Mycotic Abdominal Aortic Aneurysm Caused by *Campylobacter fetus*: A Case Report and Literature Review

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Campylobacter spp. usually cause gastrointestinal infections, but among them, *Campylobacter fetus* is a well-known organism causing mycotic abdominal aortic aneurysm (MAAA), which requires proper surgical intervention and antibiotic therapy. We report a 65-year-old man who was successfully treated by an in situ operation using a rifampicin (RFP)−bonded J-Graft for *C. fetus*−induced MAAA. We performed a review of the English literature on MAAA caused by *C. fetus* and summarized the results of the cases (28 cases). All but 2 of the patients (92.9%) were men. Blood culture and arterial wall culture were positive in 63% and 73.1% of the cases, respectively. Aneurysm rupture was seen in half of the patients, and approximately half of those patients died. Among the 18 patients who underwent in situ graft replacement, only 1 patient (5.6%) died after surgery. Antibiotic therapy was performed for more than 1 month in most cases, and overall mortality rate was 25.9% (7 of 27 cases, 3 deaths before the operation and 4 deaths after surgery). Although extra-anatomic bypass has been conventionally performed after complete resection of an MAAA, the utility of in situ surgery has generally been recognized. Our review suggests that the in situ operation can be a choice also in cases of *C. fetus*−associated MAAA. Furthermore, our case suggested the clinical utility of a newly manufactured prosthetic graft, J-Graft, for such surgical treatment.

Mycotic abdominal aortic aneurysm (MAAA), which is formed by destruction of the arterial wall as a result of infection, accounts for approximately 1% of all aortic abdominal aneurysms (AAAs).[1] Common sites of MAAA are the abdominal aorta distal to the renal artery and the thoracic aorta.[2] The risk factors include arterial injury, preceding infection, immunocompromised state, atherosclerosis, preexisting aneurysm, and aging. Some patients complain of lumbar or back pain, whereas others may present fever of unknown origin or sudden rupture of the aneurysm. The overall mortality rate in patients with MAAA has been reported to be as high as 30%.[1,3] The risk of aneurysm rupture has been reported to be as high as 50−80%,[1,4,5] and the mortality rate would be more than 70% if it is ruptured.[6]

The common causative organisms of MAAA include *Staphylococcus* spp., *Salmonella* spp., and *Streptococcus* spp.[7] Occasionally, *Campylobacter* spp.

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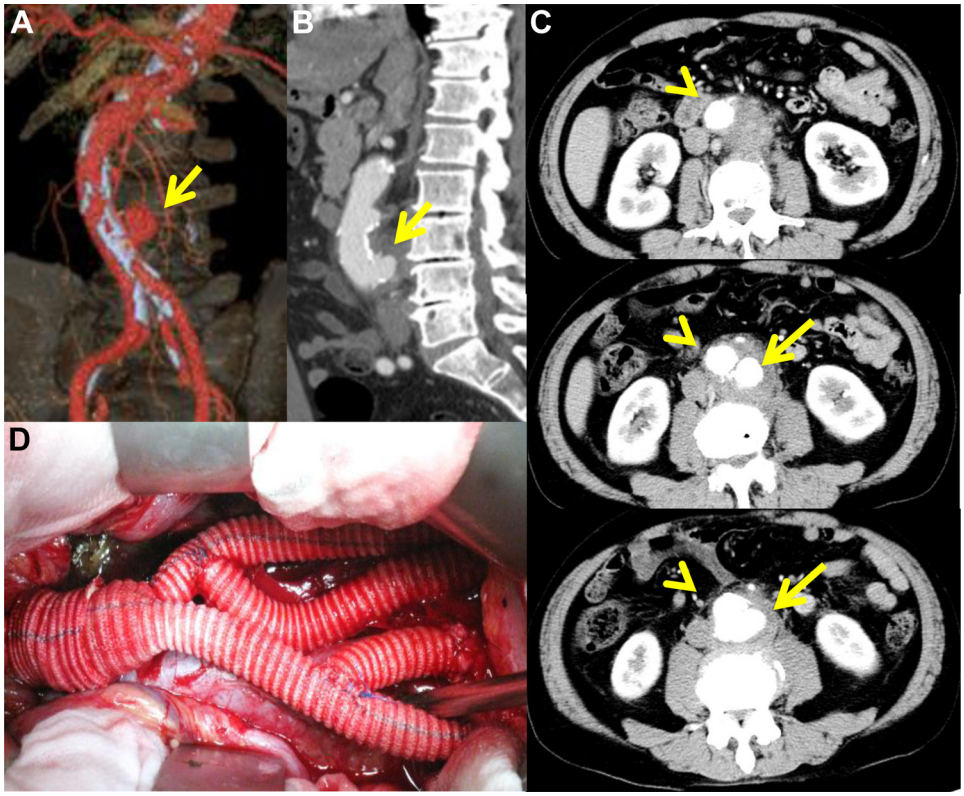

Fig. 1. Abdominal contrast-enhanced computed tomography (CECT) and gross appearance of J-Graft. (**A**) A coronal image of the three-dimensional vascular reconstruction, (**B**) a sagittal image of CECT, (**C**) Axial images of CECT, and (**D**) rifampicin (RFP)—bonded J-Graft after anastomosis. *Arrow*: infected aneurysm, *Arrowhead*: true lumen. A saccular-type abdominal aneurysm (40 × 39 mm) surrounded by a dirty fat sign was found between the inferior mesenteric artery and bifurcation of the common iliac artery (**A**—**C**). The patient underwent aortobilateral iliac reconstruction with bifurcated RFP-bonded Dacron graft (J-Graft; **D**).

are detected as pathogenic organisms of MAAA. Among them, *Campylobacter fetus* is reported to be the most common pathogen.[8] *C. fetus*—associated MAAA has rapid progression and a high mortality rate (approximately 30%),[9,10] and no survival can be expected without surgical intervention.[11,12] Because the number of reported cases is still insufficient, the appropriate strategy for *C. fetus*—associated MAAAs is inconclusive. We report a case of MAAA caused by *C. fetus*, which was successfully treated with replacement of a newly manufactured Japanese woven Dacron graft, J-Graft (Japan Lifeline Co. Ltd, Tokyo, Japan), which was preliminarily bonded with rifampicin (RFP). Furthermore, we discuss the diagnosis and treatment of *C. fetus*—associated MAAAs based on a literature review.

CASE REPORT

A 65-year-old man without any past medical history presented with a 1-week history of lumbar pain. He had an episode of diarrhea 2 weeks previously, but the etiology was unknown. There was no history of drinking raw milk or consuming uncooked meat. A urinary examination showed hematuria, and he returned home with the diagnosis of urolithiasis. The next day, he consulted an urologist and was again diagnosed as having urolithiasis although a urinary tract stone was not apparent in plain computed tomography (CT). Later that day, a physician noticed an inflammatory change around his abdominal aorta in the CT, and the patient was called immediately to visit the hospital.

On arrival, his body temperature was 38.1°C. He complained of rigor and shivering, but his vital signs were stable. Laboratory examination revealed white blood cells of 10,500/mm^3 and C-reactive protein of 13.8 mg/dL. Emergently performed contrast-enhanced CT (CECT) showed a saccular and poorly marginated AAA (40 × 39 mm; Fig. 1A—C). The aneurysm only involved the infrarenal aorta, and celiac, renal, and mesenteric arteries were confirmed to be patent by CT angiography. Ascites or free abdominal air was not obvious. After drawing 3 sets of blood culture, administration of ceftriaxone (2 g every 24 hr) and ciprofloxacin (CPFX, 300 mg every 12 hr)