Infected endovascular aneurysm repair graft complicated by vertebral osteomyelitis

Christopher Lowe, MRCS,[a] Anthony Chan, MRCS,[a] Neil Wilde, FRCR,[b] and **Simon Hardy, FRCS,[a]** *Blackburn, Lancashire, United Kingdom*

Endovascular aneurysm repair (EVAR) is now an established method of treating abdominal aortic aneurysms. Endovascular stent graft infection is a rare complication of EVAR. Diagnosis can be difficult and subsequent management challenging as a significant number of patients are unfit for further surgery and, untreated, graft infection is almost inevitably fatal. We present a case of an infected EVAR graft complicated by vertebral osteomyelitis that was treated conservatively. We discuss the diagnostic and therapeutic difficulties encountered and review the current literature on this evolving subject. (J Vasc Surg 2012;56:826-8.)

Endovascular aneurysm repair (EVAR) is now an established method of treating abdominal aortic aneurysms (AAA). Infection of open AAA repairs is a well-recognized and well-reported complication with established options for treatment. EVAR graft infection, however, is a rare complication with a reported incidence of 0.2% to 0.7%[1] and associated mortality of 17.5%.[2] Diagnosis can be challenging and subsequent management difficult as a significant number of such patients are unfit for further surgery. However, untreated EVAR graft infection is almost inevitably fatal.[1] We present a case of an infected EVAR graft complicated by vertebral osteomyelitis that was treated conservatively.

CASE REPORT

An 82-year-old male underwent an elective EVAR (Endurant stent graft system; Medtronic, Minneapolis, Minn) of a 9.2-cm infrarenal AAA and bilateral common iliac artery aneurysms in May 2011. He was discharged following an uneventful postoperative recovery. He was readmitted a week later with an acutely ischemic left leg, with occlusion of the left limb of the graft demonstrated on computed tomographic angiography (CTA) thought to be due to a mild-moderate stenosis in the external iliac artery. Following an unsuccessful embolectomy, a right to left femorofemoral crossover was performed. Prophylactic antibiotics were given at induction of anesthesia for both the initial EVAR (flucloxacillin and gentamicin) and subsequent crossover (co-amoxiclav).

Six weeks later, he represented with abdominal and lower back pain and pyrexia (initial 38.5°C, peak 39.0°C). The white cell count (WCC) was raised (initial 15.9 10^9/L, peak 23 10^9/L) as was C-reactive protein (initial 239 mg/L, peak 382 mg/L). A CTA did not demonstrate any vertebral or bowel abnormalities and no evidence of graft infection. No causative bacterium was identified from initial (taken prior to antibiotics) or subsequent blood cultures. Sputum and urine cultures were also negative. A hexamethylpropylene amine oxime nuclear white cell scan showed no focal accumulation to suggest infection graft infection. The patient gradually improved with empiric broad-spectrum antibiotics and was discharged.

Sixteen days later (2 months following his original EVAR), he was readmitted with confusion, anemia (hemoglobin of 6.2 g/dL), and raised inflammatory markers (intial WCC 15.9 10^9/L, peak 18.4 10^9/L; initial C-reactive protein 241 mg/L, peak 263 mg/L). An urgent gastroduodenoscopy to D3 did not reveal any evidence of an aortoenteric fistula, and the patient had no history of upper gastrointestinal inflammatory disease. A CTA demonstrated gas in the aneurysm sac with bony destruction of the superior endplate of L5 and the inferior endplate of L4. Abnormal soft tissue was occupying the disk space and extending beyond the anterior longitudinal ligament into the posterior aneurysm sac. The appearances were consistent with graft infection and vertebral osteomyelitis (Figs 1 and 2). A retrospective review of all prior CT images did not show any evidence of vertebral or bowel abnormalities other than osteoarthritis. Specifically, there was no evidence of vertebral erosion. Magnetic resonance imaging was not feasible due to a pacemaker. The patient also had new neurological signs with isolated weakness of left great toe dorsiflexion (Oxford scale 3/5) likely due to nerve root compression. He had become frail and severely debilitated due to chronic sepsis and also had significant cardiac comorbidities. On discussion, we felt the patient would not survive an open procedure to remove the graft or for neurosurgical intervention, and a joint decision was made to manage the patient conservatively with long-term intravenous antibiotics (gentamicin, rifampicin, clindamycin) after consultation with our microbiology department.

