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Renal Outcomes After Surgery for Acute Type A Aortic Dissection: Association of Intraoperative Cerebral Near-Infrared Spectroscopy with Acute Kidney Injury

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Abstract

Background: Acute kidney injury (AKI) is common after acute type A aortic dissection (ATAAD) repair. We investigated whether intraoperative cerebral near-infrared spectroscopy (NIRS) was associated with postoperative AKI. Methods: This retrospective single-center study included 288 adults undergoing open repair of acute aortic dissection with hypothermic circulatory arrest between 2015 and 2023, predominantly for ATAAD, including seven non-A non-B dissections. Bilateral cerebral NIRS was recorded at five predefined time points. AKI was defined using modified creatinine/renal replacement therapy-based KDIGO criteria. Multivariable logistic regression included cardiopulmonary bypass (CPB) duration, DeBakey type I, and anatomical renal artery involvement; additional adjustment examined baseline NIRS. Results: AKI occurred in 181 patients (62.8%), including stage 3 in 94 (32.6%). Left-sided NIRS was lower with AKI before CPB (69% vs. 73%, $p = 0.020$) and five minutes before CPB completion (72% vs. 75%, $p = 0.004$). In multivariable analysis ($n = 273$), CPB duration (OR 1.016/minute, 95% CI 1.009–1.022, $p < 0.001$), renal artery involvement (OR 2.041 per additional artery, 95% CI 1.167–3.569, $p = 0.012$), and left-sided NIRS near CPB completion (OR 0.965/%, 95% CI 0.938–0.992, $p = 0.011$) were associated with AKI. The NIRS association attenuated after baseline adjustment (OR 0.976, 95% CI 0.946–1.006, $p = 0.117$). In-hospital mortality was higher with AKI (27.6% vs. 7.5%, $p < 0.001$). Conclusions: AKI affected nearly two thirds of patients undergoing ATAAD repair. Lower left-sided cerebral NIRS near CPB completion was associated with AKI after adjustment for bypass duration and renal artery involvement, but the association attenuated after baseline adjustment. This exploratory finding requires prospective validation.

Keywords: acute type A aortic dissection; acute kidney injury; near-infraredspectroscopy; hypothermic circulatory arrest; bilateral antegrade cerebral perfusion; malperfusion

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1. Introduction

Acute type A aortic dissection (ATAAD) is a life-threatening surgical emergency. While open surgical repair under hypothermic circulatory arrest (HCA) is the standard of care, it carries substantial perioperative mortality [1].

Among these, acute kidney injury (AKI) is a frequent and prognostically decisive complication, with reported incidences ranging from 41% to over 70% [2,3]. Postoperative AKI profoundly increases intensive care unit (ICU) stay, renal replacement therapy (RRT) rates, stroke, and mortality [2,4]. Its multifactorial etiology includes preoperative renal malperfusion, prolonged cardiopulmonary bypass (CPB), and perioperative hemodynamic and inflammatory disturbances [3,5,6].

Cerebral near-infrared spectroscopy (NIRS) is routinely used to monitor cerebral oxygenation during aortic surgery. Associations between tissue oxygen saturation and postoperative AKI have been reported in cardiac surgery, raising the possibility that cerebral NIRS also reflects physiological factors relevant to renal injury [7–10].

Whether cerebral NIRS provides information on postoperative renal risk beyond established clinical and anatomical factors remains unclear, particularly in patients undergoing acute aortic dissection repair.

This study investigated the association between cerebral NIRS measurements obtained at predefined intraoperative time points and postoperative AKI after acute aortic dissection repair with HCA.

2. Methods

Study design and data collection: This retrospective single-center study screened 307 adults who underwent open surgical repair of acute aortic dissection with HCA at a tertiary university hospital between January 2015 and December 2023. Nineteen patients who died within 24 h after surgery were excluded; this group included both patients receiving preoperative renal replacement therapy. The final analysis cohort therefore comprised 288 patients (Figure 1). The cohort predominantly comprised patients with ATAAD but also included seven patients (2.4%) with non-A non-B dissection involving the aortic arch without ascending aortic involvement ("other", n = 5) and Type B with retrograde dissection into the aortic arch (n = 2), which were treated open surgically. Exclusion criteria were death within 24 h after surgery and preoperative dialysis-dependent end-stage renal disease. Patients were grouped according to the occurrence of postoperative AKI. Clinical, anatomical, operative, and outcome data were extracted from institutional electronic medical records and the perfusion database. The institutional ethics committee approved this study (approval number 25-4250-104) and waived the requirement for informed consent.

All 175 patients included in the previously published institutional NIRS–stroke study were also included in the present expanded cohort of 288 patients [9]. The present analysis addressed postoperative renal outcomes in this larger population.

Surgical technique and cerebral protection: Emergency repair was performed using cardiopulmonary bypass (CPB). The institutional standard was bilateral selective antegrade cerebral perfusion. Arterial cannulation was performed through an 8 mm Dacron side graft anastomosed to the right subclavian artery, providing perfusion of the right carotid territory. After opening and inspecting the aortic arch, a balloon-tipped catheter was inserted into the left common carotid artery to establish left-sided perfusion. The target mean cerebral perfusion pressure was 40–60 mmHg. Cannulation and perfusion were adapted to individual anatomy, particularly when the right subclavian or left common carotid artery was dissected; the configuration was therefore not identical in every patient.

