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CARDIOVASCULAR IMAGING
ORIGINAL ARTICLE

Risk factors associated with late aneurysmal sac expansion after endovascular abdominal aortic aneurysm repair

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PURPOSE
We aimed to identify the risk factors associated with late aneurysmal sac expansion after endovascular abdominal aortic aneurysm repair (EVAR).

METHODS
We retrospectively reviewed contrast-enhanced computed tomography (CT) images of 143 patients who were followed for ≥6 months after EVAR. Sac expansion was defined as an increase in sac diameter of 5 mm relative to the preoperative diameter. Univariate and multivariate analyses were performed to identify associated risk factors for late sac expansion after EVAR from the following variables: age, gender, device, endoleak, antiplatelet therapy, internal iliac artery embolization, and preprocedural variables (aneurysm diameter, proximal neck diameter, proximal neck length, suprarenal neck angulation, and infrarenal neck angulation).

RESULTS
Univariate analysis revealed female gender, endoleak, aneurysm diameter ≥60 mm, suprarenal neck angulation >45°, and infrarenal neck angulation >60° as factors associated with sac expansion. Multivariate analysis revealed endoleak, aneurysm diameter ≥60 mm, and infrarenal neck angulation >60° as independent predictors of sac expansion ($P < 0.05$, for all).

CONCLUSION
Our results suggest that patients with small abdominal aortic aneurysms (<60 mm) and infrarenal neck angulation ≤60° are more favorable candidates for EVAR. Intraprocedural treatments, such as prophylactic embolization of aortic branches or intrasac embolization, may reduce the risk of sac expansion in patients with larger abdominal aortic aneurysms or greater infrarenal neck angulation.

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The aim of endovascular abdominal aortic aneurysm repair (EVAR) is to prevent rupture of an abdominal aortic aneurysm (AAA) by depressurizing the aneurysm and excluding it from the systemic circulation using a stent-graft. Aneurysmal sac reduction is a reliable marker for the long-term prognosis after EVAR. Although most aneurysmal sacs shrink after EVAR, some sacs continue to expand. A relationship between aneurysm size and endoleaks was previously reported (1, 2). Most type II endoleaks spontaneously disappear over time, but 10%–25% persist for more than six months after EVAR (3–6). Persistent endoleaks with aneurysmal sac expansion are at high risk of rupture because of the continuously elevated intra-aneurysmal pressure and require a second intervention, such as embolization (7–11). However, it is difficult to predict sac expansion and persistent endoleak before performing EVAR. Although intraoperative intrasac thrombin injection and prophylactic embolization of aortic branches such as the inferior mesenteric artery and lumbar artery are reported to reduce the incidence of type II endoleak, the efficacy and clinical benefit of these procedures in terms of late postoperative aneurysm shrinkage have not been fully evaluated (12–15). Therefore, the purpose of this study was to identify the risk factors associated with late aneurysmal sac expansion after EVAR to determine possible indications for intrasac embolization and prophylactic embolization of aortic branches.

Methods

Study design and patients

This clinical study was performed with the approval of our Institutional Ethics Committee. All patients were treated at a single institution. A total of 183 patients with AAA underwent elective EVAR with commercially available bifurcated stent-graft devices between February 2008 and February 2014. The clinical data were maintained on electronic medical records. Patients were selected based on the anatomical indication criteria stipulated in the instructions for use (IFU) of the available endovascular devices. The IFU criteria were as follows: proximal neck (PN) length, ≥15 mm; PN diameter, ≥18 mm and ≤32 mm; suprarenal neck angulation, ≤45°; infrarenal neck angulation, ≤60°; no large thrombi in the sealing zone; no highly calcified stenotic access route (iliac arteries). Included in the study were 75 patients who, despite not meeting the IFU criteria, underwent EVAR because they were unsuitable candidates for open repair, mainly due to the presence of comorbidities, as well as hostile abdomen, unfitness for general anesthesia, and high risk of rupture.

Of 183 patients, 143 patients underwent regularly scheduled surveillance with contrast-enhanced computed tomography (CT) for ≥6 months

after EVAR. The other 40 patients were excluded from the study, either because contrast-enhanced CT follow-up could not be performed due to renal insufficiency, or because less than six months had elapsed after EVAR. This follow-up was performed at six months after EVAR and yearly thereafter, to measure aneurysm diameter and detect endoleaks. We reviewed all contrast-enhanced CT images obtained for the patients included in this retrospective study. The patients received a detailed explanation of the purpose and design of this study, and gave informed consent to participate. Sac expansion was defined as a 5 mm increase in sac diameter relative to the preoperative diameter, while sac reduction was defined as a decrease in sac diameter of 5 mm.

