

Outbreak of Clinically Mild Botulism Type E Illness from Home-Salted Fish in Patients Presenting with Predominantly Gastrointestinal Symptoms

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Five persons consumed home-salted fish and then presented with gastrointestinal symptoms to 3 hospitals; 2 of the patients had minimal cranial nerve palsies. Early serum samples obtained from all patients were negative for botulinum toxin. Remnant fish tested positive for botulinum toxin type E. In patients exposed to low doses of botulinum toxin type E, gastrointestinal symptoms may predominate.

Foodborne botulism is a paralytic illness that is caused by ingestion of the toxins of *Clostridium botulinum* and closely related species and is characterized by cranial palsies and descending symmetric flaccid paralysis that may include respiratory muscles. Treatment consists of supportive intensive care, with mechanical ventilation if needed, and administration of equine-origin botulinum antitoxin, which prevents progression of paralysis. There are 7 antigenically distinct botulinum toxins; types A, B, E, and, rarely, F cause most human illness [1]. Illness from type E toxin is associated with consumption of improperly prepared foods of aquatic origin, either freshwater or marine [2–5]. Although botulism is defined by neuroparalytic symptoms, gastrointestinal symptoms are more prominent in patients with types B and E intoxication than in patients with type A intoxication [1]. It is not known whether gastrointestinal symptoms are caused by botulinum toxin.

On 4 July 2005, clinicians in New Jersey alerted public

health officials about suspected botulism in a 45-year-old Egyptian-born man in New Jersey. Remarkably, the clinicians recognized the man as the index case patient of an outbreak of type E botulism that resulted from consumption of salted fish in 1992 [6].

Methods. An epidemiologic investigation included case-finding; interviews of patients, clinicians, and contacts; and collection of suspect foods. Medical charts were abstracted using a standardized instrument. A possible case of botulism was defined as only gastrointestinal symptoms (nausea, cramps, vomiting, or diarrhea) in a person who had consumed food that tested positive for botulinum toxin. A confirmed case of botulism was defined as any objective neurological findings consistent with botulism or gastrointestinal symptoms in a person in whose serum botulinum toxin was detected or only objective neurological findings consistent with botulism in a person who had consumed food that tested positive for botulinum toxin. Serum samples from case patients and food specimens were tested for botulinum toxin by mouse bioassay at the New Jersey Department of Health and Senior Services laboratories (Trenton, NJ) [7].

Results. Interviews revealed that the index case patient (patient A) placed fresh, unevicered whitefish with salt in a sealed ziplock bag, which was then left on a kitchen counter for ~1 month. He tasted the fish on 3 July 2005, noticed onset of gastrointestinal and neurologic symptoms on 4 July at 2:00 P.M., and presented to an emergency department (ED) that day at 4:00 P.M. with multiple complaints; on examination, the patient was found to have only a sluggish left pupil and dysarthria. The salted fish was then served at a party at his home on the evening of 4 July. Of those that consumed the fish, 2 women aged 49 years and 54 years (patients B and C) subsequently experienced predominantly gastrointestinal symptoms. They were taken to a second ED and were admitted but never developed neurological symptoms. The 16-year-old daughter of one of the women (patient D) had eaten the fish and driven patients C and D to the ED. She had made no complaints but was noted by the ED staff to have diplopia, because she could not dial a number on her cell phone. On examination, she was found to have sluggish pupils. Several days later, the mother-in-law of the index patient (patient E) was found to have been hospitalized with only gastrointestinal symptoms at a third hospital; she never developed neurological symptoms. The signs and symptoms of these patients are summarized in table 1. Patients A and C were the only patients to demonstrate objective neurological findings (despite extensive subjective symp-

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Table 1. Details of an outbreak of clinically mild botulism caused by consumption of botulinum toxin type E-containing home-salted fish.

Patient	Complaints at presentation	Neurological findings at presentation	Duration, ^a h				Serum toxin test results	Case classification ^b
			From exposure to symptom onset	From symptom onset to presentation at the ED	From ED presentation to antitoxin treatment	From exposure to serum sample collection		
A ^c	Nausea, vomiting, blurred vision, dysarthria, dry mouth, bilateral arm weakness, paresthesias, and fatigue	Sluggish left pupil dysarthria	<24	26	11	<72	Negative	Confirmed
B	Nausea, vomiting, abdominal pain, and dry mouth	None	<24	<24	Not treated	<28	Negative	Possible
C	Nausea, vomiting, and abdominal pain	None	<4	16	Not treated	<24	Negative	Possible
D	Abdominal pain, blurred vision, dry mouth, dysphagia, and shortness of breath	Sluggish pupils	Unknown	<24	<8	<24	Negative	Confirmed
E	Nausea, vomiting, and abdominal pain	None	<4	<84	Not treated	<24	Negative	Possible

NOTE. ED, emergency department.

