Foodborne Botulinum Type E Intoxication Associated with Dried Bean Curd: First Case Report in Taiwan

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Abstract-

**Purpose:** Botulism type E intoxication is a rare condition among human botulism. We aim to describe a first case of botulism type E intoxication in Taiwan.

**Case report:** We report a 36-year-old young man with foodborne botulism type E associated with commercially vacuum packaged dried bean curd. He developed bilateral ptosis, diplopia and dysphagia 4 days after taking the dried bean curd. Electrophysiologic findings demonstrated waxing responses to 3 Hz repetitive nerve stimulation and decreased compound muscle action potentials on peripheral nerve conduction study. A bioassay for botulism in mice demonstrated that the patient had botulism caused by type E botulinum toxin. Antibodies to C. botulinum type E were identified from his serum, confirming the diagnosis.

**Conclusions:** This is the first known case of foodborne type E botulism in Taiwan. The potential source of this foodborne botulism should consider contaminated food made of soy beans.

**Key Words:** botulism, foodborne, dried bean curd, Taiwan

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INTRODUCTION

Foodborne botulism is an uncommon intoxication in Taiwan with the mean annual incidence rate of 3 cases per year\(^1\). The pathogen, Clostridium botulinum, which is an anaerobic gram-positive organism found in soil, produces seven different toxins designated A through G. The foodborne botulism is the most common form in the human botulism. The symptoms are caused by the irreversible binding of the neurotoxin to presynaptic terminals of motor neurons with resultant blocking acetylcholine release into the neuromuscular junction. Patients with botulism typically presents as a progressive, symmetrical, flaccid, descending paralysis with prominent bulbar involvement. In addition, symptoms of autonomic dysfunction including accommodative paralysis with mydriasis, anhydrosis with severe dry mouth and throat, and orthostatic hypotension are also associated. Respiratory distress due to failure of respiratory muscles and autonomic dysfunction is occasionally complicated...
in the extreme cases. Among the seven botulinum toxins, toxin type A, which is the most frequent cause of foodborne botulism, is often found in the home-canned food and toxin type B is found in the cooked pork meats. The incubation time from taking the contaminated food to the onset of neurological symptoms is between few hours to 1 week(2).

Between January 2007 and December 2009, only 20 cases were reported to the Centers for Disease Control (CDC), Taipei, Taiwan, and in the literature(1). Since then, there has been an upsurge of cases in this half a year(3). The new breakout risk group had taken the commercially vacuum-packaged dried bean curd, a traditionally Chinese food made of soy bean. All these reported foodborne botulism were caused by toxin type A and to a lesser extent, toxin type B(4-5). None has been reported other than these two types of toxin in Taiwan yet. Herein, we describe a case of a 36-year-old man who presented with typical botulism manifestations caused by toxin type E which was associated with dried bean curd use.

CASE REPORT

A 36-year-old healthy man without previous systemic disease before sought treatment at the outpatient clinic of our hospital. He reported right upper quadrant abdomen pain accompanied with diarrhea and vomiting for 2 days. The abdomen was tender at the right upper quadrant but without rebounding pain. Routine blood test results were unremarkable; an abdominal radiograph showed copious stool and gas. He reported he ate the commercially vacuum-packaged dried bean curd before this episode. One day later, he visited emergency department for new-onset binocular diplopia, swallowing difficulty, dry mouth and cold sweating. There were no more gastrointestinal symptoms and his vital sign was stable. The respiratory pattern was normal at that time and the routine blood test was within normal limits. He was soon admitted to the neurological ward. The neurological examinations revealed bilateral ptosis and mild facial diplegia. His pupils showed sluggish responses to light stimulation. He had minimal extraocular muscle motion limitations on the vertical gaze, normal palatal/gag reflexes but bilateral vocal cord palsy. The motor examination showed nearly complete paralysis of neck extensor and proximal upper extremities but preserved normal muscle strength in the hands and feet with symmetric deep tendon reflexes. Nasogastric tube was inserted soon after admission for his severe dysphagia and swallowing difficulty. Results of head magnetic resonance image (MRI) were unremarkable and did not reveal brainstem lesions. Cerebrospinal fluid values were within normal limits. The nerve conduction study (NCV) showed normal sensory nerve function but low amplitude on motor stimulation. The repetitive nerve stimulation was incomplete due to poor cooperation of the patient. Therefore, we can only complete the 3Hz frequency repetitive stimulation of spinal accessory nerve in this patient. We cannot clearly clarify the response to be incremental or decremental changes due to poor cooperation of the patient. However, the response showed waxing and waning pattern, which is suggestive of the diagnosis of neuromuscular junction disease.

