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Acute Kidney Injury After Cardiac Surgery in Patients Without Chronic Kidney Disease: Incidence, Predictors, and Outcomes

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ABSTRACT

Acute kidney injury (AKI) is a frequent and serious complication following cardiac surgery, even in patients without preexisting chronic kidney disease (CKD). This prospective cohort study included 950 adult patients undergoing elective cardiac surgery at Assiut University Hospital between 2021 and 2024. Using KDIGO criteria, the incidence of cardiac surgery–associated AKI (CSA-AKI) was 42%, with 18.6% classified as severe (stage 3). AKI patients were significantly older, more often female, and had higher rates of hypertension, diabetes, and valvular surgery. They also experienced longer cardiopulmonary bypass and cross-clamp times, more postoperative hypotension and sepsis, and a greater need for renal replacement therapy. Mortality was 22.3% compared with 6.5% in patients without AKI. Multivariate analysis identified hypertension, diabetes, and prolonged ICU and hospital stays as independent predictors. CSA-AKI remains common and strongly associated with adverse outcomes, highlighting the need for preventive perioperative management strategies.

KEYWORDS: Acute kidney injury; Cardiac surgery; Cardiopulmonary bypass; KDIGO; Risk factors; Renal outcomes.

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INTRODUCTION

Acute kidney injury (AKI) is a frequent and serious complication following cardiac surgery, even in patients without pre-existing chronic kidney disease (CKD) [1]. Cardiac surgery-associated AKI (CSA-AKI) is linked with increased morbidity, prolonged hospital stays, higher healthcare costs, and raised short- and long- term mortality [2,3]. Despite advances in surgical techniques and perioperative care, the incidence of CSA-AKI remains significant, ranging from 5% to 30% depending on the diagnostic criteria and patient population [4,5].

Improving Global Outcomes (KDIGO) classification offers a standardized and widely accepted framework for diagnosing and staging AKI, integrating both changes in serum creatinine and alterations in urine output [6]. Adoption of KDIGO criteria has improved early recognition and stratification of CSA-AKI severity, enabling timely interventions and risk assessment [7]. Several studies have emphasized the utility of KDIGO-based definitions in predicting postoperative outcomes and guiding clinical management [8,9].

Multiple perioperative factors contribute to the development of CSA-AKI. Patient-related risk factors include advanced age, preexisting comorbidities, and baseline renal function. In contrast, procedural variables such as cardiopulmonary bypass (CPB) duration, perfusion pressure, hemodilution, and hypothermia during surgery have been implicated in renal injury [10–12]. The choice of surgical techniques, on-pump versus off-pump procedures, conventional sternotomy versus minimally invasive approaches also influences CSA-AKI incidence [13,14]. Understanding these procedural and perioperative determinants is essential for identifying modifiable factors that may reduce the risk of postoperative renal injury.

This study focuses on evaluating the incidence, risk factors, and outcomes of CSA-AKI in patients without preexisting CKD, applying KDIGO diagnostic criteria taking consideration that following urine output in early post-operative cardiac surgery patients to diagnose AKI can be misleading as oliguria commonly occurs in the immediate postoperative period and may be related more to poor perfusion than acute loss of intrinsic renal function.

Furthermore, this study examines the impact of surgical techniques, including on-pump versus off-pump procedures, as well as perioperative management variables such as CPB duration, perfusion pressure, hemodilution, and hypothermia. By focusing on these factors, this research aims to address knowledge gaps regarding modifiable procedural determinants of CSA-AKI and to provide evidence that can guide clinical practice in improving postoperative renal outcomes for this specific patient population.

PATIENTS AND METHODS

Study Design.

This prospective, observational, analytic longitudinal cohort study was conducted to evaluate the incidence, risk factors, and outcomes of acute kidney injury (AKI) in adult patients undergoing cardiac surgery. The longitudinal design enabled standardized data collection, close follow-up, and accurate application of internationally accepted KDIGO diagnostic criteria for AKI, allowing assessment of both early and late postoperative renal outcomes.

Study Setting.

The study was performed in the Department of Cardiothoracic Surgery at Assiut University Hospital, a tertiary care center providing specialized cardiac surgical services for both public and private patients. The facility performs a wide range of procedures, including coronary artery bypass grafting (CABG), valve replacement, and combined surgeries. The intensive care unit (ICU) is equipped with advanced hemodynamic monitoring, renal replacement therapy (RRT), and laboratory support, ensuring appropriate evaluation of perioperative renal outcomes.

Study Population

Inclusion criteria

Adults aged 18–80 years undergoing elective cardiac surgery (CABG, valve replacement, or combined procedures) with normal baseline renal function (absence of CKD and baseline serum creatinine ≤2 mg/dL).

Exclusion criteria

Patients undergoing surgery for congenital heart disease, requiring emergency surgery, having received iodinated contrast within 72 hours preoperatively, with preexisting CKD or serum creatinine >2 mg/dL, mechanically ventilated preoperatively, who died within 24 hours postoperatively, or with systemic comorbidities unrelated to cardiac disease.

Sample Size

A total of 950 patients who met the inclusion criteria and underwent eligible cardiac surgery between 2021 and 2024 were enrolled. This sample size ensures adequate events for multivariable analysis and provides sufficient statistical power to assess CSA-AKI incidence, risk factors, surgical variables, and postoperative outcomes.

Data Collection

Data were collected prospectively using standardized forms and entered a secure electronic database. Variables were classified as preoperative, perioperative, and postoperative.

