

ORIGINAL ARTICLE



Study on the Clinical Characteristics of Patients with Acute Kidney Injury Caused by Acute Gastroenteritis

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Abstract:

The number of cases diagnosed with acute kidney injury (AKI) associated with acute gastroenteritis (AGE) is increasing. Recent studies have highlighted that even slight elevations in serum creatinine levels are associated with poor outcomes across various clinical settings. This study aimed to examine the clinical characteristics of patients hospitalized at our institution with AKI secondary to AGE. Methods: The study subjects were patients aged 18 years or older diagnosed with AKI. Following patient selection, detailed medical histories were recorded, and comprehensive clinical examinations were performed. Recorded data included the clinical course of gastroenteritis, the timing of AKI onset, fluid status at admission, and daily measurements of urea, serum creatinine, sodium, and potassium. Other laboratory parameters included complete blood count (CBC), erythrocyte sedimentation rate (ESR), urinalysis, stool analysis (including the hanging-drop method), HIV testing, blood glucose, white blood cell count and differential, and liver function tests (serum bilirubin, total protein, albumin, SGOT, SGPT, and ALP).

Keywords: Acute Kidney Injury, Acute Gastroenteritis, and Kidney Disease Improving Global Outcomes

Introduction

AKI known earlier as ARF signifies a clinical state that arises over several days or even hours and is characterized by a reduced GFR resulting in poor ability of the body to eliminate nitrogenous waste products and other toxins ^[1]. AKI is categorized into three types based on underlying causes: They include prerenal (caused by decreased renal perfusion), intrinsic (due to renal disease), and postrenal (caused by backward extension of the pathologic process to the urinary tract). The condition can be caused by many factors such as acute fluid loss from diarrhea diseases or hemorrhage; malaria, pneumonia, and sepsis; nephrotoxic drugs; and major operation ^[2]. Of them, Acute Volume Depletion, common in diarrheal diseases, is the leading known cause of AKI. AKI appears to be a heterogeneous syndrome and may range from a minor increase in serum creatinine or an abnormality noticeable on urine examination to a severe condition that causes severe dyscrasia such as fluid and

electrolyte imbalance that may prove fatal ^[3]. AKI occurs in 5–7% of patients admitted in an acute general care setting and in 20–30% of ICU patients in which mortality ranges from 40%–50% in critical cases. This remains one of the biggest presenting clinical problems globally and is associated with increased rates of morbidity and mortality, especially in the intensive care setting ^[4]. Severe acute gastroenteritis (AGE) is a leading cause of AKI worldwide and more evidently in the tropic region due to infections that cause diarrhea. AGE frequently results in a major amount of fluid loss with consequent renal hypoperfusion and oliguria, azotemia, and hyperkalemia ^[5]. These electrolyte derangements, coupled with the catabolic state and lactic acidosis, should only serve to worsen renal function and potentially cause oliguria and renal shutdown in some patients. Awareness of a relationship between modern, moderate increases in serum creatinine and poor clinical outcomes is growing, due to which the concern with kidney

function in patients with AGE has risen.

AKI is mostly reparative, especially with prerenal kidney disease which if diagnosed at an early age the cause of hypoperfusion would have been well managed. However, the characteristics of AKI in the tropical zone are still unique and different from developed countries due to the effect of socioeconomic status, the prevalence of tropical diseases, and inequality of access to treatment^[6]. Those associated with tropical areas such as viral gastroenteritis, bacillary dysentery, cholera, and food poisoning are often responsible for AKI in such locations; hence diarrheal diseases are a main cause of AKI in the tropics^[6]. This present study aimed to evaluate the clinical and biochemical characteristics of patients with AKI resulting from gastroenteritis, focusing on the outcomes and implications for managing AKI in regions where gastrointestinal infections are prevalent. Understanding the clinical profile of such patients will provide insights into prevention, early diagnosis, and effective management strategies for AKI due to acute gastroenteritis.

Material and Methods

This prospective study was conducted in the Department of General Medicine, Medical College, and Hospital. Institutional Ethical approval was obtained for the study. After explaining the nature of the study in vernacular language, written consent was obtained from all the participants. Those voluntarily willing to participate in the study were included.

Inclusion Criteria

1. Patients above 18 years diagnosed with AKI due to acute gastroenteritis, defined by:
2. Serum creatinine increased by 0.3 mg/dl (26.5 mmol/l) within 48 hours.
3. Serum creatinine rises to 1.5 times baseline within 7 days, or
4. Urine output below 0.5 ml/kg/h for 6 hours.

Exclusion Criteria

1. AKI is caused by factors other than gastroenteritis.
2. Patients with Chronic Kidney Disease (CKD) presenting with acute gastroenteritis.

