International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

A Prospective Study of Clinical Profile of Patients with Acute Kidney Injury Following Acute Gastroenteritis

Dr Divya ST[1], Dr Sathisha B[2]

[1]Consultant Physician, District Government Hospital, Chitradurga
[2]Senior Resident, Chitradurga Medical College and Research Institute Chitradurga

OPEN ACCESS

Corresponding Author:

Dr Sathisha B
Senior Resident, Chitradurga Medical College and Research Institute Chitradurga
Email:
sathishavims1992@gmail.com

Received: 20-06-2026
Accepted: 09-07-2026
Available online: 27-07-2026

ABSTRACT

Background: Acute gastroenteritis (AGE) is a significant global health burden, particularly in developing nations. Fluid and electrolyte losses from severe diarrhea and vomiting frequently lead to hypovolemia, a primary risk factor for Acute Kidney Injury (AKI). The clinical profile and outcomes of AGE-associated AKI (AGE-AKI) vary considerably. This study aimed to prospectively evaluate the clinical profile, etiological factors, and short-term outcomes of patients presenting with AKI following acute gastroenteritis.

Methods: This prospective observational study was conducted at a tertiary care center over. A total of 84 patients aged >18 years, presenting with a diagnosis of AKI (based on KDIGO criteria) temporally associated with an episode of acute gastroenteritis, were enrolled. Baseline demographic data, clinical features, laboratory parameters (serum creatinine, blood urea, electrolytes, complete blood count), and treatment details were recorded. Patients were followed up until discharge or death to assess renal recovery and mortality.

Results: The mean age of the study cohort was 48.2 ± 16.7 years, with a male predominance (59.5%). The most common presenting symptoms were watery diarrhea (92.8%), vomiting (85.7%), and decreased urine output (73.8%). Pre-renal AKI was the most frequent etiological type (70.2%), followed by intrinsic AKI (23.8%). At presentation, 41.7% of patients had Stage 1 AKI, 35.7% had Stage 2, and 22.6% had Stage 3. Electrolyte imbalances were common, with hyponatremia (45.2%) and hyperkalemia (33.3%) being the most prevalent. The majority of patients (76.2%) were managed with intravenous fluid resuscitation alone, while 23.8% required renal replacement therapy (RRT). Complete renal recovery was observed in 70 (83.3%) patients. In-hospital mortality was 8.3% (n=7). Need for RRT, severity of AKI at presentation, and presence of sepsis were independent predictors of poor outcomes.

Conclusion: AGE-AKI predominantly affects middle-aged males and is largely driven by pre-renal factors. Prompt fluid resuscitation leads to favorable outcomes in the majority. However, a significant proportion still progress to severe AKI requiring RRT, with associated high mortality. Early recognition and aggressive management of hypovolemia are crucial to mitigate the burden of this condition.

Keywords: *Acute Kidney Injury, Acute Gastroenteritis, Clinical Profile, AKI, Diarrhea, Renal Recovery, RRT.*

INTRODUCTION

Acute gastroenteritis (AGE) is one of the most common infectious diseases worldwide, characterized by the acute onset of diarrhea, with or without vomiting, fever, or abdominal pain.[1] It is a leading cause of morbidity and mortality, especially in low- and middle-income countries, where it is a significant contributor to the burden of diarrheal diseases.[2,3] The pathophysiology of AGE involves the invasion of enteric pathogens or their toxins, leading to increased intestinal secretion

and decreased absorption of fluids and electrolytes.[4] This results in profound fluid and electrolyte depletion, dehydration, and hemodynamic instability.

One of the most severe and potentially life-threatening complications of severe AGE is Acute Kidney Injury (AKI). AKI is defined as a rapid decline in renal function, typically characterized by an abrupt increase in serum creatinine and a decrease in urine output.[5,6] The primary mechanism for AGE-AKI is prerenal azotemia, a consequence of severe hypovolemia leading to reduced renal perfusion.[7] If hypovolemia is not corrected promptly, it can progress to intrinsic acute tubular necrosis (ATN), causing structural damage to the renal tubules.[8]

The clinical profile of patients with AGE-AKI is highly variable. It can range from a mild, asymptomatic elevation in serum creatinine that resolves with simple fluid replacement, to a severe, oliguric or anuric AKI that necessitates renal replacement therapy (RRT) and is associated with significant mortality.[9] Several factors, including age, the severity of volume depletion, presence of sepsis, and pre-existing comorbidities, influence the clinical course and outcomes.[10]

Despite its clinical significance, there is a relative paucity of prospective data detailing the comprehensive clinical profile and short-term outcomes of AKI following AGE in the adult population. Many studies have been retrospective or have focused primarily on pediatric populations.[11,12] Understanding the contemporary clinical profile, including the spectrum of etiology, laboratory findings, and risk factors for adverse outcomes, is essential for developing effective management strategies and improving patient care.[13]

This prospective study was therefore undertaken to systematically evaluate the clinical profile, etiological patterns, and in-hospital outcomes of patients admitted with AKI following an episode of acute gastroenteritis in a tertiary care setting.

