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Original Article

Acute Kidney Injury following Acute Gastroenteritis at a Tertiary Care Center

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Abstract

Background

Acute kidney injury is a significant and potentially reversible cause of morbidity and mortality, particularly in developing countries where pre-renal etiologies predominate. Acute gastroenteritis is a frequent precipitating illness due to dehydration and electrolyte imbalance. This study aimed to evaluate the clinical profile, biochemical parameters, etiological factors, and outcomes of AKI following acute gastroenteritis in a tertiary care hospital in eastern Nepal.

Materials and Methods

A descriptive cross-sectional study was conducted at Nobel Medical College Teaching Hospital, Biratnagar, over one year among 71 patients aged ≥18 years who developed AKI in association with acute gastroenteritis. Data on demographics, clinical presentation, laboratory findings, and outcomes were collected and analysed using IBM SPSS Statistics version 29. AKI was diagnosed and staged according to KDIGO 2012 criteria. Descriptive statistics were used, and results were expressed as mean ± SD and percentage.

Results

The mean age of patients was 51.4 ± 17.2 years, with a male predominance (59.2%). Moderate to severe dehydration was present in 87.3% of cases. Mean serum creatinine and urea were 4.6 ± 1.8 mg/dL and 130.5 ± 54.2 mg/dL, respectively. Hyponatremia (30.99%), hyperkalemia (25.35%), and metabolic acidosis (52.11%) were common abnormalities. KDIGO Stage 1 AKI occurred in 39.44%, Stage 2 in 32.39%, and Stage 3 in 28.17%. Infectious gastroenteritis was the leading cause (73.24%), and 87.32% achieved complete recovery.

Conclusion

Acute kidney injury following acute gastroenteritis is a common clinical condition with a favorable prognosis. Dehydration and infectious etiology are the predominant contributing factors, and the majority of patients achieve complete renal recovery.

Keywords: *Acute kidney injury, Dehydration, Gastroenteritis, Water-electrolyte imbalance*

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Introduction

Acute kidney injury (AKI) represents a rapid decline in renal function over hours to days and remains an important cause of morbidity and mortality worldwide [1]. In developing countries, prerenal causes such as hypovolemia, dehydration, and infection are predominant. Among these, acute gastroenteritis, characterized by profuse diarrhea and vomiting, is a common precipitating factor leading to fluid loss and subsequent renal hypoperfusion [2]. Despite being preventable, AKI associated with gastroenteritis remains underrecognized in resource-limited settings.

In Nepal, diarrheal diseases continue to be a leading cause of hospitalization, yet limited data exist regarding their renal complications [3]. Early identification and correction of dehydration can prevent progression to renal failure and improve outcomes.

This study was conducted to evaluate the clinical and biochemical profiles, etiological spectrum, staging, and short-term outcomes of patients who developed AKI following acute gastroenteritis in a tertiary care hospital.

Materials and Methods

This hospital-based descriptive cross-sectional study was conducted in the department of Internal Medicine at Nobel Medical College Teaching Hospital, Biratnagar, over one year from February 2025 to January 2026. Ethical clearance for the study was obtained from the Institutional Review Committee of Nobel Medical College (IRC No:117/2024), and written informed consent was obtained from all participants prior to inclusion. The study included all patients aged 18 years and above who were admitted with acute gastroenteritis and developed acute kidney injury (AKI) either at presentation or during hospitalization. Patients with known chronic kidney disease, pre-existing structural renal abnormalities, nephrotoxic drug exposure before admission, or incomplete records were excluded. The sample size (n) was calculated using the formula, $n=Z^2\times p\times q/e^2$,where Z = 1.96 for a 95% confidence interval, p = 0.13 (prevalence of AKI in gastroenteritis from prior studies), q = 1 – p, and e = 10% allowable error [4]. The minimum calculated sample was 43, but all patients who fulfilled the inclusion criteria were included in the study. A total of 71 patients were enrolled to improve precision.

