Review

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Review

Review of Foodborne Botulism in the UK: 2006–2024

Corinne Francoise Laurence Amar [1,*], Burhan Ahmed [1], Jonathan Finch [1], Dunstan Rajendram [1], Vanessa Wong [2] and Gauri Godbole [2]

[1] UK Health Security Agency, Gastrointestinal Bacteria Reference Unit, 61 Colindale Avenue, London NW9 5EQ, UK

[2] UK Health Security Agency, Gastrointestinal Infections, Food Safety and One Health

* Correspondence: corinne.amar@ukhsa.gov.uk

Abstract: Food-borne botulism is a rare but serious disease caused by ingestion of botulinum neurotoxin pre-formed in food by *Clostridium botulinum.* This review provides an update of foodborne botulism in the UK from 2006 to 2024 as well as summaries of incidents. No cases were reported between 2006 and 2009, but 13 cases occurred between 2010 and 2024, including two outbreaks. No fatalities were reported. Cases were predominantly linked to imported, home-made and artisanal foods, occasionally to commercial products. Diagnostic and public health challenges include delayed clinical diagnosis, delayed sample collection, inadequate specimen volumes, and the frequent unavailability of suspected food sources, hampering epidemiological investigations. The UK has an extremely low incidence of foodborne botulism with an estimated rate of 0.001 cases per 100,000 people per year, however, the risk remains. Despite the low occurrence of this illness, food botulism is a public health emergency which requires rapid reactive work to be undertaken by multiple regulatory agencies. Continuous professional training of medical staff, up to date clinical guidance, rapid diagnostic and food investigations are essential for optimising patient outcomes and prevention.

Keywords: botulism; *Clostridium botulinum*; foodborne intoxication; botulism investigation; public health response

1. Introduction

Botulism is a rare but life-threatening paralytic disease caused by a neurotoxin produced by *Clostridium botulinum*, an anaerobic, spore-forming bacterium. There are currently eight recognized botulinum neurotoxin (BoNT) types—A to G and X—though only BoNT types A, B, E, and F are known to cause disease in humans [1]. In addition, rare strains of *Clostridium butyricum* and *Clostridium baratii* have been found to produce BoNT E and BoNT F, respectively. A botulinum-like neurotoxin gene cluster has also been identified in a strain of *Enterococcus* sp. isolated from cow faeces, but its pathogenic potential remains uncertain [2].

Once in the bloodstream, BoNT binds to the neuromuscular junction, blocking the release of acetylcholine from synaptic vesicles and thereby preventing muscle contraction. This results in a rapid, symmetrical, descending flaccid paralysis that can progress to respiratory failure. Without prompt intravenous administration of antitoxin, death may occur due to airway obstruction or respiratory arrest [3].

Foodborne botulism occurs when pre-formed BoNT is ingested through contaminated food or beverages in which *C. botulinum* has proliferated. Because the bacterium is a strict anaerobe, foods at highest risk include those stored in airtight conditions, such as canned goods, preserved foods in jars, and pickled products. The disease was first documented in the 18th and early 19th centuries in Germany, but its causative organism was identified in 1895 by the Belgian scientist Van Ermengem, following an outbreak linked to contaminated smoked ham at a funeral dinner [4]. In the UK, the first reported cases occurred in 1922, when eight individuals died within five days after consuming contaminated duck meat paste sandwiches [5].

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A review published in 2006 documented 15 incidents of foodborne botulism in the UK between 1922 and 2006 [6]. Despite its rarity, foodborne botulism remains a public health emergency due to its severity, the urgent need for antitoxin treatment, and the necessity of rapid source identification to prevent further exposure.

This review presents all recorded incidents and outbreaks of foodborne botulism in the UK from 2007 to 2024.

2. Materials and Methods

2.1. Clinical Diagnosis and Notification

Botulism is a clinical diagnosis based on characteristic neurological symptoms resulting from BoNT activity at the neuromuscular junction. It typically presents with acute bilateral cranial nerve palsies, oculobulbar weakness, and a generalized, descending, symmetrical flaccid paralysis. Patients commonly report diplopia, dysarthria, dysphonia, dysphagia, and dry mouth. As the bilateral, descending muscle weakness progresses, it can lead to loss of head control, weakness of the trunk and limbs, breathing difficulties, and, ultimately, respiratory failure. Affected patients remain afebrile and conscious, with no sensory deficits. Furthermore, paralysis of autonomic smooth muscle leads to constipation and urinary retention [3]. A detailed case history can provide further indications of botulism, such as recent consumption of high-risk foods and reports of others who consumed the same food developing similar symptoms. Early symptoms, including abdominal pain, nausea, vomiting and diarrhoea followed by constipation typically appear within a few hours to 3 days after toxin ingestion. The incubation period for foodborne botulism is usually between 12 and 36 hours [3]. Clinical diagnosis can be challenging, as botulism may be misdiagnosed as other neurological conditions such as Guillain-Barré syndrome, myasthenia gravis, stroke, psychiatric disorders, or other diseases affecting the central nervous system.