Preoperative data

Demographics (age, sex, BMI), comorbidities (hypertension, diabetes, COPD, coronary artery disease, prior stroke, previous cardiac surgery), laboratory investigations (baseline serum creatinine, urea), and Cleveland Clinic Scoring Tool (CCST) risk stratification.

Perioperative data

Type of surgery (CABG, valve replacement, or combined procedures), cardiopulmonary bypass (CPB) duration, aortic cross-clamp time, and pharmacologic support, including vasoactive medications.

Postoperative data

Renal function (daily serum creatinine and urea on postoperative days 1–2 and at discharge), hourly urine output for 48 hours (then daily), hemodynamics (mean arterial pressure, fluid balance), ICU and hospital stay, mechanical ventilation duration, requirement for RRT, and postoperative mortality (early ≤ 30 days; late >30 days until final follow-up).

Definitions

Acute Kidney Injury (AKI): defined according to KDIGO criteria: (i) increase in serum creatinine ≥0.3 mg/dL within 48 hours, (ii) increase to ≥1.5 times baseline within 7 days, or (iii) urine output <0.5 mL/kg/h for ≥6 consecutive hours.
Renal recovery: return of serum creatinine to baseline before hospital discharge.
Early mortality: death within 30 postoperative days.
Late mortality: death ≥31 days postoperatively until final follow-up.
Outcome Measures
Primary outcomes: incidence of CSA-AKI and requirements for RRT.
Secondary outcomes: postoperative mortality, length of ICU and hospital stay, and renal recovery.

Statistical Analysis

Data was analyzed using SPSS v27 (IBM Corp., Chicago, IL, USA). Normality was assessed with the Shapiro–Wilk test and histograms. Continuous parametric variables were presented as mean ± SD and compared using Student's t-test; non-parametric variables as median (IQR) using the Mann–Whitney U test. Categorical variables were expressed as counts (%) and compared using chi-square or Fisher's exact test. Multivariable logistic regression was used to identify independent predictors of CSA-AKI. A two-tailed p-value ≤0.05 was considered statistically significant.

Ethical Considerations

The study was approved by the Institutional Review Board (IRB) of Assiut Medical School. Written informed consent was obtained from all participants. The study did not interfere with standard patient care. Confidentiality was maintained using unique study codes, password-protected electronic databases, and secure physical storage. Patient identities were not disclosed in any reports or publications.

RESULTS

A total of 950 patients without preexisting chronic kidney disease were included in the study. The age of the cohort ranged from 30 to 71 years, with a mean of 51.4 ± 6.75 years (Table 1). There were 581 (61.16%) male and 369 (38.84%) female patients. The mean weight was 73.5 ± 11.25 kg (range 49–107), and the mean height was 162.9 ± 7.07 cm (range 145–182), resulting in a mean body mass index (BMI) of 27.8 ± 4.29 kg/m² (range 18.6–42.1). Comorbidities included hypertension in 227 patients (23.89%), and diabetes mellitus in 181 patients (19.05%) of which 73 (7.68%) patients had insulin-dependent diabetes. Atrial fibrillation was present in 141 patients, (14.84%) while 73 (7.68%) had a history of myocardial infarction. Heart failure was classified as class I in 22 (2.32%), class II in 197 (20.74%), class III in 711 (74.84%), and class IV in 19 (2%) patients. Chronic obstructive pulmonary disease (COPD) was reported in 87 patients (9.16%), and peripheral vascular disease was present in 248 patients (26.11%). Most patients had ischemic heart disease (747; 78.63%), followed by mitral stenosis (148; 15.58%), mitral regurgitation (90; 9.47%), aortic stenosis (61; 6.42%), tricuspid regurgitation (35; 3.68%), aortic regurgitation (21; 2.21%), and atrial septal defect (18; 1.89%). Regarding surgical procedures, 485 patients (51.05%) experienced on-pump CABG, 173 (18.21%) underwent off-pump CABG, 182 (19.16%) had valve surgery, 92 (9.68%) undertook combined procedures, and 18 (1.89%) had ASD closure.

Table 1. Demographic data, comorbidities and preoperative data of the patients studied.

Variable	Value
Age (years)	Mean ± SD: 51.4 ± 6.75, Range: 30–71
Gender	Male: 581 (61.16 %) Female: 369 (38.84 %)
Weight (kg)	Mean ± SD: 73.5 ± 11.25, Range: 49–107
Height (cm)	Mean ± SD: 162.9 ± 7.07, Range: 145–182
BMI (kg/m²)	Mean ± SD: 27.8 ± 4.29, Range: 18.6–42.1
Comorbidities	Hypertension: 227 (23.89%) Diabetes mellitus: 181 (19.05%) Insulin-dependent DM: 73 (7.68%) Atrial fibrillation: 141 (14.84%) Myocardial infarction: 73 (7.68%) Heart failure: I – 22 (2.32%), II – 197 (20.74%), III – 711 (74.84%), IV – 19 (2%) COPD: 87 (9.16%) Peripheral vascular disease: 248 (26.11%)
Diagnosis	Ischemic heart disease: 747 (78.63%) Mitral regurgitation: 90 (9.47%) Mitral stenosis: 148 (15.58%) Aortic stenosis: 61 (6.42%) Aortic regurgitation: 21 (2.21%) Tricuspid regurgitation: 35 (3.68%) Atrial septal defect: 18 (1.89%)
Type of Surgery	On-pump CABG: 485 (51.05%) Off-pump CABG: 173 (18.21%) Valve surgery: 182 (19.16%) Combined surgery: 92 (9.68%) ASD closure: 18 (1.89%)
Treatment/Medications	Aspirin: 248 (26.11%) ACE inhibitors: 252 (26.53%) Beta-blockers: 681 (71.68%) Diuretics: 252 (26.53%)
Cleveland Clinic Score	Median (IQR): 1 (0–2)
Preoperative Creatinine (mg/dL)	Mean ± SD: 0.9 ± 0.11, Range: 0.7–1.1
Ejection Fraction (%)	Mean ± SD: 47.2 ± 10.23, Range: 20–75

