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Acute Tubular Necrosis in Patients with Acute Gastroenteritis-Associated Acute Kidney Injury: Incidence, Risk Factors, and Clinical Outcomes – A Prospective Observational Study

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ABSTRACT

BACKGROUND & OBJECTIVE: Gastroenteritis is a common cause of community acquired AKI in South Asia. Often, the condition progresses to ATN as the result of protracted hypovolemia. A prospective study was conducted to assess the frequency, risk factors and short- and mid-term outcomes of ATN in a mixed ward-ICU population.

METHODOLOGY: 712 consecutive adult patients with acute gastroenteritis and AKI admitted at the Northwest General Hospital and Research Center, Peshawar, were enrolled. The diagnosis of ATN was based on clinical picture, laboratory parameters, microscopic examination of urinary sediment and the BUN:Serum Cr ratio. Gastroenteritis was the presenting feature in 83% of cases.

RESULTS: ATN was diagnosed in 236 patients (33.1%). Patients with ATN had higher rates of severe dehydration, electrolyte imbalance, delayed presentation and elevated BUN:Serum Cr ratio (P<0.001). Outcomes of the patients with ATN were much worse in terms of longer hospital stay (9.2±4.1 vs. 4.6±2.3 days, P<0.001), renal replacement therapy (11.4% vs. 1.3%, P<0.001), lower rate of renal recovery at 90 days (64.8% vs. 93.1%, P<0.001) and higher in-hospital mortality (8.5% vs. 1.5%, P<0.001).

CONCLUSION: In this region of a high disease burden, ATN develops in one third of gastroenteritis-induced AKI cases and predicts bad prognosis. Implementation of early risk stratification based on biochemical indicators and appropriate hydration strategies is urgent.

KEY WORDS: Acute tubular necrosis; acute kidney injury; acute gastroenteritis; dehydration; blood urea nitrogen-to-creatinine ratio; Pakistan

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INTRODUCTION

Acute kidney injury (AKI) is a complex clinical syndrome characterized by an abrupt decline in renal function, resulting in the accumulation of nitrogenous waste products and disturbances in fluid, electrolyte, and acid–base homeostasis. It constitutes a major global health problem because of its high incidence, substantial mortality, prolonged hospitalization, and potential progression to chronic kidney disease. The epidemiology and outcomes of AKI vary considerably across geographical regions, healthcare systems, socioeconomic settings, and patient populations.[1] These disparities are particularly evident when AKI patterns in low- and middle-income countries are compared with those observed in high-income countries.

In resource-limited countries, community-acquired AKI is relatively common and frequently develops following preventable or treatable conditions such as acute gastroenteritis, hypovolemia, sepsis, obstetric complications, infectious diseases, and exposure to nephrotoxic substances. By contrast, AKI in high-resource settings is predominantly hospital-acquired and usually occurs in older patients with multiple comorbidities, critical illness, major surgery, sepsis, or exposure to several nephrotoxic medications.[2,3] Delayed access to healthcare, inadequate availability of diagnostic facilities, poor awareness of early warning symptoms, and limited access to renal replacement therapy may further aggravate the severity and adverse consequences of AKI in low-resource regions.

Acute gastroenteritis is an important precipitating factor for community-acquired AKI, particularly in tropical and subtropical regions. It may be caused by a wide range of bacterial, viral, and parasitic pathogens and is commonly accompanied by vomiting, diarrhea, fever, and reduced oral intake. Persistent gastrointestinal losses can produce profound intravascular volume depletion, hypotension, electrolyte disturbances, and impaired renal perfusion. During the initial stages, this decline in kidney function is generally considered a functional and potentially reversible pre-renal process. Prompt recognition and appropriate fluid replacement may restore renal perfusion and prevent structural kidney damage.[4]

However, when renal hypoperfusion is severe or prolonged, compensatory mechanisms become insufficient and ischemic injury to the renal tubular epithelium may occur. This transition from pre-renal azotemia to intrinsic renal damage most commonly manifests as acute tubular necrosis (ATN), also referred to

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as acute tubular injury. ATN is among the most frequent causes of intrinsic AKI and is characterized by tubular epithelial-cell dysfunction, loss of cellular polarity, disruption of the tubular basement membrane, intratubular obstruction, inflammation, and impairment of glomerular filtration.[5]Although ischemic ATN is potentially reversible, recovery may be delayed and patients may require intensive monitoring, renal replacement therapy, or prolonged hospitalization. Severe episodes can also increase the subsequent risk of chronic kidney disease, recurrent AKI, cardiovascular complications, and death.

