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Original Article

Evaluation and Coil Embolization of the Aortic Side Branches for Prevention of Type II Endoleak after Endovascular Repair of Abdominal Aortic Aneurysm

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Objectives: Aneurysm shrinkage after EVAR is the strong factor of favorable outcomes after endovascular abdominal aortic aneurysm repair (EVAR), and type II endoleaks is the risk factor of no aneurysm shrinkage or aneurysm enlargement in the long term. In this study, we evaluate the aortic side branches relate to early postoperative type II endoleak, and performed coil embolization for those vessels for prevention of type II endoleak.
Methods: Patency and diameter of aortic side branches including inferior mesenteric artery (IMA) and lumbar artery (LA) were evaluated in 56 consecutive patients with abdominal aortic aneurysm who were scheduled for EVAR. Coil embolization with Interlock was performed in 24 patients during EVAR for all patent IMA and LA with maximal diameter more than 2.0 mm. Computed tomography was performed one week after EVAR for evaluation of endoleak.
Results: In patients with IMA more than 2.5 mm in diameter, the frequency of type II endoleak was approximately 90% regardless of the number of patent LA. In case with patent IMA less than 2.5 mm or with 2 or more patent LA larger than 2.0 mm, the frequency of type II endoleak was 46 to 67%. Coil embolization for IMA was successfully performed in 15/16 patients (94%). Coil embolization of LA was performed for patent LA larger than 2.0 mm and 29 out of 45 LA (64%) were successfully occluded. There was no perioperative complication associated with coil embolization. The frequency of type II endoleak was significantly lower in patients with coil embolization than those without coil embolization (4.2% vs 58.9%, $p<0.0001$).
Conclusion: Patent IMA and LA in diameter larger than 2.0 mm were associated with type II endoleak one week after EVAR, and coil embolization with Interlock during EVAR is safe and effective procedure to prevent type II endoleak. (This is a translation of Jpn J Vasc Surg 2016; 25: 321–328.)

Keywords: abdominal aortic aneurysm, endovascular surgery, type II endoleak, coil embolization

Introduction

Endovascular abdominal aortic aneurysm repair with stent graft (EVAR) has been widely performed because of its minimal invasiveness.[1)] Although EVAR is associated with a low perioperative mortality rate, long-term survival rate associated with it is approximately the same as that associated with vascular graft replacement (open repair). Furthermore, the re-intervention rate associated with EVAR has been reported to be significantly higher than that associated with open repair.[2)] Because most instances of re-intervention involve additional treatment for an endoleak, to achieve long-term prognosis after EVAR similar to that after open surgery, strict follow-up observation with contrast-enhanced computed tomography (CT) is required in many patients. Moreover, if aneurysm expansion, graft movement, or graft closure occurs, appropriate secondary intervention must be implemented. However, long-term postoperative follow-up with contrast-enhanced CT is associated with the problems of adverse effects on kidney function, radiation exposure, and cost.[3)] In addition, even on conducting such detailed follow-up, aneurysm rupture has been reported to occur significantly more often after EVAR than after open repair.[2)] Meanwhile, post-EVAR aneurysm shrinkage, which demonstrates that the aneurysm has been completely excluded systemic blood flow, is considered to be a strong predictor of favorable long-term prognosis.[3–5)] Many reports have indicated that a type II endoleak is a factor for the failure of aneurysm shrinkage.[6–8)] In addition, intra-arterial coil embolization for type II endoleaks following

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EVAR has not been reported to be highly effective,[9] and recurrence of type II endoleaks is common.[10] A hybrid operating theater was established in April 2015 at our facility. Using the fusion method to combine fluoroscopic images with volume-rendering images created by preoperative contrast-enhanced CT, the EVAR procedure and identification of arterial bifurcations become simple. Here we investigated which arterial branches are important causes of type II endoleaks and attempted to perform coil embolization on these aortic branches during EVAR.

