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Clinical Profile of Patients with Acute Kidney Injury Secondary to Gastroenteritis Attending a Tertiary Care Centre

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HIGHLIGHTS

- Gastroenteritis caused AKI
- Dehydration reduced renal perfusion
- Severe AKI increased dialysis needs
- Early treatment improved recovery
- Shock predicted poor outcomes

Key Words:

Acute kidney injury
Acute gastroenteritis
Dehydration
KDIGO
Renal outcomes

ABSTRACT

Introduction: Acute kidney injury (AKI) is a frequent and potentially reversible complication of acute gastroenteritis, primarily resulting from dehydration and renal hypoperfusion. Delayed recognition may lead to prolonged hospitalization, dialysis requirement, and increased mortality. **Aim & Objective:** This study evaluated the clinical profile, severity, risk factors, and outcomes of AKI secondary to acute gastroenteritis in patients attending a tertiary care centre. **Materials & Methods:** A prospective observational cross-sectional study was conducted over 18 months in the Department of General Medicine at a tertiary care teaching hospital. A total of 160 adult patients (≥ 18 years) with acute gastroenteritis who developed AKI according to KDIGO criteria were included. Patients with pre-existing chronic kidney disease, prior renal dysfunction, or AKI due to causes other than gastroenteritis were excluded. Clinical details, laboratory investigations, hydration status, AKI staging, complications, dialysis requirement, and outcomes were recorded and analyzed using appropriate statistical methods. **Results:** The mean age of the study population was 50.17 ± 16.81 years. Complete recovery was achieved in 68.8% of patients, partial recovery in 27.5%, and mortality was 3.7%. Increasing AKI severity was significantly associated with longer hospital stay, poorer clinical outcomes, and greater dialysis requirement ($p < 0.001$). Severe dehydration, septicemia, shock, and dialysis requirement were significant predictors of adverse outcomes, whereas progressive reduction in blood urea and serum creatinine reflected improvement following appropriate treatment. **Conclusion:** Acute kidney injury secondary to gastroenteritis is predominantly related to dehydration-induced renal hypoperfusion. Early diagnosis, prompt fluid resuscitation, correction of electrolyte abnormalities, and timely supportive management result in favorable renal recovery, while severe AKI, septicemia, shock, and dialysis requirement predict poor prognosis. Early intervention is essential to reduce morbidity and mortality in these patients.



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INTRODUCTION

Acute kidney injury (AKI) is a sudden decline in renal function that impairs the kidneys' ability to regulate fluid balance, electrolyte homeostasis, and acid-base status. It is characterized by a reduction in glomerular filtration rate, resulting in the accumulation of nitrogenous waste products such as urea and creatinine. In the early stages, serum creatinine levels may remain within the normal range, making reduced urine output one of the earliest clinical indicators. As the condition progresses, electrolyte abnormalities including hyperkalemia and hyponatremia, along with metabolic acidosis, commonly develop. AKI has become a major global public health concern because of its association with increased morbidity, mortality, prolonged hospitalization, and rising healthcare costs. It affects approximately 5–7% of hospitalized patients and nearly one-third of critically ill patients admitted to intensive care units. Timely diagnosis and prompt management are essential to prevent irreversible renal damage, reduce complications, and improve patient outcomes [1,2].

AKI develops secondary to a wide range of conditions such as infections, dehydration, nephrotoxic medications, obstructive uropathy, and systemic illnesses that compromise renal perfusion or directly injure the kidneys. Clinically, it presents with elevated serum creatinine, reduced urine output, electrolyte disturbances, and metabolic acidosis, while severe cases may require renal replacement therapy. Although many episodes of AKI are reversible with early intervention, delayed diagnosis increases the likelihood of persistent renal dysfunction and progression to chronic kidney disease (CKD). Hospital-acquired AKI remains a significant challenge, with studies reporting that nearly one-fifth of hospitalized patients develop AKI, and approximately 10% of these individuals eventually require dialysis. Consequently, routine monitoring of renal function and early identification of

high-risk patients have become essential components of modern clinical practice [2,3].