Suspected cases in England are notified to public health UK Health Security Agency (UKHSA). Cases are documented in the UKHSA's case and outbreak management system where demographic details, initial symptoms, symptom progression, treatment, microbiology results and outcomes are recorded. A food botulism questionnaire is completed to facilitate the food source investigation.

2.2. Microbiology Confirmation of Botulism

Clinical specimens, suspected food, and environmental samples are sent to the UK *C. botulinum* National Reference Laboratory for confirmation of the clinical diagnosis and identification of the food source responsible for intoxication.

Detection of BoNT is performed using a mouse bioassay (MBA), in which serum, faecal or food extracts are injected intraperitoneally into the animals. Symptoms of botulism are observed up to 4 days, and confirmation, along with BoNT type identification, is carried out through a neutralization test [7]. The presence of *C. botulinum* is confirmed by real-time PCR assays targeting the *BoNT* A, B, E, and F genes [8,9] and the organism is isolated using specialised media and enrichment broths [7].

3. Results

3.1. Number of Foodborne Botulism Cases Reported in the UK, 2006–2024

From 2006 to 2009, no cases were reported. From 2010 to 2024, 7 separate incidents and 2 outbreaks occurred totalising 13 cases. There were no UK fatalities. Table 1 summarises all the incidents described here.

Table 1. Summary of all incidents and outbreaks of foodborne botulism in the UK (2006-2024), (PCR: polymerase chain reaction; MBA: mouse bioassay; GP: general practitioner; ABE: *C. botulinum* anti-neurotoxin type A, B and E pool; MG: myasthenia gravis; GBS: Guillain Barre Syndrome; IVIG: intravenous immunoglobulin).

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Incident date	Number of cases	Food source of intoxication confirmed (C) or suspected (S)	C. botulinum neurotoxin type	Microbiology confirmation of clinical diagnosis	Initial treatment
Case July 2010	1	Acquired abroad, un-identified food served at wedding (S).	B	PCR from faeces (not recovered by culture).	Pneumonia.
Outbreak November 2011	3	Commercially distributed Korma sauce in glass jar (C).	A	Serum MBA from 2 of the cases, PCR and culture from faeces of the 3 cases, MBA, PCR and culture from remnant of Korma sauce, lid and emptied jar.	Delayed diagnosis in case 1, botulism in cases 2 and 3
Case July 2012	1	Olives in brine in glass jar, artisanal product from Italy (C).	B	PCR and culture from faeces, MBA, PCR and culture from olives in jar.	Botulism.
Case October 2013	1	Home preserved mushroom in vinegar (S).	Not determined	MBA with ABE polyvalent neutralisation test from serum.	Gastroenteritis,
Case December 2016	1	Home-made tuna in oil (S).	B	PCR and culture from faeces, MBA from faeces.	Botulism
Case May 2019	1	Pizza topping (S).	Not determined	MBA with ABE polyvalent neutralisation test from serum.	Guillain Barre syndrome
Outbreak September 2023	3	Sardine in oil prepared by local restaurant (C).	B	PCR and culture from faeces from all 3 cases[1].	Botulism.
Case February 2024	1	Pub meal or home-made meat bakes (S).	B	MBA from serum, PCR and culture from faeces.	Botulism.
Case September 2024	1	Roasted chicken bites (S).	A	PCR and culture from faeces.	Stroke.

1 *C. botulinum* neurotoxin type B was detected in the sardines by the French reference laboratory.

3.2. Case Summary—July 2010

A male individual in his thirties travelled abroad to attend to a wedding. The day after arrival, he developed symptoms of food poisoning followed by neurological symptoms. He was discharged by the local hospital. Six days after symptom onset, he returned to the UK, was admitted to hospital with suspected pneumonia, and received antibiotics. His condition worsened and developed classical symptoms of botulism requiring intensive care support and was treated with botulinum antitoxin. He recovered and was discharged home a month later.

There was no history of consuming homemade or preserved food. At the wedding, he ate vegetable soup, lamb meatballs, and roasted peppers, though their preparation method was unknown. He also consumed street food the day before falling ill.

