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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 caner 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 of the organism.

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

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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 type: 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, with a focus on the unusual route of infection and long-term latent *V. cholerae* 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 for approximately 1 month before admission. The endoscopic examination revealed a recurred gastric adenocarcinoma. A total gastrectomy and Roux-en-Y esophagojejunostomy were performed with dissection of the multiple adhesions around the small sac and between the transeverse colon and liver.

Four days after the operation, the patient developed fever. He also showed consolidation in the left lower chest and increasing pleural effusion on chest radiography. Thoracostomy was performed. Because *Klebsiella pneumoniae* and *Candida albicans* grew in the cultures of sputum and pleural effusion, antibiotics treatment including intravenous cefepime and clindamycin was initiated. Although the regimen was changed to meropenem, the empyema on the left side did not improve, and decortication of the left lung was performed 30 days after the operation. After decortication, no more microorganisms were isolated from pleural effusion. However, methicilin-resistant coagulase-negative staphylococci was recovered from the exudates of the tube insertion site. Despite the vancomycin treatment, the wound did not improve, and marginal resection of the tube insertion site was performed 15 days after decortication. After marginal resection, vancomycin-resistant enterococci (VRE) was recovered in the culture of exudates from the wound site. The abdominal computed tomography showed multiple abscesses in the left upper abdomen at that time. Two days after the marginal resection, the patient underwent re-exploration of the left thoracic cavity due to a hemothorax from intercostal arterial bleeding at the resection site. The laboratory findings showed white blood cell count of 19,100/μL with a neutrophil predominance (85%), increased ESR (72 mm/hr) and CRP (28.34 mg/dL). In the culture of blood and exudates from the pleural cavity obtained

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., Califonia, U.S.A.) and API 20E (bioMerieux, France). It was susceptible to amikacin, ceftazidime, ceftriaxone, cephalothin, ciprofloxacin, gentamicin, imipenem, piperacillin, tobramycin, trimethoprim-sulfamethoxazole and cefepime. The PCR for the cholera toxin gene was performed with primers, 5′-ACAGAGTGAGTACTTTGACC-3′ and 5′-ATACCATCCATATATTTGGGAG-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 nonculturable 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 Ñiño (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 resided 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 suctional 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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