Several classification systems have been developed to standardize the diagnosis and severity assessment of AKI. The RIFLE classification was the first widely accepted staging system, categorizing patients into Risk, Injury, Failure, Loss of kidney function, and End-stage renal disease based on changes in serum creatinine, glomerular filtration rate, and urine output. The Acute Kidney Injury Network (AKIN) criteria subsequently improved the sensitivity of early diagnosis. Currently, the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines integrate the strengths of both RIFLE and AKIN and represent the internationally accepted standard. According to KDIGO, AKI is diagnosed by an increase in serum creatinine of at least 0.3 mg/dL within 48 hours, an increase to 1.5 times baseline within seven days, or urine output below 0.5 mL/kg/hour for six hours or longer. Adoption of KDIGO criteria has enhanced uniformity in diagnosis, facilitated comparison among clinical studies, and improved patient management worldwide [4,5].

Based on the underlying mechanism, AKI is classified into prerenal, intrinsic renal, and postrenal types. Prerenal AKI, the most common and potentially reversible form, results from reduced renal perfusion caused by hypovolemia, hypotension, or decreased cardiac output. Intrinsic renal AKI arises from direct injury to the renal parenchyma, including acute tubular necrosis, glomerulonephritis, interstitial nephritis, or nephrotoxic drug exposure. Postrenal AKI occurs due to urinary tract obstruction that impedes urine flow. Early identification and correction of the underlying cause often allow complete recovery of renal function. However, persistent renal ischemia or delayed treatment may lead to irreversible nephron damage, chronic kidney disease, and eventual end-stage renal disease [5,6].

The epidemiology and causes of AKI vary considerably between

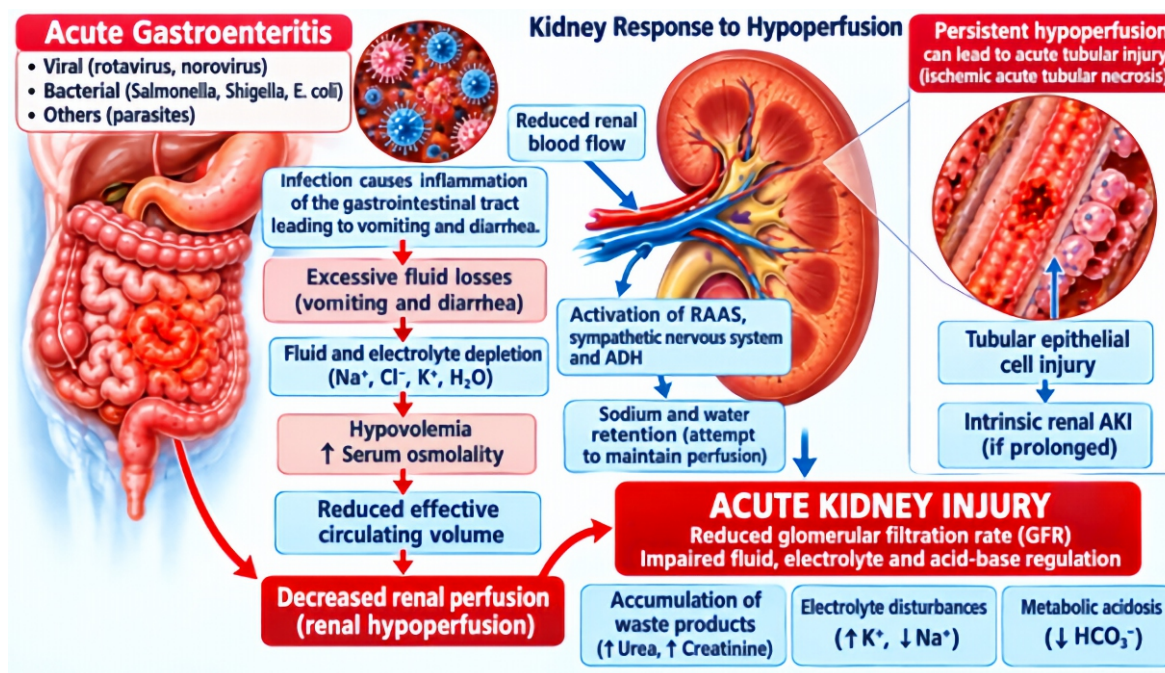


Figure 1: Pathophysiology of Acute Kidney Injury Secondary to Gastroenteritis

developed and developing countries. In low- and middle-income regions, infectious diseases, poor sanitation, inadequate access to healthcare, & limited obstetric services contribute substantially to the disease burden. Tropical infections such as malaria, leptospirosis, dengue fever, and severe diarrheal illnesses remain important causes of AKI. Among these, acute gastroenteritis is particularly significant because contaminated water supplies and poor hygiene facilitate its widespread occurrence. Severe vomiting and diarrhea cause profound fluid and electrolyte losses, leading to intravascular volume depletion, renal hypoperfusion, and prerenal AKI. Consequently, gastroenteritis-associated AKI continues to represent a major preventable cause of renal dysfunction in developing countries [6,7].

