Journal of Public Health | Vol. 28, No. 4, pp. 337–342 | doi:10.1093/pubmed/fdl053 | Advance Access Publication 17 August 2006

Food-borne botulism in the United Kingdom

Jim McLauchlin, K. A. Grant, C. L. Little

Health Protection Agency Department of Gastrointestinal Infections, Centre for Infections, 61 Colindale Avenue, London NW9 5EQ, UK
Address correspondence to Jim McLauchlin, E-mail: jim.mclauchlin@hpa.org.uk

ABSTRACT

Food-borne botulism is a rare but serious disease caused by ingestions of neurotoxin [botulinum neurotoxins (BoNTs)] produced as a result of the growth of the bacterium *Clostridium botulinum* in foods before consumption. The disease is rare in the United Kingdom, and only 62 cases have been recognized between 1922 and 2005. This report provides a brief review of *C. botulinum* and food-borne botulism as well as descriptions of the six episodes (33 cases with three deaths) of this disease that occurred in the United Kingdom between 1989 and 2005. The six incidents illustrate the importance of the risk factors of poor processing or storage of commercially prepared foods, improper home preservation of foods and travel to countries where botulism is much more common than in the United Kingdom. Even small outbreaks of food-borne botulism can precipitate a national emergency and inundate public health and acute care provision. This report provides a reminder to public health professions of the occurrence, diagnosis, treatment and control of this rare but serious food-borne disease.

Keywords botulism, *Clostridium botulinum*, epidemiology, foodborne intoxication, public health responses

Introduction

Botulism was one of the first food-borne diseases to be described and was named in 1820 after the Latin for sausage, botulus, which was historically associated with transmission.[1] The causative organism, *Clostridium botulinum*, was first cultured by van Ermengem in 1896 in Belgium. It is now understood that botulism is caused by neurotoxins [botulinum neurotoxins (BoNTs)] usually produced by *C. botulinum*, which results in paralysis by inhibiting neurotransmitter release at the neuromuscular junction.[2] Human food-borne botulism is caused by the ingestion of preformed BoNT in foods where *C. botulinum* has grown.[1] However, botulism can be transmitted by other routes, i.e. as an intestinal colonization by *C. botulinum*, which usually occurs in infants, or as a wound infection.[1] Wound botulism is now the most common presentation of this disease in the United Kingdom, and although not reported before 2000, up to the end of 2005, almost 120 cases were reported.[3–5] All of these wound infections were amongst illegal injecting drug users (IDUs) and occurred co-incidentally with a rise in soft-tissue infections due to other spore-forming anaerobic bacteria within this patient group.[6] Infant botulism is very rare in the United Kingdom, and only six cases were detected between 1978 and 2001.[7]

Food-borne botulism was first recognized in the United Kingdom in 1922, and since then, only 62 cases have been reported (Table 1). However, even small outbreaks of food-borne botulism can precipitate a national emergency. Changes in eating habits (including the consumption of minimally processed foods stored at refrigeration temperatures) together with the greater consumption of foods from areas of the world with a higher incidence of this disease make this opportune for reviewing recent UK experience of food-borne botulism. This report serves as a reminder of this rare but serious cause of food poisoning.

Clinical diagnosis

Botulism presents with an acute bilateral cranial nerve neuropathy causing dry mouth, dysphagia, slurred speech, ptosis and ophthalmoplegia. Patients are afebrile, unless a secondary infection is present, and conscious, although they may appear lethargic and have difficulty in communicating because of neuropathy. Sensory changes do not occur. Disease progression is marked by a symmetrical descending muscle weakness resulting in the loss of head control, hypotonia, generalized weakness, difficulty in breathing and eventually respiratory failure. Deep tendon reflexes diminish as the disease progresses, and constipation may also occur. If

