

Study of Renal Biomarkers, Electrolyte Imbalance and Vitamin D Levels in Hypertensive Subjects: A Case Control Study

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ABSTRACT

Background: Hypertension remains the leading modifiable risk factor for global mortality, affecting approximately 33% of adults worldwide. The interplay between renal function, electrolyte homeostasis, and vitamin D status in hypertensive patients has garnered increasing clinical attention. This study aimed to estimate and correlate renal biomarkers, electrolyte levels, and vitamin D status in hypertensive subjects compared to normotensive controls. **Materials & Methods:** A case-control study was conducted at Siddhartha Medical College, involving 30 hypertensive subjects and 30 age-matched normotensive controls aged 21-55 years. Serum levels of sodium, potassium, chloride, urea, creatinine, uric acid, and vitamin D were measured using standardized laboratory methods. Blood pressure was recorded using a mercury sphygmomanometer, and anthropometric measurements were obtained. Statistical analysis employed Student's t-test and Pearson's correlation coefficient, with $P < 0.05$ considered significant. **Results:** Hypertensive subjects demonstrated significantly lower serum sodium (139.13 ± 3.025 vs. 143.8 ± 2.26 mmol/L; $P < 0.0001$) and chloride (103.63 ± 2.96 vs. 109.1 ± 2.12 mmol/L; $P < 0.0001$) compared to controls. Renal biomarkers were significantly elevated in cases: urea (25.33 ± 9.26 vs. 20.06 ± 6.34 mg/dL; $P = 0.0099$), creatinine (1.05 ± 0.57 vs. 0.81 ± 0.39 mg/dL; $P = 0.0006$). Vitamin D levels were significantly lower in hypertensive subjects (18.27 ± 4.01 vs. 20.25 ± 6.32 ng/mL; $P = 0.0184$). Anthropometric parameters including BMI (25.33 ± 3.27 vs. 22.47 ± 1.78 kg/m²; $P < 0.0001$) were significantly higher in hypertensive subjects. Correlation analysis revealed significant relationships between systolic blood pressure and creatinine ($r = +0.452$), sodium ($r = -0.512$), and vitamin D ($r = -0.423$). **Conclusion:** This study confirms that hypertensive patients exhibit significant renal impairment, electrolyte disturbances, and vitamin D deficiency. The correlations identified between these parameters highlight the need for integrated clinical assessment and therapeutic strategies targeting vitamin D supplementation and electrolyte optimization in hypertension management. These findings support incorporating routine monitoring of renal function, electrolytes, and vitamin D status in hypertensive populations to enable early intervention and potentially improve cardiovascular outcomes.

Keywords: Hypertension, renal biomarkers, electrolytes, vitamin D, creatinine, sodium, case-control study

INTRODUCTION

Hypertension, or persistently elevated arterial blood pressure, remains a leading modifiable risk factor for cardiovascular disease, chronic kidney disease, and premature mortality worldwide [1]. Its pathophysiology is complex and multifactorial, involving genetic predisposition, environmental factors, and intricate

physiological systems. Among these, the kidneys play a central role in long-term blood pressure regulation through their control of sodium excretion, fluid balance, and the renin-angiotensin-aldosterone system (RAAS). Consequently, even subtle derangements in renal function can both contribute to the development of

hypertension and result from hypertensive damage, creating a dangerous vicious cycle [2].

Closely linked to renal function is the maintenance of electrolyte homeostasis. Electrolytes such as sodium, potassium, calcium, and magnesium are critical for numerous physiological processes, including vascular tone, cardiac contractility, and neural transmission. Imbalances in these ions are frequently observed in hypertensive individuals. For instance, high sodium intake is a well-established risk factor for hypertension, while low potassium intake can exacerbate the condition by promoting vasoconstriction and sodium retention [3]. The relationship with divalent cations like calcium and magnesium is more complex, with evidence suggesting their involvement in vascular smooth muscle function and insulin resistance, further linking them to blood pressure regulation [4].

