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Postoperative renal dysfunction independently predicts late mortality in patients undergoing aortic reconstruction

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Abstract

Objective—Preoperative chronic kidney disease (CKD) has been shown to predict postoperative renal complications and mortality after open aortic surgery; the impact of postoperative renal complications less severe than permanent dialysis are unknown. We evaluated the impact of postoperative renal dysfunction severity on survival using a regional quality improvement registry.

Methods—Patients undergoing intact open aortic reconstruction in the Vascular Study Group of New England registry (2003-2012) were stratified by severity of postoperative renal complications; none, creatinine increase of greater than 0.5 mg/dL (incCr), or any hemodialysis (HD). Predictors of renal dysfunction and impact of renal complications on survival were analyzed using multivariable methods.

Results—We included 2695 patients, of which 65% (n = 1733) underwent open abdominal aortic aneurysm repair, and 35% (n = 962) open aortoiliac reconstruction. At baseline, 15% of patients had preoperative moderate CKD and 1.2% had severe CKD. Postoperative renal complications of incCr occurred in 8.5% of patients, and 1.5% required HD. Multi-variable cumlogit regression identified severe baseline CKD (odds ratio [OR], 15; 95% confidence interval [CI], 6.4-34; $P < .001$, moderate CKD (OR, 2.8; 95% CI, 1.9-3.3; $P < .001$), suprarenal clamp use (OR, 2.2; 95% CI, 1.6-2.9; $P < .001$), perioperative vasopressor requirements (OR, 2.2; 95% CI, 1.6-2.9; $P < .001$), operating time (OR, 1.004 per minute; 95% CI, 1.003-1.006; $P < .001$), and chronic obstructive pulmonary disease (OR, 1.5; 95% CI, 1.2-1.8; $P < .001$) as independent predictors of worsening

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strata of postoperative renal dysfunction. Multivariable logistic regression analysis showed that patient age (OR, 1.06 per year; 95% CI, 1.01-1.1; $P = .01$), baseline chronic obstructive pulmonary disease (OR, 1.6; 95% CI, 1.2-2.2; $P < .01$), incCr (OR, 3.7; 95% CI, 1.8-7.4; $P = .009$), and HD (OR, 4.8; 95% CI, 1.8-12.7); $P = .009$) independently increased 30-day mortality. Risk-adjusted multivariable Cox regression showed that incCr (hazard ratio, 1.8; 95% CI, 1.3-2.6; $P < .001$) and HD (hazard ratio, 4.4; 95% CI, 2.8-6.9; $P < .001$) increased risk of late death independent of a variety of other clinical variables, including baseline CKD. The 5-year survival was lower (log-rank $P < .001$) in patients with incCr (66% ± 4%), and HD (38% ± 10%) compared with those with no renal complications (77% ± 1%).

Conclusions—Increasing severity of postoperative renal dysfunction independently predicts increased risk of late mortality after open aortic surgery. Perioperative measures to decrease renal complications may potentially prolong the survival of patients after open aortic surgery.

Renal complications after aortic reconstruction occur along a spectrum of severity from self limited nonoliguric creatinine elevation to complete renal failure requiring hemodialysis (HD). Acute renal failure after abdominal, cardiac, thoracic, and vascular operative procedures has been shown to significantly contribute to patient morbidity and increased operative mortality.[1,2] Major predictors of postoperative dialysis dependent renal failure after aortic surgery include patient features, in particular antecedent chronic kidney disease (CKD), manner of clinical presentation, and technical conduct of operation.[3-11] Postoperative HD has been shown to contribute to increased operative mortality and decreased late survival in patients undergoing thoracoabdominal aortic aneurysm repair[12-14] as well as cardiac operative procedures.[15-17] Studies evaluating the impact of renal complication after abdominal aortic surgery[3-11] have only focused on either creatinine change or HD as an outcome, have not evaluated both early and long-term impact of postoperative renal complications, or are outdated. The present study was performed to identify clinical and technical predictors of increasing severity of renal complications, and to evaluate the impact of renal complications on both perioperative mortality and long-term survival after open aortic reconstruction for both aneurysmal and occlusive disease. This study was performed using contemporary data from the Vascular Study Group of New England (VSGNE) regional quality registry.

