Abdominal aortic aneurysms

The quest for meaningful biomarkers and opportunities to improve surgical care

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CHAPTER 6

Long-term survival after acute kidney injury following ruptured abdominal aortic aneurysm repair

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ABSTRACT

Objective

Acute kidney injury (AKI) is a major complication of ruptured abdominal aortic aneurysm (RAAA). Severe AKI is associated with high morbidity and mortality in the short term. The objective of this study was to determine the association between AKI after RAAA repair and long-term survival.

Methods

We conducted a retrospective cohort study of all patients undergoing RAAA repair in three hospitals between 2004 and 2011. Outcomes were long-term survival after RAAA repair, incidence of postoperative AKI, and chronic dialysis rates. Survival rates were compared between different AKI groups (no AKI, Risk, Injury, Failure) with Kaplan-Meier survival analyses and log-rank tests. Univariable and multivariable Cox regression analyses were carried out to assess the association of survival with AKI, preoperative shock, postoperative shock, and sex. The main analysis focused on the group of patients surviving initial hospital stay.

Results

Our study encompassed 362 patients with RAAA. AKI occurred in 267 of 362 patients (74%). At discharge, 267 patients were alive (74%). Median survival in this group was 7.2 years. Survival was not significantly different between the four AKI groups ($P = .07$). However, the univariable Cox regression analysis demonstrated a significant association between Failure and reduced long-term survival compared with having no AKI (hazard ratio, 1.85; 95% confidence interval, 1.15-2.97). This association did not remain significant after multivariable adjustment. Four patients were discharged with chronic dialysis and four other patients needed chronic dialysis later after discharge.

Conclusions

This study demonstrates no significant independent association between AKI after RAAA repair and long-term survival. Only a small proportion of patients developed end-stage renal disease at a later stage in life.

INTRODUCTION

Ruptured abdominal aortic aneurysm (RAAA) carries a high mortality. Up to 80% of the patients do not survive the immediate consequences of AAA rupture.[1] One of the main complications of RAAA is acute kidney injury (AKI). Up to three-quarters of patients who survive RAAA repair suffer from AKI.[2, 3] The incidence of severe AKI after RAAA repair ranges between 16 and 36%.[2-4] The cause of AKI after RAAA is multifactorial and consists of prolonged hypovolemia leading to renal ischemia, acute-on-chronic kidney disease, general atherosclerotic disease, suprarenal aortic cross-clamping in open repair, and the use of nephrotoxic contrast agents and medication.[2, 3, 5] We previously reported that patients with severe AKI after RAAA have a higher risk of dying than patients without AKI (combined 30-day and in-hospital death rate, adjusted odds ratio, 6.36; 95% confidence interval [CI], 2.23-18.13).[3] Up to a quarter of patients with AKI after RAAA require renal replacement therapy (RRT) during admission, and 2% of patients are discharged with permanent RRT.[3]

Until recently, it was thought that AKI and chronic kidney disease were two distinct syndromes. It was common belief that long-term outcomes after recovery from AKI are benign.[6] However, new observational studies have shown an association between AKI and the development of chronic kidney disease and even increased mortality.[6] More specifically, it has been demonstrated that preoperative reduced renal function and postoperative AKI are associated with increased long-term mortality after cardiac surgery.[7, 8] Much less is known about the long-term consequences of AKI after RAAA. The few reports that have been published demonstrate an association between AKI after RAAA and increased mortality after 1 year.[2, 4] As a consequence, some authors suggest more intensive monitoring of renal function after postoperative discharge.[2, 7]

We have carried out this study to confirm these claims about long-term effects of AKI. The primary aim of our study was to assess the association between AKI after RAAA repair and long-term survival. The secondary aim was to determine the incidence of long-term end-stage renal disease. We hypothesized that AKI is associated with reduced long-term survival in patients with RAAA and is associated with increased end-stage renal disease.

METHODS

Study design & setting

This is a retrospective cohort study using both prospectively and retrospectively collected data from the Amsterdam Acute Aneurysm (AJAX) cohort database. This database consisted of consecutive patients with RAAA and was part of a prospective cohort study running parallel to the AJAX trial (ISRCTN: 66212637). Details of the AJAX trial and AJAX cohort have been published previously.[9] Patient consent was not needed for this study because of the observational study design. This paper includes all items recommended by the Strengthening the Reporting of Observational studies in Epidemiology STROBE statement.[10]

Participants & study size

This study included all consecutive patients from the AJAX cohort who underwent RAAA repair in three hospitals in Amsterdam (AMC, VUmc and OLVG hospitals) between 2004 and 2011. Patients with preoperative permanent dialysis and those who died during intervention or within 6 hours after arrival on the intensive care unit (ICU) were excluded from current analysis. Also, patients with unknown demographics or unknown short-term outcomes were excluded.

