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Original Article

Clinical impact and safety of continuous renal replacement therapy in critically ill patients with solid tumors and acute kidney injury: a retrospective cohort analysis

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Abstract: Objectives: To evaluate the efficacy and safety of continuous renal replacement therapy (CRRT) in critically ill patients with solid tumors complicated by acute kidney injury (AKI) in the intensive care unit (ICU). Methods: In this retrospective cohort study, 580 ICU patients with solid tumors and AKI were enrolled and stratified into a CRRT group (n = 300) and a non-CRRT group (n = 280). CRRT was initiated within 12 hours of ICU admission in patients meeting Kidney Disease Improving Global Outcomes stage 3 criteria. Propensity score matching (PSM) was performed to balance baseline characteristics. Primary outcomes included AKI remission, metabolic stabilization, and 90-day survival. Multivariable logistic regression and XGBoost machine learning were used to identify prognostic factors and build predictive models. Results: After PSM, baseline characteristics were well balanced. CRRT significantly improved renal and metabolic parameters within 72 hours, including reductions in serum creatinine, blood urea nitrogen, and lactate (all $P < 0.001$), with a trend toward increased urine output. The CRRT group had higher AKI remission (64.54% vs. 49.65%, $P < 0.001$) and 90-day survival rates (47.52% vs. 39.01%, $P = 0.012$), albeit with greater dialysis dependence (19.15% vs. 0.00%, $P < 0.001$). Adverse events were comparable. XGBoost models achieved AUCs of 0.78 and 0.75 for mortality and AKI remission, respectively. Conclusions: CRRT improves renal recovery, metabolic status, and survival in critically ill cancer patients with AKI, with an acceptable safety profile. Machine learning offers promising tools for individualized outcome prediction.

Keywords: Continuous renal replacement therapy, acute kidney injury, solid tumors, critical care, machine learning

Introduction

Recent advances in oncologic therapies have significantly improved survival in patients with solid tumors, resulting in a growing population undergoing complex multimodal treatments. However, this progress has been accompanied by a higher incidence of treatment-related complications, increasing intensive care unit (ICU) demand in this vulnerable group. Current epidemiological data indicate that patients with malignancies comprise approximately 20% of ICU admissions, the majority being individuals with advanced or metastatic solid tumors [1, 2]. These patients frequently present with profound immunosuppression and multi-organ dysfunction, requiring comprehensive life support measures such as mechanical ventilation, vasopressors, and renal replacement therapy (RRT) [1, 3]. A multicenter study identified lung and gastrointestinal cancers as the most common solid tumors in ICU settings, with over half of patients exhibiting distant metastases. While short-term survival during ICU stay was relatively high (77.4%), one-year survival declined sharply to 33.2%, underscoring the poor long-term prognosis associated with organ support dependency [3].

Among organ dysfunctions, acute kidney injury (AKI) is both prevalent and prognostically significant. Its etiology in cancer patients is multifactorial, including tumor-related processes, sepsis, nephrotoxic agents, and hemodynamic

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instability [4]. A recent meta-analysis reported AKI incidence rates up to 52% in critically ill cancer patients, with approximately 48% of those with solid tumors affected [5], far exceeding rates in the general ICU population.

AKI substantially worsens clinical outcomes. Its occurrence is linked to increased short-term mortality, including ICU and 28-day mortality, and has been identified as an independent predictor of death in oncology patients - 56% in solid tumors and up to 78% in hematologic malignancies [6]. These data highlight the urgent need for early recognition and timely management of AKI in cancer ICU populations to improve survival and preserve renal function.

CRRT is commonly used to manage AKI in ICU patients with solid tumors, but its clinical utility remains controversial. A retrospective study reported a 28-day survival of only 57.1% in postoperative cancer patients with stage 3 AKI receiving CRRT, raising concerns about cost-effectiveness and clinical benefit [7]. In contrast, a study from MD Anderson Cancer Center found no significant association between RRT and hospital or long-term survival in ICU patients with stage IV solid tumors and AKI [8]. Notably, most patients in that study had terminal malignancies and received RRT as part of palliative care, with AKI often stemming from end-stage cancer processes. These findings reflect the limitations of generalizability due to population heterogeneity. Moreover, many prior studies combined solid and hematologic tumors and lacked tumor-specific prognostic stratification tools [9].

This study aims to systematically assess the short- and intermediate-term outcomes of CRRT in patients with solid tumors, spanning various TNM stages and comorbidity profiles, with a focus on those presenting with AKI. By addressing current gaps, this research seeks to provide evidence-based guidance for CRRT use in patients with non-hematologic malignancies and to improve risk stratification and ICU management in this high-risk population.

Materials and methods

Study design and patient selection

This retrospective observational cohort study aimed to evaluate the impact of CRRT on renal recovery and short-term clinical outcomes in ICU patients with solid tumors who developed AKI. The study was conducted at the Longyan First Affiliated Hospital of Fujian Medical University between September 2024 and May 2025. Ethical approval was obtained from the Medical Ethics Committee of Longyan First Affiliated Hospital of Fujian Medical University (Approval No. LYREC2025-K121-01). Given the retrospective nature of the study, the requirement for informed consent was waived.

Inclusion criteria: (1) Age ≥ 18 years; (2) Histologically or clinically confirmed diagnosis of a solid tumor, primarily gastrointestinal or pulmonary malignancies.

Diagnosis of AKI during ICU stay according to the 2012 Kidney Disease Improving Global Outcomes (KDIGO) Clinical Practice Guideline for Acute Kidney Injury [10], defined as meeting at least one of the following: (1) Increase in serum creatinine by ≥ 0.3 mg/dL (26.5 μmol/L) within 48 hours; Increase in serum creatinine to ≥ 1.5 times baseline within the prior 7 days; Urine output < 0.5 mL/kg/h for ≥ 6 hours; (2) ICU length of stay ≥ 48 hours; (3) Availability of complete clinical and laboratory data.

Exclusion criteria: (1) Pre-existing end-stage renal disease (ESRD) or chronic dialysis prior to ICU admission; (2) Diagnosis of hematologic malignancy; (3) Death or discharge within 48 hours of ICU admission; (4) CRRT initiated prior to ICU admission; (5) Concurrent enrollment in interventional clinical trials.

Grouping and treatment protocol

A total of 580 patients with solid tumors complicated by AKI who were admitted to the ICU were included. Patients were stratified into a CRRT group (n = 300) and a non-CRRT group (n = 280) based on the actual treatment received. The decision to initiate CRRT was made by the attending intensivists in accordance with KDIGO stage 3 AKI criteria, considering hemodynamic stability, metabolic disturbances, and fluid overload, consistent with standard ICU practice.

CRRT was initiated in 268 patients who met one or more of the following criteria: refractory fluid overload, life-threatening hyperkalemia, severe metabolic acidosis, or uremic complica-

tions. The median time from ICU admission to CRRT initiation was 12 hours (interquartile range [IQR]: 8-18 hours). CRRT parameters, including timing and prescription, were individualized. Treatment was delivered at a prescribed effluent dose of 25 ± 5 mL/kg/h, most commonly set at 25 mL/kg/h. Regional citrate anticoagulation was employed in approximately 80% of patients, while systemic anticoagulation with unfractionated heparin was used in the remaining 20%. The median CRRT duration was 48 hours (IQR: 24-72 hours). Discontinuation was based on hemodynamic stabilization or sustained renal recovery. The mean cumulative net fluid removal over the first 72 hours was -1.0 L.

Patients who did not receive CRRT initially were managed conservatively for at least 48 hours. If renal function did not improve - defined as < 10% reduction in serum creatinine and urine output < 0.5 mL/kg/h - a multidisciplinary team re-evaluated CRRT indications. Based on actual treatment received, patients were ultimately categorized into the CRRT group (n = 300) or the non-CRRT group (n = 280).

Patients in the non-CRRT group received no RRT but were managed with standard ICU supportive measures. Fluid management targeted a daily net fluid balance between -500 mL and +500 mL. Antimicrobial therapy was guided by culture results, susceptibility testing, or empirical broad-spectrum protocols. Vasopressors were titrated to achieve target mean arterial pressure and central venous pressure. All patients received comprehensive supportive care, including respiratory support, enteral or parenteral nutrition, and correction of acid-base and electrolyte disturbances.

Data collection

All data were retrospectively extracted from the hospital's integrated clinical information systems, including the electronic medical record (EMR), laboratory information system (LIS), and nursing information system (NIS). Data extraction was independently performed by two trained researchers using a predefined standardized protocol. A senior intensivist cross-checked all entries for consistency and accuracy. In cases of discrepancy or missing data, a third investigator adjudicated.

Collected baseline variables included age, sex, body mass index (BMI), tumor type, TNM stage, and prior cancer therapies (chemotherapy, radiotherapy, targeted therapy, or immunotherapy). Disease severity on ICU admission was assessed using the Sequential Organ Failure Assessment (SOFA) score and the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, based on clinical and laboratory data documented within the first 24 hours of ICU stay. AKI severity was staged according to the 2012 KDIGO criteria, categorized as Stage 1 to Stage 3 [11]. Relevant data were obtained from LIS and nursing fluid balance records.

