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A Rare Case of Foodborne Botulism in a Pediatric Patient

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ABSTRACT

Botulism is a rare and potentially fatal condition caused by the neurotoxin produced by Clostridium botulinum, an exotoxin released by this gram-positive bacterium in conducive environments, typically found in homemade canned foods. Among the various clinical manifestations of botulism, foodborne botulism arises from the consumption of food contaminated with preformed botulinum toxin. In 2020, the NCDC Georgia reported nine cases of botulism, one of which involved a child. In our clinic, we encountered a pediatric case of foodborne botulism as part of a familial outbreak following the ingestion of homemade canned red tomatoes. Despite negative lab results for botulism toxin in all family members, it is noteworthy that sample submission for investigation was delayed until after gastric lavage and enema. Nevertheless, all four family members were successfully treated with Botulism Antitoxin (BAT). It is imperative to highlight that patient exhibiting progressive symptoms or signs indicative of botulism, such as paralysis, should receive BAT treatment irrespective of laboratory confirmation. Additionally, BAT treatment should be administered regardless of underlying medical conditions or demographic characteristics such as age, sex, or other demographic factors.

Keywords: Antitoxin; botulism; foodborne infection; neurotoxins.

INTRODUCTION

Foodborne botulism is primarily caused by the potent neurotoxins produced by Clostridium botulinum, which contaminate certain foods.[1-4] These toxins, classified from types A to G based on their antigenicity, with types A, B, and E most commonly associated with human illness, often result from small outbreaks linked to home-canned foods like fruits, vegetables, and fish.[5-8] Type A is predominant in the United States, Europe, and Asia, while Type E is more prevalent in Canada and Egypt, often associated with traditional preparations of salted fish.[9-11] Although homemade canned foods with low acidity (pH>4.5) are the typical source of botulism toxin, commercially prepared foods account for only about 10% of identified cases. Type E is responsible for approximately 50% of seafood-related outbreaks, with types A and B causing the remainder.[4] Botulism is a global concern, with the CDC in the United States reporting around 200 cases annually.[10]

The Centers for Disease Control and Prevention (CDC) in the United States recommends suspecting botulism in patients meeting three criteria:[3]

- At least one specific symptom of cranial neuropathy, such as blurred or double vision, difficulty speaking, dysphagia, or drooling;
- At least one specific sign of cranial neuropathy, including ptosis, extraocular palsy, facial paresis, fixed pupils, or descending paralysis;
- Absence of fever.

Diagnosis of botulism relies on toxin detection or C. botulinum growth in relevant specimens (serum, urine, stool, gastric aspirate, and suspected food source), ideally collected before antitoxin administration as toxin levels diminish over time. However, confirmatory tests may not yield timely results, prompting treatment based on presumptive clinical diagnosis.[3]

Antitoxin is the mainstay treatment for botulism and should be administered promptly upon clinical suspicion without awaiting diagnostic confirmation.[12,13] In the United States, two forms of botulism therapies are available:

- Equine serum heptavalent botulinum antitoxin for non-infant botulism;
- Human-derived botulism immune globulin for infant botulism.

In regions lacking equine serum heptavalent botulinum antitoxin, alternatives such as monovalent equine serum (e.g., type A, B, or E) are used.[14-16] Dosage adjustments are made according to manufacturer recommendations in such cases.

CASE

A three-year-old male patient was admitted to our hospital two days after the onset of symptoms. Earlier, his three adult family members (mother, father, and grandmother) had developed similar symptoms shortly after consuming

homemade canned tomatoes for dinner. Symptoms include nausea, vomiting, dysphagia, speech disturbance, weakness, and drowsiness. All family members, including the patient, had initially sought care at a regional hospital, where they received gastric lavage and enema. Subsequently, the parents and grandmother were transferred to an adult center in the capital with a diagnosis of foodborne botulism. In contrast, the child was transferred to our center with the same diagnosis.

