RECURRENT EPISODES OF FOOD BORNE BOTULISM IN A 7-YEAR OLD BOY

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Abstract

Objective

Botulism is the acute, descending, flaccid paralysis that results when the neurotoxin of Clostridium botulinum blocks neuromuscular transmission. C botulinum toxin is the most poisonous substance that blocks neuromuscular transmission and causes death through airway and respiratory muscle paralysis; all forms of botulism manifest neurologically as asymmetric, descending, flaccid paralysis beginning with the cranial nerve musculature. Food-borne botulism results from the ingestion of food in which C botulinum has multiplied and produced its toxin.

Patient

We report a new case of food-borne botulism in a 7 year old boy with recurrent episodes of weakness, difficulty in swallowing and speech; bilateral ptosis and mydriasis. He had positive history of the same symptoms, documented twice before. The patient's samples were sent for detection of toxin of Clostridium botulinum, and toxin of C. botulinum, type A was found in his stool sample, confirming our diagnosis. This case was unusual report because our patient has not history of canned food ingestion and also because recurrent episodes of paralysis in this case are unusual findings in botulism.

Conclusion

In this report we want to emphasize that canned-food ingestion is not necessary for diagnosis of food-borne botulism and because delayed treatment leads to increase mortality and morbidity, treatment should be initiated promptly on the basis of clinical suspicion.

Keywords: Botulism, Clostridium Botulinum, Food-borne.

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Introduction

Botulism is a neuroparalytic disorder that can be classified into the following categories: food-borne, infant, wound, unclassified and inadvertent botulism. The fourth category "unclassified" was disignated by the center for Disease Control and Prevention (CDC) for adult patients who lack an apparent food or wound source of botulinum toxin. The inadverent form most recently recognized, is an unintended consequence of treatment of certain movement disorders with botulinum toxin A(1,2). From 1973 to 1996, a median of 24 cases of food-borne botulism, 3 cases of wound and 71 cases of infant botulism were reported annually to the CDC(1). Except for infant botulism, which may have a prolonged course, onset of symptoms occurs abruptly within hours or evolves gradually over several days. Cranial nerves palsy is the most common complication of botulism, followed by symmetric, descending,

flaccid paralysis of the somatic musculature that may progress rapidly. Patients with rapidly evolving illness initially may have generalized weakness and hypotonia. Signs and symptoms in older children or adults can include diplopia, blurred vision, dry mouth, dysphagia, dysphonia, and dysarthria (3).

Foodborne botulism results when a food contaminated with spores of C botulinum is preserved or stored improperly under anaerobic conditions that permit germination, multiplication, and toxin production. Outbreaks have occurred with restaurant-prepared foods, such as patty-melts; potato salad; aluminum foil-wrapped baked potatoes; home-canned foods; bottled garlic; and cheese sauce (1,4).

The usual interval between the ingestion of food and the onset of symptoms is 18 to 36 hours, but it may be as short as a few hours or as long as 8 days. Confirmation of the diagnosis of botulism depends primarily on detection of toxin or the organism in the patient, or in the implicated food or wound.

A toxin neutralization bioassay is used to identify botulinum toxin in serum, stool, gastric aspirate, or suspect foods. In infant and wound botulism, the diagnosis is made by demonstrating C botulinum organisms or toxin in feces, wound materials, or tissue specimens (2,3).

Mortality rate from untreated botulism is high. recurrent episodes of muscle weakness is an unusual finding in botulism, being seen more in other conditions such as Myasthenia gravis, Cerebral vascular accident, tick paralysis, paralytic shellfish poisoning and psychiatric disease. The case reported here is an unusual one with recurrent episodes of muscle weakness(3).

Case report

A seven year old boy was admitted in our hospital with chief complaints of nausea, vomiting; weakness, difficulty in swallowing and speech; bilateral ptosis, mydriasis; and diplopia for one week duration. He was from suburb of Hamedan; his family was from a poor socio-economic background.

The patient had no history of fever, gastrointestinal or respiratory tract infection before his symptoms. He had a positive history of the condition, having been admitted to the hospital twice before with the same symptoms. Five months ago, in his first admission, the patient was admitted along with other members of his family (including three siblings and his parents and uncle). His parents died, his sister was discharged with diagnosis of myasthenia gravis crisis and his uncle was discharged with diagnosis of functional weakness. Four months later (one month ago) the patient was admitted again with sudden onset of bilateral mydriasis, ptosis and dysphagia and received antibiotics, improving after 2-3 days.

Before these episodic symptoms, the patient had normal growth and development with unremarkable health problems and had received routine vaccination.

In his physical examination, the patient was fully conscious without respiratory distress and his vital signs were normal. Revealed neurologic examination. Revealed bilateral mydriasis and ptosis with normal corneal reflex, extraoccular muscles' movement and fundoscopy. There was asymmetry in facial expression and gag reflex was impaired. Deep tendon reflexes were decreased bilaterally in lower extremities and absent in upper. Plantar reflexes were bilaterally downward. Cerebellar reflexes, motor and sensory systems were normal.

The patient's brain CT scan, EMG, and NCV, all were normal. CSF analysis was normal with negative culture. The patient's serum and stool samples were sent for detection of toxin of Clostridium botulinum, and toxin of C. botulinum type A was found only in his stool sample.

The patient received botulinum antitoxin (Botulism Immune Globulin Intravenous (BIGIV) and supportive treatment and he improved.

An investigation into the source of food containing the botulinum toxin led us to a jar of whey containing botulinum toxin, which proved to be the source of food borne botulism in all three episodes of disease in patient and other members of his family.

Discussion

Food-borne botulism results from the ingestion of food in which C. botulinum has multiplied and produced its toxin. Most preserved foods have been implicated in food-borne botulism but recent outbreaks associated with cheese, baked potatoes, and other foods in Iran and other countries have modified the older traditional view that food borne botulism results mainly from home-

canned foods(5,6,7); this is supported by our patient, in whom the absence of canned-food ingestion led to misdiagnoses in the first and second episodes of disease (3).

Moreover immunity to botulinum toxin does not develop in food-borne botulism, therefore, even in recurrent episodes, botulism must be taken into consideration.

It must be noted that the diagnosis of botulism is established by demonstrating the presence of botulinum toxin in serum or of C. botulinum toxin or of organisms in wound material or feces that often requires days to complete; furthermore, the success of the antitoxin treatment is strongly dependent on the time of administration; if the toxin has entered the nerve endings and disappeared from blood circulation, the treatment may be of no benefit to the patient (8). Therefore, clinical diagnosis is the foundation for early recognition of all forms of botulism(3), and treatment with antitoxin should be initiated promptly on the basis of clinical suspicion, since confirmation by lab test results can, very often, cause delays by several days.

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