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ORIGINAL ARTICLE

Perioperative factors associated with aneurysm sac size changes after endovascular aneurysm repair

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Abstract

Purpose To identify the perioperative factors associated with aneurysm size changes after endovascular aortic aneurysm repair (EVAR).

Methods Between August, 2008 and December, 2014, 187 patients underwent EVAR treatment in our institution. The subjects of this study were 135 of these patients without peripheral artery disease, who were followed up with computed tomography (CT) for 3 years. Significant aneurysm size change was defined as sac size change of more than 5 mm from the baseline.

Results Sac enlargement was identified in 25 patients (18.5%) and sac shrinkage was identified in 59 (43.7%) patients. The factors associated with sac enlargement were postoperative pulse wave velocity (OR: odds ratio 3.80, $p=0.047$), prevalence of a type 2 endoleak 1 week after surgery (OR 4.26, $p=0.022$), inner diameter (OR 1.10, $p=0.005$), and distance from the lower renal artery to the terminal aorta (OR 1.05, $p=0.017$). The factors associated with sac shrinkage were prevalence of a type 2 endoleak (OR 0.09, $p<0.001$) and preoperative pulse wave velocity (OR 0.32, $p=0.022$). The factors independently associated with type 2 endoleak were the use of an Excluder device (OR 3.99, $p=0.002$) and the length of the aneurysm (OR 1.02, $p=0.027$).

Conclusion Inner diameter, treatment length, perioperative pulse wave velocity, and type 2 endoleak were associated with sac size changes after EVAR.

Keywords EVAR · Type 2 endoleak · Pulse wave velocity · Enlargement · Shrinkage

Introduction

Endovascular repair of an abdominal aortic aneurysm is minimally invasive with lower perioperative morbidity and mortality than conventional open surgical repair [1, 2]. However, it is also associated with a risk of complications and the need for re-intervention over time, with early changes in the aneurysm sac diameter reported as a strong predictor of this [3]. Based on the uncertainty of long-term outcomes, the postoperative surveillance of patients undergoing endovascular aortic aneurysm repair (EVAR) is also debatable with regards to costs and the use of radiation and iodine contrasts [3].

The prevalence of endoleak has been reported as an independent risk factor for aneurysm sac enlargement [4–6]. Other potential risk factors have been suggested, including age, thrombus formations, medications, and comorbidities such as hyperlipidemia and coronary artery disease [7–10]. In the current clinical practice, patients with sac enlargement must be followed up closely to evaluate the need for re-intervention and risk of aneurysm rupture. The aim of this study was to evaluate the perioperative factors associated with sac enlargement and shrinkage after EVAR.

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Methods

Patients

Between August, 2008 and December, 2014, 187 patients underwent EVAR in our institution, 144 of whom were followed up with computed tomography (CT) for more than 3 years after surgery. Seventeen patients who died within the 3 years, 23 patients who were transferred to another hospital for postoperative follow-up or were admitted to a nursing home, three patients who had undergone prior conventional treatment, and three patients with traumatic injury of the aorta were excluded from the analysis. Four patients with an ABI < 0.9 were also excluded from the study because of the possibility of underestimated pulse wave velocity (PWV) measurement (Fig. 1). Enhanced CT was performed preoperatively and 1 week after EVAR. For follow-up, CT was performed annually thereafter, with contrast for patients with a persistent endoleak 1 week after surgery and those without kidney injury. Re-intervention was considered for patients with sac enlargement of more than 10 mm from the baseline or those with sac enlargement of 5 mm within 6 months. This study was approved by the Institutional Review Board of Saitama Medical Center, Jichi Medical University (S17-143).

CT measurement

Anatomical measurement of the aortic aneurysm was performed with Ziostation (Ziosoft, Inc. Tokyo, Japan). Using 3D multi-planar view, all measurements were performed in the axial view. Maximum aortic diameter, length of the aorta from the lower renal artery to the terminal aorta, neck length, number of persistent lumbar arteries, and inferior mesenteric arteries (IMA) were assessed before surgery. The existence of endoleak and the maximum aortic diameter were assessed 1 week after surgery, and the maximum aortic diameter was measured annually.