Acute gastroenteritis was defined as the passage of ≥3 loose or watery stools per day with or without vomiting, abdominal pain, or fever, lasting less than 14 days, as per the World Health Organization (WHO) criteria [5]. Dehydration status was assessed clinically and categorized as mild, moderate, or severe according to WHO guidelines, based on findings such as skin turgor, mucosal dryness, pulse volume, and urine output [6]. Acute kidney injury was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO, 2012) guidelines as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours, or a rise to ≥1.5 times the baseline within seven days, or urine output less than 0.5 mL/kg/hour for more than six hours [7]. AKI staging followed KDIGO classification: Stage 1 (1.5–1.9× baseline or ≥0.3 mg/dL increase), Stage 2 (2.0–2.9× baseline), and Stage 3 (≥3× baseline or serum creatinine ≥4.0 mg/dL). Patient with available previously done serum creatinine measurements were taken as baseline, and for patients without such examination, MDRD equation assuming a normal GFR of 75ml/min/1.73 m^2 was used to calculate the baseline creatinine. Serum creatinine, blood urea, electrolytes (sodium, potassium), bicarbonate, and haematocrit were measured at presentation and during follow-up. Hyponatremia was defined as serum sodium <135 mEq/L, and hyperkalemia as serum potassium >5.0 mEq/L [8]. Metabolic acidosis was diagnosed when serum bicarbonate was <22 mEq/L or arterial pH <7.35 [9].

Data were collected using a structured proforma including demographic details, clinical symptoms, dehydration status, laboratory investigations, and outcomes. Stool routine microscopy and culture were performed when indicated, and abdominal ultrasonography was used to exclude structural causes. Patients were managed as per institutional protocol, including oral or intravenous rehydration, electrolyte correction, antibiotics when indicated, and renal replacement therapy for severe cases. All data were entered in Microsoft Excel and analysed using IBM SPSS Statistics version 29. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequency and percentage (n, %). Strict confidentiality of patient information was maintained throughout the study.

Results

A total of 71 patients fulfilling the inclusion criteria were enrolled. The mean age was 51.4 ± 17.2 years (range 18–85 years), with males outnumbering females (male-to-female ratio 1.45:1). The mean duration of diarrhea before admission was 3.6 ± 1.9 days, and the mean duration of hospital stay was 5.2 ± 2.3 days. The age and sex distribution, presenting symptoms, and dehydration severity are summarized in Table 1.

Table 1: Age and Sex Distribution, Presenting Symptoms, and Dehydration Severity of Patients (n = 71)

Parameter	n (%)
Age Group (years)	
18–30	9 (12.68)
31–40	19 (26.76)
41–60	23 (32.39)
>60	20 (28.17)
Sex	
Male	42 (59.15)
Female	29 (40.85)
Presenting Symptoms	
Diarrhea	71 (100.00)
Vomiting	52 (73.24)
Generalized weakness	45 (63.38)
Fever	33 (46.48)
Abdominal pain	21 (29.58)
Dehydration Severity	
Mild	9 (12.68)
Moderate	39 (54.93)
Severe	23 (32.39)
Total	71 (100.00)

Biochemical investigations revealed moderate-to-severe renal impairment at presentation, along with frequent electrolyte and acid–base disturbances. Mean values and the prevalence of electrolyte abnormalities are presented in Table 2.

Table 2: Biochemical Parameters and Electrolyte/Acid–Base Abnormalities (n = 71)

Parameter	Mean ± SD	Abnormality n (%)
Biochemical Parameters		
Serum creatinine (mg/dL)	4.6 ± 1.8	—
Blood urea (mg/dL)	130.5 ± 54.2	—
Haematocrit (%)	38.7 ± 5.6	—
Electrolytes & Acid–Base		
Serum sodium (mEq/L)	133.2 ± 6.7	—
Hyponatremia (<135 mEq/L)	—	22 (30.99)
Hypernatremia (>145 mEq/L)	—	6 (8.45)
Serum potassium (mEq/L)	4.7 ± 0.9	—
Hyperkalemia (>5.0 mEq/L)	—	18 (25.35)
Serum bicarbonate (mEq/L)	19.8 ± 4.2	—
Metabolic acidosis (HCO3 <22 mEq/L or pH <7.35)	—	37 (52.11)

Hyponatremia: serum sodium <135 mEq/L; Hypernatremia: >145 mEq/L; Hyperkalemia: serum potassium >5.0 mEq/L; Metabolic acidosis: serum bicarbonate <22 mEq/L or arterial pH <7.35. According to KDIGO 2012 criteria, Stage 1 was the most frequent AKI grade, indicating that most patients sustained reversible prerenal injury. Infective gastroenteritis was the predominant etiology; Escherichia coli was the most commonly isolated organism in stool cultures, accounting for 21.13% of culture-positive cases. Drug-induced causes included NSAID, ACE inhibitor, and herbal drug use. Renal outcomes were favourable overall; no patient required long-term dialysis on follow-up, and deaths were confined to those presenting with septic shock and multiorgan dysfunction. Staging, etiological breakdown, and outcomes are detailed in Table 3.

