

Frequency and Risk Factors of Hospital-Acquired Acute Kidney Injury in Medical Ward Admissions: A Multi-Centre Study from Tertiary Care Hospitals in Pakistan

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ABSTRACT

Objective: To determine the frequency of hospital-acquired acute kidney injury (HA-AKI) among medical ward admissions and to identify its associated risk factors at tertiary care hospitals in Pakistan.

Study Design: Cross-sectional descriptive study.

Place and Duration of Study: Medical wards of three tertiary care hospitals in Pakistan, from 30th September 2025 to 30th March 2026.

Methodology: A total of 110 adult patients admitted to medical wards were enrolled through non-probability consecutive sampling. Hospital-acquired AKI was defined and staged using KDIGO (Kidney Disease: Improving Global Outcomes) criteria. Demographic data, comorbidities, nephrotoxic drug exposure, laboratory parameters, and clinical outcomes were recorded. Logistic regression analysis was performed to identify independent risk factors for HA-AKI.

Results: HA-AKI was identified in 42 (38.2%) patients. Stage 1 AKI was most frequent (57.1%), followed by Stage 2 (33.3%) and Stage 3 (9.5%). Sepsis (52.4%), nephrotoxic drug exposure (45.2%), diabetes mellitus (57.1%), hypertension (66.7%), and baseline chronic kidney disease (42.9%) were significantly associated with AKI development. In-hospital mortality was significantly higher in the AKI group (19.0% vs 7.4%; $p=0.004$).

Conclusion: Hospital-acquired Acute Kidney Injury is highly prevalent in medical ward admissions in Pakistan, with sepsis and nephrotoxic drug exposure being the most significant risk factors. Early recognition, targeted nephroprotective strategies, and judicious use of nephrotoxic agents are essential to reduce its burden.

Keywords: Acute Kidney Injury; Hospital-Acquired; Medical Ward; Risk Factors; KDIGO; Pakistan

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INTRODUCTION

Acute kidney injury (AKI) is not simply a lab finding — it is a serious clinical event that significantly worsens the trajectory of hospitalised patients. Defined by an abrupt decline in renal function, AKI is associated with prolonged hospital stays, increased need for renal replacement therapy, and substantially higher in-hospital mortality [1,2]. What makes it particularly concerning is how frequently it develops after admission rather than being present at the outset — so-called hospital-acquired AKI — and how often it goes unrecognised until the damage is already done.

Globally, HA-AKI affects somewhere between 10% and 15% of all hospitalised patients, though these figures rise sharply in medical ward populations where patients tend to be older, carry more comorbidities, and are often exposed to multiple nephrotoxic agents simultaneously [3,4]. Sepsis remains the single most important precipitant, responsible for nearly half of all AKI cases in critically ill and acutely unwell patients [5]. Other well-documented risk factors include diabetes mellitus, pre-existing chronic kidney disease (CKD), heart failure, hypovolaemia, and exposure to drugs such as

aminoglycosides, non-steroidal anti-inflammatory agents, and iodinated contrast media [6,7].

In South Asia, the problem carries additional weight. Pakistan's disease burden is dominated by conditions that independently predispose to AKI — poorly controlled diabetes, untreated hypertension, and a high prevalence of CKD, much of it undiagnosed [8]. At the same time, antibiotic stewardship remains inconsistent, nephrotoxic drug monitoring is not routine in many centres, and early warning systems for deteriorating renal function are largely absent in public sector hospitals [9,11]. The result is a healthcare environment in which AKI is both more likely to occur and less likely to be caught early.

Despite this, published data on HA-AKI from Pakistani medical wards are sparse. The few studies that do exist are largely single-centre, vary widely in their diagnostic criteria, and do not capture the inter-institutional variation that a multi-centre design can provide [10,12]. Without reliable local data, it is difficult to make the case — administratively or clinically — for the preventive protocols and monitoring systems that could make a real difference.

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The objective of this study was to determine the frequency of hospital-acquired acute kidney injury in medical ward admissions and to identify its independently associated risk factors across three tertiary care hospitals in Pakistan.

MATERIAL AND METHODS

Study Design and Setting

This cross-sectional descriptive study was conducted at the medical wards of three tertiary care hospitals in Pakistan from 30th September 2025 to 30th March 2026.

