Sac Expansion with Vasa Vasorum-Related Type II Endoleak after Endovascular Aortic Repair Managed by Translumbar Direct Sac Embolization Using Glue

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Abstract

An 84-year-old man with an abdominal aortic aneurysm with a largest diameter of 50 mm was treated using endovascular aortic repair (EVAR) within the instructions for use. Computed tomography (CT) performed 3 years after the EVAR revealed significant sac expansion. The first (3 years after EVAR) and second (4 years after EVAR) transarterial embolizations demonstrated rare type II endoleaks from the vasa vasorum as multiple fluffy contrast blushes from the aortic tributaries. As sac expansion persisted at 5 years after the EVAR, translumbar direct puncture of the sac and embolization using a mixture of 25% n-butyl cyanoacrylate and lipiodol was performed. A follow-up CT 2 years after the last embolization showed sac stability without any complications.

Key words: endovascular aortic repair (EVAR), endoleak, embolization, vasa vasorum

Introduction

Type II endoleaks after endovascular aortic repair (EVAR) are caused by retrograde perfusion of the space between the aneurysm wall and the stent-graft within aortic side branches. Such endoleaks are the most common adverse events during EVAR follow-up, and persistent type II endoleaks can contribute to sac expansion during the follow-up period, necessitating secondary interventions. Many arteries arising from the abdominal aorta can be a source of type II endoleaks, including the inferior mesenteric artery (IMA), lumbar arteries, the middle sacral artery, and accessory renal arteries. Here, we report a rare case of chronic abdominal aortic aneurysm (AAA) expansion after EVAR with persistent type II endoleak from the vasa vasorum. The sac was eventually stabilized by an n-butyl cyanoacrylate and lipiodol (NBCA-LPD) (N-butyl cyanoacrylate; Histoacryl, B. Braun, Melsungen, Germany) (Lipiodol; Guerbet Japan, Tokyo, Japan) injection administered via translumbar direct puncture after failed transarterial embolization procedures.

Case Report

Our institution’s review board approved the preparation of this report, and patient consent for publication was obtained. An 84-year-old male with AAA with a largest diameter of 50 mm was treated via EVAR with an Excluder stent-graft (W.L. Gore Associates, Inc., Flagstaff, AZ) after occlusion of the right internal iliac artery with coils. The EVAR was completed without any endoleaks, and the patient was discharged uneventfully and followed without any oral anticoagulation or antiplatelet agents; his course appeared to be stable with suspected type II endoleak. A CT performed 3 years after EVAR revealed significant growth of the aneurysm, with a largest diameter of 55 mm. The endoleak was...
The endoleak was not apparent on CT in the arterial phase (a); however, faint enhancement of the endoleak was observed in the anterior and posterior parts of the sac on CT during the delayed phase (b).

Under local anesthesia, a 5F sheath was placed through the left common femoral artery. Type I and type III endoleaks were excluded based on an aortogram. After cannulation of the superior mesenteric artery (SMA) by a guiding catheter, an angiogram from a microcatheter placed in the IMA via Riolan’s arch showed that the IMA was occluded at the ostium and that multiple small reticular arterial plexuses arising from the IMA supplied multiple fluffy and hazy contrast blushes associated with type II endoleak (Fig. 2a). Subsequently, embolization was performed via gentle injection of 1 mL of 25% NBCA-LPD. CT performed immediately after embolization revealed that NBCA-LPD was distributed not only at the endoleak channel but also within the aortic wall, proving that the type II endoleak was supplied
Fig. 3. Third reintervention by direct sac puncture. DSA from the needle (a) revealed residual patchy mottled enhancement [white arrows in (a)] of the aneurysmal cavity. A radiograph during the translumbar puncture and embolization using NBCA-LPD (b) showed accumulation and retention of the glue cast [black arrows in (b)] within the aneurysmal cavity. Note: white arrowheads = needle, black arrowheads = coil in the 4th lumbar artery, S = stent-graft

from the vasa vasorum (Fig. 2b). Consequently, embolizations of the right and left 4th lumbar artery, the left 3rd lumbar artery, and the nidus using 25% NBCA-LPD and microcoils were conducted via the left iliolumbar artery. The patient was discharged uneventfully and followed up. CT conducted 1 year after the first reintervention revealed continuing enlargement of the aneurysmal sac, which measured 60 mm, and a residual type II endoleak above the aortic bifurcation was noted.

A second transarterial embolization procedure was then performed because we speculated that the endoleak was supplied by the middle sacral artery. A selective middle sacral arteriogram obtained via the left lateral sacral artery revealed a hazy-shaped type II endoleak supplied by tiny reticular arteries arising from the middle sacral artery that were similar to those observed in previous reinterventions. These arteries and the endoleak channel were embolized using 1.6 mL of 25% NBCA-LPD. A CT conducted after the embolization showed accumulation of NBCA-LPD within the aortic wall and in part of the endoleak channel.

