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CASE SERIES

Familial Cluster of Foodborne Botulism Associated with Homemade Dairy Products: A Case Series

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Background: Foodborne botulism is a life-threatening illness caused by neurotoxins produced by Clostridium botulinum. Although dairy-related cases are rare, they remain a public health concern, especially in regions where traditional food preparation methods are common.

Case Presentation: This case series describes botulism in three family members—including a pregnant woman—following the consumption of homemade dairy products. All presented with varying degrees of neurological and respiratory symptoms. Two required intubation and intensive care. Laboratory findings confirmed neurotoxin type A in stool and gastric samples. Early administration of a horse-derived trivalent botulinum antitoxin and supportive care resulted in recovery.

Conclusion: Prompt recognition and treatment are essential for favorable outcomes. This case series underscores the exceptionally rare occurrence of foodborne botulism linked to homemade dairy products, highlighting the clinical and public health significance of early diagnosis and preventive measures.

Keywords: botulism, botulinum antitoxin, dairy products, pregnancy, food safety, diagnosis

Introduction

Foodborne botulism is a serious form of food poisoning caused by ingestion of food contaminated with a neurotoxin produced by Clostridium botulinum.[1] Ingesting as little as 30 nanograms of this toxin can result in severe neuroparalysis.[2] The toxin exerts its effects by blocking the release of acetylcholine at the neuromuscular junction, leading to flaccid paralysis.[3] Among the seven identified types of botulinum neurotoxins, types A, B, and E are most commonly implicated in foodborne cases. Toxin production occurs under specific environmental conditions—particularly when food is stored in anaerobic settings with a pH above 4.6, low salt and sugar content, and temperatures ranging from 4°C to 45°C.[4] When multiple individuals consume the same contaminated food, simultaneous symptom onset can occur within families or communities.

Symptoms typically manifest between 12 and 36 hours after ingestion.[5] Diagnosis is based on clinical evaluation supported by laboratory testing. Respiratory failure is the leading cause of mortality in botulism cases.[6]

In Iran, epidemiological data from 2020 indicated annual incidence rates of 7.1 cases per 100,000 males and 3.3 cases per 100,000 females among the native population. Contributing factors to this incidence include limited healthcare infrastructure in some regions, as well as social and environmental determinants that influence exposure risk through direct and indirect pathways.[7] The predominant sources of infection were traditionally home-processed fish, commercially canned products, and unpasteurized dairy items.[8] This report presents a familial cluster of foodborne botulism associated with the consumption of homemade dairy products—specifically, curd and yogurt. Three members of the same household consumed these products and were subsequently admitted to a public health center with symptoms consistent with botulism. The novelty of this case series lies in the identification of a familial cluster of botulism linked to homemade dairy products, an uncommon vehicle of infection that remains underrecognized in endemic regions. The inclusion of a pregnant patient provides rare clinical insight into disease progression, antitoxin safety, and favorable fetal outcomes. By comparing the clinical course of three related individuals, this report illustrates how the timing of antitoxin administration critically influences severity and prognosis.

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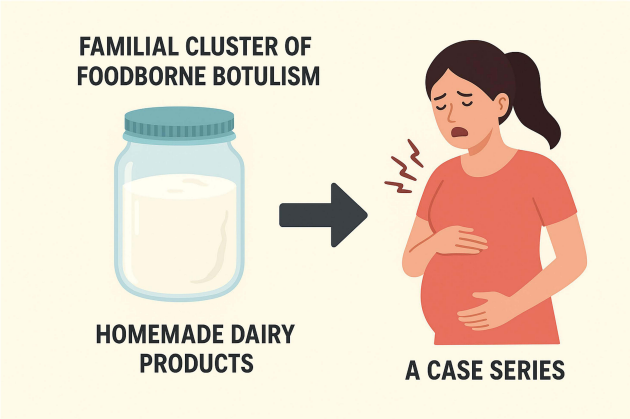
Graphical Abstract



Furthermore, the study not only highlights diagnostic and therapeutic challenges in resource-limited settings but also proposes specific public health and clinical recommendations to guide prevention and management.

