Brief Communication

Endotension as a Rare Complication to Endovascular Abdominal Aortic Aneurysm Repair

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Abstract

Endotension remains an enigmatic rare cause of endovascular abdominal aortic aneurysm repair failure leading to aneurysm growth and/or rupture. We present a patient with a long-standing endotension treated with open reconstruction and graft explantation. We also provide a unique clinical video, which gives a key view of the intrasac operative findings.

Keywords

endotension, endoleak, abdominal aortic aneurysm, open repair, graft explantation

Introduction

Endovascular abdominal aortic aneurysm repair (EVAR) has gained widespread applicability during the past decades in preference to open repair. However, there are concerns regarding the long-term efficacy of EVAR, as post-EVAR ruptures may occur at a rate of 2% to 3%.[1] They are attributed to complications, mainly endoleaks and stent-graft migration. Sometimes these complications are triggered from post-EVAR aneurysm remodeling. This may include aneurysm shrinkage or aortic elongation and/or dilatation due to progression of the aneurysmal disease in the aneurysmal neck and/or iliacs. These alterations compromise the graft's proximal and distal fixation and sealing zones with detrimental results. Endotension is defined as aneurysmal sac pressurization and expansion without a demonstrable endoleak, appearing in 1% to 5% of EVAR cases.[2] The term "sac expansion without evident leak" has been suggested as an alternative.[3] It may be asymptomatic, cause pain, or lead to rupture. Its etiology has not been fully clarified, although pressure transmission across occluding thrombus, graft transparency, or occult endoleak have been mostly proposed. Although the average sac growth rate has been reported to be 0.5 to 1.0 cm in a 2-year period, sac growth >8 mm was a risk factor for rupture in 1 report.[2] Rupture may affect up to 25% of patients with endotension.[4,5] There are limited data in the literature regarding the operative findings within the aneurysmal sac in endotension. We present a patient with endotension and provide operative imaging (Supplemental Video) to enlighten this obscure area.

Case Report

A 69-year-old man underwent EVAR for a 9.2 cm in diameter abdominal aortic aneurysm (AAA) 6 years ago (Fig. 1). A woven polyester Treovance trimodular endo-graft device with suprarenal fixation (Terumo Aortic, Sunrise, FL, USA) was inserted. Due to an incomplete follow-up, he presented with an asymptomatic aneurysm sac enlargement at 14 cm in diameter AAA. He was an active smoker, and his medical history included hypertension, hypercholesterolemia, mild obstructive pulmonary disease, and appendectomy 50 years ago. He was receiving olmesartan medoxomil 20 mg plus amlodipine 5 mg o.d., simvastatin 40 mg o.d., and acetylsalicylic acid 100 mg o.d. Preoperatively, he performed a stress echocardiography, which was normal, and a spirometry, which revealed a mild obstructive pulmonary disease. Carotid color duplex was normal. No signs of infection were apparent, and his C-reactive protein level was 0.14 mg/dL (normal value <0.5 mg/dL). He did not suffer any serious infection in the past. Despite a proximal significant migration, no apparent endoleak was found on computed tomographic angiography (CTA) establishing the diagnosis of endotension (Fig. 2). Under general endotracheal anesthesia and via a midline laparotomy, he underwent dissection of the aneurysm from the renal arteries down to the iliac bifurcations. The left renal vein was encircled twice with soft silicone rubber vessel loops on both sides, evacuated temporarily, and mobilized (Fig. 3).[6] After infusion of 5,000 IU of heparin and proximal infrarenal and distal iliac (in both internal and external iliac arteries) clamping, the sac was opened, and a

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Fig. 1. Computed tomography angiography performed 6 years ago depicting an infrarenal abdominal aortic aneurysm measuring 9.2 cm (yellow arrowheads).

straw-colored thin fluid was encountered and suctioned (Fig. 4). Afterward, a stuck solid material was noted on the graft (Supplemental Video). No occult endoleak was apparent. The graft was divided just below the uncovered stent centrally and at the level of the right iliac orifice distally. The graft was explanted along with the whole left limb, after a small incision on the left common iliac artery (Fig. 5). After axial partial division of the infrarenal neck, the proximal anastomosis with a new standard walled 22 × 11 mm bifurcated expanded polytetrafluoroethylene (ePTFE) graft (W. L. Gore & Associates, Inc., Newark, DE, USA) was performed end-to-end incorporating the proximal uncovered stent, which was left in place as it was fixed to the aortic wall (Fig. 6). The right iliac anastomosis was performed incorporating distally the residual iliac limb along with the iliac orifice. The left iliac anastomosis was performed at the iliac bifurcation. Cultures from the graft and intrasac contents were negative. We did not send any for histological examination. On the first postoperative day, the patient experienced an episode of tachycardia (heart rate, 130 beats/min) due to atrial flutter treated successfully with beta-blockers (esmolol hydrochloride) intravenously along with low-molecular-weight heparin. Afterward, his postoperative course was uneventful. He was discharged on the seventh postoperative day and advised to take a long-term beta-blocker (bisoprolol 2.5 mg o.d.) and anticoagulant therapy (rivaroxaban 20 o.d.). A first-month postoperative 3-dimensional CTA was normal (Fig. 7).

