl in those older than 60 years, with hyperuricaemia prevalence increasing from 12.5% to 74% across the same range. These findings accord with those reported by Meti et al. [28] and Murugan and Padmavathi [41]. The age-related increment in SUA most likely reflects the progressive decline in glomerular filtration rate and the vascular changes that accompany normal ageing, leading to reduced renal urate clearance. The absence of a statistically significant sex difference is consistent with the observations of Murugan and Padmavathi [41], Meti et al. [28], and Vyas et al. [42], and may partly reflect the attenuation of the sex differential in urate metabolism that occurs after the menopause.

The association between SUA and hypertension severity was particularly pronounced. Mean SUA increased from 5.21 ± 0.86 mg/dl in Stage I to 6.45 ± 1.65 mg/dl in Stage II disease ($p < 0.001$), and hyperuricaemia was more than five times more prevalent in Stage II patients (48% vs 9%). Comparable severity-related differences have been reported by Kumar et al. [27] and Meti et al. [28], and the disproportionately high SUA in more severe hypertension may reflect both greater haemodynamic stress and more pronounced renal vascular damage, reducing urate clearance in a severity-dependent fashion.

An equally striking gradient was observed with disease duration: mean SUA rose from 5.22 ± 1.36 mg/dl in patients hypertensive for fewer than 5 years to 6.96 ± 1.21 mg/dl in those with longer-standing disease ($p < 0.001$), and the prevalence of hyperuricaemia increased from 9.4% to 63.8%. These findings are consistent with the observations of Vyas et al. [42], Kumar et al. [27], and Meti et al. [28], and are attributable to the progressive nephrosclerosis and decline in renal function that characterise long-standing uncontrolled hypertension.

Taken together, the present findings demonstrate a significant and graded association between serum uric acid and essential hypertension, with SUA levels and hyperuricaemia prevalence rising in parallel with age, severity, and duration of disease. The cross-sectional design precludes causal inference, but the consistency of the findings with both mechanistic data and prospective epidemiological evidence supports a clinically meaningful relationship between SUA and the hypertensive phenotype.

CONCLUSION

In patients with essential hypertension, serum uric acid increased significantly with advancing age, greater disease severity, and longer disease duration; no significant sex-based difference was observed. Mean SUA was significantly higher in Stage II than in Stage I hypertension, and higher in patients with more than 5 years' disease duration than in those with shorter-duration disease, with correspondingly higher rates of hyperuricaemia in each instance. Serum uric acid is thus positively and significantly associated with essential hypertension and correlates with both its severity and its duration. These findings support the inclusion of routine SUA estimation in the biochemical evaluation of patients with essential hypertension, where it may serve as a practical and inexpensive marker for cardiovascular and renal risk stratification.

Conflict of interest: None declared.

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