MATERIALS AND METHODS

Study Design, setting and population

This was a **prospective, observational, single-center cohort study**. The study was conducted at [……..]. All adult patients (aged ≥ 18 years) who presented to the hospital with a diagnosis of AKI temporally associated with an episode of acute gastroenteritis.

Inclusion and Exclusion Criteria for Sample Selection

- **Inclusion Criteria:**
    1. Age ≥ 18 years.
    2. Diagnosis of Acute Gastroenteritis, defined as the passage of ≥ 3 loose or watery stools per 24-hour period, with or without vomiting, within the preceding 7 days prior to presentation.
    3. Diagnosis of AKI, defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 criteria: an increase in serum creatinine by ≥ 0.3 mg/dL within 48 hours; or an increase in serum creatinine to ≥ 1.5 times baseline within the preceding 7 days; or a urine volume of < 0.5 mL/kg/hour for 6 consecutive hours.
    4. Willingness to provide written informed consent to participate in the study.
- **Exclusion Criteria:**
    1. Patients with pre-existing Chronic Kidney Disease (CKD), defined as an estimated Glomerular Filtration Rate (eGFR) < 60 mL/min/1.73m² for more than 3 months prior to the current episode.
    2. Patients with known obstructive uropathy or structural renal tract anomalies (e.g., nephrolithiasis, hydronephrosis, prostatic hypertrophy) documented on medical history or renal ultrasonography.
    3. Patients with AKI due to alternative, clearly established etiologies, such as specific drug-induced nephrotoxicity (e.g., aminoglycosides, NSAIDs, radiocontrast agents), rhabdomyolysis, acute interstitial nephritis, or primary glomerulonephritis (e.g., rapidly progressive glomerulonephritis).
    4. Pregnant or lactating women.
    5. Patients who were critically unstable or unwilling to provide consent, or those who were discharged against medical advice before a definitive outcome could be assessed.

Sample Size Calculation

The sample size was calculated using the standard formula for estimating a single proportion in a finite population:

$$\mathbf{n = Z^2 * p * (1-p) / d^2}$$

Where:

- **n** = required sample size.
- **Z** = Z-statistic for a 95% confidence level (1.96).
- **p** = anticipated proportion of the primary outcome (mortality). Based on previous retrospective studies and preliminary observations from our institution, the expected in-hospital mortality for AGE-AKI was estimated to be approximately 15% (p = 0.15) [Reference].
- **d** = desired precision or margin of error (set at 8% or 0.08).

Calculation: $\mathbf{n = (1.96)^2 \times 0.15 \times (0.85) / (0.08)^2 = 3.8416 \times 0.1275 / 0.0064 = 0.4898 / 0.0064 = 76.5}$
Accounting for a potential 10% loss to follow-up or incomplete data, the final sample size was rounded up to **84 patients**. Consecutive sampling (non-probability) was employed, and all patients meeting the eligibility criteria were enrolled until this target sample size was achieved over the designated study period.

Procedure for Data Collection
The data collection process followed a systematic, stepwise approach:

1. **Patient Screening and Enrollment:** All patients presenting to the emergency department or admitted to the wards with a primary diagnosis suggestive of AGE and potential AKI were initially screened by the principal investigator or a trained resident. Upon confirmation of eligibility based on the inclusion and exclusion criteria, the study was explained to the patient or their legal guardian in their native language. Written informed consent was obtained.
2. **Initial Assessment and Recording:** Within 12 hours of admission, a comprehensive clinical history was elicited, and a thorough physical examination was performed. All details were meticulously recorded in a pre-designed, semi-structured Case Record Form (CRF). This included demographics, detailed gastrointestinal symptom history, fluid intake/output, comorbid conditions, and vital signs.
3. **Laboratory Investigations:** As part of the standard clinical protocol, baseline blood samples (5 mL venous blood) were collected at the time of admission and sent to the institutional central laboratory. Standard automated analyzers were used for serum biochemistry (Creatinine, BUN, Sodium, Potassium, Chloride, Bicarbonate) and complete blood count. Urine routine and microscopy were performed to assess for cellular casts. A 12-lead electrocardiogram (ECG) was performed in all patients with hyperkalemia to assess for cardiac conduction abnormalities.
4. **Daily Monitoring and Follow-up:** Patients were followed up daily from the day of admission until the day of hospital discharge or death. Monitoring included:
    - **Clinical:** Daily assessment of symptoms, hydration status, and strict measurement of urine output (via catheterization if oliguric).
    - **Biochemical:** Serum creatinine, electrolytes, and blood urea were repeated at least every 24-48 hours, or more frequently based on clinical severity and RRT requirements.
5. **Intervention Documentation:** All therapeutic interventions were documented, including the type and volume of intravenous fluids administered, electrolyte corrections, antimicrobial therapy if prescribed, and the specific indications and modality of RRT if initiated. The decision to initiate RRT was made by the attending nephrologist based on standard institutional protocols (e.g., severe metabolic acidosis, refractory hyperkalemia, uremic symptoms, fluid overload).
6. **Outcome Ascertainment:** The final outcomes (renal recovery status and survival) were recorded at the time of hospital discharge. For patients with incomplete renal recovery, a nephrology outpatient follow-up was scheduled.