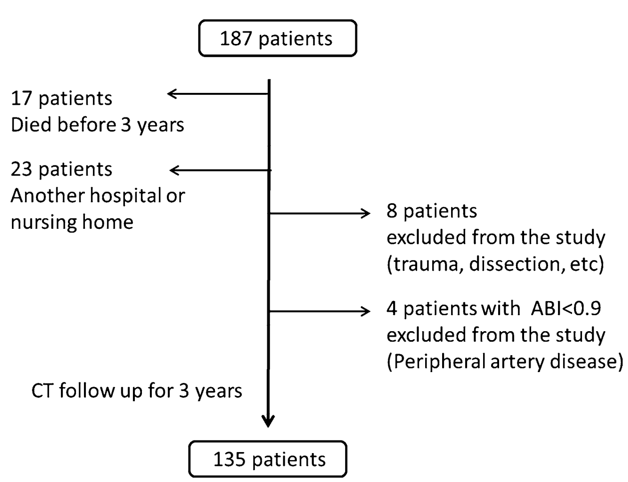

Fig. 1 Patients enrolled in the study. Patients who died within 3 years after surgery, those who were transferred to another hospital or nursing home and did not participate in follow-up, patients with dissection or trauma, and those with peripheral artery disease were excluded from the study

PWV measurement

The baPWV measurements were performed using a non-invasive device (Omron Colin, Tokyo, Japan). Pressure-sensitive transducers were placed on the arm and ankle and simultaneous recording of the two different pulse waves were measured at each point. PWV was calculated by the distance between the transducers over the body surface and the pulse transit time. [PWV (m/s) ¼ travel distance (m)/transit time (s)] [11, 12].

Data analysis

The normality of the distribution of the data was tested by the Kolmogorov–Smirnov test. To compare three groups, ANOVA was used to analyze continuous variables that were normally distributed (mean ± SD). The Kruskall–Wallis test was used if data were not normally distributed after logarithmical transformation (median, Q1-Q3). The Bonferroni post hoc test was performed for multiple comparisons. To compare two groups, continuous variables were analyzed by the Student *t* test (mean ± SD) or Mann–Whitney test, if the data were not normally distributed after logarithmical transformation (median, Q1–Q3). Categorical variables were analyzed by Fisher's exact test. Significant sac size change was defined as a decrease or increase in sac sizes of more than 5 mm from the baseline. Patients were divided into the following three groups: those with no significant change (less than 5 mm) in the sac diameter; those with significant sac shrinkage; and those with significant sac enlargement. The multivariate regression model for significant sac changes was assessed by adjusting for factors associated with aneurysm sac size changes in the univariate analysis. Since pulse wave velocity is an intermediate variable for age and aneurysm sac size changes, age was not included in the multivariate analysis when preoperative pulse wave velocity was used. $p < 0.05$ was considered significant and all analyses were performed using Stata (version 13.1, Stata Corp, College Station, TX, USA).

Results

The commercially available devices used for treatment were the Excluder (W.L. Gore & Associates, Inc. Flagstaff, AZ, USA) ($n = 39$), the Endurant (Medtronic, Minneapolis, MN, USA) ($n = 55$), the Zenith (Cook Medical, Bloomington, IN, USA) ($n = 32$), and the Powerlink (Endologix Inc, Irvine, CA, USA) ($n = 6$). Three patients were treated with a combination of the Endurant and Excluder devices and were categorized as "others" ($n = 3$). The only endoleak observed 1 week after EVAR was a type 2 endoleak and there were no patients with a thoracic aortic aneurysm. Table 1 shows the demographics of the patients with no significant change (37.8%), those with significant sac shrinkage (43.7%), and those with significant sac enlargement (18.5%), 3 years after EVAR. The patients with sac enlargement were significantly older than those with sac shrinkage. (80 ± 7.6 years vs. 75 ± 77 years, $p = 0.019$). There were no significant differences in prior medical history, medication, or laboratory data. Patients with sac enlargement had a longer distance between the lower renal artery and the terminal

