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Electrolyte Imbalance and Renal Function Alterations in Type 1 and Type 2 Diabetes Mellitus: A Hospital-Based Cross-Sectional Study in South-Eastern Nigeria

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Abstract

Diabetes mellitus predisposes patients to electrolyte imbalance and renal dysfunction, contributing to increased morbidity and mortality. Comparative data between type 1 and type 2 diabetes in South-eastern Nigeria remain limited. The aim of this study was to evaluate and compare electrolyte profiles and renal function markers in patients with type 1 and type 2 diabetes mellitus, and to determine their relationship with glycaemic control. A hospital-based cross-sectional study was conducted at ESUT Teaching Hospital, Nigeria, involving 100 participants: 25 with type 1 diabetes, 50 with type 2 diabetes, and 25 healthy controls. Fasting blood glucose, HbA1c, serum electrolytes (sodium, potassium, chloride), urea, and creatinine were analysed using standard methods. Data were evaluated using ANOVA and Spearman's correlation ($p < 0.05$). Both diabetic groups showed significantly elevated fasting blood glucose and HbA1c compared to controls ($p < 0.001$). Serum sodium levels were significantly reduced, while potassium levels were elevated, particularly in type 1 diabetes ($p < 0.05$). Serum creatinine was significantly higher in diabetic patients, indicating early renal impairment. Fasting blood glucose showed significant positive correlations with sodium and potassium levels. Diabetes mellitus is associated with significant electrolyte disturbances and early renal dysfunction linked to poor glycaemic control. Routine monitoring of these parameters is essential for early detection and improved clinical management.

Keywords: Diabetes mellitus, Electrolytes, Renal Function, Creatinine, Glycaemic control

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INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. It remains a major global public health problem and a leading contributor to morbidity and mortality worldwide. Recent epidemiological data indicate a rapidly increasing burden of diabetes, particularly in low- and middle-income countries, including sub-Saharan Africa, where limitations in early diagnosis and long-term disease management persist (Eshetu et al., 2023; Pawar et al., 2024; Kumar et al., 2024). In Nigeria, the growing prevalence of diabetes has been accompanied by a rising incidence of chronic complications that significantly impair quality of life and strain healthcare systems. Chronic hyperglycaemia induces widespread metabolic and haemodynamic disturbances that adversely affect multiple organ systems, most notably the kidneys. Diabetic kidney disease (DKD) is the leading cause of chronic kidney disease globally and is associated with increased cardiovascular risk and premature mortality (Elsawy et al., 2025; Pawar et al., 2024). The pathophysiology of DKD involves glomerular hyperfiltration, oxidative stress, inflammation, and progressive nephron loss, ultimately resulting in impaired renal excretory function and biochemical derangements detectable through routine laboratory testing. In addition to structural and functional renal damage, diabetes mellitus is frequently complicated by electrolyte imbalances. These disturbances arise from a combination of mechanisms, including glucose-induced osmotic diuresis, altered renal tubular handling of electrolytes, insulin deficiency or resistance, and acid–base abnormalities (Eshetu et al., 2023; Kumar et al., 2024). Hyponatraemia commonly occurs as a dilutional effect of hyperglycaemia-mediated osmotic shifts of water from the intracellular to the extracellular compartment, while potassium imbalance reflects impaired insulin-mediated cellular uptake and reduced renal excretion. Hyperkalaemia is particularly concerning due to its association with life-threatening cardiac arrhythmias (Holkar et al., 2024; Pawar et al., 2024). Serum urea and creatinine are widely utilized biochemical markers for the assessment of renal function. Elevations in these parameters among patients with diabetes often indicate declining glomerular filtration rate and progressive renal impairment. While urea levels may be influenced by hydration status and dietary protein intake, serum creatinine remains a relatively more stable and sensitive indicator of early renal dysfunction in diabetic populations (Elsawy et al., 2025). The combined evaluation of renal markers and electrolyte profiles therefore provides valuable insight into the metabolic and renal status of patients with diabetes, particularly when comparisons are made between type 1 and type 2 diabetes mellitus. Despite the clinical importance of electrolyte and renal abnormalities in diabetes, there remains a paucity of locally generated data from south-eastern Nigeria, especially studies that directly

