

Incidence, Risk Factors, and Economic Burden of Drug-Induced Acute Kidney Injury in a Tertiary Care Hospital

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Abstract

Original Research Article

Background: The development of DI-AKI is influenced by patient characteristics and the underlying condition of the kidneys, in addition to exposure to nephrotoxic medications. Therefore, this study aimed to determine the incidence, risk factors, clinical outcomes, and economic burden of drug-induced acute kidney injury among hospitalized patients. **Methods:** This prospective observational study was conducted in the Departments of Nephrology and Medicine, Mugda Medical College Hospital, Dhaka, Bangladesh, from January 2026 to June 2026, among 246 hospitalized patients. Demographic, clinical, medication, renal outcome, and cost data were collected and analyzed using SPSS. Drug-induced AKI, risk factors, clinical outcomes, and economic burden were assessed using the chi-square test, with $p < 0.05$ considered significant. **Results:** Among 246 patients, D-AKI occurred in 62 (25.2%), predominantly as Stage 1 (50.0%). Complete renal recovery occurred in 77.4%, while 11.3% required renal replacement therapy and 9.7% died. Antibiotics were the leading implicated drugs (40.3%), followed by diuretics (29.0%) and ACE inhibitors/ARBs (24.2%). Older age, comorbidities, polypharmacy, and nephrotoxic drug exposure were significantly associated with D-AKI ($p < 0.05$). D-AKI was also associated with longer hospitalization (14.2 vs. 8.1 days) and higher total costs (BDT 45,000 vs. 26,800; $p < 0.001$). **Conclusion:** Drug-induced AKI is a significant and potentially preventable complication associated with multiple risk factors, prolonged hospitalization, and increased healthcare costs, underscoring the need for rational prescribing and early renal monitoring.

Keywords: Drug-Induced Acute Kidney Injury, Incidence, Risk Factors, Clinical Outcomes, Economic Burden.

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INTRODUCTION

Acute Kidney Injury (AKI) is a frequently encountered clinical syndrome that involves an abrupt decline in glomerular filtration rate (GFR), which may lead to significant disturbances in blood volume and acid-base balance [1]. AKI occurs commonly among hospitalized patients, with reported incidence reaching up to 22%, while its occurrence may be as high as 65% among critically ill patients [2]. The development of AKI can result in a range of short- and long-term complications, including prolonged hospital length of stay (LOS), requirement for renal replacement therapy (RRT), increased risk of developing or progressing to chronic kidney disease (CKD), impaired health-related

quality of life, and substantially increased long-term mortality [3]. Furthermore, AKI is associated with considerable morbidity and mortality, increased likelihood of CKD development or progression, and greater healthcare costs [4].

AKI has a multifactorial etiology, with drugs representing one of its important causes and accounting for approximately 30% of AKI cases among hospitalized patients [5,6]. Drug-Induced Acute Kidney Injury (DI-AKI) is a clinical condition that occurs in association with exposure to potentially nephrotoxic medications and is particularly prevalent in hospitalized patients [7]. Although exposure to nephrotoxic medications is necessary for the development of DI-AKI, the

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nephrotoxic potential of a drug alone does not determine the occurrence of renal injury [7]. The underlying pathophysiological mechanisms of DI-AKI include Acute Tubular Necrosis (ATN), Acute Interstitial Nephritis (AIN), and Crystal-Induced Nephropathy. Different medications may produce renal injury through distinct mechanisms; for example, NSAIDs may decrease renal blood flow, promote tubular obstruction through crystal deposition, and cause direct cytotoxicity or cell-mediated immune injury, thereby contributing to AKI.

The development of DI-AKI is also influenced by characteristics of the patient and the underlying condition of the kidneys, in addition to exposure to nephrotoxic medications [7]. Extended exposure to nephrotoxic medications and concurrent administration of multiple nephrotoxic drugs may further increase the likelihood of renal injury. Individuals at greater risk of nephrotoxicity include elderly women aged over 65 years, particularly when CKD or cirrhosis is present [7]. Older age 5,6 and pre-existing CKD have also been associated with the development of AKI during NSAID exposure, with previous studies indicating that patients with abnormal baseline renal function have a 3–4-fold greater risk of deterioration in renal function than those with normal baseline renal function [8]. In addition to its initial renal effects, AKI may produce substantial short- and long-term consequences, including extended hospital LOS, requirement for RRT, increased risk of CKD progression, reduced health-related quality of life, and considerably higher long-term mortality [9].

