

## Regional Review Article

# Food-borne botulism in Argentina

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### Abstract

Botulism is a severe neuroparalytic disease caused by *Clostridium botulinum* toxins. Although the disease is uncommon it is a cause of great concern due to its high rate of mortality. Food-borne outbreaks of botulism occur worldwide and require immediate public health attention and acute care resources.

Analysis of outbreaks showed that the food products most often involved were fermented fish products in Alaska; home-canned food, oil preservation and restaurant sauce in the rest of the United States (US) and in London and; and home-canned vegetables, airtight packed food with inappropriate refrigeration, and aerosols in Argentina.

The diagnosis is based only on clinical findings matching the disease and previous exposure to suspicious food. Botulism must be immediately identified as even one case suggests the start of an epidemic and should be treated as a public health emergency. Therefore, the purpose of the following review is to recognize the risks associated with the consumption of potentially dangerous foods, and to encourage prevention by seeking to make all public health professionals aware of the dangers of this potentially lethal disease.

**Key Words:** food-borne; botulism; infant botulism; *Clostridium botulinum*

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### Introduction

Botulism is a paralytic illness caused by neurotoxins synthesized by spore-forming anaerobic bacterium named *Clostridium botulinum* (*C. botulinum*). There are seven different immunological kinds of toxins, types A to G, but human botulism is usually caused by types A, B and E, the most powerful poisons ever known. Food-borne botulism is caused by the consumption of food containing the neurotoxin produced by *C. botulinum*. It is most frequently caused by the ingestion of inappropriately processed or stored, home-canned foods, which create an anaerobic environment in which spores thrive, germinate, reproduce and synthesize the toxin. The neurotoxins block the neuromuscular stimulation by interfering with cholinergic autonomic neuromuscular plaques at presynapsis [1].

The clinical illness shows cranial nerve paralysis, followed by descending flaccid muscular paralysis, which may involve breathing muscles from the larynx and also decreased saliva secretion with dryness of buccal mucosa. The most common symptoms are blurry vision, dysphagia and dysarthria. Even though ptosis and dysarthria of victims of botulism poisoning may be confused with

the signs of encephalopathy, these patients are totally conscious and afebrile. The incubation period lasts from 12 to 36 hours after initial toxin ingestion; generally, neurological symptoms are preceded by nausea, vomiting, abdominal pain and diarrhoea. The healing process varies from weeks to months, depending on the severity of the case. The close support of an intensive care unit with mechanical ventilation and the administration of an equine trivalent antitoxin are necessary for treatment. If the antitoxin is given soon enough, it may prevent worsening of paralysis and reduce the length of the disease [2].

The severity of the symptoms depends on the amount of toxin ingested by the patient. In the case of a mild botulism attack, only nausea or minor neurological symptoms may be present, and thus an appropriate diagnosis may be missed. This may also lead to an underestimation of the true number of cases.

### Epidemiology and Transmission

Botulism is caused by the consumption of food that has been contaminated with the *C. botulinum* neurotoxin, whose spores are widely distributed in

the environment. Proper food storage requires eliminating the conditions needed for the pathogenic microorganisms to thrive and taking measures to prevent recontamination. *C. botulinum* needs an anaerobic environment that is free of growth inhibitors, that has adequate nutrients, a suitable temperature, enough water, and adequate acidity to support the growth and production of toxins.

The growth of *C. Botulinum* in food products may be prevented by control of acidity ( $\text{pH} \leq 4.6$ ), water activity ( $a_w$ ), refrigeration and chemical preservatives. The  $a_w$  may be determined by an electrical hygrometer that measures the water available for microbial growth; values beneath 0.93 stop *C. botulinum*'s growth. Most moist foods, such as fresh meat, fruits and vegetables, have high  $a_w$  values, and therefore require refrigeration (less than 4°C) to inhibit the toxin production by *C. botulinum*. Food dehydration, through sprinkling or freezing, provides one effective way to reduce  $a_w$ . Another way is to add sugar or salt to capture water molecules until osmotic equilibrium is reached. A low  $a_w$  is ensured by any of these means. The combination of packing in airtight receptacles and food fermentation leads to anaerobic conditions that allow *C. botulinum*'s spores to germinate [3].

At the beginning of the 20th century, the number of epidemics produced by commercial canned foods was reduced. However, home-canned foods represented the main cause for botulism, not only of sporadic cases but also in the outbreaks reported from restaurant food [4]. Traditional food from Alaska, especially fish, fish eggs, seals, beavers and whales, which are processed by allowing room temperature fermentation and then consumed without cooking [5], are sources of botulism in that geographic area. As the distribution of large amounts of contaminated food may affect many people, there must be constant epidemiologic surveillance established and every case of botulism should be treated as a public health emergency.