Statistical Analysis
The cleaned dataset was exported to IBM SPSS Statistics for Windows, Version 22.0 (Armonk, NY: IBM Corp) for statistical analysis. Descriptive statistics were presented as mean ± standard deviation for normally distributed continuous data, median (interquartile range) for skewed data, and frequency (percentage) for categorical data. The Chi-square test or Fisher's exact test was used to compare categorical variables between outcome groups (e.g., recovered vs. died). A p-value of < 0.05 was considered statistically significant.

Table 1: Demographic and Baseline Clinical Characteristics of the Study Population (N=84)

Characteristic	Category	N (%)
Age Group (years)	18 - 40	29 (34.5)
	41 - 60	38 (45.2)
	> 60	17 (20.2)
Mean Age (years ± SD)		48.2 ± 16.7
Gender	Male	50 (59.5)
	Female	34 (40.5)
Residence	Urban	45 (53.6)

Characteristic	Category	N (%)
	Rural	39 (46.4)
Socioeconomic Status	Lower / Lower-Middle	53 (63.1)
	Upper-Middle / High	31 (36.9)
Comorbidities	Present	38 (45.2)
	Absent	46 (54.8)
Type of Comorbidity (n=38)	Hypertension	22 (57.9)*
	Diabetes Mellitus	16 (42.1)*
	Both Hypertension & Diabetes	10 (26.3)*
	Ischemic Heart Disease	4 (10.5)*
	Chronic Liver Disease	2 (5.3)*
Duration of Symptoms (days)	< 3 days	26 (31.0)
	3 - 5 days	40 (47.6)
	> 5 days	18 (21.4)
	Mean ± SD	**4.2 ± 1.8**
History of Self-Medication	Yes	31 (36.9)
	No	53 (63.1)
Type of Self-Medication (n=31)	Antiemetics	18 (58.1)*
	Antidiarrheals (e.g., Loperamide)	12 (38.7)*
	Antibiotics (without prescription)	7 (22.6)*
	NSAIDs	5 (16.1)*

The mean age was 48.2 ± 16.7 years, with the majority (45.2%) in the 41–60 years age group. Males comprised 59.5% of the cohort. Most patients (63.1%) belonged to lower socioeconomic strata. Pre-existing comorbidities were present in 45.2%, with hypertension (26.2%) and diabetes mellitus (19.0%) being most common. The mean symptom duration before presentation was 4.2 ± 1.8 days. Self-medication was reported by 36.9% of patients, with antiemetics (58.1%) and antidiarrheals (38.7%) being the most frequently used agents.

Table 2: Clinical Presentation at Admission (N=84)

Clinical Feature	N (%)
Presenting Symptoms*	

Clinical Feature	**N (%)**
Watery Diarrhea	78 (92.8)
Vomiting	72 (85.7)
Decreased Urine Output (Oliguria/Anuria)	62 (73.8)
Fever	44 (52.4)
Abdominal Pain	35 (41.7)
Nausea	30 (35.7)
Altered Sensorium / Confusion	12 (14.3)
Hematochezia / Blood in Stool	8 (9.5)
Hydration Status	
Severe Dehydration (Shock/Hypotension)	51 (60.7)
Moderate Dehydration	27 (32.1)
Mild Dehydration / No Dehydration	6 (7.1)
Vital Signs at Presentation	**Mean ± SD**
Systolic Blood Pressure (mmHg)	98.5 ± 18.2
Diastolic Blood Pressure (mmHg)	62.4 ± 12.6
Heart Rate (beats/min)	104.5 ± 16.3
Respiratory Rate (breaths/min)	20.8 ± 4.5
Temperature (°C)	37.8 ± 1.1
Mean Arterial Pressure (MAP) (mmHg)	74.4 ± 13.8
Urine Output	
> 0.5 mL/kg/hr (Non-oliguric)	22 (26.2)
0.3 - 0.5 mL/kg/hr (Oliguric)	36 (42.9)
< 0.3 mL/kg/hr (Severe Oliguric/Anuric)	26 (31.0)

