PUBLIC HEALTH

Botulism in Canada

Epidemiology: Last November Heinz Canada voluntarily recalled one of its canned food products because some cans may not have been processed adequately to prevent contamination from *Clostridium botulinum*. A disaster may have been averted by these actions, although this fact probably did not register with most of us. Thanks to intensive surveillance and safe food-production methods, cases of botulism are so rare in Canada — 18 cases were reported in 1997[1] — that we tend to forget that the spores of *C. botulinum* are ubiquitous and that its neurotoxin is the most potent lethal substance known.[2,3] The toxin has been estimated to cause death in humans in doses as small as 0.05–0.1 μg[4] and is 15 000 to 100 000 times more toxic than sarin, the organophosphate nerve agent used in the infamous terrorist attack in Tokyo's subway system.[3]

C. botulinum is an anaerobic, gram-positive, spore-forming bacilli commonly found in soils throughout the world. Conditions of low acidity (pH > 4.5), low oxygen and high water content favour spore germination and toxin production. Seven serologically distinct neurotoxins have been identified. Most human cases of botulism are caused by types A, B and E.[3,4] The toxins bind irreversibly to presynaptic nerve endings, inhibiting acetylcholine release in the parasympathetic and sympathetic systems and at the neuromuscular junction.

There are 3 main clinical forms of botulism: foodborne, intestinal and wound related. Foodborne botulism occurs when food contaminated by the preformed neurotoxin is eaten. The most frequent source in Canada is home-prepared products such as canned foods, fermented Inuit food and improperly stored marine meat. In these cases, spores that survive an inadequate canning or refrigeration process germinate, reproduce and produce toxin. Intestinal botulism occurs when spores are ingested and germinate in the gastrointestinal tract. Infants, whose gastrointestinal tracts lack the protective bacterial flora and *Clostridium*-inhibiting bile acids found in the adult gut, are more susceptible to colonization, as are adults with abnormal gut flora, such as those with a recent history of bowel surgery or antibiotic use. The source of ingestion is unknown in about 85% of cases of infant botulism; in up to 15% of cases honey is the suspected agent.[3] Wound-related botulism occurs when anaerobic conditions within an abscess allow germination of *C. botulinum* spores. Most of these cases originate in wounds contaminated with soil or at injection sites in intravenous drug users.[4]

Clinical management: Suspect botulism in a patient with acute onset of gastrointestinal, autonomic and cranial nerve dysfunction. The sensory system and mentation are not affected. Signs and symptoms may range from subtle motor weakness or cranial nerve palsies to rapid respiratory arrest. Symmetrical, descending paralysis beginning with the cranial nerves and advancing to the upper extremities, the respiratory muscles and the lower extremities is characteristic. Constipation, a weak cry, difficulty feeding and weakening bulbar and limb muscles may signal infant botulism.

The local public health unit or a member of Health Canada's Botulism Reference Service[5] should be contacted immediately when a case is suspected. A single case of foodborne botulism represents a public health emergency and may herald the beginning of a larger outbreak. Laboratory proof of botulism is established with detection of the toxin in the patient's serum, stool or wound or through culture of *C. botulinum* from gastric aspirate, the wound or stool. However, because of the importance of early detection, botulism must first be diagnosed on the basis of the history and physical findings. Normal findings on lumbar puncture and brain-imaging studies in the presence of signs and symptoms compatible with botulism should raise the suspicion for botulism and prompt close monitoring for respiratory failure. Electrophysiologic abnormalities such as a small evoked muscle action potential in response to a single supramaximal nerve stimulus in a clinically affected muscle can provide presumptive evidence of botulism.[4]

The main treatment for severe botulism is meticulous supportive therapy, which may include mechanical ventilation. An equine polyvalent antitoxin, available through Health Canada's Special Access Program (see the lavender section of the *Compendium of Pharmaceuticals and Specialties*[6]), is recommended for cases of foodborne or wound-related botulism. To be most effective, the antitoxin must be given before much toxin has bound to presynaptic nerve endings.[3] In cases of wound-related botulism, the wound must be debrided and therapy with an appropriate antibiotic such as penicillin[7] started. Antitoxin is not indicated in cases of infant intestinal botulism; recommendations are conflicting in cases of adult intestinal botulism because of concerns about the high rate of hypersensitivity reactions (9%) to the equine antiserum.[3,7] With critical care management, the death rate is about 14%.[5] Recovery takes weeks to months and occurs when new presynaptic end plates and neuromuscular junctions are formed.

Prevention: *C. botulinum* spores are resistant to heat and may survive the home-canning process at temperatures below 120ºC. In contrast, the toxin is heat labile. Boiling food for 10 minutes to ensure thorough heating should destroy the toxin. Honey should not be fed to children during the first year of life.[4,7,8]

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References

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