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Telephone 503/731-4023
Emergencies 503/731-4030
Fax 503/731-4798
cd.summary@state.or.us

CENTER FOR DISEASE PREVENTION & EPIDEMIOLOGY • OREGON HEALTH DIVISION

HOME-CANNED ENTEROTOXIN

IT WAS A TUESDAY in late February when "Katherine," a 70-something Oregon woman with a history of diabetes, began to experience double vision and dizziness. The next day she saw an ophthalmologist, who found no obvious explanation for her condition. Meanwhile, her adult daughter "Susan," with whom she lived, was also not feeling well. The previous week, Susan had seen her physician with a complaint of dry cough, low grade fever, sore throat, and myalgias, but now on Wednesday morning she presented to the ER with a complaint of diplopia— "just like her mother," she noted. She was given antibiotics for possible sinusitis, but felt much worse by the next day, with nausea, diarrhea, worsening diplopia, and hoarseness.

Her mother was also deteriorating, and on Friday was admitted with diplopia, dizziness, dysarthria, marked dysphonia, and dysphagia. Susan herself was admitted to the same hospital later that day with most of the same symptoms as well as increasing shortness of breath and weakness in her extremities. The coincidence of both mother and daughter developing such symptoms naturally heightened concern. Susan volunteered that on Monday they had eaten home-canned beets, vintage 1986. Nonetheless, Katherine was diagnosed with a brain stem stroke, and her daughter was thought to be experiencing conversion hysteria.

Friday night and over the weekend botulism was the subject of discussion among the medical staff, but no one considered it a very plausible explanation, and no one called the health department. On Monday the infection control nurse contacted the local health department to bruit the possibility. Minutes later these concerns were shared with Health Division epidemiologists, who, after a hasty phone consultation with attending physicians, made arrangements with CDC to fly botulinum antitoxin to the patients from the nearest quarantine station (Seattle). Before the antitoxin arrived, Katherine suffered a pulmonary arrest and was intubated. Susan was airlifted to Portland and intubated shortly after arrival.

Subsequent laboratory data were consistent with foodborne botulism. When Susan was tested by electromyography using rapid (30 Hz), repetitive stimulation, muscle action potentials were augmented. Although serum collected from both patients on Friday tested negative for botulinum toxin by mouse bioassay at the public health lab, mice injected with supernatant from the jar of leftover beets developed characteristic signs and died.

The daughter was treated with supportive care and a tracheostomy. She was discharged on her 26th hospital day, still requiring ventilatory assistance, and is currently breathing on her own and undergoing physical rehabilitation. Her mother did not fare as well. After a complicated hospital course, she died on the 64th hospital day subsequent to a cardiopulmonary arrest.

BAD BURRITO?

Earlier in February, a 63-year-old man had presented to a Portland-area emergency room with a one-day history of diplopia, dysphagia, dysphonia, and the sensation of having a "thick tongue." The previous morning, he awoke with nausea and vomiting [sick]. Later that day, he developed a sore throat. He denied any recent history of home-canned food consumption.

The patient was afebrile, and his mental status was normal. His speech was slurred, and he had a slight left facial droop, mild nystagmus, and a diminished gag reflex. The admission diagnosis was probable stroke. Head CT scan was unrevealing. His cranial nerve weakness progressed, and by the second hospital day, he was unable to open his eyes or extend his tongue, evidenced bilateral ophthalmoplegia, and had developed proximal extremity weakness. Paralysis progressed, and the patient suffered a respiratory arrest. Although botulism was thought unlikely because of the unsuggestive food history, the attending physician discussed the case with Health Division epidemiologists, and serum and stool specimens were collected and sent to the public health lab. After consultation with CDC, botulinum antitoxin was obtained and administered. Because the provisional diagnosis was Guillain-Barré syndrome (Miller Fisher variant), plasmapheresis was begun, and the patient seemed to improve. He was weaned from the ventilator on the 16th hospital day and three days later transferred to a skilled nursing facility for rehabilitation. As of this writing, the patient is home and nearly fully recovered.

No toxin was identified in serum or stool, but *Clostridium botulinum* was eventually cultured from the latter. Although unconfirmed, the likely source was a commercially packaged frozen chicken and bean burrito that the patient had heated in a microwave and then held in a [not very] warming oven (~38°C, per subsequent testing) for 24 hours before tasting. It "wasn't very good,"* and was discarded after that first bite.

ABOUT BOTULISM

These are the first cases of foodborne botulism reported in Oregon since 1992, when pumpkin pie preparations went awry. (Seven cases of infant and one case of wound botulism have been reported.) Botulism is a paralytic illness caused by the action of one of several extraordinarily potent neurotoxins that prevent the release of acetylcholine at the neuromuscular junction. Once the heat-labile toxin is ingested, symptoms usually begin within 18-36 hours (range, 6 hours-10 days) after ingestion. Botulism in humans is almost always caused by toxin types A, B, or E—all produced by *C. botulinum,* an anaerobic, spore-forming, Gram-positive bacillus.

