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CLINICAL INVESTIGATION ARTERIAL INTERVENTIONS

Kidney Failure After Occlusion of Accessory Renal Arteries in Endovascular Abdominal Aneurysm Repair

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Abstract

Purpose To evaluate the incidence of acute renal failure and chronic kidney disease due to occlusion of accessory renal arteries during endovascular aneurysm repair of infrarenal abdominal aortic aneurysm.

Material and Methods We retrospectively reviewed the course of 181 patients (mean age, 71, SD ± 9 years) who underwent EVAR of infrarenal abdominal aortic aneurysm. The renal vessel anatomy was analyzed in all pre- and postoperative CT scans. Diameter and origin of accessory renal arteries were evaluated. Renal function was determined by pre- and postoperative serum creatinine and eGFR levels. Long-term follow-up (>3 months) of patients was available in 121 cases (66.9%). Acute kidney injury and chronic kidney failure were defined according to guidelines of "Kidney Disease: Improving Global Outcomes" (KDIGO).

Results In 65 of 181 patients (33.9%), 82 accessory renal arteries were identified preoperatively. In 19 of 181 patients (10.5%), one or more accessory renal arteries were covered and subsequently occluded by the implanted stent-graft device. Neither acute kidney injury (10.3% vs 12.5%; p = .785) nor chronic kidney disease (10.7% vs 15.38%; p = .452) was detected significantly more often in patients with covered accessory renal artery. The only significant predictor of acute kidney injury was the preoperative serum creatinine level (1.12 mg/dl vs. 0.98 mg/dl; p = .03). Significant predictors for chronic kidney disease were preoperative serum creatinine, eGFR, and impaired renal function ($p < .001$).

Conclusion Coverage of accessory renal artery due to stent-graft does not lead either to temporary acute kidney injury after endovascular aneurysm repair or to chronic kidney disease.

Level of Evidence Level II b.

Keywords Aneurysm · EVAR · Accessory renal arteries · Acute kidney injury · Chronic kidney disease

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Introduction

Since its first description, endovascular aneurysm repair (EVAR) has become widely accepted as a safe technique for the treatment of abdominal aortic aneurysm (AAA) [1].

Coverage of infrarenal accessory renal arteries (ARA) is often technically unavoidable and relatively common, considering the fact that up to one-third of patients have one or more ARAs [2, 3]. However, the impact of ARA occlusion on renal function during EVAR is unclear and discussed controversially [4, 5]. Some authors found that coverage of ARA is not associated with renal failure [6–9], whereas others observed a decline in renal function after EVAR due to coverage of ARA [10–12]. Our retrospective study aimed to evaluate the incidence of acute kidney injury (AKI) and chronic kidney disease (CKD) due to occlusion of ARA during EVAR procedure of infrarenal AAA in a large cohort.

Material and Methods

Patients

This consecutive study was approved by the institutional review board. Between January 2003 and January 2013, 803 patients were treated with abdominal aneurysm repair. They were informed about endovascular as well as open aneurysm repair and anatomical characteristics. A total of 435 patients underwent EVAR for abdominal aortic aneurysm. For further analysis, we excluded patients with incomplete preoperative or postoperative imaging (121/435; 27.8%), with aortic rupture (24/435; 5.5%), with solitary kidney (4/435; 0.9%), with incomplete laboratory values (43/435; 9.9%), with preoperative terminal dialysis-dependent kidney insufficiency (4/435; 0.9%), and with fenestrated or branched stent-graft devices including renal arteries (58/435; 13.3%). Finally, 181 (181/435; 41.6%) patients with an infrarenal AAA undergoing standard EVAR procedure fulfilled the inclusion criteria (Fig. 1). There were 166 men (mean age 71, SD ± 9 years) and 15 women (mean age 75, ± SD 5 years). Patients in normovolaemic status received 1 ml/kg bodyweight per hour full electrolyte solution 24 h before EVAR.

EVAR Procedures

In most patients (176/181; 97.2%), a bifurcated stent-graft was implanted, followed by tube stent-graft in 3 patients (1.7%) and mono-iliac in 2 patients (1.1%).

