CASE SERIES

JIACM 2024; 25 (3): 130-32

From Plate to Paralysis: A Rare Encounter with Food-Borne Botulism

Manjri Garg, Majaz Ahmad**, Renu Bala***, Deeksha Chaudhary****, Muskan Goyal****, Sandeep Goyal******

Abstract

Background: Botulism is a rare but fatal neuroparalytic syndrome, caused by a neurotoxin produced by bacteria of the genus Clostridium. Food-borne botulism is caused by ingestion of pre-formed botulinum neurotoxin. This syndrome courses initially with symmetrical cranial nerve palsy, may progress to descending flaccid paralysis and ultimately to respiratory arrest. Intensive management and antitoxin administration are crucial for treatment. Here, we present a case report involving five family members with varying degrees of severity of food borne botulism.

Cases description: Five members of a family, a 38-year-old lady with her three sons and one daughter came to Accident and Emergency (A and E) department with nausea, vomiting, and varying degrees of weakness after 13 hours of ingestion of common food (Kadhi Rice). It gradually progressed to bilateral symmetrical ophthalmoparesis and descending paralysis with varying severity amongst them. The mother and her 17-year-old son required mechanical ventilation. All of them recovered with conservative and supportive management with the mother having residual generalised weakness. A diagnosis of food-borne botulism with acute descending paralysis was made clinically.

Conclusion: Although a rare entity, recognising botulism based on the clinical picture is relatively easy. It is important to maintain a high clinical suspicion to avoid diagnostic delay with increased risk of sequelae and death. Anti-botulinum toxin is not required in every case of botulism, making intensive care support the corner stone of management.

Key words: Botulinum, food borne, descending paralysis, antitoxin, BoNTs.

Introduction

Botulism is a rare but fatal neuroparalytic syndrome, caused by a neurotoxin produced by bacteria of the genus *Clostridium*. Food-borne botulism is caused by ingestion of preformed botulinum neurotoxin. A reported case of food-borne botulism represents a public health emergency because of the potential severity of the disease and the possibility of mass exposure to the contaminated product. The first food-borne botulism case in India was reported in 1996 by Chaudhry *et al*[1] which was caused by a neurotoxigenic *Clostridium butyricum*. This syndrome courses initially with gastrointestinal symptoms like nausea and vomiting, followed by symmetrical cranial nerve palsies, may progress to descending flaccid paralysis and ultimately lead to respiratory arrest. The effective management of this condition hinges significantly on intensive care and the timely administration of antitoxin. Here, we present a case series of 5 family members, with varying severity of food-borne botulism.

Cases Description

Five members of a family, a 38-year-old lady with her four children presented to A and E department at Pandit B. D. Sharma PGIMS, Rohtak on 20 January 2024 with nausea, and vomiting episodes and varying degrees of weakness after 13 hours of ingestion of common food (Kadhi Rice, a curd-based dish). All the family members initially showed similar gastrointestinal symptoms characterised by multiple episodes of non-bilious, non-projectile, watery vomiting.

Case 1

The 38-year-old lady experienced throat heaviness 5 - 6 hours after the onset of vomiting. This progressed to difficulty in swallowing and speaking, accompanied by blurring of vision. Subsequently, she developed weakness in both the upper limbs, which extended to her lower limbs.. On examination in the emergency department, her Glasgow Coma Scale (GCS) score was E4V1M1. She had bilateral flaccid paralysis (Power 0/5 in all 4 limbs) with absent deep tendon and superficial reflexes, complete ophthalmoplegia, mild bilateral ptosis, and absent direct and consensual light reflexes in both pupils. Also, her corneal and gag reflexes were absent. She started having chest tightness and shortness of breath with oxygen saturation dropping to 80%

**Associate Professor, **Junior Resident, ***Assistant Professor, ****Senior Resident, *****Professor, Department of Medicine, Pandit B. D. Sharma PGIMS, Rohtak - 124 001, Haryana.*

Corresponding Author: Dr Sandeep Goyal, Professor, Department of Medicine, Pandit B. D. Sharma PGIMS, Rohtak - 124 001, Haryana. Phone: 9968969651, E-mail: sandeepgoyal20000@gmail.com.

on room air within 30-45 minutes. She was intubated and put on ventilator in the intensive care unit (ICU) . After three days, she was extubated but continued to have 4/5 power in all four limbs at two-months follow-up.

