

Aortic graft infection with enteric organism after embolization of late type II endoleak

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ABSTRACT

An 82-year-old man with a history of endovascular repair for ruptured abdominal aortic aneurysm 6 years ago presented with a type II endoleak and enlarging sac. He had successful transabdominal direct sac puncture embolization but developed fever 2 days postoperatively. Contrast-enhanced computed tomography showed a rim-enhancing collection, and sac aspiration was positive for enteric organisms, confirming endograft infection. The patient underwent graft explantation and neo-aortic reconstruction using superficial femoral veins. Three months postoperatively, computed tomography showed complete resolution of fluid collection and no signs of graft infection. This report illustrates direct puncture embolization complicated by endograft infection from enteric bacteria. (J Vasc Surg Cases and Innovative Techniques 2019;5:61-4.)

Keywords: Abdominal aortic aneurysm; Endovascular aneurysm repair; Graft infection; Endoleak embolization; Aortic reconstruction

Graft infection after endovascular aneurysm repair (EVAR) for abdominal aortic aneurysm is a serious complication with an incidence of 0.6% and mortality rate of 45.7%.¹ Most studies of graft infection have been performed on patients receiving open rather than endovascular repair.²⁻⁴ Furthermore, there are few reports of endograft infection after endoleak embolization.⁵ Given the lack of guidelines on managing this life-threatening condition, we present a case of EVAR graft infection after direct sac puncture embolization for type II endoleak that was successfully treated with neo-aortic reconstruction using superficial femoral veins. The patient's consent was received for this report.

CASE REPORT

An 82-year-old man with a history of ruptured abdominal aortic aneurysm treated with EVAR 6 years ago presented to a community hospital with 4 days of right flank pain. Initial

EVAR was performed with a Cook bifurcated endograft (Cook Medical, Bloomington, Ind) and intraoperative Palmaz stent (Cordis, Bridgewater, NJ) for type I endoleak. The patient had a history of type II endoleak treated with iliolumbar embolization 5 years ago and chronic kidney disease secondary to acute kidney injury after ruptured EVAR with a baseline creatinine concentration of 150 $\mu\text{mol/L}$.

Contrast-enhanced computed tomography (CT) showed a type II endoleak and 9.7-cm sac (Fig 1, A), which had enlarged by 1.9 cm since his last CT scan. A diagnosis of symptomatic aneurysm sac expansion secondary to type II endoleak was made. The patient was transferred to our academic vascular center for direct sac puncture embolization. Under ultrasound and CT fluoroscopic guidance, the aneurysm sac was punctured transabdominally with a trocar needle. Intraoperative angiography confirmed a type II endoleak originating from the lumbar arteries (Fig 1, B). Embolization using lipiodol and *n*-cyanoacrylate glue was completed with no obvious complications. Prophylactic antibiotics including cefazolin and metronidazole were administered.

He was transferred back to his community hospital but subsequently developed fever. Findings on noncontrast-enhanced CT were suggestive of graft infection. He was started on empirical broad-spectrum antibiotics and transferred back to our center for further investigation. On arrival, the patient was febrile but hemodynamically stable. His blood cultures were positive for *Escherichia coli*, and contrast-enhanced CT showed a rim-enhancing collection, psoas abscess, and locules of gas within the sac (Fig 2). This was consistent with endograft infection secondary to transabdominal embolization.

Under CT fluoroscopic guidance, a drainage catheter was placed in the psoas abscess through a translumbar approach; 5 mL of fluid was aspirated, and the culture was positive for *E. coli* and other enteric bacteria including *Bacteroides thetaio-*
taomicron, *Parabacteroides distasonis*, and *Eggerthella lenta*.