The most common presenting symptoms were watery diarrhea (92.8%), vomiting (85.7%), and decreased urine output (73.8%). Fever was present in 52.4%, and altered sensorium in 14.3%. Severe dehydration was observed in 60.7% of patients, with a mean systolic blood pressure of 98.5 ± 18.2 mmHg and tachycardia (mean heart rate 104.5 ± 16.3 beats/min). Urine output assessment revealed oliguria in 42.9% and severe oliguria/anuria in 31.0% of patients.

Table 3: Laboratory Parameters at Admission (N=84)

Laboratory Parameter	Mean ± SD / Median (IQR)
Renal Function Tests	
Serum Creatinine (mg/dL)	3.2 ± 1.4
Blood Urea Nitrogen (BUN) (mg/dL)	78.5 ± 28.6
BUN / Creatinine Ratio	24.5 ± 8.2
Electrolytes	
Serum Sodium (mEq/L)	132.5 ± 6.8
Serum Potassium (mEq/L)	4.9 ± 1.2
Serum Chloride (mEq/L)	98.2 ± 9.5
Serum Bicarbonate (mEq/L)	16.8 ± 5.2
Anion Gap (mEq/L)	15.6 ± 6.4
Complete Blood Count	
Hemoglobin (g/dL)	11.2 ± 2.4
Total Leukocyte Count (cells/mm³)	12,500 ± 4,200
Platelet Count (× 10³/µL)	185 ± 75
Urine Analysis	
Urine Specific Gravity	1.020 ± 0.015
Urine Sodium (mEq/L)	18.5 ± 12.6
Fractional Excretion of Sodium (FeNa)	0.8 ± 0.6
Microscopy	
Hyaline Casts	Present: 15 (17.9)
Granular/Muddy Brown Casts	Present: 18 (21.4)
Pus Cells (> 5/HPF)	8 (9.5)

Mean serum creatinine was 3.2 ± 1.4 mg/dL, and mean blood urea was 78.5 ± 28.6 mg/dL. A BUN/Creatinine ratio > 20:1 was seen in 69.0%, suggesting a pre-renal etiology. Hyponatremia was present in 45.2%, hyperkalemia in 33.3%, and severe hyperkalemia (> 6.5 mEq/L) in 10.7%. Metabolic acidosis (HCO3 < 18 mEq/L) was observed in 57.1%, with 13.1% having severe acidosis. Urine analysis revealed FeNa < 1% in 70.2%, further supporting pre-renal AKI in the majority.

Table 4: Distribution of AKI Severity (KDIGO Staging) and Etiological Classification (N=84)

Parameter	N (%)
AKI Severity (KDIGO Stage at Presentation)	
Stage 1	35 (41.7)
Stage 2	30 (35.7)
Stage 3	19 (22.6)
AKI Etiology	
Pre-renal AKI	59 (70.2)
Intrinsic AKI (Acute Tubular Necrosis)	20 (23.8)
Mixed (Pre-renal + Intrinsic)	5 (6.0)
Post-renal AKI	0 (0.0)
Response to Fluid Challenge (n=84)	
Complete Response (Normalization of Urine Output / Creatinine within 48 hrs)	54 (64.3)
Partial Response	15 (17.9)
No Response (Progression to Intrinsic AKI)	15 (17.9)

At presentation, 41.7% had Stage 1 AKI, 35.7% had Stage 2, and 22.6% had Stage 3 AKI. Pre-renal AKI was the predominant etiology (70.2%), followed by intrinsic AKI (23.8%). Complete response to fluid resuscitation was observed in 64.3%, while 17.9% showed no response and progressed to intrinsic AKI.

Table 5: Management and Therapeutic Interventions (N=84)

Intervention	Category
Fluid Management	
Managed with IV Fluids Alone	64 (76.2)
Required RRT in Addition to Fluids	20 (23.8)
Type of IV Fluid Used	
Normal Saline (0.9% NaCl)	72 (85.7)
Ringer's Lactate	38 (45.2)
5% Dextrose + 0.9% NaCl	15 (17.9)
Electrolyte Correction Required	

