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Renal dysfunction and the associated decrease in survival after elective endovascular aneurysm repair

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Abstract

Objective—The reported frequency of renal dysfunction after elective endovascular aneurysm repair (EVAR) varies widely in current surgical literature. Published research establishes pre-existing end-stage renal disease as a poor prognostic indicator. We intend to quantify the mortality effect associated with renal morbidity developed postoperatively and to identify modifiable risk factors.

Methods—All elective EVAR patients with preoperative and postoperative renal function data captured by the Vascular Quality Initiative between January 2003 and December 2014 were examined. The primary study end point was long-term mortality. Preoperative, intraoperative, and postoperative parameters were analyzed to estimate mortality stratified by renal outcome and to describe independent risk factors associated with post-EVAR renal dysfunction.

Results—This study included 14,475 elective EVAR patients, of whom 96.8% developed no post-EVAR renal dysfunction, 2.9% developed acute kidney injury, and 0.4% developed a new hemodialysis requirement. Estimated 5-year survival was significantly different between groups, 77.5% vs 53.5%, respectively, for the no dysfunction and acute kidney injury groups, whereas the

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new hemodialysis group demonstrated 22.8% 3-year estimated survival ($P < .05$). New-onset postoperative congestive heart failure (odds ratio [OR], 3.50; 95% confidence interval [CI], 1.18-10.38), return to the operating room (OR, 3.26; 95% CI, 1.49-7.13), and postoperative vasopressor requirement (OR, 2.68; 95% CI, 1.40-5.12) predicted post-EVAR renal dysfunction, whereas a preoperative estimated glomerular filtration rate (eGFR) ≥60 mL/min/1.73 m^2 was protective (OR, 0.33; 95% CI, 0.21-0.53). Volume of contrast material administered during elective EVAR varies 10-fold among surgeons in the Vascular Quality Initiative database, but the average volume administered to patients is statistically similar, regardless of preoperative eGFR. Multivariable logistic regression demonstrated nonsignificant correlation between contrast material volume and postoperative renal dysfunction.

Conclusions—Any renal dysfunction developing after elective EVAR is associated with decreased estimated long-term survival. Protecting renal function with a rational dosing metric for contrast material linked to preoperative eGFR may better guide treatment.

Endovascular aneurysm repair (EVAR) overtook open repair as the most frequent elective surgical intervention for infrarenal abdominal aortic aneurysm (AAA) in the mid-2000s, representing 74% of repairs by 2010.[1] Several clinical trials demonstrated superior midterm mortality and lower morbidity in both low- and high-risk cohorts.[2-5] These landmark trials catalog complications associated with EVAR but do not explicate morbidity risk factors.

Renal dysfunction after EVAR is a rare event. In comparison to open AAA repair, EVAR engenders approximately half the risk for acute kidney injury (AKI) and one-third the risk for new hemodialysis (HD) requirement.[6] An early series describing a single center's experience in the late 1990s placed the frequency of a new HD requirement after elective EVAR at 3.5%.[7] Subsequent registry analyses and clinical trials refined this estimate to between 0.6% and 1.1%[2,3,5,8-10]; acute and chronic kidney injuries are poorly characterized in these reports. Hua et al reported AKI appearing at a frequency of 0.9% in their National Inpatient Sample analysis, whereas Pisimisis et al identified that 17.2% of their cohort suffered a similar complication.[8,11] No study reports long-term survival data after elective EVAR-induced renal dysfunction.

Several papers propose mechanisms leading to renal dysfunction, including embolism, ischemia, contrast-induced nephropathy, and suprarenal stent graft fixation.[9,12,13] No study examined a cohort adequately powered to correlate risk factors and outcomes with precision. This study intends to describe long-term survival after elective EVAR among patients developing AKI or a new HD requirement compared with EVAR patients with no renal dysfunction postoperatively. We intend to identify variables independently predicting renal dysfunction in efforts to guide therapy and to improve procedural quality. Survival is hypothesized to decrease in concert with severity of postoperative dysfunction. Preoperative estimated glomerular filtration rate (eGFR) and volume of contrast material administered intraoperatively are expected to independently predict renal morbidity.