The clinical consequences of AKI also translate into a substantial economic burden for hospitalized patients and healthcare systems. In the United States, approximately 498,000 patients diagnosed with AKI are discharged from hospitals annually, contributing to an estimated aggregate annual hospital cost of \$4.7 to \$24.0 billion [10]. The financial burden becomes greater with increasing severity of kidney injury, requirement for dialysis, and failure to achieve kidney recovery, with hospitalization costs reported to be \$11,016 to \$42,077 higher than those for patients without AKI [11]. Despite the considerable costs associated with AKI, the economic consequences specifically attributable to drug-associated AKI (D-AKI), including its longer-term effects, remain insufficiently characterized. In addition, adverse effects related to NSAIDs impose a substantial economic burden on both individual patients and healthcare systems.

Despite the recognized contribution of medications to AKI, limited evidence is available regarding the incidence, associated risk factors, clinical outcomes, and economic burden of drug-induced AKI, particularly in resource-limited healthcare settings. Therefore, this study aimed to determine the incidence, risk factors, clinical outcomes, and economic burden of

drug-induced acute kidney injury among hospitalized patients.

Objective

- To determine the incidence, risk factors, clinical outcomes, and economic burden of drug-induced acute kidney injury among hospitalized patients.

METHODOLOGY & MATERIALS

This prospective observational study was conducted in the Departments of Nephrology and Medicine, Mugda Medical College Hospital, Dhaka, Bangladesh, from January 2026 to June 2026. A total of 246 hospitalized patients were included according to predefined inclusion criteria. Data were collected to determine the incidence, associated factors, clinical outcomes, and economic burden of drug-induced acute kidney injury.

Inclusion Criteria

- Hospitalized patients aged ≥ 18 years.
- Patients admitted to the Departments of Nephrology and Medicine during the study period.
- Patients who received one or more medications during hospitalization.
- Patients with sufficient clinical, laboratory, medication, and outcome data for assessment of AKI and its potential drug-related cause.
- Patients who provided informed consent to participate in the study.

Exclusion Criteria

- Patients with AKI clearly attributable to non-drug-related causes.
- Patients with established end-stage kidney disease or receiving maintenance dialysis.
- Patients with incomplete or insufficient clinical or laboratory records.
- Patients discharged or transferred before adequate assessment of renal outcomes.
- Patients who declined to participate in the study.

Data Collection

Data were collected using a structured data collection form through patient interviews, clinical examination, medication records, and hospital records. Demographic characteristics, residence, comorbidities, sepsis/infection, medication history, and polypharmacy (≥ 5 drugs) were recorded. Relevant clinical and laboratory data were monitored during hospitalization to identify and characterize AKI.

Assessment of Drug-Induced AKI

AKI was identified based on changes in serum creatinine and clinical assessment during hospitalization. Drug-induced AKI was considered when AKI developed following exposure to a potentially nephrotoxic or hemodynamically active medication and alternative causes were excluded based on clinical and laboratory

findings. The severity of AKI was categorized into Stage 1, Stage 2, and Stage 3 according to KDIGO guideline. The implicated drug classes, including antibiotics, diuretics, ACE inhibitors/ARBs, NSAIDs, antidiabetic drugs, contrast agents, antineoplastic drugs, and other medications, were documented.

Assessment of Associated Factors and Clinical Outcomes

Potential factors associated with drug-induced AKI included age, sex, hypertension, diabetes mellitus, chronic kidney disease, sepsis/infection, polypharmacy, NSAID exposure, diuretic exposure, and ACE inhibitor/ARB exposure. Clinical outcomes were assessed in terms of renal replacement therapy requirement, degree of renal recovery, and in-hospital mortality.