Table 1 Demographics of patients with no significant sac size change, sac shrinkage, or sac enlargement 3 years after endovascular aortic aneurysm repair (EVAR)

	No change $n = 51$	Shrinkage $n = 59$	Enlargement $n = 25$	p
Age (years)	78 ± 6.1	75 ± 7.7	80 ± 7.6	0.011
Male (%)	44 (86.3%)	52 (88.1%)	19 (76.0%)	0.33
Medical history				
Hypertension (%)	37 (72.5%)	45 (76.3%)	18 (72.0%)	0.90
Dyslipidemia (%)	22 (43.1%)	22 (37.3%)	8 (32.0%)	0.62
Diabetes (%)	3 (5.9%)	6 (10.2%)	1 (4.0%)	0.69
Medication				
Angiotensin converting enzyme inhibitor (%)	6 (11.8%)	4 (6.8%)	2 (8.0%)	0.68
Angiotensin II receptor blocker (%)	23 (45.1%)	18 (30.5%)	7 (28.0%)	0.18
Beta blocker (%)	9 (17.6%)	19 (32.2%)	10 (40.0%)	0.09
Aspirin (%)	23 (45.1%)	18 (30.5%)	13 (52.0%)	0.11
Calcium channel blocker (%)	29 (56.9%)	33 (55.9%)	13 (52.0%)	0.85
Dual antiplatelet therapy (%)	5 (9.8%)	3 (5.1%)	0 (0.0%)	0.24
HMG-CoA inhibitor (%)	28 (54.9%)	25 (42.4%)	10 (40.0%)	0.31
Warfarin (%)	4 (7.8%)	4 (6.8%)	2 (8.0%)	1
Laboratory data				
Creatinine (mg/dL)	0.85 (0.74–0.99)	0.86 (0.75-1.00)	0.83 (0.77–0.98)	0.85
Hemoglobin (g/dL)	12.9 ± 1.61	12.8 ± 1.53	12.2 ± 1.76	0.21
Platelets ($x10^4$/uL)	20.2 ± 5.82	21.3 ± 5.56	18.4 ± 5.07	0.09
Anatomy and physiology				
Neck length (mm)	35.4 ± 11.44	34.8 ± 14.38	34.7 ± 13.69	0.97
Length from lower renal artery to terminal aorta (mm)	117.2 ± 16.31	120.7 ± 19.98	131.7 ± 21.37	0.009
Inner length (mm)	74.0 ± 17.99	80.6 ± 21.61	86.8 ± 30.11	0.055
Maximum outer diameter (mm)	49.9 ± 8.39	52.2 ± 10.17	53.9 ± 7.76	0.17
Maximum inner diameter (mm)	37.2 ± 8.24	41.8 ± 10.93	45.7 ± 10.78	0.002
Patent IMA (%)	37 (72.5%)	43 (72.9%)	19 (76.0%)	1
Patent lumbar arteries	5.1 ± 2.17	4.9 ± 2.25	4.9 ± 1.91	0.87
Preoperative pulse wave velocity (m/s)	2.03 ± 0.493	1.85 ± 0.351	2.03 ± 0.502	0.07
Postoperative pulse wave velocity (m/s)	2.16 ± 0.452	1.99 ± 0.386	2.34 ± 0.612	0.012
Persistent endoleak (%)	14 (27.5%)	3 (5.1%)	12 (48.0%)	< 0.001
Mean arterial pressure (mmHg)	94 ± 12.4	92 ± 9.4	94 ± 12.2	0.60
Stent graft device (%)				0.17
Zenith	11 (21.6%)	17 (28.8%)	4 (16.0%)	
Excluder	20 (39.2%)	12 (20.3%)	7 (28.0%)	
Powerlink	4 (7.8%)	2 (3.4%)	0 (0.0%)	
Endurant	16 (31.4%)	26 (44.1%)	13 (52.0%)	
Others	0 (0.0%)	2 (3.4%)	1 (4.0%)	

