

## ***Helicobacter cinaedi* bacteremia with cellulitis after ABO-incompatible living-donor liver transplantation: Case report**

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### **Abstract**

*Helicobacter cinaedi* (*H. cinaedi*), a Gram-negative spiral-shaped bacterium, is an enterohepatic non-*Helicobacter pylori* *Helicobacter* species. We report the first case of *H. cinaedi* bacteremia with cellulitis after liver transplantation. A 48-year-old male, who had been a dog breeder for 15 years, underwent ABO-incompatible living-donor liver transplantation for hepatitis C virus-induced decompensated cirrhosis using an anti-hepatitis B core antibody-positive graft. The patient was preoperatively administered rituximab and underwent plasma exchange twice to overcome blood type incompatibility. After discharge, he had been doing well with immunosuppression therapy comprising cyclosporine, mycophenolate mofetil, and steroid according to the ABO-incompatible protocol of our institution. However, 7 mo after transplantation, he was admitted to our hospital with a diagnosis of recurrent cellulitis on the left lower extremity, and *H. cinaedi* was detected by both blood culture and polymerase chain reaction analysis. Antibiotics improved his symptoms, and he was discharged at day 30 after admission. Clinicians should be more aware of *H. cinaedi* in immunocompromised patients, such as ABO-incompatible transplant recipients.

**Key words:** *Helicobacter cinaedi*; Bacteremia; Cellulitis; Liver transplantation; Hepatitis C; Living-donor; ABO-incompatible; HBc-Ab-positive donor

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**Core tip:** This is the first case report of *Helicobacter cinaedi* infection in a liver transplant recipient. Clinicians should be aware of this microorganism when treating immunocompromised patients, such as ABO-incompatible liver transplant recipients with symptoms of cellulitis.

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## INTRODUCTION

*Helicobacter* is a genus of gram-negative bacteria possessing a characteristic spiral shape. The most well-known species of the genus is *Helicobacter pylori*, as some strains are associated with peptic ulcers, chronic gastritis, and gastric cancers. Nevertheless, several reports published during the last few decades have contributed to a better understanding of both human and animal infection with non-*Helicobacter pylori* *Helicobacter* species<sup>[1]</sup>. One such enterohepatic species is *Helicobacter cinaedi* (*H. cinaedi*), which colonizes the gastrointestinal tract mucosa of mammals, including humans<sup>[2]</sup>. Cellulitis due to *H. cinaedi* is occasionally reported in neutropenic patients with hematologic malignancies and less frequently in patients with immunocompromised conditions, such as diabetes mellitus and malnutrition. Here, we report the first case of *H. cinaedi* bacteremia with cellulitis after liver transplantation.

In Japan, although the number of deceased donor liver transplantation (DDLT) has gradually increased, living-donor liver transplantation (LDLT) is still the most frequent treatment option because of organ shortages. Therefore, the use of liver grafts from hepatitis B core antibody (HBc-Ab)-positive<sup>[3,4]</sup> or ABO-incompatible (ABO-I) living donors are permitted in Japan. The present case is also a rare case of ABO-I LDLT from an HBc-Ab-positive donor to a recipient with hepatitis C virus (HCV).

## CASE REPORT

After receiving a detailed explanation, the patient

**Table 1** Preoperative blood type, viral marker status of the recipient and donor

Blood type	Recipient B Rh (+)	Donor AB Rh (+)
Viral marker status		
HBs-Ag (C.O.I)	< 1.0	< 1.0
HBs-Ab (mIU/mL)	186.9	< 10.0
HBc-Ab (% Inh)	92.6	95.8
HBe-Ag (S/CO)	< 0.50	< 0.50
HBe-Ab (% Inh)	73	48
HBV-RNA (log copy/mL)	–	–
HCV-Ab	+	–
HCV-RNA (log IU/mL)	5.8	–
HCV genotype	2a	–

HBs-Ag: Hepatitis B surface antigen; HBs-Ab: Hepatitis B surface antibody; HBc-Ag: Hepatitis B core antigen; HBc-Ab: Hepatitis B core antibody; HBe-Ag: Hepatitis B envelope antigen; HBe-Ab: Hepatitis B envelope antibody; HCV-Ab: Hepatitis C virus antibody.

provided informed consent to publish his case details.