The Zenith device (Cook, Bloomington, Indiana, USA) was used in 54 (29.8%), the Anaconda device (Vascutek, Inchinnan, Scotland) in 48 (26.5%), the Excluder device (W. L. Gore & Associates, Flagstaff, Arizona, USA) in 45 (24.9%), the Endologix device (Endologix, Irvine, California, USA) in 15 (8.3%), the Endurant device (Medtronic, Minneapolis, Minnesota, USA) in 16 (8.8%), and other devices in 3 (1.7%) patients. During EVAR 234, 44 ml (± 94,35 ml) non-ionic contrast media was applied.

Imaging Studies

Images were acquired by multidetector CT scanners (Somatom Sensation 16 or Somatom Flash Dual Source; Siemens Healthcare GmbH, Forchheim, Germany). For arterial phase images, non-ionic contrast material (90−150 ml) was injected with a flow rate of 4 ml/sec using bolus tracking with a threshold of 100 HU. Delayed phase images were acquired 70 s after the arterial phase scan. Reconstructed transverse and coronal plane images used for analyses had a section thickness of 3.0 mm and a section increment of 3.0 mm.

Preoperative contrast-enhanced CT scans were performed 33 days (± 51 days) before EVAR. Postoperative contrast-enhanced CT scans were performed 4 days (± 2,9 days) after EVAR.

Analysis of Renal Vascular Anatomy

Analysis of the renal arterial blood supply was based on digitally stored CT datasets (syngo.via; Siemens Healthcare GmbH, Forchheim, Germany). Pre- and postoperative contrast-enhanced CT datasets were analyzed by two blinded readers (Reader 1: author # 3, 4 years; Reader 2: author # 1, 1 year of experience in diagnostic imaging) in a random order. In case of disagreement, a decision was made by author # 1, author # 3 and author # 10 (15 years of experience) in consensus. Mean diameters of ARAs were measured on preoperative CT scans using electronic callipers.

Renal infarcts were defined as locally restricted hypoperfusioned renal tissue that appeared hypodense on arterial phase imaging. Two readers (Reader 1: author # 2, 2 years; Reader 2: author # 1, 1 year of experience in diagnostic imaging) measured the renal volume using the open source software OsiriX 7.5 [13]. Absolute renal infarct volume and overall renal parenchyma volume in postoperative CT images was determined. Relative renal infarct volume compared to overall renal parenchyma volume was calculated (Fig. 2).

Measurement of Renal Function

Serum creatinine (SCr) was determined by enzymatic assays on ADVIA®1650/1800 and Dimension Vista®1500 chemistry analyzer. Estimated glomerular filtration rate (eGFR) was expressed in values normalized to standard