Jim McLauchlin, PhD
K.A. Grant, PhD
C.L. Little, PhD

Table 1 Food-borne botulism in the United Kingdom

Year	*Cases (deaths)*	*Home prepared*	*Implicated food (country of origin if outside the United Kingdom)*	*Botulinum neurotoxin (BoNT) type*
1922	8 (8)	No	Duck pâté	A
1932	2 (1)	Yes	Rabbit and pigeon broth	NK
1934	1 (0)	Yes	Jugged hare	NK
1935	5? (4)?	Yes	Vegetarian nut brawn	A
1935	1 (1)	Yes	Minced meat pie	B
1949	5 (1)	Yes	Macaroni cheese	NK
1955	2 (0)	NK	Pickled fish (Mauritius)	A
1978	4 (2)	No	Canned salmon (USA)	E
1987	1 (0)	No	Rice and vegetable shelf stable airline meal	A
1989	27 (1)	No	Hazelnut yoghurt	B
1998	2 (1)	Yes	Bottled mushrooms (Italy)	B
2003	1 (1)	Yes	Sausage (Poland)	B
2004	1 (0)	No	Hummus	NK
2004	1 (0)	NK	Not known*	A
2005	1 (0)	Yes	Home-preserved pork (Poland)	B

NK, not known.

**Returned from travel to Georgia.*

untreated, death results from airway obstruction or respiratory failure. There is a high degree of variation in severity, presumably related to exposure to different amounts of toxin; however, those patients most severely affected require prolonged acute care including assisted respiration, sometimes for several months.[1,2]

The clinical diagnosis of botulism can be difficult, and misdiagnosis with the following has been reported: Guillain–Barré syndrome, myasthenia gravis, stroke, intoxication with depressants (e.g. alcohol and carbon dioxide), poliomyelitis, central nervous system infection, brain tumour or psychiatric illness. Botulism differs from other causes of flaccid paralysis in that it affects the cranial nerves more severely; it is symmetrical and shows an absence of sensory signs and symptoms. Diagnostic procedures that may be useful in excluding other causes include CT scans to identify a space occupying lesion, lumbar puncture to identify infection and cerebrospinal fluid protein levels that are increased in Guillain–Barré syndrome but normal in botulism.[1,2] Electromyography can be a useful adjunct to investigation.[8] The incubation period for food-borne botulism is usually between 12 and 36 h.[1,2]

Treatment and patient management

The mainstay of treatment of food-borne botulism is the inactivation of toxin in the patient. This is achieved by intravenous administration of antitoxin which neutralizes free toxin that has not yet bound to nerve endings. The decision to treat must always be based on clinical observation, case history and physical findings and must not be delayed for the results of laboratory tests. Because antitoxin is the only specific therapy for botulism and because it is most effective early in the course of the disease,[9] early administration is essential. Treatment carries a significant risk because some patients are hypersensitive to serum from other animals; hence, patient assessment by a consultant neurologist as well as by an infectious disease physician should be urgently sought. Antibiotic treatment has no role in the treatment of food-borne botulism because this may increase toxin release from organisms in the gut.[2] Patients with botulism require meticulous supportive care, sometimes for many months, including feeding by enteral tube or parenteral nutrition, mechanical ventilation and treatment of secondary infections.

In the initial stages of a food-borne outbreak of botulism, clinicians must, as a matter of urgency, liaise closely with public health and microbiological colleagues because the identification of implicated foods and their withdrawal from consumption may prevent further cases occurring (see *Public health responses to food-borne botulism*).

Clostridium botulinum and BoNT

Clostridium botulinum is a group of gram-positive, spore-forming obligate anaerobic bacteria, which are defined on the basis of a single phenotypic characteristic, i.e. BoNT production. The species is classified into four distinct taxonomic lineages (designated groups I–IV) which combine organisms of high phylogenetic diversity that consequently show considerable differences in genotype and phenotype. This situation is further complicated because there are genetically very closely related *Clostridium* species that do not produce neurotoxins and are therefore not named *C. botulinum*; there is instability and horizontal gene transfer of BoNT genes such that other *Clostridium* species may produce neurotoxins; non-toxigenic '*C. botulinum*' variants containing cryptic or fragmentary copies of BoNTs occur.[10]

BoNTs are proteins that can be divided into seven antigenically distinct types, designated A–G, and are amongst the most potent toxins known.[1] Human botulism is almost always associated with *C. botulinum* from taxonomic lineages I and II, which produce toxin types A, B, E and F, type F being the least common (Table 2). Toxin types A and B are the most common causes of food-borne botulism, and type E is particularly associated with the consumption of marine products. The most common types associated with food

Table 2 Characteristics of *Clostridium botulinum* groups I and II

	Group	
	I	*II*
Neurotoxin types	A, B and F	B, E and F
Human disease	Yes	Yes
Growth temperatures		
Minimum	10	3.3
Optimum	35–40	18–25
$D_{100°C}$* of spores (min)	25	<0.1
$D_{121°C}$* of spores (min)	0.1–0.2	<0.001
Distribution in the environment	Widespread	Widespread
	Type A generally more common in Western USA Type B is more common in the Eastern USA, Europe and Asia	Type E common in marine sites worldwide

ND, not determined.

