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Original Research Article

COMPARATIVE EVALUATION OF CLINICAL PROFILE, ELECTROLYTE ABNORMALITIES, AND HOSPITAL OUTCOMES IN PATIENTS WITH ACUTE KIDNEY INJURY VERSUS CHRONIC KIDNEY DISEASE

Rajesh Sharma[1], Shah Parth Abhijeet[2]

[1]Professor, Department of General Medicine, SIMS, Hapur Uttar Pradesh India
[2]Junior Resident 1, Department of General Medicine, SIMS, Hapur Uttar Pradesh India

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Corresponding Author:
Dr. Shah Parth Abhijeet,
Junior Resident 1, Department of General Medicine, SIMS, Hapur Uttar Pradesh India
Email: pspshah100@gmail.com

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ABSTRACT

Background: Acute kidney injury (AKI) and chronic kidney disease (CKD) are important causes of renal dysfunction associated with electrolyte disturbances, metabolic abnormalities, and adverse hospital outcomes. Comparative evaluation of their clinical and biochemical characteristics may facilitate early diagnosis and appropriate management. The aim is to compare the clinical profile, electrolyte abnormalities, biochemical parameters, and hospital outcomes among patients with AKI and CKD admitted to a tertiary care hospital.

Materials and Methods: This hospital-based, prospective observational comparative study included 114 patients, divided into two groups: AKI (n=57) and CKD (n=57). Demographic characteristics, comorbidities, clinical manifestations, hematological and biochemical parameters, electrolyte abnormalities, and hospital outcomes were evaluated. Statistical analysis was performed using SPSS version 28.0, with $p<0.05$ considered statistically significant.

Results: CKD patients were significantly older than AKI patients (58.67 ± 12.31 versus 51.84 ± 14.22 years; p=0.007). Diabetes mellitus (54.39% versus 28.07%) and hypertension (73.68% versus 36.84%) were significantly more prevalent in CKD patients. Oliguria (66.67%) and dehydration (47.37%) were more frequent in AKI patients. CKD patients demonstrated significantly lower hemoglobin and serum calcium levels, with higher serum creatinine and phosphate concentrations. Hypocalcemia (68.42% versus 40.35%; p=0.003), hyperphosphatemia (73.68% versus 45.61%; p=0.002), and metabolic acidosis (70.18% versus 45.61%; p=0.008) were significantly more frequent in CKD patients. ICU admission was higher in AKI patients (42.11% versus 22.81%; p=0.028), whereas hospital stay was significantly longer in CKD patients (9.16 ± 3.36 versus 7.23 ± 2.85 days; p=0.001).

Conclusion: AKI was associated with greater acute clinical instability and critical care requirements, whereas CKD demonstrated more pronounced metabolic abnormalities and prolonged hospitalization. Early recognition and appropriate management of renal dysfunction and electrolyte disturbances remain essential for improving hospital outcomes.

Keywords: Acute Kidney Injury; Chronic Kidney Disease; Electrolyte Abnormalities; Metabolic Acidosis.

INTRODUCTION

Kidney diseases represent a major global public health challenge and contribute substantially to morbidity, mortality, healthcare expenditure, and reduced quality of life. Acute kidney injury (AKI) and chronic kidney disease (CKD) are two important forms of renal dysfunction frequently encountered in