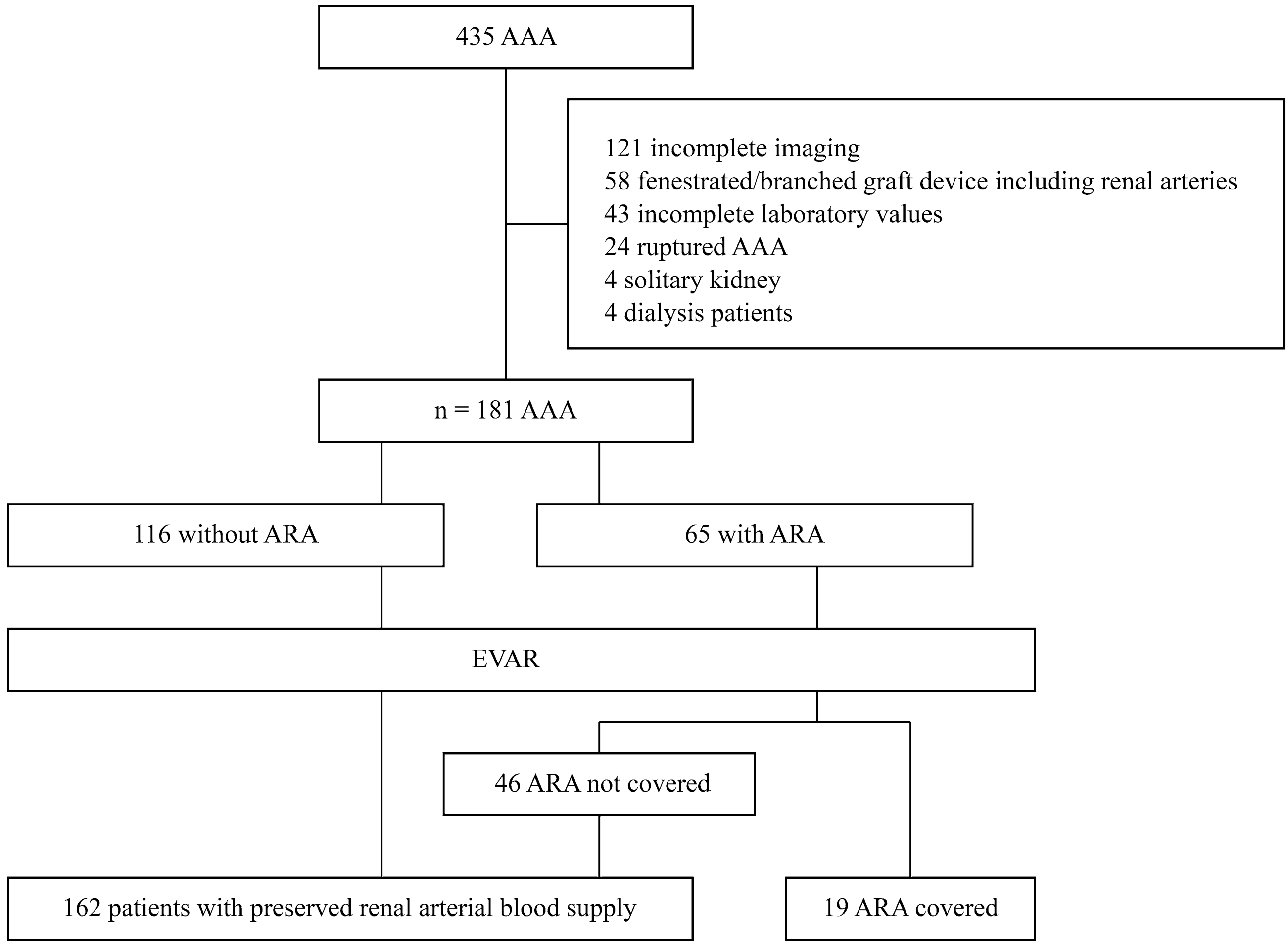

Fig. 1 Flowchart of patient cohort. Between 2003 and 2013, 435 abdominal aortic aneurysms (AAA) were treated with EVAR. Exclusion criteria were incomplete imaging, fenestrated or branched graft device, incomplete laboratory values, ruptured AAA and solitary kidney. In 65 patients of 181 patients, at least one accessory renal artery (ARA) was detected in preoperative contrast-enhanced computed tomography (CT) imaging. During EVAR in 19 patients, at least one ARA was covered

body surface area (ml/min/1.73 m^2) according to the modification of diet in renal disease (MDRD) study equation from January/2003 to January/2013 [14, 15] and according to the chronic kidney disease—epidemiology collaboration (CKD-EPI) formula from February 2013 to December 2016 [16].

Definitions

Preoperative SCr and eGFR were determined mean 3 days (± 2.9 days) before EVAR. Patients were categorized by the preoperative baseline eGFR (normal renal function, eGFR $\geq$ 60 ml/min/1.73 m^2; impaired renal function, eGFR < 60 ml/min/1.73 m^2).

AKI was defined as an increase in SCr either at least 0.3 mg/dl within 48 h or $\geq$ 50% of the baseline value within seven days after EVAR according to the kidney disease—improving global outcomes (KDIGO) guidelines [17].

CKD was defined as decrease of eGFR less than 60 ml/min/1.73 m^2 lasting longer than 3 months (>90 days) after EVAR [18].

