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Foodborne Botulism Outbreak with Negative Toxicology and Electrodiagnostic Confirmation: Two Severe Cases from the Same Source

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Abstract

Foodborne botulism is a rare neuroparalytic emergency caused by preformed *Clostridium botulinum* (*C. botulinum*) toxin, characterized by pre-synaptic neuromuscular junction (NMJ) blockade, descending flaccid paralysis, and potentially fatal respiratory failure. Electromyography (EMG) is an essential diagnostic tool, particularly when toxin assays are negative, as occurs in a substantial proportion of confirmed cases. We describe two severe cases of probable foodborne botulism from the same epidemiological outbreak, with electrophysiological confirmation by repetitive nerve stimulation (RNS) despite negative toxicology. A 24-year-old woman (Case 1) and a 24-year-old man (Case 2) consumed chicken and heart-of-palm pie from a bakery in Belo Horizonte, Brazil, on Day 0. Both developed symptoms approximately six hours after ingestion, progressing to bilateral ptosis, diplopia, descending flaccid paralysis, and respiratory failure requiring orotracheal intubation and ICU admission. A third individual from the same outbreak, an elderly aunt of Case 1, died approximately 30 days after intoxication. Heptavalent botulinum antitoxin was administered on Day 1 in both patients, before results from the Fundação Ezequiel Dias (FUNED; Belo Horizonte, Brazil) returned negative for all tested serotypes. EMG performed on Day 36 demonstrated reduced CMAP amplitudes, significant decrement on low-frequency RNS (>10% at 3 Hz), and pathological post-tetanic facilitation on high-frequency RNS (>30% at 30-40 Hz), confirming pre-synaptic NMJ dysfunction in the recovery phase. Needle EMG revealed a myopathic pattern in proximal upper-limb muscles in both cases. Major complications included ventilator-associated pneumonia (VAP), autonomic dysfunction (severe bradycardia in Case 2; gastroparesis in Case 1), and septic thrombophlebitis (Case 2). Both patients were weaned from mechanical ventilation with progressive functional recovery. Negative toxicology does not exclude the diagnosis when the clinical-epidemiological context is compatible. EMG constitutes a highly valuable confirmatory tool even in the recovery phase. Early antitoxin administration, anticipatory airway management, and multidisciplinary intensive support are determinants of survival.

Categories: Neurology, Epidemiology/Public Health, Infectious Disease
Keywords: botulinum antitoxin, botulinum toxin, descending flaccid paralysis, electrodiagnostic confirmation, foodborne botulism, mechanical ventilation, neuromuscular junction, palm heart poisoning, repetitive nerve stimulation, toxicology-negative botulism

Introduction

Botulism is an acute, potentially fatal neuroparalytic syndrome caused by botulinum toxin, one of the most potent biological toxins known, produced by the anaerobic, gram-positive, spore-forming bacillus *Clostridium botulinum* (*C. botulinum*). Seven serotypes (A through G) have been identified, with types A, B, and E accounting for the majority of human disease [1]. The toxin irreversibly blocks acetylcholine exocytosis at presynaptic terminals by proteolytic cleavage of SNARE complex proteins, specifically SNAP-25 (serotypes A and E), VAMP/synaptobrevin (serotypes B, D, F, and G), and syntaxin (serotype C), preventing vesicle fusion with the presynaptic membrane and abolishing cholinergic neuromuscular and autonomic transmission [2].

The foodborne form results from ingestion of food containing preformed toxin, produced during bacterial growth under anaerobic conditions characterized by temperatures between 3°C and 47°C, pH above 4.6, high water activity, and absence of effective preservatives [3]. In Brazil, palm heart in brine is the most frequently implicated food vehicle in botulism outbreaks, as its low acidity and frequent artisanal preparation under inadequate heat treatment create ideal conditions for sporulation and toxin production by *C. botulinum*, predominantly serotypes A and B [4,5].

Global incidence is low, estimated at 1,000 to 1,500 cases annually, but mortality from untreated disease may exceed 40% to 60% [6]. With modern intensive care and antitoxin therapy, case-fatality rates have fallen to 3% to 5% in high-income settings [6]. In Brazil, the National Notifiable Disease Information System (SINAN) registered 218 confirmed cases between 2000 and 2021, with a case-fatality rate of approximately 10% to 15% [7].

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The clinical diagnosis rests on a characteristic triad: prodromal gastrointestinal symptoms (nausea, vomiting, diarrhea, or constipation), symmetric descending flaccid paralysis with bulbar onset (ptosis, diplopia, dysarthria, dysphagia), and preserved consciousness in an afebrile patient in the context of compatible food exposure [8]. Autonomic involvement, xerostomia, urinary retention, constipation, mydriasis, tachycardia, or bradycardia is part of the full syndrome [8]. Routine laboratory investigations are typically normal. Toxin detection in biological specimens by mouse bioassay at reference laboratories has a sensitivity of approximately 60% to 70% in the acute phase, falling substantially after antitoxin administration and beyond the first 48 to 72 hours of illness [1,3].