^a Time intervals are reported as the maximum possible number of hours on the basis of hospital chart information.

^b A confirmed case was defined as any objective neurological findings consistent with botulism or gastrointestinal symptoms in a person in whose serum sample botulinum toxin was detected or only objective neurological findings consistent with botulism in a person who had consumed food that tested positive for botulinum toxin. A possible case was defined as gastrointestinal symptoms (nausea, cramps, vomiting, or diarrhea) without neurological findings in a person who consumed food that tested positive for botulinum toxin.

^c Index patient.

toms), and they were treated with botulinum antitoxin within 11 h and <8 h after presentation to the ED, respectively (table 1), with no subsequent neurological progression. The duration of time from the consumption of the implicated food to presentation to the ED is also shown in table 1.

Serum samples collected from patients from 24 to 72 h after consumption of salted fish tested negative for botulinum toxin (table 1). Stool was not available for diagnostic testing. Remnant salted fish recovered from the index patient's home tested positive for botulinum toxin type E; the sample size was insufficient for toxin quantification.

Discussion. We report an outbreak of botulism type E that resulted from consumption of home-salted fish prepared by a New Jersey resident who was the index case patient of another outbreak in 1992. The outbreak was notable for predominance of gastrointestinal symptoms, paucity of neurological findings, the absence of botulinum toxin in all of the patients' serum samples, and the dispersion of the 5 case patients to 3 EDs.

Botulinum toxin type E was detected in remnant home-salted fish that was consumed by all of the case patients. The prominence of gastrointestinal symptoms in botulism type E cases has been well-described [2–5, 8]; however, it remains unknown whether these symptoms are caused by the toxin, by other clostridial products, or by products of anaerobic spoilage independent of *C. botulinum*. In this outbreak, 3 patients, apparently unaware of their exposure to botulinum toxin, presented to EDs because of severe gastrointestinal symptoms within hours after consuming the contaminated fish. Failure

to detect toxin in the serum samples from any patient, despite prompt serum collection, suggests either that the patients ingested low toxin loads, with rapid clearance of any circulating toxin, or that the patients' gastrointestinal symptoms were caused by a substance other than botulinum toxin. The acute objective neurological symptoms in 2 cases can be ascribed only to the action of botulinum toxin. Gastrointestinal symptoms have not been reported in wound botulism, which is caused by toxin types A and B [9], nor have corresponding signs been observed in primate experiments in which pure toxin types A, B, C, or D were administered intragastrically or intravenously [10–15]. Vomiting is not reported as a prominent occurrence in animals fed purified botulinum toxin type E [16]. It is, therefore, possible that the 3 patients meeting our case definition for possible botulism did not, in fact, have botulism.

Although botulinum toxins are highly antigenic [17], serum samples from patients with convalescent foodborne botulism do not demonstrate protective antibodies [18]. A handful of repeat intoxications with the same toxin type in the same individual have been reported [19, 20], suggesting that naturally occurring foodborne botulism confers no protection. Nevertheless, because the index patient in this outbreak experienced clinically severe type E botulism in 1992, it is possible that his mild illness in this outbreak of botulism was because of naturally acquired immunity. Other patients in the 1992 outbreak of botulism included 3 relatives, but we do not know if these were the same persons who were affected in the 2005 outbreak.

The 5 patients in this outbreak of botulism received treat-

ment at the EDs in 3 hospitals, where the link of 4 patients to the index case of botulism was not initially divulged to clinicians. Although the swift investigation by New Jersey health officials and the astuteness of clinicians led to timely linking of these patients, the incident highlights the challenge of identifying clusters of mild botulism when patients are not treated at the same facility.

This investigation had several limitations. Some of the patients were reluctant to assist health authorities and initially provided limited data. Clinical data were documented in charts from 3 hospitals by various clinicians and were retrospectively abstracted. Stool samples for toxin testing and culture were not obtained from any patients.

Traditional salted fish have caused botulism in Egypt [21], and the Egyptian-born index case patient in this outbreak of botulism was also the index case patient of a small outbreak of botulism in New Jersey in 1992 that was caused by a traditional salted fish of uncertain provenance [6]. Further investigation and outreach work with Egyptian communities in North America may identify ways to decrease the risks associated with eating salted fish.

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