

## Cholera from Raw Seaweed Transported from the Philippines to California

D. J. VUGIA,<sup>1\*</sup> A. M. SHEFER,<sup>2</sup> J. DOUGLAS,<sup>3</sup> K. D. GREENE,<sup>2</sup> R. G. BRYANT,<sup>1</sup> AND S. B. WERNER<sup>1</sup>

California Department of Health Services, Berkeley,<sup>1</sup> and San Joaquin County Health Department, Stockton,<sup>3</sup> California, and Centers for Disease Control and Prevention, Atlanta, Georgia<sup>2</sup>

Received 12 July 1996/Returned for modification 3 September 1996/Accepted 17 October 1996

**In March 1994, a California woman without any recent travel developed acute, profuse, watery diarrhea. Her astute physician diagnosed cholera after ordering the appropriate stool culture, and the patient improved on an oral antibiotic. Epidemiologic investigation implicated seaweed from the Philippines that was transported by a friend to California and subsequently eaten raw as the vehicle of infection.**

While water and various foods have been implicated by epidemiologic studies as vehicles of transmission of *Vibrio cholerae* O1 (6), rarely has the implicated food crossed international borders. In the United States, most cases of cholera are associated with foreign travel where contaminated foods and drinks were consumed (5). However, four outbreaks of locally acquired cholera have been associated with food transported from other countries: two resulted from eating crab transported in luggage from Ecuador (1, 3), one followed consumption of frozen coconut milk commercially imported from Thailand (8), and one occurred after consumption of foods transported from El Salvador, although a specific food item was not implicated (2). We report a case of cholera acquired in California caused by a rather unusual food vehicle that was transported into this country: uncooked seaweed.

In March 1994, a 31-year-old California woman of Filipino descent became acutely ill with nausea, vomiting, and watery diarrhea approximately every 15 min for at least 24 h. Although she had no history of recent travel, her physician astutely ordered a fecal culture for cholera in addition to routine enteric pathogens. He prescribed ciprofloxacin, and the patient improved without hospitalization two days after starting antibiotic therapy. Her stool yielded toxigenic *V. cholerae* O1 serotype Ogawa, as confirmed by the Microbial Diseases Laboratory (MDL) of the California Department of Health Services and by the Foodborne and Diarrheal Diseases Laboratory (FDDL) of the U.S. Centers for Disease Control and Prevention (CDC).

Two days before the onset of her illness, the patient ate a dinner at home consisting of raw seaweed, fried fish, pickled pig feet, and freshly steamed rice. The seaweed was brought from the Philippines a month earlier, hand carried on an airline flight by a friend, and kept frozen at the patient's home until being defrosted, seasoned, and served uncooked. The fish was also hand carried from the Philippines by another passenger several weeks earlier and kept frozen until being fried. According to the patient, the fish was fried until well done. The pig feet were purchased raw from a local store and pickled at home. Six other persons shared this dinner, but no one else became ill. One day before her illness, the patient also attended another family gathering where sashimi, a raw fish dish, was served. This fish was bought in Hawaii the previous day

and flown in a refrigerated ice chest. Eleven persons had eaten the sashimi, and, except for the patient, all remained well.

Leftover seaweed and other fish found in the patient's freezer were tested for *Vibrio* organisms at MDL by enrichment with alkaline peptone broth and thiosulfate-citrate-bile salts-sucrose agar. No leftover fried fish, pig feet, or sashimi was available for testing. Of the food items tested, only the seaweed yielded a vibrio, a nontoxigenic *V. cholerae* non-O1 isolate.

Sera from five other persons present at the dinner where seaweed was served (the seventh person refused blood drawing) were obtained 16 days after the dinner and were tested for vibriocidal antibodies and antitoxin titers at the CDC's FDDL (9). The results, along with food consumption histories, are listed in Table 1. Of the three persons who ate the seaweed, besides the case patient, two had serologic results indicative of recent infection with *V. cholerae*, including one who had eaten seaweed but not fish.

To our knowledge, this is the first time that seaweed has been implicated as a food vehicle for cholera. Given the food exposures and the results of serology and food testing, the raw seaweed was the likely vehicle for these symptomatic and asymptomatic *V. cholerae* infections. Seaweed grows in an environment where vibrios thrive (4, 7). Isolation of *V. cholerae* non-O1 from leftover seaweed supports the notion that this vehicle could have been contaminated with *V. cholerae* O1 as well, but the latter was not recovered. There was not enough seaweed leftover for identification of species, but inquiry of others in the Filipino community suggests that it may have been a type of kelp. Consumption of raw seaweed or kelp should now be considered a potential risk factor for cholera, particularly if gathered from potentially contaminated waters.