Information on comorbid conditions - such as diabetes mellitus, hypertension, coronary artery disease, septic shock, and prior exposure to nephrotoxic agents - was extracted from admission assessments, medication orders, and discharge summaries. Laboratory and metabolic parameters, including serum creatinine (Scr), blood urea nitrogen (BUN), uric acid (UA), potassium, phosphate, and lactate, were collected at ICU admission (defined as hour 0), and again at 48 and 72 hours, with the first available value within each time window being recorded. Hourly urine output (mL/kg/h) was retrieved from nursing records and fluid input/output charts. This variable was used to assess renal function dynamics over time.

Primary outcomes included: Renal recovery: defined as a ≥ 25% reduction in serum creatinine from baseline, accompanied by an average urine output ≥ 0.5 mL/kg/h within 48 hours. This outcome was independently determined by two investigators based on laboratory and nursing data; discrepancies were resolved by a senior nephrologist; Overall survival at 28 and 90 days: verified through inpatient records and follow-up telephone interviews; ICU length of stay: calculated from the time of ICU admission to ICU discharge; Dialysis dependence at discharge: determined by reviewing discharge instructions and nephrology consultation notes. Patients requiring ongoing RRT at discharge were classified as dialysis-dependent.

Complications were identified based on explicit documentation of diagnoses and clinical interventions: Infections: confirmed by a combination of positive microbiological cultures and the

initiation or escalation of targeted antibiotic therapy. This included bloodstream infections, ventilator-associated pneumonia, and catheter-related infections, as defined by established CDC/NHSN surveillance criteria; Major bleeding: identified through clinical documentation and supported by laboratory findings (e.g., a ≥ 2 g/dL drop in hemoglobin) and transfusion records (≥ 2 units of packed red blood cells), or bleeding at critical sites such as intracranial, gastrointestinal, or retroperitoneal locations; Electrolyte disturbances: life-threatening abnormalities requiring immediate intervention, such as hyperkalemia (> 6.5 mmol/L), severe hypokalemia (< 2.5 mmol/L), hyperphosphatemia (> 2.5 mmol/L), and symptomatic hypocalcemia. These were confirmed through laboratory test results and the presence of emergency treatment orders, such as calcium infusion or insulin-glucose administration.

Statistical analysis

All statistical analyses were performed using SPSS Statistics (Version 26.0, IBM Corp., Armonk, NY, USA), R software (version 4.3.2; R Foundation for Statistical Computing, Vienna, Austria). A two-sided *P*-value < 0.05 was considered statistically significant unless otherwise specified. Normality of continuous variables was tested using the Shapiro-Wilk test. Variables with normal distribution were expressed as mean ± standard deviation (SD) and compared between groups using the independent-samples t-test. Non-normally distributed data were reported as median [interquartile range] and analyzed using the Mann-Whitney U test. Categorical variables were summarized as frequencies and percentages, and group differences were assessed using Pearson's χ^2 test or Fisher's exact test when appropriate.

Descriptive analysis and propensity score matching (PSM): To reduce treatment selection bias and control for confounding between groups, PSM was performed. Before matching, 300 patients who received CRRT were compared to 280 who did not, based on whether CRRT was administered during the ICU stay.

Baseline variables included in the matching model were age, sex, BMI, tumor type (primarily gastrointestinal or pulmonary), TNM stage, SOFA score, APACHE II score, prior oncologic therapy, and AKI stage per KDIGO guidelines. Propensity scores were estimated using clinically relevant covariates with a standardized mean difference (SMD) > 0.1. A 1:1 nearest-neighbor matching algorithm without replacement was applied, using a caliper width of 0.2 SDs of the logit-transformed propensity score. Covariate balance was confirmed by SMD < 0.1. All PSM procedures and diagnostics were conducted using R (v4.3.2) and the "MatchIt" package (v4.5.2). Matched cohorts were then used for subsequent outcome analyses.

Longitudinal analysis of renal and metabolic indices: To compare the temporal effects of CRRT on renal function and metabolism, repeated-measures ANOVA was used to analyze laboratory values at 0 h, 48 h, and 72 h. The statistical model included main effects for time and treatment group, as well as an interaction term (time × group) to examine differential trajectories. When a significant interaction or main effect was detected, Bonferroni-adjusted post hoc pairwise comparisons were conducted to determine specific timepoints at which group differences occurred.

Subgroup analyses: Prespecified subgroup analyses were conducted to assess effect modification based on key comorbidities, including diabetes mellitus, hypertension, coronary artery disease, and septic shock. Stratified logistic regression models were used to estimate odds ratios (ORs) for treatment effects within each subgroup. Interaction *p*-values were calculated to assess heterogeneity across subgroups.

Multivariable logistic regression: To identify independent predictors of AKI recovery (binary outcome), a multivariable logistic regression model was constructed using backward stepwise selection. Candidate variables included CRRT exposure, age, SOFA score, AKI stage, comorbidities (e.g., diabetes, hypertension, septic shock), hemodynamic instability, and tumor type (gastrointestinal vs. pulmonary). Statistical significance was defined as P < 0.05.

For internal validation, the matched cohort (n = 282) was randomly split into a training set (70%, n = 198) and a testing set (30%, n = 84)

using stratified sampling to maintain outcome distribution. Model construction and variable selection were performed on the training set; performance was evaluated on the testing set. Discrimination was assessed using the area under the receiver operating characteristic (ROC) curve (AUC), with AUC > 0.70 considered good. Calibration was evaluated via the Hosmer-Lemeshow test, with P > 0.05 indicating acceptable fit. Analyses used the R packages pROC, ResourceSelection, and rmda.

Machine learning models: To supplement traditional analyses, supervised machine learning models were developed using Python 3.10 and the scikit-learn library to identify the key predictors that might be related to 90-day mortality and AKI remission outcomes. Preprocessing included standardization and imputation of missing values via the K-nearest neighborsalgorithm. An eXtreme Gradient Boosting (XGBoost) classifier was trained with five-fold cross-validation.

Model performance was assessed via AUC, sensitivity, and specificity. Model interpretability was enhanced using SHapley Additive exPlanations (SHAP) to quantify individual feature contributions. Visualizations, including ROC curves and SHAP summary plots, were generated using matplotlib and shap.

Results

Comparison of baseline characteristics

As summarized in **Table 1** and **Figure 1**, baseline characteristics were well balanced postmatching. No significant differences were observed between groups in age, sex, BMI, tumor type (predominantly gastrointestinal and pulmonary), or disease severity scores (SOFA and APACHE II). Likewise, TNM stage, prior oncologic treatment, and AKI stage were comparable (all P > 0.05).

Figure 2A illustrates propensity score distributions before and after matching. Before PSM (left panel), the two groups exhibited marked differences, indicating potential selection bias. After PSM (right panel), distributions were nearly identical, demonstrating successful balance. **Figure 2B** displays standardized mean differences (SMDs) for each covariate pre- and postmatching. Prior to matching, imbalances were evident for variables such as SOFA score, APACHE II score, and AKI stage (SMD > 0.1). Post-matching, all covariates achieved SMDs < 0.1, confirming adequate covariate balance and effective control of baseline confounding.

Comparison of renal and metabolic parameters

Post-matching comparisons of renal and metabolic indicators were performed at baseline, 48 hours, and 72 hours after enrollment (**Figures 3** and **4**). At baseline, the CRRT group had more severe renal dysfunction, with higher Scr, BUN, and UA compared to the non-CRRT group (all P < 0.05). During treatment, these parameters decreased markedly in the CRRT group, with significantly greater reductions in the non-CRRT group (all P < 0.001). UA levels also decreased, though intergroup differences narrowed by 72 hours (**Figure 3**). Repeated-measures ANOVA revealed significant time-by-treatment interactions for Scr (F = 124.3), BUN (F = 101.7), and UA (F = 52.9), (all P < 0.001), indicating faster renal recovery in the CRRT group (**Table 2**). Bonferroni-adjusted post hoc tests confirmed that intra-group differences in Scr, BUN, and UA across the three timepoints (0 h, 48 h, and 72 h) were all statistically significant (P < 0.05) (**Figure 3**).

