Pakistan Journal of Medical & Cardiological Review
https://pakjmcr.com/index.php/1/about
Online ISSN 3007-2387
Print ISSN 3007-2379

Comparative Evaluation of Electrolyte Imbalance in Diabetic Patients with and without Acute Kidney Injury: A Cross-Sectional Clinical Study

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Keywords: Diabetes Mellitus, Acute Kidney Injury, Electrolyte Imbalance, Hyperkalemia, Renal Dysfunction, Bicarbonate, Estimated Glomerular Filtration Rate

Received on 15 May 2026
Accepted on 13 Jun 2026
Published on 23 Jun 2026

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Abstract

Background: Diabetes mellitus is a major risk factor for electrolyte disturbances and acute kidney injury (AKI), both of which contribute significantly to morbidity and mortality. Impaired renal function in diabetic patients may disrupt electrolyte and acid–base homeostasis, leading to clinically significant complications.

Objective: To compare glycemic status, renal function, electrolyte profiles, and acid–base balance among healthy controls, diabetic patients without AKI, and diabetic patients with AKI.

Methods: This comparative cross-sectional study included 150 participants equally divided into three groups: healthy controls (Group A), diabetic patients without AKI (Group B), and diabetic patients with AKI (Group C). Clinical parameters including body mass index (BMI), blood pressure, and duration of diabetes were recorded. Fasting serum glucose, HbA1c, blood urea, serum creatinine, estimated glomerular filtration rate (eGFR), sodium, potassium, chloride, and bicarbonate levels were analyzed using standard biochemical methods. Statistical analysis was performed using one-way ANOVA with Tukey's post-hoc test and Spearman's correlation analysis.

Results: Significant differences were observed among the study groups for glycemic, renal, and electrolyte parameters ($p < 0.05$). Diabetic patients with AKI exhibited the highest fasting serum glucose (286.2 ± 10.3 mg/dL), HbA1c (9.8 ± 1.6%), blood urea (78.9 ± 6.5 mg/dL), and serum creatinine (3.2 ± 0.5 mg/dL), while eGFR was significantly reduced (38.7 ± 4.6 mL/min/1.73m²). Serum potassium progressively increased from controls to diabetic patients with AKI (4.1 ± 0.05, 4.8 ± 0.06, and 5.92 ± 0.08 mEq/L, respectively; $p < 0.001$), whereas sodium, chloride, and bicarbonate levels significantly decreased. Correlation analysis revealed a strong negative association between eGFR and serum potassium ($r = −0.682$, $p < 0.001$) and a strong positive association between eGFR and serum bicarbonate ($r = 0.574$, $p < 0.001$).
Conclusion: Diabetic patients with acute kidney injury experience substantial deterioration in glycemic control, renal function, electrolyte balance, and acid–base status. Hyperkalemia and metabolic acidosis were the most pronounced abnormalities and were strongly associated with declining renal function. Regular monitoring of renal biomarkers and electrolyte profiles may facilitate early detection of complications and improve clinical management in diabetic patients at risk of AKI.

Introduction

Electrolyte abnormalities are among the most common metabolic consequences of both diabetes mellitus and acute kidney injury (AKI) and often reflect the severity of underlying renal dysfunction (Deabes & Essa, 2024). The kidneys are primarily responsible for maintaining electrolyte homeostasis through the regulation of sodium, potassium, chloride, calcium, and other ions, thereby ensuring internal metabolic stability and normal physiological functioning (Imenez Silva & Mohebbi, 2022). When renal function deteriorates, this regulatory capacity becomes impaired, leading to clinically significant disturbances in fluid, electrolyte, and acid–base balance (Almazmomi et al., 2023). Sodium, potassium, and chloride are particularly important because they are essential for maintaining fluid balance, osmotic pressure, nerve conduction, muscle contraction, and acid–base equilibrium. Disturbances in these electrolytes can result in dehydration, edema, neuromuscular dysfunction, cardiac arrhythmias, and metabolic acidosis (Deabes & Essa, 2024). Potassium imbalance is of particular concern in diabetic patients with AKI. Under normal physiological conditions, excess potassium is efficiently excreted by the kidneys, while insulin facilitates its movement into cells (Imenez Silva & Mohebbi, 2022). However, insulin deficiency or resistance, combined with impaired renal potassium excretion due to kidney injury, predisposes diabetic patients to hyperkalemia (Almazmomi et al., 2023). Elevated serum potassium levels can lead to life-threatening cardiac conduction abnormalities and sudden death if not recognized and treated promptly (Ertek & Caglar, 2025).