IMA Inferior mesenteric artery

aorta than those with sac shrinkage (131.7 ± 21.37 mm vs. 120.7 ± 19.98 mm, $p = 0.049$) and those without significant change (131.7 ± 21.37 mm vs. 117.2 ± 16.31 mm, $p = 0.007$). The maximum inner diameter was significantly larger in patients with sac enlargement than in those without significant change (45.7 ± 10.78 mm vs. 37.2 ± 8.24 mm, $p = 0.002$). Postoperative pulse wave velocity was faster in patients with sac enlargement than in those with sac shrinkage (2.34 ± 0.61 m/s vs. 1.99 ± 0.39 m/s, $p = 0.012$). Patients with sac enlargement had a higher prevalence of postoperative endoleak than those with sac shrinkage (48.0% vs. 5.1%, $p < 0.001$), while patients with sac shrinkage had a lower prevalence of type 2 endoleak than those without significant change (5.1% vs. 27.5%, $p = 0.004$; Table 1).

In the multivariate regression model, postoperative pulse wave velocity (OR: Odds ratio 3.80, 95% CI 1.02–10.70, $p = 0.047$), prevalence of endoleak 1 week after EVAR (OR 4.26, 95% CI 1.24–14.70, $p = 0.022$), inner diameter (OR 1.10, 95% CI 1.03–1.17, $p = 0.005$), and distance from the lower renal artery to the terminal aorta (OR 1.05, 95% CI 1.01–1.08, $p = 0.017$) were independently associated with sac enlargement. (Table 2) On the other hand, the prevalence of endoleak after EVAR (OR 0.09, 95% CI 0.03–0.35, $p < 0.001$) and preoperative pulse wave velocity (OR 0.32, 95% CI 0.12–0.84, $p = 0.022$) were independently associated with sac shrinkage after EVAR. (Table 3) Of the four devices used, Powerlink had the minimum effect on pulse wave velocity while Endurant, Excluder, and Zenith had significant or tendency towards increased pulse wave velocity after device implantation (Table 4).

Factors that were associated with persistent type 2 endoleak 1 week after EVAR were use of the Excluder device ($p = 0.002$), and length of the aneurysm ($p = 0.024$). (Table 5) In multivariate analysis, use of the Excluder device (OR 3.99, 95% CI 1.64–9.66, $p = 0.002$) and length of the aneurysm (OR 1.02, 95% CI 1.00–1.04, $p = 0.027$) were independently associated with persistent type 2 endoleak 1 week after EVAR (Table 6). Four patients with sac enlargement underwent re-intervention. Three patients without significant change underwent re-intervention for leg migration, and one patient with sac shrinkage underwent femoral–femoral bypass for leg occlusion. There were no aneurysm-related deaths during the follow-up.

Table 2 Multivariate logistic regression model for sac enlargement

	Odds ratio	95% CI	p value
Age (years)	1.04	0.94–1.14	0.46
Postoperative pulse wave velocity (m/s)	3.80	1.02–10.70	0.047
Endoleak (%)	4.26	1.24–14.70	0.022
Inner diameter (mm)	1.10	1.03–1.17	0.005
Length from lower renal artery to terminal aorta (mm)	1.05	1.01–1.08	0.017

Discussion

Early change in sac diameter after EVAR is a strong predictor of late complications. In our series, 25 (18.5%) patients had sac enlargement, 59 (43.7%) had sac shrinkage, and 51 (37.8%) had no significant change after EVAR. The prevalence of endoleak, aneurysm treatment length, and preoperative and postoperative pulse wave velocity were associated with sac size changes after EVAR. Although EVAR was associated with increased pulse wave velocity, Powerlink had a minimum effect on pulse wave velocity. Furthermore, use of the Excluder device and the length of the aneurysm were factors independently associated with residual type 2 endoleak after EVAR.