compare biochemical derangements between type 1 and type 2 diabetes mellitus. Such data are essential for guiding routine monitoring strategies, improving early detection of renal complications, and optimizing clinical management in resource-limited settings. Therefore, this study aimed to evaluate serum electrolyte profiles (sodium, potassium, and chloride) and renal function markers (urea and creatinine) among patients with type 1 and type 2 diabetes mellitus attending the Enugu State University of Science and Technology Teaching Hospital, Parklane, Nigeria, and to assess their relationship with glycaemic control.

Materials and Methods

Study design

This study was a hospital-based cross-sectional comparative study conducted at the diabetes and specialist clinics of the Enugu State University of Science and Technology (ESUT) Teaching Hospital, Parklane, Enugu, Nigeria. The study was carried out over three months from September to November 2025.

Study population and sample size

A total of 100 participants were recruited for this study. These comprised 25 patients diagnosed with type 1 diabetes mellitus (T1DM), 50 patients with type 2 diabetes mellitus (T2DM), and 25 apparently healthy non-diabetic individuals who serve as the control group. Diabetic patients were consecutively recruited during routine clinic visits, while control subjects were selected from hospital staff and patient relatives who met the eligibility criteria.

Inclusion and exclusion criteria

Adult patients (> 18 years) with a confirmed diagnosis of type 1 or type 2 diabetes mellitus attending ESUT Teaching Hospital were eligible for inclusion. Control participants were apparently healthy adults with no prior history of diabetes mellitus, renal disease, or electrolyte disorders. Participants were excluded if they had known chronic kidney disease, acute infections, hepatic disease, heart failure, pregnancy, or were receiving medications known to affect electrolyte balance or renal function, including diuretics and nephrotoxic drugs.

Ethical considerations

Ethical approval for the study was obtained from the ESUT Teaching Hospital Health Research Ethics Committee. Written informed consent was obtained from all participants prior to enrolment. Confidentiality of participants' data was ensured through anonymization using unique identification codes, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

Sample collection and processing

Following an overnight fast of 8 – 12 hours, five milliliters (5 mL) of venous blood was aseptically collected from each participant. Three milliliters were dispensed into lithium heparin tubes for the analysis of serum electrolytes, urea, and creatinine, while two milliliters were dispensed into fluoride oxalate tubes for fasting blood glucose estimation. Blood samples were centrifuged at 3000 x g for 10 minutes and the separated serum was stored at 2 – 8 °C and analysed within 24 hours.

Biochemical analysis

Fasting blood glucose was determined using the glucose oxidase-peroxidase enzymatic method as described by [Ochei and Kolhatkar \(2000\)](#). Briefly, 1000 µL of glucose reagent was added to two test tubes and 10 µL of the standard-to-standard tube, mixed, and incubated for 15 minutes at 37°C. The absorbance was then read at 505nm using a spectrophotometer (Spectrum Lab 23A, HME Global Ltd, England). Serum urea was determined using the urease-Berthelot colorimetric method ([Taylor & Vadguma, 1992](#)). Briefly, 10 µL of the sample was added to the test tube, 10 µL of the standard-to-standard tube, then the urease reagent was added to both tubes, respectively, mixed, and incubated at 37°C for 10 minutes. Then, 2.5 ml of phenol solution and sodium hypochlorite were added to both tubes mix and incubated for 15 minutes at 37°C, and a blue coloured compound was formed whose absorbance was read at 540nm using a (Spectrum Lab 23A, HME Global Ltd, England). Determination of serum creatinine by Jaffe's method ([Spencer, 1996](#)). 500 ul of sodium hydroxide was added to the test and standard tubes, respectively, after which 500 ul of picric acid was added to both tubes. 100ul of sample was added to the test tube, mix and read absorbance at 505 nm using a spectrophotometer (Spectrum Lab 23A, HME Global Ltd, England) following the manual protocol. Serum sodium (Na⁺), potassium (K⁺), and chloride (Cl⁻) concentrations were determined using an ion-selective electrode (ISE) analyzer. This method measures ion activity across selective membranes and is widely regarded for its analytical precision and reliability in clinical electrolyte assessment ([Kumar et al., 2024](#)).