Table 3: KDIGO Staging, Etiological Spectrum, and Clinical Outcome of Patients (n = 71)

Category	n (%)
KDIGO Staging	
Stage 1	28 (39.44)
Stage 2	23 (32.39)
Stage 3	20 (28.17)
Etiological Spectrum	
Infective gastroenteritis	52 (73.24)
Drug-induced	6 (8.45)
NSAID	3 (4.23)
ACE Inhibitor	2 (2.81)
Herbal drugs	1 (1.41)
Mixed / undetermined	13 (18.31)
Clinical Outcome at Discharge	
Complete recovery	62 (87.32)
Partial recovery	6 (8.45)
Death	3 (4.23)
Total	71 (100.00)

Bivariate analyses were performed using the Chi-square test or Fisher's exact test. Neither dehydration severity nor age group (≤60 vs. >60 years) showed a statistically significant association with KDIGO staging, indicating a broadly comparable distribution of AKI stages across these variables (Table 4). Although patients aged >60 years had a higher proportion of Stage 3 AKI, the difference did not reach statistical significance, likely owing to the limited size of the elderly subgroup (n = 20).

Table 4: Association of Dehydration Severity and Age Group with KDIGO Staging (n = 71)

Variable	Stage 1 n (%)	Stage 2 n (%)	Stage 3 n (%)	Total n	p-value
Dehydration Severity					
Mild	4 (44.44)	3 (33.33)	2 (22.22)	9	0.992*
Moderate	15 (38.46)	13 (33.33)	11 (28.21)	39	
Severe	9 (39.13)	7 (30.43)	7 (30.43)	23	
Total	28	23	20	71	
Age Group					
≤60 years	19 (37.25)	20 (39.22)	12 (23.53)	51	0.122*
>60 years	9 (45.00)	3 (15.00)	8 (40.00)	20	
Total	28	23	20	71	

*Chi-square test. df = degrees of freedom.

Dehydration severity was significantly associated with clinical outcome: adverse outcomes (partial recovery or death) were confined almost entirely to patients with severe dehydration. Age group did not reach statistical significance for the outcome, although older patients trended toward higher rates of adverse events. Hyperkalemia was strongly associated with both KDIGO severity and adverse outcome, with all hyperkalemic patients classified as Stage 2 or Stage 3. Metabolic acidosis showed significant associations with both KDIGO staging and outcome, confirming its role as a marker of severe renal and systemic compromise. Detailed cross-tabulations are presented in Table 5.

Discussion

Acute kidney injury (AKI) remains a major cause of morbidity and mortality worldwide and is particularly common in developing countries, where prerenal etiologies predominate due to infection, dehydration, and poor access to early healthcare [1], [2]. In the present study, AKI following acute gastroenteritis accounted for 71 cases over one year, emphasizing the clinical importance of volume depletion and fluid imbalance in the pathogenesis of renal dysfunction. The mean age of 51.4 years observed in this study reflects a predominance in middle-aged and elderly populations, similar to findings from Prakash et al. and Mehta et al., who also reported a higher incidence among older individuals owing to diminished renal reserve and higher prevalence of comorbidities [10], [11]. The male preponderance (59.15%) noted in this series corresponds with prior South Asian studies that attribute this difference to occupational exposure, delayed healthcare-seeking among females, and possible gender bias in hospital attendance [7], [12].

All patients in this study presented with diarrhea, consistent with its defining feature in acute gastroenteritis, while vomiting, weakness, and fever were frequently associated symptoms. Moderate to severe dehydration was identified in nearly 87% of patients, confirming the central role of extracellular fluid loss and prerenal hypoperfusion in AKI development. Similar findings were reported by Bhakthavatchalam et al and Jayakumar et al., who emphasized dehydration as a leading precipitant of reversible renal injury [13, 14].

Table 5: Bivariate Associations of Key Variables with KDIGO Staging and Clinical Outcome (n = 71)