Of patients who received full follow-up with contrast-enhanced CT, 27.3% (39/143) were regarded as outside the IFU criteria (Table 1). Overall, 70 patients received antiplatelet therapy and 56 patients underwent internal iliac artery (IIA) embolization. No patient had a conduit or alternative access. There were no cases of rupture, graft infection, conversion to open repair, or graft-related deaths in the follow-up period. Secondary interventions were performed in 14 patients. Eleven patients underwent embolization of a type II endoleak. Type II endoleaks were treated by transcatheter arterial embolization or direct sac embolization if the endoleak persisted for >12 months, aneurysm sac expansion was ≥10 mm, and the patient gave consent for embolization. Patient outcomes following the secondary interventions were unavailable for the current analyses. CT images obtained in the last follow-up and clinical data obtained in the follow-up period before secondary interventions were used in this study. Graft migration occurred in one patient who underwent additional placement of an iliac extender. Limb occlusion occurred in two patients, who subsequently underwent endovascular thrombolysis and bare-stent placement to achieve complete recanalization.

Table 1. Patient characteristics

	n=143
Age (years)	76.5±7.25
Gender	
Male	119 (83.2)
Female	24 (16.8)
Within the IFU	104 (72.7)
Outside the IFU	39 (27.3)
PN diameter (mm)	20.2±3.25
PN length (mm)	29.9±10.3
Suprarenal neck angulation (°)	26.3±17.6
Infrarenal neck angulation (°)	48.7±17.4
Aneurysm diameter (mm)	51.8±7.9
Device used	
Zenith®	74 (51.7)
Excluder®	58 (40.6)
Powerlink®	11 (7.7)
Antiplatelet therapy	70 (49.0)
IIA embolization	56 (39.2)
Unilateral	40
Bilateral	16
Endoleaks immediately after EVAR (CTDA)	89 (62.2)
Type I	5 (3.5)
Type II	84 (58.7)
Type III and IV	0
Secondary intervention	14 (9.8)
Embolization for type II endoleak	11
Additional SG placement for graft migration	1
Endovascular thrombolysis for limb occlusion	2
Follow-up time (months), median (mean±SE)	12 (18.7±1.08)

Data are presented as n (%) or mean±standard deviation.
IFU, instructions for use; PN, proximal neck; IIA, internal iliac artery; EVAR, endovascular abdominal aortic aneurysm repair; CTDA, CT during aortography; SG, stent-graft; SE, standard error.

Endovascular abdominal aortic aneurysm repair

Before EVAR, all patients underwent contrast-enhanced CT using a multidetector CT scanner (LightSpeed® VCT 64-slice CT; GE Healthcare) and 1.25 mm thick sections were sent to a workstation (Aquarius iNtuition viewer; Tera Recon. Inc.) to measure the maximum aneurysm diameter, PN diameter, PN length, and neck angulation before EVAR (Table 1). These prospectively collected data were recorded and stored, and were used in this study.

The EVAR procedure was performed via the femoral artery under epidural anesthesia in the interventional radiology suite. We used the following bifurcated stent-grafts: Zenith® AAA Endovascular Graft (Cook Medical Inc., n=74), Excluder® AAA Endoprosthesis (W. L. Gore & Associates, Inc., n=58), and Powerlink® (Endologix Inc., n=11). EVAR was successfully performed on all patients. Immediately after EVAR, digital subtraction angiography and CT during aortography (CTDA) were conducted to investigate the absence or presence of endoleaks. CTDA was conducted using an interventional radiology-CT system with a 64-multislice CT scanner (Infinix Celeve™-I

and Aquilion™ CX; Toshiba Medical Systems). The following parameters were used: helical pitch, 53; slice thickness acquisition, 0.5 mm; image interval, 0.5 mm; rotation time, 0.5 s; reconstruction slice thickness, 5 mm; and scanning time, approximately 5 s. The CTDA procedure was performed as follows: a 4 F pigtail catheter (Cook Medical Inc.) was inserted from the left brachial artery, with the tip of the catheter placed at the level of the superior mesenteric artery. A total of 70 mL of contrast medium (Iopamiron® [iopamidol] 300; Bayer AG) diluted three-fold with saline was infused at 5 mL/s using an automatic infuser. Biphasic CTDA was performed for the region from the bifurcation of the superior mesenteric artery to the pelvis; the first phase started 8 s after contrast infusion was initiated, and the second phase started 8 s after the first. The presence or absence of endoleaks was confirmed in both phases.