METHODS

Patients

The Vascular Quality Initiative (VQI) is a regional collaborative aimed at improving health services. The organization is governed by the Society for Vascular Surgery Patient Safety Organization and is sponsored by multiple vascular disease societies. The VQI Research Advisory Committee granted our request to review all patients receiving EVAR between January 2003 and December 2014. This study was approved by the Institutional Review Board at Dartmouth College. An informed consent exemption was granted, given that data collection occurred without patient identifiers included in a national database. All patients receiving EVAR were reviewed; those undergoing elective repair were analyzed. We compared patient demographics as well as operative and postoperative data, with particular attention to renal function and factors affecting renal function.

Renal function

Patient information captured within the VQI database includes sex, race or ethnicity, preoperative creatinine level, height, and weight; these variables allowed us to estimate glomerular filtration rate with the Chronic Kidney Disease Epidemiology Collaboration equation, the calculation deemed most precise in guiding clinical decision-making, assigned a level 1B recommendation by the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 practice guideline 1.4.3.4.[14,15] Post-operative information about change in renal function contained within the VQI database includes increase in creatinine concentration ≥0.5 mg/dL, which we define as AKI, and new temporary or permanent HD requirements.

End points

The primary end point was long-term mortality. Patient deaths are captured by the VQI database and cross-referenced with the Social Security Death Index. In constructing Fig 1, we arbitrarily selected 100 mL of contrast material as a limiting value, representing the volume administered to image the renal arteries, distal seal zones, and completed implant.

Statistical analysis

Those EVAR patients suffering an AKI or requiring new HD initiation were compared with the unaffected population. Unless otherwise noted, all figures display 95% confidence intervals (CIs). χ^2 tests compared dichotomous variables. Analysis of variance was employed to analyze parametrically distributed means, whereas Kruskal-Wallis one-way analysis of variance evaluated nonparametric data, as determined by histogram evaluation. Kaplan-Meier life tables estimated patient survival, and Cox mortality hazard modeling predicted variables associated with mortality in our cohort. Stepwise multivariable logistic regression in a backward fashion was used to determine associations between patient-, surgeon-, and hospital-level characteristics as well as factors contributing to renal dysfunction. Any variable demonstrating a P value ≥ .2 was deemed nonsignificant; these were removed sequentially beginning with the highest P value. Both the multivariable logistic regression and Cox models were run in multiple combinations to assess surgeon and center as covariates or clustering influences. Model fit was assessed with a Hosmer-

Lemeshow goodness-of-fit test, and area under the receiver operating characteristic curve indicated model discrimination. Stata 13.1 (StataCorp, College Station, Tex) was used for all analyses.

RESULTS

Cohort

Between January 2003 and December 2014, 17,213 patients receiving EVAR were recorded in the VQI database. A total of 462 patients were excluded from analysis, including 422 patients who were receiving HD at the time of intervention or had no record of their preoperative HD status, 40 patients with a functional kidney transplant, and 2276 patients undergoing emergent intervention (Fig 2). Therefore, the overall cohort for analysis included 14,475 patients undergoing elective EVAR. Of these, 3.2% (n = 470) suffered from postoperative renal dysfunction, including 414 (2.9%) patients with AKI and 56 (0.4%) patients with a new HD requirement.

Baseline characteristics

There were several statistically significant baseline differences between patients who developed renal dysfunction after elective EVAR and those who did not (Table I).

