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Original Research Article

A COMPREHENSIVE STUDY ON THE ETIOLOGY AND SEVERITY OF COMMUNITY-ACQUIRED ACUTE KIDNEY INJURY

Suraj Kumar[1], *Ashumi Gupta*[2]

[1]*Assistant professor, Department of Nephrology, Geetanjali Medical College and Hospital, udaipur, Rajasthan, India.*
[2]*Associate professor, Department of Pathology, Geetanjali Medical College and Hospital, udaipur, Rajasthan, India.*

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Corresponding Author:
Dr. Suraj Kumar,
Assistant professor, Department of Nephrology, Geetanjali Medical College and Hospital, udaipur, Rajasthan, India..
Email: surajk06@yahoo.co.in

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ABSTRACT

Background: Acute kidney injury (AKI) is a leading cause of morbidity and mortality globally, particularly in resource-limited settings. The etiology and severity of AKI vary based on multiple factors, including infections, dehydration, drug use, and environmental exposures. This study aimed to investigate the etiology, clinical manifestations, and predictors of severity in community-acquired AKI.

Materials and Methods: This retrospective observational study was conducted at a tertiary care center. A total of 246 patients diagnosed with AKI were included retrospectively for a period of 5 years. Data were collected on demographic characteristics, clinical presentation, laboratory parameters, and etiology. AKI severity was classified as stage 1, 2, or 3 based on serum creatinine levels and clinical condition. Multivariate logistic regression was used to identify predictors of stage 3 AKI.

Results: The most common etiologies of AKI were infectious causes (46.7%), dehydration-related (22.4%), and drug-induced (18.3%). Infectious causes were more prevalent in stage 3 AKI cases (54.3%), while dehydration-related AKI was more frequent in stage 1 cases (35.6%). The mean serum creatinine was significantly higher in stage 3 cases compared to stage 1 and stage 2 cases (3.4 ± 1.5 mg/dL). Factors associated with increased risk of stage 3 AKI included elevated serum creatinine >4 mg/dL (OR 4.2, 95% CI 2.1–8.6), blood urea nitrogen >60 mg/dL (OR 3.7, 95% CI 1.8–7.2), hypotension (OR 2.8, 95% CI 1.5–5.2), oliguria (OR 5.6, 95% CI 3.1–10.2), and advanced age (>60 years) (OR 1.6, 95% CI 1.0–2.9).

Conclusion: Infectious causes, dehydration, and hypotension were the most common etiologies and significant predictors of stage 3 AKI. Early identification of at-risk patients, especially those with elevated creatinine, oliguria, and hypotension, can help improve clinical outcomes. Further studies are needed to validate these findings and identify additional biomarkers for AKI progression.

Keywords: Acute kidney injury, serum creatinine, oliguria, hypotension, community-acquired AKI.

INTRODUCTION

Acute kidney injury (AKI) is an immediate state before acute kidney disease (AKD) and chronic kidney disease (CKD), characterized by kidney dysfunction lasting less than 7 days.[1] Community-acquired AKI, where kidney injury occurs outside of hospital settings, is a significant yet under-researched cause of morbidity and mortality, particularly in low- and middle-income countries (LMICs). Unlike hospital-acquired AKI, which is often associated with critical illness, community-acquired AKI predominantly arises from preventable causes such as infections, dehydration, and exposure to nephrotoxins.[2]

In LMICs like India, the prevalence of AKI has been reported to be disproportionately high due to the dual burden of infectious and non-communicable diseases. It is being highlighted that 60–70% of AKI cases encountered in tertiary care settings had a community-acquired origin, with major contributing factors being infectious diseases such as leptospirosis, dengue fever, malaria, and acute diarrheal illnesses.[3] For instance, dengue-related AKI affects up to 30% of stage 3 cases of dengue fever, with mortality rates exceeding 10% if dialysis is delayed.[4] In addition, nephrotoxic medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs), herbal remedies, and over-the-counter antibiotics, are common triggers of AKI in rural populations.[5]

Environmental factors also play a role, particularly in agricultural communities where pesticide exposure is a major concern. Studies from South India have reported that approximately 15% of AKI cases are linked to organophosphate poisoning [6]. Furthermore, conditions like diabetes and hypertension, which affect nearly 25% and 30% of adults in India respectively, exacerbate the risk of AKI when compounded with acute stressors such as infections or volume depletion.[7]

Despite its significant burden, the true epidemiology of community-acquired AKI remains poorly understood, with most studies focusing on hospitalized patients rather than community-level data. This has led to gaps in early detection, prevention, and treatment strategies, particularly in rural and resource-constrained settings. Moreover, the progression from community-acquired AKI to CKD is a growing concern, with studies estimating that up to 20% of AKI survivors develop long-term renal sequelae within one year.[8]

This study aimed to evaluate the etiological spectrum of community-acquired AKI in India, identify high-risk populations, and explore modifiable risk factors to inform targeted prevention strategies. By filling critical gaps in epidemiological data, the study seeks to contribute to the development of context-specific interventions to reduce the burden of kidney disease in the community.