clinical practice. Although both conditions involve impairment of renal function, they differ considerably in their underlying pathophysiology, clinical manifestations, biochemical abnormalities, progression, and prognosis. The increasing prevalence of diabetes mellitus, hypertension, cardiovascular disorders, infections, and exposure to nephrotoxic medications has further increased the burden of kidney-related disorders. Early identification and appropriate management are essential for preventing potentially life-threatening complications and improving patient outcomes.[1] Acute kidney injury is characterized by a sudden deterioration in renal function, resulting in impaired excretion of nitrogenous waste products, disturbances in fluid and electrolyte balance, and abnormalities in acid–base homeostasis. It commonly develops in hospitalized patients, particularly those with sepsis, dehydration, hypotension, shock, major surgical interventions, or exposure to nephrotoxic medications. The clinical presentation varies from asymptomatic elevations in serum creatinine to oliguria, anuria, fluid overload, metabolic acidosis, and life-threatening electrolyte disturbances. AKI may be reversible when the underlying cause is identified and corrected promptly; however, severe or persistent renal injury can result in prolonged hospitalization, multiorgan dysfunction, and progression toward chronic renal impairment.[2] Chronic kidney disease is characterized by persistent abnormalities of kidney structure or function for at least three months, with important implications for general health. Unlike AKI, CKD generally develops gradually and is associated with progressive nephron loss, structural renal alterations, and declining glomerular filtration. Diabetes mellitus and hypertension are major underlying conditions, while chronic glomerular diseases, obstructive nephropathy, hereditary disorders, and systemic inflammatory conditions also contribute to its development. Patients may remain asymptomatic during the initial stages but subsequently develop generalized weakness, anorexia, nausea, edema, anemia, cardiovascular complications, and metabolic abnormalities. Progressive renal dysfunction may eventually necessitate renal replacement therapy, emphasizing the importance of early diagnosis, risk assessment, and comprehensive clinical management.[3] Differentiation between AKI and CKD is essential because diagnostic approaches, treatment priorities, and long-term management strategies differ significantly. Assessment of renal dysfunction requires comprehensive evaluation of clinical history, previous laboratory investigations, urine output, serum creatinine, estimated glomerular filtration rate, and radiological findings. Renal ultrasonography provides additional information regarding kidney size, cortical echogenicity, and structural abnormalities. Reduced renal dimensions and increased cortical echogenicity may suggest chronic renal disease, whereas acute dysfunction may occur without established structural changes. Accurate classification is particularly important in hospitalized patients presenting with elevated serum creatinine, as inappropriate interpretation may delay treatment or influence clinical decisions regarding dialysis and follow-up.[4] Electrolyte disturbances are common and clinically significant complications of renal dysfunction. The kidneys maintain sodium and water balance through coordinated processes involving glomerular filtration, tubular reabsorption, and hormonal regulation. Impairment of these mechanisms may produce hyponatremia, hypernatremia, and disturbances in extracellular fluid volume. Hyponatremia may develop because of impaired free-water excretion, excessive fluid administration, or associated systemic illnesses, while hypernatremia frequently reflects inadequate water intake or excessive fluid losses. These disturbances may manifest as weakness, nausea, confusion, seizures, or altered consciousness. Careful assessment of serum sodium concentrations and hydration status is therefore essential for guiding appropriate fluid management and preventing neurological complications.[5] Potassium abnormalities represent another important concern among patients with AKI and CKD. Hyperkalemia may develop because of reduced renal potassium excretion, metabolic acidosis, tissue breakdown, or medications affecting the renin–angiotensin–aldosterone system. Severe hyperkalemia can cause potentially fatal cardiac arrhythmias and requires prompt intervention. Hypokalemia may also occur, particularly in patients receiving diuretics or experiencing gastrointestinal losses. Chronic impairment of renal function additionally contributes to abnormalities in calcium, phosphate, magnesium, and bicarbonate metabolism. These disturbances may coexist and increase the complexity of clinical management. Comprehensive biochemical monitoring is therefore necessary for identifying electrolyte-related emergencies and determining appropriate therapeutic interventions, including renal replacement therapy when indicated.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, prospective observational comparative study to evaluate and compare the clinical profile, electrolyte abnormalities, biochemical parameters, associated comorbidities, complications, and hospital outcomes among patients diagnosed with acute kidney injury (AKI) and chronic kidney disease (CKD). The study aimed to identify significant differences in clinical presentation, electrolyte disturbances, renal dysfunction, requirement for renal replacement therapy, length of hospital stay, and in-hospital mortality between the two groups. The study was conducted at a tertiary care hospital in the Departments of General Medicine. Patients admitted to medical wards and intensive care units with AKI or CKD were evaluated. A total of 114 adult patients diagnosed with AKI or CKD were included using a

consecutive sampling technique. Patients were categorized into two groups according to their established diagnosis. Group A comprised patients with acute kidney injury, whereas Group B comprised patients with chronic kidney disease. The classification was based on clinical history, previous medical records, biochemical investigations, urine output, and ultrasonographic findings.

Inclusion Criteria

Patients aged 18 years and above, irrespective of gender, with a confirmed diagnosis of AKI or CKD requiring hospitalization were eligible for inclusion. Patients presenting with electrolyte disturbances, reduced renal function, or complications associated with kidney disease were included after obtaining informed consent.

