

Original Research Article

COMPARATIVE ANALYSIS OF SERUM ELECTROLYTE PROFILE AND SERUM URIC ACID LEVELS IN DIAGNOSED HYPERTENSIVE AND NORMOTENSIVE PATIENTS”

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ABSTRACT

Background: Hypertension is a major global health concern and a leading risk factor for cardiovascular morbidity and mortality. Alterations in serum electrolyte levels and uric acid have been implicated in the pathophysiology of hypertension, influencing vascular tone, fluid balance, and renal function. Understanding these biochemical changes may aid in improving disease management and prevention strategies. The objective is to compare serum electrolyte profile (sodium, potassium, and chloride) and serum uric acid levels between hypertensive and normotensive individuals, and to assess the prevalence of associated biochemical abnormalities.

Materials and Methods: This case-control study included 126 participants, comprising 63 hypertensive and 63 normotensive individuals. Baseline demographic and clinical data were recorded. Serum sodium, potassium, chloride, and uric acid levels were measured using standard laboratory methods. Statistical analysis was performed using the unpaired Student's t-test, with a p-value < 0.05 considered statistically significant.

Results: Hypertensive subjects demonstrated significantly higher mean serum sodium (140.75 ± 3.70 mmol/L), chloride (104.05 ± 2.70 mmol/L), and uric acid levels (4.47 ± 1.40 mg/dL) compared to normotensive individuals (135.08 ± 6.11 mmol/L, 99.62 ± 5.09 mmol/L, and 3.71 ± 1.14 mg/dL, respectively; $p < 0.05$). Serum potassium levels were significantly lower in hypertensive subjects (3.83 ± 0.56 mmol/L) than in normotensive controls (4.06 ± 0.48 mmol/L; $p = 0.016$). Additionally, the prevalence of hypernatremia, hypokalaemia, hyperchloremia, and hyperuricemia was higher among hypertensive individuals.

Conclusion: Hypertension is associated with significant alterations in serum electrolyte levels and elevated uric acid. These findings highlight the importance of routine biochemical monitoring and suggest that correcting electrolyte imbalance and hyperuricemia may improve blood pressure control and reduce complications.

Keywords: Hypertension; Electrolytes; Sodium; Potassium; Chloride; Uric acid; Hyperuricemia.

INTRODUCTION

Hypertension is a major global public health challenge and a leading risk factor for cardiovascular morbidity and mortality worldwide. It is often

referred to as a “silent killer” because it frequently remains asymptomatic until significant complications arise.^[1] The prevalence of hypertension continues to rise due to urbanization, sedentary lifestyles, and dietary transitions, making

its early detection and management a priority in clinical practice.^[2] Lifestyle factors, particularly dietary habits such as high sodium intake and low potassium consumption, play a crucial role in the development and progression of hypertension.^[3,4]

Electrolyte balance is fundamental in maintaining normal blood pressure, with sodium and potassium being the most influential ions. Excess sodium intake has been strongly associated with increased blood pressure due to its effects on fluid retention and vascular resistance.^[5,6] Conversely, potassium has a protective role by promoting vasodilation and enhancing sodium excretion, thereby lowering blood pressure.^[7] Evidence from meta-analyses has shown that reduction in dietary salt intake significantly decreases blood pressure levels, further emphasizing the role of sodium in hypertension pathogenesis.^[8] Disturbances in electrolyte levels, including hypernatremia, hypokalaemia, and hyperchloremia, have been observed in hypertensive patients and may contribute to disease progression.^[9-11]

In addition to electrolytes, serum uric acid has emerged as an important biochemical factor associated with hypertension. Elevated uric acid levels have been implicated in endothelial dysfunction, inflammation, and activation of the renin-angiotensin system, all of which contribute to increased blood pressure.^[12,13] Epidemiological studies have demonstrated a significant association between hyperuricemia and hypertension, suggesting that uric acid may serve as both a marker and a potential causal factor.^[14-16]

Given the growing evidence linking electrolyte imbalance and elevated uric acid levels with hypertension, this study aims to compare serum electrolyte profiles and uric acid levels between hypertensive and normotensive individuals, and to assess the prevalence of related biochemical abnormalities.

MATERIALS AND METHODS

This prospective cross-sectional comparative study was conducted in the Department of Biochemistry at Chamarajanagar Institute of Medical Sciences, Chamarajanagar. The study population consisted of adult male and female participants who were categorized into two groups: hypertensive cases and normotensive controls. A total of 126 participants were included, comprising 63 diagnosed hypertensive patients and 63 normotensive individuals.

