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Research Article

Prediction of postoperative acute kidney injury after cardiopulmonary bypass cardiac surgery: The role of preoperative inflammatory indices and intraoperative perfusion stress

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ABSTRACT

Background: Postoperative acute kidney injury is a frequent complication after cardiac surgery performed with cardiopulmonary bypass and is closely associated with increased mortality. This study aimed to evaluate the predictive value of preoperative renal function, routinely available inflammatory indices, and intraoperative perfusion stress–related parameters for the development of postoperative acute kidney injury in adult cardiac surgery.

Methods: This single-center retrospective cohort study included 455 adult patients aged 18 years or older who underwent elective or urgent coronary artery bypass grafting, valve surgery, or combined procedures with cardiopulmonary bypass between January 1, 2025 and January 1, 2026. Postoperative acute kidney injury was defined according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria by comparing the highest serum creatinine value measured within the first 72 postoperative hours with the preoperative creatinine level obtained within 48 hours before surgery. Preoperative inflammatory indices derived from routine blood tests and intraoperative perfusion-related variables were recorded. Independent predictors of acute kidney injury were identified using parsimonious multivariable logistic regression analysis, and discriminative performance was assessed by receiver operating characteristic analysis.

Results: Postoperative acute kidney injury occurred in 216 patients (47.5%), and 12.7% developed stage two or higher acute kidney injury. In-hospital mortality was significantly higher in patients with acute kidney injury compared with those without acute kidney injury (25.5% versus 6.7%), increasing progressively with advancing severity. Advanced age, higher preoperative serum creatinine, and longer cardiopulmonary bypass duration were independently associated with postoperative acute kidney injury. For stage two or higher acute kidney injury, age and cardiopulmonary bypass duration remained independent predictors. Although inflammatory indices were associated with acute kidney injury, they did not retain independent predictive value in adjusted models. Cardiopulmonary bypass duration demonstrated the strongest discriminative performance for KDIGO stage $\geqslant$2 acute kidney injury.

Conclusions: Postoperative acute kidney injury is common after cardiac surgery with cardiopulmonary bypass and is strongly associated with increased in-hospital mortality. Age, baseline renal function, and cardiopulmonary bypass duration are key determinants of postoperative acute kidney injury, highlighting intraoperative perfusion stress as a clinically relevant and potentially modifiable risk factor.

Trial Registration: The study was registered at ClinicalTrials.gov (ID: NCT05405049).

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1. Introduction

Acute kidney injury (AKI) is a common complication after cardiac surgery performed with cardiopulmonary bypass (CPB) and represents a major clinical problem that substantially influences both early postoperative morbidity and long-term prognosis. The occurrence of AKI after cardiac surgery is closely associated with increased mortality and morbidity, prolonged intensive care unit and hospital length of stay, and higher healthcare costs. In the literature, the incidence of AKI after CPB-assisted cardiac surgery has been reported to be approximately 20–30%, and both short- and long-term mortality increase significantly, particularly among patients who develop severe AKI [1, 2].

During CPB, several mechanisms—including non-pulsatile flow, hemodilution, hypothermia, systemic inflammatory response, and ischemia–reperfusion injury—may adversely affect renal blood flow and oxygen delivery, thereby triggering kidney injury [3]. However, the observation that AKI does not develop in all patients despite similar surgical and perfusion conditions suggests that individual risk profiles, preoperative renal reserve, inflammatory status, and intraoperative perfusion stress may be decisive determinants of AKI development [4]. Therefore, early identification of high-risk patients using readily available preoperative and intraoperative parameters is clinically important to guide targeted preventive strategies.

In recent years, accessible and low-cost inflammatory indices derived from routine complete blood count and basic biochemistry tests have emerged as potential biomarkers for AKI. In particular, parameters such as the neutrophil-to-lymphocyte ratio (NLR), the systemic immune-inflammation index (SII), and the C-reactive protein-to-albumin ratio have been reported to be associated with AKI after cardiac surgery, with cohort studies and systematic reviews indicating that higher values correlate with an increased risk of AKI [5, 6]. Nevertheless, much of the existing evidence evaluates these indices in isolation, whereas studies that integrate them—within the same patient cohort and within the same multivariable modeling framework—together with intraoperative markers of perfusion stress, such as CPB duration, lactate levels, and metabolic acidosis, remain limited. Yet, AKI is a multifactorial outcome arising from the interaction between preoperative susceptibility (renal reserve and an inflammatory milieu) and intraoperative triggers (perfusion stress); simultaneous assessment of these axes may strengthen clinical risk stratification.

The aim of this retrospective cohort study was to investigate the independent predictive value of preoperative indicators of renal function and inflammatory biomarkers, together with intraoperative perfusion stress–related parameters, for postoperative AKI after cardiac surgery performed with CPB. We hypothesized that incorporating preoperative inflammatory indices and renal reserve in conjunction with intraoperative markers of perfusion stress would improve the accuracy of AKI risk prediction. A key strength of the present study is the concurrent evaluation of routinely available preoperative inflammatory indices and intraoperative perfusion stress–related markers within the same cohort and within the same multivariable modeling framework. By integrating "preoperative susceptibility" and "intraoperative exposure" axes—often examined separately in prior reports—we sought to provide a more holistic and clinically applicable framework for AKI risk stratification. This approach enables a structured evaluation of interacting risk domains within a single multivariable model.

Unlike previous studies that have primarily examined inflammatory indices or intraoperative variables in isolation, the present study integrates preoperative inflammatory biomarkers, baseline renal reserve, and intraoperative perfusion stress parameters within the same multivariable framework. By simultaneously evaluating these interacting domains, this study provides a structured risk model that reflects the multifactorial pathophysiology of AKI after CPB. Moreover, the identification of CPB duration and baseline creatinine as dominant determinants, despite the presence of inflammatory signals, refines the relative weight of modifiable versus non-modifiable risk components in perioperative risk stratification.

2. Materials and Methods

2.1. Study design, setting, and ethical approval

This single-center retrospective cohort study was conducted at a tertiary-care university hospital between January 1, 2025 and January 1, 2026. Institutional Ethics Committee approval was obtained (approval number: B.30.2.ATA.0.01.00/932). The study was registered at ClinicalTrials.gov (ID: NCT05405049) for transparency purposes in the context of a retrospective observational design. The study was performed in accordance with the principles of the Declaration of Helsinki. Due to the retrospective nature of the study and the use of anonymized data, informed consent was waived. The study period was defined a priori as January 1, 2025 to January 1, 2026, and the cohort was assembled by consecutive case identification from the institutional electronic records.