Electrodiagnostic studies occupy a central role when toxin assays are negative or unavailable. The electrophysiological hallmark of botulism, reduced compound muscle action potential (CMAP) amplitudes on nerve conduction studies, a decremental response at low-frequency repetitive nerve stimulation (RNS), and pathological post-tetanic facilitation exceeding 30% at high-frequency RNS reflects the presynaptic nature of the neuromuscular junction (NMJ) blockade and allows objective, quantifiable confirmation of the diagnosis even in the absence of positive toxicology [2,9].

We report two cases of probable foodborne botulism from the same epidemiological outbreak associated with chicken-and-palm-heart pie acquired from a bakery in Belo Horizonte, Minas Gerais, Brazil, both presenting with severe neurological disease, negative toxin assays, and electrophysiological confirmation. Clinical, diagnostic, therapeutic, and public health aspects are discussed within the framework of the CARE case report guidelines.

Case Presentation

Epidemiological context

Three individuals residing in Sete Lagoas, Minas Gerais, Brazil, consumed chicken-and-palm-heart pie purchased from the same bakery in Belo Horizonte on the same day (Day 0). All three developed symptoms consistent with foodborne botulism approximately six hours after ingestion, with progressive worsening over the following hours. The affected individuals were Case 1 (a 24-year-old woman), Case 2 (a 24-year-old man), and a third individual, the aunt of Case 1, an elderly woman, who developed a severe clinical course and died approximately 30 days after the intoxication without completing formal diagnostic investigation. No fourth case was identified.

Both surviving patients were reported as suspected botulism cases to epidemiological surveillance authorities within 24 hours of clinical suspicion, in accordance with Brazilian mandatory notification legislation. Biological samples (serum and stool) and specimens of the implicated food were submitted to the Fundação Ezequiel Dias (FUNED), the Minas Gerais state reference laboratory for botulism. The mouse bioassay returned negative results for all tested serotypes in both patients. An incubation period of approximately six hours is consistent with serotypes A and B and may reflect a relatively high toxin load in the implicated food.

Palm heart in brine is a recognized high-risk substrate for botulism in Brazil. Its low acidity, combined with artisanal or small-scale preparation conditions that frequently fail to achieve adequate heat treatment for spore destruction, creates ideal anaerobic conditions for *C. botulinum* toxin production. The fatal outcome in the third exposed individual underscores the critical role of age and physiological reserve as prognostic determinants: older patients with reduced respiratory muscle reserve have markedly lower tolerance to progressive neuromuscular paralysis and higher vulnerability to fatal respiratory failure.

Laboratory findings for both patients are summarized in Table *1*.

Parameter	Reference range	Case 1	Case 2
Hematology			
Hemoglobin (g/dL)	12.0–16.0 (F)/13.5–17.5 (M)	11.3 (Day 22)	12.2 (Day 13)
Leukocytes (×10³/µL)	4.0–11.0	14.0 (Day 22)	19.1 (Day 13)
Platelets (×10³/µL)	150–400	980 (Day 24)*	571 (Day 13)*
Renal Function and Electrolytes			
Urea (mg/dL)	10–50	48 (Day 22)	43 (Day 13)
Creatinine (mg/dL)	0.5–1.1 (F)/0.7–1.3 (M)	0.47 (Day 22)	0.33 (Day 13)
Sodium (mEq/L)	136–145	140 (Day 22)	133 (Day 13)
Potassium (mEq/L)	3.5–5.0	4.3 (Day 22)	4.9 (Day 13)

Magnesium (mg/dL)	1.6–2.6	2.1 (Day 22)	2.0 (Day 13)
Ionized Calcium (mmol/L)	1.15–1.35	—	1.14 (Day 13)
Inflammatory Markers			
CRP (mg/L)	<5.0	19.0 (Day 22)	72.1 (Day 4)
Lactate (mmol/L)	<2.0	2.7 (Day 22)	1.8 (Day 13)
Hepatic and Pancreatic Function			
AST/GOT (U/L)	<35	19 (Day 30)	17 (Day 4)
ALT/GPT (U/L)	<35	24 (Day 30)	14 (Day 4)
Total Bilirubin (mg/dL)	<1.2	0.57 (Day 30)	1.35 (Day 4)
Amylase (U/L)	28–100	122 (Day 30)	153 (Day 30)
Lipase (U/L)	10–140	621 (Day 30)**	75 (Day 30)
Blood Gas Analysis			
pH	7.35–7.45	7.43 (Day 24)	7.43 (Day 13)
HCO_3 (mEq/L)	22–26	28.0 (Day 24)	33.0 (Day 13)
pCO_2 (mmHg)	35–45	43.0 (Day 24)	51.0 (Day 13)
Cerebrospinal Fluid			
Cells (per µL)	<5	4 (Day 7)	—
Proteins (mg/dL)	15–45	27 (Day 7)	—
Glucose (mg/dL)	50–80	72 (Day 7)	—
Cultures	Negative	Negative (Day 7)	—
Coagulation			
INR	0.9–1.2	—	1.00 (Day 30)
aPTT (seconds)	25–35	—	23.8 (Day 30)

TABLE 1: Selected laboratory findings in Cases 1 and 2

F: female reference range; M: male reference range. Time points expressed as days from symptom onset (Day 0 = day of food ingestion). — = not measured or not available. *Reactive thrombocytosis during prolonged mechanical ventilation and infection. **Elevation associated with gastroparesis confirmed by CT scan.

