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Cardiorenal Syndrome Type 1 After Coronary Artery Bypass Grafting: Clinical and Metabolic Predictors of Acute Kidney Injury

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Research Article

Keywords: Cardiorenal syndrome type 1, Coronary artery bypass grafting, Acute kidney injury, Cardiopulmonary bypass, Hemodynamic instability, Metabolic acidosis

Posted Date: September 30th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10953483/v1

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Additional Declarations: No competing interests reported.

Abstract

Background

Cardiorenal syndrome type 1 (CRS-1), characterised by acute cardiac dysfunction leading to acute kidney injury (AKI), represents a clinically important but under-recognised phenotype following coronary artery bypass grafting (CABG), where it significantly increases morbidity and mortality. Early risk stratification remains challenging due to the complex interplay between cardiac dysfunction and perioperative hemodynamic and metabolic disturbances. This study aimed to identify independent predictors of postoperative AKI, the majority of which occurred in the setting of CRS-1 after elective CABG.

Methods

This retrospective multicenter cohort study included 250 adult patients undergoing elective CABG across four tertiary cardiac centers. AKI was defined using Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Preoperative, intraoperative, and early postoperative variables—including estimated glomerular filtration rate (eGFR), left ventricular ejection fraction (LVEF), intraoperative mean arterial pressure (MAP), serum lactate, and acid–base status—were extracted from electronic medical records. Multivariable logistic regression was used to identify independent predictors of postoperative AKI.

Results

Postoperative AKI occurred in 95 patients (38%), of whom 78 (82.1%) fulfilled the prespecified diagnostic criteria for cardiorenal syndrome type 1 (CRS-1). Independent predictors of AKI included preoperative eGFR < 60 mL/min/1.73 m² (adjusted odds ratio [aOR] 3.2; 95% CI 1.8–5.7; p < 0.001), LVEF < 40% (aOR 2.7; 95% CI 1.5–4.8; p = 0.002), intraoperative MAP < 65 mmHg sustained for > 20 minutes (aOR 2.9; 95% CI 1.6–5.2; p < 0.001), ICU admission serum lactate > 2.0 mmol/L (aOR 3.5; 95% CI 1.9–6.3; p < 0.001), metabolic acidosis with base excess < − 5 mmol/L (aOR 2.4; 95% CI 1.3–4.3; p = 0.004), CPB duration > 120 minutes (aOR 2.1; 95% CI 1.2–3.8; p = 0.011), and preoperative diabetes mellitus (aOR 1.9; 95% CI 1.1–3.4; p = 0.028). Patients with AKI experienced significantly higher in-hospital mortality (14.7% vs. 3.9%; p = 0.01) and prolonged intensive care unit length of stay (mean 7.8 vs. 3.9 days).

Conclusions

CRS-1-related AKI after CABG is driven by a multifactorial interaction between preoperative cardiac dysfunction, intraoperative hypotension, and early postoperative metabolic derangements. Integration of readily available clinical and metabolic parameters into perioperative monitoring may enable early risk stratification and timely initiation of cardio-renal protective strategies, thereby improving outcomes in high-risk patients.

Introduction

Cardiorenal syndrome type 1 (CRS-1) is defined as acute kidney injury (AKI) secondary to acute cardiac dysfunction and represents a frequent and clinically significant complication following coronary artery bypass grafting (CABG) [1, 2]. Despite advances in surgical techniques and perioperative management, postoperative AKI remains common, affecting approximately 20–40% of patients undergoing cardiac surgery, and is independently associated with increased in-hospital mortality, prolonged intensive care unit (ICU) stay, and higher healthcare costs [3, 4].

The pathophysiology of CRS-1 is complex and multifactorial. Traditionally, renal injury has been attributed to reduced cardiac output leading to renal hypoperfusion; however, contemporary evidence highlights additional mechanisms including venous congestion, systemic inflammatory activation, endothelial dysfunction, and oxidative stress as key contributors to renal injury in the perioperative setting [5, 6]. In patients undergoing CABG, these mechanisms may be further exacerbated by cardiopulmonary bypass–induced inflammatory response and intraoperative hemodynamic instability, particularly sustained hypotension and reduced renal perfusion pressure [7].

Because the renal medulla is highly susceptible to hypoperfusion, systemic circulatory insufficiency not only drives renal tubular injury but also precipitates global anaerobic metabolism. Consequently, early postoperative metabolic disturbances, including hyperlactatemia and metabolic acidosis, have been increasingly recognized as markers of global tissue hypoperfusion and impaired oxygen utilization, and are strongly associated with adverse renal outcomes following cardiac surgery [8, 9]. These metabolic derangements may reflect a state of systemic circulatory insufficiency that contributes to the development and progression of CRS-1 in susceptible patients.

Despite the existence of established clinical risk scores for postoperative AKI, their predictive accuracy remains suboptimal, and they often fail to incorporate dynamic intraoperative and early postoperative physiological changes. Novel biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 (KIM-1) have demonstrated potential utility; however, their limited availability and high cost restrict routine clinical application [10].

Therefore, there is increasing interest in the use of readily available perioperative variables, including estimated glomerular filtration rate (eGFR), left ventricular ejection fraction (LVEF), intraoperative mean arterial pressure (MAP), serum lactate levels, and acid–base status, as pragmatic tools for early risk stratification of AKI in cardiac surgery patients [11].

Accordingly, this multicenter retrospective cohort study aimed to identify independent clinical, hemodynamic, and metabolic predictors of postoperative AKI following elective CABG, characterize the proportion of cases fulfilling prespecified CRS-1 criteria, and evaluate their association with key postoperative outcomes, including in-hospital mortality and ICU length of stay.

Methods

Study Design and Setting

This retrospective multicenter cohort study included consecutive adult patients undergoing elective isolated on-pump coronary artery bypass grafting (CABG) between January 2022 and December 2025 across four tertiary cardiac surgery centers. The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of New Valley University (approval no. 20260430010), with formal reliance agreements from the participating institutions. Given the retrospective design and use of fully de-identified data, the requirement for informed consent was waived. The study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Study Population

Patients aged ≥ 18 years undergoing elective isolated CABG with cardiopulmonary bypass (CPB) were eligible for inclusion. Exclusion criteria included pre-existing dialysis-dependent renal failure, end-stage renal disease (ESRD), emergency or salvage procedures, concomitant valve or major aortic surgery, prior renal transplantation, active infection or sepsis at the time of surgery, exposure to nephrotoxic agents (including NSAIDs, aminoglycosides, amphotericin B, or high-dose vancomycin) or intravenous contrast within 7 days before surgery, and incomplete key perioperative or laboratory data.

A total of 250 patients met the inclusion criteria and were included in the final analysis.

Perioperative Management

All procedures were performed via median sternotomy using standard CPB techniques with non-pulsatile flow, moderate hypothermia (32–34°C), and alpha-stat blood gas management. Systemic anticoagulation was achieved using unfractionated heparin (target activated clotting time > 480 seconds). Myocardial protection was provided using intermittent cold blood cardioplegia. Perioperative management, including anesthetic strategy, hemodynamic monitoring, vasoactive support, and postoperative intensive care unit (ICU) management, followed standardized institutional protocols. Clinical decisions were made by the clinical team.

