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Comparison of Electrolyte, Urea, Creatinine and Lipid Profile Status in Hypertensive and Renal Disease Patients in Enugu State University of Science and Technology (ESUT) Teaching Hospital,



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ABSTRACT

The pathophysiological interplay between hypertension (HTN) and renal disease (RD) manifests in distinct biochemical perturbations that are critical for risk stratification and management. This study provides a comparative analysis of electrolyte homeostasis, renal function, and lipid metabolism across these conditions. A hospital-based, cross-sectional study was conducted involving 60 participants recruited from ESUT Teaching Hospital, Enugu, and categorized into four age-matched groups: HTN (n=15), RD (n=15), comorbid HTN/RD (n=15), and healthy controls (n=15). Serum electrolytes (sodium, potassium, chloride, bicarbonate), urea, creatinine, and a full lipid profile were quantified using standardized automated and manual spectrophotometric methods. The comorbid HTN/RD group exhibited the most profound dysregulation, characterized by severe azotemia with creatinine levels elevated over 50-fold ($303.43 \pm 289.62 \mu\text{mol/L}$) compared to controls ($52.00 \pm 10.28 \mu\text{mol/L}$). While electrolyte imbalances were subtle, a significant inter-group variance in sodium was observed. Lipid profiling revealed a specific atherogenic phenotype in renal and comorbid groups, marked by significant hypertriglyceridemia (1.57 ± 0.91 and $1.49 \pm 0.74 \text{ mmol/L}$, respectively, vs. $1.05 \pm 0.35 \text{ mmol/L}$ in controls; $p=0.046$), without concomitant alterations in LDL or total cholesterol. These findings delineate the most severe deterioration of renal function and a unique lipidemic pattern in comorbid patients, underscoring the necessity for integrated biochemical monitoring that includes triglyceride surveillance alongside renal parameters to mitigate the accelerated cardiorenal risk in this population.

Keywords: Electrolytes, Hypertension, Urea, Lipid profiles, Renal disease

Introduction

Hypertension stands as a predominant chronic non-communicable disease and the leading cardiovascular disorder, affecting approximately 1.4 billion adults,

representing about 33% of the population aged 30-79 years and is a critical risk factor for stroke, heart failure, and chronic kidney disease (CKD) (World Health Organisation, 2025). Clinically defined as a sustained systolic blood pressure $\geq 140 \text{ mmHg}$ and/or diastolic pressure $\geq 90 \text{ mmHg}$,

hypertension although often asymptomatic, it is a critical risk factor for severe complications including stroke, heart failure, chronic kidney disease (CKD), and premature mortality (Mills et al., 2020, Princewel et al., 2019). This elevated pressure is categorized as either primary (essential) or secondary hypertension, with both forms significantly increasing the risk of myocardial infarction, renal disease, and heart failure (Adeola et al., 2025). The interplay between hypertension and renal disease is particularly consequential. CKD itself affects over 10% of people globally (Csaba, 2022) and frequently coexists with hypertension in a bidirectional relationship that markedly accelerates morbidity and mortality (Yirga et al., 2025). The global pooled prevalence of CKD among hypertensive patients is notably high at 34.97% (Tekalign et al., 2021), with studies in Africa indicating an even greater burden in urban settings (Ajayi et al., 2021). As renal function declines, the kidney's role in maintaining homeostasis is compromised, leading to characteristic biochemical derangements (Molla et al., 2020). These include electrolyte disturbances such as imbalances in potassium, sodium, and bicarbonate, and the accumulation of nitrogenous wastes like urea and creatinine (Dhondup et al., 2017). Metabolic acidosis, a common complication of CKD, arises from this dysregulation and contributes to bone demineralization, muscle proteolysis, and disease progression (Korus et al., 2025). Furthermore, CKD induces a distinct form of dyslipidemia, which is a major driver of the elevated cardiovascular risk in this population. This profile is not characterized by high LDL cholesterol but rather by elevated triglycerides, low HDL-C, and altered lipoprotein composition (Lacount and Lisa, 2025). The pathophysiology involves the downregulation of lipoprotein lipase, delayed catabolism of triglyceride-rich lipoproteins, and functional impairments in HDL particles (Chan et al., 2009; Schuchardt et al., 2015). These collective disturbances in electrolytes, renal markers, and lipids signify advanced renal impairment and contribute to cardiovascular complications (Vaziri, 2014). Dietary factors, particularly high salt intake, which promotes fluid retention and increased blood volume, are a pivotal environmental determinant in the development of hypertension (Swift & Macgregor, 2014). Given this complex cardiorenal-metabolic interplay, integrated biochemical profiling is essential for risk stratification. However, while the general pathophysiology is well-described, there is a notable gap in direct, comparative studies that profile electrolytes, renal function, and lipids together across hypertension, renal disease, and their comorbid state within specific populations. This lack of local comparative data limits tailored management strategies. Therefore, this study aims to conduct a comparative analysis of serum electrolyte balance (sodium, potassium, chloride, bicarbonate), renal function markers (urea, creatinine), and lipid profiles in patients with hypertension, renal disease, and their comorbidities, in comparison to healthy controls at ESUT Teaching Hospital, Enugu State.