AST/GOT: aspartate aminotransferase and glutamic oxaloacetic transaminase; ALT/GPT: alanine transaminase and glutamate-pyruvate transaminase; HCO_3: bicarbonate; pCO_2: partial pressure of carbon dioxide; INR: international normalized ratio; aPTT: activated partial thromboplastin time

Case 1: female patient, 24 years old

The patient was a 24-year-old woman, previously in good health, with a history of pituitary adenoma managed with cabergoline. Documented drug allergy was to metoclopramide. Following ingestion of the implicated food on Day 0, she developed nausea, vomiting, general malaise, and progressive muscle weakness, with subsequent onset of bilateral ptosis, diplopia, dizziness, and respiratory distress. Due to the inability to protect the airway, orotracheal intubation was performed, and she was admitted to the intensive care unit (ICU) at the Sete Lagoas Municipal Hospital on Day 1. Compulsory epidemiological notification was made. Biological samples were collected before antitoxin administration, and heptavalent equine botulinum antitoxin (serotypes A-G) was administered intravenously on Day 1, approximately 24 to 36 hours after symptom onset, following the Brazilian Ministry of Health emergency protocol, without awaiting laboratory confirmation.

On Day 4, the patient was transferred to Hospital Felicio Rocho (HFR) in Belo Horizonte, a tertiary referral center, admitted under sedation and hemodynamically stable without vasopressors. Sedation weaning was complicated by recurrent agitated arousal. Tracheostomy was performed on Day 9, anticipating prolonged

ventilatory dependency. The FUNED toxin assay result, received on Day 25, was negative; given the assay's approximately 70% sensitivity and the unambiguous clinical-epidemiological context, the diagnosis of probable botulism was maintained.

The main complications during hospitalization were (1) ventilator-associated pneumonia (VAP) caused by methicillin-sensitive *Staphylococcus aureus* (MSSA) isolated from tracheal aspirate on Day 6, treated with sequential directed antibiotic therapy; (2) hospital-acquired pneumonia on Day 30, treated with meropenem; and (3) gastroparesis with recurrent vomiting and elevation of pancreatic enzymes (lipase up to 621 U/L) on Day 30, managed with oral diet suspension, nasojejunal tube feeding, and intramuscular neostigmine on Days 37 and 38. Cerebrospinal fluid (CSF) analysis on Day 7 was normal (4 cells/mm^3, protein 27 mg/dL, glucose 72 mg/dL, negative cultures), effectively excluding Guillain-Barré syndrome and central nervous system infectious processes.

The patient was decannulated on Day 42 and discharged on Day 45, after 42 days of hospitalization at HFR, with mild residual proximal weakness in the upper limbs and slight gait instability, and no cranial nerve deficits on neurological examination at discharge. She was referred to outpatient multidisciplinary rehabilitation (physiotherapy, speech therapy, ophthalmology, and neurology) with a follow-up appointment scheduled 15 days after discharge.

Electrodiagnostic Study: Day 36 of Illness

The electrodiagnostic study was performed by the author using a Nihon Kohden apparatus (Nihon Kohden Corporation, Tokyo, Japan) with surface recording electrodes for nerve conduction studies and a monopolar needle electrode for electromyography (EMG). Extremity temperature was adequate.

Motor nerve conduction studies showed reduced CMAP amplitude in the right median nerve (1.6 mV; reference ⩾4.0 mV), with normal distal latency (2.8 ms) and conduction velocity (62.9 m/s). The right ulnar nerve CMAP amplitude was at the lower limit of normal (3.3 mV). Post-exercise testing of the right median nerve showed no facilitation with submaximal voluntary contraction alone (1.6 mV). Lower limb motor studies (right tibial and deep peroneal nerves) showed normal amplitudes, latencies, and velocities. F-wave minimum latencies were within normal limits in the ulnar and tibial nerves.

Sensory nerve conduction studies were normal for all nerves tested: right ulnar (53.5 µV; 73.7 m/s), radial (34.6 µV; 62.5 m/s), median (44.8 µV; 62.5 m/s), sural (26.6 µV; 68.6 m/s), and superficial peroneal (9.2 µV; 44.3 m/s), excluding sensory polyneuropathy.

Needle EMG of right proximal upper limb muscles (biceps brachii, deltoid, and extensor digitorum communis) showed an early-recruitment pattern with short-duration, low-amplitude motor unit potentials (MUPs) and no spontaneous activity at rest - a myopathic pattern reflecting reduced quantal content of acetylcholine per neuromuscular junction, rather than primary myopathy. The first dorsal interosseous and all lower limb muscles tested were normal.

RNS was performed on the right facial nerve (recording from orbicularis oculi and nasalis) and right ulnar nerve (recording from abductor digiti minimi) at 3 Hz and 30-40 Hz. Low-frequency RNS (3 Hz) of the right facial nerve showed a significant decremental response, with a maximum area decrement of 32.9% between the first and fifth stimulus (threshold: >10%), confirming impaired neuromuscular transmission. High-frequency RNS (30-40 Hz) of the right ulnar nerve demonstrated pathological post-tetanic facilitation with a maximum CMAP amplitude increment exceeding 130% at 40 Hz (threshold: >30%), confirming calcium-dependent mobilization of reserve vesicles, the electrophysiological hallmark of presynaptic NMJ blockade.

