

Full Length Research Paper

A case of food-borne botulism by consumption of home-made canned red pepper: Importance of the botulism antitoxin therapy

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Botulism is a rare but severe neuroparalytic disease, caused by *Clostridium botulinum* toxins. Botulinum toxin is one of the most potent toxins in nature. Foodborne botulism is caused by foods particularly canned foods (home/commercial) but also by sausages, meat products and seafoods. Here we described four adult foodborne poisoning cases caused by home-made canned red pepper. Intensive care monitoring and antitoxin therapy were initialised immediately. After the patients were intubated and antitoxins administered, symptoms improved in two days for one patient, seven days in another and twenty-one days in the remaining two patients. Rapid improvement of symptoms is thought to be related to intensive care and antitoxin therapy.

Key words: Botulinum intoxication, foodborne botulism, *Clostridium botulinum*, toxin, antitoxin.

INTRODUCTION

Botulism is a rare but severe neuroparalytic disease that can be fatal, caused by neurotoxins elaborated by *Clostridium botulinum*. If it is not treated immediately and appropriately, can lead to mortality (Tamer et al., 2007). *C. botulinum* is a gram-positive, spore-forming, strictly anaerobic bacillus and its spores are widespread throughout the world (Ayse, 2006). They occur in both cultivated and forest soils, bottom sediments of streams, lakes, coastal waters and in the intestinal tracts of fish and mammals, crabs and other shellfishes (<http://www.fda.gov/Food/FoodSafety/Foodbornellness/FoodbornellnessFoodbornePathogensNaturalToxins/BadBugBook/ucm070000.htm>). There are 7 antigenically different types of toxin referred as types A,B,C,D,E,F,G. Types A,B,E and F produce human disease (Ayse, 2006). *C. botulinum* spores are heat-resistant but the toxins are heat-labile and acid-resistant. Any type of food that is not very acidic (pH above 4.6) can support growth and toxin production by *C. botulinum*. The botulinum toxin is one of the most potent toxins known in nature and a very small amount (a few nanograms) of toxin can cause

Abbreviation: SPO₂, Oxygen saturation.

Illness (<http://www.fda.gov/Food/FoodSafety/Foodbornellness/FoodbornellnessFoodbornePathogensNaturalToxins/BadBugBook/ucm070000.htm>).

C. botulinum toxins are haematogenously disseminated to peripheral cholinergic synapses where toxins bind irreversibly to the receptor sites at the neuromuscular junction and other peripheral autonomic synaptic sites, and block acetylcholine release (Tamer et al., 2007).

Botulism was recently classified into five clinical forms: classical food-borne botulism, infant botulism, wound botulism, botulism of undetermined etiology and inadvertent botulism (Ayşe, 2006). Most of the outbreaks are associated with canned foods (home/commercial), sausages, meat products and seafoods (Zineb et al., 2002).

In foodborne botulism, symptoms are usually develop 18 to 36 h after ingestion of the food containing the toxin. Publications report a clinical duration between 4 h to 8 days. The symptoms include marked lassitude, weakness, nausea, vomiting, abdominal cramps, dry mouth, vertigo, difficulty in speaking, blurring of vision and diplopia. More than 90% of patients develop at least three of these symptoms (Alvoro et al., 2000).

The clinical diagnosis of food-borne botulism can be confirmed by detailed history of patients, assaying for the

toxin in serum, feces, gastric or vomitus contents and isolation of *C. botulinum* from the feces of the patient. Currently, the most sensitive and widely used method for detecting toxin is the mouse neutralization test. The duration of this test is 48 h. and the duration of the cultures of specimens are 5 to 7 days. As the diagnosis of the disease with laboratory analysis is therefore protracted and difficult, the history of patient and clinical symptoms are the most important diagnostic factors (Jim et al., 2006).

CASE REPORT

Four women patients, aged between 27 and 48 years were admitted to the hospital 36 h after the consumption home-made canned red pepper with nausea, vomiting and abdominal cramp. The cases were referred to the National Poison Center in thirty-seventh hour. Foodborne botulism was diagnosed in these patients from their histories of canned food and clinical symptoms. Intensive care monitoring and antitoxin therapy were initialised immediately.

