European Journal of Case Reports in Internal Medicine

Foodborne Botulism: Neglected Diagnosis

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Doi: 10.12890/2019_001122 - European Journal of Case Reports in Internal Medicine - © EFIM 2019

Received: 18/04/2019
Accepted: 28/04/2019
Published: 17/05/2019

How to cite this article: Zarcos Palma N, da Cruz M, Fagundes V, Pires L. Foodborne botulism: neglected diagnosis. *EJCRIM* 2019;**6**: doi:10.12890/2019_001122.

Conflicts of Interests: The Authors declare that there are no competing interests.

ABSTRACT

Botulism is rare neuroparalytic disease caused by botulinum toxin, one of the most toxic substances known. Foodborne botulism is caused by consumption of foods contaminated with botulinum toxin. The clinical manifestations are flaccid, symmetrical, descending paralysis affecting cranial and peripheral nerves. The only specific treatment is botulinum antitoxin. We report the case of a 37-year-old man with gastrointestinal manifestations and posterior cranial nerve palsy who was diagnosed with botulism infection. Clinicians should be aware of rare causes of infection and determine the aetiology of symptoms.

LEARNING POINTS

- Botulism remains a diagnostic challenge.
- Misdiagnosis of early cases suggests sporadic cases are overlooked.
- Timely clinical diagnosis is critical for treatment decisions as botulinum antitoxin cannot reverse existing paralysis.

KEYWORDS

Botulism, botulinum toxin, *Clostridium botulinum*, foodborne

INTRODUCTION

Botulism is a rare disease, with sudden onset, caused by exposure to botulinum toxin. The first research on botulism was reported in the 1820s and described hundreds of cases of 'sausage poisoning' in a southern German town[1]. We now know that botulinum neurotoxin is ingested, inhaled or produced in the intestine or a wound, enters the vascular system and is transported to peripheral cholinergic nerve terminals, including neuromuscular junctions, postganglionic parasympathetic nerve endings and peripheral ganglia. The central nervous system is likely not involved[2].

There are eight identified toxin serotypes (A through H), all of which are antigenically distinct but structurally similar (~150 kDa zinc-endopeptidase proteins consisting of a 100 kDa heavy chain and a 50 kDa light chain). Foodborne botulism is caused by the ingestion of food contaminated with botulinum toxin. Toxin types A, B and E have all been associated with foodborne botulism[2–5].

The manifestations are the result of the toxin's inhibition of acetylcholine release at the neuromuscular junction through an enzymatic mechanism. All forms of botulism manifest as a distinct clinical syndrome of symmetrical cranial nerve palsies followed by descending symmetrical flaccid paralysis of voluntary muscles, which may progress to respiratory failure and death[6].

The mainstays of therapy are meticulous intensive care and timely treatment with botulinum antitoxin, which may limit the extent of paralysis. The only specific treatment is botulinum antitoxin, which neutralizes toxin molecules that have not yet bound to nerve endings, preventing their binding to the neuromuscular junction.

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CASE REPORT

A 37-year-old man without relevant medical history, developed epigastric pain and biliary vomiting 1 day after eating sausages at a gastronomic fair. After 2 days he noted diplopia and blurred vision and so consulted an ophthalmologist who prescribed correction glasses, but symptoms persisted. He then developed muscular weakness of his face and upper arms, and progressive onset of dysphagia and xerostomia. Due to the persistent symptoms, he presented to the emergency department 10 days after the first symptoms began. The physical examination showed bilateral ptosis, mydriasis and sluggish pupillary response to light. A cerebral CT scan did not show any lesions. The patient was not in respiratory distress, and blood gas analysis was normal.

As the patient presented with neuroparalytic syndrome with cephalocaudal progression, the diagnosis of botulism was considered, but unfortunately no antitoxin was available. As a result, he was admitted to the intensive care unit for support therapy and monitoring. He remained clinically stable, with no need for ventilatory support. The symptoms improved slowly and he was kept under clinical surveillance during hospitalization for a total of 3 weeks in the internal medicine department. Supportive treatment with mucosal hydration, was continually provided, and there were no ocular complications.

The diagnosis of botulism was confirmed by laboratory findings of type B botulinum toxin in a blood sample.

The patient's symptoms improved during hospitalization but complete neurological recovery was only achieved after several weeks. The patient showed no symptoms at his 3-month follow-up evaluation.

DISCUSSION

Foodborne botulism is caused by ingestion of food contaminated with botulinum toxin, and does not have individual predisposing factors. Most cases of botulism are sporadic and outbreaks are typically small, involving two or three cases.

The clinical presentation is varied and may be related to the type of toxin. Toxin type A causes the most severe syndrome, and has the highest proportion of patients requiring mechanical ventilation. Toxin type B appears to cause milder disease, and commonly autonomic symptoms precede muscular weakness[5]. Most cases of botulism are associated with home-preserved meats, fish and vegetables. In Europe, foodborne botulism is often related to home-preserved meat, such as sausages and ham. The spores of *Clostridium botulinum* are heat resistant, easily surviving heating to 100°C at 1 atm for 5 or more hours. However, spores can be destroyed by heating to 120°C for 5 minutes, but not all food is suitable for such heat treatment. Adequate processing and storage must be the first concern [5, 7].

Timely clinical diagnosis is critical for treatment decisions. Since botulinum antitoxin cannot reverse existing paralysis as it only neutralizes toxin molecules that have not yet bound to nerve endings, it must be administered early in the course of the disease, ideally within 24 hours after symptom onset. Antitoxin administration must be based on a high clinical suspicion for botulism and should not be delayed while awaiting the results of diagnostic studies [8–10].

Botulism is a public health emergency. In Portugal all suspected and confirmed cases must be notified to the relevant authorities[11].

CONCLUSION

This was a case of foodborne botulism caused by ingestion of toxin in contaminated food, where the diagnosis was considered later in the course of the disease. The patient was treated only with supportive therapy because botulinum antitoxin was unavailable. The rarity of the condition and the misdiagnosis of early cases have led to speculation that sporadic cases, particularly in people with mild disease, are overlooked[5].

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