Discussion

The etiology of endotension remains controversial. All of the alternative pathophysiological mechanisms lead to a common process, which is increased pressure on the aneurysmal wall.[1] These mechanisms include (1) occult very-low-flow endoleak below the sensitivity limits of the diagnostic imaging modality; (2) pressure transmission across the aortic wall thrombus at the sealing sites or at the orifices of the side branches, especially if the length of the thrombus is short and the orifices are wide; (3) increased graft fabric porosity; (4) type II endoleak from hypertrophic sac vasa vasorum; (5) seroma-like fluid gradual accumulation because of thrombus fibrinolysis; (6) hyperfibrinolysis, which could lead to reopening of branches; (7) postural-dependent intermittent endoleak; (8) enzymatic activity; (9) infection; and (10) anticoagulation.[3,4,7,8] There is no difference in endotension between the different prostheses. Some types of stent-grafts presented in case reports or case series are those

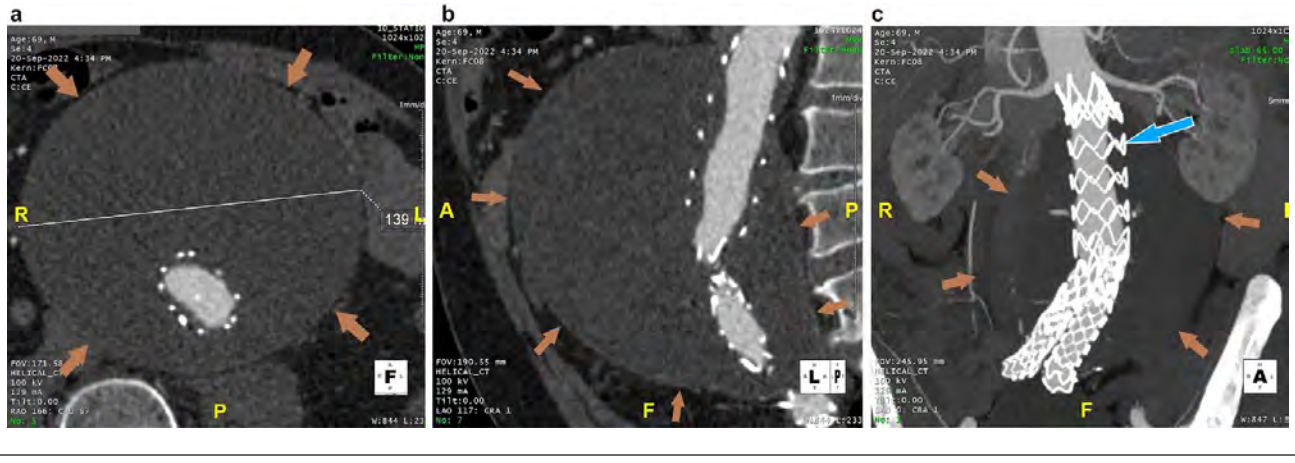

Fig. 2. Computed tomography angiography depicting the expanded abdominal aortic aneurysm 14 cm in diameter (brown arrows) in the (a) axial view, (b) sagittal view, and (c) coronal view with Treovance stent-graft (Terumo Aortic, Sunrise, FL, USA) indicated by the blue arrow.

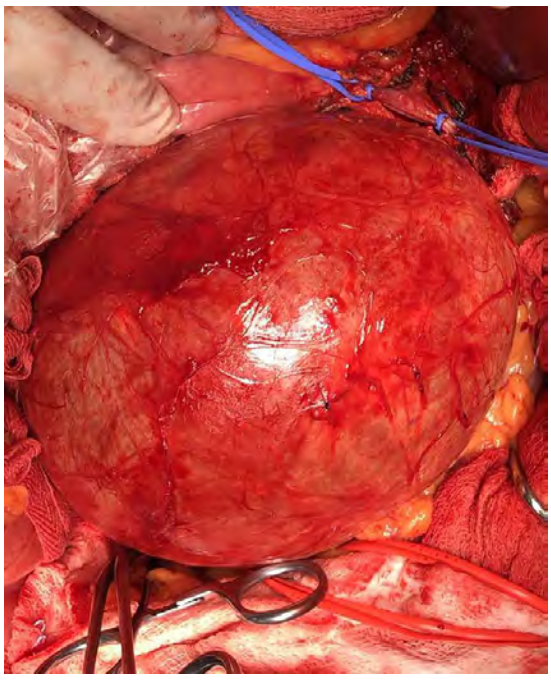

Fig. 3. Intraoperative view of the abdominal aortic aneurysm.

