CASE REPORTS

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Hypogastric artery aneurysm rupture after endovascular graft exclusion with shrinkage of the aneurysm: Significance of endotension from a "virtual," or thrombosed type II endoleak

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Type II endoleaks, resulting from retrograde branch flow, after endovascular graft aneurysm exclusion are considered benign because they usually thrombose and are commonly associated with stable or shrinking aneurysm sacs. We report a hypogastric artery aneurysm rupture from endotension from an undetected, thrombosed Type II endoleak, associated with sac shrinkage. The patient had undergone an endovascular graft repair of a 4-cm right common iliac artery and 9-cm hypogastric artery aneurysm with distal hypogastric artery coil embolization. Serial computed tomography scans revealed no endoleak and a hypogastric aneurysm thrombosis with shrinkage. Eighteen months later, the aneurysm ruptured as a result of pressurization from backbleeding, patent branches. (J Vasc Surg 2001;33:1271-4.)

The objective of transluminal endovascular grafting is to exclude aneurysms from the circulation and to prevent rupture by reducing intrasac pressure. An endoleak is the persistence of blood flow outside the graft lumen but inside the aneurysm sac or adjacent vascular segment being treated by the graft, demonstrating a failure of exclusion that may result in aneurysm rupture.[1] The significance of Type II endoleaks from retrograde branch flow remains elusive, because they frequently thrombose, or if persistent, may be associated with stable or shrinking aneurysm sacs.[2] Moreover, they may be difficult to demonstrate, requiring delayed contrast computed tomography (CT) scans. Therefore, many think that persistent Type II endoleaks without aneurysm expansion may be safely observed with CT or angiography.[3] Additionally, some have observed aneurysm rupture without a demonstrable endoleak.[4] The mechanism for this remains obscure but is postulated to be due to "endotension," from pressure transmission through thrombus, after endoleak thrombosis.[5,6]

We describe rupture of a treated, shrinking, nonpulsatile hypogastric aneurysm. The probable cause of rupture was endotension resulting from a Type II endoleak not identified on delayed contrast CT. It raises questions concerning the safety of observing patients with thrombosed Type II endoleaks, even when they are associated with shrinking aneurysms.

CASE REPORT

The patient was an 85-year-old man with a 4.2-cm abdominal aortic aneurysm (AAA), a 4-cm right common iliac artery aneurysm, and a 9-cm hypogastric artery aneurysm (Fig 1, *A*). He was a high risk for open repair because of coronary artery disease, hypertension, hypercholesterolemia, and a right nephrectomy. On July 13, 1998, he underwent an endovascular graft (EVG) repair of his right common iliac artery and hypogastric artery aneurysms. A Montefiore EVG, composed of a Palmaz stent and polytetrafluoroethylene graft, was deployed proximally in the right common iliac artery, as detailed elsewhere.[7] The proximal end of this graft was anchored in the proximal common iliac artery, and the distal end was handsewn within the common femoral artery (Fig 2). Before graft insertion, the distal clot-filled portion of the hypogastric artery aneurysm was coil embolized (Gianturco coils; Cook, Bloomington, Ill) (Fig 2). Delayed contrast CT scans at 1, 3, 6, and 12 months revealed an enlargement of the AAA from 4.2 to 5.3 cm, no change in the 4-cm right common iliac artery aneurysm, and a shrinkage of the excluded hypogastric artery aneurysm from 9 to 8.4 cm (Fig 1, *B*). All CT scans showed absence of contrast outside the graft and, therefore,

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Competition of interest: FJV and TO are part owners of Vascular Innovations, the Corporation that is attempting to commercialize the MEGS graft.

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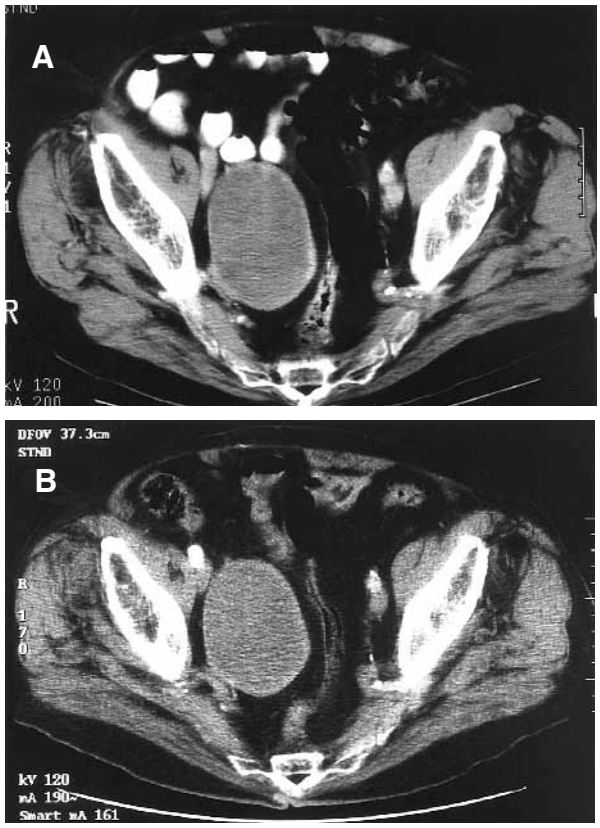