BMI: body mass index. COPD: Chronic obstructive pulmonary disease. DM: Diabetes mellitus. ACE inhibitors: Angiotensin-converting enzyme inhibitors. CABG: Coronary artery bypass grafting. ASD: Atrial septal defect. ACE inhibitors: Angiotensin-converting enzyme inhibitors.

Preoperative medications included aspirin in 248 (26.11%) patients, ACE inhibitors in 252 (26.53%), beta-blockers in 681 (71.68%), and diuretics in 252 (26.53%). The median Cleveland Clinic Score was 1 (IQR: 0–2). Preoperative serum creatinine ranged from 0.7 to 1.1 mg/dL, with a of 0.9 ± 0.11 mg/dL. Left ventricular ejection fraction (EF) ranged from 20% to 75%, with a mean of 47.2 %.

Intraoperative data of the patients studied

The Intraoperative data of the patients studied are preseted in Table 2. The mean duration of cardiac surgery was 6 ± 0.75 hours (range: 3.32–8.27 hours). Cardiopulmonary bypass (CPB) was required in 709 (74.63%) patients, with a mean CPB time of 148.2 ± 11.9 minutes (range: 85–240). Aortic cross-clamp time ranged from 40 to 180 minutes, with a mean of 104.9 ± 9.7 minutes. Inotropic support was administered in most patients: 743 (78.21%) received norepinephrine, all 950 (100%) received epinephrine, 298 (31.37%) received dobutamine, and 46 (4.84%) received Levosimendan. Diuretics were directed to 709 (74.63%) patients. Intraoperative complications included hypotension in 410 (43.16%) patients, arrhythmia in 606 (63.79%), and cardiopulmonary resuscitation (CPR) in 18 (1.89%). The median intraoperative fluid balance was 161 mL (IQR: -103.5 to 1840).

Table 2. Intraoperative Characteristics and Hemodynamic Support (n = 950).

Variable	Value
Duration of operation (hours)	Mean ± SD: 6 ± 0.75 Range: 3.32–8.27
Cardiopulmonary bypass (CPB)	Required: 709 (74.63%) CPB time (min): Mean ± SD: 148.2 ± 11.9, Range: 85–240
Aortic cross-clamp time (min)	Mean ± SD: 104.9 ± 9.7, Range: 40–180
Inotropic support	Norepinephrine: 743 (78.21%) Epinephrine: 950 (100%) Dobutamine: 298 (31.37%) Levosimendan: 46 (4.84%)
Diuretics	709 (74.63%)
Intraoperative complications	Hypotension: 410 (43.16%) Arrhythmias: 606 (63.79%) CPR required: 18 (1.89%)
Intraoperative fluid balance (mL)	Median (IQR): 161 (-103.5 – 1840)

CPB: Cardiopulmonary bypass. CPR: Cardiopulmonary resuscitation.

Postoperative treatment and complications of the patients studied

Postoperatively, inotropic or vasoactive support was commonly administered: 779 (82%) patients received norepinephrine, 941 (99.05%) received epinephrine, 283 (29.79%) received dobutamine, 63 (6.63%) received Levosimendan, 28 (2.95%) received dopamine, and 15 (1.58%) received a combination of Levosimendan and dopamine (Table 3). Other medications included NSAIDs (excluding aspirin) in 165 (17.37%) patients and diuretics in 646 (68%) patients. Postoperative complications included hypotension in 267 (28.11%) patients, sepsis in 39 (4.11%), and 100 (10.53%) patients required reoperation.

Table 3. Postoperative Treatment and Complications (n = 950).

Variable	Value
Inotropic/Vasoactive Support	Norepinephrine: 779 (82%) Epinephrine: 941 (99.05%) Dobutamine: 283 (29.79%) Levosimendan: 63 (6.63%) Dopamine: 28 (2.95%) Levosimendan & dopamine: 15 (1.58%)
Other Medications	NSAIDs (except aspirin): 165 (17.37%) Diuretics: 646 (68%)
Postoperative Complications	Hypotension: 267 (28.11%) Sepsis: 39 (4.11%) Reoperation: 100 (10.53%)

Urine output in ICU

Urine output was measured daily during the ICU stay (Table 4). The mean urine output on day 0 was 1480.7 ± 403.71 mL. It increased significantly to 2388.3 ± 385.03 mL on day 1 ($P < 0.001$), decreased significantly to 917.8 ± 163.55 mL on day 2 ($P < 0.001$), and was not significantly different from day 0 on day 3 (1470.5 ± 397.92 mL, $P = 0.591$). These findings indicate a **significant postoperative fluctuation in urine output**, with a peak on day 1 followed by a decline on day 2 and recovery by day 3.