### **Epidemiology of Botulism outbreaks**

Between 1990 and 2000, 160 food-borne botulism outbreaks were reported in the United States. Food items involved in these cases were home-canned, including soups, tomato sauce, garlic in oil, stew, potato salad, homemade sausages and, less frequently, meatballs, steak, liver pâté, bread pudding, apple pie sauce, hamburger, chili and peyote [4].

Also, in the United States, there have been outbreaks caused by food prepared in restaurants, such as the sauce, *skordalia*, made from potatoes and cheese. The *skordalia* was made with potatoes wrapped in aluminium foil that were baked and then kept in the same wrapping paper at room temperature for several days. The epidemiologic investigation of this outbreak led to identifying this method as the main hazard, a practice that needs to be eliminated from restaurants and homes. Other control measures depend on the education and awareness of people working in restaurants and the efforts made by health authorities [3,4]. Other botulism outbreaks have been related to traditional recipes of salted un-gutted fish, warmed in the sun and then sealed into barrels, such as Egyptian *fasikh*, *kapchunka*, and *mohola*, etc. [5,6].

Between 1990 and 2000, a total of 91 botulism cases occurred in Alaska. Arctic explorers and whale men have described the deaths of entire native families that ate whale meat. Even though the mortality rate for botulism in natives has decreased in recent decades, [7,8] the incidence of botulism in Alaska is among the highest of the world, and every case has been associated with the consumption of traditional native foods, including aquatic fermented mammals such as *muktuk* (the pink fat under the whale's skin), beaver tail, flipper and seal oil. Others were caused by fermented salmon and white fish heads or fermented fish eggs. Some 88% of the cases were due to the type E toxin, which is exclusively related to ingestion [9] of aquatic mammals.

McLaughlin *et al.* described a type E botulism outbreak associated with the ingestion of beached whales. On 12 July 2002, two Yupik village residents, in West Alaska, found a beached whale's skeleton on the Bering Sea coast, which seemed to have died that spring. They took the tail for consumption, chopped it and sealed it in plastic bags that were refrigerated and later distributed to friends and family. During the next three days, a total of 14 people ate the raw *muktuk*. On 17 July, a doctor from West Alaska reported three suspicious cases of botulism in that village; all of the patients had eaten the *muktuk*. Out of 14 people identified as having eaten *muktuk*, 8 (57%) had the disease. All of the seven *muktuk* samples taken tested positive for the type E botulinic toxin [10].

According to McLaughlin and Grant, in the United Kingdom the following botulism outbreaks have been identified during the last eight decades. The first outbreak reported was in 1922 with eight

cases, all fatal, due to commercial duck pâté. This outbreak was then followed by two cases, with one death, caused by rabbit and pigeon broth; one because of jugged hare; five, with four deaths, due to vegetarian nut brawn; one fatal case contracted from a minced meat pie; one fatality out of five cases caused by macaroni and cheese; two more incidents caused by pickled fish; four cases with two deaths associated with tinned salmon imported from the US (the can was contaminated from the factory environment); and another single case from a rice-and-vegetable shelf-stable airline meal, which was stored for too long under conditions that supported the development of *C. botulinum* type A toxin. The largest outbreak in the United Kingdom, with 27 cases but only one death, occurred in 1989, caused by the consumption of commercial hazelnut yogurt, an unusual vehicle, because of its low acidity, for *C. botulinum*'s growth. But the toxin was produced as a consequence of insufficient heating to kill the bacteria plus the absence of chemical preservatives. The two most recent outbreaks, in 1998 (caused by bottled mushrooms) and in 2002 (due to sausages), were associated with imported products originally from Italy and Poland. The Italian case caused two cases and one death, and the 2002 Polish case resulted in one fatal case of botulism [11].

Botulism is a major public health problem in Argentina and has been recognized since 1922, when the first outbreak was reported in Mendoza; it was caused by home-canned asparagus. Botulism outbreaks in Argentina have been mainly due to consumption of improperly processed meats and vegetables as well as home-canned food. Food involved in the outbreaks included ham, red and green chili peppers, the rodent vizcacha (*Lagostomus maximus*), eggplant, cucumbers, heart of palm, tomatoes, peaches, spinach, cheese with onions, pickled octopus, asparagus, canned fish and sweet corn, all home canned [12]. Between 1992 and 2004, forty-one food-borne botulism cases were reported, and the cause in each was storing food under conditions that benefit the toxin's growth [12]. One of the most noteworthy outbreaks occurred in January 1998 in Buenos Aires and affected several bus drivers. Out of 11 people who consumed the food, nine (82%) developed the disease. The type A neurotoxin was detected and the food related to the outbreak was *matambre* (rolled and stuffed meat). The *matambre* had been cooked in water at 78°-80°C for four hours, then packed, sealed in a plastic bag and wrongly refrigerated, which allowed the *C.*

*botulinum* bacteria to thrive and produce the neurotoxins that cause the illness [13].

