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Frequency and Outcomes of Acute Kidney Injury Among Patients Admitted to the Medical Ward of a Tertiary Care Hospital

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ABSTRACT

Objective: To determine the frequency and outcomes of acute kidney injury (AKI) among patients admitted to the medical ward of a tertiary care hospital, and to identify factors associated with in-hospital mortality.

Methods: This descriptive cross-sectional study was conducted in the Medical Ward of Akbar Niazi Teaching Hospital, Islamabad, from January 2026 to April 2026. Consecutive patients aged 18–80 years admitted to the medical ward with a hospital stay of more than 48 hours were screened for AKI, which was diagnosed and staged using KDIGO criteria. Data were analysed using SPSS version 26. Associations with in-hospital mortality were assessed using the Chi-square or Fisher's exact test, and odds ratios (OR) with 95% confidence intervals (CI) were calculated, with $p<0.05$ considered significant.

Results: Of 1470 eligible admissions, 234 patients developed AKI, giving a frequency of 15.9%. The mean age was 60.4 ± 15.2 years, and 148 (63.2%) were male. Hypertension (122, 52.1%) and diabetes (94, 40.2%) were the most common comorbidities. Stage 3 AKI was present in 126 (53.8%) patients. The mean hospital stay was 6.2 ± 3.4 days. No patient required dialysis. In-hospital mortality was 34 (14.5%). Sepsis (OR 4.91, 95% CI 2.12–11.39), septic shock (OR 9.95, 95% CI 3.20–30.95), vasopressor use (OR 15.08, 95% CI 4.24–53.58), and Stage 3 AKI (OR 4.86, 95% CI 1.93–12.24) were significantly associated with mortality (all $p<0.001$). Hypertension and diabetes were not associated with mortality.

Conclusion: AKI affected about one in six medical ward admissions and was often severe. Mortality was associated mainly with sepsis-related factors, not chronic comorbidities. Early detection of AKI and prompt sepsis management are essential to improve outcomes.

Keywords: Acute kidney injury; KDIGO; Sepsis; In-hospital mortality; Tertiary care hospital.

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Introduction

Acute kidney injury (AKI) is a prevalent and serious complication that can occur during hospitalization.[1] Large cohort studies suggest that AKI is associated with increased mortality and morbidity.[1] A multi-center study conducted in the United States showed that approximately 14% of hospitalized patients developed AKI.[1]

A global meta-analysis showed that 21.6% of adult inpatients over the world have this condition.[2] A large population-based cohort study was conducted in Olmsted County, Minnesota. This study analyzed data of 52,000 hospitalized patients. Researchers reported a higher incidence of AKI among general ward patients than among ICU patients (287 vs 186 per 100,000 person-years), showing that a large part of the AKI burden lies outside critical care.[3] In Hong Kong, a cohort of more

than 125,000 admissions, the prevalence of AKI was 9.1% in general wards.[4]

The burden of AKI is greater in low- and middle-income countries (LMICs). A single-center study from Pakistan showed an AKI prevalence of 34.7% among hospitalized patients with decompensated liver cirrhosis.[5] The identified predictors were diabetes (60%, p=0.002), nephrotoxic medication exposure (43%, p=0.01), and advanced Child–Pugh class.[5] Another study from Karachi, among patients with organophosphate poisoning, revealed an AKI frequency of 22.3%.[6]

