Acute kidney injury after open and endovascular elective repair for infrarenal abdominal aortic aneurysms

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Objective: **The aim of this study was to evaluate the incidence of acute kidney injury (AKI) after open and endovascular abdominal aortic aneurysm repair according to the Aneurysm Renal Injury Score classification.**
Methods: **We retrospectively evaluated 431 patients undergoing elective open aortic repair (OAR; n = 285) or endovascular repair (n = 146) for infrarenal aortic aneurysm. All data regarding preoperative and postoperative serum creatinine concentrations and postoperative outcomes were assessed. Univariate and multivariate logistic regression models investigated the association between AKI and different risk factors and complications.**
Results: **The incidence of AKI was significantly higher after OAR (26.3% vs 5.5%; $P < .001$). A significant share of patients who experienced AKI were restored to preoperative renal function at discharge (62.5% vs 77.5% in the endovascular and OAR groups, respectively; $P = .37$). Preoperative serum creatinine concentration was significantly higher in those patients who further developed AKI (1.25 vs 1.04 mg/dL; $P < .001$). At the multivariate analysis, AKI was significantly associated with current smoking (odds ratio [OR], 2.05; 95% confidence interval [CI], 1.19-3.52; $P = .01$), hypertension (OR, 2.46; 95% CI, 1.21-4.3; $P = .01$), chronic renal disease (OR, 2.53; 95% CI, 1.42-4.53; $P < .001$), OAR (OR, 7.3; 95% CI, 3.25-16.42; $P < .001$), and arrhythmias (OR, 3.16; 95% CI, 1.09-9.13; $P = .03$). AKI stage did not affect postoperative outcomes, except for a longer hospital stay in patients in stage 2 and stage 3 compared with stage 1.**
Conclusions: **AKI is a common but often reversible complication, especially after OAR. There is an urgent need of a common classification for AKI after aortic surgery. New diagnostic markers for AKI should be evaluated in large-scale studies to assess their reliability. (J Vasc Surg 2016;64:928-33.)**

Abdominal aortic aneurysm (AAA) is a life-threatening disease that requires surgical repair when its size is >5.5 cm or it is growing rapidly (>1 cm/y).[1] Endovascular aneurysm repair (EVAR) and open aortic repair (OAR) are the current available options. Whereas EVAR offers superior early advantages, a recent meta-analysis concluded that survival is similar after 2 years.[2] One of the most common postoperative complications after both treatments is acute kidney injury (AKI). A real incidence of AKI after aortic surgery is difficult to evaluate as many different classifications for AKI have been proposed. Two similar staging systems have been more widely adopted: the Acute Kidney Injury Network (AKIN)[3] and Risk, Injury, Failure, Loss, End-stage (RIFLE).[4] Recently, Bang et al[5] compared the incidence of AKI according to these two classification systems and concluded that AKIN showed a better prediction of mortality after any kind of AAA repair. Moreover, Twine and Boyle[6] proposed a tailor-made classification of AKI after abdominal aortic surgery, the Aneurysm Renal Injury Score (ARISe), derived from the RIFLE criteria. These classifications are summarized in Table I. Finally, new urinary markers have been recently identified,[7,8] but their application is not widespread in routine practice yet. In this paper, we aim to evaluate the incidence of AKI according to the ARISe classification in a large series of patients who underwent both OAR and EVAR.

METHODS

All patients who underwent OAR and EVAR in our institution from January 2009 until October 2015 were prospectively included in an Excel database. Data regarding demographics, aneurysm characteristics, operative details, and postoperative complications were noted. Inclusion criteria for aneurysm repair were AAA >5.5 cm or >4 cm rapidly growing (>1 cm/y) and iliac artery aneurysms >3 cm requiring aortobi-iliac or bifemoral open or endovascular repair. Exclusion criteria were symptomatic or ruptured AAA, need for suprarenal clamping, branched or fenestrated or chimney endografts, concomitant renal artery interventions (ie, reimplantation, angioplasty), and minor accessory renal artery ligations. In our division, EVAR is usually preferred in the following conditions: high risk for surgery (ie, severe cardiac or pulmonary disease; concomitant cancer needing surgery, chemotherapy, or radiotherapy), previous abdominal surgery, need for

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Table I. Comparison of acute kidney injury (AKI) staging systems