Fig 1. A, 9-cm right hypogastric artery aneurysm. **B,** Shrinkage of right hypogastric artery aneurysm to 8.4 cm a year after endovascular graft exclusion.

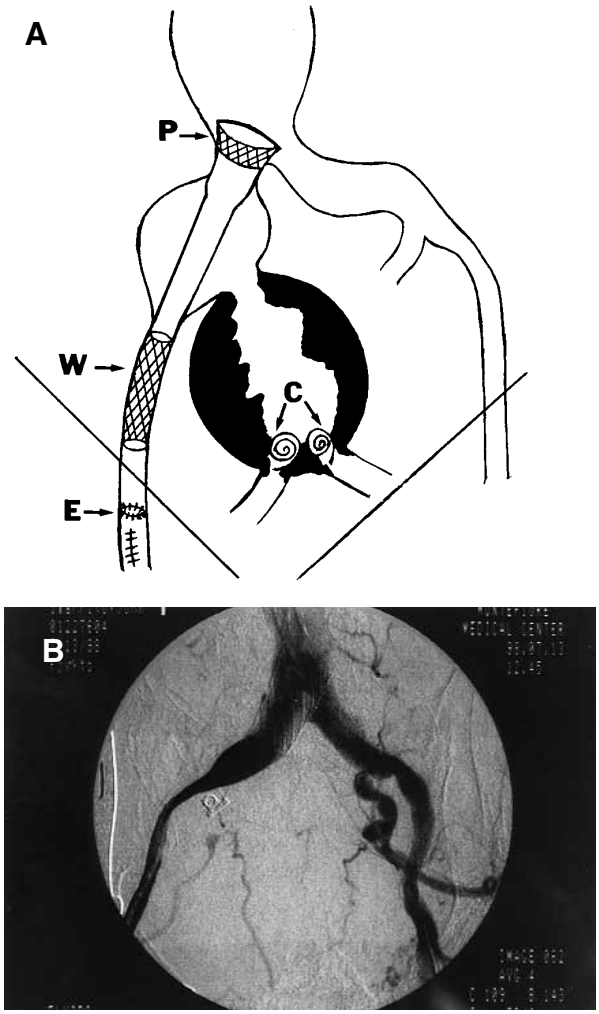

Fig 2. A, Schematic of Montefiore Endovascular Graft System (MEGS) and coil embolization repair of right common iliac artery and hypogastric artery aneurysms. **B,** Postoperative angiogram after placement of MEGS device and coil embolization for repair of right common iliac artery and hypogastric artery aneurysms. *P,* Palmaz stent; *W,* Wallstent; *E*, endoluminal anastomosis; *C,* coils.

no endoleak. In addition, the palpable hypogastric artery aneurysm was nonpulsatile on rectal examination.

Eighteen months later, the patient presented with syncope, right lower quadrant pain, and hypotension. Results of a physical examination revealed a pulsatile abdominal mass and a nonpulsatile, tender right rectal mass. Laparotomy for a ruptured aneurysm revealed a large right lower quadrant hematoma originating from a rupture site in the hypogastric artery aneurysm. When the aneurysm was opened, no bleeding was noted, the clot was removed from the sac, and backbleeding from hypogastric artery branches was observed. Branch backflow and continued sac pressure through clot were deemed the mechanisms of rupture. The branches were oversewn from within the sac, and a bifurcated Hemashield aortoiliac graft (Meadox, Oakland, NJ) was inserted. The right limb was sewn end to end into the EVG within the right external iliac artery. The left limb was anastomosed to the distal left external iliac artery, after oversewing the proximal left common iliac artery (preserving flow to the left hypogastric artery) (Fig 3). After a complicated postoperative course, the patient continues to do well 14 months later.

DISCUSSION

This case is notable for several reasons. First, it documents a rupture of an endovascularly repaired aneurysm resulting from endotension from a nondemonstrable, "virtual" Type II endoleak. Second, it raises questions about aneurysm treatment with interruption of inflow and coil placement within the sac to promote thrombosis. Third, it demonstrates the danger of Type II leaks even after apparent sealing and sac shrinkage. This raises concerns about observational management of a Type II endoleak associated with aneurysm shrinkage or size stability. Finally, it shows that rupture of a fully thrombosed, nonpulsatile aneurysm can occur.

