

Outbreak of botulism type E associated with eating traditional soup in a family group, Loghman Hakim Hospital, Tehran, Iran

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ABSTRACT

Background: Botulism is a neuroparalytic illness caused by clostridium botulinum toxin that is usually transmitted as a foodborne disease. It is manifested by cranial nerve dysfunction and descending flaccid muscle paralysis.

Patients: Eleven members of a family developed severe weakness, lethargy and other clinical manifestations compatible with botulism following the ingestion of traditional soup. Stool, gastric fluid and serum samples were submitted for toxicological evaluation with standard mouse bioassay, while, only one patient demonstrated type E toxin. All patients received 3 monovalent antitoxins A, B and E, whilst most of their symptoms improved within 12 hours.

Conclusion: This study confirmed that prompt administration of antitoxin could prevent progression of the disease and may be a life-saving approach.

Keywords: *Botulism, Antitoxin, Outbreak.*

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INTRODUCTION

Clostridium botulinum spores are found throughout the world in soil samples and marine sediments (1). Botulism is a paralytic illness caused by neurotoxin of clostridium botulinum whilst foodborne botulism may develop after having foods contaminated with botulinum toxin. It usually develops 12-36 hours following the toxin ingestion; however, cases occurred as early as 2 hours or even 10 days later were also found in the literature.

Botulinum neurotoxins predominantly affect the peripheral neuromuscular junctions and autonomic synapses and the patient manifests with weakness (2,3).

Rarely, botulism may be confused with diphtheria, organophosphate poisoning and brainstem infarction (4); however, one should keep in mind magnesium intoxication that may mimic botulism (5). Clostridium botulinum toxins are categorized in seven types (A-G). While types A, B, and E comprise most of the human cases, F type toxin was rarely reported. Type E toxin has not proteolytic effects, therefore, the contaminated food does not have abnormal taste or even physical changes (2,3,6). The sufferers initially complaint of

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nausea and a dry mouth; however, cranial nerves palsy and descending flaccid muscle paralysis may develop later. This can involve the respiratory muscles as well.

Cranial nerve dysfunction could be manifested as blurred vision, ptosis, facial weakness, nystagmus, dysphagia, dysarthria and hypoglossal weakness. The following cardinal clues have been proposed by CDC:

- Fever is absent, unless complicating infections occur
- Neurological manifestations are usually symmetrical
- Patient remains responsive (in type E, lethargy is common) (7-9)
- Sensory deficits, except blurred vision, are rare.
- Heart rate is normal or decreased in the absence of hypotension.

Prompt administration of polyvalent equine antitoxin can decrease the progression of paralysis and severity of illness but will not reverse the existing paralysis. Thus, clinicians should not wait for laboratory confirmation of the diagnosis to commence therapy.

In Iran, Lapeyssonnic (a representative of WHO) reported botulism in a group of patients admitted to Poorsina Hospital in Rasht (northern part of Iran) about 40 years ago which was confirmed both toxicologically and clinically (6). Since then, several outbreaks of botulism have been reported in our country. This report summarizes a foodborne outbreak of botulism (type E toxin) in a family group in Tehran when they had a traditional soup (Ashmast).

PATIENTS

Eleven members of a family, including 5 males and 6 females, presented with severe weakness and lethargy 0.5-3 hours following the ingestion of traditional soup (Ashmast). All were admitted to Loghman Hakim Hospital among whom 2 were

hospitalized in ICU due to the severity of their symptoms. Their illness met the criteria of CDC for botulism. Their signs and symptoms are shown in table 1.