Acute gastroenteritis (AGE) is characterized by the sudden onset of diarrhea, often accompanied by nausea, vomiting, abdominal pain, or fever, with symptoms generally lasting less than two weeks. Viral pathogens such as rotavirus and norovirus are common etiological agents, while bacterial organisms including *Salmonella*, *Shigella*, and *Escherichia coli* also contribute significantly. Disease severity ranges from mild self-limiting illness to severe dehydration with electrolyte imbalance and systemic complications. Clinical diagnosis is usually based on history and physical examination, whereas laboratory investigations such as complete blood count, serum electrolytes, renal function tests, and stool analysis are reserved for complicated or severe cases [7,8].

Dehydration is the most important mechanism linking gastroenteritis with AKI. Excessive gastrointestinal fluid losses produce hypovolemia, increased serum osmolality, and diminished renal blood flow. Depending on the extent of fluid loss, dehydration may be mild, moderate, or severe, with manifestations ranging from thirst and lethargy to hypotension, altered mental status, and circulatory collapse. Progressive renal hypoperfusion ultimately results in prerenal AKI if fluid deficits are not corrected promptly. Elderly individuals and patients with diabetes mellitus, hypertension, or cardiovascular disease are particularly susceptible because of reduced physiological reserve and impaired renal function. Gastroenteritis-associated AKI is also associated with prolonged hospitalization, increased healthcare utilization, and a higher risk of CKD. Therefore, understanding its clinical profile, laboratory abnormalities, risk factors, and outcomes is essential for facilitating early diagnosis, prompt fluid resuscitation, and improved patient prognosis [9-11].

Figure 1. Pathophysiology of Gastroenteritis-Induced Acute Kidney Injury

The present study was undertaken to assess the clinical profile, risk factors, severity, laboratory characteristics, and outcomes of patients developing acute kidney injury secondary to acute gastroenteritis at a tertiary care centre. It also evaluated the demographic and clinical features of affected patients, classified the severity of acute kidney injury according to the KDIGO guidelines, identified factors associated with its development, analyzed relevant laboratory parameters, and assessed clinical outcomes following prompt rehydration therapy and appropriate supportive management.

MATERIAL & METHODS

This prospective observational cross-sectional study was conducted at the Department of General Medicine, at a tertiary care teaching hospital from 2023 to 2026 for 18 months. Ethical approval has been obtained from the Ethical Approval Committee of a tertiary care teaching hospital.

Study Population

The study population comprised adult patients aged 18 years and above who were admitted with acute gastroenteritis and subsequently developed acute kidney injury secondary to the illness. Patients with pre-existing chronic kidney disease, known prior renal dysfunction, or acute kidney injury attributable to causes other than gastroenteritis, including septic shock unrelated to acute gastroenteritis, obstructive uropathy, glomerulonephritis, nephrotoxic drug-induced renal injury, snake bite, poisoning, and cardiac disorders, were excluded from the study.

Data Analysis

The collected data were entered and analyzed using IBM SPSS Statistics version 20. Descriptive statistics were expressed as frequencies, percentages, and mean $\pm$ standard deviation. Associations between categorical variables were evaluated using the Pearson chi-square test, while differences in proportions were assessed using the Z-test. A p-value of less than 0.05 was considered statistically significant for all statistical analyses performed in the study.

RESULTS

The study included 160 patients with a mean age of 50.17 ± 16.81 years. Regarding clinical outcomes, complete recovery was observed in the majority of patients (68.8%), while 27.5% experienced partial recovery. Mortality was low, accounting for only 3.7% of cases. These findings indicate that most patients with acute kidney injury secondary to acute gastroenteritis responded favorably to appropriate management, with complete renal recovery achieved in over two-thirds of the study population.

