EUR REV MED PHARMACOL SCI
2025; 29: 416-424

CLINICAL AND ELECTROPHYSIOLOGICAL FEATURES OF FOODBORNE BOTULISM: A RETROSPECTIVE CASE SERIES

F. AL-HUSSAIN[1], H. ALBULAIHI[2], R. ALHAMMAD[2], S. ALSUBAIE[1], A. ALHAMMAD[1], S. BASHIR[3]

[1]Department of Neurology, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia
[2]Department of Neurology & Neuromuscular Medicine, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia
[3]Neuroscience Center, King Fahad Specialist Hospital, Dammam, Saudi Arabia

Corresponding Author
Fawaz Al-Hussain, MD; e-mail: faalhussain@ksu.edu.sa

ABSTRACT – *Background:* Foodborne botulism is a rare but potentially serious and lethal disease that is considered a threat to public health systems across the globe. Botulism is a paralytic disorder caused by the neurotoxin, produced by *Clostridium botulinum*, which acts upon peripheral cholinergic nerve terminals by inhibiting acetylcholine release, subsequently causing denervation to muscle fibers.

Case Series: In April 2024, an outbreak of foodborne botulism was reported in Riyadh, Saudi Arabia, following consumption of contaminated fast food. Four patients were affected: two females and two males, aged 14 to 21 years. All patients developed neurological symptoms, including cranial nerve involvement, dysphagia, and generalized weakness. Three patients progressed to severe hypercapnic respiratory failure requiring prolonged mechanical ventilation. Electrophysiological studies demonstrated characteristic findings of presynaptic neuromuscular junction dysfunction.

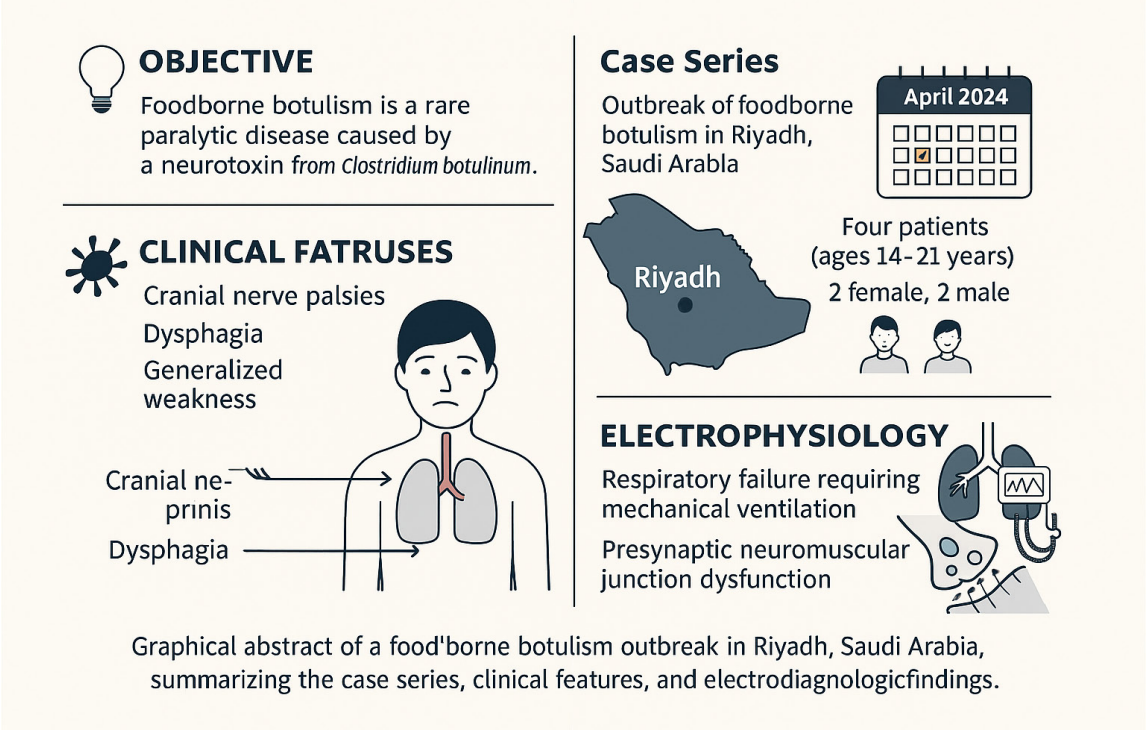
Conclusions: This case series adds to existing knowledge by providing detailed descriptions of the clinical course, neurological examination findings, and electrodiagnostic features of foodborne botulism cases in Saudi Arabia. Our findings highlight that, despite early antitoxin administration, patients can develop prolonged neuromuscular weakness requiring extended mechanical ventilation. This underscores the importance of considering botulism in the differential diagnosis of acute flaccid paralysis and the need for timely electrophysiological evaluation to guide management and prognosis.

