Research Square

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Evaluating the Impact of Early RBC Transfusion on Acute Kidney Injury Following Cardiac Surgery

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Abstract

Background

Cardiac surgery-associated acute kidney injury (CSA-AKI) is a common, clinically important complication of cardiac surgery. Red blood cell (RBC) transfusion has been associated with CSA-AKI, but whether this risk follows a linear dose-response pattern and whether a safe threshold exists during the early postoperative period remain unclear.

Methods

We used the MIMIC-IV database to identify adults admitted to an intensive care unit (ICU) after cardiac surgery. Early postoperative RBC transfusion was defined as receipt of at least one unit of packed RBCs within 24 hours of ICU admission. The primary outcome was CSA-AKI, and the secondary outcome was 7-day all-cause mortality. We used crude and multivariable logistic regression, multiple imputation for missing covariates, and overlap propensity score weighting. Prespecified restricted and subgroup analyses were conducted, together with an exploratory generalized additive model of the dose-response relationship.

Results

The analysis included 4,058 patients, of whom 1,442 (35.5%) received early postoperative RBC transfusion. The crude 7-day incidence of CSA-AKI was 35.1% among non-transfused patients and 48.9% among transfused patients. Early postoperative RBC transfusion was associated with higher odds of CSA-AKI (OR 1.77, 95% CI 1.56–2.02, $p < 0.001$). Adjusted ORs ranged from 1.29 to 1.37 across sensitivity analyses, and all associations were statistically significant ($p < 0.05$). The dose-response relationship was linear (GAM edf = 1.00, $p = 8.30 \times 10^{-5}$); adjusted ORs increased from 1.25 (95% CI 1.01–1.55) at 201–400 ml to 1.60 (95% CI 1.29–2.00) at > 800 ml. The transfused group had a higher cumulative hazard of death, but absolute mortality was low, and early transfusion was not associated with an observable short-term survival benefit.

Conclusions

Early postoperative RBC transfusion was associated with greater CSA-AKI risk in a linear, dose-dependent pattern, with no evidence of a protective threshold. Even small-volume transfusions after cardiac surgery should therefore be individualized by balancing the expected benefit of correcting anemia against potential renal risk.

Introduction

Cardiac surgery-associated acute kidney injury (CSA-AKI) is a serious complication of cardiac surgery[1], characterized by a rapid decline in kidney function[2]. Proposed contributors include ischemia-reperfusion injury, systemic inflammation, hemolysis, oxidative stress, and microcirculatory disturbances[3–5]. CSA-AKI is associated with higher in-hospital mortality, chronic kidney disease (CKD), and poor long-term outcomes[6,7]. It develops in more than 20% of patients undergoing cardiac surgery[8,9] and remains a major contributor to perioperative death and long-term morbidity[10]. Its multifactorial pathophysiology and the lack of proven preventive therapies make both clinical management and intervention development difficult[11].

Red blood cell (RBC) transfusion is often used after cardiac surgery to increase the oxygen-carrying capacity of blood, improve oxygen delivery, and alter microcirculatory flow[12]. However, free hemoglobin released from RBCs can generate reactive oxygen species, causing oxidative injury and amplifying inflammation[13]. These processes may contribute to the onset and progression of CSA-AKI. Transfusion may also affect renal function through changes in hemodynamic stability, tissue oxygenation, and inflammatory responses[14,15]. Although early postoperative RBC transfusion has been linked to CSA-AKI, large ICU-based studies examining this relationship remain scarce[16].

Using data from the Medical Information Mart for Intensive Care IV (MIMIC-IV), we examined whether early postoperative RBC transfusion was associated with CSA-AKI and other clinical outcomes after cardiac surgery. Multiple analytical approaches were used to assess the robustness of the findings and to inform postoperative transfusion decisions.

Methods

Study design and data source

We used the MIMIC-IV database, which contains hospital records from 2008 to 2022[17,18]. The authors completed the required training and obtained data-access certification (Record ID: 68629862). Because this retrospective study used deidentified data, individual informed consent was waived[19]. We reported the study in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines[20].

Study population

We included adults aged ≥ 18 years at their first ICU admission after cardiac surgery and required an ICU stay longer than 24 hours. Patients were excluded for (1) inconsistent or implausible timing indicating that RBC transfusion, AKI onset, or death preceded the ICU baseline; or (2) pre-existing AKI, defined as AKI within the 7 days before baseline. These criteria restricted the analysis to incident postoperative kidney injury.

Exposure definition

Early postoperative RBC transfusion was defined as receipt of at least 1 unit of packed RBCs within 24 hours of ICU admission. Patients were classified as exposed (RBC group) or unexposed (No RBC group).

Outcome definitions

The primary outcome was CSA-AKI, defined by the KDIGO criteria: an increase in serum creatinine of ≥ 0.3 mg/dL within 48 hours, an increase to ≥ 1.5 times baseline within 7 days, or urine output < 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for > 6 hours[21]. The secondary outcome was 7-day all-cause mortality. Death was treated as a competing event in the cumulative incidence analysis because it could occur before AKI and preclude observation of the primary outcome.

Covariates

Covariates were specified a priori and included age, sex, race, admission year, first ICU type, baseline serum creatinine, Charlson Comorbidity Index (CCI), CKD, diabetes mellitus, hypertension, heart failure, body weight (kg), total urine output (ml), vasoactive-agent use, renal replacement therapy (RRT), total crystalloid volume (ml), day-1 Sequential Organ Failure Assessment (SOFA) score, and Acute Physiology Score III (APS III).