Outcomes

The primary outcome of this study was long-term survival after RAAA repair. Secondary outcomes were the incidence of postoperative AKI according to the Risk, Injury, Failure and Loss and End-stage (RIFLE) criteria and chronic dialysis rates.[11] The RIFLE criteria define short-term AKI as Risk, Injury or Failure, and classification is based on serum creatinine (SCr) levels or urine output (Supplementary Table I). The RIFLE criteria previously have been used to classify AKI in patients undergoing RAAA repair.[3, 12] AKI categorization was based on the worst SCr or urine output. SCr and urine output were recorded during ICU admission up until 1 week.

Baseline SCr was defined according to the steps recommended by the RIFLE criteria (Supplementary Figure). In brief, baseline SCr was based on the lowest value of the premorbid SCr, the preoperative SCr, an estimated SCr (obtained through the Modification of Diet in Renal Disease equation), or an imputed value when premorbid, preoperative, and estimated Scr were unavailable (calculated as the median baseline SCr of patients with renal comorbidity). Urine output category was estimated in separate blocks of 6, 12 and 24 hours. The lowest block of urine output was used for AKI categorization (Supplementary Table I). In patients treated with RRT, the AKI category before start of RRT was used for analysis. All three hospitals used venovenous hemofiltration as temporary RRT.

The secondary outcome was the incidence of long-term end-stage renal disease. We used the incidence of chronic dialysis as a surrogate marker.

Pre- and postoperative shock markers

The Glasgow Aneurysm Score (GAS)[13] was included to serve as a surrogate marker for preoperative shock profile. The GAS includes age, presence of shock, myocardial disease, cerebrovascular disease, renal disease, and type of aneurysm repair. As markers of postoperative shock status, we used the Acute Physiology and Chronic Health Evaluation (APACHE) II score, the administration of vasopressors, and fluid balance in the first 24 hours after RAAA repair. The APACHE II score is a measurement of disease severity and includes body temperature, serum electrolyte levels, and the Glasgow Coma Scale score to predict the risk of ICU mortality. Administration of vasopressors was categorized as none (no ICU admission or no vasopressors administered), low dose (norepinephrine <2 mg/min or dopamine <500 mg/min), or high dose (any epinephrine, norepinephrine >2 mg/min, or dopamine >500 mg/min).[3] Fluid balance was categorized in <2 L, 2 to 5 L positive, or >5 L positive.

Data sources

Clinical data were drawn from the AJAX cohort database. Dates of death were retrospectively acquired from the municipal registry of death certificates in August 2016. Under Dutch law, the municipal registry can provide data for research purposes if a patient's privacy is not disproportionally affected. Long-term chronic dialysis data were retrieved from the Dutch national dialysis registry (Renine, stichting Nefrovisie) in December 2016.[14] Chronic dialysis of >28 days is registered in this registry.

Statistical methods and bias

Survival rates were estimated by Kaplan-Meier survival analysis and were compared between AKI categories (no AKI, Risk, Injury, Failure) using the log-rank test. Patients who could not be identified in the municipal registry were censored at last point of hospital contact during follow-up. To test whether the proportional hazards assumption was met, log-minus-log plots were created for the following variables: method of RAAA repair (OR vs. EVAR), GAS, APACHE II score, the administration of vasopressors, fluid balance, and sex. Subsequently, a univariable Cox proportional hazards regression analysis was carried out for all variables that showed proportionality of hazards in the log-minus-log plots. In addition, we carried out a multivariable Cox regression analysis with a simultaneous method of entry. Again, all proportional variables were included in the multivariable model. This also included the variables that were not significant in the univariable analysis.

For the purpose of including all patients in the regression model, an additional imputation model was carried out for APACHE II, GAS and baseline characteristics. As previously published, 10 datasets were created by using logistic and linear regression models.[3] The predictors in the imputation model were baseline characteristics, serum hemoglobin, level of consciousness, AKI, and death.

All analyses were carried out according to a prespecified plan with the aim of investigating the relation between AKI and long-term survival. Data were analyzed using IBM SPSS Statistics version 23 (IBM Corp, Armonk, NY). Continuous data are described by either mean and standard deviation, or median and interquartile range, depending on normality of the distribution. Categorical data are described in numbers and percentages. A *P* value of < .05 was considered statistically significant.

