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Review article

Documented outbreaks of botulism: the impact of foodborne transmission

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Abstract

Botulism is a severe neuroparalytic disease caused by Clostridium botulinum's toxins. Although the disease is uncommon, causes great concern due to its high rate of mortality; foodborne outbreaks of botulism occur worldwide and require immediate public health and acute care resources. This study had a review of outstanding outbreaks published; Journals articles related to the subject. From the outbreaks analysis we found the most involved food products were: fermented fish products in Alaska; home canning food, oil preservation and restaurant sauce in London and USA; home canned vegetables, food airtight packed with inappropriate refrigeration and aerosols in Argentina. In conclusion, the diagnosis is based only on clinical findings matching the disease and previous exposure to suspicious food. Botulism must be immediately identified as one case suggests an epidemic and should be treated as a public health emergency. Therefore the purpose of the following review is to recognize the associated risks with the consumption of potentially dangerous foods, to help work our way on prevention for every public health professional to be aware of the dangers of this potentially lethal disease.

Keywords: foodborne; botulism; Clostridium botulinum

INTRODUCTION

Botulism is a paralytic illness caused by neurotoxins synthesized by spore forming anaerobic bacterium named Clostridium 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 toxins, the most powerful poisons ever known. Consuming food containing the neurotoxin produces botulism by C. botulinum. Its most frequent cause is by ingesting home canning foods, which are stored inappropriately, creating an anaerobic environment for the spores to thrive, then 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 decrease saliva secretion with dryness of buccal mucosa. The first most common symptoms are blurry vision, dysphagia, dysarthria, dysphoria and descending symmetrical flaccid paralysis. Eventhough ptosis and dysarthria may be confused with encephalopathy signs, patients are totally conscious and afebrile. The incubation period lasts 12 to 36 hours, after the toxin ingestion; generally neurological symptoms are preceded by nausea, vomiting, abdominal pain and diarrhoea. Healing process takes weeks to months, depending on the seriousness of the case. During which it's most important a close support of intensive care unit, with mechanical ventilation if needed and equine trivalent antitoxin treatment. If the antitoxin is given on time, it may prevent worsening of paralysis and reduce the length of

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the disease[2].

The severity of the symptoms depends on the amount of toxin ingested by the patient. When slight botulism, it may not need medical attention at all, that is why when a patient shows only nausea or slight neurological symptoms, there is a possibility that the case may not be diagnosed, and along with it to underestimate the real number of cases occurred.

RESULTS

Incidents of botulism and risk factors

Botulism is caused by consumption of contaminated food with C. botulinum´s neurotoxin, whose spores are everywhere in the environment. The correct food storage requires eliminating the conditions needed for the pathogen microorganisms to thrive, and to prevent recontamination. To support growth and produce toxins, C. botulinum needs an anaerobic environment, nutrients, a suitable temperature, enough water availability and adequate acidity, free of growth inhibitors.

C. botulinum´s growth in food products may be prevented by: acidity control (pH), water activity control (aW), refrigeration and chemical preservatives. To obtain a safe product it has to be preserved in an acid environment (pH 4,6 or less), where the production of the lethal toxin is inhibited. Food with pH above 4,6 are low acidity products, therefore, a dangerous risk to catch botulism.

The aW may be determined by an electrical hygrometer that measures water available for microbial growth; values beneath 0,93 stop C. botulinum´s growth. Most of moist foods such as fresh meat, fruits and vegetables have high Aw values, and therefore require refrigeration (less than 4° C) to inhibit the toxin production by C. botulinum. One way to reduce the aW (aW is by food dehydration, through sprinkling or freezing. Another way is by adding sugar or salt to capture water molecules until osmotic equilibrium is reached. A low aW is ensured by any of these means.

Packing in airtight recipients 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, although home canned foods represent the main cause for botulism, not only of sporadic cases but also outbreaks, included the ones occurred in restaurants[4]. Traditional food from Alaska, especially fermented foods such as fish, fish eggs, seal, beaver and whale, have caused large part of botulism in that area. They are made by allowing room temperature fermentation and then consumed without cooking[5].

As the distribution of large amounts of contaminated food may affect many people, there must be a constant epidemiologic surveillance established and every case of botulism should be treated as a public health emergency. In other countries botulism has been developed as a biological weapon and can be spread by intentional food contamination or through aerosols.

