MAJOR ARTICLE

An Outbreak of Foodborne Botulism Associated with Food Sold at a Salvage Store in Texas

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Foodborne botulism is caused by potent neurotoxins of *Clostridium botulinum*. We investigated a large outbreak of foodborne botulism among church supper attendees in Texas. We conducted a cohort study of attendees and investigated the salvage store that sold the implicated foods. We identified 15 cases of botulism (40%) among 38 church supper attendees. Nine patients (60%) had botulinum toxin type A detected in stool specimens. The diagnosis was delayed in 3 cases. Fifteen (63%) of 24 attendees who ate a chili dish developed botulism (relative risk, undefined; $P < .001$). The chili dish was prepared with "brand X" or "brand Y" frozen chili, "brand Z" canned chili, and hot dogs. An unopened container of brand X chili yielded type A toxin. Brand X chili was purchased at a salvage store where perishable foods were inadequately refrigerated. Our investigation highlights the need to improve clinicians' awareness of botulism. More rigorous and more unannounced inspections may be necessary to detect food mishandling at salvage stores.

Botulism is a rare neuroparalytic illness caused by *Clostridium botulinum,* a gram-positive, anaerobic, spore-forming bacterium. *C. botulinum* strains produce 7 potent neurotoxins (types A–G), of which types A, B, and E cause most cases of botulism in humans [1]. The neurotoxins inhibit acetylcholine release into the neuromuscular junction by binding irreversibly to presynaptic nerve endings and causing the enzymatic cleavage of cholinergic vesicle shuttle proteins. Botulinum intoxication can lead to a classic clinical syndrome of cranial neuropathy and symmetric descending flaccid paralysis, which necessitates mechanical ventilatory support in ~60% of patients [2].

Foodborne botulism is caused by the consumption of food containing botulinum toxin. *C. botulinum* spores are ubiquitous in the environment, but germination and toxin production occur primarily, although not exclusively, under anaerobic, low-salt, low-sugar, and low-acid conditions at nonrefrigeration temperatures [3]. Most outbreaks of foodborne botulism are caused by home-canned foods [4]. However, several large outbreaks in recent decades have been associated with commercial or restaurant foods [5–7]. Storage at inappropriate temperatures of a widely distributed commercial food product that contains *C. botulinum* spores could lead to large outbreaks of this potentially fatal disease. We describe a large outbreak of foodborne botulism caused by commercially produced food sold at a salvage food store in the Dallas–Fort Worth metropolitan area in Texas. Salvage stores typically sell foods in bulk quantities or, occasionally, foods that are rejected by standard grocery stores, including those that have passed the expiration date.

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OUTBREAK RECOGNITION

On the evening of 29 August 2001, 4 men were admitted to a hospital in Dallas with cranial neuropathy and

progressive peripheral muscle weakness. Three had attended a choir event and supper at a church on 25 August. The fourth patient had eaten leftover food from the church supper. Officials from the Texas Department of Health (TDH; Austin) soon learned that 2 children who attended the church supper had been hospitalized on 28 and 29 August in Fort Worth for similar complaints. One of the 6 hospitalized patients required emergent mechanical ventilatory support, and 4 others underwent intubation subsequently. A preliminary clinical diagnosis of botulism was made, and the Centers for Disease Control and Prevention (CDC; Atlanta) was contacted for immediate release of botulinum antitoxin. The TDH and the CDC initiated an investigation to determine the extent of the outbreak, to identify the contaminated food, and to enact control measures.

METHODS

We conducted hypothesis-generating interviews with family members of the 6 hospitalized patients and with other church supper attendees to determine what food items were served, where the ingredients were purchased, and how the meals were prepared. Active case-finding was conducted among supper attendees and in the greater Dallas–Fort Worth area. Botulinum intoxication was defined by the detection of botulinum toxin in stool specimens. For surveillance purposes, we defined a case as laboratory-confirmed botulinum intoxication or $\geqslant$2 cranial nerve symptoms, in a Texas resident who had consumed food with laboratory-confirmed botulinum toxin contamination during August or September 2001. Area hospitals were notified of the outbreak, instructed about signs and symptoms of botulism, and asked to call local public health officials regarding possible cases. An alert was issued to physicians statewide and to epidemiologists in neighboring states and Mexico.