Missing data

We used multiple imputation by chained equations (MICE) in a sensitivity analysis to address missing covariate data. Five imputed datasets were generated using predictive mean matching for continuous variables, logistic regression for binary variables, and polytomous regression for categorical variables. Estimates were pooled according to Rubin's rules. The primary adjusted analysis used the observed complete-case dataset, whereas the imputed model served as a confirmatory sensitivity analysis (Supplementary Table 3).

Statistical analysis

Continuous variables were summarized as medians with interquartile ranges (IQRs), and categorical variables as counts and percentages. Groups were compared using the Wilcoxon rank-sum test for continuous variables and the chi-square test for

categorical variables.

We used logistic regression to examine the association between early postoperative RBC transfusion and CSA-AKI. The crude model was unadjusted; the multivariable model included all prespecified covariates. Estimates are reported as odds ratios (ORs) with 95% confidence intervals (CIs). To further address confounding, we applied overlap propensity score weighting[22]. Propensity scores were estimated with logistic regression using the covariates from the primary adjusted model. Balance before and after weighting was evaluated with absolute standardized mean differences (ASMDs), with values < 0.1 indicating adequate balance.

For the primary outcome, cumulative incidence functions were estimated with the Aalen-Johansen method, treating death as a competing event[23], and compared using Gray's test. For 7-day mortality, Nelson-Aalen cumulative hazard curves were constructed and compared with the log-rank test. All tests were two-sided, and $p < 0.05$ was considered statistically significant.

Restricted, subgroup, and dose-category analyses

Four prespecified restricted analyses were used to test the robustness of the primary finding: (1) patients whose postoperative care occurred exclusively in the Cardiovascular ICU (CVICU); (2) exclusion of patients who received renal replacement therapy (RRT) within the first 24 h; (3) a high-risk cohort defined by vasoactive-agent use within 24 h or a day-1 SOFA score ≥ 4; and (4) a cohort meeting all three criteria. The full analytical strategy, including crude, adjusted, and overlap-weighted models, was repeated in each cohort. Sample sizes and outcome counts are reported in Supplementary Table 3.

We assessed effect modification by age (< 65 vs ≥ 65 years), sex, race, CCI (< 3 vs ≥ 3), CKD, diabetes, heart failure, and early RRT use. Within each stratum, adjusted ORs and 95% CIs were estimated using the primary multivariable model and displayed in a forest plot.

We compared five transfusion-volume categories (≤ 200, 201–400, 401–600, 601–800, and > 800 ml) with no transfusion. Adjusted ORs for each category were estimated using the primary multivariable model. We tested for a linear trend by modeling the ordered dose category as an ordinal variable. Among transfused patients, a generalized additive model (GAM) with a cubic regression spline was used to estimate the continuous dose-response curve, with adjustment for all prespecified covariates.

Software

Analyses were performed in R (version 4.4.2). MIMIC-IV data were extracted with SQL queries in PostgreSQL (version 13.7.2) and Navicat Premium (version 16.1.12).

Results

Baseline characteristics

The analysis included 4,058 eligible patients (Fig. 1). Of these, 1,442 (35.5%) received RBC transfusion within 24 hours of ICU admission (RBC group), and 2,616 (64.5%) did not (No RBC group). Baseline characteristics are reported in Table 1. Most covariates differed between groups ($p < 0.001$). Compared with the No RBC group, transfused patients were older, more often female, and had a greater comorbidity burden. They also appeared more severely ill at baseline, with higher median SOFA and APS III scores and more frequent vasoactive-agent use.

Covariate balance assessment

Overlap weighting was used to reduce confounding by indication (Fig. 2). Before weighting, age, sex, CCI, body weight, urine output, SOFA score, and APS III differed between groups (ASMD > 0.1). After weighting, every ASMD was < 0.1, indicating adequate balance. Similar improvements were observed in all four restricted cohorts (Supplementary Fig. 1). We then evaluated the association between early RBC transfusion and CSA-AKI in the balanced weighted sample.

Primary analyses

The 7-day incidence of CSA-AKI was 48.9% in the RBC group and 35.1% in the No RBC group (Fig. 3). In the unadjusted model, early RBC transfusion was associated with higher odds of CSA-AKI (OR 1.77, 95% CI 1.56–2.02, $p < 0.001$). The association persisted after multivariable adjustment (OR 1.32, 95% CI 1.13–1.55, $p < 0.001$). Sensitivity analyses produced similar estimates: OR 1.32 (95% CI 1.13–1.55) after multiple imputation and OR 1.29 (95% CI 1.11–1.50, $p = 0.001$) after overlap weighting. Full model results are provided in Table 2 and Supplementary Table 2.

Restricted analyses

Results were similar in all four prespecified restricted cohorts (Table 2 and Supplementary Table 3). The adjusted OR was 1.33 (95% CI 1.13–1.55) in the CVICU-only cohort (n = 3,893), 1.33 (95% CI 1.13–1.55) after excluding patients who received RRT within 24 hours (n = 4,022), 1.35 (95% CI 1.15–1.59) in the high-risk cohort (n = 3,702), and 1.37 (95% CI 1.16–1.61) in the combined cohort (n = 3,587; all $p < 0.001$). Thus, restricting the cohort did not materially attenuate the association.