Pakistan and other South Asian countries continue to experience a considerable burden of acute gastroenteritis because of unsafe drinking water, inadequate sanitation, food contamination, overcrowding, seasonal outbreaks, and limited access to timely medical care. Gastroenteritis therefore remains an important cause of emergency presentation and hospital admission in the region.[6] Patients may seek medical attention only after several days of persistent diarrhea and vomiting, by which time substantial dehydration, oliguria, hypotension, and biochemical abnormalities may already be present. Delayed or inadequate rehydration increases the probability that reversible pre-renal dysfunction will progress to established tubular injury.

Regional studies have reported that approximately 20–30% of patients hospitalized with acute gastroenteritis may develop some degree of AKI, although the reported frequency varies according to the population studied, severity of illness, definition of AKI, comorbid conditions, and availability of early treatment.[7] A proportion of these patients may subsequently develop ATN, particularly in the presence of prolonged hypotension, severe dehydration, sepsis, advanced age, pre-existing renal impairment, diabetes mellitus, cardiovascular disease, or exposure to nephrotoxic medications. Identifying the clinical and biochemical factors associated with this progression is important because early recognition may facilitate timely fluid resuscitation, withdrawal of nephrotoxic agents, correction of electrolyte abnormalities, and referral for specialist renal care.

The diagnosis and classification of AKI are principally based on changes in serum creatinine concentration and urine output. In resource-limited settings, routine biochemical parameters—including serum urea, serum creatinine, electrolytes, and the blood urea nitrogen-to-creatinine ratio—remain central to the initial evaluation of patients with suspected AKI.[8] Nevertheless, serum creatinine is an imperfect and relatively delayed marker of renal dysfunction. Its concentration may be influenced by age, sex, muscle mass, nutritional status, volume of distribution, and baseline renal function. Furthermore, a reliable pre-illness serum creatinine value is frequently unavailable in patients presenting with community-acquired disease.

Distinguishing uncomplicated pre-renal azotemia from established ATN is clinically important but can be challenging. Pre-renal AKI is generally expected to improve following restoration of intravascular volume and renal perfusion, whereas ATN is associated with persistent renal dysfunction despite appropriate correction of hypovolemia. In everyday clinical practice, the distinction depends on a composite assessment incorporating the patient's history, duration and severity of fluid loss, hemodynamic status, response to fluid therapy, persistence of oliguria, serial changes in serum creatinine, urine sediment examination, and other available biochemical indicators.[9,10] Granular casts and renal tubular epithelial cells may support a diagnosis of tubular injury, but urine microscopy is not always performed consistently and its interpretation depends on operator expertise.

Several emerging biomarkers—including neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, interleukin-18, cystatin C, and tissue inhibitor of metalloproteinases-2 combined with insulin-like growth factor-binding protein 7—have been investigated for the earlier detection and prognostication of tubular injury. However, their cost, limited availability, uncertain diagnostic thresholds, and lack of routine laboratory infrastructure restrict their widespread clinical use in many low- and middle-income countries. Consequently, clinicians in these settings continue to rely predominantly on conventional biochemical measurements, urine output, urine sediment findings, and the clinical response to fluid resuscitation.

Despite the considerable regional burden of gastroenteritis-associated AKI, prospective data specifically examining the development of ATN among affected patients in South Asia remain limited.[11] Available investigations have often been retrospective, based on small samples, or focused on AKI from heterogeneous causes. Consequently, the frequency of ATN, its principal risk factors, patterns of renal recovery, requirement for dialysis, and short-term clinical outcomes among patients hospitalized with acute gastroenteritis remain insufficiently characterized.

The present prospective observational study was therefore conducted at Northwest General Hospital and Research Center, Peshawar, to determine the frequency of ATN among patients presenting with acute gastroenteritis-associated AKI and to evaluate its clinical and biochemical predictors. The study also aimed to examine the short-term prognosis and renal outcomes of affected patients.[12] By generating locally relevant evidence, this investigation may facilitate earlier risk stratification, improve clinical monitoring, guide timely therapeutic interventions, and contribute to the prevention of avoidable morbidity and mortality associated with gastroenteritis-induced AKI in Pakistan.