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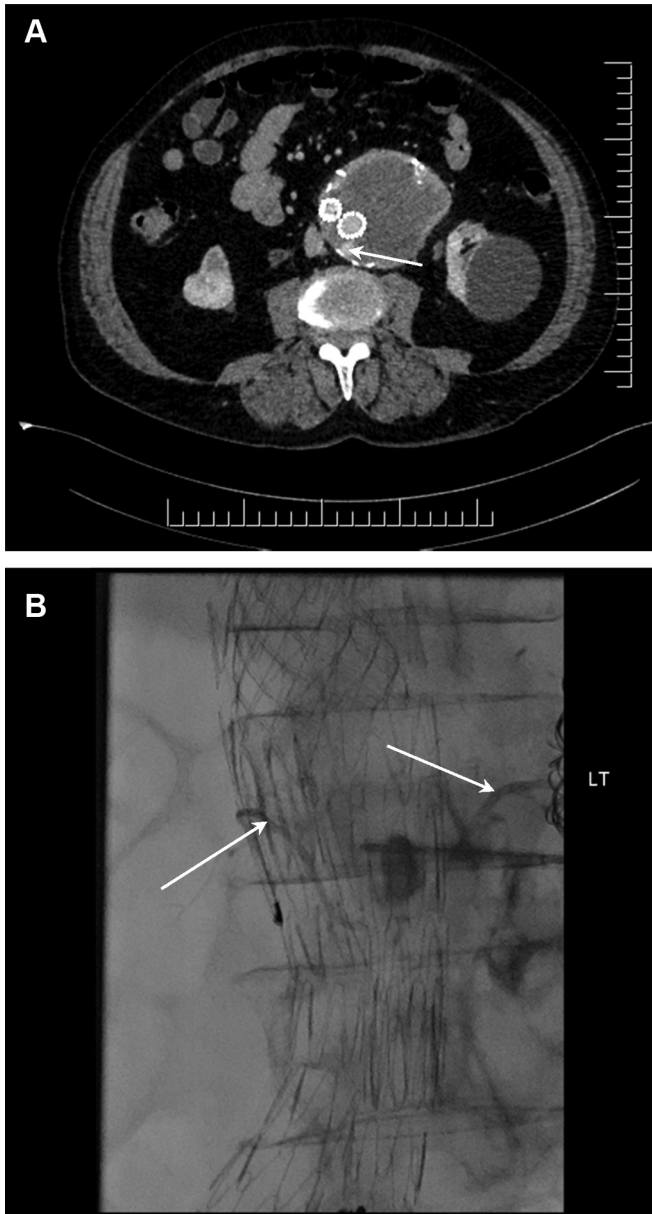


Fig 1. Type II endoleak demonstrated by (A) contrast-enhanced computed tomography (CT) scan and (B) angiography (originating from lumbar arteries).

Vein mapping showed that his femoropopliteal veins were patent, and echocardiography was negative for inducible ischemia. He was consented for endograft explantation and neo-aortic reconstruction using bilateral superficial femoral veins.

From each of the superficial femoral veins, 15 cm was harvested. The proximal ends were spliced together to create a bifurcated vein graft. A midline laparotomy was performed, and the retroperitoneum was opened. Purulent fluid with a feculent smell and dense adhesions were observed. No enterotomy was identified on full bowel examination, consistent with needle puncture of the bowel rather than fistula.

An aortic clamp site was identified just above the left renal artery. After heparinization, the aneurysm sac was opened and purulent material was washed out. We dissected out the

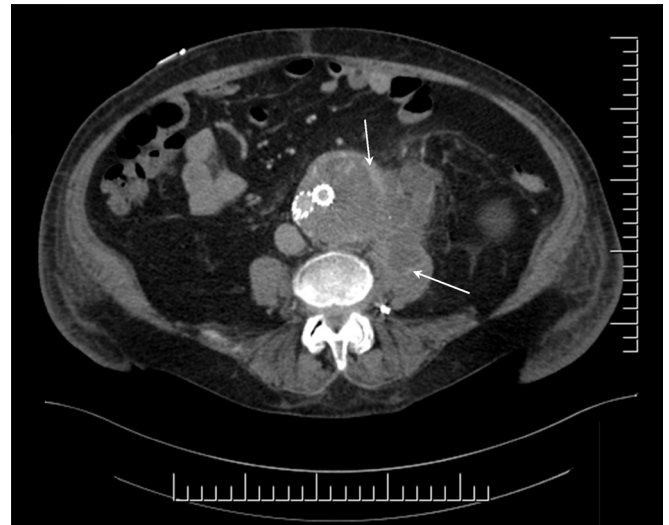


Fig 2. Aortic endograft infection on contrast-enhanced computed tomography (CT) scan demonstrated by rim-enhancing collection, psoas abscess, and locules of gas within the sac.

endograft and retrieved the Palmaz stent without difficulty. An aortobi-iliac repair was performed using the neo-aorta. All infected material was débrided from the aortic sac and sent for culture.