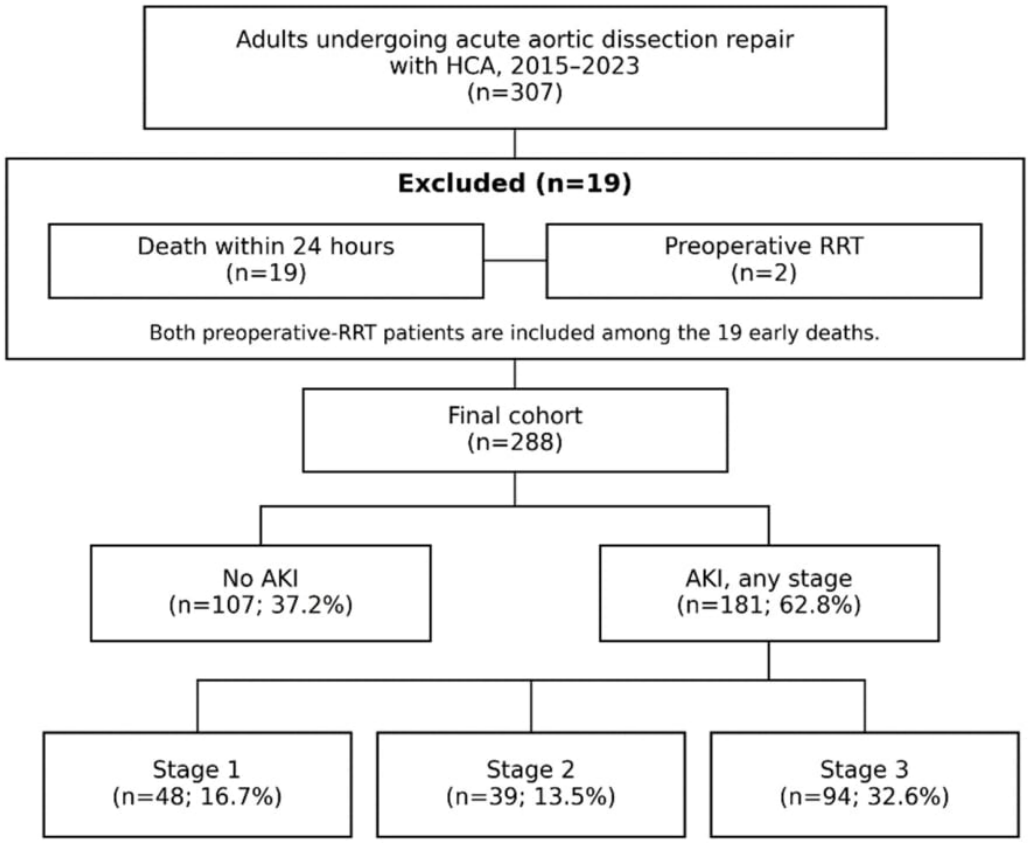


Figure 1. Flow chart of patient selection. Abbreviations: AKI, acute kidney injury; ATAAD, acute type A aortic dissection; HCA, hypothermic circulatory arrest; Pat., patients; RRT, renal replacement therapy.

The intended rectal temperature during HCA was 20–28 °C. Nasopharyngeal temperature at the arrest nadir was 20.0 °C (IQR 20.0–22.0). Acid–base management during hypothermia followed an alpha-stat strategy, using the CDI 500 Blood Parameter Monitoring System (Terumo Cardiovascular Systems, Ann Arbor, Michigan, USA), with a target base excess of −3 mmol/L. Blood gas results during hypothermia were not recorded in the study dataset. Standard institutional protocols governed transfusion thresholds and vasopressor/inotrope use.

NIRS Monitoring and Analysis:

Bilateral frontal cerebral oxygen saturation was monitored continuously using INVOS 5100C or 7100 devices (Medtronic, Dublin, Ireland). The INVOS algorithm estimates a venous-weighted regional oxygen saturation and is intended as a trend monitor rather than as an absolute measure of cerebral or renal blood flow. The displayed values were visible to the clinical team and could prompt adjustments in perfusion management. This study analyzed documented values at five predefined measurement points: MP1, 5 min before CPB initiation; MP2, 1 min before HCA; MP3, the documented minimum during HCA; MP4, 1 min after HCA; and MP5, 5 min before CPB completion. MP5 was therefore obtained while the patient was still receiving CPB support.

Derived measurements comprised relative changes from MP1 to MP3 and from MP3 to MP5, the absolute change from MP1 to MP5, and descriptive thresholds for low saturation or relative changes exceeding 20%. NIRS analyses used complete recorded measurement series. For each hemisphere, the mean, standard deviation, maximum, and minimum used in derived calculations were obtained from the five documented measurement points.

The relative range was calculated as (maximum − minimum)/mean. The NIRS analyses were exploratory.

AKI definition and renal outcomes: The primary outcome was postoperative AKI, defined using modified creatinine/RRT-based KDIGO criteria without urine output criteria. Preoperative serum creatinine served as baseline. AKI was identified by an increase of at least 0.3 mg/dL within 48 h, an increase to at least 1.5 times baseline within 7 days, or initiation of RRT. Stage 1 comprised a creatinine increase of at least 0.3 mg/dL or to 1.5–1.9 times baseline; stage 2, an increase to 2.0–2.9 times baseline; and stage 3, an increase to at least 3.0 times baseline, an acute increase to at least 4.0 mg/dL, or initiation of RRT. Patients were assigned the highest stage reached during the assessment window.

Secondary outcomes included acute postoperative RRT, dialysis dependence at discharge, in-hospital mortality, perioperative stroke, and intensive care unit and hospital length of stay.

Survival follow-up: Survival analyses used the recorded survival times and last available follow-up dates in the institutional dataset. Patients without a recorded death were censored at their last documented follow-up. Vital status after transfer or discharge was not systematically ascertained, and follow-up did not terminate at a uniform administrative closing date. Survival beyond discharge was therefore analyzed exploratorily, with follow-up duration and numbers at risk reported alongside the Kaplan–Meier estimates.

Statistical analysis: Continuous variables are presented as medians with interquartile ranges and categorical variables as counts and percentages. Comparisons used Mann–Whitney U, Fisher's exact, or chi-square tests, as appropriate. Multivariable logistic regression included CPB duration, left-sided MP5 NIRS, DeBakey type I, and renal artery involvement (0–2 arteries). Sensitivity analyses examined baseline adjustment, right-sided MP5, bilateral mean MP5 with and without corresponding baseline adjustment, and left-sided MP5 − MP1 change. Adjusted odds ratios are reported with 95% confidence intervals and *p*-values. Exploratory survival analyses used Kaplan–Meier estimates and log-rank tests. Tests were two-sided, with $p < 0.05$ indicating statistical significance; no multiplicity adjustment was applied.

3. Results

Study population and preoperative characteristics

The analysis included 288 patients (Figure 1). Median age was 61 years (IQR 53–70), and 35.4% were female (Table 1).

Table 1. Demographic data and preoperative status.

