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Case Report

Infected Abdominal Aortic Aneurysm Caused by *Campylobacter Jejuni*

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A 69-year-old man presented with abdominal pain. Enhanced computed tomography (ECT) showed abdominal aortic aneurysm (AAA) with vessel wall thickening. Follow-up ECT on day 3 of admission showed expansion of the AAA. Endovascular abdominal aortic repair (EVAR) was urgently performed. Since preoperative blood cultures revealed *Campylobacter jejuni* infection, the antibiotics imipenem/cilastatin were administered for five weeks, followed orally by Clarithromycin. The patient was discharged on postoperative day 45. There was no recurrence of the aneurysm at 9 months after EVAR. To the best of our knowledge, this is the first report of EVAR for infected AAA caused by *Campylobacter jejuni*.

Keywords: abdominal aortic aneurysm, stent graft, infected aneurysm

Introduction

Usually, infected abdominal aortic aneurysms (AAA) tend to enlarge rapidly and subsequently rupture. They are considered a critical disease because of the associated high mortality rate (23.5%–37%).[1)] We describe a rare case of infected AAA caused by *Campylobacter jejuni* which was treated with endovascular abdominal aortic repair (EVAR).

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Case Report

A 69-year-old man developed abdominal pain without any trigger. Since his symptoms did not improve, he visited an emergency room the following day. Enhanced computed tomography (ECT) revealed an AAA with a maximum diameter of 57 mm and with vessel wall thickening. Since it was a possibility of impending rupture, he was immediately hospitalized. The following day he was referred to our hospital for further evaluation (first hospital day). Physical examination revealed the following: body weight, 47.9 kg; height: 159.5 cm; blood pressure: 139/72 mmHg; body temperature: 36.7°C; heart rate: 79 beats/min, and no cardiac murmur. Blood tests indicated a white blood cell (WBC) count of 8,800/μL and C-reactive protein (CRP) level of 13.2 mg/dL. Since his abdominal pain improved after admission, we decided to manage him conservatively with rest and antihypertensive therapy. However, inflammatory changes and Gram-positive coccus infection were suspected from the results of one of the two blood culture samples collected at the previous hospital. An infected aneurysm was suspected and antibiotics (vancomycin, 1,000 mg/day) were administered. On day 3 of admission, follow-up ECT showed expansion of the maximum diameter of the AAA to 60 mm (**Fig. 1a**). Impending rupture was suspected and immediate surgical treatment was considered. The patient had a past history of panperitonitis due to perforation of the colon. In addition, the aneurysm was almost juxta renal type (**Fig. 1b**). So, because open surgery was considered high risk, we opted for EVAR, which was performed on day 4 of admission. An Endurant II stent-graft system (Medtronic, Santa Rosa, CA, USA) device was selected. Under general anesthesia, the main body (ETBF2816C145EJ) of the stent-graft was deployed from just below the left renal artery to the left common iliac artery via the left femoral artery. Then, the contralateral leg (ETLW1616C82EJ) of the stent-graft was placed into the right common iliac artery. Angiography performed after deployment of the devices revealed no type I endoleaks.

A subsequent blood culture report from the previous hospital and our hospital strongly suggested a Gram-

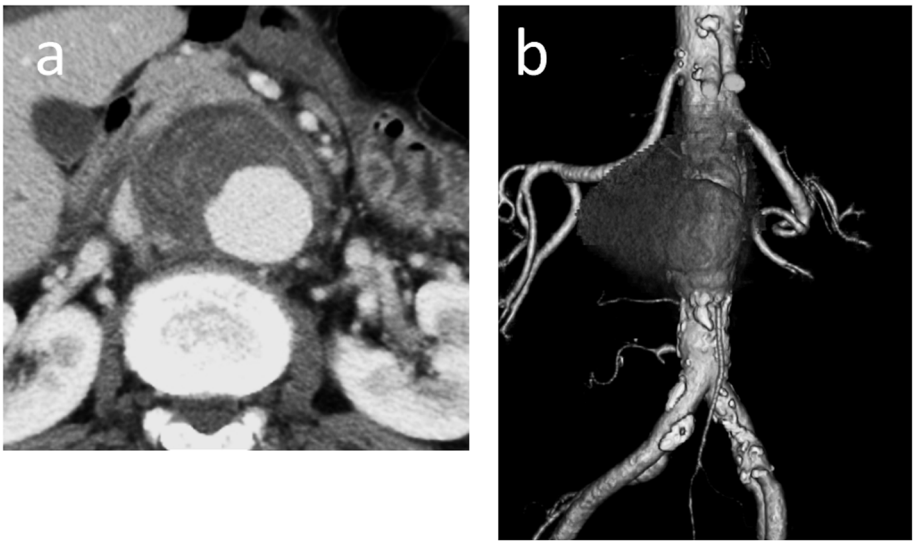

Fig. 1 Enhanced computed tomography (ECT). (**a**) ECT image on day 3 of admission. The diameter of the aneurysm had enlarged to 60 mm, with aneurysmal wall thickening. (**b**) 3D-CT image of abdominal aortic aneurysm. Aneurysmal change occurred at the same level of left renal artery.