Case Presentation

Their medical history visited the emergency room of Imam Reza Hospital in Mashhad, was as follows:

Patient 1

A 60-year-old woman with a known history of hypertension managed with oral medication was brought to the emergency department of Imam Reza Hospital, Mashhad, by emergency medical services (EMS) due to loss of consciousness and respiratory failure. She required immediate intubation for airway protection and mechanical ventilation.

On admission, her vital signs were as follows:

Temperature: 37.1°C (normal range: 36.1–37.2°C)

Heart rate: 62 beats per minute (normal: 60–100 bpm)

Blood pressure: 125/80 mmHg (normal systolic: 90–120 mmHg; diastolic: 60–80 mmHg)

Oxygen saturation (SpO_2): 71% (normal: 95–100%)

According to her son, she had experienced profound fatigue, dyspnea, nausea, and vomiting over the preceding 24 hours.

A horse-derived trivalent botulinum antitoxin was administered on hospital day 2.

Stool and nasogastric aspirate tested weakly positive for botulinum neurotoxin type A. (Explain: Diagnostic testing for botulinum neurotoxin was conducted at the National Reference Laboratory for Foodborne Diseases, Pasteur Institute of Iran. Stool and nasogastric aspirate samples were analyzed using the standard mouse bioassay (MBA), with confirmation by an enzyme-linked immunosorbent assay (ELISA) specific for type A neurotoxin. A test was considered positive if lethality occurred in mice within 48 hours and was neutralized by type A-specific antitoxin, or if ELISA optical density values exceeded the assay threshold (>0.1 OD above the negative control). The results were reported as "weakly positive", reflecting low toxin concentrations consistent with partial clearance at the time of specimen collection. All serum samples were negative, likely due to delayed sampling (>24 hours after symptom onset) and prior administration of antitoxin, which can neutralize circulating toxin and hinder its detection) (Table 1).

The serum sample tested negative for the toxin.

Routine laboratory investigations were unremarkable, and toxicology/urinalysis were negative. Arterial blood gas analysis showed marked hypercapnia and acidosis. Key laboratory and ABG findings are summarized in Table 2.

Chest X-ray: No acute pathology.

Following intubation, the patient was transferred to the intensive care unit (ICU) for ongoing ventilatory support and close monitoring.

Table 1 Clinical Characteristics and Management of the Three Patients with Foodborne Botulism

Parameter	Patient 1 (Mother, 60 y)	Patient 2 (Daughter, 32 y, Pregnant)	Patient 3 (Son, 28 y)
Time from ingestion to onset	~24 h	~24 h	~4 days
First symptoms	Fatigue, dyspnea, nausea, vomiting	Fatigue, dyspnea, nausea, vomiting	Ptosis, dysphagia, dysarthria, dysphonia
Neurological manifestations	Loss of consciousness, respiratory paralysis	Bilateral mydriasis, respiratory distress	Cranial nerve palsy (ptosis, dysarthria)
Respiratory support	Intubation and mechanical ventilation	Intubation and mechanical ventilation	Not required
Time of antitoxin administration	Day 2 of hospitalization	Day 2 of hospitalization	Immediately upon admission
Antitoxin type/dose	Horse-derived trivalent (10 mL IV, single dose)	Horse-derived trivalent (10 mL IV, single dose, no dose adjustment)	Horse-derived trivalent (10 mL IV, single dose)
Length of hospital stay	7 days (ICU)	7 days (ICU)	3 days (ward)
Prognosis	Full recovery	Full recovery, fetus unaffected	Full recovery

Table 2 Key Laboratory and Arterial Blood Gas (ABG) Findings in Three Patients with Foodborne Botulism