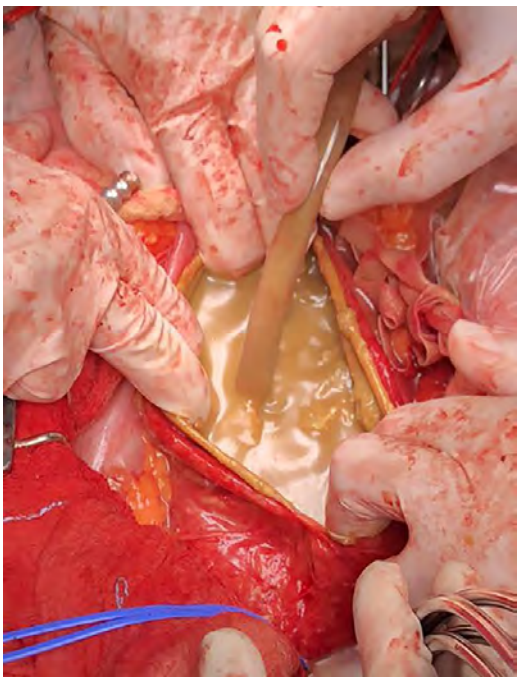

Fig. 4. After the aneurysmal sac was opened, a straw-colored thin fluid was found and suctioned.

Fig. 5. Explanted endograft.

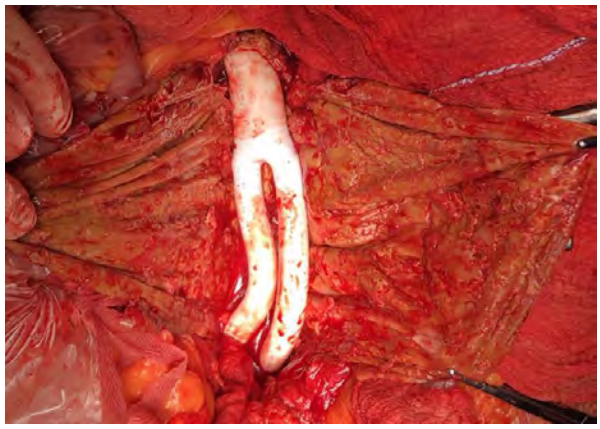

Fig. 6. A bifurcated expanded polytetrafluoroethylene graft was interposed.

that are more commonly used in their centers, and no comments have been made in the literature regarding grafts susceptible to endotension. The only exception is the first-generation EXCLUDER ePTFE grafts (W. L. Gore & Associates, Inc.), which currently have been replaced with a new-generation low-porosity ePTFE graft. This overcomes the transudation and ultrafiltration issues by including a second layer of low-permeability ePTFE. Because endotension is a rare entity, comparative studies between different kinds of grafts are lacking, and it is hard to perform.[2,9] We must emphasize that type II endoleaks may seal spontaneously, and sac expansion will be falsely attributed to endotension.[2] In addition, we highlight that occult type III endoleak is defined as a microleak through graft fabric interstices or minor graft tear.[1,10] Contrast-enhanced magnetic resonance imaging or contrast-enhanced ultrasound may reveal endoleaks not detected by formal CTA, even with markedly delayed images. Consequently, these modalities should follow a negative CTA to unmask possibly a silent endoleak.[4,7] All of these modalities including aortography depend on a clear movement of fluid or contrast within a firm defined time period, and therefore, there is a limit of resolution at which point a minor endoleak may remain unseen.[3] Currently, dual-energy CT, photon-counting detector CT, technetium-99m positron emission tomography, and 4-dimensional phase contrast