2.2. Patient population and eligibility criteria

Adult patients (≥18 years) who underwent elective or urgent cardiac surgery with cardiopulmonary bypass (CPB) between January 1, 2025 and January 1, 2026 at Atatürk University Research Hospital were screened for eligibility. Eligible procedures included coronary artery bypass grafting, valve surgery, or combined coronary and valve operations performed under CPB. Both elective and emergency surgical cases performed under CPB during the study period were included in order to reflect routine clinical practice.

Patients were consecutively identified from institutional electronic surgical records to minimize selection bias.

The following exclusion criteria were applied a priori:

1. Preoperative end-stage renal disease requiring chronic dialysis,
2. Preoperative serum creatinine >4.0 mg/dL (to avoid inclusion of advanced renal failure cases that may confound AKI adjudication),
3. Off-pump cardiac surgery,

4. Congenital or non-standard cardiac procedures (e.g., atrial septal defect repair, surgery for intracardiac mass or thrombus),
5. Emergency cardiac trauma surgery,
6. Missing preoperative or postoperative serum creatinine measurements.

2.3. Data sources and data collection

Data were retrospectively obtained from the hospital information management system, anesthesia records, and the laboratory information system. Detailed intraoperative ultrafiltration practices and perioperative diuretic administration (including dose and duration), as well as renal replacement therapy parameters, were not uniformly accessible through the electronic database and were therefore not incorporated into the analysis. Detailed intraoperative hemodynamic variables (e.g., continuous mean arterial pressure, cardiac output, pump flow targets) and vasoactive/inotropic drug doses and duration were not uniformly accessible in the electronic database and were therefore not included in the analysis. The following variables were recorded for each patient:

Demographic and clinical data: age, sex, body mass index, type of surgery (coronary artery bypass grafting, valve surgery, or combined procedures), and surgical status (elective or urgent).

Preoperative laboratory parameters: renal function indices (serum creatinine and blood urea nitrogen), metabolic parameters (fasting glucose and glycated hemoglobin), inflammatory and nutritional markers (C-reactive protein and albumin), complete blood count variables (white blood cell count, neutrophil, lymphocyte, monocyte counts, hemoglobin, hematocrit, red cell distribution width, and platelet count), and coagulation profile (international normalized ratio). The preoperative serum creatinine value was defined as the measurement obtained within the last 48 hours before surgery. Derived inflammatory indices were calculated as follows: neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, lymphocyte-to-monocyte ratio, systemic immune-inflammation index (neutrophil × platelet / lymphocyte), and the C-reactive protein-to-albumin ratio.

Preoperative echocardiography: left ventricular ejection fraction was recorded.

Intraoperative perfusion and cardiopulmonary bypass variables: During CPB, temperature management followed a standardized institutional protocol. Mild systemic hypothermia (target core temperature approximately 32–34°C) was routinely applied. Deep hypothermia and circulatory arrest techniques were not performed in any patient during the study period; therefore, no circulatory arrest cases were included in this cohort. Rewarming was performed in a controlled manner before separation from CPB. All cardiopulmonary bypass procedures were performed using a standardized institutional protocol. A conventional roller pump system was used in all cases, and non-pulsatile flow was applied throughout CPB. Membrane oxygenators without direct blood–gas interface were used uniformly. During the study period, two identical CPB machines with the same technical specifications were available in the operating rooms, and no variation in pump technology or flow mode occurred across cases. Cardiopulmonary bypass duration, aortic cross-clamp time, highest intraoperative lactate level, lowest intraoperative pH value, and minimum intraoperative hemoglobin and hematocrit values were recorded. To summarize the degree of hemodilution, the changes in hemoglobin (Δhemoglobin = preoperative hemoglobin − minimum intraoperative hemoglobin) and hematocrit (Δhematocrit = preoperative hematocrit − minimum intraoperative hematocrit) were calculated. In this study, the concept of "perfusion stress" was used as a conceptual framework and was represented by parameters including cardiopulmonary bypass duration, lactate levels, pH, and minimum intraoperative hemoglobin and hematocrit values.

2.4. Outcomes and definitions

Primary outcome: Postoperative AKI was diagnosed according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria based on changes in serum creatinine [7]. Urine output criteria were not evaluated due to limitations inherent to retrospective data collection. Baseline creatinine was defined as the last measurement obtained within 48 hours prior to surgery.

In our institution, serum creatinine is routinely measured at 12-hour intervals during the first 72 postoperative hours (six measurements in total). For AKI assessment, the highest value recorded within this 72-hour window was defined as the "postoperative peak creatinine," and KDIGO staging was determined by comparison with the preoperative baseline value.

In cases where one of the scheduled postoperative measurements was missing, patients were not excluded; AKI classification was based on the highest available creatinine value within the 72-hour period. No patient was excluded due to incomplete postoperative creatinine surveillance.

All preoperative laboratory variables required for inflammatory index calculation were available for the entire cohort.

KDIGO staging (serum creatinine criteria):

- Stage 1: increase in serum creatinine ≥0.3 mg/dL or 1.5–1.9 times baseline
- Stage 2: increase in serum creatinine 2.0–2.9 times baseline
- Stage 3: increase in serum creatinine ≥3 times baseline or ≥4.0 mg/dL

Secondary outcomes: Secondary outcomes were AKI stage and in-hospital mortality.

2.5. Statistical analysis

All analyses were performed using SPSS (IBM Corp., Armonk, NY) statistical software. The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test and visual inspection methods. Normally distributed variables are presented as mean ± standard deviation, whereas non-normally distributed variables are reported as median with interquartile range. Comparisons between two groups were conducted using the Student's t-test or the Mann–Whitney

U test, as appropriate. Categorical variables were compared using the chi-square test or Fisher's exact test when expected cell counts were low.