Persistent shrinkage of more than 5 mm is associated with a 99.7% rate of freedom from aneurysm-related deaths at 10 years, whereas sac enlargement is associated with increased surveillance costs, re-intervention rate, and risk of rupture [3, 13]. The rate of freedom from complication differs according to the degree of sac shrinkage. The rate of freedom from complication was 84.3%, 88.1%, and 94.4% for patients with no shrinkage, moderate shrinkage, and major shrinkage, respectively [3]. Several predictors have been reported for complications associated with sac shrinkage and those associated with sac enlargement [14]. Chikazawa et al. [15] reported that the use of antiplatelets, persistent type 2 endoleak, and female gender were associated with sac enlargement. On the other hand, the absence of a thoracic aortic aneurysm, no history of antiplatelet therapy, the presence of coronary artery disease, and the absence of

Table 3 Multivariate logistic regression model for sac shrinkage

	Odds ratio	95% CI	p value
Endoleak (%)	0.09	0.03–0.35	< 0.001
Preoperative pulse wave velocity (m/s)	0.32	0.12–0.84	0.022

Table 4 Preoperative and postoperative pulse wave velocity for each device used

		Preoperative pulse wave velocity (m/s)	Postoperative pulse wave velocity (m/s)	p
Endurant	$n = 55$	1.89 ± 0.406	2.10 ± 0.470	< 0.001
Excluder	$n = 36$	1.88 ± 0.361	2.13 ± 0.516	< 0.001
Powerlink	$n = 6$	2.32 ± 0.930	2.36 ± 0.659	0.86
Zenith	$n = 30$	2.06 ± 0.442	2.11 ± 0.393	0.055

Table 5 Demographics of patients with a persistent type 2 endoleak 1 week after endovascular aortic aneurysm repair (EVAR)

	Type 2 endoleak $n=106$	No endoleak $n=29$	p value
Age (years)	77 ± 7.2	76 ± 7.6	0.45
Aspirin (%)	41 (38.7%)	14 (48.3%)	0.40
Dual antiplatelet therapy (%)	7 (6.7%)	2 (6.9%)	1
Warfarin (%)	7 (6.7%)	3 (10.3%)	0.45
Excluder (%)	23 (21.7%)	15 (51.7%)	0.002
Platelet count ($\times 10^4$/uL)	20.5 ± 5.61	20.1 ± 6.26	0.73
Creatinine (mg/dL)	0.84 (0.75-1.00)	0.84 (0.71–0.98)	0.65
Preoperative pulse wave velocity (m/s)	1.94 ± 4.266	1.97 ± 5.005	0.72
Patent lumbar artery	4.8 ± 2.17	5.7 ± 1.91	0.053
Patent IMA (%)	75 (70.8%)	25 (86.2%)	0.10
Neck length (mm)	35.3 ± 13.52	33.9 ± 11.55	0.62
Length of aneurysm (mm)	77.2 ± 22.21	87.9 ± 22.33	0.024
Aneurysm diameter (mm)	51.3 ± 8.72	53.9 ± 11.18	0.17

IMA Inferior mesenteric artery

Table 6 Multivariate logistic regression for a persistent type 2 endoleak 1 week after endovascular aortic aneurysm repair (EVAR)

	Odds ratio	95% CI	p value
Excluder	3.99	1.64–9.66	0.002
Length of aneurysm (mm)	1.02	1.00–1.04	0.027

type 2 endoleak were associated with sac shrinkage. Similarly, Aoki et al. reported persistent type 2 endoleak and multiagent antiplatelet therapy as a predictor for no response to sac shrinkage 6 months after EVAR [5]. Young age, use of calcium channel blockers, statins, and the absence of calcium in the neck of the aneurysm neck have also been reported to be associated with sac shrinkage after EVAR [8–10].