Cohort 2015–2023	Total	AKI Post-Op	No AKI Post-Op	*p*-Value	Rating
No. of Patients	288	181	107	-	-
Age (years)	61 (53; 70)	61 (53; 69)	62 (55; 72)	0.347	-
Female	102 (35.4%)	62 (34.3%)	40 (37.4%)	0.612	-
BMI (kg/m^2)	27.1 (24.3; 30.5)	27.7 (24.7; 31.2)	26.0 (24.0; 29.0)	0.008	***
DeBakey I	195 (67.7%)	135 (74.6%)	60 (56.1%)	0.002	***
DeBakey II	86 (29.9%)	42 (23.2%)	44 (41.1%)	0.002	***
DeBakey III	2 (0.7%)	2 (1.1%)	0 (0%)	0.531	-
DeBakey other	5 (1.7%)	2 (1.1%)	3 (2.8%)	0.364	-

Table 1. *Cont.*

Cohort 2015–2023	Total	AKI Post-Op	No AKI Post-Op	*p*-Value	Rating
GERAADA score (%)	21.2 (12.6; 29.7)	24.3 (15.1; 30.7)	16.1 (10.5–24.5)	<0.001	***
CPR, prae proc.	15 (5.2%)	11 (6.1%)	4 (3.7%)	0.584	-
Mech. ventilation, pre-op	16 (5.6%)	10 (5.5%)	6 (5.6%)	0.999	-
FND, pre-op	54 (18.8%)	32 (17.7%)	22 (20.6%)	0.537	-
S/P cardiac surgery	11 (3.8%)	10 (5.5%)	1 (0.9%)	0.059	*
Vessel dissection status					
coronary	16 (5.6%)	15 (8.3%)	1 (0.9%)	0.007	***
innominate	98 (34.0%)	68 (37.6%)	30 (28.0%)	0.122	-
RCCA	78 (27.1%)	57 (31.5%)	21 (19.6%)	0.029	**
LCCA	68 (23.6%)	48 (26.5%)	20 (18.7%)	0.152	-
RSA	48 (16.7%)	36 (19.9%)	12 (11.2%)	0.071	*
LSA	52 (18.1%)	36 (19.9%)	16 (15.0%)	0.343	-
spinal	5 (1.7%)	5 (2.8%)	0 (0%)	0.161	-
celiac	75 (26.0%)	56 (30.9%)	19 (17.8%)	0.018	**
SMA	48 (16.7%)	35 (19.3%)	13 (12.2%)	0.141	-
RRA	42 (14.6%)	35 (19.3%)	7 (6.5%)	0.003	***
LRA	94 (32.6%)	71 (39.2%)	23 (21.5%)	0.002	***
leg right	33 (11.5%)	27 (14.9%)	6 (5.6%)	0.021	**
leg left	51 (17.8%)	37 (20.4%)	14 (13.1%)	0.150	-
Number of renal arteries involved					
Neither	172 (59.7%)	93 (51.4%)	79 (73.8%)	<0.001	***
Unilateral	96 (33.3%)	70 (38.7%)	26 (24.3%)	0.014	**
Bilateral	20 (6.9%)	18 (9.9%)	2 (1.9%)	0.008	***
Laboratory findings					
Creatinine (mg/dL)	1.0 (0.8; 1.2)	1.0 (0.8; 1.3)	1.0 (0.8; 1.2)	0.548	-
Hemoglobin (g/dL)	12.4 (11.1; 13.4)	12.4 (11.1; 13.7)	12.5 (11.1; 13.4)	0.597	-
Leucocytes ($\times 10^3/\mu L$)	11.7 (9.3; 14.5)	12.4 (9.7; 15.4)	10.8 (9.0; 13.4)	0.006	***
Platelets ($\times 10^3/\mu L$)	182 (152; 216)	181 (149; 218)	184 (158; 215)	0.712	-
ACT (s)	148 (137; 160)	149 (139; 163)	144 (134; 155)	0.027	**
Lactate (mg/dL)	10 (7; 16)	11 (7; 19)	9 (7; 15)	0.061	*
Troponin (ng/L)	7 (0.1; 20)	9 (0.1; 26)	4 (0.1; 17)	0.265	-
CK (U/L)	101 (68; 165)	107 (70; 172)	91 (62; 162)	0.122	-
CK-MB (ng/mL)	2 (1; 5)	2 (1; 5)	2 (1; 4)	0.752	-
Bilirubin (mg/dL)	0.6 (0.4; 0.8)	0.6 (0.4; 0.8)	0.6 (0.4; 0.9)	0.767	-

Data presented as [n (%)] or [median (Q_1; Q_3)]. *** $p < 0.01$/ ** $p < 0.05$/ * $p < 0.1$. The seven patients with DeBakey III and DeBakey other can be classified as non-A non-B dissection and were included in the analysis. Abbreviations: ACT, activated clotting time; BMI, body mass index; CK(-MB), creatine kinase (MB isoenzyme); CPR, cardiopulmonary resuscitation; FND, focal neurological disorder; LCCA, left common carotid artery; LRA, left renal artery; LSA, left subclavian artery; RCCA, right common carotid artery; RRA, right renal artery; RSA, right subclavian artery; SMA, superior mesenteric artery.

AKI occured in 181 patients (62.8%). Patients with AKI had higher BMI (27.7 vs. 26.0 kg/m^2, $p = 0.008$), GERAADA scores (24.3% vs. 16.1%, $p < 0.001$), and leukocyte counts (12.4 vs. 10.8 $\times 10^3/\mu L$, $p = 0.006$). DeBakey type I was more frequent (74.6% vs. 56.1%, $p = 0.002$); age, sex, and baseline creatinine did not differ significantly (Table 1). AKI stage 1 occured in 48 (16.7%), stage 2 in 39 (13.5%), and stage 3 in 94 (32.6%) patients (Figure 1).

Right and left renal artery involvement was more frequent with AKI (19.3% vs. 6.5%, $p = 0.003$; 39.2% vs. 21.5%, $p = 0.002$). AKI incidence increased from 54.1% (93/172) with neither artery involved to 72.9% (70/96) with unilateral and 90.0% (18/20) with bilateral involvement (overall distribution $p < 0.001$). Coronary, right common carotid, celiac, and right lower limb arterial involvement were also more frequent with AKI (Table 1).

Operative characteristics

Concomitant vascular procedures were more frequent with AKI (25.4% vs. 14.0%, $p = 0.025$). Total arch replacement, AVR, CABG, and descending stent/FET use did not differ significantly (Table 2).

Table 2. Intraoperative data and NIRS analysis.