Case 2

17-year-old son had dizziness, difficulty in swallowing, blurring of vision, and weakness in his upper limbs, followed by weakness in his lower limbs. At presentation, he had a GCS of E4V1M1 and an oxygen saturation of 90%. He also had bilateral flaccid paralysis with absent reflexes and complete ophthalmoplegia. He also required mechanical ventilation in the ICU amd recovered after three days.

Case 3

12-year-old son presented with acute descending weakness, characterised by flaccid paralysis in all four limbs followed by development of bilateral dilated, non-reacting pupils, and ophthalmoplegia . On presentation, he had a GCS of E4V1M1 and oxygen saturation of 82%. He also required mechanical ventilation and recovered after three days.

Case 4

18-year-old son developed a feeling of neck heaviness about an hour after vomiting. He experienced heaviness and weakness in both arms followed by weakness in both the lower limbs. On presentation, he had difficulty in raising his arms above his head and holding objects. He was able to bear weight but had slight knee buckling upon standing. Despite these symptoms, he did not experience any chest tightness, swallowing or breathing difficulty. The boy was vitally stable but had bilateral decreased tone and 4/5 power in all four limbs with mild bilateral ptosis. He was managed conservatively and showed clinical improvement within two days.

Case 5

15-year-old daughter also experienced mild heaviness in both upper limbs followed by difficulty in raising her arms above the head. There were no problems with swallowing or speaking. Neurologically, she had 4/5 power in her upper limbs, 5/5 power in lower limbs with normal pupillary and deep and superficial reflexes. She was managed conservatively.

There was no history of loose stools, any unknown bite, fever, bladder-bowel involvement in any of the member. All the routine investigations including serum electrolytes were normal. Based on the history of common food intake, afebrile gastrointestinal complaints, clustering in a family, normal potassium levels, acute flaccid descending paralysis with bilateral symmetrical cranial nerve palsy of varying severity, a clinical diagnosis of food-borne botulism was made.

Discussion

Botulism is a rare neuroparalytic disease mediated by *Clostridium botulinum*, an anaerobic, gram-positive, spore-forming bacterium. It is caused by botulinum neurotoxin (BoNTs) which is produced by bacteria under low oxygen conditions and certain combinations of storage temperature and preservative parameters. There are 7 distinct forms of botulinum toxin, types A-G. Four of these (types A, B, E and rarely F) cause human botulism. The toxin is one of the most lethal and potent neurotoxin known to humans. Food-borne botulism occurs when *C. botulinum* grows and produces toxins in food prior to consumption with case fatality being 5 - 10 %[2]. The botulinum toxin has been found in a variety of foods, including low-acid preserved vegetables, such as green beans, spinach, mushrooms, and beets; fish, including canned tuna, fermented, salted, and smoked fish; and meat products, such as ham and sausage. Following ingestion, the toxin permeates the bloodstream via the mucosa of the jejunum or ileum, ultimately spreading within the neuromuscular cholinergic synapse[3]. BoNTs specifically attach to motor neurons and autonomic cholinergic nerves. Within the peripheral cholinergic synapse, the toxin establishes an irreversible and highly selective affinity with the presynaptic receptors.

The typical manifestation of food-borne botulism involves a sudden onset of bilateral cranial neuropathies accompanied by symmetric descending weakness, typically starting between 6 and 72 hours after ingestion[4]. The cranial nerve impairment encompasses symptoms such as blurred vision (resulting from fixed pupillary dilation and affliction of cranial nerves III, IV, and VI), diplopia, nystagmus, ptosis, dysphagia, dysarthria, and facial weakness (bulbar palsy). The progression of muscle weakness typically follows a descending pattern, starting from the trunk and upper extremities to the lower extremities. Respiratory challenges, such as dyspnoea, may arise due to diaphragmatic paralysis, upper airway compromise, or a combination of both, often necessitating intubation and mechanical ventilation[5].

Notably, like in our case, most patients initially present to the emergency department with predominant gastrointestinal symptoms followed by onset of pronounced neurological changes. A systematic review also showed nausea was reported 36% of the time by patients with foodborne botulism and vomiting was reported in 50% of the patients[6].

