Letter to the Editor

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Foodborne botulism: a case report

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To the Editor

We are pleased to address a case report of a patient with a foodborne botulism. The diagnosis was assumed by clinical presentation, evolution and resolution of the clinical features after botulism antitoxin administration and later confirmed by electroneuromyography. We were unable to isolate botulinic toxin on biological samples nor in the possible culprit food. Latter we highlight the physiopathology and differential diagnosis causing neuromuscular junction impairment.

Botulism is a rare neuroparalytic syndrome, caused by a neurotoxin produced by bacteria of the genus *Clostridium*. This syndrome courses initially with symmetrical cranial nerve palsy, may progress to descending flaccid paralysis and ultimately to respiratory arrest. We present the case of a 51-year-old patient admitted in the Emergency Department with a history of headache, dizziness, vomiting, bilateral non46 reactive mydriasis with one day of evolution. The patient rapidly progressed to urinary retention, hypotonia and respiratory arrest. Food botulism was hypothesized and antitoxin immunoglobulin was administered. Diagnosis was assumed by clinical response to antitoxin immunoglobulin and electromyogram alterations. The patient evolved favorably with total functional recovery.

Although a rare entity, it is important to maintain a high suspicion in order to avoid diagnostic delay with increased risk of sequelae and death.

Introduction

Botulism is a rare neuroparalytic disease mediated by botulinum toxin. Seven botulinum toxin serotypes (from A to G) were described, produced by different species of the genus *Clostridium*. These are gram-positive, obligate anaerobic, spore-forming rods and ubiquitous bacteria.[1] Clinical forms of botulism include foodborne botulism, wound-associated botulism, infant botulism, adult intestinal toxemia, iatrogenic botulism and inhalational botulism.[2–4]

Food botulism, largely caused by home-canned food, is the most common form and usually occurs in sporadic outbreaks. Type A toxin is the most commonly identified in Europe.[5,6] Between 2013 and 2017 a total of 547 confirmed cases and 17 deaths were reported with an average of 109 cases per year (minimum 86 cases in 2017, maximum 128 cases in 2016).[7]

Symptoms range from minor symmetrical cranial nerve palsies to descending weakness and rapid respiratory arrest.

The cornerstone of treatment is a timely administration of antitoxin and intensive care support.[8]

Case report

A 51-year-old Caucasian female patient was admitted in the Emergency Department (ED) with blurred vision, headache, dizziness and vomiting with 1 day of evolution. Personal history of hypertension, obesity and hypercholesterolemia and family history of gynecological and gastrointestinal neoplasms were registered. The patient worked as farmer, but denied chronic exposure to toxic products. Concomitantly, the patient's husband reported self-limited episode of diarrhea and vomiting. A history of homemade sausage, broad beans and honey consumption was registered. No other epidemiological context was identified.

In the ED, the patient presented with fixed bilateral mydriasis and bilateral monocular diplopia, without hemodynamic instability nor fever. No other changes in the objective examination were found at the time. Later, the patient presented urinary retention. Due to suspicion of cerebrovascular etiology, the patient was admitted in the Stroke Unit and underwent cranial computed tomography and later magnetic resonance imaging that revealed an ill-defined hyperintense focus, that could be an ischemic gliotic focus, in the anterior region of the right inner capsule or an artefact image. Blood hemogram, biochemistry with thyroid function study, autoimmune and anticholinergic antibodies and liquor analysis were normal.

On the fourth day of hospitalization, the patient developed dysarthria, bilateral ptosis and bilateral plegia, that rapidly evolved to respiratory arrest, and was admitted in the Intensive Care Unit. Given the epidemiological context and rapid clinical evolution, the hypothesis of botulism was raised. Serum and feces samples were collected, and the botulinum antitoxin administrated. The possible culprit foods consumed, homemade sausage, broad beans and honey, were also collected.

The patient evolved positively after administration of the immunoglobulin. Electroneuromyography was performed, revealing a presynaptic lesion compatible with the hypothesis of botulism (Tables 1 and 2).

The patient was discharged home after 27 days of hospitalization. At the follow-up visit, the patient was asymptomatic, and no sequelae were identified. Laboratory results did not identify the presence of botulinum toxin, neither in serum, feces nor food.

