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Original Research Article

PROSPECTIVE EVALUATION OF SERUM URIC ACID LEVELS AND THEIR ASSOCIATION WITH BLOOD PRESSURE CONTROL IN PATIENTS WITH NEWLY DIAGNOSED ESSENTIAL HYPERTENSION

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ABSTRACT

Background: Serum uric acid has emerged as a potential biochemical marker associated with hypertension, vascular dysfunction, and cardiovascular risk. However, its role in predicting blood pressure control among newly diagnosed hypertensive patients remains uncertain. The aim is to evaluate serum uric acid levels and their association with blood pressure control among patients with newly diagnosed essential hypertension.

Materials and Methods: This prospective observational analytical study included 102 patients with newly diagnosed essential hypertension at a tertiary care hospital. Demographic characteristics, body mass index, serum uric acid, lipid profile, glycaemic parameters, renal function, and blood pressure were evaluated. Patients underwent follow-up assessment after initiation of antihypertensive treatment. Blood pressure control was defined as systolic blood pressure <140 mmHg and diastolic blood pressure <90 mmHg. Statistical analysis was performed using IBM SPSS Statistics version 28.0, with $p<0.05$ considered statistically significant.

Results: Among 102 patients, 76 (74.51%) achieved blood pressure control, while 26 (25.49%) remained uncontrolled. Mean serum uric acid levels were significantly higher in uncontrolled patients (6.84 ± 1.28 mg/dL) compared with controlled patients (6.00 ± 1.20 mg/dL; p=0.006). Hyperuricaemia was more prevalent in uncontrolled patients (57.69% vs. 32.89%; p=0.025). Uncontrolled hypertension increased from 11.54% in the lowest uric acid quartile to 46.15% in the highest quartile (p=0.030). Serum uric acid correlated significantly with BMI (r=0.575), triglycerides (r=0.599), and baseline systolic blood pressure (r=0.419; all $p<0.001$). However, serum uric acid was not an independent predictor after multivariable adjustment (AOR=1.46; p=0.165).

Conclusion: Elevated serum uric acid was associated with poorer blood pressure control and adverse metabolic characteristics. However, its independent predictive significance was not established, highlighting the need for larger prospective studies.

Keywords: Essential Hypertension; Serum Uric Acid; Hyperuricaemia; Blood Pressure Control; Cardiovascular Risk.

INTRODUCTION

Hypertension is one of the most prevalent noncommunicable diseases worldwide and represents a major public health concern because of its substantial contribution to cardiovascular morbidity, premature mortality, and healthcare expenditure. Essential hypertension, characterized by persistently elevated arterial blood pressure without an identifiable secondary cause, accounts for the majority of hypertension cases encountered in clinical practice. It is frequently asymptomatic during its initial stages, allowing progressive cardiovascular and renal damage to develop before clinical