To identify independent predictors associated with the development of postoperative AKI, multivariable logistic regression analysis was performed. A purposeful selection strategy was applied to obtain parsimonious models: clinically relevant variables and those associated with the outcome at $p < 0.10$ in univariable analyses were considered, and variables that did not materially improve model fit or that showed instability due to collinearity were not retained. Model calibration was evaluated using the Hosmer–Lemeshow test, and discrimination was quantified by the area under the ROC curve. Type of surgery (isolated CABG, isolated valve surgery, or combined procedures) was evaluated as a potential confounder in exploratory multivariable analyses to account for procedural heterogeneity. Surgical status (elective vs. emergency) was also evaluated as a potential confounder in exploratory analyses to assess its independent association with postoperative AKI. To reduce the risk of overfitting, model construction accounted for an events-per-variable ratio of at least 10. The potential for multicollinearity was assessed based on clinical judgment and statistical considerations, and results are reported as odds ratios with 95% confidence intervals. Discriminative performance was evaluated using receiver operating characteristic curve analysis; the area under the curve, sensitivity, specificity, and optimal cutoff value (based on the Youden index) were calculated. A two-sided p value <0.05 was considered statistically significant.

2.6. Sample size

Sample size planning was based on the expected incidence of AKI (20–30%) and the number of variables to be evaluated in logistic regression models. Given the observed AKI incidence and the number of events in the final cohort, the events-per-variable ratio remained ≥10, supporting model stability.

3. Results

During 2025, a total of 1187 patients underwent cardiac surgery at our institution. Of these, 701 patients were excluded because cardiopulmonary bypass was not used, and 486 patients who underwent surgery with cardiopulmonary bypass were initially evaluated. After excluding patients with a preoperative requirement for dialysis (n = 21), those operated on for intracardiac mass or thrombus (n = 3), patients who underwent atrial septal defect repair (n = 5), and those who underwent surgery for emergency cardiac trauma (n = 2), the final analytical cohort consisted of 455 patients (Fig. 1). According to the Kidney Disease Improving Global Outcomes criteria, postoperative acute kidney injury developed in 216 patients (47.5%), whereas 239 patients (52.5%) did not develop acute kidney injury.

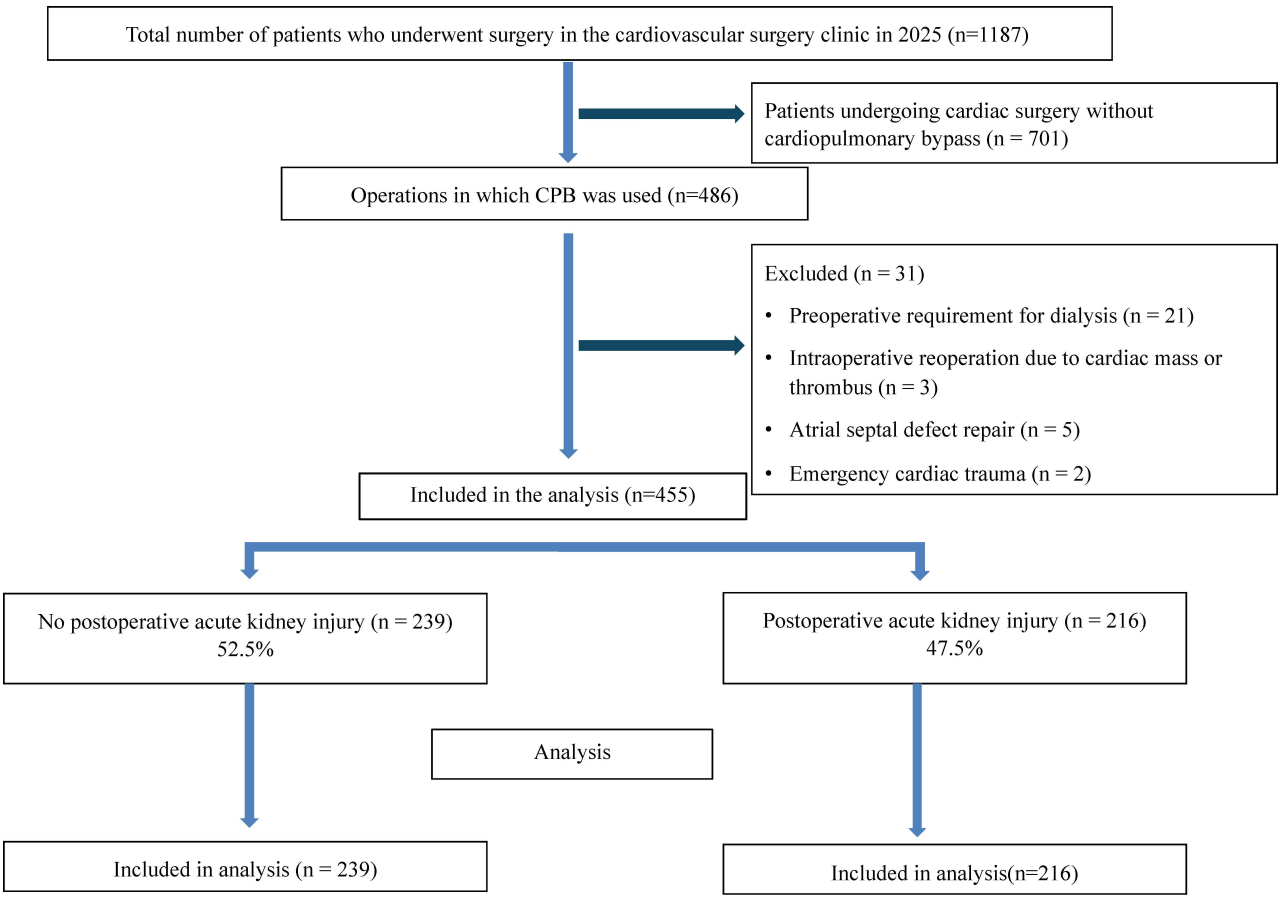

Fig. 1. Flowchart illustrating patient selection and distribution of the study cohort according to postoperative acute kidney injury status (CPB, cardiopulmonary bypass; ASD, atrial septal defect; AKI, acute kidney injury).