Quality control

All biochemical analyses were performed following standard operating procedures. Commercially prepared quality control

sera were analyzed alongside patient samples to ensure analytical accuracy and precision. All assays were calibrated according to manufacturer recommendations.

Data analysis

Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 20.0. Results were expressed as mean ± standard deviation (SD). Comparisons among the three study groups were performed using one-way analysis of variance (ANOVA), followed by Tukey post hoc tests where applicable. Spearman's rank correlation coefficient was used to assess the relationship between fasting blood glucose and electrolyte or renal function parameters. A p-value ($p < 0.05$) was considered statistically significant.

Results

The [Table 1](#) demonstrates significant differences in glycaemic indices and selected renal and electrolyte parameters across the study groups. HbA1c and fasting blood glucose were significantly higher in both type 1 and type 2 diabetic patients compared with controls ($p < 0.001$), indicating poor glycaemic control. Serum sodium levels were significantly reduced in diabetic patients relative to controls ($p < 0.045$), reflecting dilutional hyponatraemia secondary to hyperglycaemia-induced osmotic shifts. Potassium levels were significantly elevated in diabetics, particularly in type 1 diabetes ($p < 0.013$), suggesting impaired renal potassium excretion and insulin-mediated intracellular potassium uptake. Serum creatinine was significantly higher in diabetic patients ($p < 0.012$), consistent with early renal dysfunction, while urea and chloride showed no statistically significant differences. Among insulin-dependent diabetic patients, fasting blood glucose showed a significant positive correlation with serum sodium ($p = 0.002$), indicating a relationship between glycaemic status and electrolyte imbalance. Although potassium and creatinine also demonstrated positive correlations with fasting glucose, these associations were not statistically significant [Table 2](#).

In non-insulin-dependent diabetic patients, fasting blood glucose correlated positively and significantly with serum sodium ($p < 0.001$) and potassium ($p < 0.04$). Negative, non-significant correlations were observed between fasting glucose and urea as well as chloride, suggesting variable renal tubular responses in type 2 diabetes [Table 3](#).

Table 1: Biochemical measurements in type 1 and type 2 diabetes mellitus subjects

Parameters	Type 1 Diabetes (n=25)	Type 2 diabetes (n=50)	Controls (n=25)	P-value
HbA1C (%)	9.8±1.9	10.4±2.1	4.5±0.4	0.000*
FBS (mg/dl)	219.58±96.65	76.42±2.63	108±13	0.000*
Sodium (meq/l)	98±2.5	108±2.9	138±3.3	0.045*
Potassium (meq/l)	7.6±0.98	5.40±0.65	4.16±0.82	0.013*
Chloride (meq/l)	117.4±5.88	119.37±5.97	99.7±0.04	0.124
Urea (µmol/l)	4.125±1.88	7.225±4.65	3.47±1.24	0.24
Creatinine (µmol/l)	98.13±16.70	104.91±67.62	97.23±14.32	0.012*

p-value <0.05 statistically significant, ANOVA Tukey post hoc test.*

Table 2: Correlation between fasting blood sugar and electrolytes in type 1 diabetic patients

Parameters	ρ -Value	t-value	P-value
FBS with Sodium (meq/l)	1.233	15.91	0.002*
FBS with Potassium (meq/l)	1.001	12.99	0.073
FBS with Chloride (meq/l)	-0.21	14.37	0.096
FBS with Urea (µmol/l)	-0.079	10.936	0.057
FBS with Creatinine (µmol/l)	1.234	6.320	0.247

p-value <0.05 statistically significant, Spearman rho correlation.*

Table 3: Correlation between fasting blood sugar and electrolytes in type 2 diabetic patients