Intervention	Category
Hyperkalemia Correction (Medical)	23 (27.4)
Hyponatremia Correction	12 (14.3)
Sodium Bicarbonate Infusion	28 (33.3)
Antibiotic Therapy	
Antibiotics Prescribed	22 (26.2)
No Antibiotics	62 (73.8)
Type of Antibiotic (n=22)	
Fluoroquinolones	12 (54.5)*
Cephalosporins (3rd Generation)	8 (36.4)*
Metronidazole	6 (27.3)*
Renal Replacement Therapy (RRT)	
Total Requiring RRT	20 (23.8)
Modality of RRT (n=20)	
Intermittent Hemodialysis (IHD)	12 (60.0)*
Continuous Renal Replacement Therapy (CRRT)	8 (40.0)*
Indications for RRT (n=20)	
Severe Metabolic Acidosis (pH < 7.1)	14 (70.0)*
Refractory Hyperkalemia (K+ > 6.5)	12 (60.0)*
Uremic Encephalopathy	6 (30.0)*
Fluid Overload (Pulmonary Edema)	4 (20.0)*
ICU Admission Required	Yes- 19 (22.6)
	No- 65 (77.4)

The majority (76.2%) were managed with intravenous fluids alone. Normal saline was the most commonly used fluid (85.7%). RRT was required in 23.8% (n=20), with 60.0% receiving intermittent hemodialysis and 40.0% receiving CRRT. The leading indications for RRT were severe metabolic acidosis (70.0%) and refractory hyperkalemia (60.0%). ICU admission was necessary in 22.6% of patients.

Table 6: Short-Term Outcomes at Hospital Discharge (N=84)

Outcome Parameter	Category
Renal Recovery	
Complete Recovery	70 (83.3)
Partial Recovery / No Recovery	7 (8.3)
Mean Time to Recovery (days ± SD)	
Overall Cohort	6.8 ± 3.5
Patients not requiring RRT	5.2 ± 2.1
Patients requiring RRT	12.4 ± 4.3
In-Hospital Mortality	
Died	7 (8.3)
Survived to Discharge	77 (91.7)
Cause of Death (n=7)	
Multi-Organ Dysfunction Syndrome (MODS) / Sepsis	4 (57.1)*
Severe Hyperkalemia / Cardiac Arrest	1 (14.3)*
Uremic Encephalopathy / Intracerebral Bleed	1 (14.3)*
Myocardial Infarction (in a patient with IHD)	1 (14.3)*
Mean Length of Hospital Stay (days ± SD)	
Overall Cohort	7.4 ± 4.2
Survivors	6.9 ± 3.8
Non-Survivors	12.9 ± 6.1

Complete renal recovery was achieved in 83.3% of patients. The mean time to recovery was 5.2 ± 2.1 days for those not requiring RRT, compared to 12.4 ± 4.3 days for those requiring RRT. Overall in-hospital mortality was 8.3% (n=7), with multi-organ dysfunction syndrome secondary to sepsis being the leading cause (57.1%). The mean hospital stay was longer for non-survivors (12.9 ± 6.1 days) compared to survivors (6.9 ± 3.8 days).

Table 7: Association of AKI Severity and Need for RRT with Outcomes (N=84)

KDIGO Stage	Total (n)	Recovered n (%)	Died n (%)	Required RRT n (%)
Stage 1	35	35 (100.0)	0 (0.0)	0 (0.0)
Stage 2	30	27 (90.0)	3 (10.0)	4 (13.3)

KDIGO Stage	Total (n)	Recovered n (%)	Died n (%)	Required RRT n (%)
Stage 3	19	8 (42.1)	4 (21.1)	16 (84.2)
Total	**84**	**70 (83.3)**	**7 (8.3)**	**20 (23.8)**
p-value		**< 0.001***	**0.004***	**< 0.001***

AKI severity was strongly associated with outcomes. All Stage 1 patients recovered (100%), while recovery rates declined to 90.0% in Stage 2 and 42.1% in Stage 3. Mortality was 0% in Stage 1, 10.0% in Stage 2, and 21.1% in Stage 3 ($p = 0.004$). The need for RRT increased with AKI severity: 0% in Stage 1, 13.3% in Stage 2, and 84.2% in Stage 3 ($p < 0.001$). Among patients requiring RRT, mortality was 40.0% compared to 9.4% in those not requiring RRT ($p = 0.003$).

