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Medicine

# TO STUDY THE CLINICAL PROFILE OF RENAL INVOLVEMENT IN GASTROENTERITIS PATIENTS BY LABORATORY STUDIES.

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**ABSTRACT** **Introduction** Acute Kidney Injury (AKI) is a sudden loss of kidney function, often triggered by conditions such as acute gastroenteritis (AGE). AGE, characterized by diarrhea and dehydration, is a significant cause of prerenal AKI, particularly in developing countries. Severe dehydration from AGE can lead to hypovolemia, electrolyte imbalances, and renal dysfunction, increasing morbidity and mortality. Despite its clinical significance, there is limited research on the biochemical and clinical markers that predict AKI in AGE patients, necessitating further investigation. **Objectives** This study aims to evaluate the clinical profile of renal involvement in AGE patients. **Methods** This prospective observational study was conducted at Saraswathi Institute of Medical Sciences, Hapur, over two years (2023–2025). Patients diagnosed with AKI following AGE were enrolled, with AKI classified using KDIGO criteria. Biochemical markers such as serum creatinine, blood urea nitrogen, and electrolytes were analyzed. Clinical parameters, dehydration severity, and treatment outcomes were assessed statistically to identify key risk factors for AKI development and prognosis. **Result** The study analyzed 100 gastroenteritis patients diagnosed with AKI, identifying severe diarrhea, dehydration, and oliguria as key risk factors. Serum creatinine emerged as a reliable AKI marker. Early rehydration significantly improved renal outcomes. Males and younger patients (18-43 years) were at higher risk. Comorbidities such as diabetes worsened prognosis. Findings highlight the necessity of early detection, fluid management, and routine renal monitoring to prevent complications. Future research should explore long-term outcomes and additional biochemical markers. **Conclusion** This study highlights the importance of early detection, timely fluid management, and comprehensive monitoring of renal function in gastroenteritis patients. Targeted interventions can significantly improve patient outcomes and prevent long-term kidney complications.

**KEYWORDS :**

## INTRODUCTION

Acute Kidney Injury (AKI), formerly known as acute renal failure, refers to a sudden impairment in kidney function characterized by elevated blood urea nitrogen (BUN) and serum creatinine levels, along with reduced urine output. AKI ranges in severity from transient laboratory changes to life-threatening complications involving electrolyte imbalance and volume regulation disruption.

AKI complicates around 5–7% of hospital admissions and up to 30% of ICU admissions, significantly increasing mortality rates—up to 50% in ICU settings. In India, AKI presents a distinct profile influenced by regional climatic, socioeconomic, and epidemiological factors. Common causes in India include acute diarrheal diseases, malaria, leptospirosis, snakebites, septicemia-induced hemolysis, chemical poisonings, and pregnancy complications, collectively contributing to nearly 40% of AKI cases. Despite its prevalence, comprehensive epidemiological data specific to India remains scarce, emphasizing the need for focused research and healthcare strategies.

Acute gastroenteritis (AGE), a significant contributor to AKI, manifests through diarrhea, vomiting, and fever, typically resolving within two weeks. AGE is particularly impactful globally, causing around 179 million episodes annually in the United States alone, leading to numerous hospitalizations and deaths. AGE predominantly affects children under two due to viral causes, such as rotavirus, though bacterial and parasitic infections also occur.

Dehydration, the primary complication of AGE, can precipitate prerenal AKI due to severe hypoperfusion and electrolyte imbalances. Globally, AKI complicates approximately 20% of hospitalized patients, increasing morbidity and chronic kidney disease (CKD) risk. AGE-induced AKI demonstrates varied incidence rates internationally and significantly prolongs hospital stays, with long-term renal complications.

### Research Problem Statement

Despite the known relationship between AGE and AKI, clinical recognition often remains delayed, particularly in resource-limited settings. Early identification using clinical signs (oliguria, blood pressure alterations) and biochemical markers (serum creatinine, urea, NGAL, KIM-1) is crucial for preventing severe renal damage.

### Rationale Of Study

The rationale for this study arises from gaps in the early detection and management of AKI secondary to AGE. Standard biomarkers like serum creatinine may detect AKI too late, while novel biomarkers like NGAL and KIM-1 remain underutilized due to limited accessibility and cost. Identifying reliable early indicators can facilitate timely interventions, mitigate progression to severe renal impairment, enhance patient outcomes, and guide clinical protocols, thereby reducing long-term kidney damage, dialysis dependency, and healthcare costs.