Data Collection

Data were extracted retrospectively from electronic medical records, anesthesia information systems, ICU charts, and institutional surgical databases by trained investigators using a standardized case report form. Data abstractors were blinded to the primary outcome status to minimize ascertainment bias. Data collection included demographic characteristics, comorbidities, preoperative cardiac function, intraoperative variables, and early postoperative biochemical and hemodynamic parameters. Data accuracy was ensured by double-entry verification in a random 10% sample.

Variables Assessed

Continuous physiological variables were dichotomized based on established clinical guidelines and widely accepted thresholds in cardiac surgery literature to facilitate clinical applicability and bedside risk stratification. Specifically, eGFR < 60 mL/min/1.73 m² (KDIGO threshold for CKD), LVEF < 40% (threshold for severe systolic dysfunction), MAP < 65 mmHg (standard hemodynamic resuscitation target), lactate > 2.0 mmol/L (threshold for hyperlactatemia/tissue hypoperfusion), and base excess < − 5 mmol/L (threshold for significant metabolic acidosis) were selected a priori based on prior prognostic studies [9, 13, 15].

Preoperative Variables

Collected variables included age, sex, body mass index (BMI), diabetes mellitus, hypertension, baseline serum creatinine (defined as the lowest outpatient value within 30 days prior to surgery, excluding values obtained during acute illness), estimated glomerular filtration rate (eGFR) calculated using the CKD-EPI equation, and left ventricular ejection fraction (LVEF) assessed by transthoracic echocardiography.

Intraoperative Variables

Intraoperative data included cardiopulmonary bypass (CPB) duration, aortic cross-clamp time, mean arterial pressure (MAP) recorded at 5-minute intervals, cumulative duration of intraoperative hypotension (defined as MAP < 65 mmHg sustained for ≥ 20 minutes), total vasopressor and inotropic support requirements, and intraoperative red blood cell transfusion volume.

Postoperative Variables

Early postoperative variables obtained within 1 hour of ICU admission included serum lactate, arterial blood gas parameters, and base excess. Hyperlactatemia was defined as lactate > 2.0 mmol/L, and metabolic acidosis was defined as base excess < − 5 mmol/L. Requirement for mechanical circulatory support, including intra-aortic balloon pump (IABP) or extracorporeal membrane oxygenation (ECMO), was recorded. Hourly urine output was monitored for the first 24 hours.

Outcome Definitions

Primary Outcome

The primary outcome was postoperative acute kidney injury (AKI) occurring within 72 hours of surgery, defined according to kidney disease: Improving Global Outcomes (KDIGO) criteria based on changes in serum creatinine relative to baseline. Although urine output data were collected and reviewed, the primary diagnostic definition of AKI was based on serum creatinine according to the KDIGO criteria. Urine output criteria were considered supportive rather than diagnostic because protocolized

postoperative fluid resuscitation and intravenous diuretic use after CABG may substantially confound urine output as an independent marker of acute kidney injury. AKI severity staged per KDIGO (Stage 1/2/3) was also analyzed as a secondary outcome.

Because not all postoperative AKI is attributable to acute cardiac dysfunction, cases fulfilling prespecified diagnostic criteria for cardiorenal syndrome type 1 (CRS-1) were identified through blinded clinical adjudication as a predefined secondary classification. This approach enabled differentiation between hemodynamically mediated AKI consistent with CRS-1 and AKI resulting from other postoperative causes.

Cardiorenal Syndrome Type 1 (CRS-1)

CRS-1 was defined as KDIGO-defined AKI occurring in the setting of acute postoperative cardiac dysfunction or hemodynamic compromise, including at least one of the following:

(1) low cardiac output syndrome requiring continuous vasopressor or inotropic support (norepinephrine equivalent ≥ 0.05 μg/kg/min for > 24 hours),

(2) need for mechanical circulatory support (IABP or ECMO), or

(3) postoperative LVEF < 40% within 48 hours of AKI diagnosis.

Cardiac dysfunction was required to precede or occur concurrently with AKI onset within 24 hours. Alternative causes of AKI, including sepsis (Sepsis-3 criteria), obstructive uropathy, nephrotoxic drug exposure, and contrast-induced nephropathy, were systematically excluded.

Secondary Outcomes

Secondary outcomes included in-hospital mortality, ICU length of stay, total hospital length of stay, need for renal replacement therapy (RRT), and duration of mechanical ventilation.

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation or median (interquartile range) depending on distribution assessed by the Shapiro–Wilk test. Categorical variables were expressed as frequencies and percentages. Group comparisons were performed using Student's t-test or Mann–Whitney U test for continuous variables and Chi-square or Fisher's exact test for categorical variables.

Univariable logistic regression was used to identify candidate predictors of AKI. Variables with $p < 0.10$ and those considered clinically relevant a priori were entered into a multivariable logistic regression model to identify independent predictors. Prespecified variables included age, diabetes mellitus, preoperative eGFR < 60 mL/min/1.73 m², LVEF < 40%, CPB duration > 120 minutes, intraoperative hypotension, postoperative lactate > 2.0 mmol/L, and base excess < − 5 mmol/L.

Multicollinearity was assessed using variance inflation factor (VIF < 5). Model performance was evaluated using the area under the receiver operating characteristic curve (AUC-ROC) for discrimination and the Hosmer–Lemeshow test for calibration. Results were reported as adjusted odds ratios (aOR) with 95% confidence intervals (CI).

Missing data were minimal (< 5% overall) and distributed across preoperative laboratory values and intraoperative hemodynamic recordings, with no single variable exceeding 2% missingness. These were handled using complete-case analysis; sensitivity analysis using multiple imputation by chained equations yielded consistent results. Given the retrospective design, no formal a priori sample size calculation was performed; however, of the 250 included patients, 95 (38%) developed postoperative AKI, providing an events-per-variable ratio of approximately 13.6:1 (95 events for 7 predictors), supporting model stability and reducing the risk of overfitting.

Sensitivity analyses were performed to confirm robustness of findings, including: (1) excluding patients with KDIGO Stage 1 AKI, (2) using an alternative lactate cutoff (≥ 4.0 mmol/L), and (3) repeating the primary analysis with multiple imputations for missing data.

All statistical analyses were performed using IBM SPSS Statistics version 28 (IBM Corp., Armonk, NY, USA), with additional validation analyses performed using R software version 4.2 (R Foundation for Statistical Computing, Vienna, Austria). A two-sided p-value < 0.05 was considered statistically significant.

Results

Study Population and Baseline Characteristics

Between January 2022 and December 2025, a total of 312 consecutive patients undergoing elective isolated on-pump coronary artery bypass grafting (CABG) across four tertiary cardiac surgery centers were screened for eligibility. After exclusion of 62 patients based on predefined criteria, 250 patients were included in the final analysis **(Fig. 1).** Complete data were available for all primary predictors in 247/250 patients (98.8%).