Parameter (Reference Range)	Patient 1 (Mother, 60 y)	Patient 2 (Daughter, 32 y, Pregnant)	Patient 3 (Son, 28 y)
Hemoglobin (11.7–13.8 g/dL F; 13.0–17.0 g/dL M)	13.5 (normal)	11.8 (mildly low, consistent with pregnancy)	13.2 (normal)
WBC (4.5–11 $\times 10^9$/L)	6.2 (normal)	8.5 (normal)	6.8 (normal)
Platelets (150–450 $\times 10^9$/L)	250 (normal)	220 (normal)	230 (normal)
Renal function (BUN 6–21 mg/dL; Cr 0.6–1.1 mg/dL)	BUN 18; Cr 0.9 (normal)	BUN 14; Cr 0.7 (normal)	BUN 12; Cr 0.9 (normal)
Electrolytes (Na 135–145 mmol/L; K 3.6–5.2 mmol/L; Cl 96–106 mmol/L)	Na 140, K 4.2, Cl 102 (normal)	Na 138, K 4.5, Cl 100 (normal)	Na 139, K 4.4, Cl 101 (normal)
Liver enzymes (AST 19–25 U/L; ALT 19–25 U/L)	AST 22, ALT 18 (slightly low ALT, not clinically relevant)	AST 24, ALT 22 (normal)	AST 18, ALT 19 (slightly low AST, not clinically relevant)
ABG (pH 7.35–7.45; $PaCO_2$ 35–45 mmHg; HCO_3^- 22–28 mEq/L)	pH 7.20, $PaCO_2$ 65 (marked hypercapnia, acidosis)	pH 7.19, $PaCO_2$ 68 (marked hypercapnia, acidosis)	pH 7.32, $PaCO_2$ 52 (mild hypercapnia)

Notes: All toxicology screens and urinalysis were negative. Serum botulinum toxin was undetectable in all cases, while stool and gastric aspirates were weakly positive for type A neurotoxin.

Patient 2

A 32-year-old pregnant woman, the daughter of Patient 1, presented to the emergency department with progressive fatigue, blurred vision, and dyspnea. She was transported via ambulance. Clinical and ultrasound assessments confirmed normal fetal development, with an estimated gestational age of 20 weeks at the time of admission. A gynecological

consultation was performed upon admission. Daily non-stress tests (NST) and serial ultrasound examinations were carried out during hospitalization, all of which demonstrated normal fetal heart activity, growth, and movement. Fetal well-being was preserved throughout hospitalization. At discharge, a repeat gynecological evaluation confirmed continued normal pregnancy progression. Follow-up after hospitalization indicated an uneventful pregnancy course, with no maternal or neonatal complications. Shortly after presentation, her condition worsened, necessitating endotracheal intubation due to increasing respiratory distress. A trivalent botulism antitoxin was administered on hospital day 2. The delay in antitoxin administration was due to diagnostic uncertainty at admission. The initial presentation of fatigue, nausea, vomiting, blurred vision, and respiratory distress led to consideration of alternative diagnoses, including acute intoxication, myasthenia gravis, and Guillain-Barré syndrome. Only after recognition of a familial cluster and progression of neurological manifestations was botulism strongly suspected, at which point antitoxin was initiated on the second hospital day (Table 1).

Vital signs at admission were relatively stable: Temperature: 36.8°C; Heart rate: 88 beats per minute; Blood pressure: 110/70 mmHg; Oxygen saturation: 94% (on mechanical ventilation) Physical examination revealed bilateral mydriasis (dilated pupils) without additional significant findings.

Laboratory results were within normal limits except for mild physiological anemia of pregnancy. ABG revealed severe hypercapnia and acidosis. Full laboratory and ABG findings are presented in Table 2.

Stool and nasogastric aspirate samples were weakly positive for botulinum neurotoxin type A, while the serum sample tested negative. Due to the severity of her clinical presentation, the patient was admitted to the intensive care unit (ICU) for close monitoring and supportive management, including mechanical ventilation.

Patient 3

Four days after the hospitalization of his mother (Patient 1) and sister (Patient 2), a 28-year-old man presented to the emergency department with ptosis, dysphagia, dysarthria, and dysphonia. He was alert and oriented on arrival. Based on the clinical picture and the family history, a single dose of trivalent botulism antitoxin was administered promptly.

Physical examination findings were otherwise unremarkable. Stool and nasogastric (NG) aspirate samples tested weakly positive for botulinum neurotoxin type A, while the serum sample was negative (Table 1).

Routine laboratory tests were normal. ABG revealed mild hypercapnia without marked acidosis. A summary of laboratory and ABG findings is shown in Table 2.

