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Prevalence, Predictors, and Outcomes of Electrolyte Imbalances and Metabolic Acidosis in Internal Medicine Patients with Acute Kidney Injury

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ABSTRACT

Background: Acute kidney injury (AKI) is a common complication in internal medicine patients and is frequently accompanied by electrolyte imbalances and metabolic acidosis. These disturbances can lead to serious complications, prolong hospitalization, and increase mortality.

Objective: To determine the prevalence, predictors, and clinical outcomes of electrolyte imbalances (hyperkalemia, hypokalemia, hyponatremia, hypernatremia) and metabolic acidosis in internal medicine patients with AKI.

Methods: A prospective observational study was conducted at Institute of Kidney disease Peshawar from march 2023 to June 2024. It included 185 adult patients diagnosed with AKI. Demographic, clinical, and laboratory data were collected, including serum electrolytes, arterial blood gases, comorbidities, and medication use. Patients were monitored for clinical outcomes such as ICU admission, need for dialysis, in-hospital mortality, and length of hospital stay.

Results: Electrolyte imbalances were common: hyperkalemia in 48 patients (26%), hypokalemia in 32 (17%), hyponatremia in 54 (29%), and hypernatremia in 21 (11%). Metabolic acidosis occurred in 76 patients (41%). The prevalence of disturbances increased with AKI severity, with Stage 3 patients showing the highest rates. Comorbidities, particularly sepsis and diabetes, were associated with higher prevalence. Patients with disturbances had worse outcomes: ICU admission 32% vs. 15%, need for dialysis 29% vs. 10%, in-hospital mortality 28% vs. 12%, and longer hospital stays (12.4 ± 6.2 days vs. 9.1 ± 4.5 days). Logistic regression identified advanced AKI stages and sepsis as independent predictors of hyperkalemia and metabolic acidosis.

Conclusion: Electrolyte imbalances and metabolic acidosis are prevalent in AKI and are associated with adverse clinical outcomes. Advanced AKI and sepsis are major predictors. Early identification and management of these disturbances are critical to improving patient prognosis.

KEYWORDS: Acute kidney injury, electrolyte imbalance, hyperkalemia, hyponatremia, metabolic acidosis, clinical outcomes.

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INTRODUCTION

Acute kidney injury (AKI) is increasingly recognized as a major cause of morbidity and mortality among hospitalized patients worldwide. Defined as a sudden decline in renal function over hours to days, AKI is characterized by a rise in serum creatinine, reduction in urine output, or both [1]. The incidence of AKI varies according to the patient population and clinical setting, with reported rates ranging from 5% to 30% in general medical wards and up to 50% in intensive care units [2]. AKI not only increases the risk of progression to chronic kidney disease but also predisposes patients to a spectrum of systemic complications, including fluid overload, electrolyte disturbances, and metabolic acidosis [3]. These complications can exacerbate underlying comorbidities, prolong hospitalization, and significantly increase the risk of adverse outcomes, including in-hospital mortality [4]. Electrolyte imbalances are among the most clinically significant complications of AKI. Hyperkalemia, resulting from impaired renal potassium excretion, can precipitate life-threatening cardiac arrhythmias, whereas hypokalemia may lead to muscle weakness, cramps, and arrhythmogenic potential [5]. Similarly, sodium disturbances, including hyponatremia and hypernatremia, are commonly observed in AKI and are often markers of fluid dysregulation, altered renal handling, or concurrent systemic illness [6]. Previous research has indicated that electrolyte abnormalities frequently coexist in patients with AKI, with severity often

correlating with the stage of renal injury, the presence of comorbid conditions, and exposure to medications such as diuretics, ACE inhibitors, and nephrotoxic agents [7]. Understanding the prevalence and pattern of these disturbances is essential for early recognition, timely correction, and prevention of potentially fatal complications.