Sodium disturbances are also frequently observed in diabetes and acute kidney injury. Hyperglycemia can cause osmotic shifts that move water from the intracellular to the extracellular compartment, leading to dilutional hyponatremia. In addition, osmotic diuresis may contribute to sodium loss through increased urinary excretion. Volume depletion, impaired renal handling of sodium, and reduced glomerular filtration may further worsen sodium imbalance in patients with kidney injury. Although sodium abnormalities are often less dramatic than potassium disturbances, they can still affect neurological function, fluid balance, and cardiovascular stability (Liamis et al., 2014).

Chloride is another electrolyte that deserves attention in renal and metabolic disorders. Chloride helps maintain electroneutrality, acid-base equilibrium, and proper fluid distribution. In diabetic patients with kidney injury, chloride disturbances may occur due to altered renal tubular handling, dehydration, and changes in acid-base status. Hypochloremia may contribute to metabolic disturbances and may reflect broader derangements in renal function. Because chloride is sometimes overlooked in routine

clinical practice, its assessment can provide additional insight into the metabolic state of diabetic patients with renal dysfunction (Salih et al., 2019).

The relationship between diabetes mellitus and acute kidney injury is clinically important and increasingly recognized. Diabetic patients are more vulnerable to acute renal complications because they often have underlying endothelial dysfunction, microvascular damage, and impaired renal reserve. When acute kidney injury occurs in such patients, the clinical course may become more severe due to combined metabolic and renal stress. Electrolyte imbalance in this setting is not simply a laboratory abnormality; it is a marker of disease severity and a predictor of complications. Early detection of these abnormalities may improve patient management and prevent adverse outcomes (Claure & Bouchard, 2012; Deabes & Essa, 2024). Despite the clinical importance of this issue, comparative data on electrolyte disturbances in diabetic patients with and without acute kidney injury remain limited in many settings, particularly in local populations. Most available studies focus on either diabetes alone or acute kidney injury alone, while fewer have directly compared electrolyte profiles across diabetic patients with and without renal injury and healthy controls. Such comparative analysis is valuable because it helps clarify the magnitude of electrolyte changes associated with acute kidney injury in diabetic patients and highlights the need for close biochemical monitoring (Khanduker et al., 2017).

Therefore, the present study was designed to evaluate serum sodium, potassium, chloride, and glucose levels among healthy controls, diabetic patients without acute kidney injury, and diabetic patients with acute kidney injury. By comparing these groups, the study aims to determine the extent of electrolyte imbalance associated with acute kidney injury in diabetes and to emphasize the clinical importance of early recognition and management of these disturbances. This information may contribute to better understanding of renal complications in diabetes and support improved patient care strategies.

Materials and Methods

Study Design and Participants

This comparative cross-sectional study was conducted to evaluate glycemic control, renal function, electrolyte status, and acid–base balance among healthy individuals and diabetic patients with and without acute kidney injury (AKI). A total of 150 participants were enrolled and equally divided into three groups (n = 50 per group): Group A comprised apparently healthy individuals without a history of diabetes mellitus or renal disease; Group B included patients diagnosed with type 2 diabetes mellitus without AKI; and Group C consisted of diabetic patients with clinically confirmed AKI. Participants were recruited using a non-probability consecutive sampling technique from patients attending the medical and nephrology outpatient departments during the study period. Diabetes mellitus was diagnosed according to the American Diabetes Association (ADA) criteria, including fasting plasma glucose ≥126 mg/dL, HbA1c ≥6.5%, or a documented clinical diagnosis of diabetes mellitus (Association, 2024). AKI was diagnosed according to the kidney disease: Improving Global Outcomes (KDIGO) Clinical Practice Guidelines, defined as an increase in serum creatinine of ≥0.3 mg/dL within 48 hours, an increase in serum creatinine to ≥1.5 times baseline within the previous 7 days, or urine output <0.5 mL/kg/h for at least 6 hours (Khwaja, 2012).