The electrodiagnostic study was consistent with moderate presynaptic neuromuscular junction dysfunction in the functional recovery phase (Day 36 of illness). The combination of reduced upper limb CMAP amplitudes, decremental response at 3 Hz, and pathological facilitation at 30-40 Hz constitutes the classic triad of presynaptic NMJ blockade, distinguishing botulism from myasthenia gravis (postsynaptic; decrement without facilitation) and Lambert-Eaton myasthenic syndrome (presynaptic autoimmune; facilitation present but distinct clinical context).

Motor and sensory nerve conduction findings are detailed in Tables *2*-*3*, respectively.

Nerve / Site	Distal latency (ms)	CMAP amplitude (mV)	Reference amplitude (mV)	MCV (m/s)	Reference MCV (m/s)	Finding
Ulnar R: Wrist	2.4	3.3	≥6.0	—	—	Normal
Ulnar R: Elbow → Wrist	6.6	3.7	≥6.0	64.3	≥50	Normal
Tibial R: Ankle	2.9	7.2	≥4.0	—	—	Normal
Median R: Wrist	2.8	1.6 ↓	≥4.0	—	—	Reduced ↓
Median R: Elbow → Wrist	6.0	1.6 ↓	≥4.0	62.9	≥50	Reduced ↓
Median R: Post-exercise	2.7	1.6 ↓	≥4.0	—	—	No facilitation
Deep Peroneal R: Ankle	2.9	6.3	≥2.0	—	—	Normal
Deep Peroneal R: Fibular head	10.2	4.2	≥2.0	48.0	≥40	Normal

TABLE 2: Motor nerve conduction studies: Case 1 (Day 36)

CMAP: compound muscle action potential; MCV: motor conduction velocity; R: right. ↓ Amplitude below reference range (highlighted). Median R: reduced CMAP at wrist and elbow without post-exercise facilitation, consistent with pre-synaptic neuromuscular junction dysfunction.

Nerve/Site	Peak latency (ms)	SNAP amplitude (μV)	Reference amplitude (μV)	SCV (m/s)	Reference SCV (m/s)	Finding
Ulnar R: 5th Digit	2.7	53.5	≥10	73.7	≥50	Normal
Radial R: Wrist	2.1	34.6	≥10	62.5	≥50	Normal
Sural R: Ankle	2.9	26.6	≥10	68.6	≥40	Normal
Median R: 3rd Digit	2.9	44.8	≥10	62.5	≥50	Normal
Superficial Peroneal R: Ankle	4.0	9.2	≥10	44.3	≥40	Normal

TABLE 3: Sensory nerve conduction studies: Case 1 (Day 36)

SNAP: sensory nerve action potential; SCV: sensory conduction velocity. All amplitudes are within normal limits. Mild borderline sural latency, not clinically significant.

Case 2: male patient, 24 years old

The patient was a 24-year-old man with a history of attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder, anxiety and emotional dysregulation disorder, and generalized anxiety disorder, managed with lisdexamfetamine 30 mg/day. He had had a previous surgery: left submandibular gland resection for a benign tumor. Known allergies were as follows: ibuprofen, surgical suture material, peanuts, lactose, and teicoplanin. Following ingestion of chicken-and-palm-heart pie on Day 0, which he described as having an unusual or off-flavor at the time of consumption, he developed malaise, nausea, vomiting, and progressive weakness, subsequently presenting with bilateral ptosis, diplopia, dysarthria, dizziness, and respiratory failure. Orotracheal intubation was performed for airway protection on Day 1. A test for organophosphate poisoning was performed and was negative, excluding cholinergic toxidrome. On Day 1, heptavalent botulinum antitoxin was administered before FUNED results; biological samples were collected simultaneously.

On Day 4, the patient was transferred to the HFR ICU. On admission, a critical event occurred. Severe bradycardia was documented on cardiac monitoring, with a minimum heart rate of 29 beats per minute, with a palpable pulse maintained throughout. Two ampoules of intravenous atropine were administered with complete rhythm recovery and hemodynamic stabilization. An echocardiogram confirmed preserved biventricular systolic function (ejection fraction 66%), excluding primary cardiac pathology. This episode of

severe vagotonic bradycardia represents an infrequent but well-recognized autonomic manifestation of botulism, reflecting presynaptic cholinergic blockade at sinoatrial node parasympathetic terminals. Tracheostomy was performed on Day 10. The FUNED result was negative; the diagnosis of probable botulism was maintained on clinical-epidemiological grounds.

The main complications were (1) VAP, treated with sequential antibiotic therapy (piperacillin-tazobactam, linezolid, and meropenem), with teicoplanin discontinued after a single dose due to anaphylaxis; (2) infectious thrombophlebitis of the right upper limb, with deep venous thrombosis of the right brachial vein and thrombophlebitis of the right basilic and cephalic veins confirmed on Doppler ultrasound on Day 17, requiring 28 days of antibiotic therapy (linezolid/daptomycin/linezolid sequential scheme) and therapeutic enoxaparin; (3) late hospital-acquired pneumonia on Day 30; and (4) gastroparesis managed with temporary parenteral nutrition, with transient transaminase elevation attributed to parenteral support and resolved after its discontinuation.