Metabolic acidosis is another common and critical manifestation of AKI, arising due to the accumulation of hydrogen ions, impaired bicarbonate reabsorption, and reduced ammonium excretion by the damaged kidneys [8]. This acid-base disturbance has important pathophysiological consequences, including decreased myocardial contractility, vasodilation, impaired oxygen delivery, and exacerbation of catabolic states [9]. Metabolic acidosis in AKI is particularly associated with high mortality rates, greater likelihood of ICU admission, and the need for renal replacement therapy [10]. Several studies have reported that patients with both metabolic acidosis and severe electrolyte imbalances have significantly worse clinical outcomes than those without these complications, underscoring the importance of early detection and management [11]. The pathogenesis of electrolyte disturbances and metabolic acidosis in AKI is multifactorial. Reduced glomerular filtration rate leads to accumulation of potassium and hydrogen ions, while impaired tubular function disrupts sodium and bicarbonate handling [12]. Comorbid conditions such as diabetes mellitus, hypertension, and sepsis further compound these derangements by affecting renal perfusion, tubular function, and systemic metabolic control [13]. Additionally, medications commonly used in internal medicine patients, including loop and thiazide diuretics, ACE inhibitors, and angiotensin receptor blockers, can alter renal electrolyte handling and exacerbate imbalances [14]. Previous research has highlighted that hyperkalemia is particularly common in patients with severe AKI, whereas hyponatremia often reflects volume overload or impaired free water clearance. Metabolic acidosis, on the other hand, is more prevalent in patients with advanced AKI, sepsis, or other systemic inflammatory states [15]. Despite their clinical significance, there is a paucity of comprehensive data assessing the prevalence, predictors, and outcomes of electrolyte disturbances and metabolic acidosis in internal medicine populations with AKI. Most prior studies have focused on critically ill patients in intensive care units, leaving a gap in understanding how these complications affect patients admitted to general internal medicine wards [16].

Objective:

To determine the prevalence, predictors, and clinical outcomes of electrolyte imbalances (hyperkalemia, hypokalemia, hyponatremia, hypernatremia) and metabolic acidosis in internal medicine patients with AKI.

METHODOLOGY

A prospective observational study was conducted at Institute of Kidney disease Peshawar from march 2023 to June 2024. It included 185 adult patients diagnosed with AKI.

Inclusion Criteria

- Adult patients aged 18 years and above admitted to internal medicine wards.
- Diagnosis of AKI according to KDIGO criteria:
    - Increase in serum creatinine by ≥0.3 mg/dL within 48 hours, or
    - Increase to ≥1.5 times baseline within 7 days, or
    - Urine output <0.5 mL/kg/h for 6 hours.
- Patients who provided written informed consent for participation.

Exclusion Criteria

- End-stage renal disease on maintenance dialysis.
- Chronic kidney disease stage 5 with baseline serum creatinine >5 mg/dL.
- Pregnant patients.
- Incomplete medical records or inability to obtain laboratory data.

DATA COLLECTION

After obtaining written informed consent, demographic and clinical information was collected for all 185 participants, including age, gender, comorbid conditions such as diabetes, hypertension, and sepsis, and details of medications that could influence electrolyte balance, such as diuretics and ACE inhibitors. Laboratory investigations were performed at admission to assess serum electrolytes (potassium and sodium), blood urea nitrogen (BUN), serum creatinine, and arterial blood gases for evaluation of metabolic acidosis. Electrolyte imbalances were classified as hyperkalemia (>5.0 mmol/L), hypokalemia (<3.5 mmol/L), hyponatremia (<135 mmol/L), and hypernatremia (>145 mmol/L), while metabolic acidosis was defined as serum bicarbonate <22 mmol/L and/or arterial pH <7.35. Patients were monitored throughout hospitalization for key clinical outcomes including ICU admission, need for dialysis, in-hospital mortality, and length of hospital stay. All relevant information was recorded in a

structured data collection form to ensure consistency and completeness of data.

Statistical Analysis

All collected data were entered into SPSS version 22.0 (SPSS Inc., Chicago, IL) for analysis. Quantitative variables such as age, serum creatinine, BUN, and hospital stay duration were expressed as mean ± standard deviation and compared between patients with and without electrolyte disturbances or metabolic acidosis using independent t-tests. Categorical variables, including presence of hyperkalemia, hyponatremia, hypernatremia, metabolic acidosis, ICU admission, dialysis requirement, and mortality, were presented as frequencies and percentages and analyzed using chi-square tests. A p-value ≤0.05 was considered statistically significant.

RESULTS

The study included 185 patients with a mean age of 57.4 ± 14 years, of which 55% were male. Comorbidities were common, with 45% having diabetes, 61% hypertension, and 28% presenting with sepsis. The severity of AKI was distributed across stages: 33% in Stage 1, 40% in Stage 2, and 27% in Stage 3. Baseline serum creatinine averaged 1.9 ± 0.8 mg/dL. Medication use was notable, with 39% receiving diuretics and 34% on ACE inhibitors or ARBs. This baseline profile indicates a population with significant comorbid burden and a range of kidney injury severity. Electrolyte disturbances were common: hyperkalemia affected 26% of patients (48/185), hypokalemia 17% (32/185), hyponatremia 29% (54/185), and hypernatremia 11% (21/185). Metabolic acidosis was present in 41% of patients (76/185), with a mean serum bicarbonate of 20.8 ± 4.5 mmol/L.