Postoperative AKI developed in 216 of 455 patients (47.5%). Patients who developed AKI were older than those without AKI [62.5 (55–70) vs. 59.0 (50–65), p < 0.001]. No significant differences were observed between the groups with respect to sex distribution, body mass index, or preoperative ejection fraction (p = 0.906, p = 0.430, and p = 0.095, respectively). The distribution of surgical type and surgical status was also similar between patients with and without AKI (p = 0.210 and p = 0.117, respectively) (Table 1). When surgical status (elective vs. emergency) was entered into exploratory multivariable models, it was not independently associated with postoperative AKI and did not materially alter the effect estimates of age, baseline creatinine, or CPB duration.

Table 1. Demographic and surgical characteristics of the patients according to acute kidney injury status.

Variable	No AKI (n = 239)	AKI (n = 216)	p value
Age (years)	59.0 (50–65)	62.5 (55–70)	<0.001
Sex, n (%)			0.906
• Male	176 (73.6)	158 (73.1)	
• Female	63 (26.4)	58 (26.9)	
BMI (kg/m^2)	27.4 (24.7–30.1)	27.8 (24.8–31.1)	0.430
Type of surgery, n (%)			0.210
• Isolated CABG	159 (66.5)	131 (60.6)	
• Isolated valve	46 (19.2)	41 (19.0)	
• Combined group surgery	34 (14.2)	44 (20.4)	
Surgical status, n (%)			0.117
• Elective surgery	226 (94.6)	196 (90.7)	
• Emergency surgery	13 (5.4)	20 (9.3)	
Preoperative EF (%)	55 (50–55)	55 (50–55)	0.095

Data are presented as median (interquartile range) or number (percentage). Continuous variables were compared using the Mann–Whitney U test, and categorical variables were compared using the Pearson chi-square test. A p value < 0.05 was considered statistically significant.

Abbreviations: AKI, acute kidney injury; BMI, body mass index; CABG, coronary artery bypass grafting; CPB, cardiopulmonary bypass; EF, ejection fraction. Isolated CABG refers to coronary artery bypass grafting performed with cardiopulmonary bypass without concomitant valve replacement or repair. Isolated valve surgery includes cases undergoing aortic or mitral valve replacement or repair. The combined group includes patients who underwent CABG together with valve replacement or repair during the same operative session.

Preoperative lymphocyte count was lower in patients who developed AKI [1.80 (1.33–2.46) vs. 2.03 (1.50–2.51) ×10^3/μL; p = 0.016]. With respect to inflammatory markers, the AKI group had higher RDW [13.5 (13.0–14.2) vs. 13.1 (12.7–14.0)%; p = 0.009], NLR [2.58 (1.82–4.66) vs. 2.44 (1.72–3.59); p = 0.037], CRP [5.0 (2.4–18.1) vs. 3.7 (2.0–10.9) mg/L; p = 0.018], and CRP/albumin ratio [1.25 (0.59–5.00) vs. 0.88 (0.50–2.81); p = 0.012], whereas albumin levels were lower [4.0 (3.6–4.2) vs. 4.0 (3.8–4.3) g/dL; p = 0.005]. As indicators of renal reserve, serum creatinine [0.93 (0.75–1.13) vs. 0.83 (0.69–1.02) mg/dL; p < 0.001] and BUN [19.0 (15.0–24.5) vs. 16.8 (13.6–22.1) mg/dL; p = 0.002] were significantly higher in the AKI group. No significant differences were observed between groups for other hematological parameters, glucose, HbA1c, or INR (Table 2).

Patients who developed AKI had significantly longer intraoperative perfusion times; CPB duration [112 (89–152) vs. 101 (80–128) min; p < 0.001] and aortic cross-clamp time [60 (45–88) vs. 55 (39–79) min; p = 0.016] were higher compared with patients without AKI. With respect to hemodilution parameters, the AKI group had lower minimum intraoperative hemoglobin [7.3 (6.3–8.1) vs. 7.6 (6.8–8.4) g/dL; p = 0.002] and minimum hematocrit values [22.5 (19.9–24.9) vs. 23.8 (21.0–26.0)%; p = 0.003]. Maximum intraoperative lactate levels showed a trend toward higher values in the AKI group [2.6 (1.8–3.6) vs. 2.4 (1.7–3.0) mmol/L; p = 0.054], whereas no significant differences were observed between groups with respect to minimum intraoperative pH or ΔHb and ΔHct values (Table 3).

Postoperative AKI occurred in 216 of 455 patients (47.5%), of whom 58 (12.7%) were classified as KDIGO stage ≥2 (stages 2–3) (Table 4). Among patients with AKI, the distribution of KDIGO stages was as follows: stage 1, 158/216 (73.1%); stage 2, 43/216 (19.9%); and stage 3, 15/216 (6.9%). In-hospital mortality was significantly higher in patients who developed AKI compared with those who did not (55/216, 25.5% vs. 16/239, 6.7%; p < 0.001). Moreover, mortality increased markedly with increasing AKI severity; in-hospital mortality was 12.7% (20/158) in stage 1, 51.2% (22/43) in stage 2, and 86.7% (13/15) in stage 3 (Table 4).

In the parsimonious multivariable logistic regression model, age, preoperative serum creatinine, and CPB duration were independently associated with postoperative AKI. Each 1-year increase in age was associated with an approximately 3% increase in the odds of AKI (OR 1.03; 95% CI 1.01–1.05; p < 0.001). Each 0.1 mg/dL increase in preoperative creatinine was associated with a 6% higher odds of AKI (OR 1.06; 95% CI 1.01–1.11; p =

0.011). Each 10-minute increase in CPB duration was associated with an approximately 4% increase in the odds of AKI (OR 1.04; 95% CI 1.00–1.08; p = 0.048). Although NLR and minimum intraoperative hemoglobin showed an association with AKI in univariable analyses, neither remained an independent predictor in the multivariable model (p = 0.703 and p = 0.102, respectively). The model demonstrated moderate discriminative performance (AUC = 0.672) with acceptable calibration (Hosmer–Lemeshow p = 0.101) (Table 5). When type of surgery was entered into exploratory multivariable models, it was not independently associated with postoperative AKI and did not materially alter the effect estimates of CPB duration or baseline creatinine.

Table 2. Preoperative laboratory parameters and inflammatory indices according to acute kidney injury status.