Infant botulism is a toxic infection produced by germination and toxinogenesis of *C. botulinum* in an infant's bowel. The bacterial spores, widely distributed in nature, are ingested with any contaminated object on the ground, or by their own hands. Less frequently, the bacterial spores are ingested by the consumption of contaminated foods that contain honey, maize syrup, and medicinal herbs [14].

The first case of infant botulism was reported in the US by Arnon, and at present a total of 1,200 cases are known [15]. In Argentina, the first two infant botulism cases were confirmed in Mendoza [16] and Buenos Aires [17] in 1982. Between then and 2000, an additional 275 cases of child botulism were diagnosed in Argentina [15]. The geographical distribution by province was as follows: Mendoza 46 (31.7%); Buenos Aires 45 (31.0%); Neuquén 16 (11.0%); San Luis 13 (9.0%); Río Negro seven (4.8%); Chubut four (2.8%); La Pampa, three (2.1%) cases, the same as in Córdoba and San Juan provinces; Tierra del Fuego; Misiones, Salta, Santa Fe and Tucumán, one case (0.7%) [18]. Mendoza province leads the nation in terms of the number of absolute cases. In every case the neurotoxin involved was type A, the predominant type in Argentina. Age is an outstanding feature in patients; cases normally range from two weeks to six months old, with an average of ten weeks. Nevertheless, there have been cases published of a patient eight months old and another one ten days old [18]. It is worth mentioning that in Mendoza province most of the cases were breast-fed babies residing in rural areas. These areas typically have little precipitation and strong winds that provide a larger spore load and promote the spreading of the spores in the atmosphere; therefore, there is a larger probability that the spores can gain access to the digestive tract.

There have been attempts to explain botulism transmission by food in infants, but spores were only identified in honey and maize syrup. In Argentina, 227 honey samples were analyzed and in eight of them (3.5%) spores were found. Other sources of botulism in Argentina are homemade infusions. Of nine cases in San Luis, seven patients had taken herbal infusions. A study of medicinal plants in Argentina—*i.e.*, poleo, anise, sen and other “medicinal herbs” [15]—found that four of 100 samples were contaminated with *C. botulinum* type

A spores. Recently, botulism was related to sudden infant death syndrome [12].

### Control and Prevention

In the United Kingdom, every outbreak associated with commercially prepared products had evidence of poor handling and processing [11]. Food-borne botulism among Alaskan natives is an old problem that has worsened in recent decades due to the altering of traditional processing that includes the unsafe inclusion of plastic or glass packaging for fermentation [9].

*C. botulinum* is an uncommon cause of illness because the conditions required for it to thrive and produce the toxin—such as  $\text{pH} > 4.6$  (low acidity), high  $a_w$ , absence of chemical preservatives, room temperature and anaerobic environment—are not likely to occur in food products [1]. Some homemade food items involved in outbreaks had low amounts of salt, no chemical preservatives, and were bottled airtight. Such products are thought to be “more healthy” by the general public.

The type E toxin is responsible for more than 85% of the botulism cases in Alaska, as many traditional local dishes, such as salmon head, whale fat, seal oil and skin, and fish eggs, are prepared by fermentation under suitable conditions for neurotoxin type E of *C. botulinum* to thrive. The use of airtight packages for storage and fermentation is considered to be responsible for at least part of the increased incidence of food-borne botulism in Alaska [7].

On the other hand, discouraging native feeding habits to avoid botulism hazards may adversely affect the popularity of Alaska’s traditional dishes, which may have a negative effect on the population’s health. A logical solution to the Arctic’s botulism problems would be to lead public health education efforts towards an appropriate understanding of the dangers involved, and of safe practices of food preparation.

### Concluding Remarks

To reduce food-borne botulism’s probability, it is important to take into account that *C. botulinum*’s development and growth occurs only under particular conditions. Prevention may be emphasized by establishing several spore germination inhibitors, such as acidification and reduction of water content in addition to assuring correct sterilisation of every commercially canned food product [4]. Spore germination is prevented with a low pH ( $< 4.5$ ),

refrigeration ( $< 4^\circ\text{C}$ ), low water activity ( $a_w=0.93$  or less) and aerobic atmosphere. Spores are destroyed by pressurized cooking at  $120^\circ\text{C}$  for 30 minutes, while the toxins are inactivated at  $80^\circ\text{C}$  temperature for 20 minutes or  $90^\circ\text{C}$  for 10 minutes [3]. Certainly, the consumer, as well as the seller or handler of food products, may control pathogen growth by restricting one or more of the conditions needed for their development.

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