METHODS

Patient population

The VSGNE cohort is a prospectively maintained quality improvement registry that captures key clinical, technical, and perioperative outcomes data for patients undergoing vascular operative procedures. Linkage of the registry with the Social Security Death Index Masterfile allows for accurate mortality and survival analysis. Specific details about the registry and validation methods, included variables, and variable definitions can be found on line at www.vascularweb.org/regionalgroups/vsgne.[18] At the time of this study, the VSGNE registry included data from more than 75 surgeons practicing at a heterogeneous cohort (n = 22) of academic medical centers and private hospitals in the New England region. Participation in the VSGNE quality registry as a patient safety organization and use of deidentified human subject information for outcomes analysis was approved by the

institutional review board at each of the participating institutions. Given the retrospective nature of the study, absence of direct patient participation, and blinded patient data, direct informed consent was not obtained.

The VSGNE registry was queried from 2003 to 2012 to identify all patients undergoing open abdominal aortic aneurysm (AAA) repair or aortoiliac reconstruction for occlusive disease. Specific exclusions were applied to remove confounders associated with renal complications, including aneurysm rupture, unstable patients, those undergoing repair with supravisceral clamp application, and occlusive disease patients who had inflow from the thoracic aorta. The final dataset consisted of 2095 patients with 583 patients treated for occlusive disease and 1512 for aneurysms.

Study design

This retrospective review evaluated the impact of postoperative renal complications after open aortic reconstruction for AAA or occlusive disease. Patients undergoing elective AAA repair or aortoiliac reconstruction were stratified into three groups based on degree of post-operative renal dysfunction as defined by the VSGNE registry: no postoperative change in renal function, increase in creatinine, or dialysis-dependent renal failure (HD). The primary outcome measure was 30-day mortality and late survival. Secondary measures included major postoperative complications.

Variable definitions. A large number of preoperative, procedural, and postoperative data are recorded in the VSGNE registry and were used for this study. Postoperative renal complications are recorded for both the open AAA, and suprainguinal bypass datasets. Renal complications are defined as no change in renal function, increase in creatinine of 0.5 mg/dL or greater (incCr), temporary postoperative HD, and permanent HD. Given the small numbers of patients requiring postoperative HD, the temporary and permanent HD groups were grouped together as any postoperative HD to improve statistical power and achieve meaningful conclusions. Subgroup analysis showed no differences in outcomes between the individual groups and the combined cohort. The estimated glomerular filtration rate (eGFR) was calculated for all patient using the Modification of Diet in Renal Disease equation.[19] Using National Kidney Foundation definitions[20] for CKD class, CKD severity was categorized as normal to mild disease (CKD class 1 or 2; eGFR > 60 mL/min), moderate disease (CKD class 3; eGFR 30-60 mL/min), and severe disease (CKD class 4 or 5; eGFR < 30 mL/min).

Statistical analysis

Continuous data are presented as mean values ± standard deviation and categorical variables are presented as percentage frequencies within the cohort. Univariate analysis was performed by using χ^2 analysis for categorical data, or the Wilcoxon rank-sum test for nonnormally distributed continuous and ordinal variables. Ordinal multinomial logit regression analysis[21] was performed to identify predictors of increasing degree of postoperative renal dysfunction and multivariable logistic regression analysis was performed to identify predictors of 30-day mortality; the resulting odds ratios (ORs), along with 95 percent confidence intervals (CIs) are presented. A Kaplan-Meier product limit estimator

curve was plotted to compare survival functions between patients of differing degrees of postoperative renal dysfunction. The log-rank test for the equality of the survival distribution curves was used to evaluate for differences in survival by degree of renal dysfunction. A multivariable risk adjusted Cox proportional hazards model was used to obtain hazard ratios (HRs), 95% CIs, and *P* values for predictors of late death. Statistical analysis was performed using SAS 9.2 (SAS, Inc, Cary, NC). A 2-sided *P* value of <.05 was considered significant.

