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NOTES

Intracranial Infection by *Vibrio alginolyticus* following Injury in Salt Water

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A 20-year-old man presented with an epidural abscess 3 months after a seawater diving accident. Cultures of the abscess cavity obtained by surgical drainage revealed a pure culture of *Vibrio alginolyticus*. Marine vibrios may produce serious intracranial infection after head injury in salt water.

Marine vibrios have recently been recognized as potential pathogens in soft-tissue infections (2, 9). *Vibrio* species are ubiquitous in marine or brackish water habitats, where they are considered part of the resident microflora. These halophilic, motile, curved, gram-negative rods thrive in coastal waters worldwide (1, 8, 10).

Infections attributed to these organisms tend to occur in summer months when warmer temperatures promote higher counts of vibrios (6, 9). Extraintestinal infection from *Vibrio* species may arise after entry of the organism through breaks in the skin, resulting in soft-tissue infection. Abrasions or lacerations which occur in seawater are particularly prone to infection by vibrios. Cutaneous infection may be self-limited and resolve without specific antimicrobial therapy. However, severe, life-threatening infections have been described with rapidly evolving cutaneous necrosis, requiring extensive surgical debridement. Fatal infections occur as wound contamination leads to widespread dissemination and septicemia (1, 3). Fatalities are generally observed in patients with underlying disorders such as immunocompromised states, cirrhosis, or iron overload states (1, 2, 5, 9).

There are 10 recognized species of the genus *Vibrio* which have been identified as potentially pathogenic in humans (9). *Vibrio alginolyticus* has infrequently been isolated from human infections despite its widespread saprophytic existence in coastal waters. This organism is most commonly associated with self-limited wound infections and ear infections (10–12). *V. alginolyticus* septicemia and fatal infections have been reported in severely immunocompromised patients as well as in burn patients (3). Intracranial infection by this organism has not, to our knowledge, been described.

This report describes a patient with serious intracranial suppuration after open head injury caused by *V. alginolyticus*. The pathogenesis and management of infection caused by marine vibrios are briefly reviewed.

A 20-year-old male presented in February 1985 with headache and fever. He had sustained an open head injury in December 1984 while on active duty in the U.S. Navy. He had struck his forehead on a submerged object while diving off a platform along the coast of Guam. He sustained an 8-cm laceration and lost consciousness for 2 min after the incident. The laceration was repaired, and radiographs were taken revealing a comminuted fracture of the frontal bone extending through the frontal sinus, with minimal depression of the fragments. A computerized tomography scan showed the fracture site but no other evidence of intracranial pathology.

The patient underwent frontal craniotomy with exoneration of the frontal sinus and realignment of the frontal bone fragments 3 days after the accident. The subdural space was explored, revealing no evidence of hemorrhage or abscess formation. Bacterial cultures of the subdural and epidural spaces were negative. The patient did well postoperatively but did experience cerebrospinal fluid rhinorrhea, which responded to repeated lumbar punctures with decompression of the subarachnoid space. The patient did not receive any antimicrobial therapy other than cephalothin in the perioperative period.

The patient remained asymptomatic until February 1985 when intermittent fever and headache developed. A repeat computerized tomography scan revealed a displaced frontal fracture site as well as a large epidural fluid collection. The patient denied chills, weight loss, or persistent cerebrospinal fluid rhinorrhea. Physical examination was completely normal other than the bony defect and a well-healed, surgical scar in the mid-frontal area. Laboratory studies were unremarkable except for an elevated leukocyte count of 12,000/mm^3 with a normal differential count. A lumbar puncture was performed with normal results except for a protein content of 61 mg/dl. Spinal fluid cultures were negative for bacterial, fungal, or mycobacterial growth.

The patient was empirically treated with nafcillin and chloramphenicol and taken immediately to the operating room, where osteomyelitis of the frontal bone was noted. The entire frontal plate of involved bone was excised. A 25-ml collection of purulent material was recovered from the epidural space. Gram stain of this material revealed pleomorphic, curved gram-negative rods. Cefotaxime was added to the antimicrobial regimen until culture and susceptibility data were available.