	Total	AKI Post-Op	No AKI Post-Op	*p*-Value	Rating
Operative extent					
Ascending replacement without arch/desc. stent	73 (25.3%)	46 (25.4%)	27 (25.2%)	0.999	-
Ascending replacement with hemiarch	185 (64.2%)	112 (61.9%)	73 (68.2%)	0.310	-
Ascending replacement with total arch	30 (10.4%)	23 (12.7%)	7 (6.5%)	0.113	-
Any descending stent/FET	12 (4.2%)	6 (3.3%)	6 (5.6%)	0.372	-
w/vascular procedure	61 (21.1%)	46 (25.4%)	15 (14.0%)	0.025	**
w/aortic valve replacement	40 (13.9%)	26 (14.4%)	14 (13.1%)	0.861	-
w/CABG	9 (3.1%)	6 (3.3%)	3 (2.8%)	0.999	-
Cardiopulmonary bypass					
CPB time (min)	183 (155; 227)	198 (168; 242)	168 (144; 195)	<0.001	***
Cross clamp time (min)	97 (79; 126)	103 (82; 134)	90 (75; 105)	<0.001	***
Duration of HCA (min)	40 (31; 57)	43 (32; 60)	37 (28; 49)	<0.001	***
Reperfusion time (min)	68 (57; 85)	77 (62; 89)	63 (49; 75)	<0.001	***
Bicarbonate admin. (mL)	200 (120; 250)	200 (140; 300)	150 (100; 200)	<0.001	***
Diuresis (mL)	250 (150; 500)	200 (120; 500)	300 (200; 550)	0.032	**
Hemofiltration (mL)	2500 (2000; 4500)	3000 (2000; 5000)	2500 (2000; 3900)	.043	**
Cell saver volume (mL)	500 (250; 1000)	500 (250; 1000)	250 (250: 750)	0.053	*
Intra-op transfusions (IU)					
Units of pRBC	2 (1;4)	2 (1; 4)	2 (1; 3)	0.007	***
Units of PC	4 (3;5)	4 (3; 6)	3.5 (2; 4)	0.080	*
Units of FFP	6 (5; 6)	6 (6; 7)	6 (4; 6)	<0.001	***

Table 2. *Cont.*

	Total	AKI Post-Op	No AKI Post-Op	*p*-Value	Rating
Intra-op laboratory					
Hemoglobin min (g/dL)	6.7 (6; 7.4)	6.7 (6; 7.4)	6.7 (6.2; 7.4)	0.389	-
Lactate max (mg/dL)	52 (38; 71)	56 (43; 80)	44 (31; 59)	0.001	***
ACT min (s)	419 (381; 480)	415 (376; 473)	433 (396; 489)	0.027	**
ACT max (s)	772 (584; 999)	828 (597; 999)	728 (567; 999)	0.081	*
Temperature (°C)					
Lowest rectal temp. (°C)	26.5 (24.5; 27.7)	26.4 (24.6; 27.7)	26.6 (24.5; 27.6)	0.611	-
MP1–rectal, pre CPB	36.2 (35.5; 36.8)	36.2 (35.5; 36.8)	36.2 (35.5; 36.7)	0.780	-
MP2–rectal, pre HCA	27.5 (25.7; 28.9)	27.6 (25.7; 29.2)	27.4 (25.7; 28.5)	0.303	-
MP3–head, min HCA	20.0 (20.0; 22.0)	20.0 (20.0; 22.0)	20.0 (20.0; 22.0)	0.457	-
MP4–rectal, post HCA	27.0 (25.6; 28.3)	27.0 (25.7; 28.4)	27.1 (25.2; 28.1)	0.545	-
MP5–rectal, end of CPB	36.1 (35.0; 36.7)	36.1 (35.2; 36.7)	35.9 (35.0; 36.6)	0.164	-
NIRS (Left/Right) at					
MP1–pre CPB	70 (61; 78)	69 (60;77)	73 (62; 79)	0.020	**
	70 (60; 77)	70 (59.77)	70 (61; 77)	0.647	-
MP2–pre HCA	67 (61; 75)	67 (60; 74)	69 (61; 76)	0.187	-
	67 (61; 74)	67 (60; 74)	66 (61; 74)	0.897	-
MP3–min at HCA	50 (41; 61)	50 (41; 59)	52 (43; 65)	0.093	*
	56 (41; 67)	55 (41; 67)	56 (42; 69)	0.435	-
MP4–post HCA	64 (55; 73)	64 (57; 72)	65 (55; 73)	0.645	-
	66 (58; 72)	66 (59; 73)	64 (57:72)	0.222	-
MP5–5 min before CPB completion	73 (67; 81)	72 (65; 79)	75 (69; 84)	0.004	***
	73 (66;79)	72 (66; 79)	75 (67; 81)	0.188	-
NIRS analysis					
Relative range of NIRS values (L/R)	0.41 (0.29; 0.60)	0.42 (0.30; 0.62)	0.38 (0.26; 0.57)	0.097	*
	0.38 (0.23; 0.57)	0.38 (0.23; 0.60)	0.38 (0.22; 0.55)	0.575	-
SD across MP1–MP5	10.7 (7.9; 14.1)	11.0 (7.9; 14.4)	10.3 (7.8; 13.6)	0.244	-
NIRS MP3 < 50%	155 (53.8%)	102 (56.4%)	53 (49.5%)	0.273	-
NIRS drop MP1–MP3 (%, L/R)	−27 (−40; −12)	−27 (−40; −12)	−26 (−40; −11)	0.858	-
	−17 (−39; −1.4)	−17 (−40; −1.6)	−16 (−37; 0)	0.627	-
NIRS drop MP1–MP3 > 20%	182 (63.2%)	117 (64.6%)	65 (60.7%)	0.529	-
NIRS rise MP3–MP5 (%, L/R)	40 (18; 78)	38 (18; 80)	42 (17; 74)	0.725	-
	33 (10; 72)	33 (10; 77)	35 (11; 60)	0.831	-
NIRS rise MP3–MP5 > 20%	222 (77.1%)	142 (78.5%)	80 (74.8%)	0.655	-
NIRS drop and rise > 20%	168 (58.3%)	107 (59.1%)	61 (57.0%)	0.805	-

Data presented as [n (%)] or [median (Q_1; Q_3)]. *** $p < 0.01$/ ** $p < 0.05$/ * $p < 0.1$. Abbreviations: ACT, activated clotting time; CABG, coronary artery bypass grafting; CPB, cardiopulmonary bypass; desc., descending; FET, frozen elephant trunk; FFP, fresh frozen plasma; min., minimum; HCA, hypothermic circulatory arrest; MP, measure point; NIRS, near-infrared spectroscopy; PC, platelet concentrate; pRBC, packed red blood cells; SD, standard deviation; w/, with.