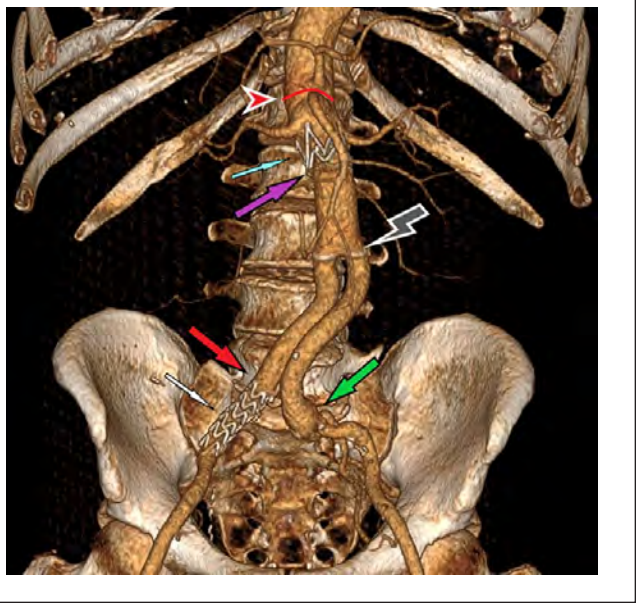

Fig. 7. Postoperative 3-dimensional computed tomography angiography depicting the reconstruction. The red line indicates the initial level of the suprarenal stent's apex before migration, the purple arrow indicates the proximal anastomosis, the red arrow indicates the right iliac anastomosis, and the green arrow indicates the left iliac anastomosis. The suprarenal bare stent was left in place and was located infrarenally due to stent-graft migration (light green arrow). The distal part of the endograft's right limb was left in place (white arrow).

magnetic resonance imaging that visualizes aortic flow dynamics are considered additional helpful imaging modalities.[2,3] Catheter angiography may be unable to identify a type I endoleak when there is no sac outflow, but if an endoleak is diagnosed, catheter angiography is essential for further treatment, which usually consists of embolization in type I and II endoleaks.

There are limited data in the literature regarding intrasac contents in endotension. Rubbery, gelatinous material and/or yellowish seroma-like contents have been reported so far.[4,9,11] They were probably the result of increased fibrinolysis and liquefaction of the intrasac thrombus due to pressurization of the sac. Ultrafiltration of plasma elements due to increased graft fabric semipermeability is another cause, but current low-porosity grafts are far more amenable to transudation as compared with older grafts.[2,4,11] The brownish color may be due to alterations of sac thrombus due to sac pressurization.[4] Dark brown liquids indicate old blood clots, but fresh red clots should raise the suspicion of an occult endoleak.[4] There is a similarity in the incidence and the intrasac contents as a semisolid, rubbery, gelatinous material that was found after open reconstruction with regular PTFE grafts in AAA patients with unexplained symptomatic sac expansion in the long term.[11]

The optimal treatment of endotension remains unclear. No consensus has been reached so far, and recent guidelines suggest proceeding with graft relining or open graft explantation if the AAA diameter increases above 10 mm.[12] They also emphasize the initial exclusion of an occult endoleak with additional imaging. Generally, alternative treatment strategies include (1) endoluminal reinforcement with "relining" using a new low-porosity second endograft alone or by filling the sac with embolic materials (detachable metallic coils and a mixture of n-butyl cyanoacrylate and lipiodol[1,2,4,13]), (2) open conversion with aneurysm sac plication, and (3) open conversion with graft explantation in fit patients.[13] A recent report indicated that the later approach carries a mortality rate of 8.3%.[4] If a type II endoleak is found intraoperatively, it should be ligated along with sac plication. Wrapping of the endograft with a new graft, open sac fenestration, and percutaneous sac aspiration have also been used, but they are not generally suggested.[8] We decided to proceed with the last option because we had to treat the concomitant graft migration and the patient was relatively young without severe comorbidity. Due to an insufficient proximal seal zone, relining required custom fenestrated devices, and treatment would be delayed for 2 to 3 months for graft creation. This was considered unacceptable because of the aneurysm's size and the fear of rupture. Otherwise, graft relining alone treats the graft transudation and fabric integrity issues and not the other possible causes of endotension. Generally, regarding endotension treatment, a sensible stepwise approach should include relining as a first option if sufficient proximal and distal seal zones are apparent. If this fails and the sac continues to expand, then sac embolization might follow. In a new aneurysm diameter increase, the next step is open sac plication in high-risk patients. Recent publications advocate for graft explantation and open reconstruction with a regular graft in low-risk patients.[1,2]

Conclusions

Endotension is a rare complication of EVAR with an ambiguous etiology and a pathophysiology that is not fully understood so far. Detailed diagnostic imaging is essential to exclude occult endoleak. Treatment is tailored to individual patients and includes observation, relining alone or with sac embolization, and open repair. Specific treatment should be guided by aneurysm sac growth rate and patient fitness for open surgery.

Declaration of Conflicting Interests

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Supplemental Material

Supplemental material for this article is available online.

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