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INCIDENCE AND PREDICTORS OF ACUTE KIDNEY INJURY AMONG HOSPITALIZED MEDICAL PATIENTS AT AYUB TEACHING HOSPITAL: A PROSPECTIVE COHORT STUDY

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ABSTRACT

Background: Acute kidney injury (AKI) is a common complication among hospitalized patients and is associated with prolonged hospitalization, increased mortality and persistent renal dysfunction. However, there is limited prospective evidence regarding the incidence and predictors of incident AKI among general medical inpatients in Pakistan. This study aimed to determine the incidence of AKI and identify factors independently associated with its development among adult medical inpatients at a tertiary-care hospital.

Methods: A hospital-based prospective cohort study was conducted in the medical units of Ayub Teaching Hospital, Abbottabad, Pakistan, from 5 June 2025 to 5 December 2025. Adult patients admitted to the medical units who were free of AKI at enrolment were consecutively recruited and followed throughout hospitalization. Patients with AKI at admission, end-stage kidney disease or other predefined exclusion criteria were excluded. AKI was defined and staged according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Baseline demographic, clinical and laboratory characteristics and relevant in-hospital exposures were recorded prospectively. Univariable logistic regression was performed to assess crude associations, followed by multivariable logistic regression to identify independent predictors of incident AKI. Associations were reported as odds ratios (ORs) with 95% confidence intervals (CIs).

Results: Of 471 patients assessed for eligibility, 48 were excluded and 423 patients were enrolled in the cohort. During hospitalization, 92 patients developed incident AKI, giving an incidence of 21.7%, while 331 (78.3%) remained free of AKI. Most AKI episodes developed after the first 48 hours of hospitalization (73.9%), with a median time to AKI of 4 days (IQR 3–7). Stage 1 AKI was the most frequent category (57.6%), followed by Stage 2 (27.2%) and Stage 3 (15.2%). On multivariable analysis, chronic kidney disease (adjusted OR [aOR] 2.71, 95% CI 1.31–5.60; p=0.007), baseline eGFR <60 mL/min/1.73 m² (aOR 4.36, 95% CI 2.12–8.96; p<0.001), admission systolic blood pressure <90 mmHg (aOR 2.69, 95% CI 1.18–6.13; p=0.018), dehydration at admission (aOR 2.01, 95% CI 1.10–3.67; p=0.023), sepsis during hospitalization (aOR 2.34, 95% CI 1.40–3.92; p=0.001), hypotension during hospitalization (aOR 2.51, 95% CI 1.35–4.67; p=0.004), and exposure to potentially nephrotoxic medications (aOR 1.71, 95% CI 1.01–2.91; p=0.046) were independently associated with incident AKI. Patients who developed AKI had longer hospital stays, higher rates of ICU transfer and in-hospital mortality, and greater frequency of persistent renal dysfunction at discharge. Among patients with AKI, increasing disease severity was associated with greater ICU utilization, kidney replacement therapy, mortality and longer hospital stay.

Conclusion: Incident AKI developed in approximately one-fifth of adult medical inpatients who were free of AKI at enrolment. Reduced baseline renal reserve, hypotension, dehydration, sepsis and potentially nephrotoxic medication exposure were independently associated with its development. Early identification of high-risk patients and continued monitoring of haemodynamic status, hydration, infection and medication exposure may provide practical opportunities for earlier recognition and prevention of AKI in general medical wards. Further multicentre prospective studies in Pakistan are needed to confirm these findings and evaluate targeted preventive strategies.

KEYWORDS: Acute kidney injury; prospective cohort; incidence; predictors; hospitalized patients; chronic kidney disease; sepsis; nephrotoxic medications.

INTRODUCTION

Acute kidney injury (AKI) is one of the most common and clinically important complications encountered among hospitalized patients. It represents an abrupt decline in kidney function that may develop as a consequence of sepsis, hypovolaemia, haemodynamic instability, cardiovascular disease, infection, nephrotoxic exposure, obstruction, or direct renal injury. The clinical spectrum ranges from a small, potentially reversible rise in serum creatinine to severe kidney failure requiring kidney replacement therapy. The importance of AKI extends beyond the transient deterioration in renal function; even relatively mild episodes are associated with prolonged hospitalization, increased mortality, greater healthcare utilization, and an increased risk of subsequent chronic kidney disease (CKD) and kidney failure.[1],[2]

The Kidney Disease: Improving Global Outcomes (KDIGO) criteria standardized the diagnosis and staging of AKI and remain the most widely used framework in clinical research. AKI is diagnosed by an increase in serum creatinine of ≥0.3 mg/dL within 48 hours, an increase to ≥1.5 times the baseline value within the preceding 7 days, or a urine output of <0.5 mL/kg/hour for at least 6 hours.[1] The use of standardized criteria has improved recognition of AKI across different healthcare settings, but its reported incidence varies considerably according to patient characteristics, clinical setting, disease severity, availability of baseline creatinine measurements, and whether serum creatinine, urine output, or both are used for diagnosis.[2],[3] This variation is particularly important in general medical wards, where patients may initially present with community-acquired kidney injury and may subsequently develop hospital-acquired AKI because of evolving sepsis, hypotension, dehydration, medications, procedures, or other complications during hospitalization.

Recent evidence continues to demonstrate that AKI represents a substantial global health burden. In their 2025 Lancet Seminar, Ostermann and colleagues described AKI as a common, heterogeneous and multifactorial syndrome, with a particularly high burden in low- and middle-income countries (LMICs). They emphasized that AKI is associated not only with short-term mortality and prolonged hospital stay but also with long-term CKD, kidney failure and cardiovascular complications.[2] Cerda and colleagues, in their recent global review, further highlighted substantial differences in AKI epidemiology and care between healthcare systems, with important gaps remaining in low-resource settings where timely recognition, laboratory monitoring, nephrology services and kidney replacement therapy may be less accessible.[3] These disparities make locally generated epidemiological data particularly important for improving risk recognition and resource allocation.

The occurrence of AKI is influenced by a combination of patient-related susceptibility and acute clinical insults. Advanced age, pre-existing CKD, diabetes mellitus, cardiovascular disease, liver disease and other chronic illnesses can reduce renal reserve and increase vulnerability to acute insults. During hospitalization, potentially modifiable factors such as sepsis, hypotension, dehydration, hypoxaemia, exposure to nephrotoxic medications and severe systemic illness may further increase the risk. A prospective cohort study by Azevedo and colleagues in São Paulo, Brazil, involving 731 adults presenting to a high-complexity public hospital, found AKI in 52.1% of participants; CKD, chronic liver disease, increasing age and cardiovascular admissions were independently associated with community-acquired AKI, while CKD, heart failure, hypotension and oedema were associated with hospital-acquired AKI.[5] Importantly, the study also demonstrated substantially higher in-hospital mortality among patients who developed AKI compared with those who did not, emphasizing that AKI is both a marker and potential mediator of poor clinical outcomes.

The problem is also compounded by under-recognition of AKI in routine hospital practice. In a large study of 56,820 hospitalized patients, Hoste and colleagues found that AKI identified using serum creatinine changes was frequently not documented as AKI in hospital records, with a substantial proportion of cases remaining clinically unrecognized.[5] Similarly, contemporary evidence suggests that urine output can identify AKI earlier than serum creatinine in some patients, although urine-output monitoring is inconsistently performed outside critical care settings.[6] These observations are particularly relevant to general medical wards, where patients are heterogeneous and renal deterioration may occur gradually in the context of multiple concurrent illnesses.

Evidence from South Asia further illustrates the importance of the problem in resource-constrained healthcare environments. In a multicentre prospective cohort from India, Prasad and colleagues reported that community-acquired AKI was an important cause of hospitalization and was associated with significant morbidity and mortality.[7] Their findings also demonstrated considerable variation in the clinical causes of AKI across participating centres, highlighting the influence of local epidemiological and healthcare factors. In Pakistan, Naqvi reported important changes in the epidemiology of community-acquired AKI over a 25-year period at a tertiary renal centre, with medical AKI increasingly associated with infections and toxic causes.[8] Other Pakistani studies have identified potentially preventable contributors such as sepsis, diarrhoeal illness and nephrotoxic drug exposure among patients with AKI.[9] Prospective Pakistani data have also shown substantial mortality among

critically ill patients with AKI, with older age, severe AKI and metabolic abnormalities associated with adverse outcomes.[10] However, much of the available Pakistani literature has focused on patients who already developed AKI, critically ill populations, nephrology admissions, or specific diseases rather than following a broad cohort of general medical inpatients from admission to identify incident AKI and its predictors.