Table 4. Urine Output in the ICU (mL/day) (n = 950).

ICU Day	Urine Output (Mean ± SD)	P-value vs Day 0
Day 0	1480.7 ± 403.71	–
Day 1	2388.3 ± 385.03	<0.001*
Day 2	917.8 ± 163.55	<0.001*
Day 3	1470.5 ± 397.92	0.591

*Data presented as mean ± SD. Statistical significance determined by comparison with Day 0.

3.4. ICU Fluid Management and Renal Replacement Therapy

The total ICU fluid input ranged from 3233 to 8855 mL, with a mean ± SD of 6167.6 ± 779.27 mL (Table 5). The total ICU fluid output ranged from 4228 to 9910 mL, with a mean ± SD of 6657.2 ± 841.37 mL. The median ICU fluid balance was -607.5 mL (IQR: -826.75 to -68.25), indicating a slightly negative fluid balance in most patients. Renal replacement therapy (RRT) was required in 78 (8.21%) patients during the ICU stay.

Table 5. ICU Fluid Management and Renal Replacement Therapy (n = 950).

Variable	Value
Total ICU Fluid Input (mL)	Mean ± SD: 6167.6 ± 779.27 Range: 3233–8855
Total ICU Fluid Output (mL)	Mean ± SD: 6657.2 ± 841.37 Range: 4228–9910
ICU Fluid Balance (mL)	Median (IQR): -607.5 (-826.75 – -68.25)
Renal Replacement Therapy (RRT)	78 (8.21%)

Incidence and KDIGO Staging of AKI

Among the 950 patients studied, 399 (42%) developed acute kidney injury (AKI) according to KDIGO criteria (Table 6). Of these, 158 (16.63%) patients were classified as stage 1, 64 (6.74%) as stage 2, and 177 (18.63%) as stage 3. These findings highlight a relatively high incidence of postoperative AKI, with a considerable proportion of patients developing moderate to severe stages (KDIGO 2–3).

Table 6. Incidence and KDIGO Staging of AKI among the Studied Patients (n = 950).

Parameter	n (%)
AKI (overall)	399 (42%)
KDIGO Stage 1	158 (16.63%)
KDIGO Stage 2	64 (6.74%)
KDIGO Stage 3	177 (18.63%)

Length of ICU and Hospital Stay

The duration of the first ICU stay among the studied patients ranged from 2 to 28 days, with a mean (±SD) of 5.1 ± 3.86 days (Table 7). The second ICU stay, observed in patients who required readmission to intensive care, ranged from 1 to 12 days, with a mean (±SD) of 5.2 ± 2.37 days. Although the mean duration of ICU stay did not differ substantially between the first and second admissions, the second ICU stay demonstrated a narrower range, suggesting a more uniform duration among readmitted patients. The overall hospital stay ranged from 4 to 30 days, with a mean (±SD) of 9.8 ± 6.88 days.

Table 7. Length of ICU and Hospital Stay of the Studied Patients (n = 950).

Parameter	Mean ± SD (days)	Range (days)
Length of ICU stay	5.1 ± 3.86	2 – 28
Length of 2nd ICU stay	5.2 ± 2.37	1 – 12
Length of hospital stay	9.8 ± 6.88	4 – 30

Outcome and causes of death among the studied patients

Out of 950 patients, **125 (13.16%)** died during the postoperative period (Table 8). Among these fatalities, the most common cause of death was **cardiogenic shock (53.6%),** followed by **sepsis (22.4%), arrhythmia (9.6%), respiratory failure (5.6%), neurological complications (4%)**, and **major bleeding (4%).** One patient (0.8%) died due to multi-organ failure.

Table 8. Outcome and causes of death among the studied patients (n = 950).

Outcome / Cause of Death	Number of Patients (n)	Percentage (%)
Alive	825	86.84
Dead	125	13.16
Cause of Death		
Cardiogenic shock	67	53.6
Sepsis	28	22.4
Arrhythmia	12	9.6
Respiratory failure	7	5.6
Neurological	5	4.0
Major bleeding	5	4.0
Multi-organ failure	1	0.8

Relation between AKI and selected demographic and diagnostic characteristics

Patients in the **AKI group were significantly older** than those without AKI (mean 53.9 ± 5.7 vs. 49.5 ± 6.8 years, $P < 0.001$) (Table 9). **Female sex** was significantly more frequent among patients who developed AKI ($P < 0.001$). There was **no significant difference in body weight** between the groups ($P = 0.094$). Regarding the underlying cardiac diagnosis, **ischemic heart disease** was significantly less frequent in the AKI group ($P < 0.001$), whereas **mitral stenosis, aortic regurgitation**, and **tricuspid regurgitation** were significantly more prevalent among patients who developed AKI ($P < 0.05$). No significant association was found for **mitral regurgitation, aortic stenosis**, or **atrial septal defect.**

Table 9. Relation between AKI and selected demographic and diagnostic characteristics.

