

BOTULISM *by Respiratory Care Student Lorna D. Ringley*



Don't give that baby honey, and when you ask, well why not, the answer is simply, because it will make him so sick that he'll have to get put on a breathing machine. This frank response, though not being to your satisfaction, is taken in stride because your mother, having more experience, knows best and her counsel needs no longwinded explanation and should never be questioned. Well being the inquisitive, Internet savvy person you are, as soon as mother ends her visit and the baby is down for the night, ok at least for the next four hours, you immediately run to your computer and Google "avoid giving baby honey". This leads you to Google "Botulism". When 1,900,012 results come up, being so determined, you prepare for a long night of blurred vision, throbbing eyes and backaches and click on the first result and this is what you find.

Definition

Botulism is commonly associated with an infant ingesting honey. It is not so much an infection but an intoxication of the body that results from a potent exotoxin that is released by *Clostridium Botulinum*. This binds irreversibly to the presynaptic membrane blocking the release of acetylcholine across the myoneural junction resulting in paralysis of cranial and peripheral nerves.

There are three types of botulism; food borne, wound, and infant botulism. Food borne is caused by ingesting inadequately prepared food low in acid. Home-canned fruits and vegetables, sausages and smoked or preserved meats have been associated with this type of botulism. Wound botulism is less common and occurs when a wound is infected with the bacteria *Clostridium botulinum*, with injection of black tar heroin being the most common and recent implication. An important distinction between these two is that food borne is limited to the quantity of toxin consumed and wound botulism will be produced continually until the infection is eliminated. Infant botulism is normally attributed to the ingestion of honey or corn syrup and results from the spores of the botulinum which then grow in the intestines and release their toxin.

Signs and symptoms

Signs and symptoms may vary slightly between the three types but the cardinal sign is acute symmetrical cranial nerve impairment. The patient may present with ptosis (drooping eyelid), diplopia (double vision) or dysarthria (difficulty speaking), followed by descending weakness or paralysis of muscles in the extremities or trunk and respiratory distress from diaphragmatic paralysis. Food borne and wound botulism symptoms may also include blurred vision, dysphagia, dry mouth, sore throat, weakness, dizziness, vomiting and diarrhea. These symptoms usually appear between 12-36 hours after consumption of contaminated food, though onset can range from 6 hours-10 days. Infants who acquire botulism usually do so between 3-20 weeks of life. Signs of botulism intoxication include; flaccid facial expression, ophthalmoplegia (paralysis of one or more muscles of the eye), lethar-

gy, hypotonic (floppy) infant syndrome, constipation, a feeble cry, depressed gag reflex, areflexia (absence of reflexes) and the inability to suck.

Population most affected

Out of an average of 110 cases of botulism a year in the United States, the 25% attributed to food borne and the 75% to infant honey ingestion has not changed much. The once rare occurrence of wound botulism is on the rise with the increase in subcutaneous and intravenous black tar heroin use. Mortality from botulism has decreased from 50% to 8% in the past 50 years, likely a consequence of education on proper home canning techniques and advances in medicine. Patients who have survived botulism may fatigue easily for years after the event and may need to continue long term therapy to aid in their recovery.

Diagnosis

The most direct way of diagnosing is by mouse inoculation test. The patients' stool, gastric content, contaminated food or blood serum is injected into the mouse and signs of botulism are assessed. The bacteria itself may also be isolated in the stool of patients with food borne or infant botulism. It is important to rule out other diseases/ syndromes such as; Guillain-Barré, myasthenia gravis, stroke, staphylococcal food poisoning, tick paralysis, chemical intoxication, carbon monoxide poisoning, fish poisoning, trichinosis and diphtheria, as they are commonly confused with botulism. The use of brain scans, a spinal fluid test, a Tensilon test

and an intramuscular electromyogram (EMG), a test of nerve and muscle function will be helpful in excluding other diagnoses.