Subjects and Methods

Elective EVAR was performed on 134 patients at our hospital between August 2009 and April 2016. Of 105 patients who underwent EVAR before introducing coil embolization of arterial branches (until March 2015), we excluded 49 patients (40 who did not undergo CT 1 week postoperatively or only underwent plain CT, six who had an iliac aneurysm, two who had a saccular aneurysm, and one who died postoperatively); thus, 56 patients were available for analysis. We investigated the vessels causing type II endoleaks. These 56 patients were served as control group. In total, 29 patients underwent EVAR from April 2015 onward. After excluding two patients with saccular and three with iliac aneurysms, 24 remained for analysis. Coil embolization was attempted for all patent inferior mesenteric arteries (IMAs) and all lumbar arteries (LAs) that were patent by $\geq$2 mm (the coil group).

The vessels causing type II endoleaks were investigated by evaluating whether the IMA and LA from the third lumbar vertebrae (L3) downward were patent on preoperative contrast-enhanced CT and by measuring their inner diameter if they were patent. The presence of any type II endoleak was confirmed on contrast-enhanced CT 1 week postoperatively. If a type II endoleak was observed, the causative artery was identified and the endoleak area was measured.

For coil embolization, preoperative contrast-enhanced CT data were directly introduced into the Artis Zeego Syngo X-Workplace (Siemens, Erlangen, Germany) before EVAR. Subsequently, images of the IMA and LA targeted for coil embolization were created and their origins were marked. The Artis Zeego device was used to perform low-dose 3D imaging over 5 s (Syngo DynaCT). With bone data as landmarks, Syngo 3D/3D fusion was used to share positional information between preoperative CT data and Syngo DinaCT data. Fusion images, obtained by combining CT data with fluoroscopic images on the Syngo X Workspace, were made available to be used. For coil embolization, angles at which the target bifurcations for coil embolization could be isolated were set on fusion images (**Fig. 1**, right). A 4-Fr RIM catheter or shepherd hook catheter was inserted until it was close to the origin of side branches, and if the bifurcation could be selected on contrast imaging (**Fig. 1**, left), a Renegade microcatheter (Boston Scientific, MA, USA) was inserted into the side branches, and selection was confirmed using contrast-enhanced imaging (**Fig. 2**, left). Embolization was performed with Interlock coils (Boston Scientific, MA, USA) as cen-

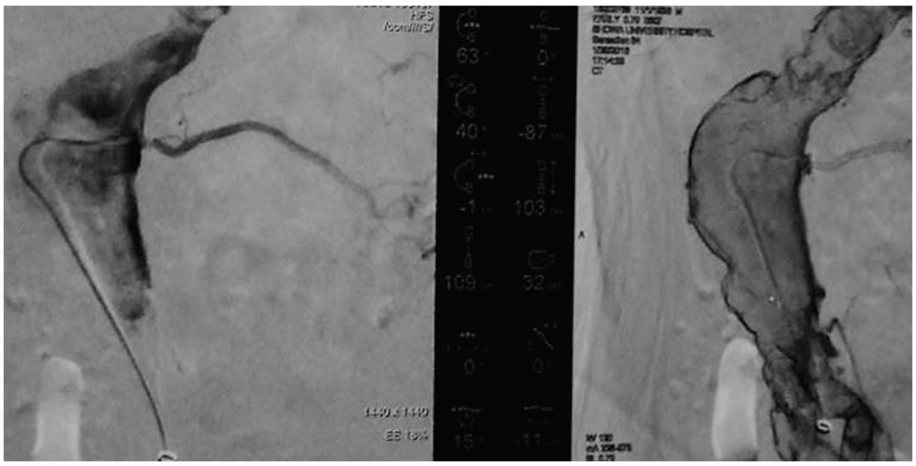

Fig. 1 Fusion (right panel) and selective angiogram (left panel). Just before the procedure, preoperative computed tomography (CT) data was imported into Singo X-Workplace of Artic Zeego and volume rendering figure was constructed. Also 3D-CT data was obtained from Artuc Zeego (Syngo DinaCT). Then, preoperative CT data was matched with Syngo DinaCT. Consequently volume rendering figure could be visualized on fluoroscopy (Fusion image). Adequate direction of fluoroscopy to identify the origin of aortic side branch was adjusted by fusion image (right panel). Then aortic side branch was selected by angiogram (left panel).

