JOURNAL OF CLINICAL MICROBIOLOGY, Mar. 2010, p. 1002–1004 Vol. 48, No. 3
0095-1137/10/$12.00 doi:10.1128/JCM.02198-09

CASE REPORTS

Acute Cholecystitis Caused by Nontoxigenic *Vibrio cholerae* O1 Inaba[▽]

Adam P. Vogt,[1] Rupali K. Doshi,[2] Judy E. Higgins,[1] Eileen M. Burd,[1] Bruce S. Ribner,[2] and Colleen S. Kraft[1,2]*

Department of Pathology and Laboratory Medicine, Emory University, Atlanta, Georgia,[1] and Division of Infectious Diseases, Emory University, Atlanta, Georgia[2]

Received 10 November 2009/Returned for modification 23 December 2009/Accepted 28 December 2009

A rare case of acute cholecystitis caused by serogroup O1 *Vibrio cholerae* in an 83-year-old man is presented. His risk factors for cholecystitis included advanced age and previous abdominal surgeries. The patient had consumed raw oysters several days before presentation. The patient had a poor outcome after admission for this infection, likely due to his underlying illnesses that complicated his hospital course.

CASE REPORT

In October 2009, an 83-year-old man presented to the emergency department at our academic tertiary referral center with severe right upper quadrant pain. The pain began 4 h after routine dialysis treatment, was episodic, and had increased in intensity without radiation. Past medical history was significant for chronic kidney disease that necessitated dialysis due to glomerulonephritis, chronic obstructive pulmonary disease, congestive heart failure, peripheral vascular disease, hypertension, and hyperlipidemia. His past surgical history was significant for a total colectomy with resultant ileostomy, left carotid endarterectomy, and aortic aneurysm repair.

On presentation, the patient denied fever, chills, nausea, and vomiting but noted an increase in ileostomy output with what was described as a "Rice Krispies" consistency. Laboratory data were as follows: white blood cell count, 10,800 cells/μl; hemoglobin, 11.3 g/dl; glucose, 82 mg/dl; creatinine, 4 mg/dl; serum sodium, 142 mmol/liter; and potassium, 4.3 mmol/liter. An electrocardiogram was performed, producing results that were unchanged from those seen previously, and the troponin-I level was within normal limits. An ultrasonogram revealed a sonographic Murphy's sign and pericholecystic fluid, which is consistent with the presence of acute acalculous cholecystitis (Fig. 1).

With his multiple comorbidities, the patient was not considered a surgical candidate for cholecystectomy. A computed tomatography-guided percutaneous cholecystostomy was performed, and 150 ml of clear green fluid was aspirated; specimens of the fluid were sent for culture.

The Gram stain of the body fluid specimen demonstrated rare Gram-negative rods and many polymorphonuclear lymphocytes. The culture was prepared using sheep blood agar (SBA), chocolate agar, colistin-nalidixic acid agar, MacConkey agar, brucella agar with hemin and vitamin K (ABAP), and phenyl ethyl alcohol agar (PEA) and incubated in 5% CO_2 at 37°C and under anaerobic conditions for ABAP and PEA. There was heavy growth of a single organism on SBA and chocolate agar that was a nonlactose fermenter on MacConkey agar. ABAP grew the same organism as the aerobic plates, and PEA had no growth. A Gram stain from the colonies growing on SBA showed curved Gram-negative rods (Fig. 2). The organism was positive for oxidase, and results obtained using a Neg Breakpoint Combo Panel Type 41 (NBC41) and a MicroScan WalkAway Plus system (Siemens Healthcare Diagnostics, Deerfield, IL) identified the organism as *Vibrio cholerae*, with 97.76% probability. Since *V. cholerae* is rarely isolated in our laboratory, the isolate was also tested using a manual API 20E Gram-negative identification panel (bioMérieux, Inc., Durham, NC), which yielded a code of 5347124, giving a presumptive identification of *V. cholerae* at 99.9%. Thiosulfate citrate bile salt sucrose agar was inoculated and yielded yellow colonies (Fig. 3). The reaction on triple sugar iron agar produced alkaline over acid (K/A), and there was no hydrogen sulfide (H_2S) production. The sample was submitted to the Georgia Public Health laboratory, where the organism was classified as *V. cholerae*, O1 serogroup, via gas chromatography. The sample was identified at the Centers for Disease Control and Prevention (Atlanta, GA) as a member of the nontoxigenic *Vibrio cholerae* O1 serogroup, Inaba serotype. The patient's stool culture remained negative for *Vibrio* species. Repeat cultures from the gallbladder drain grew *Enterococcus faecium*. Susceptibility determinations were performed using a MicroScan WalkAway Plus system as described above; the organism was susceptible to ampicillin, tetracycline, and trimethoprim-sulfamethoxazole.

