Recanalization of previously thrombosed type II endoleak with aneurysm sac expansion after systemic thrombolysis

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ABSTRACT

Patients who have undergone endovascular aneurysm repair (EVAR) need lifelong monitoring because of the risk of aneurysm rupture secondary to delayed endoleaks. Thrombolytic therapy may expose patients with previous EVAR to the risk for development of new endoleaks. We describe a case in which a single dose of intravenous tissue plasminogen activator for acute ischemic stroke was complicated by aneurysm sac expansion secondary to a recurrent endoleak. The potential for a life-threatening complication may warrant routine imaging evaluation of the stent graft after systemic tissue plasminogen activator therapy for acute ischemic stroke in patients with previous EVAR. (J Vasc Surg Cases and Innovative Techniques 2018;4:262-4.)

Keywords: AAA; EVAR; Endoleak; Acute ischemic stroke; Thrombolytic therapy; tPA

Patients who undergo endovascular aneurysm repair (EVAR) require long-term surveillance because of persistent or delayed occurrences of endoleaks and development of endotension, which may expose them to a recurrent risk of aneurysm rupture.[1] Although the incidence of aneurysm rupture after EVAR is low (approximately 0.9% according to a recent meta-analysis), the predominant majority of cases are associated with an endoleak.[2] Thus, adequate surveillance is an important component of postoperative management after EVAR.

Approximately 25% of patients will develop an endoleak at some point after EVAR,[3] the majority of which are type II endoleaks.[1,3] More than 50% of type II endoleaks are discovered at follow-up[4] and are detected >1 year after the initial surgery.[5] We currently have a limited understanding of the risk factors associated with persistent or new type II endoleaks, some of which include older age,[4-6] anatomy (aneurysm diameter,[6,7] number of patent aortic branches[6]), graft type,[4] hypogastric coil embolization,[4] and chronic anticoagulation therapy.[8-10] Two previous reports of EVAR-related complications after catheter-directed, intra-arterial administration of tissue plasminogen activator (tPA)[11] and urokinase[12] suggest that exposure to thrombolytic therapy is another potential risk factor. Here, we present a case in which intravenous (IV) tPA therapy for acute ischemic stroke (AIS) resulted in aneurysm sac enlargement associated with a recurrent type II endoleak. The patient provided consent for publication of the images and clinical details in this report.

CASE REPORT

An 82-year-old woman with no history of smoking and a past medical history significant for hypertension, coronary artery disease, and atrial fibrillation, receiving warfarin therapy for 14 months, underwent EVAR for an incidentally found infrarenal abdominal aortic aneurysm. This 6-cm fusiform aneurysm (Fig, *A*) with a 3.6-cm aortic neck, 127-degree neck angulation, and 4.5-cm (right) and 3.6-cm (left) iliac seal distances was repaired with an Endurant (Medtronic, Santa Rosa, Calif) stent graft. She had an immediate postoperative type II endoleak through the inferior mesenteric artery, which was observed. Her endoleak resolved 9 months after the procedure, and a computed tomography (CT) scan 3 years later showed an aneurysm sac size of 4.6 cm (Fig, *B*).

Four years later, the patient presented to the emergency department with acute-onset left facial droop, dysarthric speech, and left hemiparesis along with severe hypertension (227/103 mm Hg). Because of concerns for AIS, the patient was given IV tPA after lowering of her blood pressure with IV labetalol. The initial bolus dose of IV tPA (7.2 mg) was administered 1 hour and 45 minutes from symptom onset. The presenting neurologic symptoms resolved within 2 minutes of tPA administration; however, 1.5 hours later, the patient developed new-onset right flank pain. Her blood pressure at this time was 190/101 mm Hg. CT angiography showed interval expansion of the aneurysm sac to 6.4 cm with findings suggestive of a type II endoleak (Fig, *C*). There was no sign of aneurysm rupture. Subsequent angiographic evaluation could not identify a definitive source of the leak, and transarterial embolization of branch vessels was unsuccessful because of the inability to selectively access the middle colic or lumbar arteries. Aneurysm sac obliteration through direct sac puncture and the possibility of surgical graft explantation was discussed with the patient. However, in