Cumulative incidence and competing risk analysis

With death treated as a competing risk, the estimated cumulative incidence of CSA-AKI remained higher in the RBC group throughout the 7-day follow-up (approximately 50% vs 40% at day 7; Fig. 4). Death was uncommon in both groups. Nelson-Aalen analysis nevertheless showed a higher cumulative hazard of 7-day all-cause mortality in the RBC group (approximately 2% vs < 0.5% at day 7; Fig. 5). Kaplan-Meier analysis showed high 7-day survival in both cohorts (> 98%), although survival was slightly lower in the RBC group (Supplementary Fig. 2).

Subgroup analyses

Subgroup estimates are shown in Fig. 6 and Supplementary Table 5. The direction of association was consistent in most prespecified subgroups, although statistical significance varied. Associations were statistically significant among patients aged < 65 years (OR 1.454, 95% CI 1.097–1.923) or ≥ 65 years (OR 1.223, 95% CI 1.009–1.483), male patients (OR 1.455, 95% CI 1.200–1.763), White patients (OR 1.310, 95% CI 1.094–1.569), patients without CKD (OR 1.307, 95% CI 1.104–1.546), patients without heart failure (OR 1.495, 95% CI 1.247–1.792), and those with CCI ≥ 3 (OR 1.309, 95% CI 1.104–1.552). The association was not statistically significant among female patients (OR 1.090, 95% CI 0.825–1.440), Black or Asian patients, patients with pre-existing heart failure (OR 1.030, 95% CI 0.745–1.421), or those with CCI < 3 (OR 1.167, 95% CI 0.746–1.800).

Dose-response relationship

Categorical and continuous dose-response results are shown in Supplementary Table 4 and Supplementary Fig. 3. Among transfused patients, the generalized additive model yielded a significant smooth term for transfusion volume (effective degrees of freedom [edf] = 1.00, $p = 8.30 \times 10^{-5}$), consistent with a linear association after multivariable adjustment. The adjusted OR for CSA-AKI rose from 1.0 at the reference volume to approximately 2.0 at 2800 ml (Supplementary Fig. 3).

In the categorical analysis, the ≤ 200 ml group was too small for reliable inference because of quasi-complete separation. Relative to no transfusion, 201–400 ml was associated with 25% higher odds of CSA-AKI (adjusted OR 1.25, 95% CI 1.01–1.55, $p = 0.043$). Estimates for 401–600 and 601–800 ml were similar in direction but not statistically significant (adjusted ORs 1.16 and 1.16, respectively), likely because these strata were small. Volumes > 800 ml were associated with higher odds of CSA-AKI (adjusted OR 1.60, 95% CI 1.29–2.00, $p < 0.001$). Together with the continuous GAM result, the consistent direction across evaluable categories supports a dose-dependent association between transfusion volume and CSA-AKI.

Discussion

In this retrospective cohort, early postoperative RBC transfusion was independently associated with higher odds of CSA-AKI. Adjusted ORs ranged from 1.29 to 1.37 across sensitivity analyses, and the direction of association was consistent across subgroups. We found no evidence of a short-term survival benefit.

Early after cardiac surgery, hemodynamic instability is common[24], and clinicians may transfuse RBCs in an effort to improve oxygen delivery and support organ function[25]. Focusing on this early postoperative window allowed us to assess a clinically

relevant period in which transfusion practice might be modified. Our findings are consistent with previous reports linking perioperative transfusion to renal injury after cardiac surgery[26]. Because transfused and non-transfused patients differed substantially at baseline, we used overlap propensity score weighting to reduce confounding by indication. The balance achieved after weighting (Fig. 2) makes indication bias a less likely sole explanation for the observed association.

Several mechanisms could contribute to transfusion-related AKI. Stored RBCs become less deformable and may be more susceptible to post-transfusion hemolysis[27]. Free hemoglobin and iron released from damaged RBCs can increase oxidative stress and impair oxygenation of the renal medulla[28,29], while transfusion-related immune responses may intensify systemic inflammation[30]. Concurrent volume loading may also worsen renal venous congestion, particularly in patients with right heart failure[31,32]. Although this observational study cannot test these mechanisms directly, they offer a plausible pathophysiological context for the association we observed.

The dose-response analysis showed a linear association between transfusion volume and CSA-AKI (GAM effective degrees of freedom = 1.00, $p = 8.30 \times 10^{-5}$), with no evidence of a threshold (Supplementary Fig. 3). Transfusion of 201–400 ml, approximately 1–2 units, was associated with 25% higher odds of CSA-AKI (adjusted OR 1.25, 95% CI 1.01–1.55, $p = 0.043$). The ≤ 200 ml group could not be evaluated reliably because very few patients produced quasi-complete separation. Estimates for the remaining categories pointed in the same direction; volumes > 800 ml were associated with higher odds (adjusted OR 1.60, 95% CI 1.29–2.00, $p < 0.001$). These findings do not identify a lower transfusion volume at which the renal association disappears. They also accord with dose-dependent associations between transfusion exposure and renal morbidity reported by Koch[33] and Karkouti[34]. Even modest transfusion volumes should therefore be considered in light of a possible incremental AKI risk.

The mortality findings require cautious interpretation because events were rare. Although cumulative hazard differed between groups, absolute 7-day mortality was very low, with fewer than 30 deaths in the entire cohort. This difference is unlikely to represent a clinically meaningful survival effect. The higher observed mortality in the transfusion group may instead reflect its greater illness severity, including higher SOFA and APS III scores and more frequent vasopressor use. Accordingly, our analysis provides neither evidence that early RBC transfusion improves short-term survival nor sufficient evidence that transfusion itself increases mortality. This interpretation is consistent with Murphy[35], who reported greater morbidity without a survival advantage after cardiac-surgery transfusion, and Engoren[36], who found no improvement in long-term survival after adjustment for illness severity.