recognition. Persistent elevation of blood pressure contributes to endothelial dysfunction, vascular remodelling, left ventricular hypertrophy, coronary artery disease, stroke, and chronic kidney disease. Despite advances in diagnostic approaches and therapeutic interventions, achieving sustained blood pressure control remains a considerable challenge. Identification of additional biochemical markers associated with hypertension severity and treatment response may therefore improve cardiovascular risk assessment and individualized management strategies.[1] The development and progression of essential hypertension involve complex interactions between genetic susceptibility, environmental exposures, neurohormonal mechanisms, and metabolic abnormalities. Important contributing factors include advancing age, obesity, excessive dietary sodium intake, physical inactivity, insulin resistance, and dyslipidaemia. Activation of the sympathetic nervous system, alterations in renal sodium handling, and dysregulation of the renin–angiotensin–aldosterone system play important roles in maintaining elevated arterial pressure. Effective hypertension management requires accurate blood pressure measurement, evaluation of associated cardiovascular risk factors, lifestyle modification, and appropriate pharmacological treatment. However, considerable variation exists in individual responses to antihypertensive therapy. Consequently, identifying additional metabolic determinants of inadequate blood pressure control has become an important area of clinical investigation.[2] Serum uric acid has emerged as a potentially important biochemical marker associated with essential hypertension and cardiovascular dysfunction. Uric acid is the final product of purine metabolism in humans and is generated through enzymatic reactions involving xanthine oxidoreductase. Circulating uric acid concentrations depend on the balance between endogenous production, dietary purine intake, renal excretion, and intestinal elimination. Hyperuricaemia may develop because of increased uric acid production, reduced renal clearance, or a combination of both mechanisms. Several biological pathways have been proposed to explain its possible relationship with hypertension, including oxidative stress, endothelial dysfunction, reduced nitric oxide availability, vascular inflammation, and activation of the renin–angiotensin system. These mechanisms may contribute to increased vascular resistance and impaired regulation of arterial blood pressure, although the independent causal role of uric acid remains uncertain.[3] The relationship between serum uric acid and hypertension is particularly relevant because hyperuricaemia frequently coexists with metabolic abnormalities that independently influence cardiovascular risk. Insulin resistance, abdominal obesity, hypertriglyceridaemia, and altered glucose metabolism may contribute to increased circulating uric acid concentrations through changes in renal tubular transport and metabolic regulation. These disturbances can also promote vascular inflammation, impaired endothelial function, and increased arterial stiffness. Consequently, serum uric acid may represent an integrated marker of metabolic and vascular dysfunction rather than an isolated biochemical abnormality. Prospective evaluation of uric acid alongside anthropometric measurements, glycaemic indices, lipid parameters, and blood pressure characteristics is therefore necessary to clarify its clinical significance in essential hypertension.[4] Renal function represents another important component of the relationship between uric acid metabolism and blood pressure regulation. The kidneys maintain uric acid homeostasis through glomerular filtration, tubular reabsorption, and secretion. Alterations in renal haemodynamics and tubular function may simultaneously influence serum uric acid concentrations and systemic blood pressure. Conversely, sustained hypertension may contribute to renal microvascular injury and progressive impairment of renal function. Serum creatinine and estimated glomerular filtration rate are therefore important parameters when evaluating the association between hyperuricaemia and hypertension. Considering renal and metabolic characteristics together may help distinguish the independent contribution of serum uric acid from other established cardiovascular risk factors.[5] Prospective assessment provides an opportunity to examine whether baseline serum uric acid concentrations are associated with subsequent changes in blood pressure following initiation of antihypertensive treatment. Unlike a single cross-sectional evaluation, longitudinal follow-up permits assessment of blood pressure reduction, achievement of predefined treatment targets, and potential variations in therapeutic response. Categorization of patients according to baseline uric acid concentrations may also assist in identifying patterns of increasing cardiovascular risk. However, interpretation requires appropriate adjustment for demographic characteristics, baseline blood pressure, obesity, renal function, and metabolic abnormalities, because these factors may influence both serum uric acid levels and hypertension outcomes.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, prospective observational analytical study to evaluate serum uric acid levels and their association with blood pressure control among patients with newly diagnosed essential hypertension. The study aimed to determine the relationship between baseline serum uric acid concentrations, demographic characteristics, metabolic parameters, renal function, hypertension severity, and subsequent blood pressure response to antihypertensive treatment. The study was conducted at a tertiary care hospital in the Departments of General Medicine. Patients attending the outpatient department with newly diagnosed essential hypertension were screened for eligibility.

Clinical assessment, biochemical investigations, and blood pressure monitoring were performed according to a standardized study protocol. A total of 102 patients with newly diagnosed essential hypertension were enrolled using a consecutive sampling technique. Eligible patients fulfilling the inclusion and exclusion criteria were recruited after obtaining written informed consent. Participants were evaluated prospectively to assess the association between baseline serum uric acid levels and subsequent blood pressure control.

Inclusion Criteria

Patients aged 18 years and above with newly diagnosed, previously untreated essential hypertension were included. Hypertension was defined as systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, confirmed through repeated measurements on separate clinical visits. Patients willing to undergo biochemical investigations and follow-up blood pressure assessments were considered eligible.