A CTA 14 days later showed persistent pockets of gas within the aneurysm sac but no clear evidence of disease progression or resolution. His clinical condition improved, and he was able to mobilize independently, though the distal lower limb weakness did not change. After 6 weeks of intravenous antibiotics, he was discharged on lifelong oral antibiotics (rifampicin, clindamycin). At 6-month follow-up, the patient was on analgesia to control back pain but was systemically well with a normal WCC and improving

From the Department of Vascular Surgery[a] and Department of Radiology,[b] Royal Blackburn Hospital, East Lancashire Healthcare Trust.

Author conflict of interest: none.

Reprint requests: Christopher Lowe, 9 Greenbank Ave, Heaton Mersey, Stockport, Cheshire, SK4 3BU United Kingdom (e-mail: chris.lowe@doctors.org.uk).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214/$36.00

http://dx.doi.org/10.1016/j.jvs.2012.03.268

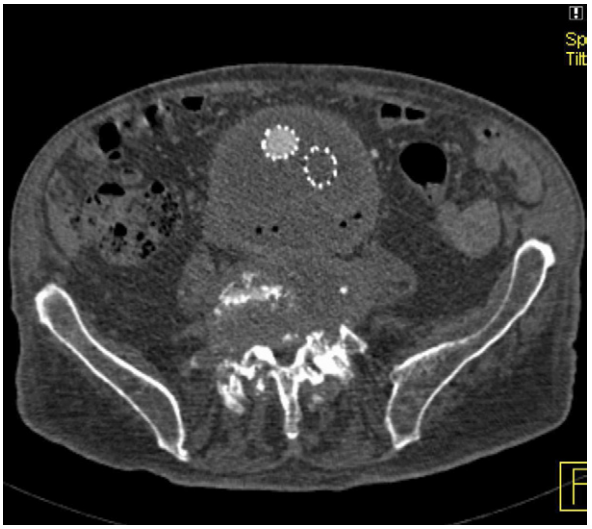

Fig 1. Axial computed tomography (CT) image demonstrating air within the aneurysm sac and vertebral osteomyelitis.

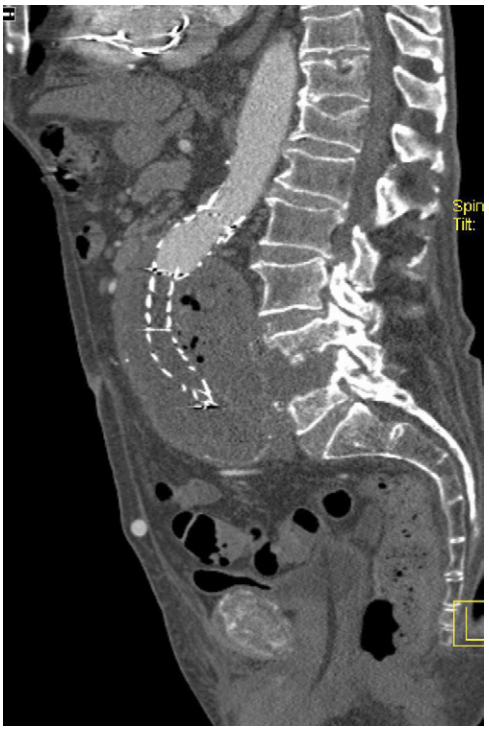

Fig 2. Coronal computed tomography (CT) image further demonstrating osteomyelitis of L4 and L5.

albumin level (24 g/L). A CTA demonstrated essentially static appearances (Fig 3).

DISCUSSION

Infection of EVAR grafts is rare with around 110 cases reported in the literature since 1997.[1] Our patient's early

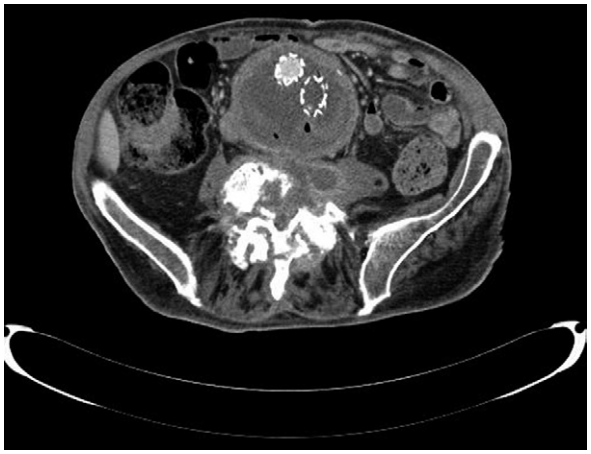

Fig 3. Axial computed tomography (CT) image at 6 months showing essentially static appearances.

