

**Educational Note**

# From Garden to Paralysis: A One Health Educational Note on Foodborne Botulism with Insights from Animal and Environmental Links

*Sohier F. Syame, Abouelhag H. A. \**

Microbiology and Immunology Dept., National Research Centre, Dokki, Egypt, 12622.

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## Abstract

**Purpose:** This educational note presents a hypothetical case of foodborne botulism to illustrate the clinical presentation, diagnosis, and management of this rare but life-threatening neuroparalytic illness. The case is expanded with a One Health perspective, exploring botulism in animals and the links between animal-source foods and human disease. The goal is to provide a teaching tool for medical, veterinary, and public health trainees.

**Key Learning Points:**

- Recognize the classic triad of afebrile, descending flaccid paralysis with bulbar symptoms and a history of consuming home-preserved or animal-derived foods.
- Understand the pathophysiology of botulinum toxin at the neuromuscular junction.
- Apply timely diagnostic and therapeutic interventions, including antitoxin administration and respiratory support.
- Appreciate the One Health dimensions: botulism in livestock, wildlife, and the potential for transmission through animal-source foods.
- Identify prevention strategies spanning safe home canning, animal feed management, slaughter inspection, and intersectoral surveillance.

**Keywords:** *Clostridium botulinum*, botulism, foodborne botulism, flaccid paralysis, botulinum antitoxin, neurotoxin, One Health, animal botulism, zoonotic potential, food safety, home canning, public health surveillance, Guillain-Barré syndrome, myasthenia gravis, descending paralysis.

## 1. Introduction:

Botulism is a neuroparalytic disorder caused by the potent neurotoxin of *Clostridium botulinum*. Although rare—approximately 20 foodborne cases per year in the United States—it carries high morbidity and mortality if not recognized and treated promptly (Centers for Disease Control and Prevention [CDC], 2021; Rao et al., 2021). The classic form is foodborne botulism resulting from ingestion of preformed toxin in improperly preserved foods, most commonly home-canned vegetables. However, animal-derived products such as contaminated milk, meat, and traditional fermented marine mammal foods have also been implicated in outbreaks (O’Mahony et al., 1990; Peck et al., 2020).

Beyond human medicine, botulism significantly affects livestock, poultry, horses, and wildlife, leading to economic losses and conservation challenges (Anniballi et al., 2013). The interconnection between animal and human botulism underscores the importance of a One Health approach that integrates human, veterinary, and environmental health.

This educational note presents a **hypothetical composite case** designed to illustrate the key clinical features, diagnostic workup, and management of foodborne botulism. It then expands the discussion to include animal botulism and the pathways by which animal-source foods can transmit the toxin or spores to humans. The note is intended for use in medical, veterinary, nursing, and public health education.

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\*Corresponding author: Abouelhag H. A.

[ha.abouelhag@nrc.sci.eg](mailto:ha.abouelhag@nrc.sci.eg)

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## 2. Illustrative Case (Hypothetical)

### Patient Presentation

A 45-year-old man with no significant past medical history presented to the emergency department with progressive double vision, dry mouth, and difficulty speaking that had begun 12 hours earlier. He reported that approximately 36 hours before symptom onset, he and his wife had consumed home-canned green beans from a batch he had prepared two months prior. The jar seal had appeared “questionable,” but the beans smelled normal. His wife ate only a small portion and remained asymptomatic.

### Physical Examination

Vital signs were normal; the patient was afebrile. Neurologic examination revealed bilateral ptosis, extraocular muscle palsies, fixed and dilated pupils, facial weakness, and a diminished gag reflex. Motor strength was symmetrically reduced (4/5) proximally, with hypoactive deep tendon reflexes. Sensation was intact. Mental status was normal.

### Diagnostic Workup

Complete blood count, basic metabolic panel, and cerebrospinal fluid analysis were unremarkable. Nerve conduction studies showed a decremental response to low-frequency repetitive stimulation, consistent with a presynaptic neuromuscular junction defect. Based on the classic triad—afebrile descending paralysis, bulbar symptoms, and a history of home-canned food—a presumptive diagnosis of foodborne botulism was made.

### Clinical Course

Within six hours of admission, the patient developed respiratory distress with a forced vital capacity drop from 4.2 L to 1.5 L, requiring endotracheal intubation and mechanical ventilation. Botulism antitoxin heptavalent (BAT) was administered approximately 48 hours after symptom onset. Serum, stool, and gastric aspirate were sent for laboratory confirmation, and the leftover green beans were collected for analysis.

Four days later, the CDC confirmed botulinum toxin type A in the patient’s serum and in the green bean sample; *C. botulinum* was cultured from stool and the green bean residue.

The patient required mechanical ventilation for 21 days, followed by inpatient rehabilitation. At six-month follow-up, he reported near-complete recovery with only mild residual fatigue and dry eyes.

## 3. Clinical Pearls for Diagnosis and Management

### 3.1 Pathophysiology

Botulinum toxin irreversibly binds to presynaptic nerve terminals at the neuromuscular junction and cholinergic autonomic synapses. It cleaves SNARE proteins (e.g., SNAP-25 for toxin type A), preventing acetylcholine release and causing flaccid paralysis. Recovery requires axonal sprouting and regeneration, accounting for the prolonged course (Pirazzini et al., 2017).