METHODOLOGY

Prospective cohort study was conducted in the medical wards, surgical wards, and adult intensive care unit of the Northwest General Hospital and Research Center, Peshawar, Pakistan, from January 1, 2024 to March 30, 2026. The study protocol was approved by the Ethics Committee of Northwest General Hospital and Research Center, Peshawar, Pakistan (Ref Approval No: NWGH-EC-2024-042). Informed written consent was obtained from all participants/their respective legal representatives before conducting the research, following the principles of the Declaration of Helsinki.[13]

All consecutive adult patients (≥ 18 years) with clinical diagnosis of acute gastroenteritis (defined as passing three or more watery stools per day for less than 14 days, with or without vomiting) and AKI were included in the study. AKI was defined according to KDIGO criteria: rise in serum creatinine by ≥ 0.3 mg/dL (≥ 26.5 μmol/L) within 48 hours, by ≥ 1.5 times of baseline within the previous 7 days, or oliguria (< 0.5 mL/kg/h for > 6 hours).[14] Patients with self-limited pre-renal AKI resolved within 24–48 hours of fluid repletion, post-renal obstruction on imaging, suspected glomerulonephritis or acute interstitial nephritis, known chronic kidney disease stages 4–5 and those unable to give consent were excluded.[15]

The sample size was calculated based on the primary outcome of estimating the incidence of ATN among patients presenting with acute gastroenteritis-associated AKI. Assuming an anticipated ATN incidence of 30% based on regional literature, a 95% confidence level, and a margin of error of 3.5%, the minimum required sample size was calculated to be 658 patients using Cochran's formula ($n = Z^2 \cdot p(1-p)/d^2$). To account for potential missing data or loss during 90-day follow-up, enrollment was extended to 712 patients. Additionally, with 236 ATN events recorded, the sample size provided >35 events per variable (EPV) for multivariable logistic regression analysis, exceeding the standard requirement of 10–15 EPV to prevent model overfitting.

ATN diagnosis was made by a panel of nephrologist, intensivist and laboratory specialist based on a composite clinical and laboratory algorithm: (1) clinical scenario of prolonged hypovolemia, sepsis, or persistent hemodynamic instability not resolving rapidly with volume expansion therapy; (2) urine microscopy revealing muddy brown granular casts and renal tubular cells; (3) elevated blood urea nitrogen-to-serum creatinine (BUN:Cr) ratio along with clinical signs of intrarenal damage; and (4) absence of alternative post-renal or glomerular causes revealed by renal ultrasonography and laboratory studies.[16,17]

Detailed information about demographic characteristics, duration of symptoms prior to admission, severity of dehydration (WHO classification), comorbidities, vital signs, biochemical parameters (complete blood count, electrolytes, BUN, serum Cr, BUN:Cr ratio, C-reactive protein), illness severity scores (where applicable in ICU), treatment (IV fluids, vasopressors, antibiotics, RRT) and complications was collected.[18] Patients were followed until discharge and then 30 and 90 days post-discharge to determine renal recovery (return of serum creatinine to ≤25% of baseline) and vital status.[19]

The data was collected using a structured questionnaire. All the information that was obtained was extensive with respect to patient demographics, the time and degree of symptoms before admission, baseline clinical information (including vital signs and WHO score for dehydration), repeated laboratory investigations, in-patient management (IV fluids, vasopressors, antibiotics, and RRT use), length of hospitalization, and outcome.

The patients were monitored during the course of hospital admission as well as after discharge at 30 and 90 days by means of clinic follow-ups or telephone calls as required by the study design. The follow-up measures comprised of:

1. Clinical assessment: The symptoms, performance status, medications used, re-admissions, and GI/renal symptoms were evaluated.
2. Laboratory parameters: These included repeat measurements of serum creatinine, blood urea nitrogen and electrolytes to assess the renal function recovery. The criteria for renal function recovery at 90 days included serum creatinine normalization to 25% of baseline levels and no dialysis.
3. Mortality and RRT dependence at 90 days.

Statistical analyses were performed using SPSS software, version 26.0 (IBM Corp.). Continuous variables are shown as mean ± standard deviation or median (interquartile range) and were compared using Student's t-test or Mann-Whitney U test accordingly. Categorical variables are given as frequencies (percentages) and compared with the chi-square test or Fisher's exact test. Independent predictors of ATN and adverse outcomes were identified using multivariable logistic regression models, reporting adjusted ORs and 95% CIs.[20] All tests were two-sided and P-value of less than 0.05 was considered statistically significant.[21] Interim analyses were not planned.