Routine investigations, including urine drug screen, urinalysis, chest X-ray, and brain CT scan, were unremarkable. ABG revealed mild hypercapnia without significant acidosis (Table 2). Given the incubation period for foodborne botulism and the shared dietary exposure to homemade dairy products, clinical suspicion of botulism was high.

Differential Diagnosis

The diagnostic process considered other causes of acute flaccid paralysis, including: Guillain-Barré syndrome – ruled out due to the absence of sensory symptoms and the pattern of weakness. Myasthenia gravis – deemed unlikely due to the sudden onset and positive response to antitoxin therapy. Toxin-mediated paralysis (eg, tick paralysis) – excluded by clinical evaluation and absence of physical findings.

Due to the delayed results of confirmatory laboratory tests (toxin assays), the diagnosis was made clinically, based on: Symptomatology; Known exposure to a contaminated food source; Positive response to botulinum antitoxin therapy; Presence of similarly affected family members.

Clinical Outcome

Patient 3 showed notable improvement within three days of hospitalization. His mother and sister also recovered after seven days of intensive care. All three patients were discharged in stable condition, with full resolution of neurological symptoms.

Discussion

This case series identified foodborne botulism caused by neurotoxin type A, following the consumption of homemade dairy products, specifically curd and yogurt. The affected individuals presented with a range of nonspecific symptoms, including nausea, fatigue, and respiratory distress, which progressed rapidly in some cases.

The mother and her pregnant daughter developed severe respiratory compromise requiring intubation and intensive care unit (ICU) admission, while the son, who received botulinum antitoxin early, exhibited milder symptoms. These observations are consistent with findings by Anderson et al, who emphasized the importance of early antitoxin administration in improving outcomes and reducing costs in botulism management.[9]

The nonspecific nature of early symptoms—such as gastrointestinal discomfort—can delay diagnosis and treatment, underscoring the need for a high index of clinical suspicion in endemic or high-risk settings.[2] The rapid progression from initial symptoms to cranial nerve involvement and respiratory failure, particularly in the mother and pregnant daughter, reflects trends reported by Chatham-Stephens et al, who noted a high incidence of cranial nerve dysfunction in foodborne botulism cases.[10]

In our cluster, the delay in antitoxin administration for Patients 1 and 2 was primarily due to diagnostic uncertainty, as their nonspecific early symptoms overlapped with more common conditions such as intoxication, stroke, myasthenia gravis, or Guillain-Barré syndrome. Additional patient-specific factors may also have contributed to disease severity. The mother's history of hypertension could have reduced her physiological reserve during acute neuromuscular respiratory failure, while the pregnant daughter's rapid deterioration was likely influenced by pregnancy-related physiological changes, including increased oxygen demand, reduced functional residual capacity, and altered immune responses. In contrast, Patient 3 received immediate treatment because the diagnosis had already been established within the family. These observations underscore the need for heightened clinical awareness, particularly in high-risk groups such as pregnant women and patients with comorbidities, to enable timely recognition and antitoxin administration, which are strongly associated with improved outcomes.

The management of Patient 2 also raises important considerations regarding antitoxin use during pregnancy. Existing evidence indicates that botulinum antitoxin, including horse-derived formulations, is considered safe for use in pregnant women, as it does not cross the placenta in significant amounts and no teratogenic effects have been reported. Conversely, while maternal botulinum neurotoxin exposure causes severe neuromuscular paralysis, the toxin itself does not cross the placenta, and adverse fetal outcomes are usually secondary to maternal hypoxia or systemic complications. Published reports, including those by Tacket et al.[11] Ghantarchyan et al[12] and CDC guidelines,[1] support the use of antitoxin in pregnancy, emphasizing that maternal benefit outweighs any theoretical risk. In our case, timely administration of the full standard dose was associated with maternal recovery and no adverse fetal effects.

