# *Listeria monocytogenes* endograft infection after fenestrated endovascular aneurysm repair—a case report

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## ABSTRACT

An extremely uncommon cause of endograft infections, *Listeria monocytogenes* graft infections are associated with high morbidity and mortality. Best managed with complete graft resection and long-term antibiotic therapy, we present a rare case of *L. monocytogenes* aortic graft infection managed successfully with direct sac drainage and lifelong suppressive antibiotic therapy. (J Vasc Surg Cases Innov Tech 2022;8:1-4.)

**Keywords:** EVAR; Fenestrated endograft; *Listeria monocytogenes*; Mycotic aneurysm

Infrarenal endovascular aneurysm repair (EVAR) and complex fenestrated endovascular aneurysm repair (FEVAR) have a unique set of well-documented procedure-related postoperative complications, namely endoleaks.[1] Less frequently described, however, are endograft infections, occurring in ~1% to 4% of cases. Although the optimal management of graft infections has included removal of the infected material with concurrent extra-anatomic or in situ bypass surgery, often this will not be feasible in medically comorbid patients.[2]

We report an extremely rare case of *Listeria monocytogenes* endograft infection after FEVAR for a large juxta-renal abdominal aortic aneurysm (AAA) managed nonoperatively with prolonged direct aortic sac drainage and long-term antibiotic therapy. The patient provided written informed consent for the report of his case details and imaging studies.

## CASE REPORT

An 80-year-old man had presented with an asymptomatic juxta-renal AAA measuring 84 × 80 mm in the maximal antero-posterior diameter. He had a history of hypercholesterolemia, hypertension, congestive heart failure, coronary artery bypass grafting in 2002, and subsequent coronary artery stenting in 2008. He had undergone FEVAR using a custom-made four-vessel fenestrated Alpha Endograft (Cook Medical, Bloomington, Ind), with stenting of the superior mesenteric artery (SMA) and bilateral renal arteries with Bard LifeStents (BD, Franklin Lakes, NJ). Intraoperatively, good apposition was noted between the aortic wall and the celiac fenestration. Thus, we decided not to proceed with stenting because of an ongoing difficulty cannulating the celiac artery.

At 6 months postoperatively, he had presented with a 1-week history of unremitting watery diarrhea, generalized malaise, and fevers. He also reported a 20-kg weight loss since his FEVAR. His abdomen was soft and not tender to palpation. His bilateral lower limb dorsalis pedis pulses were palpable. His initial inflammatory markers were significantly elevated with a white blood cell count of $20.4 \times 10^9$/L, neutrophil count of $18.6 \times 10^9$/L, and C-reactive protein of 306.6 mg/L. Intravenous (IV) ceftriaxone and metronidazole were started for presumed gastroenteritis, with rapid improvement in his symptoms and inflammatory marker levels (Fig 1). All the results from the sepsis workup were negative. No periaortic stranding, collections, or malignancies were evident on computed tomography (CT) of his chest, abdomen, and pelvis. An incidental filling defect in the SMA stent was seen and thought suspicious for thrombus. Urgent CT angiography confirmed the presence of SMA stent thrombosis of an indeterminate age, combined with impaired flow in the celiac artery and an occluded inferior mesenteric artery. With further history taking, he reported chronic postprandial abdominal pain and anorexia, suggestive of chronic mesenteric ischemia. His abdomen remained nontender. SMA thrombectomy and stent relining were performed by interventional radiology (IR) with improvement of his symptoms and appetite.

However, 1 month later, he had presented to a peripheral hospital with acute exacerbation of heart failure and ongoing weight loss. CT of the abdomen and pelvis was repeated to rule out malignancy, which demonstrated enlargement of the native AAA sac, measuring 98 × 96 mm with surrounding soft tissue hyperdensity (Fig 2). His inflammatory markers remained within acceptable limits (white blood cell count, $8.8 \times 10^9$/L; C-reactive protein, 61.7 mg/L). His serial blood cultures were negative. Positron emission tomography demonstrated diffuse moderate-to-intense $^{18}$F-fluorodeoxyglucose accumulation localized to a rim of soft tissue surrounding the native sac, consistent with sac infection (Fig 3).