A 48-year-old male, who had been a dog breeder for 15 years, underwent ABO-I LDLT for HCV-induced decompensated liver cirrhosis with an HBc-Ab-positive liver graft. His Model for End-stage Liver Disease (MELD) score was 9 at the time of LDLT. His notable medical history included was hypertension and diabetes mellitus.

The donor was the patient's 46-year-old younger brother who had no notable medical history except for resolved HBV infection. The viral marker statuses of the recipient and donor are summarized in Table 1; the results suggest that the recipient also had a history of resolved HBV infection. To overcome blood type incompatibility, 500 mg/body of rituximab (an anti-CD20 antibody) was administered to the recipient four weeks before LDLT and preoperative plasma exchange was performed twice according to our institution's protocol<sup>[5]</sup>. LDLT was performed routinely using a left lobe graft. Intraoperative liver wedge biopsy of the donor revealed no evidence of steatosis. Immediately after total hepatectomy was performed, 10000 IU of hepatitis B immunoglobulin (HBIG) was systemically infused into the recipient as anti-HBV prophylaxis. The liver graft was revascularized in the order of anastomosis of the hepatic vein to the inferior vena cava and reconstruction of the portal vein and hepatic artery. Immediately before reperfusion of the liver *via* the portal vein, 650 mg of methylprednisolone was infused intravenously. Splenectomy was performed following hepatic artery reconstruction, and a portal vein catheter was placed *via* a middle colic vein for local graft infusion therapy according to the immunosuppression protocol for ABO-I<sup>[5,6]</sup>.

In addition to routine postoperative treatment, the CD19- and CD20-positive B-cell counts, as well as isoagglutinin titers of anti-A and anti-B, were monitored frequently. At postoperative day 25, tacrolimus was stopped and cyclosporine was started

**Table 2** Case reports of *Helicobacter cinaedi* bacteremia and their associated symptoms *n* (%)

Ref.	Bacteremia, <i>n</i>	Fever	Cellulitis	Diarrhea
Kawakami <i>et al</i> <sup>[21]</sup>	46	43 (93)	8 (17.4)	4 (8.7)
Araoka <i>et al</i> <sup>[22]</sup>	63	ND	24 (38.0)	7 (10.4)
Mandai <i>et al</i> <sup>[23]</sup>	1	Yes	Yes	No
Kikuchi <i>et al</i> <sup>[24]</sup>	1	Yes	Yes	No
Kim <i>et al</i> <sup>[25]</sup>	1	Yes	Yes	No
Ishizawa <i>et al</i> <sup>[26]</sup>	1	No	Yes	No
Holst <i>et al</i> <sup>[19]</sup>	1	Yes	Yes	No
Matsumoto <i>et al</i> <sup>[16]</sup>	6	6 (100)	ND	0 (0)
Nishine <i>et al</i> <sup>[27]</sup>	1	Yes	No	No
Kitamura <i>et al</i> <sup>[17]</sup>	11	ND	11 (100)	ND
Uçkay <i>et al</i> <sup>[28]</sup>	1	Yes	No	No
Van Genderen <i>et al</i> <sup>[29]</sup>	1	Yes	Yes	No
Simons <sup>[30]</sup>	1	No	No	No
Murakami <i>et al</i> <sup>[8]</sup>	1	Yes	Yes	No
Lasry <i>et al</i> <sup>[31]</sup>	1	Yes	No	No
Hung <i>et al</i> <sup>[32]</sup>	1	Yes	No	Yes
Sullivan <i>et al</i> <sup>[33]</sup>	1	Yes	Yes	No
Tee <i>et al</i> <sup>[34]</sup>	3	ND	1 (33)	0
Mammen <i>et al</i> <sup>[35]</sup>	1	Yes	No	Yes
Burman <i>et al</i> <sup>[11]</sup>	7	5 (71.4)	4 (57.1)	1 (14.3)

ND: Not documented.

due to the possibility of thrombotic microangiopathy. Immunosuppression therapy at discharge (*i.e.*, postoperative day 63) comprised cyclosporine (130 mg/d), MMF (2000 mg/d), and PSL (15 mg/d). The doses of these drugs were gradually reduced during follow-up. He was followed almost every two weeks.

Unfortunately, four months after ABO-I LDLT, routine laboratory investigations and liver biopsy specimens showed early HCV relapse. The HCV-RNA level at that time had increased to 7.2 log IU/mL. As the patient had a history of progression of diabetic retinopathy due to interferon therapy and liver function tests at that time were almost normal, he did not start interferon therapy; he is planned to take sofosbuvir, which will be approved shortly in Japan.