Patients with AKI had longer CPB (198 vs. 168 min), cross-clamp (103 vs. 90 min), HCA (43 vs. 37 min), and reperfusion durations (77 vs. 63 min; all $p < 0.001$). Peak lactate was higher (56 vs. 44 mg/dL, $p < 0.001$), as were bicarbonate administration and hemofiltration volumes; urine output during CPB was lower. pRBC and FFP transfusion distributions were higher ($p = 0.007$ and $p < 0.001$), despite identical group medians of 2 and 6 units, respectively. Platelet transfusion did not differ significantly ($p = 0.080$). Median lowest rectal temperature was 26.5 °C (IQR 24.5–27.7); 244 patients (84.7%) reached 20–28 °C. Nasopharyngeal temperature at MP3 was 20.0 °C (IQR 20.0–22.0). Lowest rectal temperature did not differ by AKI status ($p = 0.611$).

Cerebral NIRS measurements

Left-sided NIRS was lower with AKI at MP1 (69% vs. 73%, $p = 0.020$) and MP5 (72% vs. 75%, $p = 0.004$), with overlapping MP5 distributions (IQR 65–79% vs. 69–84%). Other left-sided and all right-sided measurements did not differ significantly (Table 2).

Across no AKI, AKI stages 1–2, and stage 3, left-sided NIRS differed at MP1 ($p = 0.006$), MP3 ($p = 0.003$), and MP5 ($p = 0.014$); right-sided measurements did not (Figure 2). Derived NIRS descriptors and desaturation/recovery thresholds were not significantly associated with AKI (Table 2).

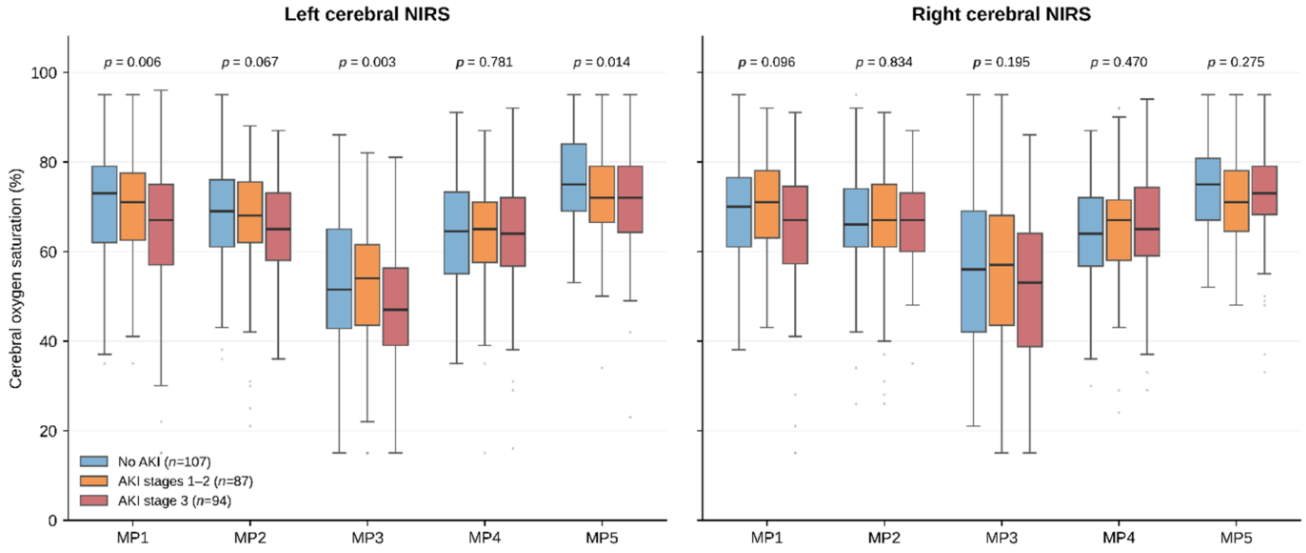


Figure 2. Bilateral cerebral NIRS at MP1–MP5 by postoperative AKI severity. Boxes show medians and interquartile ranges; whiskers extend to 1.5 × IQR, with outliers plotted individually. *p*-values: Kruskal–Wallis tests. Abbreviations: AKI, acute kidney injury; MP, measure point; NIRS, near-infrared spectroscopy.

Postoperative outcomes

Acute postoperative RRT was required in 84 patients (29.2%), all of whom had stage 3 AKI. Of these, 33 died in hospital, 30 survived after temporary RRT, and 21 were recorded as requiring RRT at discharge (Table 3).

Patients with AKI had longer ICU stays (10 vs. 5 days) and hospital stays (21 vs. 13 days), more perioperative stroke (39.8% vs. 18.7%), and higher in-hospital mortality (27.6% vs. 7.5%; all $p < 0.001$). Direct discharge home was less frequent with AKI (13.8% vs. 38.3%, $p < 0.001$) (Table 4).

Table 3. Postoperative renal outcomes.

No AKI	107 (37.2%)
AKI, any stage	181 (62.8%)
AKI stage 1	48 (16.7%)
AKI stage 2	39 (13.5%)
AKI stage 3	94 (32.6%)
Postoperative RRT -temporary RRT -discharge with RRT -in-hospital death with RRT	84 (29.2%) 30 (10.4%) 21 (7.3%) 33 (11.5%)
In-hospital death with AKI	50 (27.6%)

Data presented as [n (%)]. Abbreviations: AKI, acute kidney injury; RRT, renal replacement therapy.

Table 4. Postoperative course: in-patient stay and discharge.

	Total	AKI Post-Op	No AKI Post-Op	*p*-Value	Rating
ICU stay (days)	8 (5; 15)	10 (6; 20)	5 (3; 9)	<0.001	***
Hospital stay (days)	16 (11; 25)	21 (12; 28)	13 (11; 18)	0.001	***
Perioperative stroke	92 (31.9%)	72 (39.8%)	20 (18.7%)	<0.001	***
In-hospital mortality	58 (20.1%)	50 (27.6%)	8 (7.5%)	<0.001	***
Discharge home	66 (22.9%)	25 (13.8%)	41 (38.3%)	<0.001	***
Transfer to external hospital	130 (45.1%)	81 (44.8%)	49 (45.8%)	0.999	-
Transfer to rehabilitation clinic	30 (10.4%)	22 (12.2%)	8 (7.5%)	0.237	-
Discharge disposition missing	4 (1.4%)	3 (1.7%)	1 (0.9%)	-	-

Data presented as [n (%)] or [median (Q_1; Q_3)]. *** $p < 0.01$. Abbreviations: AKI, acute kidney injury; ICU, intensive care unit.