Similar outcomes from neighboring LMICs are reported. A nine-center registry in India of 3,711 patients with community-acquired AKI found sepsis (34.7%) and tropical infections (9.8%) as the main causes.[7] The study also reported an in-hospital mortality rate of 10.8%.[7] In Bangladesh, a prospective ward-based study was done in Dhaka.[8] The results showed that hospital-acquired AKI was mostly attributable to sepsis (44%) and fluid loss (42%). This led to an 18% inpatient mortality rate and 22% of survivors progressing to chronic kidney disease after three months.[8] A meta-analysis of 22 studies including 4,069 patients from Iran revealed that 24% of hospitalized COVID-19 patients experienced AKI.[9] The rate in normal wards (23%) was close to that in ICUs (30%). This shows that AKI is a serious problem even when people are not in critical care.[9] Reported mortality among patients with AKI ranges from as low as 1% in mild cases to as high as 66% in critically ill groups.[10] Besides that, the long-term consequences are also concerning.[10] AKI is associated with an approximately fivefold increase in one-year mortality compared to individuals without AKI.[1] Moreover, AKI is frequently under-recognized in routine ward care, which delays simple corrective measures such as optimization of volume status and withdrawal of nephrotoxic drugs. A multicenter study revealed that nearly 68% of Kidney Disease Improving Global Outcomes (KDIGO) defined AKI episodes were not diagnosed in general medical wards.[11]

The pathophysiology of AKI in hospitalized patients is usually multifactorial. In sepsis, kidney injury results not only from reduced renal perfusion but also from systemic inflammation, microvascular dysfunction and tubular epithelial cell injury, which may occur even when blood pressure is maintained.[12] Septic shock and the need for vasopressors represent the most severe end of this spectrum, reflecting refractory circulatory failure and multi-organ dysfunction, and are therefore expected to be closely linked to mortality.[12] Chronic conditions such as diabetes mellitus and hypertension reduce renal reserve through glomerular hyperfiltration, nephrosclerosis and impaired renal autoregulation, making the kidney more vulnerable to haemodynamic and toxic insults.[10] Nephrotoxic drugs, such as aminoglycosides and iodinated contrast, cause direct tubular injury, while drugs that alter intraglomerular haemodynamics, such as ACE inhibitors and angiotensin receptor blockers, reduce glomerular filtration in vulnerable patients.[10] Chronic comorbidities therefore mainly predispose to the development of AKI, whereas the severity of acute illness is more likely to determine its outcome.

The present study aimed to determine the frequency of AKI among patients admitted to the medical ward of a tertiary care hospital in Pakistan, the frequency of associated risk factors such as comorbidities, sepsis and nephrotoxic exposures, and its outcomes, including length of hospital stay, need for dialysis and in-hospital mortality. Most available Pakistani data on AKI come from intensive care units or from selected groups such as patients with cirrhosis, poisoning or stroke, and ward-based data from general medical admissions using KDIGO criteria are scarce. This work therefore aims to generate actionable evidence for a setting where systematic data are scarce.

Methodology

This descriptive cross-sectional study was conducted in the Medical Ward of Akbar Niazi Teaching Hospital, Islamabad, from January 2026 to April 2026. The ethical exemption for the study was obtained from the Ethics Review Committee of Islamabad Medical and Dental College, Islamabad. Ref no 190/IMDC/IREB-2025 dated April 2025

All patients admitted to the medical ward during the study period were screened for eligibility. Patients aged 18 to 80 years, admitted to the medical ward with a hospital stay of more than 48 hours, were included in the study through non-probability consecutive sampling and were monitored for the development of AKI during their hospital stay. The minimum stay of 48 hours was required to allow serial serum creatinine measurement as per KDIGO criteria. The study population therefore represented the general adult medical case-mix of the ward rather than a specific disease group. Patients with end-stage renal disease on maintenance dialysis, kidney transplant recipients, those with an admission serum

creatinine greater than 5.0 mg/dL (taken as a marker of pre-existing advanced chronic kidney disease, in whom an acute rise cannot be reliably attributed to AKI), and patients admitted to the intensive care unit were excluded.

The sample size was calculated using the OpenEpi sample size calculator, taking an expected AKI frequency of 21.6% among hospitalized adults,[2] a 95% confidence level and an absolute precision of 5%, which gave a minimum required sample of 261 patients. All 1470 eligible patients admitted during the study period were screened, exceeding this requirement.