Exclusion Criteria

Patients with secondary hypertension, chronic kidney disease with an estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73 m², acute kidney injury, gout, severe hepatic dysfunction, malignancy, or acute systemic illness were excluded. Pregnant women and patients receiving uric acid-lowering medications or drugs known to substantially influence uric acid metabolism before enrolment were also excluded.

Clinical and Demographic Assessment: Detailed clinical histories were recorded, including age, gender, family history of hypertension, smoking habits, alcohol consumption, dietary habits, physical activity, and associated comorbidities. Anthropometric measurements included height, weight, body mass index (BMI), and waist circumference. Clinical examination assessed pulse rate, cardiovascular status, peripheral oedema, and evidence of hypertension-related target organ involvement.

Blood Pressure Measurement: Blood pressure was measured using a calibrated automated sphygmomanometer with an appropriately sized cuff. Participants remained seated for at least five minutes before measurement, with their back supported and arm positioned at heart level. Three readings were obtained at one-minute intervals, and the average of the final two readings was recorded. Baseline SBP, DBP, pulse pressure, and mean arterial pressure (MAP) were documented. The same measurement protocol was followed during subsequent visits.

Laboratory Investigations: Venous blood samples were collected under aseptic conditions following overnight fasting. Serum uric acid was estimated using the enzymatic uricase method. Additional investigations included fasting blood glucose, glycated haemoglobin (HbA1c), serum creatinine, blood urea, serum electrolytes, and lipid profile comprising total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol. Renal function was assessed using eGFR, while urinary albumin-to-creatinine ratio was evaluated where available. Hyperuricaemia was defined as serum uric acid >7.0 mg/dL in males and >6.0 mg/dL in females.

Treatment and Follow-Up Assessment: Antihypertensive treatment was initiated according to standard clinical guidelines and the treating physician's assessment. Medication classes, dose modifications, lifestyle counselling, and treatment adherence were documented. Follow-up evaluations included SBP, DBP, changes from baseline blood pressure, medication adherence, and achievement of blood pressure targets. Potential confounding effects of antihypertensive medications, particularly diuretics, were considered during analysis.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 28.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Independent Student's t-test or Mann–Whitney U test was applied for continuous variables, whereas the Chi-square test or Fisher's exact test was used for categorical comparisons. Paired t-test or Wilcoxon signed-rank test evaluated changes in blood pressure. Pearson's or Spearman's correlation assessed relationships between serum uric acid and clinical parameters. Multivariable logistic regression was used to identify independent associations with uncontrolled hypertension, adjusting for relevant confounders. Odds ratios with 95% confidence intervals were calculated. A p-value <0.05 was considered statistically significant.

RESULTS

[Table 1] Comparison of Baseline Demographic and Clinical Characteristics According to Blood Pressure Control (N=102)

A total of 102 patients with newly diagnosed essential hypertension were evaluated, of whom 76 (74.51%) achieved blood pressure control, while 26 (25.49%) remained uncontrolled. The mean age was significantly higher in the uncontrolled group (55.09 ± 10.80 years) compared with the controlled group (50.12 ± 9.57 years; p=0.044). Similarly, mean BMI was significantly higher among uncontrolled patients (27.27 ± 3.01 vs. 25.70 ± 3.11 kg/m²; p=0.028). Diabetes mellitus was more prevalent in the uncontrolled group (30.77%) than in the controlled group (11.84%), with a statistically significant difference (p=0.035). Male sex (61.54% vs. 56.58%), current smoking (30.77% vs. 19.74%), and baseline stage 2 hypertension (46.15% vs. 28.95%) were also more frequent in the uncontrolled group, although these differences were not statistically significant (p>0.05). Waist circumference and family history of hypertension were comparable between the groups.

