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Original Article

Occurrences and Results of Acute Kidney Injury after Endovascular Aortic Abdominal Repair?

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Purpose: Acute kidney injury (AKI) is an important postoperative complication that may impact mortality and morbidity. The incidence of AKI after elective endovascular aneurysm repair (EVAR) is not known well. The aim of this study is to assess the incidence of AKI after elective EVAR and examine the impact of AKI.

Materials and Methods: Data were collected and analyzed retrospectively from 78 elective EVARs for abdominal aortic aneurysm (AAA) among 102 total cases of conventional EVAR performed in Inha University Hospital from 2009 to 2015. The primary endpoint was incidence and risk factors of AKI. Secondary endpoints included drop in estimated glomerular filtration rate (eGFR) and the mortality of AKI.

Results: We included 78 patients (17 females, 21%; mean age, 73.9±12.5 years; mean AAA diameter, 59.3±8.9 cm), 11 (14.1%) of whom developed AKI. Within 48 hours, those with AKI experienced a decrease in eGFR from 65.5±21.2 to 51.2±19.6 mL/kg/1.73 m^2, and those without AKI showed a change from 73.1±9.2 to 74.2±10.7 mL/kg/1.73 m^2. There were no patients who required dialysis during follow-up (mean, 24.2±18.0 months). Development of AKI was related to operation time (odds ratio [OR], 2.024; 95% confidence interval [CI], 1.732-4.723; P<0.010) and contrast dose (OR, 3.192; 95% CI, 2.182-4.329; P<0.010). There were no differences in mortality between the 2 groups (P=0.784).

Conclusion: The incidence of AKI after EVAR was related to operation time and contrast dose, but was not associated with medium-term mortality.

Key Words: Acute kidney injury, Aortic aneurysm, Endovascular procedures

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INTRODUCTION

Although endovascular aneurysm repair (EVAR) is now the first-line treatment for abdominal aortic aneurysm (AAA), EVAR has some weak points, including the risk of acute kidney injury (AKI) [1]. During the EVAR procedure, the patient is exposed to risks of AKI due to various circumstances. When the stent graft is suprarenal coverage [2,3] or covers the accessory renal artery [4,5], blood flow to the kidney may be blocked, resulting in AKI. Renal artery injuries, such as dissection, thrombosis, or microembolization also lead to renal injury [1]. Every intravascular procedure including EVAR requires contrast medium, which can induce contrast-induced nephropathy.

Many reports have investigated the risk factors of AKI and the relationship between AKI and patients' outcomes [1,6,7]. AKI after EVAR is associated with mortality, morbidity, and prolonged hospitalization [7]. Yet there have been

few studies that have examined AKI after intervention in Asian countries, particularly in Korea.

The aim of this study is to assess the incidence of AKI after elective EVAR in order to investigate the risk factors associated with AKI and to compare the mortality between the AKI group and no-AKI group in Koreans.

MATERIALS AND METHODS

We retrospectively enrolled 102 patients who underwent elective EVAR for AAA at Inha University Hospital from January 2009 to December 2015. Data based on a chart review for each of the 102 identified patients were generated. Based on these reviews, 24 patients who had end stage renal disease, ruptured AAA or symptomatic AAA, or a history of renal artery angioplasty were excluded, and 78 patients remained. Demographic data, comorbidity, preoperative renal function, procedural details, and postoperative complications were collected and analyzed. Blood samples were collected at 24, 48, and 72 hours after EVAR. Measurements of urine output continued for at least 48 hours or until the patients were discharged.

The estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration formula [8].

The grading of AKI was defined by the risk, injury, failure, loss of kidney function, and end-stage kidney disease (RIFLE) criteria: risk of renal dysfunction, injury to the kidney, failure of kidney function, loss of kidney function, and end-stage kidney disease. The RIFLE criteria are quantified by the GFR or serum creatinine, and urine production (Table 1) [9]. AKI can be defined as abrupt (within 7 days) and sustained decrease (more than 24 hours) in renal function. The cases with decrease over 25% of eGFR or elevation over 1.5 times comparing baseline and urine production below 0.5 mL/kg/h for 6 hours belonged to AKI group.