RESULTS

Overall, 712 patients met inclusion criteria and were included in the analysis. All of the patients enrolled had AKI associated with gastroenteritis. Gastroenteritis was the main complaint of the patients who attended the ED in 591 cases (83.0%), while in the other 121 cases (17.0%), there were patients with major complications caused by gastroenteritis (hypovolemic shock, electrolyte encephalopathy, and sepsis).

Admission biochemical parameters revealed significant differences between the groups (Table II). Higher levels of serum creatinine, blood urea nitrogen, and the BUN:Cr ratio were found in ATN group, which proves profound changes in nitrogen metabolism and

Table I: Baseline Demographic and Clinical Characteristics According to Presence or Absence of Acute Tubular Necrosis

Characteristic	ATN (n=236)	No ATN (n=476)	P Value
Age — yr	44.2 ± 17.1	41.5 ± 15.9	0.05
Male sex — no. (%)	142 (60.2)	268 (56.3)	0.32
Primary presentation acute gastroenteritis — no. (%)	196 (83.1)	395 (83.0)	0.98
Duration of symptoms >48 hr — no. (%)	172 (72.9)	205 (43.1)	<0.001
Severe dehydration — no. (%)	158 (66.9)	124 (26.1)	<0.001
Hypokalemia (<3.5 mmol/L) — no. (%)	105 (44.5)	102 (21.4)	<0.001
Hypotension on admission — no. (%)	82 (34.7)	71 (14.9)	<0.001
ICU admission — no. (%)	68 (28.8)	52 (10.9)	<0.001

tubular dysfunction inherent for established ischemic lesion.

Short-term outcomes are shown in Table III. ATN patients had significantly longer hospital and ICU stay. The need for renal replacement therapy was more than ninefold higher, renal recovery rate at 90 days was much lower and in-hospital mortality was over five times higher in ATN patients. All differences persisted after adjusting for confounding factors.

Table II: Selected Laboratory Values at Admission

Variable	ATN (n=236)	No ATN (n=476)	P Value
Serum creatinine — mg/dL	2.8 ± 1.4	1.9 ± 0.8	<0.001
Blood urea nitrogen — mg/dL	68 ± 32	42 ± 21	<0.001
BUN-to-creatinine ratio	24.3 ± 5.8	21.5 ± 5.1	<0.001
Serum potassium — mmol/L	3.3 ± 0.6	3.9 ± 0.5	<0.001

Table III: Primary and Secondary Clinical Outcomes

Outcome	ATN (n=236)	No ATN (n=476)	P Value
Hospital length of stay — days	9.2 ± 4.1	4.6 ± 2.3	<0.001
ICU length of stay (among admitted) — days	6.8 ± 3.5	3.9 ± 2.1	<0.001
Renal replacement therapy — no. (%)	27 (11.4)	6 (1.3)	<0.001
Renal recovery at 90 days — no. (%)	153 (64.8)	443 (93.1)	<0.001
In-hospital mortality — no. (%)	20 (8.5)	7 (1.5)	<0.001

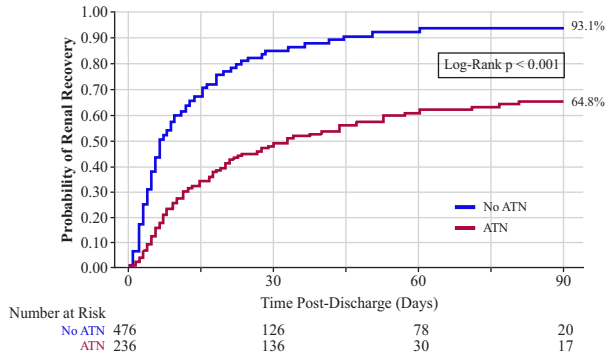

Figure 1: Kaplan–Meier estimates of the probability of renal recovery according to the presence or absence of ATN (log-rank test, P<0.001).

