



Botulism Associated with the Consumption of Home Canned Beets—A Case Report

Introduction: On April 15, 2010, the Mississippi State Department of Health (MSDH) Office of Epidemiology received a telephone call from a central Mississippi hospital requesting information on *Clostridium botulinum* toxin testing. The initial clinical report indicated that an elderly adult female had developed an acute, progressive, descending paralysis of several days duration that was consistent with botulinum intoxication. Investigation of the report led to an epidemiological link to the consumption of home canned beets. The patient received botulinum antitoxin within eleven hours of the initial report, and is currently admitted clinically improving. The beets and a serum sample from the patient ultimately tested positive for botulinum toxin type B.

Case Presentation: The patient is a 70 year old female with an underlying history of multiple medical problems including multiple myeloma, atrial fibrillation and a lower extremity peripheral neuropathy from prior chemotherapy. She was admitted to the hospital through the emergency department on April 10, 2010 with a one day history of slurred speech, confusion and dizziness and lower abdominal discomfort. Over the next 24 hours the patient complained of dysphagia, shortness of breath, and subjective weakness. By April 13, 2010 she developed full cranial nerve deficits with paralysis of the eye muscles, facial paralysis, and upper extremity weakness, but retained some strength in her lower extremities. She ultimately went into respiratory failure requiring ventilation. Based on her clinical presentation of descending flaccid paralysis, the local physician notified MSDH to facilitate *Clostridium botulinum* toxin testing.

The Centers for Disease Control and Prevention (CDC) was contacted for consultation regarding the potential testing of clinical samples and release of botulinum antitoxin. The local physician was put into contact with the CDC Botulism Officer to discuss the patient's condition and treatment. After these discussions, the initial impression was that the patient's symptoms were consistent with botulism, but an exacerbation of her multiple myeloma could not be ruled out. The decision was made to not release botulinum antitoxin until further investigation could establish an epidemiological link to potential botulism.

Figure



The local Public Health District V Health Officer and Epidemiology staff were contacted to begin the investigation. At the time, the patient was unable to give a food or exposure history. District V staff were able to locate and interview several family members. The initial report from the patient's family indicated no history of consumption of home canned foods or other at risk exposures prior to the onset of symptoms. Food history details were limited, however, because the patient lives alone. With the help of family members, the District V epidemiology nurse was able to obtain access to the patient's home to look for evidence of any foods posing a risk for botulism. The nurse was quickly able to identify an opened jar of home canned beets on the kitchen counter (Figure), establishing a potential link to botulinum intoxication.

Both the CDC Botulism Officer and the local physician were informed of the discovery. Within four hours of the initial call to MSDH, approval for the release of antitoxin was obtained, and the infusion of antitoxin was begun within 11 hours of first notification.

Prior to the antitoxin infusion, serum and gastric content specimens were collected, and along with the home canned beets, were sent to CDC for evaluation. Testing there confirmed the presence *Clostridium botulinum* toxin type B in the canned beets and in the serum sample. The District V staff determined that the canned beets were given to the patient by an elderly neighbor. The patient was given the beets two days prior to the onset of her symptoms. A home visit to the neighbor determined that the neighbor had canned beets and other vegetables that had been given to the neighbor's son and to the patient. All had been consumed previously with the exception of two additional jars of beets discovered under the neighbor's sink. These jars were disposed of by MSDH.

The patient initially improved after the antitoxin infusion, but again developed respiratory distress, likely due to aspiration, and required re-intubation and ventilation. She was ultimately transferred to a restorative care facility on April 27, and is now undergoing rehabilitation with clinical improvement.

Discussion: *Clostridium botulinum* is an anaerobic spore-forming bacterium that produces a potent neurotoxin. Of the seven identified subtypes of neurotoxin, four are known to cause human illness (A, B, E, and rarely F). Clinical illness is caused by the neurotoxin rather than the organism or spores themselves. *Clostridium botulinum* spores are ubiquitous in the environment and are recovered from soil, agricultural products such as honey, and from the gastrointestinal tract in animals.

There are four types of naturally occurring botulism:

- **Foodborne botulism**—occurs after ingestion of the preformed neurotoxin in contaminated foods. The most commonly associated foods are low acid canned foods such as meat, seafood and vegetables. Symptoms may begin within hours to days after ingestion and usually follow the classic pattern of flaccid descending paralysis with cranial nerve involvement.
- **Wound botulism**—occurs when a wound is contaminated with spores that germinate, reproduce and produce neurotoxin. The symptoms are similar to those of foodborne botulism; however symptoms may not develop until up to two weeks after exposure.
- **Infant botulism**—the most common type of botulism. It is generally seen in children <12 months of age; most commonly in children aged six weeks to six months of age. In this form the *Clostridium botulinum* spores germinate in the intestinal tract and produce toxin. Ingestion of raw honey contaminated with spores is often associated with infant botulism, and infants should not be fed raw honey before the age of one year. The most common symptoms are constipation, loss of appetite and feeding difficulty, and loss of head control. The incubation time is unknown.
- **Adult intestinal toxemia botulism**—similar to infant botulism. Ingested organisms produce toxin in the intestine of immunocompromised adults, or in adults with functional or anatomical intestinal abnormalities.

Inhalational botulism occurs through the inhalation of aerosolized neurotoxin, and may be seen in occupational exposure; an intentional bioterrorist event could also result in inhalational botulism.