Variable	AKI group (n = 399)	No AKI group (n = 551)	P value
Age (years)			
Mean ± SD	53.92 ± 5.71	49.53 ± 6.84	<0.001*
Range	39 – 71	30 – 71	
Gender			
Male	199 (49.87%)	382 (69.33%)	<0.001*
Female	200 (50.13%)	169 (30.67%)	
Weight (Kg)			
Mean ± SD	72.79 ± 11.26	74.03 ± 11.22	0.094
Range	49 – 107	49 – 107	
Diagnosis			
Ischemic heart disease	277 (69.42%)	470 (85.30%)	<0.001*
Mitral regurgitation	45 (11.28%)	45 (8.17%)	0.116
Mitral stenosis	92 (23.06%)	56 (10.16%)	<0.001*
Aortic stenosis	43 (10.78%)	18 (3.27%)	0.518

Variable	AKI group (n = 399)	No AKI group (n = 551)	P value
Aortic regurgitation	16 (4.01%)	5 (0.91%)	0.002*
Tricuspid regurgitation	21 (5.26%)	14 (2.54%)	0.035*
Atrial septal defect	4 (1.00%)	14 (2.54%)	0.096

Relation between AKI and (type of surgery and comorbidities) of the studied patients

Significant differences were observed in the types of surgery performed between the two groups (Table 10). Both **on-pump** and **off-pump CABG** procedures were significantly less frequent among patients who developed AKI (*P* = 0.002 and *P* < 0.001, respectively). Conversely, **valve surgery** was markedly more prevalent in the AKI group (29.8% vs. 11.4%, *P* < 0.001). No significant differences were found regarding **combined surgery** or **ASD closure**. Among comorbid conditions, **hypertension**, **diabetes mellitus** especially **insulin-treated diabetes,** were significantly more frequent in the AKI group (*P* < 0.001 for each). In contrast, **atrial fibrillation**, **myocardial infarction**, and **heart failure (NYHA classes I–IV)** did not differ significantly between the two groups.

Table 10. Relation between AKI and (type of surgery and comorbidities) of the studied patients (n = 950).

Variable	AKI group (n = 399)	No AKI group (n = 551)	P value
Type of surgery			
On-pump CABG	178 (44.61%)	302 (54.81%)	0.002*
Off-pump CABG	51 (12.78%)	122 (22.14%)	<0.001*
Valve surgery	119 (29.82%)	63 (11.43%)	<0.001*
Combined surgery	46 (11.53%)	46 (8.35%)	0.119
ASD closure	4 (1.00%)	14 (2.54%)	0.206
Comorbidities			
Hypertension	146 (36.59%)	81 (14.70%)	<0.001*
Diabetes mellitus	122 (30.58%)	59 (10.71%)	<0.001*
Insulin-treated DM	59 (48.76%)	14 (23.73%)	0.001*
Atrial fibrillation	58 (14.54%)	83 (15.06%)	0.822
Myocardial infarction	29 (7.27%)	44 (7.99%)	0.682
Heart failure class (NYHA)			
I	9 (2.26%)	13 (2.36%)	1.000
II	81 (20.30%)	116 (21.05%)	0.808
III	300 (75.19%)	411 (74.59%)	0.879
IV	9 (2.26%)	13 (2.36%)	1.000

DM: Diabetes mellitus; CABG: Coronary artery bypass grafting.

Relation between AKI and intraoperative data

The mean ejection fraction (EF**)** was significantly lower in the AKI group compared to the non-AKI group (43.6 ± 9.58 vs. 49.83 ± 9.89, *P* < 0.001) (Table 11). Similarly, patients who developed AKI had significantly higher Cleveland Clinic scores, longer operative durations, longer CPB times, and longer cross-clamp times (all *P* < 0.001). However, the proportion of patients requiring cardiopulmonary bypass (CPB) was not significantly different between the two groups (*P* = 0.164).

Table 11. Relation between AKI and intraoperative data of the studied patients (n = 950).

Variable	AKI group (n = 399) Means ± SD, (Range)	AKI group (n = 399) Means ± SD, (Range)	P value
EF (%)	43.6 ± 9.58 (20 – 71.8)	49.83 ± 9.89 (25.9 – 75)	<0.001*
Cleveland Clinic score	1.63 ± 1.22 (0 – 6)	0.95 ± 1.04 (0 – 5)	<0.001*
Duration of operation (h)	6.10 ± 0.68 (3.32 – 8.27)	5.85 ± 0.78 (3.41 – 7.27)	<0.001*
CPB, n (%)	307 (76.94%)	402 (72.96%)	0.164
CPB time (min)	152.55 ± 12.43 (85 – 240)	144.72 ± 10.20 (90 – 180)	<0.001*
Cross-clamp time (min)	108.19 ± 9.47 (60 – 180)	102.21 ± 9.05 (40 – 124)	<0.001*

EF: Ejection fraction; CPB: Cardiopulmonary bypass.

Relation between AKI and intraoperative treatment and complications.

There were no significant differences between the AKI and non-AKI groups regarding norepinephrine, dobutamine, diuretic use, arrhythmias, CPR, or intraoperative fluid balance (Table 12). However, episodes of intraoperative hypotension were significantly more frequent in the AKI group (47.6% vs. 39.9%, P = 0.018), indicating an association between hemodynamic instability and postoperative kidney injury.

Table 12. The relation between AKI and intraoperative treatment and complications of the studied patients.

Variable	AKI group (n = 399)	No AKI group (n = 551)	P value
Norepinephrine	315 (78.95%)	428 (77.68%)	0.640
Dobutamine	112 (28.07%)	186 (33.76%)	0.062
Diuretics	307 (76.94%)	402 (72.96%)	0.164
Episodes of hypotension	190 (47.62%)	220 (39.93%)	0.018*
Arrhythmias	242 (60.65%)	364 (66.06%)	0.087
CPR	7 (1.75%)	11 (2.00%)	0.787
Intraoperative balance (mL)	Median (IQR): 161 (−103.5 – 1782.5)	161 (−230 – 1955)	0.951

CPR: Cardiopulmonary resuscitation.