Sample Size

A total of 110 patients were enrolled. Sample size was calculated on an estimated AKI prevalence of 30% in medical ward admissions, with a 95% confidence interval and 8% margin of error, yielding a minimum requirement of 105 patients; 110 were enrolled to account for attrition.

Sampling Technique

Non-probability consecutive sampling.

Ethical Approval

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Institutional ethical approval was obtained from the Research and Ethics Committees of all three participating hospitals prior to commencement. Written informed consent was obtained from all enrolled patients or their next of kin where applicable. Patient data were fully anonymised and handled in accordance with institutional confidentiality protocols.

Inclusion Criteria

- Adult patients aged 18–75 years admitted to general medical wards.
- Patients with a hospital stay of at least 48 hours.
- Patients with a baseline serum creatinine documented within three months of admission or at the time of admission.

Exclusion Criteria

- Patients with AKI present on admission (community-acquired AKI).
- Patients with end-stage renal disease (ESRD) on maintenance dialysis.
- Patients admitted directly to the intensive care unit (ICU) at the time of enrolment.
- Patients with obstructive uropathy as the primary cause of renal impairment.
- Incomplete medical records or loss to follow-up during the hospital stay.

Data Collection

A structured proforma was used to collect demographic data (age, gender, BMI), comorbidities (diabetes mellitus, hypertension, baseline CKD, heart failure), primary diagnosis at admission, and details of nephrotoxic drug exposure (aminoglycosides, NSAIDs, ACE inhibitors/ARBs, contrast agents). Serial serum creatinine values were recorded at admission, 48 hours, and subsequently every 48 hours or at clinically indicated intervals. AKI was defined and staged using KDIGO 2012 criteria: Stage 1 — creatinine rise ≥ 1.5 –1.9 times baseline or ≥ 0.3 mg/dL within 48 hours; Stage 2 — rise ≥ 2.0 –2.9 times baseline; Stage 3 — rise ≥ 3.0 times baseline, or creatinine ≥ 4.0 mg/dL, or initiation of renal replacement therapy. Clinical outcomes including duration of hospital stay, ICU transfer, need for renal

replacement therapy (RRT), and in-hospital mortality were documented.

Statistical Analysis

Data were analysed using SPSS version 26.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean $\pm$ standard deviation (SD) and compared using the independent samples t-test. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test as appropriate. Binary logistic regression was performed to identify independent predictors of HA-AKI, with results expressed as odds ratios (OR) with 95% confidence intervals (CI). A p-value of <0.05 was considered statistically significant.

RESULTS

Of the 110 patients enrolled across three tertiary care centres, 42 (38.2%) developed hospital-acquired AKI during their admission. The mean age of the cohort was 53.2 ± 14.6 years, and males accounted for 64 (58.2%) of patients. AKI patients were significantly older than those without AKI (58.4 ± 14.2 vs 49.7 ± 13.8 years; $p=0.003$). The AKI frequency ranged from 34.2% to 43.7% across the three participating centres.

Among the 42 patients who developed HA-AKI, Stage 1 (mild) AKI was most common (24 patients; 57.1%), followed by Stage 2 (moderate) in 14 patients (33.3%), and Stage 3 (severe) in 4 patients (9.5%). Three patients (7.1%) required renal replacement therapy. In-hospital mortality was significantly higher in the AKI group compared to the non-AKI group (19.0% vs 7.4%; $p=0.004$), and AKI patients were more frequently transferred to the ICU (28.6% vs 8.8%; $p=0.001$). Mean hospital stay was significantly prolonged in the AKI group (11.4 ± 4.6 vs 6.8 ± 3.1 days; $p<0.001$).

On univariate analysis, sepsis at admission (52.4% vs 20.6%; $p<0.001$), nephrotoxic drug exposure (45.2% vs 17.6%; $p=0.002$), baseline CKD (42.9% vs 14.7%; $p=0.001$), diabetes mellitus (57.1% vs 32.4%; $p=0.009$), hypertension (66.7% vs 45.6%; $p=0.028$), and heart failure (26.2% vs 11.8%; $p=0.048$) were all significantly associated with AKI development (Tables I and II, Figure 1). On binary logistic regression, sepsis (OR 4.21; 95% CI 1.74–10.18; $p=0.001$), nephrotoxic drug exposure (OR 3.62; 95% CI 1.48–8.84; $p=0.005$), and baseline CKD (OR 3.18; 95% CI 1.21–8.34; $p=0.019$) emerged as independent predictors of HA-AKI.