Prior studies have shown the existence of residual intra-aneurysm sac pressure after EVAR [16, 17]. In their in vitro experiment with a latex aneurysm model, Kwon et al. reported that intra-aneurysm sac pressure was inversely associated with aortic compliance [17]. This supports the finding of Yeung et al. that intraluminal thrombus was associated with sac enlargement [18]. Intraluminal thrombus triggers hypoxia and inflammatory infiltration of the adjacent aortic wall, which results in a less compliant aortic wall [19–21]. In their multivariate analysis, minimal thrombus (OR: 1.47) and greater aneurysm diameter (OR: 1.3) were independent factors associated with sac shrinkage 1, 6, and 12 months after EVAR ($p < 0.05$) [18].

Pulse wave velocity, which measures vascular stiffness, is a good surrogate for aortic compliance [12]. In our series, high postoperative pulse wave velocity was independently associated with sac enlargement, whereas low preoperative pulse wave velocity was associated with sac shrinkage. Aortic stiffness is caused by vascular aging, characterized by structural changes in collagen and elastin fibers. It is also caused by changes in dynamic components resulting from endothelial dysfunction [22]. Advanced biomechanical changes in the aorta cause it to be less compliant. Thus, patients with high preoperative pulse wave velocity are more unlikely to show vascular remodeling. A less compliant aortic wall is also associated with higher intra-aneurysm sac pressure, which may interfere with the progress of sac shrinkage [17, 21]. Intra-aneurysm sac pressure, especially the pulse pressure, plays a major role in aneurysm sac remodeling, where high pressure is associated with sac enlargement and low pressure is associated with vascular remodeling and shrinkage [23–25]. As higher pulse wave velocity is associated with higher pulse pressure, an increase in postoperative pulse wave velocity may affect increased pulse pressure on the intra-aneurysm sac pressure, especially in patients with residual endoleaks [21–24, 26]. Although our sample size was small, we found that Powerlink, an endoskeleton type device, did not result in a significant increase after endovascular repair. This is supported by prior findings in thoracic endovascular aneurysm repair (TEVAR), where the endoskeleton type device did not show significant increase in pulse wave velocity after TEVAR [12].

Risk factors for persistent type 2 endoleak, a major component of high intra-aneurysm sac pressure, include the preoperative abdominal aortic aneurysm length and patent inferior mesenteric artery [4, 27]. In a study by Kray et al., sac volume regression was 21.8% in patients with an occluded inferior mesenteric artery, but 13.2% in patients with a patent inferior mesenteric artery ($p = 0.004$) [4]. In our series, the factors that were independently associated with type 2 endoleak were the length of the aneurysm (OR 1.02, 95% CI 1.00–1.04,

$p = 0.027$) and use of the Excluder device (OR 3.99, 95% CI 1.64–9.66, $p = 0.002$). Cieri et al. also reported the significance of device selection on sac behavior, observing that persistent sac shrinkage was independently associated with the Zenith device (OR 1.33, 95% CI 1.176–1.514) and Endurant (OR 1.52, 95% CI 1.108–2.092), whereas the AneuRx device was inversely associated with sac shrinkage (OR 0.57, 95% CI 0.477–0.688) [13]. Although the Excluder in our series was associated with persistent type 2 endoleak 1 week after surgery, it was not associated with sac enlargement 3 years after EVAR.

This study has several limitations. First, the results did not include those of 23 patients who were not able to re-visit the hospital, most of whom were moved to a nursing home and refused medical follow-up. Second, pulse wave velocity is influenced by the aortic diameter; namely, a larger lumen of the aorta tends to decrease the flow velocity. In our series, the inner diameter was largest in patients who showed sac enlargement; however, these patients with large diameters had the highest pulse wave velocity. Many prior studies have looked at arterial stiffness measured by pulse wave velocity in aortic aneurysms [16, 28, 29]. Our data also showed that there was no correlation between aneurysm sac size and preoperative pulse wave velocity measurement ($r = -0.102$, $p = 0.24$). Pulse wave velocity in patients with aortic aneurysms may not simply indicate arterial stiffness, but it is still a good tool to show trends in aortic compliance. Finally, this was a retrospective study with a small sample size. Our study shows only association and not the causal relationship of each variable with sac size changes. A prospective study with a much larger sample size is needed to confirm the results.