After the selection of the patient, a detailed history of the case was recorded and the patient was subjected to a thorough clinical examination.

Records included information on gastroenteritis duration, onset of renal failure, hydration status at admission, and daily blood tests for urea, serum creatinine, sodium, and potassium levels. Additional tests included CBC, ESR, urine, stool (including hanging drop), HIV, blood glucose, total and differential leukocyte counts, liver function tests (serum bilirubin, total serum protein, albumin, SGOT, SGPT, ALP), among others.

Management: Patients received fluid replacement based on dehydration severity. Complications like hyperkalemia and metabolic acidosis were managed urgently. Fluid intake and output were recorded, and fluids were adjusted accordingly. Antibiotics targeting gram-negative bacteria, especially quinolones, were initially administered, with adjustments based on culture reports or complications. Patients with persistent diarrhea received fluoroquinolones, while those with confirmed infections were given parenteral Cefotaxime or Cefoperazone. Dialysis was provided for patients with hyperkalemia, pulmonary edema, severe acidosis unresponsive to medical treatment, and those with serum creatinine levels above 8 mg/dl. Patients were monitored until discharge or death, recording any complications during hospitalization. Clinical and laboratory data were analyzed to assess factors influencing patient outcomes, whether recovery or death.

Statistical Analysis: Data analysis was performed using SPSS (version 22.0) with Chi-square tests, Small Sample t-tests, and ANOVA. A P-value <0.05 was considered statistically significant.

Results

The age-wise distribution of Acute Kidney Injury (AKI) following Acute Gastrointestinal (GI) events is given in Table 1. AKI also occurs in a significant proportion of patients older than 60 years. It is consistent with the trend of generally increased vulnerability to complications (including AKI) in older people. The mean age of the cohort was 52.35 ± 10.5 years. The overall distribution between males and females is roughly equal, but there is a trend for an increase in males' incidence, especially in the older age groups. This can be due to many causes including Lifestyle, underlying health conditions, etc, or biological factors.

Table 1: Age-wise distribution of cases of AKI following Acute GE

Age group in years	Male	Female	Total
18 – 20	4 (10.5%)	1(4.5%)	5(8.3%)
21 – 30	7(18.4%)	4(1.8%)	11(18.3%)
31 – 40	6(15.8%)	3(13.6%)	9(15.0%)
41 – 50	6(15.8%)	3(13.6%)	9(15.0%)
51 – 60	6(15.8%)	4(18.18%)	10(16.7%)
>60	9(39.5%)	7(31.8%)	16(26.7%)
Total	38(100.0%)	22(100.0%)	60(100.0%)

Table 2 provides a clear overview of the symptoms experienced by patients who developed AKI following acute GI events. Loose stools were the most common symptom, present in all cases. This is expected, given the association of AKI with acute GI infections and inflammatory conditions. Vomiting was also a frequent

symptom, indicating the severity of the GI illness. Fever and altered sensorium were observed in a significant proportion of cases, suggesting a systemic inflammatory response. Shortness of breath could be a sign of acute lung injury or other complications associated with severe illness.

Table 2: Symptom-wise distribution of cases in the study

Symptoms	Frequency	Percentage
Loose stools	60	100.0
Fever	46	76.7
Vomiting	40	66.7
Shortness of breath	10	16.7
Altered sensorium	8	13.3

The severity of AKI cases, divided by the kidney disease is shown in Table 3. Most cases (56.7%) were characterized as stage 1 AKI, being mildly elevated serum creatinine or decreased urine output. Thirty percent of patients had stage 2 AKI characterized by a greater rise in serum creatinine and persistent oliguria (AKI), a more substantial increase in serum creatinine. Only 16.7 percent of the cases progressed to stage 3 AKI requiring renal replacement therapy or the other criteria for

severe kidney injury. Keeping Stage 1 AKI can be prevented by early identification and intervention. The KDIGO classification can be used to stratify patients based on their risk for adverse outcomes and thus there can be different strategies for managing. Monitoring of patients with stage 2 and 3 AKI is necessary to recognize early changes toward worsening renal function and for the implementation of adequate interventions (dialysis).