KEYWORDS: Foodborne botulism, Electrophysiology, Botulinum toxin, Neuromuscular transmission, Case series.

INTRODUCTION

Foodborne botulism is a rare but potentially life-threatening neuroparalytic condition. It remains a significant public health concern worldwide due to its rapid progression, potential for outbreaks, and requirement for urgent supportive care. Botulism results from intoxication with botulinum neurotoxins (BoNTs), which are produced by the anaerobic, gram-positive, spore-forming bacterium *Clostridium* botulinum[1-5]. These neurotoxins target peripheral cholinergic nerve terminals, blocking acetylcholine release at the neuromuscular junction and leading to reversible flaccid paralysis[1,2].

There are several recognized forms of botulism, including foodborne, wound, infant, iatrogenic,



Graphical Abstract. Graphical abstract of a foodborne botulism outbreak in Riyadh, Saudi Arabia, summarizing the case series, clinical features, and elctrodiagnologic findings of the study.

and adult intestinal toxemia botulism, with foodborne botulism accounting for most outbreak-associated cases[3-5]. Foodborne botulism typically arises from ingestion of improperly processed, canned, or fermented foods that create an anaerobic environment favorable for spore germination and toxin production[3,4]. Risk factors include low salt, low-acid environments, absence of preservatives, and inadequate thermal processing[6,7]. *C. botulinum* growth can be inhibited by refrigeration below 4°C, heating above 121°C, low water activity, or acidity below pH 4.5[6].

Clinically, foodborne botulism is characterized by the acute onset of bilateral cranial nerve palsies, followed by symmetric descending flaccid paralysis, which can lead to respiratory failure and autonomic dysfunction if not promptly treated[8,9]. Gastrointestinal symptoms, such as nausea, vomiting, and abdominal cramps may precede neurological manifestations, often causing delays in diagnosis[8].

Although the administration of botulinum antitoxin remains the mainstay of treatment, its efficacy is limited to neutralizing circulating toxin and does not reverse established neurotoxicity. Therefore, early clinical suspicion and supportive care, including mechanical ventilation and monitoring for autonomic instability, are essential[10]. Emerging therapeutic options, such as plasmapheresis, have been discussed in isolated case reports and small series, with some suggesting potential benefit in severe or refractory presentations, although robust evidence is lacking[11-13].

Given the clinical and public health relevance of such outbreaks, this study aimed to describe the clinical presentation, disease progression, and electrophysiological findings in four patients affected by a foodborne botulism outbreak in Riyadh, Saudi Arabia, in April 2024. The objective is to contribute to the existing literature and inform diagnostic and management strategies in future suspected botulism cases.

CASE PRESENTATION

This retrospective case series study collected data from the medical record of the patients who visited King Saud University Medical City in Riyadh, Saudi Arabia from April 21st to April 24th 2024, There were a total of four patients, and detail such as meal ingestion time, onset of symptoms, vital signs, initial neurological exam findings and specific lab results [C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and creatine kinase (CK)] were recorded, as shown in Table I.

The study was approved by the Institutional Review Board of King Saud University (Ref. No. E-24-8822; approval date: 13 June 2024). As a retrospective study involving the review of medical records, all data were fully anonymized prior to analysis. Data collection for this study commen-

ced only after obtaining full approval from the IRB. The IRB waived the requirement for informed consent in accordance with institutional and national guidelines for research involving minimal risk and de-identified data.