Patient 1

The patient is 27 years old, female. Her general condition was good. She was conscious, co-operative and had spontaneous respiration. Initial vital signs were stable. The blood pressure was 120/70 mmHg, pulse rate was 60 bpm, respiration rate was 16 breaths/min. Laboratory results were within normal limits. Her arterial blood gases were normal. Oxygen saturation (SPO₂) was 99%. This patient was discharged 2 days after admission with fully recovery following antitoxin therapy.

Patient 2

This patient is 48 years old, woman. The general condition was moderate. She was conscious, co-operative, had spontaneous respiration. Her blood pressure was 130/60 mmHg, pulse rate was 80 bpm. Her chemistry tests and complete blood count results showed no abnormalities. The arterial blood gases were normal. SPO₂; 97%. But she had symptoms; as dry mouth, mydriasis, blurred vision and difficulty in speaking. This patient was monitored and antitoxin therapy was initialised. Symptoms resolved and she was discharged within 7 days.

Patient 3

A 48 year old woman, her general condition was bad. She had dry mouth, mydriasis, blurred vision, speech disorder, dysphagia, tachycardia, decreased deep tendon

reflexes, symmetrical muscle weakness and paresthesias in both extremities. On initial examination, her blood pressure was 140/70 mmHg, pulse rate was 68 bpm, temperature was 36.5°C. Arterial blood gases analysis were normal as follows; pH; 7.31, PaCO₂; 65 mmHg, PaO₂; 122 mmHg. The patient was monitored in intensive care and antitoxin therapy was administered immediately. In addition, physiotherapy was done for muscle weakness. She had hypercapnia, PaCO₂; 80 mmHg and respiratory distress, 3 days later. She was intubated immediately. The following day, her respiratory distress worsened with increasing oxygen requirement. Temperature began to rise 6 days later. The temperature was 39.5°C. Tracheal fluids were cultured and *Escherichia coli* was selected. Secondary infection (pneumonia) was diagnosed and antibiotic therapy was initialised. She had hyperthermia until tenth days. The temperature was normal, 37.0°C, 10 days later. The symptoms began to resolve within 19 days. She had no respiratory problem, temperature was 36.5°C and PaCO₂; 50 mmHg, 19 days later. She was discharged from the hospital after 30 days.

Patient 4

A 32 year old female, her general condition was bad and there were respiratory problems. The symptoms were dry mouth, mydriasis, blurred vision, speech disorder, dysphagia, tachycardia, tachypnea, decreased deep tendon reflexes, symmetrical muscle weakness and paresthesias in both arms and legs (especially the upper extremities). Initial vital signs were normal, blood pressure; 130/80 mmHg, temperature; 37.5°C, pulse rate; 76 bpm. But results of arterial blood gases analysis were as follows; pH; 7.19, PaCO₂; 88 mmHg, PaO₂; 277 mmHg. This patient had respiratory distress and was intubated and monitored in intensive care. Antitoxin therapy was initialised immediately. In addition; physiotherapy was done for muscle weakness too. The patient had fever 5 days later. The temperature was 39.5°C and pulse rate was 110 bpm. Secondary infection (pneumonia) was diagnosed after the culturing from the tracheal fluids. So, antibiotic was initialised immediately. After 9 days; she had no fever, and temperature was 36.5°C. The symptoms of this patient began to resolve within 21 days. She had no symptom, respiratory and circulatory functions showed stable and PaCO₂; 53 mmHg. So, the patient was discharged from the hospital after 30 days.

DISCUSSION

Foodborne botulism is a foodborne intoxication caused by the consumption of foods containing the neurotoxin produced by *C. botulinum*, especially canned foods (home/commercial), sausages, meat products and seafoods (Jim et al., 2006). In our case, intoxication is

related to home-made canned red pepper.