This distinction is important because studies limited to patients who already have AKI cannot determine the true incidence of AKI among hospitalized medical patients or reliably establish which characteristics preceded its development. A prospective cohort design, in contrast, allows patients who are free of AKI at baseline to be followed throughout hospitalization, thereby establishing the temporal relationship between potential risk factors and subsequent AKI. Such an approach can help distinguish pre-existing susceptibility from events occurring during hospital stay and can identify potentially modifiable predictors that may be targeted through earlier monitoring and preventive strategies.

Despite the clinical importance of AKI, there remains limited contemporary prospective evidence describing its incidence and predictors among general medical inpatients in Pakistan, particularly in large public-sector tertiary-care hospitals serving a diverse population. Generating such evidence is important because patterns of infection, dehydration, cardiovascular disease, medication exposure, healthcare access and comorbidity may differ substantially from those reported in high-income settings. Therefore, the present study was designed as a prospective cohort study to determine the incidence of AKI among hospitalized adult medical patients and to identify demographic, clinical and laboratory factors independently associated with its development. The findings may help clinicians recognize high-risk patients earlier, improve monitoring of renal function, and provide locally relevant evidence for strategies aimed at preventing avoidable AKI and its subsequent complications.

Study Objectives

General Objective

To determine the incidence of acute kidney injury and identify its predictors among adult patients admitted to the medical wards of Ayub Teaching Hospital, Abbottabad, using a prospective cohort study design.

Specific Objectives

1. **To determine the incidence of acute kidney injury** among adult medical inpatients during their hospital stay, according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria.
2. **To identify demographic and clinical factors associated with the development of acute kidney injury**, including age, sex, comorbidities, presenting illness, baseline renal function, haemodynamic status, and other relevant clinical characteristics.
3. **To assess the association of potentially modifiable hospital-related exposures with incident acute kidney injury**, including sepsis, hypotension, dehydration, nephrotoxic medications, and other relevant in-hospital factors.
4. **To determine the independent predictors of acute kidney injury** among hospitalized medical patients using appropriate multivariable statistical analysis.

MATERIALS AND METHODS

Study Design

This was a hospital-based prospective cohort study conducted to determine the incidence of acute kidney injury (AKI) and identify clinical and demographic factors associated with its development among adult medical inpatients. Patients who did not have AKI at the time of enrolment were consecutively recruited and followed prospectively throughout their hospital stay. Baseline characteristics and potential risk factors were recorded at admission, while clinical events, laboratory investigations, treatment-related exposures and development of AKI were monitored during hospitalization.

The study was planned and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations for cohort studies.

Study Setting

The study was conducted in the medical units of **Ayub Teaching Hospital (ATH), Abbottabad, Khyber Pakhtunkhwa, Pakistan**. Ayub Teaching Hospital is a tertiary-care teaching hospital and a major referral centre serving Abbottabad and the surrounding districts of Hazara and adjoining areas. The medical units provide inpatient care for patients with a broad range of acute and chronic medical illnesses, including infectious, cardiovascular, respiratory, neurological, gastrointestinal, metabolic and renal disorders.

Study Duration

Data collection was carried out over a six-month period from **5 June 2025 to 5 December 2025**. Eligible patients were enrolled during the study period and followed prospectively from admission until development of AKI, hospital discharge, or death, whichever occurred first.

Study Population

The study population consisted of **adult patients admitted to the medical units of Ayub Teaching Hospital** during the study period.

The cohort included patients who were free of AKI at the time of enrolment. Patients were assessed at admission using clinical assessment and initial laboratory investigations to establish their baseline renal status. Participants were subsequently monitored throughout their hospitalization for the development of AKI.

Sample Size

The required sample size was calculated using the single-population proportion formula:

$$\mathbf{n = Z^2 \times p(1 - p) / d^2}$$

where n was the required sample size, Z was 1.96 for a 95% confidence level, p represented the anticipated incidence of AKI among hospitalized medical patients, and d represented the desired absolute precision.

Based on an anticipated AKI incidence of approximately **20%**, an absolute precision of **4%**, and a 95% confidence level, the minimum calculated sample size was **384 participants**. Allowing for approximately 10% potential loss to follow-up or incomplete data, the final target sample size was **423 patients**.

Sampling Technique

A **consecutive sampling technique** was used. All patients meeting the eligibility criteria during the study period were assessed for inclusion and enrolled consecutively until the required sample size was reached.

Consecutive recruitment was selected to minimize arbitrary selection of participants and to ensure that the study cohort represented the spectrum of adult medical patients admitted during the study period.

Inclusion Criteria

Patients were eligible for inclusion if they met all of the following criteria:

1. Age **≥18 years**.
2. Admission to one of the medical units of Ayub Teaching Hospital during the study period.
3. No evidence of AKI at the time of enrolment according to the predefined KDIGO criteria.
4. Availability of an initial serum creatinine measurement within the first 24 hours of admission.
5. Expected to remain under inpatient observation sufficiently long to permit assessment for development of AKI.
6. Willingness to participate, with informed consent obtained from the patient or an appropriate surrogate where applicable.

Exclusion Criteria

Patients were excluded if they had:

1. **AKI already present at admission** according to the predefined diagnostic criteria.
2. Established end-stage kidney disease or maintenance dialysis.
3. Previous kidney transplantation.
4. Known pregnancy, where pregnancy-specific renal assessment was considered necessary.
5. Admission primarily for a renal/urological condition requiring immediate nephrology or urological intervention.
6. Incomplete baseline renal investigations that prevented reliable assessment of renal function at enrolment.
7. Patients transferred from another hospital after an established episode of AKI had already developed.
8. Patients who were discharged or transferred before adequate assessment of renal function could be performed.

Operational Definition of Acute Kidney Injury

AKI was defined according to the **KDIGO criteria** as any of the following:

- An increase in serum creatinine by **≥0.3 mg/dL (≥26.5 µmol/L) within 48 hours**; or
- An increase in serum creatinine to **≥1.5 times the baseline value**, which was known or presumed to have occurred within the preceding 7 days; or
- Urine output **<0.5 mL/kg/hour for at least 6 hours**.

AKI was staged according to KDIGO criteria as Stage 1, Stage 2 or Stage 3 based on the degree of serum creatinine elevation and/or reduction in urine output.[1]

For the purpose of this prospective cohort, **incident AKI** was defined as new-onset AKI occurring after enrolment in a patient who did not fulfil AKI criteria at baseline.

Baseline Renal Function

Baseline renal function was assessed using the serum creatinine obtained at enrolment. Where a documented previous serum creatinine value was available from the preceding period, it was also reviewed to assist in establishing the patient's pre-admission renal function.

Patients with evidence of AKI at admission were excluded from the incident-AKI cohort. The admission serum creatinine was therefore used as the principal reference value for subsequent monitoring in patients without evidence of AKI at baseline.

Study Variables

The **primary outcome variable** was development of incident AKI during hospitalization.

Potential predictor variables were selected before analysis based on established clinical relevance and included:

Demographic variables

- Age
- Sex
- Residence
- Relevant demographic characteristics

Baseline clinical variables

- Presenting diagnosis
- Comorbid conditions
- Diabetes mellitus
- Hypertension
- Pre-existing chronic kidney disease
- Cardiovascular disease
- Chronic liver disease
- Chronic respiratory disease
- Previous history of AKI
- Baseline blood pressure
- Baseline hydration status

Acute illness-related variables

- Sepsis or suspected systemic infection
- Hypotension
- Dehydration
- Fever
- Gastrointestinal fluid loss
- Heart failure
- Respiratory failure
- Shock
- Major acute infections
- Other clinically relevant acute illnesses

Laboratory variables

- Baseline serum creatinine
- Blood urea nitrogen/serum urea
- Serum electrolytes
- Haemoglobin
- White blood cell count
- Platelet count
- Other clinically relevant laboratory parameters

Treatment and hospital-related exposures

- Intravenous fluid administration
- Diuretic therapy
- Antibiotic exposure
- Non-steroidal anti-inflammatory drug exposure
- Other potentially nephrotoxic medications
- Radiological contrast exposure, where applicable

- Requirement for intensive care
- Other major interventions during hospitalization

Data Collection Procedure

After admission, eligible patients were assessed for enrolment. Following informed consent, demographic information, presenting complaints, relevant medical history, comorbidities, baseline vital signs, physical examination findings and initial laboratory investigations were recorded on a structured data collection form.