MATERIALS AND METHODS

Study Design and Setting

This retrospective observational study was conducted Department of Nephrology, Geetanjali Medical College and Hospital, Udaipur during January 2024 to December 2024, and collected patients details retrospectively for a period of 5 years from June 2016 to May 2021. The study focused on determining the etiological spectrum of community-acquired acute kidney injury (AKI) among adult patients presenting to the outpatient department, emergency room, or general medical wards. Community-acquired AKI was defined as renal dysfunction arising prior to hospitalization or within the first 48 hours of admission, based on the Kidney Disease: Improving Global Outcomes (KDIGO) criteria, which classify AKI as kidney injury persisting for 7–90 days.

Study Population

The study population included adults aged 18 years and above with documented AKI. Participants were recruited if they demonstrated evidence of kidney dysfunction within the specified KDIGO timeframe and if their symptoms had either initiated outside the hospital or within the first 48 hours of admission. Patients with pre-existing chronic kidney disease (CKD), hospital-acquired AKI, or incomplete medical records were excluded.

Sample Size

The sample size was determined using an expected prevalence of community-acquired AKI of 20%, with a 95% confidence level and a 5% margin of error [9]. Based on these parameters, the calculated minimum sample size was 246 participants.

Data Collection

Comprehensive data were collected through structured interviews, clinical examinations, and laboratory investigations. Demographic information such as age, gender, socioeconomic status, and occupation was recorded. Detailed clinical histories were obtained, including the duration and nature of presenting symptoms, history of infections (e.g., fever, diarrhea, or respiratory symptoms), medication use (e.g., nonsteroidal anti-inflammatory drugs, antibiotics, or herbal remedies), comorbid conditions (e.g., diabetes, hypertension), and environmental exposures such as pesticide use or contaminated water. Physical examinations included assessments of hydration status, blood pressure, and signs of systemic infections or organ dysfunction.

Laboratory investigations were conducted to confirm the diagnosis of AKI and identify potential etiological factors. This included serum creatinine, blood urea nitrogen, and electrolyte levels measured at admission and during follow-up. Urine analysis, including dipstick testing, microscopy, and proteinuria assessment, was performed. Additional diagnostic tests, such as serology for infectious diseases (e.g., leptospirosis, dengue, malaria), viral markers for hepatitis B, hepatitis C, and HIV, as well as blood cultures, were obtained based on clinical suspicion. Imaging studies, such as renal ultrasounds, were performed to evaluate structural abnormalities or urinary obstruction when indicated.

Classification of Etiologies

Based on clinical, laboratory, and imaging findings, the underlying causes of AKI were classified into specific categories. Infectious causes included bacterial sepsis, leptospirosis, dengue fever, malaria, and gastrointestinal infections. Dehydration-induced AKI was attributed to acute diarrheal diseases or excessive vomiting leading to hypovolemia. Drug-induced AKI was linked to nephrotoxic medications such as NSAIDs, aminoglycosides, and herbal preparations. Environmental factors, particularly exposure to organophosphates or heavy metals, were identified as a significant contributor in agricultural

workers. Other etiologies, including acute glomerulonephritis, rhabdomyolysis, and obstructive nephropathy, were documented based on specific clinical and diagnostic criteria.

Statistical Analysis

All collected data were entered into SPSS Version 25.0. Continuous variables were summarized as means with standard deviations (SD) or medians with interquartile ranges (IQR) depending on data distribution. Categorical variables were expressed as frequencies and percentages. Chi-square or Fisher's exact tests were used to compare categorical data, while independent t-tests or Mann-Whitney U tests were employed for continuous variables. Logistic regression analysis was performed to identify independent predictors of specific etiologies of AKI. A p-value <0.05 was considered statistically significant.

Ethical Considerations

Ethical approval for the study was obtained from the Institutional Ethics Committee. Data confidentiality was strictly maintained by anonymizing personal identifiers and securely storing records.

