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Original Article / Özgün Araştırma

Complications of Endovascular Aneurysm Repair: Mortality, Myocardial Infarction and Acute Kidney Injury

Endovasküler Anevrizma Onarımının Komplikasyonları: Mortalite, Miyokard İnfarktüsü ve Akut Böbrek Hasarı

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Abstract / Öz

Objective: Patients undergoing endovascular aneurysm repair (EVAR) have comorbidities that increase the risk of death, myocardial infarction (MI) and acute kidney injury (AKI). Our aim was to evaluate the incidence and predictors of mortality, MI and AKI after EVAR and to compare AKI incidence with Vascular Surgery Kidney Injury Predictive Score (VSKIPS).

Methods: We conducted a retrospective study of EVAR procedures performed between March 2006 and November 2013. We defined mortality at 30 days, MI as an increase in troponin level to >0.034 ng mL^{-1} in the first 72 h and AKI as an increase in creatinine level to >0.3 mg dL^{-1} in the first 48 h after surgery. Risk factors were analysed using logistic regression calculating Hosmer–Lemeshow test and the area under the receiver operating curve (AUROC).

Results: Ninety-eight patients were included in the study. The incidence of mortality, MI, and AKI was 2%, 5%, and 18%, respectively. AKI increased the risk of MI [odds ratio (OR) 24.4, p=0.006]. Preoperative serum urea level of >50 mg dL^{-1} (OR 4.97, p=0.038), general anaesthesia (OR 9.64, p=0.002) and surgery duration (OR 1.53, p=0.043) were considered independent predictors of AKI. The AUROC of the AKI model was 0.886 compared with 0.793 of VSKIPS.

Conclusion: We found the incidence of mortality, MI and AKI consistent with that of previous studies. However, we may be underestimating the last two because of the short follow-up time. AKI was an independent predictor of MI. Preoperative serum urea level of >50 mg dL^{-1}, general anaesthesia and surgery duration were considered independent predictors of AKI.

Keywords: Abdominal aortic aneurysm, endovascular procedures, anaesthesia, myocardial infarction, mortality, acute kidney injury

Amaç: Endovasküler anevrizma onarımı (EVAR) uygulanan hastalarda ölüm, miyokard enfarktüsü (MI) ve akut böbrek hasarı (AKI) riskini artıran komorbiditeler vardır. Amacımız EVAR sonrası mortalite, MI ve AKI insidansını ve prediktörlerini değerlendirmek ve AKI insidansını Vasküler Cerrahi Böbrek Hasarı Öngörü Skoru (VSKIPS) ile karşılaştırmaktı.

Yöntemler: Mart 2006 ile Kasım 2013 tarihleri arasında gerçekleştirilen EVAR prosedürlerine ilişkin retrospektif bir çalışma gerçekleştirdik. 30 günde mortalite tanımlandı. MI, ilk 72 saatte troponin seviyesinde >0,034 ng mL^{-1} değerine varan artış olarak ve AKI ise ameliyat sonrası ilk 48 saatte kreatin seviyesinde >0,3 mg dL^{-1} değerine ulaşan artış olarak tanımlandı. Risk faktörleri, lojistik regresyonu hesaplayan Hosmer-Lemeshow test ve alıcı işlem eğrisi altındaki alan (AİEAA) kullanılarak analiz edildi.

Bulgular: Doksan sekiz hasta çalışmaya dahil edildi. Mortalite, MI ve AKI insidansı sırasıyla %2, %5 ve %18 idi. AKI, MI [odds ratio (OR) 24,4, p=0,006] riskini artırdı. Preoperatif >50 mg dL^{-1} (OR 4,97, p=0,038) serum üre düzeyi, genel anestezi (OR 9,64, p=0,002) ve ameliyat süresi (OR 1,53, p=0,043) AKI'nın bağımsız prediktörleri olarak kabul edildi. AKI modelinin AİEAA değeri 0,886 ve VSKIPS ise 0,793 idi.

Sonuç: Mortalite, MI ve AKI insidansını önceki çalışmalarla uyumlu bulduk. Ancak kısa takip süresi nedeniyle son ikisini yeterli seviyede değerlendirememiş olabiliriz. AKI, MI'nın bağımsız bir prediktörü idi. Preoperatif >50 mg dL^{-1} serum üre düzeyi, genel anestezi ve ameliyat süresi AKI'nın bağımsız prediktörleri olarak kabul edildi.