RESULTS

Participants

Of 457 consecutive patients with RAAA, 407 underwent intervention. The combined in-hospital and 30-day mortality of patients who were operated was 140 (34.4%). For current analysis, 45 of 407 patients were excluded because of the following reasons: 32 died during intervention, 9 died within 6 hours after arrival on ICU, 3 had missing charts, and 1 had preoperative permanent dialysis (Figure 1). Survival data of the 362 included patients were retrieved 12.4 years after the first inclusion and 5.5 years after the last inclusion. Survival data could not be retrieved from the municipal registry for 19 of 362 patients. As previously mentioned, these 19 patients were censored at last point of hospital contact during follow-up.

Outcome data

Sixty-eight patients underwent EVAR and 294 patients underwent open repair. Characteristics of the patients are listed in Table I and have been published previously.[3] In short, AKI occurred in 267 patients (74%), with 71 patients in the Risk, 104 in the Injury, and 92 in the Failure category. Median age was 76 years (interquartile range 69-81) and 294 of 362 (81%) patients were male. As previously reported, patients who were classified with AKI had a significantly higher GAS and APACHE II score, more fluid and vasopressor administration, higher age, and more cardiac comorbidity (Supplementary Table II). In total, 267 patients survived the initial hospital stay.

Survival

By August 2016, 248 of 362 patients (69%) had died. Median survival in the group of patients surviving initial hospital stay was 7.2 years. Because the number of patients

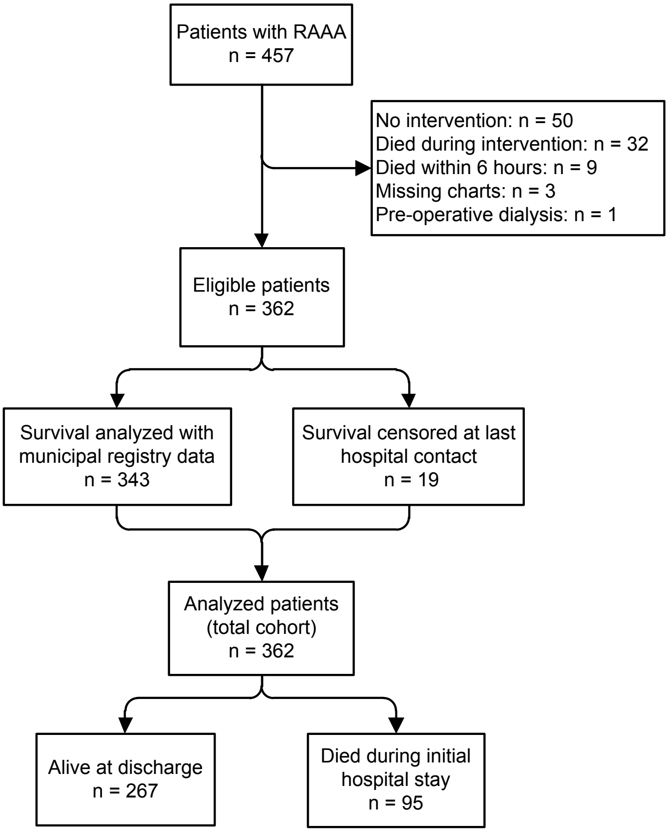


Figure 1. Flow chart of analyzed patients.
RAAA, Ruptured abdominal aortic aneurysm.

surviving longer than 10 years was too small for solid statistical analyses (2 patients alive at 12 years after repair), the survival was censored at 10 years (33 patients alive) for the Kaplan-Meier survival analyses and Cox-regression analyses. The Kaplan-Meier survival analyses are shown for the main analysis of patients surviving initial hospital stay and for the total cohort in Figures 2 and 3, respectively. In the patients surviving initial hospital stay, survival was not significantly different between the groups of no AKI, Risk, Injury and Failure (Figure 2: $P = .07$). However, survival was significantly different between these AKI groups in the total cohort (Figure 3: $P = .00$).