Assessment of Economic Burden

Healthcare resource utilization was assessed by recording the duration of hospitalization and costs

associated with medications, investigations, hospital/bed services, and total hospitalization. Costs were expressed in Bangladeshi Taka (BDT), and economic outcomes were compared between patients with and without drug-induced AKI.

Statistical Analysis

Data were entered, coded, and analyzed using SPSS software. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were presented as frequency and percentage. Associations between drug-induced AKI and categorical demographic, clinical, and medication-related factors were assessed using the chi-square test. Differences in continuous economic parameters between patients with and without drug-induced AKI were also assessed using an appropriate statistical test. A p-value of <0.05 was considered statistically significant.

RESULTS

Table 1: Demographic and Clinical Characteristics of the Study Population (n = 246)

Variable		Frequency (n)	Percentage (%)
Age group (years)	≤ 40	52	21.1
	41–60	91	37.0
	>60	103	41.9
Sex	Male	142	57.7
	Female	104	42.3
Residence	Urban	151	61.4
	Rural	95	38.6
Hypertension		96	39.0
Diabetes mellitus		74	30.1
Chronic kidney disease		38	15.4
Heart disease		46	18.7
Sepsis/infection		83	33.7
Polypharmacy (≥ 5 drugs)		128	52.0

The majority of patients were aged >60 years (103 patients, 41.9%), followed by those aged 41–60 years (91 patients, 37.0%) and ≤ 40 years (52 patients, 21.1%), with a mean age of 56.8 ± 16.7 years. Male patients predominated (142 patients, 57.7%), and most participants were from urban areas (151 patients, 61.4%). Hypertension was present in 96 patients (39.0%), while

74 patients (30.1%) had diabetes mellitus. Chronic kidney disease and heart disease were present in 38 (15.4%) and 46 (18.7%) patients, respectively. Sepsis or infection was observed in 83 patients (33.7%), while polypharmacy involving ≥ 5 drugs was reported in 128 patients (52.0%).

Table 2: Incidence, Severity, and Clinical Outcomes of Drug-Induced Acute Kidney Injury (n = 246)

Variable		Frequency (n)	Percentage (%)
Drug-induced AKI	Present	62	25.2
	Absent	184	74.8
Severity of drug-induced AKI (n = 62)	Stage 1	31	50.0
	Stage 2	19	30.6
	Stage 3	12	19.4
Renal replacement therapy required	Yes	7	11.3
Renal recovery	Complete recovery	48	77.4
	Partial recovery	9	14.5
	No recovery	5	8.1
Mortality	Death	6	9.7

Drug-induced acute kidney injury was identified in 62 patients (25.2%), while 184 patients (74.8%) did not develop drug-induced AKI. Among the 62 patients with drug-induced AKI, Stage 1 was the most common severity category (31 patients, 50.0%), followed by Stage 2 (19 patients, 30.6%) and Stage 3 (12

patients, 19.4%). Renal replacement therapy was required in 7 patients (11.3%). Complete renal recovery occurred in 48 patients (77.4%), while 9 patients (14.5%) had partial recovery and 5 patients (8.1%) had no recovery. Mortality was observed in 6 patients (9.7%).