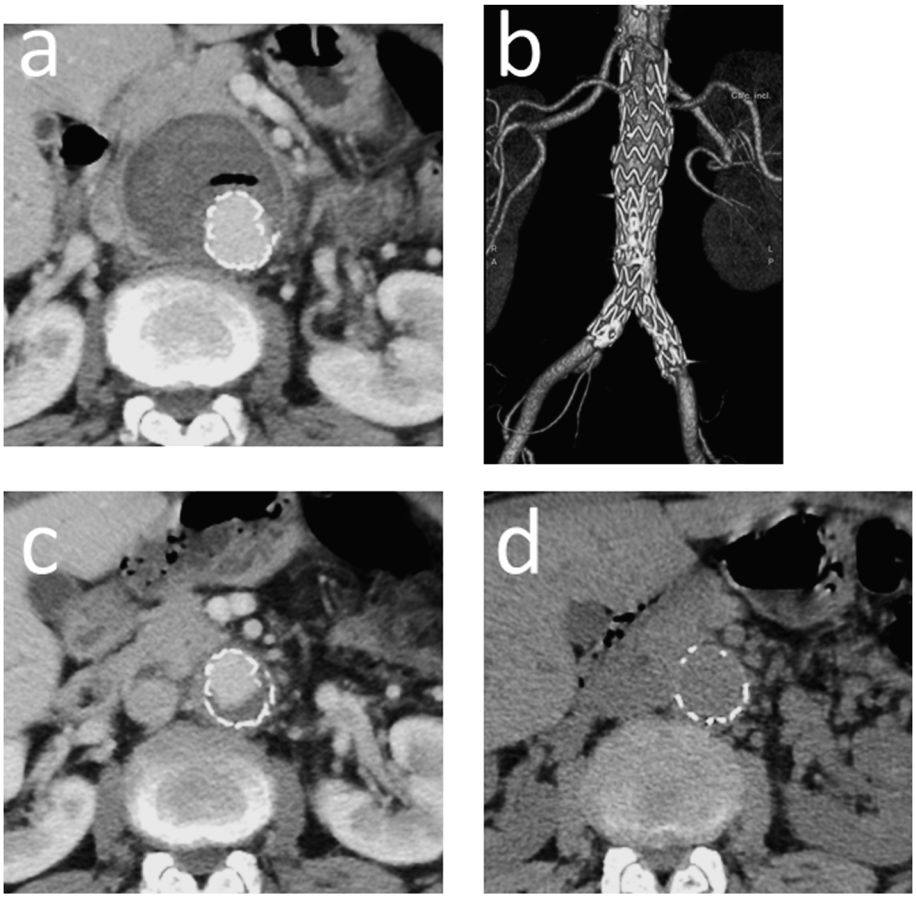

Fig. 2 Computed tomography (CT). (**a**) Enhanced CT image after endovascular abdominal aortic repair (EVAR) indicated no endoleaks. (**b**) 3D-CT image after EVAR. (**c**) Enhanced CT image at 4 months after EVAR showing shrinkage of the aneurysm. (**d**) Plain CT image at 9 months after EVAR showed no recurrence of the aneurysm.

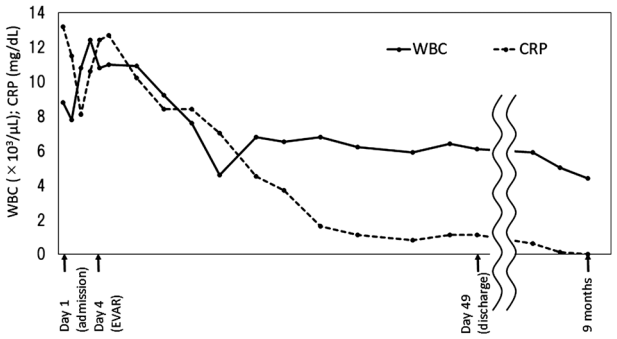

Fig. 3 Summary of the patient's perioperative course. Inflammatory markers gradually improved and became negative with treatment. CRP: C-reactive protein; WBC: white blood cells; EVAR: endovascular abdominal aortic repair