Associations with postoperative AKI

Univariable logistic regression identified associations with BMI, DeBakey type I, GERAADA score, leukocyte count, preoperative lactate, coronary and renal artery involvement, CPB duration, and HCA duration. Higher left-sided NIRS at MP1 and MP5 was associated with lower odds of AKI (Table 5).

In the principal model (n = 273; 172 AKI events), CPB duration (adjusted OR 1.016/min, 95% CI 1.009–1.022, $p < 0.001$), renal artery burden (adjusted OR 2.041/additional artery, 95% CI 1.167–3.569, $p = 0.012$), and left-sided MP5 NIRS (adjusted OR 0.965/%, 95% CI 0.938–0.992, $p = 0.011$) were associated with AKI. DeBakey type I was not independently associated (adjusted OR 1.561, 95% CI 0.805–3.029, $p = 0.188$) (Table 6).

Table 5. Univariate analysis: results for postoperative AKI.

Variable	OR (95% CI)	*p*-Value	Rating
BMI, per kg/m^2	1.088 (1.031–1.147)	0.002	***
DeBakey I	2.299 (1.384–3.819)	0.001	***
GERAADA score, per %	1.043 (1.020–1.066)	<0.001	***
Leucocytes, per $\times 10^3/\mu L$	1.092 (1.028–1.160)	0.004	***
Lactate (pre-op), per mg/dL	1.03 (1.007–1.054)	0.011	**
Coronary artery involvement	9.578 (1.247–73.582)	0.030	**
RRA involvement	3.425 (1.463–8.016)	0.005	***
LRA involvement	2.357 (1.361–4.083)	0.002	***
Number of renal arteries involved, per additional artery	2.445 (1.560–3.833)	<0.001	***
CPB time, per minute	1.016 (1.010–1.023)	<0.001	***
HCA duration, per minute	1.024 (1.011–1.037)	<0.001	***
Left NIRS at MP1, per %	0.977 (0.958–0.996)	0.016	**
Left NIRS at MP5, per %	0.963 (0.940–0.987)	0.002	***

Variables with $p < 0.05$ are shown. ORs are presented with 95% CIs from separate univariable logistic regressions; *p*-values are two-sided Wald tests. Continuous variable ORs refer to one-unit increases. Renal artery involvement was coded 0–2.*** $p < 0.01$ / ** $p < 0.05$. Abbreviations: AKI, acute kidney injury; BMI, body mass index; CI, confidence interval; CPB, cardiopulmonary bypass; HCA, hypothermic circulatory arrest; GERAADA, German Registry for Acute Aortic Dissection Type A; LRA, left renal artery; MP, measure point; NIRS, near-infrared spectroscopy; OR, odds ratio; RRA, right renal artery.

Baseline adjustment attenuated the MP5 association (adjusted OR 0.976, 95% CI 0.946–1.006, $p = 0.117$); CPB duration and renal artery involvement remained associated with AKI (Table 6).

Additional sensitivity analyses showed no association for right-sided MP5 NIRS or the MP1–MP5 change; the association for bilateral mean MP5 NIRS attenuated after baseline adjustment.

Exploratory survival analysis

Recorded follow-up was a median of 193 days (IQR 28–506; maximum 2817 days), with 83 recorded deaths and 205 censored observations. Kaplan–Meier survival differed between patients with and without AKI (log-rank $p < 0.001$; Figure 3). Compared with no AKI, survival differed for stage 2 ($p = 0.008$) and stage 3 AKI ($p < 0.001$), but not for stage 1 ($p = 0.286$; Figure 4). Numbers at risk were 49 at 2 years and 22 at 3 years. The Bavarian general population curve was included as a descriptive reference [11], without formal comparison with the patient groups.

Table 6. Multivariate logistic regression for postoperative AKI.

Variable	Principal Model: Adjusted OR (95% CI)	*p*-Value	Rating	Baseline-Adjusted Model: Adjusted OR (95% CI)	*p*-Value	Rating
CPB time, per minute	1.016 (1.009–1.022)	<0.001	***	1.016 (1.010–1.023)	<0.001	***
Left-sided NIRS at MP5, per %	0.965 (0.938–0.992)	0.011	**	0.976 (0.946–1.006)	0.117	-
DeBakey type I	1.561 (0.805–3.029)	0.188	-	1.547 (0.794–3.016)	0.200	-
Number of renal arteries involved, per additional artery	2.041 (1.167–3.569)	0.012	**	2.192 (1.240–3.875)	0.007	***
Left-sided NIRS at MP1, per %	–	–	–	0.978 (0.954–1.003)	0.081	*

Adjusted ORs with 95% CIs; two-sided Wald *p*-values. Both models: $n = 273$, 172 AKI events. Baseline adjustment adds left-sided NIRS at MP1. MP1: 5 min before CPB; MP5: 5 min before CPB completion. *** $p < 0.01$/ ** $p < 0.05$/ * $p < 0.1$. Abbreviations: AKI, acute kidney injury; CI, confidence interval; CPB, cardiopulmonary bypass; MP, measurement point; NIRS, near-infrared spectroscopy; OR, odds ratio.

