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Delayed migration of a thrombosed aortic endograft within a thrombosed aneurysm sac resulting in continued sac expansion and rupture

Edvard Skripochnik, MD, Nicos Labropoulos, PhD, *and* Shang A. Loh, MD, *Stony Brook, NY*

ABSTRACT

We present the case of delayed migration of a thrombosed aortic endograft within a thrombosed aneurysm sac that expanded and ruptured. Dilation of the aortic neck likely led to endograft migration and exposure of the occluded endograft and aneurysm sac to systemic pressure. Although no endoleak was identified, a key finding on ultrasound showed mobility of the sac thrombus. This may be an indicator of flow within the sac that may predict potential for rupture. Despite thrombosis of the aortic sac and endograft, the risk of rupture still lingers, and thus continued surveillance of occluded endografts may be prudent. (J Vasc Surg Cases and Innovative Techniques 2017;3:115-8.)

Endovascular aneurysm repair (EVAR) is the standard of care for abdominal aortic aneurysms (AAAs). Early landmark trials showed increased reintervention rate and late mortality secondary to late rupture in the EVAR group.[1,2] Higher reintervention rates were observed secondary to stent migration, thrombosis, endoleaks, and delayed rupture.[1-3] Late complications and their subsequent risk for AAA rupture necessitate long-term surveillance.

Aneurysm sac expansion is a significant indicator of endoleaks. Preoperative anatomy can be a predictor of complications after endograft deployment. Tortuous iliac arteries, severely angulated neck, short neck, and other hostile characteristics make for a risky endovascular repair, especially when it is completed outside of the instructions for use (IFU).[4] Yet, even a repair performed strictly per IFU can still be fraught with endoleaks. Natural progression of aortic disease causing further neck dilation or graft thrombosis may be inevitable in the long term.[5,6] Moreover, if a repair fails by endograft occlusion, it does not signal the end of potential complications. The natural history of occluded endografts is not clearly defined.

We present a case of a thrombosed aortic endograft with aneurysm sac thrombosis that migrated secondary to proximal neck dilation. Surveillance duplex ultrasound imaging demonstrated mobile thrombus in the sac without evidence of endoleak. Continued surveillance showed further sac expansion, which eventually resulted in rupture and death. We propose that neck dilation, migration, and systemic pressure transmission contributed to expansion of the thrombosed aneurysm sac and eventual rupture. Furthermore, changes in the quality of the aneurysm sac thrombus may be a predictor of potential rupture. Consent to present this case was obtained from the patient's family.

CASE REPORT

The patient is a 68-year-old woman with a past medical history significant for hypertension, known 4.5-cm thoracic aortic aneurysm, and severe emphysema on home oxygen who underwent EVAR 3 years earlier at an outside hospital. Repair was complicated intraoperatively by thrombosis of the contralateral limb and conversion to an aortouni-iliac graft with a femoral-femoral bypass. Three years later, she presented to our hospital with an ischemic left leg. A computed tomography angiography scan was performed, demonstrating complete thrombosis of the aortic endograft and aneurysm sac with an infected thrombosed bypass graft. A left axillary-femoral bypass was performed with excision of the infected femoral-femoral bypass. Initial follow-up ultrasound examinations of the aneurysm sac demonstrated no growth with a sac size of 4.9 cm. After delayed follow-up at 17 months, an unrelated computed tomography scan demonstrated interval aneurysm sac growth to 5.1 cm without evidence of an endoleak and stable endograft position. Given the unclear cause of the size change and the chance of measurement error, we elected to repeat the study in 6 months. Repeated duplex ultrasound (Fig 1) 6 months later

From the Division of Vascular and Endovascular Surgery, Department of Surgery, Health Sciences Center T19-090, Stony Brook Medicine.

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Correspondence: Edvard Skripochnik, MD, Division of Vascular and Endovascular Surgery, Department of Surgery, Health Sciences Center T19-090, Stony Brook Medicine, Stony Brook, NY 11794-8191 (e-mail: edvard.skripochnik@stonybrookmedicine.edu).