Strengths and limitations

Several features strengthen this study. We examined the exposure-outcome association using conventional adjustment, multiple imputation, and overlap weighting, which produced consistent estimates. We also treated death as a competing event when estimating cumulative AKI incidence, rather than assuming that all patients remained observable for a renal outcome. Finally, the relatively large real-world ICU cohort contained detailed early postoperative data, allowing adjustment for important measures of illness severity and treatment intensity.

This study also has limitations. First, its observational design leaves the possibility of residual confounding by factors not included in this analysis, including intraoperative blood loss, nadir hemoglobin, transfusion thresholds, and intraoperative hemodynamics; randomized trials are needed to address causality. Second, the single-center setting may limit generalizability, and the mechanisms linking RBC transfusion to CSA-AKI could not be examined directly. Multicenter studies are needed to confirm the findings. Third, although few key covariates were missing and multiple imputation was used, bias from missing data or residual confounding cannot be excluded. Fourth, the short follow-up and low mortality prevented assessment of long-term renal recovery and survival. Finally, formal interaction tests were not performed, and some subgroups were small; variation across strata should therefore be interpreted cautiously.

Conclusions

Early postoperative RBC transfusion was associated with a linear, dose-dependent increase in CSA-AKI risk, and we found no evidence of a protective threshold. Decisions to transfuse even small volumes during the early postoperative period should

therefore balance the expected benefits of correcting anemia against potential renal risks.

Abbreviations

AKI	Acute kidney injury
APS III	Acute Physiology Score III
ASMD	Absolute standardized mean difference
CCI	Charlson Comorbidity Index
CI	Confidence interval
CKD	Chronic kidney disease
CSA-AKI	Cardiac surgery–associated acute kidney injury
CVICU	Cardiovascular Intensive Care Unit
edf	Effective degrees of freedom
GAM	Generalized additive model
ICU	Intensive Care Unit
IQR	Interquartile range
KDIGO	Kidney Disease: Improving Global Outcomes
MICE	Multiple imputation by chained equations
MIMIC-IV	Medical Information Mart for Intensive Care IV
OR	Odds ratio
RBC	Red blood cell
RRT	Renal replacement therapy
SOFA	Sequential Organ Failure Assessment

Declarations

Acknowledgements

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Author contributions

RM and LP conceived and designed the study. RM extracted the clinical data from MIMIC-IV. XM, CM, FL, and CZ cleaned and curated the data. FW, CZ, and XM performed the statistical analyses. RM and FW drafted the manuscript. LP critically revised the manuscript for important intellectual content. All authors reviewed and approved the final manuscript.

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Data availability

The data analyzed in this study are available in the manuscript and its supplementary files. Additional information may be requested from the corresponding authors.

Ethics approval and consent to participate

Use of the MIMIC-IV database was approved by the Institutional Review Boards of the Massachusetts Institute of Technology (MIT) and Beth Israel Deaconess Medical Center. Because all MIMIC-IV data are deidentified, the requirement for individual informed consent was waived. The study was conducted in accordance with the Declaration of Helsinki. Clinical trial number: not applicable.

Consent for publication

All authors consent to publication of this work.

Competing interests

None.

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Tables

Tables 1 and 2 are available in the Supplementary Files section.