Treatment

When the diagnosis of Botulism has been made it is important to admit the patient into the intensive care unit (ICU) so the course of their disease can be followed closely. Vital capacities are routinely measured by the respiratory therapist to keep track of severity of respiratory muscle paralysis. Deep breathing exercises, with the use of incentive spirometry, are encouraged. If ingestion has occurred within several hours, vomiting is induced. Gastric lavage can also be done and a high enema given to purge any unabsorbed toxin from the bowel. When dysphagia sets in fluids are administered through an IV or a nasogastric tube is placed. If the diagnosis is made quickly, botulinum antitoxin can be administered to food borne or wound botulism patients to block the action of the toxin circulating in the blood and prevent the disease from worsening. Although the antitoxin, if given early, shortens hospital stays and reduces the number of days on a ventilator, recovery can take several weeks to months. The antitoxin has not yet been approved for use in infant botulism. Treatment of a patient with botulism, when respiratory failure has set in, is largely supportive with mechanical ventilation managed

Miss Lorna Ringley is a Senior Respiratory Care Student at the University of Texas Health Science Center in San Antonio, Texas. Her paper on Botulism was chosen from 14 papers on various topics submitted to Focus for this issue. Miss Ringley will receive a \$100 gift certificate and a gratis registration to the 2008 Focus Conference. Her school's RC Program will also receive a \$100 donation. Students are encouraged to submit their papers for the May/June issue by May 5th. Papers should be between 900 and 1250 words and should be submitted as MS Word files to our Craig Baker at BakerCT78@yahoo.com.

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Reference Values:

Adult: Total: 0.1-1.2 mg/dl, 1.7-20.5 micromol/l (SI units).
Direct (conjugated): 0.1-0.3 mg/dl, 1.7-5.1 micromol/l (SI units).
Child: Newborn: Total: 1-12 mg/dl, panic level is > 15 mg/dl, 17.1-205 micro-moles/l (SI units). Child: 0.2-0.8 mg/dl.

Direct or conjugated bilirubin is frequently the result of obstructive jaundice, either extrahepatic (from stones or tumor) or intrahepatic in origin. Conjugated bilirubin cannot escape in the bile into the intestine and thus backs up and is absorbed into the blood stream. Damaged liver cells cause a blockage of the bile sinusoid, increasing the serum level of direct bilirubin. With hepatitis and decompensated cirrhosis, both direct and indirect bilirubin may be elevated.

How do you know if your patient may have a bilirubin problem? The most obvious is jaundice, which appears as yellowing of the skin and sclera (white part of the eye). Other conditions may cause yellowing or darkening of the skin (e.g., carotinemia, Addison's disease, quinacrine ingestion), but in these conditions scleral and mucosal discoloration are absent. The most important initial step is to define whether the jaundice is predominately due to an elevation of unconjugated or of conjugated bilirubin. If jaundice is primarily due to unconjugated bilirubin, evaluation for hemolysis is appropriate. In patients with elevated conjugated bilirubin, the clinical challenge lies in distinguishing whether biliary obstruction, impaired hepatic excretion, or hepatocellular injury is the cause. Many drugs, from antibiotics to vitamins, may also have an effect on bilirubin values.

Finally, some interesting factors that can affect laboratory results. A high-fat dinner prior to testing may affect bilirubin levels. Carrots and yams may increase the serum bilirubin level (with even a jaundice-type appearance in the patient)! Hemolysis of the blood sample can give inaccurate results. The tube should not be shaken! Most of all, if your patient is jaundiced-check the chart-again!

Don Steinert is an Associate Professor in the Department of Nursing and a faculty member in the Respiratory Therapy Program at the University of the District of Columbia.

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by physicians and respiratory therapists. When wound botulism the source of the infection, the site must be cleared to prevent continuing production of the toxin.

Discussion and conclusion

While researching this disease I have once again come to respect even more the physicians who have to diagnose patients with one or more of the thousands upon thousands of possible diseases. I understand how difficult it must be to get a definitive diagnosis for a disease when tests are deemed inconclusive, symptoms are very general or end up not being characteristic of the disease at all. That is why it is even more important for respiratory therapists to maintain communication with physicians and other medical staff so that we can share our knowledge of respiratory diseases, respiratory therapy and ventilator management.