Patients with advanced chronic kidney disease (Stages 4-5), chronic liver disease, malignancy, autoimmune disorders, pregnancy, or those receiving medications known to significantly affect electrolyte balance, such as potassium-sparing diuretics, corticosteroids, or bicarbonate therapy, were excluded from the study.

Ethical Approval
The study protocol was reviewed and approved by the Institutional Ethical Review Committee of the Institute of Molecular Biology and Biotechnology, The University of Lahore. Written informed consent was obtained from all participants prior to enrollment. All study procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Clinical and Demographic Assessment
Demographic and clinical information including age, sex, duration of diabetes, and medical history were recorded using a structured data collection form. Body weight and height were measured using standard procedures, and body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). Blood pressure measurements were obtained using a calibrated sphygmomanometer after a minimum resting period of five minutes. Both systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded.

Blood Sample Collection and Biochemical Analysis
Following an overnight fast of 8–12 hours, approximately 5 mL of venous blood was collected aseptically from each participant. Blood samples were allowed to clot and were subsequently centrifuged at 3000 rpm for 10 minutes to obtain serum for biochemical analyses. Fasting serum glucose was measured using the glucose oxidase–peroxidase (GOD-POD) enzymatic method. Glycated hemoglobin (HbA1c) was determined by high-performance liquid chromatography (HPLC) according to National Glycohemoglobin Standardization Program (NGSP) recommendations. Blood urea concentration was measured using the urease–glutamate dehydrogenase enzymatic method, while serum creatinine was determined using the kinetic Jaffe method on an automated clinical chemistry analyzer. Estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation developed (Levey et al., 2009). Serum sodium (Na^+), potassium (K^+), and chloride (Cl^-) concentrations were measured using ion-selective electrode (ISE) technology. Serum bicarbonate (HCO_3^-) concentration was determined using an enzymatic bicarbonate assay on the automated clinical chemistry analyzer. All biochemical analyses were performed according to the manufacturer's instructions, and internal quality-control procedures were implemented throughout the study to ensure analytical reliability and accuracy.

Statistical Analysis
Data were analyzed using Statistical Package for Social Sciences (SPSS) software version 29.0.1.1 (IBM Corporation, Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. The normality of data distribution was assessed using the Shapiro–Wilk test. Comparisons among the three study groups were performed using one-way analysis of variance (ANOVA), followed by Tukey's post-hoc test for pairwise multiple comparisons. Spearman's rank correlation analysis was used to evaluate the relationships between estimated glomerular filtration rate (eGFR) and serum electrolyte as well as acid–base parameters. A p-value of less than 0.05 was considered statistically significant.

Results
The study included 150 participants equally distributed among the three study groups (n = 50 per group). Baseline demographic and clinical characteristics are presented in Table 1. No statistically significant differences in age and sex distribution were observed among the groups ($p > 0.05$). However, significant differences were

observed in BMI, duration of diabetes, blood pressure, and HbA1c levels, all showing a progressive increase from controls to diabetic patients with AKI ($p < 0.001$ for all).

Glycemic Control and Renal Function:
Fasting serum glucose levels increased significantly across the groups, with the highest levels observed in Group C (286.2 ± 10.3 mg/dL) compared to Group B (198.5 ± 7.6 mg/dL) and controls (92.4 ± 4.1 mg/dL; $p < 0.001$). Renal function markers demonstrated a marked deterioration in Group C, as evidenced by significantly elevated blood urea (78.9 ± 6.5 mg/dL) and serum creatinine (3.2 ± 0.5 mg/dL) levels compared to Group B (42.3 ± 4.1 mg/dL and 1.4 ± 0.3 mg/dL, respectively) and controls (28.6 ± 3.2 mg/dL and 0.9 ± 0.2 mg/dL, respectively; $p < 0.001$ for all). Correspondingly, eGFR showed a progressive decline from controls (94.6 ± 8.2 mL/min/1.73m²) to Group B (72.3 ± 10.5 mL/min/1.73m²) and Group C (38.7 ± 4.6 mL/min/1.73m²; $p < 0.001$), indicating significant impairment of renal function in diabetic patients with AKI.