[Table 2] Comparison of Serum Uric Acid Levels and Biochemical Parameters According to Blood Pressure Control (N=102)

The mean serum uric acid level was significantly higher among patients with uncontrolled hypertension (6.84 ± 1.28 mg/dL) compared with those achieving blood pressure control (6.00 ± 1.20 mg/dL; p=0.006). Hyperuricaemia was observed in 57.69% of patients with uncontrolled hypertension compared with 32.89% of controlled patients, demonstrating a statistically significant association (p=0.025). Mean triglyceride levels were also significantly elevated in the uncontrolled group (160.18 ± 30.89 mg/dL) compared with the controlled group (140.82 ± 35.78 mg/dL; p=0.011). Although fasting blood glucose (101.68 ± 13.62 vs. 98.94 ± 14.40 mg/dL) and HbA1c (5.82 ± 0.60% vs. 5.72 ± 0.46%) were higher in the uncontrolled group, these differences were not statistically significant. Similarly, LDL cholesterol and serum creatinine were slightly higher, whereas HDL cholesterol and eGFR were lower among uncontrolled patients. However, these biochemical and renal parameters did not demonstrate statistically significant between-group differences (p>0.05).

[Table 3] Association Between Baseline Serum Uric Acid Quartiles and Blood Pressure Control (N=102)

The distribution of patients according to baseline serum uric acid quartiles demonstrated a significant association between increasing uric acid concentrations and poorer blood pressure control (p=0.030). In the lowest uric acid quartile (Q1: 3.60–5.33 mg/dL), 23 of 26 patients (88.46%) achieved blood pressure control, while only 3 patients (11.54%) remained uncontrolled. In the second quartile (Q2: 5.35–6.28 mg/dL), blood pressure was controlled in 80.00% of patients, whereas 20.00% had uncontrolled hypertension. The proportion of uncontrolled hypertension increased further to 24.00% in the third quartile (Q3: 6.30–7.10 mg/dL). In the highest quartile (Q4: 7.11–10.04 mg/dL), only 53.85% achieved satisfactory blood pressure control, while 46.15% remained uncontrolled. These findings demonstrated a progressive reduction in blood pressure control rates with increasing baseline serum uric acid concentrations, with a statistically significant overall difference across the four quartiles.

[Table 4] Comparison of Baseline and Follow-Up Blood Pressure and Treatment Adherence (N=102)

The mean baseline systolic blood pressure (SBP) was 152.47 ± 10.64 mmHg in the overall study population and was significantly higher in the uncontrolled group (159.31 ± 11.67 mmHg) compared with the controlled group (150.13 ± 9.24 mmHg; p<0.001). At final follow-up, mean SBP decreased to 133.69 ± 8.32 mmHg overall, with significantly lower values among controlled patients (130.38 ± 6.35 mmHg) than uncontrolled patients (143.38 ± 5.28 mmHg; p<0.001). The mean SBP reduction was greater in the controlled group (19.75 ± 8.41 mmHg) compared with the uncontrolled group (15.93 ± 11.14 mmHg), although the difference was not statistically significant (p=0.118). Baseline diastolic blood pressure (DBP) was comparable between the groups (p=0.678), whereas final follow-up DBP was significantly lower among controlled patients (81.71 ± 4.83 vs. 84.92 ± 6.10 mmHg; p=0.020). The mean DBP reduction was 12.32 ± 5.27 mmHg in the controlled group and 9.57 ± 6.71 mmHg in the uncontrolled group (p=0.066). Treatment adherence was reported in 73.68% of controlled patients compared with 65.38% of uncontrolled patients, without a statistically significant difference (p=0.418).

[Table 5] Correlation of Serum Uric Acid with Clinical and Biochemical Parameters (N=102)

Pearson's correlation analysis demonstrated significant positive correlations between serum uric acid levels and several clinical and biochemical parameters. Serum uric acid showed a strong positive correlation with triglycerides (r=0.599; p<0.001) and a moderate positive correlation with BMI (r=0.575; p<0.001). Significant positive correlations were also observed with baseline SBP (r=0.419; p<0.001), serum creatinine (r=0.415; p<0.001), and HbA1c (r=0.295; p=0.003). Age (r=0.244; p=0.013) and baseline DBP (r=0.196; p=0.049) demonstrated weaker but statistically significant positive correlations with serum uric acid concentrations. Conversely, eGFR showed a significant negative correlation with serum uric acid (r=−0.212; p=0.033), indicating that higher uric acid concentrations were associated with lower estimated renal filtration rates. However, the correlation between serum uric acid and reduction in SBP was weak and statistically insignificant (r=0.073; p=0.465).