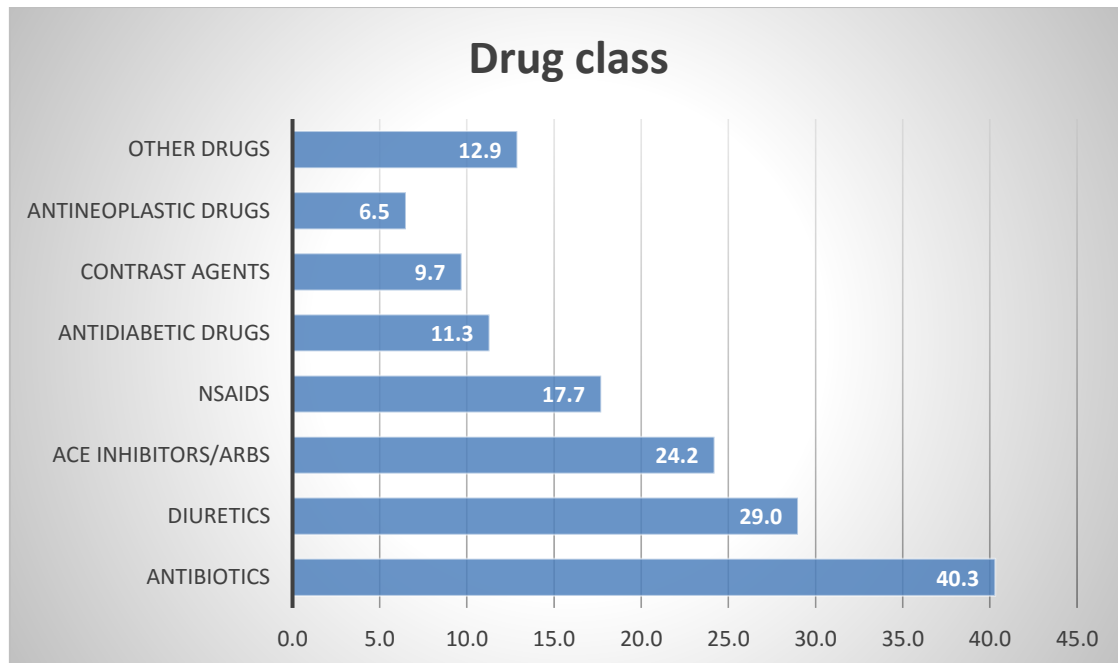


Figure 1: Distribution of Drug Classes Implicated in Drug-Induced Acute Kidney Injury (n = 62)

Among patients with drug-induced AKI, antibiotics were the most frequently implicated drug class (25 patients, 40.3%), followed by diuretics (18 patients, 29.0%) and ACE inhibitors/ARBs (15 patients, 24.2%). NSAIDs were implicated in 11 patients (17.7%),

while antidiabetic drugs, contrast agents, antineoplastic drugs, and other drugs were implicated in 7 (11.3%), 6 (9.7%), 4 (6.5%), and 8 (12.9%) patients, respectively. Percentages exceed 100% because some patients were exposed to more than one potentially nephrotoxic drug.

Table 3: Association Between Selected Risk Factors and Drug-Induced Acute Kidney Injury (n = 246)

Risk factor	D-AKI Present (n=62), n (%)	D-AKI Absent (n=184), n (%)	P-value
Age >60 years	36 (58.1)	67 (36.4)	0.003
Male sex	39 (62.9)	103 (56.0)	0.337
Hypertension	34 (54.8)	62 (33.7)	0.004
Diabetes mellitus	25 (40.3)	49 (26.6)	0.045
Chronic kidney disease	19 (30.6)	19 (10.3)	<0.001
Sepsis/infection	29 (46.8)	54 (29.3)	0.012
Polypharmacy (≥5 drugs)	43 (69.4)	85 (46.2)	0.002
NSAID exposure	16 (25.8)	18 (9.8)	0.002
Diuretic exposure	25 (40.3)	32 (17.4)	<0.001
ACE inhibitor/ARB exposure	21 (33.9)	32 (17.4)	0.007

Patients aged >60 years had a significantly higher frequency of drug-induced AKI compared with those without drug-induced AKI (36 patients, 58.1% vs. 67 patients, 36.4%; $p = 0.003$). Significant associations were also observed with hypertension (54.8% vs. 33.7%; $p = 0.004$), diabetes mellitus (40.3% vs. 26.6%; $p = 0.045$), chronic kidney disease (30.6% vs. 10.3%; $p < 0.001$), and sepsis/infection (46.8% vs. 29.3%; $p =$

0.012). Polypharmacy (69.4% vs. 46.2%; $p = 0.002$), NSAID exposure (25.8% vs. 9.8%; $p = 0.002$), diuretic exposure (40.3% vs. 17.4%; $p < 0.001$), and ACE inhibitor/ARB exposure (33.9% vs. 17.4%; $p = 0.007$) were also significantly associated with drug-induced AKI. Male sex was not significantly associated with drug-induced AKI ($p = 0.337$).