Blood urea and serum creatinine levels showed a progressive decline from admission to discharge, indicating significant improvement in renal function following timely fluid resuscitation & appropriate supportive management (**Table 1**). The mean duration of hospital stay increased progressively with the severity of acute kidney injury, from 5.14 ± 0.86 days in Stage 1 to 10.61 ± 1.63 days in Stage 3, indicating that more severe AKI was associated with significantly longer hospitalization (**Table 2**). A highly significant association was observed between AKI stage and clinical outcome ($p < 0.001$), with complete recovery decreasing as disease severity increased. All deaths and most partial recoveries occurred in Stage 3 AKI, whereas all Stage 1 patients achieved complete recovery (**Figure 2**). Hydration status showed a highly significant association with clinical outcome ($p < 0.001$), with severe dehydration associated with markedly higher rates of partial recovery and mortality. In contrast, patients with mild dehydration predominantly

experienced complete recovery and favorable outcomes (**Table 3**). Patients with complete recovery showed significant improvement in systolic blood pressure during hospitalization, whereas those who died had persistently low blood pressure, indicating hemodynamic instability. Failure of blood pressure normalization was associated with poor clinical outcomes and increased mortality (**Table 4**). Complications showed a significant association with clinical outcome (overall $p=0.0005$). While electrolyte imbalance was the most common complication and was associated with favorable recovery after timely

correction, septicemia ($p < 0.001$) and shock ($p = 0.002$) were strongly associated with partial recovery and mortality, making them major predictors of poor prognosis in gastroenteritis-associated acute kidney injury (**Figure 3**).

Dialysis requirement was significantly associated with poor clinical outcomes ($p < 0.001$). None of the patients requiring dialysis achieved complete recovery, with 75% showing partial recovery and 25% dying, indicating that the need for dialysis is a marker of severe acute kidney injury and adverse prognosis (**Table 5**).

Table 1: Trend of Blood Urea and Serum Creatinine During Hospital Stay

Parameter	Admission (Mean $\pm$ SD)	Day 3–5 (Mean $\pm$ SD)	Outcome/Discharge (Mean $\pm$ SD)
Blood Urea (mg/dL)	121.41 $\pm$ 39.54	93.06 $\pm$ 35.46	59.39 $\pm$ 31.70*
Serum Creatinine (mg/dL)	4.98 $\pm$ 1.42	3.74 $\pm$ 1.49	2.34 $\pm$ 1.55

Table 2: Hospital Stay According to AKI Stage

AKI Stage	Mean Hospital Stay (Days) $\pm$ SD
Stage 1	5.14 $\pm$ 0.86
Stage 2	6.37 $\pm$ 1.17
Stage 3	10.61 $\pm$ 1.63

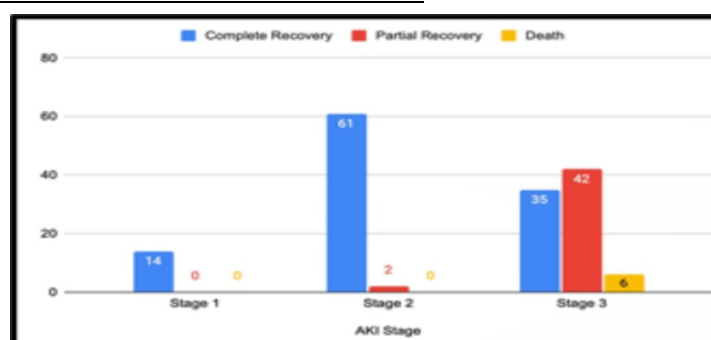


Figure 2: Column chart showing Association Between AKI Stage and Outcome Interpretation

Table 3: Hydration Status and Outcome

Hydration Status	Complete Recovery	Partial Recovery	Death	Total
Mild	14	4	0	18
Moderate	62	0	1	63
Severe	34	40	5	79
Total	110	44	6	160

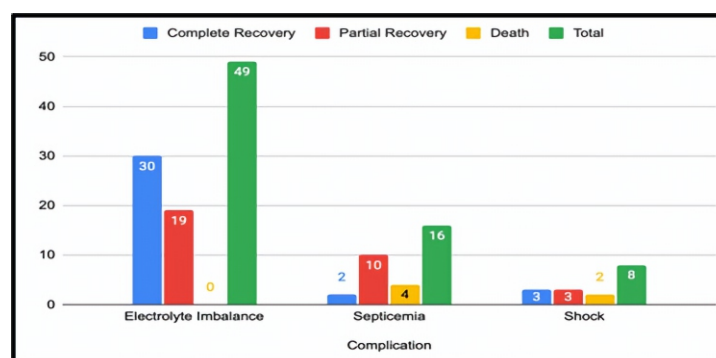


Figure 3: Column chart showing Distribution of Complications According to Outcome

