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Botulism in the Emergency Department: Clinical Characteristics, Diagnostic Challenges, and Patient Outcomes

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Abstract

Objective: Botulism is a rare but potentially life-threatening neuroparalytic disease. In recent years, the increasing use of botulinum toxin for cosmetic and therapeutic purposes has led to a rise in iatrogenic cases. This study aimed to evaluate the clinical characteristics, diagnostic process, management strategies, and outcomes of patients diagnosed with botulism in the emergency department.

Materials and Methods: This retrospective, observational, single-center study included adult patients diagnosed with botulism in the emergency department of a tertiary-care referral hospital between January 1, 2022, and January 1, 2026. The diagnosis was established on clinical grounds based on compatible neurological findings together with a documented history of botulinum toxin exposure and was confirmed by neurological consultation in all patients. Demographic characteristics, exposure history, clinical findings, laboratory parameters, treatment approaches, and outcomes were analyzed.

Results: A total of 16 patients were included, with a median age of 34 years (IQR: 32–44), and 81.2% were female. The median time from symptom onset to hospital admission was 7 days (IQR: 5–8). The majority of cases (93.7%) were associated with iatrogenic botulinum toxin exposure, with intragastric (37.5%) and cosmetic facial or cervical (37.5%) injections being equally frequent sources. The most common presenting features were neurological symptoms without impaired consciousness, particularly cranial nerve involvement such as diplopia, dysphagia, dysarthria, and ptosis (81.3%). Laboratory findings were generally within normal limits. Management consisted of pyridostigmine in seven patients (43.8%), botulinum antitoxin in three (18.8%), and observation with supportive care alone in six (37.5%). All three patients treated with antitoxin had bulbar or respiratory symptoms, and in two of them, treatment had been initiated at the referring institution. No patient required mechanical ventilation, and no mortality was observed. Most patients were discharged with clinical recovery.

Conclusion: Iatrogenic botulism represents an emerging clinical entity in emergency practice. The presence of cranial nerve dysfunction with preserved consciousness should raise suspicion for botulism, particularly in patients with recent botulinum toxin exposure. Early recognition and timely management are essential to prevent disease progression and ensure favorable outcomes. The uniformly favorable outcomes observed here should be interpreted in the context of a cohort that presented late and had predominantly mild disease and do not exclude a clinically relevant risk of respiratory failure in iatrogenic botulism.

Keywords: Botulism, botulinum toxin, emergency department, iatrogenic botulism, neuromuscular junction disease

Introduction

Botulism is a rare but potentially life-threatening disease caused by botulinum neurotoxins produced by *Clostridium botulinum* and related species [1]. These toxins inhibit acetylcholine release at presynaptic nerve terminals, leading to flaccid paralysis characterized by blurred vision, dry mouth, dysphagia, diplopia, dysarthria, dyspnea, and progressive muscle weakness[1,2]. Early symptoms may include fatigue, weakness, and dizziness, followed by gastrointestinal manifestations such as vomiting, diarrhea, constipation, and abdominal distension [3]. Although mild alterations in mental status may occasionally be observed, loss of consciousness and fever are typically not expected [1]. Due to its rarity and the nonspecific nature of its clinical presentation,

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diagnosis is often delayed, which may increase mortality in untreated cases.

Botulism presents in five main clinical forms: foodborne, wound, iatrogenic, infant, and adult intestinal toxemia [4–7]. Among these, foodborne botulism is the most common and typically results from the ingestion of improperly stored or home-canned foods in which bacterial spores have proliferated and produced toxins [8,9]. Wound botulism develops following contamination of traumatic wounds with bacterial spores [10]. In recent years, the increasing use of botulinum toxin for both cosmetic and therapeutic purposes has been accompanied by a rise in reported cases of iatrogenic botulism [11]. These cases, often associated with high doses or nonstandard preparations, highlight the changing epidemiology of the disease [12].

Symptoms usually appear within 12–36 hours after exposure to botulinum toxin, although the incubation period may range from 4 hours to 8 days [2]. Clinically, newly developed cranial nerve involvement (diplopia, ptosis, dysphagia, dysarthria), accompanied by progressive, typically descending neuromuscular weakness, is characteristic of botulism [13]. In patients presenting to the emergency department, obtaining a history of potential toxin exposure, such as the consumption of home-canned foods, the presence of wounds, or recent botulinum toxin injections, is critical for diagnosis [2,3]. Early recognition of these clinical and anamnestic clues is essential for considering botulism in the differential diagnosis and preventing diagnostic delays. However, the fact that laboratory findings are often within normal limits at initial presentation further complicates the diagnostic process [12]. As a result, botulism may be misdiagnosed as other neurological emergencies, such as myasthenia gravis, Guillain–Barré syndrome, or stroke [14].

The indications for botulinum toxin use have expanded considerably in recent years [15]. It is widely used across a broad spectrum of conditions, ranging from neurological disorders and hyperhidrosis to migraine treatment and aesthetic procedures. Although generally considered safe, inappropriate dosing, the use of unlicensed preparations, or uncontrolled administration may result in systemic toxicity [16]. This has led to an increase in cases of iatrogenic botulism, particularly with the growing popularity of cosmetic and intragastric ("gastric Botox") procedures [15–18]. These trends suggest that botulism should be considered not only as a rare disease but also as an important clinical entity in the differential diagnosis of emergency department presentations.

The rise in iatrogenic botulism has become more evident with outbreaks associated with cosmetic botulinum toxin applications [17,18]. For example, a 2025 outbreak in the United Kingdom involving 25 cases linked to cosmetic botulinum toxin injections demonstrated that all patients presented to emergency departments and that a substantial proportion required antitoxin treatment [19]. The same study identified the use of unlicensed preparations and exposure to specific practitioners as major risk factors for disease development [19]. These findings indicate that iatrogenic botulism may occur not only as isolated complications but also as clusters and outbreaks with public health implications.