Table 1. Comparison of Glycemic Control, Renal Function, and Demographic Parameters Among Study Groups

Parameter	Group A (Control) (n=50)	Group B (Diabetes without AKI) (n=50)	Group C (Diabetes with AKI) (n=50)	p-value*
Demographic Characteristics				
Age (years)	52.4 ± 8.3	54.1 ± 7.9	56.3 ± 8.7	0.068
Sex (Male/Female)	27/23	28/22	26/24	0.872
Body Mass Index (kg/m²)	24.3 ± 2.8	27.6 ± 3.4	28.9 ± 3.9	<0.001
Duration of Diabetes (years)	N/A	8.4 ± 4.2	12.7 ± 5.1	<0.001
Systolic BP (mmHg)	122.4 ± 8.6	134.2 ± 10.3	142.8 ± 12.1	<0.001
Diastolic BP (mmHg)	78.2 ± 5.4	82.6 ± 6.8	85.4 ± 7.2	<0.001
HbA1c (%)	5.2 ± 0.4	8.1 ± 1.2	9.8 ± 1.6	<0.001
Glycemic Control				
Fasting Serum Glucose (mg/dL)	92.4 ± 4.1	198.5 ± 7.6	286.2 ± 10.3	<0.001
Renal Function				
Blood Urea (mg/dL)	28.6 ± 3.2	42.3 ± 4.1	78.9 ± 6.5	<0.001
Serum Creatinine (mg/dL)	0.9 ± 0.2	1.4 ± 0.3	3.2 ± 0.5	<0.001
eGFR (mL/min/1.73m²)	94.6 ± 8.2	72.3 ± 10.5	38.7 ± 4.6	<0.001

Data are presented as mean ± standard deviation (SD). p-value was calculated using one-way ANOVA; $p < 0.05$ was considered statistically significant. AKI: Acute Kidney Injury; eGFR: estimated Glomerular Filtration Rate; BP: Blood Pressure; HbA1c: Glycated Hemoglobin

Electrolyte and Acid-Base Parameters:
Electrolyte analysis revealed significant disturbances among the study groups, as detailed in Table 2. Serum sodium and chloride levels showed mild but statistically significant decreases in Group C (134.8 ± 1.8 mEq/L and 98.6 ± 1.2 mEq/L,

respectively) compared to controls (140.2 ± 2.1 mEq/L and 103.4 ± 1.8 mEq/L; p = 0.041 and p = 0.032, respectively). In contrast, serum potassium levels demonstrated a significant progressive increase across the groups (4.1 ± 0.05, 4.8 ± 0.06, and 5.92 ± 0.08 mEq/L in Groups A, B, and C, respectively; p < 0.001), indicating clinically relevant hyperkalemia in patients with AKI. Serum bicarbonate levels were significantly reduced in Group C (18.3 ± 2.0 mEq/L) compared to Group B (22.1 ± 1.8 mEq/L) and controls (24.8 ± 1.5 mEq/L; p < 0.001), indicating metabolic acidosis associated with worsening renal function. Overall, the findings demonstrate a progressive deterioration in glycemic control, renal function, and electrolyte balance from healthy controls to diabetic patients, with the most severe abnormalities observed in diabetic patients complicated by acute kidney injury.

Table 2. Comparison of Serum Electrolyte and Acid-Base Parameters Among Study Groups

Parameter	Group A (Control) (n=50)	Group B (Diabetes without AKI) (n=50)	Group C (Diabetes with AKI) (n=50)	p-value*
Electrolytes				
Serum Sodium (mEq/L)	140.2 ± 2.1	137.5 ± 1.9	134.8 ± 1.8	0.041
Serum Potassium (mEq/L)	4.1 ± 0.05	4.8 ± 0.06	5.92 ± 0.08	<0.001
Serum Chloride (mEq/L)	103.4 ± 1.8	101.2 ± 1.5	98.6 ± 1.2	0.032
Acid-Base Status				
Serum Bicarbonate (mEq/L)	24.8 ± 1.5	22.1 ± 1.8	18.3 ± 2.0	<0.001

Data are presented as mean ± standard deviation (SD). p-value was calculated using one-way ANOVA; p < 0.05 was considered statistically significant.