Regarding metabolic parameters, baseline serum potassium, phosphate, and lactate were significantly higher in the CRRT group (all P < 0.01), suggesting more severe metabolic derangements. These values improved progressively during CRRT, with significant reductions over time (P < 0.05). Although urine output remained lower in the CRRT group throughout observation, a gradual increase was noted - from 0.62 to 0.74 mL/kg/h by 72 hours - indicating potential renal recovery. In contrast, urine output in the non-CRRT group remained largely unchanged (**Figure 4**). Repeated-measures ANOVA showed significant time-by-treatment interactions for potassium (F = 19.4), phosphate (F = 38.1), lactate (F = 25.5), and urine output (F = 6.7), (P < 0.001 for potassium, phosphate, and lactate; P = 0.003 for urine output) (**Table 2**). Similarly, post hoc analysis showed that within both the CRRT and non-CRRT groups, serum potassium, phosphate,

Table 1. Comparison of baseline characteristics (mean ± standard deviation (SD) or n (%))

Variables	Before PSM					After PSM				
	CRRT (n = 300)	Non-CRRT (n = 280)	Statistic	*P*	SMD	CRRT (n = 141)	Non-CRRT (n = 141)	Statistic	*P*	SMD
Age, Mean ± SD	60.98 ± 9.88	59.76 ± 10.96	t = 1.414	0.158	-0.112	60.62 ± 10.04	60.60 ± 11.04	t = 0.013	0.989	-0.002
BMI, Mean ± SD	22.40 ± 3.62	22.33 ± 3.42	t = 0.225	0.822	-0.019	22.24 ± 3.36	22.31 ± 3.27	t = -0.183	0.855	0.022
SOFA, Mean ± SD	9.81 ± 3.00	6.35 ± 2.69	t = 14.609	< 0.001	-1.289	7.86 ± 2.41	7.52 ± 2.61	t = 1.121	0.263	-0.129
APACHE II, Mean ± SD	22.82 ± 6.87	18.92 ± 6.55	t = 6.982	< 0.001	-0.595	20.82 ± 6.57	20.09 ± 6.41	t = 0.944	0.346	-0.114
Tumor Type, n (%)			χ^2 = 0.773	0.679				χ^2 = 2.570	0.277	
1	180 (60.00)	165 (58.93)			-0.022	74 (52.48)	84 (59.57)			0.145
2	55 (18.33)	59 (21.07)			0.067	30 (21.28)	31 (21.99)			0.017
3	65 (21.67)	56 (20.00)			-0.042	37 (26.24)	26 (18.44)			-0.201
TNM Stage, n (%)			χ^2 = 1.831	0.608				χ^2 = 3.435	0.329	
1	25 (8.33)	28 (10.00)			0.056	9 (6.38)	13 (9.22)			0.098
2	37 (12.33)	42 (15.00)			0.075	16 (11.35)	21 (14.89)			0.100
3	113 (37.67)	105 (37.50)			-0.003	61 (43.26)	47 (33.33)			-0.211
4	125 (41.67)	105 (37.50)			-0.086	55 (39.01)	60 (42.55)			0.072
Gender, n (%)			χ^2 = 3.215	0.073				χ^2 = 1.168	0.280	
Male	113 (37.67)	126 (45.00)			0.147	57 (40.43)	66 (46.81)			0.128
Female	187 (62.33)	154 (55.00)			-0.147	84 (59.57)	75 (53.19)			-0.128
Chemo, n (%)			χ^2 = 0.190	0.663				χ^2 = 0.014	0.905	
No	149 (49.67)	134 (47.86)			-0.036	67 (47.52)	66 (46.81)			-0.014
Yes	151 (50.33)	146 (52.14)			0.036	74 (52.48)	75 (53.19)			0.014
Radiotherapy, n (%)			χ^2 = 0.024	0.877				χ^2 = 1.220	0.269	
No	191 (63.67)	180 (64.29)			0.013	83 (58.87)	92 (65.25)			0.134
Yes	109 (36.33)	100 (35.71)			-0.013	58 (41.13)	49 (34.75)			-0.134
Targeted Therapy, n (%)			χ^2 = 1.559	0.212				χ^2 = 0.076	0.783	
No	230 (76.67)	202 (72.14)			-0.101	107 (75.89)	105 (74.47)			-0.033
Yes	70 (23.33)	78 (27.86)			0.101	34 (24.11)	36 (25.53)			0.033
Immunotherapy, n (%)			χ^2 = 0.260	0.610				χ^2 = 1.781	0.182	
No	245 (81.67)	224 (80.00)			-0.042	117 (82.98)	108 (76.60)			-0.151
Yes	55 (18.33)	56 (20.00)			0.042	24 (17.02)	33 (23.40)			0.151
DM, n (%)			χ^2 = 3.509	0.061				χ^2 = 0.271	0.602	
No	197 (65.67)	204 (72.86)			0.162	101 (71.63)	97 (68.79)			-0.061
Yes	103 (34.33)	76 (27.14)			-0.162	40 (28.37)	44 (31.21)			0.061
HTN, n (%)			χ^2 = 0.422	0.516				χ^2 = 1.085	0.298	
No	210 (70.00)	189 (67.50)			-0.053	103 (73.05)	95 (67.38)			-0.121
Yes	90 (30.00)	91 (32.50)			0.053	38 (26.95)	46 (32.62)			0.121

CHD, n (%)			$\chi^2 = 3.085$	0.079				$\chi^2 = 0.033$	0.857	
No	253 (84.33)	250 (89.29)			0.160	123 (87.23)	124 (87.94)			0.022
Yes	47 (15.67)	30 (10.71)			-0.160	18 (12.77)	17 (12.06)			-0.022
Septic Shock, n (%)			$\chi^2 = 0.675$	0.411				$\chi^2 = 1.652$	0.199	
No	213 (71.00)	190 (67.86)			-0.067	102 (72.34)	92 (65.25)			-0.149
Yes	87 (29.00)	90 (32.14)			0.067	39 (27.66)	49 (34.75)			0.149
Sepsis, n (%)			$\chi^2 = 0.221$	0.638				$\chi^2 = 1.717$	0.190	
No	142 (47.33)	138 (49.29)			0.039	64 (45.39)	75 (53.19)			0.156
Yes	158 (52.67)	142 (50.71)			-0.039	77 (54.61)	66 (46.81)			-0.156
Tumor Lysis Syndrome, n (%)			$\chi^2 = 0.119$	0.730				$\chi^2 = 0.085$	0.771	
No	235 (78.33)	216 (77.14)			-0.028	110 (78.01)	112 (79.43)			0.035
Yes	65 (21.67)	64 (22.86)			0.028	31 (21.99)	29 (20.57)			-0.035
Hemodynamic Disorder, n (%)			$\chi^2 = 2.079$	0.149				$\chi^2 = 0.228$	0.633	
No	131 (43.67)	139 (49.64)			0.120	63 (44.68)	67 (47.52)			0.057
Yes	169 (56.33)	141 (50.36)			-0.120	78 (55.32)	74 (52.48)			-0.057
Nephrotoxic History, n (%)			$\chi^2 = 0.546$	0.460				$\chi^2 = 0.357$	0.550	
No	239 (79.67)	216 (77.14)			-0.060	115 (81.56)	111 (78.72)			-0.069
Yes	61 (20.33)	64 (22.86)			0.060	26 (18.44)	30 (21.28)			0.069
AKI Stage, n (%)			$\chi^2 = 22.060$	< 0.001				$\chi^2 = 0.273$	0.872	
1	47 (15.67)	25 (8.93)			-0.236	19 (13.48)	18 (12.77)			-0.021
2	62 (20.67)	105 (37.50)			0.348	40 (28.37)	44 (31.21)			0.061
3	191 (63.67)	150 (53.57)			-0.202	82 (58.16)	79 (56.03)			-0.043

t: t-test, χ^2: Chi-square test, SD: standard deviation; PSM, Propensity score matching.