Stage	*AKIN*[3]	*RIFLE*[4]	*ARISe*[6]
1	SCr increase >0.3 mg/dL or 150%-200% from baseline in <48 hours	SCr increase 150%-200% from baseline in <7 days	SCr increase >0.3 mg/dL or <50% from baseline in <48 hours
2	SCr increase 200%-300% from baseline in <48 hours	SCr increase 200%-300% from baseline in <7 days	SCr increase 50%-99% from baseline in <7 days
3	SCr increase >300% from baseline in <48 hours or >4 mg/dL with an acute increase of at least 0.5 mg/dL	SCr increase >300% from baseline in <7 days or >4 mg/dL with an acute increase of at least 0.5 mg/dL	SCr increase >100% from baseline in <7 days
4		Need for renal replacement for >4 weeks	Need for temporary renal replacement
5		Need for renal replacement for >3 months	Need for permanent renal replacement

AKIN, Acute Kidney Injury Network; *ARISe,* Aneurysm Renal Injury Score; *RIFLE,* Risk, Injury, Failure, Loss, End-stage; *SCr,* serum creatinine.

prolonged dual antiplatelet therapy due to recent myocardial revascularization, and patient's choice. All patients underwent routine blood tests, including determination of serum creatinine (SCr) concentration, on the day before surgery, and this parameter was considered the basal SCr value. We then considered the highest SCr value in the first 7 postoperative days or the requirement for renal replacement therapy, according to the ARISe classification,[6] and patients were divided into the five categories of this staging system. Urine output was not available in some patients, so we decided not to consider this parameter. In those patients who suffered from any stage of AKI, we reported the last SCr value available before the discharge to evaluate the reversibility of AKI. All subjects gave informed consent for the intervention. Data were collected as part of routine service evaluation and no patient-identifiable data are presented, so a local ethical committee approval was not deemed necessary for this retrospective study. The primary study end point was the incidence of AKI according to the ARISe classification. The secondary end points included the impact of AKI on postoperative outcomes and mortality, the restoration rate at the preoperative renal function after AKI, and the influence of pre-existing comorbidities on the development of AKI.

Statistical analysis. Descriptive statistics were performed using frequencies and percentages for qualitative variables, means with standard deviation, and minimum-maximum values for quantitative variables. The patients' characteristics were analyzed by the χ^2 test (or Fisher exact test as appropriate) for categorical variables and by the Student *t*-test for continuous ones, after normality distribution assessment by Shapiro-Wilk test. The main outcome was the postoperative occurrence of AKI (dependent variable); its potential association with different risk factors and complications (independent variables) was investigated by univariate and multivariate logistic regression models. The covariates included in the final multivariate model were selected through the Hosmer and Lemeshow procedure by inserting variables with a univariate P value $< .25$ as the main criterion, with age and gender as potential confounders. Results are expressed as odds ratios (ORs) with 95% confidence intervals (CIs), and a two-tailed P value $\leq .05$ was considered significant for all analyses.

Table II. Demographics and pre-existing comorbidities and drugs

	All	*EVAR*	*OAR*	P *value*
Male sex	405 (94)	136 (93)	269 (94)	.35
Age, years	73.1 ± 7.2	76.7 ± 6.9	71.3 ± 6.7	**<.001**
Current smoking	136 (32)	36 (25)	100 (35)	**.03**
History of smoking	236 (55)	78 (53)	158 (55)	.69
Diabetes	74 (17)	22 (15)	52 (18)	.41
Hypertension	323 (75)	112 (77)	211 (74)	.54
Dyslipidemia	160 (37)	51 (35)	109 (38)	.50
CAD	112 (26)	40 (27)	72 (25)	.63
SCr, mg/dL	1.1 ± 0.43	1.09 ± 0.4	1.1 ± 0.4	1
COPD	118 (27)	43 (29)	75 (26)	.49
History of cancer	77 (18)	39 (27)	38 (13)	**<.01**
Antithrombotic agents	331 (77)	116 (79)	215 (76)	.38
Statins	198 (46)	64 (44)	134 (47)	.53
Beta-blockers	150 (35)	40 (27)	110 (39)	**.02**
ACE inhibitors/ sartans	255 (59)	96 (66)	159 (56)	**.05**
Calcium channel blockers	118 (27)	31 (21)	87 (31)	**.04**

ACE, Angiotensin-converting enzyme; *CAD,* coronary artery disease; *COPD,* chronic obstructive pulmonary disease; *EVAR,* endovascular aneurysm repair; *OAR,* open aortic repair; *SCr,* serum creatinine.
Categorical variables are presented as number (%). Continuous variables are presented as mean ± standard deviation. Boldface entries indicate statistically significant values.