The aerobic culture of the epidural space and frontal bone tissue revealed heavy growth of *V. alginolyticus* in a pure culture. The organism grew readily on MacConkey medium as well as thiosulfate-citrate bile salts-sucrose medium (Difco Laboratories, Detroit, Mich.). The isolate was identified by an API 20E system (Analytab Products Inc.,

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TABLE 1. Antimicrobial susceptibility results of the isolate *V. alginolyticus*

Antimicrobial agent	MIC (μg/ml)[a]
Ampicillin	>16
Tetracycline	0.5
Chloramphenicol	1
Cephalothin	>16
Cefamandole	8
Cefoxitin	>16
Gentamicin	1
Amikacin	2
Cefotaxime	<1
Cefoperazone	<2
Trimethoprim-sulfamethoxazole	<0.5-9.5
Piperacillin	<8

[a] MIC by microtiter dilution technique; inoculum size was 10^4 organisms.

Plainview, N.Y.). Anaerobic, fungal, and mycobacterial cultures were all negative. Antimicrobial susceptibility testing was performed by using an automated microtiter dilution technique (American Microscan Systems, Mahwah, N.J.). Results of these studies are given in Table 1. The patient was treated with a 4-week course of intravenous chloramphenicol at a dosage of 50 mg/kg per day without complication. The patient recovered without neurologic sequelae and was discharged without medication other than phenytoin for seizure prophylaxis. The patient was seen in follow-up 6 months later and was completely asymptomatic. Serial computerized tomography scans demonstrated resolution of the epidural abscess collection.

The pathogenic potential of *Vibrio* species in soft-tissue infection has been well illustrated in numerous reports (1–3, 5, 10, 11). These organisms do not appear to be capable of cutaneous invasion through intact skin; nonetheless, serious and potentially life-threatening infections may occur if they gain entrance through cuts or abrasions. Soft-tissue infections are most commonly attributed to *Vibrio vulnificus*, *Vibrio parahaemolyticus*, or *Vibrio damsela*. *V. alginolyticus* has been reported as the causative agent in wound infection (2, 10), otitis (5, 11), and bacteremia (3). The organism is not known to produce a diarrheal illness.

V. alginolyticus is readily distinguished from *Vibrio cholerae* by its inability to grow on sodium chloride-free media. *V. alginolyticus* is oxidase positive and gives negative arginine dihydrolase and variable ornithine decarboxylase reactions. The organism is distinguished from closely related species such as *V. vulnificus* and *V. parahaemolyticus* by biochemical methods. *V. alginolyticus* produces acid from sucrose but not from lactose or arabinose. The organism gives a negative *o*-nitrophenyl-β-D-galactophyranoside reactions but positive Voges-Proskauer reactions. Voges-Proskauer reaction mixtures must be supplemented with 3% NaCl for valid results (1). *V. vulnificus* is Voges-Proskauer negative and is *o*-nitrophenyl-β-D-galactopyranoside positive, whereas *V. parahaemolyticus* is negative for both reactions. *V. vulnificus* has the potential to produce severe soft-tissue infections and septicemia. *V. parahaemolyticus* is primarily an enteric pathogen, but the organism has also been implicated as a cause of soft-tissue infection (1). Little is known about the invasive potential or virulence of *V. alginolyticus*. The organism is known to produce extracellular protease (4). This protease or other substance may act as a toxin enhancing the virulence of the organism. Extracellular toxins probably contribute to the virulence of other *Vibrio* species in soft-tissue infections, such as *V. vulnificus* (7; D. R. Maneval, A. C. Wright, G. M. Marley, R. Petnick, and J. G. Morris, Program Abstr. Intersci. Conf. Antimicrob. Agents Chemother. 24th, Washington, D.C., abstr. no. 605, 1984).

Treatment for soft-tissue infections with *Vibrio* organisms is based upon early recognition, antimicrobial therapy, and surgical drainage. The organisms grow readily on routine culture media such as sheep blood and MacConkey agars. It is unnecessary to use selective media such as thiosulfate-citrate-bile salts-sucrose from extraintestinal specimens where the organism is usually isolated in pure cultures.

Vibrios are usually susceptible to tetracycline, chloramphenicol, aminoglycosides, and a number of the newer cephalosporin antimicrobial agents. Surgical debridement is warranted if necrosis or abscess formation is evident.

Our patient provides clear evidence that *V. alginolyticus* is capable of producing serious intracranial infection after head injury. The possibility of vibrio infection should be considered in the management of head injuries occurring in salt water.

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