Initial nerve conduction studies were conducted after a clinical diagnosis of botulism was made. These studies were performed by two investigators (HMA and RMA) who were involved in the study. Compound muscle action potentials were recorded following distal stimulation of ulnar, median, and tibial nerves at specific muscle locations. Peroneal compound muscle action potentials were also recorded. The spinal accessory nerve was stimulated when possible. Post-activation CMAP amplitudes were measured to calculate the percentage increment. Slow repetitive nerve stimulation (RNS) was performed to assess decremental response, with a positive response defined as more than 10%. Fast (50 Hz) RNS was not conducted due to a lack of sedation. All motor studies used a supramaximal stimulus (pulse duration 0.1ms), with proper skin temperature maintained at ≥33°C. The Natus Keypoint electromyography (EMG) machine was utilized for these studies.

Patient 1

Patient 1 was a 21-year-old female who presented two days after exposure with diplopia and ptosis, without gastrointestinal symptoms (Table I). Neurological examination revealed dysphonia, dysphagia, dyspnea, orthopnea, proximal upper extremity weakness, and signs of autonomic dysfunction (**Supplementary Table I**). Deep tendon reflexes were preserved in the lower limbs but diminished in the upper limbs. Sensory testing was intact, and no cranial nerve palsies were noted.

Follow-up neurological examinations on days 6-9 demonstrated preserved ocular motility, intact motor strength, and normalization of most reflexes (Table II). Mechanical ventilation was not required. Her extraocular movements were regular, and ptosis was absent on examination. She did not receive adjunctive therapies such as intravenous immunoglobulin (IVIG) or plasmapheresis. Botulinum antitoxin was administered on day 4 post-exposure (Table III).

Initial electrodiagnostic studies performed on days 9-11 showed normal sensory responses, a modest CMAP increment post-exercise (APB 32%; ADM 12%), and no decrement with 3Hz repetitive nerve stimulation (Table IV). Follow-up studies on days 16-19 revealed stable findings with continued clinical improvement (Table V).

Patient 2

Patient 2, a 14-year-old female and the sibling of patient 1, presented 22 hours after exposure with progressive oculobulbar weakness, including ptosis

Table I. Clinical features and laboratory findings for each patient diagnosed with foodborne botulism, including demographic data, presenting symptoms, neurological signs, vital signs, and relevant laboratory results at presentation.

Variable	Patient 1	Patient 2	Patient 3	Patient 4
Age/Sex	21F	14F	19M	19M
Exposure to onset (days)	2	<1	1	2
GI symptoms	None	None	None	Vomiting
Key neurological symptoms	Diplopia, ptosis, dysphagia, dyspnea, proximal UE weakness, autonomic dysfunction	Ptosis, oculobulbar weakness, dysphagia, dyspnea, autonomic instability	Diplopia, ptosis, dysphagia, dysarthria, generalized weakness	Ptosis, ophthalmoplegia, dysphagia, dysphonia, respiratory failure
Mechanical ventilation (hours post-onset)	NA	25	64	24
Reflexes	UE ↓, LE normal	UE absent, LE normal	Normal	Normal
Botulinum toxin	Positive	Positive	Positive	NA
CRP (mg/L)	62	18.9	NA	1.6
ESR (mm/hr)	62.1	2	NA	5
CK (U/L)	47	45	30	417

CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, CK: creatine kinase. UE: Upper Extremity, LE: Lower Extremity, GI: Gastrointestinal.

Table II. Follow-up neurological examination findings for all four patients conducted between days 6 and 9 after the onset of neurological symptoms, including assessments of cranial nerve function, muscle strength, and reflexes.