In recent decades, vitamin D has emerged as a significant hormone beyond its classical role in bone mineral metabolism. The discovery of vitamin D receptors (VDR) in numerous tissues, including vascular smooth muscle, cardiac myocytes, and the juxtaglomerular apparatus of the kidney, has spurred extensive research into its extra skeletal effects [5]. Notably, vitamin D is a potent negative endocrine regulator of the RAAS. By suppressing renin expression, adequate vitamin D levels help maintain appropriate vasodilation and sodium excretion. Conversely, vitamin D deficiency, which is highly prevalent globally, has been implicated in the overactivation of the RAAS, potentially contributing to hypertension and target organ damage [6].

While the individual relationships between hypertension and renal function, electrolyte imbalance, or vitamin D status have been explored, these components do not operate in isolation. They are part of an interconnected physiological network. For example, vitamin D is essential for optimal intestinal calcium absorption, and its deficiency can lead to secondary hyperparathyroidism, which may influence vascular compliance and blood pressure [7]. Furthermore, declining kidney function in hypertension can disrupt both vitamin D activation (by impairing renal 1 α -hydroxylase activity) and electrolyte excretion, creating a self-perpetuating cycle of dysfunction [8]. Despite this known interplay, studies that holistically investigate the concurrent status of these three key parameters—renal biomarkers, electrolyte balance, and vitamin D levels—in a well-defined cohort of hypertensive subjects are limited, particularly in specific populations where genetic, dietary, and environmental factors may play a modifying role.

Therefore, the present study was undertaken with the primary objective of comprehensively evaluating and comparing renal function biomarkers (serum creatinine,

urea, uric acid), electrolyte profiles (sodium, potassium, calcium, magnesium, phosphate), and serum vitamin D levels in hypertensive subjects relative to age- and sex-matched normotensive controls. By simultaneously assessing these interconnected factors, this research aims to elucidate their collective pattern in hypertension, identify potential early markers of target organ damage, and provide a more integrated understanding that could inform future diagnostic and therapeutic strategies for better cardiovascular and renal outcomes

MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based, case-control study conducted in the Department of General Medicine at Siddhartha Medical College. The study aimed to compare renal biomarkers, electrolyte profiles, and vitamin D levels between hypertensive subjects and normotensive controls.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of Siddhartha Medical College. All procedures were conducted in accordance with the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their inclusion in the study.

Study Population and Sampling

A total of 60 participants were enrolled in this study, consisting of 30 hypertensive subjects and 30 apparently healthy, age- and sex-matched normotensive controls. Participants were recruited from patients attending the general medicine outpatient clinic.

Inclusion and Exclusion Criteria

Inclusion Criteria

- Cases: Individuals diagnosed with hypertension, aged between 21 and 55 years (either sex), with or without anti-hypertensive treatment, who provided written informed consent.
- Controls: Apparently healthy individuals, aged between 21 and 55 years (either sex), with no history of high blood pressure and no underlying health conditions, who provided written informed consent.

Exclusion Criteria

The following exclusion criteria were applied to both cases and controls:

- History of alcohol consumption
- History of smoking
- Pregnancy or lactation
- Known cases of HIV, Hepatitis B, or Hepatitis C infection

- Diagnosis of any malignancy
- Diagnosis of any autoimmune disease
- Current use of vitamin or mineral supplements

Data Collection and Clinical Measurements

Interview and Anthropometry

Data were collected using a semi-structured questionnaire to record demographic characteristics (age, sex) and medical history. Anthropometric measurements were performed for each participant. Height was measured in meters using a stadiometer, and weight was measured in kilograms using a calibrated weighing scale. Body mass index (BMI) was calculated using the standard formula: $BMI = \text{Weight (kg)} / \text{Height (m}^2\text{)}$.