We conducted a cohort study involving attendees of the 25 August 2001 church supper and persons who ate leftover food from the event to identify the specific food exposure. For the cohort study, we defined a case as laboratory-confirmed botulinum intoxication or $\geqslant$2 cranial nerve or gastrointestinal symptoms in the 2 weeks after the church supper in a person who consumed food prepared for that event. A written questionnaire assessed clinical history and exposure to foods served at the church supper. Persons who ate food served at the church supper were asked to provide stool samples for botulinum toxin testing and *C. botulinum* culture.

Commercial food products used in preparing the church supper were traced back to the retail store and manufacturing plant. The TDH, the CDC, and the Food Safety and Inspection Service staff of the US Department of Agriculture interviewed retail store employees and examined storage conditions and food sale practices. We inspected the chili manufacturing plant's production methods and reviewed product distribution details.

Laboratory testing. Stool samples were obtained from hospitalized patients. In addition, stool specimens were requested from all persons who attended the church supper, regardless of the presence of symptoms. All specimens from humans were forwarded to the TDH laboratory in Austin for detection of botulinum toxin using the mouse toxicity and neutralization bioassay and culture for *C. botulinum* [1]. Leftover foods were tested at the TDH laboratory for the presence of botulinum toxin [1]. Using methods outlined in the *Bacteriological Analytical Manual, Revision A* [8], the US Food and Drug Administration's Office of Regulatory Affairs Southeast Regional Laboratory (Atlanta) tested unopened original containers of food for detection and quantification of botulinum toxin using the mouse bioassay and ELISA [9]. All stool specimens and nonintact food samples were cultured for *C. botulinum* using standard culture techniques.

Statistical analysis. Univariate analysis was performed using SAS software, version 8 (SAS Institute), to determine the association between exposure to church supper foods and the development of botulism.

RESULTS

Active case finding. We identified 16 cases of botulism in the greater Dallas–Fort Worth area; onset of symptoms occurred during the period of 25 August through 1 September 2001 (table 1 and figure 1). Fifteen patients were from the church cohort. An additional case of botulism occurred in a Fort Worth resident (hereafter referred to as the "outlier") who had cranial neuropathy and a descending flaccid paralysis. He had not attended the church supper but had consumed a commercial chili product that was confirmed to contain botulinum toxin. Nine patients had laboratory-confirmed botulinum intoxication. There were no deaths. Neurologic symptoms included dysphagia (69% of patients), blurred vision (56%), slurred speech (50%), and double vision (38%). Gastrointestinal symptoms were abdominal pain (56% of patients), diarrhea (44%), nausea (31%), and vomiting (31%). Botulinum toxin was detected in the stool specimen of 1 asymptomatic person. Ten patients (63%) were hospitalized, 6 (38%) of whom required mechanical ventilatory support. Nine patients (56%) received bivalent AB botulinum antitoxin. No cases of botulism were detected in other parts of Texas or in neighboring states during August and September 2001.

Of the 15 symptomatic patients, 5 did not seek medical care; they were detected by the investigative team with the use of the outbreak questionnaire and the collection of stool specimens. Two of these 5 were confirmed to have botulinum toxin

Table 1. Characteristics of patients with botulism, by clinical severity, in Texas, August through September 2001.

Patient	Age, years	Symptom type	Clinical severity	Laboratory confirmation	Delay in diagnosis
1	3	CN, GI	Patient required intubation	Type A toxin in stool specimen	Yes
2	7	CN, GI	Patient required intubation	Toxin type A in stool specimen	Yes
3[a]	17	CN	Patient required intubation	Symptoms, epidemiologic link	Yes
4	29	CN, GI	Patient required intubation	Toxin type A in stool specimen	No
5	42	CN, GI	Patient required intubation	Toxin type A in stool specimen	No
6	49	CN, GI	Patient required intubation	Toxin type A in stool specimen	No
7	25	GI	Patient was hospitalized	Symptoms, epidemiologic link	No
8	45	CN	Patient was hospitalized	Toxin type A in stool specimen	No
9	53	CN, GI	Patient was hospitalized	Symptoms, epidemiologic link	No
10	62	CN, GI	Patient was hospitalized	Symptoms, epidemiologic link	Yes
11	17	GI	Patient was not hospitalized	Toxin type A in stool specimen	NA[b]
12	45	CN, GI	Patient was not hospitalized	Symptoms, epidemiologic link	NA[b]
13	50	GI	Patient was not hospitalized	Symptoms, epidemiologic link	NA[b]
14	52	CN	Patient was not hospitalized	Toxin type A in stool specimen	NA[b]
15	78	GI	Patient was not hospitalized	Symptoms, epidemiologic link	NA[b]
16	72	Asymptomatic	Patient was not hospitalized	Toxin type A in stool specimen	NA[b]