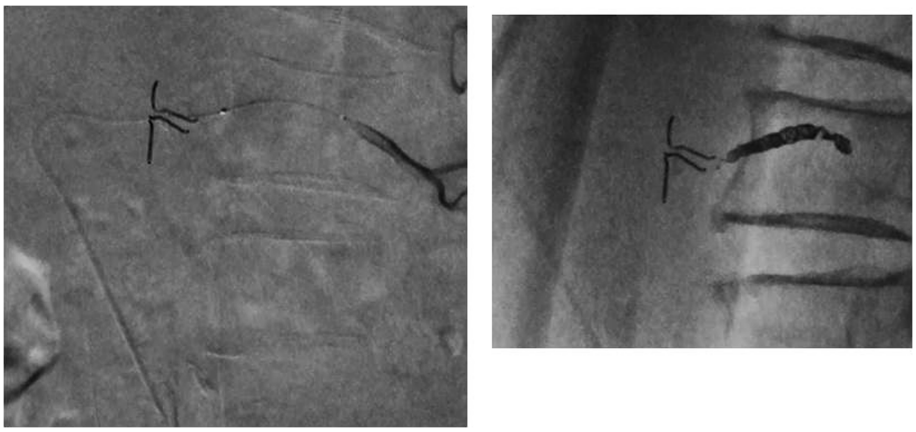

Fig. 2 Selective angiogram with microcatheter (left panel) and coil embolization (right panel). Microcatheter was advanced into the aortic side branch and selection of target branch was comfirmed by angiogram (left panel). Then target branch was coil embolized with Interlock as close as its origin (right panel).

trally as possible. For the IMA in particular, we ensured that coil embolization was performed proximal side of the first bifurcation. The procedure time required for one vessel was limited up to 10 min, and coil embolization was stopped once 100 mL of contrast medium was used. The success rates of coil embolization for the IMA and LA were evaluated, and the successful and unsuccessful LA embolization procedures were compared. The presence of any endoleak was confirmed using contrast-enhanced CT at 1 week postoperatively, and results were compared with those of the control group. We then investigated whether any complication thought to be postoperative embolism had occurred.

Statistical analysis was performed using JMP Pro 11 (SAS, Inc., NC, USA). The χ^2 test or Student's t-test was performed, and the level of significance was set at $<5\%$.

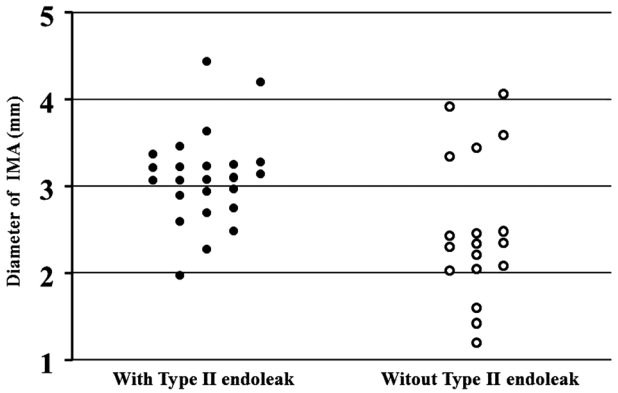

Fig. 3 Diameter of IMA with or without type II endoleak. The diameter of IMA with Type II endoleak was significantly larger than those without Type II endoleak. IMA: inferior mesenteric artery

Results

The IMA was patent preoperatively in 42 patients, and type II endoleaks were noted at 1 week postoperatively in 24 of them. IMA occlusion was observed following EVAR in 18 patients (42.9%). The IMA inner diameter was significantly greater in patients with type II endoleaks than in those in whom the IMA was occluded following EVAR (3.1 ± 0.5 vs. 2.5 ± 0.8 mm, respectively; $p<0.001$) and significantly more patient had type II endoleaks in patients with an IMA of $\geq$2.5-mm diameter than in those with an IMA of $<$2.5-mm diameter [3/16 (18.8%) vs. 21/26 (80.8%), respectively; $p<0.0001$; **Fig. 3**].