Botulism outbreaks epidemiology

From 1990 to 2000, 160 botulism food borne outbreaks were informed in USA. Food involved in the cases were home canned: soups, tomatoe sauce, garlic in oil, stew, potato salad, home made sausages, and less frequently meatballs, steak, liver paté, bread pudding, apple pie sauce, hamburger, chilli and peyote[4].

Also, in USA, there have been outbreaks informed, produced by food prepared at restaurants such as the sauce called "skordalia", and made of potato and cheese sauce. The "skordalia" sauce was made with potatoes rapped in aluminum foil while baked and then kept in the same rapping paper at room temperature for several days. The epidemiologic investigation of this outbreak led to identify this method as the main hazard; that must be eliminated from restaurants and homes. Other control measures depend on the education and consciousness of people working in restaurants and the efforts made by health authorities[3,4]. Other botulism outbreaks have been related with traditional recipes of salted ungutted fish, warmed in the sun and then sealed into barrels, such as the egyptian "fesaikh" and the "kapchunka", the "mohola", amongst others. The environment was clearly sufficient to allow germination and grow of C. botulinum type E, probably in the fish viscera[5,6].

From 1990 till 2000, a total of 91 botulism cases occurred in Alaska. Artic explorers and whale men have described the death of entire native fami-

lies that ate whale meat. Even though the mortality rate for botulism in natives has decreased in the last decades[7,8] the incidence of botulism in Alaska is amongst the highest of the world, and every case has been associated with consumption of traditional native foods, included aquatic fermented mammals such as muktuk (pink fat under the whale's skin), beaver tail, flipper and seal oil. Others were caused by fish like fermented salmon head and white fish; or fermented fish eggs. The 88% of the cases were due to type E toxin, which is exclusively related to aquatic mammal's ingestion[9].

McLaughlin J., described a type E botulism outbreak associated with ingestion of beached whales. On July 12th of 2002, two Yupik village residents, on 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 distributed to friends and family. Between the 13th and 15th of july, a total of 14 people ate the raw muktuk. On July 17th, a physician from west Alaska informed 3 suspicious cases of botulism in that village; all of the patients had eaten the muktuk. Out of 14 people identified as having ate muktuk, 8 had the disease. All of the seven-muktuk samples taken tested positive for type E botulinic toxin[10].

According to McLauchlin & Grant, in the United Kingdom, the following botulism outbreaks have been identified along the last 8 decades: the first outbreak informed was in 1922 with 8 cases, all fatal, due to commercial duck paté; followed by 2 cases with 1 death by rabbit and pigeon broth; 1 because of jugged hare; 5 with 4 deaths due to vegetarian nut brawn; 1 fatal case by minced meat pie; 1 fatality out of 5 cases caused by macaroni cheese; 2 more incidents caused by pickled fish; 4 cases associated with tinned salmon imported from USA (the can was contaminated from the factory environment) causing 2 deaths; another single case from 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 UK, with 27 cases and only 1 death occurred in 1989, caused by the consumption of commercial hazelnut yogurt, an unusual vehicle for C. botulinum's growth because of its low acidity. 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 by sausages, were associated with imported products originally from Italy and Poland respectively. The first one caused 2 cases and 1 death, and in 2002 one fatal case of botulism[11].

Botulism is an important public health problem in Argentina, known since 1922, when the first outbreak was informed in Mendoza, caused by home canned asparagus. Botulism outbreaks in our country have been mainly due to eating meat and vegetables wrongly packed and home canned food. Food involved in the outbreaks included ham, red and green chili peppers, "vizcacha" (rodent specie: Lagostomus maximus), eggplant, cucumbers, inside of palm tree, tomatoes, peaches, spinach, cheese with onions, pickled octopus, asparagus, canned fish, home canning and sweet corn[12]. From 1992 to 2004, 41 botulism food borne cases were informed, and in every one of them the cause was due to storing food under conditions that benefit the toxin´s growth12. One of the most important outbreaks occurred in January of 1998 in Buenos Aires and affected several bus drivers. Out of 11 people who consumed the food[9].

developed the disease. 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 y 80° C for 4 hours, then packed, sealed in a plastic bag and wrongly refrigerated, which led the C. botulinum bacteria to thrive and produce the neurotoxins that cause the illness[13].

Infants botulism (< 1 year) are susceptible to botulism through ingesting spores of C. botulimun, which then germinate, multiply, and produce the neurotoxin in the in the bowel. The bacterial spores, widely distributed in nature, are ingested with the dummy or any other contaminated object on the ground including their own hands, and less frequently by consuming contaminated food that contain the bacterial spores (honey, maize syrup, medicinal herbs contaminated)[14].

