Development of endotension after multiple rounds of thrombolysis after endovascular aneurysm repair

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Endoleaks, defined as blood flow outside the graft but inside the aneurysm sac, are a common complication after endovascular aneurysm repair. Sometimes however, for reasons not fully understood, expansion of the aneurysm sac can occur with no identifiable endoleak, a phenomenon termed endotension, or a type V endoleak. We describe a case of endotension in a 71-year-old man that developed after recurrent stent graft thrombosis requiring thrombolysis 3 years after the initial endovascular implantation. To our knowledge, this is the first description in the literature of endotension after multiple rounds of thrombolytic treatment. (J Vasc Surg Cases 2015;1:24-7.)

The goal of endovascular aneurysm repair (EVAR) is exclusion of the aneurysm sac, which, if successful, may result in evidence of the aneurysm sac shrinking on follow-up imaging. Although EVAR has many benefits over open surgery, it also has its own specific potential complications. If aneurysm sac expansion occurs postoperatively, it is usually due to the development of an endoleak, defined as blood flow outside the graft into the aneurysm sac. Endoleaks are one of the commonest complications after EVAR. The culprit blood flow can usually be seen on imaging, and the endoleak is appropriately classified: type I involves proximal or distal seal zones, type II involves patent branch vessels, type III refers to component junction or fabric failure, and type IV refers to fabric porosity; finally, persistent sac growth with no identifiable leak is termed type V endoleak or endotension. The etiology of endotension is still a topic of debate, but there are several theories of its pathogenesis.

Another complication of EVAR is stent graft thrombosis, often resulting in emergency presentation with acute limb ischemia. In the absence of any absolute contraindications, graft thrombosis can often be successfully treated by the use of thrombolytic agents, such as recombinant tissue plasminogen activating factor (r-tPA). Thrombolytics create a generalized lytic state, such that target thromboemboli and protective hemostatic thrombi are both broken down, resulting in restoration of luminal patency and the potential for unwanted bleeding, respectively.

r-tPA is a recombinant serine protease with a half-life of 4 to 6 minutes, which preferentially activates plasminogen bound to fibrin and thus theoretically confines fibrinolysis to formed thrombus, such as that found in an occluded stent graft, rather than systemic activation.

In this case study, we present an example of the development of endotension preceded and thus potentially caused by repeated treatment of stent graft thrombosis with thrombolysis, 3 years after EVAR. The patient was notified of this interesting case and consented to publication.

CASE REPORT

In 2011, a 69-year-old man underwent elective EVAR with a Cook Flex bifurcated graft (Cook Medical, Bloomington, Ind) for a 5.9-cm infrarenal abdominal aortic aneurysm (AAA) and made an uneventful recovery (Fig 1, a). A postoperative computed tomography angiogram (CTA) at 6 weeks showed good position of the graft, with no endoleaks or graft kinks (Fig 1, b). A duplex ultrasound scan at the 6-month follow-up showed appropriate reduction of the AAA to 5.4 cm, with no observable endoleaks or limb narrowing.

The patient presented again 17 months after the initial implant, complaining of a 2-week history of worsening exertional right buttock and lower limb pain, consistent with new intermittent claudication but also associated with numbness and paresthesia in the lower limb. On examination, he had a palpable but
decreased femoral pulse and absent distal pulses on the right side, which were previously present.

A CTA showed recurrent occlusion of the right limb of the graft, with no graft displacement or kink. Of note, his anticoagulation therapy had been subtherapeutic for the previous 2 to 4 weeks. He again received thrombolysis via right common femoral access with 10-mg bolus of r-tPA, followed by 1 mg/h for 18 hours (Fig 2, b).

Acute abdominal pain developed after this procedure. A further CTA showed complete resolution of the thrombus and patent graft and vessels, but with a large increase in the AAA diameter from 5.4 cm to 6.6 cm, with evidence of retroperitoneal stranding but no frank hematoma. This raised the possibility of acute endotension (Fig 3, a). He was closely monitored, and a repeat CT scan 72 hours later showed some shrinkage of the aneurysm sac to 6.4 cm, with resolution of the previous retroperitoneal findings likely secondary to cessation of thrombolysis.

Because the patient’s symptoms resolved and he remained hemodynamically stable, he was continued on conservative management and was discharged once his international normalized ratio was within the therapeutic range. Follow-up with duplex ultrasound imaging 6 weeks later showed a widely patent graft with a

Fig 1. a, Completion angiogram at the time of endograft implantation. b, The postoperative axial computed tomography angiography (CTA) displays shrinkage of the aneurysm sac to 5.4 cm.

Fig 2. a, Computed tomography angiography (CTA) axial image shows occlusive thrombus in the right limb (arrow). b, Completion angiogram after the second treatment with thrombolysis shows patent graft limbs.
6.3-cm AAA sac and no evidence of endoleak. A subsequent duplex assessment at 29 months showed a sac size of 4.6 cm with no endoleak (Fig 3, a).

DISCUSSION

The goal of EVAR is to successfully repair an AAA and thus prevent death due to rupture. However, complications from EVAR, such as endoleak, may occur, and if persistent can cause AAA expansion with resultant rupture. This also applies to endotension, the least understood of all endoleaks. Our patient demonstrated this complication after the need for multiple rounds of thrombolysis. To our knowledge, no cases of the development of endotension after thrombolysis treatment are documented in the literature.

As stated previously, the pathophysiology underlying the development of endotension remains unclear. The three main theories are:

- Direct pressure transmission from the aortic lumen due to thrombus formation or undetected endoleak;
- Porosity of the graft causing microleaks and transudation;
- Pressure build up from accumulation of sac contents such as thrombus fibrinolysis/hygroma and enzymatic degradation.

The endotension in our patient can be supported by a combination of the latter two theories. Endotension generally manifests as delayed aneurysm expansion after EVAR, and the case described here appears to be related to the use of thrombolysis. Despite extensive imaging studies, no endoleaks were found to account for enlarging sac size. In one case study, four patients presented with endotension after EVAR with clear, highly viscous fluid in the aneurysm sac but no bleeding, which led to the diagnosis of aneurysm sac hygroma and endotension. Analysis of sac contents revealed prominent local active hyperfibrinolysis and coagulation factors. The authors postulated that local active factors may cause rebleeding, liquefaction, and continued sac expansion.

We propose that the presence of thrombolytic agents in the area of the graft may have contributed to an increased porosity of the graft, which allowed increased transudation into the aneurysm sac and thus sac expansion with no identifiable endoleak. Because the patient’s symptoms resolved after the second round of thrombolysis, no treatment for endotension/sac expansion was performed.

Because the cause of endotension is not yet clear, management is highly variable. A sac that reduces in size after initial enlargement is considered free of endotension, and conservative treatment may be most appropriate. Although some studies suggest open conversion surgery, others suggest conservative management is generally appropriate, assuming the patient is asymptomatic and the sac size is stable. Therefore, until more is known about the natural history and management of endotension, rigorous surveillance is important in these patients.

CONCLUSIONS

The pathogenesis and natural history of endotension are not yet clearly defined. Recurrent rounds of thrombolytic therapy after EVAR may lead to the development of endotension. Conservative management may be most appropriate in asymptomatic patients; however, close surveillance is essential to monitor the patient for rupture and other complications that may require further intervention.

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