The relation between AKI and postoperative complications, and RRT

Postoperative complications were notably more frequent among patients who developed AKI (Table 13). Episodes of hypotension, sepsis, and the need for RRT occurred significantly more often in the AKI group (P < 0.001 for all). In contrast, the rate of reoperation did not differ significantly between the two groups.

Table 13. Relation between AKI and postoperative complications and RRT of the studied patients.

Variable	AKI group (n = 399)	No AKI group (n = 551)	P value
Episodes of hypotension	151 (37.84%)	116 (21.05%)	<0.001*
Reoperation	40 (10.03%)	60 (10.89%)	0.668
Sepsis	32 (8.02%)	7 (1.27%)	<0.001*
RRT	78 (19.55%)	0 (0%)	<0.001*

RT: Renal replacement therapy

Relation between AKI and (ICU fluid balance and SCr at ICU and hospital discharge)

Patients in the AKI group had a significantly higher ICU fluid balance compared to those without AKI (*P* = 0.013), indicating greater fluid retention (Table 14). Serum creatinine levels at both ICU discharge and hospital discharge were markedly higher among AKI patients (*P* < 0.001), reflecting persistent renal impairment. Only patients in the AKI group required a second ICU stay, with a mean duration of 5.2 ± 2.37 days.

Table 14. Relation between AKI and (ICU fluid balance and SCr at ICU and hospital discharge) of the studied patients.

Variable	AKI group (n = 399)	No AKI group (n = 551)	P value
Length of 2nd ICU stay (days)	5.2 ± 2.37 (Range: 1–12)	—	—
ICU fluid balance (mL)	Median: -500 (IQR: -772 – 15.5)	Median: -659 (IQR: -876 – -120.5)	0.013*
SCr at ICU discharge (mg/dL)	Median: 2.03 (IQR: 1.3 – 3)	Median: 0.92 (IQR: 0.83 – 1.02)	<0.001*
SCr at hospital discharge (mg/dL)	Median: 1.2 (IQR: 1.04 – 2.38)	Median: 0.9 (IQR: 0.8 – 1)	<0.001*

ICU: Intensive care unit; SCr: Serum creatinine.

Relation between AKI and (length of ICU stay, length of hospital stay and outcome

Patients who developed AKI had significantly longer ICU and hospital stays compared to those without AKI (*P* < 0.001 for both) (Table 15). Mortality was also markedly higher in the AKI group, with 22.3% of AKI patients dying compared to only 6.54% in the non-AKI group (*P* < 0.001), underscoring the strong association between AKI and adverse postoperative outcomes.

Table 15. Relation between AKI and (length of ICU stay, length of hospital stay, and outcome) of the studied patients.

Variable	**AKI group (n = 399)**	**No AKI group (n = 551)**	**P value**
Length of ICU stay (days)	5.88 ± 4.15	4.49 ± 3.49	<0.001*
Length of hospital stay (days)	12.37 ± 7.25	7.86 ± 6.04	<0.001*
Outcome			
Alive	310 (77.7%)	515 (93.46%)	<0.001*
Dead	89 (22.3%)	36 (6.54%)	<0.001*

ICU: Intensive care unit.

The relation between AKI and the causes of mortality

Sepsis and major bleeding were significantly more common causes of death among patients with AKI compared to those without (P = 0.017 and P = 0.044, respectively) (Table 16). Other causes of mortality, including cardiogenic shock, arrhythmia, neurological events, and respiratory failure, showed no statistically significant differences between the two groups.

Table 16. The relation between AKI and the causes of mortality among the studied patients.

Cause of Death	**AKI group (n = 89)**	**No AKI group (n = 36)**	**P value**
Sepsis	25 (28.1%)	3 (8.3%)	0.017*
Cardiogenic shock	45 (50.6%)	22 (61.1%)	0.273
Arrhythmia	9 (10.1%)	3 (8.3%)	0.776
Neurological causes	4 (4.5%)	1 (2.8%)	1.000
Respiratory failure	4 (4.5%)	3 (8.3%)	0.396
Major bleeding	2 (2.2%)	4 (11.1%)	0.044*

Univariate and multivariate regression analysis of risk factors associated with AKI

Univariate analysis identified several variables significantly associated with the development of AKI, including age, female gender, hypertension, diabetes mellitus, longer CPB and cross-clamp times, and prolonged ICU and hospital stays (P < 0.05) (Table 17). In the multivariate regression model, hypertension, diabetes mellitus, length of ICU stay, and length of hospital stay remained independent predictors of AKI (P < 0.05), confirming their strong influence on postoperative renal outcomes.