Statistical Analysis

All patients (n = 181) with available pre- and postoperative SCr values were included in statistical analysis. Continuous data are presented as mean (± SD); categorical data as absolute frequencies (%). The impact of baseline characteristics and further clinical parameters on renal function (no AKI vs. AKI) and CKD after EVAR was analyzed using logistic regression models. Odds Ratios with corresponding 95% confidence intervals are presented as effect estimates. A p value < 0.05 was considered statistically significant. All analyses were performed using R version

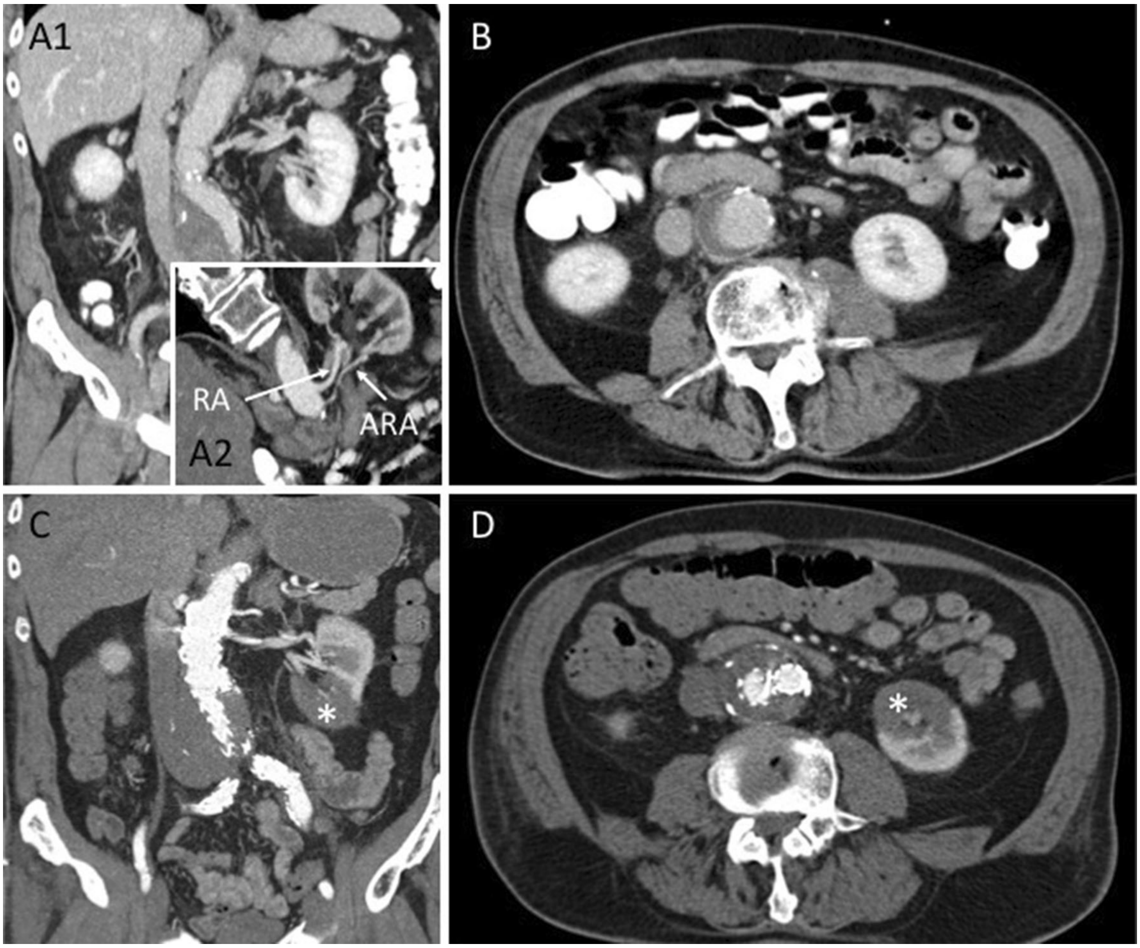

Fig. 2 CT imaging analysis of a patient with maximal aortic diameter 5.1 cm, Endurant device (Medtronic, Inc., Minneapolis, Minnesota, USA). We analyzed preoperative CT images (A1; A2; B) and discovered an ARA supplying the inferior segment of the left kidney. Postoperative CT images (C; D) showed a renal infarction (*) due to ARA coverage by the stent-graft device. Abbreviations: renal artery (RA); accessory renal artery (ARA)

3.5.2 (R Foundation for Statistical Computing, www.r-project.org) [19].