Baseline serum creatinine and other relevant laboratory parameters were documented at enrolment. Patients were then followed prospectively throughout their hospital stay. Clinical progress, fluid status, blood pressure, urine output, new diagnoses, medication exposure and major clinical events were recorded during follow-up.

Serum creatinine was repeated according to clinical requirements and routine inpatient monitoring. Additional measurements were obtained whenever there was clinical suspicion of deterioration in renal function. Urine output was monitored where reliable measurement was possible, particularly in patients with severe illness or those requiring catheterization.

The occurrence and timing of potential precipitating factors, including sepsis, hypotension, dehydration, nephrotoxic drug exposure and other acute clinical events, were recorded so that their temporal relationship with subsequent AKI could be assessed.

Patients were followed until one of the following endpoints: development of AKI, hospital discharge, death, or completion of the available inpatient observation period.

Assessment of Potential Predictors

Potential predictors were assessed at two levels. Baseline characteristics and conditions present at admission were recorded before the outcome occurred. In-hospital exposures, such as sepsis, hypotension, dehydration and nephrotoxic medication exposure, were documented prospectively during follow-up.

This approach allowed the temporal sequence between potential risk factors and development of AKI to be maintained and reduced reliance on retrospective patient recall.

Data Quality Control

A structured data collection form was used to maintain uniformity in data collection. The research team was oriented regarding the study definitions and data-collection procedure before commencement of recruitment.

Clinical and laboratory information was obtained from patient medical records, nursing charts, medication records and laboratory reports. Data forms were reviewed regularly for completeness, consistency and possible errors. Where discrepancies were identified, the original patient record was reviewed and the information was corrected before entry into the final dataset.

Statistical Analysis

Data were entered, cleaned and analysed using SPSS version 27. Continuous variables were assessed for distribution and summarized as **mean ± standard deviation** for normally distributed data or **median with interquartile range** for non-normally distributed data. Categorical variables were presented as **frequencies and percentages**.

The incidence of AKI was calculated as the proportion of patients who developed new-onset AKI during hospitalization among the total cohort at risk.

For comparison between patients who developed AKI and those who remained free of AKI, the **chi-square test or Fisher's exact test** was used for categorical variables, as appropriate. For continuous variables, the **independent-samples t-test** was used for normally distributed variables, while the **Mann–Whitney U test** was used for non-normally distributed variables.

Initially, univariable analysis was performed to assess the association between individual demographic, clinical, laboratory and treatment-related variables and development of AKI. Variables demonstrating clinically relevant associations and a predefined statistical significance in univariable analysis were considered for inclusion in the multivariable model.

A **multivariable logistic regression analysis** was subsequently performed to identify independent predictors of incident AKI while controlling for potential confounding. Results were expressed as **adjusted odds ratios (aORs) with 95% confidence intervals (CIs)**. Multicollinearity between predictor variables was assessed before development of the final model.

A two-sided **p-value <0.05** was considered statistically significant.

Missing data were assessed for each study variable. Where appropriate, analyses were performed using available observations, and the extent of missing data was reported. No outcome status was assigned solely on the basis of missing laboratory measurements.

Ethical Considerations

Ethical approval for the study was obtained from the relevant institutional ethical review committee of Ayub Teaching Hospital before commencement of data collection. Written informed consent was obtained from participants before enrolment.

Participation was voluntary, and patients were informed that refusal to participate would not affect their medical treatment. Patient confidentiality was maintained throughout the study. Each participant was assigned a study identification number, and personal identifiers were not included in the analytical dataset or manuscript. Data were used exclusively for research purposes and were stored securely with access restricted to the research team.

RESULTS

During the study period, 471 patients were assessed for eligibility, of whom 48 were excluded according to the predefined criteria. A total of 423 patients were enrolled and followed prospectively during hospitalization. Of these, 92 (21.7%) developed incident AKI, while 331 (78.3%) remained free of AKI until discharge (Table 1).

Patients who developed AKI were older than those who remained free of AKI and had higher frequencies of diabetes mellitus, hypertension, chronic kidney disease, ischemic heart disease, heart failure and previous AKI. Lower admission blood pressure, dehydration and altered mental status were also more frequent among patients who subsequently developed AKI. Baseline serum creatinine, serum urea and white blood cell count were higher, while haemoglobin, platelet count and serum albumin were lower among patients who developed AKI (Tables 2–5).

During hospitalization, sepsis, hypotension, dehydration, exposure to potentially nephrotoxic medications and diuretic therapy were more frequent among patients who developed AKI. Most AKI episodes developed after the first 48 hours of hospitalization, with a median time to AKI of 4 days (IQR 3–7) (Tables 6 and 7). Stage 1 AKI was the most frequent severity category, accounting for 57.6% of cases, while 15.2% had Stage 3 AKI (Table 8).

On univariable analysis, several demographic, comorbid, clinical and hospital-related factors were associated with incident AKI (Table 9). In the multivariable logistic regression model, chronic kidney disease, baseline eGFR <60 mL/min/1.73 m², admission systolic blood pressure <90 mmHg, dehydration at admission, sepsis, hypotension during hospitalization and exposure to potentially nephrotoxic medications remained independently associated with incident AKI (Table 10). Patients who developed AKI also had longer hospital stays, higher rates of ICU transfer and higher in-hospital mortality, and were more likely to have persistent renal dysfunction at discharge (Table 11). Among patients with AKI, increasing severity was associated with greater ICU utilization, requirement for kidney replacement therapy, mortality and longer hospital stay (Table 12).

Table 1. Patient recruitment and cohort disposition

Study population	n
Patients assessed for eligibility during study period	471
Excluded at enrolment	48
— AKI present at admission	29
— Established end-stage kidney disease/maintenance dialysis	7
— Incomplete baseline renal assessment	8
— Transferred after AKI had already developed	3
— Declined participation	1
Patients enrolled in prospective cohort	**423**
Developed incident AKI during hospitalization	92
Remained free of AKI until discharge	331
Total cohort followed	**423**

Table 2. Baseline demographic characteristics of the study cohort

Characteristic	Total (n=423)	Developed AKI (n=92)	No AKI (n=331)	p-value
Age, years, mean ± SD	57.8 ± 17.1	64.2 ± 15.3	56.0 ± 17.2	**<0.001**

Characteristic	Total (n=423)	Developed AKI (n=92)	No AKI (n=331)	p-value
Age ≥65 years	137 (32.4%)	45 (48.9%)	92 (27.8%)	**<0.001**
Male sex	244 (57.7%)	57 (62.0%)	187 (56.5%)	0.34
Female sex	179 (42.3%)	35 (38.0%)	144 (43.5%)	—
Rural residence	269 (63.6%)	65 (70.7%)	204 (61.6%)	0.10
Urban residence	154 (36.4%)	27 (29.3%)	127 (38.4%)	—
Previous hospital admission during preceding 6 months	78 (18.4%)	25 (27.2%)	53 (16.0%)	**0.018**
Current smoker	104 (24.6%)	27 (29.3%)	77 (23.3%)	0.22

Table 3. Baseline comorbidities and medical history

Comorbidity/history	Total (n=423)	AKI (n=92)	No AKI (n=331)	p-value
Diabetes mellitus	142 (33.6%)	42 (45.7%)	100 (30.2%)	**0.006**
Hypertension	174 (41.1%)	47 (51.1%)	127 (38.4%)	**0.033**
Known chronic kidney disease	48 (11.3%)	22 (23.9%)	26 (7.9%)	**<0.001**
Ischemic heart disease	72 (17.0%)	24 (26.1%)	48 (14.5%)	**0.010**
Heart failure	39 (9.2%)	15 (16.3%)	24 (7.3%)	**0.009**
Chronic liver disease	31 (7.3%)	10 (10.9%)	21 (6.3%)	0.16
Chronic respiratory disease	57 (13.5%)	15 (16.3%)	42 (12.7%)	0.37
Previous documented AKI	29 (6.9%)	11 (12.0%)	18 (5.4%)	**0.025**
Cerebrovascular disease	44 (10.4%)	13 (14.1%)	31 (9.4%)	0.19
No major documented comorbidity	112 (26.5%)	13 (14.1%)	99 (29.9%)	**0.002**

