Are We Recognizing Most Clinical Botulism?

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Botulism is an uncommon disease resulting from the ingestion of a preformed protein neurotoxin secreted by *Clostridium botulinum*. Six antigenic types are known, four (ABEF) causing disease in man, and two (CD) in animals.

Although the derivation of the word "botulism" comes from the Latin *botulus*, meaning sausage, most cases occurring in the U. S. during the past 70 years have been associated with vegetables or fish rather than sausage or meat.

Types A and B botulism are usually associated with home preserved vegetables or fruit, while type E botulism occurs almost exclusively with fish or marine products.

Only one outbreak of type F botulism has been reported in the U. S. and was associated with home processed venison jerky.¹

In addition to the association of specific foods for different types of botulism, types A, B, and E show a unique geographic distribution in the U. S. Ninety-one percent of all type A botulism reported between the years 1899 and 1967 occurred west of the Mississippi River, while 23 of 34 reported outbreaks of type B botulism were found in the Eastern States.

Outbreaks of type E botulism occurred most commonly in Alaska and the Great Lakes region. Predilection of geographic areas for different types of botulism is supported by soil surveys, which show a predominance of type A spores in the Western U. S. and type B spores in the Northeast and Central States.²

Spores of type E *Clostridium botulinum* have been demonstrated along Atlantic, Gulf and Pacific Coasts of North America and the Great Lakes, with the highest spore concentrations in the shallow, off-shore fishing areas, particularly near the mouths of rivers.³,⁶

Surveys of fish caught in the Great Lakes showed that 9% of all fish contained spores of type E *C. botulinum*, with a high of 56% of the fish in Green Bay containing the organism.⁷ Most commercially available fish and shellfish have been shown to contain spores from type E *C. botulinum*.

Processing of the fish may spread contamination from the intestines of a few to many other fish. However, since type E *C. botulinum* spores are relatively more heat-labile than other *C. botulinum* types, smoking or light cooking may be sufficient to inactivate most spores.

Despite this sensitivity to heat, 1% of freshly smoked fish from Lake Michigan were found to have detectable *C. botulinum*.⁸

Since type E *C. botulinum* may germinate and produce toxin at refrigerator temperatures, lethal amounts of toxin can accumulate when contaminated fish are held at low temperatures under anaerobic conditions.⁹

Symptoms from clinical botulism developed directly from interruption of neuromuscular transmission, first apparent in the cranial nerves, and later progressing to other somatic muscles with a symmetrical, descending weakness or paralysis.

Because the toxin has a specific effect only at the neuromuscular junction, there are usually no abnormalities in the sensory system or in mental abilities.

The most common symptoms found in 145 cases were blurred vision, photophobia or diplopia, dysphagia, nausea and vomiting with dysphonia and generalized weakness.

Respiratory impairment was by far the most common sign in this series, followed by eye muscle weakness.
involvement, dilated fixed pupils, dry mouth and specific muscle weakness or paralysis.\(^1\)

In some outbreaks of type B and particularly in type E botulism, the first symptoms of the onset of the disease were related to the gastrointestinal tract, with nausea, severe vomiting and subternal burning. These symptoms were so marked in several outbreaks that cranial nerve involvement was not recognized and the patients were considered to have high intestinal obstruction.\(^1,11-12\)

Perhaps more significant, the presence of hypotension without shock, epigastric distress and tachycardia initially suggested the true poisoning may be under-reported. Fortunately, the toxin is inactivated if heated to 80°C for 30 minutes.

The diagnosis of botulism is established by demonstrating the toxin in the patient’s serum, the isolation of the organism, or demonstration of the toxin in a food ingested by the patient.

Isolation of the organism may be difficult since considerable variation exists in cultural characteristics of the different types. The most specific method to prove botulism is demonstration of the toxin in the patient’s serum. This is done by injecting the suspect’s serum into susceptible mice. If the mice die, aliquots of the serum are mixed with antiserum to the four human types in protection tests.\(^13\)

Although toxin has been found in serum up to ten days following ingestion of contaminated food, serum samples taken early in the illness give the most reliable results.

The treatment of botulism is specific antitoxin. Antitoxin must be started on suspicion, since confirmation by laboratory tests is usually too late.

The results of antitoxin therapy vary with the antigenetic type of the disease, being most effective with type E, and least effective with type A.

Trivalent A, B, and C antitoxin is available from the National Communicable Disease Center (NCDC) and should be used when there is suspicion of botulism of unknown type. The NCDC also distributes monovalent type E antitoxin.

Lederle Laboratories produces a bivalent A and B antiserum which is also available through the NCDC. The only available type F antiserum is made by the State Serum Institute of Denmark; and is distributed through the NCDC as a polyvalent antiserum containing ABEF.

Help, in the form of antiserum and laboratory assistance to establish the diagnosis of botulism, is available at all hours from the NCDC in Atlanta, Georgia (telephone 404-633-3311).

For a more complete list of telephone numbers for emergency assistance and an excellent summary of the clinical experience in the U. S. between 1899-1967, refer to reference 1, available without charge from the NCDC.

References

5. Eklund, M. W. and Pysky, F. T. Distribution of Clostridium botulinum on the Pacific Coast of the United States, Conference on Toxic Micro-organisms sponsored by the Joint United States-Japan Cooperation on Development and Utilization of Natural Resources (USNR), Honolulu, Hawaii, October, 1966 to be published.