Figure 1. Study flow diagram

Figure 1. Description: Patient Screening, Enrollment, and Cohort Stratification Flowchart. A total of 312 consecutive adult patients undergoing elective isolated on-pump coronary artery bypass grafting (CABG) between January 2022 and December 2025 across four tertiary cardiac surgery centers were screened for eligibility. Following the application of predefined exclusion criteria, 62 patients were excluded, resulting in a final analytical cohort of 250 patients. The cohort was stratified by the development of postoperative acute kidney injury (AKI) within 72 hours (n = 95, 38% vs. n = 155, 62%). Subsequent clinical adjudication classified 78 AKI cases (82%) as cardiorenal syndrome type 1 (CRS-1).

Abbreviations: AKI, acute kidney injury; CABG, coronary artery bypass grafting; CRS-1, cardiorenal syndrome type 1; ESRD, end-stage renal disease.

Postoperative acute kidney injury (AKI) occurred in 95 patients (38.0%) within 72 hours of surgery, while 155 patients (62.0%) did not develop AKI. Following systematic adjudication of alternative etiologies, 78 cases (82.1% of AKI cases) were classified as cardiorenal syndrome type 1 (CRS-1), whereas 17 patients (17.9%) demonstrated mixed or non-cardiac etiologies of renal dysfunction.

Baseline demographic and clinical characteristics are summarized in Table 1. Patients who developed AKI were significantly older (mean age 68.4 ± 9.2 vs. 63.1 ± 10.5 years; $p < 0.001$) and had a higher prevalence of diabetes mellitus (62.1% vs. 37.4%; $p = 0.001$), hypertension (76.8% vs. 61.9%; $p = 0.004$), and pre-existing chronic kidney disease (eGFR < 60 mL/min/1.73 m²: 32.6% vs. 12.25%; $p < 0.001$) compared with the non-AKI cohort. Preoperative renal dysfunction was substantially more frequent among AKI patients, with a significantly lower baseline estimated glomerular filtration rate (eGFR) (mean 58.6 ± 16.1vs. 78.9 ± 18.4 mL/min/1.73 m²; $p < 0.001$).

Table 1
Baseline Demographic and Preoperative Characteristics

Variable	**AKI Group (n = 95)**	**No AKI Group (n = 155)**	**p-value**
Age, years	68.4 ± 9.2	63.1 ± 10.5	**< 0.001**
Male sex, n (%)	71 (74.7%)	118 (76.1%)	0.790
Body mass index, kg/m²	29.1 ± 4.3	28.4 ± 4.1	0.210
Diabetes mellitus, n (%)	59 (62.1%)	58 (37.4%)	**< 0.001**
Hypertension, n (%)	73 (76.8%)	96 (61.9%)	**0.018**
Chronic kidney disease, n (%)	31 (32.6%)	19 (12.25%)	**< 0.001**
Baseline serum creatinine, mg/dL	1.39 ± 0.42	1.02 ± 0.28	**< 0.001**
eGFR, mL/min/1.73 m²	58.6 ± 16.1	78.9 ± 18.4	**< 0.001**
eGFR < 60 mL/min/1.73 m², n (%)	42 (44.2%)	24 (15.5%)	**< 0.001**
LVEF, %	42.1 ± 11.3	50.3 ± 7.5	**< 0.001**
LVEF < 40%, n (%)	37 (38.9)	23 (14.8)	**< 0.001**
Preoperative anemia, n (%)	33 (34.7%)	29 (18.7%)	**0.006**
EuroSCORE II	3.9 ± 1.8	2.4 ± 1.3	**< 0.001**

Data are presented as mean ± standard deviation for continuous variables, and as count (percentage) for categorical variables. Bolded p-values indicate statistical significance at the $p < 0.05$ threshold.
Abbreviations: AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; EuroSCORE II, European System for Cardiac Operative Risk Evaluation II; LVEF, left ventricular ejection fraction.

Table 1. **Baseline Demographic and Preoperative Characteristics**

Table 1 **Description**: Data are presented as mean ± standard deviation for continuous variables, and as count (percentage) for categorical variables. Bolded p-values indicate statistical significance at the $p < 0.05$ threshold. *Abbreviations: AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; EuroSCORE II, European System for Cardiac Operative Risk Evaluation II; LVEF, left ventricular ejection fraction.*

Additionally, patients who developed AKI demonstrated significantly reduced baseline cardiac function, reflected by lower preoperative left ventricular ejection fraction (LVEF) (mean 42.1% ± 11.3% vs. 50.3% ± 7.5%; $p < 0.001$). No significant differences were observed regarding sex distribution or body mass index between groups.

Intraoperative Characteristics

Intraoperative variables are presented in Table 2. Patients who developed postoperative AKI experienced significantly longer cardiopulmonary bypass (CPB) duration (mean 128 ± 31 vs. 102 ± 24 minutes; $p < 0.001$) and prolonged aortic cross-clamp times (median 78 [IQR 65–95] vs. 62 [IQR 51–74] minutes; $p < 0.001$) compared with patients without AKI.

Table 2
Intraoperative Characteristics

Variable	AKI Group (n = 95)	No AKI Group (n = 155)	p-value
CPB duration, min	128 ± 31	102 ± 24	**< 0.001**
CPB duration > 120 min, n (%)	51 (53.7)	29 (18.7)	**< 0.001**
Aortic cross-clamp time, min	79 ± 18	63 ± 14	**< 0.001**
MAP < 65 mmHg > 20 min, n (%)	48 (50.5)	26 (16.8)	**< 0.001**
Lowest intraoperative MAP, mmHg	57 ± 8	64 ± 7	**< 0.001**
Vasopressor/inotropic support, n (%)	57 (60.0)	41 (26.5)	**< 0.001**
Intraoperative RBC transfusion, mL	420 ± 190	250 ± 120	**< 0.001**
Perioperative IABP use, n (%)	21 (22.1%)	11 (7.1%)	**< 0.001**

Data are presented as mean ± standard deviation for continuous variables, and as count (percentage) for categorical variables. Bolded p-values indicate statistical significance at the $p < 0.05$ threshold. *Abbreviations: AKI, acute kidney injury; CPB, cardiopulmonary bypass; IABP, intra-aortic balloon pump; MAP, mean arterial pressure; RBC, red blood cell.*

Table 2. **Intraoperative Characteristics**

Table 2 **Description** : Data are presented as mean ± standard deviation for continuous variables, and as count (percentage) for categorical variables. Bolded p-values indicate statistical significance at the $p < 0.05$ threshold. *Abbreviations: AKI, acute kidney injury; CPB, cardiopulmonary bypass; IABP, intra-aortic balloon pump; MAP, mean arterial pressure; RBC, red blood cell.*

Episodes of intraoperative hypotension were markedly more frequent and prolonged in the AKI group. Specifically, sustained mean arterial pressure (MAP) < 65 mmHg for more than 20 minutes occurred significantly more often among patients developing AKI (50.5% vs. 16.8%; $p < 0.001$). Furthermore, AKI patients required greater intraoperative red blood cell transfusion volumes (420 ± 190 mL vs. 250 ± 120 mL).