In conclusion, the predictive factors for sac enlargement may help us decide on the best therapeutic approach for patients with abdominal aortic aneurysm. Identifying patients at higher risk of sac enlargement may allow us to avoid repeated intervention and the medical costs associated with frequent surveillance after surgery. Although further study is needed, perioperative pulse wave velocity and management of type 2 endoleaks should be investigated as a therapeutic approach to achieve better surgical outcomes for patients undergoing EVAR.

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Compliance with ethical standards

Conflict of interest We have no conflicts of interest to disclose.

References

1. Prinssen M, Verhoeven EL, Buth J, Cuypers PW, van Sambeek MR, Balm R, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med. 2004;351:1607–18.
2. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. Lancet. 2004;364:843–8.
3. Bastos Goncalves F, Baderkhan H, Verhagen HJ, Wanhainen A, Bjorck M, Stolker RJ, et al. Early sac shrinkage predicts a low risk of late complications after endovascular aortic aneurysm repair. Br J Surg. 2014;101:802–10.
4. Kray J, Kirk S, Franko J, Chew DK. Role of type II endoleak in sac regression after endovascular repair of infrarenal abdominal aortic aneurysms. J Vasc Surg. 2015;61:869–74.
5. Aoki A, Suezawa T, Sangawa K, Tago M. Effect of type II endoleaks and antiplatelet therapy on abdominal aortic aneurysm shrinkage after endovascular repair. J Vasc Surg. 2011;54:947–51.
6. Alvarez Marcos F, Llaneza Coto JM, Franco Meijide FJ, Zanabili Al-Sibbai AA, Vilarino Rico J, Alonso Perez M, et al. Effect of antiplatelet therapy on aneurysmal sac expansion associated with type II endoleaks after endovascular aneurysm repair. J Vasc Surg. 2017;66:396–403.
7. Tsuyuki Y, Matsushita S, Dohi S, Yamamoto T, Tambara K, Inaba H, et al. Factors for sac size change of abdominal aortic aneurysm after endovascular repair. Ann Thorac Cardiovasc Surg. 2014;20:1016–20.
8. Pini R, Faggioli G, Mascoli C, Gallitto E, Freyrie A, Gargiulo M, et al. Influence of statin therapy on type 2 endoleak evolution. Ann Vasc Surg. 2015;29:1167–73.
9. Kaladji A, Cardon A, Abouliatim I, Campillo-Gimenez B, Heautot JF, Verhoye JP. Preoperative predictive factors of aneurysmal regression using the reporting standards for endovascular aortic aneurysm repair. J Vasc Surg. 2012;55:1287–95.
10. Bailey MA, Sohrabi S, Flood K, Griffin KJ, Rashid ST, Johnson AB, et al. Calcium channel blockers enhance sac shrinkage after endovascular aneurysm repair. J Vasc Surg. 2012;55:1593–9.
11. Munakata M, Konno S, Miura Y, Yoshinaga K. Prognostic significance of the brachial-ankle pulse wave velocity in patients with essential hypertension: final results of the J-TOPP study. Hypertens Res. 2012;35:839–42.
12. Hori D, Akiyoshi K, Yuri K, Nishi S, Nonaka T, Yamamoto T, et al. Effect of endoskeleton stent graft design on pulse wave velocity in patients undergoing endovascular repair of the aortic arch. Gen Thorac Cardiovasc Surg. 2017;65:506–11.
13. Cieri E, De Rango P, Isernia G, Simonte G, Verzini F, Parlani G, et al. Effect of stentgraft model on aneurysm shrinkage in 1450 endovascular aortic repairs. Eur J Vasc Endovasc Surg. 2013;46:192–200.
14. Lalys F, Daoudal A, Gindre J, Goksu C, Lucas A, Kaladji A. Influencing factors of sac shrinkage after endovascular aneurysm repair. J Vasc Surg. 2017;65:1830–38.
15. Chikazawa G, Hiraoka A, Totsugawa T, Tamura K, Ishida A, Sakaguchi T, et al. Influencing factors for abdominal aortic aneurysm sac shrinkage and enlargement after EVAR: clinical reviews before introduction of preoperative coil embolization. Ann Vasc Dis. 2014;7:280–5.
16. Moulakakis KG, Mylonas SN, Kakisis J, Kadoglou NP, Papadakis I, Sfyroeras GS, et al. Arterial stiffness alterations and inflammatory response following endovascular aortic repair: based on a presentation at the 2013 VEITH Symposium, November 19–23, 2013 (New York, NY, USA). Aorta (Stamford, Conn) 2015;3:75–80.
17. Kwon ST, Rectenwald JE, Baek S. Intrasac pressure changes and vascular remodeling after endovascular repair of abdominal aortic aneurysms: review and biomechanical model simulation. J Biomech Eng. 2011;133:011011. https://doi.org/10.1115/1.4003134.