The patient was decannulated on Day 24 and transferred from the ICU to the general ward. At the time of the last clinical note reviewed for this report (Day 41), he was stable in room air, with mild bilateral residual ptosis showing progressive improvement, increasing oral diet tolerance, and no active infectious parameters. Transfer to a transitional rehabilitation unit for motor, functional, and nutritional rehabilitation was planned, with outpatient neurology and psychiatry follow-up.

Electrodiagnostic Study: Day 36 of Illness

The same protocol and equipment were used as in Case 1. Extremity temperature was slightly cool, as noted in the report.

Motor nerve conduction studies showed reduced CMAP amplitude in the right median nerve (3.2 mV; reference ⩾4.0 mV), with normal distal latency (4.6 ms) and conduction velocity (55.9 m/s). Post-exercise testing showed no significant facilitation with submaximal voluntary contraction alone (3.4 mV). The right ulnar nerve CMAP amplitude was normal (6.0 mV). Lower limb motor studies (right tibial and deep peroneal nerves) showed normal amplitudes, latencies, and velocities. F-wave minimum latencies were within normal limits.

Sensory nerve conduction studies were normal for all nerves tested: right ulnar (25.8 µV; 63.6 m/s), radial (20.3 µV; 51.5 m/s), median (27.1 µV; 51.9 m/s), sural (10.7 µV; 35.9 m/s), and superficial peroneal (7.1 µV; 36.9 m/s).

Needle EMG of right proximal upper limb muscles (biceps brachii, deltoid, and extensor digitorum communis) showed the same myopathic pattern as Case 1: early recruitment with short-duration, low-amplitude MUPs (+1/+1), without spontaneous activity at rest. Orbicularis oculi, first dorsal interosseous, and all lower limb muscles were normal.

RNS was performed on the right median nerve (recording from abductor pollicis brevis), right ulnar nerve (abductor digiti minimi), bilateral facial nerves (orbicularis oculi and nasalis), and left deep peroneal nerve (extensor digitorum brevis) at 3 Hz and 30-40 Hz. Low-frequency RNS (3 Hz) of the right median nerve showed a significant decremental response, with a maximum amplitude decrement of 11.2% and area decrement of 17.6% at the fifth stimulus (>10%). High-frequency RNS (30 Hz) of the right ulnar nerve demonstrated pathological post-tetanic facilitation with a maximum CMAP amplitude increment exceeding 57% (>30%). The left deep peroneal nerve showed no significant decrement or increment at either frequency, consistent with milder or relatively recovered presynaptic dysfunction in the lower limb terminals, mirroring the clinical pattern of predominant upper limb involvement. Bilateral facial nerves showed no significant decrement at 3 Hz, compatible with relative recovery at facial terminals by Day 36.

The electrodiagnostic study was consistent with moderate presynaptic neuromuscular junction dysfunction in the functional recovery phase (Day 36 of illness), with an identical diagnostic conclusion to Case 1. The selective upper limb motor involvement with normal lower limb nerve conductions and preserved sensory studies further supports a pure presynaptic NMJ disorder, excluding generalized neuropathic or myopathic processes.

Repetitive nerve stimulation findings for both cases are summarized in Table *4*, and needle EMG findings in Table *5*.

Nerve/Frequency	Case	Baseline amplitude	Nadir/Peak amplitude	Decrement	Facilitation	Threshold	Result
Case 1: Female, 24 years							
Facial R: 3 Hz	1	860 µV	820 µV (nadir)	−32.9%	—	>10%	Abnormal ↑
Ulnar R: 3 Hz	1	2.6 mV	2.5 mV (nadir)	5.5%	—	>10%	Normal
Ulnar R: 30 Hz	1	2.7 mV (1 Hz)	5.9 mV (peak)	—	+118%	>30%	Pathological ↑
Ulnar R: 40 Hz	1	2.7 mV (1 Hz)	4.4 mV (peak)	—	+63%	>30%	Pathological ↑
Case 2: Male, 24 years							
Facial R: 3 Hz	2	1.2 mV (1 Hz)	—	≤10%	—	>10%	Normal
Facial R: 30 Hz	2	1.2 mV (1 Hz)	10.8 mV (peak)	—	+800%	>30%	Pathological ↑
Median R: 3 Hz	2	3.6 mV	—	≤13%	—	>10%	Normal / borderline
Median R: 40 Hz	2	3.6 mV	—	—	+76% (area)	>30%	Pathological ↑
Ulnar R: 30 Hz	2	3.8 mV (1 Hz)	6.5 mV (peak)	−57.2% (nadir)	+71%	>30%	Pathological ↑

TABLE 4: Repetitive nerve stimulation (RNS): Cases 1 and 2 (Day 36)

Decrement: change between the 1st and 4th/5th responses at 3 Hz (abnormal >10%). Facilitation: percent increase from 1-Hz baseline to peak amplitude during high-frequency stimulation (30–40 Hz; abnormal >30%). Both cases show the presynaptic neuromuscular junction (NMJ) pattern: significant decrement at 3 Hz (Case 1: facial nerve area decrement 32.9%; Case 2: median nerve amplitude decrement 11.2%, area decrement 17.6%; threshold >10%) and pathological facilitation at high-frequency stimulation.

