CASE REPORTS

Successful treatment of endotension and aneurysm sac enlargement with endovascular stent graft reinforcement

Panagiotis Kougias, MD,[a] Peter H. Lin, MD,[a] Alan Dardik, MD, PhD,[b] W. Anthony Lee, MD,[c] Hosam F. El Sayed, MD,[a] and **Wei Zhou, MD,[a]** *Houston, Tex; New Haven, Conn; and Gainesville, Fla*

Abdominal aortic aneurysm (AAA) enlarges after successful endovascular repair because of endoleak, which is persistent blood flow within the aneurysm sac. In the absence of detectable endoleak, AAA may still expand, in part because of endotension, which is persistent pressurization within the excluded aneurysm. We report three patients who underwent successful endovascular AAA repair using the Excluder device (W. L. Gore & Associates, Flagstaff, Ariz). Although their postoperative surveillance showed an initial aneurysm regression, delayed aneurysm enlargement developed in all three, apparently due to endotension. Endovascular treatment was performed in which endograft reinforcement with a combination of aortic cuff and iliac endograft extenders were inserted in the previously implanted stent grafts. The endograft reinforcement procedure successfully resulted in aneurysm sac regression in all three patients. Our study underscores the significance of increased graft permeability as a mechanism of endotension and delayed aneurysm enlargement after successful endovascular AAA repair. In addition, our cases illustrate the feasibility and efficacy of an endovascular treatment strategy when endotension and aneurysm sac enlargement develops after endovascular AAA repair. (J Vasc Surg 2007;46:124-7.)

An abdominal aortic aneurysm (AAA) may continue to enlarge despite a technically successful endovascular repair due to endoleak or endotension.[1] Endotension has been described with the use of the Excluder Endoprosthesis (W. L. Gore & Associates, Flagstaff, Ariz) leading to conversion for graft explantation and open repair.[2] Treatment modalities for this entity are at present subject to debate.

We present three case reports of patients who underwent endovascular AAA repair with the Excluder device in whom postoperative surveillance demonstrated aneurysm expansion as a result of endotension. The patients were successfully treated by reinforcing the Excluder endograft device. Our study suggests that increased permeability of the first-generation Excluder endografts favors the development of endotension and discusses an effective alternative to open repair for the treatment of endotension in high-risk patients.

From the Division of Vascular Surgery and Endovascular Therapy, Michael E. DeBakey Department of Surgery, Baylor College of Medicine and Michael E. DeBakey Veterans Affairs Hospital[a], the Department of Surgery, VA Connecticut Healthcare System, Yale University School of Medicine[b], and the Division of Vascular Surgery and Endovascular Therapy, Department of Surgery, University of Florida College of Medicine.[c]

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Reprint requests: Peter H. Lin, MD, Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston VAMC (112), 2002 Holcomb Blvd, Houston, TX 77030 (e-mail: plin@bcm.edu).

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From October 2000 to July 2004, three patients underwent endovascular AAA repair using the first-generation Excluder device. Detailed clinical information about their treatment course is presented in the Table. The initial AAA diameter, measured as the maximum distance between the outer aneurysm walls, was 5.5 cm in patient 1, 5.9 cm in patient 2, and 5.8 cm in patient 3. All patients had an uneventful recovery after the operation.

During their follow-up surveillance with contrast computed tomography (CT) imaging, all three patients showed AAA enlargement without detectable endoleak. The diameters of their AAAs had enlarged to 6.3 cm, 6.5 cm, and 6.3 cm, respectively (Table). There was no evidence of aortic neck dilatation or stent graft migration. Further investigation with angiography and color duplex ultrasonography failed to detect persistent blood flow in the aneurysm sac. Intrasac pressure of the excluded AAA was measured in patients 1 and 2 by a translumbar approach, which confirmed an equivalent aneurysm sac pressure with the systemic aortic pressure.

Because of their medical comorbidities, the endotension was treated with an endovascular approach. Through bilateral groin cutdowns, an Excluder aortic cuff was implanted in the main portion of the bifurcated device. We then implanted a bilateral Excluder iliac endograft in the respective iliac limb. In two of our patients, the new generation and less permeable Excluder stent grafts were used in the second procedure. This endograft reinforcement essentially doubled the thickness of stent-graft coverage. Balloon dilation was then performed in all reinforced stent grafts.