negative rod (*Helicobacter cinaedi*) infection rather than Gram-positive coccus. Finally, *Campylobacter jejuni (C. jejuni)* was confirmed as the causative agent by mass spectrometry. Then, we changed the antibiotic to imipenem/cilastatin (2 g/day). Postoperative ECT showed no endoleaks (**Figs. 2a** and **2b**). Ten days postoperatively, he had pain in the left leg. ECT revealed occlusion of the left leg of the stent-graft because of deformation. Since his symptom was only intermittent claudication and the risk of graft infection was a concern, antiplatelet therapy was commenced without performing revascularization. After administration of imipenem/cilastatin for 5 weeks, WBC count and CRP levels improved to near normal levels (**Fig. 3**). The antibiotic was changed to Clarithromycin (400 mg/day) administered orally. The patient was discharged 45 days postoperatively. Follow-up CT revealed disappearance of the aneurysm (**Figs. 2c** and **2d**). Nine months after EVAR, there has been no recurrence of the infected aneurysm.

Discussion

Staphylococcus and *Salmonella* species are common causative agents of infected aneurysms.[1] In our case, Gram-positive cocci were first suspected as being the causative agent based on blood culture reports. Subsequently, the Gram-negative rod *Helicobacter cinaedi* was more strongly suspected as the causative agent based on interim reports. Finally, *C. jejuni* was confirmed as the causative agent by mass spectrometry using VITEK MS (SYSMEX bioMérieux Co., Ltd., Tokyo, Japan). Each bacterial species has characteristic mass spectral pattern. Mass spectrometry is a new method for identification of bacteria by matching the mass spectrum pattern contained in the database. In addition, mass spectrometry can provide direct identification of bacteria in positive blood culture bottles. It contributes to rapid diagnosis of the causative agent. Although there are several reports regarding infected aneurysms caused by *Campylobacter* species, the majority are caused by *Campylobacter fetus (C. fetus)*.[2–5] *C. fetus* is known to spread throughout the body by the hematogenous route and to have an affinity for vascular endothelium. For such reason, it tends to infect circulatory organs.[5] On the other hand, *C. jejuni* is well known as a causative agent of enteritis. The symptoms of enteritis caused by *C. jejuni* are similar to those of other bacterial food poisoning, with abdominal pain, fever, vomiting, diarrhea, etc. However, *C. jejuni* rarely causes bacteremia,[6] and also rarely causes infected aneurysm. There is only one previous report in the English literature describing infected AAA caused by *C. jejuni*.[7] In our case, there were no preceding risk factors for an infected aneurysm (such as enteritis, pneumonia, and dental treatment, etc.). The patient was also not on steroids or other immunosuppressive agents, and did not have immunodeficiency. For the above reasons, the source of infection was unknown. However, *C. jejuni* has been previously reported to cause bacteremia in a healthy person.[6]

The approaches to treatment of infected AAA are as follows: 1) Vascular replacement with rifampicin-impregnated artificial graft with omental wrapping[4]; 2) Closure of the abdominal aorta with extra-anatomic revascularization[8]; 3) EVAR.[9,10] Although graft replacement is considered as radical surgery, it was considered too difficult and high risk in our case, because the aneurysm was located just below the renal artery and we anticipated severe adhesions due to the history of panperitonitis. As a result, we selected EVAR as the treatment procedure. Although the proximal neck of the aneurysm was short and of a reversed tapering shape, the aortic lumen was not dilated by a thick mural thrombus. If type I a endoleak was appeared, proximal extension was considered even if the left renal artery was sacrificed. For these reasons, we judged EVAR as being possible. Recently, EVAR has become one of the approaches for infected aneurysms, although implantation of artificial grafts in an infected field is controversial. Kan et al. reported that EVAR seems a possible alternative method for treating infected aortic aneurysms if the patient does not present with aneurysm rupture and fever.[10] Our case had a good postoperative course. There were no endoleaks and shrinkage of the aneurysm was successfully achieved.

There is no consensus regarding the period of antibiotic administration for infected AAA. We administered sensitive antibiotics for 5 weeks according to the guidelines of treatment of infective endocarditis. Thereafter, we switched to oral antibiotics, with reduction in inflammatory marker levels (WBC and CRP) and no recurrence of infection.

Conclusion

We experienced a rare case of infected AAA caused by *C. jejuni*. This is the first report of successful treatment with EVAR for infected AAA caused by *C. jejuni*.

Disclosure Statement

None of the authors has any conflicts of interest to declare.

Author Contributions

Writing: YY
Critical review and revision: all authors

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