Anahtar Kelimeler: Abdominal aort anevrizması, endovasküler prosedürler, anestezi, miyokard infarktüsü, mortalite, akut böbrek hasarı

Introduction

Abdominal aortic aneurysms (AAAs) are arterial dilatations or widening of the abdominal aorta with a diameter of ≥3 cm in either anteroposterior or transverse planes (1-3). AAA accounts for 65% of aortic aneurysms and 90% of them are infrarenal (1).

Endovascular aneurysm repair (EVAR) of AAAs was first described in 1991 by Parodi and was designed as a less invasive approach than open surgical repair (OSR), without aortic clamping. EVAR aimed to reduce morbidity and mortality and promote haemodynamic stability. Studies have shown improvements in perioperative complications such as acute myocar-

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dial infarction (MI), acute kidney injury (AKI), mesenteric ischaemia and pneumonia (4, 5). It has become the first-line treatment for many patients and has enabled aneurysm repair in some patients considered unfit for OSR, such as older patients with severe comorbidities. Therefore, perioperative cardiac events should not be disregarded (4, 5). According to the European Society of Cardiology (ESC)/European Society of Anaesthesiology (ESA) guidelines, EVAR is an intermediate cardiac risk procedure, with a 1%-5% incidence of cardiac events (MI or cardiac death) (6). The 30-day mortality after EVAR has been shown to be significantly lower than that after OSR; however, the difference was mitigated when considering medium- and long-term mortality (2).

Acute kidney injury is a known complication after EVAR, independently increasing medium-term morbidity and mortality (7). Its incidence after EVAR is as high as 20% in some studies (7). Although EVAR would attenuate the perioperative renal injury associated with OSR, studies have shown that in the long term, renal function deteriorates more quickly after EVAR than after OSR (8). The aetiology of AKI after EVAR is probably multifactorial and several mechanisms may be involved, other than the repeated renal contrast agent injury. Microembolisation into the renal vasculature, suprarenal bare stent fixation with the risk of renal artery trauma, accessory renal artery occlusion and inflammatory and ischaemic response after endovascular manipulation have been suggested to play a part (3, 7, 9).

In 2015, Kashani et al. (10) presented a risk prediction model for AKI in patients undergoing vascular surgery. Two clinical multivariate models for the Vascular Surgery Kidney Injury Predictive Score (VSKIPS) were developed. Model 1 was restricted to perioperative variables (preoperative glomerular filtration rate, history of previous vascular intervention and preoperative exposure to diuretics or beta-blockers), whereas model 2 included all the above and also age and intraoperative variables [duration of the procedure, fluid balance, fresh-frozen plasma (FFP) and platelet transfusion]. Both models had a fair performance predicting the occurrence of postoperative AKI after major open vascular surgery of the descending thoracic or abdominal aorta (10).

Anaesthetic technique for EVAR procedures may include general anaesthesia (GA), regional anaesthesia (RA) (subarachnoid block, epidural block and combined spinal and epidural anaesthesia) and combined general and regional anaesthesia or local anaesthesia (LA) with or without sedation. There is some evidence suggesting that patients receiving LA or RA show fewer systemic complications (cardiac, renal and respiratory), lower hospital and intensive care unit (ICU) length of stay (LOS), as well as an improvement in 30-day mortality compared with those receiving GA (4, 5, 11, 12). However, there is still controversy regarding recommended anaesthetic technique for EVAR procedures being the choice made according to the patient's comorbidities, anaesthesiologist's preference and surgical requirements (4, 5, 11-13).

The aim of this study was to evaluate the incidence and predictors of mortality, MI and AKI after EVAR and to compare it with VSKIPS models.