Discussion

Botulism should be suspected on a patient who develops acute and afebrile symmetric cranial nerve palsy (typically bulbar

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Table 1

Ulnar nerve electroneuromyography—a successive compound motor action potential (CMAP) from the ulnar nerve are displayed after ten stimuli at 2 Hz. A decremental response (a decline in the response amplitude) is seen. It is maximal at 12% by the seventh response

Stim freq: 2 Hz Stim dur: 0.1 ms Time: 15:16:27			No. in train: 10 Stim rjct: 0.5 ms Stop watch: 0.11		
Pot No. peek	**Peek amp (mV)**	**Amp decr (%)**	**Area (mV ms)**	**Area decr (%)**	**Stim level (mA)**
1	5.40	0	15.00	0	97.6
2	4.88	10	14.10	6	97.6
3	4.85	10	13.40	11	97.6
4	4.88	10	13.50	10	97.6
5	5.34	1	13.00	13	97.6
6	5.41	0	13.80	8	97.6
7	4.74	12	13.80	8	97.6
8	5.11	5	14.00	7	97.6
9	5.30	2	13.70	8	97.6
10	4.97	8	13.40	11	97.6

Table 2

Radial nerve electroneuromyography—a successive compound motor action potential (CMAP) from the radial nerve are displayed after ten stimuli at 2 Hz. A decremental response (a decline in the response amplitude) is seen. It is maximal at 5% by the eighth and nineth response

Stim freq: 2 Hz Stim dur: 0.1 ms Time: 15:18:46			No. in train: 10 Stim rjct: 0.5 ms Stop watch: 0.12		
Pot No. peek	**Peek amp (mV)**	**Amp decr (%)**	**Area (mV ms)**	**Area decr (%)**	**Stim level (mA)**
1	7.27	0	22.20	0	79.1
2	7.07	3	21.80	2	79.1
3	7.00	4	22.10	0	79.1
4	7.01	4	22.30	0	79.1
5	6.95	4	21.80	2	79.1
6	7.07	3	22,30	0	79.1
7	6.95	4	21.90	1	79.1
8	6.91	5	21.70	2	79.1
9	6.91	5	22.30	0	79.1
10	7.01	4	22.30	0	79.1

palsies) followed by bilateral flaccid paralysis of voluntary muscles, and ultimately, respiratory arrest. Is mediated by a neurotoxin, the botulism toxin, a zinc-dependent endoprotease which has a tropism for cholinergic neuromuscular junctions. Independently of the clinic form, the toxin is disseminated by bloodstream to terminals of peripheral cholinergic nerves (including neuromuscular junctions, parasympathetic postganglionic nerve endings and peripheral ganglia). There, the release of acetylcholine on the presynaptic nerve endings is inhibited by blocking the soluble N-ethylmaleimide-sensitive-fusion-attachment protein receptors, leading to paralysis while preserving sensory function and consciousness.[9]

The botulism diagnosis is based on clinical presentation, and epidemiological context.

Gastrointestinal symptoms are common in food botulism (nausea, vomit, abdominal pain, diarrhea, xerostomia) and may precede neurological syndromes.

Initially, suspicion of stroke (basilar occlusion syndrome) was raised, but excluded by the clinical evolution and imaging techniques.

Other differential diagnosis that should be excluded are Guillain-Barré syndrome (that usually start with sensory complains, areflexia and ascending neuropathy, which are absent in botulism), Miller Fisher variant (that may present with oculomotor dysfunction associated with cranial neuropathies and ataxia, absent in botulism) and Myasthenia gravis (MG) or Lambert-Eaton syndrome (LE) (rarely fulminant and associated with presence of antibodies, MG lack autonomic symptoms and the LE is associated with cancer, more frequently small cell lung carcinoma).[10,11] The electromyography patterns may be helpful in distinguishing these causes. In botulism, compound muscle action potential (CMAP) amplitudes are decrease if the presynaptic block is severe enough. Repetitive nerve stimulation at frequencies of 2 to 5 Hz deplete readily available stores of acetylcholine from neuromuscular junction and decreases CMAP amplitude even further, a finding termed decremental response, which is usually greater than 10%. However, in more severe cases, the baseline CMAP may be to low to see a decremental response. Repetitive nerve stimulation at high rates (20–50 Hz) increase the CMAP amplitude in botulism and LE, but not in MG.[12]

It is also important to exclude toxic causes of neuromuscular junction disorders (organophosphate and carbamate poisoning, tick paralysis, snake venom, drugs, hypocalcemia and hypermagnesemia) and shellfish and puffer fish intoxication, excluded by anamneses, a careful clinic examination and blood analysis.[12]

Conclusion

The identification of toxin, although desired, is not necessary to reach a definitive diagnosis. Therefore, it is important to maintain

a high suspicion and treatment must be started with administration of antitoxin and intensive care support before the time-consuming workup is complete.

We present a case of food botulism in which we highlight the rapid clinical evolution to a critical and deadly state and total clinical response to specific therapeutic, without founding the culprit food contaminated by botulinum toxin.

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