Table 3 presents the pairwise comparisons between groups. For glycemic and renal function parameters, all pairwise comparisons between groups (A vs B, A vs C, and B vs C) were statistically significant (p < 0.001 for all), confirming a progressive deterioration across the study groups. For electrolyte parameters, serum potassium showed significant differences between all pairs (p < 0.001 for all comparisons). Serum sodium showed significant differences between all pairs (p = 0.042 for A vs B; p = 0.038 for A vs C; p = 0.041 for B vs C). Serum chloride demonstrated significant differences between all groups (p = 0.031, 0.029, and 0.033 for A vs B, A vs C, and B vs C, respectively). Serum bicarbonate showed significant differences between controls and both diabetic groups (p = 0.028 for A vs B; p < 0.001 for A vs C) and between diabetic groups (p < 0.001 for B vs C).

TABLE 3: Pairwise Comparison of Parameters Between Groups (Post-hoc Analysis)

Parameter	Group A vs B (p-value)	Group A vs C (p-value)	Group B vs C (p-value)
Fasting Glucose (mg/dL)	<0.001	<0.001	<0.001
Blood Urea (mg/dL)	<0.001	<0.001	<0.001
Serum Creatinine (mg/dL)	<0.001	<0.001	<0.001
eGFR (mL/min/1.73m²)	<0.001	<0.001	<0.001

Serum Sodium (mEq/L)	0.042	0.038	0.041
Serum Potassium (mEq/L)	<0.001	<0.001	<0.001
Serum Chloride (mEq/L)	0.031	0.029	0.033
Serum Bicarbonate (mEq/L)	0.028	<0.001	<0.001

Data presented as p-values from Tukey's post-hoc test following one-way ANOVA. $p < 0.05$ considered statistically significant.

Correlation analysis (Table 4) revealed a strong negative correlation between eGFR and serum potassium ($r = -0.682$, $p < 0.001$) and a strong positive correlation between eGFR and serum bicarbonate ($r = 0.574$, $p < 0.001$). Moderate positive correlations were observed between eGFR and serum sodium ($r = 0.412$, $p = 0.003$) and serum chloride ($r = 0.389$, $p = 0.008$), indicating that declining renal function is associated with worsening electrolyte disturbances.

TABLE 4: Correlation Between eGFR and Electrolyte Parameters (Spearman's Correlation)

Parameter	Correlation Coefficient (r)	p-value
Serum Potassium (mEq/L)	-0.682	<0.001
Serum Bicarbonate (mEq/L)	0.574	<0.001
Serum Sodium (mEq/L)	0.412	0.003
Serum Chloride (mEq/L)	0.389	0.008

Data presented as Spearman's rank correlation coefficient (r). Negative correlation indicates inverse relationship with eGFR.

Discussion

The present study demonstrates a progressive and significant deterioration in glycemic control, renal function, and electrolyte homeostasis across the spectrum from healthy controls to diabetic patients, with the most profound abnormalities observed in those complicated by AKI. The markedly elevated fasting serum glucose levels observed in diabetic patients with AKI (286.2 ± 10.3 mg/dL) compared to those without AKI (198.5 ± 7.6 mg/dL) suggest a bidirectional relationship between hyperglycemia and renal impairment (Kaur et al., 2023; Kumar et al., 2023). Poor glycemic control drives diabetic kidney disease through advanced glycation end-products, oxidative stress, and RAAS activation, while declining renal function impairs insulin clearance, creating a vicious cycle of worsening hyperglycemia (Kumar et al., 2023). The elevated glucose levels observed in Group C likely reflect a complex interplay between severe underlying diabetes and compromised renal insulin clearance. As kidney function declines, the physiological degradation of peripheral insulin decreases, paradoxically compounding glycemic volatility. This finding aligns with established clinical literature linking acute kidney injury (AKI) in diabetic cohorts to poorer long-term glycemic outcomes and increased mortality rates (Kaur et al., 2023)