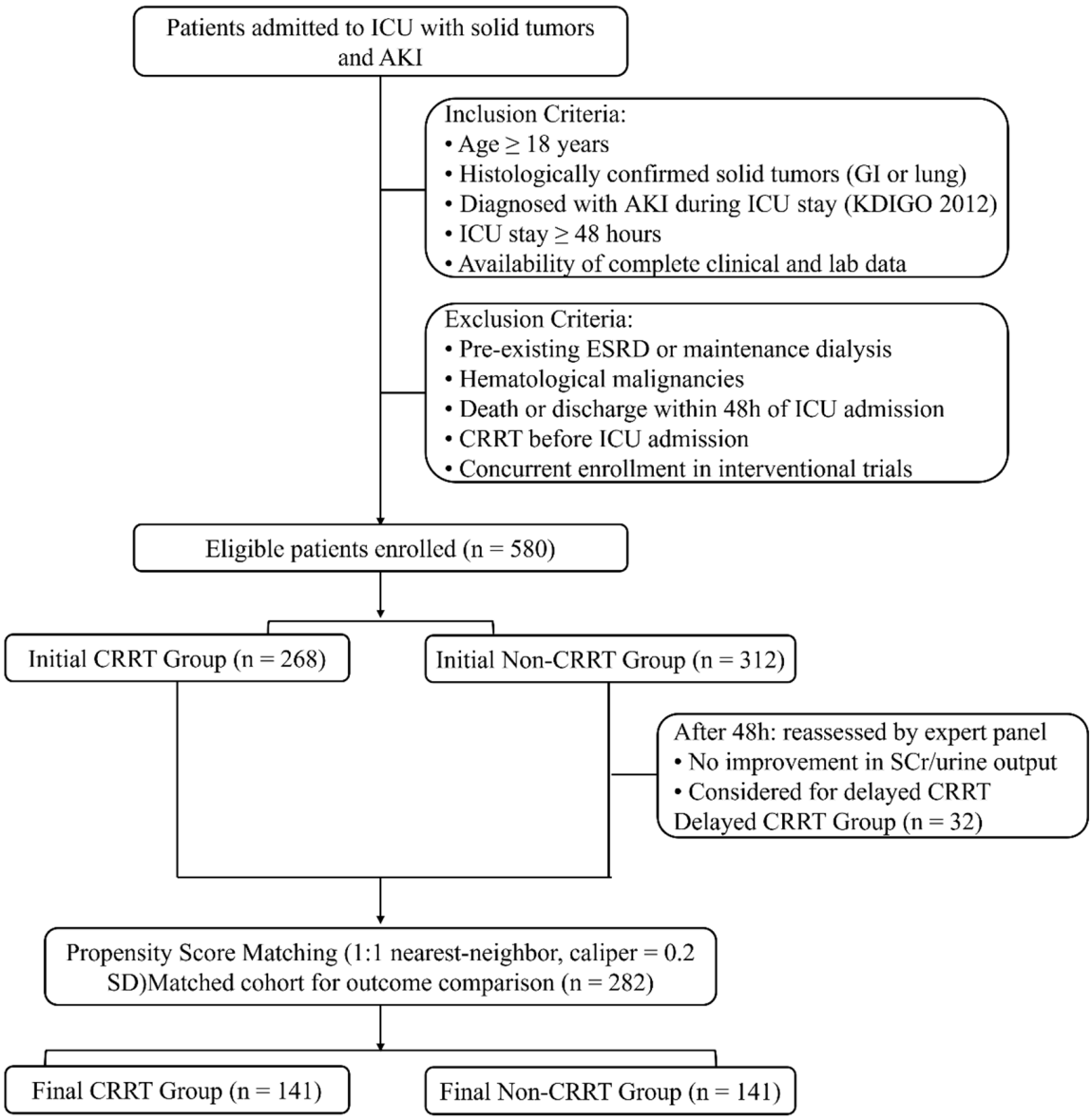


Figure 1. Flowchart of study design and patient selection.

and lactate levels significantly decreased from 0 h to 48 h and from 48 h to 72 h ($P < 0.05$), while urine output significantly increased over time ($P < 0.05$) (**Figure 4**).

Overall, these findings indicate that CRRT not only facilitates rapid correction of metabolic abnormalities but may also promote residual renal function recovery.

Comparison of clinical outcomes

Patients who received CRRT had significantly longer ICU stays than those in the non-CRRT group (14.45 ± 3.18 vs. 9.99 ± 2.23 days, $P < 0.001$), consistent with its use in more severely ill individuals. Although 28-day survival did not differ significantly between groups (63.12% vs. 40.43%, $P = 0.149$), 90-day survival was significantly higher in the CRRT group (47.52% vs. 39.01%, $P = 0.012$), suggesting potential mid-to-long-term benefits. Renal recovery was also more frequent in the CRRT group, which demonstrated a significantly higher AKI remission rate (64.54% vs. 49.65%, $P < 0.001$). However, dialysis dependence at discharge was more common among CRRT recipients (19.15% vs.

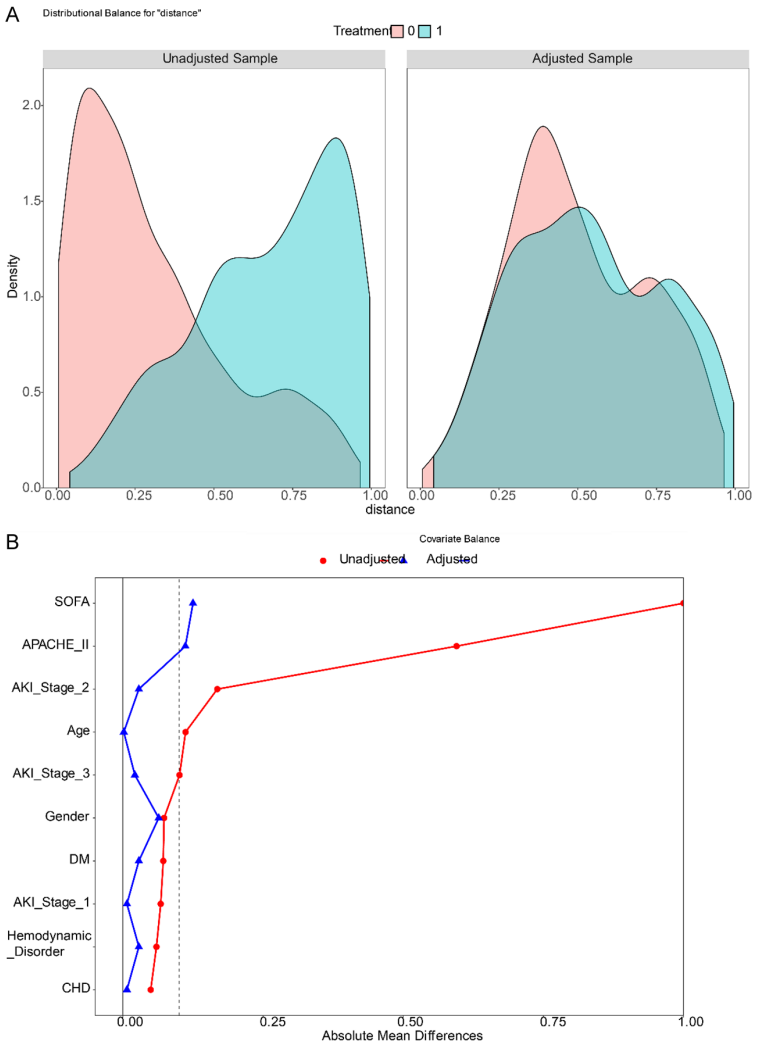


Figure 2. Propensity score matching (PSM) analysis. A: Distribution of propensity scores before and after matching. B: Standardized mean differences of baseline covariates pre- and post-matching.

0.00%, P < 0.001), indicating that while more patients survived, a subset had persistent renal dysfunction.

With respect to safety, no significant differences were observed in infection or major bleeding events between groups (both P > 0.05), supporting the tolerability of CRRT in this population. Notably, the incidence of severe electrolyte disturbances was significantly lower in the CRRT group (14.18% vs. 34.04%, P < 0.001), underscoring CRRT's role in maintaining metabolic stability (**Table 3**). Overall, CRRT was associated with improved renal recovery, better correction of metabolic derangements, and enhanced 90-day survival, without an increased risk of infection or bleeding. These findings support the potential clinical benefit of CRRT in selected ICU patients with solid tumor-associated AKI.

Subgroup analyses

Among the 282 propensity score-matched patients, the AKI remission rate remained significantly higher in the CRRT group (P = 0.012). Stratified subgroup analyses demonstrated that the association between CRRT and AKI remission was consistent across several clinically relevant populations. Specifically, CRRT was significantly associated with improved AKI remission in patients without diabetes (P = 0.007), and in those with hypertension (P = 0.027), coronary artery disease (P = 0.012), tumor lysis syndrome (P = 0.010), hemodynamic instability (P = 0.009), and prior nephrotoxic exposure (P = 0.035). A notable benefit was also observed among patients with sepsis (P = 0.008). However, none of the subgroup interaction terms reached statistical significance (P for interaction = 0.265; **Figure 5**).

Multivariate analysis of factors associated with AKI recovery

To identify independent predictors of AKI remission - defined as a ≥ 25% reduction in serum creatinine and urine output ≥ 0.5 mL/kg/h sustained for 48 hours - a multivariate logistic regression analysis was conducted (**Figure 6A**). CRRT emerged as an independent protective factor for AKI remission (P = 0.005), even after controlling for potential confounders. Conversely, elevated baseline serum creatinine (P = 0.004), the presence of sepsis (P = 0.036), and hemodynamic instability (P = 0.011) were significantly associated with reduced odds of renal recovery. Nephrotoxic exposure (P =