Methods

After receiving approval from the institutional ethics committee, we performed a retrospective study including all adult patients undergoing EVAR between March 2006 and November 2013 at a university hospital. We collected the following data: demographic characteristics, American Society of Anaesthesiology (ASA) status classification, previous medical history, usual medication, pre- and postoperative analytic study, type of anaesthesia, intraoperative monitoring, anaesthesia and procedure duration, intra- or postoperative blood transfusions during hospital stay, aneurysm characteristics, type of endovascular stent graft, ICU and hospital LOS, incidence of MI (defined as an increase in troponin level to >0.034 ng mL^{-1} in the first 72 h after surgery), occurrence of AKI (defined as an increase in creatinine level to >0.3 mg dL^{-1} in the first 48 h after surgery, according to the KDIGO classification) (14) and 30-day mortality. For the AKI analysis, we excluded patients with preoperative chronic renal failure.

Statistical analysis

Statistical analysis was performed using the IBM Statistical Package for the Social Sciences software for Windows version 22.0 (IBM SPSS Statistics; Armonk, NY, USA). Descriptive analysis, independent t, Mann-Whitney U, Fisher and chi-square tests were performed. Since we analysed three outcomes (mortality, MI and AKI), we used Bonferroni correction to decrease the probability of a type I error, which resulted in a p value of <0.017 being statistically significant. Univariate and multivariate logistic regressions were used to calculate the odds ratio (OR) and its 95% confidence interval (CI). In the multivariate logistic regression, we used the forward method including all variables with $p<0.05$ to identify the independent predictors of the outcomes. The Hosmer-Lemeshow test for the goodness-of-fit and the area under the receiver operating curve (AUROC) to measure the predictive discrimination of the model were also analysed.

Results

Patient characteristics are presented in Table 1. The majority (98%) of the aneurysms were infrarenal, and approximately half of them (52%) involved the iliac arteries. Pre-operatively, 56 patients were medicated with antiplatelet therapy, 45 in monotherapy and 11 with dual therapy. Postoperatively, 72 patients required antiplatelet therapy (41 aspirin, 24 clopidogrel, 1 ticlopidine, 6 aspirin plus clopidogrel) and 17 patients started anticoagulants after surgery.

Table 2 summarises the procedure and postoperative variables. Of the 79 RAs performed, 5 were subarachnoid blocks, 33 epidural blocks and 41 combined spinal and epidural blocks. The three combined anaesthesia were GA with epidural block. During the procedure, all patients had ASA

Table 1. Summary of patients' characteristics

	n=98
Sex	
Male	93 (95)
Female	5 (5)
Age, years	75.0±6.8
ASA physical status	
II	29 (30)
III	57 (58)
IV	10 (10)
V	2 (2)
Comorbidities	
Arterial hypertension	88 (90)
Dyslipidaemia	66 (68)
Coronary disease	40 (41)
Cardiac arrhythmia	27 (28)
Obesity	26 (27)
CHF	24 (25)
COPD	23 (24)
DM	20 (21)
CVD	12 (12)
CRF	11 (11)
PAOD	9 (9)
Usual medication	
Statin	58 (73)
Diuretic	35 (44)
β-blocker	31 (39)
Antiplatelet therapy	56 (69)
Anticoagulation therapy	9 (11)
Digoxin	2 (3)
Aneurysm characteristics	
Diameter (cm)	6.1 [5.4–7.0]
Length (cm)	5.5 [5.0–7.0]
Iliac artery involvement	44 (52)
Renal artery involvement	2 (2)
Preoperative analytic study	
Haemoglobin (g dL^{-1})	13.3±1.9
Haematocrit (%)	40.4 [36.5–43.6]
Platelets (109/L)	182.0 [154.0–218.8]
Creatinine (mg dL^{-1})	1.2 [1.0–1.5]
Urea (mg dL^{-1})	49.0 [38.0–61.0]

N (%), mean ± SD: standard deviation or median; IQR: interquartile range [P25–P75]; ASA: American Society of Anaesthesiology; HBP: high blood pressure; CHF: congestive heart failure; COPD: chronic obstructive pulmonary disease; DM: diabetes mellitus; CVD: cerebrovascular disease; CRF: chronic renal failure; PAOD: peripheral arterial occlusive disease.