Table 17. Univariate and multivariate regression analysis of risk factors associated with AKI

	Univariate Analysis			**Multivariate Analysis**		
Variable	Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Age	1.1148	1.09 – 1.14	<0.001*	1.0577	0.99 – 1.12	0.061
Gender (female)	2.2717	1.74 – 2.97	<0.001*	1.6114	0.74 – 3.49	0.227
Preoperative creatinine	0.5363	0.16 – 1.76	0.305	—	—	—
Hypertension (HTN)	3.3485	2.45 – 4.57	<0.001*	3.7605	1.67 – 8.49	0.001*
Diabetes mellitus (DM)	3.6728	2.60 – 5.18	<0.001*	3.4039	1.24 – 9.31	0.017*
Atrial fibrillation	0.9591	0.67 – 1.38	0.822	—	—	—
Myocardial infarction (MI)	0.9031	0.55 – 1.47	0.682	—	—	—
Heart failure (HF)	1.0349	0.81 – 1.33	0.788	—	—	—
Chronic obstructive pulmonary disease (COPD)	0.9723	0.62 – 1.52	0.902	—	—	—
Peripheral vascular disease (PVD)	0.8129	0.60 – 1.09	0.171	—	—	—
Cardiopulmonary bypass (CPB)	1.2368	0.92 – 1.67	0.164	—	—	—
CPB time (min)	1.0747	1.05 – 1.09	<0.001*	1.0449	0.98 – 1.10	0.112
Cross-clamp time (min)	1.0874	1.07 – 1.11	<0.001*	1.0036	0.96 – 1.05	0.867
Duration of operation (h)	1.6181	1.33 – 1.96	<0.001*	0.2574	0.01 – 5.19	0.376

	Univariate Analysis			Multivariate Analysis		
Length of ICU stay (days)	22.6808	13.56 – 37.93	<0.001*	5.2816	2.79 – 9.99	<0.001*
Length of hospital stay (days)	4.0609	3.29 – 5.02	<0.001*	1.8481	1.37 – 2.49	<0.001*

CI: Confidence interval; HTN: Hypertension; DM: Diabetes mellitus; MI: Myocardial infarction; HF: Heart failure; COPD: Chronic obstructive pulmonary disease; PVD: Peripheral vascular disease; CPB: Cardiopulmonary bypass; ICU: Intensive care unit.

DISCUSSION

Reported CSA-AKI incidence varies widely due to differences in baseline risk, operative complexity, and (crucially) the definition used. Narrative and consensus reviews consistently place overall AKI in the 20–30% range when older criteria are used, with dialysis-requiring AKI typically 2–5% [15-17]. When KDIGO criteria are applied rigorously, rates climb, particularly in mixed (CABG + valve) case-mix cohorts [15-17]. This is mirrored by our cohort's high incidence and substantive proportion of severe (stage 3) AKI.

Definition of sensitivity. KDIGO detects smaller creatinine rises and integrates urine output, outperforming RIFLE/AKIN for sensitivity and prognostication [15,16]. Centers adopting KDIGO early and consistently, as in our study, identify more AKI, especially stage 1, clarifying why our rate exceeds those of series that used older definitions. Procedural complexity and perfusion exposure. Valve surgery and combined CABG+valve procedures, longer CPB and cross-clamp times, and intraoperative/early postoperative hemodynamic instability are repeatedly linked to higher AKI risk [16,18]. Our cohort contained a substantial proportion of such exposures, plausibly elevating incidence. Clinically, the message is less about "counting more AKI" and more about capturing clinically relevant renal stress early enough to intervene, because even mild KDIGO stage 1 events carry prognostic weight (see Outcomes below) [19].

In this prospective cohort study of 950 adult patients without chronic kidney disease undergoing various cardiac surgeries, the incidence of acute kidney injury (AKI) reached 42%, with 18.6% of patients developing severe forms (KDIGO stage 3). These findings underscore that CSA-AKI (cardiac surgery–associated acute kidney injury) continues to be one of the most prevalent and serious complications following cardiac surgery. Comparable studies have reported an incidence ranging between 25% and 45%, depending on the patient population, surgical type, and diagnostic criteria used [20–22]. Patients who developed AKI in our cohort experienced significantly longer ICU and hospital stays, higher rates of postoperative complications, and greater mortality (22.3% vs. 6.5%) compared with patients who maintained normal renal function. These results are consistent with previous research demonstrating that even mild postoperative increases in serum creatinine are associated with a substantial rise in morbidity, prolonged hospitalization, and early mortality [23,24].

Risk Factors and Predictors of AKI

Multivariate analyses identified several clinical and intraoperative parameters associated with AKI development. Hypertension, diabetes mellitus, prolonged ICU stay, and longer hospital stay emerged as independent predictors in the final multivariate model. These findings align with several previous investigations reporting that pre-existing hypertension and diabetes significantly predispose patients to CSA-AKI [25–27]. Both comorbidities are known to induce microvascular and endothelial damage, increase oxidative stress, and reduce renal autoregulatory capacity, making the kidneys more susceptible to ischemic and inflammatory injury during and after cardiopulmonary bypass (CPB) [28,29].

Although age, female sex, and CPB duration showed significant associations in univariate analysis, these variables did not hold independent significance after adjustment. Nevertheless, their contribution to renal vulnerability should not be underestimated. Older age and female gender have been previously correlated with lower nephron mass, impaired autoregulation, and increased inflammatory response to CPB [30,31].

Surgical and Intraoperative Influences

Cardiopulmonary bypass and aortic cross-clamping are central factors in CSA-AKI pathogenesis. In our study, patients with AKI had significantly longer CPB and cross-clamp times. This observation supports the concept that prolonged exposure to extracorporeal circulation increases renal hypoperfusion, hemolysis, and systemic inflammation, which together contribute to ischemia–reperfusion injury [32–34]. Other authors have similarly demonstrated that each additional 30 minutes of CPB time increases AKI risk by approximately 20–30% [35].