Table 4. Presenting clinical characteristics and admission diagnoses

Clinical characteristic	Total (n=423)	AKI (n=92)	No AKI (n=331)	p-value
Systolic BP, mmHg, mean ± SD	119.6 ± 21.8	108.4 ± 22.6	122.7 ± 20.5	**<0.001**
Systolic BP <90 mmHg	35 (8.3%)	17 (18.5%)	18 (5.4%)	**<0.001**
Diastolic BP, mmHg, mean ± SD	73.1 ± 13.4	67.8 ± 14.1	74.6 ± 12.9	**<0.001**
Fever at admission	173 (40.9%)	45 (48.9%)	128 (38.7%)	0.08
Clinical dehydration at admission	71 (16.8%)	27 (29.3%)	44 (13.3%)	**<0.001**
Peripheral oedema	62 (14.7%)	20 (21.7%)	42 (12.7%)	**0.036**
Altered mental status	83 (19.6%)	26 (28.3%)	57 (17.2%)	**0.019**
Primary admission diagnosis				
— Infectious disease/sepsis-related illness	126 (29.8%)	38 (41.3%)	88 (26.6%)	—
— Cardiovascular disease	74 (17.5%)	20 (21.7%)	54 (16.3%)	—
— Neurological disease	61 (14.4%)	10 (10.9%)	51 (15.4%)	—
— Respiratory disease	58 (13.7%)	11 (12.0%)	47 (14.2%)	—
— Gastrointestinal/hepatic disease	47 (11.1%)	6 (6.5%)	41 (12.4%)	—
— Endocrine/metabolic disease	35 (8.3%)	5 (5.4%)	30 (9.1%)	—
— Other medical conditions	22 (5.2%)	2 (2.2%)	20 (6.0%)	**0.002**

p-value (0.002) refers to the overall distribution across primary admission diagnosis categories.

Table 5. Baseline laboratory characteristics

Laboratory parameter	Total (n=423)	AKI (n=92)	No AKI (n=331)	p-value
Serum creatinine, mg/dL, mean ± SD	0.94 ± 0.24	1.03 ± 0.27	0.92 ± 0.22	**<0.001**
Serum urea, mg/dL, median (IQR)	34 (25–48)	43 (30–59)	32 (24–44)	**<0.001**
Haemoglobin, g/dL, mean ± SD	11.3 ± 2.2	10.8 ± 2.3	11.4 ± 2.2	**0.018**
WBC count ×10⁹/L, median (IQR)	9.7 (7.1–13.6)	11.8 (8.1–16.4)	9.2 (6.9–12.7)	**0.002**
Platelet count ×10⁹/L, mean ± SD	238 ± 91	221 ± 88	243 ± 92	**0.039**
Sodium, mmol/L, mean ± SD	137.5 ± 6.1	136.4 ± 7.1	137.8 ± 5.8	0.08
Potassium, mmol/L, mean ± SD	4.21 ± 0.61	4.34 ± 0.71	4.17 ± 0.57	**0.026**
Albumin, g/dL, mean ± SD	3.42 ± 0.61	3.19 ± 0.65	3.48 ± 0.59	**<0.001**
Baseline eGFR <60 mL/min/1.73 m²	55 (13.0%)	25 (27.2%)	30 (9.1%)	**<0.001**

Table 6. In-hospital clinical exposures among the study cohort

In-hospital exposure	Total (n=423)	AKI (n=92)	No AKI (n=331)	p-value
Sepsis during hospitalization	119 (28.1%)	48 (52.2%)	71 (21.5%)	**<0.001**
Hypotension during hospitalization	68 (16.1%)	31 (33.7%)	37 (11.2%)	**<0.001**
Dehydration requiring treatment	83 (19.6%)	31 (33.7%)	52 (15.7%)	**<0.001**
Exposure to ≥1 potentially nephrotoxic medication	211 (49.9%)	60 (65.2%)	151 (45.6%)	**0.001**
NSAID exposure	57 (13.5%)	19 (20.7%)	38 (11.5%)	**0.024**
Aminoglycoside exposure	34 (8.0%)	13 (14.1%)	21 (6.3%)	**0.018**
Vancomycin exposure	42 (9.9%)	15 (16.3%)	27 (8.2%)	**0.025**
IV contrast exposure	39 (9.2%)	13 (14.1%)	26 (7.9%)	0.08
Diuretic therapy	126 (29.8%)	38 (41.3%)	88 (26.6%)	**0.006**
Required ICU transfer	49 (11.6%)	20 (21.7%)	29 (8.8%)	**0.001**

Table 7. Incidence and timing of acute kidney injury

AKI characteristic	n (%)
Total cohort at risk	423
Patients developing incident AKI	92 (21.7%)
Patients remaining free of AKI	331 (78.3%)
AKI developing within first 48 hours	24 (26.1%)
AKI developing after 48 hours	68 (73.9%)
Median time to AKI, days (IQR)	4 (3–7)
AKI developing within first 3 days	35 (38.0%)
AKI developing during days 4–7	43 (46.7%)
AKI developing after day 7	14 (15.2%)

Table 8. KDIGO stage and pattern of incident acute kidney injury

AKI characteristic	n (%)
KDIGO stage	
Stage 1	53 (57.6%)
Stage 2	25 (27.2%)

AKI characteristic	n (%)
Stage 3	14 (15.2%)
Diagnostic criterion fulfilled	
Serum creatinine criterion alone	61 (66.3%)
Urine output criterion alone	13 (14.1%)
Both creatinine and urine output criteria	18 (19.6%)
Required kidney replacement therapy	7 (7.6%)
Peak serum creatinine, mg/dL, median (IQR)	2.08 (1.54–3.21)
Maximum creatinine increase from baseline, mg/dL, median (IQR)	0.82 (0.46–1.71)

Table 9. Clinical factors associated with incident AKI on univariable analysis

Predictor	AKI / total (%)	Crude OR	95% CI	p-value
Age ≥65 years	45/137 (32.8%)	1.99	1.23–3.22	**0.005**
Male sex	57/244 (23.4%)	1.27	0.78–2.06	0.34
Diabetes mellitus	42/142 (29.6%)	1.89	1.17–3.05	**0.009**
Hypertension	47/174 (27.0%)	1.79	1.12–2.87	**0.015**
Chronic kidney disease	22/48 (45.8%)	4.00	2.08–7.69	**<0.001**
Ischemic heart disease	24/72 (33.3%)	2.01	1.11–3.64	**0.021**
Heart failure	15/39 (38.5%)	2.72	1.33–5.56	**0.006**
Previous AKI	11/29 (37.9%)	2.85	1.27–6.39	**0.011**
Baseline eGFR <60	25/55 (45.5%)	8.02	4.18–15.38	**<0.001**
Admission SBP <90 mmHg	17/35 (48.6%)	4.22	2.03–8.78	**<0.001**
Dehydration at admission	27/71 (38.0%)	2.96	1.70–5.16	**<0.001**
Sepsis during hospitalization	48/119 (40.3%)	2.91	1.83–4.62	**<0.001**
Hypotension during hospitalization	31/68 (45.6%)	3.83	2.18–6.73	**<0.001**
Nephrotoxic medication exposure	60/211 (28.4%)	2.24	1.37–3.66	**0.001**
NSAID exposure	19/57 (33.3%)	1.99	1.07–3.70	**0.030**
Diuretic therapy	38/126 (30.2%)	1.73	1.07–2.79	**0.025**
ICU transfer	20/49 (40.8%)	2.77	1.46–5.25	**0.002**

Table 10. Independent predictors of incident acute kidney injury on multivariable logistic regression

Predictor	Adjusted OR	95% CI	p-value
Age ≥65 years	1.62	0.94–2.79	0.081
Diabetes mellitus	1.48	0.86–2.55	0.154
Chronic kidney disease	2.71	1.31–5.60	**0.007**
Baseline eGFR <60 mL/min/1.73 m²	4.36	2.12–8.96	**<0.001**
Admission SBP <90 mmHg	2.69	1.18–6.13	**0.018**
Dehydration at admission	2.01	1.10–3.67	**0.023**
Sepsis during hospitalization	2.34	1.40–3.92	**0.001**
Hypotension during hospitalization	2.51	1.35–4.67	**0.004**
Nephrotoxic medication exposure	1.71	1.01–2.91	**0.046**
Heart failure	1.69	0.79–3.62	0.175

Multivariable logistic regression model adjusted for clinically relevant demographic, comorbidity, baseline renal function and in-hospital variables. Statistical significance was considered at p<0.05.

