Helicobacter cinaedi as a cause of primary aortic infections and the challenges of diagnosis and optimal treatment

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ABSTRACT

Helicobacter cinaedi is primarily seen and diagnosed in immunocompromised patients. We present cases of primary mycotic aortitis in three immunocompetent and one immunocompromised patient caused by *H cinaedi*. Bacterial identification was made through perioperative samples using 16S rRNA gene sequencing, as routine culture techniques were negative owing to the slow turnaround time of *H cinaedi*. Successful management was achieved with a neoaortoiliac system and prolonged intravenous antibiotic therapy. All four patients had a history of having sex with men, highlighting the need to further investigate the transmission of *H cinaedi* in immunocompetent patients and its association with aortitis. (J Vasc Surg Cases Innov Tech 2025;11:101744.)

Keywords: Aortitis; *Helicobacter cinaedi*; NAIS procedure; 16S rRNA gene sequencing

Infectious aortitis carries significant morbidity and mortality rates, with *Staphylococcus aureus* and *Salmonella* spp. being the most implicated pathogens.[1] *Helicobacter cinaedi*, a gram-negative spiral rod, was once considered an opportunistic pathogen, but is now known to infect immunocompetent individuals, mostly men having sex with men.[2,3] We present four cases of *H cinaedi* aortitis successfully treated with long-term IV antibiotics and autologous venous reconstruction using a neoaortoiliac system (NAIS) procedure. This case series respects the SCARE criteria. Written consents were received from all patients and all cases are summarized in Table. An exemption from the Comité d'éthique de la recherche du Centre Hospitalier Universitaire de Québec-Université Laval (CER-CHU-UL) was received for this study.

CASE REPORT 1

A 51-year-old man with known hypothyroidism consulted for abdominal pain and asthenia. Initially misdiagnosed with constipation, his symptoms worsened over a week, leading to a computed tomography scan showing an ulcerated lesion at the infrarenal aorta with surrounding soft tissue infiltration. Physical examination showed mild abdominal tenderness, normal vitals and lab tests showed normal white blood cell count and elevated C-reactive protein (50.7 mg/L). A positron emission tomography (PET) scan showed circumferential periaortic hypermetabolism (Fig 1). The patient was started on piperacillin-tazobactam and doxycycline because there were concerns of a Q fever infection and a NAIS procedure was performed. Tissue biopsy 16S rRNA gene sequencing later identified *H* cinaedi, prompting a change to IV ceftriaxone and ciprofloxacin for a total of 7 weeks.

CASE REPORT 2

A 73-year-old man presented with acute lower abdominal pain radiating to his back and chills. His past medical history was remarkable for a 3.0 cm infrarenal abdominal aortic aneurysm (AAA). On admission, his temperature was 38.3°C with normal vital signs. The patient had a history of sleeping with other men and manipulating dead animal carcasses. Physical examination was positive for a tender, pulsatile, abdominal mass. Laboratory tests showed leukocytosis (11.1×10^9 cells/L) and elevated C-reactive protein (104.0 mg/L). On computed tomography angiography (CTA), the AAA had increased in size from 3.0 to 4.5 cm over 2 years and was associated with significant retroperitoneal tissue infiltration, raising high suspicion for aortitis (Fig 2). Empirical piperacillin-tazobactam was initiated and patient underwent NAIS. Perioperative cultures demonstrated *S. pneumoniae* and piperacillin-tazobactam was switched for ceftriaxone. However, 16S rRNA gene sequencing on surgical tissue biopsy later identified *H cinaedi* prompting a change to a 6-week antibiotic treatment using meropenem.

