restricted to plasma for fractionation. Some protection against blood donations from HEV-infected persons may occur because HEV and malaria are coendemic to many countries. Our findings showed a higher HEV seroprevalence among donors with prior malaria or diarrhea deferrals; thus, malaria- and diarrhea-related screening questions may reduce contributions from donors with travel-associated HEV infection.

Our findings showed HEV exposure in travelers and nontravelers, suggesting the possibility of imported and locally acquired HEV in Australia. Prior HEV exposure was higher in donors who were temporarily excluded from donating blood on previous donation attempts, suggesting the current management strategy in Australia is partially effective in minimizing any risk of HEV transmission through blood transfusion. However, the presence of HEV IgG in donors who reported no overseas travel and/or no prior related deferrals, coupled with the knowledge that asymptomatic infection is possible, suggests that additional safety precautions may be warranted.

Acknowledgments

We thank Australian Red Cross Blood Service staff in Donor Services and Manufacturing, especially A. Fadel, L. Lycett, B. Fisher, and R. Rodda. We also thank L. Danzig and J. Linnen for assistance with transcription-mediated amplification assay testing; and J. Fryk, P. Kiely, and H. Yang for technical assistance.

The Australian government fully funds the Australian Red Cross Blood Service for the provision of blood products and services to the Australian community. This study was conducted under approval from the Blood Service Human Research Ethics Committee.

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DOI: http://dx.doi.org/10.3201/eid2011.140412

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Helicobacter cinaedi Infection of Abdominal Aortic Aneurysm, Japan

To the Editor: Infected abdominal aortic aneurysm (IAAA) is uncommon, but life-threatening; the mortality rate ranges from 25% to 30% (*1*,*2*). Identification of the pathogen is essential for diagnosis and treatment. Previous studies have shown that species of the genera *Salmonella*, *Staphylococcus*, and *Streptococcus* are the most common pathogens associated with IAAA, but a causative organism is not identified in 14%–40% of patients (*1*,*2*). *Helicobacter cinaedi* has mainly been isolated from immunocompromised patients with bacteremia, cellulitis, and septic arthritis (*3*,*4*). Here, we report 3 cases of IAAA caused by *H. cinaedi* detected by 16S ribosomal RNA (16S rRNA) gene analysis.

The 3 patients (case-patients 1–3) were referred to Tohoku University

Hospital, Sendai, Japan, for surgical treatment of IAAA in 2013. None had a history of disease known to cause immunodeficiency. Because their abdominal aneurysms enlarged rapidly, all 3 patients underwent resection of the aneurysm and extensive local debridement and irrigation. Histopathologic examination of the surgical specimens revealed severe atherosclerosis and inflammation, consistent with a diagnosis of IAAA. For each case-patient, blood culture (BacT/ALERT; bioMérieux Industry, Tokyo, Japan) was negative, as was culture of surgically removed tissue on HK semisolid agar (Kyokuto Pharmaceutical Industrial Co., Ltd., Tokyo, Japan) at 35°C under aerobic conditions for 7 days for enrichment of microorganisms, and on chocolate agar at 35°C under 5% CO_2 for 48 h. We then used 16S rRNA gene analysis to identify a pathogen. We extracted DNA from resected tissues using a QIAamp DNA Mini kit (QIAGEN K.K., Tokyo, Japan), amplified it using PCR, and sequenced it using universal primers for 16S rRNA (*5*). We used the EzTaxon-e Database for sequence analysis (http://eztaxon-e.ezbiocloud.net/), which revealed that the 16S rRNA gene sequence of bacteria in the aneurysmal tissues was identical to that of *H. cinaedi*.

For case-patient 3, we cultured microaerophilic tissue at 35°C using Trypticase Soy Agar II with 5% sheep blood (Kyokuto Pharmaceutical Industrial Co.) and an Anaero Pouch-MicroAero (Mitsubishi Gas Chemical Co., Inc., Tokyo, Japan) to detect *H. cinaedi*. We observed bacterial colonies, after Gram staining, which showed gram-negative spiral rods. By 16S rRNA gene analysis, we confirmed that the isolate was *H. cinaedi*.

For each of the 3 case-patients, species identification was further confirmed by sequence analysis of 23S ribosomal RNA (23S rRNA) (DNA Data Bank of Japan: http://blast.ddbj.nig.ac.jp/blastn?lang = ja) and amplification of the *gyrB* gene region that is specific to *H. cinaedi* (*6*,*7*). In samples from the 3 patients, there were mutations of the 23S rRNA gene and amino acid substitutions in GyrA related to macrolide and fluoroquinolone resistance, respectively (*6*,*8*). After identifying the pathogen, we selected antimicrobial agents based on the reported drug susceptibility profile of *H. cinaedi* (*6*,*8*). The patients survived and are being followed up as outpatients. Clinical and molecular characteristics of the 3 cases of IAAA with *H. cinaedi* infection are shown in the Table.