RESULTS

We identified 2695 patients who underwent open aortic surgery (n = 1733 with AAA and n = 962 with occlusive disease) during the study period. Postoperative renal morbidity results were notable for no renal complication in 2416 (90%), elevated creatinine in 232 (8.6%), and dialysis in 47 (1.7%). Clinical features which predicted renal complications on univariate analysis are presented in Table I; operative time, suprarenal clamp application, operative blood loss, intraoperative blood transfusion, and vasopressor requirements all strongly predicted renal complications on univariate analysis (Table II). On univariate analysis, renal complications were more common in patients undergoing aneurysm repair with higher rates of elevated creatinine (10% vs 6%; $P = .001$) and any dialysis (2.5% vs 0.4%; $P = .001$).

Postoperative renal dysfunction was associated with increased risk of perioperative mortality (1% no complication vs 9% incCr vs 23% dialysis; $P < .001$; Table III). A significant increase in postoperative morbidity after open aortic surgery was also observed in patients with an increasing degree of renal dysfunction (Table III). Patients with renal complications had a significantly higher median duration of stay (6 days no complications vs 10 days incCr vs 22 days dialysis; $P < .001$) and were less likely to be discharged to home (80% no complications vs 64% incCr vs 29% dialysis; $P < .001$).

Multivariable cumlogit regression analysis identified several independent predictors of renal complications. Baseline severe (OR, 15; 95% CI, 6.4-34; $P < .001$) and moderate CKD (OR, 2.8; 95% CI, 1.9-3.8; $P < .001$) were the strongest predictors of postoperative renal complications. Other independent predictors of renal complications included application of a suprarenal aortic clamp, vasopressor requirement, preoperative chronic obstructive pulmonary disease, and operative time per minute (Table IV). Multivariable logistic regression analysis showed that patient age (OR, 1.06 per year; 95% CI, 1.01-1.1; $P = .01$), baseline chronic obstructive pulmonary disease (OR, 1.6; 95% CI, 1.2-2.2; $P < .01$), incCr (OR, 3.7; 95% CI, 1.8-7.4; $P = .009$), and HD (OR, 4.8; 95% CI, 1.8-12.7; $P = .009$) were associated independently with 30-day mortality in a model risk adjusted for urgency, baseline CKD (moderate and severe), congestive heart failure, hypertension, diabetes mellitus, and coronary artery disease.

Long-term survival and associated Kaplan-Meier life table are depicted in Fig. Both incCr and dialysis-dependent renal failure were associated with reduced 1-year (96 ± 0% no complications vs 84 ± 2% incCr vs 65 ± 6% dialysis; log-rank, $P < .001$) and 5-year (77 ± 1% no complications vs 66 ± 4% incCr vs 38 ± 10% dialysis; log-rank, $P < .001$) survival rates. Cox proportional hazards regression analysis showed that an increased postoperative

creatinine (HR 1.8; 95% CI, 1.3-2.6; $P < .001$) and any postoperative dialysis (HR, 4.4; 95% CI, 2.8-6.9; $P < .001$) increased the risk of late mortality independent of preoperative renal dysfunction (moderate or severe CKD). Other independent predictors of late mortality included age (per year), severe baseline CKD, chronic obstructive pulmonary disease, congestive heart failure, postoperative myocardial infarction, and urgent operations (Table V).

DISCUSSION

Renal complications after operative procedures lead to increased morbidity, mortality, and resource utilization. This observation has been universal across many specialties and procedure types.[1,2] As such, the evaluation of renal complications and its predictors has important value in clinical decision making, operative conduct, clinical management, and long-term outcomes. This is especially true for patients with vascular disease, because acute renal failure rates are highest among patients undergoing vascular operative procedures.[1] In the present study, we evaluated the impact of postoperative renal complication on perioperative and late outcomes after open aortic surgery for AAA and aortoiliac occlusive disease. Unique to this study, outcomes were risk adjusted for baseline CKD severity, which has been reported widely to increase the risk of renal complications. Our study findings were notable for the fact that early mortality and late survival were associated incrementally with any degree of postoperative renal complications, independent of baseline CKD and other predictors of mortality.