Comparing EVAR patients without postoperative renal dysfunction vs those with AKI or new HD, unaffected patients were younger (mean, 73.6 years vs 76.4 and 76.7 years; $P < .001$), were less likely to be female (18.6% vs 24.6% and 37.5%; $P < .001$), and had higher preoperative eGFRs (68.6 mL/min/1.73 m^2 vs 52.3 and 41.7 mL/min/1.73 m^2; $P < .001$). EVAR patients without postoperative renal dysfunction also had a lower prevalence of hypertension (83.6% vs 88.2% and 92.9%; $P < .008$), diabetes (20.2% vs 23.2% and 37.5%; $P = .002$), and congestive heart failure (CHF; 10.9% vs 18.9% and 19.6%; $P < .001$). Other demographic and preoperative comorbidity details appear in Table I.

Survival

Estimated survival at 5 years ± standard error of the mean in the group without renal dysfunction was 77.5% ± 1.0% (Fig 3); notably, 51 patients in this group have 0 days of recorded follow-up and are immediately censored, accounting for the listed number at risk in the no dysfunction group of 13,954 rather than the number diagrammed in Fig 2, 14,005. Patients suffering postoperative AKI demonstrate a 53.5% ± 5.9% 5-year estimated survival, whereas those developing a new HD requirement exhibit 22.8% ± 8.5% 3-year estimated survival; data for the new HD group were not estimable past 3 years.

A Cox proportional hazards model adjusting for preoperative, intraoperative, and postoperative variables found AKI to confer a 2.32 (95% CI, 1.78-3.02; $P < .001$) greater risk of death relative to the no renal dysfunction group, whereas a new HD requirement increased mortality risk by a factor of 4.91 (95% CI, 2.79-8.62; $P < .001$). This model produces a Harrell C statistic value equal to 0.749. The component variables appear in the Supplementary Table (online only).

Preoperative eGFR significantly influenced mortality in our model; the Supplementary Fig (online only) is a Kaplan-Meier plot incorporating only patients with a preoperative eGFR value >60 mL/min/1.73 m^2. Those patients with initially normal renal function who develop an AKI are estimated to survive in significantly lower proportion at 2 years than those who demonstrate no renal dysfunction (77.5% ± 4.8% [95% CI, 75.0%-89.5%] vs 93.4% ± 0.4% [95% CI, 92.6%-94.1%]). Ten patients with a normal preoperative eGFR developed a new HD requirement after EVAR and were not included in this figure as only two survived beyond 1 year.

Renal dysfunction

Factors significantly correlated with postoperative renal dysfunction appear in Table II. AKI and new HD patients were grouped together and compared with those not developing postoperative renal dysfunction.

Several factors independently predict postoperative renal dysfunction, particularly new-onset CHF (odds ratio [OR], 3.50; 95% CI, 1.18-10.38; $P = .02$) and a return to the operating room (OR, 3.26; 95% CI, 1.49-7.13; $P = .003$), whereas a preoperative eGFR ≥60 mL/min/1.73 m^2 was protective (OR, 0.33; 95% CI, 0.21-0.53; $P < .001$; Table II).

This observation led us to consider modifiable risk factors associated with renal dysfunction, specifically intraoperative administration of contrast material. Fig 1 depicts surgeon-specific mean contrast material volumes for elective EVAR. Of the 862 surgeons reporting cases in the VQI database, 447 (51.9%) performing 53.7% of all elective EVARs averaged >100 mL of contrast material per elective EVAR, whereas 404 (47.0%) performing 46.3% of all EVARs averaged <100 mL; 11 (1.3%) performing 0.08% of all EVARs reported no contrast material volume values for any cases.

Preoperative eGFR for patients demonstrating no post-operative renal dysfunction was significantly higher than for those suffering AKI or requiring new HD ($P < .001$; Fig 4, *a*). Mean intraoperative contrast material volume was not significantly different between groups stratified by renal outcome ($P = .13$; Fig 4, *b*).