	Patient			
	1	**2**	**3**	**4**
Pupils	3 mm, ERL	3 mm, ERL	3 mm, ERL	3 mm, sluggish reaction to light
Ocular Motility Estimated Range of Motion				
Up gaze	100% No limitations	20% bilaterally	70% bilaterally	80% bilaterally
Down gaze	100% No limitations	30% bilaterally	60% bilaterally	70% bilaterally
Abduction	100% No limitations	40% bilaterally	80% bilaterally	50% bilaterally
Adduction	100% No limitations	100% No limitations	80% bilaterally	60% bilaterally
Asymmetry in EOM limitations	NA	Yes	Yes	Yes
Ptosis	None	Partial bilateral	Complete bilateral	Complete bilateral
Fatigability in ptosis and/or ophthalmoplegia	NA	Yes	Yes	Yes
Facial sensation	Intact	Intact	Intact	Intact
Facial weakness	None	Yes	Yes	Yes
Trapezius	5	3-	4	4
Neck flexion	4	1	4	1
Neck extension	5	1	4	4-
Deltoid (right/left)	4-	2	2/3	1
Elbow flexion (right/left)	5	3	4+	4/3
Elbow extension (right/left)	4+	3	4	4+/4
Wrist flexion	5	3	4	4
Wrist extension	4-	4	3	3
Finger flexion (FDS, FDP)	4+	4-	4+	4+
Finger extension	3	2	3	3
APB	3	3	4	4
Hip flexion (right/left)	3	2	4/4+	2
Hip extension (right/left)	5	3	4	3/4
Hip adduction	5	2	4-	1
Hip abduction	5	2	4	4-
Knee flexion	5	4	4	4
Knee extension	5	3	5	4-
Ankle DF	5	3	4+	4+
Ankle PF	5	4	4+	5
Ankle inversion	5	4	5	5
Ankle eversion	5	4	5	5
EHL	4+/5	4	4	4
Asymmetry in power strength	Yes	No	No	Yes
DTR				
Triceps	2/2	0/0	0/0	2/1
Biceps	0/0	0/0	0/0	0/0
Brachioradialis	1/1	0/0	0/0	0/1
Patellar	2/2	2/2	0/0	1/1
Ankle	2/2	2/2	0/0	2/2

EOM: Extraocular movements, NA: not applicable, FDS: Flexor digitorum superficialis, FDP: Flexor digitorum profundus, APB: Abductor policis brevis, DF: Dorsiflexion, PF: Plantarflexion, EHL: Extensor hallucis longus, DTR: Deep tendon reflex. ERL: Equal and Reactive to Light.

Table III. Interval in days from exposure to contaminated food to administration of anti-botulinum toxin for each patient included in the case series.

Treatment	Patient			
	1	**2**	**3**	**4**
Interval from exposure to anti-botulinum toxin, days	4	4	5	6
Therapies received for Suspected Myasthenic Crisis or GBS				
IVIG	none	none	none	3 sessions (0.4 g/kg per dose)
Plasmapheresis	none	5 sessions	one session	none
Therapies received for botulism				
Plasmapheresis	none	5 sessions	none	5 sessions
3,4, DAP	no	no	no	yes
Outcomes				
Presentation-to-intubation; hours	NA	8	52	4
Duration of mechanical ventilation, days	NA	57	32	51

IVIG: intravenous immunoglobulin, GBS: Guillain-Barré Syndrome. DAP: diaminopyridine.

and limited eye movements (Table I). She developed dysphagia, dysphonia, and dyspnea, although no gastrointestinal symptoms were reported.

Neurological examination revealed cranial nerve involvement, reduced motor strength, particularly in the neck and upper limbs, and areflexia in the upper limbs, alongside autonomic instability with notable fluctuations in blood pressure and heart rate (**Supplementary Table I**). Deep tendon reflexes were absent in the upper limbs but preserved in the lower limbs. Follow-up neurological examinations demonstrated partial recovery of extraocular movements and muscle strength, although fatigue and ptosis persisted (Table II). She required mechanical ventilation within 8 hours of presentation (Table III) and received botulinum antitoxin on day 4 post-exposure, followed by five sessions of plasmapheresis for management.

Initial electrophysiological studies showed low baseline CMAP amplitudes with minimal post-exercise increment (APB 16%, ADM 18%) (Table IV). Follow-up testing revealed persistently low CMAPs but notable improvement in tibialis anterior (TA) responses, with a 67% increment (Table V). She remained on mechanical ventilation for a total of 57 days.

Patient 3

Patient 3 was a 19-year-old male who presented one day post-exposure with diplopia and ptosis (Table I). He exhibited extensive cranial nerve dysfunction, including ophthalmoplegia, as well as dysphagia, dysarthria, dyspnea, and generalized muscle weakness. No gastrointestinal symptoms were reported.