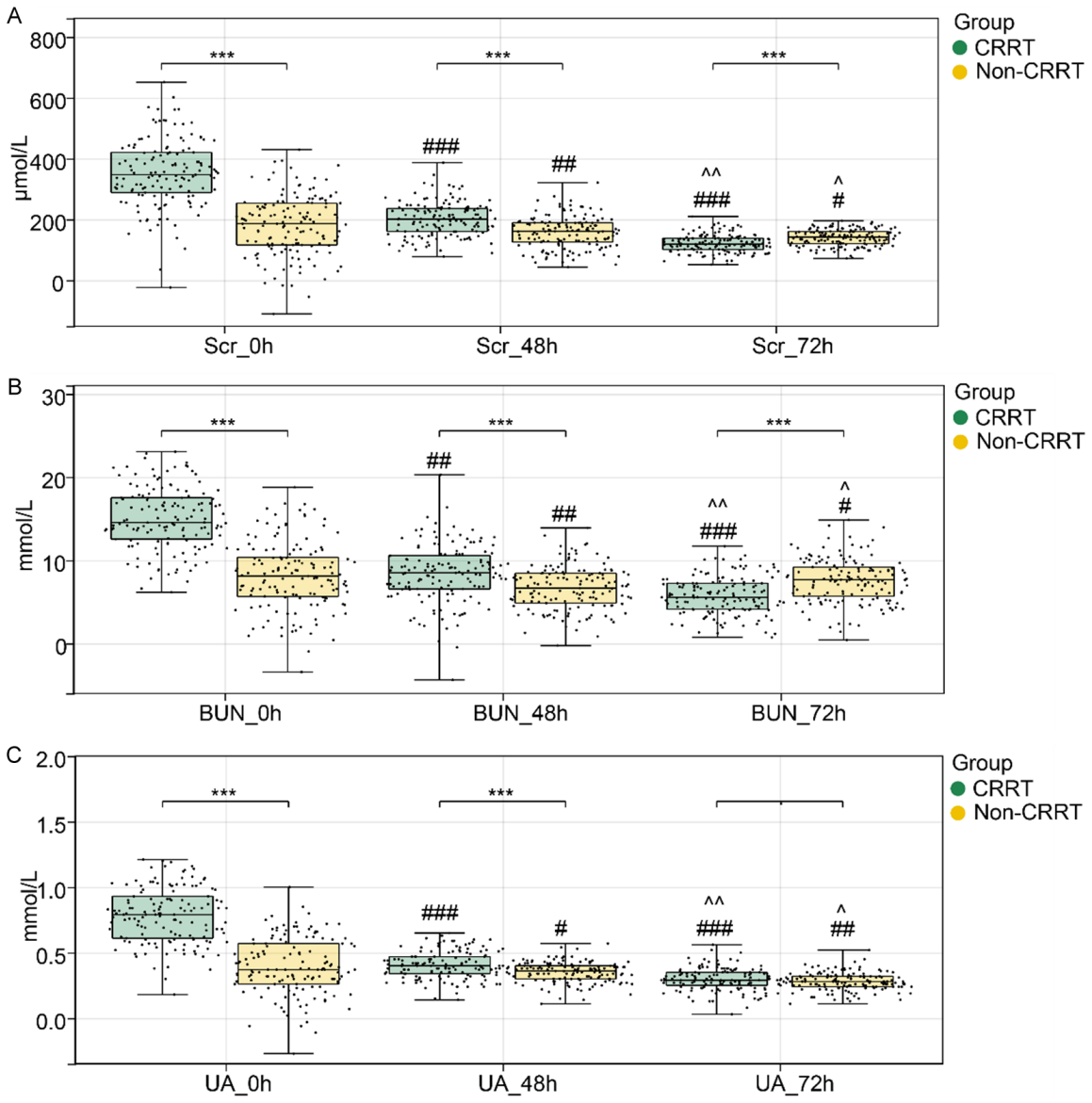


Figure 3. Longitudinal changes in renal function parameters in continuous renal replacement therapy (CRRT) and non-CRRT groups. A: Serum creatinine; B: Blood urea nitrogen (BUN); C: Uric acid levels at 0 h, 48 h, and 72 h. ***P < 0.001. #P < 0.05, ##P < 0.01, ### P < 0.001 indicate significant differences compared to 0 h within each group. ^P < 0.05, ^^P < 0.01 indicate significant differences compared to 48 h within each group.

0.053) and elevated lactate levels (P = 0.073) did not reach statistical significance but showed a trend toward negative association.

The logistic regression model demonstrated strong discriminatory ability, with an AUC of 0.80 (95% CI, 0.74-0.86) in the training cohort and 0.72 (95% CI, 0.60-0.83) in the validation cohort (**Figure 6B**). Calibration was confirmed using the Hosmer-Lemeshow goodness-of-fit test (training: P = 0.468; validation: P = 0.498; **Figure 6C**). Decision curve analysis indicated favorable clinical utility across a range of threshold probabilities (**Figure 6D**), supporting the model's applicability for individualized decision-making.

Machine learning-based prediction of clinical outcomes

To complement traditional analyses, supervised machine learning models were developed using the XGBoost algorithm to predict two key outcomes: 90-day mortality and AKI remis-

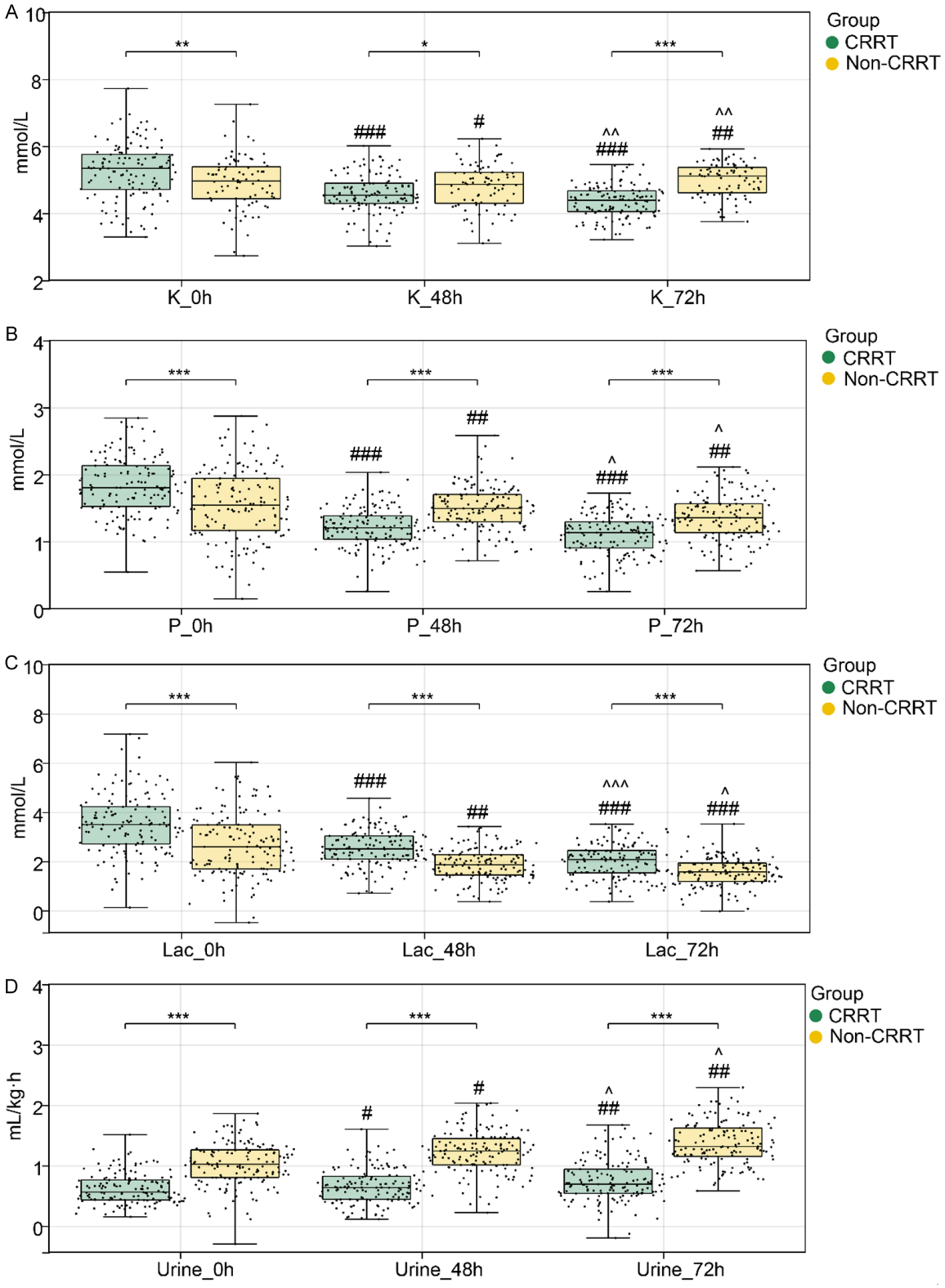


Figure 4. Temporal changes in metabolic parameters and urine output in continuous renal replacement therapy (CRRT) and non-CRRT groups. A: Serum potassium; B: Serum phosphate; C: Serum lactate; D: Urine output at 0 h, 48 h, and 72 h. *P < 0.05, **P < 0.01, ***P < 0.001. #P < 0.05, ##P < 0.01, ###P < 0.001 indicate significant differences compared to 0 h within each group. ^P < 0.05 indicates significant differences compared to 48 h within each group.

Table 2. Comparison of renal and metabolic parameters between groups (CRRT vs. Non-CRRT, n = 141 per group)

Variable	Group × Time Interaction (F)	*P*-value
Serum Creatinine (Scr)	124.3	< 0.001
Blood Urea Nitrogen (BUN)	101.7	< 0.001
Uric Acid (UA)	52.9	< 0.001
K	19.4	< 0.001
P	38.1	< 0.001
Lactate	25.5	< 0.001
Urine Output	6.7	0.003

sion. Model interpretability was enhanced using SHAP (**Figure 7**).