In our patient, the hypogastric artery aneurysm rupture resulted from endotension associated with an undetectable or virtual Type II endoleak from hypogastric artery branches. When the clot was opened and removed

from the aneurysm, retrograde flow was seen from these branches.

Branch vessel or Type II endoleaks often thrombose and cause no harm. In addition, some propose that even if they persist without associated sac expansion, they may be safely observed.[3] With regard to aneurysm size, reduction is considered the criterion of successful treatment,[8] yet in our patient, the hypogastric artery aneurysm ruptured despite its shrinkage and the absence of a demonstrable endoleak on CT scan.

Recently, Baum et al[9] demonstrated that Type II endoleaks can transmit systemic pressure to the aneurysm sac. Furthermore, the behavior of AAAs treated with exclusion and bypass graft facilitates our present understanding of the significance of Type II endoleaks. In 1218 patients with excluded AAAs followed up for 14 years, 48 (4%) had persistent sac flow, and 27 required surgery. Seven patients had rupture of their aneurysms. These ruptures resulted from branch backbleeding, which was proven during a second operation. Furthermore, Darling et al[10] contend that sac expansion may be the only reliable sign to diagnose branch patency into the aneurysm. Finally, Politz et al[11] reported a case of an AAA rupture after an AneuRx graft repair. The rupture was caused by a Type II endoleak associated with patent lumbar and accessory renal arteries. These reports, plus our case, demonstrate that retrograde branch leaks can cause high sac pressures and aneurysm rupture. To our knowledge, our case represents the first report of an aneurysm rupture from endotension from a radiographically nondemonstrable Type II endoleak associated with sac shrinkage. It is possible, of course, that the hypogastric artery aneurysm expanded between the last CT and the time of rupture. Nevertheless, this is disturbing because it means that aneurysm shrinkage does not always mean that further observation is safe, although it may be in many cases.[2] In our case, the increase in size of the aortic aneurysm occurred because the endograft did not extend into it; we left it untreated at the first procedure.

With regard to the absence of a detectable endoleak in our patient, high intrasac pressure was probably maintained by a no-flow, virtual endoleak that could not be identified with conventional imaging.[12] Continued sac pressurization without the presence of an endoleak is the evolving concept of endotension of the Grade III variety (high pressure, no flow, sealed leak).[5] This phenomenon may result from pressure transmission through thrombus that is sealing the aneurysm from flowing blood.[6]

Even thrombosed endoleaks may be dangerous and associated with variable sac pressures.[13] This is consistent with our experimental demonstration that coiling with thrombosis of endoleaks fails to reduce systolic intra-aneurysmal pressures.[14]

Greenberg and Green[15] have suggested that successful aneurysm exclusion should result in a nonpulsatile mass. In our case, however, an initially pulsatile aneurysm became nonpulsatile after exclusion, yet went on to rupture from endotension. Other investigators have shown, in a bench-top model, that every endoleak, even small ones, can cause elevated pressures within an aneurysm sac.[16] Therefore, when clinically feasible in the future, follow-up examinations after stent-graft placement should focus on direct pressure measurements, which may supplant radiographic imaging. In this regard, Baum et al[9] reported two techniques to measure intrasac pressures. One involves catherization of the sac via a patent inferior mesenteric artery accessed via the superior mesenteric artery. The second, which involves direct translumbar sac puncture, is particularly promising and may facilitate effective treatment and elimination of the endoleak.[9] Without such direct pressure measurements, current surveillance programs do not always detect aneurysms at risk of rupture after EVG repair.[4] Accordingly, this is an area in which better methods of follow-up and treatment are required.

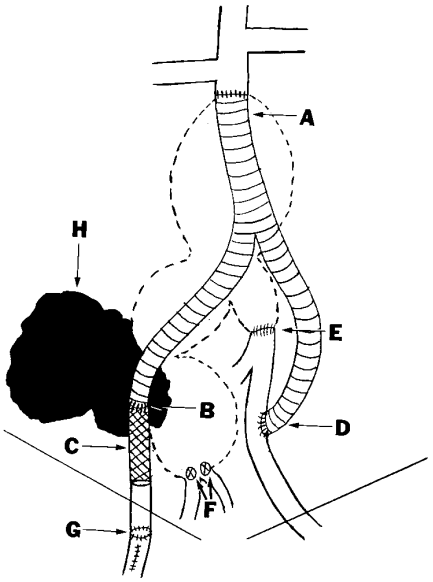

Fig 3. Schematic of aortoiliac bypass graft. *A,* Bifurcated aortoiliac graft; *B,* right iliac limb sewn end to end to previous MEGS device; *C,* previous Wallstent; *D,* left iliac limb sewn end to side to native left external iliac artery; *E,* left common iliac artery oversewn; *F,* right hypogastric artery branches suture ligated; *G,* previous endoluminal anastomosis; *H,* hematoma.