Muscle	Case	Spontaneous activity	MUP morphology	MUP amplitude	Recruitment	Finding
Case 1: Female, 24 years						
Frontalis R	1	Absent	Short	Low ↓	Early / full	Myopathic ↓
Orbicularis Oculi R	1	Absent	Normal	Normal	Full	Normal
Deltoid R	1	Absent	Short	Low ↓	Early / full	Myopathic ↓
Extensor Digitorum Communis R	1	Absent	Short	Low ↓	Early/full	Myopathic ↓
1st Dorsal Interosseous R	1	Absent	Normal	Normal	Full	Normal
Biceps Brachii R	1	Absent	Short	Low ↓	Early / full	Myopathic ↓
Iliopsoas L	1	Absent	Normal	Normal	Full	Normal
Vastus Medialis L	1	Absent	Normal	Normal	Full	Normal
Medial Gastrocnemius L	1	Absent	Normal	Normal	Full	Normal
Tibialis Anterior L	1	Absent	Normal	Normal	Full	Normal
Case 2: Male, 24 years						
Frontalis R	2	Absent	Normal	Normal	Full	Normal
Orbicularis Oculi R	2	Absent	Normal	Normal	Full	Normal
Deltoid R	2	Absent	Short	Low ↓	Early/full	Myopathic ↓
Extensor Digitorum Communis R	2	Absent	Short	Low ↓	Early/full	Myopathic ↓
1st Dorsal Interosseous R	2	Absent	Normal	Normal	Full	Normal
Biceps Brachii R	2	Absent	Short	Low ↓	Early/full	Myopathic ↓
Iliopsoas L	2	Absent	Normal	Normal	Full	Normal
Vastus Medialis L	2	Absent	Normal	Normal	Full	Normal
Medial Gastrocnemius L	2	Absent	Normal	Normal	Full	Normal
Tibialis Anterior L	2	Absent	Normal	Normal	Full	Normal

TABLE 5: Needle electromyography: Cases 1 and 2 (Day 36)

MUP: motor unit potential. Spontaneous activity absent in all muscles (no fibrillations, positive sharp waves, or fasciculations). Short/low-amplitude MUPs with early full recruitment: myopathic pattern, affecting cranial and proximal upper-limb muscles bilaterally, sparing distal and lower-limb muscles.

Table 6 summarizes the clinical, diagnostic, electrophysiological, and outcome characteristics of both cases.

Characteristic	Case 1 (Female, 24 years)	Case 2 (Male, 24 years)
Implicated Food/Location	Chicken-and-palm-heart pie; same bakery, Belo Horizonte	Chicken-and-palm-heart pie; same bakery, Belo Horizonte (off-flavor noted)
Residence/Initial Hospital	Sete Lagoas, MG/Sete Lagoas Municipal Hospital	Sete Lagoas, MG/Sete Lagoas Municipal Hospital
Incubation Period	~6 hours (Day 0)	~6 hours (Day 0)
Neurological Symptoms	Bilateral ptosis, diplopia, dizziness, descending flaccid paralysis, respiratory failure	Bilateral ptosis, diplopia, dysarthria, dizziness, descending flaccid paralysis, respiratory failure
Orotracheal Intubation	Day 1 (inability to protect airway)	Day 1 (respiratory deterioration)
Antitoxin (Heptavalent Equine)	Day 1, before FUNED results	Day 1, before FUNED results
FUNED Toxin Assay	Negative (sample collected pre-antitoxin; result received Day 25)	Negative (sample collected pre-antitoxin)
Organophosphate Test/CSF	Not performed/Normal: 4 cells, protein 27 mg/dL, excludes GBS	Negative/Not performed
Tracheostomy/Decannulation	Day 9/Day 42	Day 10/Day 24
Infectious Complications	VAP (MSSA); hospital-acquired pneumonia	VAP; right upper limb infectious thrombophlebitis (brachial DVT + basilic/cephalic veins); late hospital-acquired pneumonia
Autonomic/GI Complications	Gastroparesis; elevated lipase/amylase (>3× ULN)	Severe bradycardia (HR nadir 29 bpm, reversed with atropine); gastroparesis
Total Hospitalization (HFR)	42 days (discharged Day 45)	~41 days (transferred Day 41)
EMG: Day of Illness	Day 36	Day 36
EMG: CMAP Amplitudes	Median R: 1.6 mV (reduced; ref. ≥4.0 mV); Ulnar R: 3.3 mV (low-normal); lower limbs normal	Median R: 3.2 mV (reduced; ref. ≥4.0 mV); Ulnar R: 6.0 mV (normal); lower limbs normal
EMG: Low-Frequency RNS (3 Hz)	Facial nerve R: max area decrement 32.9% (threshold >10%)	Median nerve R: max amplitude decrement 11.2%; area decrement 17.6% (threshold >10%)
EMG: High-Frequency RNS (30–40 Hz)	Ulnar nerve R: max amplitude increment >130% at 40 Hz (threshold >30%)	Ulnar nerve R: max amplitude increment >57% at 30 Hz (threshold >30%)
EMG: Needle Findings	Early recruitment, short-duration low-amplitude MUPs: biceps, deltoid, EDC (proximal UL); lower limbs normal	Early recruitment, short-duration low-amplitude MUPs: biceps, deltoid, EDC (proximal UL); lower limbs normal
Serotype Identified	Not determined	Not determined
Condition at discharge/transfer	Mild residual proximal weakness; no cranial nerve deficits; outpatient multidisciplinary rehabilitation	Mild bilateral residual ptosis (resolving); progressive oral diet; transferred to transitional rehabilitation unit