Further questioning revealed two instances where the patient had ingested baked oysters in an Oysters Rockefeller dish, as well as raw oysters at a local restaurant, 10 and 3 days prior to presentation, respectively. Each of the meals was shared with family and friends; however, only the pa-

* Corresponding author. Mailing address: Department of Pathology and Laboratory Medicine, Emory University Hospital, 1364 Clifton Rd., NE, Atlanta, GA 30322. Phone: (404) 712-0197. Fax: (404) 712-4632. E-mail: cskendr@emory.edu.

▽ Published ahead of print on 6 January 2010.

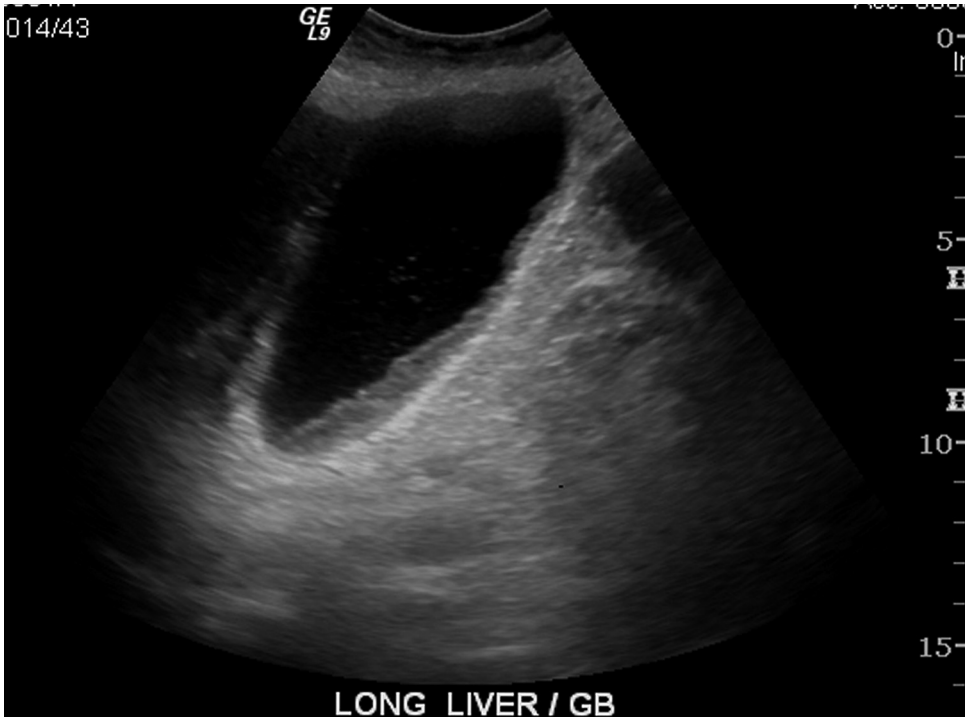

FIG. 1. Abdominal ultrasonogram showing the presence of pericholecystic fluid and absence of calculi, consistent with acalculous cholecystitis.

tient consumed oysters on both occasions and became symptomatic.

