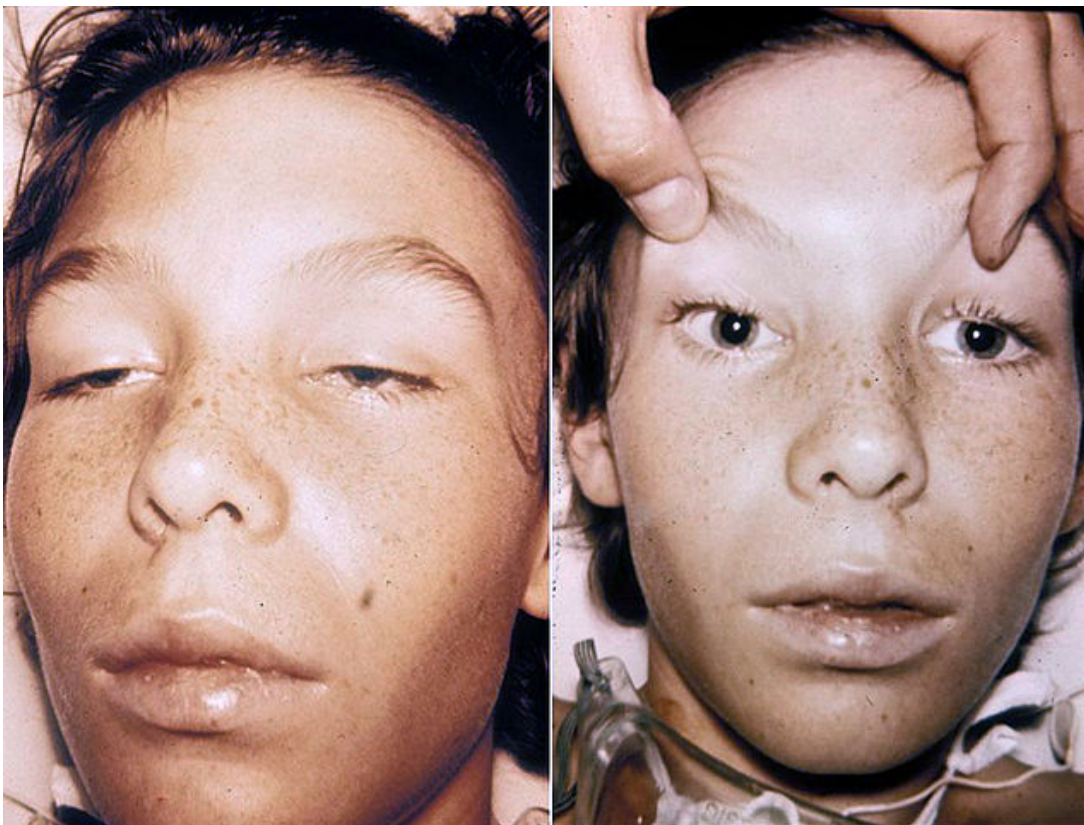


Infant Botulism — Diagnosis and Treatment

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Botulism is a neuromuscular paralysis secondary to a neurotoxin secreted from *Clostridium botulinum* bacteria. It is a rare syndrome that results from eating food that has been contaminated with the spore-forming *C. botulinum* toxin. The spores germinate and colonize the infant's intestine and secrete the neurotoxin. Other forms of botulism include wound botulism, where the toxin is secreted in infected wounds; inhalation botulism, seen in, e.g., cases of biological warfare; and iatrogenic botulism, which occasionally occurs in people using botulinum toxin for cosmetic purposes.



Introduction

Botulism is a disease of neuromuscular paralysis, secondary to a neurotoxin secreted by the *C. botulinum* bacteria. Infant botulism is the most common form of botulism worldwide. Other forms of botulism that affect older age groups include:

- Wound botulism, which is common among military personnel. Inoculation of the spores deep in the tissues creates an excellent environment for germination and toxin secretion. Polymicrobial bacterial infection can also be present in wound botulism. Wound debridement, antitoxin therapy, antibiotics,

and tetanus toxoid are recommended for management of this form of botulism.

- Other foodborne botulism, which can occur after eating home-canned foods containing bacterial toxins.

Epidemiology

Although infant botulism is more common in children between the ages of 2 and 8 months, it can occur any time up to the age of 1. It is a rare disease that affects up to 100 children annually in the United States.