Of the patients who underwent EVAR before the introduction of coil embolization, all LAs from L3 downward were preoperatively occluded in four patients. At least one LA was patent in 52 patients (one LA in five, two in 12, three in seven, four in 17, five in 10, and six in one). Thus, a total of 174 LAs were patent, and 152 of them were occluded after EVAR. For 22 patients with type II endoleaks related with LAs, a significantly more LAs from L3 downward were patent (3.9 ± 1.3 and 2.6 ± 1.6 LAs in patients with and without a type II endoleak, respectively; $p=0.003$), the LA inner diameter was significantly greater (2.3 ± 0.6 vs. 1.8 ± 0.4 mm; $p<0.001$), and LAs of $\geq$2.0-mm diameter caused type II endoleaks significantly more frequently than did LAs of $<$2.0-mm diameter [12/55 (21.8%) vs. 10/122 (8.2%); $p=0.0110$; **Fig. 4**].

Evaluation of the frequency of type II endoleaks per combination of IMA (occluded, or patent with diameter $<$2.5 or $\geq$2.5 mm) and the number of patent LAs that were at least 2 mm showed that the frequency of type II endoleaks was approximately 90% if the IMA with diameter $>$2.5 mm patent regardless the number of patent LAs and approximately 50% if the diameter of IMA was

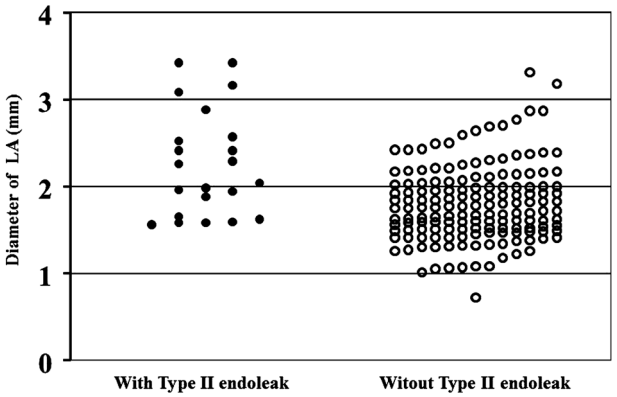

Fig. 4 Diameter of LA with or without type II endoleak. The diameter of LA with Type II endoleak was significantly larger than those without Type II endoleak. LA: lumbar artery

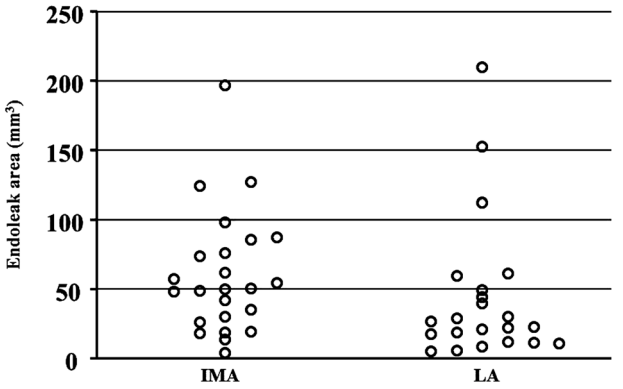

Fig. 5 Endoleak area of IMA and LA. Endoleak area did not differ between the two groups. However, the frequency of the patients with endoleak area 50 mm² was significantly higher in patients with endoleak from inferior mesenteric artery. IMA: inferior mesenteric artery; LA: lumbar artery

Table 1 Frequency of type II endoleak according to the patency of IMA and number of patent LA

	Number of patent LA with diameter≧2.0 mm	
	none or 1	2 or more
Occluded IMA	8.3% (1/12)	50.0% (1/2)
Patent IMA with diameter<2.5 mm	46.2% (6/13)	66.7% (2/3)
Patent IMA with diameter≧2.5 mm	87.5% (14/16)	66.7% (2/3)

The frequency of type II endoleak was about 90% when IMA with diameter≧2.5 mm was patent regardless the number of patent LA with diameter≧2.0 mm. When IMA with diameter<2.5 mm was patent or 2 or more LA with diameter≧2.0 mm were patent, type II endoleak was detected about half of patients. IMA: inferior mesenteric artery; LA: lumbar artery

<2.5 mm and at least two LAs were patent (**Table 1**).