Log-minus-log testing demonstrated proportionality of hazards for all included variables except for the method of repair (open repair vs EVAR). Therefore, this variable was not included in the subsequent regression analyses. Tables II and III present the results of the

Table I. Baseline characteristics of 362 patients with a ruptured abdominal aortic aneurysm (RAAA)

Variable	Value
Age, years	76 (69-81)
Sex, male/female	294/68
Cardiac comorbidity	42% (152/360)
Pulmonary comorbidity	23% (83/359)
Renal comorbidity	12% (42/359)
Cerebrovascular comorbidity	16% (58/360)
Diabetes	10% (37/356)
Hypertension	43% (154/358)
BMI	25.6 (23.4-27.8)
Lowest preoperative in-hospital systolic blood pressure	98 (74-128)
Preoperative cardiopulmonary resuscitation	8% (27/351)
Premorbid SCr level (available in 45 patients), µmol/L	84 (64-108)
Preoperative SCr level (available in 359 patients), µmol/L	104 (85-132)
Estimated SCr level, µmol/L	98 (95-100)
Baseline SCr level, µmol/L	96 (74-100)
EVAR or open repair	68/294
Preoperative GAS	91 (81-103)
APACHE II score	19 (15-24)
Use of vasopressors 24 hours after intervention	
None	33% (120/362)
Low dose (norepinephrine <2 µgram/min or dopamine <500 µg/min)	45% (162/362)
High dose (epinephrine, norepinephrine >2 µg/min or dopamine >500 µg/min)	22% (80/362)
Fluid balance first 24 hours after intervention	
<2 L positive	36% (132/362)
>2 L and <5 L positive	36% (130/362)
>5 L positive	28% (100/362)

APACHE II, Acute Physiology and Chronic Health Evaluation II; BMI, body mass index; EVAR, endovascular aneurysm repair; GAS, Glasgow Aneurysm Score; SCr, serum creatinine.
Continuous data are presented as median (interquartile range). Categorical data are presented as % (n/N).

univariable and multivariable Cox regression analyses for the patients surviving initial hospital stay and for the total cohort. In the patients surviving initial hospital stay, Failure was associated with reduced survival in the univariable analysis compared with no AKI (hazard ratio [HR], 1.85; 95% CI, 1.15-2.97; Table II). However, none of the AKI categories, including Failure, remained significantly associated with reduced survival after multivariable adjustment. Only GAS remained independently associated with reduced survival. In the total cohort of patients undergoing RAAA repair, those who were categorized as Injury and Failure had significantly reduced survival in comparison to those

Table II. Cox regression analysis: Patients surviving initial hospital stay

	Univariable analysis		Multivariable analysis	
	HR (95% CI)	Significance (*P* value)	HR (95% CI)	Significance (*P* value)
No AKI	Reference	Reference	Reference	Reference
AKI: Risk	1.13 (.71- 1.79)	.62	.97 (.59 - 1.58)	.90
AKI: Injury	1.31 (.87 - 1.98)	.19	1.034 (.67 - 1.59)	.88
AKI: Failure	1.85 (1.15 - 2.97)	.01	1.24 (.73 - 2.10)	.42
GAS	1.03 (1.02 - 1.04)	.00	1.03 (1.02 - 1.04)	.00
APACHE II	1.06 (1.03 - 1.09)	.00	1.02 (.98 - 1.06)	.26
Fluid balance < 2 L	Reference	Reference	Reference	Reference
Fluid balance 2-5 L	1.50 (1.05 - 2.15)	.03	1.28 (.88 - 1.84)	.20
Fluid balance > 5L	1.59 (1.01 - 2.50)	.04	1.60 (.93 - 2.76)	.09
No vasopressors	Reference	Reference	Reference	Reference
Vasopressors low	1.29 (.91 - 1.84)	.15	1.08 (.75 - 1.58)	.67
Vasopressors high	1.02 (.57 - 1.81)	.95	.68 (.36 - 1.28)	.23
Female sex	1.60 (1.06 - 2.41)	.03	1.27 (.81 - 1.98)	.29

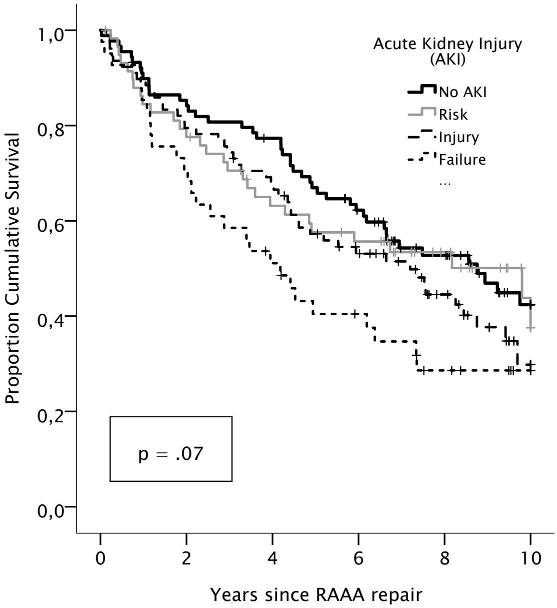
AKI, Acute kidney injury; APACHE II, Acute Physiology and Chronic Health Evaluation II; CI, confidence interval; GAS, Glasgow Aneurysm Score; HR, hazard ratio.

