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

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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

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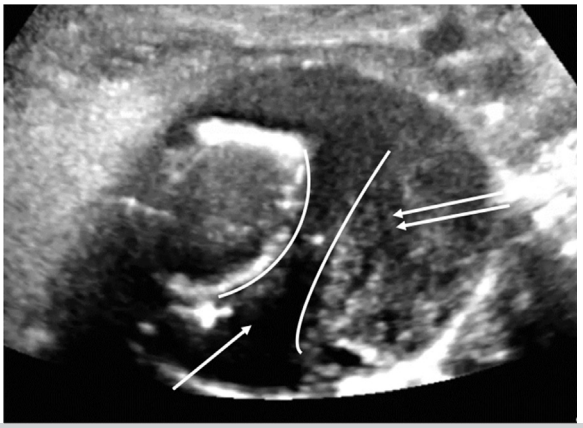

Fig 1. Ultrasound of the abdominal aorta in transverse view at 2 years after presentation showing the occluded endograft surrounded by an aneurysm sac with heterogeneous thrombus. On video imaging, there was mobility of the entire aorta, aneurysm sac, and sac thrombus. The *single arrow* and *white outline*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

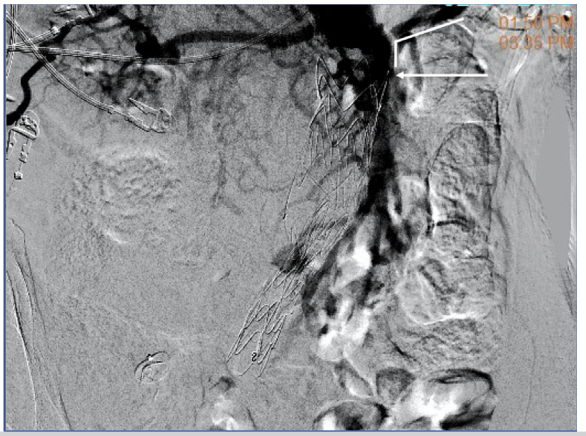

Fig 2. Anteroposterior view aortogram of occluded endograft performed 2 years after presentation. Endograft migration of 5 mm distally (*arrow*) from the left renal artery (*white outline*) with no endoleak visualized.

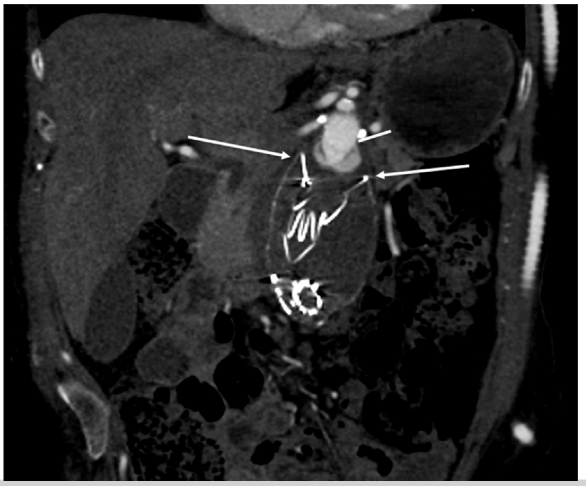

Fig 3. Coronal view computed tomography angiogram of the abdominal aorta at 2.5 years after presentation showing endograft migration with suprarenal struts (*arrows*) >5 mm distal to the lowest renal artery (*white line*). The aneurysm sac measures 6.1 cm, and the aortic neck is 3.6 cm. No endoleak visualized.

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