The use of mechanical circulatory support was also significantly higher in the AKI cohort, particularly intra-aortic balloon pump (IABP) support initiated perioperatively 21 (22.1%) vs. 11 (7.1%).

Early Postoperative Metabolic and Hemodynamic Parameters

Early postoperative metabolic profiles differed substantially between groups Table 3. Patients who developed AKI had a significantly higher prevalence of hyperlactatemia upon ICU admission (69 [72.6%] vs. 38 [24.5%]; $p < 0.001$) and more pronounced metabolic acidosis, reflected by more negative base excess values.

Table 3
Early Postoperative Metabolic and Hemodynamic Parameters

Variable	AKI Group (n = 95)	No AKI Group (n = 155)	p-value
ICU admission lactate, mmol/L	3.8 ± 1.5	1.9 ± 0.8	< 0.001
Lactate > 2.0 mmol/L, n (%)	69 (72.6%)	38 (24.5%)	< 0.001
Base excess, mmol/L	−6.1 ± 3.0	−2.8 ± 2.1	< 0.001
Base excess < − 5 mmol/L, n (%)	53 (55.8%)	27 (17.4%)	< 0.001
Mechanical circulatory support, n (%)	18 (18.9%)	5 (3.2%)	< 0.001
Vasopressor support > 24 h, n (%)	46 (48.4%)	18 (11.6%)	< 0.001
Urine output first 24 h, mL	980 ± 310	1650 ± 420	< 0.001

Data obtained within 1 hour following intensive care unit (ICU) admission. Continuous variables are expressed as mean ± standard deviation, and categorical variables are expressed as counts and percentages. Bolded p-values indicate statistical significance at the $p < 0.05$ threshold. *Abbreviations: AKI, acute kidney injury; ICU, intensive care unit.*

Table 3. **Early Postoperative Metabolic and Hemodynamic Parameters**

Table 3 **Description**: Data obtained within 1 hour following intensive care unit (ICU) admission. Continuous variables are expressed as mean ± standard deviation, and categorical variables are expressed as counts and percentages. Bolded p-values indicate statistical significance at the $p < 0.05$ threshold. *Abbreviations: AKI, acute kidney injury; ICU, intensive care unit.*

Hyperlactatemia (serum lactate > 2.0 mmol/L) was observed in 72.6% of AKI patients and was strongly associated with postoperative renal dysfunction. Similarly, metabolic acidosis at ICU admission (base excess < − 5 mmol/L) was significantly more common among AKI patients (55.8% vs. 17.4%; $p < 0.001$).

Patients developing AKI also demonstrated lower postoperative urine output during the first 24 postoperative hours (980 ± 310 mL/24 h vs. 1650 ± 420 mL/24 h; $p < 0.001$) and required prolonged vasopressor support more frequently (48.4% vs. 11.6%; $p < 0.001$).

Incidence and Severity of Acute Kidney Injury

According to KDIGO staging criteria, stage 1 AKI occurred in 54 patients (56.8%), stage 2 in 28 patients (29.5%), and stage 3 in 13 patients (13.7%) **(Fig. 2)**. Renal replacement therapy (RRT) was required in 5 patients (5.3% of AKI cases; 2.0% of the overall cohort), all of whom belonged to the CRS-1 subgroup and demonstrated severe postoperative hemodynamic compromise.

Figure 2. KDIGO Staging Distribution of Postoperative AKI

Figure 2. Description: Distribution of postoperative acute kidney injury (AKI) severity according to the kidney disease: Improving Global Outcomes (KDIGO) criteria (N = 95). Data are presented as percentages and absolute frequencies (n).

Among the 78 patients classified as CRS-1, post-hoc exploratory stratification based on hemodynamic severity identified:

- **-** Isolated cardiac dysfunction without prolonged vasopressor dependency (Type 1a) in 22 patients (28.2%),
- - Vasopressor-dependent CRS-1 (Type 1b) in 38 patients (48.7%), and
- - CRS-1 requiring mechanical circulatory support (Type 1c) in 18 patients (23.1%).

Patients with Type 1c CRS-1 demonstrated the highest rates of severe AKI (KDIGO stage 3: 44.4% vs. 10.5% Type 1b vs. 4.5% Type 1a; $p < 0.001$), prolonged ICU stay (mean 12.3 ± 5.1 vs. 7.8 ± 3.2 vs. 5.1 ± 2.4 days; $p < 0.001$), and in-hospital mortality (33.3% vs. 15.8% vs. 4.5%; $p = 0.008$).

Predictors of Postoperative AKI

Univariable logistic regression analysis identified several variables associated with postoperative AKI, including advanced age, diabetes mellitus, reduced eGFR, impaired LVEF, prolonged CPB duration, intraoperative hypotension, postoperative hyperlactatemia, metabolic acidosis, and perioperative vasopressor requirement.

In multivariable logistic regression analysis (Table 4), independent predictors of postoperative AKI included: Preoperative eGFR < 60 mL/min/1.73 m² (adjusted odds ratio [aOR] 3.2; 95% confidence interval [CI] 1.8–5.7; p < 0.001), LVEF < 40% (aOR 2.7; 95% CI 1.5–4.8; p = 0.002), Intraoperative MAP < 65 mmHg sustained for > 20 minutes (aOR 2.9; 95% CI 1.6–5.2; p < 0.001), ICU admission lactate > 2.0 mmol/L (aOR 3.5; 95% CI 1.9–6.3; p < 0.001), metabolic acidosis with base excess < − 5 mmol/L (aOR 2.4; 95% CI 1.3–4.3; p = 0.004),CPB duration > 120 minutes (aOR 2.1; 95% CI 1.2–3.8; p = 0.011), and Preoperative diabetes mellitus (aOR 1.9; 95% CI 1.1–3.4; p = 0.028).

Table 4
Multivariable Logistic Regression Analysis for Predictors of Postoperative AKI

Variable	Adjusted Odds Ratio (aOR)	95% Confidence Interval	P-value
eGFR < 60 mL/min/1.73 m²	3.2	1.8–5.7	**< 0.001**
LVEF < 40%	2.7	1.5–4.8	**0.002**
MAP < 65 mmHg > 20 min	2.9	1.6–5.2	**< 0.001**
Lactate > 2.0 mmol/L	3.5	1.9–6.3	**< 0.001**
Base excess < − 5 mmol/L	2.4	1.3–4.3	**0.004**
CPB duration > 120 min	2.1	1.2–3.8	**0.011**
Diabetes mellitus	1.9	1.1–3.4	**0.028**
Model discrimination: AUC-ROC = 0.84 (95% CI 0.78–0.89) Hosmer–Lemeshow goodness-of-fit test: p = 0.41			

No clinically significant multicollinearity was detected among candidate predictors (all variance inflation factors < 2.5).

Table 4. **Multivariable Logistic Regression Analysis for Predictors of Postoperative AKI**

Table 4 **Description**: Model discrimination: AUC-ROC = 0.84 (95% CI 0.78–0.89). Hosmer–Lemeshow goodness-of-fit test: p = 0.41.