After adequate hemostasis, the aortic sac was sutured over the vein graft, and the retroperitoneum was closed over the aorta. The laparotomy was closed, and the patient was transferred to the cardiovascular intensive care unit. Culture of the operative specimen was positive for *B. thetaiotaomicron* and *P. distasonis*, and the patient continued on intravenous fluconazole and meropenem.

A CT scan on postoperative day 7 showed a patent graft with no signs of ongoing infection. His recovery was complicated by delirium and fever secondary to pneumonia. On postoperative day 19, contrast-enhanced CT scan showed a sac size of 5.4 cm with minor wall enhancement, and no free gas was detected. The patient was discharged with graduated compression stockings when his delirium and pneumonia resolved.

One month later, he was seen for follow-up and resumed daily activities. His incisions were healed and pulses were palpable. His intravenous antibiotics were transitioned to oral amoxicillin-clavulanic acid for long-term infection suppression. Three months postoperatively, CT showed complete resolution of the fluid collection and no signs of ongoing graft infection (Fig 3). The patient is alive 18 months postoperatively with no issues related to his aortic reconstruction.

DISCUSSION

Graft infection is a rare complication after EVAR, and no guidelines exist for managing this serious condition. The only other published case of endograft infection after direct puncture embolization for type II endoleak was reported by Harlock et al.⁶ However, the authors used a translumbar approach and the responsible organism



Fig 3. Noncontrast-enhanced computed tomography (CT) scan demonstrating no signs of endograft infection 3 months postoperatively.

was *Propionibacterium acnes*, a skin rather than an enteric bacterium. Because transabdominal puncture involves accessing the aorta through the abdomen, there is risk of inadvertent bowel injury and seeding of the endograft with enteric organisms.⁷ Our report illustrates the importance of querying graft infection in EVAR patients who develop fever after transabdominal embolization.

Transarterial and direct puncture techniques demonstrate similar efficacy in treating type II endoleak and preventing sac expansion.^{8,9} In this case, we performed transabdominal direct puncture embolization because the patient presented again with type II endoleak after transarterial embolization and his endoleak was anatomically suitable for a transabdominal approach. Our institution is experienced with transabdominal embolizations, having performed 33 for type II endoleak between 2011 and 2016 with a technical success rate of 97%.¹⁰

The benefits of transabdominal embolization over a translumbar technique include better toleration of surgery with supine positioning and easier cannulation of lumbar artery origins.¹⁰ A transcaval approach may have lowered the probability of bowel injury, but it presents unique risks, including inadvertent intracaval embolization and aortocaval fistula.¹¹ Direct repair with minilaparotomy is a more invasive approach for patients

with persistent type II endoleak who have failed to respond to other embolization methods.¹² This technique may be considered if the patient presents again with endoleak in the future.

Endograft infection can be managed medically or surgically. Because of high mortality rates, conservative management with antibiotics is reserved for patients who cannot tolerate surgery.¹³ Surgical management of aortic graft infection generally consists of graft explantation, débridement of infected tissue, and aortic revascularization. Traditionally, extra-anatomic bypass has been the standard of care,¹⁴ but recent evidence demonstrates that in situ reconstruction provides improved mortality, amputation, and long-term patency rates.¹⁵

Prosthetic, autogenous, and cryopreserved grafts are available for in situ reconstruction.¹⁶ Autogenous aortic reconstruction using the femoral-popliteal vein, first described by Clagett et al,¹⁷ provides better long-term patency, reinfection, and mortality rates than other methods.¹⁸

Applying neoaortic reconstruction using superficial femoral veins to our patient allowed complete resolution of endograft infection and recovery of quality of life within 3 months. Smeds et al⁷ published a case series of 206 patients demonstrating similar feasibility of this technique for managing endograft infection.

CONCLUSIONS

Seeding of the aneurysm sac with enteric organisms is a rare complication after transabdominal direct sac puncture embolization of post-EVAR type II endoleak. Neoaortic reconstruction using autogenous superficial femoral veins is a feasible treatment option for this complication.

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