Exclusion Criteria

Patients with acute kidney injury superimposed on established chronic kidney disease, renal transplant recipients, patients receiving maintenance dialysis, and individuals with incomplete clinical or laboratory records were excluded. Pregnant women and patients unwilling to provide informed consent were also excluded to minimize potential confounding factors.

Methodology: Acute kidney injury was diagnosed according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria, including an increase in serum creatinine ≥0.3 mg/dL within 48 hours, an increase ≥1.5 times baseline within seven days, or urine output <0.5 mL/kg/hour for at least six hours. Chronic kidney disease was diagnosed by persistent abnormalities of kidney structure or function for at least three months, including estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2 or evidence of kidney damage. AKI severity and CKD stages were classified according to KDIGO recommendations.

Clinical Assessment: Detailed demographic and clinical information was recorded using a structured proforma. Parameters included age, gender, body mass index, socioeconomic background, smoking history, alcohol consumption, duration of symptoms, and previous kidney disease. Associated comorbidities, including diabetes mellitus, hypertension, cardiovascular disease, chronic liver disease, and infections, were documented. Clinical manifestations such as oliguria, anuria, generalized weakness, nausea, vomiting, peripheral edema, breathlessness, altered sensorium, and reduced appetite were evaluated. Blood pressure, heart rate, respiratory rate, oxygen saturation, hydration status, and Glasgow Coma Scale score were recorded at admission.

Laboratory Investigations: Venous blood samples were collected under aseptic conditions for complete blood count, hemoglobin, total leukocyte count, platelet count, blood urea, serum creatinine, sodium, potassium, chloride, bicarbonate, total calcium, magnesium, phosphate, and albumin. Additional investigations included random blood glucose, liver function tests, urinalysis, urine protein, and urine albumin-to-creatinine ratio wherever indicated. Serum creatinine and blood urea levels were monitored during hospitalization. Renal ultrasonography was performed to assess kidney size, cortical echogenicity, corticomedullary differentiation, and urinary tract obstruction.

Assessment of Electrolyte Abnormalities: Electrolyte disturbances were assessed at admission and during hospitalization. Hyponatremia and hypernatremia were defined as serum sodium <135 mEq/L and >145 mEq/L, respectively. Hypokalemia was defined as serum potassium <3.5 mEq/L, while hyperkalemia was defined as >5.5 mEq/L. Hypocalcemia was identified using albumin-corrected serum calcium <8.5 mg/dL. Hyperphosphatemia was defined as serum phosphate >4.5 mg/dL, and metabolic acidosis was assessed using serum bicarbonate <22 mEq/L. The frequency, severity, and coexistence of electrolyte abnormalities were compared between both groups.

Assessment of Hospital Outcomes: Patients were monitored throughout hospitalization for clinical progression and complications. Major outcomes included requirement for intensive care, initiation of renal replacement therapy, mechanical ventilation, cardiovascular complications, electrolyte-related emergencies, length of hospital stay, and in-hospital mortality. Improvement or deterioration in renal function was evaluated using serial serum creatinine measurements and urine output. Patients were categorized according to survival status at discharge, and associations between electrolyte disturbances and adverse hospital outcomes were analyzed.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 28.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. The independent Student's t-test or Mann–Whitney U test was applied for continuous variables, as appropriate. Categorical variables were compared using the Chi-square test or Fisher's exact test. Multivariable logistic regression analysis was used to identify independent predictors of adverse hospital outcomes, where event counts permitted. Odds ratios with 95% confidence intervals were calculated. A p-value <0.05 was considered statistically significant.

RESULTS

[Table 1] Comparison of Baseline Demographic Characteristics and Associated Comorbidities Between AKI and CKD Patients

[Table 1] presents the baseline demographic characteristics and associated comorbidities among 114 patients, comprising 57 patients each in the AKI and CKD groups. The mean age was significantly higher in CKD patients (58.67 ± 12.31 years) compared with AKI patients (51.84 ± 14.22 years; p=0.007). Male predominance was observed in both groups, accounting for 61.40% of AKI patients and

59.65% of CKD patients, without a significant difference (p=0.848). The mean BMI was comparable between the groups (24.43 ± 3.68 versus 25.16 ± 4.05 kg/m²; p=0.316). Diabetes mellitus (54.39% versus 28.07%; p=0.004), hypertension (73.68% versus 36.84%; p<0.001), and cardiovascular disease (31.58% versus 15.79%; p=0.047) were significantly more prevalent among CKD patients. Conversely, infection or sepsis at admission (43.86% versus 17.54%; p=0.002) and recent nephrotoxic drug exposure (28.07% versus 8.77%; p=0.008) were significantly more frequent in AKI patients, suggesting differences in the clinical factors associated with both renal conditions.