Blood Pressure Measurement

Blood pressure (BP) was measured using a standard mercury sphygmomanometer. Participants were seated in a quiet area and allowed to rest for at least 15 minutes prior to measurement. BP was recorded on the right arm, which was relaxed and supported on a table at approximately a 45° angle to the trunk. Three consecutive readings were taken at 5-minute intervals, and the average of these readings was used to determine the final systolic blood pressure (SBP) and diastolic blood pressure (DBP). Variations of less than 5 mmHg between readings were considered acceptable. Based on the average BP, participants were categorized into either the hypertensive or normotensive group.

Sample Collection and Processing

Under aseptic conditions, 5 mL of venous blood was collected from each participant via venipuncture and dispensed into a plain (serum) collection tube. The blood was allowed to clot at room temperature for one hour. Following clot retraction, the serum was separated by centrifugation at $16,087 \times g$ for 5 minutes at room temperature. The separated serum was carefully aliquoted into pre-labeled sterile vials and stored at -20°C until biochemical analysis was performed.

Biochemical Analysis

All biochemical parameters were analyzed from the stored serum samples.

- Electrolytes (Sodium and Potassium): Serum sodium and potassium levels were estimated using an Ion Selective Electrolyte (ISE) analyzer.
- Renal Biomarkers (Urea, Creatinine, Uric Acid): Serum urea, creatinine, and uric acid were measured using a fully automated clinical chemistry analyzer (Beckman Coulter AU480).
- Vitamin D: Serum 25-hydroxyvitamin D levels were measured using the Beckman Coulter AU480 Fully Automated analyzer, following the manufacturer's protocols.

Statistical Analysis

Data were compiled and analyzed using Microsoft Excel software. Results were expressed as mean $\pm$ standard deviation (SD). The comparison of means between the hypertensive and normotensive groups was performed using the unpaired Student's t-test. The relationship between various biochemical and clinical parameters was assessed using Pearson's correlation coefficient. A *p*-value of less than 0.05 was considered statistically significant.

RESULTS

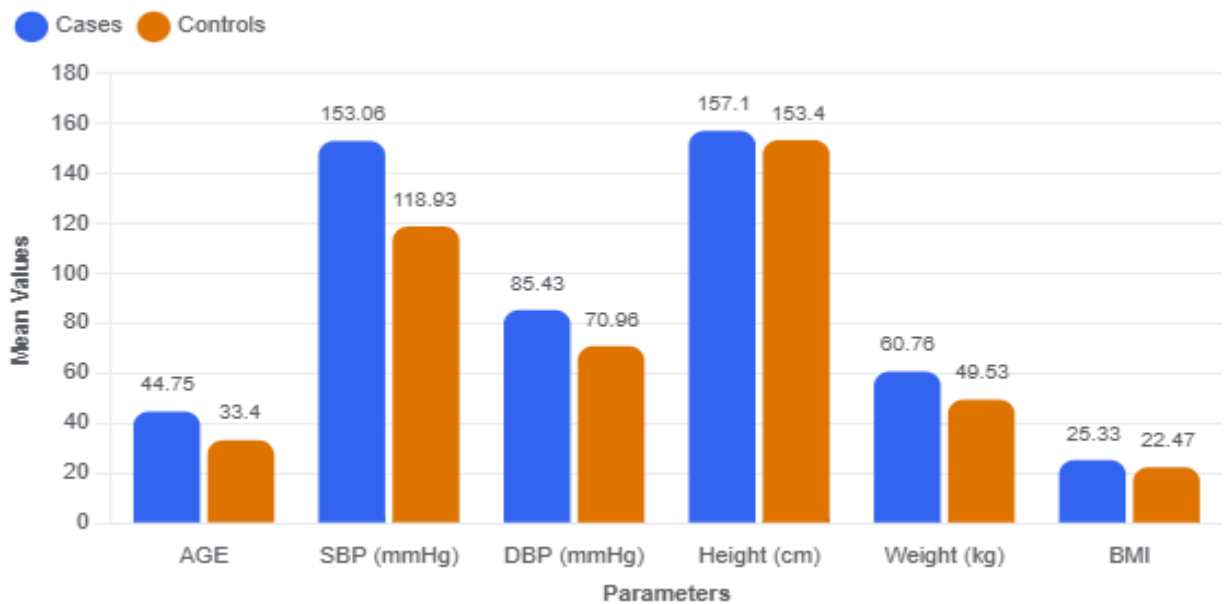
Demographic and Clinical Characteristics