A follow-up CT performed 1 year after the second reintervention revealed growth of the aneurysm, which measured 65 mm in diameter, and residual endoleak (Fig. 4a). For the third reintervention, we recommended CT-guided direct puncture of the sac under local anesthesia in the prone position. A 19G polypropylene coaxial needle (ELASTER, Hakko, Tokyo, Japan) was percutaneously inserted into the endoleak channel. Baseline intrasac digital subtraction angiogram (DSA) was then conducted by manual injection of 10 mL contrast medium to verify the endoleak channel (Fig. 4a). The endoleak channel was visualized as patchy with mottled enhancement, and inflow and outflow vessels, including the vasa vasorum, were not visualized on the angiography (Fig. 3a). A microcatheter was coaxially advanced through the needle toward the desired portion of the endoleak channel followed by glue injection. Exchanging the microcatheter through the needle, this process was repeated until the glue cast was distributed across the entire endoleak channel. A total of 35 mL of 25% NBCA-LPD was injected in 3 sessions, which was followed by tract embolization with NBCA-LPD (Fig. 3b). After this procedure, the patient was discharged uneventfully. A follow-up CT 2 years after the final intervention showed that the aneurysm diameter was stable (Fig. 4b) compared with the CT 1 day after the procedure (Fig. 4a).

Discussion

The vasa vasorum of the aorta is a microvascular structure that forms an arterial plexus within an adventitial layer and originates from the lumbar or mesenteric arteries [1]. Physiologically, it feeds the aortic wall [2]. Marked dilatation of the vasa vasorum within the aortic wall has been reported during exploration of the enlarged AAA sac after EVAR [3] and in histological specimens of the aortic wall after the placement of a stent-graft in experimental studies [3, 4]. In one report [4], the hypoxic environment in the aortic wall after EVAR was found to evoke proliferation of the vasa va-
sorum not only in the outer layer of the aorta but also in the neointima, predominantly during the early phase after stent-graft placement. Another possible cause of proliferation of the vasa vasorum is degenerated contents within the cavity, which are derived from a chronic thrombus produced via fibrinolytic and recoagulation processes [3]. These thrombus-derived contents can provoke inflammation of the aorta and proliferation of the vessel. The vasa vasorum type II endoleak may reasonably be formed via the neovascularization of the thrombus within the lumen through inflammatory or organizational processes or through extravasation from fragile proliferating microvessels into the lumen, which may simulate the formation of a chronic expanding hematoma [5].

Similar to this case, other reported postcontrast CT findings of a rare endoleak from the vasa vasorum were more apparent during the delayed phase and presented as peripheral faint enhancements [6, 7]. On selective digital subtraction angiogram, this rare endoleak appears fluffy and hazy, with patchy contrast blushes supplied from small reticular arterial plexuses arising from aortic small tributaries [6, 8]; this description is similar to that provided in previous reports [6, 8], and these findings may be pathognomonic for vasa vasorum type II endoleaks.

The treatment options for type II endoleak include transarterial embolization, translumbar embolization, and surgical interventions. Transarterial feeder occlusion is associated with a significant incidence of type II endoleak recurrence and sac expansion, even after embolization. During the embolization of type II endoleak, the nidus must be completely obliterated to achieve stability [9]. In the described case, the type II endoleaks from the vasa vasorum were supplied by multiple reticular arteries and formed a separated complex nidus; therefore, it is easy to imagine that obliteration of the entire nidus by transarterial embolization alone would be rather difficult and unsuccessful. The translumbar approach provides direct access to the endoleak cavity, potentially allowing for occlusion of the entire endoleak using NBCA-LPD despite the complexity of the nidus supplied by the vasa vasorum. However, classical type II endoleaks from aortic tributaries may also be complicated by these vasa vasorum type II endoleaks. The question of which treatment is most appropriate or whether a combination of transarterial and translumbar embolization is effective to control this complex endoleak should be investigated further.

Other liquid embolic agents, including Onyx (Medtronic, Santa Rosa, CA) or a mixture of n-butyl cyanoacrylate, lipiodol, and ethanol (NLE) [10], can be used to occlude the endoleak channel. A large amount of Onyx is required to occlude the entire endoleak channel within the aneurysm, which can cause a beam-hardening artifact on follow-up CT scans. Another disadvantage of Onyx is its higher cost. NLE is a recently reported embolic agent [10] that has a less adhesive nature than NBCA-LPD, making it easier to inject a large amount of NLE though the microcatheter or needle; however, its durability and effectiveness have not yet been proven at this time of writing.

Conclusion

Rare type II endoleaks from the vasa vasorum were detected on delayed-phase contrast-enhanced CT and demonstrated by selective angiograms. These endoleaks contributed to sac expansion, which was successfully controlled after sac embolization using NBCA-LPD with translumbar direct puncture.
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References