[Table 2] Comparison of Clinical Presentation and Ultrasonographic Findings Between AKI and CKD Patients

[Table 2] demonstrates significant differences in clinical manifestations and renal ultrasonographic findings between the two groups. Oliguria was significantly more frequent among AKI patients (66.67%) compared with CKD patients (35.09%; p<0.001), whereas generalized weakness (75.44% versus 52.63%; p=0.011) and peripheral edema (68.42% versus 36.84%; p<0.001) were significantly more common in CKD patients. Clinical dehydration was reported in 47.37% of AKI patients compared with 14.04% of CKD patients (p<0.001), while systolic hypotension at admission was more frequent in AKI patients (26.32% versus 8.77%; p=0.014). Nausea or vomiting, breathlessness, and altered sensorium showed no statistically significant differences between the groups (p>0.05). Ultrasonographic examination revealed reduced renal size in 64.91% of CKD patients compared with 7.02% of AKI patients, while increased renal cortical echogenicity was observed in 78.95% and 21.05%, respectively, with both differences being highly significant (p<0.001).

[Table 3] Comparison of Hematological, Renal Function, and Biochemical Parameters Between AKI and CKD Patients

[Table 3] compares the hematological, renal function, and biochemical parameters of patients with AKI and CKD. The mean hemoglobin concentration was significantly lower in CKD patients (8.92 ± 1.73 g/dL) than in AKI patients (10.76 ± 1.89 g/dL; p<0.001), whereas the total leukocyte count was significantly higher in AKI patients (13.24 ± 4.15 versus 9.78 ± 3.34 ×10³/µL; p<0.001). CKD patients exhibited significantly higher mean blood urea (96.73 ± 30.84 versus 83.42 ± 27.61 mg/dL; p=0.017) and serum creatinine concentrations (5.04 ± 1.86 versus 3.62 ± 1.44 mg/dL; p<0.001). Serum bicarbonate was significantly lower in CKD patients (17.41 ± 4.16 versus 19.62 ± 4.56 mEq/L; p=0.008), indicating more pronounced metabolic acid–base disturbance. Corrected serum calcium was significantly lower in CKD patients (7.86 ± 0.78 versus 8.47 ± 0.91 mg/dL; p<0.001), while serum phosphate (5.83 ± 1.58 versus 4.79 ± 1.35 mg/dL; p<0.001) and magnesium (2.41 ± 0.51 versus 2.15 ± 0.42 mg/dL; p=0.004) were significantly higher. However, differences in mean serum sodium, potassium, and albumin concentrations were not statistically significant (p>0.05), suggesting that CKD patients had more pronounced anemia, azotemia, and mineral metabolism abnormalities.

[Table 4] Comparison of Electrolyte Abnormalities and Acid–Base Disturbances Between AKI and CKD Patients

[Table 4] illustrates the distribution of electrolyte abnormalities and acid–base disturbances among patients with AKI and CKD. Hyponatremia was observed in 50.88% of AKI patients and 42.11% of CKD patients (p=0.348), while hypernatremia was reported in 12.28% and 7.02%, respectively (p=0.341), without statistically significant differences. Hypokalemia was comparatively more frequent in AKI patients (15.79% versus 7.02%; p=0.141), whereas hyperkalemia was more common in CKD patients (52.63% versus 36.84%; p=0.090), although neither difference reached statistical significance. Hypocalcemia was significantly more prevalent among CKD patients (68.42%) compared with AKI patients (40.35%; p=0.003), and a similar pattern was observed for hyperphosphatemia (73.68% versus 45.61%; p=0.002). Metabolic acidosis was significantly more frequent in CKD patients (70.18% versus 45.61%; p=0.008), reflecting greater acid–base disturbances in this group. Furthermore, at least two concurrent electrolyte abnormalities were identified in 66.67% of CKD patients compared with 40.35% of AKI patients (p=0.005), demonstrating a significantly greater burden of multiple electrolyte disturbances among hospitalized CKD patients.