Table 11. Hospital outcomes according to development of acute kidney injury

Outcome	AKI (n=92)	No AKI (n=331)	p-value
Length of hospital stay, days, median (IQR)	9 (6–14)	6 (4–9)	**<0.001**
Hospital stay ≥10 days	39 (42.4%)	65 (19.6%)	**<0.001**
ICU transfer	20 (21.7%)	29 (8.8%)	**0.001**
In-hospital mortality	15 (16.3%)	20 (6.0%)	**0.003**
Discharged alive	77 (83.7%)	311 (94.0%)	—
Discharged with persistent renal dysfunction*	18 (19.6%)	9 (2.7%)	**<0.001**

*Persistent renal dysfunction was defined as serum creatinine remaining above the patient's baseline value at the time of discharge among patients in whom a discharge creatinine measurement was available.

Table 12. Clinical profile according to severity of acute kidney injury

Characteristic	Stage 1 (n=53)	Stage 2 (n=25)	Stage 3 (n=14)	p-value
Age ≥65 years	23 (43.4%)	13 (52.0%)	9 (64.3%)	0.20
Diabetes mellitus	21 (39.6%)	12 (48.0%)	7 (50.0%)	0.60
Chronic kidney disease	9 (17.0%)	7 (28.0%)	6 (42.9%)	**0.049**
Sepsis	24 (45.3%)	15 (60.0%)	9 (64.3%)	0.28
Hypotension	13 (24.5%)	10 (40.0%)	8 (57.1%)	**0.040**
Nephrotoxic medication exposure	32 (60.4%)	17 (68.0%)	11 (78.6%)	0.41
ICU transfer	7 (13.2%)	7 (28.0%)	6 (42.9%)	**0.018**
Kidney replacement therapy	0	1 (4.0%)	6 (42.9%)	**<0.001**
In-hospital mortality	5 (9.4%)	5 (20.0%)	5 (35.7%)	**0.018**
Median hospital stay, days (IQR)	8 (5–11)	11 (7–16)	14 (9–19)	**0.003**

AKI, acute kidney injury; BP, blood pressure; CI, confidence interval; eGFR, estimated glomerular filtration rate; ICU, intensive care unit; IQR, interquartile range; KDIGO, Kidney Disease: Improving Global Outcomes; OR, odds ratio; SBP, systolic blood pressure; SD, standard deviation; WBC, white blood cell. Values in bold indicate statistically significant p-values (p<0.05).

Pictorial depictions

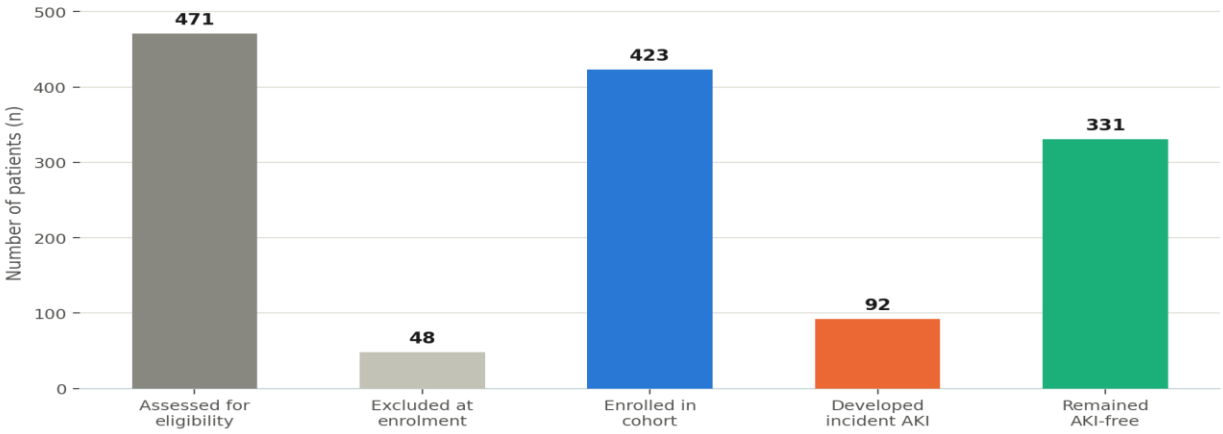

Figure 1. Patient recruitment and cohort disposition

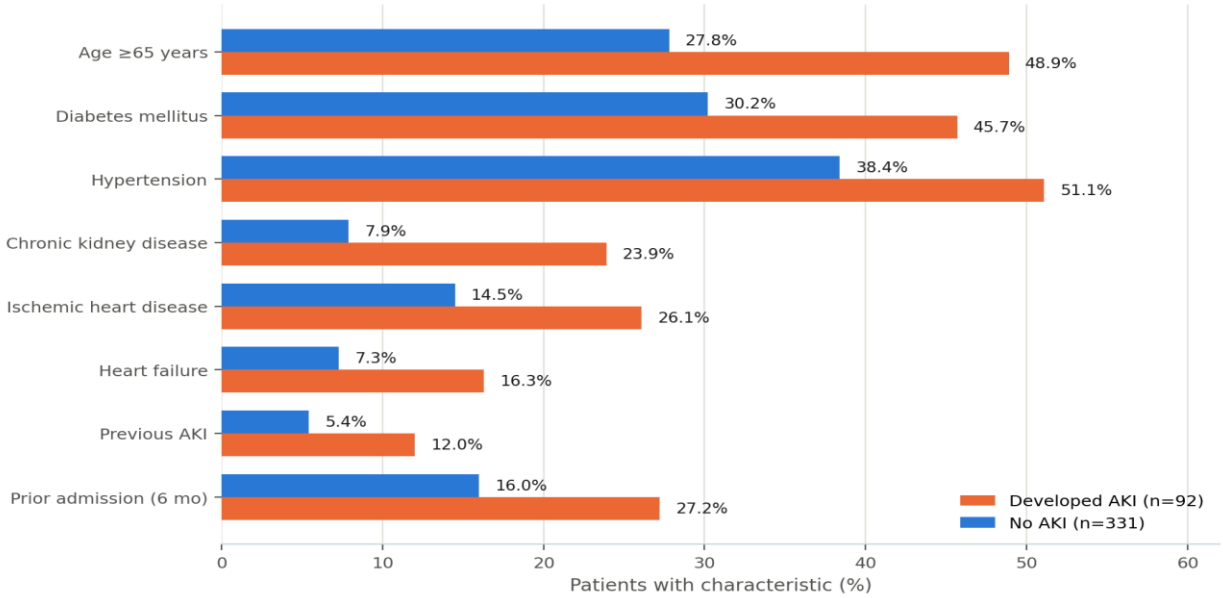

Figure 2. Baseline demographic and comorbid predictors of incident AKI (AKI vs no AKI)