All of the patients took prophylaxes for contrast-induced nephropathy (CIN). The prophylaxes included N-acetylcysteine (600 mg 3 times per day for 3 days) and preprocedural and postprocedural hydration with at least 1 L of normal saline or half saline. For AKI, we continued hydration and N-acetylcystein till improvement of AKI [10].

Standard follow-up procedures including a laboratory exam were conducted at 30 days, 6 months, and 12 months after EVAR with an image study, such as duplex or computed tomography (CT). We performed the duplex and contrast-enhanced ultrasound in case in case with chronic renal failure or history of AKI.

The primary endpoint of this study was the occurrence of AKI after elective EVAR and the risk factors of the AKI, and the secondary endpoint was the comparison of eGFR, serum creatinine, and mortality between the AKI and no-AKI groups.

IBM SPSS Stastistic ver. 24.0 software (IBM Co., Armonk, NY, USA) was used for statistical analyses. Fisher's exact test and Student's t-test were used for univariate analyses and the logistic regression test for multivariate analyses. A P-value less than 0.05 was considered statistically significant.

RESULTS

A total of 78 patients were enrolled after exclusions. AKI occurred in 11 patients; 9 patients were at risk of renal dysfunction, and 2 patients had kidney injury. No patient was categorized as having loss of kidney function or end-stage kidney disease (Table 1).

We compared 2 groups according to the patient demographics, comorbidities, preoperative renal functions, and preoperative aneurysmal diameters. There were no statistically differences between the 2 groups (Table 2). In the procedural details, the procedural time and contrast dose affected the occurrence of AKI. The AKI group had longer procedural times than the no-AKI group (128.1 minutes in the AKI group vs. 109.5 minutes in the no-AKI group, P=0.025) and received more contrast agent (108.3 mL of contrast agent in the AKI group vs. 89.6 cc in the no-AKI group [P=0.018]) (Table 2). In the multivariate analysis, the preoperative eGFR, procedural time, and contrast dose were factors affecting the occurrence of AKI (Table 3).

Postoperative eGFR and creatinine were compared be-

Table 1. Criteria and occlusion of AKI after EVAR

Group	eGFR decrease	S_{Cr}	Urine production	Patient (n=78)
Patients at risk	>25% or	>1.5 times over baseline or	<0.5 mL/kg/h for 6 h	9 (11.5)
Kidney injury	>50% or	>2 times over baseline or	<0.5 mL/kg/h for 12 h	2 (2.6)
Kidney failure	>75% or	>3 times or >4 mg/dL	<0.3 mL/kg/h for 24 h	0
Loss of kidney function		Complete loss over 4 wk		0
End-stage renal disease		Complete loess over 3 mo		0

Values are presented as number (%).
AKI, acute kidney injury; EVAR, endovascular aneurysmal repair; eGFR, estimated glomerular filtration rate; S_{Cr}, serum creatinine.

Table 2. Patient characteristics and procedural details between AKI and no-AKI groups

Variable	AKI (n=11)	No-AKI (n=67)	P-value
Demographics			
Age (y)	73.59±10.45	72.64±8.64	0.657
Male	8 (72.7)	53 (79.1)	0.282
Comorbidity			
Smoking	4 (36.4)	28 (41.8)	0.754
Hypertension	8 (72.7)	52 (77.6)	0.817
Coronary artery disease	2 (18.2)	8 (11.9)	0.791
Diabetes mellitus	5 (45.5)	26 (38.8)	0.548
Cerebrovascular disease	2 (18.2)	7 (10.4)	0.884
Hyperlipidemia	6 (54.5)	41 (61.2)	0.841
Preoperative renal function			
eGFR (mL/kg/1.73 m^2)	65.5±21.2	73.1±9.2	0.045
S_{Cr} (mg/dL)	1.2±0.4	1.0±0.5	0.048
Aneurysmal diameter (mm)	57.4±9.2	59.7±10.7	0.735
Suprarenal fixation device	10 (90.9)	62 (92.5)	0.884
Accessory renal artery occlusion	1 (9.1)	4 (6.0)	0.549
Endoleak in completion angiogram	6 (54.5)	28 (41.8)	0.269
Type I	3	5	
Type II	2	12	
Type III	0	1	
Type IV	1	10	
Additional procedure after angiogram			0.150
Aortic or limb extension	3	6	
Additional ballooning	3	22	
Procedural time (min)	128.1±48.2	109.5±37.3	0.025
Contrast dose (mL)	108.3±251.5	89.6±55.3	0.018