CASE REPORT 3

A 72-year-old man with hypertension, hypothyroidism, and a recent inferior myocardial infarction presented with a month-long history of progressive abdominal pain. Initially thought to be diverticulitis, he was treated with ciprofloxacin and metronidazole. CTA showed inflammation surrounding a 17-mm left common iliac aneurysm. Laboratory tests showed no

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Table. Case summary of four patients with *Helicobacter cinaedi* aortitis

Characteristics	Case/patient 1	Case/patient 2	Case/patient 3	Case/patient 4
Age, years/sex	51/Male	73/Male	72/Male	77/Male
Past medical history	HTN, hypothyroidism	3.0 cm AAA	HTN, DLP, hypothyroidism, IgM MGUS, recent myocardial infarction	HIV-positive status, DLP, history of 3 cancers (gastric, bladder, prostate)
Risk factors for aortitis	MSM, contact with farm animals and animal carcasses	MSM, contact with animal carcasses, recent colonoscopy	MSM	MSM, forest stay
Chief complaint	Abdominal pain and asthenia	Fever	Abdominal pain	Abdominal pain and asthenia
Blood culture	Negative	Negative	Negative	Negative
Management	NAIS + IV antibiotics	NAIS + IV antibiotics	NAIS + IV antibiotics	NAIS + IV antibiotics
Antimicrobial therapy				
Before surgery	None	Piperacillin-Tazobactam	None	None
After surgery	Piperacillin-tazobactam + doxycycline	Imipenem + voriconazole → ceftriaxone	Piperacillin-tazobactam + tigecycline	Meropenem + doxycycline
At discharge (after pathogen identification)	Ceftriaxone + ciprofloxacin	Meropenem	Ciprofloxacin	Meropenem
Duration of treatment	7 weeks	6 weeks	7 weeks	6 weeks
Outcome	Alive and well at the 6-year follow-up	Alive and well at the 3-year follow-up	Alive and well at the 1-year follow-up	Alive and well at the 6-month follow-up

AAA, Abdominal aortic aneurysm; *DLP*, dyslipidemia; *IV*, intravenous; *MGUS*, monoclonal gammopathy of undetermined significance; *MSM*, men who have sex with men; *NAIS*, neoaortoiliac system.

leukocytosis but an elevated C-reactive protein (50.7 mg/L). PET scan confirmed a mycotic aneurysm (Fig 3). Subsequently, a NAIS procedure was performed. After surgery, the patient was treated initially with piperacillin-tazobactam and tigecycline. Later, 16S rRNA gene sequencing revealed *H cinaedi* as the causative agent, prompting a change to intravenous ciprofloxacin for a total of 7 weeks.

CASE REPORT 4

A 77-year-old HIV-positive man with an undetectable viral load and a history of gastric lymphoma, presented with 2 weeks of abdominal pain and fever. A CTA revealed an infrarenal aortic pseudoaneurysm. On admission, his symptoms had subsided, his vitals were stable, and all lab tests were within normal range. PET scan revealed intense uptake around the pseudoaneurysm, confirming infection (Fig 4), and the patient underwent a NAIS procedure. Postoperatively, meropenem and doxycycline were initiated, but later 16S rRNA gene sequencing identified *H cinaedi*. The antibiotic regiment was adjusted to meropenem alone for 6 weeks.

DISCUSSION

We describe four patients with aortitis caused by *H cinaedi* identified postoperatively by 16S rRNA gene sequencing on tissue biopsy. *H cinaedi* has been reported predominantly in Japan, where it has been implicated in cases of infected ascending aorta, AAA, and endocarditis. Recent studies have detected *H cinaedi* in the walls of clinically noninfectious AAAs and have shown the bacterium's association with atherosclerosis.[4,5] This raises questions about *H cinaedi*'s causality in certain cases of aortitis, especially when a well-established pathogen is found concomitantly, as in case 2 of our series. However, it is crucial to note that *H cinaedi*'s role in these cases may be coincidental rather than causal, necessitating further research to delineate its pathogenic role. Owing to the low virulence of *H cinaedi*, infections tend to progress indolently,[6] and, to date, no cases of aneurysm rupture attributed to this bacterium have been documented.[7] Despite the indolent nature, we opted for NAIS for all patients, because it offers long-term durability and minimizes the risk of reinfection.