We found that progression to any HD after surgery increased 30þ day mortality to 23%. This is within the ranges (20%-77%)[1,4-6,9-11] reported in the literature, however, likely on the lower end owing to the fact that we included patients who required temporary renal replacement therapy into our HD cohort to increase the number of patients with HD to improve statistical power. In a large, multicenter, prospective study, Siddiqui et al[1] reported a mortality rate of 42% at 90 days in patients with postoperative HD. In this large administrative dataset study, the impact of other renal complications such as incCr was not evaluated. More recently, using the 2002 National Inpatient Sample database, Wald et al[9] reported an operative mortality similar to ours of 19% in patients who developed acute renal failure and HD after elective open AAA repair or endovascular aneurysm repair. In that study, acute renal failure independently predicted operative mortality (OR, 11.3; 95% CI, 7.6-16.8; $P = .01$). Two large, single-center reports[10,11] evaluating clinical and technical details also reported that postoperative HD was an independent predictor of 30-day mortality after aortic reconstruction. Our results showed similarly that HD independently increases the risk of postoperative mortality (OR, 4.8; 95% CI, 1.8-12.7; $P = .009$); the large range for the 95% CI in our data suggests a lower power of our study with only 33 patients developing HD. Differences in magnitude of effect may be related to differences in risk adjustment.

An important observation from our data is that even a modest degree of acute tubular necrosis eventuated in a three- to four-fold increased risk of 30-day mortality. A change in creatinine value of 0.5 mg/dL or greater has been shown to impact survival of hospitalized[22,23] patients as well as patients after cardiac operative procedures.[16] In a careful analysis of 9210 patients admitted to a single tertiary care hospital, Chertow et al[22] reported

an independently increased risk of mortality (OR, 6.5; 95% CI, 5.0-8.5; $P < .05$) for medical and surgical patients developing an increase in creatinine of 0.5 mg/dL or greater during admission. Similar to our observation, these investigators also noted that such a modest increase in serum creatinine marginally increased hospital length of stay by 3 to 5 days ($P < .001$).The impact of an increase in creatinine on in-hospital mortality was further validated in a recent meta-analysis[23] of 8 studies including 78,855 medical and surgical patients. Coca et al[23] showed that the relative risk for 30-day mortality was two-fold greater among patients who had an increase in creatinine value from 0.3 to 0.4 mg/L, and that an increase in creatinine of greater than 0.5 mg/dL was associated with a three- to five-fold increase in 30-day mortality. Other smaller, single-center reports have shown that early (within 3 days) postoperative changes in creatinine or renal function have adverse impact on outcomes of after aortic surgery, even if temporary.[7,10,11] In agreement with observations from these cohort studies, our findings also suggest an independent increase in 30-day mortality (OR, 3.7; 95% CI, 1.8-7.4; $P = .009$) among patients with a moderate degree of incCr.

Our study also showed that the impact of renal dysfunction extends beyond the early postoperative period and both incCr (HR, 1.8; 95% CI, 1.3-2.6; $P < .001$) and HD (HR, 4.4; 95% CI, 2.8-6.9; $P < .001$) after aortic surgery were independent predictors of late death. Our modeling suggested that this mortality increase is independent of other major confounders which impact late survival (Table V). Careful review of our Kaplan-Meier survival curves (Fig), however, suggests that for patients with incCr the independent late mortality risk is likely the result of higher early mortality. The survival curves for patients with incCr diverge early and after about 0.5 years run parallel to each other, suggesting that increased procedural mortality drives late survival differences Welten et al[11] evaluated 952 patients undergoing open AAA repair at a single high-volume center for impact of early postoperative changes in renal function. Patients were stratified as having no change, a temporary (<3 days) decrease, or sustained (>3 days) decrease in renal function. Patients with sustained renal dysfunction had a 5-year survival of approximately 60%, which is higher than our reported rate of nearly 40%; however, their cohort of patients with sustained decrease in renal function was composed of patients with both increased creatinine and HD. These author reported that approximately one-half of late mortality was attributed to cardiovascular causes. Our study was unable to capture the cause of late death; however, given the similarities in patient population and comorbid conditions, our patients likely had similar causes of late death. On risk-adjusted hazards analysis, Welten et al[11] observed that only progression to HD independently reduced late survival (HR, 2.6; 95 CI, 1.5-4.6; $P = .05$). Thus, it is consistent that postoperative HD has an independent negative impact on late survival, whereas incCr likely impacts late survival by increased perioperative risks beyond 30 days.