Despite this, data on the clinical presentation, follow-up, and management of botulism in the emergency setting remain limited. Most of the available evidence originates either from outbreak investigations and public health reports, which are designed for epidemiological case finding rather than for describing bedside decision-making, or from isolated case reports [11,15,19]. In particular, the emergency department course of iatrogenic botulism related to intragastric botulinum toxin injection performed for weight reduction, a procedure that has expanded rapidly in Türkiye and has already generated internationally reported clusters, has rarely been described in a consecutive clinical series [15,18]. The present study addresses this gap by presenting a consecutive, single-center, four-year emergency department series of botulism cases, with a particular focus on iatrogenic exposures. We describe the diagnostic pathways followed in these patients and the treatment approaches applied in practice, including botulinum antitoxin, pyridostigmine, and observation. In this study, we aimed to evaluate the clinical characteristics, diagnostic and treatment processes, and outcomes of patients with botulism presenting to the emergency department.

Materials and Methods

Study Design and Setting

This retrospective, observational, single-center study was conducted in accordance with the principles outlined in the Declaration of Helsinki. Ethical approval was obtained from the Institutional Review Board of Başakşehir Çam and Sakura City Hospital, Istanbul, Türkiye (Date: 29.04.2026 – Decision: 187/2026). The study cohort comprised all patients diagnosed with botulism who were admitted to the emergency department of this tertiary-care referral hospital between January 1, 2022, and January 1, 2026.

Adult patients aged 18 years and older who were diagnosed with botulism and managed in the emergency department during the study period were included. A total of 16 patients met the inclusion criteria and were included in the final analysis. Of these, 15 cases were classified as iatrogenic botulism, whereas one case was attributed to a non-iatrogenic cause. Patients were excluded if they were under 18 years of age, had incomplete or missing

clinical data, or did not have a confirmed diagnosis of botulism based on clinical findings and specialist evaluation.

Case Definition and Diagnostic Process

Because laboratory confirmation of botulinum neurotoxin is not routinely available in our setting, the diagnosis was established clinically, in line with current clinical guidelines [20]. A case was defined as the acute onset of compatible neurological findings, namely cranial nerve involvement (diplopia, ptosis, blurred vision, dysarthria, or dysphagia), with or without symmetrical descending muscle weakness, occurring in an afebrile patient without impairment of consciousness and in association with documented exposure to botulinum toxin (cosmetic, therapeutic, or intragastric administration) or, in the single non-iatrogenic case, ingestion of a suspected food source. All patients were first assessed by an emergency physician and subsequently evaluated by a neurologist during the index emergency department visit, and the final diagnosis was reached by consensus between the emergency physician and the consulting neurologist. Alternative causes of acute neuromuscular weakness were excluded by neurological examination, routine laboratory testing, and cranial imaging; further investigations, such as cranial magnetic resonance imaging or cerebrospinal fluid examination, were performed at the discretion of the consulting neurologist when the presentation was considered atypical. Neither electrodiagnostic testing nor laboratory toxin detection was routinely applied as a diagnostic criterion, and this is addressed in the Study Limitations section. Patients in whom the diagnosis remained unconfirmed after specialist evaluation were not included.

Management Strategy

No standardized institutional treatment protocol for botulism was in place during the study period. Management decisions were made jointly by the treating emergency physician and the consulting neurologist and, in the case of botulinum antitoxin, in coordination with the infectious diseases department and the public health authority responsible for antitoxin supply in Türkiye. Decisions were therefore based on clinical severity and the interval since symptom onset. In general, botulinum antitoxin was considered for patients with bulbar or respiratory involvement, such as dysphagia, dyspnea, or orthopnea, or with objective or progressive limb weakness, that is, features indicating a risk of further deterioration, provided that these patients presented sufficiently early in the disease course for antitoxin to be expected to limit further toxin binding. Pyridostigmine was administered empirically to selected patients with persistent but nonprogressive symptoms, predominantly those presenting late, when antitoxin was no longer considered likely to alter the disease course. Patients with mild, stable, and nonprogressive findings were managed with observation and supportive care alone. It should be noted that a proportion of our patients were referred from other institutions with an established suspicion of botulinum intoxication, and in these patients, the decision to administer antitoxin had already been made and treatment initiated at the referring center; in such cases, treatment was continued at our hospital. Irrespective of the treatment group, all patients underwent serial neurological examinations and monitoring of respiratory status, with immediate availability of intensive care in the event of deterioration.

Data Collection

Patient data were retrospectively obtained from the hospital information management system. Iatrogenic botulism was defined as the presence of compatible clinical findings following recent botulinum toxin administration for therapeutic or cosmetic purposes. The collected variables included demographic characteristics (age, sex, and comorbidities), etiological exposure (categorized as iatrogenic, foodborne, or other), presenting symptoms (neurological, gastrointestinal, respiratory, and cardiac), and relevant time intervals (time from symptom onset to emergency department admission and from admission to diagnosis). Additional data included initial diagnoses, including misdiagnoses, neurological examination findings, vital signs at presentation, laboratory parameters (hematological, biochemical, coagulation, and blood gas values), and imaging findings. Treatment-related variables, such as antitoxin administration and its timing, were also recorded, along with the need for intensive care unit admission, requirement for mechanical ventilation, length of hospital stay, and clinical outcomes, including discharge, treatment refusal, and mortality.

Statistical Analyses

Statistical analyses were performed using SPSS software (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as medians and interquartile ranges, whereas categorical variables were presented as frequencies and percentages. The normality of continuous variables was assessed using the Shapiro–Wilk test. Given the small sample size and the nonnormal distribution of several variables, nonparametric tests were used for comparative analyses. Continuous variables were compared using the Mann–Whitney U test, and categorical variables were analyzed using Fisher's exact test. Associations between continuous variables were examined using Spearman's rank correlation coefficient (rho). Because of the exploratory nature of these analyses and the limited sample size, no adjustment for multiple comparisons was performed, and the results of subgroup and correlation analyses

were regarded as hypothesis-generating. A p value of <0.05 was considered statistically significant.

Results

Demographic and Clinical Characteristics

A total of 16 patients diagnosed with botulism were included in the study (Table 1). The median age was 34 years (interquartile range [IQR]: 32–44; range: 24–67). Thirteen patients (81.2%) were female, and three (18.8%) were male. The median time from symptom onset to hospital admission was 7 days (IQR: 5–8; range: 0–9).