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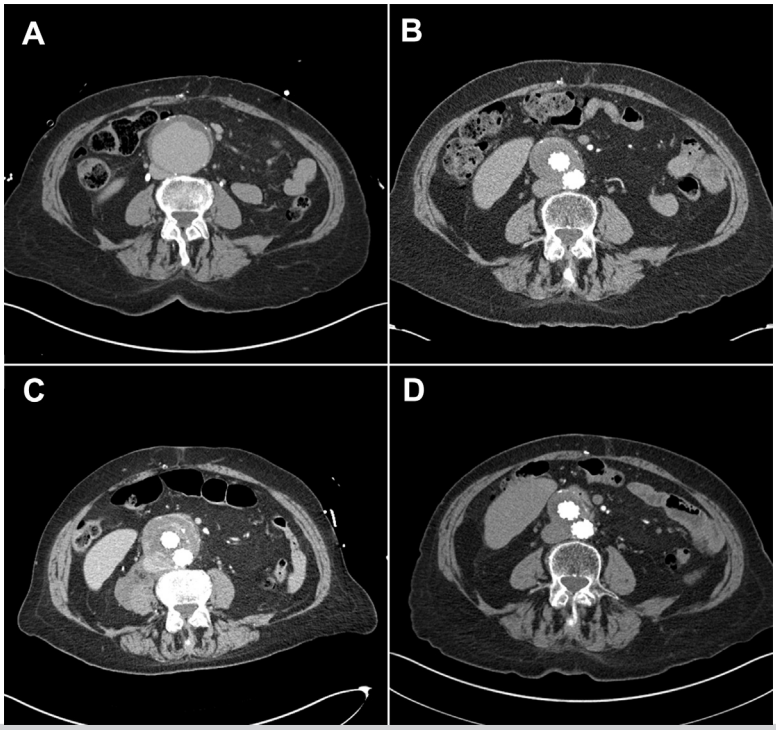

Fig. Computed tomography (CT) evaluation of aneurysm before and after endovascular aneurysm repair (EVAR), before and after tissue plasminogen activator (tPA) administration. **A,** Non-contrast-enhanced axial CT image of infrarenal abdominal aortic aneurysm before surgery. **B,** Intravenous (IV) contrast-enhanced axial CT image 3 years after EVAR. **C,** IV contrast-enhanced axial CT image taken after tPA administration. Note the heterogeneous hyperdensity within the aneurysm sac suggestive of an endoleak. The aneurysm sac has expanded compared with the previous image. **D,** IV contrast-enhanced axial CT image taken 5 months after tPA administration. The aneurysm sac has shrunk in size.

considering the morbidity associated with additional interventions, the patient wished to proceed with nonoperative management.

The abdominal pain resolved, and repeated CT scans on hospital days 2 and 5 demonstrated a stable aneurysm sac size. The patient was discharged home on hospital day 6. A repeated CT scan 5 days after discharge was again stable. Antiplatelet and anticoagulation therapy was initially held but resumed 2 weeks after discharge because of a transient ischemic attack. Additional CT scans 1 month and 5 months after discharge demonstrated a decreased aneurysm sac size to 4.8 cm (Fig, *D*), and no further evidence of endoleak was detected on ultrasound.

DISCUSSION

Previous reports of EVAR-related complications after thrombolytic therapy occurred in the setting of catheter-directed, intra-arterial administration of tPA[11] or urokinase[12] for the treatment of acute limb ischemia. This case suggests that systemic IV tPA therapy for AIS can also expose post-EVAR patients to the risk for EVAR-related complications. Our patient had a stable residual aneurysm sac and no evidence of endoleak for >3 years after EVAR. This was also evident on a duplex ultrasound study 2 months before the described complication after tPA therapy. The temporal relationship between tPA therapy and sac expansion associated with a type II endoleak led us to conclude that sac expansion was due to recanalization of her previous endoleak. The patient's concurrent severe hypertension probably contributed to the acute symptomatic sac expansion.

The Society for Vascular Surgery recommends treatment of endoleaks associated with aneurysm sac expansion ≥5 mm.[1] Catheter-directed embolization of inflow vessels was attempted but unsuccessful. The decision not to pursue aggressive treatment was deemed appropriate in the setting of resolving symptoms and a stable aneurysm sac size on repeated imaging. The aneurysm was monitored closely with repeated CT imaging until confirmation of a reduced residual sac size.

Although there were brief interruptions, the recurrent endoleak in this patient resolved despite continued warfarin therapy. The role of chronic anticoagulation therapy in persistence or new occurrence of endoleaks is unclear. Johnson et al[13] showed that warfarin had no effect on the incidence of endoleak after EVAR. A recent meta-analysis, however, showed that warfarin therapy almost doubled the risk of type II endoleaks.[9] Although some reported a higher rate of persistent type II

endoleaks in patients receiving warfarin,[10] others found that the rate of spontaneous resolution of type II endoleaks was unaffected by warfarin.[8] More important, as this case illustrates, discontinuation of anticoagulation therapy may not be feasible in some patients.

There are currently no guidelines on the use of systemic tPA therapy for AIS in patients with previous EVAR. Providers must base their decision on relative contraindications to tPA therapy, including recent major surgery and arterial or venous puncture of noncompressible vessels.[14,15] Despite the potential for complications, however, previous EVAR should not preclude the patient from receiving tPA for AIS. Patients should be closely monitored, and CT angiography of the aorta should be performed immediately in the setting of new-onset abdominal pain.

CONCLUSIONS

This case illustrates the potential for aneurysm sac expansion after a single dose of IV tPA therapy for AIS in patients with previous EVAR. The potential for a life-threatening complication may warrant routine imaging evaluation of the EVAR after tPA therapy for aneurysm sac expansion. If aneurysm sac expansion is noted after tPA therapy, the decision to treat should be individualized, but expectant management as a first step may be reasonable in the absence of a rupture.

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