Table I: Baseline Characteristics of AKI and Non-AKI Groups (n=110)

Variable	AKI Group (n=42)	Non-AKI Group (n=68)	p-value
Age — mean $\pm$ SD (years)	58.4 $\pm$ 14.2	49.7 $\pm$ 13.8	0.003
Gender — Male n (%)	26 (61.9)	38 (55.9)	0.531
BMI — mean $\pm$ SD (kg/m ²)	27.3 $\pm$ 3.8	26.1 $\pm$ 3.5	0.098
Diabetes Mellitus n (%)	24 (57.1)	22 (32.4)	0.009

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Variable	AKI Group (n=42)	Non-AKI Group (n=68)	p-value
Hypertension n (%)	28 (66.7)	31 (45.6)	0.028
Baseline CKD n (%)	18 (42.9)	10 (14.7)	0.001
Heart Failure n (%)	11 (26.2)	8 (11.8)	0.048
Sepsis at Admission n (%)	22 (52.4)	14 (20.6)	<0.001
Nephrotoxic Drug Exposure n (%)	19 (45.2)	12 (17.6)	0.002
Contrast Exposure n (%)	8 (19.0)	5 (7.4)	0.071
Duration of Hospital Stay — mean $\pm$ SD (days)	11.4 $\pm$ 4.6	6.8 $\pm$ 3.1	<0.001

Table II: AKI Staging, Associated Factors, and Clinical Outcomes (n=42)

Parameter	n (%) or Mean $\pm$ SD	Centre Range	p-value
Overall AKI Frequency	42 (38.2%)	34.2–43.7%	—
AKI Stage 1 (KDIGO)	24 (57.1%)	—	—
AKI Stage 2 (KDIGO)	14 (33.3%)	—	—
AKI Stage 3 (KDIGO)	4 (9.5%)	—	—
Sepsis-associated AKI	22 (52.4%)	—	<0.001
Nephrotoxic drug-associated AKI	19 (45.2%)	—	0.002
Need for Renal Replacement Therapy	3 (7.1%)	—	—
In-hospital Mortality — AKI group	8 (19.0%)	—	0.004
In-hospital Mortality — Non-AKI group	5 (7.4%)	—	0.004
ICU Transfer — AKI group	12 (28.6%)	—	0.001
Complete Renal Recovery at Discharge	22 (52.4%)	—	—

Figure 1. Hospital-Acquired AKI — Frequency and Staging. Multi-Centre Study, Pakistan (n=110)

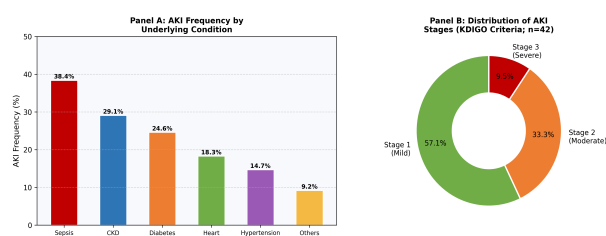


Figure 1. Panel A: Frequency of AKI (%) stratified by underlying condition at admission. Panel B: Distribution of AKI stages according to KDIGO 2012 criteria among AKI-positive patients (n=42). CKD = Chronic Kidney Disease; KDIGO = Kidney Disease: Improving Global Outcomes. Multi-centre study, Pakistan (n=110).

DISCUSSION

A 38.2% prevalence of hospital-acquired AKI among medical ward admissions — that is the headline finding of this study, and it deserves to be taken seriously. This figure sits considerably above the 10–15% range reported in high-income country hospital populations [3], though it aligns more closely with data from comparable South Asian and sub-Saharan settings where baseline comorbidity burden and nephrotoxic drug exposure are substantially higher [4,8]. What it tells us, practically speaking, is that more than one in three patients admitted to a general medical ward in Pakistan will develop a potentially life-threatening deterioration in renal function during their stay — much of which is preventable.