Figures

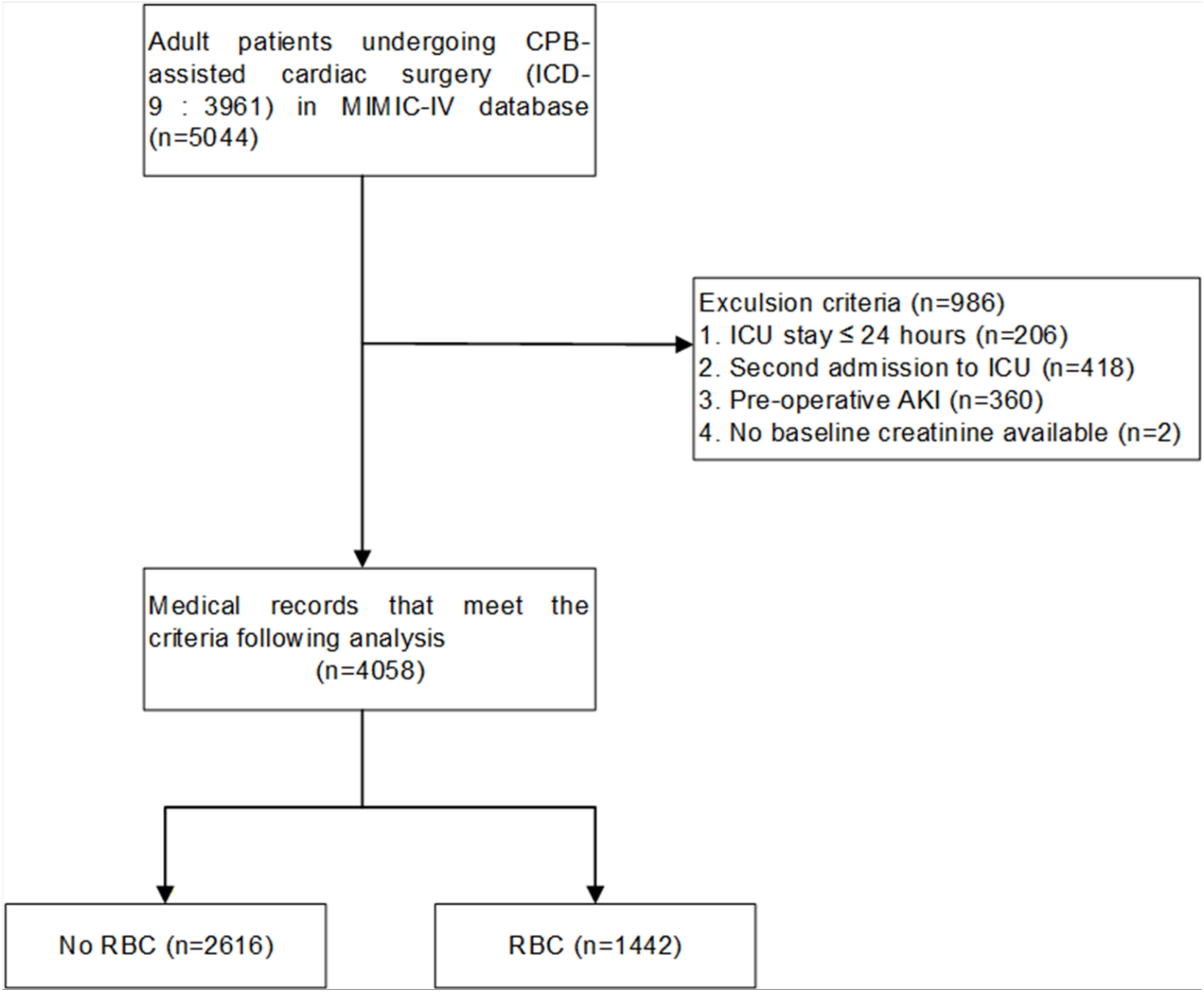


Figure 1

Study flowchart. ICU, intensive care unit; MIMIC-IV, Medical Information Mart for Intensive Care IV; AKI, acute kidney injury.

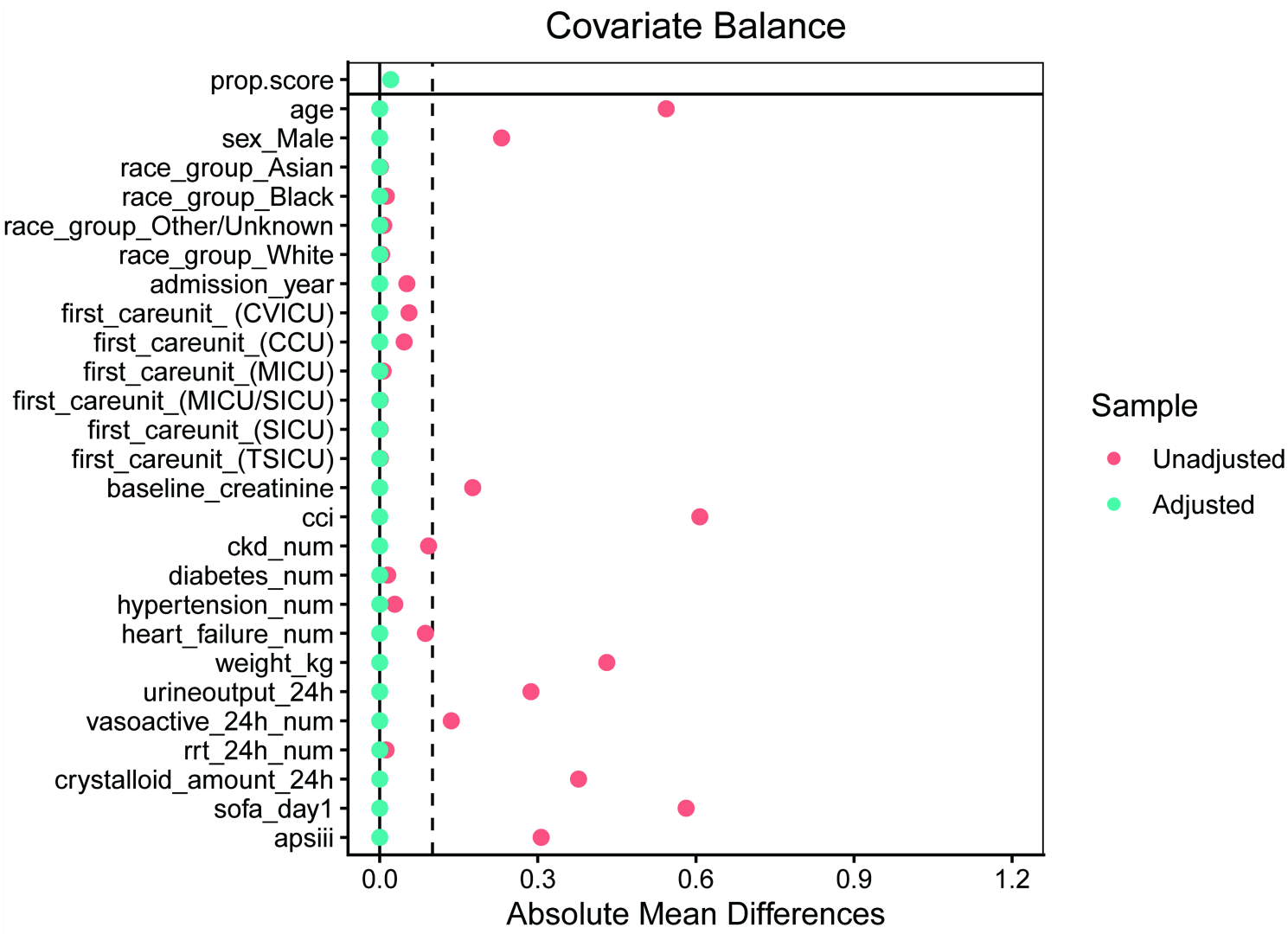


Figure 2

Covariate balance before and after overlap propensity score weighting.