[Table 5] Comparison of Hospital Outcomes, Complications, and Mortality Between AKI and CKD Patients

[Table 5] presents the comparative hospital outcomes, complications, and mortality among AKI and CKD patients. Intensive care unit admission was significantly more frequent in AKI patients (42.11%) compared with CKD patients (22.81%; p=0.028), indicating a greater requirement for critical care support in the AKI group. Renal replacement therapy was initiated in 31.58% of AKI patients and 47.37% of CKD patients (p=0.085), while mechanical ventilation was required in 26.32% and 12.28%, respectively (p=0.058), with neither difference reaching statistical significance. Cardiovascular complications were comparable between AKI and CKD patients (19.30% versus 17.54%; p=0.809), whereas electrolyte-related emergencies were numerically more frequent in CKD patients (43.86% versus 29.82%; p=0.120). The mean duration of hospitalization was significantly longer among CKD patients (9.16 ± 3.36 days) than AKI patients (7.23 ± 2.85 days; p=0.001). Although in-hospital mortality was higher in AKI patients (19.30% versus 10.53%) and discharge survival was higher in CKD patients (89.47% versus 80.70%), these differences were not statistically significant (p=0.189).

Table 1: Comparison of Baseline Demographic Characteristics and Associated Comorbidities Between AKI and CKD Patients (N=114)

Parameter	AKI (n=57)	CKD (n=57)	p-value
Age (years), mean ± SD	51.84 ± 14.22	58.67 ± 12.31	0.007
Male gender	35 (61.40%)	34 (59.65%)	0.848
Female gender	22 (38.60%)	23 (40.35%)	0.848
BMI (kg/m²), mean ± SD	24.43 ± 3.68	25.16 ± 4.05	0.316
Diabetes mellitus	16 (28.07%)	31 (54.39%)	0.004
Hypertension	21 (36.84%)	42 (73.68%)	<0.001
Cardiovascular disease	9 (15.79%)	18 (31.58%)	0.047
Infection/sepsis at admission	25 (43.86%)	10 (17.54%)	0.002
Recent nephrotoxic drug exposure	16 (28.07%)	5 (8.77%)	0.008

Table 2: Comparison of Clinical Presentation and Ultrasonographic Findings Between AKI and CKD Patients (N=114)

Clinical parameter	AKI (n=57)	CKD (n=57)	p-value
Oliguria	38 (66.67%)	20 (35.09%)	<0.001
Generalized weakness	30 (52.63%)	43 (75.44%)	0.011
Nausea or vomiting	34 (59.65%)	32 (56.14%)	0.704
Peripheral edema	21 (36.84%)	39 (68.42%)	<0.001
Breathlessness	20 (35.09%)	29 (50.88%)	0.089
Altered sensorium	13 (22.81%)	7 (12.28%)	0.140
Clinical dehydration	27 (47.37%)	8 (14.04%)	<0.001
Systolic BP ≤90 mmHg at admission	15 (26.32%)	5 (8.77%)	0.014
Reduced renal size on ultrasonography	4 (7.02%)	37 (64.91%)	<0.001
Increased renal cortical echogenicity	12 (21.05%)	45 (78.95%)	<0.001

Table 3: Comparison of Hematological, Renal Function, and Biochemical Parameters Between AKI and CKD Patients (N=114)

Laboratory parameter	AKI (n=57), mean ± SD	CKD (n=57), mean ± SD	p-value
Hemoglobin (g/dL)	10.76 ± 1.89	8.92 ± 1.73	<0.001
Total leukocyte count (×10³/µL)	13.24 ± 4.15	9.78 ± 3.34	<0.001
Blood urea (mg/dL)	83.42 ± 27.61	96.73 ± 30.84	0.017
Serum creatinine (mg/dL)	3.62 ± 1.44	5.04 ± 1.86	<0.001
Serum sodium (mEq/L)	132.86 ± 6.34	134.10 ± 5.59	0.270
Serum potassium (mEq/L)	5.07 ± 0.97	5.31 ± 0.91	0.176
Serum bicarbonate (mEq/L)	19.62 ± 4.56	17.41 ± 4.16	0.008
Corrected serum calcium (mg/dL)	8.47 ± 0.91	7.86 ± 0.78	<0.001
Serum phosphate (mg/dL)	4.79 ± 1.35	5.83 ± 1.58	<0.001
Serum magnesium (mg/dL)	2.15 ± 0.42	2.41 ± 0.51	0.004
Serum albumin (g/dL)	3.28 ± 0.53	3.12 ± 0.56	0.120