The new cases have been reported about outbreaks related to the baked potatoes, garlic in oil, sautéed onion, tomatoes, eggplant, lobster, green beans, spinach, asparagus, mushrooms, canned corn, home-made sauce, home-made salted or fermented fish, tuna fish, chicken and chicken livers, shellfishes and meats (rabbit, goat, pork)(<http://www.fda.gov/Food/FoodSafety/Foodbornellness/FoodbornellnessFoodbornePathogensNaturalToxins/BadBugBook/ucm070000.htm>).

The prevention of foodborne botulism is achieved by processing food in such a way as to kill spores, inhibit bacterial growth and denature preformed toxin. Canned foods are normally safe for consumption if they are too acidic, sterilised, heated (75 to 80°C), cooked or preserved in otherway. Fresh foods are no hazard for botulism (WHO, 1999).

Onset of symptoms in foodborne botulism is usually 18 to 36 h after ingestion of the food containing the toxin, although in past cases reported in the literature onset varied from 4 h to 8 days. More than 90% of patients have at least three symptoms. The initial symptoms of the foodborne botulism include marked lassitude, weakness, nausea, vomiting, abdominal cramps, dry mouth, vertigo, difficulty in speaking, blurring of vision, diplopia, mydriasis and nystagmus (Alvoro et al., 2000).

All of our patients had at least three of these following symptoms, nausea, vomiting, abdominal pain, lassitude and weakness within 36 h. Three of the patients also developed dry mouth, vertigo, difficulty in speaking, blurring of vision, diplopia, mydriasis and nystagmus.

Botulinum toxin causes dilated, fixed or poorly reactive pupils, tachycardia, hypertension, orthostatic hypotension and urinary retention. These may be followed by ptosis, ophthalmoplegia, dysarthria and dysphagia (Nilgun et al., 2004). The abnormalities of the cranial nerve are followed by a symmetrical descending pattern of weakness and paralysis. After the cranial nerves, the toxin affects the upper extremities, the respiratory muscles and the lower extremities. Respiratory depression is caused by respiratory muscle paralysis. It may lead to ventilatory failure and death (Nilgun et al., 2004; Michael, 1998).

After ingestion of suspect or contaminated food gastric lavage should be performed and activated charcoal should be given within 1 h. But in our cases, the patients came to the hospital after 36 h. Supportive treatment, especially adequate mechanical ventilation and botulism antitoxin therapy are the most important steps in the management of severe botulism. Ventilatory support may be required for a long period of weeks or months. Patients should be closely monitored since respiratory arrest can occur abruptly (Michael, 1998).

Botulism antitoxin that was prepared as a trivalent antitoxin for toxin A, B, E. (7500 IU of type A, 5500 IU of type B, and 8500 IU of type E antitoxins) was given to all of our patients by intravenously after the test for serum sensitivity. The test was done intradermally by injecting

0.1 ml. of a 1:10 dilution of antitoxin in saline (WHO, 1999).

The report of Jim et al. (2006) provides a brief review of *C. botulinum* and food-borne botulism that occurred in the United Kingdom between 1989 and 2005. In this report, the six incidents illustrate (33 patients with three deaths) the importance of the risk factors of poor processing or storage of commercially prepared foods, improper home preservation of foods and traveling to countries where botulism is much more common than in the United Kingdom. The symptoms were acute bilateral cranial nerve neuropathy, dry mouth, dysphagia, slurred speech, ptosis and ophthalmoplegia. Patients were afebrile, unless a secondary infection was present, sensory changes did not occur. Disease progression was marked by a symmetrical descending muscle weakness resulting in the loss of head control, hypotonia, generalized weak-ness, difficulty in breathing and eventually respiratory failure. If untreated, deaths were due to airway obstruction or respiratory failure. Inactivation of toxin in the patients were achieved by intravenous administration of antitoxin (Jim et al., 2006).