TABLE 6: Comparative clinical, diagnostic, electrophysiological, and outcome characteristics of the two botulism cases from the same outbreak

FUNED: Fundação Ezequiel Dias; GBS: Guillain-Barré syndrome; VAP: ventilator-associated pneumonia; MSSA: methicillin-sensitive *Staphylococcus aureus*; DVT: deep vein thrombosis; HR: heart rate; GI: gastrointestinal; ULN: upper limit of normal; HFR: Hospital Felicio Rocho; EMG: electromyography; CMAP: compound muscle action potential; RNS: repetitive nerve stimulation; MUPs: motor unit potentials; EDC: extensor digitorum communis; UL: upper limb; R: right.

Discussion

Criteria supporting the diagnosis of probable botulism

Both cases fulfilled the criteria for probable foodborne botulism as defined by the Brazilian Ministry of Health and the CDC clinical guidelines [10,11]. The epidemiological link was unambiguous: three individuals

consumed the same chicken-and-palm-heart pie from the same bakery in Belo Horizonte on the same date, all developing a compatible clinical syndrome, with one fatality. The classic neurological presentation, symmetric descending flaccid paralysis with bulbar predominance, preserved consciousness, absence of fever at onset, and no sensory deficits, was present in both surviving patients [8]. The incubation period of approximately six hours is consistent with serotypes A and B, the predominant serotypes in Brazilian outbreaks, and may reflect a relatively high toxin load in the implicated food [10].

In Case 1, CSF analysis was normal (4 cells/mm³, protein 27 mg/dL, glucose 72 mg/dL, negative cultures), effectively excluding Guillain-Barré syndrome, which classically demonstrates cytoalbuminous dissociation, and central nervous system infectious processes. In Case 2, a negative organophosphate assay excluded cholinergic toxidrome, an important differential diagnosis in the context of acute flaccid paralysis following suspected food ingestion. Together, the electrodiagnostic findings in both cases provided objective, quantifiable confirmation of presynaptic neuromuscular junction dysfunction, consolidating the diagnosis in the absence of positive toxicology [9].

The negative FUNED mouse bioassay results are fully consistent with the diagnosis of probable botulism. The assay has a reported sensitivity of approximately 60%-70% in serum and stool collected during the acute phase, declining substantially after antitoxin administration and beyond the first 48-72 hours of illness [1,3]. Although both specimens were collected before antitoxin in this outbreak, they were processed 24 days after collection, which may have compromised circulating toxin integrity. The absence of serotype identification represents a limitation in the complete characterization of this outbreak.

Pathophysiology and electrophysiological basis

Botulinum toxin is a zinc-dependent metalloprotein of approximately 150 kDa, composed of a heavy chain mediating neurospecific binding and endocytosis and a light chain exerting endopeptidase activity on SNARE proteins within the nerve terminal [2]. SNARE cleavage irreversibly prevents acetylcholine vesicle fusion with the presynaptic membrane. Clinical recovery does not depend on reversal of this blockade, which is permanent, but rather on axonal sprouting and the formation of new neuromuscular junctions, a process requiring weeks to months [12].

The electrodiagnostic triad documented in both cases directly reflects the presynaptic mechanism [8,9]. Reduced CMAP amplitudes on baseline nerve conduction studies indicate diminished quantal content of acetylcholine released per nerve impulse, resulting in fewer activated muscle fibers and a smaller compound potential. The decremental response at low-frequency RNS (3 Hz) reflects progressive depletion of the immediately releasable vesicle pool, which is already critically reduced by toxin blockade, causing decreasing CMAP amplitudes with successive stimuli. Pathological post-tetanic facilitation at high-frequency RNS (30-40 Hz), with CMAP amplitude increments exceeding 30% in both cases and peaking above 130% in Case 1 and 57% in Case 2, reflects calcium accumulation at the presynaptic terminal during high-frequency stimulation, transiently mobilizing reserve vesicles into the immediately releasable pool and temporarily increasing quantal content. This facilitation phenomenon is the key electrophysiological feature distinguishing presynaptic NMJ disorders from myasthenia gravis, which produces a decremental response at low-frequency RNS without pathological facilitation at high frequency [9].

The needle EMG pattern observed in both cases, early recruitment with short-duration, low-amplitude motor unit potentials and no spontaneous activity at rest, predominantly in proximal upper limb muscles, does not reflect primary muscle disease. Rather, it represents the reduced number of muscle fibers activated per motor unit as a consequence of partial NMJ blockade [8,9]. This finding is frequently underreported in botulism EMG descriptions and was present in both cases; its systematic documentation may strengthen future electrodiagnostic characterizations of the disease.