Materials and Methods

Study Area

The present study was conducted at the Enugu State University Teaching Hospital, Parklane, Enugu.

Study Design

A cross-sectional study was conducted at Enugu State University Teaching Hospital. Sixty participants were recruited: 15 hypertensive patients, 15 renal disease patients, 15 with both conditions, and 15 apparently healthy controls.

Ethical Approval

Ethical clearance for sample collection was granted by the Ethics and Scientific Committee of Enugu State University Teaching Hospital (ESUTH) with the help of our head of department prior to the commencement of the study. Informed consent was obtained from each subject who participated the study, prior to the sample collection. Furthermore, the study was conducted in accordance with the Declaration of Helsinki.

Sample Collection

Six milliliters of blood were collected aseptically via venipuncture and dispensed into a plain bottle. The sample was allowed to clot for one hour, after which the serum was separated by centrifugation at $5000 \times g$ for 5 min at room temperature. The serum was then carefully withdrawn into a pre-labeled tube and stored at -20°C until analysis. Serum electrolytes (sodium, potassium, bicarbonate, chloride), urea, and creatinine were measured using standard biochemical methods (Jelliffe, 2008; Hosten, 1990; Eisenman, 1967). Lipid profile (total cholesterol, triglycerides, HDL, LDL, VLDL) was determined enzymatically (Allain et al., 1974; Richmond, 1973; Burstein et al., 1970; Assmann et al., 1984).

Blood Pressure Measurements

Blood pressure was measured using a mercury sphygmomanometer (Kenz Model 605P, Suzuken Co., Ltd.) Participants were allowed to sit and rest in a quiet area for at least 15 minutes before the measurements were taken. Blood pressure was measured on the right arm with the arm relaxed and supported on the table at a 45° angle to the trunk. A total of three measurements taken at 5-minute intervals were averaged for diastolic blood pressure (DBP) and systolic blood pressure (SBP). Variations of < 5 mmHg were permitted. Based on their average SBP and DBP values, the study participants were divided into two groups (hypertensive and non-hypertensive groups).

Inclusion and Exclusion Criteria

Inclusion criteria for patient groups were adults aged 18 years and above with a confirmed diagnosis as outlined above. The key exclusion criterion for all participants was the refusal to provide informed consent.

Laboratory Diagnostic Method

Biochemical Analysis

Serum samples were analyzed for electrolytes, renal function parameters, and lipid profile using a combination of automated analysis and standardized manual spectrophotometric methods, following established principles and manufacturer's protocols.

Renal Function Parameters

Serum Creatinine

Serum creatinine was determined using the manual kinetic Jaffe's Method (Jelliffe 2008). The assay was performed by reacting 100 μ L of the serum sample with an alkaline picrate solution (500 μ L of 0.4 M sodium hydroxide and 500 μ L of 10 mM picric acid). The reaction mixture was incubated at room temperature, and the change in absorbance at 505 nm was measured kinetically over 120 seconds using a spectrophotometer. Creatinine concentration was calculated by comparing the kinetic absorbance change of the test sample to that of a known creatinine standard processed concurrently.