Results

Incidence of Accessory Renal Arteries

Sixty-five of 181 patients (35.9%) had 82 accessory renal arteries. One accessory renal artery was found in 51 patients (28.2%), two in 12 patients (6.6%), three in one patient (0.6%), and four in one patient (0.6%). In 27 patients (14.9%), ARAs arose above the main renal artery, in 14 patients (7.7%) at the level of the main renal artery and in 35 patients (19.3%) below the main renal artery. The mean diameter of all ARA was 0.27 cm (range 0.12–0.45 cm).

Coverage of ARA due to Stent-Graft

In 19 patients (10.5%) 20 accessory renal arteries were occluded by the aortic stent-graft (ARA covered). In the remaining 162 patients (89.5%), the renal arterial blood supply was completely preserved after EVAR (preserved renal arterial blood supply). No coverage occurred if the ARA arose below renal arteries but above the aortic stent-graft.

Acute Kidney Injury After EVAR

Overall 16 of 181 patients (9.9%) developed AKI after EVAR (Table 1). There was no significant impact of ARA covered after EVAR ($p = 0.785$). Considering the amount of contrast media applied during EVAR ($p = 0.083$) or during EVAR, preoperative and postoperative CT imaging ($p = 0.169$) had no significant impact on AKI. The only

Table 1 Acute kidney injury

	No AKI after EVAR (n = 165)	AKI after EVAR (n = 16)	Odds ratio	p Value
General characteristics				
Age (years)	70.87 (SD ± 8.85)	74.75 (SD ± 9.1)	1.06 (95% CI 0.99,1.13)	.099
Gender			0.72 (95% CI 0.04, 3.98)	.758
Male	151 (91%)	15 (9%)		
Female	14 (93%)	1 (7%)		
Current smoker	32 (97%)	1 (3%)	0.28 (95% CI 0.02, 1.45)	.222
Hypertension	116 (89%)	14 (11%)	2.96 (95% CI 0.79, 19.28)	.162
Diabetes	36 (92%)	3 (8%)	0.83 (95% CI 0.18, 2.74)	.776
Hyperlipidemia	59 (94%)	4 (6%)	0.6 (95% CI 0.16, 18.81)	.393
Hyperuricemia	8 (100%)	0	**	
Aneurysm diameter (cm)	5.46 (SD ± 1.01)	5.8 (SD ± 1.38)	1.33 (95% CI 0.83,2.07)	.213
ARA				
ARA detected before EVAR	59 (91%)	6 (9%)	1.08 (95% CI 0.35, 3.05)	.890
Mean number of ARA before EVAR	0.45 (SD ± 0.68)	0.50 (SD ± 0.82)	1.11 (95% CI 0.5, 2.12)	.777
ARA covered after EVAR	17 (89%)	2 (11%)	1.24 (95% CI 0.19, 4.98)	.785
Mean number of covered ARA	0.11 (SD ± .33)	0.12 (SD ± .34)	***	
Diameter of the largest ARA covered (cm)	0.29 (SD ± 0.08)	0.18 (SD ± .01)	***	
Absolute renal infarct volume (cm^2)	30.35 (SD ± 17.14)	2.94 (SD ± .12)	***	
Relative renal infarct volume (%)	8.41 (SD ± 4,02)	1.2 (SD ± .11)	***	
Renal function before EVAR				
Impaired renal function before EVAR	66 (87%)	10 (13%)	2.5 (95% CI .89, 7.66)	.090
eGFR before EVAR (ml/min/1.73 m^2)	65.17 (SD ± 17.23)	56.88 (SD ± 19.8)	0.97 (95% CI 0.93,1)	.074
Serum creatinine before EVAR (mg/dl)	0.98 (SD ± 0.22)	1.12 (SD ± 0.36)	1.23 (95% CI 1.02, 1.49)#	.030*
Contrast media				
During EVAR (angiography) (ml)	230.6 (SD ± 93.55)	274.06 (SD ± 96.49)	1.05 (95% CI 0.99, 1.10)##	.083
Preoperative Angio-CT, during EVAR and postoperative Angio-CT (ml)	465.96 (SD ± 112.66)	506.56 (SD ± 103.74)	1.03 (95% CI 0.99, 1.08)##	.169

#OR calculated per 0.1 units change

##OR calculated per 10 units change

*Significant

**No odds ratio calculable due to quasi-separated data

***No odds ratio calculable due to insufficient number of events

significant predictor on AKI was serum creatinine before EVAR (1.12 mg/dl vs. 0.98 mg/dl; $p = 0.030$).