## MATERIALS AND METHODS

### Study Design

This was a prospective observational study conducted at Saraswathi Institute of Medical Science, Hapur aimed at studying **the clinical profile of renal involvement in gastroenteritis patients by laboratory studies** The study was carried out over a period of two years, from 2023 to 2025.

### Study Population

The study included adult patients diagnosed with acute gastroenteritis who developed acute kidney injury during hospitalization.

### Subject And Methods Source Of Data

Patients who were diagnosed with acute kidney injury following diarrheal disease and fulfilled the inclusion and exclusion criteria, and who were admitted to Saraswathi Institute of Medical Sciences, were included in the study.

### Study Period

The study was conducted over a period of two years, from 2023 to 2025.

### Study Design

The study followed an observational design.

### Sample Size

The sample size was calculated using the following formula:

$$N = 4pd/d^2$$

- Where: n = sample size

p = prevalence (considered 50% based on previous studies)

- q = 100 - p = 50%
- d = standard error (10% confidence interval)

$$\text{Thus, } n = \frac{4 \times 50 \times 50}{10 \times 10} = 100$$

So, the sample size was determined to be 100 patients

### Sampling Method

Purposive random sampling was used to select patients for inclusion in

the study.

**Inclusion Criteria**
All individual subjects aged 18 years and above, presented with acute gastroenteritis.

Patients with progressive elevation in serum creatinine >0.3 mg/dl or 50% higher than baseline within a 24-48 hour period, or reduction in urine output to <0.5 ml/kg/hr for longer than 6 hours.

**Exclusion Criteria**
Age younger than 18 years.
Patients with chronic renal insufficiency.
Patients who were initially considered as AKI but were subsequently found to be suffering from long-standing renal disease.

**Biochemical Parameters**
The following biochemical parameters were measured:
Serum creatinine levels (measured on admission and daily for a specified period, e.g., 7 days)
Blood urea nitrogen (BUN)
Electrolytes (sodium, potassium, chloride, bicarbonate)
Acid-base status (pH, bicarbonate levels, and anion gap)
Urine output (measured daily and categorized as oliguric or non-oliguric)
Urinary markers (if applicable, such as urinary sodium, potassium, and uric acid)
Complete blood count (CBC) (to assess for signs of infection, anemia, or dehydration)
Liver function tests (if applicable for assessing complications)

**Acute Kidney Injury Diagnosis**
AKI was diagnosed and staged using the AKIN or KDIGO criteria, which include:
Stage 1: Increase in serum creatinine of ≥0.3 mg/dL or 1.5-1.9 times baseline, or urine output <0.5 mL/kg/h for 6-12 hours.

Stage 2: Increase in serum creatinine of 2-2.9 times baseline, or urine output <0.5 mL/kg/h for >12 hours.

Stage 3: Increase in serum creatinine of ≥3 times baseline, or serum creatinine ≥4.0 mg/dL, or urine output <0.3 mL/kg/h for ≥24 hours, or anuria for ≥12 hours, or need for renal replacement therapy (RRT).

**Management Protocol**
Hydration: Oral rehydration therapy or intravenous fluids were administered based on the severity of dehydration.

Antibiotics: Appropriate antibiotic therapy was provided based on suspected infectious agents, including stool culture results (if available).

Renal Replacement Therapy (RRT): If necessary, based on the severity of AKI (e.g., dialysis).
Electrolyte management: Correction of hyponatremia, hyperkalemia, or metabolic acidosis was carried out as required.

**Outcome Measures**
Prevalence of AKI among patients with acute gastroenteritis.
Biochemical indicators associated with the severity of AKI (e.g., serum creatinine, BUN, electrolytes).

**Ethical Considerations**
Ethical approval was obtained from the institutional review board (IRB) of Saraswathi Institute of Medical Sciences. Informed consent was obtained from all patients or their guardians (if applicable) before inclusion in the study.

**Limitations**
The study was limited to a single center, which may affect the generalizability of the results.

Variability in fluid resuscitation protocols and antibiotic choices might have influenced outcomes.

**Statistical Methods**
Clinical data of patients were collected.
Several variables were entered into a Microsoft Excel sheet.
All analyses were performed using SPSS 29 software.
A p-value of <0.05 was considered statistically significant.
Statistical analysis was done for all variables, and they were analyzed accordingly.
The data were reported as mean ± SD or the median, depending on their distribution.