Table 1. Demographic, clinical, and laboratory characteristics of patients with botulism (n=16)

Variable	n=16
Demographics	
Age, years — median (IQR)	34 (32–44)
Age, years — min–max	24–67
Female, n (%)	13 (81.2)
Male, n (%)	3 (18.8)
Time from exposure to admission, days — median (IQR)*	7 (5–8)
Time from exposure to admission, days — min–max*	0–9
Etiology (source of exposure)	
Gastric botulinum toxin, n (%)	6 (37.5)
Cosmetic botulinum toxin (face/neck), n (%)	6 (37.5)
Axillary (hyperhidrosis) botulinum toxin, n (%)	2 (12.5)
Canned mushrooms, n (%)	1 (6.3)
Masseter botulinum toxin, n (%)	1 (6.3)
Mechanism of intoxication	
Accidental, n (%)	1 (6.3)
Iatrogenic, n (%)	15 (93.7)
Clinical symptoms	
Altered consciousness, n (%)	3 (18.8)
Gastrointestinal symptoms, n (%)	5 (31.3)
Cardiac symptoms, n (%)	1 (6.3)
Respiratory symptoms, n (%)	3 (18.8)
Neurological symptoms (excluding consciousness), n (%)	13 (81.3)
Comorbidities	
Diabetes mellitus, n (%)	1 (6.3)
Hypertension, n (%)	0 (0.0)
Asthma/COPD, n (%)	1 (6.3)

Table 1. Continue

Variable	n=16
Vital parameters at presentation	
GCS — median (IQR)	15 (15–15)
Systolic BP, mmHg — median (IQR)	122 (109–127)
Diastolic BP, mmHg — median (IQR)	67 (61–74)
MAP, mmHg — median (IQR)	80 (74–88)
Temperature, °C — median (IQR)	36.5 (36.1–36.5)
Heart rate, /min — median (IQR)	81 (73–90)
SpO_2, % — median (IQR)	98 (98–99)
Respiratory rate, /min — median (IQR)	16 (15–17)
Laboratory findings	
Leukocytes, /mm^3 — median (IQR)	6,540 (6,140–8,220)
Hemoglobin, g/dL — median (IQR)	12.7 (11.5–14.4)
Hematocrit, % — median (IQR)	38.5 (35.3–43.2)
MCV, fL — median (IQR)	85.1 (81.7–87.8)
Platelets, /mm^3 — median (IQR)	261,500 (217,750–304,750)
CRP, mg/dL — median (IQR)	0.8 (0.4–5.2)
Glucose, mg/dL — median (IQR)	92 (88–96)
Urea, mg/dL — median (IQR)	23.6 (18.4–27.9)
Creatinine, mg/dL — median (IQR)	0.71 (0.67–0.77)
Sodium, mEq/L — median (IQR)	140 (139–141)
Potassium, mEq/L — median (IQR)	4.2 (4.0–4.5)
Chloride, mEq/L — median (IQR)	105 (104–106)
Calcium, mg/dL — median (IQR)	9.0 (8.6–9.6)
ALT, U/L — median (IQR)	17 (12–21)
AST, U/L — median (IQR)	19 (15–32)
aPTT, s — median (IQR)	28.5 (25.8–30.8)
INR — median (IQR)	1.1 (1.0–1.1)
pH — median (IQR)	7.39 (7.37–7.42)
HCO_3 , mEq/L — median (IQR)	25.8 (24.3–27.8)
Lactate, mmol/L — median (IQR)	1.1 (0.9–1.2)
Emergency department / inpatient treatment	
Botulinum antitoxin, n (%)	3 (18.8)
Pyridostigmine, n (%)	7 (43.8)
Observation, n (%)	6 (37.5)
Outcomes	
Endotracheal intubation, n (%)	0 (0.0)
Length of hospital stay, days — median (IQR)	2.5 (2–3)
Length of hospital stay, days — min–max	1–5
APACHE-II — median (IQR)	5 (2–10)

Table 1. Continue

Variable	n=16
Outcomes	
Discharged, n (%)	14 (87.5)
Treatment refusal, n (%)	2 (12.5)
In-hospital mortality, n (%)	0 (0.0)

Data are presented as median (interquartile range, IQR), min–max, or n (%). GCS: Glasgow Coma Scale; BP: blood pressure; MAP: mean arterial pressure; SpO_2: peripheral oxygen saturation; MCV: mean corpuscular volume; CRP: C-reactive protein; ALT: alanine aminotransferase; AST: aspartate aminotransferase; aPTT: activated partial thromboplastin time; INR: international normalized ratio; HCO_3 : bicarbonate; COPD: chronic obstructive pulmonary disease; APACHE-II: Acute Physiology and Chronic Health Evaluation II. **Time from exposure to admission was available for 13 of 16 patients (missing in 3).*

Etiology

According to the source of exposure, the most common etiologies were gastric botulinum toxin injections (n=6; 37.5%) and cosmetic botulinum toxin applications involving the face and neck (n=6; 37.5%). These were followed by hyperhidrosis treatment (n=2; 12.5%), foodborne botulism related to canned mushrooms (n=1; 6.3%), and masseter botulinum toxin injection (n=1; 6.3%). Overall, 15 patients (93.7%) had iatrogenic exposure, whereas one patient (6.3%) had foodborne botulism (Table 1). No concurrent medication use, alcohol consumption, substance use, or pregnancy was identified in any patient.

Diagnostic Process

All 16 patients were evaluated by a neurologist during the index emergency department visit, and in every case, the diagnosis was established clinically based on compatible neurological findings and a documented exposure history. No patient had laboratory confirmation of botulinum neurotoxin. Cranial imaging and routine laboratory testing, performed to exclude alternative causes of acute neuromuscular weakness, did not reveal findings that accounted for the clinical picture in any patient. Electrodiagnostic testing was not used as a diagnostic criterion; when it was obtained at the discretion of the consulting neurologist, the findings were normal and did not alter the clinical diagnosis.

Clinical Presentation

The most common presenting features were neurological symptoms without impaired consciousness, including diplopia, dysphagia, dysarthria, ptosis, and motor weakness (n=13; 81.3%). Gastrointestinal symptoms (nausea, vomiting, and diarrhea) were observed in 5 patients (31.3%), altered consciousness in 3 patients (18.8%), respiratory symptoms in 3 patients (18.8%), and cardiac symptoms in 1 patient (6.3%).