with no AKI in the univariable analysis (Injury: HR, 1.65; 95% CI, 1.15-2.36; Failure: HR, 3.47; 95% CI, 2.43-4.95). From the AKI categories, only Failure remained significantly associated in the multivariable analysis of the total cohort (HR, 1.76; 95% CI, 1.17-2.63; Table III).

RRT

During admission, 83 of 362 patients received temporary RRT (23%; 95% CI, 19%-28%). The decision to start RRT was based on the judgement of the treating physicians. Of the 267 discharged patients, four patients were discharged with chronic RRT. In these four patients, AKI was categorized as Failure, and RRT was initiated the first postoperative day. RRT was continued intermittently until discharge in all four. The time between RAAA repair and discharge ranged from 19 to 167 days. In these patients, baseline SCr concentration before rupture was unknown, and the first measured SCr level ranged from 98 to 410 μmol/L.

Four other patients developed end-stage renal disease requiring chronic RRT at a later stage in life (starting 3.1 to 8.7 years after discharge). In these four patients, AKI during initial hospital stay was categorized as Failure in two, Injury in one, and Risk in another one. Only one patient (who was categorized as Failure) received RRT during initial hospital stay. All eight patients receiving chronic RRT never discontinued with RRT.



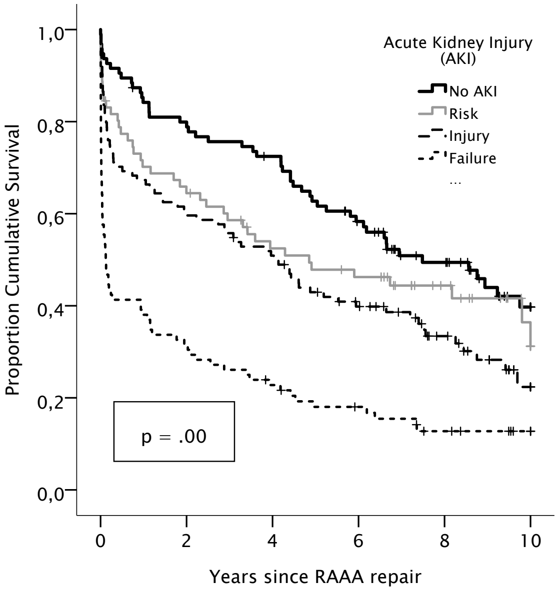
Number at risk						
No AKI	89	75	67	50	34	17
Risk	59	45	34	29	17	7
Injury	78	62	52	37	22	6
Failure	41	29	20	14	8	3

AKI, Acute kidney injury; RAAA, ruptured abdominal aortic aneurysm.

Figure 2. Kaplan-Meier survival analysis in patients surviving initial hospital stay.

DISCUSSION

Our study shows that survival was not significantly different between patients who had no AKI, and those who had AKI defined as Risk, Injury, or Failure. However, AKI defined as Failure was significantly associated with reduced 10-year survival compared with no AKI in the univariable Cox regression analysis. This association did not remain significant after adjusting for confounding factors. Only GAS, which is mainly composed of patient's age, remained independently associated with reduced survival. The results are in contrast with previous studies showing an independent association between AKI and reduced short-term survival after RAAA. Our results also show that only a small proportion of patients recovering from AKI suffer from end-stage renal disease at a later stage in life.



Number at risk						
No AKI	95	75	67	50	34	17
Risk	71	45	34	29	17	7
Injury	104	62	52	37	22	6
Failure	92	29	20	14	8	3

AKI, Acute kidney injury; RAAA, ruptured abdominal aortic aneurysm.

Figure 3. Kaplan-Meier survival analysis in the total cohort number at risk.