Although intraoperative hypotension, vasopressor use, and diuretic administration were common in our cohort, these variables did not independently predict AKI after multivariable adjustment. The same has been observed by Lassnigg et al. [8] and Rosner et al. [2], who suggested that it is not the use of vasopressors per se, but rather the underlying hemodynamic instability, that drives renal injury.

Off-pump CABG was proportionally less common in the AKI group, while valve surgery was over-represented. Although off-pump techniques can reduce mild creatinine rises in some populations, the weight of evidence suggests that meticulous perfusion/hemodynamic management is more important for kidney protection than pump avoidance per se, especially in mixed valve/CABG programs [16, 18].

Postoperative Course and Outcomes

Postoperative complications were notably more frequent among AKI patients, particularly hypotensive episodes and sepsis, which were both significantly associated with renal injury and mortality. Sepsis is known to exacerbate renal hypoperfusion through inflammatory cytokine release, microcirculatory dysfunction, and direct tubular damage [36]. The strong association between AKI and mortality in our study (22.3%) reflects global data, where mortality rates in severe CSA-AKI can exceed 30–50% [37,38].

Additionally, it was observed that RRT was required in nearly 20% of AKI patients, similar to the 10–20% range reported in comparable prospective cohorts [39, 40]. This high rate of dialysis underscores the clinical severity of renal dysfunction in this population and its major implications for resource utilization.

Pathophysiological Considerations
CSA-AKI is a complex, multifactorial process involving both ischemic and inflammatory mechanisms. Renal ischemia–reperfusion injury during CPB leads to oxidative stress, activation of neutrophils, and endothelial dysfunction [41]. The release of free hemoglobin and iron from hemolysis further amplifies oxidative injury [42]. Moreover, systemic inflammatory responses, complement activation, and microembolization contribute to tubular and vascular damage [43].

In patients with hypertension and diabetes, chronic endothelial damage and impaired nitric oxide production exacerbate the susceptibility of the renal microcirculation to ischemia [44]. Postoperative hemodynamic instability (especially sustained MAP <65 mmHg), venous congestion (CVP >10 mmHg), and positive fluid balance, observed more frequently in the AKI group, may have perpetuated renal venous congestion and decreased glomerular filtration pressure [45, 46].

Clinical Implications
The findings highlight several important clinical messages.
First, preoperative identification and optimization of high-risk patients—particularly those with hypertension, diabetes, or reduced cardiac function—are essential with universal kidney health assessment (including proteinuria), individualized optimization (e.g., hold ACEI/ARB when hypotension risk is high), and allow clear liquids up to 2 hours pre-anesthesia in appropriate patients to avoid dehydration.

Secondly, intraoperative optimization—particularly implement a CPB bundle—maintain DO_2 targets (>280–300 mL/min/m² where feasible), avoid sustained hypotension, prevent severe anemia/hemodilution, control glucose (<180 mg/dL), avoid excessive ultrafiltration, and minimize nephrotoxin exposure; use individualized goal-directed hemodynamics [16, 18, 47]. — may play a crucial role in reducing the incidence of AKI following cardiac surgery.

Third, postoperative Mild AKI matters. Small creatinine rises (0.3–0.5 mg/dL) independently increase 30-day mortality [19]. The clinical implication is that stage 1 KDIGO AKI warrants the same early-response posture as more severe stages, with a focus on preventing progression. RRT marks extreme risk. Although only a minority of cardiac surgery patients require dialysis, mortality in this group often approaches or exceeds 50% [15, 16]. Our finding that a sizeable fraction of AKI cases progressed to stage 3 and that RRT clustered within AKI patients—explains a substantial portion of the excess mortality signal. Also avoid routine diuretics and NSAIDs (Nephrotoxins); NSAIDs were used in 17% post-op. Given their impact on afferent arteriolar tone and autoregulation, avoiding non-aspirin NSAIDs in the peri-operative period is a pragmatic, high-yield change [18, 47]; use point-of-care ultrasound (POCUS) to assess volume/venous congestion; recognize that creatinine/urine output lag true injury and consider urinary stress biomarkers (TIMP-2•IGFBP7) to trigger a KDIGO-style care bundle in at-risk patients [47].

While multiple pharmacological interventions have been tested (e.g., *N-acetylcysteine*, sodium bicarbonate, statins), none have consistently shown benefit across studies [48, 49]. Therefore, attention to hemodynamic and volume optimization remains the most effective preventive measure at present [50, 51].

LIMITATIONS
This study has several limitations. As a single-center study, the findings may not fully reflect practices and outcomes in other institutions with different surgical protocols and patient demographics. Second, while we identified important clinical predictors, we were unable to incorporate novel biomarkers such as NGAL or [TIMP-2]: [IGFBP7], which have been shown to enable earlier detection of subclinical injury [16, 52]. Third, although our prospective design minimized missing data, residual confounding remains possible, particularly regarding intraoperative fluid and hemodynamic management. Finally, the observational nature of the study precludes causal inference, though associations are biologically plausible and consistent with mechanistic studies.

CONCLUSION
CSA-AKI was frequent and clinically consequential in this setting, with a clear severity–outcome gradient and high RRT utilization. Readily identifiable comorbidities (hypertension, diabetes) and modifiable peri-operative factors underpin risk. Standardized, pathway-based prevention and early detection—emphasizing hemodynamic stewardship (arterial pressure and venous congestion), oxygen-delivery targets during bypass, judicious transfusion, nephrotoxin avoidance, and rapid post-operative response—are warranted to reduce AKI severity and improve outcomes.

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