Table 4: Systolic Blood Pressure at Admission and Outcome

Outcome	Mean SBP at Admission (mmHg)	Mean SBP at Outcome (mmHg)
Complete Recovery	104.65	118.56
Partial Recovery	105.80	111.14
Death	91.33	75.63

Table 5: Dialysis Requirement and Outcome

Dialysis Requirement	Complete Recovery	Partial Recovery	Death	Total
No	110	35	3	148
Yes	0	9	3	12
Total	110	44	6	160

DISCUSSION

The present study demonstrated that the mean age of patients with acute kidney injury (AKI) secondary to gastroenteritis was 50.17 ± 16.81 years, indicating that the condition predominantly affected middle-aged and older adults. Increasing age is an established risk factor for AKI because of the physiological decline in renal reserve, reduced ability of the kidneys to concentrate urine, diminished compensatory response to hypovolemia, and the higher prevalence of comorbid conditions such as diabetes mellitus and hypertension. These factors increase susceptibility to renal hypoperfusion during episodes of severe vomiting and diarrhea, thereby predisposing patients to kidney injury. **Bogari MH, et. al; 2023**, observed a significantly higher incidence of AKI among patients older than 60 years with acute gastroenteritis, while **Cheung AK, et. al; 2021**, also reported a comparable mean age among patients with gastroenteritis-associated AKI. Differences in age distribution across studies are likely related to variations in population characteristics, healthcare accessibility, and inclusion criteria [12,13].

Most patients in the present study achieved complete recovery following appropriate treatment, confirming that gastroenteritis-associated AKI is largely reversible when diagnosed early and managed with prompt fluid resuscitation, correction of electrolyte abnormalities, and supportive care. The predominance of prerenal AKI resulting from dehydration explains the favorable prognosis, as restoration of intravascular volume rapidly improves renal perfusion and glomerular filtration. **Cheung AK, et. al; 2021** and **Umakanth A, et. al; 2022**, who documented successful recovery in the majority of patients managed conservatively, with relatively low mortality rates. Progressive reductions in blood urea and serum creatinine levels during hospitalization further reflected improvement in renal function following treatment. Comparable biochemical recovery has also been reported by **Umakanth A, et. al; 2022** and **Sahay M. 2024**, emphasized that timely correction of dehydration prevents progression to irreversible renal damage. Differences in recovery rates among studies may be explained by variations in disease severity, duration of illness before presentation, presence of sepsis or shock, underlying renal reserve, and access to intensive care or dialysis services [13-15]. The study also demonstrated that increasing severity of AKI was associated with prolonged hospitalization, highlighting the greater clinical burden among

patients with advanced disease. Individuals with higher KDIGO stages required longer hospital stays because of severe dehydration, electrolyte disturbances, septicemia, shock, intensive monitoring, and, in some cases, renal replacement therapy. **Mehta K, et. al; 2018** and **Bogari MH, et. al; 2023**, observed that severe AKI resulted in prolonged hospitalization, increased complication rates, and poorer outcomes. Furthermore, disease severity at presentation showed a significant association with clinical outcome. Patients with Stage 1 AKI generally achieved complete recovery, whereas Stage 3 AKI was associated with partial recovery and increased mortality. **Mehta K, et. al; 2018** and **Anvar MI & Raghavendra B. 2018**, demonstrated that mortality and multiorgan dysfunction increased progressively with worsening KDIGO stage. Advanced AKI reflects more extensive renal ischemia, tubular injury, metabolic derangements, and systemic complications, thereby reducing the likelihood of complete renal recovery. Variations in outcomes between studies may be attributed to differences in patient demographics, prevalence of sepsis, timing of presentation, availability of dialysis facilities, and healthcare infrastructure [12,16,17]. Hemodynamic stability emerged as a critical determinant of both AKI severity and patient outcome. Patients who received early fluid replacement and maintained adequate systolic blood pressure experienced better recovery of renal function, whereas persistent hypotension was associated with advanced AKI and poorer prognosis. Severe fluid loss due to diarrhea and vomiting results in intravascular volume depletion, decreased renal perfusion, and reduced glomerular filtration rate. Early restoration of circulating volume prevents progression from reversible prerenal azotemia to intrinsic renal injury, particularly acute tubular necrosis. **JAMAL N, et. al; 2022**, identified hypotension and shock as important predictors of AKI development and adverse outcomes, while **Sahay M. 2024**, demonstrated that delayed correction of hypovolemia significantly increased the risk of acute tubular necrosis and mortality. Likewise, severe dehydration showed a strong association with unfavorable outcomes because prolonged volume depletion contributes to renal hypoperfusion, electrolyte disturbances, metabolic acidosis, and systemic complications. **Bogari MH, et. al; 2023**, similarly recognized dehydration as one of the strongest predictors of AKI among patients with acute gastroenteritis [12,15,18].