When IMA and LA were compared as the source of post-operative type II endoleaks, 24/42 (57.1%) IMA and 22/174 (12.6%) LAs those were patent preoperatively caused type II endoleaks. Thus, IMA were significantly more likely to cause type II endoleaks ($p<0.0001$). No significant difference was observed between IMAs and LAs for the endoleak area (IMA: 60 ± 44 mm², LA: 44 ± 5 mm²; $p=0.259$). However, the frequency of endoleaks with an area of ≥50 mm² was significantly higher for IMAs (54.2%) than for LAs (22.7%; $p=0.029$; **Fig. 5**).

We attempted to perform coil embolization on the patent IMAs in 16/24 patients in the coil group in which the mean vascular diameter was 3.23 ± 0.59 mm. Coil embolization was successfully performed in 15 of these 16 patients (93.8%). The patient in whom coil embolization was unsuccessful exhibited stenosis with calcification at the IMA origin, and although a 0.035-inch Radifocus guidewire could be inserted into the IMA, a microcatheter could not be inserted. This IMA was confirmed to be occluded on postoperative contrast-enhanced CT. In the coil group, a total of 67 LAs were patent and the mean vascular diameter was 2.18 ± 0.53 mm. Coil embolization was attempted in 45 LAs with ≥2-mm diameter and was successful in 29 (64.4%). The numbers of LAs that were patent preoperatively and could not be coil embolized or those origin were not covered with stent graft by postoperative contrast-enhanced CT was 0 in eight patients, one in seven (only one with LA diameter of ≥2 mm), and two in nine (number of LAs with diameter of ≥2 mm: none in three patients, one in two, and two in four). No patient exhibited patency of three or more LAs of ≥2-mm diameter. When LA inner diameters and the diameters of the aorta at the level of LA origin were compared between successful and unsuccessful LA coil embolization cases, we found that the inner LA diameters were significantly greater in successful patients, and that the long axis diameter of the aorta (32.6 ± 7.7 vs. 39.1 ± 10.1 mm; $p=0.0203$) and mean diameter of the aorta (31.6 ± 7.5 vs. 37.6 ± 9.5 mm; $p=0.0244$) were significantly smaller in the successful cases that in the unsuccessful cases. No significant difference was observed between the successful and unsuccessful groups with regards to the lumbar vertebra height of the LA or whether it was on the left or right side. No patient in whom coil embolization was attempted exhibited postoperative renal dysfunction, peripheral embolism, or elevated serum alanine aminotransferase or lactate dehydrogenase levels.

Comparison of the coil and control groups indicated no significant differences in patient backgrounds, devices implanted, or the frequency of internal iliac artery coil embolization, except that the patients in the coil group had significantly larger constitutes that those in the control group. Operation time of coil embolization group tended to be longer about 20 min than control group. A type II endoleak was detected on contrast-enhanced CT

Table 2 Comparison between coil embolization group and control group

	Coil embolization group n=24	Control group n=56	p value
Age (year)	76.0±7.8	77.5±5.5	0.3368
Female gender	4 (16.7%)	18 (32.1%)	0.1828
Height (cm)	164.9±8.8	159.3±9.8	0.0220
Weight (kg)	64.4±11.2	57.6±11.8	0.0216
Body surface area (m^2)	1.70±0.17	1.57±0.21	0.0129
Device			
Endurant	10 (41.7%)	26 (45.6%)	
Excluder	7 (29.2%)	18 (32.1%)	
Powerlink	4 (16.7%)	8 (14.3%)	
Zenith	3 (12.5%)	4 (7.1%)	0.8621
Internal iliac artery coil embolization	5 (20.8%)	12 (24.0%)	0.7604
Operation time (min)	145±41	127±40	0.0882
Type II endoleak by CT 1 week after EVAR	1 (4.2%)	33 (58.9%)	<0.0001

There were no significant difference between the two groups for background, device and frequency of internal iliac artery coil embolization except coil embolization group was significantly larger constitution. Operation time tended to be longer in coil embolization group. The frequency of type II endoleak detected 1 week after EVAR was significantly lower in coil embolization group. EVAR: endovascular abdominal aortic aneurysm repair

performed on postoperative day 7 only in one patient in the coil group, which was a significantly lower than control group (**Table 2**). This patient also had a clear type IV endoleak, however there was patent LA, we decided as type II endoleak.