The other interesting aspect of this investigation is that this case of cholera was acquired in California by a person who had no recent foreign travel. If it were not for her astute physician asking that stool studies be set up for *Vibrio* species that cause cholera, this case would have been missed. In patients with acute, profuse, watery diarrhea in whom cholera is suspected, clinicians should also request specific stool studies for vibrios (e.g., using thiosulfate-citrate-bile salts-sucrose agar) since routine stool cultures do not usually detect *V. cholerae*. While antibiotics can shorten the duration of illness, primary treatment for cholera remains prompt replacement of fluid and electrolytes.

For patients with suspected cholera, questions about consumption of foods from abroad may be as revealing as questions about recent travel. The practice of transporting perish-

\* Corresponding author. Mailing address: Division of Communicable Disease Control, California Department of Health Services, 2151 Berkeley Way, Berkeley, CA 94704. Phone: (510) 540-2566. Fax: (510) 540-2570.

TABLE 1. Food history and vibriocidal (*V. cholerae* Inaba or Ogawa) and antitoxin antibody titers<sup>a</sup> of six persons attending the dinner where seaweed was served

Person	Ate:			Titer <sup>a</sup>	
	Pig feet	Fish	Seaweed	Vibriocidal	Antitoxin
1	Yes	No	No	40	200
2	Yes	No	No	160	200
3	Yes	Yes	Yes	<20	200
4	Yes	Yes	Yes	1,280	200
5	Yes	No	Yes	1,280	12,800
Case	Yes	Yes	Yes	ND	ND

<sup>a</sup> Vibriocidal antibodies were considered positive at  $\geq 1,280$  while antitoxin titers were considered positive at  $\geq 400$ . ND, not done.

able seafood in travelers' luggage has caused two previous outbreaks of cholera in the United States (1, 3). While not illegal, this practice should be discouraged.

#### REFERENCES

1. Centers for Disease Control and Prevention. 1991. Cholera—New York, 1991. *Morbidity and Mortality Weekly Report* 40:516–518.
2. Centers for Disease Control and Prevention. 1995. Cholera associated with food transported from El Salvador—Indiana, 1994. *Morbidity and Mortality Weekly Report* 44:385–386.
3. Finelli, L., D. Swerdlow, K. Mertz, H. Ragazzoni, and K. Spitalny. 1992. Outbreak of cholera associated with crab brought from an area with epidemic disease. *J. Infect. Dis.* 166:1433–1435.
4. Janda, J. M., C. Powers, R. G. Bryant, and S. L. Abbott. 1988. Current perspectives on the epidemiology and pathogenesis of clinically significant *Vibrio* spp. *Clin. Microbiol. Rev.* 1:245–267.
5. Mahon, B. E., E. D. Mintz, K. D. Greene, J. G. Wells, and R. V. Tauxe. 1996. Reported cholera in the United States, 1992–1994. A reflection of global changes in cholera epidemiology. *JAMA* 276:307–312.
6. Mintz, E. D., T. Popovic, and P. A. Blake. 1994. Transmission of *Vibrio cholerae* O1, p. 345–356. In I. K. Wachsmuth, P. A. Blake, and Ø. Olsvik (ed.), *Vibrio cholerae* and cholera: molecular to global perspectives. American Society for Microbiology, Washington, D.C.
7. Sims, J. K., J. A. Brock, R. Fujioka, L. Killion, L. Nakagawa, and S. Greco. 1993. *Vibrio* in stinging seaweed: potential infection. *Hawaii Med. J.* 52(10): 274–275.
8. Taylor, J. L., J. Tuttle, T. Pramukul, K. O'Brien, T. J. Barrett, B. Jolbitado, Y. L. Lim, D. Vugia, J. G. Morris, Jr., R. V. Tauxe, and D. M. Dwyer. 1993. An outbreak of cholera in Maryland associated with imported commercial frozen fresh coconut milk. *J. Infect. Dis.* 167:1330–1335.
9. Young, C. R., I. K. Wachsmuth, Ø. Olsvik, and J. C. Feeley. 1986. Immune response to *Vibrio cholerae*, p. 363–370. In N. R. Rose, H. Friedman, and J. L. Fahey (ed.), *Manual of clinical laboratory immunology*, 3rd ed. American Society for Microbiology, Washington, D.C.