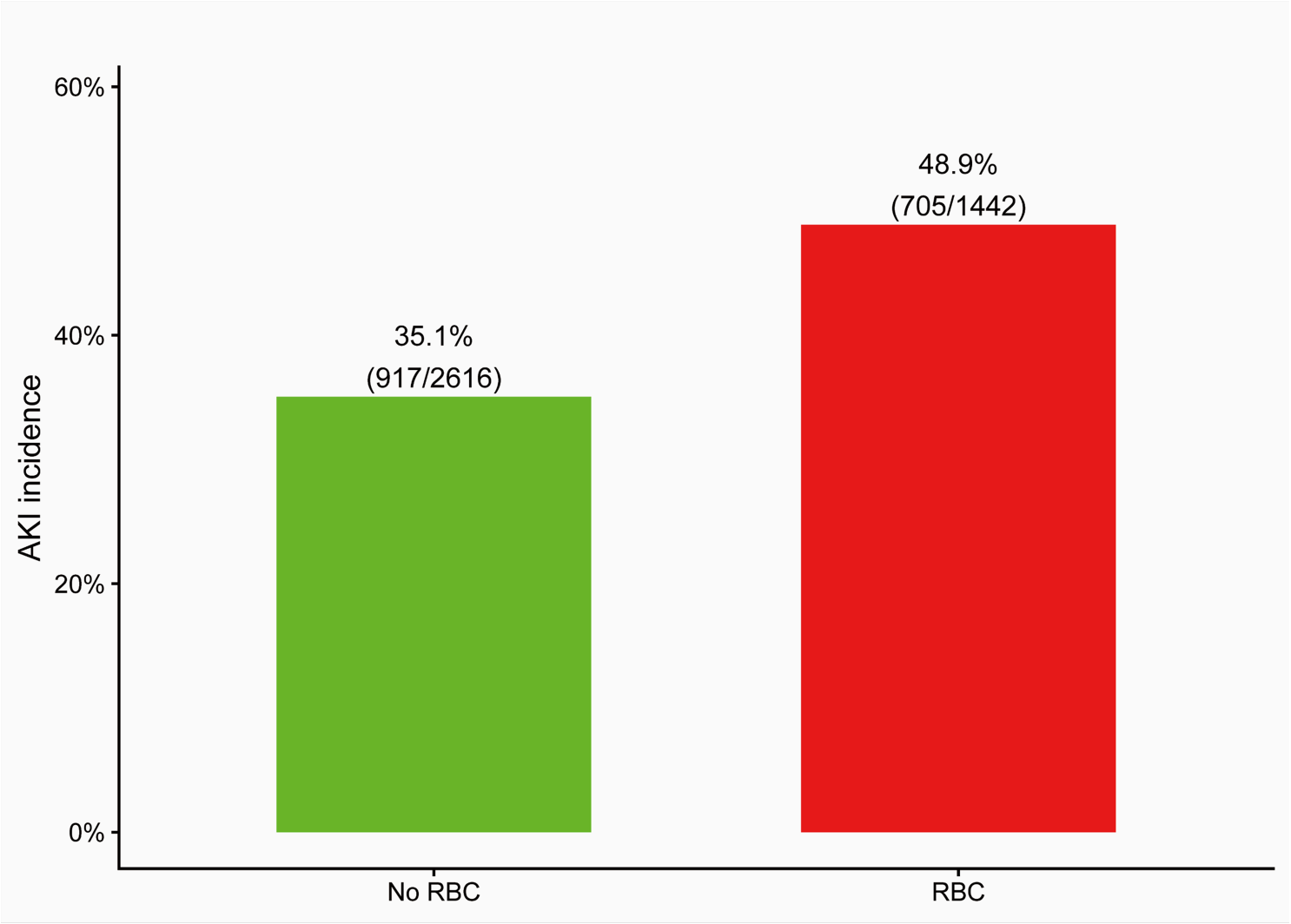


Figure 3

Crude incidence of 7-day CSA-AKI by early postoperative RBC transfusion status.

