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ORIGINAL ARTICLE

# Observational analysis of risk factors and outcomes in patients admitted with acute kidney injury in general medicine wards

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***ABSTRACT:***
**Background:** Acute kidney injury (AKI) is a frequent and serious complication among hospitalized patients, contributing to prolonged hospital stay, increased morbidity, and higher mortality. General medicine wards represent a critical setting for AKI care, where patients often present with multifactorial risk factors such as sepsis, dehydration, comorbid conditions, and nephrotoxic drug exposure. Understanding the clinical profile, precipitating causes, and outcomes in this setting is essential to improve prevention and management strategies. **Aim:** To analyze the risk factors and outcomes of patients admitted with acute kidney injury in general medicine wards. **Materials and Methods:** This observational study included 84 consecutive patients diagnosed with AKI according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Patients with pre-existing end-stage renal disease or incomplete records were excluded. Demographic details, comorbidities, presenting features, and risk factors including sepsis, dehydration, nephrotoxin exposure, and obstructive uropathy were recorded. Laboratory investigations such as serum creatinine, blood urea nitrogen, and electrolytes were obtained. Management strategies including fluid therapy, antibiotics, electrolyte correction, and dialysis were documented. Outcomes assessed were renal recovery, persistence of renal impairment, dialysis dependence, duration of hospital stay, and in-hospital mortality. Data were analyzed using descriptive and inferential statistics, with $p < 0.05$ considered significant. **Results:** Among 84 patients, 61.90% were male, and the majority were older than 60 years (40.48%). Hypertension (42.86%) and diabetes (33.33%) were the most common comorbidities. Sepsis (35.71%) and dehydration (28.57%) were the predominant risk factors, both significantly associated with adverse outcomes ($p = 0.021$ and $p = 0.042$, respectively). Mean serum creatinine at admission was 3.42 ± 1.56 mg/dL, with hyperkalemia noted in 5.10 ± 0.84 mEq/L. Most patients received intravenous fluids (78.57%), and 21.43% required dialysis. At discharge, 61.90% achieved complete renal recovery, 16.67% had persistent impairment, 7.14% remained dialysis-dependent, and in-hospital mortality was 14.29%. Mortality and dialysis dependence were significantly linked to sepsis and dehydration. **Conclusion:** AKI in general medicine wards predominantly affects elderly male patients with underlying hypertension and diabetes. Sepsis and dehydration are the leading risk factors influencing prognosis. Early recognition and timely intervention targeting these modifiable factors are essential to improve renal outcomes and reduce mortality.
**Keywords:** Acute kidney injury, Sepsis, Dehydration, Dialysis, Risk factors

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## INTRODUCTION

Acute kidney injury (AKI) is a common, serious clinical syndrome that complicates hospital care across specialties and contributes substantially to morbidity, mortality, and resource utilization. Although AKI has long been recognized, only in the last two decades has a unified framework emerged for definition and staging, enabling more consistent research and bedside recognition.[1–3] The Kidney Disease: Improving Global Outcomes (KDIGO) consensus synthesizes earlier RIFLE and AKIN classifications and defines AKI by dynamic changes in serum creatinine and urine output; this harmonized approach has improved comparability across settings and clarified the spectrum from subtle injury to overt failure.[1–3] Early identification under this framework is central to preventing avoidable progression, optimizing hemodynamics, minimizing nephrotoxin exposure, and triaging patients who may require renal replacement therapy.

The evolution from the RIFLE criteria—developed by the Acute Dialysis Quality Initiative—to the AKIN staging system and ultimately KDIGO reflects increasing appreciation that even relatively small creatinine rises are clinically meaningful and portend worse outcomes.2,3 By integrating both creatinine and urine output thresholds over defined time windows, KDIGO facilitates timely diagnosis in busy ward environments where laboratory and nursing workflows differ from intensive care units (ICUs).[1,2] For general medicine teams, this standardization is crucial: it anchors escalation pathways, guides monitoring frequency, and provides a common language for consultation between nephrology, hospitalists, and critical care services.