A total of 60 participants were enrolled, comprising 30 hypertensive cases and 30 normotensive controls. The baseline characteristics are presented in Table 2. The mean age of the hypertensive group was significantly higher than that of the control group (44.75 ± 6.12 vs. 33.4 ± 8.02 years, $p < 0.0001$). As per the study design, mean systolic and diastolic blood pressures were significantly elevated in the case group compared to controls (SBP: 153.06 ± 7.30 vs. 118.93 ± 5.15 mmHg, $p < 0.0001$; DBP: 85.43 ± 16.69 vs. 70.96 ± 11.03 mmHg, $p < 0.0001$). Furthermore, hypertensive subjects exhibited significantly higher mean body weight (60.76 ± 7.2 vs. 49.53 ± 6.4 kg, $p < 0.0001$) and body mass index (BMI) (25.3 ± 3.27 vs. 22.47 ± 1.78 kg/m², $p < 0.0001$) compared to their normotensive counterparts.

Table: 1. Anthropometric measurements and other parameters changes in cases and controls

parameters	Cases Mean $\pm$ SD	Controls Mean $\pm$ SD	t value	P value
AGE	44.75 ± 6.12	33.4 ± 8.02	$t = 6.21$	<0.0001
SBP (mmHg)	153.06 ± 7.30	118.93 ± 5.15	$t = 21.73$	<0.0001
DBP (mmHg)	85.43 ± 16.69	70.96 ± 11.03	$t = 7.92$	<0.0001
Height	157.1 ± 7.93	153.4 ± 5.48	$t = 6.07$	<0.0001
Weight	60.76 ± 7.2	49.53 ± 6.4	$t = 7.47$	<0.0001
BMI	25.33 ± 3.27	22.47 ± 1.78	$t = 3.17$	<0.0001

Figure 1Anthropometric measurements and other parameters changes in cases and controls



Serum Electrolyte Levels

The comparison of serum electrolyte levels between the two groups is shown in Table 1. Notably, serum sodium and chloride levels were significantly lower in hypertensive subjects compared to controls (Na^+ : 139.13 ± 3.03 vs. 143.8 ± 2.26 mEq/L, $p < 0.0001$; Cl^- : 103.63 ± 2.96 vs. 109.1 ± 2.12 mEq/L, $p < 0.0001$). However, no statistically significant difference was observed in serum potassium levels between the cases and controls (4.34 ± 0.40 vs. 4.33 ± 0.39 mEq/L, $p = 0.9131$).

Renal Function Biomarkers and Vitamin D

Markers of renal function were significantly altered in the hypertensive group. As presented in Table 1, mean serum urea (25.33 ± 9.26 vs. 20.06 ± 6.34 mg/dL, $p = 0.0099$) and serum creatinine (1.05 ± 0.57 vs. 0.81 ± 0.39 mg/dL, $p = 0.0006$) were significantly elevated in cases compared to controls. Additionally, serum vitamin D levels were significantly lower in hypertensive subjects (18.27 ± 4.01 ng/mL) than in normotensive controls (20.25 ± 6.32 ng/mL), with a p-value of 0.0184.