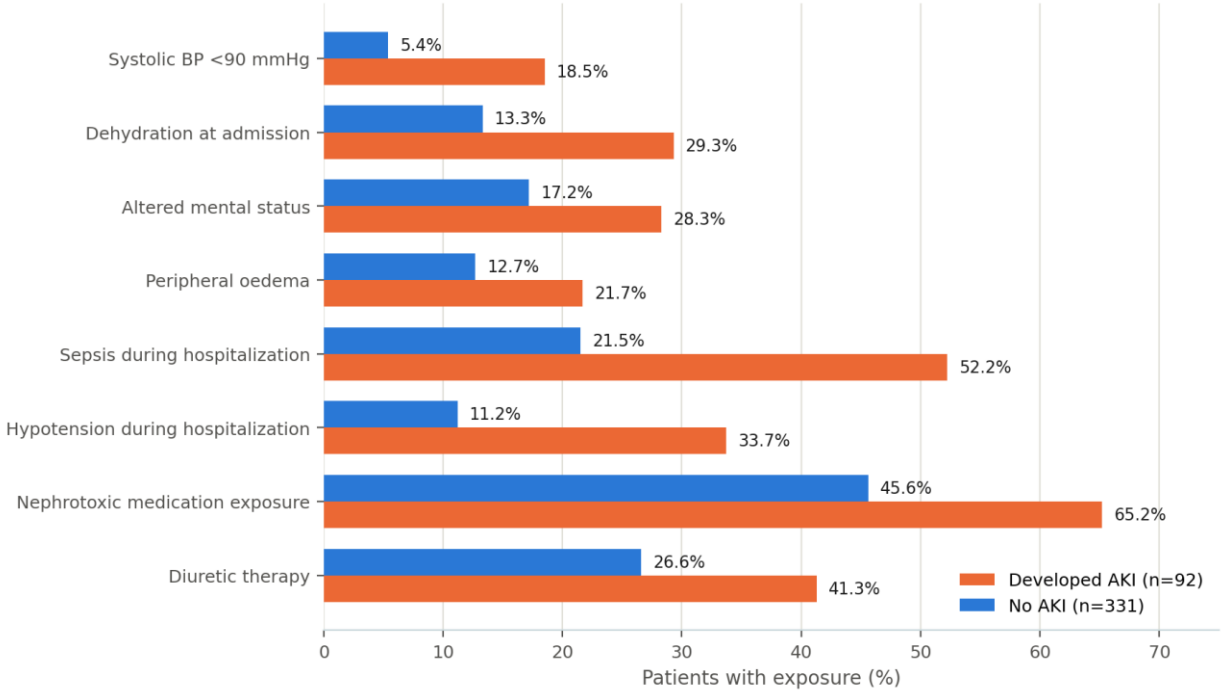

Figure 3. Presenting clinical features and in-hospital exposures (AKI vs no AKI)

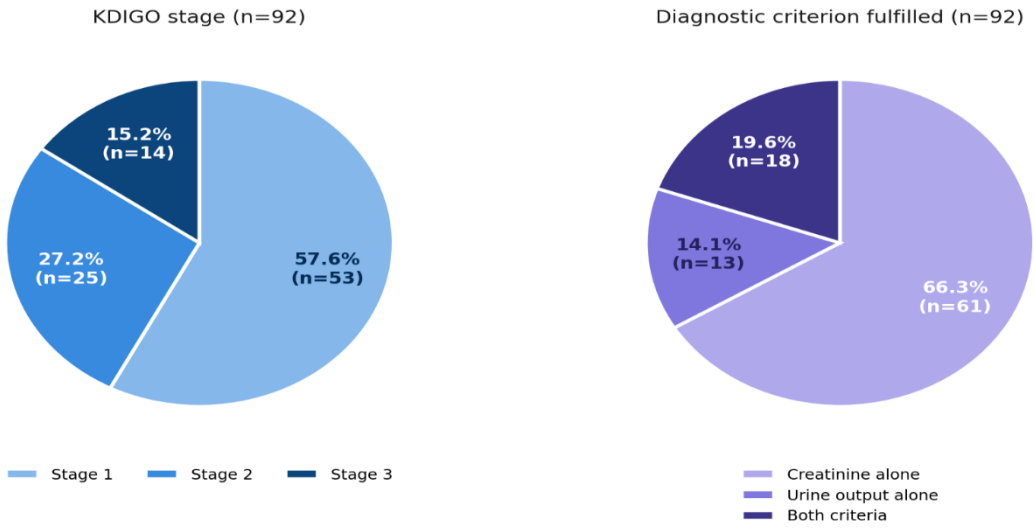

Figure 4. KDIGO severity stage and diagnostic criterion fulfilled among incident AKI cases

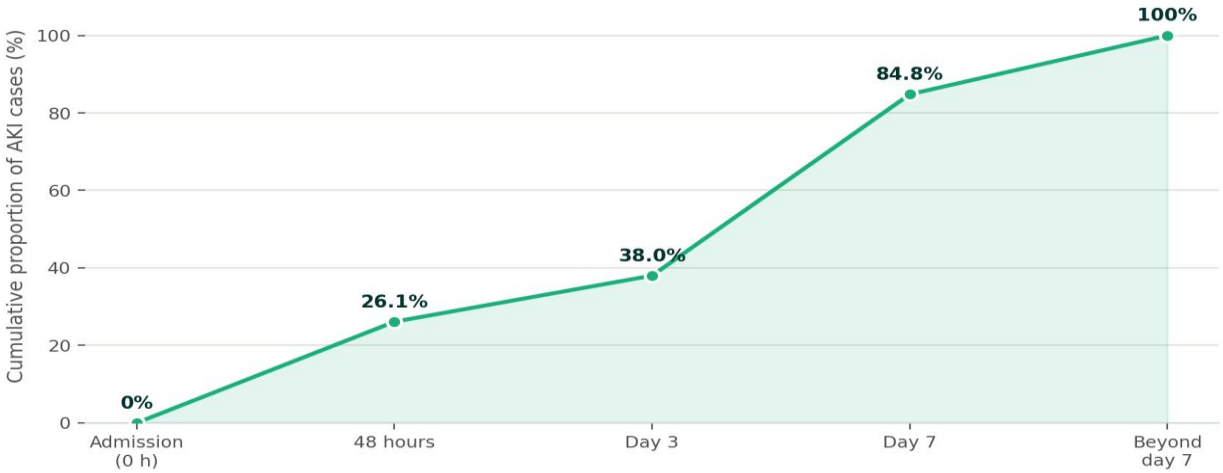

Figure 5. Cumulative time-to-onset of incident AKI

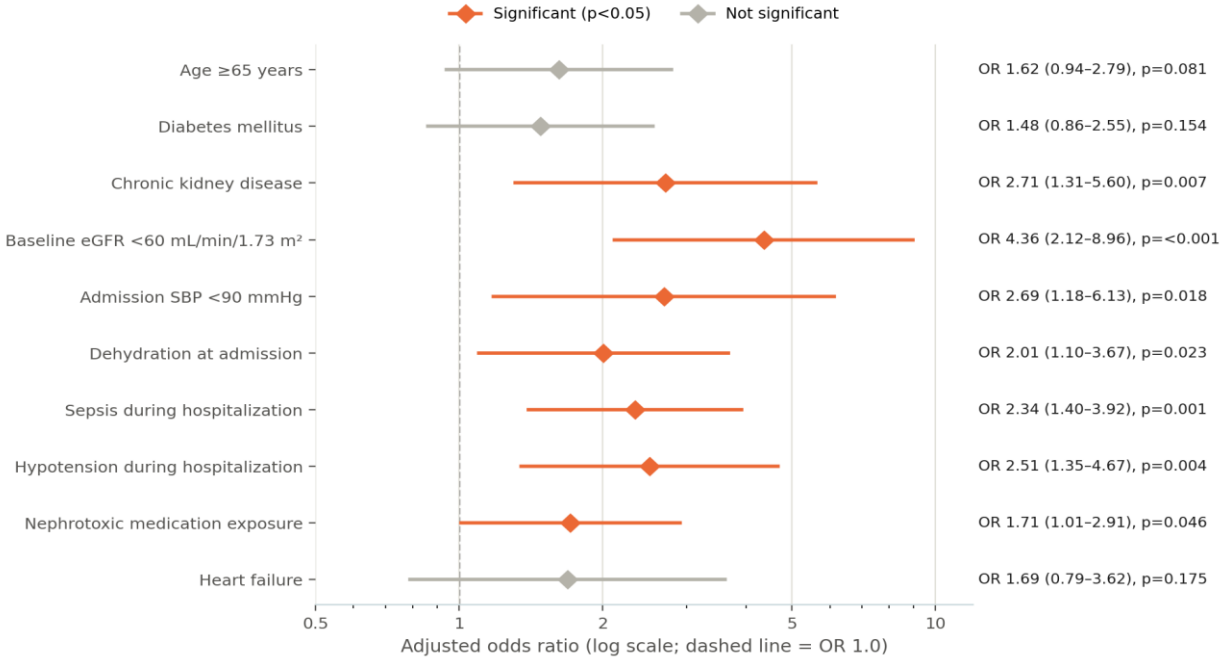

Figure 6. Independent predictors of incident AKI — multivariable logistic regression (forest plot)