Although the high negative culture rate for pathogens causing IAAA had been explained by prolonged preoperative antimicrobial drug therapy (*2*), another possibility is that *H. cinaedi* may be a causative organism. Earlier research has suggested that *H. cinaedi* infections can remain undiagnosed or be incorrectly diagnosed because of difficulty in isolating this microorganism (*9*). *H. cinaedi* grows slowly under microaerophilic conditions, but no current standard laboratory methods result in a diagnosis of this pathogen (*6*,*7*,*9*). We isolated *H. cinaedi* from surgically removed tissue from case-patient 3 by microaerophilic culture after taking this pathogen into consideration. For diagnosis of *H. cinaedi* infections, methods leading to accurate identification by clinical microbiological laboratories are needed. Currently, *H. cinaedi* is identified by molecular analysis of the 16S rRNA gene (*6*,*7*,*10*). In addition, matrix-assisted laser desorption/ionization–time-of-flight mass spectrometry (MALDI-TOF MS) (*10*), may become a useful tool for this purpose.

Standard breakpoints of antimicrobial drugs for *H. cinaedi* have not been defined, but all isolates in this study had mutations that indicated resistance to macrolides and fluoroquinolones. For adequate treatment for *H. cinaedi* infections, guidelines for selection of antimicrobial drugs and surveillance of its antimicrobial susceptibility profile are required.

During November 2012–November 2013, 8 patients underwent their first operation for IAAA at the university hospital. We used 16S rRNA gene analysis of surgical tissues and culture of blood and tissue specimens to detect pathogens (data not shown). Identification of *H. cinaedi* in 3 of 8 patients suggests that it could be a prevalent pathogen related to IAAA. Taking such information into consideration could affect the prognosis of many patients. Accordingly, tissue should be cultured while considering *H. cinaedi* infection in patients with IAAA. *H. cinaedi* colonizes the gastrointestinal tract, and bacterial translocation may lead to bacteremia associated with mucosal damage (*4*). However, the route of transmission and reason most *H. cinaedi* infections have been reported in Japan are unclear. To clarify the relationship between *H. cinaedi* and IAAA, further clinical and epidemiologic studies are needed. Meanwhile, we recommend clinical consideration of *H. cinaedi* infection, use of appropriate laboratory procedures to identify cases, and development of treatment guidelines.

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DOI: http://dx.doi.org/10.3201/eid2011.140440

Table. Clinical characteristics of 3 patients with *Helicobacter cinaedi* infected abdominal aortic aneurysms and molecular characteristics of isolates, Japan*

Characteristic	Case-patient 1	Case-patient 2	Case-patient 3
Age, y/sex	64/M	59/M	62/M
Underlying diseases	Hypertension, hyperlipidemia	None	History of myocardial infarction
Risk factors for infection	None	None	None
Clinical signs and symptoms before surgery	Fever, back pain	Fever, abdominal pain	Low back pain
CT results			
Site of aneurysm	Infrarenal abdominal, bilateral common iliac, internal iliac, L femoral, aortic arch†	Infrarenal abdominal, bilateral common iliac	Infrarenal abdominal
Inflammatory findings around aneurysms	+	+	+
Maximum leukocyte count/μL)/C-reactive protein, mg/dL before operation	10,600/25.3	9,100/6.05	7,050/ 5.29
Surgical management	In situ grafting	In situ grafting	In situ grafting
Microbiological diagnosis			
Blood culture	–	–	–
Tissue culture	–	–	+‡
rRNA gene sequence similarity, %§			
16S	99.8	99.6	99.6
23S	99.8	99.8	99.8
Amplification of *gyrB* specific to *H. cinaedi*	+	+	+
Aneurysms in which *H. cinaedi* was identified	Infrarenal abdominal, L common iliac, R internal iliac, L femoral	Infrarenal abdominal	Infrarenal abdominal
MLST	ST15 (CC7)	ST10 (CC9)	ST10 (CC9)
Mutation of 23S rRNA gene and amino acid substitutions in GyrA	2018 A→G and T84I D88G	2018 A→G and T84I	2018 A→G and T84I
Antimicrobial therapy dosage and duration			
Before admission	Ceftriaxone, 2 g/d, and levofloxacin, 500 mg/d, for 2 d	Piperacillin/tazobactam, 4.5 g/d for 12 d; faropenem sodium hydrate, 600 mg/d for 10 d	Oral antimicrobial agent, 4 d
After admission	Doripenem, 1.5 g/d for 22 d, and vancomycin, 3.0 g/d, for 14 d	Piperacillin/tazobactam, 4.5 g/d for 28 d	Doripenem, 1.5 g/d for 28 d
After identification of pathogen	Sulbactam/ampicillin, 3.0 g/d, and minocycline, 100 mg/d for 25 d	Continuation of piperacillin/tazobactam	Continuation of doripenem
At discharge	Oral amoxicillin, 1,500 mg/d, and minocycline, 200 mg/d, until follow-up visit	Oral amoxicillin, 1,500 mg/d, and minocycline, 200 mg/d, until follow-up visit	Oral amoxicillin, 1,500 mg/d, and minocycline, 200 mg/d, until follow-up visit
Postoperative complications	None	None	None
Outcome	Survived	Survived	Survived