Foodborne botulism was thus suspected and a bioassay for botulism was arranged by CDC, in which mice were injected with a sample prepared from the patient’s serum. The mice showed muscle weakness that was normalized by anti-serum against botulinum toxin type E. However, no pathogen was isolated from the patient’s feces.

Antitoxins to botulinum toxin types A, B, and E were then administered within 24 hours after admission. The patient recovered gradually from his predominantly proximal generalized weakness 2 days after antitoxins use. The nasogastric tube was successfully removed 2 weeks after admission and he was discharged on the same day, when he had nearly full muscle power of extremities. Regarding the source of contamination, the history revealed that a small amount of commercially vacuum-packaged dried bean curd had been taken. He denied exposed to honey, sea-food or other home-made canned food and there is no wound on his extremities. However, examination of foods at his home, including the dried bean curd, failed to identify the pathogen.
DISCUSSION

Our study is the first report to demonstrate a patient of botulism type E intoxication in Taiwan that possibly resulted from consumption of dried bean curd. Although we did not identify the bacterial pathogen, the Clostridium species, in the feces of the patient, the rapid response (within two days) of antitoxin and the positive bioassay test confirmed the diagnosis. Among the seven antigenically distinct botulinum toxins, types A, B, E, and, rarely, F cause most human illness\(^{(2)}\). As compared to toxin type A and B that are often associated with home-made canned food, botulinum toxin type E is often associated with consumption of improperly prepared foods of aquatic origin, either freshwater or marine\(^{(4,5)}\). Clinically, although botulism is characterized by motor symptoms of descending paralysis, gastrointestinal symptoms are more prominent in patients with types B and E intoxication than in patients with type A intoxication\(^{(1,4,8)}\). In addition, the gastrointestinal discomforts have not been reported in wound botulism, which is mostly caused by toxin types A and B, nor have similar signs been observed in animal studies in which pure toxin types A, B, C, or D were administered intragastrically or intravenously\(^{(7-9)}\). Compatible with these aforementioned literatures, prominent abdominal pain and diarrhea are noted preceding the neurological symptoms in our index patient. The mechanism why more gastrointestinal symptoms are caused by botulinum toxin E than other types remains unclear. Paradoxically, toxin type A and type E both cleave the same molecule, synaptosome-associated protein with relative molecular mass 25K (SNAP-25), in the presynaptic nerve terminal, which intuitively leads to predict that these two toxins may have identical effects on neurotransmitter release and clinical phenotypes\(^{(10)}\). However, the blockade of exocytosis is much more severe in the case of toxin E than toxin A in in vitro studies\(^{(11)}\). The mechanism includes that toxin A and toxin E may differentially affect the ability of SNAP-25 to bind the plasma membrane syntaxins, providing a possible explanation for the clinical differences in the botulinum intoxication\(^{(12)}\).

Another characteristic of our patient is that the possible contaminated food caused by toxin type E is the dried bean curd. It was originally thought that type E botulism was caused by fish or sea products in most countries\(^{(4,5)}\). However, Fu et al has recently reported that type E botulism in eastern countries is different from that in other areas of the world\(^{(13)}\). The foods most commonly associated with the disease are fermented grain and beans as well as raw meat. In 1990s, there was a suspected outbreak of type E botulism poisoning in the central coastal region of China and the most of the contaminated food were made of soy beans, including dou jiang (soy bean milk) and chou dou fu (fermented bean curd)\(^{(13)}\). These findings confirmed our observations that the possible contaminated food in our index patient was the dried bean curd, another kind of traditional food made of soy beans.

In conclusion, we identify the first patient of foodborne botulinum caused by toxin type E in Taiwan. The possible contaminated food is the commercially vacuum-packaged dried bean curd. Further investigation and outreach work may identify ways to decrease the risks associated with eating the soy-bean made food.

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REFERENCES