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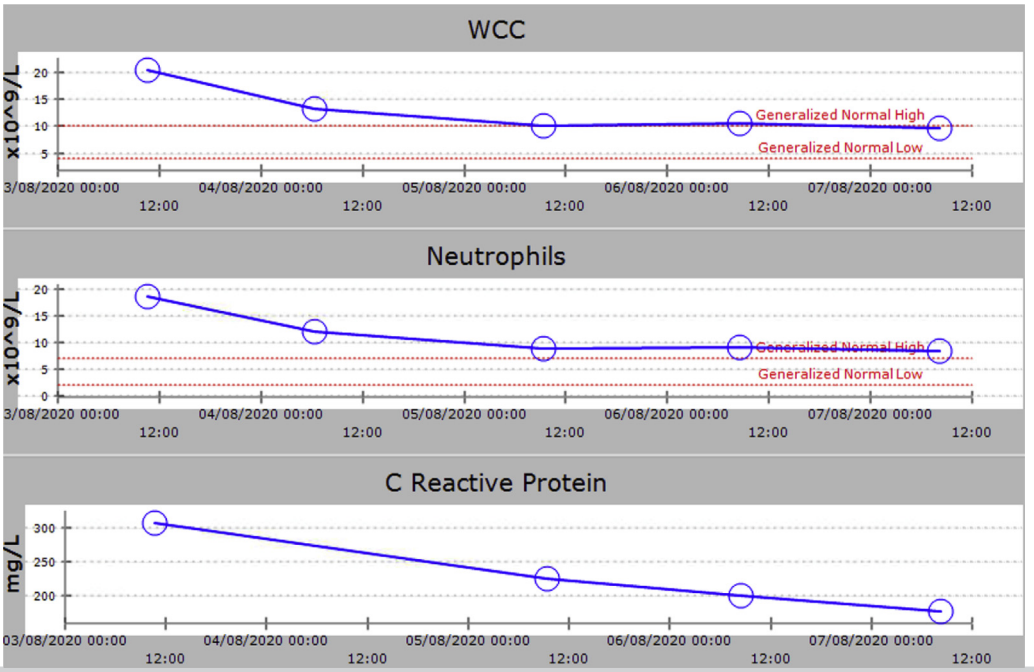


**Fig 1.** Graphic representation of the rapid improvement in inflammatory markers from presentation to after administration of intravenous (IV) antibiotics for presumed gastroenteritis. *WCC,* White blood cell count.

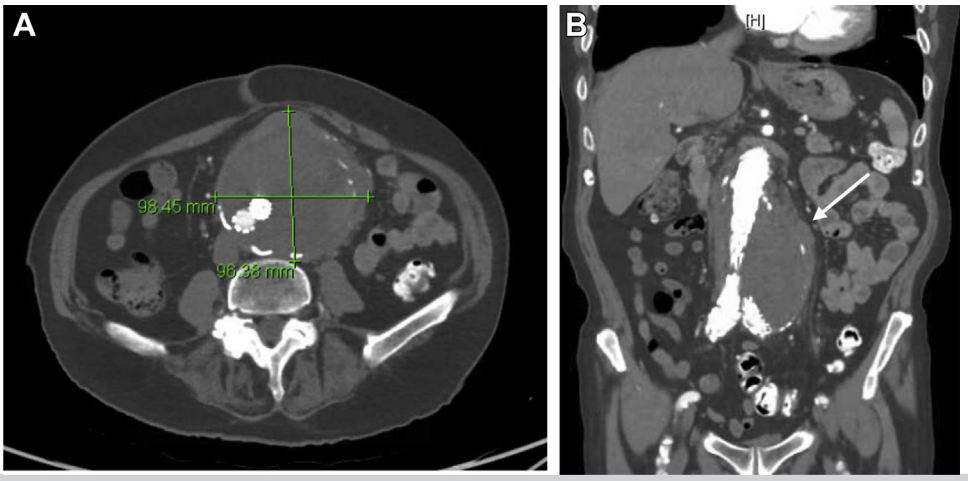


**Fig 2.** Computed tomography (CT) angiogram performed ~7 months after the initial fenestrated endovascular aneurysm repair (FEVAR) demonstrating an increase in the aortic sac size, measuring ≤98 × 96 mm (previously 82 mm; **A**) with a hyperdense soft tissue rim surrounding the previously repaired fusiform infrarenal aneurysm (*arrow*; **B**).

Broad-spectrum antibiotic coverage with IV piperacillin-tazobactam and vancomycin was started. Percutaneous drainage of the aneurysm sac via a retroperitoneal approach using a 12F multipurpose drain (Cook Medical) was performed by IR (Fig 4). Frank purulent material was aspirated and sent for microscopy, culture, and sensitivity examination. The results were positive for *L. monocytogenes*, sensitive to penicillin. He was transitioned to IV benzylpenicillin 2.4 g every 4 hours for 6 weeks, with a plan for lifelong amoxicillin of 1 g three times daily after the 6 weeks of IV therapy. The drain was removed 8 days after >24 hours with no output. He was discharged with a prescription for IV antibiotics, with overall improvement in his heart failure symptoms and appetite. The complexity of the aortic reconstruction required and his comorbidities precluded him from undergoing graft explantation as an alternative treatment option. The guarded prognosis of a graft infection and the limited options for invasive management were discussed with the patient.