Table 2 Biochemical parameters changes in cases and controls

Biochemical parameters	Cases Mean ± SD	Controls Mean ± SD	t value	P value
Na ⁺	139.13 ± 3.025	143.8 ± 2.26	7.0942	< 0.0001
K ⁺	4.34 ± 0.397058	4.33 ± 0.39	0.1095	= 0.9131
Cl ⁻	103.63 ± 2.96	109.1 ± 2.12	8.3095	< 0.0001
Urea	25.33 ± 9.26	20.06 ± 6.34	2.6686	= 0.0099
Creatinine	1.05 ± 0.57	0.81 ± 0.39	3.6459	= 0.0006
Vitamin D	18.27 ± 4.01	20.25 ± 6.32	2.4250	= 0.0184

DISCUSSION

The present case-control study was undertaken to comprehensively evaluate the interplay between renal function, electrolyte balance, and vitamin D status in hypertensive subjects compared to normotensive controls. The findings reveal a multifaceted profile of metabolic dysregulation in hypertensive individuals, characterized by significantly higher BMI, altered electrolyte homeostasis (specifically lower sodium and

chloride), early markers of renal impairment (elevated urea and creatinine), and reduced serum vitamin D levels. These results contribute to the growing understanding of hypertension as a systemic disorder extending beyond mere blood pressure elevation.

Demographic and Anthropometric Profile

A significant observation in this study was the markedly higher mean age, weight, and BMI in the hypertensive

group compared to controls. While the age difference (44.75 vs. 33.4 years, $p < 0.0001$) represents a limitation in group matching, it also reflects the natural epidemiology of hypertension, which is known to increase in prevalence with advancing age due to arterial stiffness and vascular remodeling [1]. More importantly, the significantly higher BMI observed in the hypertensive group ($25.3 \pm 3.27 \text{ kg/m}^2$) places them in the overweight category, in contrast to the normal BMI of the control group ($22.47 \pm 1.78 \text{ kg/m}^2$). This finding strongly corroborates the well-established link between excess adiposity and hypertension. The mechanistic basis for this association is multifactorial, involving activation of the sympathetic nervous system, stimulation of the renin-angiotensin-aldosterone system (RAAS) by adipocyte-derived factors (such as angiotensinogen), and physical compression of the kidneys by visceral fat, leading to altered pressure natriuresis [2, 3]. The higher weight observed in our cases (60.76 vs. 49.53 kg) further underscores the contribution of overall body mass to increased metabolic demand and vascular resistance.

Electrolyte Alterations: The Sodium and Chloride Paradox

Perhaps the most intriguing finding of this study pertains to the serum electrolyte profile. Contrary to the conventional expectation that hypertension is associated with high sodium, our results demonstrated significantly lower serum sodium and chloride levels in hypertensive subjects compared to controls ($p < 0.0001$). This seemingly paradoxical finding warrants careful interpretation.

First, it is critical to distinguish between dietary sodium intake and serum sodium concentration. Serum sodium is primarily a reflection of water homeostasis (hydration status) rather than sodium balance per se. Low serum sodium (approaching hyponatremia) in hypertensive patients could be explained by several factors. One plausible explanation is the use of diuretic medications (e.g., thiazides), which are a mainstay of antihypertensive therapy and are known to cause natriuresis and subsequent hyponatremia [4]. Although the study included patients "with or without antihypertensive treatment," the specific medication history was not detailed in the methodology, making this a potential confounding variable.

Second, the significantly lower chloride levels ($103.63 \text{ vs. } 109.1 \text{ mEq/L}$, $p < 0.0001$) parallel the sodium findings, which is expected as hyperchloremia often accompanies hyponatremia, and vice versa. However, chloride itself has emerging importance in hypertension. It is a key anion involved in renal vascular tone and the regulation of renin release. Some studies suggest that chloride may be a more critical determinant of salt-

sensitive hypertension than sodium, as it is necessary for the full pressor response to a high-salt diet [5].