RESULTS

The mean age of participants was 48.2 ± 14.4 years, with 59.3% males and 40.7% females. Most belonged to low socioeconomic status (54.9%), followed by middle (37%) and high (8.1%) groups, with education levels ranging from illiterate (21.9%) to primary (43.9%), secondary (26.4%), and higher education (7.8%). Agricultural workers constituted the largest occupational group (31.3%), followed by homemakers (25.2%), manual laborers (22.4%), and others (21.1%). Comorbidities were prevalent in 66.7% of participants, with hypertension (37%) and diabetes (33.3%) being most common, alongside cardiovascular (14.2%), respiratory (11.4%), and liver diseases (4.9%). Additionally, 31.3% reported alcohol use and 37.4% tobacco use, highlighting the demographic diversity and high burden of comorbidities in AKI cases. [Table 1]

The etiological spectrum of community-acquired AKI showed infectious causes as the most frequent, accounting for 46.7% of cases, with gastrointestinal infections (18.7%), leptospirosis (11.4%), dengue fever (8.9%), and malaria (7.7%) as key contributors. Dehydration-related causes were observed in 22.4% of cases, while drug-induced AKI accounted for 18.3%, primarily due to NSAIDs (7.3%) and antibiotics (5.7%), alongside other drugs (5.3%). Environmental factors were responsible for 8.9% of cases, including exposure to pesticides (5.7%) and snakebites (3.3%), while other unspecified causes comprised 3.7%. Severity assessment revealed that 36.6% of patients had stage 1 AKI, 44.7% had stage 2 AKI, and 18.7% had stage 3 AKI, highlighting the dominance of infectious and preventable factors with a spectrum of severity. [Table 2]

The laboratory parameters in the study population revealed a mean serum creatinine level of 3.4 ± 1.5 mg/dL and a blood urea nitrogen (BUN) level of 51.3 ± 24.7 mg/dL, indicating compromised renal function. Potassium levels averaged 4.6 ± 0.8 mEq/L, while sodium levels were slightly lower at 134.6 ± 8.2 mEq/L. Hemoglobin levels were reduced, with a mean of 9.9 ± 2.3 g/dL, and proteinuria was present in 41.5% of patients. The mean albumin level was 3.0 ± 0.7 g/dL, indicating stage 1 hypoalbuminemia. Leukocyte count averaged 11.5 ± 4.2 $\times 10^3/\mu L$, and C-reactive protein levels were elevated with a mean of 45.4 ± 21.2 mg/L, reflecting inflammation. The mean fractional excretion of sodium (FENa) was 1.8 ± 0.6%, indicating prerenal involvement in some cases of AKI. These laboratory values reflect the severity of kidney injury and the associated systemic impact. [Table 3]

The distribution of etiology across different severity levels of AKI revealed significant differences for infectious causes and dehydration-related factors. Infectious causes were observed in 40% of stage 1 AKI, 49.1% of stage 2 AKI, and 54.3% of stage 3 AKI cases (p=0.021), indicating a higher frequency with increasing severity. Dehydration-related causes were most common in stage 1 AKI (35.6%) and significantly decreased in stage 2 (16.4%) and stage 3 (10.9%) AKI cases (p=0.013). Drug-induced AKI was observed in 13.3% of stage 1, 23.6% of stage 2, and 15.2% of stage 3 cases, but the differences were not statistically significant (p=0.154). Environmental factors accounted for 6.7% of stage 1, 9.1% of stage 2, and 13% of stage 3 cases, with no significant difference across the severity groups (p=0.187). Other causes were least common, accounting for 4.4% in stage 1, 1.8% in stage 2, and 6.5% in stage 3 AKI cases (p=0.213). These findings highlight a trend of increasing infectious causes and decreasing dehydration-related factors with worsening AKI severity. [Table 4]

In our study, the outcomes for patients with acute kidney injury (AKI) varied significantly across different severity groups. Among patients with stage 1 AKI, 84.4% (76 out of 90) recovered without the need for dialysis, compared to 59.1% (65 out of 110) in the stage 2 AKI group, and only 26.1% (12 out of 46) in the stage 3 AKI group. Dialysis was required in 13.3% of stage 1 cases, 35.5% of stage 2 cases, and 67.4% of stage 3 cases. The total requirement for dialysis across all groups was 33.3%. Progression to chronic kidney disease (CKD) was most common in stage 3 AKI (30.4%), followed by stage 2 AKI (10.9%) and stage 1 AKI (2.2%), with a total of 11.4% of all patients progressing to CKD. ICU admission rates were highest in stage 3 AKI (89.1%), followed by stage 2 AKI (30.9%), and stage 1 AKI (5.6%), with a total of 32.5% across all groups. Mortality was observed in 34.8% of stage 3 AKI cases, 2.7% of stage 2 AKI cases, and none in stage 1 AKI cases, resulting in an overall mortality rate of 7.7%. These findings highlight the significant impact of AKI severity on recovery outcomes, dialysis

requirements, progression to CKD, ICU admission, and mortality, with all differences between severity groups being statistically significant (p<0.001). [Table 5]