However, 7 days later, he had presented again with signs of sepsis (ie, worsening fever, rigor, shortness of breath, and

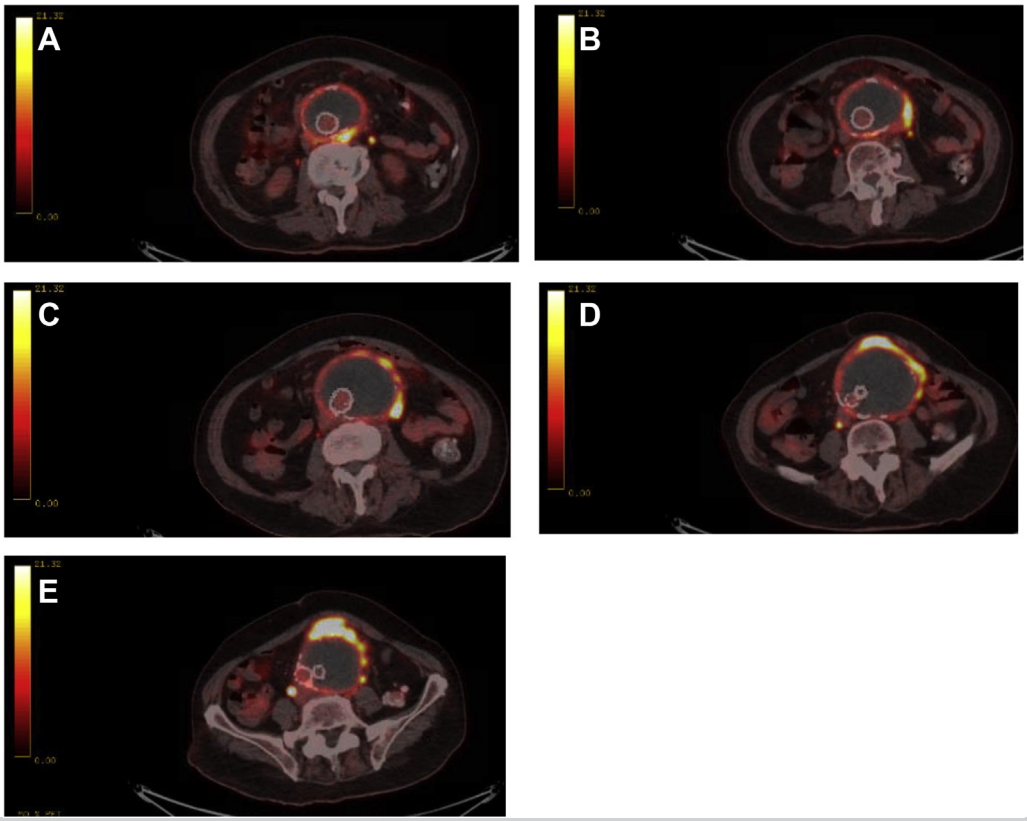


**Fig 3.** Serial axial positron emission tomography slices from cranially to caudally (**A-E**) demonstrating diffuse moderate to intense heterogeneous $^{18}$F-fluorodeoxyglucose uptake localized to a rim of soft tissue around the infrarenal abdominal aortic aneurysm (AAA) with prominence at the posterosuperior, left lateral, and anteroinferior aspects of the aneurysm.

hypotension). CT angiography demonstrated an unchanged appearance of the aortic sac, without evidence of an endoleak or an increase in sac size. Multidisciplinary discussions between the vascular, IR, and infectious diseases teams resulted in the decision to reinsert the drain and to leave it in situ for ≥4 weeks, in accordance with the limited available literature.[3] A 10F multipurpose percutaneous drain (Cook Medical) was inserted, and additional purulent material positive for *L. monocytogenes* was cultured. After insertion of the drain, his symptoms rapidly improved, and he was discharged on day 5 with long-term IV benzylpenicillin and the drain left in situ.

At his 4-week outpatient follow-up visit, he had experienced a considerable recovery with improvement in his appetite, a 5-kg weight gain, and complete resolution of his fever, diarrhea, and abdominal symptoms. Thus, the drain was removed, and he completed a 6-week course of IV antibiotic therapy and was transitioned to lifelong oral amoxicillin. He had continued to progress well at 10 months after initial drain insertion.