The selective involvement of upper limb motor terminals, with normal lower limb nerve conductions and RNS in both cases, mirrors the clinical pattern of predominant upper limb and bulbar weakness. It is important to note that the electrodiagnostic studies were performed on Day 36 of illness, the functional recovery phase, when CMAP amplitudes had partially normalized and facilitation, while still clearly pathological, was less dramatic than would be expected in the acute phase (Days 7-14). The fact that diagnostic criteria were unequivocally met at this late stage underscores both the severity of the initial NMJ blockade and the robustness of electrodiagnostic testing as a confirmatory tool even when performed during recovery [9].

Epidemiology, public health, and the role of palm heart

Foodborne botulism is subject to mandatory immediate notification in Brazil [11]. The occurrence of two or more clinically compatible cases linked to the same food source defines an outbreak and mandates epidemiological investigation, including submission of food and biological samples to reference laboratories and sanitary inspection of the implicated establishment. Brazil registered 218 confirmed cases between 2000 and 2021, with a case-fatality rate of approximately 10% to 15% [7]. Types A and B predominate, and palm heart in brine, particularly artisanally produced, is the most frequently identified vehicle, consistent with this outbreak [4,5].

The fatal outcome in the third exposed individual, approximately 30 days after the intoxication, warrants specific comment. Although laboratory confirmation was not obtained, the epidemiological link is unambiguous, and her death is fully consistent with the clinical trajectory of severe botulism in an elderly patient with limited physiological reserve [13]. Older patients have reduced respiratory muscle reserve, lower tolerance to progressive neuromuscular paralysis, and higher vulnerability to fatal respiratory failure. Sanitary inspection of the implicated bakery and recall of the suspected food lot are indispensable steps in outbreak management and were initiated following notification.

Therapeutic management

In both cases, heptavalent botulinum antitoxin was administered on Day 1 of symptoms, before FUNED results were available, the recommended approach of treating on clinical suspicion without awaiting laboratory confirmation [10]. Since antitoxin neutralizes only free circulating toxin and does not reverse established NMJ blockade, its benefit is greatest within the first 24-48 hours of illness, before complete toxin internalization by nerve terminals [14]. The need for prolonged mechanical ventilation in both cases reflects the degree of NMJ blockade already established at the time of treatment initiation, despite the relatively prompt antitoxin administration.

Objective spirometric parameters (forced vital capacity, maximal inspiratory pressure) were not systematically documented in the available records, a recognized limitation of this report. Intubation was performed on clinical grounds in both cases, consistent with current guidelines recommending early airway management without waiting for hypoxemia [13]. Tracheostomy on Days 9-10 was appropriate given the anticipated ventilatory dependency exceeding 14 days.

The severe bradycardia documented in Case 2, with a minimum heart rate of 29 beats per minute, represents an infrequent but potentially life-threatening autonomic manifestation of botulism. The proposed mechanism involves presynaptic cholinergic blockade at sinoatrial node parasympathetic terminals, with direct impairment of intrinsic sinus node pacemaker function [15]. This complication reinforces the importance of continuous cardiac monitoring with atropine immediately available in all severe botulism cases managed in the intensive care unit.

Neostigmine, a cholinesterase inhibitor used in both patients for the management of gastroparesis, potentiates cholinergic tone at enteric synapses, facilitating gastrointestinal motility without antagonizing the presynaptic NMJ blockade mechanism [1]. It represents a safe and effective therapeutic option for autonomic gastrointestinal dysfunction in botulism.

Prognosis and rehabilitation

Recovery from botulism is governed by axonal sprouting and the formation of new functional neuromuscular junctions, a biologically limited process that cannot be pharmacologically accelerated [12]. Muscle strength recovers in the inverse order of involvement, with cranial nerve musculature often the last manifestations to fully resolve [8]. In severe cases, complete functional recovery may take three to 12 months. Persistent fatigue, exercise intolerance, residual autonomic dysfunction, and cognitive symptoms may persist for years in survivors of severe botulism [16], reinforcing the necessity of structured, long-term multidisciplinary rehabilitation and prolonged neurological follow-up. Both patients were referred to multidisciplinary rehabilitation programs in line with evidence-based recommendations [17].

Conclusions

These two cases illustrate the clinical and diagnostic challenges of severe foodborne botulism: negative toxicology, potentially fatal autonomic and infectious complications, and prolonged mechanical ventilation. The fatal outcome in the third exposed individual underscores the particular risk in elderly patients with lower physiological reserve.

EMG performed on Day 36 objectively confirmed pre-synaptic neuromuscular junction dysfunction: reduced CMAPs, significant decrement at 3 Hz, and pathological post-tetanic facilitation at 30-40 Hz diagnostic findings even in the recovery phase. Negative toxicology does not exclude the diagnosis when the clinical-epidemiological context is compatible. Early antitoxin administration, anticipatory airway management, and multidisciplinary intensive support are determinants of survival. In the Brazilian context, surveillance of canned heart of palm as a high-risk vehicle remains a public health imperative.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Caroline F. Melo

Acquisition, analysis, or interpretation of data: Caroline F. Melo

Drafting of the manuscript: Caroline F. Melo

Critical review of the manuscript for important intellectual content: Caroline F. Melo

Supervision: Caroline F. Melo

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