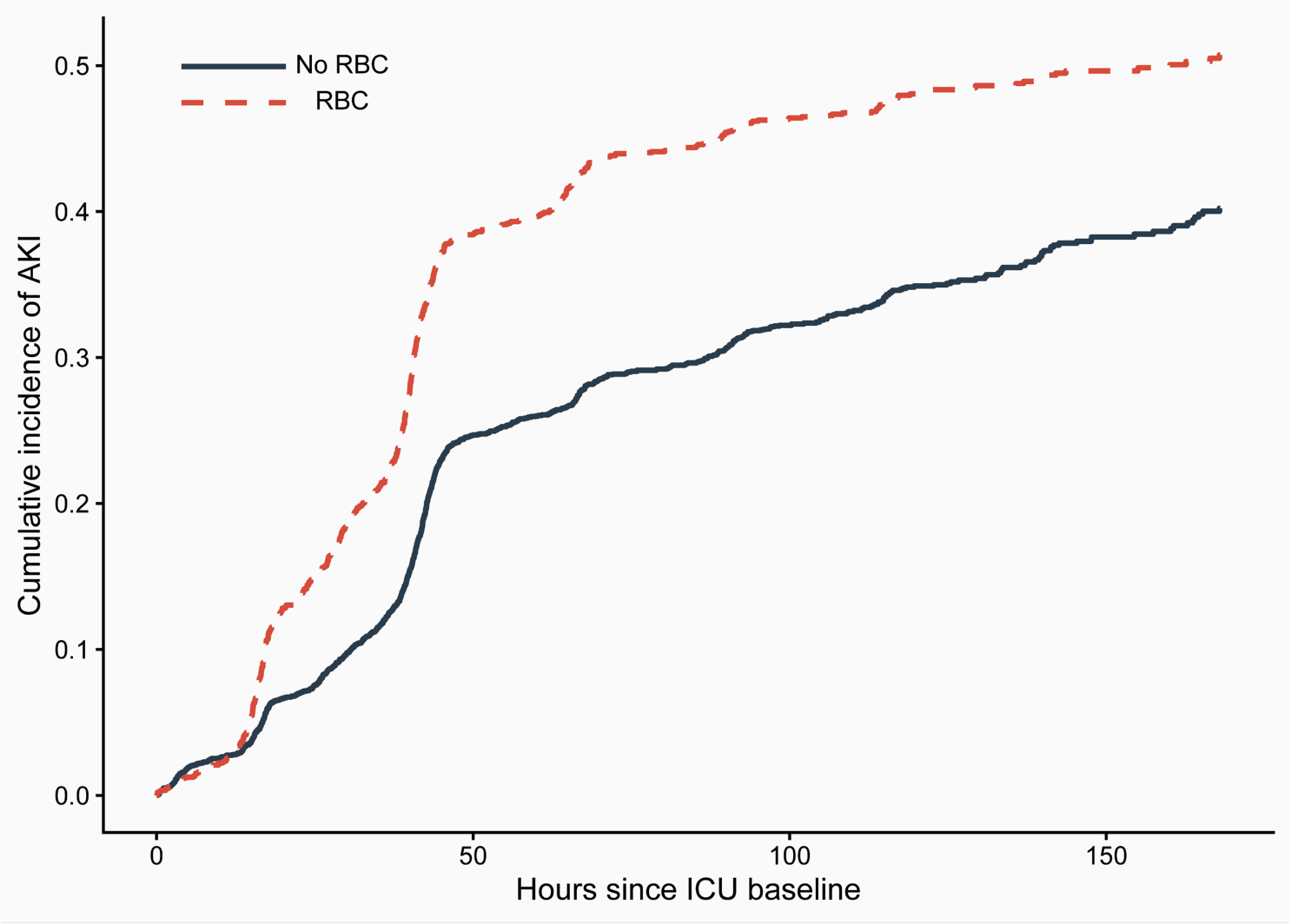


Figure 4

Crude incidence of 7-day CSA-AKI by early postoperative RBC transfusion status.