## DISCUSSION

Since their inception, EVAR and FEVAR have transformed the management of complex aneurysms, especially for high-risk patients deemed otherwise unsuitable for open surgical repair.[4,5] However, the use of endografts has resulted in its own set of unique complications such as endoleaks and graft infections.[1] Infected aortic endografts have remained relatively uncommon and mostly result due to seeding from systemic bacteremia. Less common causes of endograft infections include septic emboli, invasion of neighboring infectious foci, IV drug use, invasive vascular procedures, and trauma. Typically, organisms of the skin flora and gastrointestinal tract such as staphylococci, *Enterococcus coli*, and *Pseudomonas aeruginosa* are responsible for endograft infections.[2,6,7] Graft infections occurring within the first 4 months after insertion will usually be associated with more virulent organisms.[8] Although extremely rare, *L. monocytogenes* has been implicated in endograft infections with seven reported cases worldwide to date.[7]

An opportunistic gram-positive bacillus usually found in soil, *L. monocytogenes*' surface proteins give it the ability to cross the intestinal, placental, and blood-brain barriers. Infection of endograft material often presents as nonspecific constitutional symptoms, resulting in a delayed diagnosis.[9,10] Although the reported data set of

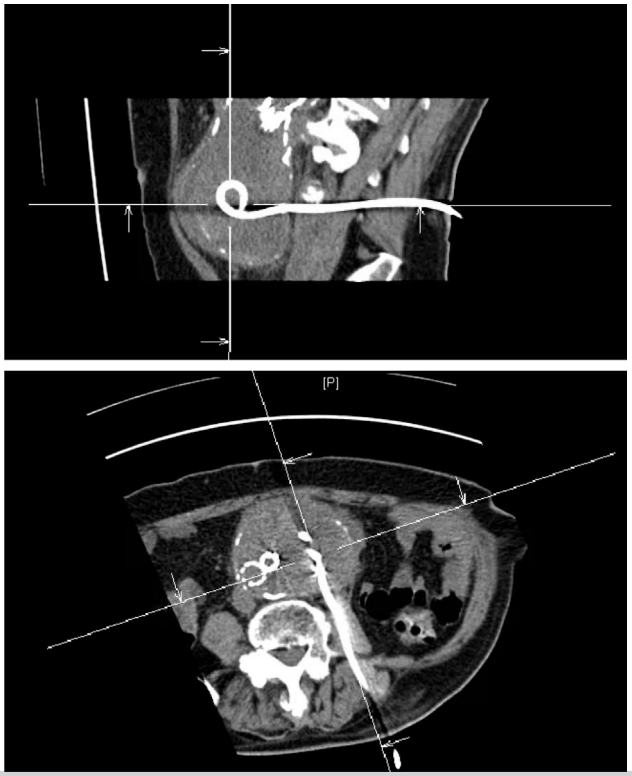


**Fig 4.** Computed tomography (CT)-guided percutaneous drainage of aneurysmal aortic sac using a lateral approach. A 0.035-in. J-wire was introduced into the aortic sac. Using the Seldinger technique, a 12F drain (Cook Medical) was introduced over the wire, with its position confirmed by CT.

*L. monocytogenes*–related endograft infections is small, the management principles remain the same as those for other endograft infections—antibiotic therapy, drainage of infected sac material, and surgical excision.

Explantation of the infected prosthesis remains the mainstay recommendation. However, given that many of these patients had initially undergone EVAR or FEVAR because they were insufficiently fit for open repair, these patients will often be deemed unsuitable for subsequent explantation and aortic reconstruction.[11-13] This is especially true for cases of FEVAR explantation, which will have the additional morbidity associated with visceral vessel reconstruction. Prolonged courses of a combination of IV and oral antibiotic therapy are indicated for all endograft infections, with the antibiotic therapy ideally targeted to the isolated pathogen.[2,9,14] CT-guided percutaneous aortic sac drainage and irrigation have been used with good effect in a number of cases, with drains remaining in situ for ≤28 days.[3,4,14,15]

*L. monocytogenes* endograft infection is a rare, yet serious, complication after EVAR, with significant morbidity and mortality. Given the rarity of *L. monocytogenes* mycotic aneurysms and endograft infections, limited pathogen-specific outcomes are available. Thus, management should be guided by basic principles and guidelines in a multidisciplinary setting.[2,14]

## CONCLUSIONS

We have demonstrated the successful management with prolonged direct sac drainage and lifelong antibiotic suppression of a complicated endograft infection in an elderly, comorbid patient unfit for graft explantation.

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