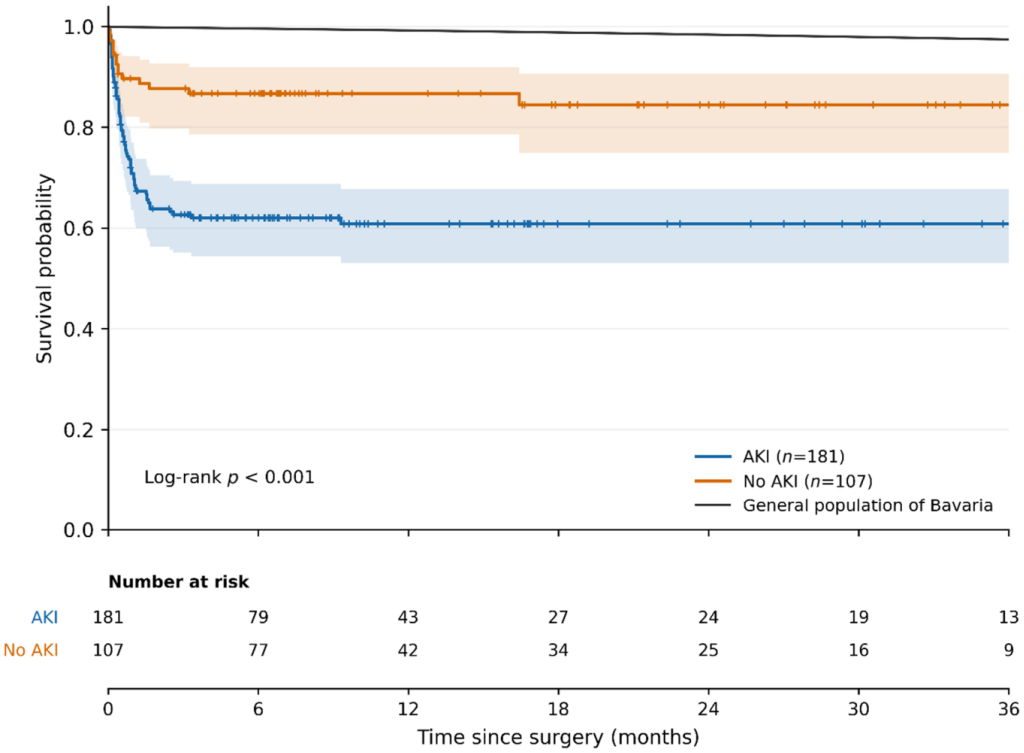


Figure 3. Postoperative survival and follow-up data after open surgery in ATAAD. AKI any stage vs. no AKI. Kaplan–Meier survival estimates with censoring. Shading: 95% confidence intervals; ticks: censoring. Numbers at risk are shown below; *p*-values are from log-rank tests. Comparison with the overall population survival as imposed by official data. Abbreviations: AKI, acute kidney injury.

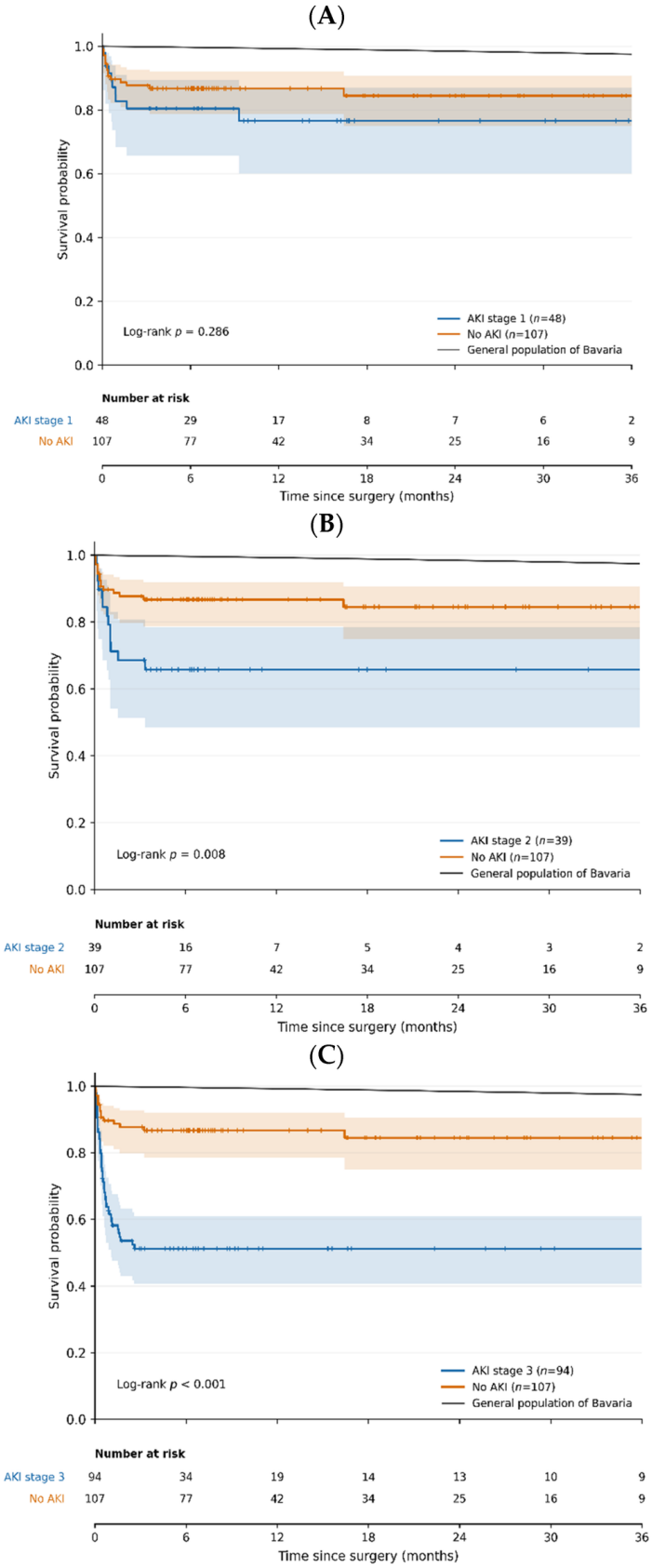


Figure 4. Postoperative survival and follow-up data after open surgery in ATAAD. (**A**–**C**): AKI stage 1/2/3 vs. no AKI. Kaplan–Meier survival estimates with censoring. Shading: 95% confidence intervals; ticks: censoring. Numbers at risk are shown below; *p*-values are from log-rank tests. Comparison with the overall population survival as imposed by official data. Abbreviations: AKI, acute kidney injury.

4. Discussion

In this predominantly ATAAD cohort, AKI affected nearly two thirds of patients and was associated with greater postoperative morbidity and higher in-hospital mortality. CPB duration and anatomical renal artery involvement remained associated with AKI in the adjusted analyses. Lower left-sided cerebral NIRS at MP5 was associated with AKI in the principal model, but the association attenuated after adjustment for baseline saturation. The findings therefore support an exploratory relationship between cerebral oxygenation and renal risk, while leaving uncertain whether MP5 provides information beyond baseline NIRS.

The incidence and clinical burden of AKI are consistent with previous studies of acute aortic dissection repair [2–4]. Comparisons between cohorts nevertheless depend on operative complexity, case mix, and the criteria used to identify renal injury. Our modified KDIGO definition incorporated serum creatinine and acute RRT but not urine output. The substantial requirement for RRT illustrates the severity of renal complications in this cohort; however, patients who died while receiving RRT must be distinguished from survivors discharged with ongoing renal support. The higher rates of stroke and mortality accompanying AKI indicate a broader adverse postoperative course rather than proving that renal injury caused these outcomes.