presentation (abdominal and lower back pain, with low grade sepsis within 4 months of repair) was not atypical; Hobbs and colleagues suggest a bimodal distribution in presentation either within 4 months of initial surgery or more than 12 months following surgery. They also proposed two distinct but not exclusive scenarios of presentation, either with sepsis or signs of aortoenteric fistula.[1]

Sources of EVAR infection may include hematogenous spread from a distant source (such as the urinary tract), contamination at initial implantation, and from an aortoenteric fistula. Causative organisms are usually derived from skin flora or the gastro-enteric tract with *Staphylococcus aureas* and *Escherichia coli* most prevalent.[2] In this case, we have not been able to fully elucidate if this was a primary graft infection or if there was an underlying discitis/osteomyelitis that extended anteriorly to involve the aneurysm sac. Of note, the patient had a long-term urinary catheter and a history of recurrent *E coli* urinary tract infections – this may have been a possible source of sepsis. Additionally, it is possible that the attempted embolectomy may have introduced infection. Percutaneous drainage may have been a feasible option to gain some more informative bacteriology and has been used successfully for treatment in other cases.[2] This remains as a possible option for further treatment in this case.

It is interesting to note that our patient had repeatedly negative CT findings. Sensitivity of CT for diagnosis of open AAA graft infection is reported to be 56% to 64%,[3] while the sensitivity of white cell scanning is given as 60% to 100%.[4] However, low grade infection in a nonperfused aneurysm sac may not be detected.

There is agreement that infected grafts should be explanted if the patient's condition allows, and operative risk appears to be the main indication for conservative management. In Setacci's report,[2] overall mortality from EVAR infection was reported to be 17.5%. Mortality for conservative treatment was 38.8% compared with 14.6% (graft excision and extra-anatomical bypass) and 7.4% (graft exci-

sion and in situ reconstruction) for surgical treatment. In a case series of six patients with infected aortic endografts, Sharif et al reported 100% mortality in those managed conservatively.[5] In a recent series of 12 patients with infected EVAR/TEVAR grafts, six patients were treated conservatively with antibiotic therapy alone. The rationale for conservative versus surgical treatment was not detailed. Three patients died during treatment or follow-up, two of which had been treated conservatively.[6] Results from the EVAR-2 trial show patients assessed as fit for EVAR should also be fit for open repair.[7] However, some patients may have undergone EVAR outside of these recommendations or as an emergency. In addition, patients may develop comorbidities following EVAR that subsequently make them unfit for open surgery.

CONCLUSIONS

Infection of an EVAR graft remains a relatively rare complication but should be considered in any patient who presents with signs of sepsis or back pain. Graft infection can be complicated by involvement of nearby structures, including the lumbar spine, spinal cord, and nerve roots. Neurological symptoms may be signs of underlying graft infection and should be investigated urgently. Management of infected EVAR grafts remains problematic as patients may not be fit for open surgery, and conservative treatment is associated with a high mortality. We must emphasize that our follow-up is too short to comment definitively on the efficacy of the conservative approach. However, long-term antibiotic therapy may be the only treatment option in such cases.

REFERENCES

1. Hobbs SD, Kumar S, Gilling-Smith G. Epidemiology and diagnosis of endograft infection. J Cardiovasc Surg 2010;51:5-14.
2. Setacci C, De Donato G, Setacci F, Chisci F, Perulli A, Falzerano G, et al. Management of abdominal endograft infection. J Cardiovasc Surg 2010; 51:33-41.
3. Fukuchi K, Ishida Y, Higashi M, Tsunekawa T, Ogino H, Minatoya K, et al. Detection of aortic graft infection by fluorodeoxyglucose positron emission tomography: comparison with computed tomographic findings. J Vasc Surg 2005;42:919-25.
4. Fiorani P, Speziale F, Rizzo L, De Santis F, Massimi GJ, Taurino M, et al. Detection of aortic graft infection with leukocytes labeled with technetium 99m-hexametazime. J Vasc Surg 1993;17:87-95.
5. Sharif MA, Lee B, Lau LL, Ellis PK, Collins AJ, Blair PH, et al. Prosthetic stent graft infection after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2007;46:446-8.
6. Cernohorsky P, Reijnen MMPJ, Tielliu IFJ, van Sterkenburg SMM, van den Dungen JJAM, Zeebregts CJ. The relevance of aortic endograft prosthetic infection. J Vasc Surg 2007;54:327-33.
7. EVAR trial participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2): randomised controlled trial. Lancet 2005;365:2187-92.

Submitted Jan 31, 2012; accepted Mar 29, 2012.