Within 52 hours of presentation, he developed respiratory failure and required mechanical ventilation (Table III). Initially, he was misdiagnosed with a myasthenic crisis and received one session of plasmapheresis before establishing the correct diagnosis. Botulinum antitoxin was administered on day 5 post-exposure.

Neurological examination revealed ophthalmoplegia, facial weakness, flaccid limb weakness, and reduced deep tendon reflexes (Supplementary Table I). Initial electrophysiological studies demonstrated reduced CMAP amplitudes with post-exercise increment (APB 27%; ADM 35%) and myopathic features on EMG (Table IV). Follow-up studies showed persistently abnormal findings with limited improvement (Table V). He remained intubated for a total of 32 days.

Patient 4

Patient 4 was a 19-year-old male who presented 48 hours post-exposure with vomiting, followed by cranial nerve deficits including bilateral ptosis, ophthalmoplegia, dysphagia, and dysphonia (Table I). Follow-up examinations on days 6-9 demonstrated partial recovery of ocular movements and improved muscle strength (Table II). Within 4 hours of admission, he developed severe autonomic dysfunction and respiratory failure, necessitating mechanical ventilation (Table III).

Table IV. Results of initial electrodiagnostic studies, including nerve conduction studies (NCS), electromyography (EMG), and repetitive nerve stimulation (RNS), performed between days 9 and 11 post-onset for all patients.

NCS parameters	Patient			
	1	2	3	4
Baseline compound muscle action potential amplitudes				
APB	6.5	2.5	5.2	3
ADM	8.3	2.2	6.6	4.6
Trapezius	2.5	NR	1.8	NT
TA	4	NR	2.4	1.6
AHL	12.5	4.8	7.6	2.9
EDB	5.8	1.7	6	3.6
(Percent) increment following 10 seconds isometric exercise				
APB	32	16	27	17
ADM	12	18	35	2
Trapezius	4	NR	31	NT
TA	0	NR	13	0
Decremental response with 3 Hz RNS				
Median	No	No	No	No
Ulnar	No	No	No	No
Peroneal (TA)	No	No	No	No
Spinal accessory	No	No	No	No
EMG findings				
Deltoid	Normal	NA	Myopathic	Myopathic
FDI	Normal	Normal	Normal	Myopathic
Vastus	Normal	NA	Myopathic	Myopathic
TA	Normal	Myopathic	Myopathic	Myopathic

AHL: Abductor hallucis longus. ADM: Abductor digitorum minimi. APB: Abductor policis brevis. FDI: First dorsal interosseous. TA: Tibialis anterior. NR: Not recordable. NT: Not tested. NA: Not applicable. EDB: Extensor digitorum brevis.

Initially, he was treated with intravenous immunoglobulin (IVIG) for suspected Guillain-Barré syndrome, receiving three sessions at 0.4 g/kg, before a diagnosis of botulism was established. Botulinum antitoxin was administered on day 6 post-exposure, followed by five sessions of plasmapheresis. Neurological examination revealed severe ophthalmoplegia and ptosis with fatigability, diffuse proximal and distal muscle weakness, and significant autonomic instability (**Supplementary Table I**).

Initial electrodiagnostic testing revealed low CMAP amplitudes with minimal post-exercise facilitation (APB, 17%; ADM, 2%) (Table IV). Follow-up studies showed slight improvement in CMAP responses (Table V). He required prolonged ventilatory support for a total of 51 days.

DISCUSSION

In the present case series, the most frequent presenting symptoms were acute oculobulbar deficits such as flaccid dysarthria, muffled dysphonia, dysphagia, asymmetric ophthalmoplegia, and ptosis, complete and bilateral in two out of three affected patients. These findings are consistent with prior reports, where cranial nerve involvement is often the earliest and most prominent sign[4-6]. Proximal muscle weakness predominated

over distal weakness, and upper limbs were more affected than lower limbs in all cases. This distribution aligns with previously reported patterns of descending symmetric weakness[7].

Notably, reduced deep tendon reflexes in the upper extremities, with preservation in the lower limbs, were observed in half of our patients at presentation. Patients 3 and 4 initially had normal reflexes, which progressively diminished over 6-9 days. Such a pattern of evolving hyporeflexia, particularly when isolated to the upper limbs, may serve as a diagnostic clue in early or unrecognized outbreaks. Although not specific, this feature has also been documented in differential diagnoses such as myasthenia gravis, Guillain-Barré Syndrome (GBS), and Lambert-Eaton Myasthenic Syndrome (LEMS)[8-10].