Renal artery involvement was an important anatomical correlate of AKI. The increasing incidence with unilateral and bilateral involvement suggests that renal branch vessel burden adds information beyond a general description of dissection extent. Previous studies have linked preoperative renal malperfusion to postoperative AKI and operative mortality [12,13]. Our variable differs from confirmed malperfusion: it denotes dissection involving the renal artery origin, without separate adjudication of dynamic obstruction or functional perfusion impairment. These mechanisms cannot be inferred from anatomical involvement alone. DeBakey type I was associated with AKI in univariable analysis but not after adjustment for renal artery burden. This attenuation is consistent with overlapping anatomical information and does not establish that dissection extent is clinically unimportant.

The association with longer CPB duration agrees with previous studies identifying operative and bypass-related factors in postoperative renal injury [3,5,6]. CPB duration may reflect both procedural complexity and cumulative exposure to hemodilution, inflammatory activation, and disturbances in perfusion. Higher peak lactate and lower intraoperative urine output in the AKI group were compatible with greater physiological stress, although neither establishes the specific mechanism of renal injury. Studies examining perfusion pressure, pump flow, and urine output during CPB support further assessment of these potentially relevant factors [14,15]. Our data do not show that shortening bypass or altering an individual perfusion target would independently reduce AKI.

The NIRS findings should be interpreted within this multifactorial setting. Cerebral oximetry measures regional tissue oxygenation, which may share determinants with renal oxygen delivery, but cerebral saturation is not a direct measure of renal perfusion [7]. Choi et al. reported an association between oxygen desaturation measured over the renal region and AKI after cardiac surgery [8]. In ATAAD repair, Zhao et al. found that muscular desaturation relative to baseline was associated with AKI, whereas the cerebral desaturation thresholds they examined were not [10]. These studies support interest in tissue oxygenation while demonstrating that findings from different monitoring sites and measurement definitions are not interchangeable. Our phase-specific cerebral measurements extend this question rather than establish cerebral NIRS as a renal surrogate.

Baseline adjustment is particularly relevant because left-sided saturation was already lower before CPB in patients who subsequently developed AKI. The attenuation of the

MP5 estimate after inclusion of MP1 suggests that the later measurement partly reflects oxygenation characteristics present before bypass. Its confidence interval after adjustment included no association; this does not exclude a small effect, but it limits claims of additional prognostic information. Moreover, MP5 was recorded while CPB was still ongoing. It should therefore not be interpreted as a measurement after weaning or as evidence of impaired native cardiac output. Time-matched pump flow, hemoglobin, arterial carbon dioxide tension, perfusion pressure, and vasoactive support would be needed to clarify the physiology underlying the association.

The apparent laterality also requires cautious interpretation. A significant association on the left and a non-significant association on the right do not themselves demonstrate a difference between hemispheres. Left-sided MP5 was selected following exploratory comparisons rather than prespecified as the primary NIRS exposure. Bilateral antegrade cerebral perfusion was the institutional standard, and cerebral oximetry has established applications in detecting perfusion abnormalities during aortic surgery [16–18]. However, right-sided arterial inflow does not by itself establish preferential right hemispheric oxygenation; Hillebrand et al. reported balanced cerebral oxygenation with right axillary cannulation [19]. Anatomical variation, sensor-related factors, and sampling variability remain possible explanations for the observed pattern.

The modest separation and substantial overlap of MP5 values limit direct clinical application. Neither a renal risk threshold nor an improvement in prediction beyond clinical and anatomical factors was established. NIRS remains useful for its intended cerebral monitoring role, including assessment of potentially correctable perfusion disturbances [16], but these results do not justify a NIRS-guided renal protection algorithm. Prospective evaluation should examine bilateral measurements, changes relative to baseline, and continuous desaturation burden alongside renal artery anatomy and contemporaneous hemodynamic data.

The survival analyses reinforce the adverse outcome pattern associated with AKI but require qualification. The separation of Kaplan–Meier curves includes early postoperative deaths and cannot establish an independent effect on survival after discharge. Follow-up was incomplete, with only 49 patients remaining at risk at two years. Stage-specific comparisons were exploratory, and the Bavarian general population curve served as a descriptive reference rather than a matched control. In-hospital mortality is consequently the most securely ascertained mortality endpoint in this dataset.

Several limitations should be acknowledged. The retrospective single-center design permits residual confounding, and exclusion of deaths within 24 h limits applicability to the earliest postoperative course. Anatomical renal artery involvement was not equivalent to adjudicated malperfusion, and subsequent endovascular or angiological reinterventions after discharge were not systematically captured. Creatinine/RRT-based staging without urine output criteria may classify AKI differently from full KDIGO assessment. Renal biomarkers such as NGAL, which have been investigated in ATAAD surgery, were unavailable [20].

NIRS analysis was based on five documented measurements rather than exported continuous recordings, precluding the assessment of desaturation duration or cumulative burden. Readings were visible to clinicians and could trigger treatment, which may alter observed associations. Two INVOS generations were used, and signal acquisition and extracranial contributions may affect comparability [7]. Selection of the left-sided exposure, multiple exploratory comparisons without multiplicity adjustment, and the absence of internal or external validation limit inference about prediction.

5. Conclusions

AKI affected nearly two thirds of patients and was accompanied by longer hospitalization, and higher in-hospital mortality. Renal artery involvement and prolonged CPB remained associated with AKI after adjustment. The modest left-sided NIRS association weakened when baseline saturation was considered, leaving its additional prognostic contribution uncertain. These findings support assessing cerebral oxygenation alongside anatomical renal risk and operative factors in future prospective studies.

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Abbreviations

ACT	Activated clotting time
AKI	Acute kidney injury
ATAAD	Acute type A aortic dissection
AVR	Aortic valve replacement
BMI	Body mass index
CABG	Coronary artery bypass grafting
CI	Confidence interval
CK(-MB)	Creatine kinase (MB isoenzyme)
CPB	Cardiopulmonary bypass
CPR	Cardiopulmonary resuscitation
FET	Frozen elephant trunk
FFP	Fresh frozen plasma
FND	Focal neurological disorder
GERAADA	German Registry for Acute Aortic Dissection Type A
HCA	Hypothermic circulatory arrest
ICU	Intensive care unit
IQR	Interquartile range
KDIGO	Kidney Disease: Improving Global Outcomes
LCCA	Left common carotid artery

LRA	Left renal artery
LSA	Left subclavian artery
MP	Measure point
NIRS	Near-infrared spectroscopy
OR	Odds ratio
PC	Platelet concentrate
pRBC	Packed red blood cells
RCCA	Right common carotid artery
RRA	Right renal artery
RRT	Renal replacement therapy
RSA	Right subclavian artery
SD	Standard deviation
SMA	Superior mesenteric artery

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