Parameters	ρ -Value	t-value	P-value
FBS with Sodium (meq/l)	1.095	12.61	0.001*
FBS with Potassium (meq/l)	1.001	16.21	0.04*
FBS with Chloride (meq/l)	-0.373	17.71	0.134
FBS with Urea (µmol/l)	-0.194	10.936	0.96
FBS with Creatinine (µmol/l)	1.193	49.5	0.065

p-value <0.05 statistically significant, Spearman rho correlation.*

Discussion

The present study demonstrates significant electrolyte disturbances and evidence of renal dysfunction among patients with diabetes mellitus compared with non-diabetic controls. Poor glycaemic control, as reflected by elevated fasting blood glucose and HbA1c levels, is consistent with reports indicating that inadequate glycaemic regulation is a major driver of metabolic and electrolyte derangements in diabetes (Eshetu et al., 2023; Pawar et al., 2024). The significantly reduced serum sodium observed among diabetic patients suggests dilutional hyponatraemia secondary to hyperglycaemia-induced osmotic shifts. Elevated plasma glucose increases extracellular osmolality, drawing water from the intracellular compartment and diluting serum sodium concentration, a mechanism widely described in diabetic populations (Eshetu et al., 2023; Kuamr et al., 2024). The significant association between fasting blood glucose and sodium further supports this physiological relationship. Hyperkalaemia, particularly pronounced in type 1 diabetes, may be attributed to insulin deficiency, which impairs intracellular potassium uptake, as well as early renal dysfunction that limits potassium excretion (Pawar et al., 2024; Holker et al., 2024). Insulin plays a central role in potassium homeostasis, and its absence or resistance in diabetes predisposes patients to elevated serum potassium levels, increasing the risk of cardiac arrhythmias (Kumar et al., 2024; Holker et al., 2024). Elevated serum creatinine

observed in diabetic patients indicates early renal impairment consistent with diabetic kidney disease. Chronic hyperglycaemia induces glomerular hyperfiltration, oxidative stress, and mesangial expansion, ultimately leading to reduced glomerular filtration rate and creatinine retention (Elsawy et al., 2025; Pawar et al., 2024). Although urea levels were not significantly altered, creatinine appears to be a more sensitive marker of early renal dysfunction in this cohort (Eshetu et al., 2023; Elsayy et al., 2025). Overall, the findings underscore the close interplay between glycaemic control, electrolyte balance, and renal function in diabetes mellitus. Routine monitoring of electrolytes and renal markers is therefore essential for early identification of complications and prevention of adverse clinical outcomes (Pawar et al., 2024; Kumar et al., 2024). This study has several limitations that should be considered when interpreting the findings. First, the cross-sectional design limits the ability to establish causal relationships between glycaemic control, electrolyte disturbances, and renal dysfunction. Longitudinal studies would be more appropriate to assess temporal changes and progression of biochemical abnormalities in diabetes mellitus. The relatively small sample size, particularly among patients with type 1 diabetes mellitus, may limit the generalizability of the findings to the broader diabetic population. Additionally, the study was conducted in a single tertiary healthcare facility, which may

introduce selection bias and restrict the applicability of the results to other settings or regions.

Conclusion

This study demonstrates that patients with diabetes mellitus attending ESUT Teaching Hospital exhibit significant electrolyte disturbances and evidence of early renal dysfunction, particularly hyponatraemia, hyperkalaemia, and elevated serum creatinine. These abnormalities correlate with poor glycaemic control and underscore the need for routine biochemical monitoring in diabetic care. Early identification and correction of electrolyte and renal abnormalities may reduce the burden of diabetes-related morbidity and mortality.

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Conflict of interest

The authors declare no conflict of interest in the publication of this manuscript

Informed consent

Not applicable

Authors contribution

UGWUENE, Francis O.: Participated in designing and carrying out the experiment

JOSHUA, Parker E.: Contributed to designing and carrying out the experiment and in revision of the manuscript

MUANKANG, Junior Tegha K.: Participated in the writing and reviewing of the manuscript

ORHONIGBE, Innocent O.: Participated in the review and followed up on the writing of the manuscript

OKA, Samson: Participated in the review and followed up on the writing of the manuscript

EZE, Tochukwu R.: Participated in reviewing and following up on the writing of the manuscript

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