**Vibrio cholerae** Bacteremia Associated with Gastrectomy

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Bacteremia due to *Vibrio cholerae* is rare. Each of 15 cases previously reported in the English language literature occurred in the setting of immune deficiency. We describe an instance of non-serogroup O1 *V. cholerae* septicemia in an otherwise healthy patient. Susceptibility to such infection may have been enhanced by a prior gastrectomy for duodenal ulcer.

Non-serogroup O1 *Vibrio cholerae* has been associated with a variety of gastrointestinal infections ranging from mild watery diarrhea to dysentery (4). The more virulent strains associated with fulminating watery diarrhea (cholera) are distinguished by in vitro agglutination by specific antisera (4). Although classical (agglutinating) strains are rarely associated with extraintestinal infection (5), non-O1 *V. cholerae* has been reported to cause bacteremia in immunocompromised hosts. We have recently encountered *V. cholerae* gastroenteritis and septicemia in an otherwise healthy patient who had previously undergone gastrectomy for a duodenal ulcer.

A 66-year-old male was hospitalized for fever, rigors, watery diarrhea, and diffuse abdominal pain of 4 days’ duration. Twenty years previously, he underwent a Billroth II gastrectomy for duodenal ulcer and a cholecystectomy for choledolithiasis but had experienced no intervening gastrointestinal illness. He had not traveled from Israel in recent years and denied exposure to well water, shellfish, or individuals with acute gastrointestinal symptomatology. The patient lives in a coastal city; 4 days before the onset of symptoms, he had been swimming in the Mediterranean Sea. No other family members were ill at the time of the patient’s illness.

On physical examination, the rectal temperature was 39.6°C and the pulse was 108/min. The abdomen was diffusely tender, with hyperactive bowel sounds. There was no lymphadenopathy or splenomegaly. Laboratory studies disclosed a blood hemoglobin concentration of 13.7 g/dl and a leukocyte count of 15,300/mm³, with 76% neutrophils, 2% band forms, 20% lymphocytes, 1% monocytes, and 1% eosinophils. Concentrations in serum of sodium, potassium, urea, alanine aminotransferase, alkaline phosphatase, bilirubin, and amylase were normal. Upper gastrointestinal barium and ultrasound studies were consistent with a prior Billroth II gastrectomy and cholecystectomy but were otherwise unrevealing.

Stool and blood specimens were submitted for culture on the day of admission, and a regimen of intravenous gentamicin (80 mg every 8 h) and ampicillin (1 g every 6 h) was initiated. Each of five blood specimens produced oxidase-positive, curved, motile, gram-negative bacilli following 24-h incubation in BACTEC 6B medium (Johnston Laboratories, Inc., Towson, Md.). Identical isolates were recovered from each of two stool specimens by picking oxidase-positive colonies from 5% human blood agar (tryptose blood agar base; Difco Laboratories, Detroit, Mich.). The organism failed to agglutinate with polyvalent *V. cholerae* O1 antisera (Israel Ministry of Health) but was identified as *V. cholerae* in both API (API International, SA, Montalieu-Vercieu, France) and Sensititre AP 70 (Sensititre Ltd., West Sussex, United Kingdom). When a standard disk diffusion technique was used, the isolate was susceptible to chloramphenicol, cephalothin, sulfamethoxazole-trimethoprim, and gentamicin and resistant to ampicillin and mezlocillin.

When the identity and antibiogram of the isolate were established, therapy was changed to intravenous chloramphenicol, 0.5 g every 6 h. The patient recovered without further incident and was discharged on hospital day 7.

Subsequent studies revealed normal concentrations of blood immunoglobulins and T-lymphocyte subsets (fluorescence-activated cell sorter; Beckman SA, Geneva, Switzerland). Intradermal reactions with streptokinase, streptodornase, and *Candida albicans* antigen were negative.

The identity of the organism was confirmed as non-serogroup O1 *V. cholerae*, serovar 23, by the laboratories of the Israel Health Ministry and by R. Sakazaki of the National Institutes of Health, Tokyo, Japan.

Autochthonous cholera has not been reported from Israel for 17 years, although tourists have occasionally returned from Egypt with the disease. Bacteremia due to non-serogroup O1 *V. cholerae* has not been previously encountered in the Middle East. Each of the 15 cases previously reported in the English language literature was associated with hepatic cirrhosis, leukemia, malnutrition, corticosteroid therapy, or other immune compromise (4, 8). The organism was recovered from both stool and blood specimens in only one previous case (8).

Hypochlorhydria is known to predispose to infection by *Salmonella* spp., *Shigella* spp., *Brucella* spp., *Escherichia coli*, and *V. cholerae* as well as by parasites, such as *Giardia* sp., *Strongyloides* sp., and *Diphyllobothrium latum* (1, 3). The artificially induced hypochlorhydria associated with cinemidine and similar drugs may also increase the risk for such infections (7). Indeed, neutralization of gastric acid reduces the minimal infecting inoculum of *V. cholerae* (1, 2) and increases both the incidence and severity of cholera (6).

Although the environmental source of the septicemia of our patient was not established, we suspect that the unusual nature of his infection reflected either anatomic derangement or a lack of effective gastric acid due to prior gastrectomy.

**LITERATURE CITED**