Variable	No AKI (n = 239)	AKI (n = 216)	p value
WBC (×10³/μL)	8.10 (6.49–9.88)	8.20 (6.65–10.00)	0.737
Neutrophil (×10³/μL)	5.14 (3.79–6.69)	5.37 (4.00–6.97)	0.300
Lymphocyte (×10³/μL)	2.03 (1.50–2.51)	1.80 (1.33–2.46)	0.016
Monocyte (×10³/μL)	0.59 (0.45–0.72)	0.58 (0.44–0.75)	0.899
Hemoglobin (g/dL)	13.9 (12.2–15.0)	13.3 (12.0–15.0)	0.341
Hematocrit (%)	41.2 (37.0–45.0)	40.1 (36.1–44.0)	0.278
RDW (%)	13.1 (12.7–14.0)	13.5 (13.0–14.2)	0.009
Platelet (×10³/μL)	229 (192–287)	230 (186–276)	0.593
NLR	2.44 (1.72–3.59)	2.58 (1.82–4.66)	0.037
PLR	115.4 (87.0–162.5)	125.6 (87.4–173.4)	0.096
LMR	3.70 (2.58–4.78)	3.26 (2.11–4.40)	0.024
SII	556 (380–904)	622 (402–1167)	0.108
CRP (mg/L)	3.7 (2.0–10.9)	5.0 (2.4–18.1)	0.018
Albumin (g/dL)	4.0 (3.8–4.3)	4.0 (3.6–4.2)	0.005
CRP/Albumin ratio	0.88 (0.50–2.81)	1.25 (0.59–5.00)	0.012
Creatinine (mg/dL)	0.83 (0.69–1.02)	0.93 (0.75–1.13)	<0.001
BUN (mg/dL)	16.8 (13.6–22.1)	19.0 (15.0–24.5)	0.002
Glucose (mg/dL)	106 (91–144)	112 (90–150)	0.446
HbA1c (%)	5.8 (5.5–6.7)	6.0 (5.6–7.2)	0.125
INR	1.04 (0.96–1.15)	1.04 (0.97–1.15)	0.572

Data are presented as median (interquartile range). Groups were compared using the Mann–Whitney U test (two-sided). A p value < 0.05 was considered statistically significant. SII = neutrophil × platelet / lymphocyte; NLR = neutrophil/lymphocyte; PLR = platelet/lymphocyte; LMR = lymphocyte/monocyte.

Abbreviations: AKI, acute kidney injury; WBC, white blood cell count; RDW, red cell distribution width; CRP, C-reactive protein; INR, international normalized ratio; BUN, blood urea nitrogen.

Table 3. Intraoperative perfusion and hemodilution parameters according to AKI status.

Variable	No AKI (n = 239)	AKI (n = 216)	p value
CPB duration (min)	101 (80–128)	112 (89–152)	<0.001
Aortic cross-clamp time (min)	55 (39–79)	60 (45–88)	0.016
Minimum intraoperative pH	7.34 (7.30–7.37)	7.34 (7.29–7.37)	0.903
Maximum intraoperative lactate (mmol/L)	2.4 (1.7–3.0)	2.6 (1.8–3.6)	0.054
Minimum intraoperative Hb (g/dL)	7.6 (6.8–8.4)	7.3 (6.3–8.1)	0.002
Minimum intraoperative Hct (%)	23.8 (21.0–26.0)	22.5 (19.9–24.9)	0.003
ΔHb (g/dL)	6.0 (4.9–7.1)	6.2 (5.0–7.2)	0.328
ΔHct (%)	17.0 (13.4–20.4)	17.9 (13.7–20.8)	0.253

Data are presented as median (interquartile range). Groups were compared using the Mann–Whitney U test (two-sided). A p value < 0.05 was considered statistically significant. ΔHb = preoperative Hb − minimum intraoperative Hb; ΔHct = preoperative Hct − minimum intraoperative Hct.

Abbreviations: AKI, acute kidney injury; CPB, cardiopulmonary bypass; Hb, hemoglobin; Hct, hematocrit.

Table 4. Incidence of postoperative AKI, KDIGO stages, and in-hospital mortality.

Outcome	Total (N = 455)	No AKI (n = 239)	AKI (n = 216)	p value
AKI, n (%)	216 (47.5)	0 (0)	216 (100)	NA
AKI stage ≥2, n (%)	58 (12.7)	0 (0)	58 (26.9)	NA
AKI stage (among AKI patients only), n (%)				NA
• Stage 1	—	—	158 (73.1)	
• Stage 2	—	—	43 (19.9)	
• Stage 3	—	—	15 (6.9)	
In-hospital mortality, n (%)	71 (15.6)	16 (6.7)	55 (25.5)	<0.001
In-hospital mortality by AKI stage (among AKI patients only)				NA
• Stage 1	—	—	20/158 (12.7)	
• Stage 2	—	—	22/43 (51.2)	
• Stage 3	—	—	13/15 (86.7)	

Data are presented as n (%). In-hospital mortality was compared between the AKI and no AKI groups using the Pearson chi-square test (Fisher's exact test was used when expected cell counts were <5). A p value < 0.05 was considered statistically significant. AKI stages were classified according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria.

Abbreviations: AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes; NA: Not applicable.

Table 5. Parsimonious logistic regression model for postoperative acute kidney injury (KDIGO) (N = 455).

Variable (scale)	Univariable OR (95% CI)	p value	Multivariable OR (95% CI)	p value
Age (per 1 year)	1.03 (1.02–1.05)	<0.001	1.03 (1.01–1.05)	<0.001
Preoperative creatinine (per 0.1 mg/dL)	1.07 (1.02–1.12)	0.004	1.06 (1.01–1.11)	0.011
CPB duration (per 10 min)	1.05 (1.02–1.09)	0.004	1.04 (1.00–1.08)	0.048
NLR (per 1 unit)	1.05 (1.00–1.10)	0.070	1.01 (0.96–1.06)	0.703
Intraoperative minimum hemoglobin (per 1 g/dL)	0.83 (0.72–0.95)	0.006	0.89 (0.77–1.02)	0.102

Odds ratios (ORs) with 95% confidence intervals (CIs) are reported. Continuous variables were scaled for clinical interpretability (creatinine per 0.1 mg/dL; CPB duration per 10 min). The multivariable model was constructed in a parsimonious manner based on clinical relevance and univariable screening. Model discrimination was moderate (AUC = 0.672), and calibration was acceptable (Hosmer–Lemeshow $\chi^2 = 13.34$, df = 8; p = 0.101).