For 90-day mortality prediction, the model achieved strong performance with an AUC of 0.78 (95% CI, 0.71-0.85; **Figure 7A**). SHAP summary plots identified serum lactate concentration, AKI stage, urine output at ICU admission, age, and lactate clearance as the top contributors (**Figure 7B**), highlighting the prognostic importance of metabolic derangement, renal dysfunction, and physiological reserve.

In predicting AKI remission, the model achieved an AUC of 0.75 (95% CI, 0.67-0.82; **Figure 7C**). Key predictors included AKI stage, receipt of CRRT, baseline serum creatinine, urine output, and BUN levels (**Figure 7D**). The concordance between machine learning outputs and established clinical risk factors supports the relevance of XGBoost-based tools for individualized outcome prediction in critically ill patients with solid tumors.

Discussion

This study comprehensively evaluated the impact of CRRT on renal recovery, metabolic homeostasis, and clinical outcomes in ICU patients with solid tumors and AKI, using propensity score matching to minimize baseline confounding. Our findings demonstrate that CRRT significantly improved renal function markers, including Scr, BUN, and UA, as well as key metabolic parameters such as serum potassium, phosphate, and lactate. Moreover, CRRT was associated with a higher 90-day survival rate, without a corresponding increase in infection or bleeding risk. However, CRRT initiation was linked to a prolonged ICU stay. Subgroup analyses revealed consistent therapeutic benefits across a range of comorbid conditions. Both multivariate logistic regression and machine learning models independently identified CRRT as a significant predictor of AKI remission.

As a continuous and hemodynamically stable modality, CRRT offers several advantages over intermittent hemodialysis (IHD), particularly for patients with cardiovascular instability [12]. Its capacity for gradual solute and fluid removal facilitates the maintenance of hemodynamic stability, making it the preferred modality for critically ill and unstable patients [13, 14]. Consistent with KDIGO guidelines, which recommend CRRT as the first-line therapy in hemodynamically unstable patients [13, 15], - and our results support its clinical utility in this setting. In our cohort, patients who received CRRT showed significant improvements in renal and metabolic profiles, reflecting the therapy's ability to achieve continuous solute clearance and precise fluid control. CRRT promotes sustained removal of uremic toxins and effective correction of electrolyte and acid-base imbalances [16-18]. Prior studies have emphasized its role in managing complex electrolyte disturbances, effectively replicating the excretory functions of native kidneys [19]. This mechanistic foundation likely explains the observed reductions in serum creatinine, BUN, potassium, and phosphate, as well as improvements in arterial pH and base excess. In addition, CRRT enhances clearance of lactate and other metabolic byproducts, contributing to systemic metabolic stabilization. Emerging evidence supports its efficacy in lowering serum potassium, phosphate, and lactate levels while maintaining acid-base homeostasis through bicarbonate-buffered dialysate. These effects are essential for cardiovascular stabilization and may help reduce the risk of life-threatening complications such as arrhythmias and hypotension [20].

Despite the physiological advantages of CRRT, overall mortality among critically ill patients with AKI remains high. To date, randomized controlled trials have not consistently demonstrated a short-term survival benefit of CRRT compared with IHD [19]. As noted by Wald et

Table 3. Comparison Key clinical outcomes

Variables	CRRT (n = 141)	Non-CRRT (n = 141)	Statistic	*P*
ICU Stay Days, Mean ± SD	14.45 ± 3.18	9.99 ± 2.23	t = 13.63	< 0.001
Survival 28 d, n (%)	89 (63.12)	57 (40.43)	χ^2 = 2.08	0.149
Survival 90 d, n (%)	67 (47.52)	55 (39.01)	χ^2 = 6.38	0.012
AKI Recovery, n (%)	91 (64.54)	70 (49.65)	χ^2 = 29.86	< 0.001
Dialysis Dep, n (%)	27 (19.15)	0 (0.00)	χ^2 = 29.86	< 0.001
Infection, n (%)	30 (21.28)	34 (24.11)	χ^2 = 0.32	0.570
Bleed, n (%)	17 (12.06)	9 (6.38)	χ^2 = 2.71	0.100
Electrolyte Disorder, n (%)	20 (14.18)	48 (34.04)	χ^2 = 15.19	< 0.001

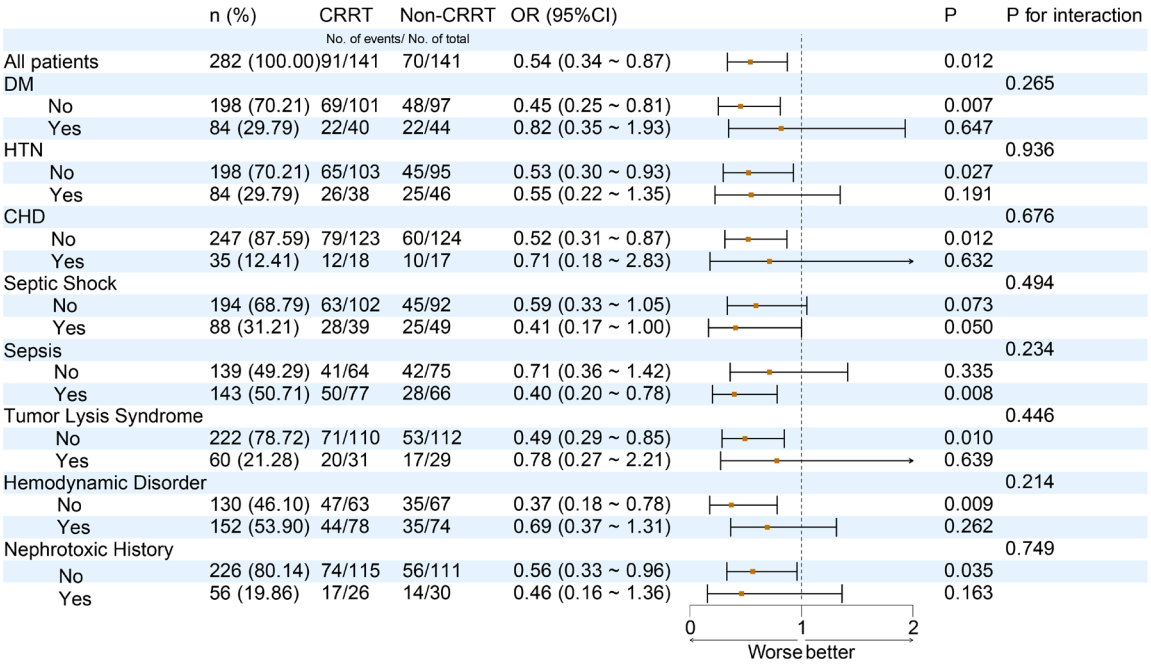


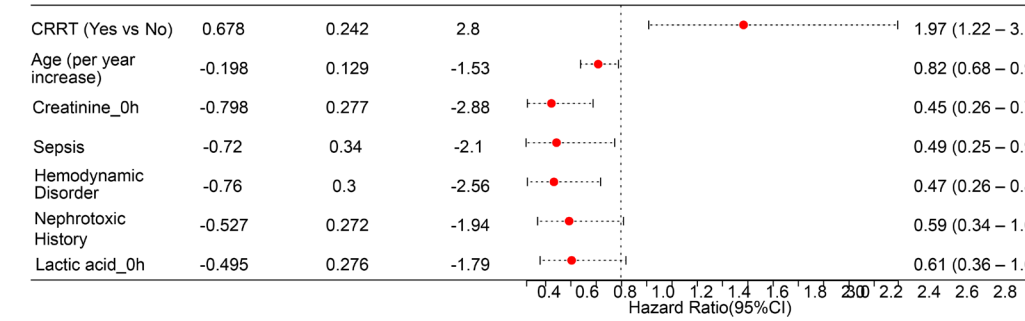
Figure 5. Subgroup analysis of the effect of continuous renal replacement therapy (CRRT) on acute kidney injury (AKI) remission across various comorbidities (after propensity score matching).

al., although CRRT is widely used in the management of severe AKI, most studies have failed to show a significant mortality advantage [21]. Nonetheless, CRRT is hypothesized to exert additional benefits beyond renal support, particularly through immunomodulatory mechanisms. Continuous blood purification techniques may facilitate partial removal of pro-inflammatory cytokines and endotoxins, thereby attenuating systemic inflammation in the setting of sepsis and septic shock [22]. Supporting this hypothesis, a recent meta-analysis found that CRRT using the oXiris hemofilter significantly reduced 28-day mortality, lowered serum lactate levels, and improved SOFA scores in septic patients, although no significant effect on 90-day mortality was observed [22].