All patients showed features of autonomic dysfunction, including fluctuations in heart rate and blood pressure. Autonomic involvement is a well-recognized manifestation of botulism and may include dilated, poorly reactive pupils, dry mouth, orthostatic hypotension, abnormal sweating, and urinary retention[11-13]. In a study on botulism patients, over 70% of them exhibited signs of dysautonomia, which often correlated with disease severity and the need for intensive care[14].

Three of the four patients in our series required mechanical ventilation within 52 hours of presentation, consistent with prior literature indicating early and severe respiratory compromise. Respiratory failure in botulism is multifactorial, stemming from upper airway collapse, diaphragmatic and intercostal muscle weakness, and impaired autonomic regulation of breathing[15,16]. A systematic review reported that nearly half of botulism patients develop respiratory compromise within 48 hours, necessitating prolonged intubation and ICU-level care[17].

Electrophysiological studies played a crucial role in our diagnostic workup. Findings of reduced CMAPs with post-exercise facilitation were observed, indicative of a presynaptic neuromuscular junction disorder. These findings are hallmark features of botulism and help distinguish it from GBS or myasthenia gravis[18-20]. The classical diagnostic criterion includes a greater than 100% increment in CMAP amplitude following high-frequency repetitive nerve stimulation or brief

Table V. Follow-up electro diagnostic studies: nerve conduction studies (NCS), electromyography (EMG), and repetitive nerve stimulation (RNS) (Days 16-19).

NCS parameters	Patient			
	1	2	3	4
Baseline compound muscle action potential amplitudes				
APB	6	3.9	5	5.2
ADM	9.2	5.2	5.1	11.4
Trapezius	2.4	NT	1.6	NT
TA	2.7	1.68	2.1	2.2
EDB	5.2	0.9	5.5	3.1
(Percent) increment following 10 seconds isometric exercise				
APB	10	0	14	25
ADM	14	0	6	0
Trapezius	0	0	0	0
TA	0	67	9	5
Decremental response with 3 Hz RNS				
Median	Yes	NT	No	No
Ulnar	No	No	No	No
Peroneal (TA)	No	NT	No	No
Spinal accessory	No	NT	NT	No

ADM: Abductor digitorum minimi. APB: Abductor policis brevis. TA: Tibialis anterior. NT: Not tested. EDB: EDB: Extensor digitorum brevis.

maximal voluntary contraction[21]. However, more recent studies have shown that a lower threshold of ≥60% or even ≥25% can still provide high sensitivity and specificity, especially in outbreak settings[22-24]. Nonetheless, caution is advised, as some healthy individuals may exhibit similar facilitation responses[25].

Early clinical recognition remains a critical challenge, particularly in the absence of a known outbreak. In our series, patients 3 and 4 were initially misdiagnosed and treated as myasthenia gravis and GBS, respectively. A high index of suspicion is necessary when encountering acute cranial neuropathies with descending paralysis. The presentation of two siblings (patients 1 and 2) ultimately raised concern for foodborne botulism, prompting immediate public health reporting and therapeutic intervention.

Clustering of similar neurological symptoms among individuals who share a common exposure is considered highly suggestive of botulism, especially when no definitive diagnostic test is available in the early stages[26]. Once suspicion was established, antitoxin was administered promptly, in accordance with CDC and WHO guidelines[27], and the Ministry of Health rapidly identified a shared contaminated food source, confirming the outbreak.

In addition to standard antitoxin therapy, plasmapheresis was trialed in two severely affected patients who showed limited improvement after antitoxin administration. Though not widely adopted in botulism management, several case reports and small series have proposed that plasmapheresis may offer adjunctive benefits by enhancing toxin clearance or modulating immune response[24-28]. While our observations suggest possible autonomic stabilization after plasmapheresis, controlled studies are needed to validate its efficacy in this setting.