Comorbidities

Most patients had no significant comorbidities. Diabetes mellitus and asthma/chronic obstructive pulmonary disease were each present in one patient (6.3%). Additionally, one patient had a history of cervical disc herniation, and another had osteoporosis.

Vital Signs

At presentation, the median Glasgow Coma Scale score was 15 (IQR: 15–15). The median systolic blood pressure was 122 mmHg (IQR: 109–127), diastolic blood pressure was 67 mmHg (IQR: 61–74), and mean arterial pressure was 80 mmHg (IQR: 74–88). The median body temperature was 36.5°C (IQR: 36.1–36.5), heart rate was 81 beats/min (IQR: 73–90), peripheral oxygen saturation was 98% (IQR: 98–99), and respiratory rate was 16 breaths/min (IQR: 15–17).

Laboratory Findings

Hematological analysis showed a median leukocyte count of 6,540/mm^3 (IQR: 6,140–8,220), hemoglobin level of 12.7 g/dL (IQR: 11.5–14.4), hematocrit of 38.5% (IQR: 35.3–43.2), mean corpuscular volume of 85.1 fL (IQR: 81.7–87.8), and platelet count of 261,500/mm^3 (IQR: 217,750–304,750).

Biochemical analysis revealed a median C-reactive protein level of 0.8 mg/dL (IQR: 0.4–5.2), glucose level of 92 mg/dL (IQR: 88–96), urea level of 23.6 mg/dL (IQR: 18.4–27.9), and creatinine level of 0.71 mg/dL (IQR: 0.67–0.77). Median electrolyte levels were as follows: sodium, 140 mEq/L (IQR: 139–141); potassium, 4.2 mEq/L (IQR: 4.0–4.5); chloride, 105 mEq/L (IQR: 104–106); and calcium, 9.0 mg/dL (IQR: 8.6–9.6). The median alanine aminotransferase level was 17 U/L (IQR: 12–21), and the median aspartate aminotransferase level was 19 U/L (IQR: 15–32).

Coagulation parameters showed a median activated partial thromboplastin time (aPTT) of 28.5 seconds (IQR: 25.8–30.8) and an international normalized ratio (INR) of 1.1 (IQR: 1.0–1.1). Blood gas analysis demonstrated a median pH of 7.39 (IQR: 7.37–7.42), bicarbonate level of 25.8 mEq/L (IQR: 24.3–27.8), and lactate level of 1.1 mmol/L (IQR: 0.9–1.2).

According to the Shapiro–Wilk test, several variables deviated from a normal distribution; therefore, nonparametric tests were used for comparative analyses.

Treatment and Outcomes

Regarding management, 7 patients (43.8%) received pyridostigmine, 3 patients (18.8%) received botulinum antitoxin, and 6 patients (37.5%) were managed with observation (Table 1). All three patients who received botulinum antitoxin had bulbar or

respiratory symptoms at presentation: dysphagia was reported by two patients, and dyspnea or orthopnea by two; one of them, who had been exposed to approximately three vials of a botulinum toxin type A preparation at multiple injection sites, additionally had nasal speech and objective proximal limb weakness (upper extremity muscle strength, 3/5). In two of these three patients, antitoxin had been initiated at the referring institution before transfer and was continued at our hospital, whereas in the third, it was initiated on the day of admission, within three days of symptom onset; in this patient, limb muscle strength improved to 4/5 within 24 hours. No patient required endotracheal intubation. The median length of hospital stay was 2.5 days (IQR: 2–3; range: 1–5). The median APACHE II score was 5 (IQR: 2–10). Fourteen patients (87.5%) were discharged, whereas two patients (12.5%) refused treatment. No mortality was observed during the study period.

Comparative Analyses

Treatment vs. Observation Groups

Patients were categorized into two groups: those receiving treatment (antitoxin or pyridostigmine; n=10) and those managed with observation (n=6) (Table 2).

There was no significant difference in age (median: 33.5 [IQR: 28.5–41.2] vs. 41.5 [IQR: 34.0–49.8]; p=0.102), APACHE II score (p=0.114), or length of hospital stay (p=0.382) between the groups.

Etiological distribution differed significantly between the groups (p=0.035), with cosmetic botulinum toxin exposure predominating in the treatment group (50.0%), whereas gastric botulinum toxin exposure was more common in the observation group (83.3%).

Patients in the treatment group had significantly lower systolic blood pressure (p=0.039), diastolic blood pressure (p=0.045), and mean arterial pressure (p=0.011) than those in the observation group. Blood gas analysis revealed a significantly higher pH in the treatment group (p=0.009). As treatment was allocated on clinical grounds rather than randomly, these differences are interpreted with caution in the Discussion.

Glucose and urea levels showed borderline differences between the groups, whereas no significant differences were observed in other hematological, biochemical, or coagulation parameters.

Neurological Symptom-Based Comparison

Patients were divided into those with neurological symptoms (n=13) and those without neurological symptoms (n=3) (Table 3). No significant differences were observed between the groups in terms of age, APACHE II score, or length of hospital stay.

Among laboratory parameters, only serum chloride levels were significantly lower in patients with neurological symptoms (p=0.044). No other significant differences were identified.

Correlation Analysis

Using Spearman's rank correlation analysis, platelet count was positively correlated with length of hospital stay (rho=0.508, p=0.045). Similarly, aPTT showed a significant positive correlation with length of hospital stay (rho=0.538, p=0.031) (Table 4).

A moderate positive correlation was observed between calcium levels and length of hospital stay (rho=0.449; p=0.081), whereas a moderate negative correlation was found between INR and length of hospital stay (rho=-0.456; p=0.076), both at a trend level. No significant correlations were observed for other variables.

Discussion

In this study, we evaluated the etiological characteristics, clinical findings at emergency department presentation, neurological involvement, and the follow-up and treatment processes of patients who developed botulism following exposure to botulinum toxin for various reasons. In this retrospective study, the vast majority of cases were found to be attributable to iatrogenic botulism. To our knowledge, this is one of the few consecutive emergency department series in which iatrogenic botulism, rather than foodborne disease, accounted for almost all cases and in which intragastric botulinum toxin injection performed for weight reduction was as frequent a source as cosmetic facial injection. Its contribution is therefore primarily practical: it describes how these patients present at the front door of the hospital, which alternative diagnoses are considered, how treatment is selected in the absence of a standardized protocol, and what the corresponding outcomes are.

