Symptomatic Aneurysm Rupture Without Bleeding Secondary to Endotension 4 Years After Endovascular Repair of an Abdominal Aortic Aneurysm

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**Purpose:** To demonstrate the absence of bleeding from a ruptured abdominal aortic aneurysm (AAA) without evidence of endoleak following endovascular aneurysm repair (EVAR).

**Case Report:** A 72-year-old woman developed aneurysm enlargement 4 years after EVAR of an infrarenal AAA. During surgical exploration for abdominal pain and presumed aneurysm rupture, the ruptured aneurysm sac was found to be filled with gelatinous material without evidence of thrombus or active bleeding.

**Conclusion:** This case provides insight into the natural history of endotension and indicates that conservative management even in the face of an expanding aneurysm is a valid management option for selected patients.

**Key words:** abdominal aortic aneurysm, endovascular aneurysm repair, stent-graft, endoleak, endotension, aneurysm expansion, rupture, hygroma

Abdominal aortic aneurysms (AAA) may continue to expand after endovascular aneurysm repair (EVAR) either because of persistent or recurrent sac pressurization (endotension) or because of accumulated extracellular material. Pressurization may be due to blood flow into the sac (endoleak), or it may occur in the absence of endoleak (presumably due to transmission of pressure through thrombus). Rupture of an AAA in the absence of endoleak is uncommon, and treatment strategies for this entity are at present subject to debate.

Recently, we encountered a patient who presented with aneurysm rupture despite cessation of blood flow into the aneurysm. Our experience in this case suggests that rupture in the absence of endoleak can be a purely mechanical phenomenon and may be observed if the patient is free of symptoms indicative of hemorrhage.

**CASE REPORT**

A 72-year-old woman underwent EVAR of a 5.4-cm infrarenal AAA using a first-generation Excluder Endoprosthesis (W.L. Gore & Associates, Flagstaff, AZ, USA) in 2003. Periodic surveillance demonstrated no change in the aneurysm size until the 3-year computed tomography (CT) scan, which showed an increase in the size of the aneurysm to 5.8 cm in maximum diameter. No evidence of endoleak was evident in any of the follow-up CT scans. A few weeks later, she developed abdominal pain and was referred to our facility, where a new CT scan documented an increase...
in the aneurysm size to 7.0 cm and showed evidence of a contained leak in the left retroperitoneum (Fig. 1). Her hemoglobin was 10.2 and her vital signs normal.

She was taken to the operating room with the presumed diagnosis of ruptured aortic aneurysm. A retroperitoneal exposure was performed because of her history of hysterectomy, bowel resection, and multiple operations for lysis of adhesions. During the dissection, a fair amount of gelatinous material was encountered in the retroperitoneum. There was no evidence of blood or recent thrombus. An opening of ~2 cm was evident in the aneurysm sac. Control was obtained, and the sac was completely opened, confirming the presence of gelatinous material and the absence of fresh thrombus inside the sac (Fig. 2). Since the endograft seal was excellent and no endoleak was identified, the thought of leaving the old endograft in place and terminating the procedure was temporarily entertained. However, the natural history of aortic remodeling and endograft migration after the sac has been opened is not clear, and the potential exists for rapid exsanguination should an endoleak develop in the future. Hence, the endograft was partially removed, and the aneurysm was repaired with a bifurcated prosthesis, which was anastomosed in normal aorta proximally and in the endograft limbs distally. The patient tolerated the procedure well and recovered uneventfully.

**DISCUSSION**

Continuous expansion and ultimate rupture of the aneurysm sac after EVAR in the absence of endoleak may be an event of limited clinical significance, as this case indicates. In this context, it underscores the value of observation as a sole treatment strategy in patients who present with expanding aneurysm after successful initial repair and have undergone testing to rule out the presence of endoleak. Sac enlargement is most often linked to the presence of endotension and continuous sac pressurization. Although this might have been the case in our patient, we were not able to confirm the presence of elevated
intrasac pressures due to the urgent nature of the repair. It is notable that our patient sustained a sac growth of 1.2 cm over the course of 3 weeks prior to rupture. Such a rapid size increase in the absence of major endoleak would seem unlikely; however, we did not recover any thrombus from the aneurysm sac to indicate that significant endoleak was the underlying etiology.

The mechanism of sac enlargement without detectable endoleak remains a matter of conjecture. Several theories have been proposed, including increased graft porosity, direct pressure transmission from the endograft lumen to the aneurysm sac, and low-flow endoleak with subsequent enzymatic degradation of the accumulated thrombus that presumably weakens the aortic wall. A combination of the two latter reasons is most likely responsible for the continuous aneurysm expansion in our patient.

The clinical significance of rupture in the absence of endoleak remains poorly understood. Kong and colleagues reviewed data from the multicenter phase I and II clinical trials of the Excluder endograft and reported no endotension-related aneurysm rupture. Mennander et al. expectantly treated 5 patients with endotension but did not prove endoleak; 3 of these patients suffered aneurysm rupture during the follow-up period, but none had a clinical picture indicative of hemorrhage. During exploratory laparotomy for presumed aneurysm rupture and abdominal pain in 1 of these 3 patients, a large amount of gelatinous material was evacuated from the retroperitoneum, but there was no evidence of bleeding. Risberg et al. also found gelatinous material (hygroma) in several AAA patients they treated for sac expansion months to years after EVAR, confirming that sac enlargement is not always associated with blood flow inside the sac. Continuous transudation through the graft may result in sac rupture without hemodynamic sequelae.

Managing the patient with an enlarging aneurysm sac after endovascular aneurysm repair can be challenging. Clearly, the first task is to prove or disprove the presence of associated endoleak. A contract-enhanced CT scan and/or duplex ultrasonography are the most common initial imaging modalities. Angiography can also be utilized if these techniques fail to reveal the presence or type of suspected endoleak. If an endoleak is not found, the presence of endotension can be confirmed by intra-aneurysm pressure measurement using either direct translumbar puncture or an indirect approach via the mesenteric or internal iliac arteries. If endotension is confirmed, then most authors would favor treatment. For patients who are medically fit to undergo an open procedure, conversion to open aneurysmorrhaphy should be considered. Alternatively, van Nes et al. proposed endoscopic aneurysm sac fenestration as a treatment option for growing aneurysms. More recently, our group has reported a less invasive technique that utilizes stent-graft reinforcement with deployment of new graft components to re-line the endograft and minimize porosity-related endotension. This technique is particularly applicable to patients treated with the first-generation Excluder device, for which endotension due to increased porosity is a well recognized problem.

Our case indicates that the natural history of an expanding aneurysm sac in the absence of endoleak is rather benign, and conservative treatment with close observation only is an appropriate alternative. For symptomatic patients in whom endoleak has not been reliably ruled out, a more aggressive treatment approach is reasonable.

Conclusion

Aneurysm expansion due to endotension several years after EVAR can lead to sac rupture in the absence of extraluminal blood flow. Conservative management may be appropriate for asymptomatic patients with documented endotension and enlarging aneurysms on serial CT scans; however, more data are needed to fully elucidate the natural history of endotension and the most appropriate therapeutic strategy.

REFERENCES


