Non–Serogroup O:1 Vibrio cholerae Bacteremia and Cerebritis

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We describe a case of non–serogroup O:1 Vibrio cholerae bacteremia and cerebritis in a 41-year-old Thai man with alcoholism who presented with fever and cellulitis of the right ankle. He was successfully treated with parenteral cefotaxime and then was switched to treatment with oral ciprofloxacin.

Non–serogroup O:1 (hereafter known as “non-O:1”) V. cholerae is morphologically and biochemically indistinguishable from serogroup O:1 (hereafter known as “O:1”) V. cholerae, but it does not agglutinate with Vibrio serogroup O:1 antiserum [1]. These non-O:1 strains of bacteria previously have been named “noncholera vibrios” or “nonagglutinable vibrios.” They are commonly found in brackish water [2], and they have been isolated from various domestic animals [3, 4]. There has been increasing recognition that non-O:1 V. cholerae can cause disease in humans. Non-O:1 V. cholerae has been associated with a spectrum of gastrointestinal illnesses, which range from mild watery diarrhea to febrile enteritis with mucous and bloody diarrhea [5, 6]. In contrast to O:1 V. cholerae, which only rarely causes extraintestinal infection [7], non-O:1 V. cholerae has been associated with systemic infection, such as septicemia, skin infection, pneumonia, sinusitis, peritonitis, and biliary tract infection, particularly in the immunocompromised host [8].

We report a case of non-O:1 V. cholerae bacteremia associated with cerebritis in a patient with alcoholism. To our knowledge, this is the first case of non-O:1 V. cholerae cerebritis to be reported from Thailand and the first to be reported in an adult.

Case report. A 41-year-old man with a 20-year history of excessive alcohol consumption was admitted to Chulalongkorn University Hospital, Bangkok, Thailand. He had experienced fever with chills and right leg pain for 4 days. He denied having cough, abdominal pain, diarrhea, dysuria, or other organ-specific symptoms.

Physical examination revealed a temperature of 39.3°C, blood pressure of 120/60 mm Hg, a heart rate of 126 beats/min, and a respiratory rate of 20 breaths/min. The patient was lethargic and confused. Pale and icteric sclerae were noted. He had signs of chronic liver disease, including bilateral parotid gland enlargement, spider nevi, and palmar erythema. There was mild-to-moderate hepatosplenomegaly, but no ascites were noted. Cellulitis of the right ankle was noted without regional lymphadenopathy. Neither focal neurologic signs nor nuchal rigidity was noted, but flapping tremor was observed. A complete blood count showed a hematocrit of 26.4%, a WBC count of 16,500 cells/mm³ (with 76% polymorphonuclear cells, 8% lymphocytes, and 16% monocytes), and a platelet count of 118,000 cells/mm³. The results of liver function tests were as follows: total bilirubin, 26.6 mg/dL; direct bilirubin, 18.85 mg/dL; alkaline phosphatase, 276 U/dL; aspartate transaminase, 73 U/dL; alanine transaminase, 47 U/dL; and albumin, 2.6 g/dL. At the time of admission, 3 sets of blood samples were drawn for culture before administration of iv cefotaxime, 1 g given q8h.

Four days after admission to the hospital, the patient became deeply confused and developed left hemiparesis. CT of the head revealed 3 small enhancing lesions at the right frontal, frontotemporal cortex, and left insula lobes. Also found was a low-density area that involved the basal ganglia and the thalamic region bilaterally, and that extended upward to the periventricular white matter and the right frontal area (figure 1). These findings suggested cerebritis. A lumbar puncture was not done because of marked cerebral edema.

Antibiotic treatment was changed to iv cefotaxime, 2 g given q6h. Subsequently, all blood cultures done at the time of admission to the hospital were found to have yielded non-O:1 V. cholerae, and the organism was found to be susceptible to ampicillin, cotrimoxazole, tetracycline, third-generation cephalosporins, imipenem, and fluoroquinolones. The patient developed an urticarial rash after 5 days of treatment, so treatment was switched from cefotaxime to iv ciprofloxacin, 400 mg given q8h. Cellulitis subsided after ~1 week of treatment. The patient then gradually improved; he fully recovered after 2 weeks of treatment, at which time antibiotic treatment was changed to oral ciprofloxacin. He remained healthy, and ciprofloxacin was discontinued after 2 months. Serial CT scans of the brain were obtained at ~3 weeks and at 2 months after the initiation of treatment.
antibiotic treatment; the scans showed a gradual improvement, and findings were eventually completely normal.

**Discussion.** Four species of the genus *Vibrio* are pathogenic in humans. These include *V. cholerae* (both the O:1 and the non-O:1 strains) and 3 halophilic *Vibrio* species (*V. parahaemolyticus*, *V. alginolyticus*, and *V. vulnificus*) [9]. Of these species, only *V. vulnificus* has a propensity for bloodstream invasion, and this occurs mostly in immunocompromised hosts. Non-O:1 *V. cholerae*, which is a species of motile, gram-negative, curved, rod-shaped bacteria, is nonhalophilic. However, it requires trace amounts of sodium chloride for growth and can thus exist in a variety of water sources that range from fresh water to salt water. A strong epidemiologic association between cases of non-O:1 *V. cholerae* gastroenteritis and the consumption of raw oysters has been reported [6, 10, 11]. In contrast to O:1 *V. cholerae*, non-O:1 *V. cholerae* can cause a wide spectrum of diarrheal illnesses that vary from mild watery diarrhea to severe mucous and bloody diarrhea. No clinical features distinguish the severe diarrheal illnesses caused by enterotoxin-producing non-O:1 *V. cholerae* from those caused by classic O:1 *V. cholerae* [6]. Again, in contrast to O:1 *V. cholerae*, non-O:1 *V. cholerae* has often been reported to cause extraintestinal infection. Previous reports have documented isolation of the organism from wounds [7, 12, 13], the ear [6, 13], sputum [13, 14], the biliary tract [13, 15], peritoneal fluid [13, 16], urine [17] and CSF [13, 18–20].