Figure 7. Hospital outcomes and length of stay, AKI vs no AKI

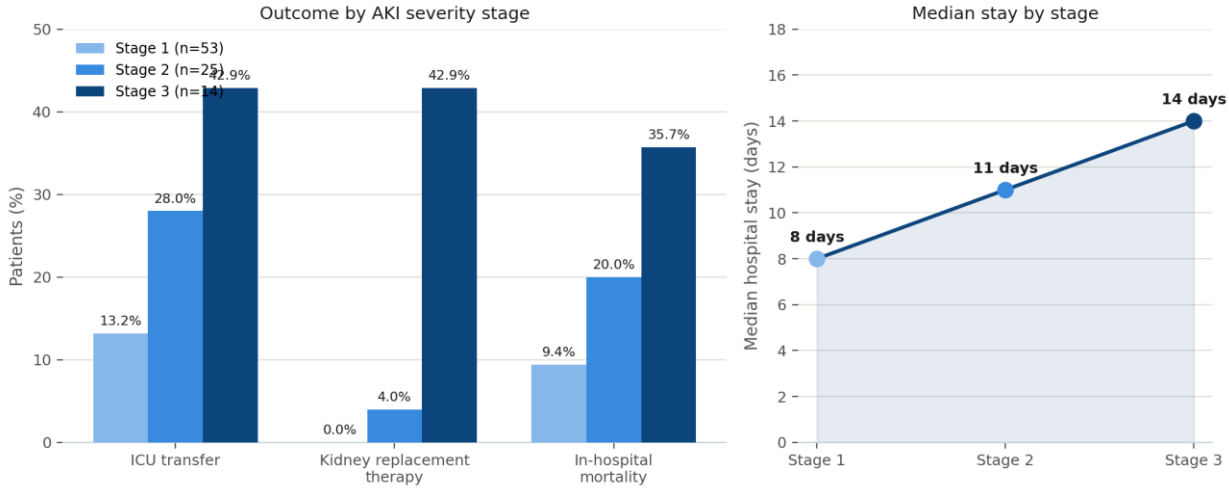

Figure 8. Clinical outcomes and length of stay by AKI severity stage

DISCUSSION

This prospective cohort study found that 92 of 423 adult medical inpatients who were free of AKI at enrolment developed AKI during hospitalization, giving an incidence of 21.7%. The occurrence of AKI was not confined to patients with severe disease at admission. Rather, renal injury developed throughout the hospital stay, with 73.9% of episodes occurring after the first 48 hours and a median time to AKI of 4 days. Patients who developed AKI were older and had a greater burden of diabetes mellitus, hypertension, chronic kidney disease, ischemic heart disease, heart failure and previous AKI. Lower blood pressure, dehydration and altered mental status were also more frequent at admission. Most importantly, after adjustment for potential confounding, chronic kidney disease, baseline eGFR <60 mL/min/1.73 m², admission systolic blood pressure <90 mmHg, dehydration at admission, sepsis, hypotension during hospitalization and exposure to potentially nephrotoxic medications remained independently associated with incident AKI. These findings indicate that AKI among general medical inpatients results from the interaction between reduced renal reserve and acute potentially reversible insults rather than from a single clinical factor.

The incidence of 21.7% observed in this cohort is clinically important and is broadly comparable with the incidence reported in other large studies of hospitalized adults, although direct comparison should be made cautiously because populations and methods differ. In the multicentre cohort of 56,056 hospitalized adults, Takeuchi et al. reported that 23.1% developed AKI during hospitalization.[11] The close similarity between their estimate and our finding is notable despite differences in healthcare systems and study methodology. In contrast, Azevedo et al. reported a much higher frequency of AKI, 52.1%, among 731 adults presenting to a high-complexity public hospital in São Paulo.[4] However, their study included both community-acquired and hospital-acquired AKI, whereas our cohort excluded patients who already fulfilled AKI criteria at admission. Therefore, the lower incidence in our study is not unexpected and more specifically reflects the burden of new-onset AKI among patients who entered hospital without established AKI. This distinction is important when comparing epidemiological estimates because including prevalent AKI at presentation can substantially increase the overall frequency of AKI in hospitalized populations. Cerda et al. have similarly emphasized that differences in case definition, timing of ascertainment, patient characteristics and healthcare setting contribute substantially to the wide variation in reported AKI epidemiology worldwide.[3]

The timing of AKI in our cohort provides an additional important finding. Almost three-quarters of episodes developed after the first 48 hours, while only 26.1% occurred during the first 48 hours. The median time to AKI was 4 days, and 84.8% of episodes occurred within the first 7 days. This pattern supports the relevance of events occurring during hospitalization, rather than considering AKI solely as a manifestation of the presenting illness. Azevedo et al. similarly distinguished community-acquired from hospital-acquired AKI and found that CKD, heart failure, hypotension and edema were associated with hospital-acquired AKI.[4] In our cohort, sepsis, hypotension, dehydration and nephrotoxic medication exposure were particularly frequent among patients who developed AKI. This reinforces the importance of continued renal monitoring after admission, particularly during the early inpatient period when the patient's clinical condition and treatment are rapidly changing. Ostermann et

al. have emphasized that AKI is a dynamic and multifactorial syndrome in hospitalized patients, often arising from the combined effect of patient susceptibility and acute physiological insults.[2]

Pre-existing renal vulnerability was one of the clearest findings in the present study. Known CKD was present in 23.9% of patients who developed AKI compared with 7.9% of those who remained free of AKI, and CKD remained independently associated with incident AKI in the adjusted model. Baseline eGFR <60 mL/min/1.73 m² was also strongly associated with AKI and remained the strongest renal-function-related predictor in the multivariable analysis. These findings are consistent with the concept of reduced renal reserve, whereby patients with pre-existing renal impairment have less capacity to tolerate additional haemodynamic, infectious or toxic insults. Azevedo et al. identified CKD as an independent risk factor for both community-acquired and hospital-acquired AKI in their prospective cohort, while Takeuchi et al. also demonstrated that patients who developed AKI had substantially lower baseline eGFR than those without AKI.[4,11] The consistency of these findings across different populations strengthens the observation that baseline kidney function should be considered an important part of AKI risk assessment at admission.

The higher frequency of diabetes, hypertension, ischemic heart disease and heart failure among patients with AKI further supports the role of underlying comorbidity. On univariable analysis, each of these conditions was associated with incident AKI, although diabetes and heart failure did not remain statistically significant in the final multivariable model. This attenuation after adjustment suggests that their effect may partly operate through other factors such as reduced renal reserve, haemodynamic instability and severity of acute illness. Takeuchi et al. found diabetes to be independently associated with incident AKI in a large multicentre cohort, although the magnitude of association was modest after adjustment.[11] Similarly, Azevedo et al. found heart failure to be associated with hospital-acquired AKI.[4] The differences between individual studies are understandable because comorbidities often cluster together and their apparent effect can change when baseline renal function and acute illness are considered simultaneously.

Age showed a similar pattern. Patients who developed AKI were considerably older than those who did not, and age ≥65 years was significantly associated with AKI on univariable analysis. However, the association was attenuated after adjustment and did not remain statistically significant. This suggests that older age may function primarily as a marker of accumulated vulnerability rather than as an independent determinant in itself. Older patients are more likely to have CKD, cardiovascular disease, impaired physiological reserve and polypharmacy, all of which can increase susceptibility to acute renal insults. Azevedo et al. identified age as an independent determinant of community-acquired AKI, while the large cohort reported by Takeuchi et al. also showed a higher mean age among patients who developed AKI.[4,11] The lack of independent significance of age in our final model may therefore reflect the fact that some of its effect was captured by more proximal clinical and renal risk factors.

Hypotension and dehydration were among the most clinically relevant findings in our study. Patients with admission systolic blood pressure below 90 mmHg had substantially higher odds of developing AKI, and admission hypotension remained independently associated with the outcome. Dehydration at admission was similarly associated with incident AKI after adjustment. These findings are clinically plausible because impaired renal perfusion is an important pathway through which volume depletion and haemodynamic instability can precipitate kidney injury, particularly in patients with limited baseline renal reserve. Azevedo et al. reported dehydration in 29% and hypotension in 15% of patients at admission and identified hypotension as an independent risk factor for hospital-acquired AKI.[4] In the Pakistani setting, Arshad and Ayaz found several potentially preventable contributors among patients with AKI, including diarrhoeal illness and other causes of volume depletion.[9] Our findings therefore have direct practical relevance to general medical wards, where early recognition of dehydration and hypotension and timely correction of reversible haemodynamic abnormalities may represent realistic opportunities to reduce AKI risk.

