Mycotic Abdominal Aneurysm Caused by *Campylobacter Fetus*: A Case Report for Surgical Management

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We report a rare case of mycotic abdominal aortic aneurysm associated with *Campylobacter fetus*. A 72-year-old male admitted to the hospital because of pain in the right lower quadrant with pyrexia. The enhanced abdominal computed tomography (CT) examination showed abdominal aortic aneurysm (AAA) measuring 50 mm in maximum diameter and a high-density area of soft tissue density from the right lateral wall to the anterior wall of the aorta. However, since the patient showed no significant signs of defervescence after antibiotics administration, we performed emergency surgery on the patient based on the diagnosis of impending rupture of mycotic AAA. The aneurysm was resected in situ reconstruction using a bifurcated albumin-coated knitted Dacron graft was performed. The cultures of blood and aneurysmal wall grew *Campylobacter fetus*, allowing early diagnosis and appropriate surgical management in this case, and the patient is making satisfactory progress. This is the fifth report of mycotic AAA characterizing culture positive for *Campylobacter fetus* in blood and tissue culture of the aortic aneurysm wall.

**Key words:** mycotic abdominal aneurysm, *Campylobacter fetus*, vascular surgery

INTRODUCTION

*Campylobacter fetus infection* associated with endocarditis and meningitis and a decline in the patient’s general condition is becoming more prevalent in patients. With mycotic abdominal aortic aneurysm (AAA) involvement, *Campylobacter fetus* is difficult to diagnosis early, and there is a high risk of aneurysm rupture due to rapid dilation. This is one of several case reports on a patient with mycotic AAA positive for *Campylobacter fetus* in the blood and tissue cultures of aneurysm wall samples.

CASE REPORT

A 72-year-old man with hypertension and no history of overseas voyage or animal contact was admitted to the hospital for surgical treatment. The patient developed symptoms of right lower quadrant abdominal pain, vomiting, and fever of 38°C after the admission. Pulsating abdominal mass was not palpable, while tenderness in the right lower quadrant without muscular defense was present. There was hyperinflammatory reaction indicated by white blood cell count of 10,300/µl (Neutrophil 76.5%) and C-reactive protein (CRP), 1.6 mg/dL. Liver function, renal function, and electrolytes were all normal. We performed an enhanced abdominal computed tomography (CT) examination of the patient for suspected acute appendicitis; however, there were no swollen appendix, just findings of AAA with 50 mm in maximum diameter and a deposition pattern of high-density area of soft tissue from the right lateral wall to the anterior wall of the aorta instead (Fig. 1). Blood cultures were positive for *Campylobacter fetus*.

Clinical findings and CT images revealed an impending rupture of mycotic AAA, and the patient underwent...
surgery in which we found infrarenal AAA with a 50-mm maximum diameter, a hyperinflammatory state, and hypertrophy of the adventitia of the anterior and right lateral wall. The entire retroperitoneum was edematous. Intraoperative findings were compatible with the preoperative CT examination. The Intramural thrombus was removed carefully, and the aneurysm wall was resected. Following an adequate washing procedure with a combination of povidone iodine solution and physiological saline, we replaced by a Y-shaped woven Dacron graft (16 × 8mm, Hemashield) without Omental wrapping, since there was no evidence of abscess formation.

Postoperative germ culture of the resected aneurysm wall was positive for \textit{Campylobacter fetus}. The pathological findings showed extensive calcification and hypertrophy of tunica intima due to atheroma formation with cholesterin slits. The elastic lamina in the media was damaged and eventually the fibrous lesions extended to the tunica adventitia. Although the adventitia had hemorrhaged, with infiltration of neutrophils and lymphocytes, there was no evidence of abscess. Neither bacteria or mold was detected. All these findings suggest a mycotic AAA caused by \textit{Campylobacter fetus} infection of the atherosclerotic aorta.

The patient was treated with Meropenem 1.5 mg/day for 14 days after the surgery (co-administrated with immunoglobulin in the first 3 days), replaced by Ciprofloxacin hydrochloride (CPFX) 600 mg/day for the next 17 days. The patient began Levofloxacin (LVFX) treatment on day 30 after the surgery and was discharged on day 38. At one year follow-up, the patient had a negative CT scan and was symptom-free.