Seven months after transplantation, he was hospitalized with complaints of high fever and swelling in the left lower extremity, which is compatible with cellulitis, without any signs of trauma. On admission, hemoglobin level was 11.9 g/dL, white blood cell count was 13000/ $\mu$ L with 80.5% neutrophils, and platelet count was 364000/ $\mu$ L. C-reactive protein level was elevated to 6.50 mg/dL. Blood culture was not analyzed at that time. Thereafter, cefazolin was empirically administered for seven days. His symptoms were relieved immediately, and he was discharged at day 10 after admission.

However, one week later, he was readmitted with a diagnosis of recurrent cellulitis on the left lower extremity. Blood culture was analyzed at this time, and cefazolin was empirically administered again. Although left lower leg swelling improved immediately, subfever was prolonged and gram-negative spiral bacteria were confirmed by both aerobic and anaerobic vials of two sets of blood cultures at day 5 after admission.

Considering the possibility of *Campylobacter* infection according to the results of gram-negative spiral bacteria, cefazolin was replaced with ciprofloxacin. The results of the API Campy kit (Sysmex bioMerieux Co., Ltd., Kobe, Japan) indicated that the causative microorganism was *H. cinaedi* with a 68.5% probability; 16S rRNA gene sequencing was performed for further identification. According to a search of the Basic Local Alignment Search Tool (BLAST) database (<http://www.ncbi.nlm.nih.gov/blast/>), the sequence of this isolate exhibited 99% similarity with that of *H. cinaedi*. As the swelling of the left lower extremity and high fever occurred simultaneously, we diagnosed *H. cinaedi* bacteremia with recurrent cellulitis. According to the result of antibiotic susceptibility testing (disk diffusion test), the microorganism was susceptible to tetracycline, third generation cefem, and carbapenem, and, on the contrary, resistant to first generation cefem and new quinolone antibiotics. Therefore, ciprofloxacin was switched to minocycline at day 20 after admission because of reports of increasing quinolone-resistant *H. cinaedi*. Thereafter, his subfever resolved, and he was discharged at day 30 after the latest admission. He has been on minocycline for more than 3 mo and is currently being followed up at our institution, without recurrence.

## DISCUSSION

We reported a case of bacteremia with cellulitis caused by *H. cinaedi* after LDLT for HCV-induced decompensated liver cirrhosis, using an HBc-Ab-positive liver graft. To our knowledge, this is the first case report of *H. cinaedi* infection in a liver transplant recipient; meanwhile, there is only one case report of ABO-I LDLT from an HBc-Ab-positive donor to an HCV recipient<sup>[7]</sup>. In the field of solid organ transplantation, only one case of *H. cinaedi* infection after renal transplant has been reported<sup>[8]</sup>. *H. cinaedi* was originally isolated as a *Campylobacter*-like organism from rectal swabs obtained from homosexual men infected with HIV in 1984<sup>[9]</sup>. Regarding the isolation of *H. cinaedi*, it takes  $4.1 \pm 1.60$  d to identify this species after blood culture. Therefore, at least 5 d of incubation is required to avoid overlooking the microorganism.

Some cases of *H. cinaedi* infection have been reported during the last few decades. In these reports, this microorganism is described as causing diverse symptoms, including erysipelas, cellulitis, arthritis, and neonatal meningitis, as well as gastroenteritis and proctitis<sup>[10-14]</sup>. A review of the literature on cases of bacteremia by *H. cinaedi* documenting the incidence of each symptom is shown in Table 2. Interestingly, cellulitis was observed in 56/150 cases (37.3%), whereas diarrhea was only reported in 14/150 cases (9.3%); thus, cellulitis is the predominant symptom caused by this microorganism compared with other gram-negative enteric bacilli, such as *Campylobacter* spp.

Regarding *H. cinaedi* pathogenesis, the secondary involvement of the skin and subcutaneous tissues in bacteremia is thought to be caused by its toxic factors<sup>[15]</sup>. In addition, immunodeficiency may allow continuous bacterial translocation resulting in high recurrence. In our case, recurrent cellulitis accompanied by bacteremia led to the diagnosis of *H. cinaedi* infection.