Serum was collected on UK hospital admission (six days after symptom onset), and faeces only seven days after his return due to severe constipation. *C. botulinum* type B was detected by PCR from faecal enrichment broths, but the organism could not be isolated and mouse bioassays from serum and faecal extracts were negative for BoNT. No food samples were available for testing. The final diagnosis was botulism, based on clinical symptoms and faecal PCR results. No public health response was initiated, as the suspected source was thought to be food consumed abroad.

3.3. Outbreak Summary—November 2011

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In November 2011, on two consecutive days, the *C. botulinum* UK reference laboratory was alerted to two suspected botulism cases in siblings, aged 5 and 7 years. Both were clinically diagnosed with botulism, they required ventilation and received botulinum antitoxin. Their 3-year-old sister, initially asymptomatic, developed signs of botulism six days after the first child was admitted to hospital. She was treated with antitoxin and discharged six days after admission [10].

A detailed food history covering three days before symptom onset identified potential sources. Samples collected from the home's refrigerator, cupboards and recycling bins included a number of foods suspected on the basis of biological plausibility. These included remnants of branded korma sauce with cooked chicken from the rubbish bin, an empty korma sauce jar and lid from the recycling bin, and an unopened jar of the same brand from the kitchen cupboard. The chicken korma had been consumed 2 days before the first child became ill [10].

Mouse bioassay (MBA) of serum from the 5- and 7-year-olds showed classic botulism symptoms, but there was insufficient sample for neutralisation testing. *C. botulinum* type A was detected in the rectal washout of the 5-year-old and the faeces of the 7-year-old, though BoNT was not detected by MBA. The 3-year-old also tested positive for *C. botulinum* type A in faeces. Her serum was untestable due to haemolysis and collection in EDTA, which does not meet UK Animals (Scientific Procedures) Act 1986 refinement obligations.

BoNT type A and *C. botulinum* type A were detected in the chicken/korma sauce remnants, the empty jar, and its lid. The unopened jar tested negative.

The implicated korma sauce batch was withdrawn in the UK and Ireland, with warnings issued to consumers, medical professionals, and the public. Similar alerts were disseminated across Europe. Investigation on the growth of *C. botulinum* and subsequent production of neurotoxin in the Korma jar was inconclusive [10]. Examination for loss of vacuum in 2 jars from the implicated batch showed they had received impact damage on the lid which released vacuum. This could have raised the pH of the product and consequently allow germination of spores in the implicated jar; however, this was never shown to be conclusive, and the source and cause of contamination remained unknown. Furthermore, 86 jars from different batches sampled at random from different retailers were tested for their content's pH which was in all products below 4.3.

3.4. Case Summary—July 2012

In July 2012, an adult female visited her GP with signs of mild bulbar palsy and blurring of vision. The next day, she deteriorated and was admitted to intensive care with botulism. She received botulinum immunoglobulin three days after admission (five days post-onset). She recovered well and was discharged a few days later.

Two days before symptom onset, the patient dined with friends, consuming a single olive in brine from an Italian artisanal brand purchased at a deli. The other guests avoided the olives due to their unusual smell.

Serum collected six days after symptom onset was unsuitable for testing due to delayed collection. However, *C. botulinum* type B was detected in faeces collected on day nine by culture and PCR.

The hosts later provided the opened jar of olives to the hospital and testing at the *C. botulinum* NRL confirmed the presence of BoNT type B by MBA and *C. botulinum* type B by PCR and culture. The pH was 6.65.

A Rapid Alert System for Food and Feed (RASFF) notification prompted an Italian investigation, revealing that 60 jars from the implicated batch had been sent to a single UK supplier, which distributed them to three delis [11]. The UK Food Authority issued a Food Alert for Action, but no jars from the batch were found, suggesting all had been sold [12]. As a precaution, the suppliers withdrew other batches of the same product. After investigation, it was found that the controlling factors, i.e. salt concentration, were insufficient to prevent growth and toxin production of *C. botulinum* [11].

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3.5. Case Summary—October 2013

In October 2013 a 40-year-old male Polish national visiting the UK, went to a general practitioner for symptoms of food poisoning. He was prescribed azithromycin for gastroenteritis but deteriorated rapidly the same day. He was diagnosed and treated with botulinum antitoxin. His discharge date was not communicated to the Reference laboratory.

The day before symptoms began, he had consumed home-preserved wild mushrooms brought from Poland. Picked three weeks earlier, they were transported in a polythene bag and stored in vinegar, which may have lacked sufficient acidity to prevent *C. botulinum* growth.

