Original Research Article

Study of Electrolyte Abnormalities and Their Relationship with Clinical Outcomes in Chronic Kidney Disease Patients

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Abstract

Background: Electrolyte disturbances are common in chronic kidney disease (CKD) and may contribute to adverse clinical outcomes. This study assessed the pattern of electrolyte abnormalities and their relationship with outcomes in CKD patients.

Materials and Methods: This prospective observational study included 150 adult CKD patients. Clinical characteristics, CKD stage, serum electrolytes, and relevant outcomes were assessed and analyzed.

Results: The mean age was 56.8 ± 13.4 years, and 60.7% were males. Overall, 74.7% had at least one electrolyte abnormality. Low bicarbonate (44.7%), hyperphosphatemia (40.7%), hypocalcemia (38.7%), hyponatremia (32.7%), and hyperkalemia (28.0%) were most frequent. Hyperkalemia increased from 9.8% in stage 3 to 44.4% in stage 5 CKD ($p<0.001$). Serum potassium ($r=-0.39$) and phosphate ($r=-0.45$) correlated negatively with eGFR, while bicarbonate correlated positively ($r=0.42$; all $p<0.001$). Electrolyte abnormalities, particularly hyperkalemia, were significantly associated with adverse clinical outcomes.

Conclusion: Electrolyte abnormalities were frequent and increased with advancing CKD. Regular monitoring and timely correction may help identify high-risk patients and improve clinical management.

Keywords: Chronic kidney disease; electrolyte abnormalities; hyperkalemia; eGFR; clinical outcomes.

Introduction

Chronic kidney disease (CKD) is a major global health problem characterized by progressive loss of kidney function and is associated with substantial morbidity, cardiovascular complications, and mortality[1]. The kidneys play a central role in maintaining fluid, electrolyte, and acid–base homeostasis through glomerular filtration, tubular reabsorption, and secretion. With progressive loss of functioning nephrons, these regulatory mechanisms become impaired, making electrolyte disturbances increasingly common, particularly in advanced CKD.[2]Electrolyte abnormalities in CKD are multifactorial and may result from reduced renal clearance, altered tubular handling, impaired water balance, dietary factors, comorbid illnesses, and medications.[2] Sodium and potassium disturbances are particularly important because of their potential neurological and cardiovascular consequences. Impaired urinary concentrating and diluting capacity predisposes patients to dysnatremia, whereas declining renal potassium excretion increases susceptibility to hyperkalemia.[2,3]Potassium abnormalities are of particular clinical importance because potassium is essential for normal cellular membrane potential and cardiac electrophysiology. Both hyperkalemia and hypokalemia can alter myocardial excitability and conduction, potentially resulting in serious cardiac arrhythmias.[4,5] Hyperkalemia becomes increasingly frequent with declining kidney function and may be further

aggravated by diabetes mellitus, cardiovascular disease, metabolic acidosis, acute deterioration in kidney function, and the use of renin–angiotensin–aldosterone system inhibitors.[6,7] Sarafidis et al. demonstrated a considerable burden of hyperkalemia among predialysis patients with advanced CKD and identified renal function and medication-related factors as important determinants.[8]Importantly, potassium abnormalities have been associated with adverse clinical outcomes. Nakhoul et al., in a cohort of 36,359 patients with stage 3–4 CKD, reported potassium concentrations below 3.5 mmol/L in approximately 3% and above 5.0 mmol/L in approximately 11%, with both abnormalities associated with increased mortality.[9] Similarly, Luo et al. demonstrated a U-shaped relationship between serum potassium concentration and mortality, major cardiovascular events, and hospitalization among patients with reduced kidney function.[10] A large CKD Prognosis Consortium meta-analysis further demonstrated that both low and high potassium concentrations were associated with adverse outcomes across different levels of kidney function.[11]Sodium abnormalities also have important prognostic implications in CKD. Impairment of free-water excretion and urinary concentrating ability predisposes patients to both hyponatremia and hypernatremia.[3] A systematic review and meta-analysis by Xie et al. demonstrated that dysnatremia, particularly hyponatremia, was associated with increased all-cause mortality among patients with CKD.[12]