Model discrimination for prediction of postoperative AKI was good, with an apparent area under the receiver operating characteristic curve (AUC-ROC) of 0.84 (95% CI 0.78–0.89), indicating good discriminative ability (Fig. 3). It should be noted that this apparent AUC is uncorrected for potential overfitting.

Sensitivity analyses excluding patients with baseline chronic kidney disease demonstrated consistent findings without significant attenuation of effect sizes. Results remained consistent when using an alternative lactate cutoff (≥ 4.0 mmol/L) or after multiple imputation for missing data.

Clinical Outcomes

Clinical outcomes are summarized in Table 5. Patients developing postoperative AKI experienced significantly worse postoperative outcomes compared with the non-AKI cohort.

Table 5
Postoperative Clinical Outcomes

Outcome	AKI Group (n = 95)	No AKI Group (n = 155)	p-value
In-hospital mortality, n (%)	14 (14.7%)	6 (3.9%)	**0.010**
ICU stay, days	7.8 ± 3.6	3.9 ± 1.8	**< 0.001**
Total hospital stay, days	14.2 ± 5.1	8.7 ± 3.2	**< 0.001**
Mechanical ventilation duration, h	22 ± 11	11 ± 6	**< 0.001**
Renal replacement therapy, n (%)	5 (5.3)	0 (0)	**0.003**
Persistent AKI (> 48 h), n (%)	39 (41.1)	—	—

Data are presented as mean ± standard deviation for continuous variables, and as count (percentage) for categorical variables. Bolded p-values indicate statistical significance at the p < 0.05 threshold. *Abbreviations: AKI, acute kidney injury; ICU, intensive care unit.*

In-hospital mortality was significantly higher among AKI patients (14/95 [14.7%] vs. 6/155 [3.9%]; p = 0.01). AKI was also associated with prolonged ICU stay (mean 7.8 ± 3.6 vs. 3.9 ± 1.8 days; p < 0.001), longer total hospital stay (mean 14.2 ± 5.1 vs. 8.7 ± 3.2 days; p < 0.001), prolonged duration of mechanical ventilation (22 ± 11 vs. 11 ± 6 hours; p < 0.001), and increased requirement for renal replacement therapy (5.3% vs. 0%; p = 0.008).

Within the CRS-1 subgroup, patients requiring mechanical circulatory support (Type 1c phenotype) demonstrated the poorest prognosis, including the highest incidence of severe AKI, prolonged ventilatory support, and mortality (33.3% vs. 15.8% Type 1b vs. 4.5% Type 1a; p = 0.008).

Figure 3. ROC Curve for Prediction Model

Figure 3. Description: Receiver operating characteristic (ROC) curve for the multivariable prediction model of postoperative acute kidney injury (AKI). The model demonstrated good discriminative ability with an area under the curve (AUC) of 0.84 (95% CI: 0.78–0.89). The dashed diagonal line represents chance performance (AUC = 0.50).

Table 5. **Postoperative Clinical Outcomes**

Table 5 **Description**: Data are presented as mean ± standard deviation for continuous variables, and as count (percentage) for categorical variables. Bolded p-values indicate statistical significance at the p < 0.05 threshold. *Abbreviations: AKI, acute kidney injury; ICU, intensive care unit.*

Discussion

In this multicenter retrospective cohort study of 250 patients undergoing elective on-pump CABG, we identified seven independent clinical and metabolic predictors of postoperative AKI, predominantly occurring in the setting of CRS-1. Notably, 78 of the 95 AKI cases (82.1%) fulfilled the prespecified diagnostic criteria for cardiorenal syndrome type 1 (CRS-1), indicating that CRS-1 represented the predominant AKI phenotype in our cohort. Postoperative AKI occurred in 38% of patients, with CRS-1 accounting for 82% of cases. Preoperative eGFR < 60 mL/min/1.73 m², LVEF < 40%, intraoperative mean arterial pressure (MAP) < 65 mmHg sustained for > 20 minutes, ICU admission hyperlactatemia (lactate > 2.0 mmol/L), persistent metabolic acidosis (base excess < − 5 mmol/L), CPB duration > 120 minutes, and preoperative diabetes mellitus were each independently associated with a 1.9-to-3.5-fold increased odds of AKI.

The predictive model demonstrated good discrimination (AUC 0.84) and acceptable calibration. Importantly, AKI was associated with a nearly four-fold higher in-hospital mortality (14.7% vs. 3.9%) and significantly prolonged ICU and hospital length of stay. These findings underscore the multifactorial pathophysiology of postoperative AKI, which predominantly occurred in the setting of CRS-1 in our cohort, and highlight readily available bedside parameters that can be integrated into perioperative risk stratification.

Comparison with Previous Studies and Interpretation of Findings

Our observed AKI incidence of 38% aligns with the widely reported range of 20–40% following cardiac surgery [3, 4, 12]. Previous studies largely evaluated cardiac surgery-associated acute kidney injury (CS-AKI) irrespective of the underlying mechanism, whereas the present study systematically adjudicated the proportion of postoperative AKI attributable to cardiorenal syndrome type 1 (CRS-1). The predominance of CRS-1 (82% of AKI cases) in our cohort reflects the expected pathophysiology in elective CABG, where acute cardiac dysfunction and hemodynamic compromise—rather than sepsis or nephrotoxin exposure—predominantly drive renal injury. This observation is consistent with the conceptual framework proposed by Ronco et al., which emphasizes the interplay among reduced cardiac output, venous congestion, and neurohormonal activation in CRS-1 pathogenesis [1, 2].

It is important to acknowledge the semantic nuances of applying the term 'CRS-1' to the post-cardiopulmonary bypass setting. Classic CRS-1 describes AKI secondary to abrupt primary worsening of cardiac function. In contrast, post-CABG AKI is highly multifactorial, driven not only by reduced cardiac output but also by CPB-induced systemic inflammation, endothelial dysfunction, and ischemia-reperfusion injury. Therefore, our use of the term 'CRS-1' refers specifically to the hemodynamically mediated phenotype of Cardiovascular Surgery-Associated AKI (CS-AKI), where acute post-cardiotomy myocardial stunning or low cardiac output syndrome is the predominant clinical driver of renal hypoperfusion, aligning with the broader cardio-renal interaction framework [29].

Preoperative renal dysfunction (eGFR < 60 mL/min/1.73 m²) emerged as the strongest baseline predictor (adjusted odds ratio [aOR] 3.2). This finding is biologically plausible: even mild chronic kidney disease reduces renal functional reserve and impairs autoregulatory capacity, rendering the kidney more vulnerable to perioperative insults such as hypotension, inflammation, or oxidative stress [7, 13]. Our results support the use of baseline eGFR—rather than serum creatinine alone—for preoperative risk assessment.