**D (decimal reduction) values are the time taken to kill 90% of organisms at a specific temperature.*

poisoning are type A in the United States (except for Alaska where type E predominates[11]) and type B for most of Europe.[12] Some of the physiological properties of *C. botulinum* groups I and II are summarized in Table 2, and of note is their considerable resistance to heat of the spores (especially by those in group I), which is important when considering survival through food processes.

During the disease process, BoNT is absorbed into the circulation and taken up by nervous tissue. At the neuromuscular junction, the toxin blocks the release of acetylcholine, which inhibits neuronal transmission resulting in flaccid paralysis. The toxin is not dislodged by the administration of antitoxin; hence, recovery only occurs when new terminals grow from the affected nerve end plate, a process that may take weeks or months.[2]

Laboratory diagnosis

As stated above, a diagnosis of botulism relies on urgent clinical observation: laboratory analysis may be able to subsequently confirm but not refute a clinical diagnosis. However, because BoNT production is the cardinal feature for the identification of *C. botulinum*, the detection of toxins provides confirmation of a clinical diagnosis as well as identifying the bacterial species. Criteria for a confirmed laboratory diagnosis of food-borne botulism[13] include the detection of BoNT in serum, stool, gastric or intestinal contents or patient's food and/or the isolation of *C. botulinum* from stool, gastric or intestinal contents. *Clostridium botulinum* should also be recovered from implicated foods, but because this organism is common in the environment, spores (usually in small numbers) will naturally occur in food without presenting a disease risk. If appropriate clinical samples are not available, the toxin concentration is below the detection limit, or toxin degradation has occurred during transit; it may not be possible to detect BoNT to confirm the clinical diagnosis. In some outbreaks, concentrations of BoNT below the detection level occur in 30–40% of patients.[14] In addition, the timing of specimen collection is important because toxin is present in serum or faeces in >50% of cases within 1 day of onset but <25% after 3 days: *C. botulinum* is present in the faeces of >70% of cases within 2 days and 40% after 10 days.[9] Traditionally, neurotoxin detection has relied on the use of an *in vivo* mouse bioassay to detect BoNT activity.[1,2] All clinical specimens for BoNT detection must be collected before the administration of botulinum antitoxin.[1,2]

The mouse bioassay to detect BoNT takes 4 days to complete, and the prolonged anaerobic culture and identification of the bacterium can take several weeks to complete. Considerable progress has been made by the authors in the development of real-time PCR-based assays for BoNT A, B, E and F gene fragments, and these have been shown to be applicable directly to clinical material (faeces) as well as to enrichment cultures, bacterial colonies growing *in vitro* and food[15] (K.A. Grant, HPA unpublished results). Although these assays will not provide a complete substitute for the bioassay, data from this laboratory have shown a very high correlation between the results of these two approaches and that PCR-based assays provided faster diagnostic turnaround (especially when seeking organisms in food) and a reduction in the number of *in vivo* tests needing to be performed.[15,16]

Recent experience of food-borne botulism in the United Kingdom

As summarized in Table 1, food-borne botulism was first recognized in the United Kingdom in 1922; however, since 1989, there has been six incidents (33 cases with three deaths) detected. These six incidents will now be considered in more detail.