DISCUSSION

Current vascular surgical practice is associated with AKI in 2.9% of elective EVAR patients and a new HD requirement in 0.4% as captured by the VQI database. These results compare favorably with clinical trial data and outperform early single-center series.[2,3,5,7-11] The 5-year 77.5% survival demonstrated by the study group not suffering renal morbidity aligns with long-term results from the Dutch Randomized Endovascular Aneurysm Management (DREAM) trial.[10] Although it is an infrequent event, those patients unfortunate enough to have development or exacerbation of renal dysfunction after EVAR also demonstrate a distinct survival disadvantage. Decreased estimated life expectancy is associated with more severe renal dysfunction, as hypothesized. AKI correlates with a reduction in estimated 5-year survival by an absolute value of 24% in comparison to the unaffected cohort. One-quarter of the patients in the AKI group are estimated to die within 1 year after EVAR based on Fig 2; this is a novel result not published elsewhere to our knowledge. This study is the

first to quantify the long-term mortality effect associated with post-EVAR renal dysfunction. Post-operative creatinine concentration elevation after elective EVAR is not a benign finding.

More than half of patients initiating HD after EVAR are estimated to die within 6 months (Fig 3). Repair in this population may not prevent aneurysm-related mortality and may precipitate end-stage renal disease (ESRD)-related mortality. AAA treatment guidelines suggest 5.5 cm as the optimal repair threshold for repair among average-risk patients but allow that repair may be indicated in women and other populations at smaller AAA diameters.[16] Conversely, patients deemed at higher risk are not necessarily treated at 5.5 cm; certainly, patients projected to require new post-EVAR HD should be in this latter category. Identifying those patients potentially requiring HD after EVAR and either protecting them with extraordinary perioperative care or forgoing aneurysm repair altogether is necessary to mitigate procedure-related harm. Should patients be unfortunate enough to require HD after EVAR, a conversation with the patient and the patient's family members about goals of care is warranted.

Univariate analysis finds patients with AKI and a new HD requirement to be older, to harbor more preoperative comorbidities, and to present with larger aneurysms in comparison to the no renal dysfunction group. Although these differences are statistically significant because of a large cohort, the clinical implication is unclear. To this point, our Cox proportional hazard model controlled for preoperative, intraoperative, and postoperative variables. This model demonstrated acceptable mortality discrimination with fewer variables independently influencing survival based on a C statistic of 0.749. CHF is well established as an ominous surgical comorbidity.[17,18] It is unsurprising that preoperative CHF increases an EVAR patient's odds of dying 1.79-fold. Normal renal function is protective against mortality after elective EVAR. Several reports have previously explored the association between preoperative renal function and postoperative mortality. Patients with pre-existing ESRD perform poorly after aneurysm repair, carotid surgery, and lower extremity revascularization.[19-21] Particularly in AAA patients, pre-existing ESRD confers 54-fold greater odds of death after EVAR.[8] A potential criticism of our study is that those patients with normal preoperative renal function might still demonstrate statistically indistinguishable estimated survival when stratified by renal outcome; the Supplementary Fig (online only) suggests that this is not the case. Patients with normal preoperative eGFR who develop AKI demonstrate significantly reduced estimated survival. Strategies protecting kidney function should be quality markers in EVAR based on these mortality effects.

A multivariable logistic regression developed from preoperative, intraoperative, and postoperative variables contained within the VQI data set identified independent predictors of post-EVAR renal dysfunction with excellent discrimination based on an area under the receiver operating characteristic curve equal to 0.832. As with mortality, development of CHF after EVAR portends renal dysfunction. Suprarenal fixation is an independent predictor of post-EVAR renal dysfunction in our model. Published data are equivocal, with some groups finding a significant difference between suprarenal and infrarenal endograft fixation's effect on renal morbidity[22] and others not.[11] Additional parameters, including a return to the operating room as well as postoperative vasopressor or transfusion requirement,

are also biologically plausible contributors to renal dysfunction. Longer hospital stay, particularly at a higher care level, although an independent predictor of renal dysfunction, may simply correspond with more severe illness or represent a collinear variable. Grams et al examined 3.6 million veterans undergoing major cardiac, general, otolaryngologic, thoracic, vascular, urologic, and orthopedic surgery during 8 years, including 11.8% who suffered AKI by KDIGO criteria; those developing AKI stayed in the hospital nearly twice as long and were readmitted nearly twice as often as those who maintained normal renal function.[23] Alternatively, prolonged exposure to factors present within the hospital environment itself, but not captured by the VQI database, may explain diminished renal function with longer stay.

