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SUPPLEMENT ARTICLE

Outbreak of Foodborne Botulism in an Immigrant Community: Overcoming Delayed Disease Recognition, Ambiguous Epidemiologic Links, and Cultural Barriers to Identify the Cause

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We describe a botulism outbreak involving 4 Middle Eastern men complicated by delayed diagnosis, ambiguous epidemiologic links among patients, and illness onset dates inconsistent with a point-source exposure. Homemade turshi, a fermented vegetable dish, was the likely cause. Patients ate turshi at 2 locations on different days over 1 month.

Keywords. botulism; foodborne illness; outbreak.

Botulism is a rare illness characterized by a descending flaccid paralysis that can lead to respiratory failure and death [1]. Myasthenia gravis and the Miller-Fisher variant of Guillain-Barré syndrome (GBS) are the illnesses most commonly confused with botulism; cases of both are typically sporadic [1]. Laboratory confirmation of botulism requires testing of food or clinical specimens obtained when toxin is circulating, which is typically early in the illness course and before antitoxin is administered [2].

A suspected case of botulism is a public health emergency because a single case could herald an outbreak. Typically, outbreaks are associated with a point-source exposure; most illnesses begin within 48 hours of exposure and not later than 10 days [3]. Illnesses beginning >10 days apart or among unlinked persons raise concern about a contaminated food that is shelf-stable and may be widely distributed. In these instances, the urgency of identifying and removing the food is especially high because of the increased likelihood of additional cases [4].

On 17 November 2013, a resident physician at hospital A in Amarillo, Texas, admitted a 24-year-old Middle Eastern man with descending paralysis diagnosed as myasthenia gravis (patient 1). On 5 December he admitted a 32-year-old Middle Eastern man with an acute illness diagnosed as GBS (patient 2). Conversing with hospital visitors, the physician learned that the 2 men were friends, and that a third friend (patient 3) had been admitted to a different medical team at hospital A with a diagnosis of atypical myasthenia gravis. Suspecting a common etiology, the physician consulted medical textbooks and decided botulism was a possibility. He contacted local, state, and federal public health authorities on 6 December. Soon thereafter, the physician described the cases to a colleague at a nearby hospital (hospital B) who said he too was caring for a Middle Eastern man with descending paralysis diagnosed as GBS (patient 4).

THE INVESTIGATION

After extensive interviewing, illness onset dates for patients 1 and 4 were determined to be in early November, and those for patients 2 and 3 more than 3 weeks later. On physical examination, all had signs and symptoms typical of foodborne botulism, including gastrointestinal complaints, bilateral cranial nerve deficits, and descending paralysis. Patients 1 and 4 were improving by the time botulism was suspected, so botulinum antitoxin was not administered. Specimens for patients 1 and 4 were collected 30 and 32 days after illness onset; specimens from patients 2 and 3 were collected 12 and 8 days after illness onset, after antitoxin was administered. Neither botulinum toxin nor *Clostridium botulinum* was detected in any patient stool or serum specimens.

We sent a public health alert to area healthcare providers and reviewed medical records flagged for botulism by syndromic surveillance; no additional cases were identified. We interviewed patients about epidemiologic links, shared exposures, and foods consumed to identify clues to the contaminated product. By the time of the investigation, hospital A had discharged patient 1 to family in Augusta, Georgia, where he was readmitted for persistent weakness.

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During early interviews, epidemiologic links between patients that would identify a common food exposure were difficult to establish. Patients 2 and 3 admitted being roommates in apartment A but patient 1 initially denied living there because he was not listed on the lease. Patient 3 was a long-haul truck driver who was out of town during the 6 weeks before patient 1 became ill and stayed at apartment A for only 1 day before his own illness. Patients 2 and 3 were adamant that an association between them and patient 4 was impossible since patient 4 belonged to a different Middle Eastern ethnic group. Patient 4 lived in a different apartment building in Amarillo, apartment B, and had been jailed in the 7 days before hospitalization. A search of apartment A did not yield any foods consumed by all 3 roommates and botulinum toxin was not detected in any of the 4 foods collected from the home.