Chronic Kidney Disease After EVAR

For long-term observation, all patients with follow-up controls for at least 3 months after EVAR were included ($n = 121$). The mean follow-up time of these patients was 24.7 months (± 32,7 months) after EVAR. Finally, 65 of 121 patients (53.7%) developed CKD after EVAR (Table 2). There was no significant impact of ARA covered after EVAR ($p = 0.452$), mean number of covered ARA ($p = 0.363$), absolute ($p = 0.131$), and relative ($p = 0.262$) renal infarct volume. Significant predictors on CKD are preoperative impaired renal function ($p < 0.001$), eGFR ($p < 0.001$), serum creatinine before EVAR ($p < 0.001$), and the diameter of the largest ARA covered ($p = 0.049$). The amount of contrast media applied during EVAR ($p = 0.560$) or during preoperative, postoperative Angio-CT and EVAR ($p = 0.828$) showed no significant impact on the occurrence of CKD.

Table 2 Chronic kidney disease

	No CKD after EVAR (n = 56)	CKD after EVAR (n = 65)	Odds ratio	p Value
ARA				
ARA detected before EVAR	20 (39%)	31 (61%)	1.64 (95% CI 0.79, 3.44)	.185
Mean number of ARA before EVAR	0.43 (SD ± 0.63)	0.62 (SD ± 0.78)	1.47 (95% CI 0.88, 2.56)	.159
ARA covered after EVAR	6 (38%)	10 (62%)	1.52 (95% CI 0.52, 4.73)	.452
Mean number of covered ARA	0.11 (SD ± 0.31)	0.17 (SD ± 0.42)	1.6 (95% CI 0.6, 4.75)	.363
Diameter of the largest ARA covered	0.35 (SD ± 0.06)	0.25 (SD ± 0.07)	0.12 (95% CI 0.01, 0.99)[#]	.049*
Absolute renal infarct volume (cm^2)	38.12 (SD ± 16.68)	21.76 (SD ± 19.93)	0.95 (95% CI 0.90, 1.01)	.131
Relative renal infarct volume (%)	9.51 (SD ± 4.19)	6.7 (SD ± 5.08)	0.88 (95% CI 0.69, 1.11)	.262
Renal function				
Impaired renal function before EVAR*	5 (11%)	42 (89%)	18.63 (95% CI 7.04,59.5)	< .001*
eGFR before EVAR (ml/min/1.73 m^2)	75.45 (SD ± 14.51)	56.65 (SD ± 12.76)	0.9 (95% CI 0.86,0.93)	< .001*
Serum creatinine before EVAR (mg/dl)	0.85 (SD ± 0.13)	1.08 (SD ± 0.2)	2.49 (95% CI 1.76,3.52)[#]	< .001*
AKI	2 (25%)	6 (75%)	2.75 (95% CI 0.6,19.29)	.228
Contrast media (ml)				
During EVAR (angiography)	228.79 (SD ± 101.37)	239.23 (SD ± 96.38)	1.01 (95% CI 0.98, 1.05)[##]	.560
Preoperative Angio-CT, during EVAR and postoperative Angio-CT (ml)	464.41 (SD ± 125.97)	469.08 (SD ± 111.8)	1.00 (95% CI 0.97, 1.03)[##]	.828

[#]OR calculated per 0.1 units change

[##]OR calculated per 10 units change

*Significant

Discussion

We found that ARA coverage did neither lead to a significant decline in renal function immediately after EVAR (p = 0.785) nor to a significant decline in renal function in long-term observation (p = 0.452). However, in patients with preoperative impaired serum creatinine, AKI was observed more frequently. Impaired renal function is supposed to be a risk factor for CKD ($p < 0.001$). No correlation between the absolute (p = 0.131) and relative (p = 0.262) renal infarct volume was found. Nevertheless, the diameter of ARA covered had an impact on the occurrence of CKD. One of our study's strengths was the measurement of laboratory values which provided comparable laboratory parameters over a period of more than 10 years. Another strength was that we carried out precise anatomical data acquisition by having two readers performing radiological analysis.