NOTE. CN, cranial nerve (i.e., blurred vision, diplopia, dysarthria, and dysphagia); GI, gastrointestinal (i.e., abdominal cramps, nausea, vomiting, and diarrhea); NA, not applicable.

[a] Outlier.

[b] Patient did not seek medical attention. Diagnosis was made by epidemiology investigative team.

in stool specimens; one patient had diarrhea and abdominal cramps, and the other had mild diplopia. Two of the 3 patients who were identified using the outbreak questionnaire and who did not have laboratory-confirmed illness had nausea and diarrhea; the third had blurred vision and diarrhea.

Among the 10 patients who sought medical care, 6 had severe neurologic impairment, requiring mechanical ventilatory support. Two of the remaining 4 patients had multiple cranial nerve and gastrointestinal symptoms; the other 2 patients had gastrointestinal symptoms. The diagnosis of botulism was delayed in 4 persons who presented with classic neurologic symptoms. Two children received diagnoses of botulism >24 h after initial presentation to medical care; the early symptoms of one included dysarthria, dysphagia, weakness, and diarrhea, and the early symptoms of the other included blurred vision and diplopia. An adult patient from the church cohort was diagnosed with hypertensive urgency and was hospitalized for 5 days with blurry vision and dysphagia. In addition, the 17-year-old outlier sought treatment at hospital emergency departments 4 times over the course of 3 days with classic, progressive multiple cranial neuropathies and descending paralysis before botulism was diagnosed.

Among church supper attendees, we attempted to quantify the relationship between "dose," defined as the amount of chili consumed, and the severity of illness, in addition to the association between the timing of chili consumption and the development of botulism. However, we were unable to obtain accurate information on how much chili was eaten from the 6 most severely affected patients, all of whom required mechanical ventilatory support and sedation.

Cohort study of church supper. Forty persons attended the church supper or ate leftovers from the supper. Questionnaires were completed for 38 persons, including 37 who attended the supper and 1 who did not attend but who ate leftovers. Fifteen persons (39%), including the asymptomatic

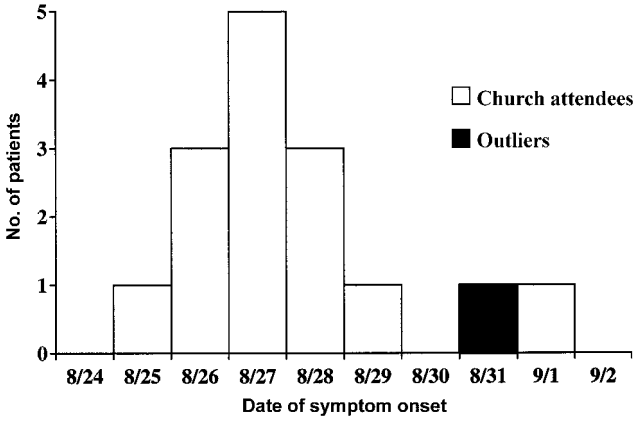

Figure 1. Cases of botulism, by date of symptom onset (month/day) in Texas, August through September 2001.

patient, met the case definition for botulism. The supper menu was limited to fried chicken from a restaurant and a chili dish prepared at home from commercial products. Fifteen (50%) of the 30 persons who ate any food served at the supper became cases, compared with none of the 8 persons who ate no food (relative risk [RR], undefined; $P = .011$, by Fisher's exact test). Fifteen (63%) of 24 persons who ate the chili dish became cases, compared with none of the 14 persons who did not eat chili (RR, undefined; $P < .001$, by Fisher's exact test). The attack rate among those who ate chicken but not chili was 0% (0 of 6 persons). No significant association was detected between the amount of chili consumed and the severity of illness.