Table 4: Comparison of Electrolyte Abnormalities and Acid–Base Disturbances Between AKI and CKD Patients (N=114)

Electrolyte abnormality	AKI (n=57)	CKD (n=57)	p-value
Hyponatremia	29 (50.88%)	24 (42.11%)	0.348
Hypernatremia	7 (12.28%)	4 (7.02%)	0.341
Hypokalemia	9 (15.79%)	4 (7.02%)	0.141
Hyperkalemia	21 (36.84%)	30 (52.63%)	0.090
Hypocalcemia	23 (40.35%)	39 (68.42%)	0.003
Hyperphosphatemia	26 (45.61%)	42 (73.68%)	0.002
Metabolic acidosis	26 (45.61%)	40 (70.18%)	0.008
At least two concurrent electrolyte abnormalities	23 (40.35%)	38 (66.67%)	0.005

Table 5: Comparison of Hospital Outcomes, Complications, and Mortality Between AKI and CKD Patients (N=114)

Hospital outcome	AKI (n=57)	CKD (n=57)	p-value
ICU admission	24 (42.11%)	13 (22.81%)	0.028
Initiation of renal replacement therapy	18 (31.58%)	27 (47.37%)	0.085
Requirement for mechanical ventilation	15 (26.32%)	7 (12.28%)	0.058
Cardiovascular complications	11 (19.30%)	10 (17.54%)	0.809
Electrolyte-related emergencies	17 (29.82%)	25 (43.86%)	0.120
Length of hospital stay (days), mean ± SD	7.23 ± 2.85	9.16 ± 3.36	0.001
In-hospital mortality	11 (19.30%)	6 (10.53%)	0.189
Discharged alive	46 (80.70%)	51 (89.47%)	0.189

DISCUSSION

The present study comparatively evaluated the clinical profile, electrolyte abnormalities, biochemical parameters, and hospital outcomes among 114 hospitalized patients, comprising 57 patients with acute kidney injury (AKI) and 57 patients with chronic kidney disease (CKD). The mean age was significantly higher among CKD patients (58.67 ± 12.31 years) than AKI patients