Statistical analyses were performed with Stata/MP 13 statistical software (Stata Corp, College Station, Tex).

RESULTS

In the study period, 431 patients met the inclusion criteria. Of these, 285 (66%) underwent OAR and 146 (34%) EVAR. Demographics and preoperative data are summarized in Table II. EVAR patients were older and less frequently current smokers. Patients with history of cancer were rather treated with stent grafts; calcium channel blockers and beta-blockers were more commonly used by OAR patients, whereas angiotensin-converting enzyme inhibitors/sartans were more commonly used by EVAR patients. Interestingly, preoperative SCr concentration was almost identical in both groups. Mortality within 30 days did not significantly differ between EVAR and

Table III. Postoperative outcomes

	All	*EVAR*	*OAR*	P *value*
Thirty-day mortality	6 (1.4)	1 (0.7)	5 (1.7)	.37
POD	7.5 ± 6.6	4.3 ± 2.5	9.2 ± 7.4	**<.001**
AKI	83 (19.3)	8 (5.5)	75 (26.3)	**<.001**
AKI stage (n = 83)				
Stage 1	60 (72.3)	8 (100)	52 (69.3)	.183
Stage 2	20 (24.1)		20 (26.7)	
Stage 3	3 (3.6)		3 (4)	
AKI restoration at discharge	60/79[a] (75.9)	5/8 (62.5)	55/71[a] (77.5)	.37
MI	3 (0.7)	1 (0.7)	2 (0.7)	.98
Arrhythmias	20 (4.6)	3 (2)	17 (6)	.07
Stroke	2 (0.5)	—	2 (0.7)	.31
Pneumonia	46 (10.7)	3 (2)	43 (15.1)	**<.001**

AKI, Acute kidney injury; *EVAR,* endovascular aneurysm repair; *MI,* myocardial infarction; *OAR,* open aortic repair; *POD,* postoperative day.
Categorical variables are presented as number (%). Continuous variables are presented as mean ± standard deviation. Boldface entries indicate statistically significant values.
[a]Four in-hospital deaths were excluded.

Table IV. Comparison of preoperative and postoperative serum creatinine (*SCr*) concentration according to the onset of acute kidney injury (*AKI*)

	All (N = 431)	*No AKI (n = 348)*	*AKI (n = 83)*	P
Preoperative SCr, mg/dL	1.08 ± 0.41 (0.56-4.57)	1.04 ± 0.35	1.25 ± 0.57	**<.001**
Postoperative SCr peak, mg/dL	1.26 ± 0.62 (0.55-5.09)	1.06 ± 0.37	2.09 ± 0.74	**<.001**

Boldface entries indicate statistically significant values.

OAR patients (0.7% vs 1.7%; $P = .37$). Postoperative days were significantly reduced after EVAR (4 vs 9; $P < .001$). AKI was much more common after OAR (26.3% vs 5.5%; $P < .001$), with a prevalence of stage 1 in both groups (100% vs 69% in EVAR and OAR, respectively). No patients in the OAR group required temporary or permanent renal replacement therapy (stage 4 and stage 5 of the ARISe classification). We then evaluated SCr values at discharge only in patients who developed AKI, after exclusion of those who died within 30 days (n = 4, all in the OAR group). Both groups had a high proportion of restoration to preoperative SCr concentration at discharge (62.5% vs 77.5% in the EVAR and OAR groups, respectively; $P = .37$). Table III summarizes these postoperative outcomes. We then analyzed renal outcomes in detail and divided the study population into two groups according to the development of AKI (Table IV), and we found that preoperative SCr concentration was significantly higher in those patients who further developed AKI (1.25 vs 1.04 mg/dL; $P < .001$). We then performed a univariate analysis comparing patients with AKI (AKI+, n = 83) vs patients without AKI (n = 348; Table V). Those who developed AKI were more frequently current smokers as well as affected by hypertension and chronic renal disease (CRD). AKI+ patients had longer hospital stay (13.6 vs 9 days; $P < .001$) and higher rate of early mortality (4.8% vs 0.6%; $P = .01$), arrhythmias (12% vs 2.9%; $P < .001$), and pneumonia (21.7% vs 8%; $P < .001$). We then tried to evaluate the impact of AKI stage by comparing stage 1 (low AKI, n = 60) vs stage 2 and stage 3 (high AKI, n = 23). However, there were no significant differences between these two groups, besides a longer hospital stay in the second one (18.1 vs 11.9 days; $P = .01$). These data are summarized in the Supplementary Table (online only). Results from multivariate analysis are presented in Table VI. Current smoking (OR, 2.05; 95% CI, 1.19-3.52; $P = .01$), hypertension (OR, 2.46; 95% CI, 1.21-4.3; $P = .01$), CRD (OR, 2.53; 95% CI, 1.42-4.53; $P < .001$), OAR (OR, 7.3; 95% CI, 3.25-16.42; $P < .001$), and arrhythmias (OR, 3.16; 95% CI, 1.09-9.13; $P = .03$) were significantly associated with an increased risk for development of AKI. Again, the multivariate analysis showed no significant differences between low and high AKI, as defined before, except for a longer hospital stay in the second group (Table VII).