The largest outbreak of food-borne botulism in the United Kingdom occurred in 1989 with 27 cases and one fatality associated with the consumption of commercially prepared hazelnut yoghurt.[17] This was a very unusual food vehicle because low-acid foods, such as yoghurt, do not support the growth of *C. botulinum* and therefore prevent the production

of BoNT. However, in this instance, canned hazelnut conserve was prepared by mixing pre-roasted hazelnuts, water, starch, aspartate and other ingredients and heated at ≥90°C for 30 min. The canned preserve was stored at room temperature for several months where the growth of *C. botulinum* and BoNT production occurred. The hazelnut conserve was added to yoghurt before dispensing in cartons that were given a 25-day 'sell by' date. The outbreak was recognized by the admission to hospital of patients (including those from the same family) with symptoms compatible with botulism to different hospitals in Northwest England. The outbreak was controlled within 4 days of the first patient being admitted to hospital by the withdrawal of the implicated brand of hazelnut yoghurt from retail sale following the identification of this as a common food consumption by most patients. Subsequent laboratory tests showed that *C. botulinum* type B and BoNTB were detected in the faeces of one of the patients, opened and unopened cartons of yoghurt and the canned hazelnut conserve.

The next incident of food-borne botulism occurred in 1998.[18] A husband and wife of Italian origins brought back to England from Italy and consumed home-preserved mushrooms in oil. Both family members developed botulism (one died), and *C. botulinum* type B and BoNTB were detected in serum and faeces of one patient as well as in the bottled mushrooms. The bacterium alone was recovered from the faeces of the patient who died.

In 2003, a male Polish national living in the United Kingdom developed botulism and subsequently died. BoNTB was detected in serum samples collected before death. The patient shared a meal with a second Polish national, which included a home-prepared meat product ('bigosh') brought from Poland. The second Polish national returned to his home country and was diagnosed with botulism.

Two single food-borne botulism incidents occurred in 2004. The first was based on clinical diagnosis alone in a male adult patient who consumed commercially prepared hummus which had been inappropriately stored in the patients home at room temperature for several weeks. The food was described as 'off' which is why no other family members consumed this product. Because of delays in investigating this patient, appropriate samples were not collected from the patient sufficiently early to confirm the clinical diagnosis. The patient recovered and was discharged without any specific anti-botulinum therapy. The second case was a female patient who returned to England from Georgia. *Clostridium botulinum* type A was recovered from the patients faeces ~10–14 days after the onset of illness which commenced as the patient was returning to the United Kingdom. A food history was taken, and multiple traditionally prepared foods had been consumed just before leaving Georgia. This patient was also discharged without any specific anti-botulinum therapy.

The final incident occurred in a male Polish national living in England in 2005, who developed botulism within 24 h of consuming home-preserved pork originally prepared in Poland. *Clostridium botulinum* type B and BoNTB were recovered from the patient's faeces and from the jar of home-preserved pork. The patient was treated with antitoxin and made a complete recovery. The preserved pork had been home slaughtered, bottled and stored at room temperature for several months in Poland before bringing to England for consumption.

Public health responses to food-borne botulism

A single incident of food-borne botulism constitutes a public health emergency; therefore, urgent action must be taken by public health professionals as soon as a suspect case is identified. Do not wait for the start of the next working day. These actions must include establishing a food history from affected patients because this can allow the identification of the vehicle of intoxication and its withdrawal, thus preventing further cases occurring. If it is not possible to obtain a food history from affected patients, this should be urgently sought from family members or other contacts. Food and food remnants from the patients home or from other sites where affected patients eat food should be collected, and suspect foods should be sent without delay to a specialist laboratory with expertise in the detection of *C. botulinum* and its neurotoxins. It is important to raise an awareness among front-line medical professionals who might come into contact with further cases and who should prepare for intensive and prolonged periods of acute care of affected individuals. Those involved with food regulation must also be informed to achieve rapid withdrawal of contaminated foods and generate public warnings to avoid the consumption of specific products as soon as information on their identity becomes available. Finally, those supplying antitoxin, which is the mainstay of treatment, should be alerted to a likely increased requirement.

The 27 cases associated with hazelnut yoghurt in 1989[17] illustrate the effectiveness of the introduction of control measures. In this outbreak, preliminary investigations showed that seven of the initial eight patients recognized had consumed a specific brand of hazelnut yoghurt which, 3 days after the report of the first case, was withdrawn, and a public warning to avoid its consumption was made. The severity and rapidity of the onset of illness necessitated the introduction

of these appropriate interventions, which were done before the availability of results from analytical epidemiology or specialist microbiology.