AKI was diagnosed according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours, an increase in serum creatinine to ≥1.5 times baseline within the prior 7 days, or a urine output of <0.5 mL/kg/hour for more than 6 hours. AKI was staged as Stage 1 (serum creatinine 1.5–1.9 times baseline or an increase of ≥0.3 mg/dL, or urine output <0.5 mL/kg/hour for 6–12 hours), Stage 2 (serum creatinine 2.0–2.9 times baseline, or urine output <0.5 mL/kg/hour for ≥12 hours) and Stage 3 (serum creatinine ≥3.0 times baseline, serum creatinine ≥4.0 mg/dL, initiation of renal replacement therapy, or urine output <0.3 mL/kg/hour for ≥24 hours or anuria for ≥12 hours). Baseline serum creatinine was taken as the most recent value available before admission; where this was not available, the admission serum creatinine was used as the reference value. Data were collected using a structured proforma after obtaining informed consent. Information regarding demographic characteristics, comorbidities, clinical findings, laboratory investigations, medication exposure, and hospital outcomes was obtained from patient files, nursing records, laboratory reports, and the hospital electronic record system.

Hypertension was defined as a documented diagnosis or use of antihypertensive drugs, and diabetes mellitus as a documented diagnosis, HbA1c ≥6.5%, or use of insulin or oral hypoglycaemic agents. Sepsis was defined according to the Sepsis-3 criteria as suspected or proven infection with a SOFA score of ≥2; where a full SOFA score was not available, a qSOFA score of ≥2 with infection was used. Nephrotoxic exposure was defined as exposure to at least one known nephrotoxic agent (e.g., aminoglycosides, vancomycin, NSAIDs or iodinated contrast) within 72 hours before the onset of AKI; use of ACE inhibitors or angiotensin receptor blockers was recorded separately.

The primary outcome was the frequency of AKI among patients admitted to the medical ward. Secondary outcomes included the frequency of risk factors, AKI severity, dialysis requirement, length of hospital stay, and in-hospital mortality.

Data were entered and analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Associations between clinical variables and in-hospital mortality were assessed using the Chi-square test or Fisher's Exact test where appropriate, and odds ratios (OR) with 95% confidence intervals (CI) were calculated. Because of the small number of deaths, multivariable analysis was not performed, and the associations reported are unadjusted. A p-value of less than 0.05 was considered statistically significant.

Results

During the study period from January 2026 to April 2026, a total of 1470 eligible patients were admitted to the medical ward and screened. Among them, 234 patients developed AKI, resulting in a frequency of 15.9%. The mean age of the patients was 60.4 ± 15.2 years, and males constituted the majority of the study population, 148 (63.2%). Hypertension, 122 (52.1%), and diabetes mellitus, 94 (40.2%), were the most frequently observed comorbidities. The mean hospital stay was 6.2 ± 3.4 days. Detailed baseline characteristics and associated clinical factors are presented in Table I.

Table 1: Baseline Characteristics and Clinical Profile of AKI Patients .(n = 234)

Variable	Frequency / Value
Mean age (years)	60.4 ± 15.2
Male gender	148 (63.2%)
Female gender	86 (36.8%)
Mean hospital stay (days)	6.2 ± 3.4
Hypertension	122 (52.1%)
Diabetes Mellitus	94 (40.2%)
Ischemic Heart Disease	16 (6.8%)
Sepsis	32 (13.7%)
Septic Shock	14 (6.0%)
Vasopressor Use	12 (5.1%)
ACE inhibitors/ARBs Use	18 (7.7%)
Aminoglycoside Exposure	6 (2.6%)
IV Contrast Exposure	2 (0.9%)

More than half of the patients had severe renal injury, with Stage 3 AKI observed in 126 (53.8%) patients. No patient required dialysis. Overall, in-hospital mortality was 34 (14.5%). The severity distribution of AKI and patient outcomes are summarized in Table II.