This case series highlights the importance of early recognition, timely antitoxin administration, and comprehensive supportive care in managing foodborne botulism. It also emphasizes the role of electrophysiology in differential diagnosis and the potential utility of adjunctive therapies in select cases.

Limitations

Despite the informative clinical and electrophysiological findings presented in this case series, several limitations must be acknowledged. The retrospective nature of the study is a key limitation, as it inherently introduces the potential for selection bias, incomplete data capture, and variability in the timing of assessments and interventions. Additionally, the small sample size and single-center setting limit the generalizability of the findings. While our observations suggest potential benefits of adjunctive therapies such as plasmapheresis, controlled prospective studies are required to validate these findings and better define their role in the clinical management of botulism.

CONCLUSIONS

While botulinum antitoxin remains the primary and most evidence-based treatment for botulism, there is limited data supporting additional disease-specific therapies. In this case series, due to the persistent severity of symptoms in cases #2 and #4 despite the timely administration of antitoxin, adjunctive plasmapheresis was trialed. Notable clinical improvement, particularly in autonomic parameters, such as heart rate and blood pressure, was observed following plasmapheresis. Although this intervention cannot be considered standard care based on current evidence, our findings, in alignment with prior reports[24-28], suggest a potential benefit of plasmapheresis in cases of severe or refractory botulism, warranting further investigation.

Conflict of Interest

The authors affirm that there are no conflicts of interest to disclose.

Ethics Approval

This study was approved by the Institutional Review Board of King Saud University (Ref. No. E-24-8822; approval date: 13 June 2024). Data collection for this study commenced only after obtaining full approval from the IRB and in compliance with the Declaration of Helsinki and its latest amendments.

Informed Consent

As a retrospective study involving the review of medical records, all data were fully anonymized prior to analysis. The IRB waived the requirement for informed consent in accordance with institutional and national guidelines for research involving minimal risk and de-identified data.

Authors' Contributions

F. Al-Hussain: conceptualization, methodology, investigation, drafting of the manuscript and final approval of the manuscript. H. Albulaihi: data curation, investigation, review and editing. R. Alhammad: analysis, visualization, review and editing. S. Alsubaie: resources, project administration, data curation. A. Alhammad: investigation, validation, review and editing. S. Bashir: review and editing.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