The risk of *H cinaedi* infection is greater in immunocompromised patients.[8] Our case series describes a strong association between *H cinaedi* and men who have sex with men. Studies suggest that asymptomatic carriers may contribute to the transmission of *H cinaedi* through a fecal-oral route; the bacterium has been isolated from stool specimens.[8,9] Additionally, *H cinaedi* has been identified in urine samples, further indicating

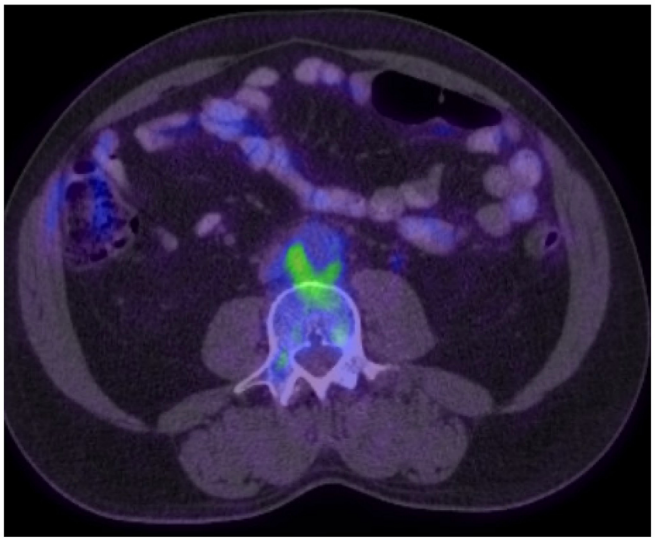

Fig 1. Positron emission tomography (PET) scan showing periaortic tissue infiltration confirming the diagnosis of aortitis in case 1.

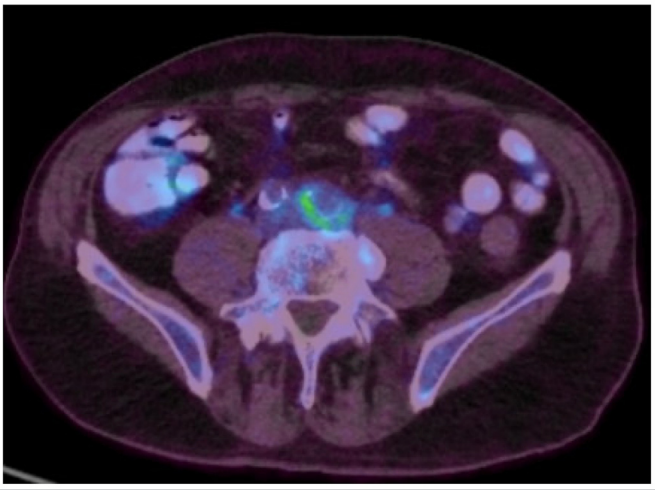

Fig 3. Positron emission tomography (PET) scan showing a 2-cm common iliac aneurysm with infiltration confirming aortitis in case 3.

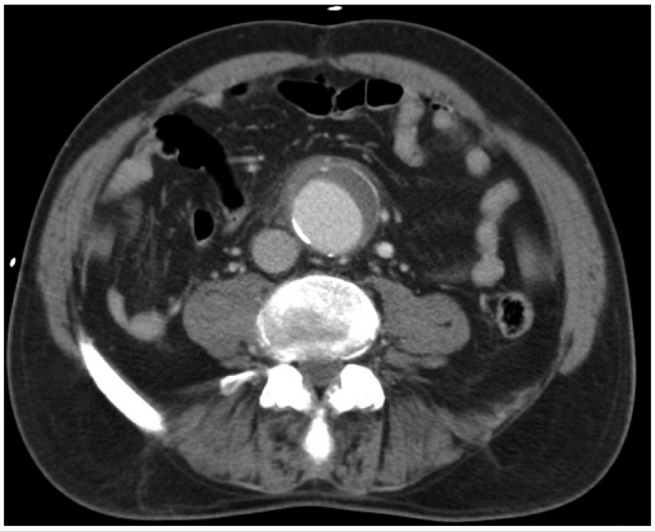

Fig 2. Computed tomography angiography (CTA) showing a 4.5-cm abdominal aortic aneurysm (AAA) with retroperitoneal tissue infiltration, suggestive of aortitis in case 2.