The first case of food borne botulism was reported in India in 1996; 34 students of a residential school in rural Gujrat complained of abdominal pain, nausea, chest pain, and difficulty in breathing. Patients reported that 24 hours before onset of symptoms, they had eaten ladoo (a local sweet), curd, buttermilk, sevu (crisp made of gram flour), and pickle. Anaerobic culture of left over sevu yielded *C. butyricum*[1]. Similarly, Agarwal *et al*[7] in 2004 reported food-borne botulism in 2 members of a family with a history of eating canned meat products, along with preserved curd.

Botulism should be suspected in a patient who is responsive, afebrile with normal or low heart rate with acute symmetric cranial nerve palsy (typically bulbar palsies) followed by bilateral flaccid paralysis of voluntary muscles and respiratory arrest. It has a distinct clinical profile, but its differential diagnosis includes conditions such as snake bite, myasthenia gravis, Lambert-Eaton myasthenic syndrome, Tick paralysis, Guillain-Barré syndrome, Miller-Fisher variant, and poliomyelitis but they have distinguishing features that help differentiate them from botulism. Other potential confounding conditions include magnesium intoxication, diphtheria, organophosphate poisoning, or brainstem infarction. A careful examination and consideration of these factors are essential for accurate diagnosis and appropriate management.

Confirmation of botulism diagnosis relies on identifying toxins in serum, stool, vomitus, or the implicated food source. However, this process involves several days for the growth and identification of the causative organism. Stool examination in our cases showed gram-positive cocci with no identification of organism. Stool culture reports which were obtained later did not show any growth of *Clostridium* on anerobic culture. Thus, syndrome-based clinical suspicion and diagnosis is of utmost importance. It is important to note that the decision to administer antitoxins should be based on a presumptive clinical diagnosis, and any delay in diagnosis should not impede the initiation of therapy.[3]

Antitoxin stands as the primary therapeutic intervention for botulism but intensive care in ICU holds the key for survival. Antitoxins exclusively neutralise circulating toxins but have no effect on toxins already bound to nerve terminals, emphasizing that supportive management is the key to management. We managed our case in intensive care with supportive management with persistent mild residual paralysis in 1 patient.

Conclusion

Botulism can cause a variety of symptoms, ranging from minor gastroenteritis to symmetrical cranial nerve palsies, to descending weakness and rapid respiratory arrest and death. Rapid neurologic dysfunction and respiratory difficulties start between 6 and 72 hours after ingestion. Overall Botulism is a clinical diagnosis and a high degree of clinical suspicion is needed in any patient who is responsive, afebrile with acute gastrointestinal symptom, acute symmetric cranial nerve palsy or descending flaccid paralysis. Anti-botulinum toxin is not required in every case of botulism, making intensive care support the corner stone of management.

References

1. Chaudhry R, Dhawan B, Kumar D *et al*. Outbreak of suspected Clostridium butyricum botulism in India. *Emer Infectio Dis* 1998; 4 (3): 506-7.
2. Botulism (Online). 2023 (Cited 2023 Sep 25); Available From: URL: https://www.who.int/news-room/fact-sheets/detail/botulism.
3. Rao AK, Sobel J, Chatham-Stephens K. Clinical guidelines for diagnosis and treatment of botulism. *MMWR Recomm Rep* 2021; 70 (2): 1-30.
4. Bleck TP. Clostridium botulinum. Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases. Mandell GL, Bennett JE, Dolin R (Eds.). Philadelphia: Elsevier 2005; 2822-9.
5. Varma JK, Katsitadze G, Moiscrafishvili M *et al*. Signs and symptoms predictive of death in patients with foodborne botulism – Republic of Georgia, 1980 - 2002. *Clin Infect Dis* 2004; 39 (3): 357-62.
6. Chatham-Stephens K, Fleck-Derderian S, Johnson SD *et al*. Clinical features of foodborne and wound botulism: a systematic review of the literature, 1932 - 2015. *Clin Infect Dis* 2018; 66: S11-6.
7. Agarwal AK, Goel A, Kohli A *et al*. Food-borne botulism. *JAPI* 2004; 52: 677-8.