Table II: Severity and Outcomes of Acute Kidney Injury	
Variable	**Frequency (%)**
Stage 1 AKI	42 (17.9%)
Stage 2 AKI	66 (28.2%)
Stage 3 AKI	126 (53.8%)
Requirement for Dialysis	0 (0%)
Discharged Alive	200 (85.5%)
In-hospital Mortality	34 (14.5%)

Categorical variables were compared using the Chi-square test or Fisher's Exact test where appropriate. Fisher's Exact test was applied for variables with expected cell counts less than 5, including sepsis, septic shock and vasopressor requirement.

Sepsis, septic shock, vasopressor requirement, and Stage 3 AKI demonstrated statistically significant associations with in-hospital mortality (all $p<0.001$). The strongest associations were observed for vasopressor use (OR 15.08, 95% CI 4.24–53.58) and septic shock (OR 9.95, 95% CI 3.20–30.95), followed by sepsis (OR 4.91, 95% CI 2.12–11.39) and Stage 3 AKI (OR 4.86, 95% CI 1.93–12.24); the wide confidence intervals reflect the small number of deaths. However, hypertension and diabetes mellitus were not significantly associated with mortality in the present study. A comparison of factors associated with mortality is shown in Table III.

OR, odds ratio for in-hospital mortality; CI, confidence interval. Fisher's Exact test was used for sepsis, septic shock and vasopressor use; Pearson Chi-square test was used for Stage 3 AKI, diabetes mellitus and hypertension.

Discussion

Our study showed that AKI occurred in 15.9% of medical ward admissions. This showed that almost one in six patients admitted to medical ward developed AKI during their stay. The frequency reported in our study lies within the range reported worldwide. Different hospital cohorts have reported KDIGO-defined AKI in 17% to 31% of inpatients.[13] A study from Italy consisting of more than 87,000 patients reported AKI frequency of 20.6%.[14] In comparison our figure is slightly lower. The most likely reason for this is our study design. We excluded ICU patients, dialysis-dependent patients, and those with a creatinine above 5 mg/dL. We measured AKI on the general ward, not in critical care.

Our results are also comparable to Pakistani data. A study of patients admitted with acute stroke in Peshawar reported a frequency of 15.4% AKI.[15] However, some other Pakistani studies reported higher figures. A single-center study among patients with decompensated cirrhosis reported an AKI frequency of 34.7%.[5] Another study from Karachi, among patients with organophosphate poisoning, reported a frequency of 22.3%.[6] ICU-based studies report even higher numbers. An ICU study from Islamabad found AKI in 68.6% of patients, and severity was significantly associated with mortality.[16] These differences are mainly driven by the setting. Ward studies report lower frequencies than ICU studies. The severity of illness in the sampled population matters more than the disease itself.

The demographic profile of our patients was typical. The mean age of the patients was 60 years. Most patients were male (63%). This pattern matches local and international reports.[16,17] Hypertension (52%) and diabetes (40%) were reported as most common comorbidities. This is expected, as both conditions predispose the kidney to injury by reducing renal reserve.

The most striking finding in our study was the severity of AKI. Our results reported that more than half of the patients (53.8%) had Stage 3 disease. This is unusual in previous literature. In well-monitored cohorts, mild Stage 1 disease is usually the most common severity reported. We interpret this finding with caution. It likely reflects late presentation and poor early detection. Studies show that up to 68% of AKI episodes go unrecognized in routine care.[11] In our setting, patients often present late. The cases that are detected are therefore the more advanced ones. The severity skew may reflect access and detection, not biology alone.[11] In addition, our inclusion criterion of a hospital stay longer than 48 hours may have preferentially captured patients with more advanced or slower-resolving AKI, as patients with mild, rapidly reversible AKI are more likely to be discharged early. Despite the high proportion of Stage 3 AKI, no patient required dialysis; this is likely explained by the exclusion

Table III: Factors Associated with In-Hospital Mortality.				
Variable	**Survivors (n=200)**	**Non-Survivors (n=34)**	**OR (95% CI)**	**p-value**
Sepsis	20 (10.0%)	12 (35.3%)	4.91 (2.12–11.39)	<0.001
Septic Shock	6 (3.0%)	8 (23.5%)	9.95 (3.20–30.95)	<0.001
Vasopressor Use	4 (2.0%)	8 (23.5%)	15.08 (4.24–53.58)	<0.001
Stage 3 AKI	98 (49.0%)	28 (82.4%)	4.86 (1.93–12.24)	<0.001
Diabetes Mellitus	78 (39.0%)	16 (47.1%)	1.39 (0.67–2.89)	0.376
Hypertension	100 (50.0%)	22 (64.7%)	1.83 (0.86–3.90)	0.113

of ICU patients and of those with an admission creatinine above 5 mg/dL.

