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

Kohei Mishima, Hideaki Obara, Kayoko Sugita, Masahiro Shinoda, Minoru Kitago, Yuta Abe, Taizo Hibi, Hiroshi Yagi, Kentaro Matsubara, Takehiko Mori, Yaoko Takano, Hiroshi Fujiwara, Osamu Itano, Naoki Hasegawa, Satoshi Iwata, Yuku Kitagawa

Kohei Mishima, Hideaki Obara, Masahiro Shinoda, Minoru Kitago, Yuta Abe, Taizo Hibi, Hiroshi Yagi, Kentaro Matsubara, Osamu Itano, Yuko Kitagawa, Department of Surgery, Keio University School of Medicine, Tokyo 160-8582, Japan

Kayoko Sugita, Yaoko Takano, Hiroshi Fujiwara, Naoki Hasegawa, Center for Infectious Diseases and Infection Control, Keio University School of Medicine, Tokyo 160-8582, Japan

Takehiko Mori, Division of Hematology, the Department of Medicine, Keio University School of Medicine, Tokyo 160-8582, Japan

Satoshi Iwata, Department of Infectious Diseases, Keio University School of Medicine, Tokyo 160-8582, Japan

**Author contributions:** Mishima K and Obara H wrote this paper; Sugita K performed analysis of blood culture and gene sequence; all other members equally contributed to medical treatment.

**Ethics approval:** The study was reviewed and approved by the Keio University School of Medicine Institutional Review Board.

**Informed consent statement:** All study participants, or their legal guardian, provided informed written consent prior to study enrollment.

**Conflict-of-interest statement:** All authors declare no conflicts of interest.

**Open-Access:** This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/4.0/

**Correspondence to:** Hideaki Obara, MD, PhD, Department of Surgery, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo 160-8582, Japan. obara@z3.keio.jp

Telephone: +81-3-33531211
Fax: +81-3-33554707

Received: March 3, 2015
Peer-review started: March 4, 2015
First decision: April 13, 2015
Revised: May 9, 2015
Accepted: May 21, 2015
Article in press: May 21, 2015
Published online: July 7, 2015

**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

© The Author(s) 2015. Published by Baishideng Publishing Group Inc. All rights reserved.

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.

---

**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[1]. One such enterohepatic species is Helicobacter cinaedi (H. cinaedi), which colonizes the gastrointestinal tract mucosa of mammals, including humans[2]. 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 ABO-incompatible living-donor liver transplantation: Case report. World J Gastroenterol 2015; 21(25): 7911-7915 Available from: URL: http://www.wjgnet.com/1007-9327/full/v21/i25/7911.htm DOI: http://dx.doi.org/10.3748/wjg.v21.i25.7911

---

**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/CD)     | < 0.50            | < 0.50          |
| Hbc-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-Ab: 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.

---

**CASE REPORT**

After receiving a detailed explanation, the patient 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[5], LDLT was performed routinely using a left lobe graft. Intraoperative liver wedge biopsy of the donor revealed no evidence of steatosis. Immediately after total heptectomy 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 perfusion 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[5,6].