Type II endoleak was visualized in 38 patients on digital subtraction angiography and in 84 patients on CTDA immediately after EVAR (Table 1). Minor type I endoleak was observed in five patients immediately after EVAR. All type I endoleaks disappeared three months after EVAR. Type III and IV endoleaks were not seen.

Follow-up contrast-enhanced CT

All patients underwent regularly scheduled surveillance with three-phase CT for ≥6 months after EVAR. CT was performed at six months after EVAR and yearly thereafter. All images were obtained using a multidetector CT scanner (LightSpeed® VCT 64-slice CT; GE Healthcare) with a 64×0.625 mm detector configuration, 0.625 mm detector-row width, and 0.5 s rotation time. Interspaced helical data sets were collected from 64 detector rows.

The three-phase CT protocol comprised unenhanced CT followed by contrast-enhanced CT in the arterial and delayed phases. All acquisitions were obtained during inspiratory breath-holds. Scanning was performed in the craniocaudal direction, from the upper margin of the liver to the lower border of the ischia at thin collimation (0.625 mm) and 120 kV with automatic tube current modulation. Axial images (5 mm thick) were sent to the picture archiving and communication system.

Using an automatic injector (Dual Shot GX; Nemoto Kyorindo Co.), each patient was intravenously administered with 100 mL of nonionic contrast medium containing 300 mg iodine/mL (Omnipaque® [iohexol] 300; Daiichi Sankyo Co.), at a rate of 3 mL/s through a 20-gauge angiocatheter placed in the antecubital vein. Automatic bolus tracking was achieved using SmartPrep software (GE Healthcare). Unenhanced scans of the whole abdomen were initially obtained and a region-of-interest cursor for bolus tracking was placed in the aorta at the level of the celiac axis. Arterial phase acquisition was performed after a trigger delay of 12 s after the trigger threshold value (set at 200 HU) was reached. Delayed phase images were automatically acquired 30 s after completion of the arterial phase. The scan duration was approximately 4–6 s in each phase. The endoleaks were monitored in both phases. The presence or absence of endoleaks was determined, and AAA sac diameter was measured on the CT images obtained at each follow-up visit. The last follow-up CT images were directly compared with the preoperative CT images obtained before EVAR.

Statistical analysis

Univariate and multivariate analyses were performed to identify possible clinical or anatomical predictors of sac expansion and reduction in the long-term follow-up. The predictive variables used for this analysis were age, gender, device used, presence of an endoleak, antiplatelet therapy, IIA embolization, and preprocedural variables (maximum aneurysm diameter, PN diameter, PN length, suprarenal neck angulation, and infrarenal neck angulation). PN diameter (≥18 mm vs. <18 mm), PN length (≥15 mm vs. <15 mm), and neck angulation (suprarenal: >45° vs. ≤45°; infrarenal: >60° vs. ≤60°) were classified according to the IFU criteria. Univariate analysis was performed with the Kaplan-Meier method followed by the log-rank test. Multivariate analysis was performed using the Cox proportional hazards model to determine the hazard ratio (HR) and 95% confidence interval (CI). The stepwise forward selection method was used to select variables included in the multivariate model. The log likelihood value and Akaike information criterion were used to find the model of best fit for the results of Cox regression analysis. In addition, chi-square test was used to compare categorical variables (incidences of sac expansion, sac reduction, sac stabilization, and type II endoleak) between patients whose stent-graft was used within the IFU, and those outside the IFU. Values, including the preprocedural variables, are presented as number and percentage or mean±standard deviation.

All statistical analyses were performed using Excel Statistical Analysis 2012 (Social Survey Research Information Co., Ltd.). Values of $P < 0.05$ were considered statistically significant.

Results

A total of 183 patients with AAA (151 men, 32 women; mean age, 76.5 years; range, 58–93 years) underwent elective EVAR. Of 183 patients, 143 (119 men, 24 women; mean age, 76.5 years; range, 58–93 years) were followed for ≥6 months after EVAR and their follow-up CT images were reviewed. The median follow-up time was 12 months (Table 1). Sac expansion was found in 22 patients, sac reduction in 44 patients, and sac stabilization in 77 patients (Table 2). A type II endoleak was found in 35 patients (24.5%) at their last follow-up examination, which included 68.2% of patients with sac expansion (15/22), 2.27% of patients with sac reduction (1/44), and 24.7% of patients with sac stabilization (19/77). Type I, III, and IV endoleaks were not found.