**DISCUSSION**

The incidence of mycotic AAA is relatively rare, between 1% and 3% of patients with AAA. Since the disease is characterized by a rapid decline in the patient’s condition, the risk of aneurysmal rupture is estimated to be between 50% and 80%.

According to the clinical criteria introduced by Müller et al., mycotic aneurysm can be diagnosed when there is 1) a positive aortic wall germ culture (including surrounding tissue or contents of the intra aneurysm, for example, the intramural thrombus) and clinical signs or 2) a negative germ culture plus rupture of AAA, unusual clinical signs during surgery (infection) or ongoing antibiotic treatment. Our diagnosis of mycotic AAA was based on criteria 1) because we were not able to define the route of infection in the patient (In spite of the careful anamnesis). However, a preoperative blood culture tested positive for \textit{Campylobacter fetus}. Therefore, we concluded that the progress of infection in the aortic wall that began with bacteremia was the cause of the mycotic AAA. The patient did have two out of three of the symptoms of mycotic AAA: pyrexia and abdominal pain, though he did not have the third, an abdominal mass. In the image diagnostics, the CT scan is highly effective in
evaluation of the time-dependent changes in aneurysmal expansion. Macedo has reported that relatively high number of cases which shows saccular aneurysm with lobular lumen is indicative of mycotic aneurysm. In addition, the increase of peri-aortic fat density and presence of edema the aorta are significant findings that should be carefully evaluated. The former is also considered being a key factor in the estimation of changes in the retroperitoneum caused by the inflammatory reaction. Our case presented with these findings, which eventually led to the early diagnosis of impending rupture of mycotic AAA, and immediate surgical treatment to prevent the rupture of the aneurysm.

The pathogenic bacteria complicating mycotic AAA are usually salmonella, staphylococcus or streptococcus; however, *Campylobacter fetus* has also been reported to complicate mycotic AAA.

*Campylobacter fetus*, a helical, microaerophilic, Gram-negative rod, is reported to cause miscarriage in livestock, especially in cattle and sheep. In humans, it is associated with a decline in the physical condition, eventually leading to endocarditis and meningitis. We have found 15 cases of *Campylobacter fetus* infection inducing mycotic AAA (13, male; 2, female; average age, 64.4 years; range, 45–84 years). Subjective symptoms were 10 cases of pyrexia (66.6%), 6 cases of abdominal pain (40%), 6 cases of diarrhea (40%), 3 cases of nausea (20%), 3 cases of low back pain (20%), and 1 case of weight loss and anorexia (6.7%). In treatments, 8 cases for anatomic reconstruction and 4 cases for extra-anatomical bypass had been performed. As the result, 4 cases died in aneurysm rupture and 3 cases was not applied surgical treatment among them. There were differences in the period of antibiotics administration between each case ranged from 7 days to 7 months.

Normally extra-anatomical bypass or anatomic reconstruction, such as prosthetic graft replacement, is applied to mycotic infrarenal AAA. While extra-anatomical bypass was commonly performed in the past, anatomic reconstruction, resection of aneurysm wall, and covering of the prosthetic graft with omental wrapping have been introduced with favorable outcomes. In our case, we selected in-situ prosthetic graft replacement because pus discharge was not observed during surgery. We considered omental wrapping; however, the omentum was severe atrophied from the inflammation, and we could not perform the procedure. We also removed the intramural thrombus carefully and washed the area with warm physiological saline to prevent infection.

Even though there is no standard-of-care for the term of prophylatic use of oral antibiotics, treatment is often continued for several months after any evidence of infection. However, we usually stop antibiotic treatment when the biochemical test for blood inflammatory response markers is negative. The patient tested negative for CRP after 10 weeks of antibiotic therapy; thus, the treatment was stopped.

For mycotic AAA, it is usually advisable to treat the
bacterial infection with antibiotics first, as in this case, and consider surgery when the infection is under control. However, surgery is indicated in patients with a rapidly expanding aneurysm or in non-responders to antibiotics. In this case, since the follow-up CT scan revealed a dense, fatty area with exacerbation of the adjacent soft-tissue mass, we operated to prevent an aneurysm rupture.

One year after surgery, the patient has been returning for regular check-ups with no evidence of the disease, though we will continue long-term follow-up to monitor any recurrence of local or systemic infection.

References