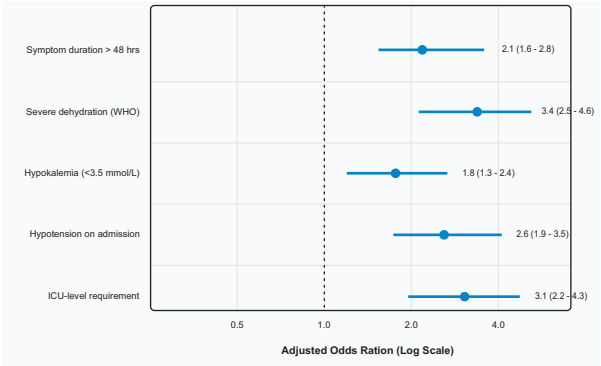

Figure 2: Multivariable-adjusted odds ratios for key risk factors associated with development of ATN.

DISCUSSION

In the large prospective study of hospitalized patients with acute gastroenteritis and AKI, ATN occurred in approximately one third of patients and was strongly associated with bad clinical outcomes. The predominant role of gastroenteritis (83%) as the etiology highlights the importance of this preventable mechanism of kidney damage in South Asian region.[22]

Our results regarding the frequency of ATN are consistent with regional data from Pakistan and India where diarrhea is a significant cause of community

Table IV: Multivariable Logistic Regression Analysis for Independent Risk Factors Associated with ATN Development

Variable	Univariate OR (95% CI)	Adjusted OR (95% CI)	P Value
Duration of symptoms >48 hr	3.51 (2.48–4.97)	2.64 (1.81–3.85)	<0.001*
Severe dehydration (WHO)	5.75 (4.06–8.14)	3.82 (2.61–5.59)	<0.001*
Hypokalemia (<3.5 mmol/L)	2.94 (2.08–4.15)	1.95 (1.33–2.86)	0.001*
Hypotension on admission	3.03 (2.09–4.39)	1.88 (1.24–2.85)	0.003
ICU admission requirement	3.30 (2.21–4.93)	1.72 (1.08–2.74)	0.022
Age (per 10-year increase)	1.10 (0.99–1.22)	1.05 (0.94–1.17)	0.41
Male sex	1.17 (0.85–1.62)	1.08 (0.76–1.53)	0.672

acquired AKI. The progression rate of AKI to ATN in our study cohort was relatively high, which may reflect the high rates of severe dehydration and late presentation in our catchment area.[23]

Using the clinical approach with routine diagnostic parameters, including examination of urinary sediment for granular casts and evaluation of trends of blood urea nitrogen and serum creatinine, proved to be very effective for stratifying the degree of injury and diagnosis of established tubular necrosis.[24,25]

ATN patients had significantly worse outcomes, including longer hospital stay, resource utilization (RRT), lower rate of renal recovery and higher mortality.[26] These results are consistent with AKI literature, where established tubular injury is proven to predict bad prognosis compared to rapidly reversible pre-renal azotemia.[27]

Risk factors for ATN identified using multivariate analysis include severe dehydration, prolonged duration of symptoms, hypokalemia and ICU requirement.[28] All these factors are potentially modifiable by public health interventions (e.g. promoting oral rehydration) and hospital protocols focusing on timely fluid resuscitation and electrolyte correction.[29]

Further outcomes may be problematic as ATN increases the risk of chronic kidney disease development.[30] Our 90-day recovery data (65% in ATN vs. 93% without ATN) are consistent with other studies of hospitalized patients.[31]

The strengths of this study are its prospective design, mixed ward-ICU population, high follow-up rate, and clinical stratification of patients using biochemical and microscopic indices. Limitations are the single-center design and lack of kidney biopsies used routinely as a gold standard in acute dehydrating diseases. Future multicenter studies should evaluate protocolized fluid and electrolyte management strategies.

CONCLUSION

In hospitalized patients, ATN complicates a significant number of AKI cases induced by gastroenteritis and predicts bad outcomes. Proper bedside assessment using routine biochemical parameters, serial creatinine measurement, and active urine sediment analysis, along with adequate hydration and electrolyte correction are key strategies to tackle the problem in high-burden regions.

Ethical approval:

Ethical approval was taken from institutional review board of Northwest General Hospital & Research Center Peshawar with the IRB number NWGH-EC-2024-042, Dated 05-01-2024.

Conflict of Interest:

Authors declare no conflict of interest.

Financial Disclosure:

None

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HA: Data Collection and manuscript drafting.

HA & KAK: Data Analysis and critical review.

SUQ, HA, KAK: Supervision & Manuscript drafting & proof reading.

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