Third, it is noteworthy that potassium levels were nearly identical between groups ($p = 0.9131$). This contrasts with some studies that report hypokalemia in hypertension, often secondary to secondary hyperaldosteronism or diuretic use [6]. The absence of hypokalemia in our hypertensive cohort suggests that either mineralocorticoid excess is not a dominant feature in this population, or that dietary potassium intake is adequate. This finding aligns with the 2025 study referenced earlier, which also reported a complex picture of electrolyte changes rather than a simple pattern of hypokalemia [7].

Evidence of Early Renal Impairment

The significantly elevated serum urea ($25.33 \text{ vs. } 20.06 \text{ mg/dL}$, $p = 0.0099$) and creatinine ($1.05 \text{ vs. } 0.81 \text{ mg/dL}$, $p = 0.0006$) in the hypertensive group provide clear evidence of early renal dysfunction, a condition often termed hypertensive nephrosclerosis. The kidney is both a victim and a contributor in hypertension. Chronic exposure to elevated systemic pressures is transmitted to the glomerular capillaries, leading to glomerular hyperfiltration, endothelial injury, and eventually, glomerulosclerosis and tubulointerstitial fibrosis [8].

Even though the mean creatinine of 1.05 mg/dL in the cases might still be considered within the normal reference range for some laboratories, the statistically significant difference compared to controls is clinically meaningful. It represents a loss of renal reserve and indicates that hypertensive damage begins long before creatinine levels become frankly abnormal. This finding underscores the importance of using more sensitive markers, such as estimated Glomerular Filtration Rate (eGFR) or cystatin C, in future studies to quantify the degree of renal impairment more accurately. The elevated uric acid (though not measured here) is another common finding in this context, as noted in the 2025 study, and contributes to both hypertension and renal injury through inflammatory and oxidative stress pathways [7, 9].

Vitamin D Deficiency and Hypertension

Our finding of significantly lower serum vitamin D levels in hypertensive subjects ($18.27 \pm 4.01 \text{ ng/mL}$) compared to controls ($20.25 \pm 6.32 \text{ ng/mL}$, $p = 0.0184$) adds to the robust body of literature linking vitamin D status to cardiovascular health. The mean level of 18.27 ng/mL falls within the range commonly classified as "insufficient" or "deficient" ($<20 \text{ ng/mL}$), while the control group's level hovers near the threshold of sufficiency.

The biological plausibility of this association is strong and centers on the RAAS. Vitamin D is a potent negative

endocrine regulator of renin expression. In the juxtaglomerular cells of the kidney, the active form of vitamin D (1,25-dihydroxyvitamin D) binds to the vitamin D receptor (VDR) and suppresses renin gene transcription by blocking the activity of the cAMP response element binding protein (CREB) [10]. Consequently, vitamin D deficiency leads to disinhibition of the RAAS, resulting in increased renin, angiotensin II, and aldosterone production, which in turn promotes vasoconstriction, sodium retention, and hypertension [11].

Furthermore, vitamin D deficiency is often associated with obesity, as vitamin D is fat-soluble and sequestered in adipose tissue, reducing its bioavailability [12]. This aligns with our observation of higher BMI in the hypertensive group, suggesting a potential synergistic effect where obesity exacerbates both vitamin D deficiency and hypertension. The lower vitamin D levels may also contribute to the electrolyte alterations observed, as vitamin D is essential for intestinal calcium absorption, and disruptions in calcium metabolism can influence vascular smooth muscle contractility [13].

Comparison with Existing Literature

Our results are broadly consistent with the major findings of the 2025 Nigerian case-control study, which reported significant differences in renal biomarkers, electrolytes, and vitamin D between hypertensives and controls [7]. However, a notable divergence is in the sodium findings; the 2025 study reported higher sodium in hypertensives, whereas we observed lower sodium. This discrepancy may be attributable to differences in dietary habits, hydration status, the prevalence of diuretic use in our cohort, or genetic variations in sodium handling. It highlights the importance of conducting population-specific research, as the pathophysiology of hypertension may manifest differently across ethnic and geographic groups.