Table 3: AKI staging (KDIGO) guidelines ^[3]

Stage	Serum creatinine	Urine output	No. of patients (%)
1	1.5–1.9 times baseline OR ≥ 0.3 mg/dl (≥ 26.5 μ mol/l) increase	<0.5 ml/kg/h for 6–12 hours	31(56.7%)
2	2.0–2.9 times baseline	<0.5 ml/kg/h for ≥ 12 hours	19(31.7%)
3	3.0 times baseline OR Increase in serum creatinine to ≥ 4.0 mg/dl (≥ 353.6 μ mol/l) OR Initiation of renal replacement therapy OR In patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m ²	<0.3 ml/kg/h for ≥ 24 hours OR Anuria for ≥ 12 hours	10(16.7%)

In this study, common complications of Acute Kidney Injury included hypotension and hyperkalemia. Other observed complications were metabolic acidosis, encephalopathy, pulmonary edema, anemia, multi-organ dysfunction syndrome (MODS), hypokalemia, and hyponatremia. Hemodialysis was required in 10% of patients, with a mortality rate of 3.3%.

Discussion

Acute Kidney Injury (AKI, formerly Acute Renal Failure) is a clinical syndrome characterized by a sudden decrease in glomerular filtration rate leading to failure of the clearance of nitrogenous waste products (urea and creatinine) and other uremic toxins. AKI causes are typically classified into three main categories: Depending on the cause, the three forms of acute renal failure are prerenal (decreased renal perfusion), intrinsic (injury to the kidney tissue), and postrenal (obstruction of the urinary tract) ^[7]. Acute fluid loss from diarrhea or hemorrhage, sepsis, viral hepatitis or malaria, pneumonia, infections, snake bites, heart failure, nephrotoxic drugs, malignancies, systemic lupus erythematosus (SLE), hypertension, major surgery, and radiocontrast agents are all common triggers. According to Lewington AJ et al, ^[8] 21.6% of hospitalized adult patients will develop AKI during their stay which is associated with high resource use and adverse outcomes. A rise as small as 0.3 mg/dL in serum creatinine is associated with 70 percent and 6.5 times greater mortality risk, and 6.5 times greater overall risk of death. Before AKI, optimizing hemodynamic status and volume status is beneficial to AKI; diuretics do not demonstrably reduce AKI progression or outcomes ^[9]. The mean patient age was 52.35 ± 10.5 years, the majority of patients were 61–70 years (26.7%) or 51–60 years (16.7%), with a male predominance. In earlier studies, AKI was caused by sepsis and acute gastroenteritis in Satri V et al. ^[10] and acute gastroenteritis alone was the cause of AKI in Eswarappa M. et al. ^[11]

In this study, we found that vasopressor support was required in 10(16.7%) patients, and 5% required blood transfusion. 11.7% of cases required CPAP support. 66.7% recovered in one week after the treatment initiation and 25% required up to 10 days for recovery. In a similar

study Pajai AE et al. noted that 18.9% of patients required vasopressor support, 7.5% needed transfusions of blood or blood products, 11.3% received CPAP support, and two patients required mechanical ventilation. Recovery within 7 days of treatment was seen in 50.9% of patients, while 45.3% required more than 7 days for recovery. In this study, 31.7% of patients died and 3.3 % of patients required renal replacement therapy (RRT). Requiring vasopressors and RRT were significantly associated with delayed initiation of definitive therapy. Inbanathan et al. ^[12] observed 30% of patients required hemodialysis of these patients 70% were managed conservatively. Within one week, discharge occurred in 52%, between one and three weeks in 37%, three weeks in 11%, with 4% mortality. The prognosis in AKI is closely associated with the cause and duration of renal failure before intervention, especially with age, multiorgan failure, circulatory failure, vasopressor use, and the need for RRT. Factors that affect patient survival in acute kidney injury include the severity of the injury and the underlying disease as well as age, degree of comorbidities, and associated complications, including intravascular overload, hyperkalemia, and other metabolic complications, and systemic life-threatening complications, such as cardiac arrhythmia, myocardial infarction, pulmonary embolism, gastrointestinal ulcers, seizures, coma, hemolysis, bleeding tendencies, and severe infections ^[13]. Irrespective of cause, management of AKI is a risk factor for the development of complications, a 10-fold increased risk of chronic kidney disease, and a 3-fold risk of end-stage renal disease. ^[14, 15] Failure to detect AKI and treat it timely and/or adequately generally has very grave consequences. Its morbidity is high and the associated loss of kidney function is permanent ^[16]. Early detection depends to a great extent on a high degree of suspicion that AKI is occurring in the appropriate context. Diagnosis is completed by a combination of a history, a thorough exam, an accurate assessment of kidney function, proper imaging, and, when indicated, a kidney biopsy.

Conclusion

In conclusion, AKI following acute GI events is a serious condition that can lead to significant morbidity and mortality. Early recognition, prompt management, and timely intervention are crucial to improve patient outcomes. Future

research should focus on identifying risk factors, developing effective prevention strategies, and optimizing treatment approaches to minimize the impact of AKI on patient health.

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