[Table 6] Multivariable Logistic Regression Analysis of Factors Associated with Uncontrolled Hypertension (N=102)

Multivariable logistic regression analysis was performed to identify factors independently associated with uncontrolled hypertension after adjusting for relevant demographic and clinical variables. Higher baseline systolic blood pressure emerged as a statistically significant independent predictor of uncontrolled hypertension, with each 1 mmHg increase associated with 7% higher adjusted odds of uncontrolled blood pressure (AOR=1.07; 95% CI: 1.01–1.13; p=0.012). Serum uric acid demonstrated a positive association with uncontrolled hypertension (AOR=1.46; 95% CI: 0.86–2.48), suggesting 46% higher adjusted odds for every 1 mg/dL increase, although this association did not reach statistical significance (p=0.165). Increasing age (AOR=1.03; p=0.253) and BMI (AOR=1.01; p=0.927) also showed nonsignificant positive associations with uncontrolled hypertension. Male sex was not independently associated with uncontrolled blood pressure (AOR=0.89; p=0.828). Treatment adherence demonstrated a potentially protective association (AOR=0.37; 95% CI: 0.12–1.12; p=0.079), although statistical significance was not achieved.

Table 1: Comparison of Baseline Demographic and Clinical Characteristics According to Blood Pressure Control (N=102)

Parameter	Controlled (n=76)	Uncontrolled (n=26)	p-value
Age (years)	50.12 ± 9.57	55.09 ± 10.80	0.044
BMI (kg/m²)	25.70 ± 3.11	27.27 ± 3.01	0.028
Waist circumference (cm)	92.65 ± 9.25	95.23 ± 9.69	0.243
Male sex	43 (56.58%)	16 (61.54%)	0.658
Family history of hypertension	40 (52.63%)	13 (50.00%)	0.817
Current smoker	15 (19.74%)	8 (30.77%)	0.245
Diabetes mellitus	9 (11.84%)	8 (30.77%)	0.035
BMI ≥25 kg/m²	46 (60.53%)	19 (73.08%)	0.251
Stage 2 hypertension at baseline	22 (28.95%)	12 (46.15%)	0.108

Table 2: Comparison of Serum Uric Acid Levels and Biochemical Parameters According to Blood Pressure Control (N=102)

Parameter	Controlled (n=76)	Uncontrolled (n=26)	p-value
Serum uric acid (mg/dL)	6.00 ± 1.20	6.84 ± 1.28	0.006
Hyperuricaemia	25 (32.89%)	15 (57.69%)	0.025
Fasting blood glucose (mg/dL)	98.94 ± 14.40	101.68 ± 13.62	0.388
HbA1c (%)	5.72 ± 0.46	5.82 ± 0.60	0.454
Triglycerides (mg/dL)	140.82 ± 35.78	160.18 ± 30.89	0.011
LDL cholesterol (mg/dL)	118.83 ± 24.39	125.27 ± 18.51	0.165
HDL cholesterol (mg/dL)	44.77 ± 7.17	43.29 ± 8.02	0.408
Serum creatinine (mg/dL)	0.93 ± 0.11	0.95 ± 0.10	0.283
eGFR (mL/min/1.73 m²)	94.69 ± 10.29	92.61 ± 10.29	0.378

Table 3: Association Between Baseline Serum Uric Acid Quartiles and Blood Pressure Control (N=102)

Serum uric acid quartile	Total (n)	Controlled n (%)	Uncontrolled n (%)	p-value
Q1 (3.60–5.33 mg/dL)	26	23 (88.46%)	3 (11.54%)	0.030
Q2 (5.35–6.28 mg/dL)	25	20 (80.00%)	5 (20.00%)	—
Q3 (6.30–7.10 mg/dL)	25	19 (76.00%)	6 (24.00%)	—
Q4 (7.11–10.04 mg/dL)	26	14 (53.85%)	12 (46.15%)	—

Table 4: Comparison of Baseline and Follow-Up Blood Pressure and Treatment Adherence (N=102)