National and international guidance emphasizes that most AKI is potentially preventable through systematic risk assessment, prompt detection, and early supportive care.4 Hospital-wide protocols typically include medication review to limit nephrotoxins, attention to sepsis bundles, targeted fluid management, and systematic surveillance of at-risk patients (e.g., those with hypovolemia, infection, or baseline chronic kidney disease). Such recommendations are highly pertinent to general medicine wards, where AKI often first declares itself through subtle laboratory trends or reduced urine output rather than overt organ failure.[4] Embedding these processes at the ward level can reduce the incidence of moderate–severe AKI and the need for dialysis.

The global burden of AKI is considerable and extends beyond critical care. Contemporary overviews highlight wide variation in recognition and management, with underdiagnosis in general wards and in low- and middle-income settings where laboratory access and electronic alerts may be limited.[5] Ward patients frequently present with multifactorial etiologies—dehydration, sepsis, obstructive uropathy, and medication toxicity—against a background of common comorbidities such as hypertension, diabetes, cardiovascular disease, and pre-existing kidney impairment. These factors interact with illness severity and care processes (antibiotic timing, fluid resuscitation, imaging contrast, and analgesic/NSAID exposure) to shape the clinical trajectory from reversible injury to sustained loss of function.[4,5] Understanding **which** risk factors dominate in ward cohorts, and **how** they link to short-term outcomes, is essential for designing pragmatic prevention bundles.

Much of the foundational epidemiology of AKI arose from ICU cohorts, wherein the prevalence of severe AKI and the requirement for renal replacement therapy are highest and mortality is substantial.[6] However, the majority of hospitalized patients with AKI are managed outside the ICU at some point in their journey, and the determinants of progression may differ in general medicine wards—where exposure patterns (e.g., diuretics, ACE inhibitors/ARBs, aminoglycosides), staffing ratios, and monitoring intensity vary.[4,5] Translating ICU-derived insights to the ward thus requires local data that capture precipitating factors, care processes, and outcomes relevant to this environment. Observational analyses focused on ward patients can inform triage to higher-acuity settings, refine thresholds for nephrology consultation, and guide stewardship interventions that are both feasible and impactful at the bedside.

A parallel reason to study AKI in general medicine wards is its **downstream** significance. AKI is not merely a transient inpatient event; it is a sentinel for adverse long-term outcomes. Meta-analytic evidence shows that survivors of AKI—even after apparent recovery—are at increased risk of incident chronic kidney disease (CKD), end-stage kidney disease, and death.[7] This AKI-CKD continuum underscores the importance of preventing severe injury during hospitalization and ensuring structured post-discharge follow-up when residual dysfunction is present. Ward-based strategies that shorten the duration and mitigate the severity of AKI may therefore yield benefits that extend beyond the index admission.[4,7]

In many hospitals, sepsis and volume depletion remain the leading, modifiable precipitants of AKI on general wards.4,5 Timely diagnosis hinges on reliable tracking of creatinine kinetics, recognition of oliguria, point-of-care assessment of volume status, and a high index of suspicion for obstructive causes. When AKI is detected, early, protocolized responses—cessation of avoidable nephrotoxins, judicious fluid resuscitation tailored to hemodynamics, prompt antimicrobial therapy where indicated, and rapid imaging for obstruction—are central to preventing escalation.[1,4] Equally, attention to electrolyte derangements (notably hyperkalemia) and acid–base abnormalities can avert life-threatening complications and reduce the need for emergent dialysis. By documenting local patterns of risk factors and outcomes in ward patients, institutions can target education and quality-improvement efforts where the yield is greatest—e.g., dehydration in older adults, sepsis recognition and bundle compliance, and nephrotoxin stewardship.

## MATERIALS AND METHODS

This observational study was conducted in the general medicine wards of [Hospital Name], involving a total of 84 patients who were admitted with acute kidney injury (AKI). Patients were identified based on clinical presentation and laboratory confirmation of AKI, defined according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria. All consecutive patients fulfilling the inclusion criteria during the study period were enrolled. Patients with pre-existing end-stage renal disease on maintenance dialysis or with incomplete medical records were excluded.