Preoperative LVEF < 40% (aOR 2.7) was another independent predictor. Reduced ejection fraction predisposes to low cardiac output syndrome, which diminishes renal perfusion pressure and may activate systemic inflammatory and neurohormonal cascades that directly injure tubular epithelium [5, 27]. Notably, one-third of CRS-1 patients requiring vasopressor support had preserved LVEF, suggesting that subtle or transient systolic dysfunction—not captured by a single preoperative echocardiogram—may still contribute to AKI. This finding underscores the value of dynamic postoperative hemodynamic assessment over static baseline measures.

Intraoperative hypotension (MAP < 65 mmHg for > 20 minutes; aOR 2.9) has been consistently associated with postoperative AKI in both cardiac and non-cardiac surgery [7, 11, 15–17]. Our study extends this evidence by quantifying a specific threshold (65 mmHg) and duration (> 20 minutes) that independently predict AKI after CABG. The underlying mechanism is likely multifactorial: reduced renal perfusion pressure directly impairs glomerular filtration, while prolonged hypotension may exacerbate cardiopulmonary bypass–induced systemic inflammation and endothelial dysfunction [14, 26].

Postoperative hyperlactatemia (lactate > 2.0 mmol/L; aOR 3.5) was the strongest independent predictor overall. While lactate serves as a marker of global tissue hypoperfusion, in the cardiac surgery context it may also reflect impaired splanchnic perfusion, reduced hepatic clearance, or adrenergic stimulation [9, 24]. Importantly, hyperlactatemia was present in nearly three-quarters of AKI patients, and the association remained robust after adjustment for hypotension and other confounders. This suggests that lactate provides incremental prognostic information beyond blood pressure, potentially capturing a state of "occult hypoperfusion" that may precede or accompany renal injury [18, 19]. Sensitivity analysis using a higher lactate cutoff (≥ 4.0 mmol/L) yielded consistent results, indicating that even mild hyperlactatemia carries clinical relevance.

Persistent metabolic acidosis (base excess < − 5 mmol/L; aOR 2.4) was an independent predictor that has received limited attention in cardiac surgery literature. Although secondary to hyperlactatemia, metabolic acidosis may directly contribute to AKI through tubular cell injury, impaired intracellular pH regulation, and activation of pro-inflammatory pathways [20, 25]. As base excess is routinely available from arterial blood gas analysis, it may serve as an early warning marker of hemodynamically mediated postoperative AKI.

Clinical Implications

Our findings offer several practical implications for perioperative management:

- **Early risk stratification**: Integrating the seven identified predictors (eGFR, LVEF, intraoperative hypotension, lactate, base excess, diabetes and CPB duration) into a simple bedside risk score could enable identification of high-risk patients before AKI becomes clinically apparent. Such a tool would be inexpensive and widely applicable, leveraging routine laboratory and hemodynamic data.
- **Intraoperative hemodynamic targets**: Our data support maintaining MAP ≥ 65 mmHg throughout the procedure whenever feasible. Although the optimal "renal perfusion pressure" target remains debated, avoiding prolonged hypotension below this threshold appears beneficial for renal protection [15, 16].
- **Postoperative monitoring**: ICU admission lactate and base excess should be regarded as critical early warning signs. Patients with lactate > 2.0 mmol/L or base excess < − 5 mmol/L—even in the absence of overt hypotension—warrant intensified AKI surveillance, including frequent serum creatinine monitoring and avoidance of additional nephrotoxic exposures [18, 19, 24].
- **Hemodynamic severity subtyping of CRS-1**: Because most AKI cases represented CRS-1, the following clinical implications primarily apply to the hemodynamically mediated phenotype of postoperative AKI. Our exploratory classification into Type 1a (isolated cardiac dysfunction), Type 1b (vasopressor-dependent), and Type 1c (mechanical circulatory support) revealed a gradient of worsening outcomes. This simple bedside framework may help characterize hemodynamic severity and guide closer renal monitoring; however, its therapeutic implications remain exploratory and require prospective validation.

Strengths and Limitations

Given the retrospective design, no formal a priori sample size calculation was performed; however, of the 250 included patients, 95 (38%) developed postoperative AKI, providing an events-per-variable ratio of approximately 13.6:1 (95 events for 7 predictors included in the final multivariable model), supporting model stability and reducing the risk of overfitting.

Several limitations warrant consideration. First, the retrospective design precludes causal inference; observed associations may be influenced by unmeasured confounders (e.g., specific inotrope use, fluid balance, central venous pressure, or renal resistive index). Second, although urine output data were collected, the primary AKI definition relied on serum creatinine-based KDIGO criteria because postoperative fluid administration and diuretic therapy after CABG can substantially confound urine output measurements. While this approach was chosen because post-CABG patients frequently receive protocolized fluid resuscitation and intravenous diuretics which confound urine output, it may have underestimated AKI severity in some patients [21, 31]. Third, lactate and base excess were measured at a single time point (ICU admission). While this captures the initial nadir of perfusion, it does not account for lactate clearance or the dynamic metabolic recovery over the first 6 to 12 hours, which are often more

powerful predictors of tissue perfusion recovery and clinical outcomes in the critical care setting [30]. Fourth, we did not evaluate novel AKI biomarkers (e.g., NGAL, KIM-1, cystatin C), which could enable earlier detection and offer deeper pathophysiological insights [10, 22, 23, 28]. Fifth, despite consecutive enrollment, the retrospective nature may have introduced selection bias. Sixth, our cohort comprised exclusively elective on-pump CABG patients; findings may not generalize to off-pump CABG, valve surgery, or emergency procedures. Seventh, our proposed CRS-1 subtyping (Types 1a–1c) represents a continuum of hemodynamic failure and vasopressor dependency rather than distinct biological phenotypes; this exploratory classification requires prospective validation to determine if it truly identifies distinct pathophysiological trajectories or merely reflects the severity of post-cardiotomy shock. Finally, our predictive model was not internally validated using bootstrap resampling, meaning the reported AUC may be optimistic due to overfitting. External validation in an independent cohort is therefore strictly required before our predictive model can be recommended for routine clinical implementation.

Future Directions

Prospective multicenter studies should validate our predictive model across diverse cardiac surgery populations. Incorporating serial lactate and base excess measurements, combined with dynamic assessments of renal perfusion (e.g., Doppler-derived renal resistive index or near-infrared spectroscopy), may enhance real-time risk prediction. Randomized controlled trials are needed to determine whether lactate- or acidosis-guided interventions—such as targeted fluid resuscitation, inotrope titration, or early renal replacement therapy—can attenuate AKI progression. Additionally, the proposed phenotypic CRS-1 classification (Types 1a–1c) should be prospectively validated to evaluate its utility in guiding personalized therapy.

Conclusions

Postoperative AKI after elective CABG is common. In our cohort, more than 80% of AKI cases fulfilled prespecified criteria for cardiorenal syndrome type 1 (CRS-1), highlighting the major contribution of cardio-renal interactions in postoperative renal dysfunction. AKI was independently predicted by readily available preoperative (eGFR < 60 mL/min/1.73 m², LVEF < 40%, diabetes mellitus), intraoperative (MAP < 65 mmHg for > 20 min, CPB duration > 120 min), and early postoperative (lactate > 2.0 mmol/L, base excess < − 5 mmol/L) parameters. The exploratory CRS-1 subtyping framework may provide additional characterization of hemodynamic severity but requires prospective validation before clinical implementation.