Our two patients had dry mouth, mydriasis, blurred vision, speech disorder, dysphagia, dysarthria, tachycardia and tachypnea. They had weakness and paralysis in the extremities (speciality upper extremities) and respiratory muscles. There were respiratory problems and hypercapnia. The general conditions were bad. One of these patients; in initial arterial blood gases results were normal, pCO₂; 65 mmHg and pH; 7.31, in 3rd day, pCO₂; 80 mmHg and pH; 7.21 and in 19th day, pCO₂; 50 mmHg and pH; 7.37. Another patient in day one had this; pCO₂; 88 mmHg and pH; 7.19 and had this in day, pCO₂; 53 mmHg and pH; 7.40.

If the mechanical ventilation is required for a long period of time (weeks to months), risks of medical complications (respiratory infections, acute respiratory distress syndrome etc.) increase significantly (Michael, 1998).

Our two patients had fever and respiratory distress. One of them had hypercapnia and respiratory distress, 3 days later. She was intubated immediately. This patient had fever 6 days later. The temperature was 39.5°C. Secondary infection (pneumonia) was diagnosed after the culturing and antibiotic therapy was initialised. She had hyperthermia until tenth days. Another patient had hypercapnia and respiratory distress in the first day. She was intubated and monitored in intensive care. The patient had fever 5 days later. Temperature was 39.5°C and pulse rate 110 bpm. Antibiotic was initialised immediately for secondary infection. She had no fever 9 days later.

Antitoxin is very effective in preventing progression of disease and shortening the duration of ventilatory failure. Antitoxin increases secretion of acetylcholine at nerve terminals and accelerates neuromuscular transmission. Antitoxin does not neutralize the toxin but can reduce

progression of disease (Jun, 2001; Amita et al., 2005).

In a case report of Tamer et al. (2007), a nine-year-old child diagnosed with botulinum intoxication caused by pickle and sausage from school cafeteria, before the symptoms began. In his first clinical examination: ataxia, speech disorder, fatigue, dry mouth, double vision, hypotonicity at the extremities, reduction of retching and swallowing reflex were observed as major symptoms. Oral food and drink were stopped to prevent aspiration. Respiratory support and botulinum antitoxin therapy were administered. Symptoms and hypotonicity recovered, respiratory and circulatory functions were stable after 7 days. Other people who ate the same foods had no findings (Tamer et al., 2007).

In a case report of Nilgun et al. (2004), the authors describe the follow up of the clinical pattern in a patient, 40 years of age, diagnosed with botulinum intoxication caused by home-made canned green beans. The patient had diplopia, blurred vision, weakness and paresthesias in all four extremities, generalised hypotonia difficulty of swallowing and breathing, bifascial paralysis, bilateral ptosis, bilateral total external ophthalmoplegia, bilateral mydriasis, dysphonia and dysphagia. All deep tendon reflexes were abolished. The patient had been taken to intensive care and antitoxin therapy was initialised immediately. Nerve conduction studies of sensory nerves were in normal limits. Electromyographic studies showed that there was no voluntary motor activity in fascial muscles while there was a decline in the recruitment pattern of extremity muscles. There was no change in the neurological examination of the patient 7 days later. The clinical improvement began gradually after this first week. Her clinical improvement became more clear in the second month's control and her neurological exam was found to be normal in the third month. Electrodiagnostic tests were repeated in the second week and in the third month. In the second week, the repetitive nerve stimulation test showed a mild decrement while it was normal in the third month and she was discharged from hospital in three months (Nilgun et al., 2004).

We conclude that the rapid improvement of symptoms in our and other cases is related to intensive care, ventilatory support and especially to use of antitoxin therapy. Diagnosis of the foodborne botulism based on

history and clinical presentation is most important. Following such diagnosis antitoxin therapy should be initiated immediately, even if the diagnosis is mostly determined on the clinical situation.

These cases provide a reminder to national poison center, public health and infectious disease, and microbiology professions about control, diagnosis and treatment of this rare but serious foodborne disease. Foodborne botulism is a national, emergency and public health problem for which acute treatment and care should be provided. Public education about botulism and its prevention is important.

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