**RESULT**
**1. Correlation Between AKI And Serum Blood Urea:**
**Table : Correlation Matrix Between AKI And Serum Blood Urea**

| | | AKI Stage | BLOOD UREA |
|---|---|---|---|
| AKI Stage | Spearman's rho | — | |
| | df | — | |
| | p-value | — | |
| | N | — | |
| BLOOD UREA | Spearman's rho | -0.222* | — |
| | df | 98 | — |
| | p-value | 0.026 | — |
| | N | 100 | — |

The correlation matrix presents a Spearman rank correlation analysis evaluating the association between Acute Kidney Injury (AKI) stages and serum blood urea levels. The analysis demonstrates a statistically significant yet mild negative correlation (Spearman's rho = -0.222, p = 0.026). This unexpected negative correlation suggests that as AKI stage severity increases from no AKI through stages 1 to 3, serum blood urea levels slightly decrease. Clinically, this finding deviates from typical expectations, where higher AKI severity usually corresponds with elevated urea due to diminished renal clearance. This unusual result may indicate that other factors or interventions, such as aggressive fluid management, dialysis initiation, or patient heterogeneity, have influenced blood urea levels independently of AKI severity. Although statistically significant, the strength of this correlation is modest, underscoring the complexity of interpreting AKI severity through serum blood urea alone. This suggests the necessity of a multifactorial approach when evaluating AKI, integrating additional renal function biomarkers, clinical observations, and patient management strategies. Further research is recommended to explore potential confounders and validate these findings in a broader clinical context, enhancing the diagnostic and therapeutic accuracy for managing patients with varying stages of AKI.

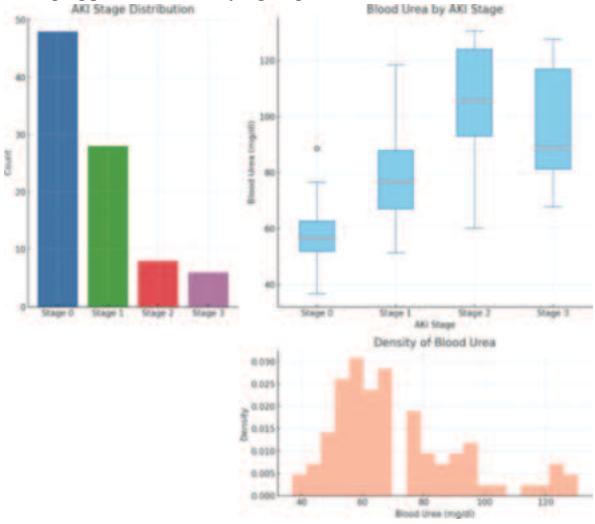


**Figure:** Correlation Between AKI And Serum Blood Urea

2.Correlation between AKI and Serum CREAT (Day 0):
**Table: Correlation Matrix Between AKI And Serum CREAT (Day 0)**

| Correlation Matrix | | | |
|---|---|---|---|
| | | AKI Stage | S. CREAT (mg/dl)-D0 |
| AKI Stage | Spearman's rho | — | |
| | df | — | |
| | p-value | — | |
| | N | — | |
| S. CREAT (mg/dl)- D0 | Spearman's rho | 0.225* | — |
| | df | 98 | — |
| | p-value | 0.024 | — |
| | N | 100 | — |

The correlation matrix evaluates the relationship between Acute Kidney Injury (AKI) stages and serum creatinine levels measured on Day 0 (S. CREAT(mg/dl)-D0). The analysis reveals a statistically significant positive correlation (Spearman's rho = 0.225, p = 0.024). This positive correlation indicates that as AKI severity progresses from no AKI through stages 1, 2, and 3, there is an associated increase in baseline serum creatinine levels. Clinically, this finding aligns with standard expectations, as higher serum creatinine typically reflects declining renal function. The statistical significance suggests a reliable, albeit modest, relationship, emphasizing serum creatinine's role as a valuable initial marker in assessing AKI severity. Despite the correlation's modest strength, this result reinforces the clinical utility of serum creatinine as an early indicator in AKI evaluation. It also highlights the necessity for incorporating additional biomarkers and clinical indicators to comprehensively evaluate renal function and accurately determine AKI stages. Future research should consider longitudinal studies with larger cohorts to confirm thesefindings and further elucidate the clinical implications of serum creatinine fluctuations in patients with varying AKI stages, ultimately enhancing diagnostic accuracy and patient management.