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of New Valley University (approval no. 20260430010). Given the retrospective

design and use of fully de-identified data, the requirement for informed consent was waived by the ethics committee.

Consent to Publish declaration

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data availability

The datasets generated and/or analyzed during the current study are not publicly available due to privacy and ethical restrictions but may be available from the corresponding author on reasonable request, subject to applicable institutional and ethical requirements.

Authors' contributions

Ahmed Hamdy Lamloum: Conceptualization, study design, data collection, data analysis, interpretation of the results, and manuscript drafting. Hosam AlamEdien: Data collection, clinical assessment, interpretation of the results, and critical revision of the manuscript. Ahmed M. Helmy: Data collection, statistical analysis, interpretation of the results, and critical revision of the manuscript. Doaa Elwasly: Data collection, clinical assessment, interpretation of the results, and critical revision of the manuscript. All authors read and approved the final manuscript.

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Figures

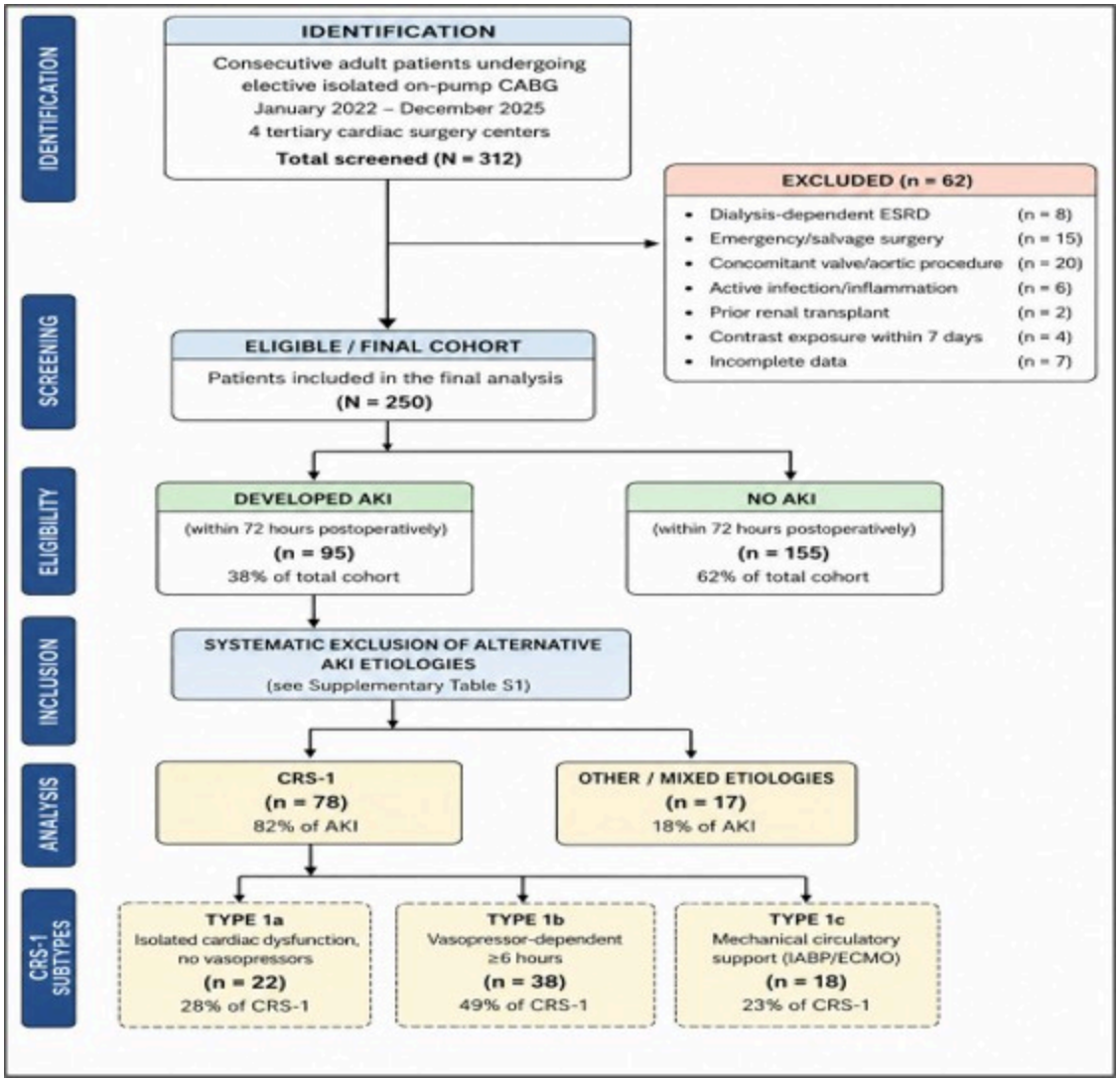

Figure 1

Study flow diagram

Figure 1. Description: Patient Screening, Enrollment, and Cohort Stratification Flowchart. A total of 312 consecutive adult patients undergoing elective isolated on-pump coronary artery bypass grafting (CABG) between January 2022 and December 2025 across four tertiary cardiac surgery centers were screened for eligibility. Following the application of predefined exclusion criteria, 62 patients were excluded, resulting in a final analytical cohort of 250 patients. The cohort was stratified by the development of postoperative acute kidney injury (AKI) within 72 hours (n = 95, 38% vs. n = 155, 62%). Subsequent clinical adjudication classified 78 AKI cases (82%) as cardiorenal syndrome type 1 (CRS-1).
Abbreviations: AKI, acute kidney injury; CABG, coronary artery bypass grafting; CRS-1, cardiorenal syndrome type 1; ESRD, end-stage renal disease.