Preparation of implicated food. On 25 August, the Saturday afternoon of the church supper, a church congregant purchased four 5-lb (2.27-kg) plastic tubs of frozen chili, an unknown number each of "brand X" and "brand Y," from a salvage store in Fort Worth. The congregant reported that she had prepared the chili dish by thawing a single frozen plastic tub of chili, but she could not remember which brand she used. She heated the contents of the plastic tub in a pot and removed the pot from the stove as soon as the chili began to boil. The congregant drove 5 min to the church kitchen, where she placed the pot on a burner set to low heat. Hot dogs and "brand Z" commercial chili from an aluminum can were later added to the pot. After the church supper, the remaining chili dish was taken to the cook's home, where several family members ate it over the subsequent 4 days.

One day before the onset of his symptoms, the 17-year-old outlier ate frozen brand X chili, which he had heated but not boiled. The patient reported to his mother that the chili appeared to be spoiled, with a fungus-like appearance in one section of the container. He proceeded to eat the chili despite the indication of spoilage and his mother's advice against consumption. The brand X chili had been purchased by his mother at the Fort Worth salvage store on 24 August, 1 day before the church congregant purchased brands X and Y chili at the same store.

Laboratory investigation. Botulinum toxin type A was detected by mouse bioassay in stool samples obtained from 9 of 14 symptomatic patients from the church cohort. Botulinum toxin type A was identified in the stool of 1 of 2 asymptomatic church supper attendees who submitted specimens. No toxin was identified in the outlier's stool sample. Toxin type A was also identified in the chili leftovers from the church supper and in an opened container of frozen brand X chili eaten by the outlier. An unopened container of frozen brand X chili obtained from the cook in the church cohort had a pH of 5.47. The concentration of botulinum toxin in the previously unopened brand X chili container was 10,000 mouse lethal doses/g and 160 ng of type A toxin/g of chili. *C. botulinum* organisms were isolated by culture of the food and were verified as type A toxin–producing organisms using ELISA.

Traceback and investigation of food source. The outlier's mother and the cook in the church cohort purchased brand X chili on Friday, 24 August, and Saturday, 25 August, respectively, at the same Fort Worth salvage store. The salvage store sold foods in conditions often rejected by standard grocery stores. Such products included products in dented cans, repackaged foods, and products that had passed their expiration date. Employees at the Fort Worth salvage store sorted through large volumes of damaged foods and selected items for sale to the public. Informal rules were followed to determine which foods could be sold. We found no evidence that standardized training or use of written manuals had guided the selection process.

During an unannounced outbreak-related inspection on a Saturday afternoon, gross mishandling of foods was observed. On Saturdays, the store routinely offered foods at discounts exceeding those of weekdays. Bulging cans, dented cans, and opened plastic containers of food were on display for sale. Foods that required refrigeration or freezing were displayed in carts at room temperature for customers to browse through and buy. Employees reported that the items displayed at ambient temperatures for >7 hours were routinely placed back in refrigerator storage for sale on later days. Other violations included the use of the warehouse area for retail sales and inadequate labeling of repackaged foods. The salvage store was regulated and had been routinely inspected by the TDH. Because routine inspections before the outbreak were usually conducted during weekdays, the mishandling of food observed during Saturday sales had not been detected previously.

Because there was a lack of invoices for products received at the salvage store, the distributors, the dates of receipt, and the condition on delivery of specific lots of frozen brands X and Y chili could not be determined. Because it is not a canned product, brand X chili did not undergo retort cooking or similar spore-killing processes. Inspections of the chili manufacturing plant did not reveal poor production practices or product mishandling. We did not detect botulinum toxin in 2 containers of brand X chili obtained from a first-line distributor. No other brand X chili containers from the implicated lot were found at other distributors or retailers. Brand X chili was distributed to Louisiana and Oklahoma; no cases of botulism were reported in either state in August and September 2001.