After the first round of interviews, we could not determine whether all 4 ate the same contaminated food or whether additional persons could be at risk. During 24 hours of subsequent interviews conducted over 6 days, a clearer picture of events emerged (Figure 1). Fermented foods are known to be risky for botulism when prepared improperly. While inquiring about consumption of fermented foods, we learned that on 1 November, patients 1 and 4 ate homemade turshi, a fermented vegetable dish, at apartment B. Two days later, they ate breakfast with friends at apartment B. Turshi was not served at the breakfast but afterward, patient 1 took leftover turshi from apartment B to apartment A and put it in the refrigerator where, days later, he noted the turshi had a foul odor and placed it back in the refrigerator without eating it. Patients 2 and 3 consumed this turshi separately after patient 1 had been hospitalized. At the time of our interviews, patient 3 was intubated and severely paralyzed, except for his fingers and toes. When we queried him about the refrigerated turshi, he wrote, "I eat this turshi" on a notepad, a breakthrough moment after days of inconclusive interviews (Figure 2). Four additional persons who attended the apartment B breakfast also took turshi to their homes and consumed it, but denied any illness and refused to be evaluated, yielding an attack rate of 50%.

To prepare the turshi, patient 4 fermented broccoli, cauliflower, salt, and cabbage for 3 weeks at room temperature in a plastic container. He boiled beets and commercially canned garlic, then refrigerated these for 1 week in a metal pot, after which the mixture was drained and added to the fermented vegetables in the plastic container, along with vinegar. The mixture was refrigerated for 3 days to complete preparation; turshi is served cold. No turshi remained for testing. This was patient 4's first time making turshi.

DISCUSSION

An astute physician recognized an outbreak of botulism and investigators determined homemade turshi was the likely cause. Turshi was consumed at 2 locations on 3 days spanning nearly 1 month by persons not all known to each other. Repeated interviews were essential for elucidating the details, using information from other patients during interviews to build upon the story. Turshi was the only food eaten by all 4 patients, illness onsets correlate well with dates turshi was eaten, and its preparation included steps during which a nonacidic, warm, and anaerobic environment conducive for botulinum toxin production might have occurred [5, 6]. The attack rate of 50% is intriguing, and may be due to uneven distribution of toxin in food, unreported mild illness, or other factors [7].

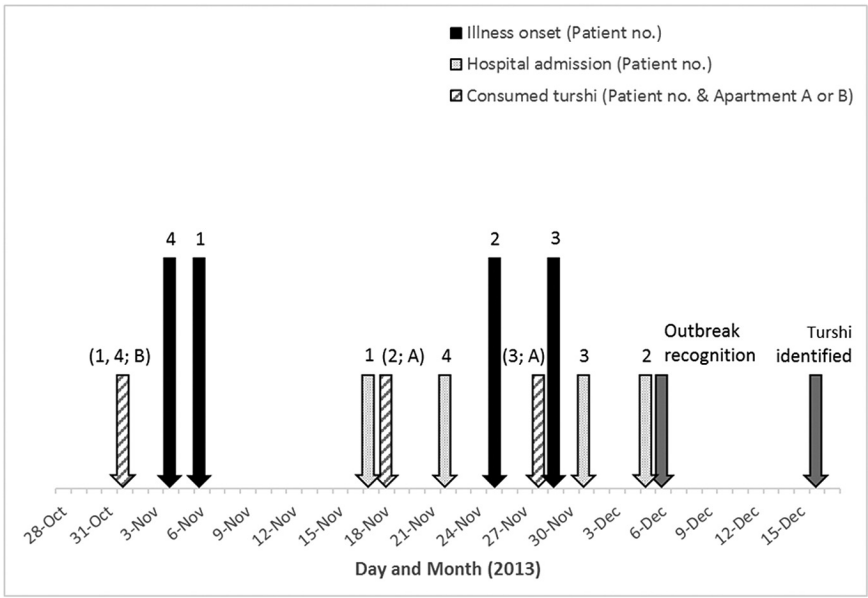

Figure 1. Select events of botulism outbreak investigation—Amarillo, Texas, November–December 2013.