CKD additionally affects calcium, phosphate, magnesium, chloride, and bicarbonate homeostasis.[2] Altered calcium–phosphate metabolism contributes to CKD-related mineral and bone abnormalities, while impaired renal acid excretion predisposes to metabolic acidosis. Abnormal serum bicarbonate concentrations have been associated with increased mortality and adverse renal outcomes in CKD.[13,14]Thus, electrolyte disturbances in CKD are not merely biochemical abnormalities but may reflect disease severity and contribute to cardiovascular complications, hospitalization, progression of kidney disease, requirement for dialysis, and mortality. Therefore, the present study was undertaken to determine the pattern and frequency of electrolyte abnormalities in patients with CKD and to evaluate their relationship with clinical outcomes, thereby facilitating early identification and appropriate management of high-risk patients.

Methodology:

This hospital-based prospective observational study was conducted in the Department of General Medicine. The study included patients diagnosed with chronic kidney disease (CKD).A total of 150 patients with CKD were enrolled consecutively during the study period after fulfilling the eligibility criteria. CKD was defined as abnormalities of kidney structure or function persisting for ≥3 months with implications for health.

Study duration- 3 months

Sample size:

The sample size was calculated based on the prevalence of electrolyte abnormality reported in the reference study by Packham et al. [15], using the formula:

$$n = \frac{Z_{1-\alpha/2}^2 \times p(1-p)}{d^2}$$

$$n = \frac{(1.96)^2 \times 0.40 \times 0.60}{(0.08)^2}$$

$$n = \frac{3.8416 \times 0.24}{0.0064} = 144.06$$

Thus, the minimum required sample size was approximately 145 patients. It was rounded up to 150 patients for the final study.

Inclusion Criteria

Patients aged ≥18 years with established CKD who provided written informed consent were included.

Exclusion Criteria

Patients with acute kidney injury without underlying CKD, acute gastrointestinal losses causing transient electrolyte disturbances, major endocrine disorders affecting electrolyte balance, and those unwilling to participate were excluded.

Data Collection

Demographic and clinical details including age, sex, duration and etiology of CKD, comorbidities, medication history, and relevant clinical findings were recorded using a structured proforma.

Laboratory Evaluation

Venous blood samples were collected under standard aseptic precautions. Investigations included complete blood count, serum urea, creatinine, sodium, potassium, chloride, bicarbonate, calcium, phosphate, and magnesium. Renal function was assessed using estimated glomerular filtration rate (eGFR), and patients were categorized according to CKD stage. Electrolyte abnormalities were defined according to the institutional laboratory reference ranges.

Clinical Outcomes

Patients were assessed for relevant clinical outcomes, including hospitalization, duration of hospital stay, cardiovascular or neurological complications, requirement for dialysis, worsening renal function, need for intensive care, and in-

hospital mortality. Electrolyte abnormalities were evaluated in relation to CKD severity and these clinical outcomes.

Statistical Analysis

Data were analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation or median (interquartile range), while categorical variables were presented as frequency and percentage. Normality was assessed using the Shapiro–Wilk test. Independent t-test/Mann–Whitney U test and chi-square/Fisher's exact test were used as appropriate. Multivariable logistic regression was used to identify factors independently associated with adverse outcomes. A p-value <0.05 was considered statistically significant.

Results:

A total of 150 CKD patients were included, with a mean age of 56.8 ± 13.4 years; 60.7% were males. Hypertension (77.3%) and diabetes mellitus (54.7%) were the major comorbidities. Mean serum creatinine was 4.62 ± 2.71 mg/dL and eGFR was 21.8 ± 14.7 mL/min/1.73 m². Stage 5 CKD was most common (42.0%), followed by stage 4 (30.7%) and stage 3 (27.3%) (Table 1).

The mean serum sodium, potassium, bicarbonate, corrected calcium, and phosphate levels were 136.1 ± 5.8 mEq/L, 4.82 ± 0.89 mEq/L, 20.8 ± 4.7 mEq/L, 8.42 ± 0.83 mg/dL, and 4.91 ± 1.42 mg/dL, respectively (Table 2). Overall, 74.7% of patients had at least one electrolyte abnormality. Low bicarbonate (44.7%), hyperphosphatemia (40.7%), hypocalcemia (38.7%), hyponatremia (32.7%), and hyperkalemia (28.0%) were the most frequent abnormalities (Table 3).