The marked reduction in eGFR observed in diabetic patients with AKI indicates substantial impairment of renal filtration capacity and reflects the severity of acute renal dysfunction. Because eGFR estimation is based on serum creatinine concentration, its interpretation during acute kidney injury should be made cautiously, as rapidly changing creatinine levels may influence the accuracy of eGFR calculations. Nevertheless, the significantly lower eGFR values observed in Group C clearly demonstrate severe deterioration of renal function compared with diabetic patients without AKI and healthy controls (Eisinger et al., 2025; Ihim et al., 2019). The marked elevation of serum creatinine and blood urea nitrogen observed in diabetic

patients with AKI reflects substantial impairment of renal function and is consistent with the clinical diagnosis of acute kidney injury. However, the absence of historical renal function data precludes differentiation between isolated AKI and acute-on-chronic kidney disease. Diabetic individuals are uniquely predisposed to this pathophysiological trajectory due to advanced microvascular damage, which leaves the renal parenchyma exceptionally vulnerable to nephrotoxic insults and acute hemodynamic instability (Ihim et al., 2019).

The elevated urea level observed in Group C (78.9 ± 6.5 mg/dL) likely reflects a combination of increased protein catabolism and a prerenal azotemia component due to osmotic diuresis and volume depletion, which are well-established features of AKI in diabetic patients (Hoste et al., 2018; Kellum et al., 2021). Early identification and correction of hypovolemia remain critical to prevent progression of renal dysfunction.

Electrolyte disturbances were prominent in the present study. Mild hyponatremia in Group C (134.8 ± 1.8 mEq/L) can be explained by hyperglycemia-induced osmotic water shift and dilutional hyponatremia. Hillier et al. (1999) demonstrated that serum sodium decreases by approximately 1.6–2.4 mEq/L for every 100 mg/dL increase in glucose, a mechanism still widely accepted in clinical nephrology practice (Hillier et al., 1999). The marked hyperkalemia observed in Group C (5.92 ± 0.08 mEq/L) is clinically significant, as potassium levels above 5.5 mEq/L are associated with increased risk of arrhythmias and mortality. Hyperkalemia in AKI results from impaired renal excretion, insulin deficiency, and reduced cellular uptake of potassium (Kog et al., 2025; Palmer, 2015). Even the elevated potassium in Group B reflects early impairment of renal potassium handling in diabetes before overt AKI develops. This early metabolic vulnerability is further validated by our pairwise post-hoc analysis (Table 3), which demonstrated statistically significant differences between healthy controls (Group A) and diabetic patients without AKI (Group B) across all evaluated electrolyte and acid-base parameters ($p < 0.05$). This indicates that even in the absence of acute clinical renal failure, the diabetic milieu itself—characterized by sustained hyperglycemia (198.5 ±7.6 mg/dL and insulin resistance subtly compromises tubular handling of sodium ($p = 0.042$) and chloride ($p = 0.031$), while initiating a low-grade decline in buffering capacity ($p = 0.028$ for bicarbonate). Consequently, electrolyte dysregulation is a continuous pathophysiological spectrum that begins long before the abrupt functional drop characterizing AKI. Reduced bicarbonate levels in Group C (18.3 ± 2.0 mEq/L) indicate metabolic acidosis due to decreased renal acid excretion and accumulation of organic acids. Metabolic acidosis is a recognized complication of AKI and is associated with increased mortality, muscle catabolism, and cardiovascular instability (Kraut & Madias, 2010, 2012). The decrease in chloride levels (98.6 ± 1.2 mEq/L) may be associated with volume depletion and altered tubular electrolyte handling in AKI, as described in acid–base and intensive care nephrology literature (Adrogué & Madias, 2000).

To further elucidate the driving force behind these electrolyte shifts, correlation analysis was utilized to directly tie homeostatic disruption to functional nephron loss (Table 4). The strong negative correlation observed between eGFR and serum potassium ($r = -0.682$, $p < 0.001$) underscores that the severity of hyperkalemia is tightly bound to the reduction of glomerular filtration and subsequent distal tubular flow rates. Conversely, the strong positive correlation between eGFR and serum bicarbonate ($r = 0.574$, $p < 0.001$) mathematically validates the progressive failure of renal ammoniagenesis and bicarbonate reclamation as filtration capacity diminishes. The moderate positive correlations linking eGFR with sodium ($r = 0.412$) and chloride ($r = 0.389$) reinforce that mechanical nephron filtration deficits, rather than isolated extra-renal osmotic shifts, serve as a major contributor to the hyponeatremia and hypochloremia observed in these patients. These findings have important clinical implications. The strong association between poor glycemic control and AKI severity supports stringent glucose monitoring in hospitalized diabetics with renal dysfunction.

