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. 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.

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. Most outbreaks of foodborne botulism are caused by home-canned foods. However, several large outbreaks in recent decades have been associated with commercial or restaurant foods. 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.

OUTBREAK RECOGNITION

On the evening of 29 August 2001, 4 men were admitted to a hospital in Dallas with cranial neuropathy and symmetric descending flaccid paralysis. The diagnosis was delayed in 3 cases. Fifteen (63%) of 24 attendees who ate a chili dish developed botulism. 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 IN HUMANS

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. 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.

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. Most outbreaks of foodborne botulism are caused by home-canned foods. However, several large outbreaks in recent decades have been associated with commercial or restaurant foods. 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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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 of botulism from food served at a church supper to be laboratory-confirmed botulinum intoxication or ≥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 ≥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 non-intact 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.

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

\textbf{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.

\textsuperscript{a} Outlier.

\textsuperscript{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.

\textbf{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.](https://academic.oup.com/cid/article-abstract/37/11/1490/372419)
Botulism from Food Sold at a Salvage Store

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.

Acknowledgments

Our deepest appreciation goes to the patients and families, who demonstrated remarkable patience and strength during very trying times. Many thanks go to Brad Walsh, Seri Lang, James Zoretic, James Perdue, and Peter Wolf, for their tireless assistance in the field; and to Sesonne Alexander and Maurice Padilla, for their excellent technical assistance in the laboratory.
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