Serum Urea Measurement

Serum urea concentration was determined using a manual urease-Berthelot method (Berthelot's method) (Hosten, 1990). The principle involves the enzymatic hydrolysis of urea by urease to produce ammonia, which then reacts with phenol and hypochlorite under alkaline conditions to form a blue indophenol complex. The assay was performed by incubating 10 μ L of serum with 100 μ L of a urease reagent at 37°C for 10 minutes. Subsequently, 2.5 mL of phenol solution and 2.5 mL of alkaline hypochlorite solution were added. The reaction mixture was incubated for a further 15 minutes at 37°C to develop the blue colour. The absorbance of the resulting complex was measured at 540 nm using a spectrophotometer. A blank (distilled water) and a known urea standard were processed concurrently with each batch of samples.

Serum Bicarbonate Measurement

Serum bicarbonate concentration was determined using a modified Van Slyke titrimetric method. The assay is based on the reaction of bicarbonate with a known excess of hydrochloric acid, releasing carbon dioxide. The remaining unreacted acid is then titrated with a standardized sodium hydroxide solution to a phenol red endpoint (faint pink colour). The procedure was performed by adding 200 μ L of serum to a mixture containing 2 mL of distilled water and 1.0 mL of a standardized 0.01N hydrochloric acid solution. Two drops of phenol red indicator were added. The solution was mixed thoroughly to liberate CO₂ and then titrated with 0.01N sodium hydroxide until a stable, faint pink endpoint was observed. A blank titration, omitting the serum sample, was performed to determine the total titratable acid. The bicarbonate concentration was calculated based on the difference in titrant volume between the blank and the test,

reflecting the amount of acid consumed by the serum bicarbonate.

Determination of Electrolyte (ion-selective electrode)

Serum electrolytes (sodium, potassium, chloride, and bicarbonate) were measured using the Ion-Selective Electrode (ISE) module of an automated clinical chemistry analyzer (Eisenman, 1967). The ISE module of the AVL9180/Roche/Hitachi cobas c system is intended for the quantitative determination of electrolytes in serum using ion-selective electrodes. The method is based on the principle of potentiometry, where ion-selective membranes generate an electrical potential proportional to the activity of the specific ion in the serum sample. The analyser was calibrated according to the manufacturer's specifications before each run. All serum samples, along with quality control materials, were thoroughly mixed and processed according to the instrument's standard operating procedure to ensure accurate results.

Lipid Profile Analysis

The serum lipid profile, comprising triglycerides, total cholesterol, and high-density lipoprotein cholesterol (HDL-C), was determined by enzymatic colorimetric methods using commercial kits (Biosystem Laboratories, Spain).

Total Cholesterol Measurement

Total cholesterol was determined by an enzymatic colorimetric method. The assay involves the enzymatic hydrolysis of cholesterol esters by cholesterol esterase and the subsequent oxidation of free cholesterol by cholesterol oxidase. The hydrogen peroxide produced then reacts with 4-aminoantipyrine and phenol in the presence of peroxidase to form a red quinoneimine dye, the absorbance of which is measured spectrophotometrically and is proportional to the total cholesterol concentration.

Triacylglycerol

Plasma triacylglycerol concentrations were determined by enzymatic colorimetric assay as described previously (Tinder, 1969) and modified by Richmond (1973). This enzymatic assay involves the hydrolysis of triglycerides by lipase to produce glycerol. The glycerol was then oxidized in a coupled enzymatic reaction, leading to the production of a colored chromogen. The absorbance of this product was measured between 530-550 nm.

HDL-C Estimation (precipitation method)

HDL-cholesterol and LDL-cholesterol were determined enzymatically after precipitation of other lipoproteins as described by Burstein (Burstein et al., 1970) and Assmann (Assmann et al., 1984) respectively, using kits from Biosystem Laboratories (Spain). Precipitating reagents such as divalent cations and polyanions are used to remove all lipoproteins except HDL. The enzymatic method for total cholesterol (cholesterol oxidase) is used to quantify HDL-C. LDL was calculated using the Friedewald equation.

