» **Case Report** «

An Infected Abdominal Aortic Aneurysm Caused by *Helicobacter cinaedi*

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We present a rare case of an infected abdominal aortic aneurysm due to *Helicobacter cinaedi* that was detected by blood culture. A 79-year-old man with lumbago and left lower quadrant pain was admitted for the treatment of an infected abdominal aortic aneurysm. *H. cinaedi* was isolated from a blood culture, which was obtained on admission. The aneurysm was successfully treated with antibiotic therapy, aneurysmectomy, debridement, replacement of a bifurcated Dacron prosthesis, and omental wrapping. Our present case suggests that *H. cinaedi* should be considered as the causative agent of an infected aortic aneurysm.

Keywords: *Helicobacter cinaedi*, infected abdominal aortic aneurysm, operation

Introduction

Various organisms can cause infection in abdominal aortic aneurysms (AAAs). Recently, *Helicobacter cinaedi* was reported as a pathogen associated with infected AAAs.¹⁾ However, an *H. cinaedi* infection may remain undiagnosed because of the difficulty in isolating this bacterium. Here, we present a rare case of an infected AAA due to *H. cinaedi* that was detected by blood culture.

Case Report

A 79-year-old man with a history of hypertension was referred to a gastroenterologist because of lumbago and left lower quadrant pain. Biochemistry examinations revealed a white blood cell count of 9900/ μ L and a C-reactive protein level of 9.67 mg/dL. Antibiotic therapy with cefcapene pivoxil hydrochloride hydrate (100 mg orally thrice daily) was

administered because diverticulitis was suspected. Subsequently, contrast-enhanced computed tomography (CT) was performed, because his symptoms did not resolve after 2 weeks of antibiotics. CT revealed an infrarenal AAA (35 mm) with an irregular aortic wall and periaortic soft tissue infiltration (Fig. 1). Therefore, the patient was hospitalized with a diagnosis of infected AAA.

On admission, his temperature was 37.1°C, and he had a white blood cell count of 5600/ μ L and a C-reactive protein level of 2.12 mg/dL. Empirical therapy with intravenous sulbactam/ampicillin (0.5 g/1.0 g, every 12 h) was started. After 1 week of antibiotics, the patient underwent surgery. His preoperative body temperature was 36.7°C, white blood cell count 6100/ μ L, and C-reactive protein level 1.15 mg/dL. The infected AAA was managed with aneurysmectomy, debridement, replacement of a bifurcated Dacron prosthesis, and omental wrapping. The retroperitoneal space was edematous, but pus was not evident in the wall of the aortic aneurysm.

A blood culture specimen after a 95-h incubation demonstrated bacterial growth. A microscopic examination revealed gram-negative, motile, spiral bacteria. The organism formed thinly spread colonies on chocolate and blood agars after 5 days of incubation under microaerophilic conditions. Biochemical test results showed a nitrate reductase-positive and catalase-positive organism. *H. cinaedi* was identified using the API-Campy system (bioMérieux, Craponne, France), with a 68.5% probability of a correct identification.

The patient's current antibiotics were replaced with intravenous gentamicin (40 mg every 8 h for 2 weeks) and intravenous ceftriaxone (2 g every 12 h for 4 weeks) postoperatively. After 4 weeks of parenteral antibiotic therapy, daily oral antibiotic therapy with sultamicillin (1145 mg orally) was started. Antibiotic therapy was terminated 3 months postoperatively, when the patient's C-reactive protein level had returned to within normal limits. The patient has remained asymptomatic for 1 year.

Discussion

Cases of infected aortic aneurysms represent 1.3%–1.8% of all cases of aortic aneurysms, and this type of aneurysm

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Fig. 1 Contrast-enhanced computed tomography images showing a 35-mm (A), saccular, abdominal aortic aneurysm with surrounding inflamed soft tissue (B).

is a life-threatening condition, with a high mortality rate if the infection is not treated immediately.^{2,3)} The common causes of infection in AAAs include *Salmonella* spp., *Staphylococcus* sp., *Streptococcus* sp., *Escherichia coli*, *Campylobacter* sp., and others.⁴⁾ However, the causative agent is not always identified. Oderich et al. reported that aneurysm wall culture was positive in 64% of the patients and blood culture was positive in 78% of the patients.⁵⁾ Kakuta et al. reported on three cases of infected AAA caused by *H. cinaedi* detected by 16S ribosomal ribonucleic acid gene analysis,¹⁾ however, *H. cinaedi* identified by blood culture has not been previously reported as a causative agent of infected AAAs. We determined that *H. cinaedi* was the causative agent, even though culturing this organism is difficult because of its slow growth. We identified *H. cinaedi* using the API-Campy system with a 68.5% probability of a correct identification. We did not use genetic analysis because of the cost-benefit imbalance.

H. cinaedi was first isolated from rectal swabs of homosexual men with intestinal symptoms,⁶⁾ and it has since been isolated from immunocompromised patients with various diseases.⁷⁾ However, there have also been

increasing numbers of reports of *H. cinaedi* bacteremia in immunocompetent patients.⁸⁾ Khan et al. reported a possible association of *H. cinaedi* with atrial arrhythmias and atherosclerosis.⁸⁾ They also reported a pathogenic role for the organism in experimental atherosclerosis models.⁹⁾ Since the organism has been suggested to be involved in arteriosclerosis, it may become a more widely recognized cause of infective AAA.

An infected AAA is likely to rupture if aggressive surgical treatment is not performed. Accordingly, patients diagnosed with infections in AAAs have a clear indication for surgery. The shape of an infected abdominal aneurysm is saccular and therefore it is likely to rupture. Even if inflammation subsides with antibiotics therapy, surgery is necessary. However, despite surgical treatment, controlling the infection can still be difficult, and patients may die due to infected vascular prostheses, septicemia, and/or aneurysm development at the anastomotic site. Thus, the treatment strategy must be carefully considered. The surgical procedures for an infected AAA consist of aneurysmectomy and debridement, and may also include extra-anatomical bypass surgery, avoiding the focus of the infection. For the present patient, however, we performed *in situ* vascular prosthesis replacement and omental wrapping because the infection was being controlled with the preoperative antibiotic therapy. Recently, many studies have examined *in situ* reconstruction as the treatment of choice. Hsu et al. performed this procedure for infected AAAs and reported good clinical outcomes, with an early mortality rate of 11% and a vascular prosthesis infection rate of 10%.¹⁰⁾ Lee et al. retrospectively compared extra-anatomic bypasses with *in situ* bypass surgery and concluded that *in situ* graft revascularization is viable in afebrile patients or those who respond well to preoperative antibiotic therapy. Extra-anatomic bypass grafting and infected infrarenal abdominal aneurysm resection has a similar long-term survival rate and should be considered in patients who are unsuitable for *in situ* graft revascularization.¹¹⁾

Conclusion

In summary, a patient with an infected AAA caused by *H. cinaedi* that was isolated from a blood culture was managed with antibiotic therapy, aneurysmectomy, debridement, replacement of a bifurcated Dacron prosthesis, and omental wrapping. Our present case suggests that *H. cinaedi* should be considered as the causative agent of an infected aortic aneurysm.

Disclosure Statement

The authors have no conflict of interest to declare.

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