Parameter	Overall (N=102)	Controlled (n=76)	Uncontrolled (n=26)	p-value
Baseline SBP (mmHg)	152.47 ± 10.64	150.13 ± 9.24	159.31 ± 11.67	<0.001
Final follow-up SBP (mmHg)	133.69 ± 8.32	130.38 ± 6.35	143.38 ± 5.28	<0.001
Reduction in SBP (mmHg)	18.77 ± 9.27	19.75 ± 8.41	15.93 ± 11.14	0.118
Baseline DBP (mmHg)	94.15 ± 5.51	94.03 ± 5.82	94.49 ± 4.55	0.678
Final follow-up DBP (mmHg)	82.53 ± 5.34	81.71 ± 4.83	84.92 ± 6.10	0.020
Reduction in DBP (mmHg)	11.62 ± 5.76	12.32 ± 5.27	9.57 ± 6.71	0.066
Reported treatment adherence	73 (71.57%)	56 (73.68%)	17 (65.38%)	0.418

Table 5: Correlation of Serum Uric Acid with Clinical and Biochemical Parameters (N=102)

Parameter	Pearson's r	p-value
Age (years)	0.244	0.013
BMI (kg/m²)	0.575	<0.001
Baseline SBP (mmHg)	0.419	<0.001
Baseline DBP (mmHg)	0.196	0.049
HbA1c (%)	0.295	0.003
Triglycerides (mg/dL)	0.599	<0.001
Serum creatinine (mg/dL)	0.415	<0.001
eGFR (mL/min/1.73 m²)	−0.212	0.033
Reduction in SBP (mmHg)	0.073	0.465

Table 6: Multivariable Logistic Regression Analysis of Factors Associated with Uncontrolled Hypertension (N=102)

Covariate	Adjusted odds ratio	95% confidence interval	p-value
Serum uric acid (per 1 mg/dL)	1.46	0.86–2.48	0.165
Age (per year)	1.03	0.98–1.09	0.253
Male sex	0.89	0.31–2.53	0.828
BMI (per 1 kg/m²)	1.01	0.82–1.24	0.927
Baseline SBP (per 1 mmHg)	1.07	1.01–1.13	0.012
Treatment adherence (yes vs no)	0.37	0.12–1.12	0.079

DISCUSSION

The present study evaluated 102 patients with newly diagnosed essential hypertension, of whom 76 (74.51%) achieved satisfactory blood pressure control, whereas 26 (25.49%) remained uncontrolled. The mean age was significantly higher among uncontrolled patients (55.09 ± 10.80 years) compared with controlled patients (50.12 ± 9.57 years; p=0.044). Similarly, BMI was significantly elevated