In our study, the observed survival advantage in the CRRT group may reflect several contributing factors, including advances in technology, differences in patient selection, and variations in statistical adjustment. Furthermore, among patients with acute respiratory distress syndrome (ARDS) receiving extracorporeal membrane oxygenation (ECMO), the addition of CRRT has been associated with reductions in inflammatory biomarkers such as interleukin-6 (IL-6) and procalcitonin (PCT), mitigating sys-

A

Variables	β	S.E	Z	OR (95% CI)	P
CRRT (Yes vs No)	0.678	0.242	2.8	1.97 (1.22 – 3.19)	0.005
Age (per year increase)	-0.198	0.129	-1.53	0.82 (0.68 – 0.98)	0.125
Creatinine_0h	-0.798	0.277	-2.88	0.45 (0.26 – 0.78)	0.004
Sepsis	-0.72	0.34	-2.1	0.49 (0.25 – 0.95)	0.036
Hemodynamic Disorder	-0.76	0.3	-2.56	0.47 (0.26 – 0.84)	0.011
Nephrotoxic History	-0.527	0.272	-1.94	0.59 (0.34 – 1.02)	0.053
Lactic acid_0h	-0.495	0.276	-1.79	0.61 (0.36 – 1.04)	0.073



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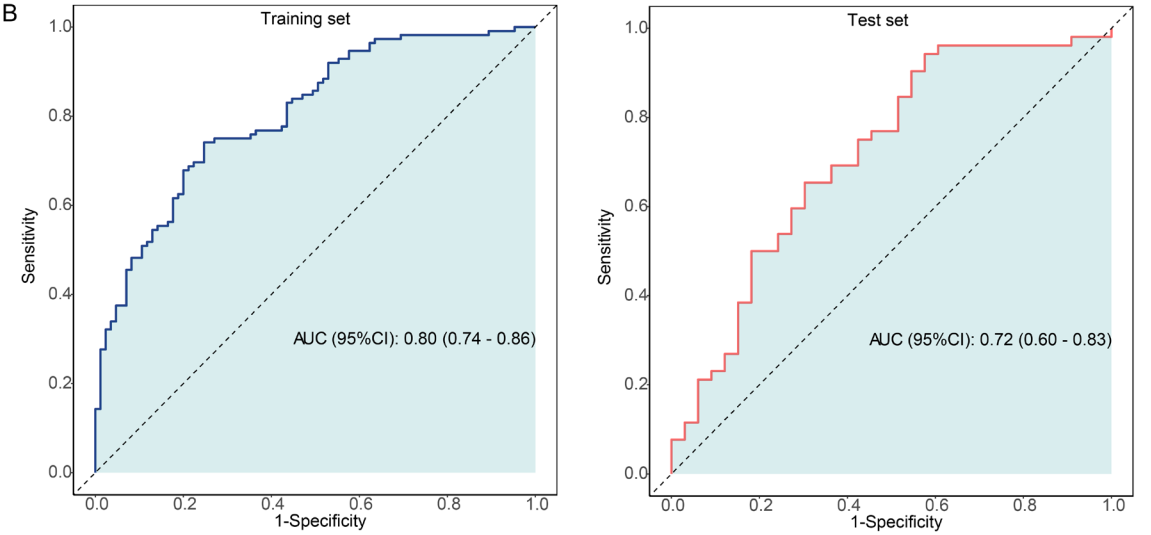

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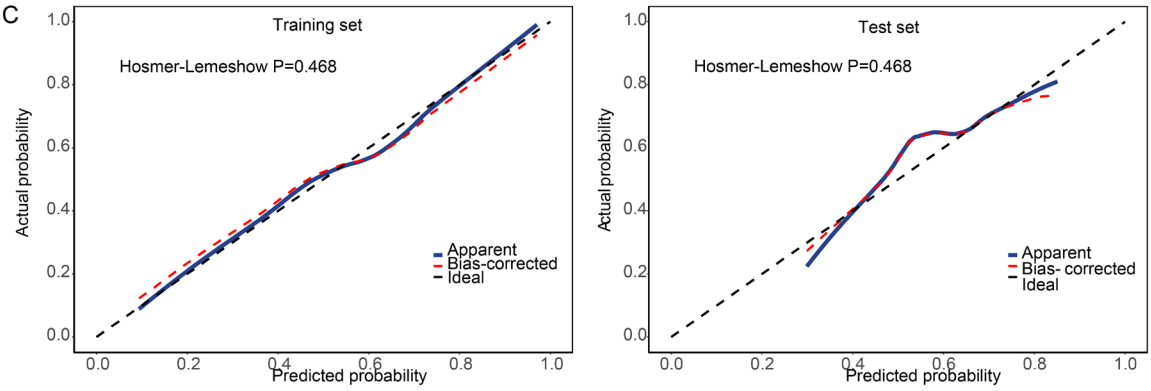

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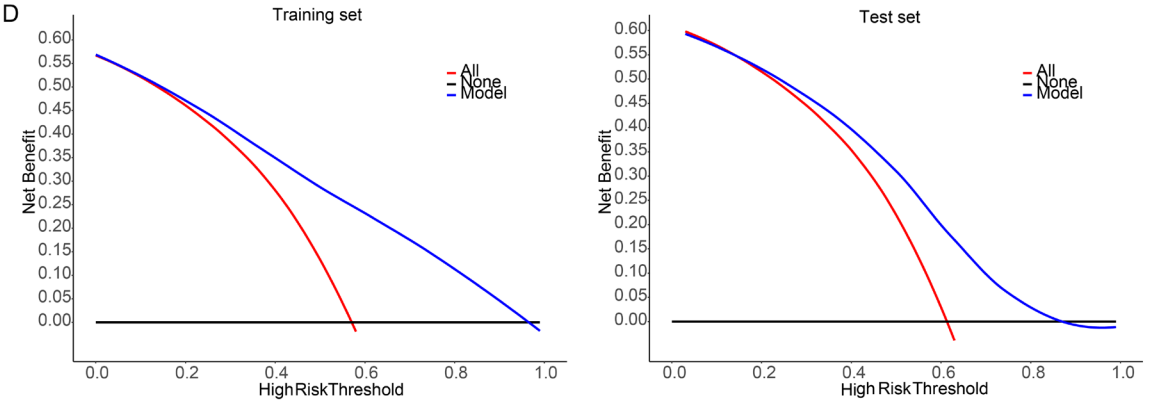

Figure 6. Multivariate logistic regression analysis of factors associated with AKI recovery. A: Forest plot depicting the multivariable model assessing predictors of AKI recovery. B: Receiver operating characteristic (ROC) curve with an area under the curve (AUC) for training and test sets. C: Hosmer-Lemeshow calibration plot, indicating good model fit. D: Decision curve analysis (DCA) illustrating the clinical net benefit of the model across various threshold probabilities.