Interpretation

To our knowledge, this study is the first to investigate the effects of AKI on long-term survival (median, 7.2 years) after RAAA repair. A possible reason for the fact that our results conflict with those reported in other RAAA studies may be that other RAAA studies did not adjust for patients surviving initial hospital stay and reported 12-month survival.[2,4]

Up to a quarter of patients with RAAA received RRT during admission. Yet fortunately, only 1.5% of patients were discharged with RRT and only 3% developed end-stage renal disease requiring permanent RRT. This is in accordance with other studies showing that two-thirds of discharged patients with AKI after RAAA have complete recovery of kidney function after 1 year.[2] However, those who remain on RRT often have a reduced quality of life and consume greater health care resources than the general population.[15]

Table III. Cox regression analysis: Total cohort

	Univariable analysis		Multivariable analysis	
	HR (95% CI)	**Significance (P value)**	**HR (95% CI)**	**Significance (P value)**
No AKI	Reference	Reference	Reference	Reference
AKI: Risk	1.32 (.87 - 1.99)	.189	.91 (.59 - 1.39)	.66
AKI: Injury	1.65 (1.15 - 2.36)	.01	1.08 (.74 - 1.58)	.68
AKI: Failure	3.47 (2.43 - 4.95)	.00	1.76 (1.17 - 2.63)	.01
GAS	1.03 (1.03 - 1.04)	.00	1.03 (1.02 - 1.04)	.00
APACHE II	1.09 (1.07 - 1.11)	.00	1.02 (.99 - 1.05)	.15
Fluid balance < 2 L	Reference	Reference	Reference	Reference
Fluid balance 2-5 L	1.94 (1.41 - 2.66)	.00	1.58 (1.14 - 2.17)	.01
Fluid balance > 5L	3.25 (2.34 - 4.51)	.00	2.27 (1.52 - 3.39)	.00
No vasopressors	Reference	Reference	Reference	Reference
Vasopressors low	1.27 (.94 - 1.72)	.123	1.02 (.742 - 1.39)	.93
Vasopressors high	2.73 (1.94 - 3.84)	.00	1.43 (.96 - 2.14)	.08
Female sex	1.62 (1.19 - 2.19)	.00	1.38 (1.00 - 1.91)	.05

AKI, Acute kidney injury; APACHE II, Acute Physiology and Chronic Health Evaluation II; CI, confidence interval; GAS, Glasgow Aneurysm Score; HR, hazard ratio.

In patients with RAAA who survive initial hospital stay, the fact that none of the AKI categories is independently associated with long-term mortality or end-stage renal disease could imply that either the kidneys have good recovery ability after AKI or AKI does not irreversibly damage the renal tissue, or the patients die of other health-related problems before the renal problems become apparent. It appears that AKI is most threatening in the short term (as we have previously demonstrated) but has no lasting effect on long-term survival.

The results of the log-rank test and the univariable Cox regression could appear somewhat contradicting. After all, the log-rank test showed no significant survival difference between the AKI groups, whereas the univariable Cox regression analysis demonstrated a significant association between Failure and reduced long-term survival. To understand this difference, it is important to realize that the first analysis compared the survival curves of the four AKI groups and the latter looked to the cohort as a whole and tested which variables significantly affected survival. In addition, this regression analysis compared Failure with no AKI and, in contrast to the log-rank test, did not compare all four groups with each other. The outcome of the univariable analysis suggests that persons with Failure have reduced long-term survival in comparison to those without AKI. However, this significant association could not be confirmed in our multivariable analysis.

Strengths, limitations and generalizability

A strength of this study is the fact that the cohort is larger than those in the studies reporting on midterm survival after post-RAAA AKI.[3, 4] An obvious limitation of the study is its retrospective design. Missing survival data were minimized by retrieving survival data from the municipal registry. Unfortunately, these data could not be retrieved for 19 patients (5%), mainly because of emigration and multiple persons with identical names and dates of birth in the registry. With respect to the imputed baseline characteristics, the amount of missing data that was imputed varied between 0% and 8% (Supplementary Table II).

The vast majority of patients in our study were treated by open repair (294/362). In the course of time, an increasing number of patients with RAAA are being treated with EVAR in daily practice. It is known that patients undergoing EVAR have a significantly lower incidence of AKI than those undergoing open repair, after both acute and elective AAA repair.[4, 16, 17] Therefore, it could be speculated that patients with RAAA nowadays have a better renal outcome than reported in our current study.