Regarding our patient's background, it has been reported that *H. cinaedi* bacteremia is rare but can occur in immunocompromised hosts by Matsumoto, Goto, who evaluated the prevalence of *H. cinaedi* as a bacteremia-causing pathogen by analyzing blood culture samples<sup>[16]</sup>. *H. cinaedi* infection is observed occasionally in patients with alcoholism, diabetes, and malignancy and less commonly in patients with no recognized host defense defect<sup>[17-19]</sup>. As this microorganism is presumably transmitted from animals to human *via* the fecal-oral route, our patient's work as dog breeder for 15 years may be associated with the infection route of *H. cinaedi*. In addition, splenectomy and the immunosuppression protocol for ABO-I comprising rituximab (anti-CD20 antibody), tacrolimus / cyclosporine, MMF, and PSL might have been associated with pathogenesis by strongly affecting the patient's immunity.

Of the drugs mentioned above, rituximab is a key drug for suppressing humoral immunity in ABO-I LDLT. In Japan, where LDLT has been developed more than DDLT because of a lack of brain-dead donors, donors are mostly limited to close family members. Therefore, ABO-I LDLT use in Japan is more common than in other countries. In ABO-I LDLT, B-cells and alloantibodies become pathogenic in terms of antibody-mediated rejection in addition to cell-mediated rejection, which is also observed in ABO-compatible LDLT. Rituximab is a monoclonal antibody usually used to treat B-cell non-Hodgkin lymphoma. In ABO-I LDLT, the effectiveness of rituximab is mostly explained by its depletion of specific antidonor antibodies and elimination of circulating and presumably tissue CD20<sup>+</sup> B-cells<sup>[20]</sup>. As the effect of rituximab persists for several months, serious fungal, bacterial, and new or reactivated viral infections can occur after treatment. The long-term effectiveness of rituximab may explain why cellulitis occurred in our patient, who was taking only cyclosporine when the pathogenesis of cellulitis occurred.

There are currently no clear guidelines in the literature concerning the choice or duration of antibiotic therapy for *H. cinaedi* infection. A large review of 23 cases of bacteremia reported that penicillins, tetracycline, and aminoglycosides are more effective than are cephalosporins, erythromycin, or ciprofloxacin<sup>[10]</sup>. Quinolones alone may not completely eradicate *H. cinaedi*, which explains the frequent reports of recurrent disease after quinolone monotherapy. In our case, recurrent cellulitis was observed in spite of the use of cefazolin for one week; therefore, oral minocycline was

continued for more than three months.

In conclusion, this is the first case report of *H. cinaedi* infection in a liver transplant recipient. Clinicians should be aware of this microorganism when treating immunocompromised patients such as ABO-I transplant recipients with symptoms of cellulitis.

## COMMENTS

### Case characteristics

A 48-year-old male who was diagnosed with recurrent cellulitis on the left lower extremity after liver transplantation.

### Clinical diagnosis

*Helicobacter cinaedi* (*H. cinaedi*) bacteremia with cellulitis.

### Differential diagnosis

*Campylobacter* species infection.

### Laboratory diagnosis

Spiral bacteria were confirmed by both aerobic and anaerobic vials of blood cultures, and 16S rRNA gene sequencing of this isolate exhibited 99% similarity with that of *H. cinaedi*.

### Imaging diagnosis

There are no imaging methods to confirm the diagnosis of *Helicobacter cinaedi* infection.

### Pathological diagnosis

There are no pathological methods to confirm the diagnosis of *Helicobacter cinaedi* infection.

### Treatment

At first, cefazolin was empirically administered for one week. Finally, oral minocycline was continued for more than three months after the diagnosis of *Helicobacter cinaedi* bacteremia with cellulitis was obtained.

### Related reports

To date, this is the first case report of *Helicobacter cinaedi* bacteremia with cellulitis after liver transplantation.

### Term explanation

*Helicobacter cinaedi* is one of the non-*Helicobacter pylori* *Helicobacter* species that colonizes the gastrointestinal tract mucosa of mammals, including humans.

### Experience and lessons

Review of literature showed that cellulitis is the predominant symptom caused by *Helicobacter cinaedi*. Clinicians should be aware of this microorganism when treating immunocompromised patients such as ABO-I liver living-donor transplant recipients with symptoms of cellulitis.

### Peer-review

This is the first case report of *Helicobacter cinaedi* infection in a liver transplant recipient. Due to the limited volume of patients, the underlying cause of bacteremia and cellulitis is unknown.

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