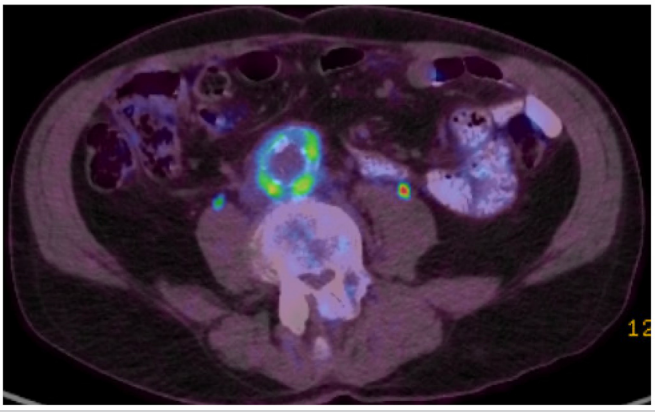

Fig 4. Positron emission tomography (PET) scan showing periaortic infiltration confirming an aortitis in case 4.

its potential for undetected spread.[9] Bacterial translocation from the intestinal tract or mucosal damage has been proposed as the most likely mechanism for *H cinaedi* bacteremia and vascular infections.[10] Other routes of transmission remain unclear, although zoonotic transmission and nosocomial dissemination have been suggested, even in individuals with competent immune systems. Case 2 had two distinct risk factors of *H. cineadi*—sleeping with other men and manipulation of dead animals.[2,11]

Diagnosing *H cinaedi* is challenging owing to its microaerophilic nature and slow growth.[12] Prolonged incubation times are recommended when *H cinaedi* bacteremia is suspected. Blood culture positivity rate is low, ranging from 0% to 22%,[7,13] showing the challenging diagnosis of new pathogens in aortitis. Advances in detection techniques, such as polymerase chain reaction-based methods on tissue biopsy targeting 16S rRNA or gyrB have, significantly improved the identification of elusive pathogens.[2,12,14] At our center, tissue biopsy for culture and gene sequencing is a standard of care for aortic infection. The use of 16S rRNA sequencing for definitive diagnosis proved to be essential in all patients in the series. In two cases, gene sequencing identified *H cinaedi* despite the presence of other pathogens on culture, demonstrating its critical role in achieving an accurate diagnosis.

The clinical management of *H cinaedi* infections is further complicated by the lack of established guidelines for antibiotic susceptibility testing or duration of

treatment. Nevertheless, carbapenems, tetracycline, and aminoglycosides have demonstrated superior efficacy compared with penicillin and cephalosporin, although resistance to fluoroquinolones and macrolides is well-documented.[7,13] When *H cinaedi* infection is suspected, initial empirical therapy with meropenem is suggested in the literature.[15] Postsurgical antimicrobial treatment typically involves a minimum of 6 weeks of intravenous therapy, with some experts advocating for extended oral therapy lasting from 6 months to lifelong therapy.[16,17] Surgical intervention remains a critical component of treatment, with open repair considered the gold standard for managing infected AAAs.[18,19]

CONCLUSIONS

This case series recognizes *H cinaedi* as a cause of infectious aortitis, previously under-recognized in immunocompetent individuals, and shows the changing face of aortic infections. Its diagnosis is challenging requiring a dedicated history of risk factors and perioperative tissue biopsy for gene sequencing. The potential of men having sex with men or zoonotic transmissions, the role of asymptomatic carriers, and the development of targeted therapies are areas that warrant continued investigation for optimal management.

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