Another known limitation in this research field is the application of a wide range of criteria to define AKI.[18] This is illustrated by the fact that van Beek et al[3] use the RIFLE criteria, Kopolovic et al[2] use the Acute Kidney Injury Network classification system, and Ambler et al[4] use the Aneurysm Renal Injury Score. Other suggested classification systems include the Kidney Disease: Improving Global Outcomes criteria.[18] For this study, we have decided to define AKI with the widely accepted and validated RIFLE criteria because the previously obtained data–at the time–were collected with the aim of being analyzed with the RIFLE criteria. A strength of this study is the fact that both SCr and urine production were included for AKI identification. It is unlikely that cases of AKI have remained unobserved as a consequence of dilution of SCr concentration due to fluid resuscitation, as urine output was also included for AKI categorization. As recently reconfirmed, it is of great importance to use both SCr level and urine output because SCr level alone fails to identify AKI in many patients.[19]

The nonstandardized initiation of RRT could have played a role as a confounding factor, especially on renal recovery in the short term. It is possible that patients with early RRT initiation had better renal recovery. However, the influence of early RRT initiation on long-term renal outcome could not be determined with the current study.

Our results need confirmation from other studies, preferably large cohorts of patients with RAAA, with information on postoperative renal function and long-term survival data. To correct for short-term in-hospital mortality, it is important that future studies carry out a separate analysis of patients surviving the initial hospital stay.

Conclusions

This study demonstrates no significant independent association between AKI after RAAA repair and reduced 10-year survival. Moreover, only a small proportion of patients suffer from end-stage renal disease at a later stage in life. In addition, the occurrence of end-stage renal disease does not seem to be related to the severity of postoperative AKI. These results suggest that kidneys have good recovery ability after AKI or that AKI does not irreversibly damage the renal tissue in patients with RAAA who survive initial hospital stay.

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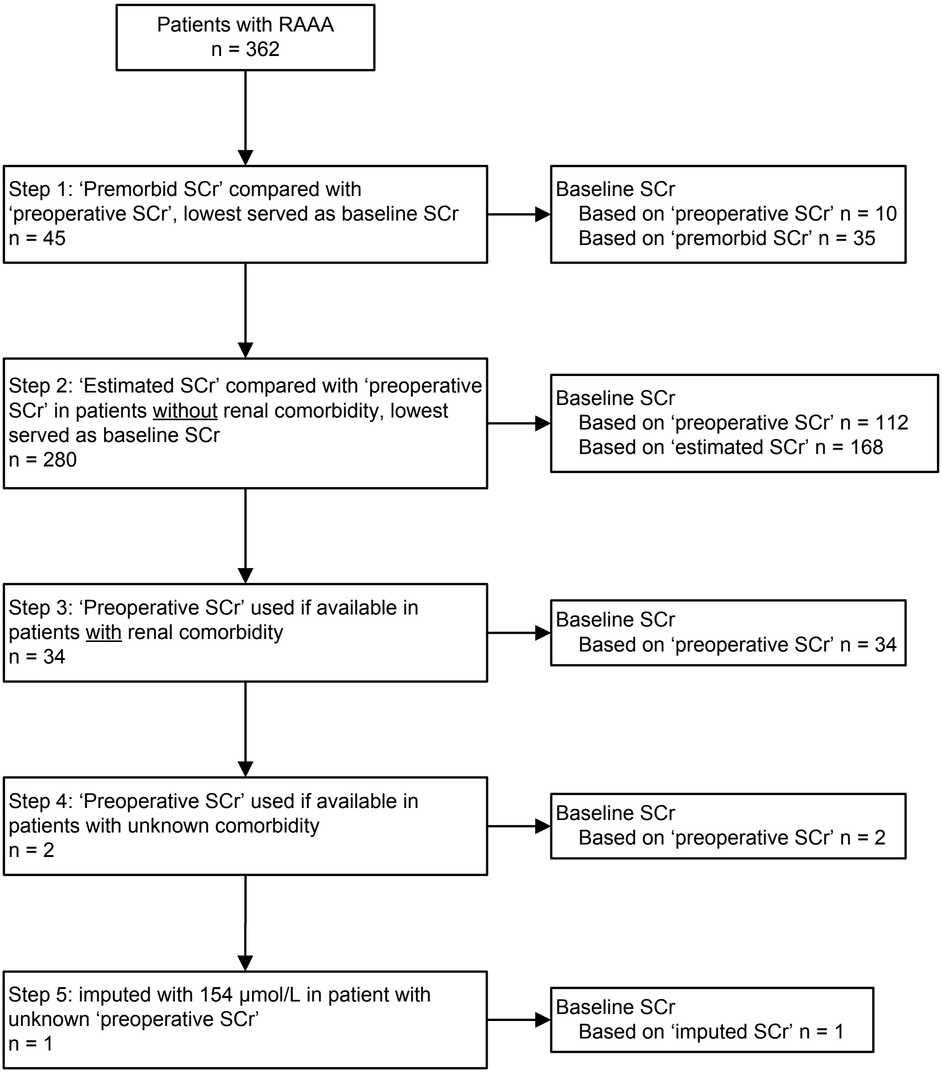
SUPPLEMENTS



Supplementary Figure. Method of baseline serum creatinine (SCr) approximation.
RAAA, Ruptured abdominal aortic aneurysm.