Sepsis was another major determinant in the present cohort. Sepsis occurred in 52.2% of patients who developed AKI compared with 21.5% of those who did not, and it remained independently associated with incident AKI after adjustment. This finding is in agreement with the established relationship between systemic infection and renal injury. Arshad and Ayaz, in a Pakistani tertiary-care study, reported that sepsis was present in approximately 45% of patients with AKI, while Naqvi's long-term Pakistani experience also demonstrated the important role of infectious causes in the changing epidemiology of medical AKI.[9,8] More recently, Prasad et al., using data from the ISN-AKI registry in India, demonstrated that infection-related illness remained an important component of AKI presentation in South Asia.[7] The high frequency of sepsis in the present study is particularly relevant to tertiary-care hospitals in Pakistan, where patients often present with advanced infection and multiple coexisting physiological disturbances. Although our observational design does not allow us to conclude that sepsis directly

caused the AKI in every case, the persistence of the association after adjustment supports its importance as a clinical risk marker and potential target for early intervention.

Exposure to potentially nephrotoxic medication was also independently associated with incident AKI. In our cohort, nearly half of all patients received at least one potentially nephrotoxic medication, and exposure was more frequent among those who subsequently developed AKI. NSAIDs, aminoglycosides and vancomycin were individually more common in the AKI group, although the adjusted analysis focused on overall nephrotoxic medication exposure. These findings are consistent with previous evidence. In a cohort of 11,311 adults, Goldstein and colleagues found that high nephrotoxin exposure was associated with subsequent AKI, including more severe AKI, even after attempts to account for confounding.[12] In a more recent cohort of patients with drug-induced AKI, Garcia et al. reported that vancomycin was the most frequently implicated nephrotoxin and emphasized the importance of early recognition and medication adjustment.[13] The present finding should nevertheless be interpreted with caution because treatment decisions are influenced by the severity and nature of the underlying illness. Therefore, nephrotoxic exposure may partly represent confounding by indication rather than a completely independent drug effect. Even with this limitation, medication review, renal dose adjustment and avoidance of unnecessary nephrotoxic combinations remain reasonable preventive measures, particularly in patients with CKD, dehydration or haemodynamic instability.

The severity pattern of AKI in our cohort was predominantly mild, with Stage 1 accounting for 57.6% of cases, followed by Stage 2 in 27.2% and Stage 3 in 15.2%. Although most episodes were Stage 1, the clinical consequences of AKI were considerable. Patients with AKI had a longer median hospital stay than those without AKI, were more likely to require ICU transfer, had higher in-hospital mortality and were more likely to leave hospital with persistent renal dysfunction. These findings are consistent with the broader literature showing that AKI is associated with adverse short-term outcomes even when the initial episode is not severe. Takeuchi et al. reported AKI in 23% of hospitalized adults and found that patients with AKI had longer hospital stays than those without AKI; they also demonstrated that non-recovery of kidney function at discharge remained an important clinical outcome among survivors.[11] In Azevedo et al.'s prospective cohort, in-hospital mortality was 25.2% among patients with AKI compared with 11.1% among those without AKI.[4] The corresponding mortality difference in our cohort was smaller in absolute terms, but the direction of association was similar, with mortality of 16.3% versus 6.0% among patients with and without AKI, respectively. The consistency of this association across different healthcare settings supports the importance of AKI as a clinically meaningful complication rather than simply a laboratory abnormality.

Our analysis according to AKI severity further demonstrated a stepwise increase in adverse outcomes. Patients with Stage 3 AKI had the highest rates of ICU transfer, kidney replacement therapy and mortality and had the longest hospital stay. Six of the seven patients who required kidney replacement therapy had Stage 3 AKI, and mortality increased from 9.4% in Stage 1 to 35.7% in Stage 3. Similar severity-dependent relationships have been described in large hospitalized cohorts, including the study by Takeuchi et al., in which increasing AKI severity was associated with poorer renal recovery.[5] These findings are clinically important because Stage 1 AKI represented the largest proportion of cases in our study. Early recognition at this stage may provide an opportunity to prevent progression to more severe renal injury, greater resource utilization and adverse outcomes.

The finding that 19.6% of patients with AKI had persistent renal dysfunction at discharge is also noteworthy. This is consistent with the growing recognition that AKI does not necessarily resolve completely before hospital discharge and may represent an important transition point toward subsequent CKD. Takeuchi et al. reported non-recovery in 30.8% of AKI survivors in their large multicentre cohort, although differences in AKI ascertainment and definitions of recovery make direct numerical comparison inappropriate.[5] The relatively lower proportion in our study may partly reflect the shorter follow-up available in an inpatient cohort and the fact that discharge creatinine measurements were not available for every patient. Nevertheless, the finding highlights the need for attention to renal function at discharge rather than considering the episode resolved once the acute illness has improved.

Taken together, the present findings suggest that incident AKI in medical wards is potentially amenable to earlier recognition. Patients with CKD or reduced baseline eGFR represent an identifiable high-risk group, while hypotension, dehydration and sepsis are clinically apparent factors that can be monitored during routine care. Nephrotoxic medication exposure is another area where medication review may be useful. The fact that most episodes occurred after the first 48 hours further supports continued renal surveillance during hospitalization rather than limiting assessment to the admission creatinine value. In a resource-constrained setting, such measures may be particularly relevant because they rely largely on careful clinical assessment, timely laboratory monitoring and rational medication prescribing rather than expensive diagnostic technologies.

STRENGTHS

The prospective design is an important strength of this study. By excluding patients who had AKI at enrolment and following participants from an AKI-free baseline, the study specifically assessed incident AKI and established a clearer temporal relationship between baseline characteristics, in-hospital exposures and subsequent renal deterioration. The study was also conducted among general medical inpatients rather than a selected nephrology or intensive care population, thereby providing a more representative picture of AKI risk in routine medical practice at a tertiary-care public hospital. In addition, the use of KDIGO criteria and assessment of both creatinine and urine-output criteria allowed a standardized definition of AKI.

LIMITATIONS

Several limitations should be considered when interpreting these findings. First, this was a single-centre study conducted at a tertiary-care hospital, and the results may not be directly generalizable to other hospitals or regions of Pakistan. Second, serum creatinine was repeated according to clinical requirements and routine inpatient practice rather than at fixed research-defined intervals. This may have resulted in under-detection of short-lived or mild episodes, particularly among patients who underwent fewer repeat measurements. Third, urine-output monitoring was dependent on reliable measurement in a general ward setting, and this may have led to under-recognition of urine-output-defined AKI. Fourth, despite prospective data collection, this remains an observational study and therefore the reported associations should not be interpreted as proof of causation. This is particularly relevant to nephrotoxic medication exposure and ICU transfer, both of which may be influenced by the severity of the underlying illness. Finally, baseline renal function was primarily assessed using admission creatinine, with previous measurements considered when available; therefore, some degree of misclassification of underlying CKD or baseline renal reserve cannot be excluded.

CONCLUSION

In this prospective cohort of adult medical inpatients who were free of AKI at admission, incident AKI developed in approximately one-fifth of patients during hospitalization. Reduced baseline renal function, chronic kidney disease, admission hypotension, dehydration, sepsis, hypotension during hospitalization, and exposure to potentially nephrotoxic medications were independently associated with the development of AKI. AKI was also associated with longer hospital stay, greater need for intensive care, higher in-hospital mortality, and persistent renal dysfunction at discharge.

These findings highlight the importance of identifying patients at increased risk of AKI at the time of admission and maintaining close monitoring throughout hospitalization, particularly for changes in haemodynamic status, hydration, infection and medication exposure. Early recognition of these risk factors may provide practical opportunities to reduce the development and progression of AKI among general medical inpatients. Further multicentre prospective studies in Pakistan are needed to confirm these findings and to evaluate preventive strategies aimed at reducing the burden and adverse outcomes of AKI.

Additional information

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Authors contribution

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