Discussion

The perioperative mortality rate associated with EVAR was significantly lower than that associated with open repair. However, aneurysm related complication rate is higher with EVAR than open repair, especially in mid- to long-term period. Therefore the advantages in early outcomes disappeared after 4 years of follow-up observations and no significant difference was observed in the overall mortality rate. Moreover, it has been reported that the frequency of secondary intervention is significantly higher after EVAR than after open repair, and that EVAR results in significantly more aneurysm ruptures.[2] The purpose of EVAR is the prevention of aneurysm rupture by eliminate the systemic blood pressure transmission to the aneurysm wall with stent graft. Postoperative aneurysm shrinkage has been considered to be a strong predictor of favorable long-term prognosis.[3–5] Therefore, we investigated factors related to aneurysm shrinkage previously, we found that taking multiple antiplatelet agents and type II endoleaks detected by contrast-enhanced CT evaluated 6 months after EVAR were risk factors for the failure of aneurysm shrinkage.[11] In 1998, Engellau et al.[12] used magnetic resonance imaging (MRI) and magnetic resonance angiography and found that thrombi in the aneurysm sac gradually organized after EVAR but complete organization occurred in only 40% of patients after 1 year. In 2012, Cornelissen et al.[13] used MRI to investigate the patients without aneurysm shrinkage 2 years after EVAR and found that the thrombus in the aneurysm sac inside the aneurysm was not organized in half of the patients. Furthermore, in 2003, Vallabhaneni et al.[14] used thrombi collected during open repair to investigate the correlation between the degree of thrombus organization and pressure transmission and found that although organized thrombi did not transmit pressure, thrombi that were not organized transmitted pressure. Because these results suggested that thrombus organization in the aneurysm sac is necessary for aneurysm shrinkage, we investigated the effects of antifibrinolytic therapy with tranexamic acid for promoting thrombus organization in aneurysm sac. Results showed that although antifibrinolytic therapy with tranexamic acid reduced aneurysms by approximately the same extent regardless of whether multiple antiplatelet agents were being administered, the presence of a type II endoleak disturbed the aneurysm shrinkage despite antifibrinolytic therapy with tranexamic acid.[15] Many studies have demonstrated that type II endoleaks are a factor for the failure of aneurysm shrinkage after EVAR.[6–8] Furthermore, once the aneurysm starts to expand due to type II endoleak, transarterial coil embolization of the responsible artery has little effect.[9,10] Therefore type II endoleaks should be prevented to achieve favorable long-term prognosis following EVAR.

Type II these endoleak has been reported to be significantly frequent in elderly patients, and centrally female patients and that a history of smoking and peripheral vascular lesions decreases the risk of type II endoleaks.[16]

In terms of aneurysm morphology, a large maximum diameter is a risk factor for type II endoleaks[17] and mural thrombus around the entire circumference decreases the risk of type II endoleaks.[16] We investigated major artery branches that caused type II endoleaks and found that risk factors were patency of the IMA (particularly IMAs with large diameter), as well as the number of patent LAs. This finding was consistent with the findings of various reports from 2011 to 2016.[16–21] We also compared IMAs and LAs and found that the endoleak area of the IMA tended to be large in many patients. In 2014, Demehri et al.[22] reported that when the endoleak cavity is larger, the aneurysm tend to expand. Therefore, it appears that the IMA is an important causative vessel of type II endoleaks.