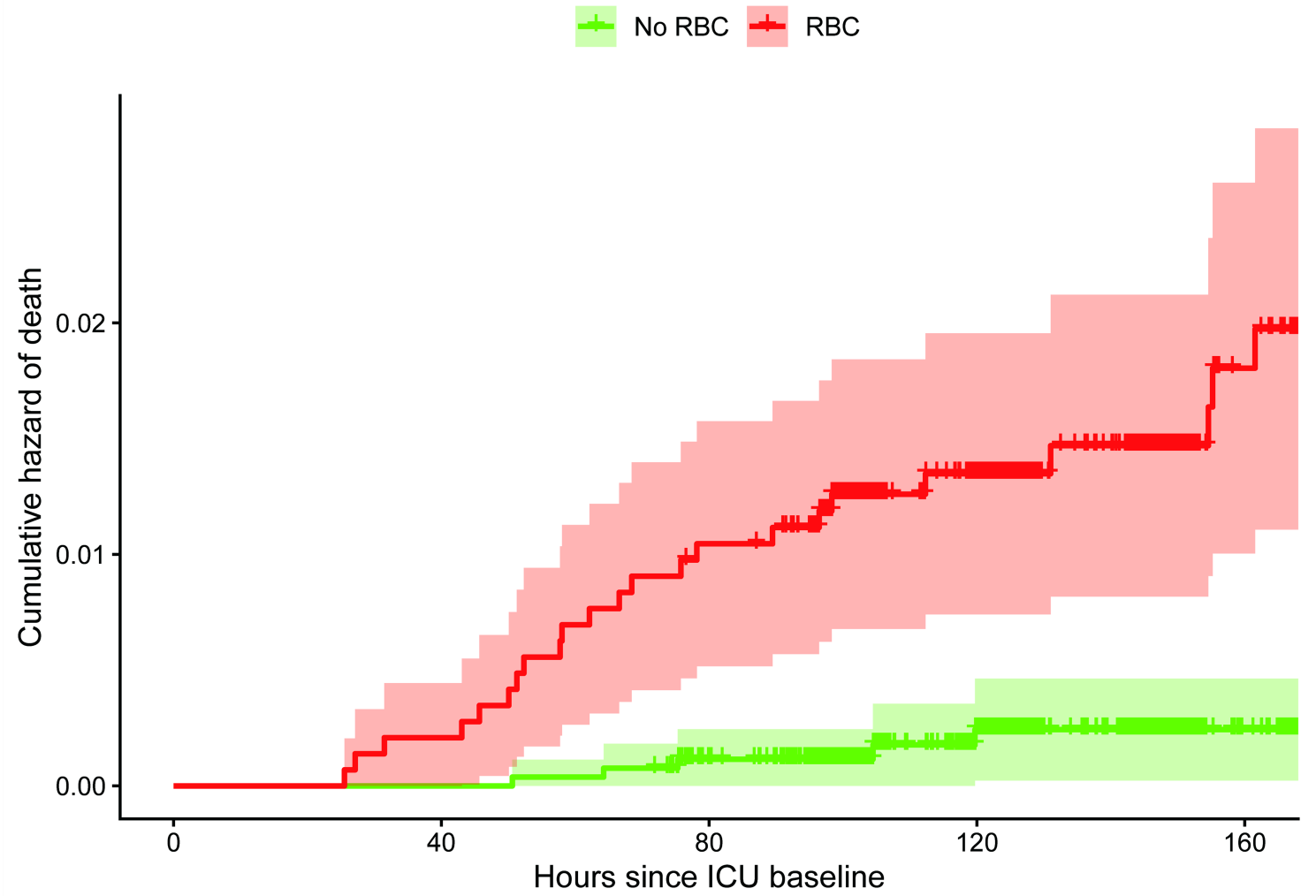


Figure 5

Nelson-Aalen cumulative hazard curves for 7-day all-cause mortality according to early postoperative RBC transfusion status.

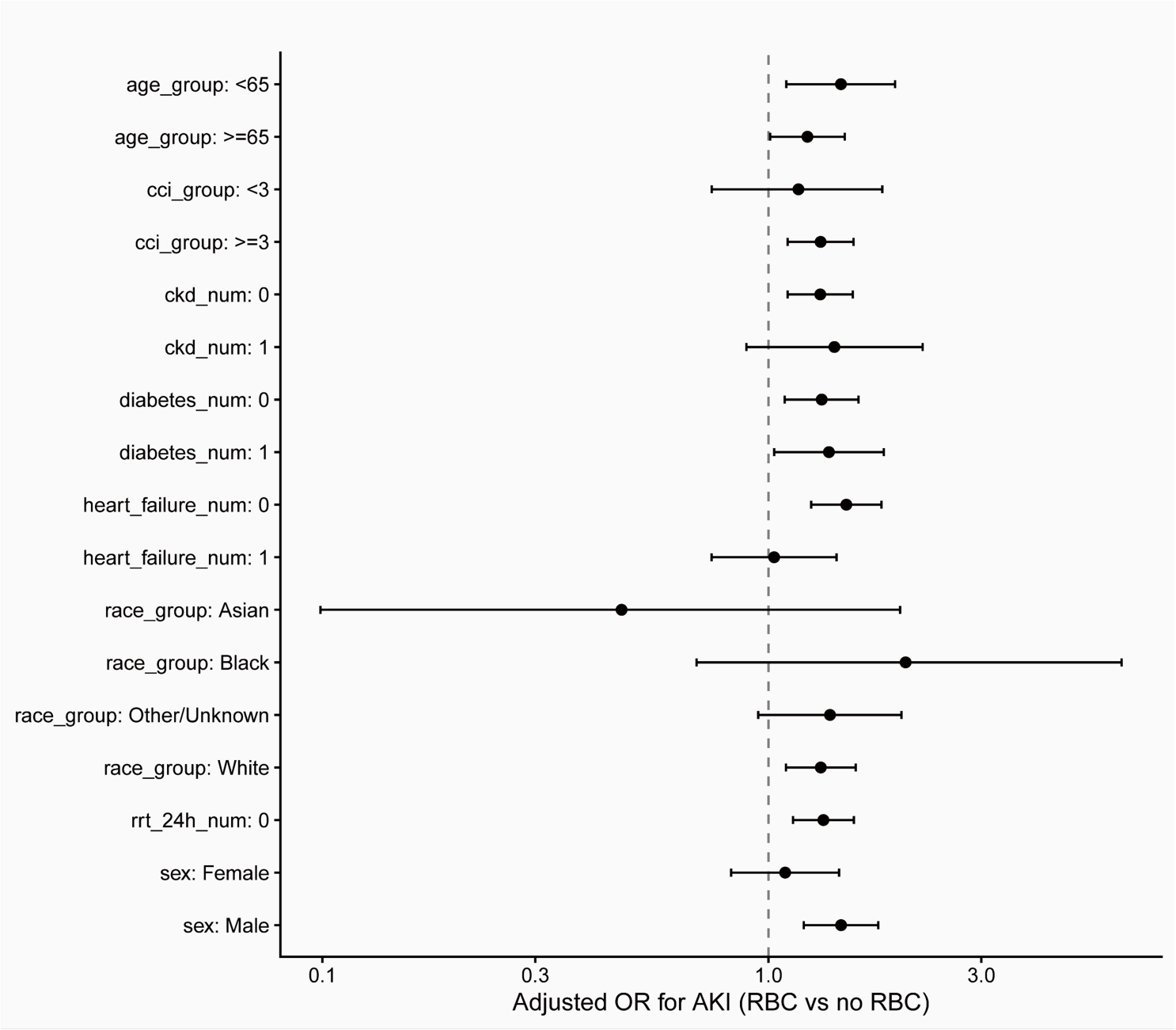


Figure 6

Forest plot of adjusted odds ratios for the association between early postoperative RBC transfusion and 7-day CSA-AKI across prespecified subgroups. CCI, Charlson Comorbidity Index; CKD, chronic kidney disease; RRT, renal replacement therapy.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Table1.BaselinecharacteristicsofpatientswithandwithoutearlypostoperativeRBCtransfusion.xlsx
- Table2.AssociationbetweenearlypostoperativeRBCtransfusionand7dayCSAAKIacrossprimaryandrestrictedanalyticmodels.xlsx