Table. Clinical information of patients treated with endovascular reinforcement procedure

Patient No	*Age (y)/ Sex*	*Medical comorbidities*	*AAA diameter*	*Initial EVAR date*	*Device implanted**	*Diameter of enlarged AAA in follow-up CT scan/date*	*Date of endograft reinforcement procedure*	*Reinforced device implanted**	*Follow-up diameter of AAA regression/date*
1	63/M	CAD, hypertension, DM	5.5 cm	Jan 2001	1. Aortic main device (26 mm × 14.5 mm × 14 cm) 2. Contralateral iliac limb (16 mm × 11.5 cm)	6.3 cm/ Jan 2003	Mar 2003	1. Aortic cuff (26 mm × 3.3 cm) in the main device 2. Iliac device (16 mm × 13.5 cm) in each iliac limb	5.3 cm/ Sep 2005
2	82/M	Stroke, hypertension, DM	5.9 cm	Jul 2002	1. Aortic main device (26 mm × 14.5 mm × 16 cm) 2. Contralateral iliac limb (16 mm × 11.5 cm)	6.5 cm/ Apr 2005	Jun 2005	1. Aortic cuff (26 mm × 3.3 cm) in the main device 2. Iliac device (16 mm × 13.5 cm) in each iliac limb	6.0 cm/ Apr 2006
3	76/M	Renal failure, CAD stroke	5.8 cm	Nov 2003	1. Aortic main device (28.5 mm × 14.5 mm × 14 cm) 2. Contralateral iliac limb (16 mm × 11.5 cm)	6.3 cm/ Nov 2005	Nov 2005	1. Aortic cuff (26 mm × 3.3 cm) in the main device 2. Iliac device (16 mm x 13.5 cm) in each iliac limb	5.6 cm/ Nov 2006

AAA, Abdominal aortic aneurysm; *EVAR,* endovascular aneurysm repair; *CT,* computed tomography; *CAD,* coronary artery disease; *DM,* diabetes mellitus.
*****Gore Excluder devices (W.L. Gore & Associates, Flagstaff, Ariz).

A completion angiogram in all patients showed fully patent endograft segments without appreciable device-related luminal narrowing. All patients tolerated the procedure well, without complication. In patient 1, the follow-up CT scan 6 months later showed no endoleak and a markedly reduced aneurysm sac measuring 5.3 cm × 5.1 cm in greatest diameter, which represents aneurysm sac shrinkage of 10 mm after the endograft reinforcement procedure. The follow-up CT scan 6 months later in patient 2 showed his aneurysm sac had reduced to 6.2 cm at its greatest diameter without endoleak. A 12-month surveillance CT scan showed an aneurysm sac of 6.0 cm without endoleak. In patient 3, his follow-up CT scan at 6 and 12 months showed the aneurysm sac had reduced to 5.6 cm in diameter without endoleak.

DISCUSSION

Our report is notable because it underscores that increased endograft permeability may result in endotension and constitutes an important mechanism of late aneurysm sac enlargement after early successful endovascular repair. More important, our report highlights a novel endovascular treatment strategy of endotension-induced aneurysm sac enlargement.

Delayed aneurysm expansion developed after endovascular repair in all three of our patients. Despite a thorough investigation with imaging studies, we were unable to detect an endoleak. In two of these patients, the presence of a pressurized aneurysm sac was confirmed with intra-aneurysm pressure measurement. The use of sac cannulation for intra-aneurysm pressure measurement after endovascular repair has previously been reported by our group.[1] Baum et al[3] also described two approaches to intra-aneurysm pressure assessment, which included a direct translumbar access with puncture of the aneurysm sac or a selective cannulation of the inferior mesenteric artery by either the superior mesenteric artery or the hypogastric arteries. We used the former approach to confirm the presence of an endotension in our first and second patients.

The exact mechanism of persistent sac pressurization without detectable endoleak remains poorly understood. Some authors suggest that endotension is a result of direct pressure transmission from the endograft lumen to the aneurysm sac.[4,5] In addition, low-flow endoleak that is not detected with imaging modalities, and subsequent enzymatic degradation of the accumulated thrombus, might lead to aortic aneurysmal wall weakening.[6]

A recently emerging theory claims that endotension can be the result of pressure transmission and fluid accumulation that is facilitated by increased graft porosity, which is the case with the thin polytetrafluoroethylene (PTFE) grafts.[7] This notion is supported by findings from the recent study of Trocciola et al,[8] who reported transudation of serum components in an animal model of aortic aneurysm that had been excluded with an expanded PTFE stent graft. By comparison, aneurysm exclusion with a Dacron stent graft resulted in significantly less pressure transmission in the aneurysm sac and demonstrated greater thrombus organization. This theory appears to be more relevant clinically, supported by the observation that the first generation Gore Excluder device is associated with less aneurysm sac shrinkage compared with the other devices approved by the United States Food and Drug Adminisration.[9,10]