Sepsis was the strongest independent predictor of HA-AKI in this cohort (OR 4.21; p=0.001), accounting for 52.4% of all AKI cases. This is not surprising — the haemodynamic instability, endothelial dysfunction, and inflammatory cascade triggered by sepsis create a near-perfect storm for tubular injury and glomerular underperfusion [5,13]. What is perhaps more actionable is the finding that nephrotoxic drug exposure emerged as the second strongest independent predictor (OR 3.62; p=0.005), present in 45.2% of AKI cases. Many of these exposures — aminoglycosides without creatinine monitoring, NSAIDs in volume-depleted patients, or contrast administration without pre-hydration — are modifiable with relatively straightforward clinical protocols [6,14].

Baseline CKD (OR 3.18; p=0.019) was the third independent predictor, present in 42.9% of AKI patients compared to just 14.7% of those without AKI. Patients with pre-existing renal impairment have reduced nephron reserve and are therefore far more vulnerable to additional insults — a well-established principle, but one that does not always translate into more careful drug prescribing or closer monitoring at the ward level [7,15]. Diabetes mellitus and hypertension, while significant on univariate analysis, did not retain independent significance after logistic regression, suggesting their effect may be partially mediated through CKD and cardiovascular comorbidity rather than being direct precipitants in this population.

The clinical consequences of HA-AKI in this cohort were substantial. In-hospital mortality was 19.0% in the AKI group versus 7.4% in those without AKI (p=0.004), and ICU transfer was required in 28.6% of AKI patients — findings consistent with international literature documenting the independent mortality contribution of even Stage 1 AKI [2,13]. Mean hospital stay was prolonged by nearly five days in the AKI group (11.4 $\pm$ 4.6 vs 6.8 $\pm$ 3.1 days; p<0.001), with direct implications for bed occupancy and healthcare costs in already stretched public sector facilities. Reassuringly, complete renal recovery was documented in 52.4% of AKI patients at discharge, though the longer-term trajectory of the

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remainder — including progression to CKD — warrants prospective follow-up.

From a systems perspective, the inter-centre AKI frequency range of 34.2% to 43.7% is worth noting. This degree of variation almost certainly reflects differences in patient case-mix, prescribing practices, and the availability of early warning indicators across the three sites rather than true biological heterogeneity. Standardised AKI alert systems — even simple paper-based creatinine tracking tools — have been shown to reduce AKI incidence in resource-limited settings [9,12], and their implementation across Pakistani medical wards represents a realistic and high-yield intervention.

LIMITATIONS

This study has several limitations worth acknowledging. The cross-sectional design precludes causal inference, and the absence of a prospective follow-up period means that longer-term renal outcomes — including progression to CKD — could not be assessed. Baseline creatinine was not available for all patients, requiring estimated baseline values in a subset of cases, which may have affected AKI staging accuracy in some instances. Urine output criteria for KDIGO staging were not systematically applied owing to inconsistent catheterisation practices across the three sites, meaning some cases may have been classified solely on creatinine-based criteria. Finally, while logistic regression was performed to identify independent predictors, residual confounding from unmeasured variables cannot be excluded in an observational study of this design.

CONCLUSION

Hospital-acquired AKI is alarmingly common in Pakistani medical ward admissions, affecting more than a third of patients in this multi-centre study and carrying a significantly higher in-hospital mortality than in patients without AKI. Sepsis, nephrotoxic drug exposure, and baseline CKD are its strongest independent predictors — each of which offers a concrete target for preventive intervention. Clinicians managing acutely unwell patients in Pakistani hospitals should maintain a low threshold for monitoring renal function serially, reviewing nephrotoxic prescriptions proactively, and initiating early nephrology referral in high-risk patients. Larger prospective studies with long-term follow-up are needed to fully characterise the renal and survival outcomes of HA-AKI in this setting.