18. Yeung JJ, Hernandez-Boussard TM, Song TK, Dalman RL, Lee JT. Preoperative thrombus volume predicts sac regression after endovascular aneurysm repair. J Endovasc Ther. 2009;16:380–8.
19. Vorp DA, Lee PC, Wang DH, Makaroun MS, Nemoto EM, Ogawa S, et al. Association of intraluminal thrombus in abdominal aortic aneurysm with local hypoxia and wall weakening. J Vasc Surg. 2001;34:291–9.
20. Kazi M, Thyberg J, Religa P, Roy J, Eriksson P, Hedin U, et al. Influence of intraluminal thrombus on structural and cellular composition of abdominal aortic aneurysm wall. J Vasc Surg. 2003;38:1283–92.
21. Georgakarakos E, Georgiadis GS, Ioannou CV, Kapoulas KC, Trellopoulos G, Lazarides M. Aneurysm sac shrinkage after endovascular treatment of the aorta: beyond sac pressure and endoleaks. Vasc Med. 2012;17:168–73.
22. Janic M, Lunder M, Sabovic M. Arterial stiffness and cardiovascular therapy. Biomed Res Int. 2014;2014:621437. https://doi.org/10.1155/2014/621437.
23. Dias NV, Ivancev K, Resch TA, Malina M, Sonesson B. Endoleaks after endovascular aneurysm repair lead to nonuniform intra-aneurysm sac pressure. J Vasc Surg. 2007;46:197–203.
24. Dias NV, Ivancev K, Malina M, Resch T, Lindblad B, Sonesson B. Intra-aneurysm sac pressure measurements after endovascular aneurysm repair: differences between shrinking, unchanged, and expanding aneurysms with and without endoleaks. J Vasc Surg. 2004;39:1229–35.
25. Dias NV, Ivancev K, Kolbel T, Resch T, Malina M, Sonesson B. Intra-aneurysm sac pressure in patients with unchanged AAA diameter after EVAR. Eur J Vasc Endovasc Surg. 2010;39:35–41.
26. Hinnen JW, Koning OH, van Bockel JH, Hamming JF. Aneurysm sac pressure after EVAR: the role of endoleak. Eur J Vasc Endovasc Surg. 2007;34:432–41 **(discussion 42–3)**.
27. Phan DD, Meyer F, Pech M, Halloul Z. Length of abdominal aortic aneurysm and incidence of endoleaks type II after endovascular repair. Wien Klin Wochenschr. 2015;127:851–7.
28. Gray C, Goodman P, Badger SA, O'Malley MK, O'Donohoe MK, McDonnell CO. Endovascular aneurysm repair increases aortic arterial stiffness when compared to open repair of abdominal aortic aneurysms. Vasc Endovasc Surg. 2016;50:317–20.
29. Lantelme P, Dzudie A, Milon H, Bricca G, Legedz L, Chevalier JM, et al. Effect of abdominal aortic grafts on aortic stiffness and central hemodynamics. J Hypertens. 2009;27:1268–76.