Table 4: Economic Burden and Hospital Resource Utilization Associated with Drug-Induced Acute Kidney Injury (n = 246)

Economic parameter	D-AKI (n = 62)	No D-AKI (n = 184)	p-value
Length of hospital stay (days)	14.2 ± 6.8	8.1 ± 4.2	<0.001
Medication cost (BDT)	18,450 ± 7,620	11,280 ± 5,140	<0.001
Investigation cost (BDT)	9,850 ± 4,210	5,920 ± 2,830	<0.001
Hospital/bed cost (BDT)	16,700 ± 7,430	9,600 ± 4,850	<0.001
Total hospitalization cost (BDT)	45,000 ± 15,800	26,800 ± 10,200	<0.001
Renal replacement therapy, n (%)	7 (11.3)	0 (0.0)	<0.001

Patients with drug-induced AKI had a significantly longer mean hospital stay than those without drug-induced AKI (14.2 ± 6.8 vs. 8.1 ± 4.2 days; $p < 0.001$). The mean medication cost was also higher among patients with drug-induced AKI (BDT 18,450 ± 7,620 vs. BDT 11,280 ± 5,140; $p < 0.001$), as were investigation costs (BDT 9,850 ± 4,210 vs. BDT 5,920 ± 2,830; $p < 0.001$) and hospital/bed costs (BDT 16,700 ± 7,430 vs. BDT 9,600 ± 4,850; $p < 0.001$). Consequently, the mean total hospitalization cost was significantly higher among patients with drug-induced AKI than among those without (BDT 45,000 ± 15,800 vs. BDT 26,800 ± 10,200; $p < 0.001$). Renal replacement therapy was required in 7 patients (11.3%) with drug-induced AKI, compared with none of the patients without drug-induced AKI ($p < 0.001$).

DISCUSSION

In this prospective observational study conducted at Mugda Medical College Hospital, a notable proportion of hospitalized patients developed drug-induced acute kidney injury, with Stage 1 being the most common severity category. Older age, hypertension, diabetes mellitus, chronic kidney disease, sepsis/infection, polypharmacy, and exposure to NSAIDs, diuretics, and ACE inhibitors/ARBs were significantly associated with drug-induced AKI, while affected patients experienced greater healthcare resource utilization and hospitalization costs, highlighting its important clinical and economic burden among hospitalized patients.

The present study demonstrated that drug-induced acute kidney injury (D-AKI) represents an important clinical problem among hospitalized patients, with a substantial burden of comorbidity and medication exposure in the study population. Older adults predominated, with 41.9% of participants aged >60 years and a mean age of 56.8 ± 16.7 years, broadly consistent with the older age profile reported by Rey *et al.*, [12], who observed a mean age of 72.1 ± 14.6 years among hospitalized patients with D-AKI. The somewhat younger age distribution in the present cohort may reflect differences in study populations, healthcare settings, and inclusion criteria. Male patients accounted for 57.7% of the present cohort, comparable to the male predominance reported by Laville *et al.*, [13]. Comorbidities were also frequent, including hypertension (39.0%), diabetes mellitus (30.1%), CKD (15.4%), and heart disease

(18.7%), highlighting the clinical vulnerability of patients exposed to potentially nephrotoxic medications. This is consistent with Rey *et al.*, [12], who identified CKD as an independent risk factor for D-AKI, and with Laville *et al.*, [13], who reported increased risk of drug-related AKI among patients with CKD, diabetes, cardiovascular disease, and reduced kidney function. Polypharmacy was particularly common in the present study, affecting 52.0% of participants, consistent with Laville *et al.*, [13], who reported that approximately 80% of their CKD cohort received at least five medications and identified polypharmacy as an important factor associated with drug-related AKI. The relatively high prevalence of sepsis or infection (33.7%) further reflects the presence of acute systemic stressors that may increase susceptibility to renal injury in patients receiving multiple medications. Collectively, these findings support the established observation that advanced age, pre-existing kidney disease, comorbidity burden, acute illness, and polypharmacy frequently coexist in patients vulnerable to drug-induced renal injury.