In the management of endoleaks, the efficacy of coil embolization has yet to be established. It is thought that tightly packing coils within an aneurysm allows solid thrombus formation, thereby reducing the risk of rupture. However, we have previously shown that after coil embolization and CT evidence of complete thrombosis of endoleaks, intra-aneurysmal pressure does not decrease significantly.[14] Endotension persists and is transmitted through thrombus, exposing the aneurysmal wall to high pressurization. In our case, placement of coils within the aneurysm sac was an error. Placement of the coils within the nonaneurysmal hypogastric artery branches would have been more effective in preventing retrograde pressure transmission to the sac and subsequent rupture.

REFERENCES

1. White GH, Yu W, May J. Endoleak—a proposed new terminology to describe incomplete aneurysm exclusion by an endoluminal graft. J Endovasc Surg 1996;3:124-5.
2. Resch T, Ivancev K, Lindh M, Nyman U, Brunkwall J, Malina M, et al. Persistent collateral perfusion of abdominal aortic aneurysm after endovascular repair does not lead to progressive change in aneurysm diameter. J Vasc Surg 1998;28:242-9.
3. Jacobowitz GR, Rosen RJ, Riles TS. The significance and management of the leaking endograft. Semin Vasc Surg 1999;12:199-206.
4. Zarins CK, White RA, Fogarty TJ. Aneurysm rupture after endovascular repair using the AneuRx stent graft. J Vasc Surg 2000;31:960-70.
5. Gilling-Smith G, Brennan J, Harris P, Bakran A, Gould D, McWillam R. Endotension after endovascular aneurysm repair: definition, classification and strategies for surveillance and intervention. J Endovasc Surg 1999;6:305-7.
6. White GH, May J, Petrasek P, Waugh R, Stephen M, Harris J. Endotension: an explanation for continued AAA growth after successful endoluminal repair. J Endovasc Surg 1999;6:308-15.
7. Sahgal A, Veith FJ, Lipsitz E, Ohki T, Suggs WD, Rozenblit AM, et al. Diameter changes in isolated iliac artery aneurysms 1 to 6 years after endovascular graft repair. J Vasc Surg 2001;33:289-95.
8. May J, White GH, Yu W, Waugh RC, Stephen MS, Harris JP. A prospective study of changes in morphology and dimensions of abdominal aortic aneurysms following endoluminal repair: a preliminary report. J Endovasc Surg 1995;2:343-7.
9. Baum RA, Carpenter JP, Cope C, Golden MA, Velazquez OC, Neschis DG, et al. Aneurysm sac pressure measurements after endovascular repair of abdominal aortic aneurysms. J Vasc Surg 2001; 33:32-41.
10. Darling RC, Ozsvath K, Chang BB, Kreienberg PB, Paty PS, Lloyd WE, et al. The incidence, natural history, and outcome of secondary intervention for persistent collateral flow in the excluded abdominal aortic aneurysm. J Vasc Surg 1999;30:968-76.
11. Politz JK, Newman VS, Stewart MT. Late abdominal aortic aneurysm rupture after AneuRx repair: a report of three cases. J Vasc Surg 2000; 31:599-606.
12. Gilling-Smith GL, Chong CK, How TV, Brennan JA, Harris PL. Endovascular repair of abdominal aortic aneurysm: how dangerous are side-branch endoleaks? J Endovasc Surg 1999;6:90-1.
13. Wain RA, Marin ML, Ohki T, Sanchez LA, Ross LT, Rozenblit A, et al. Endoleaks after endovascular graft treatment of aortic aneurysms: classification, risk factors, and outcome. J Vasc Surg 1998;27: 69-80.
14. Marty B, Sanchez LA, Ohki T, Wain RA, Fades PL, Cyanmon J, et al. Endoleak after endovascular graft repair of experimental aortic aneurysms: does coil embolization with angiographic "seal" lower intraaneurysmal pressure? J Vasc Surg 1998;27:454-62.
15. Greenberg R, Green R. A clinical perspective on the management of endoleaks after abdominal aortic endovascular aneurysm repair. J Vasc Surg 2000;31:836-7.
16. Schurink GW, Aarts NJ, Wilde J, van Baalen JM, Chuter TA, Schultze Kool LJ, et al. Endoleakage after stent-graft treatment of abdominal aneurysm: implications on pressure and imaging—an in vitro study. J Vasc Surg 1998;28:234-41.

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