*CT, computed tomography; +, positive; –, negative; L, left; R, right; MLST, multilocus sequence typing; ST, sequence type; CC, clonal complex; A, adenine; G, guanine; T, threonine; I, isoleucine; D, aspartic acid; G, glycine.
†Aortic arch was replaced 5 weeks after the abdominal operation.
‡Species unidentifiable under microaerophilic conditions.
§Compared with the type strain of *H. cinaedi* (CCUG 18818).

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Foodborne Transmission of Hepatitis E Virus from Raw Pork Liver Sausage, France

To the Editor: The number of sporadic autochthonous cases of acute hepatitis E is increasing in many industrialized countries (*1*). These cases involve hepatitis E virus (HEV) genotypes 3 and 4, which are zoonotic. Although risk for foodborne transmission from pork is now recognized, we report here direct HEV transmission through ingestion of raw pig liver sausages (figatellu [plural: figatelli]) in southeastern France.

The index case-patient was a 45-year-old woman from Hyères (southeastern France) who had no underlying medical condition. She visited her general practitioner on December 17, 2013, reporting 3 days of weakness. Acute hepatitis was diagnosed 2 days later on the basis of elevated liver enzymes (alanine aminotransferase 1,265 IU/L [reference <35 IU/L]) and bilirubin (65 μmol/L [reference <17 μmol/L]). Serum markers for acute hepatitis A, B, and C; cytomegalovirus; and Epstein-Barr virus were negative. Jaundice appeared on December 19, and the patient was referred to the Medical Unit of Hyères for additional investigations. A serum sample collected on December 20 tested positive for HEV RNA; viral load was 3.3 $\log_{10}$ IU/mL (Ceeram, La Chapelle sur Erdre, France), and IgM and IgG against HEV were found (Wantai, Beijing, China), which led to the diagnosis of acute hepatitis E. The HEV genotype was 3f, as determined from the phylogenetic analysis of a portion of the open reading frame (ORF) 2 (*2*). The index case-patient recovered by the end of January; HEV viremia was undetectable on January 17, 2014.

The index case-patient and her family regularly ate figatelli (raw pork liver sausages) made in Corsica. The patient had most recently eaten figatelli at a lunch with 8 family members on October 28, 2013, seven weeks before illness onset. After receiving informed consent, we conducted laboratory investigations of samples from the other family members; tests included HEV serology and HEV RNA detection in serum and fecal samples. Samples were obtained from family members during January 8–21, 2014 (41–54 days after the lunch). Positive HEV IgM and detectable HEV RNA were found in the serum of the index case-patient's daughter, who was asymptomatic. Because the sample was tested 10 weeks after the family lunch, the daughter's HEV viral load was too low to enable sequence characterization and clustering of HEV strains. Three other family members were IgG positive for HEV, indicating previous HEV infection. Leftover sausages had been kept frozen and were available for HEV testing.

HEV RNA was detectable from the leftover sausages, and HEV sequences were amplified in 2 different genomic regions (ORF1: RNA-dependent RNA polymerase and ORF2), as described previously (*2*). Comparison with the index case-patient's sequences showed 100% nt identity for both regions (Figure). Samples of food and samples from the index case-patient were analyzed in 2 independent laboratories to avoid any cross-contamination. The level of contamination of the figatellu was ≈4.8 10^4 copies of HEV RNA/g of sausage (*3*).

Figatellu, a dried sausage, contains 30% pork liver and no heating step occurs during its manufacture. Usually deep cooking is recommended on the package, but consumers might not follow the cooking recommendation; also, figatelli can be sold in small local shops with no label. In the instance reported here, the figatellu was sold without any warning label and was eaten raw.

That HEV was transmitted through ingestion of contaminated food is supported by the following evidence. First, 3 case reports have provided direct evidence of HEV transmission through ingestion of contaminated animal food products with identical or near identical sequences between the patients and the contaminated food they ate. Two cases occurred in the early 2000s in Japan through consumption of grilled wild boar (*4*) or sashimi of Sika deer (*5*); the third, reported recently in Spain, was transmitted through ingestion of pig meat (*6*). Second, HEV widely infects domestic pigs and wild boar (*7*). Third, swine and human HEV strains have genetic similarities and, in