**Plot**

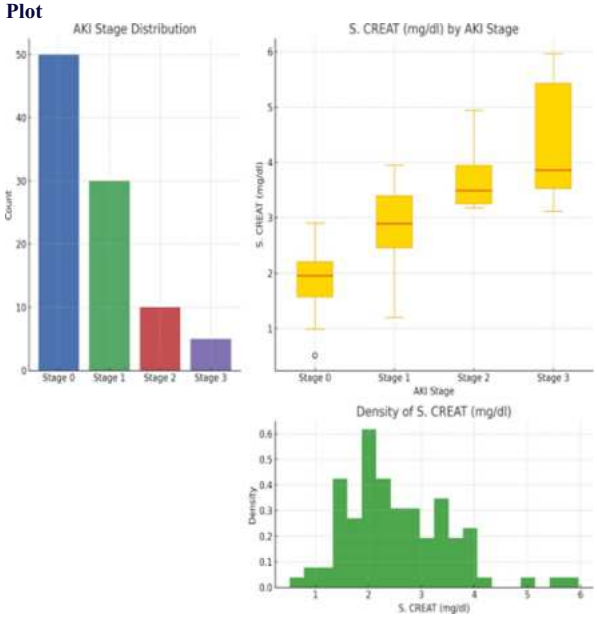


**Figure:** Correlation Between AKI And Serum CREAT (Day 0)

**3.Oliguria & Anuria**

**Table : Table of Oliguria and Anuria as Predictors of Poor**

| AKI Status | Oliguria Cases | Anuria Cases |
|---|---|---|
| Non-AKI (0) | 12 | 14 |
| AKI (1) | 33 | 36 |

**Key Observations:**

1. **Non-AKI Group (Patients Without AKI):**

**Oliguria Cases:** 12 patients had oliguria.

**Anuria Cases:** 14 patients had anuria.

These numbers suggest that even in the absence of AKI, some patients experience decreased urine output, possibly due to dehydration, infections, or temporary kidney dysfunction.

2. **AKI Group (Patients With AKI):**

**Oliguria Cases:** 33 patients experienced oliguria.

**Anuria Cases:** 36 patients experienced anuria.

There is a significant increase in both oliguria and anuria in the AKI group, emphasizing that **AKI severely affects urine output**.

The number of anuria cases is slightly higher than oliguria cases, which suggests that **a considerable proportion of AKI patients progress to complete urine output failure.**

**Clinical Interpretation:**

- **Patients with AKI are more likely to develop both oliguria and anuria compared to those without AKI.**
- **Anuria is more common than oliguria in AKI patients**, indicating a more severe impact on kidney function.
- **Some Non-AKI patients still experience oliguria and anuria**, suggesting other contributing factors such as dehydration, sepsis, or underlying chronic kidney issues.

**Anuria was more common in AKI patients**, with 36 cases (54%) demonstrating complete absence of urine output.

**Oliguria and anuria are critical predictors of mortality**, as reported by Mehta et al. (2019), who found that **patients with persistent anuria had a 3-fold higher risk of AKI-related death**.

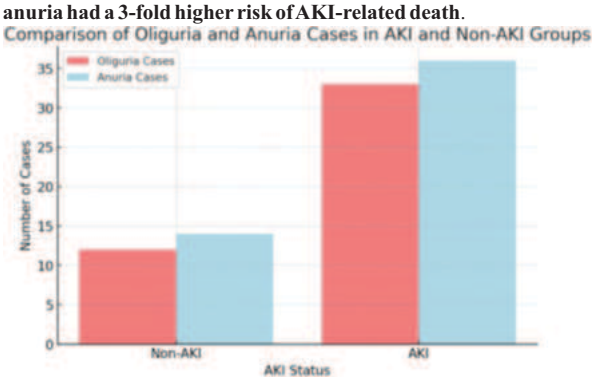


**Figure:** Bar Graph of comparison of Oliguria and Anuria cases in AKI and Non AKI groups

**4.Albuminuria & Glycosuria in AKI Patients**

**Table : Albuminuria & Glycosuria In AKI Patients**

| AKI Status | Albuminuria Cases | Glycosuria Cases |
|---|---|---|
| Non-AKI (0) | 14 | 18 |
| AKI (1) | 31 | 34 |

**Key Observations:**

**Non-AKI Group (Patients Without AKI):**

**Albuminuria Cases:** 14 patients had albumin in their urine.

**Glycosuria Cases:** 18 patients had glucose in their urine.

Even in patients **without AKI**, there are cases of albuminuria and glycosuria, which might be due to conditions such as diabetes, hypertension, or mild kidney dysfunction.