In-hospital death occurred in 14.5% of the AKI patients, and mortality was higher in patients with severe AKI, with Stage 3 disease carrying nearly fivefold higher odds of death (OR 4.86). A Brazilian study reported 25% mortality in patients with AKI.[17] Risk increased with AKI stage. This matches cohorts where Stage 3 disease carries a much higher mortality than Stage 1.[18] The main factor associated with death was sepsis. Sepsis, septic shock, and vasopressor use were all significantly associated with mortality, with the strongest associations for vasopressor use (OR 15.08) and septic shock (OR 9.95). This agrees with the wider literature.[19] A large study consisting of over 27,000 patients from five international medical databases reported that a sepsis-associated AKI carries a mortality above 25%.[19] In the literature, AKI in the setting of sepsis has been identified as an independent predictor of death.[20] However, septic shock and vasopressor use are overlapping manifestations of the same process, and our analysis was unadjusted; our findings should therefore be interpreted as associations rather than independent effects.

Our mortality figures are also consistent with recent Pakistani data. In a medical-ward study from Abbottabad, AKI developed in 38.7% of patients with sepsis, and mortality was 31.0% among those with AKI compared with 9.8% among those without AKI.[21] A nephrology-unit series from Rawalpindi identified sepsis as the commonest cause of AKI, with an in-hospital mortality of 7.3%.[22] Our overall mortality of 14.5% lies between these figures, which is consistent with a mixed medical population in which only 13.7% of patients had sepsis, while the much higher odds of death in our patients with sepsis, septic shock or vasopressor requirement mirror the excess mortality of sepsis-associated AKI reported in these studies.

In contrast, hypertension and diabetes were not associated with mortality. These are risk factors for developing AKI. They are not the main cause of death. Death appeared to be related to acute illness severity rather than chronic disease, although our study may have been underpowered to detect modest effects of these comorbidities.

This study adds ward-level data on KDIGO-defined AKI from a general medical population in Pakistan. Most published Pakistani data on AKI come from intensive care units or from selected groups such as patients with cirrhosis, poisoning, stroke or sepsis. Our findings show that AKI is common outside critical care, that a large proportion of cases are detected at an advanced stage, and that mortality in this setting is linked mainly to sepsis-related factors rather than chronic comorbidities. These data provide a local baseline for AKI surveillance and for quality-improvement measures such as routine creatinine monitoring in medical wards.

This study has several limitations. It was a single-center study using non-probability consecutive sampling, which limits generalizability. The exclusion of ICU patients, dialysis-dependent patients and those with a creatinine above 5 mg/dL restricts the findings to ward-based AKI and explains the absence of dialysis, while the requirement of a hospital stay longer than 48 hours may have favoured more advanced AKI. Baseline creatinine was not available for every patient, which may affect staging accuracy. The small number of deaths (n=34) precluded multivariable or stratified analysis, so the associations reported are unadjusted, and sparse exposures such as aminoglycosides and IV contrast could not be tested against mortality. Finally, follow-up ended at discharge, so renal recovery and progression to chronic kidney disease could not be assessed.

Conclusion

AKI occurred in about one in six medical ward admissions (15.9%), and more than half of the cases were Stage 3 at detection. In-hospital mortality was 14.5% and was associated with sepsis, septic shock, vasopressor use and Stage 3 AKI, but not with hypertension or diabetes. Routine creatinine monitoring for early detection of AKI and prompt management of sepsis may improve outcomes in medical wards.

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