Of 12 variables assessed, univariate analysis revealed female gender ($P = 0.002$), presence of an endoleak ($P < 0.001$), maximum aneurysm diameter ≥60 mm ($P = 0.012$), suprarenal neck angulation >45° ($P = 0.001$), and infrarenal neck angulation >60° ($P < 0.001$) to be associated with sac expansion (Table 2). In multivariate analysis, the presence of an endoleak ($P < 0.001$; HR, 5.61; 95% CI, 2.18–14.45), maximum aneurysm diameter ≥60 mm ($P = 0.002$; HR, 6.30; 95% CI, 1.94–20.50), and infrarenal neck angulation >60°

Table 2. Univariate analysis of factors possibly associated with sac expansion or reduction

			Expansion		Reduction		Stabilized
		n	n (%)	*P*	n (%)	*P*	n (%)
Total		143	22 (15.4)		44 (30.8)		77 (53.8)
Age	≥80 years	57	11 (19.3)	0.260	12 (21.1)	0.115	34 (59.6)
	<80 years	86	11 (12.8)		32 (37.2)		43 (50.0)
Gender	Male	119	16 (13.4)	0.002	39 (32.8)	0.894	64 (53.8)
	Female	24	6 (25.0)		5 (20.8)		13 (54.2)
Device	Zenith®	74	10 (13.5)	0.350	28 (37.8)	0.258	36 (48.6)
	Excluder®	58	11 (19.0)		15 (25.9)		32 (55.2)
	Powerlink®	11	1 (9.1)		1 (9.1)		9 (81.8)
Endoleak	Yes	35	15 (42.9)	<0.001	1 (2.9)	<0.001	19 (54.3)
	No	108	7 (6.5)		43 (39.8)		58 (53.7)
Antiplatelet therapy	Yes	70	14 (20.0)	0.259	15 (21.4)	0.016	41 (58.6)
	No	73	8 (11.0)		29 (39.7)		36 (49.3)
IIA embolization	Yes	56	12 (21.4)	0.366	16 (28.6)	0.307	28 (50.0)
	No	87	10 (11.5)		28 (32.2)		49 (56.3)
Aneurysm diameter	<50 mm	89	14 (15.7)	0.829	26 (29.2)	0.766	49 (55.1)
	≥50 mm	54	8 (14.8)		18 (33.3)		28 (51.9)
Aneurysm diameter	<60 mm	120	16 (13.3)	0.012	38 (31.7)	0.856	66 (55.0)
	≥60 mm	23	6 (26.1)		6 (26.1)		11 (47.8)
PN diameter	<18 mm	25	5 (20.0)	0.872	11 (44.0)	0.549	9 (36.0)
	≥18 mm	118	17 (14.4)		33 (28.0)		68 (57.6)
PN length	<15 mm	3	0 (0)	0.573	1 (33.3)	0.801	2 (66.7)
	≥15 mm	140	22 (15.7)		43 (30.7)		75 (53.6)
Suprarenal neck angulation	≤45°	125	16 (12.8)	0.001	43 (34.4)	0.100	66 (52.8)
	>45°	18	6 (33.3)		1 (5.6)		11 (61.1)
Infrarenal neck angulation	≤60°	113	12 (10.6)	<0.001	39 (34.5)	0.293	62 (54.9)
	>60°	30	10 (33.3)		5 (16.7)		15 (50.0)

IIA, internal iliac artery; PN, proximal neck.

(*P* = 0.038; HR, 2.83; 95% CI, 1.06–7.58) were independent risk factors for sac expansion (Table 3). Age, device used, antiplatelet therapy, IIA embolization, PN diameter, and PN length were not associated with sac expansion.

Univariate analysis showed that the absence of an endoleak and the absence of antiplatelet therapy were associated with sac reduction (Table 2). Multivariate analysis showed that the presence of an endoleak and antiplatelet therapy were significantly related to a lack of reduction in aneurysm size (Table 4).