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, n | Fever | Cellulitis | Diarrhea |
|------------|---------------|-------|------------|----------|
| Kawakami et al[31] | 46            | 43 (93) | 8 (17.4) | 4 (8.7)  |
| Arakawa et al[32]  | 63            | ND    | 24 (38.0) | 7 (10.4) |
| Mandal et al[33]   | 1             | Yes   | Yes        | No       |
| Kikuchi et al[34]  | 1             | Yes   | Yes        | No       |
| Kim et al[35]      | 1             | Yes   | Yes        | No       |
| Ishizawa et al[36] | 1             | No    | Yes        | No       |
| Holst et al[37]    | 1             | Yes   | Yes        | No       |
| Matsumoto et al[38] | 6          | 6 (100) | ND       | 0 (0)    |
| Nishine et al[39]  | 1             | Yes   | No         | No       |
| Kitamura et al[40] | 11            | ND    | 11 (100)   | ND       |
| Ucâk et al[41]     | 1             | Yes   | No         | No       |
| Van Genderen et al[42] | 1     | Yes   | Yes        | No       |
| Simons[43]         | 1             | No    | No         | No       |
| Murakami et al[44] | 1             | Yes   | Yes        | No       |
| Lasry et al[45]    | 1             | Yes   | No         | No       |
| Hung et al[46]     | 1             | Yes   | No         | Yes      |
| Sullivan et al[47] | 1             | Yes   | Yes        | No       |
| Tec et al[48]      | 3             | ND    | 1 (33)     | 0        |
| Mannen et al[49]   | 1             | Yes   | No         | Yes      |
| Burman et al[50]   | 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/µL with 80.5% neutrophils, and platelet count was 364000/µL. C-reactive protein level was elevated to 6.50 mg/dL. Blood culture was not analyzed at that time. Therefore, 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 BioMeirieux 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[7]. In the field of solid organ transplantation, only one case of H. cinaedi infection after renal transplant has been reported[8]. H. cinaedi was originally isolated as a Campylobacter-like organism from rectal swabs obtained from homosexual men infected with HIV in 1984[9]. Regarding the isolation of H. cinaedi, it takes 4.1 ± 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[10–14]. 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.
Mishima K et al. H. cinaedi after liver transplantation

Regarding H. cinaedi pathogenesis, the secondary involvement of the skin and subcutaneous tissues in bacteremia is thought to be caused by its toxic factors. 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. H. cinaedi infection is observed occasionally in patients with alcoholism, diabetes, and malignancy and less commonly in patients with no recognized host defense defect. As this microorganism is presumably transmitted from animals to human via the fecal-oral route, our patient’s work as a 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+ B-cells. 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, tetra- cycline, and aminoglycosides are more effective than cephalosporins, erythromycin, or ciprofloxacin. 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.

**REFERENCES**

1. Flahou B, Haesebrouck F, Smet A, Yonezawa H, Osaki T, Kamiya S. Gastric and enterohepatic non-Helicobacter pylori Helicobacters. Helicobacter 2013; 18 Suppl 1: 66-72 [PMID: 24011248 DOI: 10.1111/hel.12072]
2. Solnick JV, Schauer DB. Emergence of diverse Helicobacter species in the pathogenesis of gastric and enterohepatic diseases. Clin Microbiol Rev 2001; 14: 59-97 [PMID: 11148003 DOI: 10.1128/cmrr.14.1.59-97.2001]
3. Nadig SN, Bratton CF, Karp SJ. Marginal donors in liver transplantation: expanding the donor pool. J Surg Educ 2007; 64: 46-50 [PMID: 17320866 DOI: 10.1016/j.jsurg.2006.08.001]
4. Cholouttias E, Papatheodoridis GV, Burroughs AK. Liver grafts from anti-hepatitis B core positive donors: a systematic review. J Hepatol 2010; 52: 272-279 [PMID: 20034693 DOI: 10.1016/j.jhep.2009.11.009]
5. Tanabe M, Shimazu M, Wakabayashi G, Hoshino K, Kawachi S, Kadomura T, Seki H, Morikawa Y, Kitajima M. Intraportal
infusion therapy as a novel approach to adult ABO-incompatible liver transplantation. *Transplantation* 2002; 73: 1959-1961 [PMID: 12131697]

6. Abe M, Wakahagi S, Obara H, Shinoda M, Ito, H. Kitagawa W, Nakabayashi G, Shimazu M, Kitajima M. Current progress in ABO-incompatible liver transplantation. *Eur J Clin Invest* 2010; 40: 943-949 [PMID: 20636381 DOI: 10.1111/j.1365-2362.2010.02339.x]