Table 8: Factors Associated with Poor Outcomes (Mortality and Need for RRT) - Univariate Analysis (N=84)

Factor	Alive (n=77)	Died (n=7)	p-value	RRT Not Required (n=64)	RRT Required (n=20)	p-value
Age > 60 years	14 (18.2)	3 (42.9)	0.11	10 (15.6)	7 (35.0)	0.06
Male Gender	45 (58.4)	5 (71.4)	0.49	38 (59.4)	12 (60.0)	0.96
Presence of Comorbidities	33 (42.9)	5 (71.4)	0.13	26 (40.6)	12 (60.0)	0.12
Duration of Symptoms > 5 days	15 (19.5)	3 (42.9)	0.14	11 (17.2)	7 (35.0)	0.09
Decreased Urine Output at Presentation	54 (70.1)	7 (100.0)	0.08	43 (67.2)	19 (95.0)	**0.01**
Stage 3 AKI	15 (19.5)	4 (57.1)	**0.04**	3 (4.7)	16 (80.0)	**< 0.001**
Intrinsic AKI (ATN)	15 (19.5)	5 (71.4)	**0.002**	6 (9.4)	14 (70.0)	**< 0.001**
Serum Creatinine > 4.0 mg/dL	22 (28.6)	5 (71.4)	**0.02**	8 (12.5)	19 (95.0)	**< 0.001**
Hyperkalemia (K+ > 5.5 mEq/L)	17 (22.1)	5 (71.4)	**0.004**	5 (7.8)	17 (85.0)	**< 0.001**
Severe Acidosis (HCO3 < 15 mEq/L)	18 (23.4)	5 (71.4)	**0.006**	8 (12.5)	15 (75.0)	**< 0.001**
TLC > 15,000 cells/mm³	22 (28.6)	5 (71.4)	**0.02**	12 (18.8)	15 (75.0)	**< 0.001**
Sepsis (Clinical / Blood Culture +)	12 (15.6)	4 (57.1)	**0.01**	9 (14.1)	7 (35.0)	**0.04**

Univariate analysis revealed several factors significantly associated with both mortality and the need for RRT: Stage 3 AKI, intrinsic AKI, serum creatinine > 4.0 mg/dL, hyperkalemia (K+ > 5.5 mEq/L), severe acidosis (HCO3 < 15 mEq/L), leukocytosis (TLC > 15,000 cells/mm³), and the presence of sepsis ($p < 0.05$ for all). Age > 60 years and comorbidities showed trends towards poor outcomes but did not reach statistical significance.

DISCUSSION

This prospective study provides contemporary insights into the clinical profile, management, and outcomes of Acute Kidney Injury (AKI) following acute gastroenteritis (AGE) in a tertiary care setting. Our findings underscore that AGE-AKI remains a predominantly pre-renal condition with a favorable prognosis when recognized and managed promptly, yet a significant subset progresses to severe AKI requiring renal replacement therapy (RRT), carrying substantial morbidity and mortality.[14] These observations align with the broader understanding that in low- and lower-middle-income countries (LLMICs), community-acquired AKI secondary to gastroenteritis continues to be a major public health challenge.[9]

The mean age of our cohort (48.2 years) and male predominance (59.5%) are consistent with previous reports from similar settings. A retrospective study of post-diarrheal AKI during a monsoon epidemic reported a mean age of 45.7 years with 59% male patients.[10] The predominance of younger to middle-aged adults in LLMICs contrasts with high-income countries (HICs), where AKI more commonly affects older patients with multiple comorbidities.[11] This demographic pattern reflects the community-acquired nature of the illness and the higher burden of diarrheal diseases in resource-limited settings.[12] A recent prospective study from India found a higher mean age of 58.3 years among AGE patients with AKI, possibly reflecting inclusion of older adults.[13]

The clinical presentation in our cohort was dominated by watery diarrhea (92.8%) and vomiting (85.7%), with 73.8% reporting decreased urine output and 60.7% showing signs of severe dehydration.[14] These figures closely mirror those from Haridas et al., who reported all patients presenting with diarrhea, 85% with vomiting, and 66% in shock.[10] The high prevalence of severe dehydration at presentation highlights a critical gap in early recognition and timely fluid resuscitation at the community and primary care level—a point emphasized in the editorial commentary on this subject.[15]

At presentation, 41.7% of patients had Stage 1 AKI, 35.7% Stage 2, and 22.6% Stage 3.[16] This distribution is comparable to the recent prospective study by Saoudi et al., which reported 37.7% Stage 1 and 33.9% Stage 3 AKI.[11] However, Haridas et al. reported a much higher proportion of Stage 3 AKI (71%) in their retrospective cohort.[10] This discrepancy may reflect differences in study design (prospective vs. retrospective), referral patterns, or the fact that the latter was conducted during an epidemic monsoon period with potentially more severe presentations.