Botulinum toxin injections have become widely used in clinical practice, with proven efficacy in cosmetic procedures, hyperhidrosis treatment, chronic migraine, urological conditions such as overactive bladder, obesity-related interventions, and the management of neurological disorders, including spasticity and dystonia [21–23]. Following administration, systemic dissemination of the toxin, particularly if it enters the circulation, may lead to peripheral neuromuscular blockade and result in a clinical picture consistent with botulism [23]. However, the expanding range of indications and challenges in individual dose adjustment have contributed to an increasing number of iatrogenic botulism cases worldwide [18,19,24]. In addition, exposure to unlicensed or nonstandard botulinum toxin preparations represents an important risk factor for the development of such cases [19].

Table 2. Comparison of patients managed with treatment versus observation

Variable	Treatment (n=10)	Observation (n=6)	Test statistic	p
Demographic and clinical				
Age, years	33.5 (28.5–41.2)	41.5 (34.0–49.8)	U=14.5	0.102
APACHE-II	8.5 (4.2–11.8)	2.0 (1.2–4.2)	U=45.0	0.114
Length of stay, days	3 (2–3)	2.0 (2.0–2.8)	U=38.0	0.382
Time from exposure, days†	7.0 (6.0–7.5)	7.0 (4.8–7.8)	U=22.0	0.941
Vital parameters				
Systolic BP, mmHg	119.0 (104.2–122.5)	128.0 (124.0–133.5)	U=10.5	0.039 *
Diastolic BP, mmHg	61.5 (59.2–69.5)	72.0 (69.2–77.0)	U=11.0	0.045 *
MAP, mmHg	74.5 (72.0–80.0)	89.0 (87.2–90.8)	U=6.0	0.011 *
Temperature, °C	36.5 (36.0–36.7)	36.2 (36.1–36.5)	U=34.5	0.660
Heart rate, /min	77.0 (66.2–86.5)	87.0 (78.2–91.2)	U=16.5	0.158
Respiratory rate, /min	16 (14–16)	17.0 (15.5–18.5)	U=16.0	0.136
Laboratory findings				
Leukocytes, /mm^3	6,990 (6,300–8,400)	6,375 (5,995–6,522)	U=36.5	0.515
Hemoglobin, g/dL	12.3 (11.7–13.7)	13.8 (11.4–15.0)	U=26.0	0.704
Hematocrit, %	38.0 (35.5–41.2)	42.0 (34.6–45.8)	U=24.0	0.551
MCV, fL	85.1 (82.0–87.1)	85.3 (81.8–88.2)	U=28.0	0.871
Platelets, /mm^3	276,500 (230,250–314,000)	236,000 (214,000–292,500)	U=39.0	0.356
Neutrophils, /mm^3	4,020 (3,292–4,667)	4,030 (3,530–6,480)	U=27.0	0.792
Lymphocytes, /mm^3	2,725 (1,947–3,027)	1,765 (1,530–1,955)	U=39.0	0.368
CRP, mg/dL	0.6 (0.1–2.8)	2.9 (0.7–5.8)	U=18.0	0.211
Glucose, mg/dL	89.5 (82.0–93.5)	97.5 (92.5–104.0)	U=11.5	0.050 ‡
Urea, mg/dL	19.7 (17.7–24.2)	26.7 (24.9–29.9)	U=14.0	0.093 ‡
Creatinine, mg/dL	0.71 (0.68–0.76)	0.73 (0.67–0.77)	U=30.0	1.000
Sodium, mEq/L	139.5 (138.2–140.8)	140.5 (139.2–141.8)	U=23.0	0.476
Potassium, mEq/L	4.1 (4.0–4.4)	4.3 (3.9–4.4)	U=28.0	0.875
Chloride, mEq/L	104 (104–105)	106.0 (103.5–107.8)	U=20.5	0.322
Calcium, mg/dL	9.1 (8.9–9.5)	8.8 (8.6–9.5)	U=36.5	0.515
Magnesium, mg/dL	2.02 (1.94–2.25)	2.02 (1.91–2.10)	U=33.0	0.786
Albumin, g/L	42 (41–45)	41 (38–45)	U=34.0	0.702
CK, U/L	91.0 (73.5–113.8)	75.5 (50.2–121.8)	U=37.0	0.492
ALT, U/L	16.0 (12.2–20.5)	17.0 (12.8–33.2)	U=26.5	0.744
AST, U/L	19.0 (15.8–27.8)	18.0 (13.8–33.5)	U=29.5	1.000
aPTT, s	28.5 (26.9–30.1)	27.4 (25.4–31.5)	U=30.5	1.000
INR	1.00 (1.00–1.10)	1.10 (1.02–1.18)	U=20.0	0.277
pH	7.420 (7.397–7.437)	7.364 (7.344–7.381)	U=54.5	0.009 **
HCO_3 , mEq/L	24.8 (24.2–26.4)	27.4 (26.4–27.8)	U=19.0	0.254
Lactate, mmol/L	1.00 (0.80–1.20)	1.20 (0.97–1.27)	U=19.0	0.249
Ionized Ca^2 , mmol/L	1.19 (1.18–1.22)	1.17 (1.07–1.23)	U=35.5	0.584
Base excess, mEq/L	0.0 (–0.3–1.2)	2.0 (1.2–2.6)	U=21.0	0.356

*Data are presented as median (interquartile range, IQR). Comparisons were performed using the Mann–Whitney U test. The treatment group received botulinum antitoxin and/or pyridostigmine; the observation group received supportive care only. * $p < 0.05$; ** $p < 0.01$; ‡ borderline ($0.05 \leq p < 0.10$). †Time from exposure to admission was available for 7 patients in the treatment group and 6 in the observation group (3 patients with missing data).*