Of 104 patients within the IFU, sac expansion occurred in 11 patients (10.6%) and sac reduction occurred in 36 patients (34.6%). Of 39 patients outside the IFU, sac expansion occurred in 11 patients (28.2%) and sac reduction occurred in 8 patients (20.5%). The incidence of sac expansion was significantly different between patients within and outside the IFU (*P* = 0.009) (Table 5). A type II endoleak was found in 23.1% of patients within the IFU (24/104) and 28.2% of patients outside the IFU (11/39), which was not significantly different (*P* = 0.525).

Discussion

Endovascular repair aims to prevent aortic rupture. AAA sac expansion represents treatment failure because it leaves the patient at risk of death resulting from rupture. Aneurysmal sac reduction is a reliable marker for the prognosis of EVAR. In our study, we observed aneurysm sac expansion of ≥5 mm in 15.4% of patients and sac reduction or sac stabilization in 84.6% of patients after EVAR. Identifying predictors of sac stability or shrinkage can help reassure both the patient and the physician that the AAA is unlikely to rupture after EVAR. The patients in our study received three different types of stent-graft and the type of stent-graft was not associated with sac shrinkage or expansion.

In our study the incidence of sac expansion was significantly greater in patients outside the IFU than in

Table 3. Multivariate analysis of factors associated with sac expansion

			HR	95% CI	*P*
Gender (female vs. male)			2.63	0.85–8.07	0.092
Presence of endoleak (yes vs. no)			5.61	2.18–14.45	<0.001
Aneurysm diameter (≥60 mm vs. <60 mm)			6.30	1.94–20.50	0.002
Infrarenal neck angulation (>60° vs. ≤60°)			2.83	1.06–7.58	0.038
	Log likelihood value	AIC	Chi-square	df	*P*
Sac expansion	–77.35	162.71	39.88	4	0.0000

HR, hazard ratio; CI, confidence interval; AIC, Akaike information criterion; df, degrees of freedom.

Table 4. Multivariate analysis of factors associated with sac reduction

			HR	95% CI	*P*
Age (≥80 years vs. <80 years)			0.56	0.29–1.08	0.083
Presence of endoleak (yes vs. no)			0.061	0.0084–0.446	0.006
Antiplatelet drug (yes vs. no)			0.51	0.27–0.97	0.039
	Log likelihood value	AIC	Chi-square	df	*P*
Sac reduction	–180.74	367.48	21.91	3	0.0001

HR, hazard ratio; CI, confidence interval; AIC, Akaike information criterion; df, degrees of freedom.

Table 5. Incidence of sac expansion, reduction, or stabilization and endoleaks between patients within and outside the IFU

Group	Total n=143	Within the IFU n=104	Outside the IFU n=39	*P*
Sac				
Expansion	22	11 (10.6)	11 (28.2)	0.009
Reduction	44	36 (34.6)	8 (20.5)	0.104
Stabilized	77	57 (54.8)	20 (51.3)	0.706
Endoleak				
Presence of an endoleak	35	24 (23.1)	11 (28.2)	0.525

Data are presented as n (%).
IFU, instructions for use.

patients within the IFU (28.2% vs. 10.6%). Therefore, the sac diameter should be regularly monitored after EVAR if the stent-graft is used outside the IFU. The presence of an endoleak after EVAR is of great concern. If the endoleak is accompanied by sac expansion, continuous intra-aneurysmal pressure increases the risk of AAA rupture (7, 8). It was reported that most endoleaks, initially identified as a type II endoleak, spontaneously seal within several months (3, 4). In our study, a type II endoleak occurred in 58.7% of patients immediately after EVAR, and decreased to 24.5% of patients at the last follow-up examination. Of note, a type II endoleak occurred in 68.2% of patients with sac expansion compared with 2.27% of patients with sac reduction. The univariate analysis also revealed that the presence of an endoleak was associated with sac expansion. Therefore, persistent endoleaks are strongly associated with sac expansion. Ward et al. (14) reported that preoperative embolization of the inferior mesenteric artery was associated with lower incidences of type II endoleaks and aneurysm sac enlargement. However, based on our data, we are unable to predict which types of type II endoleaks are important in terms of sac expansion or require preprocedural or intraprocedural embolization to minimize the risk of late sac expansion. Nevertheless, we found that preoperative anatomical factors and a persistent endoleak after EVAR were associated with sac expansion.