DISCUSSION

In our prospective cohort of patients with unruptured infrarenal AAA, the incidence of AKI was significantly higher after OAR than after EVAR (26.3% vs 5.5%). Interestingly, most patients who developed AKI had a restoration to their preoperative renal function at discharge in both groups (77.5% vs 62.5%, respectively). The higher incidence of AKI in the OAR group is comparable with some data from the literature,[9] even though a recent meta-analysis[2] showed no statistical differences between the two treatments. However, a comparison among different studies reporting the incidence of AKI after aortic repair is always affected by a wide heterogeneity in the use of definitions and classifications.[10] There is a lack of

Table V. Univariate analysis

	AKI (n = 83)	*No AKI (n = 348)*	P
Age, years	72.8 ± 6.6	73.2 ± 7.4	.65
Male sex	96.4	93.4	.3
Current smoking	43.4	28.7	**.01**
History of smoking	53	55.2	.72
Diabetes	19.3	16.7	.57
Hypertension	85.5	72.4	**.01**
Dyslipidemia	41	36.2	.42
CAD	25.3	26.1	.87
CRD	38.6	19.2	**<.001**
COPD	34.9	25.6	.09
History of cancer	14.5	18.7	.37
Antithrombotic agents	73.5	77.8	.4
Statins	45.8	46	.97
Beta-blockers	31.3	35.7	.45
ACE inhibitors/sartans	59	59.2	.98
Calcium channel blockers	33.7	25.9	.15
POD	13.6 ± 1.1	9 ± 0.3	**<.001**
Thirty-day mortality	4.8	0.6	**.01**
Mortality, follow-up	6.1	3.1	.2
MI	1.2	0.6	.47
Arrhythmias	12	2.9	**<.001**
Pneumonia	21.7	8	**<.001**

ACE, Angiotensin-converting enzyme; *AKI,* acute kidney injury; *CAD,* coronary artery disease; *COPD,* chronic obstructive pulmonary disease; *CRD,* chronic renal disease; *MI,* myocardial infarction; *POD,* postoperative day.
Categorical variables are presented as percentages. Continuous variables are presented as mean ± standard deviation. Boldface entries indicate statistically significant values.

Table VI. Multivariate analysis

	OR	*95% CI*	P
Male sex	0.56	0.15-2.07	.38
Age	1.02	0.98-1.06	.36
Smoking	**2.05**	1.19-3.52	**.01**
Hypertension	**2.46**	1.21-4.99	**.01**
CRD	**2.53**	1.42-4.53	**<.01**
COPD	1.74	0.99-3.07	.05
OAR	**7.3**	3.25-16.42	**<.001**
POD	1.01	0.97-1.05	.71
Arrhythmias	**3.16**	1.09-9.13	**.03**
Pneumonia	1.98	0.89-4.41	.09

CI, Confidence interval; *COPD,* chronic obstructive pulmonary disease; *CRD,* chronic renal disease; *OAR,* open aortic repair; *OR,* odds ratio; *POD,* postoperative day.
Boldface entries indicate statistically significant values.

consensus on which parameters should be taken into account and how long clinicians should monitor renal function after surgical treatment. In general, the two most commonly used classification systems are the AKIN[3] and the RIFLE[4] criteria. They both use SCr concentration and urine output modifications, but AKIN considers a smaller change of SCr concentration during a shorter time window. Bang et al[5] compared these two classifications to assess their ability to predict the risk of mortality after both endovascular and open repair for infrarenal AAA, and their results evidenced that AKIN criteria better