Table 3. Comparison of patients with versus without neurological symptoms

Variable	Neurological (+) (n=13)	Neurological (–) (n=3)	Test statistic	p
Demographic and clinical				
Age, years	34.0 (29.0–44.5)	42.0 (34.0–67.0)	U=10.0	0.201
APACHE-II	5.0 (1.5–10.5)	5.0 (0.0–12.0)	U=20.5	0.893
Length of stay, days	2.0 (2.0–3.0)	3.0 (2.0–4.0)	U=13.5	0.420
Time from exposure, days†	7.0 (7.0–7.8)	5.0 (2.5–6.5)	U=20.5	0.383
Vital parameters				
Systolic BP, mmHg	123 (108–129)	118 (104–132)	U=22.5	0.687
Diastolic BP, mmHg	69 (61–76)	62 (56–70)	U=26.5	0.346
MAP, mmHg	80 (73–89)	80 (72–90)	U=19.5	1.000
Temperature, °C	36.2 (36.0–36.5)	36.5 (36.5–37.2)	U=8.0	0.122
Heart rate, /min	80 (70–89)	92 (70–92)	U=14.0	0.459
Respiratory rate, /min	16 (14–18)	16 (15–18)	U=17.5	0.788
Laboratory findings				
Leukocytes, /mm^3	6,570 (6,060–9,395)	6,280 (4,920–6,540)	U=29.5	0.179
Hemoglobin, g/dL	13.2 (11.1–14.9)	12.5 (12.2–12.8)	U=21.0	0.840
Hematocrit, %	40.5 (34.6–44.5)	37.9 (35.3–38.8)	U=24.5	0.501
MCV, fL	84.9 (80.8–86.8)	91.0 (82.0–91.9)	U=9.5	0.179
Platelets, /mm^3	237,000 (214,000–305,500)	288,000 (258,000–322,000)	U=13.0	0.382
Neutrophils, /mm^3	4,070 (3,580–5,555)	3,020 (2,950–42,500)	U=24.0	0.545
Lymphocytes, /mm^3	2,180 (1,470–3,345)	1,870 (1,620–2,850)	U=23.0	0.638
CRP, mg/dL	1.0 (0.6–10.2)	2.6 (0.2–4.9)	U=15.0	0.584
Glucose, mg/dL	91 (83–97)	96 (92–106)	U=9.5	0.179
Urea, mg/dL	20.8 (17.8–28.9)	24.4 (22.9–37.3)	U=13.0	0.382
Creatinine, mg/dL	0.73 (0.67–0.87)	0.71 (0.64–0.72)	U=25.5	0.420
Sodium, mEq/L	139 (138–142)	141 (140–142)	U=11.0	0.253
Potassium, mEq/L	4.20 (3.81–4.46)	4.14 (4.10–4.47)	U=17.0	0.737
Chloride, mEq/L	104 (103–105)	107 (106–108)	U=4.5	0.044 *
Calcium, mg/dL	9.00 (8.75–9.68)	8.64 (8.60–9.76)	U=23.5	0.590
Magnesium, mg/dL	2.00 (1.87–2.28)	2.10 (1.93–2.16)	U=17.0	0.737
Albumin, g/L	43 (39–48)	41 (37–46)	U=24.0	0.545
CK, U/L	81 (69–135)	95 (60–136)	U=19.0	0.946
ALT, U/L	15 (12–22)	19 (10–62)	U=17.5	0.788
AST, U/L	18 (14–26)	37 (14–38)	U=11.5	0.282
aPTT, s	28.3 (25.5–31.4)	28.8 (27.2–31.5)	U=15.0	0.545
INR	1.1 (1.0–1.2)	1.0 (1.0–1.0)	U=30.0	0.158
pH	7.390 (7.349–7.434)	7.420 (7.386–7.420)	U=16.5	0.687
HCO_3 , mEq/L	26.2 (23.9–27.9)	25.3 (24.4–27.8)	U=19.0	0.946
Lactate, mmol/L	1.1 (0.9–1.4)	1.2 (0.8–1.2)	U=20.5	0.893
Ionized Ca^2 , mmol/L	1.18 (1.10–1.23)	1.21 (1.15–1.24)	U=16.0	0.638
Base excess, mEq/L	0.9 (–0.8–3.7)	2.0 (1.3–2.8)	U=8.0	0.466

*Data are presented as median (interquartile range, IQR). Comparisons were performed using the Mann–Whitney U test. Neurological symptoms refer to cranial nerve or motor involvement excluding altered consciousness. * p<0.05. †Time from exposure to admission was available for 10 patients in the Neurological(+) group and 3 in the Neurological(–) group (3 patients with missing data).*

Table 4. Correlations between clinical/laboratory variables and length of hospital stay.

Variable	n	r_s	p	Interpretation
Demographic				
Age	16	0.000	1.000	No correlation
Time from exposure, days	8	0.140	0.741	Weak positive
Vital parameters				
Systolic BP	16	–0.196	0.466	Weak negative
Diastolic BP	16	0.172	0.523	Weak positive
MAP	16	0.034	0.900	No correlation
Temperature	16	–0.101	0.710	Weak negative
Heart rate	16	0.002	0.995	No correlation
Laboratory findings				
Leukocytes	16	0.365	0.164	Moderate positive
Hemoglobin	16	–0.058	0.832	No correlation
Hematocrit	16	–0.121	0.655	Weak negative
MCV	16	0.238	0.375	Weak positive
Platelets	16	0.508	0.045	Strong positive *
Neutrophils	16	0.218	0.417	Weak positive
Lymphocytes	16	0.063	0.816	No correlation
CRP	14	0.389	0.170	Moderate positive
Glucose	16	0.082	0.762	No correlation
Urea	16	–0.338	0.200	Moderate negative
Creatinine	16	–0.313	0.237	Moderate negative
Sodium	16	0.263	0.326	Weak positive
Potassium	16	0.368	0.161	Moderate positive
Chloride	16	–0.072	0.791	No correlation
Calcium	16	0.449	0.081	Moderate positive ‡
Magnesium	16	0.149	0.581	Weak positive
Albumin	16	0.212	0.432	Weak positive
CK	16	0.137	0.612	Weak positive
ALT	16	0.344	0.192	Moderate positive
AST	16	0.257	0.336	Weak positive
aPTT	16	0.538	0.031	Strong positive *
INR	16	–0.456	0.076	Moderate negative ‡
pH	16	0.110	0.684	Weak positive
HCO_3^-	16	–0.055	0.839	No correlation
Lactate	16	–0.090	0.740	No correlation
Ionized Ca^2	16	–0.076	0.780	No correlation
Base excess	14	0.186	0.525	Weak positive