Table 2. Intra- and postoperative variables

	n=98
Type of anaesthesia	
BGA	17 (18)
RA	79 (81)
Combined anaesthesia (GA+RA)	3 (3)
Sedation/local anaesthesia	1 (1)
Type of endovascular stent graft	
Zenith cook	68 (80)
Endurant medtronic	10 (12)
Others	7 (8)
Anaesthesia duration, hours	5.0 [4.0–6.0]
Surgery duration, hours	4.0 [3.0–4.5]
RBC transfusion	26 (27)
Postoperative destination	
ICU	73 (77)
Intermediate care unit	2 (2)
Hospital ward	19 (20)
Postoperative analytic study	
Haemoglobin (g dL^{-1})	11.1±1.5
Haemoglobin min (g dL^{-1})	10.0±1.7
Haematocrit (%)	33.3±4.4
Haematocrit min (%)	30.3±5.1
Platelets (10^9/L)	135.0 [112.0–155.5]
Platelets min (10^9/L)	124.0 [103.5–146.0]
Creatinine (mg dL^{-1})	1.0 [0.8–1.2]
Creatinine max (mg dL^{-1})	1.2 [1.0-1.6]
Urea (mg dL^{-1})	34.0 [26.0-43.5]
Length of ICU stay, days	1 [1-2]
Length of hospital stay, days	5 [4-7]
Postoperative complications	
Stroke	1 (1)
MI	5 (5)
AKI	15 (15)
Acute pulmonary oedema	1 (1)
Death	2 (2)

N (%), mean ± SD: standard deviation or median; IQR: interquartile range [P25-P75]; BGA: balanced general anaesthesia; RA: regional anaesthesia; GA: general anaesthesia; RBC: red blood cells; ICU: intensive care unit; min: minimum; max: maximum; MI: myocardial infarction; AKI: acute kidney injury

standard monitoring and 59 of them had invasive blood pressure monitoring. Red blood cell (RBC) transfusion was given intraoperatively in 26 patients. One patient received two units of platelets, and another patient received five units of FFP. Postoperatively, nine patients received RBC transfusion during hospital stay.

Table 3. Perioperative variables according to AKI occurrence

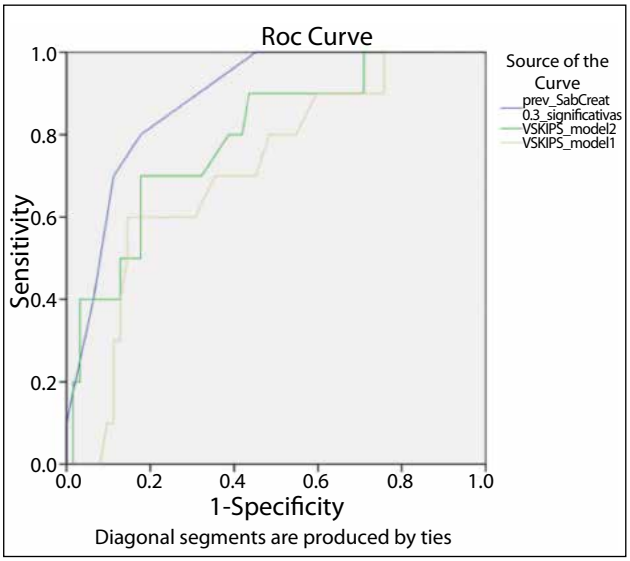
	No AKI (n=68)	AKI (n=15)	p
Male sex[1]	64 (94)	15 (100)	0.339[a]
Age[2]	74.2±6.9	78.0±5.6	0.097[b]
ASA physical status II/III[1]	59 (87)	13 (87)	1.0[a]
ASA physical status IV/V[1]	9 (13)	2 (13)	
HBP[1]	62 (91)	14 (93)	1.0[a]
Dyslipidaemia[1]	48 (68)	8 (53)	0.197[a]
Coronary Heart Disease[1]	28 (41)	7 (47)	0.697[a]
Obesity[1]	21 (31)	5 (33)	1.0[a]
COPD[1]	18 (26)	4 (27)	1.0[a]
CHF[1]	17 (25)	6 (40)	0.240[a]
DM[1]	15 (22)	5 (33)	0.341[a]
CVD[1]	8 (12)	2 (13)	1.0[a]
CRF[1]	8 (12)	3 (20)	0.409[a]
PAOD[1]	4 (6)	2 (13)	0.296[a]
Diuretic medication[1]	25 (37)	7 (47)	0.476[a]
β-blocker medication[1]	22 (32)	4 (27)	0.767[a]
Statin medication[1]	42 (62)	8 (53)	0.546[a]
Anticoagulation medication[1]	5 (7)	4 (27)	0.029[a]
ACEI/ARA medication[1]	28 (41)	4 (27)	0.386[a]
NSAID medication[2]	15 (19)	0 (0)	1.0[a]
Aneurysm diameter (mm)[3]	60 [53–70]	68 [64–82]	0.023[c]
Preoperative haemoglobin (g dL^{-1})[2]	13.5±1.9	12.6±2.1	0.273[b]
Preoperative haemoglobin <10 g dL^{-11}	2 (3)	3 (20)	0.049[a]
Preoperative creatinine (mg dL^{-1})[3]	1.15 [0.96–1.41]	1.40 [1.20–1.87]	0.005[c]
Preoperative creatinine >1.2 mg dL^{-11}	25 (37)	11 (73)	0.010[a]
Preoperative urea (mg dL^{-1})[3]	46 [38–59]	57 [52–79]	0.003[c]
Preoperative urea >50 mg dL^{-11}	24 (36)	12 (80)	0.003[a]
General anaesthesia[1]	8 (12)	8 (53)	0.001[a]
Regional anaesthesia[1]	59 (87)	6 (40)	0.001[a]
Surgery duration (hours)[3]	4.0 [3.0–4.5]	4.0 [4.0–6.1]	0.057[c]
Intraoperative RBC transfusion[1]	18 (26)	6 (40)	0.350[a]
Postoperative haemoglobin (g dL^{-1})[2]	10.2±1.6	8.4±0.9	<0.001[b]
Postoperative haemoglobin <10 g dL^{-12}	26 (38)	14 (93)	<0.001[a]
Postoperative RBC transfusion[2]	3 (4)	4 (27)	0.018[a]
Myocardial infarction	1 (1)	4 (27)	0.003[a]
Lenth of stay (days)[3]	4.5 [4.0–6.0]	12.0 [6.0–19.0]	<0.001[c]
Hospital mortality[2]	0 (0)	2 (13)	0.031[a]