Drug-induced AKI was identified in 25.2% (62/246) of the present study population, indicating a substantial contribution of medication exposure to AKI. This proportion was lower than the 37.5% reported by Liu *et al.*, [14] among hospital-acquired AKI cases across 23 academic hospitals in China, although both studies demonstrate that drug exposure constitutes an important contributor to AKI in hospitalized patients. The severity distribution in the present study was characterized by a predominance of Stage 1 AKI (50.0%), followed by Stage 2 (30.6%) and Stage 3 (19.4%). This pattern was broadly consistent with Liu *et al.*, [14], who reported Stage 1 as the predominant category (72.4%), although the present cohort had comparatively larger proportions of Stage 2 and Stage 3 disease. Similarly, Iavecchia *et al.*, [15] reported predominance of the less severe RIFLE categories, with Risk and Injury accounting for most drug-related episodes and Failure representing approximately one-fifth, closely paralleling the severity distribution observed in the present study. Renal outcomes in the present cohort were comparatively favorable, with complete recovery occurring in 77.4% of patients, compared with 45.7% in Liu *et al.*, [14] and 62.1% in Iavecchia *et al.*, [15]. Partial recovery occurred in 14.5%, while only 8.1% had no recovery, compared with 19.4% and 34.9%, respectively, in the

corresponding categories reported by Liu *et al.*, [14] and 25.5% and 12.4% in Iavecchia *et al.*, [15]. Renal replacement therapy was required in 11.3% of patients, somewhat higher than the 4.1% reported by Iavecchia *et al.*, [15]. In-hospital mortality was 9.7%, lower than the 13.9% reported by Liu *et al.*, [14] and substantially lower than the 22.4% reported by Iavecchia *et al.*, [15]. These differences may reflect variation in baseline characteristics, severity of AKI, underlying illness, implicated medications, timing of recognition, and management strategies across study settings. Nevertheless, the predominance of less severe AKI and the high rate of complete renal recovery in the present study suggest that early recognition and appropriate management of drug-related renal injury may be associated with favorable renal outcomes.

The distribution of implicated medications further demonstrated the multifactorial nature of D-AKI in the present cohort. Antibiotics were the most frequently implicated drug class (40.3%), followed by diuretics (29.0%), ACE inhibitors/ARBs (24.2%), and NSAIDs (17.7%). These findings are broadly consistent with Cui *et al.*, [16], who evaluated 228 patients with D-AKI and identified antimicrobial agents as the leading contributors (32.5%), followed by NSAIDs (27.6%). Although the proportion of antibiotic-associated cases was higher in the present study, both studies emphasize the prominent role of antimicrobial exposure in drug-related renal injury. Cui *et al.*, [16] also identified renin-angiotensin system inhibitors and anticancer agents among the implicated medications, corresponding with the range of drug classes observed in the present cohort. Similarly, Kunitsu *et al.*, [17], in a large French CKD cohort, identified diuretics and renin-angiotensin system inhibitors as important medication-related factors associated with AKI, supporting the relatively high frequencies of diuretic and ACE inhibitor/ARB exposure observed in the present study. Antidiabetic drugs, contrast agents, antineoplastic drugs, and other medications accounted for smaller proportions of implicated exposures. Because several patients were exposed to more than one potentially nephrotoxic medication, the findings also emphasize that D-AKI may arise through multiple simultaneous drug-related mechanisms rather than from a single offending agent. Overall, the medication profile observed in the present study is consistent with previous evidence identifying antibiotics, NSAIDs, diuretics, and renin-angiotensin system inhibitors as clinically important contributors to drug-associated renal injury.