The prevalence of major electrolyte abnormalities increased significantly with advancing CKD stage (Table 4, Figure 1). From stage 3 to stage 5, hyponatremia increased from 14.6% to 47.6% (p=0.002), hyperkalemia from 9.8% to 44.4% (p<0.001), hypocalcemia from 19.5% to 54.0% (p=0.002), hyperphosphatemia from 17.1% to 60.3% (p<0.001), and low bicarbonate from 22.0% to 61.9% (p<0.001).

Electrolyte abnormalities were significantly associated with adverse clinical outcomes (Table 5, Figure 2). Adverse outcomes occurred in 59.2% of patients with hyponatremia, 66.7% with hyperkalemia, 53.4% with hypocalcemia, 57.4% with hyperphosphatemia, and 58.2% with low bicarbonate (all p<0.05).

eGFR correlated positively with sodium (r=0.24), calcium (r=0.34), and bicarbonate (r=0.42) and negatively with potassium (r=−0.39) and phosphate (r=−0.45). Potassium and phosphate correlated positively with length of hospital stay, whereas bicarbonate showed a negative correlation (Table 6, Figure 3). Overall, worsening renal function was associated with increasing electrolyte disturbances and poorer clinical outcomes.

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants (N = 150)

Parameter	Result
Age (years), mean ± SD	56.8 ± 13.4
Male	91 (60.7%)
Female	59 (39.3%)
Diabetes mellitus	82 (54.7%)
Hypertension	116 (77.3%)
Cardiovascular disease	38 (25.3%)
Duration of CKD (years), median (IQR)	4.0 (2.0–7.0)
Serum creatinine (mg/dL), mean ± SD	4.62 ± 2.71
eGFR (mL/min/1.73 m²), mean ± SD	21.8 ± 14.7
CKD stage 3	41 (27.3%)
CKD stage 4	46 (30.7%)
CKD stage 5	63 (42.0%)

Table 2. Serum Electrolyte Profile of Study Participants (N = 150)

Parameter	Mean ± SD	Reference range
Sodium (mEq/L)	136.1 ± 5.8	135–145
Potassium (mEq/L)	4.82 ± 0.89	3.5–5.0
Chloride (mEq/L)	101.2 ± 6.1	98–106
Bicarbonate (mEq/L)	20.8 ± 4.7	22–29
Corrected calcium (mg/dL)	8.42 ± 0.83	8.5–10.5
Phosphate (mg/dL)	4.91 ± 1.42	2.5–4.5
Magnesium (mg/dL)	2.18 ± 0.46	1.7–2.4

Table 3. Frequency and Pattern of Electrolyte Abnormalities (N = 150)

Electrolyte abnormality	n (%)
Any electrolyte abnormality	112 (74.7%)
Hyponatremia	49 (32.7%)
Hypernatremia	7 (4.7%)
Hypokalemia	14 (9.3%)
Hyperkalemia	42 (28.0%)
Hypochloremia	31 (20.7%)
Hyperchloremia	12 (8.0%)
Hypocalcemia	58 (38.7%)
Hypercalcemia	5 (3.3%)
Hypophosphatemia	8 (5.3%)
Hyperphosphatemia	61 (40.7%)
Hypomagnesemia	12 (8.0%)
Hypermagnesemia	20 (13.3%)
Low bicarbonate (<22 mEq/L)	67 (44.7%)

Table 4. Major Electrolyte Abnormalities According to CKD Stage

Abnormality	Stage 3 (n=41)	Stage 4 (n=46)	Stage 5 (n=63)	p-value
Hyponatremia	6 (14.6 %)	13 (28.3 %)	30 (47.6 %)	0.002
Hyperkalemia	4 (9.8%)	10 (21.7 %)	28 (44.4 %)	<0.001
Hypocalcemia	8 (19.5 %)	16 (34.8 %)	34 (54.0 %)	0.002
Hyperphosphatemia	7 (17.1 %)	16 (34.8 %)	38 (60.3 %)	<0.001
Low bicarbonate	9 (22.0 %)	19 (41.3 %)	39 (61.9 %)	<0.001
Any electrolyte abnormality	22 (53.7 %)	34 (73.9 %)	56 (88.9 %)	<0.001

