INTRODUCTION

The species Vibrio cholerae is Gram-negative bacilli and has been classified according to the carbohydrate determinants of its somatic O antigens (1). Approximately 200 serotypes have been defined and are classified broadly into two types: those agglutinate in antisera to the O1 group antigen (O1 V. cholerae) and those do not agglutinate in antisera to the O1 group antigen (non-O1 V. cholerae) (2). Infections caused by V. cholerae of serogroups other than O1 or O139 usually manifest with sporadic diarrhea; however, in immunocompromised patients such as those with liver disease, renal failure, or hematologic malignancy, the infection can cause severe extraintestinal diseases such as wound infection and sepsis (3-6). Here we report, the first case of V. cholerae infection associated with pleural effusion in a long-term latent carrier state.

CASE REPORT

The patient was a 62-yr-old man who had undergone curative subtotal gastrectomy for gastric cancer 14 yr ago. He presented himself with signs of cachexia and complained of heartburn and epigastric discomfort from stomach cancer before admission. The suspected route of infection is directly from the gastrointestinal tract through the previous surgical wounds. After antibiotic treatment, no more V. cholerae was isolated and the patient was well discharged from the hospital. This is the first report of V. cholerae infection associated with pleural effusion in a long-term latent carrier of the organism.

Vibrio cholerae non-O1, non-O139 Isolated from Pleural Effusion Following Total Gastrectomy

We isolated non-O1, non-O139 Vibrio cholerae from pleural effusion in a patient with recurred advanced gastric cancer after total gastrectomy. We also recovered the organism from the patient’s stool culture. The patient did not experience gastrointestinal symptoms such as diarrhea except heartburn and epigastric discomfort from stomach cancer before admission. The suspected route of infection is directly from the gastrointestinal tract through the previous surgical wounds. After antibiotic treatment, no more V. cholerae was isolated and the patient was well discharged from the hospital. This is the first report of V. cholerae infection associated with pleural effusion in a long-term latent carrier state.

Key Words: Vibrio cholerae; Pleural Effusion; Gastrectomy; Carrier State

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Vibrio cholerae non-O1, non-O139 in Pleural Effusion

during exploration, both VRE and V. cholerae were recovered. The patient was treated with linezolid, imipenem and levofloxacin. VRE and V. cholerae were also isolated in the patient’s stool. No more pathogens were isolated on follow-up cultures of pleural effusion, rectal swab and stool, and the laboratory findings were normalized.

V. cholerae isolated from the pleural effusion and stool formed white beta-hemolytic colonies on sheep blood agar and yellow colonies on thiosulfate-citrate-bile sucrose agar. It was identified as V. cholerae by the MicroScan Gram-negative Combo panel (Dade International Inc., California, U.S.A.) and API 20E (bioMerieux, France). It was susceptible to amikacin, cefazidime, ceftiraxone, cefalothin, ciprofloxacin, gentamicin, imipenem, piperacillin, tobramycin, trimethoprim-sulfamethoxazole and cefepime. The PCR for the cholera toxin gene was performed with primers, 5′-ACAGAGTGAGTAGAACGATTGAC-3′ and 5′-ATACCATCCATATATTGAGGAG-3′, which did not yield a PCR product at 307 bp. By agglutination test for serogrouping, the isolate was finally confirmed as a non-cholera toxin-producing, non-O1, non-O139 V. cholerae.

**DISCUSSION**

The majority of infections by V. cholerae are associated with the intake of contaminated food. However, V. cholerae including O1 and O139, which requires salt for growth, is a normal flora of water, and enters into a dormant, viable but non-culturable stage when conditions are unfavorable. The sporadic and erratic occurrence of cholera epidemics has been associated with certain conditions that increase proliferation of plankton, such as flood, El Niño (7).

The stomach acidity is the main barrier against the infection of V. cholerae. The use of antacids, histamine receptor blockers, and proton pump inhibitors increases the risk of cholera and predisposes patients to a more severe disease because of reduced gastric acidity (8), and this is also the case in patients with chronic gastritis secondary to *Helicobacter pylori* infection and those who have undergone gastrectomy. Although there was a report of a carrier of V. cholerae in the gallbladder up to 12 yr (9), a long-term carrier of V. cholerae is extremely rare, compared to those of *Salmonella typhi*.

The present patient did not develop gastrointestinal symptoms, except heartburn and epigastric discomfort due to stomach cancer before admission. Therefore, it could be more probable for the bacteria to have been residing in the gallbladder or intestinal tract rather than recent infection. It was supported by the fact that it took one month to yield V. cholerae after total gastrectomy during the present admission. Considering the fact that rectal swab and stool cultures yielded the same bacteria as well as VRE and that there was no apparent history of intake of suspicious food, the patient was thought to be a long-term carrier. The previous subtotal gastrectomy and antacid drugs might have increased the susceptibility to V. cholerae. Although we could not identify the route of infection associated with pleural effusion, it was presumed that V. cholerae in multiple abscesses in the left upper abdomen was translocated to the pleural cavity during the surgical treatment for hemothorax.

Based on in vitro susceptibility tests, V. cholerae (non-O1) strains are generally susceptible to most of antimicrobial agents (10). Although the treatment of choice for V. cholerae is tetracycline, the patient was successfully treated with linezolid, imipenem and levofloxacin. The present case represents the first case of V. cholerae infection associated with pleural effusion in a long-term carrier of the organism.

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**REFERENCES**

1. Heiden D. Cholera. West J Med 2000; 173: 288.
2. Murray PR. Vibrio. In: Murray PR, Baron EJ, eds. Manual of clinical microbiology. 7th ed. Washington D.C.: American Society for Microbiology 1999; 497-505.
3. Bhattacharya MK, Dutta D, Bhattacharya SK, Deb A, Makhopadhyay AK, Nair GB, Shimada T, Takeda Y, Chowdhury A, Mahalanabis D. Association of a disease approximating cholera caused by *Vibrio cholerae* of serogroups other than O1 and O139. Epidemiol Infect 1998; 120: 1-5.
4. Ko WC, Chuang YC, Huang GC, Hsu SY. Infections due to non-O1 Vibrio cholerae in southern Taiwan: predominance in cirrhotic patients. Clin Infect Dis 1998; 27: 774-80.
5. Lee HK, Shin OR, Lee DG, Chae HS, Kim JT, Kang CS. A case of *Vibrio cholerae* non-O1/O139 gastroenteritis. Korean J Lab Med 2004; 24: 386-8.
6. Uhm JS, Oh SB, Lee SH, Kim SI, Kim YR, Park YJ, Kang MW. A case of skin and soft tissue infection caused by Non-O1, non-O139 *Vibrio cholerae* in a patient with liver cirrhosis. Infect Chemother 2005; 37: 104-6.
7. Colwell RR. Global climate and infectious disease: the cholera paradigm. Science 1996; 274: 2025-31.
8. Martinsen TC, Bergh K, Waldum HL. Gastric juice: a barrier against infectious diseases. Basic Clin Pharmacol Toxicol 2005; 96: 94-102.
9. Azarun JC, Kobari K, Barua D, Alvero M, Gomez CZ, Dixon JJ, Nakano EL, Suplido R, Ledesma L. A long-term carrier of *cholera*: a cholera Dolores. Bull World Health Org 1967; 37: 745-9.
10. Keretta JA, Paul AC, Kinibukaran VB, Jesudason MV, Moses PD. Non-01 Vibrio cho lerae septicemia and meningitis in a neonate. Indian J Pediatr 2002; 69: 909-10.