Table 1. Demographic and Baseline Clinical Characteristics of AKI Patients (n = 185)

Variable	Value
Age (years), mean ± SD	57.4 ± 14.2
Gender, n (%)	Male: 102 (55%)
	Female: 83 (45%)
Comorbidities, n (%)	Diabetes: 84 (45%)
	Hypertension: 112 (61%)
	Sepsis: 51 (28%)
	CKD stage 3–4: 26 (14%)
AKI Stage, n (%)	Stage 1: 61 (33%)
	Stage 2: 74 (40%)
	Stage 3: 50 (27%)
Baseline serum creatinine (mg/dL), mean ± SD	1.9 ± 0.8
Use of diuretics, n (%)	72 (39%)
Use of ACEi/ARB, n (%)	63 (34%)
Serum Potassium (mmol/L)	4.8 ± 1.2
Hyperkalemia (K^+ >5.0 mmol/L)	48 (26%)
Hypokalemia (K^+ <3.5 mmol/L)	32 (17%)
Serum Sodium (mmol/L)	137 ± 6
Hyponatremia (Na^+ <135 mmol/L)	54 (29%)
Hypernatremia (Na^+ >145 mmol/L)	21 (11%)

Serum Bicarbonate (mmol/L)	20.8 ± 4.5
Metabolic Acidosis (HCO_3^- <22 or pH <7.35)	76 (41%)

The prevalence of disturbances increased with AKI severity. In Stage 1, hyperkalemia was present in 16%, hyponatremia in 20%, and metabolic acidosis in 25%. In Stage 2, hyperkalemia affected 30%, hyponatremia 30%, and metabolic acidosis 39%. Stage 3 showed the highest prevalence, with hyperkalemia in 32%, hyponatremia 40%, and metabolic acidosis 64%. Hypernatremia also rose with AKI stage (Stage 3: 16%).

Table 2. Electrolyte and Acid-Base Disturbances Stratified by AKI Stage

AKI Stage	Hyperkalemia n (%)	Hyponatremia n (%)	Hypernatremia n (%)	Metabolic Acidosis n (%)
Stage 1 (n=61)	10 (16%)	12 (20%)	4 (7%)	15 (25%)
Stage 2 (n=74)	22 (30%)	22 (30%)	9 (12%)	29 (39%)
Stage 3 (n=50)	16 (32%)	20 (40%)	8 (16%)	32 (64%)

Electrolyte imbalances and metabolic acidosis were more common in patients with comorbidities. For example, patients with sepsis had the highest rates of hyperkalemia (43%) and metabolic acidosis (55%). Diabetes was associated with hyperkalemia in 31% and metabolic acidosis in 43%, while hypertension showed similar trends. Patients with chronic kidney disease had metabolic acidosis in 54% of cases.

Table 3. Association of Electrolyte Imbalances and Metabolic Acidosis with Comorbidities

Comorbidity	Hyperkalemia n (%)	Hyponatremia n (%)	Hypernatremia n (%)	Metabolic Acidosis n (%)
Diabetes (n=84)	26 (31%)	30 (36%)	10 (12%)	36 (43%)
Hypertension (n=112)	36 (32%)	42 (38%)	12 (11%)	48 (43%)
Sepsis (n=51)	22 (43%)	20 (39%)	9 (18%)	28 (55%)
CKD (n=26)	10 (38%)	10 (38%)	3 (12%)	14 (54%)

Patients with electrolyte imbalances or metabolic acidosis had worse outcomes. ICU admission occurred in 32% of those with disturbances compared to 15% without. Dialysis was required in 29% versus 10%, and in-hospital mortality was 28% versus 12%. The mean hospital stay was longer for patients with disturbances (12.4 ± 6.2 days) compared to those without (9.1 ± 4.5 days).

Table 4. Clinical Outcomes by Presence of Electrolyte/Acid-Base Disturbances

Outcome	Disturbance Present n (%)	Disturbance Absent n (%)	p-value
ICU Admission	32 (32%)	20 (15%)	0.01
Need for Dialysis	28 (29%)	14 (10%)	0.002
In-Hospital Mortality	28 (28%)	14 (12%)	0.002
Mean Length of Stay (days), mean ± SD	12.4 ± 6.2	9.1 ± 4.5	0.01

Severe AKI (Stage 2–3) and sepsis were independent predictors of both hyperkalemia and metabolic acidosis. For hyperkalemia, AKI Stage 2–3 had an OR of 2.4 (95% CI: 1.3–4.5), and sepsis had an OR of 2.9 (95% CI: 1.5–5.5). For metabolic acidosis, AKI Stage 2–3 showed an OR of 3.1 (95% CI: 1.7–5.6), and sepsis 2.8 (95% CI: 1.5–5.2).