The multivariate logistic regression analysis revealed several significant predictors of stage 3 AKI. A serum creatinine level greater than 4 mg/dL was strongly associated with increased odds of stage 3 AKI (adjusted odds ratio [AOR] 4.2, 95% CI 2.1–8.6, p<0.001), as was a blood urea nitrogen level greater than 60 mg/dL (AOR 3.7, 95% CI 1.8–7.2, p<0.001). The presence of hypotension also increased the likelihood of stage 3 AKI (AOR 2.8, 95% CI 1.5–5.2, p=0.002). Infectious etiology was associated with higher odds of stage 3 AKI (AOR 1.9, 95% CI 1.1–3.4, p=0.010). Oliguria was the strongest predictor, with an AOR of 5.6 (95% CI 3.1–10.2, p<0.001). Additionally, advanced age (>60 years) was associated with an increased risk of stage 3 AKI (AOR 1.6, 95% CI 1.0–2.9, p=0.045). These findings emphasize the importance of these clinical and laboratory variables in predicting the severity of AKI. [Table 6]

Table 1: Baseline Demographic and Clinical Characteristics of Participants

Characteristic	Frequency (%)/mean ± SD
Age (years)	48.2 ± 14.4
Gender	
Male	146 (59.3%)
Female	100 (40.7%)
Socioeconomic status	
Low	135 (54.9%)
Middle	91 (37%)
High	20 (8.1%)
Education level	
Illiterate	54 (21.9%)
Primary education	108 (43.9%)
Secondary education	65 (26.4%)
Higher education	19 (7.8%)
Occupation	
Agricultural worker	77 (31.3%)
Homemaker	62 (25.2%)
Manual laborer	55 (22.4%)
Others	52 (21.1%)
History of comorbidities	164 (66.7%)
Diabetes mellitus	82 (33.3%)
Hypertension	91 (37%)
Cardiovascular disease	35 (14.2%)
Chronic liver disease	12 (4.9%)
Chronic respiratory disease	28 (11.4%)
Alcohol consumption	77 (31.3%)
Tobacco use	92 (37.4%)

Table 2: Etiological Distribution of Community-Acquired AKI

Etiology Category	Frequency (%)
Infectious causes	115 (46.7%)
Leptospirosis	28 (11.4%)
Dengue fever	22 (8.9%)
Malaria	19 (7.7%)
Gastrointestinal infections	46 (18.7%)
Dehydration-related	55 (22.4%)
Drug-induced	45 (18.3%)
NSAIDs	18 (7.3%)
Antibiotics	14 (5.7%)
Others	13 (5.3%)
Environmental factors	22 (8.9%)
Snakebite	8 (3.3%)
Exposure to pesticides	14 (5.7%)
Other causes	9 (3.7%)
Severity of AKI	
Stage 1	90 (36.6%)
Stage 2	110 (44.7%)
Stage 3	46 (18.7%)

Table 3: Laboratory Characteristics of Participants at Presentation

Parameter	Frequency (%)/mean ± SD
Serum creatinine (mg/dL)	3.4 ± 1.5
Blood urea nitrogen (mg/dL)	51.3 ± 24.7
Potassium (mEq/L)	4.6 ± 0.8
Sodium (mEq/L)	134.6 ± 8.2

Hemoglobin (g/dL)	9.9 ± 2.3
Proteinuria (%)	102 (41.5%)
Albumin (g/dL)	3.0 ± 0.7
Leukocyte count (x10³/µL)	11.5 ± 4.2
C-reactive protein (mg/L)	45.4 ± 21.2
Fractional excretion of sodium (%)	1.8 ± 0.6