For the embolization of these major artery branches considered to cause type II endoleaks, Parry et al.[23] studied embolization of the IMA and LA in 2002 and found that the procedure was successful in 13/16 (81%) and 8/13 (62%) patients, respectively, and they found that no endoleak observed in the embolization group. In 2014, Burbelko et al.[24] used an Amplatzer vascular plug before EVAR for embolization on 33 IMAs and seven LAs of ≥2.5-mm diameter and found that all target vessels were embolized successfully and no endoleak was observed in embolization group. We found that coil embolization during EVAR was possible for 94% of IMAs and 64% of LAs, with only one type II endoleak observed postoperatively. The embolization success rate of IMA was similar with previous reports. Although the success rate for the LA was similar to that reported by Parry et al.,[23] it was lower than that in other reports, such as that by Burbelko et al.[24] Although the reason for this is unclear, it could have been related to whether the procedure was performed preoperatively or intraoperatively, whether the device used was a coil or an Amplatzer vascular plug, and our learning curve.

Embolization for the IMA alone has also been attempted. In 2013, Ward et al.[25] performed IMA embolization successfully in 108 patients. They reported that although type II endoleaks were significantly less common than in the group that did not undergo embolization, type II endoleaks still were observed in 34.3% of the embolization group. In 2014, Müller-Wille et al.[26] performed IMA embolization using an Amplatzer Vascular Plug type 4 in 31 patients and achieved complete occlusion in 29 (93.5%). They reported a significant decrease in the number of complicated IMA and LA endoleaks, which are a cause of aneurysm expansion.[10] In our study, we also found that IMA and LA embolization resulted in an extremely low frequency of type II endoleaks (4.2%). Therefore, although IMA appears to be a more important causative vessel of type II endoleaks and embolization of the IMA alone should be reasonably effective, embolization of both the IMA and LA is more useful for preventing type II endoleaks. In this study, the number of patent LAs after EVAR because of the failure of coil embolization was two or less. This suggested that type II endoleaks could be prevented even when two or fewer LAs were patent. However, among patients treated before introducing coil embolization, type II endoleaks were noted in 2/14 in the patients with occluded IMA. In these 2 patients, one LA of ≥2-mm diameter in one patient and two LAs of ≥2-mm diameter in another were patent, this topic requires verification in future studies using a larger number of patients. Although there was a risk of complications, such as distal embolism due to coil embolization procedure, the complication rate can be reduced with careful manipulation of micro catheter and micro guidewire under good quality fluoroscopy in the hybrid operating theater.

The promotion of thrombus organization inside the aneurysm is another possible method of preventing type II endoleaks. Aneurysm sac embolization using thrombin and Gelfoam,[27] coil placement within the aneurysm,[28] and intra-aneurysm coil and fibrin glue[29] were reported as the useful method to promote the thrombus formation in the aneurysm sac. Even the complication rate of all these procedures has been reported to be low, we selected the branch coil embolization because considering the risk of complications, such as bifurcation embolism, associated with the injection of embolization material into the aneurysm sac. However, it might be worth performing these procedures in patients in whom major artery branches coil embolization was unsuccessful.

We evaluated early postoperative type II endoleaks using contrast-enhanced CT. Normally, persistent type II endoleaks that remain for more than 6 months postoperatively are thought to influence aneurysm expansion or failure of reduction in aneurysm size. If no endoleak is noted on early postoperative CT and proximal and distal sealing zone length is long enough, prognosis has been reported to be favorable.[30] Thus, coil embolization during EVAR appears likely to result in a favorable prognosis. In future, we plan to investigate the aneurysm shrinkage and long-term prognosis.

Conclusion

The frequency of early type II endoleaks after EVAR was >80% when an IMA of ≥2.5-mm diameter was patent and approximately 50% when an IMA of <2.5-mm diameter or at least two LAs of ≤2-mm diameter were patent. Coil embolization during EVAR could be performed on 15/16 (93.8%) IMAs and 29/45 (64.4%) LAs. No complication thought to be related with coil embolization was observed. In the group that underwent coil embolization, the frequency of early postoperative type II

endoleaks was significantly low. Thus, it appears that IMA and LA coil embolization during EVAR can be useful for preventing type II endoleaks.

Disclosure Statement

None of the authors or co-authors have any conflicts of interest to declare.

Additional Remarks

A summary of this report was presented at the 44th Annual Meeting of the Japanese Society for Vascular Surgery on May 25, 2016, in Tokyo.

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