Normal preoperative eGFR protects against renal dysfunction. Increases in medullary filtrate viscosity stemming from contrast media contribute to nephron damage during glomerular filtration, leading to renal damage.[24,25] Diminished eGFR suggests that contrast media loads will remain in contact with nephrons for a longer period at greater concentration, further jeopardizing function. Surgeons interpret the oft-repeated admonition to “limit contrast” differently, as evidenced by the 10-fold variation in mean volume administered for elective EVAR depicted by Fig 1. Despite differences in preoperative renal function (Fig 4, *a*), Fig 4, *b* shows that all elective EVAR patients receive the same average volume of contrast material. Interestingly, contrast material volume demonstrated a statistically nonsignificant correlation with renal dysfunction and was removed during the construction of our multivariate model because of a P value > .2, possibly due to lack of a standardized dosing strategy. A metric aimed at guiding administration of contrast material has been found to prevent post-EVAR renal dysfunction in other interventional specialties.

Nyman et al describe the “grams iodine to eGFR ratio” as an a priori estimate predicting safe dosing of contrast material in ST-segment elevation myocardial infarction patients.[26] A 1-to-1 ratio of grams of iodine to eGFR forecasted a 3% frequency of contrast-induced nephropathy with excellent model discrimination down to an eGFR equaling 20 mL/min in nondiabetic patients. Conceptualizing dose of contrast material based on grams of iodine administered, rather than on media volume, as well as eGFR with a body surface area-dependent equation is an important mathematical step. A large series examining cardiac intervention patients developed similar models with unacceptable discrimination when these two concepts were omitted.[27] Infrarenal endovascular AAA repair occurs frequently enough to provide an adequate cohort for the preceding analyses. Evolving process improvement for infrarenal AAA must guide other aortic procedures, particularly complex AAA repair with branched or fenestrated endografts. Imaging of and intervening on aortic branch vessels geometrically increases case complexity and potential harm to the patient. Two recent publications highlight the necessity of proactively addressing renal sequelae.

Sailer et al concluded no relationship between contrast material volume and kidney injury in a multivariable model of AKI after complex EVAR in 157 patients.[28] They showed that 43 of 156 (28%) patients undergoing branch or fenestrated repairs developed AKI with a univariate statistically significant difference in contrast material volumes between the two groups. The event rate in their cohort is too low to support the proposed nine covariate multivariable logistic regression, which is further confounded by more than double the

number of accessory renal arteries covered in the AKI group in comparison to the no AKI group. Importantly, the frequency of AKI demonstrated by this and other work is 9 to 10 times greater than the 2.9% we report in elective EVAR.[28,29] The extrapolated mortality implications are ominous and must be managed to afford patients full benefit from minimally invasive aortic repair technology.