The absence of potassium difference in our study contrasts with some earlier work, such as the 2023 study in elderly hypertensives which identified ionized magnesium deficiency [14]. This reinforces the concept that hypertension is a heterogeneous disorder, and the specific biochemical profile can vary significantly based on age, treatment status, and comorbidities.

Strengths of the Study

This study has several strengths. First, it simultaneously evaluates renal biomarkers, electrolyte levels, and vitamin D status in hypertensive subjects compared with normotensive controls, providing an integrated assessment of these interrelated biochemical parameters. Second, the inclusion of a control group allows comparative analysis of biochemical and anthropometric differences between hypertensive and normotensive

individuals. Third, standardized laboratory methods and an automated clinical chemistry analyzer were used to assess renal biomarkers and vitamin D levels, supporting consistency in biochemical measurements. Fourth, blood pressure was measured using three consecutive readings, with the average used for analysis, which helps improve the reliability of blood pressure assessment. Finally, the study explores the relationships between blood pressure, renal function, electrolyte balance, and vitamin D status, providing a basis for further research into the underlying mechanisms and potential clinical implications of these associations.

Limitations and Future Directions

The findings of this study must be interpreted in light of several limitations. First, the significant age difference between cases and controls is a major confounding variable, as age independently affects renal function and blood pressure. Future studies should employ stricter age-matching or use multivariate analysis to adjust for age. Second, the sample size (n=60) is relatively modest, which may limit the generalizability of the findings and the power to detect smaller differences. Third, the lack of detailed medication history (specifically the use of diuretics, ACE inhibitors, or ARBs) prevents us from determining whether the electrolyte changes are disease-related or treatment-induced. Fourth, we measured total serum calcium and magnesium but did not assess ionized fractions, which are the biologically active forms. Fifth, the study did not include dietary assessments, which could provide valuable context for the electrolyte findings. Finally, the cross-sectional case-control design can only demonstrate associations, not causation.

Future research should focus on larger, prospective cohort studies with age-matched controls. Incorporating measurements of PTH (parathyroid hormone), which links vitamin D and calcium metabolism, and 24-hour urinary electrolyte excretion would provide a more complete picture of mineral homeostasis. Interventional trials examining whether vitamin D supplementation can improve blood pressure control or slow renal decline in hypertensive patients with deficiency would also be valuable.

CONCLUSION

In conclusion, this study demonstrates that hypertension is associated with a complex biochemical phenotype characterized by elevated BMI, early renal impairment (elevated urea and creatinine), reduced vitamin D levels, and a distinct electrolyte profile marked by lower serum sodium and chloride. The significant vitamin D deficiency observed in the hypertensive group supports its potential role in the pathogenesis of hypertension, likely through RAAS dysregulation. These findings advocate for the routine monitoring of renal function and

vitamin D status in hypertensive patients and suggest that a comprehensive biochemical assessment may aid in risk stratification and the development of personalized therapeutic strategies.

DECLARATIONS

Ethical Considerations

This study received ethical approval from the Institutional Ethical Committee of Government Siddhartha Medical College prior to initiation. All procedures performed were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent

Written informed consent was obtained from all individual participants included in the study. The consent form explained the purpose of the study, the procedures involved, potential risks and benefits, and the voluntary nature of participation. Participants were informed of their right to withdraw from the study at any time without affecting their medical care.

Confidentiality

All participant data were anonymized prior to analysis. Personal identifiers were removed and replaced with unique study codes to ensure patient privacy and compliance with ethical guidelines for medical research. Data were stored securely with access restricted to the research team.

Author Information

The first author completed this research at The Government Siddhartha Medical College and Hospital, Vijayawada, Andhra Pradesh. This work was submitted in partial fulfillment of research requirements for the UGSRS program.