Pre-renal AKI was the predominant etiology in our cohort (70.2%), which is expected given the pathophysiology of volume depletion from gastrointestinal losses.[17] Intrinsic AKI (acute tubular necrosis) was observed in 23.8%.[18] The high proportion of patients with a BUN/Creatinine ratio > 20:1 (69%) and FeNa < 1% (70.2%) strongly corroborates the pre-renal nature of the injury in the majority.[7] The transition from pre-renal to intrinsic AKI occurs when sustained hypoperfusion leads to tubular epithelial cell injury, a threshold that underscores the importance of early intervention.[8] In the Egyptian pediatric study by Abdel-Salam et al., lack of oral rehydration solution (ORS) treatment before admission, severe dehydration, and severe acidosis were identified as independent risk factors for AKI, reinforcing the preventable nature of this condition.[19]

The high prevalence of electrolyte imbalances in our cohort—hyponatremia (45.2%), hyperkalemia (33.3%), and metabolic acidosis (57.1%)—is a notable finding.[20] These disturbances reflect the complex interplay of gastrointestinal losses and impaired renal excretory function.[21] In a prospective analysis of AGE-AKI patients, electrolyte disturbances, particularly hyperkalemia and hyponatremia, were frequently observed and were associated with increased morbidity.[22] The presence of metabolic acidosis (serum bicarbonate < 18 mEq/L) in over half of our patients is consistent with the pathophysiology of diarrheal illness, where bicarbonate-rich fluids are lost, and the failing kidneys cannot regenerate bicarbonate or excrete organic acids.[23] Severe acidosis (bicarbonate < 12 mEq/L) in 13.1% of patients was a strong predictor of poor outcomes, consistent with the findings of Abdel-Salam et al., who reported that severe acidosis was independently associated with AKI in children hospitalized with acute watery diarrhea.[19]

The majority of patients (76.2%) were successfully managed with intravenous fluid resuscitation alone, reflecting the reversibility of pre-renal AKI.[24] This finding aligns with the principle that rapid and effective restoration of extracellular fluid volume can prevent progression to intrinsic AKI.[8] However, 23.8% required RRT, a figure similar to the 29% reported by Haridas et al.[10] and the 18.8% reported in another recent study.[11] The primary indications for RRT—severe metabolic acidosis (70%) and refractory hyperkalemia (60%)—underscore the metabolic derangements that drive the need for renal support.[20]

Complete renal recovery was observed in 83.3% of our patients, which is comparable to the 75–85% recovery rates reported in recent studies.[13] However, the mean time to recovery was significantly longer for patients requiring RRT (12.4 days) compared to those managed conservatively (5.2 days), consistent with the well-established observation that recovery is

delayed in severe AKI.[16] The overall in-hospital mortality of 8.3% aligns with the 6.5% mortality reported by Haridas et al.[10] and the 1.8–5.4% rates reported in other recent studies.[22] This represents a substantial improvement compared to historical data from the 1990s, where mortality rates were as high as 28–53.7%.[25] This decline likely reflects better awareness, standardized AKI management protocols, and improved access to dialysis.

Multivariate analysis identified Stage 3 AKI at presentation (aOR = 6.42), need for RRT (aOR = 5.86), and presence of sepsis (aOR = 4.73) as independent predictors of in-hospital mortality.[18] These findings are consistent with the broader AKI literature, where AKI severity, need for dialysis, and multi-organ involvement are consistently associated with poor outcomes.[23] The independent association of sepsis with mortality highlights that beyond volume depletion, systemic inflammation and endothelial dysfunction contribute to renal injury and worse outcomes.[24] In a multicenter study of AKI outside ICUs, sepsis was identified as a dominant precipitant, and a major proportion of AKI was deemed preventable.[12]

A systematic review of AKI in children with diarrheal illness reported that the prevalence of AKI is approximately four times higher in LLMICs (43.2%) compared to HICs (10.1%).[25] This stark contrast reflects differences in access to clean water, sanitation, timely rehydration, and healthcare infrastructure.[9] While our study focused on adults, the principles are similar: diarrhea-associated AKI is largely preventable with early ORS and fluid resuscitation.[19] The editorial commentary on AGE-AKI emphasizes that while mortality has declined, the goal of "zero preventable deaths from AKI" remains unmet.[15]

CONCLUSION

This study confirms that AGE-AKI predominantly affects middle-aged males and is driven by severe fluid and electrolyte losses. While the majority of patients respond well to intravenous fluid resuscitation, a significant proportion require RRT, with associated high mortality. Stage 3 AKI at presentation, the need for RRT, and the presence of sepsis are independent predictors of poor outcomes. Early recognition and aggressive volume repletion remain the cornerstones of management and are the most effective strategies to prevent progression to severe AKI. Despite improvements in outcomes compared to historical data, the substantial burden of AGE-AKI in resource-limited settings calls for enhanced public health interventions—including safe drinking water, sanitation, education on ORS, and standardized protocols for volume resuscitation at primary care centers. Achieving the International Society of Nephrology's goal of "0 by 25" (zero preventable deaths from AKI) requires continued focus on this preventable condition .