Discussion. Risk for acalculous cholecystitis is increased in patients who have a history of abdominal surgery (5). The most common causative organisms are Gram-negative enteric flora, in particular, *Escherichia coli*, *Enterobacter cloacae*, *Enterococcus* spp., and *Klebsiella pneumoniae*. Infection is frequently polymicrobial. Cases have also been associated with *Salmonella*, *Staphylococcus*, and *Brucella* species (7, 10). Consumption of raw oysters has been associated with acquisition of *Vibrio* species, in particular, *V. cholerae* and *V. parahaemolyticus*, with the latter affecting patients who consume uncooked seafood in various forms.

Initially, the patient clinically improved when treated with

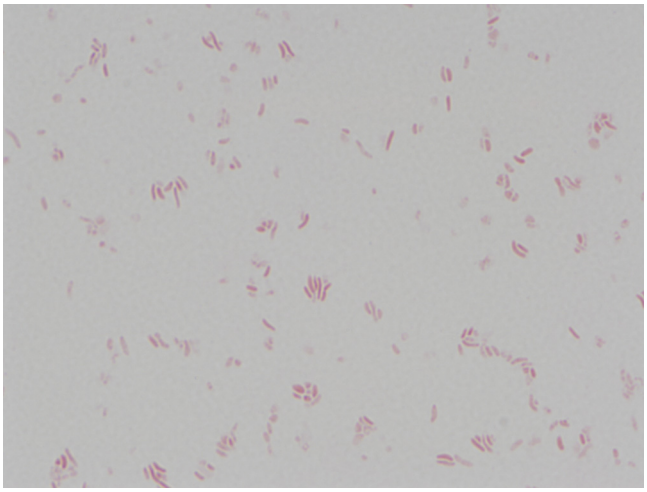

FIG. 2. A Gram stain of the biliary fluid isolate, showing slightly curved Gram-negative rods.

FIG. 3. Yellow colonies growing on thiosulfate citrate bile salt sucrose agar indicate fermentation of sucrose, which is characteristic of *V. cholerae* and *V. alginolyticus.* The isolate was identified biochemically as *V. cholerae*.

broad-spectrum antibiotics, namely, piperacillin-tazobactam, which was subsequently replaced by ceftriaxone. The patient's hospital course was complicated by rapid atrial fibrillation, transient hypotension, delirium, and the development of an ischemic limb, and the patient and his family opted for a palliative course of action, including eventual cessation of hemodialysis. He was discharged to a hospice 1 month after being admitted to the hospital.

Only 2 cases of *Vibrio cholerae* O1 serogroup causing cholecystitis have previously been reported in the literature. Both cases involved previously healthy individuals (2, 4). There have been other reports of individuals with *V. cholerae* cholecystitis (non-O1), with traditional risk factors (cholelithiasis, pregnancy) for cholecystitis (6). This is the first case reported involving a patient who has traditional risk factors for acalculous cholecystitis, including age (>50 years old) and previous abdominal surgeries (5).

Vibrio species are reportable infections under the guidelines of the Centers for Disease Control and Prevention Food-borne Diseases Active Surveillance Network (http://www.cdc.gov/FoodNet). The preliminary overall incidence of laboratory-confirmed *Vibrio* species in 2008 was reported to be 0.29 per 100,000 people in the United States (3). The only other reportable cause of bacterial infection as rare as *Vibrio* species was *Listeria*, also at an incidence of 0.29 per 100,000 persons (3). No cases of acute cholecystitis due to the *V. cholerae* O1 serogroup have been reported in the literature or surveillance data since 1996.