In this report, supportive care, including mechanical ventilation, played a critical role in patient survival. The pregnant woman required intensive respiratory support, yet no adverse fetal outcomes were observed. This aligns with findings from Yang et al, who demonstrated that ICU-based supportive care is essential for minimizing complications and improving survival in severe botulism cases.[3]

The third patient's presentation—limited to ptosis and mild dysarthria—highlights the value of early intervention. Prompt administration of antitoxin likely prevented further progression of the illness. Similar findings were reported by O'Horo et al, who stressed the importance of early recognition and treatment in preventing life-threatening complications during botulism outbreaks.[13]

Additionally, challenges in diagnostic capacity and timely treatment access, particularly in resource-limited regions, were noted in our setting. In our patients, toxin was detected in stool and gastric aspirates but not in serum samples. This finding is consistent with previous reports, which note that serum toxin is often undetectable if blood samples are obtained more than 24 hours after ingestion or following the administration of antitoxin. Circulating neurotoxin is rapidly bound to presynaptic terminals, and neutralization by antitoxin further reduces measurable levels. In contrast, stool and gastric aspirates may remain positive for longer periods, even when toxin concentrations are low, as observed in this cluster. These diagnostic dynamics highlight the importance of collecting multiple specimen types and interpreting negative serum assays with caution in clinically suspected botulism, as also emphasized in CDC guidelines.[1] These issues are consistent with findings by Okunromade et al, who highlighted systemic limitations affecting botulism diagnosis and response, particularly in underserved communities.[14]

The involvement of homemade yogurt and curd as vehicles for toxin exposure in this cluster highlights the risks associated with traditional dairy preparation and storage practices. Although rare, dairy products can support C. botulinum growth when fermentation fails to adequately acidify the product (final pH above 4.6) or when improper storage—such as unrefrigerated or anaerobic conditions—occurs.[4] Inadequate salt content, insufficient starter cultures, or prolonged storage at ambient temperature may further increase the risk of spore germination and toxin formation.[2] Previous reports have identified similar associations between unpasteurized dairy and botulism outbreaks, underscoring the importance of public health education on safe food handling and storage.

Based on these cases, several clinical recommendations can be emphasized. First, clinicians should maintain a high index of suspicion for botulism in patients presenting with acute cranial nerve dysfunction, bulbar palsy, or unexplained respiratory compromise, particularly when a shared dietary exposure is identified. Second, empirical administration of botulinum antitoxin should not be delayed pending confirmatory testing, as early treatment is strongly associated with improved outcomes. Third, supportive management — especially timely airway protection and mechanical ventilation — remains critical to survival in severe cases. Finally, clinicians should recognize that high-risk groups, such as pregnant women and individuals with comorbidities, may experience more rapid clinical deterioration and therefore warrant heightened vigilance and expedited treatment. These recommendations are consistent with current CDC guidelines and highlight actionable measures that can improve patient outcomes in resource-limited settings.

This report has several limitations, including the absence of food or environmental testing, lack of ancillary studies (eg, CSF or electrophysiology), small sample size, and potential recall bias regarding symptom onset and dietary exposure. In addition, serum toxin was undetectable in our patients, likely due to delayed sampling and prior antitoxin use, though this could not be fully clarified. These factors should be considered when interpreting our findings.

Conclusion

This study underscores the critical challenges in diagnosing and managing foodborne botulism in resource-limited healthcare settings. Delayed presentation, vague early symptoms, and restricted access to diagnostic tests and antitoxin therapy can complicate timely interventions and increase morbidity. Botulism remains a potentially fatal neuroparalytic illness, with symptoms that can rapidly involve respiratory and autonomic systems. The severity of illness may vary depending on the type of neurotoxin and the timing of antitoxin administration. Early recognition, prompt antitoxin treatment, and comprehensive supportive care are crucial to improving patient outcomes.

Public health education focusing on the risks of home-prepared food products, especially unpasteurized or improperly stored dairy, is essential. Increasing awareness among both the public and healthcare professionals, improving diagnostic capabilities, and ensuring equitable access to antitoxin are vital measures for reducing the burden of foodborne botulism, particularly in areas with traditional food practices.

Ethical Approval

This report does not include any personal information that could identify the patient; therefore, it is exempt from requiring ethical approval.

Consent for Publication

Upon recovery, written informed consent was obtained from each patient for inclusion in this report. A copy of the consent form is available for the editor-in-chief's review upon request.

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Disclosure

The authors declare that they have no competing interests in this work.

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