Statistical Analysis

Data were analyzed using a statistical software package (e.g., SPSS version 22.0, IBM). Continuous variables were presented as mean ± standard deviation, and categorical data as frequencies and percentages. Differences in biochemical parameters across the four study groups were compared using a one-way Analysis of Variance (ANOVA), followed by a post-hoc test (Duncan) for pairwise comparisons. A p-value < 0.05 was considered statistically significant.

Results

The serum levels of electrolytes, urea, and creatinine across the four study groups are summarized in Table 1. Statistical analysis revealed significant (p < 0.05) differences among the groups for sodium, urea, and creatinine. In contrast, no significant differences were observed for potassium, bicarbonate, or chloride levels.

The biochemical parameters assessed in this study are summarized in Table 1. The results indicate distinct profiles across the four study groups. The Hypertensive/Renal group demonstrated the most significant biochemical alterations. The mean serum urea concentration in this group (13.66 mmol/L) was approximately three times higher than the mean levels in the Control (4.46 mmol/L), Hypertensive (5.49 mmol/L), and Renal (4.90 mmol/L) groups. Most notably, serum creatinine was markedly elevated in the Hypertensive/Renal group (303.43 μmol/L), with a mean value several times higher than that of the control, hypertensive, and renal disease groups. In contrast, electrolyte levels, including sodium, potassium, chloride, and bicarbonate, showed comparatively minor variations across all groups. The serum lipid profiles of the study participants are summarized in Table 1. Analysis of the data revealed that triglyceride levels were the most notably altered parameter among the groups.

Table 1. Serum electrolytes, urea, creatinine and lipid levels in the study groups

Parameters	Hypertensive	Renals	Hy/Renals	Control
Sodium mmol/l	133.07 ± 4.62	137.53 ± 5.83	137.80 ± 6.29	135.06 ± 3.51
Potassium	4.03 ± 0.65	4.58 ± 0.86	4.51 ± 0.94	4.25 ± 0.37
Bicarbonate	24.39 ± 3.92	24.33 ± 2.31	22.59 ± 3.65	25.83 ± 2.63
Chloride	101.69 ± 5.35	100.93 ± 6.16	102.67 ± 7.06	97.87 ± 4.75
Urea	5.49 ± 2.52	4.90 ± 1.98	13.66 ± 8.88	4.46 ± 1.9
Creatinine	89.02 ± 23.53	54.53 ± 28.73	303.43 ± 289.67	52.00 ± 10.28
TC (mmol/L)	4.51 ± 1.34	4.63 ± 1.41	4.51 ± 1.44	4.61 ± 1.14
HDL (mmol/L)	1.16 ± 0.58	1.30 ± 0.47	1.21 ± 0.59	1.29 ± 0.33
LDL (mmol/L)	2.89 ± 1.14	2.59 ± 1.09	2.57 ± 1.32	2.85 ± 0.89
VLDL (mmol/L)	0.51 ± 0.18	0.71 ± 0.41	0.72 ± 0.36	0.48 ± 0.14
TG (mmol/L)	1.04 ± 0.43	1.57 ± 0.91	1.49 ± 0.74	1.05 ± 0.35

Data presented as Mean ± Standard Deviation. p-values in bold indicate statistical significance (p < 0.05). Abbreviations: HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; VLDL-C, Very Low-Density Lipoprotein Cholesterol

The most prominent finding was observed in the **Renal and Hypertensive/Renal groups**, which demonstrated significantly elevated levels of triglycerides and VLDL-C compared to the control and hypertensive groups. In contrast, the mean serum levels of Total Cholesterol, HDL-C, and LDL-C showed no significant difference Table 1.

