JOURNAL OF CLINICAL MICROBIOLOGY, Feb. 1984, p. 197–199
0095-1137/84/020197-03$02.00/0

Vol. 19, No. 2

Wound Infections Caused by *Vibrio vulnificus*, a Marine Vibrio, in Inland Areas of the United States

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Received 15 August 1983/Accepted 31 October 1983

Vibrio vulnificus is a halophilic marine vibrio which may produce infection in wounds exposed to seawater or raw shellfish. The Centers for Disease Control has received two isolates from wounds exposed to inland waters, a New Mexico creek and an Oklahoma reservoir. Halophilic organisms were recovered from both the creek and the reservoir, and the water in both sites was found to be brackish. Both clinical isolates of *V. vulnificus* grew in salt concentrations as low as those found in the creek and reservoir. These cases illustrate the potential for pathogenic halophilic *Vibrio* species to live in brackish inland waters and produce infections in patients living in inland areas of the United States.

The importance of *Vibrio* species other than *Vibrio cholerae* O serogroup 1 as human pathogens has only recently been appreciated. These species, which include non-O1 *V. cholerae*, *V. parahaemolyticus*, *V. alginolyticus*, *V. hollisae*, *V. damsela*, *V. mimicus*, *V. fluvialis*, *V. metschnikovii*, and *V. vulnificus*, produce a variety of intestinal and extraintestinal infections (4, 6, 11, 13, 16). Several of these species have been associated with wound infections: *V. parahaemolyticus*, *V. damsela*, *V. alginolyticus*, and *V. vulnificus* (4, 11).

V. vulnificus is a particularly virulent vibrio which most commonly produces either wound infections or primary sepsis, both associated with a high mortality rate (C. O. Tacket and P. A. Blake, Program Abstr. Intersci. Conf. Antimicrob. Agents Chemother. 22nd, Miami Beach, Fla., abstr. no. 890, 1982). The organism is halophilic, grows well in 6% NaCl (5), and has been recovered from various marine environments (8, 12). Although patients with infected wounds usually report recent exposure of the wound to seawater or to raw shellfish (3; 22nd ICAAC, abstr. no. 890), the Centers for Disease Control (CDC) has received two *V. vulnificus* isolates from infected wounds exposed only to inland waters in the United States, a New Mexico creek and an Oklahoma reservoir. These cases show that the organism exists in brackish inland waters as well as in coastal salt water and should be considered a possible pathogen in wounds exposed to any body of brackish water.

CASE REPORTS

Patient 1. On 17 August 1976, a previously healthy 13-year-old boy was involved in a motor vehicle accident after swimming in Berrendo Creek near Roswell, N.M. He sustained a serious laceration of his scalp. Perhaps because he was confused, he went back to the creek and jumped in. He was later taken to a local emergency room, where a large scalp laceration in the left parietooccipital region was repaired. He had not lost consciousness, and skull X rays did not reveal a fracture. He was hospitalized and, a few hours later, he became less responsive, vomited, and developed hypertension, bradycardia, and dilation of his left pupil. An emergency burr hole was made, revealing a large epidural hematoma. He was transferred to another hospital in Albuquerque, N.M.

He was immediately taken to the operating room, where the complex scalp laceration was explored and a craniotomy was performed. A large epidural hematoma was evacuated and the wound was closed with wire sutures.

On the first postoperative day, his temperature rose to 100.8°F (38.2°C). He was awake, alert, and oriented. On day 3, his temperature again rose to 100.8°F (38.2°C). Gram-negative rods were seen on Gram stain of aspirated subgaleal fluid. The wound was considered satisfactory, however, and without drainage. On day 4, his temperature rose to 100.0°F (37.78°C); he was ambulatory and neurologically normal. There was another collection of subgaleal fluid, and 30 ml of serosanguinous fluid was aspirated from the wound. He received intramuscular gentamicin. Thereafter he remained afebrile but with some drainage from the wound, although the edges of the wound were healing well. Gentamicin was discontinued, and he was sent home on oral ampicillin on 27 August, 10 days after the accident.