A. Dehydration Severity vs Clinical Outcome				
	Complete Recovery, n (%)	Partial Rec / Death, n (%)	Total, n	p-value
Mild	9 (100.00)	0 (0.00)	9	<0.001†
Moderate	38 (97.44)	1 (2.56)	39	
Severe	15 (65.22)	8 (34.78)	23	
Total	62	9	71	
	Complete Recovery, n (%)	Partial Rec / Death, n (%)	Total, n	p-value
≤60 years	47 (92.16)	4 (7.84)	51	0.105*
>60 years	15 (75.00)	5 (25.00)	20	
Total	62	9	71	
	Stage 1, n (%)	Stage 2, n (%)	Stage 3, n (%)	p-value
Absent	28 (52.83)	18 (33.96)	7 (13.21)	<0.001†
Present	0 (0.00)	5 (27.78)	13 (72.22)	
	Complete Recovery, n (%)	Partial Rec / Death, n (%)	Total, n	p-value
Absent	50 (94.34)	3 (5.66)	53	0.006*
Present	12 (66.67)	6 (33.33)	18	
Total	62	9	71	
	Stage 1, n (%)	Stage 2, n (%)	Stage 3, n (%)	p-value
Absent	21 (61.76)	9 (26.47)	4 (11.76)	<0.001†
Present	7 (18.92)	14 (37.84)	16 (43.24)	
	Complete Recovery, n (%)	Partial Rec / Death, n (%)	Total, n	p-value
Absent	33 (97.06)	1 (2.94)	34	0.029*
Present	29 (78.38)	8 (21.62)	37	
Total	62	9	71	

†Chi-square test. *Fisher's exact test.

The mean hospital stay of 5.2 days observed here aligns with the expected recovery period for prerenal AKI, provided prompt rehydration and supportive care are instituted. Biochemical abnormalities in the present study, a mean serum creatinine of 4.6 mg/dL and blood urea of 130.5 mg/dL, indicate moderate to severe renal impairment at presentation, in agreement with previous Nepalese and Indian studies [15], [16]. Electrolyte disturbances were frequent, with hyponatremia (30.99%), hyperkalemia (25.35%), and metabolic acidosis (52.11%) being most common. These derangements result from gastrointestinal losses, impaired renal clearance, and redistribution abnormalities. Studies from

the region report that hyponatremia is common (≈30–40%) in patients with diarrheal illness and AKI, with potassium abnormalities (either hypo- or hyperkalemia) seen in a variable proportion (≈10–50%) depending on cohort and timing of measurement [17], [18], [19]. Metabolic acidosis observed in more than half of cases corroborates earlier findings from Prakash et al. and Mehta et al., which demonstrated the high frequency of acidosis in prerenal azotaemia secondary to dehydration [16], [20]. The KDIGO classification revealed that Stage 1 AKI constituted 39.44%, Stage 2 32.39%, and Stage 3 28.17% of cases. This pattern reflects early hospital presentation and effective rehydration in many cases, preventing progression to higher stages. Comparable distributions were reported by Khwaja et al. and Waikar et al., reinforcing the usefulness of KDIGO criteria in assessing disease severity and predicting outcomes [7], [21]. The predominance of Stage 1 and 2 AKI also indicates that most patients developed reversible prerenal injury rather than intrinsic renal damage.

Infective gastroenteritis was the leading etiology in this study (73.24%), consistent with the findings of Chhetri et al., who reported acute gastroenteritis as the primary cause of AKI in nearly three-fourths of patients admitted to a tertiary hospital in Kathmandu [15]. Regional studies of diarrheal disease etiology have identified *Escherichia coli*, *Shigella* spp., and *Campylobacter* spp. as common enteric pathogens, supporting the likelihood of *E. coli* predominance in infection-associated AKI [22], [23]. The smaller proportions of drug-induced (8.45%) and mixed or undetermined causes (18.31%) highlight the multifactorial nature of renal insult in some patients, possibly due to concurrent use of nephrotoxic antibiotics, sepsis, or pre-existing comorbidities such as diabetes and hypertension. Comparable etiologic trends have been observed in prior South Asian literature [18], [24].

The overall outcome of AKI in this study was favourable, with complete recovery in 87.32% of patients, partial recovery in 8.45%, and mortality in 4.23%. These results are consistent with the predominantly prerenal nature of the insult and the reversibility of renal dysfunction once fluid and electrolyte deficits are corrected. Mortality in our cohort is lower than that reported by Liaño et al. and Mehta et al., where higher rates (10–20%) were associated with intrinsic renal disease, delayed referral, and multiorgan failure [25], [26]. In our series, death occurred mainly in patients presenting with severe dehydration, septic shock, and advanced KDIGO Stage 3 AKI. The strong correlation between advanced stage and adverse outcome parallels the findings of Hoste et al. and Coca et al., who demonstrated that each incremental rise in AKI stage confers an increased risk of death [27], [28].