Differential diagnoses include Guillain-Barré Syndrome, Myasthenia Gravis, Miller Fischer variant Guillain-Barré Syndrome, CNS infections, and cerebrovascular accident (CVA). Diagnosis should be made based on clinical findings with consideration of environmental exposures. Antitoxin is most effective when given within 24 hours of symptom onset. Evaluation for treatment should not be delayed for laboratory confirmation. If a clinician is considering botulism in the differential diagnosis, they should immediately contact the MSDH Office of Epidemiology to arrange for a consultation with a CDC Botulism Officer. Call 601-576-7725 during normal business hours or 601-576-7400 for nights, holidays and weekends.

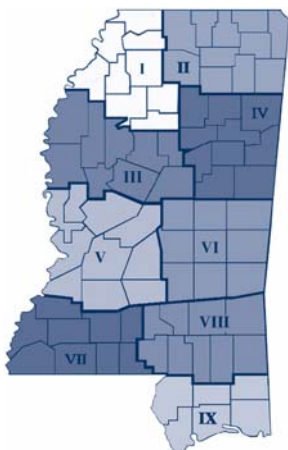
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Mississippi

Provisional Reportable Disease Statistics

April 2010



		Public Health District									State Totals*			
		I	II	III	IV	V	VI	VII	VIII	IX	Apr 2010	Apr 2009	YTD 2010	YTD 2009
Sexually Transmitted Diseases	Primary & Secondary Syphilis	1	0	1	1	3	6	0	5	6	23	22	63	70
	Total Early Syphilis	2	0	6	1	14	6	0	12	8	49	51	172	166
	Gonorrhea	57	41	95	36	146	60	24	55	68	582	653	2040	2470
	Chlamydia	252	194	310	183	525	212	94	224	231	2225	2266	7455	8215
	HIV Disease	2	4	5	4	18	1	1	3	4	42	46	174	216
Mycobacterial Diseases	Pulmonary Tuberculosis (TB)	1	0	1	1	5	0	0	0	0	8	12	23	25
	Extrapulmonary TB	0	0	0	0	1	0	0	0	0	1	3	4	5
	Mycobacteria Other Than TB	1	3	2	2	10	1	0	0	4	23	31	154	117
Vaccine Preventable Diseases	Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0
	Pertussis	0	0	0	0	1	0	0	0	0	1	2	17	25
	Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	0
	Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0
	Measles	0	0	0	0	0	0	0	0	0	0	0	0	0
	Mumps	0	0	0	0	0	0	0	0	0	0	0	0	0
	Hepatitis B (acute)	0	0	0	1	3	0	0	0	0	4	1	9	7
	Invasive <i>H. influenzae b</i> disease	0	0	0	0	0	0	0	0	0	0	0	0	0
	Invasive Meningococcal disease	0	0	0	0	0	0	0	0	0	0	1	2	2
Enteric Diseases	Hepatitis A (acute)	0	0	0	0	0	0	0	0	0	0	1	0	5
	Salmonellosis	2	5	1	2	3	3	1	3	5	25	27	81	125
	Shigellosis	2	0	0	0	1	0	0	0	0	3	1	10	9
	Campylobacteriosis	1	0	1	0	0	1	0	1	0	4	9	30	36
	<i>E. coli</i> O157:H7/HUS	0	0	1	0	0	0	0	0	0	1	2	5	5
Zoonotic Diseases	Animal Rabies (bats)	0	0	0	0	0	0	0	0	0	0	1	0	1
	Lyme disease	0	0	0	0	0	0	0	0	0	0	0	0	0
	Rocky Mountain spotted fever	0	0	0	0	0	0	0	0	0	0	1	0	1
	West Nile virus	0	0	0	0	0	0	0	0	0	0	0	1	1

*Totals include reports from Department of Corrections and those not reported from a specific District.

2009-2010 Influenza Update

The official CDC reporting period for the 2009-2010 influenza season ended the week of May 22, 2010. The season was significant for the emergence of 2009 H1N1 influenza A, first identified as causing infections in late April 2009 and resulting in an influenza pandemic by early June 2009. MSDH instituted enhanced influenza surveillance to identify cases of 2009 H1N1 and monitor influenza-like illness (ILI) activity throughout the state. The first Mississippi case of 2009 H1N1 was confirmed in mid-May 2009; ILI activity continued at low levels throughout most of the summer months. In mid-August 2009, ILI activity began to increase, reaching a peak in the week of August 30-September 5, 2009 with a rate of 15.5% of all non-trauma patients seen at Mississippi's ILI sentinel surveillance sites exhibiting symptoms consistent with an ILI (fever $\geq 100^{\circ}$ F, and cough and/or sore throat). This peak was much earlier than previous influenza seasons, which traditionally peak in late December through mid-February. Mississippi reported "widespread" geographic occurrence of influenza from August 30-October 24th. ILI activity dropped below baseline in late October and continued to be below baseline until the end of the flu season.

During the 2009-2010 influenza season, there were 27 flu-related deaths reported to MSDH. These confirmed influenza deaths are only a small portion of all the influenza deaths that occurred during the season. Most deaths from influenza occur from complications of the virus and are diagnosed and treated largely using clinical signs and symptoms. Fourteen (52%) were 2009 H1N1 influenza A infections confirmed by PCR. The remainder of the deaths were associated with influenza A infections that were not subtyped; seven (26%) were influenza A by rapid antigen tests, three (11%) were influenza A by viral culture, and three (11%) were unsubtypeable influenza A by PCR. Four (15%) of the 27 deaths were pediatric deaths (in children less than 18 years of age). The deaths ranged in age from 13 days to 77 years, with a median age of 52.

Eighty-nine percent (524/589) of positive influenza samples in the Public Health Laboratory (PHL) during the 2009-2010 season were 2009 H1N1. The last positive sample for the season was an influenza B on April 20, 2010. MSDH will continue routine influenza surveillance through the summer to detect potential future waves of pandemic influenza.