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Vibrio cholerae Non-Serogroup O1 Cystitis

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We report a case of a patient who developed cystitis caused by non-serogroup O1 *Vibrio cholerae* after swimming in the Chesapeake Bay. Treatment was empirical, with complete symptomatic resolution. Genitourinary tract infections by *Vibrio* spp. are uncommon but should be considered when cystitis occurs after saltwater exposure in appropriate geographic regions.

Vibrio cholerae strains which do not agglutinate in O group 1 antiserum usually do not produce an enterotoxin and infrequently have been associated with human illness (1, 5, 8, 10). Non-serogroup O1 *V. cholerae* strains are gram-negative, curved bacilli which possess a single polar flagellum; they are halophilic, can ferment carbohydrates, and are oxidase positive. Outbreaks of human disease have been associated with ingestion of or exposure to contaminated foods or water (1, 5, 8, 10). Non-O1 *V. cholerae* strains are ubiquitous in the Chesapeake Bay and other coastal waters and may be isolated from water or sediment samples at densities which vary with water temperature. The organisms are found in samples within a narrow range of salinity (0.4 to 1.7%) present in the upper bay or in tributaries contributing fresh water. There is no association with sewage content or fecal coliform counts (3, 6). We report a case of cystitis caused by non-O1 *V. cholerae* and suggest that the spectrum of gram-negative organisms responsible for cystitis in geographic regions where exposure to *Vibrio* species is a possibility should include non-O1 *V. cholerae*.

A 21-year-old woman presented to the adult emergency room at the Johns Hopkins Hospital in Baltimore, Md., complaining of 7 h of dysuria, urgency, and frequency. She denied hematuria, flank pain, abdominal pain, nausea, vomiting, and fever. There had been no vaginal discharge or pruritis. She was sexually active and had engaged in sexual intercourse on the night prior to presentation. Her last menstrual period had occurred appropriately and was of normal duration. A physical examination revealed an oral temperature of 36.9°C, a blood pressure of 130/72, and a pulse of 82 which was regular. The abdomen was soft, with normoactive bowel sounds and minimal suprapubic tenderness only. There was no organomegaly or costovertebral angle tenderness. The leukocyte count was 13,300/mm^3, with a differential including 6% bands, 67% neutrophils, 23% lymphocytes, and 4% monocytes. A clean-catch, midstream urine specimen had a pH of 6.5, was cloudy yellow, and had 3+ protein and large quantities of heme; a microscopic examination revealed greater than 10 leukocytes and 3 to 5 erythrocytes per high-power field. A Gram stain of the urine revealed many leukocytes and otherwise unspecified gram-negative bacilli. A diagnosis of cystitis was made, and urine cultures were sent to the microbiology laboratory. She was treated with a single dose of trimethoprim-sulfamethoxazole (160 and 800 mg, respectively). A subsequent epidemiologic investigation revealed that she became asymptomatic and neglected a follow-up examination. Additionally, it was discovered that on the day prior to presentation, the patient had been swimming in an area of the Chesapeake Bay known to contain non-serogroup O1 *V. cholerae*.

In our laboratory, initial media routinely used for urine culturing include Trypticase soy agar (BBL Microbiology Systems, Cockeysville, Md.) with 5% sheep blood and MacConkey agar, and in this case the urine culture grew greater than 10^5 CFU of an oxidase-positive, gram-negative bacillus per ml which was initially identified by cellular fatty acid analysis (Fig. 1) (MIS; Microbial ID, Newark, Del.) as *V. mimicus* or *V. cholerae* (7). Identification as *V. cholerae* was confirmed by fermentation of glucose and sucrose, decarboxylation of lysine and ornithine, growth in both 0 and 1% NaCl nutrient broth, production of indole, and utilization of citrate as a sole carbon source. In addition, the isolate was negative for H_2S production, urea hydrolysis, phenylalanine deaminase, arginine dihydrolase, and fermentation of adonitol, arabinose, inositol, lactose, and sorbitol. Serotyping, performed by J. Glenn Morris (Center for Vaccine Development, University of Maryland School of Medicine, Baltimore), confirmed the organism as non-serogroup O1 *V. cholerae*. It was susceptible to gentamicin (≤1 μg/ml), tobramycin (≤1 μg/ml), ticarcillin (≤8 μg/ml), trimethoprim-sulfamethoxazole (≤0.4 and 8 μg/ml, respectively), ciprofloxacin (≤0.25 μg/ml), tetracycline (≤2 μg/ml), nitrofurantoin (≤32 μg/ml), chloramphenicol (8 μg/ml), cephalothin (8 μg/ml), and ampicillin (4 μg/ml) and resistant to sulfamethoxazole (≥128 μg/ml), as determined by the standard agar dilution technique.

