Aortic rupture due to pneumococcal infection in aortoiliac stents

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We report a rare case of pneumococcal aortitis secondary to endovascular bare-metal stent infection. The patient was a 70-year-old man presenting with back pain 1 year after aortoiliac implantation of bare-metal kissing stents. Final diagnosis was microbial aortitis due to Streptococcus pneumoniae involving the stents that resulted in a contained aortic rupture requiring urgent surgical treatment. Emergency extra-anatomic revascularization, excision of the infected tissues, and appropriate antibiotic therapy led to a favorable outcome. A high index of suspicion is required in such a situation because the mortality rate is very high in the absence of appropriate treatment. (J Vasc Surg 2011;53:1711–3.)

Endovascular bare-metal stent infections have rarely been reported. We report a case of microbial aortitis due to Streptococcus pneumoniae that occurred 1 year after endovascular aortoiliac stent implantation. This resulted in a contained aortic rupture that required urgent surgical treatment.

CASE REPORT

A 70-year-old man was admitted to our institution with a 3-week history of continuous lower back pain associated with chills. His medical history included diabetes mellitus requiring insulin, heavy smoking, and pulmonary tuberculosis, but no history of pneumococcal infection. His surgical history included a recurrent hepatic abscess due to Escherichia coli 2 years before admission that required surgical drainage, cholecystectomy, and 3 months of antibiotic treatment. Peripheral vascular disease had been treated 1 year before admission by bilateral angioplasty and aortoiliac bare-metal stenting (10 × 37 Express Stent; Boston Scientific Corp, Natick, Mass) associated with right external iliac artery stenting (8 × 40 mm Lifesent; Bard Peripheral Vascular Inc, Tempe, Ariz).

Neither an infectious process was noticed nor antibiotic treatment initiated in the year between the stenting and the admission. On admission, the patient was afebrile and complained of isolated spontaneous lumbar pain. Hemodynamic variables were stable, and the physical examination was unremarkable. Laboratory tests showed a mildly elevated white blood cell count of 12.10×10^9/L, a decreased hemoglobin value of 109 g/L, and an elevated C-reactive protein level reaching 300 mg/L.

A contrast-enhanced computed tomography (CT) scan revealed a 7.3-cm × 3.7-cm contrast leak below the aortic bifurcation, surrounded by retroperitoneal hematoma. The inferior mesenteric and iliac arteries were patent, but the right internal iliac artery was occluded (Fig 1). The suspected diagnosis was a contained aortic rupture due to aortoiliac stent infection. Emergency management included transfer to the surgical intensive care unit, initiation of wide-spectrum antibiotic therapy, and surgical treatment. The operation was performed 12 hours after the CT scan, and included four different procedures.

First, the patient underwent axillofemoral grafting. Second, exploratory laparotomy was performed to control the infrarenal aorta, inferior mesenteric artery, and iliac bifurcations. Third, the retroperitoneum was incised toward the aortic bifurcation, revealing a retroperitoneal hematoma associated with inflammatory tissue and purulent fluid. Finally, the stents were removed, the aortic bifurcation was excised, the aorta was sewn below the inferior mesenteric artery, and the origin of the right external artery and the distal left common iliac artery were sewn below the distal edge of each stent. Because the omentum was hypotrophic, an omental patch was not performed.

The perioperative aortic wall sputum grew Streptococcus pneumoniae that was sensitive to penicillin. Histologic examination confirmed the diagnosis of infectious aortitis. The patient was treated with intravenous penicillin A and gentamicin for 3 days, followed by intravenous penicillin A for 2 weeks and oral oxacillin for 4 additional weeks. Results of the etiologic investigation, including oral examination, transesophageal echocardiography, chest radiograph, and whole-body CT scan, were negative.

The patient’s postoperative course was marked by subcutaneous wound sepsis treated by surgical debridement and negative pressure therapy. A systematic CT scan confirmed graft patency and that the retroperitoneal infection was controlled (Fig 2). The patient was discharged 1 month after surgery and remained asymptomatic after a 6-month follow-up.