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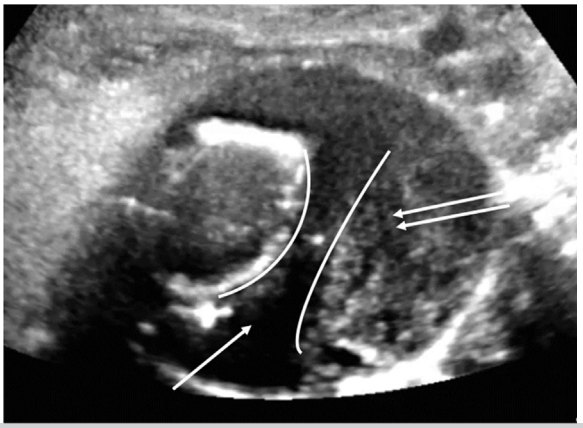

Fig 1. Ultrasound of the abdominal aorta in transverse view at 2 years after presentation showing the occluded endograft surrounded by an aneurysm sac with heterogeneous thrombus. On video imaging, there was mobility of the entire aorta, aneurysm sac, and sac thrombus. The *single arrow* and *white outline* show the region of suspicious thrombus, which was hypoechoic and mobile. The *double arrows* show the hyperechoic region of organized thrombus.

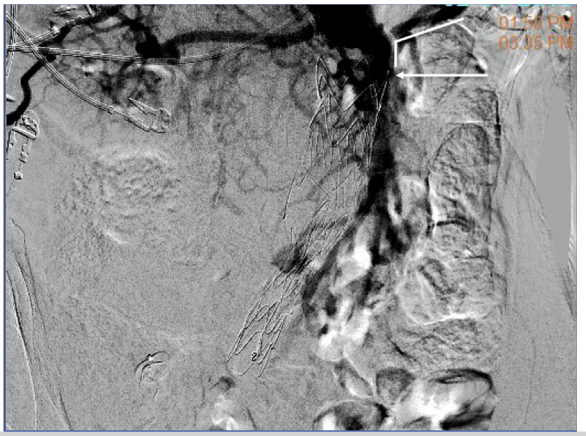

Fig 2. Anteroposterior view aortogram of occluded endograft performed 2 years after presentation. Endograft migration of 5 mm distally (*arrow*) from the left renal artery (*white outline*) with no endoleak visualized.

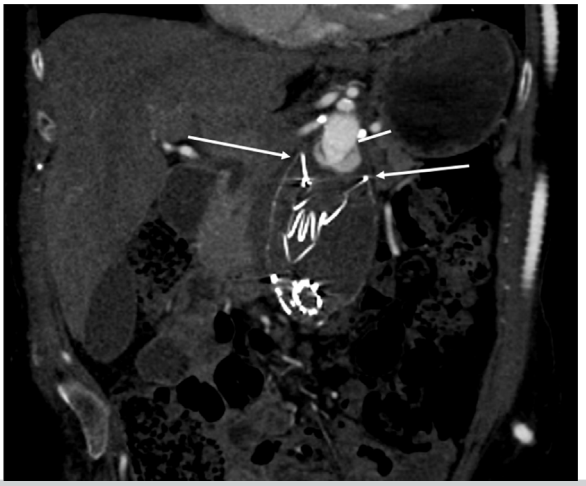

Fig 3. Coronal view computed tomography angiogram of the abdominal aorta at 2.5 years after presentation showing endograft migration with suprarenal struts (*arrows*) >5 mm distal to the lowest renal artery (*white line*). The aneurysm sac measures 6.1 cm, and the aortic neck is 3.6 cm. No endoleak visualized.

demonstrated continued sac growth to 5.6 cm. During the ultrasound examination, motion was detected within the thrombus surrounding the endograft with differing areas of echogenicity within the thrombus. Aortography (Fig 2) was then performed with no evidence of any endoleak but with noted distal migration of the endograft by 5 mm. After extensive discussion with the patient about the lack of findings and the uncertainty about the risk and severity of rupture for a thrombosed AAA, the patient elected for observation. Another computed tomography angiography scan 6 months later (Fig 3) showed aneurysm sac growth to 6.1 cm and further distal migration of the thrombosed endograft with infrarenal aortic neck dilation to 3.6 cm. An extensive discussion was held with the patient and her family regarding repair options. She was offered an open aortobifemoral aneurysm repair with graft explantation or aortic ligation with several extra-anatomic bypass options. An endovascular approach was offered as well: an attempt at rechanneling the occluded aortouni-iliac endograft, proximal extension with a thoracic-sized cuff, proximal fixation with the Aptus (Aptus Endosystems, Sunnyvale, Calif) endoanchors, and Viabahn (W. L. Gore & Associates, Flagstaff, Ariz) stent graft extension to the external iliac artery. She was at high risk for open repair, given her severe emphysema and poor aortic quality. The endovascular option represented an extremely technically challenging repair with a significant chance of failure. After multiple discussions, the patient elected not to undergo repair. She presented 2 months later with a ruptured aneurysm (Fig 4), was given comfort care, and died.