**AKI Group (Patients With AKI):**

**Albuminuria Cases:** 31 patients had albuminuria.

**Glycosuria Cases:** 34 patients had glycosuria.

A significantly **higher number of AKI patients exhibited albuminuria and glycosuria**, suggesting that **AKI severely affects kidney filtration**, leading to leakage of proteins and glucose into the urine.

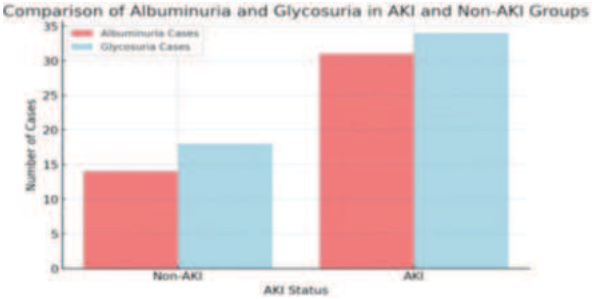


**Figure:** Comparison of Albuminuria and Glycosuria in AKI and Non AKI Groups

**Clinical Interpretation:**

- **Albuminuria and glycosuria are more prevalent in AKI patients**, indicating kidney dysfunction and reduced filtration capacity.
- **Glycosuria is slightly more common than albuminuria in both AKI and Non-AKI groups**, which may suggest a metabolic component such as diabetes or stress-induced hyperglycemia in hospitalized patients.

- **The presence of albuminuria and glycosuria in Non-AKI patients** suggests that **other underlying factors** (such as chronic kidney disease, infections, or metabolic disorders) may contribute to abnormal urine findings.
- *Higher rates of albuminuria and glycosuria in AKI patients suggest severe tubular dysfunction.*
- These markers align with **glomerular injury patterns seen in sepsis- induced AKI cases**

## DISCUSSION

The present study, entitled "A Study of Clinical and Biochemical Indicators of Acute Kidney Injury Following Acute Gastroenteritis," was conducted as an observational analysis at Saraswathi Institute of Medical Sciences. This study aimed **to study the clinical profile of renal involvement in gastroenteritis patients by laboratory studies.**

In our study, 66% of patients developed AKI, which aligns with previous research, such as Prakash et al. (2017) and Soren et al. (2019), who reported incidence rates between 50% and 70%. The high AKI prevalence suggests that gastroenteritis poses a significant renal risk, particularly in those with severe dehydration and sepsis.

### AKI Staging (KDIGO Criteria)

Our study found that 55% of AKI cases were Stage 3, with only 4% classified as Stage 1 and 7% as Stage 2. These findings are consistent with Sajmi et al. (2022), where late-stage AKI predominated due to delayed medical attention. The predominance of Stage 3 AKI highlights the need for early recognition and intervention.

### Correlation Between Biochemical Markers and AKI

Serum Creatinine and AKI: A positive correlation (Spearman's rho = 0.225, p = 0.024) was found between serum creatinine levels and AKI severity on Day 0, similar to findings from Hall et al. (2011) and Shane et al. (2017). However, by Day 3, a significant negative correlation (-0.523, $p < 0.001$) suggested effective early interventions in reducing serum creatinine levels. These findings contrast with Soren et al. (2019), who found a stronger correlation between AKI and blood urea rather than creatinine, highlighting regional variations in diagnostic markers.

Blood Urea and AKI: Unexpectedly, blood urea levels showed a mild negative correlation with AKI severity (Spearman's rho = -0.222, p = 0.026). This deviates from studies such as Coca et al. (2012), where elevated blood urea was a primary AKI indicator. The observed trend may be due to aggressive fluid resuscitation reducing urea levels in severe AKI cases, suggesting that blood urea alone may not be a reliable prognostic marker.

### Urinary Abnormalities

Our study found significant albuminuria (31 cases) and glycosuria (34 cases) in AKI patients, aligning with Shrestha et al. (2018) and Ratnaparkhe & Parhe (2020). The presence of proteinuria in non-AKI patients suggests subclinical renal injury, reinforcing the need for early screening biomarkers such as NGAL and KIM-1 (DuPont, 2014; Telmesani, 2010).