[1]N (%), [2]mean ± standard deviation, [3]median and interquartile range [P25-P75].
[a]Fisher or Qui-square test, [b]Student t test, [c]Mann–Whitney U test. AKI: acute kidney injury; ASA: American Society of Anaesthesiology; HBP: high blood pressure; CHF: congestive heart failure; COPD: chronic obstructive pulmonary disease; DM: diabetes mellitus; CVD: cerebrovascular disease; CRF: chronic renal failure; PAOD: peripheral arterial occlusive disease; ACEI/ARA: angiotensin converting enzyme inhibitor/receptor antagonist; RBC: red blood cells

Table 4. Univariate and multivariate analyses of AKI

	Simple OR [95% CI]	p	Adjusted OR [95% CI]	p
Preoperative anticoagulant medication	4.58 [1.06–19.78]	0.041	-	-
Preoperative haemoglobin <10 g dL^{-1}	8.25 [1.24–54.72]	0.029	-	-
Preoperative creatinine >1.2 mg dL^{-1}	7.43 [1.36–16.44]	0.015	-	-
Preoperative urea >50 mg dL^{-1}	7.17 [1.84–27.93]	0.005	4.97 [1.10–22.52]	0.038
General anaesthesia	8.57 [2.45–30.05]	0.001	9.64 [2.26–41.12]	0.002
Surgery duration (hours)	1.48 [1.05–2.09]	0.027	1.53 [1.01–2.32]	0.043
Postoperative haemoglobin (g dL^{-1})	0.423 [0.26–0.69]	0.001	-	-
Postoperative RBC transfusion	7.88 [1.55–40.12]	0.013	-	-
Perioperative myocardial infarction	24.36 [2.49–238.72]	0.006	-	-

AKI: acute kidney injury; RBC: red blood cells



Score	AUROC	Hosmer–Lemeshow
VSKIPS model 1	0.715	-
VSKIPS model 2	0.793	-
Study predictors	0.886	0.239

Figure 1. Acute kidney injury prediction
AUROC: area under the receiver operating curve; VSKIPS: vascular surgery kidney injury predictive score

The incidence of 30-day mortality was 2% (2 of 98). Patients who died had higher ASA physical status (p=0.011), were more frequently under anticoagulation medication pre-operatively (p=0.008) and had lower preoperative haemoglobin (9.2±0.6 vs. 13.4±1.8, p=0.002).