*r_s, Spearman rank correlation coefficient. Dependent variable: length of hospital stay (days). * $p<0.05$; ‡ borderline ($0.05 \leq p<0.10$). Oxygen saturation (SpO_2) was excluded from the analysis due to insufficient data (n=7) and one suspected data entry error.*

The etiological distribution observed in our cohort differs substantially from that reported in classical botulism series. In large reviews of foodborne and wound botulism, improperly preserved home-canned foods and injection drug use account for the overwhelming majority of cases, and affected patients are, on average, older and have a more balanced sex distribution [5,6,8–10]. In our cohort, by contrast, 15 of 16 patients (93.7%) had iatrogenic exposure, 81.2% were women, and the median age was 34 years, a profile that mirrors the demographic characteristics of the population undergoing aesthetic and weight-reduction procedures. Comparable patterns have been reported recently: the multinational outbreak that followed intragastric botulinum neurotoxin injections performed in Türkiye in 2023 predominantly affected young women [15]; in the 2025 outbreak in England linked to cosmetic injections of unlicensed products, all 25 identified cases presented to emergency departments [19]; and Eser et al. [18] described iatrogenic botulism after gastric and axillary administration in a Turkish cohort with similar characteristics. The equal contribution of intragastric (37.5%) and cosmetic facial or cervical (37.5%) injections in the present series suggests that, in settings where intragastric botulinum toxin is widely offered, this indication has become as important a source of iatrogenic botulism as aesthetic facial use, a shift that has not yet been reflected in most emergency medicine literature.

Previous studies have consistently reported that botulism typically presents with cranial nerve involvement and preserved consciousness, with symptoms such as diplopia, ptosis, dysarthria, and dysphagia being the most common initial findings [1,3]. In line with the existing literature, the majority of patients in our cohort presented with prominent neurological symptoms without impaired consciousness, predominantly involving cranial nerve dysfunction. Gastrointestinal symptoms were observed in a subset of patients, whereas altered consciousness and respiratory symptoms were relatively uncommon, supporting the classical clinical spectrum of botulism [3]. The absence of fever and the relative preservation of mental status in most patients may help differentiate botulism from other acute neurological conditions, particularly in emergency settings, where misdiagnosis can delay appropriate management. The frequency of cranial nerve involvement in our series (81.3%) is comparable to that reported in systematic reviews of foodborne and wound botulism, in which such findings are present in the great majority of patients [6]. The interval between symptom onset and presentation, however, differed markedly: the median of 7 days observed here is substantially longer than the 12–36-hour incubation period usually cited [2] and longer than that observed in outbreak settings, where active public health case finding accelerates presentation [15,19]. A plausible explanation is that patients with iatrogenic exposure initially attribute their symptoms to the procedure itself and seek care only when the symptoms fail to resolve.

In addition to the characteristic neurological presentation, laboratory findings in botulism are generally expected to remain within normal limits, reflecting the primarily neurotoxic rather than inflammatory or myopathic nature of the disease [25]. In our cohort, most laboratory parameters were within normal ranges, supporting this observation. Although gastrointestinal symptoms such as nausea, vomiting, or diarrhea may occur and can occasionally be associated with mild metabolic disturbances, marked biochemical abnormalities are typically not expected [2,3].

This pattern is particularly helpful in differentiating botulism from other neuromuscular and neurological conditions. Unlike disorders such as hypokalemic periodic paralysis, which is characterized by episodic weakness associated with low serum potassium levels, or inflammatory myopathies, botulism is not associated with significant laboratory abnormalities or elevated creatine kinase levels [25,26]. Furthermore, whereas Guillain–Barré syndrome typically presents with ascending weakness, botulism is characterized by a descending pattern of paralysis [27]. In contrast to variants such as Miller Fisher syndrome, in which cranial nerve involvement is accompanied by sensory deficits, deep sensation is generally preserved in botulism [28]. These distinctions are particularly important in emergency settings, where episodic or acute neuromuscular weakness may initially suggest alternative diagnoses and contribute to delayed recognition of botulism.

The diagnostic process in this cohort illustrates these difficulties in practice. Because the initial complaints, such as blurred vision, diplopia, ptosis, dysphagia, generalized weakness, and, in some patients, nausea and vomiting, are nonspecific, and because routine laboratory tests and cranial imaging are typically unremarkable, botulism tends to be considered only after alternative diagnoses have been entertained. Conditions commonly considered first in such patients include myasthenia gravis, Guillain–Barré syndrome and its Miller Fisher variant, brainstem ischemia, ophthalmological disease, electrolyte disturbances, and functional neurological disorders [14,25,26]. Electrodiagnostic testing is of limited help in this situation: in our cohort, the studies that were performed were normal and did not modify the clinical diagnosis, which is consistent with the recognized insensitivity of routine nerve conduction studies and needle electromyography in mild botulism, particularly when high-frequency repetitive nerve stimulation is not performed [20,25]. In our setting, the decisive diagnostic step was therefore not a laboratory or electrophysiological test but the exposure history combined with specialist neurological assessment. This has a direct

practical implication: in a patient with acute, afebrile, symmetrical cranial nerve dysfunction and preserved consciousness, explicit questioning about botulinum toxin administration is essential, including intragastric procedures, which patients may not spontaneously report as medical interventions, and cosmetic injections performed outside licensed medical settings.

In our cohort, different management strategies were observed, including supportive care alone, pyridostigmine administration, and botulinum antitoxin treatment. Notably, patients who received antitoxin presented earlier in the disease course and exhibited symptoms suggestive of potential clinical progression, such as dysphagia and dyspnea. In contrast, patients managed with supportive care or pyridostigmine typically presented later, often several days after symptom onset, and had relatively milder clinical manifestations. Two features of the antitoxin group deserve comment. First, the indication in practice was symptom-based rather than dose-based: bulbar or respiratory symptoms and, in the most severely affected patient, objective proximal weakness, rather than the reported number of units injected, determined the decision. Second, in two of the three patients, the decision had already been made at the referring institution, so our role was to continue treatment and monitor for deterioration. This is a common situation for tertiary referral centers and means that treatment allocation in such cohorts is only partly under the control of the receiving hospital, a point that also limits any comparison between treated and untreated patients.