Serum collected three days after onset tested positive for botulism by MBA. Due to limited sample volume, confirmation was done using an ABE polyvalent antiserum, preventing BoNT type identification. Vomit and rectal wash collected six days after onset tested negative by culture and PCR, likely due to late collection.

The family had discarded all food, including the mushrooms, leaving no samples for testing.

3.6. Case Summary—December 2016

A 31-year-old male Italian national was admitted to intensive care in December 2016 with a three-day history of vomiting, followed by blurred vision. He developed classical features of botulism and was treated with botulinum antitoxin two days after admission. He recovered and was discharged a week later.

The patient, a former heroin injector, admitted to snorting cocaine on the same day he consumed home-made tuna in oil, sent by post from Italy. The food smelled bad, and he vomited 30 minutes after consumption

Early serum samples were discarded by the hospital laboratory. A serum sample taken two days after admission (five days after symptom onset) tested negative by MBA. However, *C. botulinum* type B and BoNT type B were detected in a faecal sample collected seven days after onset via culture, PCR and MBA, confirming foodborne botulism.

The jar of tuna had been opened weeks earlier and stored unrefrigerated in a cupboard. It was unavailable for testing. Another similar unopened jar tested negative for *C. botulinum* and BoNT. The pH was 5.75.

3.7. Case Summary—May 2019

In May 2019, a 28-year-old male was admitted to intensive care with clinical symptoms of botulism. Five days earlier, he had brief but severe diarrhoea after eating a pizza, followed three days later by neurological symptoms. Initially treated with immunoglobulin for presumed Guillain-Barré syndrome, he later developed respiratory distress, requiring intubation and ventilation. Botulism was then diagnosed, and he received botulinum antitoxin on day seven after symptom onset. He was discharged, but the date was not reported to the reference laboratory.

Serum sample collected on admission, i.e., five days after onset, was sent to the reference laboratory. Because of insufficient volume, MBA was confirmed botulism via neutralisation with an ABE polyvalent antiserum and the BoNT type could not be determined. Faeces collected seven days after onset tested negative for *C. botulinum* by culture and PCR.

The patient did not submit a full food history, and no pizza remnants were available for testing.

3.8. Outbreak Summary—September 2023

In September 2023, the French authorities notified the UK of a botulism outbreak linked to contaminated home-made sardines in oil served at a restaurant in southern France. At least 15 people from multiple countries were affected, with 10 hospitalised, eight requiring intensive care, and one fatality [13].

Five British adult male nationals were known to have been exposed. Three developed gastrointestinal and mild neurological symptoms, including muscle fatigue and diplopia, one to 3

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days after exposure to the sardines. All 3 were hospitalised and treated with botulinum antitoxin after returning home. All were discharged after brief hospital stays. One other individual remained asymptomatic and did not seek medical care, while another was treated in Spain where he was on holiday.

Serum was collected from the three hospitalised patients, but serum from only one patient, which was negative for BoNT, was sent with sufficient volume to enable testing. *C. botulinum* type B was detected by PCR and cultured from faecal samples from all three patients.

The French reference laboratory confirmed the sardines as the source of C. botulinum neurotoxin type B, with microbiological confirmation in several clinical cases. Local health authorities inspected the restaurant, and all products were recalled [13].

3.9. Case Summary—February 2024

In February 2024, a 64-year-old woman developed heartburn, which progressed to vomiting and difficulty swallowing a day after consuming a steak and ale pie at a pub. Over the next three days, she deteriorated and was eventually diagnosed with botulism. She was treated botulinum antitoxin four days after admission, 11 days after her symptoms first appeared. She recovered and was discharged five weeks later.

BoNT type B was detected in serum collected on day 11, and *C. botulinum* type B was detected by PCR and isolated from stool samples taken on day 16.

A food questionnaire revealed that the patient had concerns about the steak and ale pie's appearance, smell, and taste. However, it had been cooked for 15 minutes at 80–82°C, a temperature sufficient to destroy any BoNT present. She also reported consuming meat pâté on toast at the same pub that day. Within the three days before symptom onset, she purchased duck and chicken meat from a supermarket and ate home-made duck potato bake and spaghetti Bolognese. However, it was unclear from the questionnaire whether she used the purchased meat to prepare these dishes or if they were separate meals. No food remnants were available for testing, leaving the source of infection uncertain.

3.10. Case Summary—September 2024

In September 2024, a 70-year-old woman became unwell after eating chicken bites snack that she described as "off" the previous day. She initially developed diarrhoea and blurred vision, eventually developed neurological signs. Initially treated for a stroke, botulism was then diagnosed, and she was treated with antitoxin on the day of admission.