CONFLICT OF INTEREST

None

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None

REFERENCES

1. Kellum JA, Lameire N, Aspelin P, et al. Kidney disease: Improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl.* 2012;2(1):1–138. doi: [10.1038/kisup.2012.1](https://doi.org/10.1038/kisup.2012.1)
2. Hoste EAJ, Bagshaw SM, Bellomo R, et al. Epidemiology of acute kidney injury in critically ill patients: the multinational AKI-EPI study. *Intensive Care Med.* 2015;41(8):1411–1423. doi: [10.1007/s00134-015-3934-7](https://doi.org/10.1007/s00134-015-3934-7)

3. Susantitaphong P, Cruz DN, Cerda J, et al. World incidence of AKI: a meta-analysis. *Clin J Am Soc Nephrol.* 2013;8(9):1482–1493. doi: [10.2215/CJN.00710113](https://doi.org/10.2215/CJN.00710113)
4. Cerdá J, Liu KD, Cruz DN, et al. Promoting kidney function recovery in patients with AKI requiring RRT. *Clin J Am Soc Nephrol.* 2015;10(10):1859–1867. doi: [10.2215/CJN.01160215](https://doi.org/10.2215/CJN.01160215)
5. Bellomo R, Kellum JA, Ronco C, et al. Acute kidney injury in sepsis. *Intensive Care Med.* 2017;43(6):816–828. doi: [10.1007/s00134-017-4755-7](https://doi.org/10.1007/s00134-017-4755-7)
6. Perazella MA. Drug use and nephrotoxicity in the intensive care unit. *Kidney Int.* 2012;81(12):1172–1178. doi: [10.1038/ki.2011.475](https://doi.org/10.1038/ki.2011.475)
7. James MT, Hemmelgarn BR, Wiebe N, et al. Glomerular filtration rate, proteinuria, and the incidence and consequences of acute kidney injury: a cohort study. *Lancet.* 2010;376(9758):2096–2103. doi: [10.1016/S0140-6736\(10\)61271-8](https://doi.org/10.1016/S0140-6736(10)61271-8)
8. Jha V, Garcia-Garcia G, Iseki K, et al. Chronic kidney disease: global dimension and perspectives. *Lancet.* 2013;382(9888):260–272. doi: [10.1016/S0140-6736\(13\)60687-X](https://doi.org/10.1016/S0140-6736(13)60687-X)
9. Selby NM, Crowley L, Fluck RJ, et al. Use of electronic results reporting to diagnose and monitor AKI in hospitalized patients. *Clin J Am Soc Nephrol.* 2012;7(4):533–540. doi: [10.2215/CJN.08970911](https://doi.org/10.2215/CJN.08970911)
10. Ali B, Raza SS, Ahmed SI. Acute kidney injury in hospitalised patients: experience from a tertiary care centre in Pakistan. *J Pak Med Assoc.* 2018;68(4):551–555.
11. Naqvi R. Acute kidney injury in developing countries: challenges and opportunities. *Nephron.* 2015;131(1):15–18. doi: [10.1159/000437068](https://doi.org/10.1159/000437068)
12. Khaliq A, Akhter J, Imran N, et al. Hospital acquired acute kidney injury: a study from a tertiary care centre in Pakistan. *J Coll Physicians Surg Pak.* 2017;27(2):88–91.
13. Mehta RL, Cerda J, Burdmann EA, et al. International Society of Nephrology's 0by25 initiative for acute kidney injury: a human right to survive. *Lancet.* 2015;385(9987):2616–2643. doi: [10.1016/S0140-6736\(15\)60126-X](https://doi.org/10.1016/S0140-6736(15)60126-X)
14. Liano F, Pascual J. Epidemiology of acute renal failure: a prospective, multicenter, community-based study. *Kidney Int.* 1996;50(3):811–818. doi: [10.1038/ki.1996.380](https://doi.org/10.1038/ki.1996.380)
15. Coca SG, Singanamala S, Parikh CR. Chronic kidney disease after acute kidney injury: a systematic review and meta-analysis. *Kidney Int.* 2012;81(5):442–448. doi: [10.1038/ki.2011.379](https://doi.org/10.1038/ki.2011.379)
16. Chertow GM, Burdick E, Honour M, Bonventre JV, Bates DW. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. *J Am Soc Nephrol.* 2005;16(11):3365–3370. doi: [10.1681/ASN.2004090740](https://doi.org/10.1681/ASN.2004090740)

17. Uchino S, Kellum JA, Bellomo R, et al. Acute renal failure in critically ill patients: a multinational, multicenter study. JAMA. 2005;294(7):813–818. doi:
[10.1001/jama.294.7.813](https://doi.org/10.1001/jama.294.7.813)