Complications significantly influenced prognosis in the present study, with electrolyte imbalance representing the most frequent complication, followed by septicemia and shock. Electrolyte disturbances, particularly hyponatremia and hypokalemia, resulted from excessive gastrointestinal fluid losses and were common among patients with gastroenteritis-associated AKI. Comparable findings were reported by **Umakanth A, et. al; 2022** and **Shah SP, et. al; 2026**, who documented high frequencies of hypokalemia and hyponatremia in similar patient populations. Although electrolyte abnormalities contributed substantially to morbidity, they generally responded well to timely diagnosis and correction.

Septicemia emerged as one of the strongest predictors of adverse outcomes, with the majority of deaths occurring in patients with systemic infection. **Umakanth A, et. al; 2022** and **Shah SP, et. al; 2026**, confirmed sepsis as a major determinant of mortality because inflammatory responses, endothelial dysfunction, and impaired renal microcirculation accelerate renal injury. Shock also demonstrated a significant association with mortality, as prolonged hypotension worsens renal ischemia and promotes irreversible tubular damage. Previous studies have likewise emphasized the importance of aggressive management of septicemia and circulatory failure to improve survival [13,14,19]. The requirement for dialysis was closely associated with poor clinical outcomes and reflected the severity of renal injury rather than the direct cause of mortality. Patients requiring renal replacement therapy generally had advanced KDIGO Stage 3 AKI, persistent oliguria, severe azotemia, refractory electrolyte disturbances, metabolic acidosis, and fluid overload. **Anvar MI, et. al; 2018**, observed that patients requiring dialysis experienced more severe illness and delayed recovery, while **Patil U, et. al; 2018**, demonstrated significantly higher morbidity and mortality among individuals receiving renal replacement therapy. These findings reinforced the importance of early recognition of dehydration, maintenance of hemodynamic stability, and prompt treatment of AKI before progression to advanced stages. Early intervention not only preserves renal function but also reduces the need for dialysis, shortens hospital stay, and improves overall survival and clinical outcomes [16,20].

CONCLUSION

The present study demonstrated that acute kidney injury is a frequent complication of gastroenteritis, primarily resulting from dehydration-induced renal hypoperfusion, and is associated with prolonged hospitalization and poorer clinical outcomes. Most patients presented with Stage 2 or 3 AKI, with severe cases showing a greater need for renal replacement therapy, incomplete renal recovery, and higher mortality. Nevertheless, most patients achieved satisfactory recovery following early fluid resuscitation, electrolyte correction, and supportive care. Prompt diagnosis, timely referral, adequate hydration, and close clinical monitoring are essential to improve outcomes and minimize renal complications.

LIMITATIONS & FUTURE PERSPECTIVES

The study's limitations include a single-centre setting, a relatively small sample size, and a short study duration, which may limit the broader applicability of the results. Future studies should incorporate multicentre designs with larger populations to enhance validity, assess long-term outcomes, and investigate advanced diagnostic & management approaches. Such efforts will improve overall patient care & help minimize complications.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical health-care, diagnosis, and treatment outcomes. By highlighting real-care applications. It emphasizes the importance of translating scientific observations into meaningful improvements in patient world relevance, the study contributes to evidence based medical practice and supports informed clinical decision making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

AKI: Acute Kidney Injury

KDIGO: Kidney Disease: Improving Global Outcomes

CKD: Chronic Kidney Disease

GIT: Gastrointestinal Tract

eGFR: Estimated Glomerular Filtration Rate

ICU: Intensive Care Unit

SCr: Serum Creatinine

BUN: Blood Urea Nitrogen

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research directions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

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