Stool and serum samples were collected the following day. *C. botulinum* type A was detected by PCR and isolated from the stool, though the serum was not tested for unclear reasons.

The patient reported that the commercially distributed roasted chicken bites had a foul smell and that she was the only one who consumed them. Her husband brought the remnant to the hospital, but the clinician on duty deemed it irrelevant to the case and discarded it.

4. Discussion

The data presented in this study updates the last major UK review by McLauchlin et al. (2006) [6] which examined foodborne botulism cases up to that year. The earlier review found that foodborne botulism in the UK was historically linked to imported or artisanal products, with a small number of incidents associated with home-prepared foods. Our findings largely confirm this trend, with cases primarily linked to imported or artisanal foods such as olives and homemade preserved foods. However, newer cases include a branded korma sauce, possibly pub food and a packet of chicken bites, highlighting the potential for contamination in commercial food either after purchase or during food production.

A recurring issue in the reviewed cases is the difficulty in confirming the food source. In most incidents, food remnants were either unavailable or had already been discarded by the time

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investigations began. This pattern was observed in cases from 2013, 2016, 2019 and 2024, where food histories were suggestive but lacked microbiological confirmation. The rapid disposal of implicated foods before public health authorities become involved limits the ability to trace and potentially prevent future cases. Additionally, specimens' collection is often delayed, leading to reduced detection sensitivity. Serum testing frequently yields negative results due to late sampling or insufficient volume provided, as seen in the 2010 and 2016 cases. Stool testing by PCR and culture appears more reliable, as evidenced by the investigation of 7 out of the 9 incidents described here, however, the detection of *C. botulinum* in faeces alone is compatible with a clinical diagnosis of botulism but does not evidence an intoxication. A further concern is the potential worsening of patient conditions due to inappropriate treatment. The administration of antibiotics in suspected botulism cases, can lead to increased toxin release as bacterial lysis occurs in the gut. This underscores the need for heightened awareness among clinicians regarding appropriate management strategies.

The absence of fatalities in the UK cases suggests effective clinical management and timely administration of botulinum antitoxin for all cases described here. However, significant diagnostic and public health challenges persist, including delayed diagnosis, sample collection, inadequate specimens for testing, and the frequent unavailability of suspected food sources, all of which hinder epidemiological investigations.

The findings of this review highlight the rarity of foodborne botulism in the UK, with only 13 cases reported between 2010 and 2024. This contrasts with higher case numbers in some other European countries. According to the European Centre for Disease Prevention and Control (ECDC), 84 confirmed botulism cases were reported across the European Union and European Economic Area in 2022, resulting in an overall notification rate of 0.02 cases per 100,000 population. This figure included all types of botulism; however, foodborne botulism was noted as the most common form of the disease frequently caused by inadequately processed, often home-canned, preserved, or fermented foods. In the 2022 ECDC report, the mode of transmission was identified for 35 cases, with 33 (94%) attributed to foodborne sources, primarily caused by BoNT type A and linked to the consumption of canned food, fish, meat, and vegetables. The 2021 ECDC report shows similar trends [14,15]. In comparison, the average annual rate of foodborne botulism in the UK from 2006 to 2024 is approximately 0.001 cases per 100,000 population (or about 1 case per 100 million people per year), highlighting its extreme rarity. Stringent food safety regulations, along with a lower prevalence of home preserved food or home canning may likely contribute to the low incidence. Despite this, the UK is not immune to foodborne botulism risks. Imported and artisanal foods, as well as potential lapses in commercial food production remain potential sources of contamination. This risk is exemplified by the 2011 korma sauce outbreak [10], where it was hypothesised that vacuum loss and pH changes may have allowed *C. botulinum* to proliferate. The absence of cases linked to domestically produced home-canned foods does not eliminate the risk, as globalization of food supply chains continues to introduce new exposures. The clinical risk assessment for food botulism in the UK used to heavily focus on history of consumption of foreign food, however the more recent cases, post COVID pandemic, were associated with local food consumption. Therefore, the clinical and public health risk assessment protocols in England have been updated to consider food botulism in a cases with the appropriate clinical presentation regardless of local or imported food consumption.

5. Conclusions

Foodborne botulism remains an uncommon but serious public health threat in the UK. The overall case burden is low compared to some other European countries, but challenges in diagnostic confirmation and food traceback persist. Improving awareness amongst clinicians to enable them to identify this rare but serious medical emergency, importance of early sample collection and appropriate treatment strategies is crucial for improving case outcomes and controlling outbreaks.

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