Values are presented as mean±standard deviation, number (%), or number only.
AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; S_{Cr}, serum creatinine.

Table 3. Univariate and multivariate analyses of risk factors of AKI

Variable	Univariate analysis		Multivariate analysis	
	OR (CI)	P-value	OR (CI)	P-value
Suprarenal fixation device	0.856 (0.432-1.659)	0.884	0.918 (0.735-1.421)	0.985
Preoperative eGFR	1.524 (1.058-3.489)	0.045	1.804 (1.261-5.427)	0.021
Diabetes mellitus	1.651 (0.615-2.273)	0.548	1.434 (0.488-1.932)	0.448
Hypertension	1.124 (0.192-1.846)	0.817	1.267 (0.292-1.943)	0.253
Endoleak in completion angiogram	1.322 (0.853-1.625)	0.765	1.089 (0.894-1.234)	0.822
Procedural time	2.123 (1.123-3.920)	0.025	2.024 (1.732-4.723)	<0.010
Contrast dose	3.201 (1.815-6.475)	0.018	3.192 (2.182-4.329)	<0.010

AKI, acute kidney injury; OR, odds ratio; CI, confidence interval; eGFR, estimated glomerular filtration rate.

tween the AKI and no-AKI groups. The mean eGFR before EVAR was 65.5±21.2 mL/kg/1.73 m^2 in the AKI group and 73.1±9.2 mL/kg/1.73 m^2 in the no-AKI group. The post-operative mean eGFR was 42.7±13.3 mL/kg/1.73 m^2 (24 hours), 51.2±19.6 mL/kg/1.73 m^2 (48 hours), and 62.8±18.2 mL/kg/1.73 m^2 (1 month) in the AKI patients and 72.3±8.5 mL/kg/1.73 m^2 (24 hours), 74.2±10.7 mL/kg/1.73 m^2 (48 hours), and 72.6±10.3 mL/kg/1.73 m^2 (1 month) in the no-AKI patients. Mean serum creatinine (S_{Cr}) was 1.2±0.4 mg/dL in AKI patients, and this was elevated to 1.7±0.7 mg/dL

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Table 4. Change of eGFR and S_{Cr} after EVAR during 1month

Variable	Basal	24 hours	48 hours	1-month
AKI				
eGFR (mL/kg/1.73 m^2)	65.5±21.2	42.7±13.3	51.2±19.6	62.8±18.2
S_{Cr} (mg/dL)	1.2±0.4	1.7±0.7	1.4±0.6	1.3±0.9
Urine output (mL/kg/h)	1.1±0.6	0.9±0.4	1.0±0.5	NA
No-AKI				
eGFR (mL/kg/1.73 m^2)	73.1±9.2	72.3±8.5	74.2±10.7	72.6±10.3
S_{Cr} (mg/dL)	1.0±0.5	1.1±0.4	1.0±0.6	1.0±0.6
Urine output (mL/kg/h)	1.2±0.5	1.2±0.6	1.3±0.5	NA

Values are presented as mean±standard deviation.
eGFR, estimated glomerular filtration rate; S_{Cr}, serum creatinine; EVAR, endovascular aneurysm repair; AKI, acute kidney injury; NA, not available.