DISCUSSION

This outbreak of foodborne botulism in Texas resulted in 15 illnesses and is the largest such outbreak in the United States since 1994. The correct diagnosis was delayed by 1–7 days in 4 patients who had classic symptoms of botulism. Misdiagnosis has resulted in delayed detection and investigation of botulism outbreaks in the past [7]. Botulinum toxin is an agent of biological warfare, and *C. botulinum* is a high-priority agent on

the CDC's *List of Critical Biological Agents* [10]. This outbreak and the potential for intentional release of botulinum toxin underscore the continuing need to improve awareness among clinicians of the symptoms and signs of botulism. Early recognition of botulism cases can be critical for providing prompt treatment with antitoxin, identifying the source of the intoxication, and controlling the extent of outbreaks. Physicians should notify their local and state health departments regarding suspected cases of botulism. State health officials initiate any necessary epidemiologic investigation and facilitate the physician's liaison with the CDC. The CDC maintains a 24-h consultation service to assist physicians with diagnosis and management of this rare disease. Equine botulinum antitoxin for types A, B, and E can prevent the progression of neurologic dysfunction if it is administered early in the course of illness [11]. The CDC makes antitoxin available after consultation with the treating physician, but delayed administration of botulinum antitoxin reduces the effectiveness of therapy [12].

As noted during previous foodborne botulism outbreaks, not all persons who eat food contaminated with botulinum toxin become ill [5, 13]. The relatively low attack rate of 63% among those who ate the chili dish served at the church supper may be explained by uneven distribution of toxin in the chili, a dose-response relationship, or unrecognized host factors that confer resistance to ingested botulinum toxin. Because only the less severely ill patients reported the amount of chili eaten, the existence of a dose-response relationship could not be determined. We documented the presence of botulinum toxin in stool specimens obtained from 1 person who had only gastrointestinal symptoms and 1 person who had no symptoms at all. Asymptomatic individuals with electromyographic evidence or laboratory confirmation of botulism were identified in the 1994 El Paso, Texas, outbreak associated with consumption of contaminated baked potatoes [5]. These outbreaks highlight the fact that the clinical spectrum of botulism includes asymptomatic intoxication, in addition to mild gastroenteritis and cranial nerve symptoms.

All 16 cases in this outbreak were associated with consumption of contaminated brand X chili. Consumers may believe that consumption of a precooked product could not cause botulism, even if the product is left at room temperature. *C. botulinum* spores are ubiquitous in the environment and may have been present in the chili during production at the manufacturing plant. Because the brand X chili did not undergo retort cooking or similar spore-killing processes, the presence of spores is not surprising. With a documented pH of 5.47, the chili was not acidic enough to prevent the germination of *C. botulinum* and other bacterial spores. The only barrier to *C. botulinum* growth and toxin production in brand X chili was adequate refrigeration. Additional barriers to spore germination include increased salinity, decreased water activity, and a pH of <4.6 [14]. Manufacturers of ready-to-eat products, such as frozen chili, should consider the incorporation of multiple barriers to prevent the germination of clostridial spores.

Storage of the chili at inappropriate temperatures, which allowed spore germination and toxin production, may have occurred at any point between production and consumption. Given the absence of any botulism cases linked to brand X chili outside the Dallas–Fort Worth area, the absence of toxin in 2 chili tubs obtained from a distributor, and the lack of serious violations at the manufacturing plant, it is unlikely that toxin production occurred at the manufacturing plant. It is also unlikely that the consumers mishandled the chili sufficiently to allow toxin production, because the cook for the church purchased the frozen chili only a few hours before the event and kept it refrigerated until cooking it. All illness in this outbreak was related to chili purchased at the salvage store, where some foods requiring refrigeration were kept at room temperature on Saturdays. Such foods could have undergone many cycles of thawing and freezing. On the basis of the gross mishandling of other refrigerated products and the strong epidemiologic link, we conclude that storage of the chili at inappropriate temperatures, allowing for production of botulinum toxin, most likely occurred at the salvage store.

To our knowledge, this is the first outbreak linked to food sold at a salvage store. We surveyed food regulatory agencies in all 50 states to determine whether salvage stores are regulated and inspected by individual states. Of 28 states responding to the survey, 26, including Texas, regulate and inspect salvage stores. Although only 4 states have adopted the 1984 Model Food Salvage Code prepared by the Association of Food and Drug Officials and the US Department of Health and Human Services, several states have based their regulations on the model code [15]. Despite routine inspections of the Fort Worth salvage store, gross mishandling may have taken place, especially during Saturday sales, because inspections usually took place during weekdays. Although most states mandate the inspection of salvage stores, we recommend that states conduct a full review of regulations governing salvage stores and of the adequacy of inspections. In light of the dangerous practices noted at the Fort Worth salvage store, more frequent and rigorous unannounced inspections, including inspections on weekends, may be necessary to detect and prevent similar food mishandling.

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