in the uncontrolled group (27.27 ± 3.01 vs. 25.70 ± 3.11 kg/m²; p=0.028), while diabetes mellitus was more prevalent among uncontrolled patients (30.77% vs. 11.84%; p=0.035). Overall, hyperuricaemia was present in 39.22% of the study population. These findings can be compared with Poudel et al. (2014), who evaluated 205 newly diagnosed essential hypertensive patients and an equal number of normotensive controls. They reported hyperuricaemia in 28.80% of hypertensive patients compared with 13.70% of normotensive individuals, with an odds ratio of 2.555 (95% CI: 1.549–4.213; p<0.001). Their mean serum uric acid concentrations were 290.05 ± 87.05 µmol/L and 245.24 ± 99.38 µmol/L, respectively. Although their study compared hypertensive and normotensive subjects rather than subsequent blood pressure control, both studies demonstrate a considerable burden of hyperuricaemia among newly diagnosed hypertensive patients.[7] In the present study, mean serum uric acid concentrations were significantly higher among patients with uncontrolled hypertension (6.84 ± 1.28 mg/dL) compared with those achieving blood pressure control (6.00 ± 1.20 mg/dL; p=0.006). Hyperuricaemia was also more frequent in the uncontrolled group (57.69%) than in the controlled group (32.89%; p=0.025), suggesting an association between elevated serum uric acid and inadequate blood pressure control. Similar observations were reported by Cicero et al. (2017) in the Brisighella Heart Study, which included 146 newly diagnosed hypertensive individuals, 394 treated but uncontrolled hypertensive patients, 470 controlled hypertensive patients, and 324 normotensive subjects. Serum uric acid concentrations were significantly higher among untreated and uncontrolled hypertensive patients compared with controlled hypertensive and normotensive participants. Their logistic regression analysis demonstrated that serum uric acid was independently associated with poorer blood pressure control (OR=1.277; 95% CI: 1.134–1.600 per mg/dL). They also reported significantly greater arterial stiffness among uncontrolled hypertensive patients (p<0.001). These findings support the unadjusted association observed in the present study, although the adjusted association in our analysis did not achieve statistical significance.[8] The biochemical findings of the present study demonstrated a significant relationship between metabolic abnormalities and inadequate blood pressure control. Mean triglyceride concentrations were significantly higher among uncontrolled patients (160.18 ± 30.89 mg/dL) compared with controlled patients (140.82 ± 35.78 mg/dL; p=0.011). Serum uric acid also demonstrated significant positive correlations with triglycerides (r=0.599; p<0.001), BMI (r=0.575; p<0.001), and HbA1c (r=0.295; p=0.003). These observations are consistent with Cibičková et al. (2017), who conducted a cross-sectional study involving 833 patients with dyslipidaemia to assess relationships between serum uric acid and metabolic syndrome parameters. They observed that serum uric acid was positively associated with triglyceride concentrations, low HDL cholesterol, and anthropometric indicators of metabolic risk. The relationship with typical dyslipidaemic features of metabolic syndrome was stronger than that with insulin resistance-related parameters, and patients exhibiting the hypertriglyceridaemic waist phenotype had higher serum uric acid concentrations. The comparable metabolic associations suggest that elevated serum uric acid may reflect a broader cardiometabolic risk profile in hypertensive individuals, rather than representing an isolated biochemical abnormality.[9] The present study demonstrated a progressive deterioration in blood pressure control with increasing baseline serum uric acid quartiles. In the lowest quartile (3.60–5.33 mg/dL), 88.46% of patients achieved blood pressure control, while only 11.54% remained uncontrolled. In contrast, the highest quartile (7.11–10.04 mg/dL) demonstrated a substantially greater proportion of uncontrolled hypertension (46.15%), with only 53.85% achieving satisfactory control. The overall association across the four quartiles was statistically significant (p=0.030). Comparable dose-dependent findings were reported by Zheng et al. (2017) in a longitudinal population-based study involving 5,105 initially non-hypertensive Chinese adults followed for nine years. During follow-up, 2,259 participants developed hypertension, corresponding to an overall cumulative incidence of 44.30%. The incidence increased progressively across serum uric acid quartiles, from 36.30% in the first quartile to 42.40%, 44.10%, and 54.50% in the second, third, and fourth quartiles, respectively (p for trend <0.001). Although their outcome was incident hypertension rather than treatment control, the consistent direction of the quartile relationship supports the potential value of baseline serum uric acid for cardiovascular risk stratification.[10] The present study demonstrated a reduction in blood pressure following antihypertensive management, with mean overall systolic blood pressure decreasing from 152.47 ± 10.64 mmHg at baseline to 133.69 ± 8.32 mmHg at final follow-up. The mean reduction in systolic blood pressure was greater in controlled patients (19.75 ± 8.41 mmHg) than uncontrolled patients (15.93 ± 11.14 mmHg), although the between-group difference was not statistically significant (p=0.118). Similarly, the mean diastolic blood pressure reduction was 12.32 ± 5.27 mmHg and 9.57 ± 6.71 mmHg, respectively (p=0.066). The possible contribution of uric acid to blood pressure regulation was investigated by Feig et al. (2008) in a randomized, double-blind, placebo-controlled crossover trial involving 30 adolescents with newly diagnosed essential hypertension and serum uric acid levels ≥6 mg/dL. Allopurinol treatment produced a mean systolic blood pressure reduction of 6.9 mmHg compared with 2.0 mmHg during placebo treatment (p=0.009). Diastolic blood pressure decreased by 5.1