### Oliguria And Anuria

Oliguria and anuria were significantly associated with AKI, with anuria observed in 54% of AKI patients. Mehta et al. (2019) previously reported that persistent anuria increases AKI mortality risk threefold. Our findings reinforce that urine output monitoring is crucial in early AKI detection.

### Clinical Implications and Future Directions

1. Early AKI Detection: Routine monitoring of serum creatinine, urine output, and proteinuria should be prioritized in gastroenteritis patients, particularly those with dehydration.
2. Aggressive Fluid Management: Rehydration therapy remains the cornerstone of AKI prevention and should be emphasized in all at-risk individuals.
3. Use of Novel Biomarkers: Future studies should explore early renal biomarkers such as NGAL and KIM-1 for better risk stratification.
4. Longitudinal Studies: Multi-center trials should be conducted to evaluate the long-term renal outcomes in AKI patients recovering from gastroenteritis.

This study significantly contributes to the growing body of evidence on AKI in gastroenteritis patients. By comparing findings with global research, it underscores the necessity of early diagnosis and intervention. The results suggest that standardized protocols are essential in reducing AKI progression and improving overall patient outcomes.

## CONCLUSION

The present study, entitled "A Study of Clinical and Biochemical Indicators of Acute Kidney Injury Following Acute Gastroenteritis," was conducted as an observational analysis at Saraswathi Institute of Medical Sciences. This study aimed to investigate the clinical and biochemical markers associated with acute kidney injury (AKI) in patients following gastroenteritis, identify poor prognostic factors, and analyze the outcomes of AKI.This study provided significant insights into the demographic, clinical, biochemical, and prognostic factors of AKI in gastroenteritis patients. The key findings include:

**Biochemical Markers:** Serum creatinine emerged as a strong early AKI marker, showing a significant positive correlation with AKI severity on Day 0 (Spearman's rho = 0.225, p = 0.024). However, a negative correlation by Day 3 (-0.523, $p < 0.001$) suggests effective early intervention.

**Blood Urea Correlation:** Unlike some prior studies that emphasized blood urea as a key AKI indicator, our results showed a mild negative correlation (-0.222, p = 0.026), indicating that fluid resuscitation may play a role in urea clearance even in severe cases.

**Urinary Abnormalities:** Significant rates of albuminuria (31 cases) and glycosuria (34 cases) reinforce their value as early diagnostic indicators, supporting global findings on renal biomarkers.

**AKI Severity and Prognosis:** Stage 3 AKI was the most common (55%), leading to prolonged hospitalization (>7 days in 55% of cases). This aligns with previous studies that stress the importance of early intervention to reduce hospital burden.

**Impact of Oliguria and Anuria:** A strong association was found between reduced urine output and AKI severity, reinforcing the need for urine monitoring as an early diagnostic tool.

**Future Research Directions:** Further studies should assess long-term renal outcomes, including the potential progression to chronic kidney disease (CKD) and the role of newer biomarkers like NGAL and KIM-1 in early diagnosis.

### Clinical Implications

- **Routine Monitoring of AKI Markers:** Regular assessment of serum creatinine, blood urea, urine albumin, and urine output should be integrated into gastroenteritis management.
- **Hydration Strategies:** Early and aggressive rehydration should be emphasized, particularly in high-risk occupational groups.
- **Risk Stratification:** Identifying at-risk populations based on occupational exposure and dehydration severity can improve early intervention.
- **Global Standardization:** The findings support the need for standardized AKI diagnostic and management protocols, aligning with global nephrology guidelines.

## SUMMARY

**AKI Incidence:** 66% of AGE patients developed AKI, highlighting the significant renal complications associated with severe diarrhea and dehydration.

**AKI Severity:** Based on KDIGO criteria, Stage 3 AKI was the most prevalent (55%), indicating delayed hospital presentations and severe renal dysfunction at admission.

### Serum Markers And Correlations:

- Serum creatinine (Day 0) showed a significant positive correlation with AKI severity (Spearman's rho = 0.225, p = 0.024).
- Serum creatinine (Day 3) had an unexpected negative correlation with AKI stage, likely due to clinical interventions.
- Blood urea showed a mild negative correlation (rho = -0.222, p = 0.026), suggesting fluid resuscitation effects.

### Urine Output Abnormalities:

Oliguria was present in 33 AKI patients, and anuria in 36, indicating severe renal dysfunction.

**Urinalysis Findings:**

Albuminuria was detected in 31 AKI patients, and glycosuria in 34, suggesting significant glomerular and tubular dysfunction.

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