Limitations

As this study derives from an administrative database, patient characteristics are not available in granular detail, limiting our ability to infer outcomes. Our definition of AKI falls short of the widely accepted KDIGO, AKIN (Acute Kidney Injury Network), and RIFLE (risk, injury, failure, loss of kidney function, end-stage renal disease) criteria.[30-32] Each incorporates several other clinical characteristics to define AKI beyond change in baseline creatinine concentration; these variables are not present in the VQI database. Furthermore, a recent meta-analysis suggests under-reporting of renal morbidity after AAA repair.[33] It is also possible that perioperative hydration strategies used for different patients may have had an effect on postoperative renal function after EVAR. Preintervention hydration is essential to preventing contrast-induced nephropathy[34]; these data are not captured by the VQI database. Furthermore, variation in strategies abound. Recent clinical trials do not support the efficacy of sodium bicarbonate and *N*-acetylcysteine over crystalloid in preventing renal morbidity.[35-38] The Prevention of Serious Adverse Events Following Angiography (PRESERVE) trial is a large, multicenter randomized trial that is actively recruiting and aimed at defining the optimal approach to protecting renal function[39]; final data collection is anticipated in March 2016.[40] Regardless, preintervention hydration strategies influence renal dysfunction and are not captured in the VQI database. Aneurysm anatomy is not addressed beyond anteroposterior diameter, a variable with statistically significant univariate difference between the study groups in our study. Although the 7-mm difference in average size implies increased technical challenge, we do not believe this to be a clinically relevant finding. However, more critical anatomic data, including neck and landing zone characteristics as well as accessory renal artery involvement, are not available. Improved preoperative planning with three-dimensional reconstruction[41] and intraoperative adjuncts, like computed tomography angiography fusion imaging[42] and steerable robotic catheters,[43] influence procedural execution. The role for each of these technologies in preventing comorbidity after EVAR is evolving. Finally, follow-up within VQI data sets is a known systematic issue. In the EVAR data set we employed, 47 patients have 0 days of follow-up but are identified in the Social Security Death Index cross-reference as nondeceased. Furthermore, 2215 patients have 1 day of follow-up; 2209 are similarly listed as nondeceased. This issue is currently being addressed by a Patient Safety Organization-wide initiative.

CONCLUSIONS

Renal dysfunction developing after elective EVAR is associated with decreased estimated long-term survival. Protecting renal function with a rational dosing metric for contrast material linked to preoperative eGFR may better guide treatment. Renal outcomes should be a quality metric for elective EVAR.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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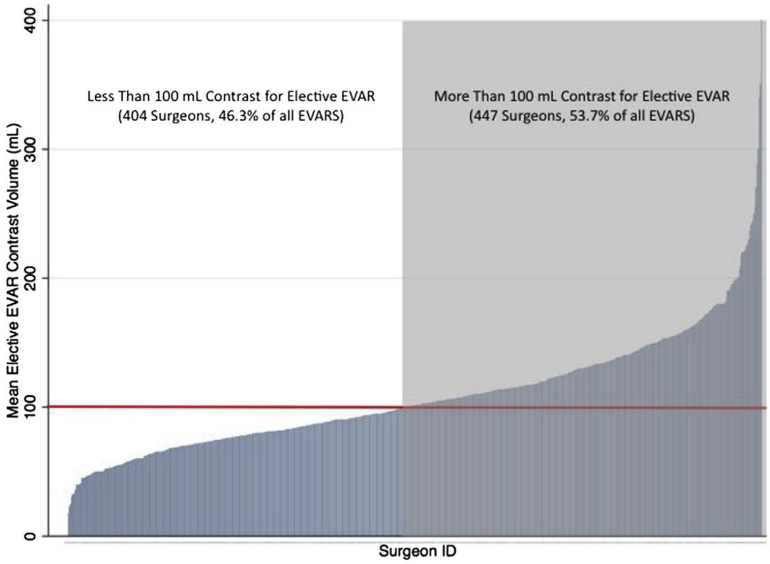

Fig 1.

Mean contrast material volume per surgeon. *EVAR*, Endovascular aneurysm repair.