These findings likely reflect real-world clinical decision-making rather than treatment efficacy, as antitoxin administration is generally prioritized in patients at risk of progression, particularly when respiratory or bulbar involvement is suspected [20]. Early administration of botulinum antitoxin, ideally within the first 24–48 hours after symptom onset, is recommended to limit disease progression and improve clinical outcomes [20]. Conversely, delayed presentation and milder symptoms may lead clinicians to adopt a more conservative approach. Although pyridostigmine has been used as a symptomatic treatment in selected cases, its role in botulism remains unclear and controversial and is not supported by current clinical guidelines, given the presynaptic mechanism of the disease and the limited and inconsistent clinical evidence [20,29].

The comparisons between the treatment and observation groups must therefore be interpreted with considerable caution. Patients were not allocated at random; allocation was determined by clinical severity, symptom progression, and the interval since exposure. The differences observed between the two groups, namely lower systolic, diastolic, and mean arterial pressures and higher pH in the treated group, are more plausibly markers of the clinical profile that prompted treatment or chance findings arising from multiple comparisons in a small sample than consequences of treatment itself. The same reasoning applies to the significant difference in etiological distribution between the groups, which reflects the observation that patients with intragastric exposure presented later and with milder findings. Consequently, no inference regarding the comparative effectiveness of antitoxin, pyridostigmine, or observation can be drawn from these data, and the present study should not be used to support or discourage any of these strategies.

Early recognition of botulism and timely initiation of appropriate management are critical determinants of clinical outcomes. Previous studies have demonstrated that early administration of botulinum antitoxin is associated with reduced mortality and improved clinical outcomes [20]. In addition, advances in supportive care, particularly respiratory management, have significantly decreased mortality rates in recent decades [1,3,20]. In our cohort, no mortality was observed, and all patients who received appropriate management were discharged with clinical recovery.

The absence of both endotracheal intubation and mortality is among the most notable findings of this study, and several explanations should be considered before this is interpreted as evidence of effective management. First, and probably most importantly, the cohort was shaped by its own natural history: the median interval from symptom onset to admission was 7 days, and patients who were still ambulatory and self-presenting a week after the onset of neuroparalytic symptoms necessarily represented the milder end of the disease spectrum, since respiratory compromise in botulism typically develops within the first days after exposure [3,20]. Second, the toxin burden after localized intramuscular or intragastric injection is generally lower than that associated with foodborne disease, in which large amounts of preformed toxin are ingested; accordingly, foodborne series report frequent mechanical ventilation, whereas most published iatrogenic cases recover without ventilatory support [6,11,17]. Third, our patients were young (median age, 34 years), predominantly female, and largely free of cardiopulmonary comorbidities, with a median APACHE II score of 5, indicating limited physiological derangement at presentation. Fourth, awareness of iatrogenic botulism has increased considerably in Türkiye since the widely reported 2023 intragastric botulinum toxin cluster [15,18], which is likely to have shortened the time from admission to recognition and ensured that patients were monitored for respiratory deterioration from the outset. Fifth, the three patients with bulbar or respiratory involvement, that is, the group genuinely at risk of progression, all received antitoxin, in two cases already at the referring institution,

and all patients were observed at a tertiary center with immediate access to intensive care.

The statistical interpretation of a zero-event rate also deserves emphasis. With 16 patients and no observed intubations or deaths, the upper limit of the 95% confidence interval for these outcomes is approximately 19% [30]. The present data are therefore fully compatible with a clinically important risk of respiratory failure in iatrogenic botulism and should not be interpreted as evidence that the condition is benign. Similarly, the correlations between length of hospital stay and platelet count or aPTT should be regarded as hypothesis-generating only: all of these values were within the reference range, no biologically plausible mechanism links them to the duration of a neuroparalytic illness, and with 16 patients and multiple correlations tested without adjustment, findings at the $p<0.05$ level are expected to occur by chance. The same caution applies to the isolated difference in serum chloride between patients with and without neurological symptoms.

Study Limitations

This study has several limitations. First, the sample size was small and derived from a single tertiary center, which limits both statistical power and external validity; the subgroup and correlation analyses, in particular, should be regarded as exploratory. Second, the diagnosis was established on clinical grounds and by specialist consensus, without laboratory confirmation of botulinum neurotoxin or routine electrodiagnostic testing, so misclassification of mimicking conditions cannot be excluded with certainty. Third, the retrospective design carries the risk of information bias and incomplete documentation, particularly regarding the exact dose, preparation, and licensing status of the injected botulinum toxin, which were rarely recorded. Fourth, treatment was not randomized, and the treatment and observation groups differed in clinical severity and timing of presentation, precluding any conclusion regarding comparative effectiveness; moreover, in some patients, antitoxin had already been started at the referring institution, so the indication and timing of the first dose were not determined by a uniform protocol. Fifth, patients were not followed systematically after discharge, so the time to complete symptom resolution and possible long-term sequelae are unknown. Finally, patients with milder disease who did not seek emergency care and those who presented to other institutions were not captured, which may have biased the cohort toward a particular severity profile.

Conclusion

In conclusion, botulism should be considered in patients presenting with acute cranial nerve dysfunction and preserved consciousness, particularly in the context of recent botulinum toxin exposure. The predominance of iatrogenic cases and the equal contribution of intragastric and cosmetic injections highlight the growing clinical relevance of this condition in modern emergency practice. Early recognition and timely administration of antitoxin are critical to preventing disease progression and ensuring favorable outcomes. Increased awareness among clinicians, especially in emergency settings, may help reduce diagnostic delays and improve patient management. The favorable outcomes observed in this cohort, however, were achieved in patients who presented late and had predominantly mild disease, and they should not be generalized to patients presenting early after high-dose exposure, in whom close respiratory monitoring remains essential.

Ethics

Ethics Committee Approval: This study was approved by The Local Ethics Committee of Çam and Sakura City Hospital (Date: 29.04.2026 – Decision: 187/2026).

Informed Consent: Written informed consents were obtained from patients who participated in this study.

Conflict of Interest: The authors declare that there is no conflict of interest.

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