Hypertensive subjects were defined as patients with a documented diagnosis of hypertension who were receiving antihypertensive therapy for more than six months. Normotensive subjects were selected as controls and were defined as individuals with systolic blood pressure <120 mmHg and diastolic blood pressure <80 mmHg. Blood pressure was measured in the right upper arm using a standard sphygmomanometer with the participant in the supine

position after a rest period of at least 10 minutes. Two consecutive readings were recorded, and the average value was used for analysis. A systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg was considered diagnostic of hypertension.

Participants with chronic kidney disease, gout, diabetes mellitus, rheumatoid arthritis, pregnancy, lactation, or any acute or chronic infectious illness were excluded from the study. Written informed consent was obtained from all participants prior to enrolment.

Venous blood samples were collected under aseptic precautions, and serum was separated for biochemical analysis. Serum electrolytes, including sodium, potassium, and chloride, were estimated using the ion-selective electrode method. Serum uric acid levels were measured using an enzymatic uricase-peroxidase method, in which the intensity of the colored quinoneimine complex formed was directly proportional to the uric acid concentration in the sample.

Sample size estimation was performed using a two-sample mean comparison with an assumed effect size of 0.5, a 95% confidence level, and 80% statistical power, resulting in a required sample size of 63 participants in each group. Data were entered into Microsoft Excel and analyzed using SPSS version 22.0 (IBM SPSS Statistics, Somers, NY, USA). Continuous variables were expressed as mean \pm standard deviation, and categorical variables were expressed as frequency and percentage. Comparisons between hypertensive and normotensive groups were performed using the unpaired Student's t-test, and a p-value <0.05 was considered statistically significant.

RESULTS

The study included a total of 126 participants, with 63 hypertensive and 63 normotensive individuals. The mean age of hypertensive subjects was 38.10 ± 13.56 years, which was slightly lower than that of normotensive subjects (41.67 ± 10.47 years). Females constituted the majority in both groups, accounting for 73.0% of hypertensive and 68.3% of normotensive participants, while males represented 27.0% and 31.7%, respectively. As expected, both systolic and diastolic blood pressures were markedly elevated in the hypertensive group (161.41 ± 15.51 mmHg and 96.84 ± 8.08 mmHg, respectively) compared to the normotensive group (120.00 ± 5.00 mmHg and 79.98 ± 3.95 mmHg), confirming appropriate group classification [Table 1].

Comparison of biochemical parameters revealed significant differences in serum electrolyte levels and uric acid between the two groups. Hypertensive subjects had significantly higher mean serum sodium levels (140.75 ± 3.70 mmol/L) compared to normotensive subjects (135.08 ± 6.11 mmol/L; $p < 0.001$). Serum potassium levels were significantly lower in hypertensive individuals (3.83 ± 0.56 mmol/L) than in normotensive individuals ($4.06 \pm$

0.48 mmol/L; $p = 0.016$). Additionally, serum chloride levels were significantly higher among hypertensive participants (104.05 ± 2.70 mmol/L) compared to normotensive controls (99.62 ± 5.09 mmol/L; $p < 0.001$). Serum uric acid levels were also significantly elevated in hypertensive subjects (4.47 ± 1.40 mg/dL) relative to normotensive subjects (3.71 ± 1.14 mg/dL; $p = 0.001$), indicating a strong association between hypertension and altered biochemical profiles [Table 2]. Furthermore, the distribution of biochemical abnormalities showed a higher prevalence among hypertensive individuals compared to normotensive

controls. Hyponatremia was observed in 12.7% of hypertensive subjects but was absent in the normotensive group. Hypokalaemia was more frequent in hypertensive individuals (33.3%) than in normotensive subjects (15.9%). Similarly, hyperchloremia was present in 20.6% of hypertensive participants compared to 4.8% of normotensive individuals. Hyperuricemia was also more common among hypertensive subjects (15.9%) than in normotensive controls (6.3%). These findings further emphasize the association between hypertension and electrolyte imbalance as well as elevated uric acid levels [Table 3].