The incidence of MI was 5% (5 of 98). Pre- and intraoperative variables were similar between the two groups. Patients having MI had higher postoperative creatinine level [2.3 (1.9-6.1) vs. 1.1 (0.9-1.5), p=0.002]. After multivariate analysis, only postoperative AKI was identified as an independent predictor of MI (adjusted OR 24.4, 95% CI, 2.5-238.7; p=0.006).

The incidence of AKI was 18% (15 of 83). Table 3 displays the perioperative data according to the occurrence of AKI. Patient characteristics were similar between the two groups. Univariate and multivariate logistic regression can be seen in Table 4. Preoperative serum urea level of >50 mg dL^{-1} (OR 4.97, p=0.038), GA (OR 9.64, p=0.002) and surgery duration (OR 1.53, p=0.043) were considered independent predictors of AKI in multivariate analysis. Hosmer-Lemeshow test value was 0.239 and AUROC was 0.886, whereas VSKIPS was 0.793 (Figure 1). Patients with AKI had higher hospital LOS [5 (4-6) vs. 12 (6-19) days, p<0.001].

Discussion

In our study, the 30-day mortality after EVAR was 2%. There are two meta-analyses comparing EVAR with OSR. In the meta-analysis conducted by Stather et al. (15) the 30-day mortality rate was found to be 1.4%. The difference in mortality may be because our patients were older, patients had a higher ASA classification or our study included only elective EVAR of Randomized Controlled Trials. Thomas et al. (16) performed a meta-analysis including observational studies and concluded that global 30-day mortality of EVAR was 4.2% but decreased to 1.4% if only elective cases were considered. Egorova et al. (13) elaborated a perioperative risk scoring system based on the predictors of 30-day mortality: renal failure, lower extremity ischaemia, age of >75 years, liver disease, congestive heart failure, female sex, neurological condition, chronic pulmonary condition, surgeon EVAR experience of <3 cases and hospital annual volume of <7 cases. The performance of that risk score in our sample was poor (AUROC, 0.570) perhaps because of the sample size. Although many surgeons perform EVAR at our hospital, none has less than three cases of experience. During the first 2 years, hospital annual volume was around 7 cases, but since

2008, there have been 12-18 cases per year. The two cases of mortality were after 2009.

According to the European Society for Vascular Surgery, cardiac events are a major cause of morbidity and mortality after non-cardiac surgery causing 10%-40% of perioperative deaths (6). Despite the prevalence of cardiovascular risk factors in our population, we may say that none of our deaths was caused by MI.

The incidence of MI in our study was 5% in the first 72 h after surgery. The ESC/ESA guidelines on non-cardiac surgery predict 1%-5% of cardiac events (cardiac death and MI) until 30 days postoperatively (6). It is possible that we may be underestimating the occurrence of MI in our study because of the shorter follow-up time. The incidence of MI after elective EVAR in the meta-analysis presented by Stather et al. (15) was 6.8% similar to 6.3% found in the meta-analysis of Thomas et al. (16) but neither specified the follow-up time for this parameter.

The ESC/ESA guidelines linked chronic renal disease with increased risk of cardiovascular disease, it being an independent risk factor for adverse postoperative cardiovascular outcomes, including MI, stroke and progression of heart failure (6). In our study, postoperative AKI was an independent predictor of MI. In a 2015 prospective cohort study, AKI (defined according to the KDIGO classification) predicted the risk of chronic non-fatal MI in patients with type 2 diabetes (17). However, this study did not define the cause of AKI and was restricted to diabetic patients. The reasons why AKI can predict MI are unknown; one possible explanation is the systemic inflammation, but it is possible that AKI constitutes a marker of renal and overall frailty (17).

The incidence of AKI was 18% in our study, which is consistent with that of previous studies (19%-29%) (7, 18). Incidence may vary across studies for various reasons, one of them being the difference in classification of AKI.