(51.84 ± 14.22 years; p=0.007). Diabetes mellitus (54.39% versus 28.07%; p=0.004), hypertension (73.68% versus 36.84%; p<0.001), and cardiovascular disease (31.58% versus 15.79%; p=0.047) were significantly more prevalent in the CKD group. Singh et al. (2013), in the SEEK-India study involving 5,588 participants, reported a CKD prevalence of 17.20%, with hypertension and diabetes mellitus present in 43.10% and 18.80% of the overall screened population, respectively. Although their population-based estimates were not directly comparable with those of hospitalized CKD patients, both studies highlighted the importance of hypertension and diabetes in the clinical assessment of kidney disease. The greater burden of chronic comorbidities observed in the present CKD group may reflect the cumulative effects of longstanding metabolic and vascular disorders on renal function.[7] Regarding the precipitating factors and clinical presentation of AKI, infection or sepsis was significantly more frequent among AKI patients (43.86%) compared with CKD patients (17.54%; p=0.002). Recent nephrotoxic drug exposure was also more common in AKI patients (28.07% versus 8.77%; p=0.008). Clinical dehydration was observed in 47.37% of AKI patients compared with 14.04% of CKD patients (p<0.001), whereas systolic hypotension was recorded in 26.32% and 8.77%, respectively (p=0.014). Mehta et al. (2016), in a multinational study involving 4,018 patients with AKI from 72 countries, identified hypotension in 40.00% and dehydration in 38.00% of patients as important contributors to AKI. Their findings were broadly consistent with the present study, particularly regarding the importance of volume depletion and hemodynamic instability in acute deterioration of renal function. Differences in the observed frequencies may be related to the severity of illness, underlying etiologies, and clinical characteristics of the studied populations.[8] Clinical manifestations and renal ultrasonographic findings demonstrated important differences between AKI and CKD patients. Oliguria was significantly more frequent in the AKI group (66.67% versus 35.09%; p<0.001), whereas generalized weakness (75.44% versus 52.63%; p=0.011) and peripheral edema (68.42% versus 36.84%; p<0.001) were more common in CKD patients. Reduced renal size was identified in 64.91% of CKD patients compared with 7.02% of AKI patients, while increased cortical echogenicity was recorded in 78.95% and 21.05%, respectively (both p<0.001). Ozmen et al. (2010), in a comparative ultrasonographic investigation involving 127 patients with renal failure and 33 healthy volunteers, demonstrated that renal length and parenchymal thickness were significantly greater in acute renal failure than in chronic renal failure (p<0.0001). Their findings supported the ultrasonographic differences observed in the present study, particularly the association of reduced renal dimensions with chronic renal dysfunction. Nevertheless, renal ultrasonography should be interpreted alongside clinical history and biochemical investigations because structural findings alone cannot reliably distinguish all cases of AKI from CKD.[9] The comparison of hematological and biochemical parameters revealed significantly lower mean hemoglobin levels among CKD patients (8.92 ± 1.73 g/dL) compared with AKI patients (10.76 ± 1.89 g/dL; p<0.001). Serum creatinine (5.04 ± 1.86 versus 3.62 ± 1.44 mg/dL; p<0.001) and blood urea concentrations (96.73 ± 30.84 versus 83.42 ± 27.61 mg/dL; p=0.017) were also significantly higher in CKD patients. Conversely, the total leukocyte count was higher among AKI patients (13.24 ± 4.15 versus 9.78 ± 3.34 $\times 10^3/\mu L$; p<0.001), potentially reflecting the greater frequency of acute infections in this group. Stauffer and Fan (2014), using data from the United States National Health and Nutrition Examination Survey, reported an anemia prevalence of 15.40% among individuals with CKD, increasing from 8.40% in stage 1 to 53.40% in stage 5 CKD. Although their study measured anemia prevalence rather than the difference in mean hemoglobin between AKI and CKD, the findings were consistent with the greater hematological impairment observed in the present CKD group. Reduced erythropoietin production, chronic inflammation, nutritional deficiencies, and prolonged renal dysfunction may contribute to the development and severity of anemia in CKD patients.[10] Electrolyte and mineral metabolism abnormalities were more pronounced among CKD patients, particularly hypocalcemia and hyperphosphatemia. In the present study, mean corrected serum calcium was significantly lower in CKD patients (7.86 ± 0.78 mg/dL) compared with AKI patients (8.47 ± 0.91 mg/dL; p<0.001), whereas mean serum phosphate was significantly higher (5.83 ± 1.58 versus 4.79 ± 1.35 mg/dL; p<0.001). Hypocalcemia was detected in 68.42% of CKD patients compared with 40.35% of AKI patients (p=0.003), while hyperphosphatemia occurred in 73.68% and 45.61%, respectively (p=0.002). Valson et al. (2014), in a hospital-based cross-sectional study involving 710 patients with stage 4 and 5 CKD in South India, reported hypocalcemia in 66.30%, hyperphosphatemia in 59.00%, hyperparathyroidism in 89.30%, and vitamin D insufficiency or deficiency in 74.70% of patients. The prevalence of hypocalcemia in their study was comparable to the 68.42% observed in the present CKD group, whereas hyperphosphatemia was more frequent in the present study.[11] Serum potassium abnormalities were frequently encountered in both study groups, although their distribution differed. Hyperkalemia was observed in 52.63% of CKD patients compared with 36.84% of AKI patients (p=0.090), whereas hypokalemia was recorded in 7.02% and 15.79%, respectively (p=0.141). Although these differences were not statistically significant, the higher numerical frequency of hyperkalemia among CKD patients indicated the clinical importance of potassium monitoring in individuals with reduced renal function. Panuccio et al. (2021), in a longitudinal