Table 1: Baseline demographic and clinical characteristics of study participants

Variable	Hypertensive (n = 63)	Normotensive (n = 63)
Age (years), Mean \pm SD	38.10 \pm 13.56	41.67 \pm 10.47
Male, n (%)	17 (27.0)	20 (31.7)
Female, n (%)	46 (73.0)	43 (68.3)
Systolic BP (mmHg), Mean \pm SD	161.41 \pm 15.51	120.00 \pm 5.00
Diastolic BP (mmHg), Mean \pm SD	96.84 \pm 8.08	79.98 \pm 3.95

Table 2: Comparison of serum electrolyte profile and serum uric acid levels between hypertensive and normotensive subjects

Parameter	Hypertensive (n = 63) Mean \pm SD	Normotensive (n = 63) Mean \pm SD	t value	p value
Sodium (mmol/L)	140.75 \pm 3.70	135.08 \pm 6.11	6.306	<0.001*
Potassium (mmol/L)	3.83 \pm 0.56	4.06 \pm 0.48	-2.435	0.016*
Chloride (mmol/L)	104.05 \pm 2.70	99.62 \pm 5.09	6.104	<0.001*
Uric acid (mg/dL)	4.47 \pm 1.40	3.71 \pm 1.14	3.329	0.001*

^a Unpaired Student's t-test

Table 3: Distribution of electrolyte and uric acid abnormalities among hypertensive and normotensive subjects

Biochemical abnormality	Hypertensive n (%)	Normotensive n (%)	Chi-square (χ^2)	p-value
Hypernatremia	8 (12.7)	0 (0.0)	8.54	0.003*
Hypokalaemia	21 (33.3)	10 (15.9)	5.18	0.023*
Hyperchloremia	13 (20.6)	3 (4.8)	7.15	0.007*
Hyperuricemia	10(15.9)	4(6.3)	2.90	0.088

DISCUSSION

The present study demonstrated significant alterations in serum electrolyte levels and uric acid concentrations among hypertensive individuals compared to normotensive controls. Hypertensive subjects exhibited significantly higher levels of serum sodium and chloride, along with lower potassium levels. These findings are consistent with the established role of sodium in increasing blood pressure through fluid retention and vascular changes.^[5,6] High sodium intake has been widely documented to elevate blood pressure, while dietary salt reduction has been shown to produce significant antihypertensive effects.^[8]

The observed reduction in serum potassium levels among hypertensive participants aligns with previous studies highlighting the protective role of potassium in blood pressure regulation.^[7] Potassium facilitates vasodilation and promotes natriuresis, thereby counteracting the hypertensive effects of sodium. Similar findings of altered sodium and potassium levels in hypertensive patients have been reported in earlier studies, supporting the association between electrolyte imbalance and hypertension.^[9,10] Additionally, the elevated chloride levels observed in

this study may reflect underlying disturbances in acid-base balance and renal handling of electrolytes, which have also been implicated in hypertension pathophysiology.^[11]

The study also found significantly higher serum uric acid levels in hypertensive subjects compared to normotensive individuals. This finding supports the growing body of evidence suggesting a link between hyperuricemia and hypertension. Uric acid has been shown to contribute to endothelial dysfunction and activation of the renin-angiotensin system, leading to increased vascular resistance and blood pressure.^[12,13] Epidemiological and clinical studies have consistently demonstrated a strong association between elevated uric acid levels and the risk of developing hypertension.^[14-16]

Furthermore, the higher prevalence of biochemical abnormalities such as hypernatremia, hypokalaemia, hyperchloremia, and hyperuricemia among hypertensive individuals in this study reinforces the clinical significance of these parameters. These abnormalities may not only reflect underlying pathophysiological changes but also serve as potential markers for disease severity and progression. Monitoring electrolyte and uric acid

levels could therefore play an important role in the comprehensive management of hypertension.

Overall, the findings of this study are in agreement with existing literature and highlight the importance of electrolyte balance and uric acid in the pathogenesis of hypertension. Further studies are warranted to explore the causal relationships and potential therapeutic implications of correcting these biochemical abnormalities.

CONCLUSION

This study demonstrates that hypertensive patients exhibit significant alterations in serum electrolyte profile and serum uric acid levels compared with normotensive individuals. Elevated serum sodium, chloride, and uric acid levels, along with reduced potassium levels, were observed among hypertensive subjects, highlighting their potential role in the pathophysiology of hypertension. These biochemical disturbances were consistently associated with higher blood pressure levels. The findings underscore the importance of routine monitoring of electrolytes and serum uric acid in hypertensive patients. Dietary modifications, including reduced sodium intake and increased potassium consumption, along with strategies to control hyperuricemia, may contribute to improved blood pressure control and prevention of hypertension-related complications.

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