Table 1. Signs and symptoms of eleven cases admitted to Loghman Hakim Hospital due to botulism

Signs and symptoms	Number of sufferers (%)
Gastrointestinal	
Constipation	4(36.6)
Nausea	6(54.5)
Vomiting	3(27.3)
Neurologic	
Dry mouth	9(81.8)
Diplopia	5(45.5)
Dysarthria	6(54.5)
Blurred vision	7(63.6)
Ptosis	2(18.2)
Decreased gag reflex	3(27.3)
Fixed and dilated pupils	2(18.2)
Weakness	10(90.9)
Lethargy	8(72.7)
Others	
Dizziness	10(90.9)
Sore throat	2(18.2)

Immediately after admission, serum, stool and gastric specimens were collected and submitted to Pasteur Reference Laboratory for botulinium toxin detection on the same day, using the standard mouse bioassay technique. Unfortunately, sample of the traditional soup (Ashmast) was not available. Type E toxin was detected in serum and stool samples of one patient. All the other samples were negative for botulinium toxin. Electrophysiological studies revealed abnormalities compatible with botulism (figure 1). Based on clinical findings, all patients received three monovalent A, B and E antitoxins. Twelve hours later, lethargy, weakness, dysarthria, diplopia and blurred vision improved significantly, whereas fixed and dilated pupils and decreased gag reflex did not alleviate completely. Electrophysiological studies (ELMST: Eaten Lambert Myasthenic Syndrome Test) was

performed on the second day of admission on 7 patients and revealed normal motor and sensory results (median nerve: R=51, L=52m/s; peroneal nerve: R=41, L=41m/s; and normal F-wave and H-reflexes latency). ELMST of peripheral muscles showed a 10-12% increment in neuromuscular junction responses, normal EMG of distal and proximal regions and normal bulbar muscles. Fortunately, all patients were discharged with good condition after one week.

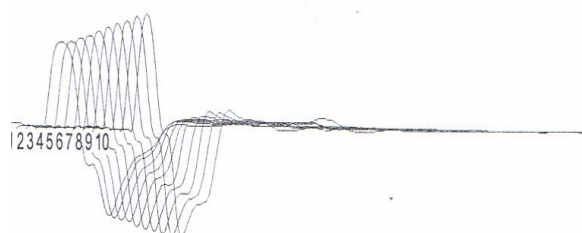


Figure 1. Incremental response of neuromuscular junction, compatible with botulism

DISCUSSION

Foodborne botulism is frequently recognized in outbreaks, whereas the other forms are sporadic. Home-canned vegetables, fruits and food products are now the most common sources. This outbreak reports foodborne botulism associated with an apparently normal traditional food product (Ashmast). Although all samples were immediately collected, only one yielded positive results for toxin. Bear in mind the limited sensitivity of mouse bioassay technique for botulinium toxin and the fact that test requires up to 4 days for completion, clinicians should not wait for laboratory confirmation to commence appropriate therapy. Severe weakness was the most common clinical manifestation among our patients. This is in agreement with another outbreak of type E botulism in Alaska in which weakness was reported among all 18 cases (100%), meanwhile, other manifestations were more or less the same (10). Nevertheless, sex, age, race and

socioeconomic status are not influencing factors in the epidemiology of botulism; however, seasonal distribution has been noted since outbreaks are more common in winter, just like our study. Improper food handling and storage in the winter may be a contributory factor. During 1990 and 1993, packaging raw vegetables in non-refrigerated airtight plastic containers was associated with botulism outbreaks in the United States (11). This may in part explain the outbreak of botulism type E in our patients since they used spinach which was stored in this way. Other food products such as peyote tea, hazelnut flavoring added to the yogurt, sweat cream cheese, and sautéed onion may also cause similar outbreaks (2). Additionally, yogurt and sautéed onion was used to prepare this traditional food and may play a causative role in our outbreak.

Among toxins, type E has the least incubation period, a finding that is in accordance with the short incubation period of 0.5-3 hours of our patients (9).

The Iranians should be instructed about the appropriate techniques of storage and preparation of traditional foods, otherwise, these products may entail hazardous complications. Prior investigators have reported that boiling food products for 10 minutes or using microwave oven for 3-5 minutes could inactivate the toxins (3).

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