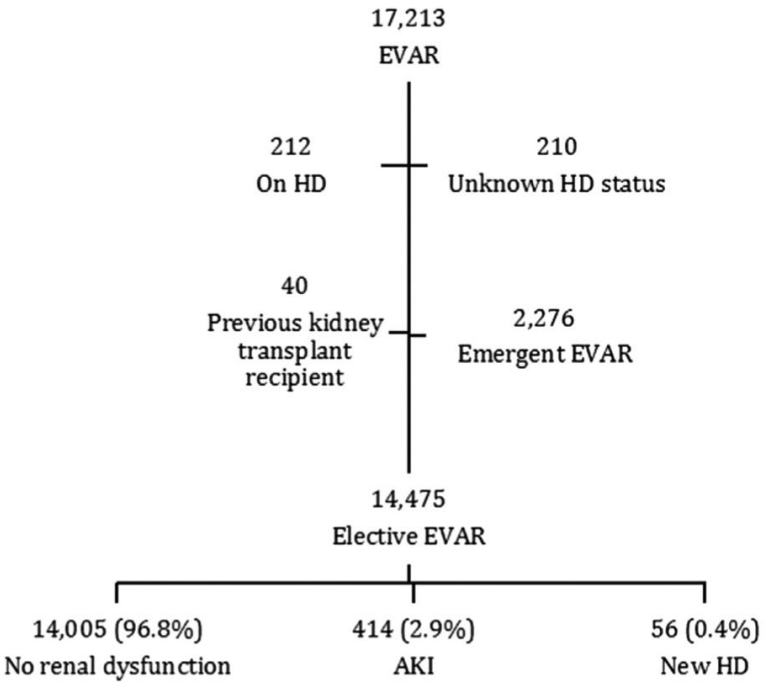

Fig 2.

Patient cohort. *AKI*, Acute kidney injury; *EVAR*, endovascular aneurysm repair; *HD*, hemodialysis.

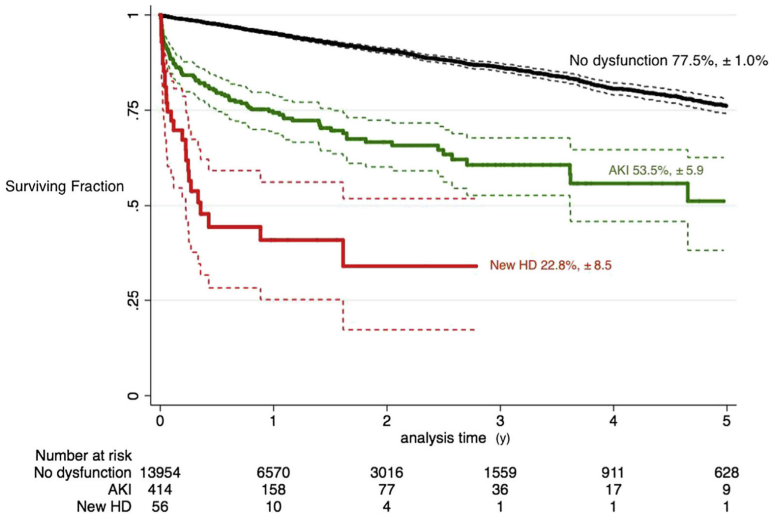

Fig 3.

Actuarial survival stratified by renal outcome. Reported as a 5-year estimate ± standard error of the mean with 95% confidence interval (CI) bounds. *AKI*, Acute kidney injury; *HD*, hemodialysis.

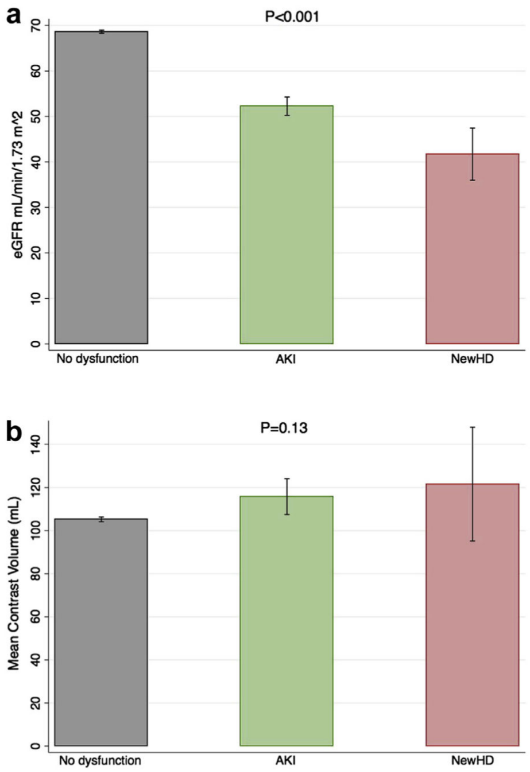

Fig 4.