The pathophysiological mechanism underlying AKI in acute gastroenteritis primarily involves hypovolemia-induced renal hypoperfusion and activation of neurohormonal pathways causing renal vasoconstriction. In prolonged hypoperfusion, ischemic tubular injury may occur, leading to intrinsic renal failure. Prompt restoration of circulatory volume reverses this process in most cases, explaining the high recovery rate in our cohort. Early recognition of dehydration, judicious rehydration therapy, and monitoring of renal function are therefore crucial in prevention and management [29]. The pattern of biochemical abnormalities in this study supports the concept of prerenal azotemia as the predominant mechanism: elevated urea and creatinine levels, accompanied by hyponatremia and metabolic acidosis, are classical findings in hypovolemic states [30]. Electrolyte correction and maintenance of acid–base balance remains essential components of therapy. The mean hospital stay and recovery profile further reflect favourable outcomes with timely management.

Bivariate analyses revealed that dehydration severity was not significantly associated with KDIGO staging ($\chi^2 = 0.262$, df = 4, $p = 0.992$), suggesting that even moderate fluid losses suffice to precipitate AKI across all severity stages in this cohort, a finding consistent with Bogari et al., who reported that renal injury could develop even in patients with clinically mild dehydration[4] However, dehydration severity was significantly associated with clinical outcome ($\chi^2 = 15.063$, df = 2, $p < 0.001$): all patients with mild dehydration achieved complete recovery, compared with 34.8% adverse outcomes in those with severe dehydration. This gradient mirrors findings from Almuradi et al. in a Pakistani cohort of 300 patients with acute gastroenteritis, where conservative management with timely fluid resuscitation and electrolyte correction led to recovery in 74.4% of patients, while delayed presentation and severe disease were associated with progression to dialysis and death [31].

No significant association was found between

age group (≤60 vs. >60 years) and KDIGO staging ($\chi^2 = 4.203$, $p = 0.122$) or clinical outcome (OR = 3.917, $p = 0.105$), though patients aged >60 years trended toward higher adverse outcome rates (25.0% vs. 7.8%). The limited sample size of elderly patients (n = 20) likely constrained statistical power. Regional commentary in the *Indian Journal of Nephrology* has highlighted that gastroenteritis-associated AKI remains a major contributor to the overall AKI burden in low- and middle-income countries and is largely preventable through early fluid resuscitation, while Feest et al. established that severe AKI incidence rises steeply with age in community-based populations [32], [33]. These trends warrant prospective evaluation in larger Nepali cohorts.

Hyperkalemia (>5.0 mEq/L) and metabolic acidosis were the strongest independent correlates of both AKI severity and adverse outcome in this study. All 18 hyperkalemic patients were classified as Stage 2 or Stage 3, with 72.2% in Stage 3, and 33.3% experienced partial recovery or death, compared with 5.7% of non-hyperkalemic patients (OR = 8.333, $p = 0.006$). Metabolic acidosis was similarly associated with KDIGO staging ($\chi^2 = 15.187$, $p < 0.001$) and outcome (OR = 9.103, $p = 0.029$), with 21.6% adverse outcomes in acidotic patients versus 2.9% in non-acidotic patients. These findings corroborate Hu et al., who demonstrated that metabolic acidosis at admission independently predicted both AKI development and in-hospital mortality in a large retrospective cohort [34]. Similarly, Takia et al., in a study of children with severe dehydration, found that higher grades of non-anion-gap metabolic acidemia were significantly associated with dyselectrolytemias, including hyperkalemia, and with progression to AKI [35]. In the resource-limited context of district hospitals in Nepal, where arterial blood gas analysis is not routinely available, serum potassium and bicarbonate represent practical, affordable triage markers for identifying patients at high risk of Stage 3 AKI and death, and should guide early escalation of care.

Our results are in line with studies conducted in other developing regions, demonstrating that AKI due to diarrheal illnesses remains largely preventable.[36] Strengthening community health education, ensuring access to clean drinking water, and promoting early oral rehydration therapy are critical preventive measures. Additionally, the establishment of hospital-based AKI surveillance systems, as recommended by the International Society of Nephrology's "0by25" initiative, could enhance early detection and reduce mortality in resource-limited settings [11]. This was a single-center, hospital-based study with a limited sample size, which may not represent the general population. Advanced diagnostic tests and long-term follow-up were not available, which may have underestimated intrinsic renal disease and delayed sequelae.

Conclusion

Acute kidney injury following acute gastroenteritis is a common clinical condition in this setting, predominantly affecting middle-aged males with moderate to severe dehydration. Infectious gastroenteritis was the leading etiology, and most patients presented with KDIGO Stage 1 AKI. Metabolic acidosis, hyponatremia, and hyperkalemia were frequent biochemical derangements. Complete renal recovery was achieved in the majority of patients, with mortality confined to those with Stage 3 AKI and septic shock, confirming the largely reversible nature of this condition when managed in a tertiary care setting.

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