Abbreviations: AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes; CPB, cardiopulmonary bypass; NLR, neutrophil-to-lymphocyte ratio; OR, odds ratio; CI, confidence interval; AUC, area under the curve.

In ROC analysis for the prediction of KDIGO stage ≥2 AKI, CPB duration demonstrated the highest discriminative ability (AUC 0.672; 95% CI 0.599–0.746; p < 0.001). Using the Youden index, an optimal cut-off of ≥123 minutes yielded a sensitivity of 60.3% and a specificity of 68.8% (Table 6). NLR (AUC 0.587; p = 0.045) and preoperative creatinine (AUC 0.584; p = 0.046) showed more limited discriminative performance; however, for NLR, a cut-off of ≥5.28 was characterized by low sensitivity but high specificity (86.9%), suggesting a predominantly "rule-in" rather than screening utility.

In the multivariable parsimonious logistic regression analysis for the KDIGO stage ≥2 AKI (stages 2–3) outcome, age (OR 1.03 per year; 95% CI 1.00–1.06; p = 0.039) and CPB duration (OR 1.07 per 10 minutes; 95% CI 1.02–1.12; p = 0.002) were identified as independent predictors (Table 7). Although NLR and minimum intraoperative hemoglobin were associated with the outcome in univariable analyses, neither retained independent significance in the multivariable model. The model demonstrated moderate discriminative performance (AUC = 0.698) and good calibration (Hosmer–Lemeshow p = 0.681).

Table 6. Receiver operating characteristic (ROC) analysis for prediction of KDIGO stage ⩾2 acute kidney injury (stages 2–3) (N = 455).

Parameter	AUC (95% CI)	p value	Optimal cut-off (Youden)	Sensitivity (%)	Specificity (%)
NLR	0.587 (0.502–0.672)	0.045	≥ 5.28	34.5	86.9
Preoperative creatinine (mg/dL)	0.584 (0.501–0.667)	0.046	≥ 0.94	58.6	60.5
CPB duration (min)	0.672 (0.599–0.746)	<0.001	≥ 123	60.3	68.8

The 95% confidence intervals for AUC were calculated using the DeLong method. Optimal cut-off values were determined by the Youden index (sensitivity + specificity − 1). For all ROC analyses, higher values were assumed to be associated with a higher risk of the outcome.

Abbreviations: AUC, area under the curve; CI, confidence interval; AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes; NLR, neutrophil-to-lymphocyte ratio; CPB, cardiopulmonary bypass.

Table 7. Parsimonious logistic regression model for prediction of KDIGO stage ⩾2 acute kidney injury (stages 2–3) (N = 455).

Variable (scale)	Univariable OR (95% CI)	p value	Multivariable OR (95% CI)	p value
Age (per 1 year)	1.03 (1.01–1.06)	0.012	1.03 (1.00–1.06)	0.039
Preoperative creatinine (per 0.1 mg/dL)	1.02 (0.97–1.08)	0.438	0.99 (0.93–1.06)	0.802
CPB duration (per 10 min)	1.08 (1.04–1.13)	<0.001	1.07 (1.02–1.12)	0.002
NLR (per 1 unit)	1.07 (1.01–1.13)	0.027	1.03 (0.97–1.10)	0.276
Intraoperative minimum hb (per 1 g/dL)	0.80 (0.65–0.98)	0.035	0.91 (0.73–1.12)	0.370

Odds ratios (ORs) with 95% confidence intervals (CIs) are reported. Continuous variables were scaled for clinical interpretability (creatinine per 0.1 mg/dL; CPB duration per 10 min). The parsimonious multivariable model was constructed from clinically relevant variables and/or those with $p < 0.10$ in univariable analyses. Model performance showed good discrimination (AUC = 0.698) and excellent calibration (Hosmer–Lemeshow $\chi^2 = 5.70$, df = 8; p = 0.681).

Abbreviations: AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes; CPB, cardiopulmonary bypass; Hb, hemoglobin; NLR, neutrophil-to-lymphocyte ratio; OR, odds ratio; CI, confidence interval; AUC, area under the curve.

4. Discussion

In this single-center retrospective cohort study, the incidence of postoperative AKI after adult cardiac surgery performed with CPB was 47.5%, and in-hospital mortality increased markedly with increasing AKI severity. The reported incidence of AKI after cardiac surgery varies widely in the literature (approximately 5%–40%, depending on the definition used, patient case-mix, surgical procedure, and surveillance strategy), and the AKI definition and the frequency of creatinine measurements are among the primary drivers of this variability [8, 9]. In this context, the routine measurement of postoperative creatinine every 12 hours in our institution and the use of the highest creatinine value within the first 72 postoperative hours as the "peak creatinine" may have increased the sensitivity for AKI detection. Accordingly, comparisons of AKI incidence across studies should account for both the operational definition and surveillance intensity, as higher-frequency creatinine sampling tends to preferentially increase detection of KDIGO stage 1 events. This methodological nuance may partly explain why our observed AKI rate appears higher than rates reported in cohorts with less frequent postoperative creatinine monitoring. The KDIGO criteria are based on changes in serum creatinine (an increase of ≥0.3 mg/dL within 48 hours or ≥1.5-fold within 7 days), and more frequent measurement strategies may particularly increase the detection of stage 1 AKI [10]. Consistent with this, the predominance of stage 1 AKI in our cohort (73.1%) aligns with the hypothesis that our surveillance strategy enhanced the likelihood of capturing early or mild creatinine changes. The relatively high incidence of AKI observed in our cohort further underscores the clinical relevance of systematic surveillance and risk stratification in CPB-assisted cardiac surgery.

In our study, the mortality rate was approximately fourfold higher in patients who developed AKI compared with those without AKI (25.5% vs. 6.7%), and mortality increased dramatically with advancing KDIGO stage (stage 1: 12.7%, stage 2: 51.2%, stage 3: 86.7%). These findings support the concept that AKI is not merely a biochemical abnormality, but rather a clinically meaningful outcome that strongly determines prognosis within the spectrum of perioperative multiorgan dysfunction. A graded association between increasing KDIGO stage and mortality has been consistently demonstrated in previous cardiac surgery cohorts, with stage 2–3 AKI showing a particularly strong relationship with short-term mortality [11]. Accordingly, the observed incidence of clinically relevant KDIGO stage ≥2 AKI of 12.7% in our cohort indicates a substantial burden of severe AKI and underscores the need for targeted risk mitigation strategies.