Not all *V. cholerae* isolates cause disease. In fact, the majority of serogroups are not pathogenic; in the O1 serogroup, nontoxigenic strains are more prevalent than toxigenic O1 strains (8). There are 3 serotypes of *V. cholerae* O1 serogroup (Ogawa, Inaba, and Hikojima) that are characterized by the presence of somatic antigens. They are all thought to present similar clinical manifestations of disease. Although the nonpathogenic strains comprise the majority of serogroups, they can become pathogenic due to horizontal transfer of virulence genes from bacteriophages or on pathogenicity islands. For example, in addition to the traditional cholera toxin-producing O1 and O139 serogroups, toxin-producing strains have been identified in O41, O75, and O141 serogroups in recent years (9).

In the present case, *V. cholerae* was correctly identified by both the MicroScan automated microbial identification system and the manual API identification panel. Rapid, accurate identification of the etiologic agent(s) is especially important for patients like this who are unable to tolerate cholecystectomy and rely on percutaneous drainage and antibiotic therapy for treatment. Susceptibilities to ampicillin, tetracyclines, folate pathway inhibitors, and chloramphenicol have interpretative guidelines through CLSI for the treatment of *V. cholerae* and should be reported (1).

Our case report demonstrates that despite the *V. cholerae* O1 serogroup being nontoxigenic, these strains can cause significant illness in the susceptible patient, and their accurate diagnosis for proper clinical management is crucial.

REFERENCES

1. **Abbot, S. L., J. M. Janda, J. A. Johnson, and J. J. Farmer.** 2007. *Vibrio* and related organisms, p. 723–733. *In* P. R. Murray, E. J. Barron, J. H. Jorgensen, M. L. Landry, and M. A. Pfaller (ed.), Manual of clinical microbiology, 9th ed. ASM Press, Washington, DC.
2. **Asnis, D. S., R. Golub, and A. Bresciani.** 1996. *Vibrio cholerae* O1 isolated in the gallbladder of a patient presenting with cholecystitis. Am. J. Gastroenterol. **91:**2241–2242.
3. **Centers for Disease Control and Prevention.** 2009. Preliminary FoodNet data on the incidence of infection with pathogens transmitted commonly through food—10 states, 2008. MMWR **58:**333–337.
4. **Gomez, N. A., C. J. Leon, and J. Gutierrez.** 1995. Acute cholecystitis due to *Vibrio cholerae*. Surg. Endosc. **9:**730–732.
5. **Kalliafas, S., D. W. Ziegler, L. Flancbaum, and P. S. Choban.** 1998. Acute acalculous cholecystitis: incidence, risk factors, diagnosis, and outcome. Am. Surg. **64:**471–475.
6. **Peterson, E. M., P. Jemison-Smith, L. M. de la Maza, and D. Miller.** 1982. Cholecystitis: its occurrence with cholelithiasis associated with a non-O1 *Vibrio cholerae*. Arch. Pathol. Lab. Med. **106:**300–301.
7. **Ryan, K. J., and C. G. Ray.** 2004. Sherris medical microbiology, 4th ed., McGraw-Hill, New York, NY.
8. **Seas, C., and E. Gotuzzo.** 2009. *Vibrio cholerae*, p. 2777–2785. *In* G. L. Mandell, J. E. Bennett, and R. Dolin (ed.), Mandell, Douglas and Bennett's principles and practice of infectious diseases, 7th ed. Churchill Livingstone, Philadelphia, PA.
9. **Tobin-D'Angelo, M., A. R. Smith, S. N. Bulens, S. Thomas, M. Hodel, H. Izumiya, M. Arakawa, M. Masatomo, H. Watanabe, C. Marin, M. B. Pars, K. Greene, K. Cooper, D. Haydel, B. Cheryl, P. Yu, and E. Mintz.** 2008. Severe diarrhea caused by cholera toxin-producing *Vibrio cholerae* serogroup O75 infections acquired in the southeastern United States. Clin. Infect. Dis. **47:**1035–1040.
10. **West, B. C., R. Silberman, and W. N. Otterson.** 1998. Acalculous cholecystitis and septicemia caused by non-O1 *Vibrio cholerae*: first reported case and review of biliary infections with *Vibrio cholerae*. Diagn. Microbiol. Infect. Dis. **30:**187–191.