In univariate analysis, female gender, presence of an endoleak, aneurysm diameter ≥60 mm, suprarenal neck angulation >45°, and infrarenal neck angulation >60° were associated with sac expansion. However, because the presence of an endoleak might be a confounding factor in these associations, we performed multivariate analysis using the stepwise forward selection method to eliminate potential confounding factors. Based on the results of this multivariate analysis, we concluded that the presence of an endoleak, maximum aneurysm diameter ≥60 mm, and infrarenal neck angulation >60° were independent risk factors for sac expansion. These findings suggest that patients with AAAs <60 mm in diameter and infrarenal neck angulation ≤60° are more favorable candidates for EVAR. In patients with larger AAAs or greater infrarenal neck angulation, preprocedural or intraprocedural treatments such as prophylactic embolization of aortic branches or intrasac embolization could potentially reduce the risk of sac expansion.

Some research groups have tried to identify clinical and anatomical factors associated with sac shrinkage or expansion after EVAR; however, it remains inconclusive whether AAA diameter is involved in sac reduction or expansion. In a study of 100 patients with AAA, Yeung et al. (16) reported minimal thrombus and greater AAA diameter as independent predictors of sac regression, and the presence of neck plaque and endoleak as independent predictors of sac expansion at one, six, and 12 months after EVAR (all $P < 0.05$). Boult et al. (17) reported that the predictors of clinical failure or need for reintervention included greater AAA diameter, neck angulation ≥45°, and short infrarenal neck. The results of the present study revealed maximum aneurysm diameter ≥60 mm as a significant and independent predictor of sac expansion after EVAR. Schanzer et al. (18) reported that independent predictors

of AAA sac enlargement included endoleak, age ≥80 years, aortic neck diameter ≥28 mm, aortic neck angle >60°, and common iliac artery diameter >20 mm in their multi-institutional study. Our single institution study also revealed the presence of an endoleak and infrarenal neck angulation >60° as significant and independent predictors of sac expansion.

In a study of 57 patients with AAA, Aoki et al. (19) reported multi-agent antiplatelet therapy and type II endoleaks to be significantly associated with the absence of aneurysm shrinkage at six months after EVAR. They defined aneurysm shrinkage as a decrease in sac diameter of ≥4 mm. Thrombus formation in an aneurysm sac depends on the relationship between coagulation and fibrinolysis. Antiplatelet therapy suppresses blood coagulation and thrombus formation in the sac and may prevent thrombus organization. In our study, antiplatelet therapy was not predictive of sac expansion, while the absence of antiplatelet therapy was predictive of sac reduction, and the presence of antiplatelet therapy was significantly related to a lack of reduction in aneurysm size.

In the present study, follow-up imaging after EVAR was routinely performed with contrast-enhanced CT, which has a reported sensitivity as high as 92% (20). Contrast-enhanced CT cannot identify all endoleaks; aneurysm sac expansion without endoleak visualized on contrast-enhanced CT is termed endotension. Endotension may represent a microleak or a low-flow endoleak, which are undetectable by contrast-enhanced CT (21, 22). Other imaging modalities, including ultrasonography, magnetic resonance imaging, and scintigraphy (23), may be able to reveal these types of endoleaks. The sensitivity of contrast-enhanced ultrasonography for detecting endoleaks was reported to be 81%–98% (24, 25). Magnetic resonance imaging was also reported as being more sensitive than CT angiography for the detection of endoleaks (26, 27).

Our study has several limitations. It was a retrospective study and the follow-up times were not the same for all patients. Furthermore, several potentially important variables, including inferior mesenteric artery and lumbar artery diameter (28), and neck calcification were not included in the analyses. Another limitation is that AAA volume may be a more accurate predictor than diameter for sac expansion or regression after EVAR and may have been more appropriate as an objective standard (29). However, it is very difficult and labor-intensive to analyze thin-slice CT images obtained at each follow-up using a dedicated three-dimensional workstation to provide reliable results, and to measure the sac volume at each follow-up. Therefore, the sac diameter may be a better standard for long-term follow-up, because it is easy to measure and compare by follow-up CT angiography.

In conclusion, the presence of an endoleak, maximum aneurysm diameter ≥60 mm, and infrarenal neck angulation >60° were independent risk factors associated with late sac expansion after EVAR. Patients with AAAs <60 mm in diameter and infrarenal neck angulation ≤60° may be favorable candidates for EVAR. To reduce the risk of sac expansion in patients with larger AAAs or greater infrarenal neck angulation, preprocedural or intraprocedural treatments such as prophylactic embolization of aortic branches or intrasac embolization may be beneficial.

Conflict of interest disclosure

The authors declared no conflicts of interest.

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