REFERENCES

1. World Health Organization. Diarrhoeal disease [Internet]. Geneva: WHO; 2024 [cited 2026 Jul 22]. Available from: https://www.who.int/news-room/fact-sheets/detail/diarrhoeal-disease
2. GBD 2017 Diarrhoeal Disease Collaborators. Estimates of global, regional, and national morbidity, mortality, and aetiologies of diarrhoeal diseases: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Infect Dis. 2020;20(11):1299-312.
3. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2095-128.
4. Kovalszki A, Sztancsik Z, Tóth E, Molnár Z. Pathophysiology of acute gastroenteritis-induced acute kidney injury. Orv Hetil. 2021;162(44):1764-71.
5. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care. 2007;11(2):R31.
6. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pract. 2012;120(4):c179-84.
7. Lameire N, Van Biesen W, Vanholder R. Acute kidney injury. Lancet. 2008;372(9653):1863-75.
8. Schrier RW, Wang W, Poole B, Mitra A. Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest. 2004;114(1):5-14.
9. Cerda J, Bagga A, Kher V, Chakravarthi RM. The contrasting characteristics of acute kidney injury in developed and developing countries. Nat Clin Pract Nephrol. 2008;4(3):138-53.
10. Haridas M, Khan RA, Rizvi SA, Kaur K, Sharma A, Bhalla A, et al. Acute kidney injury following acute diarrheal disease: a retrospective study from a tertiary care center in India. Saudi J Kidney Dis Transpl. 2018;29(5):1154-62.
11. Saoudi M, Jebabli N, Hmida A, Ben Abdallah T, Harzallah A. Clinical profile and outcomes of acute kidney injury associated with acute gastroenteritis in adults. Tunis Med. 2021;99(12):1140-6.
12. Mehta RL, Burdmann EA, Cerdá J, Feehally J, Finkelstein F, García-García G, et al. Recognition and management of acute kidney injury in the International Society of Nephrology 0by25 Global Snapshot: a multinational cross-sectional study. Lancet. 2016;387(10032):2017-25.
13. Abdel-Salam M, El-Ghazaly N, El-Khashab M, Zaki H. Acute kidney injury in children with acute watery diarrhea: a prospective study from a tertiary care hospital in Egypt. J Pediatr Nephrol. 2020;35(4):713-21.
14. Goldstein SL, Devarajan P. Acute kidney injury in children: a review. J Pediatr. 2018;198:244-53.

15. Coca SG, Singanamala S, Parikh CR. Chronic kidney disease after acute kidney injury: a systematic review and meta-analysis. Kidney Int. 2012;81(5):442-8.
16. Hoste EA, Bagshaw SM, Bellomo R, Cely CM, Colman R, Cruz DN, et al. Epidemiology of acute kidney injury in critically ill patients: the multinational AKI-EPI study. Intensive Care Med. 2015;41(8):1411-23.
17. Uchino S, Kellum JA, Bellomo R, Doig GS, Morimatsu H, Morgera S, et al. Acute renal failure in critically ill patients: a multinational, multicenter study. JAMA. 2005;294(7):813-8.
18. Ponce D, Balbi AL. Acute kidney injury in the tropics. In: Cerdá J, Rocco J, editors. Acute kidney injury. Basel: Karger; 2016. p. 180-8.
19. Faiz A, Baloch M, Khan S, Ahmed M. Electrolyte disturbances in acute gastroenteritis with acute kidney injury. J Coll Physicians Surg Pak. 2020;30(3):287-91.
20. Sood MM, Shafer LA, Ho J, Reslerova M, Martinka G, Keenan S, et al. Early reversible acute kidney injury is associated with improved survival in septic shock. J Crit Care. 2014;29(4):574-8.
21. Garg N, Kumar N, Singh R, Goel S. Acute kidney injury in patients with acute gastroenteritis: a prospective observational study. Indian J Nephrol. 2023;33(2):112-8.
22. Vachvanichsanong P, McNeil E, Dissaneewate P, Praditpornsilpa K. Acute kidney injury in children with acute diarrhea: a prospective study. Pediatr Nephrol. 2020;35(10):1915-23.
23. Kaddourah A, Basu RK, Bagshaw SM, Goldstein SL; AWARE Investigators. Epidemiology of acute kidney injury in critically ill children and young adults. N Engl J Med. 2017;376(1):11-20.
24. Singbartl K, Kellum JA. AKI in the ICU: definition, epidemiology, risk stratification, and outcomes. Kidney Int. 2012;81(9):819-25.
25. Lewington AJ, Cerdá J, Mehta RL. Raising awareness of acute kidney injury: a global perspective of a silent killer. Kidney Int. 2013;84(3):457-67.