In parsimonious multivariable models, age, preoperative serum creatinine, and CPB duration were independently associated with postoperative AKI. This triad reflects the fundamental components of AKI pathophysiology in cardiac surgery, namely a "susceptible substrate plus intraoperative triggers/exposure": advanced age and elevated baseline creatinine indicate limited renal reserve, whereas prolonged CPB duration increases the exposure dose to injurious mechanisms such as nonpulsatile flow, hemodilution, systemic inflammatory response, and ischemia–reperfusion injury [9]. From a clinical perspective, the modifiable nature of CPB duration is of particular importance. The association between prolonged CPB duration and increased AKI risk has been consistently reported in large-scale analyses and meta-analyses, with longer CPB times conferring a higher risk of AKI across different types of cardiac surgery [12]. In our study, the persistence of age and CPB duration as independent predictors in the model for KDIGO stage ≥2 AKI further supports the concept of a cumulative "exposure dose" effect. Nevertheless, the moderate AUC values observed in ROC analyses (AUC = 0.672 for any AKI and AUC = 0.698 for stage ≥2 AKI) indicate that risk stratification based on a single parameter is inherently limited and highlight the need for multidimensional, externally validated risk models, ideally incorporating biomarker data. Accordingly, CPB duration should not be regarded as a standalone decision-making threshold, but rather as a variable that meaningfully contributes to perioperative risk stratification when interpreted in conjunction with other clinical factors. Although procedural heterogeneity may theoretically influence AKI risk, type of surgery per se was not independently associated with AKI in our cohort, suggesting that perfusion-related exposure variables may be more

influential than surgical category. Similarly, although emergency surgical status may theoretically confer additional risk due to recent contrast exposure or hemodynamic instability, surgical status was not independently associated with AKI in adjusted analyses in our cohort. Importantly, CPB flow mode and pump technology were standardized across the cohort (non-pulsatile roller pump with membrane oxygenator), thereby minimizing potential confounding related to perfusion modality. Importantly, temperature management was standardized (mild hypothermia protocol without circulatory arrest), thereby reducing potential variability related to intraoperative thermal strategies.

With respect to preoperative inflammatory indices, higher NLR, CRP, CRP-to-albumin ratio, and RDW values, together with lower albumin and lymphocyte levels in patients who developed AKI, support an association between AKI development and an underlying state of inflammatory–metabolic vulnerability. These findings are consistent with the existing literature: systematic reviews and meta-analyses evaluating perioperative NLR have reported that elevated NLR may be associated with an increased risk of AKI after cardiac surgery [5,13]. Similarly, RDW—reflecting inflammation, oxidative stress, and disturbed erythrocyte homeostasis—has been linked to adverse renal outcomes in cardiac surgical populations [14]. In addition, the CRP-to-albumin ratio has gained increasing attention as a composite index capturing both inflammatory burden and nutritional/catabolic status, with studies suggesting its potential value in predicting postoperative complications and AKI after coronary artery bypass grafting [15]. However, the loss of independent significance of NLR and RDW in our multivariable models suggests that these inflammatory indices may partly reflect AKI risk through their association with baseline renal reserve and intraoperative exposure, particularly CPB duration. This pattern is biologically plausible: preoperative inflammatory indices may capture a systemic vulnerability phenotype, whereas the development of AKI after CPB is ultimately driven by the interaction between susceptibility and intraoperative exposure dose (e.g., duration and intensity of perfusion-related stress). Therefore, once dominant exposure variables such as CPB duration and baseline renal function are incorporated into the same model, the incremental independent contribution of inflammatory indices may attenuate, despite their clear unadjusted associations. In other words, while inflammatory indices may identify a "high-risk phenotype," their incremental contribution to clinical decision-making in this cohort was limited once dominant clinical determinants were included in the model. Notably, in ROC analysis for KDIGO stage ≥2 AKI, NLR demonstrated low sensitivity but high specificity, indicating that NLR may serve as a potential rule-in signal in selected subgroups, rather than as a standalone screening tool [5]. This finding is particularly relevant in light of previous studies that have reported independent associations between inflammatory indices and AKI after CPB; our results suggest that when baseline renal reserve and intraoperative exposure intensity are simultaneously accounted for, the incremental predictive value of isolated inflammatory markers may diminish.

The association between intraoperative hemodilution/anemia-related parameters and AKI is also noteworthy. In our study, minimum intraoperative Hb and Hct values were lower in the AKI group; however, these variables lost borderline significance in the multivariable model. The potentially deleterious renal effects of hemodilution have been extensively discussed in the literature: low hematocrit/hemoglobin during CPB may reduce renal oxygen delivery, exacerbate medullary hypoxia, and thereby increase the risk of AKI [16, 17]. Accordingly, the impact of hemodilution may be best interpreted as a component of the broader "perfusion stress" axis, which is closely intertwined with CPB duration and perfusion dynamics. Indeed, pathophysiological studies examining the relationship between renal oxygenation and flow targets during CPB suggest that perfusion strategies may exert meaningful effects on renal oxygenation [9]. Therefore, kidney-protective perioperative management in clinical practice likely requires a multifaceted approach, including minimizing CPB duration in high-risk patients, limiting hemodilution, individualizing perfusion pressure and flow targets, closely monitoring global perfusion markers such as lactate and pH, and avoiding nephrotoxic exposures when feasible. It should be acknowledged, however, that lactate represents a downstream metabolic consequence of global hypoperfusion and does not allow precise differentiation of the underlying hemodynamic mechanism (e.g., hypotension, reduced oxygen delivery, microcirculatory dysfunction). Therefore, the concept of "perfusion stress" in this study should be interpreted as a composite surrogate framework rather than a direct measurement of intraoperative hemodynamic instability. It should also be noted that intraoperative ultrafiltration strategies and perioperative diuretic administration may influence fluid balance and renal function. Because these variables were not systematically available in the electronic dataset, their potential contribution to AKI risk could not be evaluated in this study.

An important contribution of this study is the simultaneous integration of routinely available preoperative inflammatory indices with intraoperative perfusion stress markers in a single multivariable framework. This design reflects the multifactorial nature of AKI after CPB and provides a practical structure for perioperative risk phenotyping using variables readily accessible in routine care.