Two reports of non-O:1 *V. cholerae* bacteremia have been published in Thailand. The first report, from Siriraj Hospital (Bangkok, Thailand), described a study of the clinical features of 20 patients with bacteremia due to other *Vibrio* species [21]. Of these patients, 10 were infected with non-O:1 *V. cholerae* (3 were infected with *V. vulnificus*, and 7 were infected with *Vibrio* species). No detailed characteristics of any underlying diseases or of the clinical features for each patient with non-O:1 *V. cholerae* infection were described. However, there was no patient with cerebritis in this study.

The second report was from the Department of Pediatrics of Chulalongkorn University Hospital, Bangkok, Thailand [22]. The patient, a 15-year-old girl who had β-thalassemia/hemoglobin E disease, had undergone a splenectomy 3 years prior to admission to the hospital. She presented with primary peritonitis and bacteremia of 2-days’ duration. Exploratory laparotomy disclosed 50 mL of peritoneal fluid that yielded non-O:1 *V. cholerae*. The patient’s postoperative course was uneventful, and she became afebrile on the fifth day of hospitalization.

A total of 6 case reports of non-O:1 *V. cholerae* bacteremia

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**Table 1. Summary of 6 case reports, published in the English-language literature, of non–serogroup O:1 *Vibrio cholerae* bacteremia with associated CNS infection.**

<table>
<thead>
<tr>
<th>Patient, year [reference]</th>
<th>Age of patient</th>
<th>Where <em>V. cholerae</em> was acquired</th>
<th>Underlying disease</th>
<th>Presence of diarrhea</th>
<th>Outcome</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, 1974 [19]</td>
<td>64 y</td>
<td>North Carolina</td>
<td>Probable cirrhosis (esophageal varices)</td>
<td>No</td>
<td>Died</td>
<td>Cellulitis; positive CSF culture result; coma</td>
</tr>
<tr>
<td>2, 1975 [23]</td>
<td>49 y</td>
<td>Spain</td>
<td>Cirrhosis</td>
<td>NA</td>
<td>Survived</td>
<td>—</td>
</tr>
<tr>
<td>3, 1981 [18]</td>
<td>3 wk</td>
<td>Maryland</td>
<td>None</td>
<td>No</td>
<td>Survived</td>
<td>Positive CSF culture result; ventriculitis</td>
</tr>
<tr>
<td>4, 1985 [24]</td>
<td>6 wk</td>
<td>Texas</td>
<td>None</td>
<td>No</td>
<td>Survived</td>
<td>Pneumonia, cerebral abscess</td>
</tr>
<tr>
<td>5, 1993 [20]</td>
<td>6 wk</td>
<td>Arizona</td>
<td>None</td>
<td>No</td>
<td>Died</td>
<td>Positive CSF culture result; meningitis</td>
</tr>
<tr>
<td>6, 1994 [25]</td>
<td>58 y</td>
<td>Taiwan</td>
<td>Diabetes mellitus, cirrhosis</td>
<td>Yes</td>
<td>Survived</td>
<td>Purpuric vesiculobullous eruptions, meningitis</td>
</tr>
</tbody>
</table>

**NOTE.** NA, not available.
with associated CNS infection have appeared in the English-language literature (table 1) [18–20, 23–25]. Three of the 6 cases occurred in infants. The 6 cases included 4 cases of meningitis or meningoencephalitis, 1 case of ventriculitis, and 1 case of cerebral abscess. All of the adult patients had meningitis. No multifocal parenchymatous involvement of the brain that was consistent with cerebritis was observed in any of the adult patients described. The presence of cirrhosis was noted in all adult patients. In contrast to the adults, the infants appeared to be immunocompetent. Acquisition of the organisms occurs either through direct contact with brackish water or salt water, or through consumption of oysters, crabs, or fish that inhabited such water. Data that showed an association between these cases and the consumption of raw oysters or other seafood were available for only 1 patient (patient 6). The other 4 cases were associated with the patient having a previous history of either traveling to or visiting inland lakes or coastal areas where brackish water had been found (as in the cases of patients 1 and 3–5). Therefore, the spread of non-O:1 V. cholerae to infants was probably a result of bathing infants in natural water. In both adults and infants, the case-fatality rate was 33.33%.

Our patient did not report a history of consumption of raw oysters prior to his illness. The occurrence of focal neurological deficits in association with the CT findings of the brain, which revealed multiple enhancing and nonenhancing low-density lesions in both the subcortical and the cortical areas, suggested cerebritis. The patient made a complete clinical recovery. Cerebritis may reflect the virulence of non-O:1 V. cholerae in this unusual body site and/or the poor penetration of antibiotics into the brain parenchyma.

To the best of our knowledge, this is the first case report of non-O:1 V. cholerae bacteremia and cerebritis in a Thai patient with alcoholism. It may also be the first reported case in an adult patient.

References