The first child botulism case was informed in USA by Arnon, and a total sum of 1200 cases nowadays[15]. In Argentina, on 1982, the first two child botulism cases were confirmed in Mendoza[16] and Buenos Aires[17].

Until the year 2000 there were 275 diagnosed cases of child botulism in Argentina[15]. The geographical distribution by province was: Mendoza 46 (31.7%), Buenos Aires 45 (31.0%), Neuquén 16 (11.0%), San Luis 13 (9.0%), Río Negro 7 (4.8%), Chubut 4 (2.8%), La Pampa 3 (2.1%) cases same as in Córdoba and San Juan; Tierra del Fuego, Misiones, Salta, Santa Fe and Tucumán 1 case (0.7%).[18]. Mendoza is the province with more botulism cases in terms of population density. In every case the neurotoxin involved was type A, the predominant type in Argentina. Age is an outstanding feature in-patients: that goes from 2 weeks to 6 months old, with a 10 week average. Nevertheless, there have cases published of a patient 8 months old and another one 10 days old18. It is worth mentioning, that in Mendoza province most of the cases were breast fed lactating children, who lived in rural areas. These areas are typically of few precipitation and strong winds that provide a bigger load and spreading of the spores in the atmosphere, and therefore, a larger probability for the spores to access the digestive tract.

There have been attempts to explain botulism transmission by food in lactating infants, but spores were only identified in honey and maize syrup. In our country, 227 honey samples were analyzed and in 8 of them (3.5%) spores were found. Other sources for child botulism in Argentina are home made infusions. Of 9 cases occurred in San Luis, 7 had taken herbal infusions. A study of medicinal plants in our country, found in 4 out of 100 samples C. botulinum type A, it was identified in poleo, anise, sen and other "medicinal herbs" contaminated[15]. Recently, child botulism was related to sudden infant death syndrome[12].

DISCUSSION

In previous decades, conditions to support the survival of C. botulinum's spores and their development in food were less likely to happen. However, due to home canning and processing of food, new packing of commercial food products, new nutritious preferences or new cooking techniques in family foods that allow C. botulinum's growth, seem to predict that food borne botulism will continue to affect humans in the future[1].

Food borne botulism in Alaska's natives is almost an old problem that has worsened in recent decades by unsafe altering traditional processing, when including plastic or glass packaging for fermentation[9].

In United Kingdom, the outbreaks associated with commercially prepared products all had evidence of poor handling and processing[11]. C. botulinum is uncommon to cause illness because the conditions required for it to thrive and produce the toxin, such as -pH > 4.6 (low acidity), high aW, absence of chemical preservatives, room temperature and anaerobic environment, are not likely to occur in food products[1]. Some home made food products, involved in outbreaks had low amounts of salt, no chemical preservatives, and were airtight bottled, with no barrier to prevent C. botulinum's spores to germinate. Such kind of products are thought to be "more healthy" by general public.

Type E toxin is responsible for more than 85% of botulism cases in Alaska, as many traditional local dishes, such as salmon head, whale fat, seal oil and skin, and fish eggs, are made 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 increase of food borne botulism incidence in Alaska[7].

On the other hand, discouraging natives feeding habits for botulism hazard may lead to a lack of culture and eventually, diminish popularity on Alaska's traditional dishes, which would have a negative effect on the population's health. A logical solution to Arctic's botulism problem would be to lead public health educational efforts towards an appropriate understanding of the dangers involved, which may help to reduce them by supporting important sanitary precautions.

To reduce food borne botulism's probability it's important to take into account that C. botulinum's development and growth occurs only under particular conditions that include an anaerobic environment, low in salts and acids and room temperature. Prevention may be emphasized by establishing several spore germination inhibitors, such as acidification and reducing the water activity, in every commercial food canned product with low oxygen level[4]. Spore germination is prevented with a low pH (< 4.5), refrigeration (< 4 ℃), low water activity (aw = 0.93 or less) and aerobic atmosphere. Spores are de-

stroyed by pressurized cooking at 120° C for 30 minutes, while the toxins are inactivated at 80℃ temperature for 20 minutes or 90℃ for 10 minutes[3]. Certainly, the consumer, the handler or seller of food products, may control pathogens growth by restricting one or more of the conditions needed for their development.

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