REFERENCES

1) Sobel J, Tucker N, McLaughlin J, Maslanka S. Foodborne botulism in the United States, 1990–2000. Emerg Infect Dis 2004; 10: 1606-1611.
2) Choudhry SA, Anwar MJ, Afzal M, Shah A. Foodborne botulism, I only had nacho cheese: a case report. Cureus 2017; 9: e1666.
3) Maharjan N, Chirumamilla Y, Karki B, Berrou M, Mcdonald PJ. Foodborne botulism type F: a rare presentation of neurologic manifestation and successful management. Cureus 2024; 16: e62964.
4) Hill SE, Iqbal R, Cadiz CL, Le J. Foodborne botulism treated with heptavalent botulism antitoxin. Ann Pharmacother 2013; 47: e12.
5) Rawson AM, Dempster AW, Humphreys CM, Minton NP. Pathogenicity and virulence of Clostridium botulinum. Virulence 2023; 14: 2205251.
6) National Center for Infectious Diseases US, Division of Bacterial and Mycotic Diseases. Botulism in the United States, 1899-1996: Handbook for epidemiologists, clinicians, and laboratory workers. 1998. Available from: https://stacks.cdc.gov/view/cdc/667. Accessed August 16, 2024.
7) Parrera GS, Astacio H, Tunga P, Anderson DM, Hall CL, Richardson JS. Use of botulism antitoxin heptavalent (A-G) (equine) (BAT) in clinical study subjects and patients: a 15-year systematic safety review. Toxins Basel 2021; 14: 19.
8) Sobel J. Botulism. Clin Infect Dis 2005; 41: 1167-1173.
9) Machamer JB, Vazquez-Cintron EJ, Stenslik MJ, Pagarigan KT, Bradford AB, Ondeck CA, McNutt PM. Neuromuscular recovery from botulism involves multiple forms of compensatory plasticity. Front Cell Neurosci 2023; 17: 1226194.
10) Wakerley BR, Yuki N. Pharyngeal-cervical-brachial variant of Guillain-Barré syndrome. J Neurol Neurosurg Psychiatry 2014; 85: 339-344.
11) Jenzer G, Mumenthaler M, Ludin HP, Robert F. Autonomic dysfunction in botulism B. Neurology 1975; 25: 150.
12) Rao AK, Lin NH, Griese SE, Chatham-Stephens K, Badell ML, Sobel J. Clinical criteria to trigger suspicion for botulism: an evidence-based tool. Clin Infect Dis 2017; 66: S38-S42.
13) Carpentier VT, Weiss N, Salem JE, Joussain C, Levy J, Mariani LL, Montastruc F, Mahé J, Lebrun-Vignes B, Bensmail D, Denys P, Genêt F, Bihan K. Real-life prognosis of neurological complications of botulinum toxin: a nationwide pharmacovigilance study. Ann Phys Rehabil Med 2025; 68: 101924.
14) Rao AK, Sobel J, Chatham-Stephens K, Luquez C. Clinical guidelines for diagnosis and treatment of botulism, 2021. MMWR Recomm Rep 2021; 70: 1-30.
15) Silkina MV, Kartseva AS, Riabko AK, Makarova MA, Rogozin MM, Romanenko YO, Shemyakin IG, Dyatlov IA, Firstova VV. New approach to generating of human monoclonal antibodies specific to the proteolytic domain of botulinum neurotoxin A. Bioimpacts 2024;14: 27680.
16) Lonati D, Schicchi A, Crevani M, Buscaglia E, Scaravaggi G, Maida F, Cirronis M, Petrolini VM, Locatelli CA. Foodborne botulism: clinical diagnosis and medical treatment. Toxins Basel 2020; 12: 509.
17) Witoonpanich R, Vichayanrat E, Tantisiriwit K, Rattanasiri S, Ingsathit A. Electrodiagnosis of botulism and clinico-electrophysiological correlation. Clin Neurophysiol 2009; 120: 1135-1138.
18) Cherington M. Clinical spectrum of botulism. Muscle Nerve 1998; 21: 701-710.
19) Oh SJ, Kurokawa K, Claussen GC, Ryan HF Jr. Electrophysiological diagnostic criteria of Lambert-Eaton myasthenic syndrome. Muscle Nerve 2005; 32: 515-520.
20) Oh SJ. Distinguishing features of the repetitive nerve stimulation test between Lambert-Eaton myasthenic syndrome and myasthenia gravis. J Clin Neuromuscul Dis 2017; 19: 66-75.
21) Potulska-Chromik A, Zakrzewska-Pniewska B, Szmidt-Sałkowska E, Lewandowski J, Siński M, Przyjałkowski W, Kostera-Pruszczyk A. Long lasting dysautonomia due to botulinum toxin B poisoning: clinical-laboratory follow up and difficulties in initial diagnosis. BMC Res Notes 2013; 6: 438.
22) Albers JW, Fink JK. Porphyric neuropathy. Muscle Nerve 2004; 30: 410-422.
23) Gerischer LM, Scheibe F, Nümann A, Köhnlein M, Stölzel U, Meisel A. Acute porphyrias - A neurological perspective. Brain Behav 2021; 11: e2389.
24) Chatham-Stephens K, Fleck-Derderian S, Johnson SD, Sobel J, Rao AK, Meaney-Delman D. Clinical Features of Foodborne and Wound Botulism: A Systematic Review of the Literature, 1932-2015. Clin Infect Dis 2017; 66(swuppl_1): S11-S16.
25) Lindström M, Korkeala H. Laboratory diagnostics of botulism. Clin Microbiol Rev 2006; 19: 298-314.
26) Cosentino G, Alfonsi E, Lonati D, Locatelli CA, Valentino F, Brighina F. Positive ice pack test in a case of foodborne botulism: a clinical note. J Neurol 2012; 259: 2486-2487.
27) Elangovan C. Assessment of neuromuscular transmission. Medscape 2023. Available from: https://emedicine.medscape.com/article/1140870-overview#a. Accessed August 16, 2024.
28) Atabek ME, Yavuz H, Oran B, Karaaslan S, Erkul I. Plasmapheresis as an adjunct treatment in severe botulism. Intensive Care Med 2002; 28: 814.