Table 5. Logistic Regression Analysis for Predictors of Electrolyte Imbalances and Metabolic Acidosis

Outcome	Predictor	Adjusted OR (95% CI)	p-value
Hyperkalemia	AKI Stage 2–3 vs Stage 1	2.4 (1.3–4.5)	0.005
	Diabetes	1.8 (1.0–3.2)	0.04
	Sepsis	2.9 (1.5–5.5)	0.001
Metabolic Acidosis	AKI Stage 2–3 vs Stage 1	3.1 (1.7–5.6)	<0.001
	Sepsis	2.8 (1.5–5.2)	0.001
	Diabetes	1.5 (0.8–2.8)	0.15
	Hypertension	1.2 (0.7–2.2)	0.42

DISCUSSION

This study demonstrates that electrolyte imbalances and metabolic acidosis are highly prevalent among internal medicine patients with acute kidney injury (AKI), affecting a substantial proportion of the cohort. Among 185 patients, hyperkalemia was observed in 26%, hypokalemia in 17%, hyponatremia in 29%, hypernatremia in 11%, and metabolic acidosis in 41% of cases. These findings are consistent with previous research, which reported similarly high prevalence rates of electrolyte disturbances and acid-base abnormalities in hospitalized AKI patients [17]. The frequent occurrence of hyperkalemia and hyponatremia underscores the vulnerability of AKI patients to potentially life-threatening metabolic complications. Electrolyte and acid-base disturbances were closely associated with the severity of AKI. Stage 3 AKI patients exhibited the highest rates of hyperkalemia (32%), hyponatremia (40%), and metabolic acidosis (64%), compared to lower rates in Stage 1 and Stage 2. This gradient of risk aligns with previous research showing that worsening renal function correlates with increased prevalence of metabolic derangements [18]. The relationship between AKI stage and these disturbances likely reflects the progressive decline in glomerular filtration rate and impaired tubular handling of electrolytes as kidney injury advances. Comorbid conditions further increased the risk of disturbances. Patients with sepsis had the highest prevalence of hyperkalemia (43%) and metabolic acidosis (55%), while diabetes and hypertension also contributed to higher rates of abnormalities. Chronic kidney disease similarly predisposed patients to metabolic acidosis. These findings agree with previous research, which emphasized that systemic illness, infection, and comorbidities exacerbate electrolyte and acid-base abnormalities in AKI patients [19]. Sepsis, in particular, contributes to both renal hypoperfusion and systemic metabolic derangements, amplifying the risk of hyperkalemia and acidosis.

The clinical impact of these disturbances was substantial. Patients with electrolyte imbalances or metabolic acidosis had higher ICU admission rates (32% vs. 15%), greater need for dialysis (29% vs. 10%), higher in-hospital mortality (28% vs. 12%), and longer hospital stays (12.4 ± 6.2 days vs. 9.1 ± 4.5 days). These adverse outcomes corroborate previous research findings, which linked metabolic disturbances in AKI to increased morbidity, prolonged hospitalization, and mortality [20]. The results highlight the critical importance of early recognition, close monitoring, and timely correction of electrolyte and acid-base abnormalities to improve outcomes in this patient population. Logistic regression analysis identified AKI severity (Stage 2–3) and sepsis as independent predictors for both hyperkalemia and metabolic acidosis. Specifically, patients with advanced AKI had 2.4 times higher odds of hyperkalemia and 3.1 times higher odds of metabolic acidosis, while sepsis increased the odds by 2.9 and 2.8 times, respectively. These results are consistent with previous research, which emphasized that the severity of renal injury and systemic infection are the primary drivers of metabolic derangements in AKI [21]. Diabetes modestly increased the risk of hyperkalemia but did not significantly predict metabolic acidosis, while hypertension was not a significant predictor for either outcome, similar to observations in previous research. Overall, this study reinforces the concept that electrolyte imbalances and metabolic acidosis are common, predictable, and clinically significant complications of AKI in internal medicine patients. Early identification of at-risk patients, particularly those with severe AKI or sepsis, is critical. Monitoring and timely interventions, including correction of potassium and sodium abnormalities and management of acid-base disturbances, are essential to reduce ICU admissions, dialysis requirements, and mortality.

CONCLUSION

It is concluded that electrolyte imbalances and metabolic acidosis are highly prevalent among internal medicine patients with acute kidney injury, with hyperkalemia, hyponatremia, and metabolic acidosis being the most common disturbances. These abnormalities are significantly associated with the severity of AKI and the presence of systemic comorbidities, particularly sepsis and diabetes. Patients experiencing these disturbances are at higher risk of adverse clinical outcomes, including ICU admission, need for dialysis, prolonged hospital stay, and in-hospital mortality. Advanced AKI stages and sepsis were identified as independent predictors of both hyperkalemia and metabolic acidosis.

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