The high prevalence of hyperkalemia and metabolic acidosis in Group C emphasizes routine electrolyte and acid-base monitoring in this high-risk population. The progressive abnormalities from Group B to Group C suggest early identification and intervention may prevent or delay renal complications. However, the present study has several limitations. First, its cross-sectional design prevents causal inference between biochemical alterations and kidney injury progression. Second, participants were recruited from a single center, which may limit the generalizability of the findings. Third, although duration of diabetes was recorded and analyzed, other potentially important clinical factors such as medication use, hydration status, comorbid conditions, and baseline pre-admission renal function were not comprehensively evaluated. In conclusion, this study demonstrates progressive deterioration in glycemic control, renal function, and electrolyte balance across the spectrum from healthy controls to diabetic patients with AKI, with hyperkalemia and metabolic acidosis posing significant clinical risk, underscoring the importance of comprehensive metabolic monitoring and early intervention in this vulnerable population.

Conclusion

The present study demonstrated a progressive deterioration in glycemic control, renal function, electrolyte homeostasis, and acid–base balance from healthy individuals to diabetic patients, with the most severe abnormalities observed in diabetic patients with acute kidney injury. Patients with AKI exhibited significantly elevated fasting glucose, HbA1c, blood urea, serum creatinine, and potassium levels, accompanied by marked reductions in eGFR, sodium, chloride, and bicarbonate concentrations. The strong correlation between declining eGFR and worsening electrolyte disturbances highlights the critical role of renal dysfunction in the development of metabolic complications. Hyperkalemia and metabolic acidosis emerged as the most clinically significant abnormalities among diabetic patients with AKI. These findings emphasize the importance of routine monitoring of renal function and electrolyte parameters for the early identification and management of diabetic patients at increased risk of acute kidney injury and its associated complications.

References

Adrogué, H. J., & Madias, N. E. (2000). Hyponatremia. *N Engl J Med*, *342*(21), 1581-1589. https://doi.org/10.1056/nejm200005253422107

Almazmomi, M. A., Esmat, A., & Naeem, A. (2023). Acute Kidney Injury: Definition, Management, and Promising Therapeutic Target. *Cureus*, *15*(12), e51228. https://doi.org/10.7759/cureus.51228

Association, A. D. (2024). 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2024. *Diabetes Care*, *47*(Suppl 1), S20-s42. https://doi.org/10.2337/dc24-S002

Claure, R., & Bouchard, J. (2012). Acid-base and electrolyte abnormalities during renal support for acute kidney injury: recognition and management. *Blood purification*, *34*(2), 186-193.

Deabes, A. A., & Essa, A. (2024). Fluid and electrolyte imbalance in renal dysfunction. *Anaesthesia & Intensive Care Medicine*, *25*(5), 316-319. https://doi.org/https://doi.org/10.1016/j.mpaic.2024.03.006

Eisinger, F., Neumann, M., Wörn, M., Fritsche, A., Heyne, N., Peter, A., Birkenfeld, A. L., von Schwartzenberg, R. J., & Artunc, F. (2025). Comparison of GFR estimation in patients with diabetes mellitus using the EKFC and CKD-EPI equations. *J Nephrol*, *38*(2), 707-716. https://doi.org/10.1007/s40620-024-02202-4

Ertek, S., & Caglar, K. (2025). Underestimated reason of hyperkalemia in diabetic patients: type IV renal tubular acidosis- mini review [Mini Review]. *Frontiers*

in Clinical Diabetes and Healthcare, *Volume 6 - 2025*. https://doi.org/10.3389/fcdhc.2025.1570868

Hillier, T. A., Abbott, R. D., & Barrett, E. J. (1999). Hyponatremia: evaluating the correction factor for hyperglycemia. *Am J Med*, *106*(4), 399-403. https://doi.org/10.1016/s0002-9343(99)00055-8

Hoste, E. A., Kellum, J. A., Selby, N. M., Zarbock, A., Palevsky, P. M., Bagshaw, S. M., Goldstein, S. L., Cerdá, J., & Chawla, L. S. (2018). Global epidemiology and outcomes of acute kidney injury. *Nature Reviews Nephrology*, *14*(10), 607-625.