Several demographic, clinical, and medication-related factors were significantly associated with D-AKI in the present study. Patients aged >60 years were significantly more frequent in the D-AKI group than in the non-D-AKI group (58.1% vs. 36.4%; $p=0.003$), while hypertension, diabetes mellitus, and CKD were also significantly more common among patients with D-AKI. These findings are consistent with Yu *et al.*, [18],

who reported associations between D-AKI and older age, hypertension, diabetes, CKD, polypharmacy, ARB use, NSAID use, and diuretic use. In their multivariable analysis, NSAID use, diuretic use, and CKD remained independently associated with D-AKI, underscoring the importance of these factors. Similarly, Lin *et al.*, [19] demonstrated that NSAID-associated AKI risk increased substantially with advancing age and that CKD, cardiovascular disease, diabetes, and hypertension were independently associated with AKI. In the present study, sepsis or infection was also significantly more frequent among patients with D-AKI (46.8% vs. 29.3%; $p=0.012$), suggesting that acute systemic illness may increase susceptibility to renal injury in the presence of nephrotoxic or hemodynamically active medications. Polypharmacy showed a significant association with D-AKI (69.4% vs. 46.2%; $p=0.002$), further supporting the concern that exposure to multiple medications may increase the opportunity for nephrotoxic effects and drug-drug interactions. Significant associations were also observed for NSAID exposure (25.8% vs. 9.8%; $p=0.002$), diuretic exposure (40.3% vs. 17.4%; $p<0.001$), and ACE inhibitor/ARB exposure (33.9% vs. 17.4%; $p=0.007$), closely corresponding to medication-related risk factors reported in previous studies. [18,19] Male sex was not significantly associated with D-AKI in the present cohort ($p=0.337$). Taken together, these findings indicate that D-AKI is likely influenced by the interaction of patient vulnerability, underlying disease, acute illness, polypharmacy, and exposure to medications capable of causing direct nephrotoxicity or altering renal hemodynamics.

The economic findings demonstrate that D-AKI imposes a substantial additional burden on hospital resources. Patients with D-AKI had a significantly longer mean hospital stay than those without D-AKI (14.2 ± 6.8 vs. 8.1 ± 4.2 days; $p<0.001$), consistent with Stottlemeyer *et al.*, [20], who, in a systematic review of 25 studies, identified prolonged hospitalization as a major driver of the economic burden associated with drug-associated AKI, with reported increases in hospital stay ranging from 5 to 13.8 days. Similarly, Silver *et al.*, [21] found that AKI was associated with additional hospital days and increased hospitalization costs, with a substantially greater burden among patients requiring dialysis. In the present study, total hospitalization costs were significantly higher among patients with D-AKI than among those without D-AKI (BDT $45,000 \pm 15,800$ vs. $26,800 \pm 10,200$; $p<0.001$). Medication, investigation, and hospital/bed costs were all significantly greater in the D-AKI group, supporting the multifaceted cost burden described by Stottlemeyer *et al.*, [20], in which pharmacy services, laboratory investigations, routine hospital care, radiology, intensive care, and other hospital services contributed to excess expenditure. Furthermore, renal replacement therapy was required in 11.3% of patients with D-AKI compared with none of those without D-AKI ($p<0.001$), representing an additional marker of resource utilization and disease severity. This finding is

consistent with Silver *et al.*, [21], who demonstrated that dialysis-requiring AKI was associated with substantially greater hospitalization costs and longer hospital stays than AKI without dialysis. Thus, the present findings indicate that the economic consequences of D-AKI extend beyond the renal complication itself, encompassing prolonged hospitalization, increased medication and diagnostic requirements, greater inpatient care costs, and the need for renal replacement therapy. From a healthcare perspective, these findings underscore the potential value of strategies aimed at identifying high-risk patients, minimizing unnecessary polypharmacy, monitoring renal function during exposure to potentially nephrotoxic medications, and promptly recognizing early renal injury.

Limitations of the study

The study had a few limitations:

- The study was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings to other healthcare settings.
- The relatively short study period limited the assessment of long-term renal outcomes and the economic consequences of drug-induced AKI.

CONCLUSION

Drug-induced acute kidney injury is an important and potentially preventable complication of pharmacotherapy that can adversely affect patient outcomes and healthcare resources. D-AKI was associated with older age, comorbidities, polypharmacy, and potentially nephrotoxic medications, with antibiotics being the most frequently implicated class. Although most patients achieved complete renal recovery, D-AKI was associated with prolonged hospitalization, increased healthcare costs, and the need for renal replacement therapy. Early identification of high-risk patients, rational prescribing, and renal function monitoring may help reduce its clinical and economic burden.

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