The most common form of human disease caused by non-serogroup O1 *V. cholerae* is gastroenteritis clinically suggestive of an invasive rather than a toxin-associated pathogenesis, although cholera toxin production has been occasionally demonstrated (1, 5, 8, 10). Extraintestinal infections, including septicemia, cellulitis, meningitis, aspiration pneumonia, otitis media, and ascending cholangitis, have been reported (1, 5, 9; G. Prats, B. Mirelis, R. Pericas, and G. Verger, Letter, Ann. Intern. Med. **82**:842–849, 1975). The source of infection in these cases is not always well established but is frequently associated with exposure to salt water. Moreover, patients with a severe underlying illness are predisposed to severe infections and, on occasion, a fatal outcome (1, 5, 9). Although the recovery of non-O1 *V. cholerae* from urine has been documented, to our knowledge the clinical circumstances surrounding these cases have not been reported (4).

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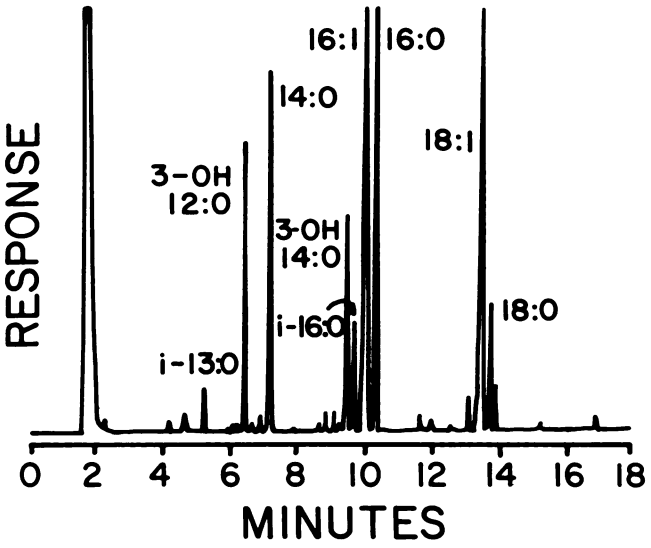

FIG. 1. Gas chromatogram of the esterified fatty acids of non-serogroup O1 *V. cholerae*. The number to the left of the colon refers to the number of carbon atoms; the number to the right refers to the number of double bonds; i indicates a methyl branch at the iso-carbon atom; 3-OH refers to a hydroxyl group at the carbon-3 atom.

Despite the fact that gram-negative organisms are frequent agents of cystitis, non-serogroup O1 *V. cholerae* strains have not been included in the spectrum of organisms capable of causing cystitis. The current report describes a well-documented case of cystitis caused by non-O1 *V. cholerae*. The ability of this organism to cause urinary tract infections is not surprising, since its invasiveness and ability to cause extraintestinal infections associated with saltwater exposure are documented (5, 9). A single previous report of *V. vulnificus* endometritis in a young woman who had engaged in sexual intercourse in waters known to harbor *V. vulnificus* has many similarities to the current case (11). Under these circumstances, inoculation of the genital and urinary tracts with unusual organisms, such as non-O1 *V. cholerae*, *V. vulnificus*, or other estuarine and marine bacterial inhabitants, can lead to infection (2). In our institution, when gram-negative bacilli are seen in urine Gram stains, single-dose trimethoprim-sulfamethoxazole therapy is commonly chosen because of frequent patient noncompliance with longer, more-expensive regimens and high efficacy. Although the isolate in this case was susceptible to the antibiotic used, it is conceivable that an infection occurring in a compromised host would require more intensive antimicrobial therapy with several agents, avoiding antimicrobial agents to which the organism is resistant, especially if a systemic infection occurs. Thus, although a careful examination of urine cultures on media sufficient to support the growth of *V. cholerae* (blood agar and MacConkey agar; *V. cholerae* grows poorly on eosin-methylene blue agar) usually will reveal any vibrios present and a careful history usually will reveal a recent source of infection, identification remains important because of the potentially disastrous effects of a systemic infection in debilitated and immunocompromised patients. These factors indicate the need for considering non-O1 *V. cholerae* as a causative agent of urinary tract infections in patients exposed to salt water.

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