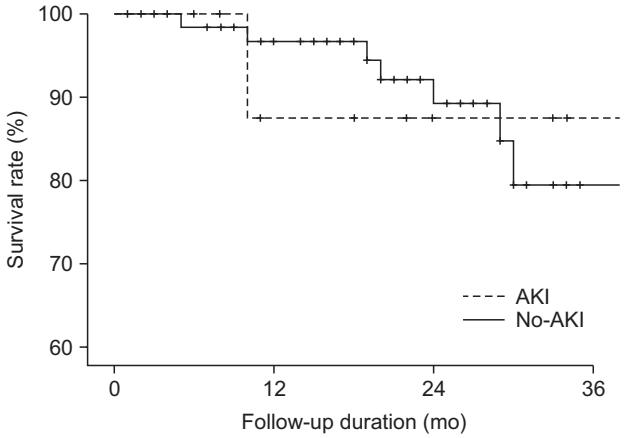

Fig. 1. Comparison of survival rates between acute kidney injury (AKI) and no-AKI groups. There is no difference between 2 groups.

(24 hours after EVAR), 1.4±0.6 mg/dL (48 hours later), and 1.3±0.9 mg/dL (1 month later) (Table 4).

In the AKI patients, there were no patients who required dialysis during follow-up. For the follow-up period (mean±standard deviation: 24.2±18.0 months), there was no significant difference in survival rate between the 2 groups at mid-term follow-up (Log rank=0.784, Fig. 1).

DISCUSSION

This study showed an AKI incidence of 14.1% after EVAR similar to other studies. Previous data showed the incidence of 1% to 19% for elective EVAR [1,7]. However, criteria of AKI in previous study were not uniform. The recent study with same criteria comparing this study showed incidence of 18.8%. In DREAM trial, AKI by S_{Cr} elevation over 20% comparing baseline occurred at 8% [6].

There are several mechanisms that can lead to AKI after EVAR, including microembolization, suprarenal fixation, accessory renal artery occlusion, and CIN, as well as the inflammatory and ischemic responses associated with the intervention [1]. In our study, main cause of AKI after EVAR maybe is CIN because contrast dose was associated with AKI. Recently the stent-graft and endovascular technique are progressive so that complications related to guidewire and catheter decreased, such as dissection or embolism.

In this study, only 3 factors affected AKI after EVAR: preoperative eGFR, procedural time, and contrast dose. Recent trial showed that base creatine level and eGFR weren't associated with AKI except cholesterolemia. However, there was difference of preoperative eGFR and creatine between 2 groups in that study. Also in this study suprarenal fixation device, such as Endurant (Medtronics Inc., Minneapolis, MN, USA) and Zenith (Cook Medicals Inc., Bloomingtons, IN, USA) did not affect AKI after EVAR like other studies [11,12]. Contrast dose and procedural time were not associated with AKI after EVAR unlike our study [1,7]. Nonetheless, the possibilty of AKI after EVAR increased in case of use the amout of contrast in our study, and thusit is neccesary to evaluate the relation between contrast dose and occurrence of AKI after EVAR with a large sample of data. There were no association between occurrence of AKI and mortality over a mean follow-up of 24 months. There is an abundance of data strongly suggesting that AKI after EVAR does impact mortality and cardiovascular event like other vascular surgery [7,13]. Additionally, following major surgery, long-term survival is worse among patients who have AKI after the procedure. For this reason, it is necessary to be beware of AKI after EVAR during follow-up. In our study, main cause of AKI after EVAR was CIN as previous mentioned. We recommend the non-contrast CT and contrast enhanced ultrasound for follow-up image modality in patients with history of AKI after EVAR.

This study is retrospective, and so it may have selection

bias for gathering samples. Also, the small patient population can be a limitation. Despite the limitations, our study shows that clinicians' efforts to reduce the use of contrast media and procedure time are vital to reduce the risk of AKI in AAA patients that are undergoing EVAR, and are with low eGFR.

In conclusion, AKI after EVAR is a relatively common complication (14.1%). In particular, patients with low preoperative eGFR, we must make efforts to decrease the procedural time and volume of contrast medium with regard to avoid AKI after EVAR.

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