We found that GA and surgery duration increased the risk of AKI. In a study using the multicentre EUROSTAR registry (EUROpean collaborators on Stent graft Techniques for AAA Repair), (19) there were fewer systemic complications (cardiac, pulmonary, renal and sepsis) for LA with sedation than for GA (6.6% vs. 13.0%, p=0.0015) and for RA than for GA (9.5% vs. 13%, p=0.0007). There is a potential bias that patients undergoing GA had more complex procedures (p=0.011), more additional procedures (p<0.001) and longer procedure duration (p<0.001), and that could be the case in our study (19). Additionally, longer procedures could mean more contrast that may contribute to the postoperative AKI.

Preoperative urea level of >50 mg dL^{-1} increased the risk of postoperative AKI. This finding may indicate the need to optimise renal function and euvolemia pre-operatively, avoid nephrotoxic drugs, carefully watch the amount of contrast administered and perform contrast nephropathy prophylaxis whenever indicated. This outcome may open future perspectives as a possible predictor of complications.

We compared our findings with the VSKIPS models (model 1: AUROC, 0.715 and model 2: AUROC, 0.793) (10). Our study had several variables in common with VSKIPS (age, preoperative exposure to diuretics and beta-blockers, duration of the procedure and plasma and platelet transfusion), but we did not use history of previous vascular intervention or fluid balance as we did not have that information available in our data. Our findings were concordant with this previous study, as model 2 performed better than model 1 (AUROC, 0.715 and 0.793, respectively). However, VSKIPS defined AKI with the Acute Kidney Injury Network criteria (using serum creatinine levels and hourly urine output), whereas our study was based on the KDIGO classification (defined as an increase in creatinine level to >0.3 mg dL^{-1} in the first 48 h after surgery) (14).

According to the European Society of Intensive Care Medicine (ESICM), the ICU and hospital LOSs are important for health finance evaluations but not as indicators of clinical outcome because they depend on hospital and healthcare policy as well as on physician performance (14). Better planning to avoid AKI may influence both hospital and ICU LOS.

Study limitations

It was impossible to collect any information regarding the amount of contrast or AKI preventive strategies used during the procedure. We cannot exclude the possibility that a part of the documented AKI may be related to contrast-induced nephropathy. We do not have available information regarding the need for renal replacement therapy. Another limitation is that we only evaluated the short-term mortality. According to the ESA/ESICM, (14) the mortality should be reported until 90 days and preferably 1 year after surgery, although short-term mortality may remain relevant as a treatment safety outcome. We did not register surgery-related complications, including the presence of endoleak, aneurysm rupture or conversion to OSR, and the technical difficulties or success rates of the intervention were not taken into account.

Conclusion

We found the incidence of mortality, MI and AKI consistent with that of previous studies. Preoperative serum urea level of >50 mg dL^{-1}, GA and surgery duration were considered independent predictors of AKI. AKI was an independent predictor of MI. The VSKIPS models developed for major open vascular surgery showed a fair performance for EVAR patients.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Centro Hospitalar São João (CES 76-14; 13.03.2014).

Informed Consent: Ethics committee did not require written informed consent for a retrospective study since data was analysed without patient identification.

Peer-review: Externally peer-reviewed.

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Hasta Onamı: Retrospektif çalışma hastanın kimlik bilgileri olmadan yapıldığından, Etik komite yazılı bilgilendirilmiş onam talep etmemiştir.