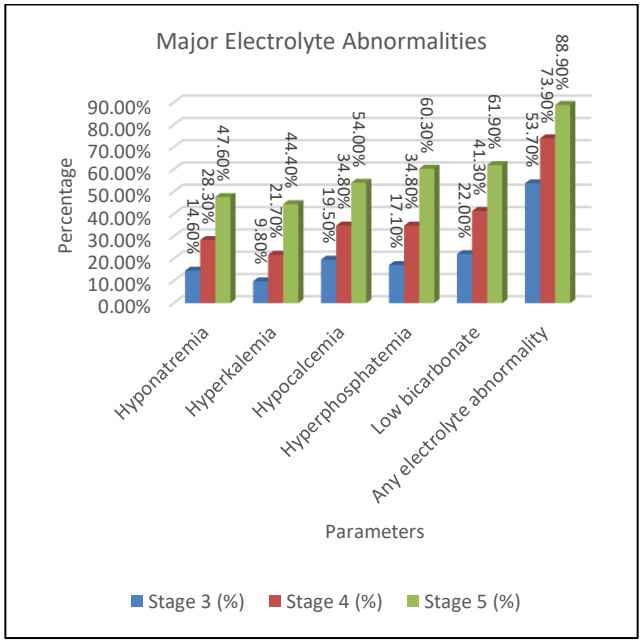

Figure 1. Major Electrolyte Abnormalities According to CKD Stage

Table 5. Association of Electrolyte Abnormalities With Adverse Clinical Outcomes

Electrolyte abnormality	Patients, n	Adverse outcome, n (%)	No adverse outcome, n (%)	p-value
Hyponatremia	49	29 (59.2 %)	20 (40.8 %)	0.004
Hyperkalemia	42	28 (66.7 %)	14 (33.3 %)	<0.001
Hypocalcemia	58	31 (53.4 %)	27 (46.6 %)	0.018
Hyperphosphatemia	61	35 (57.4 %)	26 (42.6 %)	0.006
Low bicarbonate	67	39 (58.2 %)	28 (41.8 %)	0.002

Any electrolyte abnormality	112	57 (50.9 %)	55 (49.1 %)	<0.0 01

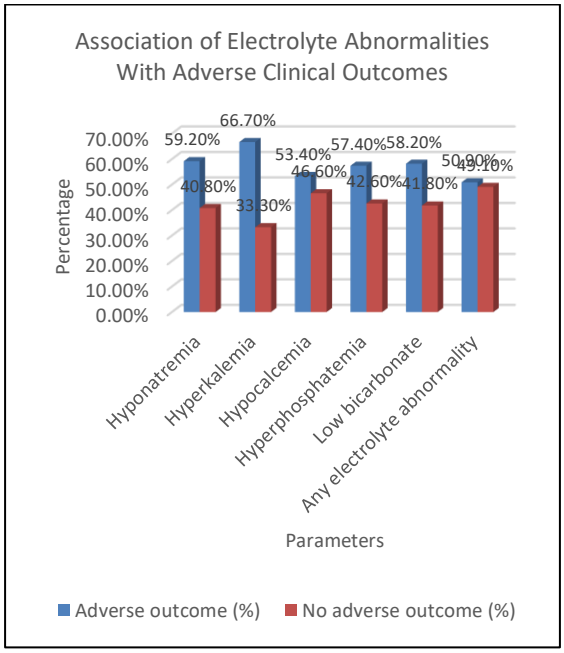

Figure 2. Association of Electrolyte Abnormalities With Adverse Clinical Outcomes

Table 6. Correlation of Serum Electrolytes With Renal Function and Clinical Outcome Parameters