DISCUSSION

In recent decades, percutaneous transluminal angioplasty and the placement of endovascular stents in the peripheral vasculature have become a prevalent treatment option for atherosclerotic disease. There have been scattered reports of infection of bare-metal stents. In 2007,
Hogg et al\textsuperscript{1} reported 2 cases and reviewed 33 others. Since then, <10 additional cases have been reported,\textsuperscript{2} making infection a rare complication of bare-metal stent placement. Infection that developed <14 days and \textit{Staphylococcus aureus} involvement were described in 70\% of cases, suggesting procedural contamination.\textsuperscript{1} In our patient, however, delayed infection and \textit{S pneumoniae} involvement excluded procedural contamination and suggested complete microbial aortitis.

Microbial aortitis is defined as infection of a normal or arteriosclerotic aorta. It may occur spontaneously or complicate the postoperative course of an aortic graft.\textsuperscript{3,8} After open aortofemoral grafting, postoperative graft infection occurs in 1\% to 5\% of patients.\textsuperscript{9,10} However, the rate is much lower, typically <0.5\%, after endovascular aortoiliac stent graft implantation.\textsuperscript{7,10-13} Furthermore, \textit{S pneumoniae} is an uncommon cause of aortitis\textsuperscript{6,7} with <50 cases reported to date.\textsuperscript{6,7} To our knowledge, this is the first case reported after aortoiliac stenting.

There are three possible mechanisms of vascular stent infection, including procedural contamination from bacteria present on the skin or in the wall of an abdominal aortic aneurysm, anatomic bacterial translation from an adjacent infected or colonized area, and hematogenous or lymphatic bacterial dissemination from a remote bacterial source.\textsuperscript{3,5,6} The latter mechanism has been described in stent infection\textsuperscript{1} and also in primary pneumococcal aortitis,\textsuperscript{8} and was probably the source in our patient. The primary infection could have been occult that had resolved spontaneously, making the remote bacterial source difficult to identify once the aortitis had developed.

Fig 1. A preoperative computed tomography scan showed the stents, the ruptured aortic bifurcation, the contrast leak, and the retroperitoneal hematoma.

Fig 2. A postoperative computed tomography scan showed the absence of an intraperitoneal collection, a patent inferior mesenteric artery, the interrupted abdominal aorta, and the axillofemoral graft.
Pneumococcal aortitis has rarely been reported, making an analysis of risk factors difficult. In previous reports, older age, male sex, atherosclerotic disease, a history of smoking, arterial hypertension, dyslipidemia, and diabetes have frequently been associated with pneumococcal aortitis. The role of immunosuppression has also been mentioned. Classic manifestations of aortitis include fever and back or abdominal pain. Only one-half of the patients with aortitis have a palpable mass, one-third have coexisting lumbar or thoracic osteomyelitis, and one-half present with positive blood cultures. Interestingly in our patient, aortic rupture occurred without previous aortic dilation or aneurysm. This unusual feature challenges the physiopathologic role of the underlying bare-metal stent in the acceleration of the aortic rupture or the deceleration of the aortic dilation in response to aortic wall bacterial infection. However, a previous pseudoaneurysm could not be ruled out in the absence of a CT scan between the stenting and the rupture.

Optimal management of aortitis from stent infections includes surgical resection of the infected graft and aortic wall, distal revascularization, and prolonged antibiotic administration. In situ autogenous tissue and extra-anatomic graft replacement have both been successfully used. We decided to perform an extra-anatomic prosthetic graft replacement before aortic ligation to prioritize an aseptic over a septic procedure and to avoid lower limb ischemia. There are no current guidelines for the duration of antimicrobial treatment after removal of an infected prosthesis. The local expert consensus of a microbiologist and vascular surgeons suggests that at least 6 weeks of treatment is required.

The mortality rate associated with nonsurgical management of microbial aortitis and aortoiliac stent infection is nearly 90%. Failure to diagnose before rupture, nonsurgical management, shock during the surgical procedure, and persistent positive blood cultures are associated with a greater likelihood of death. However, aggressive management that combines surgical and medical approaches results in long-term survival rates of 70%. The mortality rate associated with nonsurgical management, shock during the surgical procedure, and persistent positive blood cultures are associated with a greater likelihood of death. However, aggressive management that combines surgical and medical approaches results in long-term survival rates of 70%.

CONCLUSIONS

Infection is a rare complication of endovascular stents. However, recent expansion of endovascular techniques has resulted in a dramatic increase in the number of aortoiliac stents implanted every year, and postoperative stent infections may progress in the same way. Emergency excision of the infected tissue, extra-anatomic revascularization, and appropriate antibiotic therapy result in a favorable outcome.

REFERENCES