The patient had not had any recent exposure to seawater or raw shellfish. The water in Berrendo Creek was described as brackish, although Roswell is about 500 miles from the sea.

The Enteric Bacteriology Laboratory, Center for Infectious Diseases, Centers for Disease Control (CDC), identified the gram-negative rod from the subgaleal fluid aspirated on day 3 as *V. vulnificus*.

Patient 2. On 9 August 1981, a 65-year-old man went fishing in the knee-deep water of the Great Salt Plains Reservoir near Vining, Okla. That evening he developed pain and swelling in his left hand, arm and shoulder, with fever, headache, and chills. There was no puncture site, although an insect bite was suspected. He was seen by his physician, who noted a fever of 104.0°F (40.0°C), tachycardia, and evidence of systemic toxicity. His left hand was red and

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tender with brawny edema. The next morning the fever had resolved, but the hand was more tender and swollen, and small purulent vesicles were present around the first and second metacarpal area. He was treated for cellulitis with cephalexin and warm soaks. Blood cultures obtained on 10 August before antimicrobial therapy were negative. The infection resolved completely within 3 days. The patient had a history of porcine aortic valve prosthesis and was taking oral anticoagulants. He had not travelled to a coastal area or been exposed to raw shellfish.

Cultures of the hand wound obtained on 10 August grew *V. vulnificus*; the identification was confirmed by the Enteric Bacteriology Laboratory at the CDC.

MATERIALS AND METHODS

In September 1981, we returned to Berrendo Creek and, in August 1982, to Great Salt Plains Reservoir and obtained water specimens for culture and salinity measurements. Four Moore swabs were suspended in each body of water and left for 24 h (1). After 24 h, each swab was removed from the water and placed in Venkatraman and Ramakrishnan medium or alkaline peptone broth. Additional specimens were obtained for salinity measurements. These specimens were sent at ambient temperature to the CDC.

A loopful of water from each of the transport broths was streaked onto thiosulfate citrate bile-salts sucrose agar and incubated overnight at 37°C. Another loopful was inoculated into alkaline peptone broth and incubated for 6 h at 37°C before it was plated onto thiosulfate citrate bile-salts sucrose agar and incubated overnight at 37°C.

We tested the salt requirements of the *V. vulnificus* isolates from patients 1 and 2. Each isolate was inoculated into 1% peptone broth with 0.2% NaCl and 1% peptone broth with 0.4% NaCl and incubated overnight at 37°C.

Antimicrobial susceptibility tests were performed by the Kirby-Bauer disk method.

RESULTS

The New Mexico creek water sample yielded a halophilic vibrio from one specimen plated directly onto thiosulfate citrate bile-salts sucrose agar and from the other three specimens after enrichment in alkaline peptone broth. The organism differed from a type strain of *V. vulnificus* (ATCC 27562) by several reactions (Table 1). This vibrio is unlike any other in the CDC reference laboratory. The salinity of the creek was 0.4%.

The Oklahoma reservoir water sample also yielded a halophilic, gram-negative rod after direct plating onto thiosulfate citrate bile-salts sucrose agar. The isolate was different from *V. vulnificus* and from the vibrio from the New Mexico creek (Table 1). The salinity of the reservoir was 0.2%.

The *V. vulnificus* isolates from both patients grew in broth containing 0.4% NaCl and in broth containing 0.2% NaCl.

Both clinical isolates were susceptible to penicillin, ampicillin, carbenicillin, cefalexin, chloramphenicol, tetracycline, sulfadiazine, kanamycin, streptomycin, gentamicin, and nalidixic acid.