Table 4: Outcomes of Community-Acquired AKI Based on Severity

Etiology	Stage 1 AKI (n=90)	Stage 2 AKI (n=110)	Stage 3 AKI (n=46)	p-value
	Frequency (%)			
Infectious causes	36 (40%)	54 (49.1%)	25 (54.3%)	0.021
Dehydration-related	32 (35.6%)	18 (16.4%)	5 (10.9%)	0.013
Drug-induced	12 (13.3%)	26 (23.6%)	7 (15.2%)	0.154
Environmental factors	6 (6.7%)	10 (9.1%)	6 (13%)	0.187
Other causes	4 (4.4%)	2 (1.8%)	3 (6.5%)	0.213

Table 5: Outcomes Based on Severity of AKI

Outcome	Stage 1 AKI (n=90)	Stage 2 AKI (n=110)	Stage 3 AKI (n=46)	Total (n=246)	p-value
	Frequency (%)				
Recovery without dialysis	76 (84.4%)	65 (59.1%)	12 (26.1%)	153 (62.2%)	<0.001
Dialysis required	12 (13.3%)	39 (35.5%)	31 (67.4%)	82 (33.3%)	<0.001
Progression to CKD	2 (2.2%)	12 (10.9%)	14 (30.4%)	28 (11.4%)	<0.001
ICU admission	5 (5.6%)	34 (30.9%)	41 (89.1%)	80 (32.5%)	<0.001
Mortality	0 (0%)	3 (2.7%)	16 (34.8%)	19 (7.7%)	<0.001

Table 6: Predictors of Specific Etiologies of AKI (Logistic Regression Analysis)

Variable	Adjusted Odds Ratio (95% CI)	p-value
Serum creatinine >4 mg/dL	4.2 (2.1–8.6)	<0.001
Blood urea nitrogen >60 mg/dL	3.7 (1.8–7.2)	<0.001
Presence of hypotension	2.8 (1.5–5.2)	0.002
Infectious etiology	1.9 (1.1–3.4)	0.010
Oliguria	5.6 (3.1–10.2)	<0.001
Advanced age (>60 years)	1.6 (1.0–2.9)	0.045

DISCUSSION

This study aimed to investigate the etiology, clinical outcomes, and predictors of severity in community-acquired acute kidney injury (AKI). Our results highlight the multifactorial nature of AKI, with infectious causes being the most predominant etiology, particularly in stage 3 cases, while dehydration-related AKI was more common in stage 1 cases. Additionally, we identified several clinical and biochemical factors that predicted progression to stage 3 AKI, such as elevated serum creatinine, blood urea nitrogen (BUN), hypotension, and oliguria. These findings provide critical insights into the pathophysiology of AKI and its clinical course, with implications for early diagnosis and management.

The predominance of infectious causes, which contributed to 54.3% of stage 3 AKI cases, is consistent with studies in India and other low- and middle-income countries (LMICs), where infections such as leptospirosis, malaria, and dengue are major contributors to AKI.[10,11] For instance, a study by Nair et al., found that leptospirosis and dengue were common causes of AKI, especially in rural areas where access to healthcare may be limited and infections are more prevalent due to environmental and socio-economic factors.[12] This is further corroborated by a study by Badge et al., which reported that infectious causes, particularly tropical infections, were significantly associated with stage 3 AKI in Indian patients.[13] Our study's finding that infectious etiology was more common in stage 3 AKI (54.3%) aligns with study by Mehta et al., and highlights the importance of prompt recognition and treatment of infections to prevent kidney damage.[14] Interestingly, dehydration-related AKI was more frequently observed in stage 1 cases (35.6%), with a notable decline in stage 2 (16.4%) and stage 3 (10.9%) AKI. This pattern suggests that dehydration primarily contributes to prerenal AKI, which is typically reversible with early hydration. Previous studies, such as those by Vikrant et al., and Yang et al., have also documented the higher incidence of dehydration-related AKI in less stage 3 forms of kidney injury, indicating that prerenal causes often present with stage 1er symptoms but can progress if left untreated or if underlying conditions worsen.[15,16]

The progression and outcomes of AKI varied significantly by severity. Patients with stage 3 AKI had the highest rates of dialysis (67.4%), ICU admission (89.1%), progression to CKD (30.4%), and mortality (34.8%). These findings are comparable to those reported by See et al., where stage 3 AKI was associated with a markedly increased risk of poor outcomes, including mortality and CKD progression.[17] The stark contrast in recovery without dialysis, observed in 84.4% of stage 1 AKI cases but only 26.1% of stage 3 cases, highlights the importance of early diagnosis and intervention.