Parameter	Correlated variable	Correlation coefficient (r/ρ)	p-value
Serum sodium	eGFR	+0.24	0.003
Serum potassium	eGFR	−0.39	<0.001
Serum calcium	eGFR	+0.34	<0.001
Serum phosphate	eGFR	−0.45	<0.001
Serum bicarbonate	eGFR	+0.42	<0.001
Serum potassium	Length of hospital stay	+0.28	0.001
Serum phosphate	Length of hospital stay	+0.26	0.002
Serum bicarbonate	Length of hospital stay	−0.31	<0.001

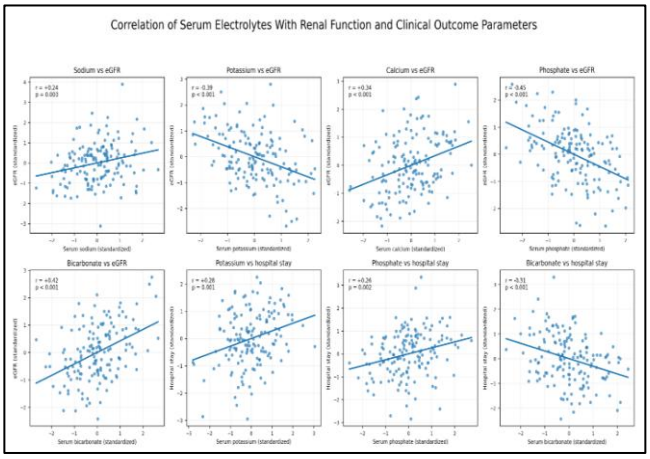

Figure 3. Correlation of Serum Electrolytes With Renal Function and Clinical Outcome Parameters

Discussion

The present study included 150 CKD patients with a mean age of 56.8 ± 13.4 years; 60.7% were males. Hypertension (77.3%) and diabetes mellitus (54.7%) were the predominant comorbidities. Mean serum creatinine was 4.62 ± 2.71 mg/dL and eGFR was 21.8 ± 14.7 mL/min/1.73 m², with 42.0% having stage 5 CKD. Similarly, Packham et al. [15] studied 753 hyperkalemic patients, of whom approximately 60% were male, 59.9% had diabetes, and 74.5% had eGFR <60 mL/min/1.73 m², demonstrating the substantial burden of renal impairment and associated comorbidities in patients prone to electrolyte disturbances.In our study, 74.7% of patients had at least one electrolyte abnormality. Low bicarbonate was most common (44.7%), followed by hyperphosphatemia (40.7%), hypocalcemia (38.7%), hyponatremia (32.7%), and hyperkalemia (28.0%). Mean bicarbonate was low (20.8 ± 4.7 mEq/L), corrected calcium was 8.42 ± 0.83 mg/dL, and phosphate was elevated (4.91 ± 1.42 mg/dL). These findings demonstrate the progressive impairment of electrolyte, mineral, and acid–base homeostasis accompanying CKD.

Hyperkalemia was present in 28.0% of our patients and increased significantly from 9.8% in stage 3 to 21.7% in stage 4 and 44.4% in stage 5 CKD (p<0.001). Serum potassium was inversely correlated with eGFR (r=−0.39, p<0.001). Similarly, Miao et al. [16], in a post hoc analysis of the RENAAL trial, found that increased serum potassium was associated with poorer renal outcomes and that the potassium-raising effect of

losartan partly offset its renoprotective effect. These findings support the clinical importance of monitoring potassium as renal function declines.

Pitt et al. [17] studied 105 high-risk heart failure patients in the PEARL-HF trial and reported that RLY5016 produced a 0.45 mEq/L greater reduction in serum potassium than placebo ($p<0.001$). Hyperkalemia occurred in 7.3% versus 24.5% of RLY5016 and placebo recipients, respectively, while 91% versus 74% could be titrated to spironolactone 50 mg/day. Buysse et al. [18] subsequently highlighted these PEARL-HF findings and concluded that RLY5016 could prevent hyperkalemia and facilitate continuation of potassium-raising cardiovascular therapy. These observations are relevant to our 28.0% prevalence of hyperkalemia, particularly the 44.4% prevalence in stage 5 CKD.