In addition to the significant impact on mortality, recent evidence suggests that postoperative dialysis-dependent renal failure is increasing in incidence.[1] This increase is likely related to increasing age and medical comorbidities of patients undergoing operative procedures.[1] In this large retrospective cohort study using Ontario's universal health care database from 1995 to 2009, the incidence of HD after all elective operative procedures increased three-fold by the later end of the study; this increase in HD incidence was observed mainly among the increased number of patients undergoing cardiac and vascular procedures. In the present

study, we did not analyze secular trends in renal complications because the VSGNE has expanded during the study period to include a significant number of new hospitals and physicians. Furthermore, the practice of vascular surgery has evolved significantly to include endovascular procedures, which has shifted older and higher risk patients to less invasive therapies.

We have found that the renal complication rates in our study occurred at similar frequency to that reported in the literature.[9,10,24-27] Using the National Inpatient Sample dataset from 2002, Wald et al[9] reported that non-HD acute renal failure occurred in 6.7% and HD occurred in 0.9%, similar to our observation of incCr in 8.5% and HD in 1.5%. Ellenberger et al[10] defined incCr identical to our study (>0.5 mg/dL) and reported a, higher incidence of 15%. Welten et al[11] reported that only 57% of patients had no change in renal function; however, 27% experienced a temporary decrease in function, and 17% developed a permanent decrease. In the more recent, prospective, randomized, controlled trials[24,25] evaluating the efficacy of endovascular aneurysm repair compared with open AAA in low- to moderate-risk patients, HD rates after open repair were approximately 1%, which is in keeping with our observation of 1.5%. A lesser degree of renal dysfunction was not evaluated in these trials. Siddiqui et al[1] observed that the risk of HD in patients undergoing cardiac or vascular procedures was 1.2%.

Given that renal complications occur along a spectrum of severity, we chose to perform an ordinal regression analysis to identify independent predictors of increasing severity of renal complications. Results of this analysis identified that baseline CKD using the National Kidney Foundation guidelines was the strongest predictor of any degree of postoperative renal complication. This observation is in keeping with most publications on this topic. The existence of preoperative CKD, elevated creatinine value, or depressed eGFR has been shown most consistently to independently predict postoperative renal failure after aortic surgery.[3-14,26,27] Recent work from our group evaluating the impact of CKD on outcomes of 8701 contemporary aneurysm repairs showed that the risk of acute renal failure and operative mortality correlated with severity of preoperative renal dysfunction.[26] The present study similarly shows that the risk of postoperative renal complication correlates with severity of baseline CKD; patients with severe CKD had a 15-fold greater risk and those with moderate CKD had roughly three times the risk.

Although patient-specific factors are generally acknowledged to increase the risk of renal failure, our data indicate that operative variables under a surgeon’s control are also important features in risk of renal complications. We observed independent risk associated with procedure time, suprarenal clamp application, and perioperative vasopressor requirements. Procedure time depends on many factors, however, and is often prolonged owing to technical challenges or unanticipated intraoperative findings, limited experience, or complications. Furthermore, a longer or more difficult procedure likely impacts hemodynamic stability, volume loss, blood loss, alterations in patient physiology, and perioperative vasopressor needs. As such, our observation of increased renal complications with longer procedure time and greater perioperative vasopressors requirements likely reflects many process, patient, and technical factors. It is, therefore, imperative to perform aortic procedures with operative expediency and efficiency, and to avoid excessive blood turnover or hemodynamic shifts,