Botulism should be considered in the differential diagnosis of Guillain-Barré syndrome, myasthenia gravis, stroke, anticholinergic and other drug toxicities, and tick paralysis, among other neuromuscular maladies. In botulism, the cranial

* surprise, surprise, surprise!

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nerves are generally the first to be affected, and patients typically present with bilateral, descending paralysis. Clinicians should be mindful of the "4 D's": diplopia, dysphagia, dysarthria, and xerostomia.[1] Blurred vision and ptosis are also common, and many patients initially complain of having a "thick tongue." Mild cases can be difficult to diagnose. In a Georgia outbreak, for example, one case presented with blurred vision and was diagnosed with astigmatism.[2] In more severe cases, paralysis of the extremities and of the muscles of respiration ensues. Convalescence can be protracted (often weeks to months on a ventilator), but full recovery is common. Fatal complications (e.g., pulmonary embolism) often stem from the prolonged inactivity.[2]

As with so many diseases today, prompt and accurate diagnosis can improve the prognosis. Antitoxin given *early* in the course of illness can reduce the duration and severity of illness. In the United States, antitoxin is only available from federal quarantine stations and can only be obtained after consultation with state and CDC epidemiologists. Patients with suspected botulism should have their respiratory functions closely monitored, with serial measurements of vital capacity. Intubation may be required.

The diagnosis of botulism can be difficult to confirm. No specific tests are available commercially. The EMG finding of muscle action potential augmentation with rapid, repetitive stimulation is suggestive of botulism. Serum (≥5 ml), stool (≥15 g, fresh), foods, and gastric aspirates can be tested for toxin by injecting extracts into mice, which develop characteristic signs and die if toxin is present. Stool can also be cultured for *C. botulinum*. **Treatment with antitoxin should never be delayed pending laboratory confirmation.** Available diagnostic tests are insensitive. Serum tested within 2 days after toxin ingestion is positive only about 60% of the time, and sensitivity declines rapidly.[3] Moreover, these tests take several days to complete. The antitoxin only acts on unbound toxin; once bound, it's just a waiting game as neural function returns.

FOLLOW THE LINKS

Botulism* was described and linked to eating blood sausage in the 18th century; but since World War I, home-canned foods have dominated the foodborne botulism scene. Alaska routinely reports more foodborne botulism cases than any other state, because of the preference of Alaskan Natives for seal muktuk, "stink heads" (fermented fish heads), and other traditional delicacies. In the lower 48, recent vehicles have included home-canned salsa and asparagus. Products kept in oil (e.g., garlic[4] and onions[5]), have been recently identified as vehicles, as have baked potatoes wrapped in aluminum foil and held at room temperature.[6] The common themes are foods contaminated with soil bacteria and then incubated in an anerobic environment: "spoiled, oiled, or foiled." Novel vehicles continue to be identified, however, so diagnosis should not depend on a history of eating a "classic" food item. Safe canning recipes, incidentally, are available from Extension Services and other sources.

REPORTING SAVES LIVES

If botulism is being even half seriously considered, you should be discussing the case with the health department. There are at least 3 reasons† to call public health officials **immediately** (day or night) whenever foodborne botulism is suspected. First, prompt administration of antitoxin is important for the patient, and you can only get it through us. Second, most clinicians will never see a case, but we have had occasion to consult on several cases; some of us have even seen multiple patients with botulism. We can offer suggestions for diagnostic testing, including specimen collection and processing through the public health lab. Third, foodborne botulism is a true public health emergency. While the potential for widespread disaster is obvious with commercial products, even homemade foods can be distributed to numerous households. In outbreaks involving multiple victims, patients may present in a variety of clinical settings—even in multple states, emphasizing the need to involve public health agencies from the outset. We take seriously our obligation to identify possible sources and get them off the shelves.

REFERENCES

1. Hughes JM, Blumenthal JR, Merson MH, Lombard GL, Dowell VR, Gangarosa EJ. Clinical features of types A and B food-borne botulism. Ann Intern Med 1981; 98:442-445.
2. Townes JM, Cieslak PR, Hatheway CL, et al. An outbreak of type A botulism associated with a commercial cheese sauce. Ann Intern Med 1996; 125:558-563. (Congratulations John and Helen!)
3. Woodruff BA, Griffin PM, McCroskey LM, et al. Clinical and laboratory camparison of botulism from toxin types A, B, and E in the United States, 1975-1988. J Infect Dis 1992; 166:1281-1286.
4. St. Louis ME, Peck SHS, Bowering D, et al. Botulism from chopped garlic: delayed recognition of a major outbreak. Ann Intern Med 1988; 108:363-368.
5. Macdonald KL, Spengler RF, Hatheway CL, Hargrett NT, Cohen ML. Type A botulism from sauteed onions. JAMA 1985; 253:1275-1278.
6. Seals JE, Snyder JD, Edell TA, et al. Restaurant-associated type A botulism: transmission by potato salad. Am J Epidemiol 1981; 113:436-444.

* from Latin *botulus*; sometimes from Polish *kielbasa*.

† and we're not even counting ORS 433.004(2).