DISCUSSION

The contributing factors that led to aneurysm rupture in this case included preoperative hostile neck, postoperative neck dilation, stent migration, and systemic pressurization of the sac and thrombus. A review of the preoperative imaging revealed that the aneurysm was repaired off IFU because of unfavorable neck anatomy. Aortic diameter at the renal arteries was 19 mm but quickly increased to 32 mm over a 12-mm distance. The suprarenal aorta was dilated to 27 mm in the area of fixation. Together this made the neck prone to further dilation with inferior fixation. Neck dilation placed this graft at risk of caudal migration. A systematic review of post-EVAR patients showed that aortic neck dilation was associated with a 26% rate of type I endoleak, migration, and reintervention compared with 2% without aortic neck dilation.[7]

Stent migration with sac expansion may still be a risk in the setting of a thrombosed endograft. Sac thrombus is

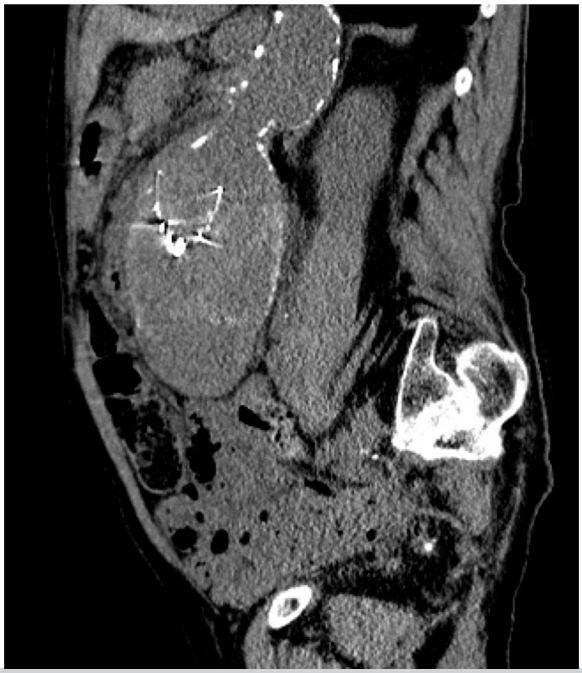

Fig 4. Sagittal view of non-contrast-enhanced computed tomography showing the endograft position at the time of rupture.

not thought to have a protective effect against rupture. Reports have shown that with thrombosis of native aortas, there is only a minor reduction in pressure on the aneurysmal vessel.[8,9] The contribution of type V endoleak to AAA rupture after EVAR is still controversial but has been shown to be a risk factor.[10] Initial theories suggested that direct systemic pressure on the endograft can be transmitted to the aneurysm wall.[11,12] Yet even thrombosed sac rupture with a good endograft seal does not necessarily result in a life-threatening event. There are several documented cases of sac rupture without hemorrhage or hemodynamic compromise with findings of thrombotic contents within the abdomen on exploration.[13] There may be many more instances of thrombosed sac rupture that go undetected with survival of the patient. The difference between aneurysm sac thrombus that may lead to hemorrhage and that which goes unnoticed is unknown.

The characterization of aneurysm sac thrombus with ultrasound imaging may be a useful tool in predicting risk of rupture with hemorrhage, once it is fully understood. We were able to identify an area of sac thrombus in our patient, namely, near the endograft (Fig 1), which was mobile and thus not as organized as would be expected after years of sac exclusion and thrombosis. Either a persistent slow undetectable leak or an intermittent endoleak maintained fresh thrombus within the sac. Lorelli et al[14] postulated that an undetectable leak may allow enzymatic degradation of sac thrombus and lead to further growth of the aneurysmal wall. Given the nature of a thrombus, it may have absorbed blood from the endoleak to form a softer, more gelatinous consistency. Transmission of fluid pressure through a gelatinous or liquefied thrombus is significantly greater compared with a solid thrombus and may be enough to cause expansion. These subtle changes to the thrombus may have caused significant pressure transmission and further weakening of the aortic wall.

The natural history of an expanding thrombosed sac within an occluded endograft is unclear. Causes of continued sac expansion, such as delayed endoleak or unidentified intermittent endoleak, have not yet been elucidated. Close evaluation of sac thrombus for concerning characteristics including heterogeneity, mobility, and hypoechogenicity can be helpful in gauging risk. Thrombus mobility within an otherwise thrombosed sac may serve as an indicator of blood flow within the sac. Further investigation into the nature of a mobile component to a thrombosed sac should be considered. Early detection may offer a potential for elective repair. However, the indications for reintervention in this clinical situation are also not clear. Overall, even in the setting of a thrombosed endograft, continued surveillance following the standard yearly protocol is warranted.

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