7. Umemura A, Nitta H, Sasaki A, Takahara T, Hasegawa Y, Wakabayashi G. ABO-Incompatible Living Donor Liver Transplantation from Hepatitis B Core Antibody Positive Donor to Hepatitis C Liver Cirrhosis Recipient: A Case Report. *Case Rep Transplant* 2014; 2014: 507621 [PMID: 25045572 DOI: 10.1155/2014/507621]

8. Murakami H, Goto M, Ono E, Sawa E, Iwata M, Okuzumi K, Yamaguchi K, Takahashi T. Isolation of Helicobacter cinaedi from blood of an immunocompromised patient in Japan. *J Infect Chemother* 2003; 9: 344-347 [PMID: 14691657 DOI: 10.1007/s10156-003-0265-3]

9. Quinn TC, Goodell SE, Fennell C, Wang SP, Schuffer MD, Holmes KK, Stamm WE. Infections with Campylobacter jejuni and Campylobacter-like organisms in homosexual men. *Ann Intern Med* 1984; 101: 187-192 [PMID: 6547580]

10. Kiehlbauch JA, Tauxe RV, Baker CN, Wachsmuth IK. Helicobacter cinaedi-associated bacteremia and cellulitis in immunocompromised patients. *Ann Intern Med* 1994; 121: 90-93 [PMID: 8017741]

11. Burman WJ, Cohn DL, Reves RR, Wilson ML. Multifocal cellulitis and monocarticular arthritis as manifestations of Helicobacter cinaedi bacteremia. *Clin Infect Dis* 1995; 20: 564-570 [PMID: 7756476]

12. Totten PA, Fennell CL, Tenover FC, Wezenberg JM, Perine PL, Stamm WE, Holmes KK. Campylobacter cinaedi (sp. nov.) and Campylobacter fennelliae (sp. nov.): two new Campylobacter species associated with enteric disease in homosexual men. *J Infect Dis* 1985; 151: 131-139 [PMID: 3965584]

13. Grayson ML, Tee W, Dwyer B. Gastroenteritis associated with Campylobacter cinaedi. *Med J Aust* 1989; 150: 214-215 [PMID: 2716603]

14. Orlick SL, Welch DF, Kuhls TL. Septicemia and meningitis caused by Helicobacter cinaedi in a neonate. *J Clin Microbiol* 1993; 31: 569-571 [PMID: 8458951]

15. van der Ven AJ, Dellinger EA, Cohn DL, Reves RR, Wilson ML. Multifocal cellulitis and monocarticular arthritis as manifestations of Helicobacter cinaedi bacteremia. *Clin Infect Dis* 1995; 20: 564-570 [PMID: 7756476]

16. Matsumoto T, Goto M, Murakami H, Tanaka T, Nishiyama H, Ono E, Okada C, Sawa E, Yagoshi M, Yoneyama A, Okuzumi K, Tateda K, Misawa N, Yamaguchi K. Multicenter study to evaluate clinical characteristics of bacteremia caused by Helicobacter cinaedi (H. cinaedi). *Kansenshogaku Zasshi* 2005; 69: 859-866 [PMID: 16051580]

17. Minauchi K, Takahashi S, Sakai T, Kondo M, Shihayama K, Arakawa Y, Mukai M. The nosocomial transmission of Helicobacter cinaedi infections in immunocompromised patients. *Intern Med* 2010; 49: 1733-1739 [PMID: 20720350 DOI: 10.2169/internalmedicine.49.3649]

18. Holst H, Andresen K, Blom H, Hjøllyng T, Ingemarsson K, Christensen JJ. A Case of Helicobacter cinaedi Bacteremia in a Previously Healthy Person with Cellulitis. *Open Microbiol J* 2008; 2: 29-31 [PMID: 19088906 DOI: 10.2174/1874825800802010029]

19. Pescevitz MD. Rituximab, an anti-cd20 monoclonal antibody: history and mechanism of action. *Am J Transplant* 2006; 6: 859-866 [PMID: 16611321 DOI: 10.1111/j.1660-1641.2006.01288.x]