4.1. Clinical implications

The present findings have several practical implications. First, the integration of baseline renal function and intraoperative perfusion-related variables into a unified predictive framework supports the importance of combined preoperative risk assessment and intraoperative optimization strategies in reducing postoperative AKI risk. Patients identified as high-risk based on elevated baseline creatinine or anticipated prolonged CPB duration may benefit from intensified renal-protective strategies, including strict hemodynamic optimization, avoidance of nephrotoxic agents, careful fluid balance management, and closer postoperative surveillance.

Second, although preoperative inflammatory indices did not retain independent significance in multivariable models, their univariable association with AKI suggests that they may serve as early markers of systemic vulnerability. In routine practice, these inexpensive and readily available laboratory parameters may assist in preliminary risk stratification before surgery.

Finally, the findings emphasize that AKI after CPB is driven by the interaction between patient susceptibility and intraoperative exposure. Therefore, perioperative teams should adopt a multimodal prevention strategy rather than focusing on a single risk domain.

Taken together, these findings clarify the relative contribution of inflammatory burden versus intraoperative exposure in AKI development and position CPB duration within a clinically actionable risk framework.

In conclusion, our findings indicate that postoperative AKI is closely associated not only with preoperative susceptibility but also with intraoperative perfusion stress, and that age, baseline renal function, and CPB duration represent core variables for perioperative risk stratification. Although inflammatory indices may convey a meaningful biological signal, they did not remain independent predictors in this cohort and appeared to function primarily as adjunctive markers for risk phenotyping. Given the moderate model performance and the lack of external validation, the generalizability of this approach requires multicenter prospective validation studies, ideally incorporating biomarker integration.

4.2. Limitations

This study has several important limitations. First, the single-center retrospective design precludes causal inference and carries a risk of residual confounding due to unmeasured or incompletely recorded variables. Second, the definition of AKI was based solely on serum creatinine criteria; urine output criteria could not be incorporated because of the retrospective nature of the study and limitations in available records. Third, routine measurement of serum creatinine every 12 hours in the postoperative period may have contributed to a higher detection rate of early or mild AKI, and the use of the highest creatinine value within the first 72 postoperative hours, while increasing sensitivity, may have affected clinical comparability across studies. Fourth, the lack of detailed characterization of surgical subtypes (e.g., number of bypass grafts, valve pathology, or repair versus replacement) limited a more granular assessment of procedure-specific risk. Furthermore, coronary artery bypass grafting, valve surgery, and combined procedures were analyzed together rather than through separate subgroup-specific multivariable models. Although type of surgery was evaluated as a potential confounder and was not independently associated with AKI, the absence of formal subgroup analyses may limit procedure-specific interpretation. The relatively limited number of KDIGO stage ≥2 AKI events further constrained the feasibility of stable subgroup modeling. In addition, the preoperative comorbidity burden of patients (e.g., diabetes mellitus, hypertension, chronic kidney disease, chronic obstructive pulmonary disease) could not be comprehensively reported, and residual confounding therefore cannot be fully excluded. Furthermore, detailed data regarding the timing and volume of preoperative coronary angiography and contrast exposure—particularly in emergency cases—were not uniformly available in the retrospective records and could not be incorporated into the analyses. Therefore, residual confounding related to potential contrast-associated renal injury cannot be entirely excluded. Fifth, detailed intraoperative hemodynamic data (including continuous mean arterial pressure, cardiac output, pump flow targets, and oxygen delivery indices) as well as vasoactive and inotropic drug doses and duration were not available in the electronic database and therefore could not be incorporated into the analyses. Given that vasoactive agents at certain doses may influence renal perfusion through vasoconstrictive effects, residual confounding related to unmeasured hemodynamic instability cannot be excluded. Accordingly, the "perfusion stress" construct used in this study should be interpreted as a surrogate conceptual model rather than a comprehensive hemodynamic assessment. Additionally, intraoperative ultrafiltration practices and perioperative diuretic use (including type, dose, and duration) were not retrievable from the electronic system and could not be analyzed. Given that both ultrafiltration and diuretic administration may influence renal perfusion, intravascular volume status, and serum creatinine dynamics, residual confounding related to these factors cannot be excluded. Finally, although the regression models were constructed in a parsimonious manner, external validation was not performed and model performance remained moderate; consequently, multicenter prospective validation is required to confirm the generalizability of these findings.

5. Conclusions

Postoperative AKI is common after cardiac surgery performed with CPB, and in-hospital mortality increases dramatically with escalating AKI severity. In this cohort, the independent determinants of postoperative AKI were age, preoperative creatinine, and CPB duration, whereas age and CPB duration emerged as key predictors of KDIGO stage ≥2 AKI. Although preoperative inflammatory indices appeared to be associated with AKI, they did not remain independent predictors in multivariable models. Overall, these findings support the clinical value of a risk stratification framework centered on "preoperative renal reserve plus intraoperative perfusion stress" and underscore the importance of kidney-protective strategies in high-risk patients, including minimizing CPB duration and optimizing perfusion and hemodilution targets. Prospective multicenter studies incorporating biomarker data are warranted to validate this approach. These findings reinforce the conceptual model of "susceptibility plus exposure" in cardiac surgery–associated AKI and support targeted intraoperative optimization strategies in high-risk individuals.

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Conflict of Interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this manuscript.

Data Availability

The datasets generated and/or analyzed during the current study are not publicly available but are available from the corresponding author upon reasonable request.

AI Assistance

No AI-based tools were used in the preparation of this manuscript.

Ethics Approval and Consent to Participate

This single-center retrospective cohort study was conducted at Atatürk University Faculty of Medicine Hospital between 01.01.2025 and 01.01.2026. Institutional Ethics Committee approval was obtained (Approval Number: B.30.2.ATA.0.01.00/932; Date: 30.01.2026), and the study was registered at ClinicalTrials.gov (ID: NCT05405049). The ClinicalTrials.gov registration was completed to enhance transparency in the context of a retrospective design (at the time the data analysis plan was established); the study represents a non-interventional observational analysis. The study was performed in accordance with the principles of the Declaration of Helsinki. Due to the retrospective nature of the study, informed consent was not obtained from participants.

Author Contributions

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