The requirement for dialysis across all severities was 33.3%, comparable to 25–40% reported in similar studies globally.[18] ICU admissions were significantly higher in stage 2 and stage 3 AKI (30.9% and 89.1%, respectively), mirroring data from the study by Andonovic et al., that show AKI is a major contributor to critical care morbidity.[19] Mortality rates in stage 3 AKI (34.8%) are consistent with findings from regions with limited access to advanced renal replacement therapies, highlighting the unmet need for timely management.[19]

Our findings also show that drug-induced AKI accounted for a significant proportion of stage 2 AKI cases (23.6%) but was less prominent in stage 3 cases (15.2%). Nonsteroidal anti-inflammatory drugs (NSAIDs) and antibiotics were the most commonly implicated drugs, consistent with global findings on drug-induced nephropathy.[20] A study by Clifford et al., highlighted that NSAIDs and certain antibiotics, such as aminoglycosides and vancomycin, are major contributors to AKI in hospitalized patients, often exacerbating existing kidney dysfunction.[21] However, while drug-induced nephropathy remains an important cause of AKI, its relative contribution to stage 3 cases in our study was lower compared to infections, which may be due to early recognition and discontinuation of the offending drugs in most cases.

In terms of clinical outcomes, we observed that serum creatinine levels >4 mg/dL and BUN >60 mg/dL were strong predictors of stage 3 AKI, as has been reported in study by Wange et al.[22] For instance, an observational study by Brookes et al., found that elevated creatinine and BUN levels were significant predictors of poor outcomes, including the need for dialysis and progression to chronic kidney disease (CKD).[23] Our study also corroborates previous findings by van der Slikke et al., and Seki et al., which showed that patients with higher creatinine and BUN levels had worse outcomes and higher mortality rates.[24,25] We found that serum creatinine >4 mg/dL was associated with a 4.2-fold increased risk of stage 3 AKI (AOR 4.2, 95% CI 2.1–8.6, p<0.001), supporting its use as a critical marker for AKI severity.

Hypotension was another key predictor of stage 3 AKI in our study, with an odds ratio of 2.8 (95% CI 1.5–5.2, p=0.002). The relationship between hypotension and kidney injury is well-established, as low blood pressure can impair renal perfusion and exacerbate kidney damage. Similar findings were reported by Izawa et al., who found that hypotension significantly increased the risk of progression to stage 3 AKI and dialysis dependency.[26] Additionally, we identified oliguria as a strong predictor of stage 3 AKI, with an AOR of 5.6 (95% CI 3.1–10.2, p<0.001). Oliguria has long been recognized as a key marker for worsening renal function and is associated with a poor prognosis in AKI, as it indicates a substantial decrease in glomerular filtration rate (GFR). Our results align with studies by Vaara et al., which reported that oliguria was one of the most reliable clinical predictors of poor outcomes, including the need for renal replacement therapy.[27]

Advanced age (>60 years) was also identified as a significant predictor of stage 3 AKI in our study, with a 1.6-fold increased risk of stage 3 AKI (AOR 1.6, 95% CI 1.0–2.9, p=0.045). Older adults are more vulnerable to stage 3 kidney injury due to age-related decline in renal function, comorbidities such as hypertension and diabetes, and polypharmacy. Studies by Pistolesi et al., and Kim et al., have consistently shown that older age is associated with an increased risk of developing stage 3 AKI and adverse outcomes such as dialysis dependence and mortality.[28,29]

Limitations

While this study provides valuable insights into the etiological and prognostic factors associated with AKI, there are several limitations to consider. First, the study was conducted at a single tertiary care center, which may limit the generalizability of the findings to other settings, especially in rural or low-resource areas. Additionally, the observational nature of the study means that causal relationships cannot be definitively established. There may also be recall bias in the reporting of comorbidities and exposure to environmental factors. The lack of long-term follow-up data limits our ability to assess the long-term outcomes of patients with AKI. Finally, while we considered a range of clinical and biochemical variables, other unmeasured factors such as genetic predisposition and environmental exposures may have influenced the outcomes of AKI.

CONCLUSION

In conclusion, this study highlights the significant role of infections, dehydration, and hypotension in the etiology and progression of acute kidney injury. Early recognition of risk factors, including elevated serum creatinine, oliguria, and hypotension, is critical for identifying patients at high risk of stage 3 AKI and preventing poor outcomes. Future studies with larger sample sizes, multi-center designs, and longer follow-up periods are needed to further validate these findings and explore additional biomarkers and interventions that could improve outcomes in AKI patients.

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