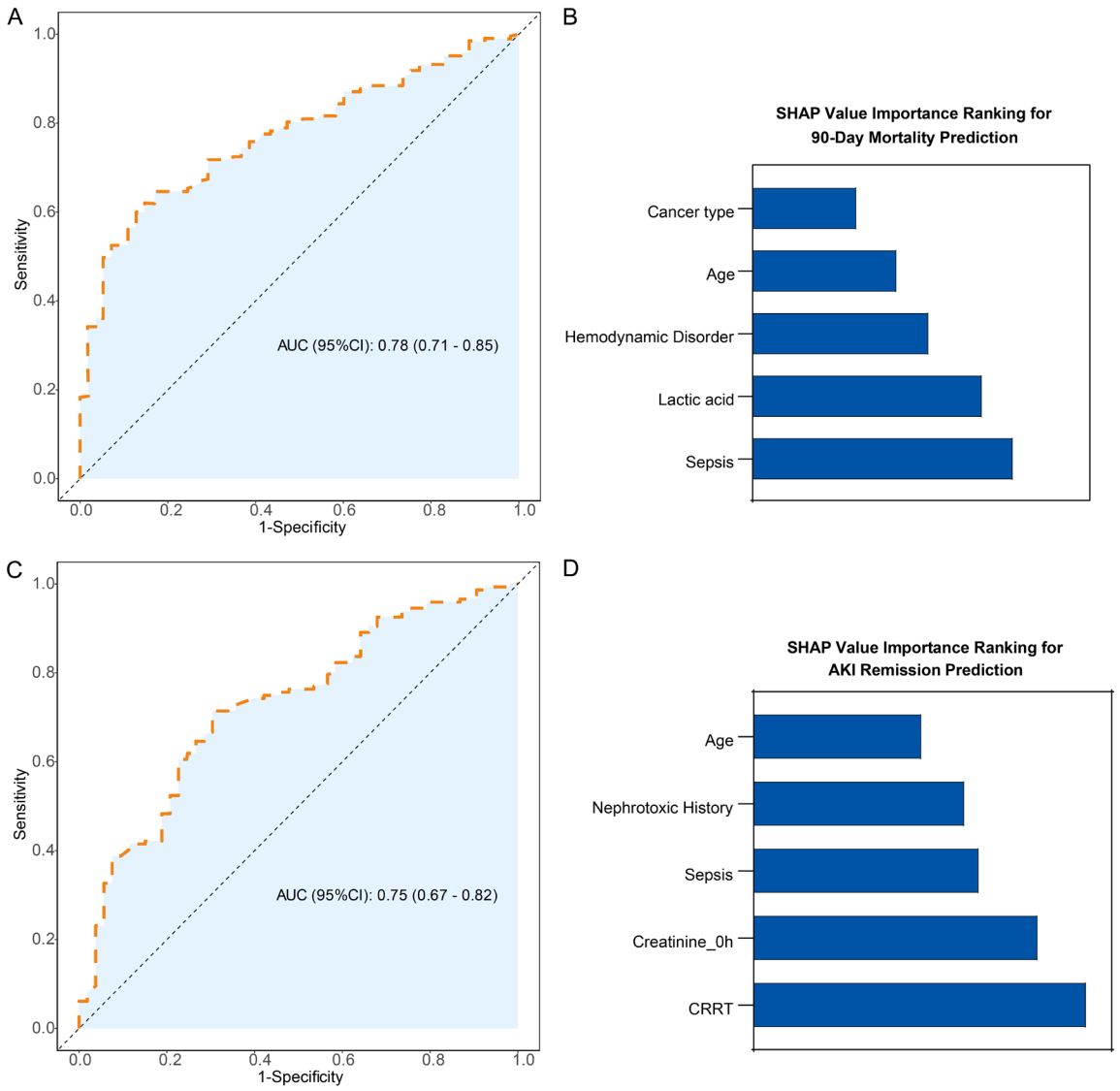

Figure 7. Machine learning model outputs. A: ROC curve for predicting 90-day mortality (AUC = 0.78). B: Shapley Additive explanations (SHAP) summary plot for mortality predictors. C: ROC curve for AKI remission prediction (AUC = 0.75). D: SHAP summary plot for remission predictors.

temic inflammatory response syndrome (SIRS) and decreasing the risk of multiorgan failure.

Beyond immunomodulation, CRRT's ability to sustain the clearance of metabolic by-products may contribute to a more favorable internal milieu, particularly in oncology patients. Preclinical studies have shown that elevated lactate levels promote epithelial-mesenchymal transition (EMT) in tumor cells and suppress immune cell activity. Thus, CRRT-mediated lactate clearance may indirectly enhance antitumor immunity and support host defense.

In our cohort, the CRRT group exhibited a significantly higher rate of complete AKI remis-

sion, consistent with prior clinical evidence. For instance, in patients with left ventricular dysfunction and septic shock, early CRRT initiation - even prior to overt AKI onset - was associated with significantly lower ICU mortality [23], suggesting that timely renal support can improve outcomes. Similarly, Wald et al. reported greater likelihood of renal recovery and dialysis independence among CRRT survivors [21]. Our findings align with these reports, indicating improved renal recovery rates in patients treated with CRRT. Although CRRT may not uniformly reduce overall mortality, it appears to promote renal recovery and enhance survival in selected critically ill populations. These results highlight the life-supporting role of CRRT in the early management of severe AKI and support current KDIGO and international guidelines that recommend CRRT as the preferred modality for renal support in hemodynamically unstable patients [20, 24].

Interestingly, our study revealed a paradoxical finding: the CRRT group had a higher rate of dialysis dependence at follow-up (19.15% vs. 0%). This warrants further investigation. One possible explanation is that a subset of survivors sustained irreversible renal injury - such as acute tubular necrosis or progression to interstitial fibrosis - which may be influenced by the timing, dose, or duration of CRRT. For example, a study on AKI following lung transplantation found that although 64.3% of patients receiving CRRT recovered renal function, 12.5% progressed to end-stage kidney disease [25].

To better understand the long-term renal consequences of CRRT, future studies should include renal biopsy data and incorporate molecular biomarkers of tubular injury, such as neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 (KIM-1). Subgroup analyses in our study revealed consistent benefits of CRRT across high-risk populations, including patients with diabetes, hypertension, and sepsis, all of whom showed significantly higher AKI resolution rates, with no evidence of significant interaction effects. Previous research suggests that in sepsis, CRRT may enhance renal perfusion and mitigate kidney injury by removing endotoxins (e.g., lipopolysaccharide) and inflammatory mediators (e.g., TNF-α, IL-6) [26].

Additionally, the observed benefits in patients with tumor lysis syndrome likely reflect CRRT's capacity to rapidly eliminate nephrotoxic solutes such as uric acid and phosphate, thereby preventing acute uric acid nephropathy and further renal compromise [27].

Our XGBoost-based machine learning model demonstrated strong predictive performance for 90-day clinical outcomes, highlighting the growing role of artificial intelligence in prognostication for critically ill patients with AKI. Recent studies suggest that ensemble learning algorithms such as XGBoost often outperform traditional statistical models in predictive accuracy. For example, one study reported a concordance index (C-index) of 0.8248 for XGBoost in AKI-related mortality prediction, significantly exceeding that of the Cox proportional hazards model [28]. Consistently, our model, which integrated a wide array of clinical and biochemical variables, achieved similarly high discriminative performance. Feature importance analysis identified serum lactate levels, AKI stage, urine output at ICU admission, age, lactate clearance, receipt of CRRT, baseline serum creatinine, and BUN as the most influential predictors of renal recovery [29]. These factors collectively reflect the severity of initial renal injury, systemic inflammatory burden, and the intensity of supportive interventions. Mechanistically, they align with established contributors to AKI pathophysiology, including ischemia-reperfusion injury, inflammation, and impaired perfusion. Notably, SHAP analysis ranked CRRT as the second most important predictor after AKI stage, reinforcing its therapeutic relevance.

XGBoost's strength lies in its ability to model complex, nonlinear relationships and process high-dimensional, heterogeneous data. This capacity enables the identification of latent prognostic patterns often missed by conventional methods, allowing for more accurate and individualized risk stratification. Our findings support the integration of machine learning tools into clinical decision-making frameworks for patients with severe AKI.

Elevated serum lactate is a well-recognized marker of tissue hypoperfusion and cellular hypoxia, indicating systemic and renal micro-

circulatory dysfunction. Persistent hyperlactatemia not only reflects ongoing metabolic stress but also contributes to tubular injury through mitochondrial dysfunction and inflammatory pathways [30]. Multiple studies have linked elevated baseline lactate levels with higher risks of AKI progression and mortality [31]. In this context, early initiation of CRRT may help attenuate lactate accumulation by optimizing fluid balance, improving organ perfusion, and facilitating the clearance of inflammatory mediators. This intervention could potentially disrupt the feedback loop of hypoxia-induced renal injury.

AKI staging, as defined by KDIGO criteria, remains a key marker of parenchymal damage and prognostic trajectory. Severe AKI (Stage 3) is strongly associated with increased mortality, and persistent oliguria is linked to greater RRT requirements, longer ICU stays, and worse outcomes [32]. In our cohort, high SHAP values associated with early-stage AKI emphasize the therapeutic window during which CRRT may be most beneficial in halting irreversible renal injury and promoting recovery.

A major strength of this study was the use of propensity score matching, which effectively balanced baseline characteristics between the CRRT and non-CRRT groups, thereby minimizing treatment allocation bias. Furthermore, the integration of multivariable logistic regression and machine learning approaches enabled a robust assessment of CRRT efficacy and the identification of mechanistically plausible predictors, enhancing both analytical rigor and clinical relevance.

However, several limitations should be acknowledged. First, the study was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. The machine learning models were exploratory and hypothesis-generating in nature, based solely on retrospectively collected data. These models were not externally validated and should not be used for clinical decision-making without further prospective evaluation. Second, the non-randomized design precludes definitive causal inference. While PSM mitigates confounding by observed variables, unmeasured confounders cannot be fully excluded. Third, we did not stratify outcomes based on specific CRRT parameters (e.g., filter type, anticoagulation strategy, ultrafiltration dose, or membrane characteristics), all of which may influence efficacy. Moreover, the timing of CRRT initiation - such as AKI stage or lactate level at onset - was not evaluated, despite its potential impact on outcomes.

Future studies should aim to validate these findings in larger, multicenter cohorts and incorporate randomized controlled designs. Detailed evaluation of CRRT indications, initiation timing, dosing strategies, and personalized parameters will be essential to inform precision renal support strategies in critically ill oncology patients.

In conclusion, this study provides robust evidence that CRRT is a safe and effective strategy for improving renal function and long-term survival in critically ill patients with solid tumors and AKI. Its benefits likely stem from enhanced metabolic regulation and inflammatory control. Early initiation should be prioritized in high-risk subgroups, such as those with elevated lactate, sepsis, or TLS.

Additionally, machine learning algorithms offer valuable support for individualized prognostication and treatment decisions. These tools can help identify patients most likely to benefit from CRRT.

Despite its benefits, the potential risk of long-term dialysis dependence warrants attention, highlighting the importance of renal rehabilitation and structured follow-up. Overall, our findings support the integration of CRRT into ICU care pathways for oncology patients with AKI and may inform refinement of existing clinical guidelines.

Disclosure of conflict of interest

None.

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