mmHg and 2.4 mmHg, respectively (p=0.05). Furthermore, 20 of 30 participants achieved normal blood pressure while receiving allopurinol compared with only one during placebo treatment. These findings suggest a potential role for uric acid in blood pressure regulation in selected younger patients, although the population and intervention differed substantially from those of the present study.[11] Despite the association between elevated serum uric acid and poorer blood pressure control in the present study, the relationship between uric acid reduction and improvement in blood pressure requires careful interpretation. Baseline serum uric acid was significantly associated with uncontrolled hypertension in unadjusted comparisons (p=0.006), whereas its correlation with the magnitude of systolic blood pressure reduction was weak and statistically insignificant (r=0.073; p=0.465). Moreover, treatment adherence was reported in 73.68% of controlled patients compared with 65.38% of uncontrolled patients, without a statistically significant difference (p=0.418). These findings can be considered alongside McMullan et al. (2017), who conducted a randomized controlled trial involving 149 overweight or obese adults assigned to allopurinol, probenecid, or placebo. After eight weeks, mean serum uric acid concentrations were 2.9, 3.5, and 5.6 mg/dL, respectively. Despite substantial uric acid reduction, changes in 24-hour systolic blood pressure were −0.4 ± 6.1 mmHg with allopurinol, −1.6 ± 10.1 mmHg with probenecid, and +0.5 ± 6.0 mmHg with placebo, with no significant treatment effect (p=0.58).[12] Correlation analysis in the present study identified significant positive relationships between serum uric acid and baseline systolic blood pressure (r=0.419; p<0.001), diastolic blood pressure (r=0.196; p=0.049), and serum creatinine (r=0.415; p<0.001). Conversely, eGFR demonstrated a significant negative correlation with serum uric acid concentrations (r=−0.212; p=0.033). These observations indicate that higher uric acid concentrations were associated with greater baseline blood pressure and less favourable renal biochemical indices. Similar findings regarding blood pressure characteristics were reported by Çağlı et al. (2015), who evaluated 300 untreated patients with newly diagnosed essential hypertension, with a mean age of 57.3 ± 13.6 years. They reported significant positive correlations between log-transformed serum uric acid and 24-hour systolic blood pressure variability (r=0.246), nighttime systolic blood pressure variability (r=0.280), and nighttime diastolic blood pressure variability (r=0.353), with p<0.001 for all associations. Multivariable analysis also demonstrated independent associations between uric acid and these blood pressure variability indices.[13] Multivariable logistic regression analysis in the present study demonstrated that baseline systolic blood pressure was the only statistically significant independent predictor of uncontrolled hypertension (AOR=1.07; 95% CI: 1.01–1.13; p=0.012). Although elevated serum uric acid was associated with increased adjusted odds of uncontrolled hypertension (AOR=1.46; 95% CI: 0.86–2.48), this relationship did not achieve statistical significance (p=0.165). Age, BMI, sex, and treatment adherence were also not independently significant predictors. These findings differ partly from those reported by Sung et al. (2017), who evaluated 96,606 Korean individuals in a large longitudinal cohort, documenting approximately 10,405 incident hypertension cases. After multivariable adjustment, the highest serum uric acid quartile was associated with significantly increased hypertension risk compared with the lowest quartile, with adjusted hazard ratios of 1.29 (95% CI: 1.19–1.38) in males and 1.24 (95% CI: 1.09–1.42) in females. Their study also identified increasing serum uric acid concentrations over time as a predictor of hypertension development. The difference from the present analysis may reflect variations in sample size, study population, outcome definitions, and statistical power.[14]

CONCLUSION

The present study demonstrated a significant association between elevated serum uric acid levels and inadequate blood pressure control among patients with newly diagnosed essential hypertension. Higher serum uric acid concentrations were associated with increased BMI, triglyceride levels, baseline systolic blood pressure, and adverse metabolic parameters. However, serum uric acid was not an independent predictor of uncontrolled hypertension after multivariable adjustment, whereas baseline systolic blood pressure remained statistically significant. Serum uric acid may serve as an additional marker for cardiovascular risk assessment, although larger prospective studies are required to establish its independent prognostic significance.

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