DISCUSSION

Wound infections with *V. vulnificus* are usually acquired while handling raw shellfish or after exposure to seawater (22nd ICAAC, abstr. no. 890). Therefore, isolation of *V. vulnificus* from wounds sustained in southeastern New Mexico and northern Oklahoma did not conform to the expected epidemiology of these infections. The two cases reported here demonstrate that brackish inland waters may also be a reservoir for *V. vulnificus*.

V. vulnificus is commonly found in coastal sea waters (8, 12), and we have presented strong evidence that it exists in brackish inland waters; nevertheless, infections of wounds with *V. vulnificus* are not commonly recognized in the United States. In 1981 and 1982, the CDC received only nine

TABLE 1. Characteristics of clinical *V. vulnificus* isolates and environmental isolates

Test	Reaction of *V. vulnificus* isolate from:		Reaction of isolate from:		
	Patient 1	Patient 2	New Mexico creek	Oklahoma reservoir	ATCC 27562 type strain
Lysine decarboxylase	+	+	−	−	+
Ornithine decarboxylase	+	+	−	+	+
Acid from:					
Lactose	+	+	−	−	+
Sucrose	−	−	+	−	−
D-Mannitol	−	−	+	−	−
Salicin	+	+	−	−	+
Sorbitol	−	−	+	−	−
L-Rhamnose	−	−	+	−	−
Melibiose	+	+	−	−	−
α-Methyl-D-glucoside	−	−	+	−	−
ONPG[a] test	+	+	−	−	+
Growth in NaCl (%):					
0	−	−	−	−	−
1	+	+	+	+	+
3.5	+	+	+	+	+
6	+	−	+	+	+
8	−	−	+	+	−

[a] ONGP, *o*-Nitrophenyl-β-D-galactopyranoside.

isolates from patients with infected wounds. Since the taxonomy of the organism has been clarified only recently (2), the apparently low incidence of infections may be partially explained by failure to identify it. The organism grows well on standard media used for wound cultures and may be identified with commercially available batteries of biochemical tests commonly used by clinical laboratories.

Although *V. vulnificus* is a rare pathogen, the organism is particularly virulent once established in a wound (22nd ICAAC, abstr. no. 890). The two patients reported here had unusually mild infections that resolved without complications. The mechanism of virulence of *V. vulnificus* is unknown but may be related to its ability to resist phagocytosis (9) or to produce extracellular toxins or enzymes (10, 14). All environmental and clinical strains of *V. vulnificus* examined in one study were Kanagawa positive (caused hemolysis on Wagatsuma agar) (7), a characteristic that for *V. parahaemolyticus* is associated with enteropathogenicity but not with the ability to cause wound infections. These and other factors may contribute to the marked cellulitis, lymphangitis, and characteristic vesicular and bullous lesions associated with *V. vulnificus* wound infections (as in patient 2) and the necrosis that occurs in the most severe infections.

Wound infections due to *V. vulnificus* are most common in late summer (3). Both cases reported here occurred in August. The seasonal variation may be because of the increase in water sports at that time of year or the ecology of the organism, which is present in larger numbers during the warmer months (8).

Although we did not recover *V. vulnificus* from either environmental source, both contained adequate concentrations of salt to support the growth of the two clinical *V. vulnificus* isolates. Perhaps other environmental conditions, such as pH, water temperature, or the presence of competing organisms, interfered with the growth of *V. vulnificus* at the time the water was sampled. Recovery of autochthonous halophilic vibrios from inland waters in New Mexico and Oklahoma suggests that other potentially pathogenic halophilic *Vibrio* species can live in brackish inland waters under certain conditions. Indeed, another halophilic vibrio, *V. parahaemolyticus*, was responsible for traumatic panophthalmitis in a patient whose eye was exposed to water from a brackish pond in inland Georgia (15). The halophilic vibrios should be considered among the potential pathogens in wound infections that occur after exposure to salt or brackish waters, even in inland areas.

ACKNOWLEDGMENTS

We gratefully acknowledge the assistance of John J. Farmer III and Gold and Rorabaugh, who cared for these patients.

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