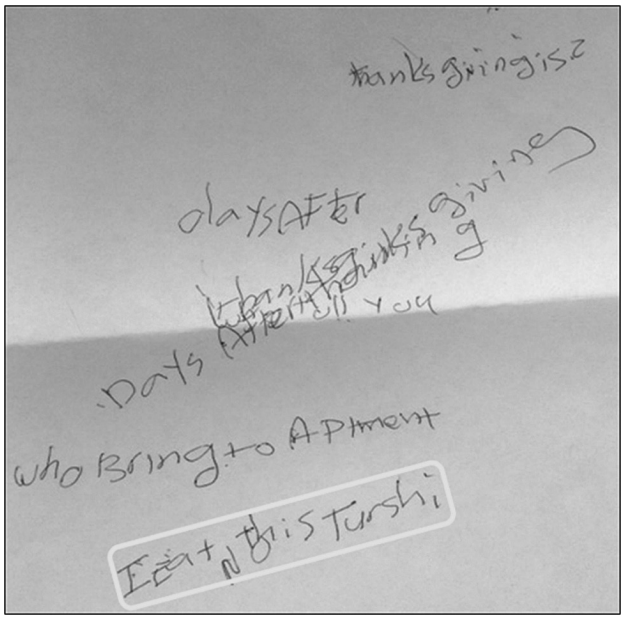

Figure 2. Sample of communication with patient 3 including "I eat this turshi"—Amarillo, Texas, 15 December 2013.

When healthcare workers recognize the signs and symptoms of botulism early, patients are treated sooner and have improved outcomes [2], and specimens can be collected early enough in illness to confirm botulism. All patients exhibited descending paralysis typical of botulism but each was misdiagnosed. Patients were in 3 hospitals and under the care of 4 clinical teams by the time of the investigation, further impeding recognition of the outbreak. Earlier consideration of botulism in patients 1 and 4 would have triggered a public health investigation sooner and might have prevented illness in patients 2 and 3. The occurrence of cranial nerve deficits followed by descending paralysis in >1 epidemiologically linked person, even without laboratory confirmation, is indicative of foodborne botulism.

Identification and removal of contaminated food hinges on identifying epidemiologic links between individuals. Early in the investigation, epidemiologic links between patients were unclear. We were concerned that the contaminated food could have been purchased at a store or consumed at a restaurant and that additional cases might be occurring. After the link among patients was recognized, we narrowed the focus to home-prepared foods.

Obtaining food histories is a challenging part of foodborne outbreak investigations because interviews are often conducted days to weeks after the contaminated food is consumed. In this outbreak, food histories were obtained from 2 patients >1 month after illness onset. Although patients understood and spoke English to varying degrees, language barriers still presented a challenge, particularly with regard to identification of ethnic foods unfamiliar to the investigators. Patient 3 had the most recent illness onset and likely the best recall and understanding of English, yet communication was exceedingly difficult because of intubation. We speculate that some of the vagueness of information initially collected was caused by reluctance of the patients, all immigrants, to identify other immigrants to authorities.

In conclusion, this investigation demonstrates the difficulties in recognizing botulism, confirming the diagnosis, and implicating a contaminated food that is unfamiliar to investigators. Botulism should be considered when myasthenia gravis and GBS are suspected; multiple cases of descending paralysis are almost pathognomonic for botulism [8]. Physicians obtaining a history can recognize outbreaks by asking whether similar illnesses have occurred among persons known to each other. Public health authorities should be notified immediately for expert clinical consultation, provision of botulinum antitoxin, investigation of possible exposures, and assistance with confirmatory testing.

Notes

Disclaimer. The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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References

1. Sobel J. Botulism. Clin Infect Dis **2005**; 41:1067–173.
2. Sobel J. Diagnosis and treatment of botulism: a century later, clinical suspicion remains the cornerstone. Clin Infect Dis **2009**; 48:1674–5.
3. Hughes JM, Blumenthal JR, Merson MH, Lombard GL, Dowell VR Jr, Gangarosa EJ. Clinical features of types A and B food-borne botulism. Ann Intern Med **1981**; 95:442–5.
4. Shapiro RL, Hatheway C, Swerdlow DL. Botulism in the United States: a clinical and epidemiologic review. Ann Intern Med **1998**; 129:221–8.
5. Wainright RB, Middaugh JP, Hatheway CL, et al. Food-borne botulism in Alaska, 1947–1985: epidemiology and clinical findings. J Infect Dis **1988**; 157:1158–62.
6. US Food and Drug Administration. Fish and fishery products hazards and controls guidance. 4th ed. **2011**. Available at: https://www.fda.gov/downloads/Food/GuidanceRegulation/UCM251970.pdf. Accessed May 2017.
7. Kalluri P, Crowe C, Reller M, et al. An outbreak of foodborne botulism associated with food sold at a salvage store in Texas. Clin Infect Dis **2003**; 37:1490–5.
8. Sobel J. Botulism. In: Lutwick LI, Lutwick SM, ed. Bioterror: the weaponization of infectious disease. Totowa, NJ: Humana Press, **2009**.