a, Mean preoperative estimated glomerular filtration rate (*eGFR*) stratified by renal outcome ($P < .001$; *error bars* show 95% confidence interval [CI]). **b,** Mean volume of contrast material administered intraoperatively ($P = .13$). *AKI*, Acute kidney injury; *HD*, hemodialysis.

Table I

Baseline characteristics

Renal outcome	*No dysfunction*	*AKI*	*New HD*	P *value*
No. (%)	14,005 (96.8)	414 (2.9)	56 (0.4)	
Age, years	73.6 (73.4-73.7)	76.4 (75.7-77.2)	76.7 (74.9-78.6)	<.001
Women	18.6	24.6	37.5	<.001
White race	92.7	91.8	91.1	.38
eGFR, mL/min/1.73 m^2	68.6 (68.3-68.9)	52.3 (50.3-54.3)	41.7 (36.1-47.3)	<.001
Hypertension	83.6	88.2	92.9	.008
Diabetes	20.2	23.2	37.5	.002
Tobacco abuse	86.3	85.5	82.1	.61
CAD	29.8	37.0	26.8	.007
Past CABG or PCI	35.0	41.7	37.5	.02
Preoperative statin	69.9	69.6	75.0	.53
CHF	10.9	18.9	19.6	<.001
COPD	31.7	41.6	44.6	<.001
Previous AAA repair	3.6	3.6	5.4	.77
BMI, kg/m^2	28.0 (27.9-28.1)	28.2 (27.6-28.8)	28.3 (26.7-29.9)	.06
AAA maximum AP diameter, mm	55.3 (55.0-55.5)	59.1 (57.7-60.6)	61.7 (57.1-66.2)	<.001

AAA, Abdominal aortic aneurysm; *AKI*, acute kidney injury; *AP*, anteroposterior; *BMI*, body mass index; *CABG*, coronary artery bypass graft; *CAD*, coronary artery disease; *CHF*, congestive heart failure; *COPD*, chronic obstructive pulmonary disease; *eGFR*, estimated glomerular filtration rate; *HD*, hemodialysis; *PCI*, percutaneous coronary intervention.

Categorical variables are presented as percentages. Continuous variables are presented as mean (95% confidence interval [CI]).

Table II

Multivariable logistic regression with factors predicting postoperative renal dysfunction

	OR	*95% CI*	P *value*
New CHF postoperatively	3.50	1.18-10.38	.02
Return to operating room	3.26	1.49-7.13	.003
Postoperative vasopressors	2.68	1.40-5.12	.003
Suprarenal fixation	1.89	1.13-3.14	.02
ACE/ARB prescription postoperatively	1.42	0.98-2.07	.06
Transfusion postoperatively	1.18	1.05-1.33	.004
ICU stay, days	1.11	1.05-1.18	<.001
Length of stay, days	1.01	1.00-1.02	.03
Procedure time, minutes	1.00	1.00-1.01	.001
eGFR ≥60 mL/min/1.73 m^2	0.33	0.21-0.53	<.001

ACE/ARB, Angiotensin-converting enzyme/angiotensin receptor blocker; *CHF*, congestive heart failure; *CI*, confidence interval; *eGFR*, estimated glomerular filtration rate; *ICU*, intensive care unit; *OR*, odds ratio.

Area under receiver operating characteristic curve = 0.832.