Discussion

This study sought to compare the status of key biochemical parameters in hypertensive, renal disease, and comorbid patients at the Enugu State University Teaching Hospital. Our findings reveal a distinct biochemical profile, particularly in patients with coexisting hypertension and renal disease, characterized by severe renal impairment and dyslipidemia. Clinical investigations of renal function using parameters such as urea and creatinine are crucial for identifying renal dysfunction. In the present study, urea and creatinine levels were significantly higher in the hypertensive subjects compared with the control group, a finding that aligns with previous studies (Gupta and Khosla, 2021; Oluboyo, 2020; Emokpae, 2013). The most striking finding was the profound elevation of these markers specifically in the Hypertensive/Renal group. This suggests

a synergistic detrimental effect where hypertension accelerates kidney damage, and declining renal function exacerbates hypertension, creating a vicious cycle that rapidly compromises glomerular filtration rate (GFR) (Adeola et al., 2025). The relatively normal levels in the isolated disease groups may indicate earlier-stage pathology or effective therapeutic control, highlighting the comorbid state as a critical inflection point for renal decline. Regarding electrolytes, the data showed a significant difference in sodium levels. This finding contrasts with some previous studies that have reported an association between hypertension and elevated plasma sodium (Adeola et al., 2025; Uchejeso et al., 2016) and underscores the complex, and potentially population-specific, dysregulation of sodium balance in cardiorenal disease (Adeola et al., 2025). Furthermore, according to the ‘vasodysfunction theory’, salt loading leads to inadequate decreases in systemic vascular resistance (Morris et al., 2016). Therefore, treatments for essential hypertension should focus on reducing plasma sodium levels by decreasing dietary salt intake, and increasing sodium excretion (Rust et al., 2019). Regarding other electrolytes, the lack of significant hyperkalemia across patient groups is clinically noteworthy. This may reflect preserved renal potassium excretion in our

cohort or the effects of medications. Similarly, the lower bicarbonate levels in the Hypertensive/Renal group compared to control group, though not statistically significant, may indicate a trend toward metabolic acidosis, a known consequence of advancing renal disease due to impaired acid excretion (Korus et al., 2025). Our lipid profile analysis identified hypertriglyceridemia as the primary lipid abnormality in the Renal and Hypertensive/Renal groups, consistent with the classic dyslipidemia of chronic kidney disease (CKD), which is characterized by elevated triacylglycerol and reduced HDL cholesterol (Lacount et al., 2025). This pattern, characterized by elevated triglycerides without concomitant rises in LDL or total cholesterol, differs from typical cardiovascular risk profiles (Lacount et al., 2025). The finding that hypertension alone did not significantly alter the lipid profile, while renal involvement did, reinforces the concept that declining kidney function itself is a primary driver of this metabolic disturbance (Oh et al., 2025). This lipid profile is highly atherogenic and contributes to the accelerated cardiovascular disease burden observed in CKD patients (Princewel et al., 2019).

Conclusion

This study demonstrates that patients with coexisting hypertension and renal disease at ESUT Teaching Hospital exhibit the most severe biochemical derangements, marked by significant renal impairment and atherogenic dyslipidemia. The findings underscore that these conditions do not exist in isolation but interact to amplify patient risk. Therefore, an integrated management approach that includes aggressive blood pressure control, regular monitoring of renal function, and targeted management of lipid abnormalities, particularly hypertriglyceridemia, is essential to mitigate the high risk of progressive kidney failure and cardiovascular events in this population.

Limitations of the Study

This study has several limitations. Its cross-sectional design allows for the identification of associations but not the determination of causality. The sample size, while adequate for initial analysis, was relatively small and recruited from a single centre, which may limit the generalizability of the findings. Furthermore, the lack of data on specific medications (e.g., types of diuretics, statins), duration of disease, and dietary habits limits our ability to fully interpret some of the results, particularly the electrolyte and lipid levels. Despite these limitations, the present study has key implications for clinical practice, such as identifying new targets for treatment or improving existing treatments, as well as setting a template for future research.

Recommendation

In light of the significant renal impairment and dyslipidemia observed, particularly in patients with comorbid conditions, it is recommended that clinical management strategies for hypertensive patients in this setting be enhanced to include integrated screening of renal function and a full lipid panel, the adoption of tailored pharmaceutical plans that address the characteristic dyslipidemia of renal disease, and the consistent provision of lifestyle modification counseling focused on sodium and dietary fat intake. Furthermore, to

build upon these findings, future longitudinal studies with larger cohorts and detailed data on medication and diet are essential to elucidate the causal pathways of this cardiorenal metabolic interplay.

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Conflicts of Interest

The authors declare no conflicts of interest.

Authors' Contributions

All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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