20. Kamikawa Y. Clinical and bacteriological examination in hospital of Helicobacter cinaedi (H. cinaedi). *Kansenshogaku Zasshi* 2014; 88: 417-422 [PMID: 25199374]

21. Arakawa Y. Recurrence of bacteremia caused by Helicobacter cinaedi in an immunocompetent patient. *J Infect Chemother* 2014; 20: 732-734 [PMID: 25131293]

22. Kikuchi H, Asako K, Tansho S, Ueda T, Koshio O, Ubagai T, Asahara M, Kawahara S, Ono Y. Recurrent Helicobacter cinaedi bacteremia and cellulitis in a patient with systemic lupus erythematosus. *Intern Med* 2012; 51: 3185-3188 [PMID: 23154730]

23. Kim SK, Cho EJ, Sung H, An P, Park SJ, Kim MN, Nam GB. A case of Helicobacter cinaedi bacteremia in an asplenic patient. *Ann Lab Med* 2012; 32: 433-437 [PMID: 23130344 DOI: 10.3343/alm.2012.32.6.433]

24. Ishizawa J, Mori T, Tsukada Y, Matsuji E, Yokoyama K, Shimizu T, Sugita K, Murata M, Iwata S, Okamoto S. Recurrent cellulitis due to Helicobacter cinaedi after chemotherapy for malignant lymphoma. *Rinsho Ketsueki* 2012; 53: 623-627 [PMID: 22790638]

25. Nishine H, Kasi S, Yoshikawa M, Otsuka Y, Tokuda H. A case of recurrent Helicobacter cinaedi-associated bacteremia in a small cell lung cancer patient during chemotherapy. *Nihon Kokyuki Gakkai Zasshi* 2007; 45: 26-30 [PMID: 17313023]

26. Ugkay I, Garbino J, Dietrich PY, Ninet B, Rohner P, Jacomo V. Recurrent bacteremia with Helicobacter cinaedi: case report and review of the literature. *BMC Infect Dis* 2006; 6: 86 [PMID: 16719920]

27. Van Gendern PJ, Goessens WH, Petit PL. Helicobacter cinaedi-associated bacteremia and erysipelas in an immunocompetent host: a diagnostic challenge. *Scand J Infect Dis* 2005; 37: 382-385 [PMID: 16051580]

28. Simons E, Spacke LA, Lederman HM, Winkelstein JA. Helicobacter cinaedi bacteremia presenting as macules in an afebrile patient with X-linked agammaglobulinemia. *Infection* 2004; 32: 367-368 [PMID: 15597229]

29. Lasry S, Simon J, Marais A, Pouchot J, Vincencieux P, Boussougant Y. Helicobacter cinaedi septic arthritis and bacteremia in an immunocompetent patient. *Clin Infect Dis* 2005; 31: 201-202 [PMID: 10913427]

30. Hung CC, Hsueh PR, Chen MY, Teng LJ, Chen YC, Luh KT, Chung CY. Bacteremia caused by Helicobacter cinaedi in an AIDS patients. *J Formos Med Assoc* 1997; 96: 558-560 [PMID: 9262063]

31. Sullivan AK, Nelson MR, Walsh J, Gazzard BG. Recurrent Helicobacter cinaedi cellulitis and bacteremia in a patient with HIV infection. *Int J STD AIDS* 1997; 8: 59-60 [PMID: 9043985]

32. Tee W, Street AC, Spelman D, Munckhof W, Mijch A. Helicobacter cinaedi bacteremia: varied clinical manifestations in three homosexual males. *Scand J Infect Dis* 1996; 28: 199-203 [PMID: 8792493]

33. Mammen MP, Aronson NE, Edenden WJ, Endy TP. Recurrent Helicobacter cinaedi bacteremia in a patient infected with human immunodeficiency virus: case report. *Clin Infect Dis* 1995; 21: 1055 [PMID: 8645814]

*P- Reviewer:* Inomata Y, Ito Y  *S- Editor:* Yu J  *L- Editor:* A  *E- Editor:* Wang CH