### 3.2 Key Diagnostic Features

- **History:** Ingestion of home-canned or preserved food (especially low-acid vegetables, fish, or meat); injection drug use (wound botulism); or travel to endemic areas.
- **Symptoms:** Acute, afebrile, descending flaccid paralysis; bulbar signs (diplopia, dysarthria, dysphagia, xerostomia); no sensory deficits.
- **Laboratory:** Normal CSF; presynaptic pattern on EMG; definitive diagnosis via mouse bioassay or mass spectrometry.

### 3.3 Treatment Priorities

1. **Airway protection:** Monitor forced vital capacity; intubate early if signs of respiratory compromise.
2. **Antitoxin:** Heptavalent antitoxin neutralizes unbound toxin; administer as soon as possible.
3. **Supportive care:** Mechanical ventilation, physical therapy, nutritional support, and prevention of secondary infections (Rao et al., 2021).

### 3.4 Differential Diagnosis

- Guillain-Barré syndrome (ascending weakness, albuminocytologic dissociation)
- Myasthenia gravis (fluctuating weakness, positive edrophonium test)
- Brainstem stroke (focal deficits, asymmetric)
- Tick paralysis (ascending, tick found on skin)

## 4. One Health Perspectives: Animal Botulism and Links to Human Food

### 4.1 Botulism in Animals

Animal botulism occurs worldwide and has significant economic and conservation impacts. Key features are summarized below.

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\*Corresponding author: *Abouelhag H. A.*

*ha.abouelhag@nrc.sci.eg*

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Species	Common Types	Typical Sources	Clinical Signs
<b>Poultry / waterfowl</b>	C, E	Decaying carcasses, invertebrates in wetlands	“Limberneck,” inability to fly or swim (Rocke & Bollinger, 2007)
<b>Horses</b>	B, C	Contaminated hay, silage; carcasses in feed	Progressive weakness, dysphagia, recumbency; high mortality (Whitlock & Buckley, 2015)
<b>Cattle</b>	C, D	Poultry litter, contaminated silage, carcasses	“Downer cow” syndrome, progressive paralysis (Anniballi et al., 2013)

**Prevention in animals** includes vaccination (type B toxoid for horses; types C and D for cattle), proper carcass disposal, and ensuring that silage and feed are free from animal carcasses (Anniballi et al., 2013).

#### 4.2 Transmission from Animal-Source Foods to Humans

Although botulism is not a classical zoonosis, animal-derived foods can serve as vehicles for toxin or spores. Important pathways include:

- **Dairy:** In a 1989 UK outbreak, 27 people developed type B botulism from pasteurized milk; the source was silage contaminated with a carcass fed to dairy cattle (O’Mahony et al., 1990).
- **Meat:** Two cases of type A botulism in the US were linked to commercially canned beef stew; contamination likely occurred before processing (Sobel, 2005).
- **Traditional fermented products:** In Alaska, over 250 cases of type E botulism (1950–2016) were associated with fermented seal, whale, and fish heads (Rao et al., 2021).
- **Spores in animal products:** Animals may carry *C. botulinum* spores in the intestine or on carcasses; spores can survive cooking and, under anaerobic storage (e.g., vacuum packaging), germinate and produce toxin (Peck et al., 2020).

#### 4.3 One Health Implications for Prevention

Effective prevention requires collaboration across sectors:

- **Animal feed safety:** Regulate silage and feed to prevent carcass contamination; avoid using poultry litter as cattle feed where botulism is endemic.
- **Slaughter inspection:** Exclude animals with neurological signs from the food supply.
- **Surveillance:** Share data on animal botulism outbreaks with public health authorities as early warning signals.
- **Public education:** Teach safe home-canning practices; for communities using traditional animal-based foods, promote proper fermentation, refrigeration, and cooking to inactivate toxin.

#### 5. Teaching Discussion Questions

1. What historical clue in this case was most critical to suspecting botulism?
2. Why is early intubation prioritized over waiting for confirmatory laboratory results?
3. How does the pathophysiology of botulinum toxin explain the descending pattern of paralysis and the lack of sensory deficits?
4. What are the key differences between foodborne botulism and Guillain-Barré syndrome in presentation and diagnostic testing?
5. Describe two ways that botulism in animals can lead to human illness.
6. What components of a One Health approach would you implement in a region with recurrent botulism outbreaks?

### Conclusion

This educational note used a hypothetical case of foodborne botulism to highlight the clinical recognition, timely management, and public health principles essential for reducing the burden of this rare but severe disease. By extending the discussion to animal botulism and the links through animal-source foods, the note reinforces the value of a One Health approach. Clinicians, veterinarians, and public health practitioners should collaborate on surveillance, prevention, and education to address botulism across the human-animal-environment interface.

\*Corresponding author: Abouelhag H. A.

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**Disclaimer:** This educational note presents a hypothetical composite case designed for teaching purposes. Any resemblance to actual persons or events is coincidental. All clinical details are representative of typical presentations described in the literature.

### Data Availability Statement

No original datasets were generated for this review article. All cited data and findings are available within the original research publications referenced in the manuscript, accessible via the provided Digital Object Identifiers (DOIs) or through respective journal platforms.