Weir et al. (2015) [19] studied 237 CKD patients with hyperkalemia receiving RAAS inhibitors and found that patiromer reduced serum potassium by 1.01 mmol/L, with 76% achieving the target potassium range. During randomized withdrawal, recurrent hyperkalemia occurred in 60% of placebo-treated patients compared with 15% continuing patiromer ($p<0.001$). These findings complement our observation that hyperkalemia becomes progressively more common with worsening CKD and is inversely related to eGFR.

Kosiborod et al. [20] evaluated 258 hyperkalemic patients in the HARMONIZE trial. Sodium zirconium cyclosilicate reduced mean potassium from approximately 5.6 to 4.5 mEq/L within 48 hours, with 84% achieving normokalemia within 24 hours and 98% within 48 hours. During maintenance, mean potassium was 4.8, 4.5, and 4.4 mEq/L with 5, 10, and 15 g doses, respectively, compared with 5.1 mEq/L with placebo. These results demonstrate the clinical relevance and treatability of hyperkalemia, which affected 28.0% of our CKD population.

Packham et al. [15] evaluated sodium zirconium cyclosilicate in 753 patients with a baseline potassium of approximately 5.3 mmol/L. At 48 hours, potassium decreased to approximately 4.9, 4.8, and 4.6 mmol/L with 2.5, 5, and 10 g doses, respectively. Importantly, 74.5% had eGFR <60 mL/min/1.73 m^2, supporting renal impairment as an important setting for hyperkalemia. This corresponds with our significant inverse relationship between potassium and eGFR ($r=-0.39$, $p<0.001$).

The frequency of any electrolyte abnormality increased significantly from 53.7% in stage 3 to 73.9% in stage 4 and 88.9% in stage 5 ($p<0.001$). Hyponatremia increased from 14.6% to 47.6%, hyperkalemia from 9.8% to 44.4%, hypocalcemia from 19.5% to 54.0%, hyperphosphatemia from 17.1% to 60.3%, and low bicarbonate from 22.0% to 61.9% between stages 3 and 5. This progressive pattern reflects declining renal capacity for electrolyte excretion, mineral regulation, and acid–base homeostasis.

Worsening renal function was significantly associated with multiple electrolyte disturbances. eGFR correlated positively with sodium ($r=+0.24$, $p=0.003$), calcium ($r=+0.34$, $p<0.001$), and bicarbonate ($r=+0.42$, $p<0.001$) and inversely with potassium ($r=-0.39$, $p<0.001$) and phosphate ($r=-0.45$, $p<0.001$). The potassium relationship agrees with Miao et al. [16] and is further supported by the CKD populations studied in OPAL-HK and the sodium zirconium cyclosilicate trials.

Adverse outcomes occurred in 59.2% of patients with hyponatremia, 66.7% with hyperkalemia, 53.4% with hypocalcemia, 57.4% with hyperphosphatemia, and 58.2% with low bicarbonate, with all associations being statistically significant. Any electrolyte abnormality was associated with an adverse outcome in 50.9% ($p<0.001$). Potassium and phosphate also correlated positively with hospital stay ($r=+0.28$, $p=0.001$ and $r=+0.26$, $p=0.002$, respectively), whereas bicarbonate correlated inversely ($r=-0.31$, $p<0.001$). The particularly high adverse-outcome rate associated with hyperkalemia is consistent with Miao et al. [16], who demonstrated poorer renal outcomes with increased serum potassium.

Conclusion

Electrolyte abnormalities were common among patients with CKD and increased significantly with advancing disease severity. Hyperkalemia, hyponatremia, hypocalcemia, hyperphosphatemia, and low bicarbonate were associated with adverse clinical outcomes, with hyperkalemia showing particular prognostic importance. Regular electrolyte monitoring and timely correction of abnormalities may facilitate early risk identification and improve clinical management of CKD patients.

Limitations

The study was limited by its relatively small sample size and single-center design, which may restrict the generalizability of the findings. In addition, the observational design precluded establishment of a causal relationship between electrolyte abnormalities and clinical outcomes.

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