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References

1. Lorentz MN, Boni CL, Soares RR. Anesthesia for endovascular surgery of the abdominal aorta. Rev Bras Anestesiol 2008; 58: 525-32. [CrossRef]
2. Paravastu SC, Jayarajasingam R, Cottam R, Palfreyman SJ, Michaels JA, Thomas SM. Endovascular repair of abdominal aortic aneurysm. Cochrane Database Syst Rev 2014; 23: CD004178.
3. Moll FL, Powell JT, Fraedrich G, Verzini F, Haulon S, Waltham M, et al. Management of abdominal aortic aneurysms clinical practice guidelines of the European society for vascular surgery. Eur J Vasc Endovasc Surg 2011; 41(Suppl 1): S1-58.
4. Elisha S, Nagelhout J, Heiner J, Gabot M. Anesthesia case management for endovascular aortic aneurysm repair. AANA J 2014; 82: 145-52.
5. Wylie SJ, Wong GT, Chan YC, Irwin MG. Endovascular aneurysm repair: a perioperative perspective. Acta Anaesthesiol Scand 2012; 56: 941-9. [CrossRef]
6. Kristensen SD, Knuuti J, Saraste A, Anker S, Botker HE, De Hert S, et al. 2014 ESC/ESA Guidelines on non-cardiac surgery: cardiovascular assessment and management. Kardiol Pol 2014; 72: 857-918. [CrossRef]
7. Saratzis A, Melas N, Mahmood A, Sarafidis P. Incidence of Acute Kidney Injury (AKI) after Endovascular Abdominal Aortic Aneurysm Repair (EVAR) and Impact on Outcome. Eur J Vasc Endovasc Surg 2015; 49: 534-40. [CrossRef]
8. Mills JL, Sr., Duong ST, Leon LR, Jr., Goshima KR, Ihnat DM, Wendel CS, et al. Comparison of the effects of open and endovascular aortic aneurysm repair on long-term renal function using chronic kidney disease staging based on glomerular filtration rate. J Vasc Surg 2008; 47: 1141-9. [CrossRef]
9. Saratzis AN, Goodyear S, Sur H, Saedon M, Imray C, Mahmood A. Acute kidney injury after endovascular repair of abdominal aortic aneurysm. J Endovasc Ther 2013; 20: 315-30. [CrossRef]
10. Kashani K, Steuernagle JHT, Akhoundi A, Alsara A, Hanson AC, Kor DJ. Vascular Surgery Kidney Injury Predictive Score: A Historical Cohort Study. J Cardiothorac Vasc Anesth 2015; 29: 1588-95. [CrossRef]
11. Franz R, Hartman J, Wright M. Comparison of anesthesia technique on outcomes of endovascular repair of abdominal aortic aneurysms: a five-year review of monitored anesthesia care with local anesthesia vs. general or regional anesthesia. J Cardiovasc Surg (Torino) 2011; 52: 567-77.
12. Smaka TJ, Cobas M, Velazquez OC, Lubarsky DA. Perioperative management of endovascular abdominal aortic aneurysm repair: update 2010. J Cardiothorac Vasc Anesth 2011; 25: 166-76. [CrossRef]
13. Egorova N, Giacovelli JK, Gelijns A, Mureebe L, Greco G, Morrissey N, et al. Defining high-risk patients for endovascular aneurysm repair. J Vasc Surg 2009; 50: 1271-9. [CrossRef]
14. Jammer I, Wickboldt N, Sander M, Smith A, Schultz MJ, Pelosi P, et al. Standards for definitions and use of outcome measures for clinical effectiveness research in perioperative medicine: European Perioperative Clinical Outcome (EPCO) definitions: a statement from the ESA-ESICM joint taskforce on perioperative outcome measures. Eur J Anaesthesiol 2015; 32: 88-105. [CrossRef]
15. Stather PW, Sidloff D, Dattani N, Choke E, Bown MJ, Sayers RD. Systematic review and meta-analysis of the early and late outcomes of open and endovascular repair of abdominal aortic aneurysm. Br J Surg 2013; 100: 863-72. [CrossRef]
16. Thomas DM, Hulten EA, Ellis ST, Anderson DM, Anderson N, McRae F, et al. Open versus Endovascular Repair of Abdominal Aortic Aneurysm in the Elective and Emergent Setting in a Pooled Population of 37,781 Patients: A Systematic Review and Meta-Analysis. ISRN Cardiol 2014: 149243.
17. Monseu M, Gand E, Saulnier PJ, Ragot S, Piguel X, Zaoui P, et al. Acute Kidney Injury Predicts Major Adverse Outcomes in Diabetes: Synergic Impact With Low Glomerular Filtration Rate and Albuminuria. Diabetes Care 2015; 38: 2333-40. [CrossRef]
18. Sailer AM, Nelemans PJ, van Berlo C, Yazar O, de Haan MW, Fleischmann D, et al. Endovascular treatment of complex aortic aneurysms: prevalence of acute kidney injury and effect on long-term renal function. Eur Radiol 2016; 26: 1613-9. [CrossRef]
19. Ruppert V, Leurs LJ, Steckmeier B, Buth J, Umscheid T. Influence of anesthesia type on outcome after endovascular aortic aneurysm repair: an analysis based on EUROSTAR data. J Vasc Surg 2006; 44: 16-21. [CrossRef]