Author Contributions

- **Dr. M. Prasad:** Conceptualization, methodology design, supervision, manuscript review and editing
- **Lakshmi Gayatri P:** Investigation, patient recruitment, data collection, laboratory analysis
- **Dr. Salma Mahaboob R, Sarala M, Y Chand Basha SK, Chowdeswari N, Navneetha Lakshmi A:** Formal data analysis, statistical evaluation, manuscript preparation
- **All Authors:** Critical review of manuscript, approval of final version for submission

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Conflict of Interest

The authors declare that they have no competing interests. No financial or personal relationships with other people or organizations that could inappropriately influence this work exist.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

REFERENCES

1. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. *Nat Rev Nephrol.* 2020;16(4):223-237. doi:10.1038/s41581-019-0244-2
2. Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME. Obesity-induced hypertension: interaction of neurohumoral and renal mechanisms. *Circ Res.* 2015;116(6):991-1006. doi:10.1161/CIRCRESAHA.116.305697
3. Elattar S, Chand S, Salem A, et al. Obesity and Hypertension: Etiology and the Effects of Diet, Bariatric Surgery, and Antiobesity Drugs. *Cardiol Rev.* 2025; Published online April 23, 2025. doi:10.1097/CRD.0000000000000937
4. Dendorfer U, Inselmann G. Drug-related disorders of water and electrolyte metabolism. *Internist (Berl).* 2006;47(11):1121-1122, 1124-1126, 1128. doi:10.1007/s00108-006-1719-3
5. McCallum L, Lip S, Padmanabhan S. The hidden hand of chloride in hypertension. *Pflugers Arch.* 2015;467(3):595-603. doi:10.1007/s00424-015-1690-8
6. Vaidya A, Mulatero P, Baudrand R, Adler GK. The expanding spectrum of primary aldosteronism: implications for diagnosis, pathogenesis, and treatment. *Endocr Rev.*

- 2018;39(6):1057-1088. doi:10.1210/er.2018-00139
7. Oluboyo A, Omon E, Oluboyo B, Odewusi O, Edet O. Correlation of renal biomarkers, electrolyte imbalances and vitamin D levels in hypertensive subjects. *Med Int.* 2025;5(2). doi:10.3892/[mi.2025.XXX](#)
 8. Memuni S, Rubattu S, Pierelli G, Tocci G, Fofi C, Volpe M. Hypertension and kidneys: unraveling the complex molecular mechanisms linking hypertensive renal disease. *ActaPhysiol (Oxf).* 2014;212(2):115-127. doi:10.1111/apha.12336
 9. Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. *N Engl J Med.* 2008;359(17):1811-1821. doi:10.1056/NEJMra0800885
 10. Li YC, Kong J, Wei M, Chen ZF, Liu SQ, Cao LP. 1,25-Dihydroxyvitamin D(3) is a negative endocrine regulator of the renin-angiotensin system. *J Clin Invest.* 2002;110(2):229-238. doi:10.1172/JCI15219
 11. Loh HH, Azizan EA, Sukor N. Connecting the dots: renin-angiotensin-aldosterone system, vitamin D, and hypertension. *J Hypertens.* 2025; Published online May 2, 2025. doi:10.1097/HJH.0000000000004046
 12. Al Argan RJ, Alqatari SG, Alwaheed AJ, Hasan MA. Vitamin D deficiency in obesity: Epidemiological evidence, biological mechanisms, and clinical considerations. *Obes Med.* 2026;59:100680. doi:10.1016/j.obmed.2025.100680
 13. Villa-Etchegoyen C, Lombarte M, Matamoros N, Belizán JM, Cormick G. Mechanisms involved in the relationship between low calcium intake and high blood pressure. *Nutrients.* 2019;11(5):1112. doi:10.3390/nu11051112
 14. Dominguez LJ, Veronese N, Barbagallo M. Magnesium and hypertension in old age. *Nutrients.* 2021;13(1):139. doi:10.3390/nu13010139