The patient's condition was assessed as severe upon admission to our center. Vital signs revealed a body temperature of 36°C, heart rate of 136 bpm, respiratory rate of 35 breaths per minute, blood pressure of 80/47 mmHg, and oxygen saturation of 90% on room air. The patient exhibited severe intoxication due to foodborne botulism, manifested by acute alteration of consciousness with somnolence. Physical examination revealed adynamia, apathy, drowsiness, mumbled speech, decreased muscle tone, and an indifferent facial expression. Mild bilateral ptosis, horizontal nystagmus, and limited eyeball movement were observed. The patient responded to strong external tactile stimuli but displayed no meningeal signs or pathologic reflexes. Physical signs of dehydration were evident, including pale, dry skin with delayed capillary refill time, decreased skin turgor, and central cyanosis. Examination revealed diminished heart sounds with the systolic murmur of the apex and base, breathing was superficial bilaterally, and the cough reflex was slightly suppressed.

In the capital hospital, the patient received antitoxin therapy, comprising Type A (10000 IU/dose, 2.86 ml), Type B (5000 IU/dose, 4.2 ml), and Type E (10000 IU/dose, 6.3 ml), along with additional supportive care including intravenous infusion, oxygenation via a simple face mask, intermittent aspiration of upper airway secretions and saliva, conjunctival care with eye drops, and placement of nasogastric and Foley catheters. Although a urine, serum, and gastric aspirate sample were obtained for laboratory testing, the suspected food source was unavailable for analysis. National Center for Disease Control (NCDC) laboratory results were negative for all family members.

During the patient's hospitalization, electrocardiogram (ECG) abnormalities related to the heart conduction system were detected, including tachycardia and signs of septal ischemia. Subsequent investigations revealed an incomplete bundle branch block and mild antero-septal ischemia. However, cardiac ultrasound revealed no pathological changes, and the patient remained hemodynamically stable throughout the hospital stay.

Following antitoxin administration, there was no further progression of symptoms in any of the four patients, and existing symptoms began to regress within a week. The patient remained hospitalized for two weeks, including one week in the intensive care unit (ICU). Prompt diagnosis, early administration of antitoxin without waiting for laboratory confirmation, and supportive care led to the improved condition of the patient and family members, who were discharged from the hospital without neurological sequelae.

DISCUSSION

Considering the shared consumption of home-prepared canned food by all family members on the same day, the rapid onset of symptoms within a short incubation period (up to 3 days), simultaneous acute disease onset without fever, and the development of similar neurological symptoms, such as descending flaccid paralysis, in all four family members, the clinical diagnosis of foodborne botulism is strongly supported. Despite negative lab results for botulism toxin, which can occur in such cases, delayed sample collection after gastric lavage and enema in another hospital may have resulted in the internalization of an already absorbed fraction of the exotoxin in presynaptic neurons. However, given the non-timely results of confirmatory tests, antitoxin administration was based on the presumptive clinical diagnosis of botulism.

Antitoxin remains the primary therapeutic option for botulism and should be promptly administered upon clinical suspicion, especially when symptoms are progressing rapidly. Delays in treatment while awaiting diagnostic results should be avoided.[12,13] Prompt identification of botulism based on clinical signs, as the CDC recommends, underscores the importance of immediate antitoxin administration. Unlike wound botulism, antibiotics are generally not recommended in treating foodborne botulism unless there are signs of bacterial super or co-infection.[3]

The observed ECG changes can be attributed to impaired postganglionic release of acetylcholine in the parasympathetic nervous system, resulting in unopposed sympathetic system stimulation due to botulism exotoxin.[3,16] Application of botulism antitoxin halted disease progression in all patients, with no further development of neurological signs or symptoms.

CONCLUSIONS

Patients with suspected botulism, especially those experiencing progressing symptoms, such as paralysis, should receive Botulism Antitoxin (BAT) treatment without delay, irrespective of lab confirmation or any demographic factors, including underlying medical conditions, age, or sex.

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