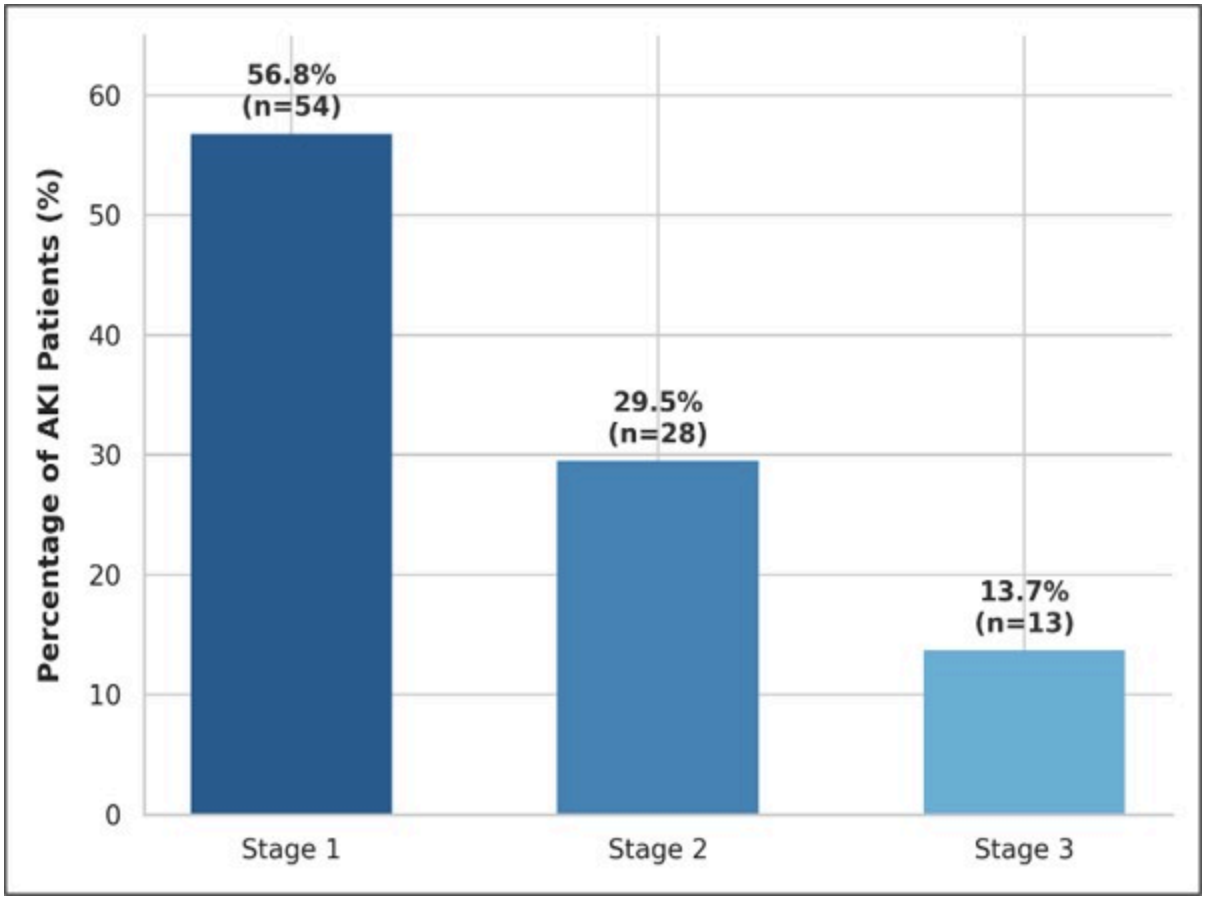

Figure 2

KDIGO Staging Distribution of Postoperative AKI

Figure 2. Description: Distribution of postoperative acute kidney injury (AKI) severity according to the kidney disease: Improving Global Outcomes (KDIGO) criteria (N = 95). Data are presented as percentages and absolute frequencies (n).

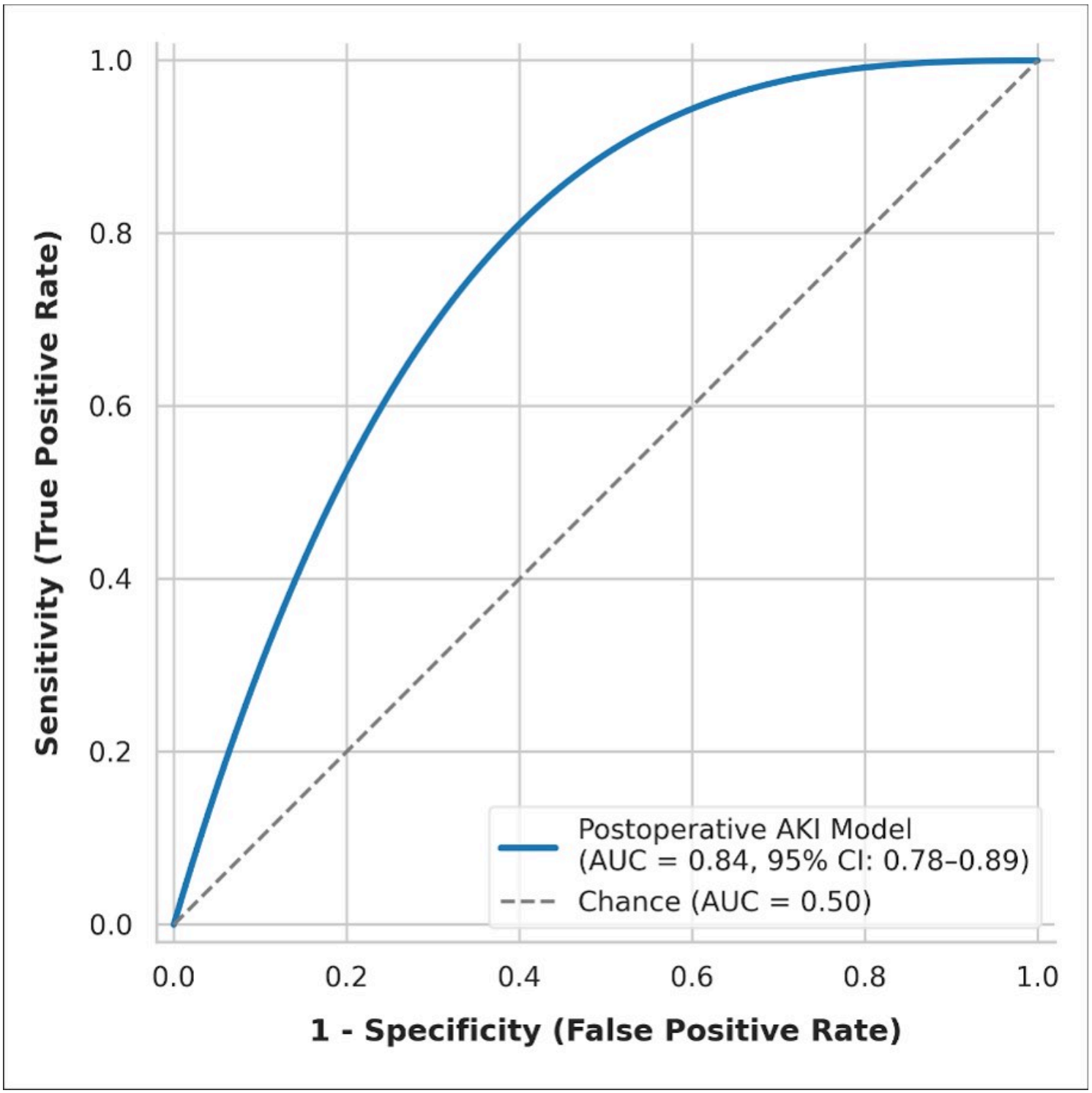

Figure 3

ROC Curve for Prediction Model

Figure 3. Description: Receiver operating characteristic (ROC) curve for the multivariable prediction model of postoperative acute kidney injury (AKI). The model demonstrated good discriminative ability with an area under the curve (AUC) of 0.84 (95% CI: 0.78–0.89). The dashed diagonal line represents chance performance (AUC = 0.50).