Greenberg et al. compared 40 patients having ARA covered with 29 patients having ARA preservation and found no significant difference regarding renal insufficiency and ARA size [6]. Karmacharya et al. drew the conclusion that ARA coverage in 45 cases was not clinically relevant since no significant difference in serum creatinine or creatinine clearance was discovered [7]. Kim et al. examined 32 patients with accessory renal artery and finally recommended to cover accessory renal arteries in patients with normal renal function in order to ensure a proximal seal of the stent-graft [8]. Malgor et al. evaluated renal function by using serum creatinine and eGFR. They agreed with previous mentioned studies that ARA coverage during EVAR can be seen as safe method having no impact on renal function. Follow-up controls up to 37 months were included [9]. Dzieciuchowitz et al. discovered a temporary decline of renal function after coverage of ARAs in six patients from which patients recovered within 90 days. They stated that ARA coverage seems to be a safe procedure [11]. The latest study from 2017 by Ikeda et al. found a significant change in renal function (p = 0.0001) in case of occlusion of ARA during EVAR. In this study renal function was evaluated using serum creatinine values collected short-term after EVAR (up to 3 days) [12]. Sadeghi et al. figured out a distinct decline in eGFR comparing patients with covered and without covered ARA one week and six months after EVAR. In particular, patients with normal renal function before EVAR showed a decrease in eGFR after ARA coverage [10], whereas in our study, we found that preoperative higher serum creatinine values correlated with incidence of AKI (0.98 mg/dl vs 1.12 mg/dl; p = 0.03). Impaired renal function, serum creatinine, and eGFR before EVAR correlated with the occurrence of CKD ($p < 0.001$). Current guidelines suggest preserving

ARAs with a diameter > 3 mm and ARAs supplying at least one-third of the renal parenchyma [20–22] or suggest covering small ARA if necessary [23]. Greenberg et al. already found out that glomerular filtration rate values do not correlate with the diameter of covered ARAs [6]. Long-term observation showed that CKD as of 3 months after EVAR occurred more often in patients with smaller covered ARA (0.35 cm vs. 0.25 cm; $p = 0.049$). However, absolute and relative renal infarct volume did not relate significantly with incidence of CKD ($p = 0.131$ and $p = 0.262$). A further factor that was supposed to have an impact on renal function is the amount of contrast media used during the intervention. Ikeda et al. considered contrast dose to be a predictor of renal deterioration after TEVAR ($p = 0.042$) but not after EVAR ($p = 0.48$) [12]. In our cohort, the amount of contrast media during EVAR did not correlate with the incidence of AKI (230.6 ml vs. 274.06 ml; $p = 0.083$) or CKD (228.79 ml vs. 239.23 ml; $p = 0.56$).

According to our study results, coverage of accessory renal arteries in patients with infrarenal AAA is an individual decision but can be performed if necessary. Nevertheless, renal function of every patient should be measured and controlled, so that patients with renal dysfunction can be recognized early in order to counteract chronic renal decompensation and pay attention to possible later complications such as acute on chronic renal failure.

The substantial limitation of our study is its retrospective design. Patients were excluded because of missing values or imaging which lead to a reduced number of cases. Besides, follow-up examinations for long-term observation were variable which could lead to selection bias in follow-up examinations. However, the number of patients was equivalent to current studies. In addition, included patients had to fulfill strict criteria minimizing disruptive factors. Apart from this, the only indicator to evaluate renal function is serum creatinine and eGFR. Oliguria and urine output were not taken into account. SCr, as well as eGFR, is influenced by renal independent variables, nutritional status, for example [24]. In the future, other renal biomarkers could enable more precise information value [25].

Conclusion

There has been contradicting evidence regarding the coverage of ARA influencing renal function. In this cohort, we found that ARA coverage did neither lead to a significant decline in renal function immediately after EVAR nor a considerable decline in renal function in long-term observation. ARA coverage during EVAR can be a possible approach as an individual treatment decision in patients with infrarenal AAA.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical Approval Approval from the local ethics committee of the University Hospital Regensburg was obtained. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. For this type of study, formal consent is not required.

Informed Constant This study has obtained IRB approval from the Local Ethics Committee of the University Hospital Regensburg, and the need for informed consent was waived.

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