which could lead to hemodynamic instability and pressor requirements postoperatively. Although suprarenal clamp application is often necessary, our data suggest an increased risk of renal complication in such patients. Suprarenal clamp application was used in one-quarter of the patients in our study. This is within expectations and similar rates have been reported by others.[10,11,27-29] Renal ischemia times of less than 40 to 60 minutes have been reported as safe limits,[10-12,28,29] and clamp times in our cohort were well below this threshold at 18 minutes in patients who developed incCr and 30 minutes in patients who developed HD. This suggests that renal ischemia time in conjunction with other factors such as atheroembolism, intrinsic kidney disease, concomitant renal artery stenosis, use or lack thereof of renal protective strategies, or other unknown factors likely increased the renal complication risk associated with suprarenal clamp. A limitation of our study is that the VSGNE dataset does not capture anatomic details that help to govern the conduct of aortic reconstruction, such as quality of anticipated clamp site, the presence and severity of concomitant renal artery disease, and whether renal artery reconstruction was performed.

Our study has several other limitations related to the dataset from which the data were drawn. First is that the study is retrospective in nature and therefore subject to systematic error. Given the self-reported completion of outcomes data, the data are subject to selection bias, which may have resulted in actual renal complication rates higher than reported herein. Despite this, our study showed strong statistical associations between exposure and outcome measures. Misclassification of patients with renal complication into the group without renal complications would result in a regression towards the mean; therefore, in the absence of this our statistical analysis would have shown even stronger statistical associations.

The definition of acute kidney injury and renal complications in the literature is variable and several renal injury classification tools have been developed to establish consistency,[30,31] however, are not used consistently and were not used in our dataset. Neither of these renal complication scores consider glomerular filtration, a more accurate measure of renal function. As such there is controversy in defining postoperative renal dysfunction with the exception of HD, which is universally accepted. In the present study, we defined renal injury according to the limitations imposed by the VSGNE dataset. Based on availability of clinical features and laboratory data, we were able to calculate preoperative eGFR to classify patient risk. We were unable to assess postoperative renal function because these data are not available in the dataset; however, they would have helped to define more accurately any postoperative renal complications. The outcomes registered in the VSGNE are reflective of immediate postoperative outcomes; therefore, patients who developed renal complications in a delayed manner were misclassified. Furthermore, patients whose creatinine returned to baseline could not be identified. Despite these limitations, our findings suggest that renal complications that occur postoperatively after aortic surgery is associated with increased 30-day mortality and has a lasting impact on late survival.

CONCLUSIONS

Despite limitations in study design and assessment of renal injury, our study has identified key predictors of renal complications in patients after aortic surgery. We have also shown that an increasing degree of postoperative renal dysfunction correlates with operative

mortality and decreased long-term survival. These findings can be used in clinical decision making and to enact changes in operative conduct, and patient care after aortic reconstruction to help reduce the incidence of renal complications.

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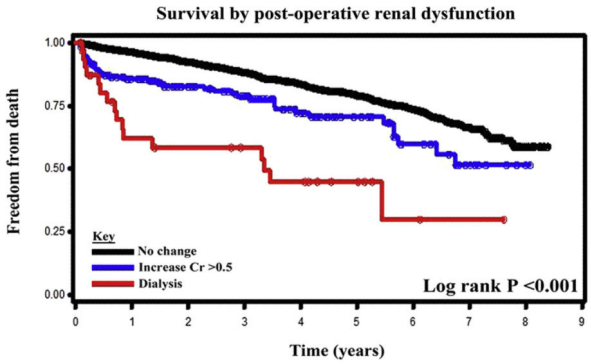

YEAR:	0	1	2	3	4	5
No renal complication						
Survival (%)	100	96	91	87	82	77
Number at risk	2062	1517	1107	869	668	485
Standard error (%)	0	0	1	1	1	1
Increase in creatinine > 0.5 mg/dL						
Survival (%)	100	84	80	75	70	66
Number at risk	229	161	115	96	66	41
Standard error (%)	0	2	3	3	4	4
Any post-operative dialysis						
Survival (%)	100	65	58	54	47	38
Number at risk	47	26	22	16	14	4
Standard error (%)	0	6	7	8	8	10

Fig.