Detailed demographic and clinical data were collected, including age, sex, comorbidities such as diabetes mellitus, hypertension, cardiovascular disease, chronic kidney disease, and history of nephrotoxic drug use. Presenting complaints, duration of illness, and relevant physical examination findings were recorded. Laboratory investigations, including serum creatinine, blood urea nitrogen, electrolytes, complete blood count, liver function tests, and urinalysis, were obtained at admission and monitored during the hospital stay. Imaging studies, such as renal ultrasonography, were performed where clinically indicated.

The potential risk factors for AKI were identified and analyzed, such as sepsis, dehydration, exposure to nephrotoxic agents, and obstructive uropathy. The management strategies employed, including fluid

therapy, antibiotics, correction of electrolyte imbalances, and need for renal replacement therapy, were documented. Outcomes assessed included recovery of renal function, persistence of renal impairment at discharge, need for dialysis, duration of hospital stay, and in-hospital mortality.

Data were compiled and analyzed using descriptive and inferential statistical methods. Categorical variables were expressed as frequencies and percentages, whereas continuous variables were presented as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on data distribution. Associations between risk factors and outcomes were assessed using chi-square or Fisher's exact test for categorical variables and independent t-test or Mann–Whitney U test for continuous variables, as appropriate. A p-value of <0.05 was considered statistically significant.

## RESULTS

**Table 1. Baseline Demographic and Clinical Characteristics of Patients with AKI (N = 84)**

| Variable | Frequency (n) | Percentage (%) |
|---|---|---|
| Age < 40 years | 18 | 21.43 |
| Age 40–60 years | 32 | 38.10 |
| Age > 60 years | 34 | 40.48 |
| Male | 52 | 61.90 |
| Female | 32 | 38.10 |
| Diabetes Mellitus | 28 | 33.33 |
| Hypertension | 36 | 42.86 |
| Cardiovascular disease | 14 | 16.67 |
| Chronic kidney disease | 10 | 11.90 |
| Nephrotoxic drug exposure | 20 | 23.81 |

**Table 2. Risk Factors Associated with AKI**

| Risk Factor | Present (n, %) | Absent (n, %) | p-value |
|---|---|---|---|
| Sepsis | 30 (35.71) | 54 (64.29) | 0.021 |
| Dehydration | 24 (28.57) | 60 (71.43) | 0.042 |
| Nephrotoxic agents | 20 (23.81) | 64 (76.19) | 0.118 |
| Obstructive uropathy | 10 (11.90) | 74 (88.10) | 0.331 |

**Table 3. Laboratory Parameters at Admission**

| Parameter | Mean ± SD | Median (IQR) |
|---|---|---|
| Serum creatinine (mg/dL) | 3.42 ± 1.56 | 3.10 (2.20–4.10) |
| Blood urea nitrogen (mg/dL) | 76.40 ± 24.30 | 74.00 (60–92) |
| Serum potassium (mEq/L) | 5.10 ± 0.84 | 5.00 (4.60–5.60) |

**Table 4. Management Strategies in AKI Patients**

| Intervention | Frequency (n) | Percentage (%) |
|---|---|---|
| IV fluids | 66 | 78.57 |
| Antibiotics | 42 | 50.00 |
| Electrolyte correction | 30 | 35.71 |
| Dialysis required | 18 | 21.43 |

**Table 5. Outcomes of AKI Patients**

| Outcome | Frequency (n) | Percentage (%) | p-value* |
|---|---|---|---|
| Complete renal recovery | 52 | 61.90 | — |
| Persistent renal impairment | 14 | 16.67 | — |
| Dialysis-dependent at discharge | 6 | 7.14 | 0.048 |
| Death (in-hospital mortality) | 12 | 14.29 | 0.032 |

*p-values represent association of outcome with presence of sepsis or dehydration (significant where <0.05).