Table VII. Multivariate analysis according to acute kidney injury (AKI) stage (high/low)

	OR	*95% CI*	P
Age	0.99	0.91-1.07	.76
CRD	0.33	0.09-1.17	.09
POD	**1.07**	1.01-1.13	**.03**
Mortality at follow-up	5.86	0.85-40.60	.07
Antiplatelet	0.31	0.09-1.03	.06
ACE inhibitors/sartans	3.13	0.93-10.60	.07
Calcium channel blockers	2.86	0.85-9.62	.09

ACE, Angiotensin-converting enzyme; *CI,* confidence interval; *CRD,* chronic renal disease; *OR,* odds ratio; *POD,* postoperative day.
Boldface entries indicate statistically significant values.

correlate with early mortality. However, some authors use other parameters to estimate AKI (ie, estimated glomerular filtrate rate)[11,12]; moreover, new markers have been evaluated in aortic surgery, such as urinary cystatin C[8,11] and urinary liver-type fatty acid-binding protein,[7] which both appear to be promising tools, although they are still not completely validated for routine practice.

We decided to apply the classification recently proposed by Twine and Boyle (ARISe classification),[6] which is tailored to abdominal aortic repair (both endovascular and open). In accordance with that, a small rise in SCr levels (ie, 0.3 mg/dL or <50% of baseline) during the first postoperative 48 hours identifies stage 1; 50% to 99% and >100% rise within 7 days correspond to stage 2 and stage 3, respectively; last, the need for temporary or permanent renal replacement therapy refers to stage 4 and stage 5, respectively. This probably explains the relatively high rate of AKI in our cohort of OAR patients (26%), with a high prevalence of stage 1 (100% for EVAR and 69% for OAR), as it considers a very small rise in SCr concentration that can happen even in a normal postoperative recovery. For example, if we applied the RIFLE criteria as originally described by Bellomo et al,[4] not all the patients in stage 1 (according to the ARISe classification) would be identified as AKI+, as the first stage of this classification ("risk") considers a 1.5-fold (ie, >50%) increase in SCr concentration. Having said that, the overall incidence of AKI would have been 5.3%, which is in line with data reported by Wald et al (ie, 6.7%)[13] but considerably lower if compared with the study of Tallgren et al (22%).[11] Furthermore, most patients were restored to preoperative SCr levels at discharge, which means that although it is frequent, AKI seems to be a rapidly reversible process. Specifically, this return to basal renal function was markedly evident in patients in stage 1 (85%). Consequently, we can suppose that the ARISe classification, albeit tailored to aortic surgery, probably overestimates the real clinical impact of AKI. Unfortunately, we did not report long-term renal outcomes as this was a retrospective analysis of a prospectively collected database, the main aim of which was not to establish renal outcomes after EVAR and OAR. Therefore, because we could not collect SCr values after discharge in most patients, we did not include an analysis of long-term impact of AKI.

Another interesting finding of this study is that overall, higher preoperative SCr levels correspond to a higher risk of postoperative AKI. We could not perform a separate analysis for OAR and EVAR groups because of the small number of patients with AKI in the EVAR group (n = 8). Parmer et al[14] evaluated AKI after both EVAR and OAR in patients with CRD and found that there were no differences between the two treatments, even if they considered 1.5 mg/dL as the threshold for CRD, whereas we use 1.2 mg/dL. Wald et al[13] also found a five-fold increased risk of AKI after OAR and EVAR in patients with pre-existing CRD. At the univariate analysis, AKI was associated with longer hospital stay, higher mortality rate, arrhythmias, and pneumonia. Furthermore, smoking, hypertension, and CRD were significant predictors of AKI. At the multivariate analysis, most of these associations remained significant. Surprisingly, age did not affect the incidence of AKI. This is in contrast with the results reported by Tallgren et al,[11] who found that AKI+ patients were significantly older than those who did not develop this complication. We observed a higher mortality rate in AKI+ patients (4.8% vs 0.6%; $P = .01$). Four of five patients who died after OAR had AKI, whereas the only death in the EVAR group was not associated with AKI. Overall, mortality was not statistically different after the two treatments (0.7% vs 1.7% after EVAR and OAR, respectively; $P = .37$). Postoperative AKI is generally a risk factor for mortality after different kinds of surgery.[15,16] Regarding abdominal aortic surgery, Wald et al[13] already highlighted the higher risk of mortality (after both EVAR and OAR) in the presence of AKI (OR, 11.3%). Nevertheless, we could not include early mortality in the multivariate analysis because of the small number of cases in both groups. In agreement with Tallgren et al,[11] arrhythmias were strictly associated with AKI; this could be mainly due to a common etiology, ie, a status of hypovolemia, especially after OAR, which could be one of the causes of both pathologic processes. Conversely, there is not a clear explanation for the association between AKI and pneumonia. We defined this complication as a pulmonary consolidation on radiography with positive bronchoaspirate. Pneumonia more often occurred after OAR,[17] mostly in those patients requiring longer invasive ventilation. In some of these patients, a renal impairment could be included in a wider multiorgan failure, which can partly explain the concomitance of these two complications. Finally, we tried to estimate the impact of AKI stage on different outcomes, but no significant differences occurred between stage 1 and stages 2 and 3, except for a longer hospital stay in the latter group.