Supplementary Table I. The Risk, Injury, Failure, Loss, and End-stage (RIFLE) criteria

Category	RIFLE SCr criteria[a]	RIFLE urine output criteria
Risk	Increase ≥1.5 times from baseline or GFR decrease >25%	<0.5 mL/kg/h ≥6 hours
Injury	Increase ≥2 times from baseline or GFR decrease >50%	<0.5 mL/kg/h ≥12 hours
Failure	Increase ≥ 3 times from baseline or GFR decrease >75% or ≥354 µmol/L with an acute increase ≥44 µmol/L from baseline	<0.3 mL/kg/h ≥24 hours or anuria ≥12 hours
Loss	Complete loss of kidney function >4 weeks	
End-stage	Complete loss of kidney function >3 months	

GFR, Glomerular filtration rate; SCr, serum creatinine. aBased on change between two SCr values within 7 days after admission.

Supplementary Table II. Preoperative, perioperative, and postoperative characteristics of patients with and without acute kidney injury (AKI) defined according to the Risk, Injury, Failure, Loss, and End-stage (RIFLE) criteria

	No AKI (n = 95)	AKI (n = 267)	P	Missing data	Imputed
Preoperative					
Age	73 (66-78)	76 (70-81)	<.01[a]	0	
Cardiac comorbidity	32% (30/95)	46% (122/265)	.01[b]	1% (2/362)	*
Pulmonary comorbidity	25% (24/95)	22% (59/264)	.56[b]	1% (3/362)	*
Renal comorbidity	11% (10/95)	12% (32/264)	.68[b]	1% (3/362)	*
Cerebrovascular comorbidity	19% (18/95)	15% (40/265)	.38[b]	1% (2/362)	*
Diabetes	11% (10/93)	10% (27/263)	.89[b]	2% (6/362)	
Hypertension	38% (35/93)	45% (119/265)	.22[b]	1% (4/362)	
BMI	26.0 (23.9-27.8)	25.4 (23.4-27.6)	.62[a]	39% (141/362)	
Lowest in-hospital SBP	110 (80-140)	90 (70-120)	<.01[a]	4% (13/362)	*
CPR	5% (5/92)	9% (22/259)	.34[b]	3% (11/362)	*
Baseline SCr level, μmol/L	90 (73-100)	96 (74-100)	.09[a]	0	d
GAS	83 (75-98)	94 (84-105)	<.01[a]	4% (16/362)	*
Perioperative					
If open repairr, suprarenal aortic cross clamping	26% (18/70)	41% (88/217)	.03[b]	2% (7/294)	
If EVAR, contrast agent used, mL	127 (100-233)	180 (120-250)	.21[a]	60% (41/68)	
Duration of intervention[c]	2:53 (2:25-3:25)	2:55 (2:23-3:59)	.21[a]	10% (35/362)	
Blood loss, L	1.4 (0.5-3.5)	2.9 (1.0-5.7)	<.01[a]	41% (148/362)	
Postoperative					
APACHE II at ICU admission	15 (12-18)	20 (17-25)	<.01[a]	8% (30/362)	*
Use of vasopressors first 24 hours after intervention					
None (or no ICU admission)	47% (45/95)	28% (75/267)	<.01[b]	0	
Low dose	50% (47/95)	43% (115/267)		0	
High dose	3% (3/95)	29% (77/267)		0	
Fluid balance first 24 hours after intervention					
<2 L positive	59% (56/95)	28% (76/267)	<.01[b]	0	
>2 L and <5 L	34% (32/95)	37% (98/267)		0	
>5 L positive	7% (7/95)	35% (93/267)		0	

APACHE II, Acute Physiology and Chronic Health Evaluation II; BMI, body mass index; CPR, cardiopulmonary resuscitation; EVAR, endovascular aneurysm repair; GAS, Glasgow Aneurysm Score; ICU, intensive care unit; SBP, systolic blood pressure; SCr, serum creatinine.

Continuous data are presented as median (interquartile range). Categorical data are presented as % (n/N). *Imputed value. [a] Mann-Whitney U test. [b] χ^2 test. [c] Time expressed as hours:minutes. [d] One imputed value according to the SCr approximation recommendations of the RIFLE criteria.