Discussion

Main finding of this study

This study provides a brief update of *C. botulinum* and food-borne botulism as well as descriptions of the six episodes of this rare disease that occurred in the United Kingdom between 1989 and 2005.

What is already known on this topic

The six recent episodes of food-borne botulism illustrate the likely contributory risk factors of poor processing or storage of commercial products, home preservation or travel to areas where botulism is much more common than in the United Kingdom. The risk factors of importance with the two instances associated with commercially prepared foods were inappropriate storage (hummus) or poor treatment of a food ingredient (canned hazelnuts) which was insufficient to kill this bacterium and did not prevent its multiplications (and toxin production) during storage. The remaining four incidents were associated with products from southern or eastern Europe which have previously reported much higher rates of food-borne botulism.[12] The increased reporting of this disease is due to considerably more common practices of home or small-scale preservation[19] where safe food preservation is inherently less under control than good commercial manufacturing processes. Poland has historically reported high rates of botulism, e.g. during 1961 and 1998, between 81 and 738 cases per year were reported.[20] Varma and colleagues[21] also suggested that economic decline (particularly in eastern Europe) may also be a contributing factor favouring food-borne botulism which results in a lack of reliable energy sources, access to clean water and food shortages compelling persons to preserve food for a larger proportion of their diet. Varma and colleagues[21] further reported that Georgia now has the highest national reported rate of food-borne botulism worldwide, with annual rates up to 3.6 cases per 100 000 of the general population occurring.

Three of the recent episodes in the United Kingdom were associated with the import of food from other European countries. Within the EU, there is a single market, thereby allowing free movement of foodstuffs between Member States. Food imported into the EU for placing on the market must comply with the relevant requirements of food law or conditions recognized by the Community to be at least equivalent thereto, or where a specific agreement exists between the Community and the exporting country, with these requirements [(EC) Regulation 178/2002]. A reasonable amount of any food on sale in any EU country may be brought back from another Member State for personal use and not to sell to other people. Personal imports of meat, meat products, milk and milk products are not allowed from countries outside the EU, with the exception of Andorra, Norway and San Marino, whereas reasonable quantities of food that do not contain anything of animal origin are allowed. There are also certain restrictions on fishery products, shellfish, eggs, egg products or honey. Information on personal imports is provided on the Food Standards Agency and Department of the Environment Food and Rural Affairs websites. Illegal imports present unknown risks to human health because they have avoided health and hygiene inspections and could potentially introduce diseases to the United Kingdom, including botulism.

What this study adds

This study serves as a reminder to public health professionals of this rare but serious cause of food poisoning. The six incidents of food-borne botulism in the United Kingdom since 1989 illustrate the importance of recognizing the risk factors of poor processing or storage of commercially prepared foods, improper home preservation of foods and travel to countries where botulism is much more common. Imported foods for personal use (some of which are illegal) where controls on preventing the growth of *C. botulinum* are more likely to be unsuccessful also represent a further risk.

In addition to the risk factors outlined above, there is an additional possibility of why food-borne botulism should be considered in the future. One of the properties of *C. botulinum* group II is the ability to multiply at refrigeration temperatures (Table 2). Food manufacturers, particularly those of minimally processed ready-to-eat foods with extended refrigerated shelf lives, should be aware of this possibility and include strategies to prevent the growth of this bacterium.

Limitations of this study

This study summarizes the most recently reported cases of food-borne botulism. Because this disease can present diagnostic difficulties and is unlikely to be frequently encountered in the United Kingdom, there may be under-ascertainment especially in cases with mild presentations, although these are probably rare. In addition, this report has not considered wound or infant botulism (which requires somewhat different public health control strategies), the former being the most common presentation of this disease in the United Kingdom.

Conclusions

Public health professions should be aware of the diagnosis, treatment and control of food-borne botulism that is a rare but serious cause of food poisoning in the United Kingdom. This disease can constitute a public health emergency and requires urgent investigation and implementation of appropriate interventions. Large outbreaks have occurred in other countries,[22] and at the time of writing (Spring 2006), >100 cases associated with the consumption of preserved bamboo shoots occurred in Thailand.[23] Because of the intensive and prolonged acute care needed by patients, even small outbreaks of food-borne botulism can precipitate a national emergency and inundate public health and acute care provision.

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