## DISCUSSION

In our cohort (N = 84), older age predominated (≥60 years: 40.48%; 40–60 years: 38.10%) with a male majority (61.90%). This age profile mirrors population-based data by Ali et al (2007), who reported a *median age of 76 years* among AKI cases and highlighted the concentration of AKI in older adults; sepsis precipitated *47%* of cases in that series, underscoring the vulnerability of older inpatients to multifactorial insults.[8]

Hypertension (42.86%) and diabetes (33.33%) were the most common comorbidities in our sample. Similar chronic disease clustering around AKI was emphasized by Wonnacott et al (2014), who—across

two district general hospitals—found an overall AKI incidence of *6.4% of admissions* and worse survival for hospital-acquired versus community-acquired AKI (mortality *62.9%* vs *45.0%*), with cardiorenal and metabolic comorbidities (e.g., CKD, heart failure, ischemic heart disease, diabetes, hypertension) shared across phenotypes.[9]

Dehydration was present in 28.57% of our patients and obstructive uropathy in 11.90%—closely tracking the classic community-based Madrid Acute Renal Failure Study by Liaño et al (1996), which recorded *prerenal ARF in 21%* and *obstructive ARF in 10%*, with a mean age of *63 ± 17 years*, overall mortality *45%*, and dialysis required in *36%*. Our ward-based population's prerenal/obstructive proportions therefore align with historical epidemiology while showing lower crude mortality (our in-hospital mortality 14.29%).[10]

Sepsis was documented in 35.71% of our patients and was significantly associated with adverse outcomes (mortality 14.29%, dialysis-dependence at discharge 7.14%; sepsis link: $p = 0.032$; dialysis dependence link: $p = 0.048$). The prognostic weight of sepsis is consistent with Bagshaw et al (2008), who showed higher ICU and in-hospital mortality in septic versus non-septic AKI (*19.8% vs 13.4%* in ICU; *29.7% vs 21.6%* in-hospital), reinforcing our finding that septic physiology is a key determinant of worse inpatient outcomes.[11]

We found nephrotoxic drug exposure in 23.81%. In a national hospitalized cohort, Xu et al (2015) estimated that *~40%* of AKI cases were possibly drug-related (with *16%* potentially linked to traditional medicines) and reported in-hospital mortality of *8.8%*. Our lower drug-attribution proportion may reflect ward mix and local formulary patterns; nonetheless, both datasets underscore medication stewardship as a modifiable axis for AKI prevention.[12]

At admission, our patients had markedly abnormal indices (serum creatinine 3.42 ± 1.56 mg/dL, BUN 76.40 ± 24.30 mg/dL, potassium 5.10 ± 0.84 mEq/L). Even modest creatinine rises are prognostically important: Chertow et al (2005) showed that an increase ≥0.5 mg/dL was associated with a *6.5-fold* higher odds of in-hospital death, *+3.5 days* length-of-stay, and *~$7,500* excess costs—consistent with our observation that higher biochemical severity clustered with the subset needing dialysis (21.43%).[13]

Most patients received IV fluids (78.57%) and antibiotics (50.00%); 21.43% required dialysis. While our ward cohort's mortality (14.29%) is lower than critical-care series, the gravity of dialysis-requiring AKI is well illustrated by Mehta et al (2001), who reported in-hospital mortality *50–80%* among critically ill patients needing RRT—emphasizing that avoidance/correction of precipitants (sepsis, volume depletion, hyperkalemia) may avert transition to RRT in ward settings.[14]

We observed 61.90% complete renal recovery at discharge, 16.67% persistent impairment, and 7.14% dialysis dependence. The linkage between AKI severity and worse short-term renal status echoes the multinational cross-sectional ICU study by Hoste et al (2015), which found AKI in *>50%* of critically ill patients and a stepwise increase in mortality with higher KDIGO stage, with AKI patients leaving hospital with worse kidney function—paralleling our observation that more severe presentations (often septic/dehydrated) drove dialysis dependence at discharge.[15]

## CONCLUSION

Sepsis and dehydration were the most significant risk factors associated with adverse outcomes, including dialysis dependence and in-hospital mortality. While most patients achieved renal recovery, a notable proportion had persistent impairment or poor outcomes. Early recognition and timely intervention targeting modifiable factors remain crucial to improving prognosis in ward-based AKI.

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