study involving 752 patients with stages 2–5 CKD, reported that 27.00% had serum potassium concentrations above 5.0 mmol/L at baseline, increasing to 30.00% at the final assessment. Furthermore, 33.00% of their patients had metabolic acidosis at baseline, and reduced bicarbonate concentrations were independently associated with hyperkalemia during follow-up. The present study demonstrated a higher frequency of hyperkalemia despite using a stricter threshold of potassium above 5.5 mEq/L. However, the findings were not directly comparable because the present population consisted of hospitalized patients, whereas the published cohort was followed in nephrological care.[12] Metabolic acidosis and multiple concurrent electrolyte abnormalities represented additional important findings in the present study. Metabolic acidosis was significantly more frequent among CKD patients (70.18%) compared with AKI patients (45.61%; p=0.008), and mean serum bicarbonate was significantly lower in CKD patients (17.41 ± 4.16 versus 19.62 ± 4.56 mEq/L; p=0.008). Furthermore, at least two concurrent electrolyte abnormalities were identified in 66.67% of CKD patients compared with 40.35% of AKI patients (p=0.005). Cook et al. (2021), in a retrospective analysis of patients with stages 3–5 CKD and hyperkalemia, reported that the prevalence of metabolic acidosis, defined as serum bicarbonate below 22 mEq/L, ranged from 25.00% to 29.00% among patients with potassium levels above 5.0 mEq/L and from 33.00% to 39.00% among those with potassium levels above 5.5 mEq/L. The substantially higher frequency of metabolic acidosis in the present CKD group might reflect differences in hospitalization status, severity of renal dysfunction, and associated clinical complications. The coexistence of multiple electrolyte and acid–base disturbances in CKD highlights the importance of comprehensive biochemical evaluation rather than the assessment of individual electrolyte abnormalities alone.[13] The comparison of hospital outcomes revealed important differences in critical care requirements between AKI and CKD patients. Intensive care unit admission was significantly more frequent among AKI patients (42.11%) than CKD patients (22.81%; p=0.028). Mechanical ventilation was required in 26.32% of AKI patients compared with 12.28% of CKD patients (p=0.058), whereas cardiovascular complications occurred in 19.30% and 17.54%, respectively (p=0.809). Renal replacement therapy was initiated in 31.58% of AKI patients and 47.37% of CKD patients (p=0.085). Hoste et al. (2006), in a retrospective cohort analysis involving 5,383 patients admitted to seven intensive care units, reported that AKI occurred in 67.00% of ICU admissions, with maximum RIFLE risk, injury, and failure categories accounting for 12.00%, 27.00%, and 28.00%, respectively. Hospital mortality increased across the severity categories, reaching 26.30% in patients classified as RIFLE failure compared with 5.50% among patients without AKI. Although their ICU-based population differed from the present mixed-ward and ICU population, the findings demonstrated the substantial clinical burden associated with acute renal dysfunction.[14] In-hospital mortality and length of hospitalization were important indicators of disease burden and clinical outcomes in the present study. The mean hospital stay was significantly longer among CKD patients (9.16 ± 3.36 days) compared with AKI patients (7.23 ± 2.85 days; p=0.001). In contrast, in-hospital mortality was numerically higher among AKI patients (19.30%) than CKD patients (10.53%), although the difference was not statistically significant (p=0.189). Patel et al. (2024), in a prospective observational study involving 230 patients with AKI at a tertiary care hospital in North India, reported an overall mortality rate of 19.50%, which was remarkably close to the 19.30% observed in the present AKI group. Their study also documented oliguria in 56.50%, hyperkalemia in 34.70%, and metabolic acidosis in 22.60% of AKI patients, compared with corresponding frequencies of 66.67%, 36.84%, and 45.61% in the present AKI group. These comparisons demonstrated broadly similar frequencies of oliguria and hyperkalemia, although metabolic acidosis was more frequent in the present study. The longer hospitalization among CKD patients may reflect the need for management of chronic comorbidities, anemia, electrolyte disturbances, and dialysis-related requirements, whereas the greater critical care requirements and numerically higher mortality among AKI patients may reflect acute physiological instability.[15]

CONCLUSION

The present study demonstrated significant differences in clinical presentation, biochemical parameters, electrolyte abnormalities, and hospital outcomes between patients with acute kidney injury (AKI) and chronic kidney disease (CKD). AKI patients exhibited higher frequencies of oliguria, dehydration, infection, and intensive care requirements, whereas CKD patients had a greater burden of diabetes, hypertension, anemia, hypocalcemia, hyperphosphatemia, and metabolic acidosis. CKD patients experienced significantly longer hospital stays, while in-hospital mortality was numerically higher among AKI patients without reaching statistical significance.

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