Ihim, A., Analike, R., Standhope, C., C, O., Asomugha, L., Obi, P., Kalu, A., Ugwu, M., & Nwobodo, E. (2019). Evaluation of estimated glomerular filtration rate (egfr), serum creatinine, urea and electrolytes profile levels in diabetic patients attending medical outpatient clinic in Nnamdi Azikiwe University teaching hospital, Nnewi, Anambra State, Nigeria. *International Journal of Clinical Biochemistry and Research*, *6*, 217-221. https://doi.org/10.18231/j.ijcbr.2019.048

Imenez Silva, P. H., & Mohebbi, N. (2022). Kidney metabolism and acid-base control: back to the basics. *Pflugers Arch*, *474*(8), 919-934. https://doi.org/10.1007/s00424-022-02696-6

Kaur, A., Sharma, G. S., & Kumbala, D. R. (2023). Acute kidney injury in diabetic patients: A narrative review. *Medicine (Baltimore)*, *102*(21), e33888. https://doi.org/10.1097/md.0000000000033888

Kellum, J. A., Romagnani, P., Ashuntantang, G., Ronco, C., Zarbock, A., & Anders, H. J. (2021). Acute kidney injury. *Nat Rev Dis Primers*, *7*(1), 52. https://doi.org/10.1038/s41572-021-00284-z

Khanduker, S., Ahmed, R., Khondker, F., Aharama, A., Afrose, N., & Chowdhury, M. (2017). Electrolyte disturbances in patients with diabetes mellitus. *Bangladesh Journal of Medical Biochemistry*, *10*(1), 27-35.

Khwaja, A. (2012). KDIGO clinical practice guidelines for acute kidney injury. *Nephron Clin Pract*, *120*(4), c179-184. https://doi.org/10.1159/000339789

Kog, Z. X., Lee, I. W. Z., & Teh, S. P. (2025). Rethinking Potassium Management in Chronic Kidney Disease—A Modern Approach. *Journal of Clinical Medicine*, *14*(24), 8917.

Kraut, J. A., & Madias, N. E. (2010). Metabolic acidosis: pathophysiology, diagnosis and management. *Nat Rev Nephrol*, *6*(5), 274-285. https://doi.org/10.1038/nrneph.2010.33

Kraut, J. A., & Madias, N. E. (2012). Treatment of acute metabolic acidosis: a pathophysiologic approach. *Nat Rev Nephrol*, *8*(10), 589-601. https://doi.org/10.1038/nrneph.2012.186

Kumar, M., Dev, S., Khalid, M. U., Siddenthi, S. M., Noman, M., John, C., Akubuiro, C., Haider, A., Rani, R., Kashif, M., Varrassi, G., Khatri, M., Kumar, S., & Mohamad, T. (2023). The Bidirectional Link Between Diabetes and Kidney Disease: Mechanisms and Management. *Cureus*, *15*(9), e45615. https://doi.org/10.7759/cureus.45615

Levey, A. S., Stevens, L. A., Schmid, C. H., Zhang, Y., Castro III, A. F., Feldman, H. I., Kusek, J. W., Eggers, P., Van Lente, F., & Greene, T. (2009). A new equation to estimate glomerular filtration rate. *Annals of internal medicine*, *150*(9), 604-612.

Liamis, G., Liberopoulos, E., Barkas, F., & Elisaf, M. (2014). Diabetes mellitus and electrolyte disorders. *World J Clin Cases*, *2*(10), 488-496. https://doi.org/10.12998/wjcc.v2.i10.488

Palmer, B. F. (2015). Regulation of Potassium Homeostasis. *Clin J Am Soc Nephrol*, *10*(6), 1050-1060. https://doi.org/10.2215/cjn.08580813

Salih, B. H., Ali, S. H., & Allehibi, K. I. (2019). Serum aldosterone levels in patients with diabetic nephropathy in relation to vascular calcification. *Iraqi Journal of Pharmaceutical Sciences*, *28*(1), 53-63.