Survival. Kaplan-Meier curves plotting the fraction of the study population alive over time for patients with no postoperative renal dysfunction, increase in creatinine >0.5 mg/dL, or dialysis dependent renal failure. The standard error of the curve representing postoperative hemodialysis (HD) is greater than 10% beyond 5 years, and may be an unreliable estimate of survival beyond that time point.

Table I

Clinical and demographic features

	No renal complication (n = 2416), %	*Creatinine increase >0.5 mg/dL (n = 232), %*	*Any dialysis (n = 47), %*	**P** *value*
Age, years	68 ± 9	70 ± 9	71 ± 7	<.001
Male gender	68	75	64	.06
HTN	82	90	90	.009
Diabetes	18	21	23	.03
CAD	29	33	36	.2
CHF	7	12	19	.003
Chronic obstructive pulmonary disease	34	43	45	.005
CKD				
Moderate (eGFR = 30-60 mL/min)	13	32	31	<.001
Severe (eGFR < 30 mL/min)	0	5	13	<.001
Urgent	13	14	23	.06
Prior aortic surgery	4	6	5	.3
Family history of aneurysm	16	10	20	.08

CAD, Coronary artery disease; *CHF*, congestive heart failure; *CKD*, chronic kidney disease; *eGFR*, estimated glomerular filtration rate; *HTN*, hypertension.

Table II

Procedural

Variable	*No renal complication (n = 2416), %*	*Creatinine increase >0.5 mg/dL (n = 232), %*	*Any dialysis (n = 47), %*	**P** *value*
Operative time, minutes	220 ± 90	250 ± 100	320 ± 130	<.001
Suprarenal clamp	26	46	59	<.001
Renal ischemia (median [IQR]), minutes	0 [0-7]	12 [0-27]	26 [1-50]	<.001
Blood loss, mL	1000 ± 900	1600 ± 1400	2700 ± 2600	<.001
Blood transfusion	16	27	60	<.001
Vasopressors	29	48	77	<.001

IQR, Interquartile range.

Table III

Perioperative outcomes

Outcome	*No renal complication (n = 2416), %*	*Creatinine increase >0.5 mg/dL (n = 232), %*	*Any dialysis (n = 47), %*	P *value*
Death	1	9	23	<.001
Myocardial infarction	4	12	30	<.001
Respiratory complications	7	32	66	<.001
Wound complications	3	11	17	<.001
Length of stay (median [IQR]), days	6 [3-9]	10 [5-15]	22 [6-38]	<.001
Discharge to home	80	64	29	<.001

IQR, Interquartile range.

Table IV

Independent predictors of renal complications*

Outcome	*HR*	*95% CI*	P *value*
Severe CKD	15	6.4-34	<.001
Moderate CKD	2.8	1.9-3.8	<.001
Suprarenal clamp	2.2	1.6-2.9	<.001
Chronic obstructive pulmonary disease	1.5	1.2-1.8	.02
Procedure time, minutes	1.004	1.003-1.006	<.001
Vasopressor requirement	2.2	1.6-2.9	<.001

CI, Confidence interval; *CKD*, chronic kidney disease; *HR*, hazard ratio.

*Multivariable cumlogit regression model for increase in creatinine or postoperative dialysis.

Table V

Independent predictors of late mortality*

Outcome	*HR*	*95% CI*	P *value*
Increased creatinine >0.5 mg/dL	1.8	1.3-2.6	<.001
Any postoperative dialysis	4.4	2.8-6.9	<.001
Severe CKD	2.1	1.1-3.9	.03
Age (per year)	1.07	1.05-1.08	<.001
Urgent operation	1.7	1.4-2.1	<.001
Chronic obstructive pulmonary disease	1.5	1.2-1.8	<.001
Postoperative MI	1.7	1.2-2.4	<.001
CHF	1.5	1.2-1.8	<.001

CHF, Any degree of congestive heart failure; *CI*, confidence interval; *CKD*, chronic kidney disease; *HR*, hazard ratio; *MI*, myocardial infarction.

*Model risk adjusted for gender, moderate CKD, postoperative MI, and gender.