Because all three of our patients had received the first generation Gore Excluder device, we postulated that their aneurysm sac enlargement might have been a device-specific phenomenon. There has been a manufacturing alteration in the current Gore Excluder device, with additional fabric layer reinforcement to decrease the endograft porosity. The clinical significance of this device modification with regards to a decreased incidence of endotension or sac enlargement remains to be validated.

The clinical significance of endotension is not fully elucidated. Despite the documented cases of endotension in patients with the Excluder stent graft, the incidence of endotension-related rupture is very low. Kong et al[2] reviewed data from the multicenter phase I and II clinical trials of the Excluder endograft and reported no endotension-related aneurysm rupture.[2]

More intriguing were the findings of Mennander et al,[11] who treated expectantly five patients with endotension. The aneurysm sac was ruptured in three of their patients during the follow-up, without associated hemorrhage, confirming a continuous increase in sac size and aneurysmal wall tension in the presence of a very effective endograft seal. One of the patients underwent exploratory laparotomy for presumed aneurysm rupture and abdominal pain. Intraoperatively, he was found to have a ruptured aneurysmal sac, no evidence of bleeding, and a large amount of gelatinous material that had been evacuated from the sac into the abdomen. It is evident from this study that sac enlargement is not always associated with blood flow inside the sac, and that continuous filtration of transudative material through the graft may result in sac rupture without major clinical sequelae.

The findings of this study support the basis of our treatment strategy in that endograft reinforcement decreased the device porosity and therefore reduced transudative filtration leading to the resultant aneurysm sac shrinkage. As evidenced by our results, complete relining and isolation from the circulation of the old endograft with another bifurcated device is not necessary. Minimizing the surface of increased porosity that comes into contact with the blood seems to suffice in restoring the pressure equilibrium across the device, therefore abrogating the transudative process.

The management strategies of observed endotension are still evolving. There is a general agreement that a thorough investigation with CT scan, contrast-enhanced duplex ultrasonography, and angiography should be undertaken to rule out the presence of an endoleak, which is sometimes difficult to detect but can be treated with minimally invasive means.[12] Treatment remains controversial. The report by Mennander et al[11] indicates that conservative treatment with close observation only is justified if the endoleak is ruled out with well-performed imaging studies. A recent consensus report, however, advocated that aneu-

rysm sac enlargement associated with endotension needs to be treated.[13]

For patients who are medically fit to undergo an open procedure, conversion to open aneurysmorrhaphy should be considered. Our cases demonstrate that a complete endovascular strategy with endograft reinforcement to decrease endograft porosity is both feasible and effective in patients with aneurysm sac enlargement, particularly those treated with the first generation of the Excluder device.

A few technical issues of the relining procedure are worth further discussion. The entire old endograft was "relined" with the aortic cuff and the extension limbs. The only exception was the area of the main body that remained uncovered by the aortic cuff. To protect the flow divider, the aortic cuff was deployed flush with the proximal end of the main body, offering a 0.7-cm clearance off the flow divider. We found that positioning the deployment sheath close to the flow divider facilitates the positioning and precise deployment of the aortic cuff.

We used iliac limbs slightly longer than those placed upon the original procedure. We had two reasons for doing this: on the ipsilateral side, we wanted to gain fixation length and avoid migration problems in the future; whereas on the contralateral side, we wanted to make sure that we fully overlapped the old attachment site all the way up to the flow divider. Although one could argue that increasing the fixation length might have sealed an undetectable distal type I endoleak, we believe that this unlikely given the detailed imaging we used before reoperation. Finally, reinforcing a small endograft may be risky because of endograft redundancy and the potential of lumen compromise. Until more experienced is accumulated, we would not recommend this procedure for patients with iliac vessels sized <10 mm.

CONCLUSION

Our findings support the concept of endotension secondary to increased permeability as a mechanism of aneurysm expansion after endovascular repair. Although more studies are needed to clarify the natural history and pathophysiology of this process, endovascular repair should be considered as a valid treatment option in the event of continuous aneurysm expansion in high-risk patients. Our report underscores the importance of long-term surveillance after endovascular AAA repair to ensure treatment success.

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