This study has several limitations. This is a retrospective analysis of a prospective cohort of patients; therefore, the main purpose of our data collection was not to assess renal outcomes after EVAR or OAR. Second, we used SCr concentration only as a parameter to define AKI, whereas in the literature, some other markers appeared to be more appropriate.[7,8,11,12] Notwithstanding this, SCr is a routine blood test that would be requested in any case after aortic surgery, making it much cheaper than other markers. However, further prospective studies purposely designed for AKI evaluation should investigate and compare different markers of renal injury. Another limitation of the study is the low number of AKI+ patients in the EVAR group, which negatively affects a statistical comparison between the two types of intervention. Finally, many potentially interesting data that have been evaluated in previous works are lacking: intensive care unit stays,[18] postoperative hemodynamic parameters,[11] intraoperative and perioperative fluid management,[19,20] contrast agent use,[5] blood losses,[5] impact of suprarenal fixation,[21] and long-term assessment of renal function.[22] A detailed report of all these parameters would have added many points of consideration to the discussion.

CONCLUSIONS

AKI is a common but transient complication, especially after open abdominal aortic surgery and in patients with pre-existing renal impairment. This complication is usually associated with other postoperative adverse outcomes and leads to higher rates of mortality. There is an urgent need of consensus to overcome the plethora of definitions and classifications of AKI, especially in abdominal aortic surgery, because the current systems may not properly reflect the actual burden of this complication. Further studies must assess which parameters should be considered reliable to evaluate the impact of AKI after any kind of abdominal aortic repair.

AUTHOR CONTRIBUTIONS

Conception and design: CC
Analysis and interpretation: CC
Data collection: CC, SQ, EF
Writing the article: CC
Critical revision of the article: GV, PR
Final approval of the article: CC, GV, SQ, EF, GS, FB, PR
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Supplementary Table (online only). Univariate analysis according to acute kidney injury (AKI) stage (low/high)

	Low, stage 1 (n = 60)	*High, stages 2 and 3 (n = 23)*	P
Age, years	73.1 ± 6.5	72 ± 6.9	.47
Male sex	95	100	.27
Current smoking	43.3	43.5	.99
History of smoking	53.3	52.2	.92
Diabetes	23.3	8.7	.21
Hypertension	83.3	91.3	.5
Dyslipidemia	45	30.4	.23
CAD	26.7	21.7	.64
CRF	43.3	26.1	.15
COPD	36.7	30.4	.59
History of cancer	15	13	1
Antithrombotic agents	78.3	60.9	.1
Statins	48.3	39.1	.45
Beta-blockers	33.3	26.1	.52
ACE inhibitors/sartans	55	69.6	.23
Calcium channel blockers	30	43.5	.24
POD	11.9 ± 7.1	18.1 ± 13.5	**.01**
Thirty-day mortality	5	4	1
Mortality, follow-up	3.4	13	.13
MI	1.7	—	1
Arrhythmias	11.7	13	1
Pneumonia	18.3	30.4	.23
Reinterventions	15.4	17.4	1

ACE, Angiotensin-converting enzyme; *CAD*, coronary artery disease; *COPD*, chronic obstructive pulmonary disease; *CRF*, chronic renal failure; *MI*, myocardial infarction; *POD*, postoperative day.

Categorical variables are presented as percentages. Continuous variables are presented as mean ± standard deviation.