Etiology & Risk factors

The disease arises from **neurotoxin secreted from *Clostridium Botulinum* bacteria that is acquired through various mechanisms such as:**

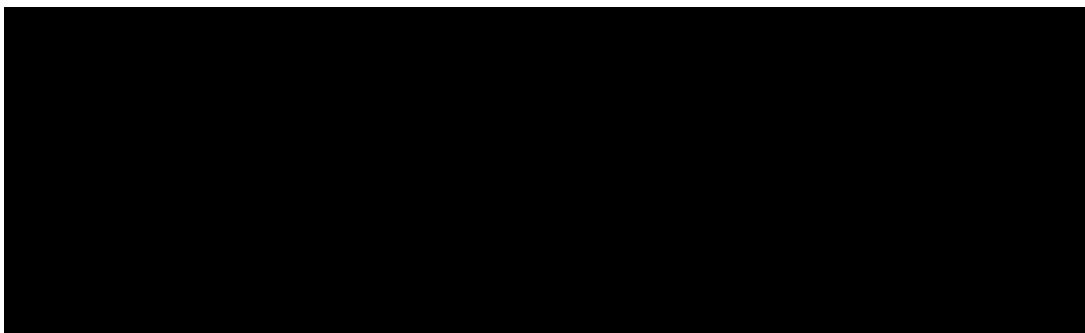
1. Ingestion of honey. Honey is a source of bacterial spores that germinate in the infant's alimentary tract and begin producing the neurotoxin in vivo.
2. Ingestion of dust containing toxin-producing bacteria spores.
3. The introduction of milk formula and solid foods increase infants' vulnerability to the bacteria.

Microbiology of Botulism

C. botulinum is a group of gram-positive anaerobic bacilli that can form spores during unfavorable environmental conditions. The spores are heat resistant up to 100°C for hours, and therefore can be easily transmitted via food. With low acidity, oxygen, and appropriate temperatures, the spores germinate into a toxin-producing bacteria.

The bacteria are commonly isolated from the soil and can be found on the surfaces of vegetables and fruits. The toxin produced by *C. botulinum* is the most potent toxin known; a very low level of the toxin can be lethal. It is a heat-labile protein that can be easily denatured by temperatures above 80°C. However, it can resist the gastric acidity and proteolytic enzymes of the gut. It is easily absorbed through the stomach or the intestinal mucosa to the bloodstream.

The botulinum toxin affects the presynaptic cholinergic transmission of sensory, motor, and autonomic nerve fibers, leading to neuromuscular paralysis. It reaches the central nervous system through systemic spread or axonal transport and can inhibit the release of dopamine, serotonin, somatostatin, gamma-aminobutyric acid, and noradrenaline from presynaptic neurons.



Clinical Picture of Botulism

The disease has an incubation period of between 3 and 30 days, after which patients present with **symmetric descending weakness with bilateral cranial neuropathies without fever or mental dysfunction**. Weakened muscles supplied by cranial nerves followed by weakness of the limbs and diaphragm are characteristic.

Some cases have gastrointestinal symptoms as the most predominant symptoms. In infants younger than 12 months of age, colonization of the intestine with the bacteria presents early on with **constipation**; this may be the first sign. This is followed by progressive **hypotonia and loss of deep tendon reflexes**. Cranial neuropathies present with a **weak cry, poor sucking and feeding, drooling, ptosis, and pupillary paralysis**. Autonomic dysfunction may be present with **dry mouth, dry eye, and fluctuating heart rate and blood pressure**.

Ingestion of the preformed toxin in foodborne botulism presents first after a few hours with:

- Nausea, vomiting, diarrhea, dry mouth, and pain
- Symmetric descending weakness of the trunk and limbs
- Blurring of vision, diplopia, and ptosis (these neurological manifestations are due to the involvement of the cranial nerves III, IV, and VI)
- Other cranial nerve involvement, which leads to dysarthria, dysphagia, and facial palsy
- Diaphragmatic paralysis; this is a common symptom, and patients eventually need intubation and mechanical ventilation
- Urine retention; this is also a common symptom and results from smooth muscle paralysis

Differential Diagnosis of Botulism

Other forms of neuromuscular paralysis include Guillain-Barré syndrome, tick paralysis, myasthenia gravis, poliomyelitis, antibiotic-associated paralysis, and Lambert-Eaton myasthenic syndrome (LEMS).

Infant botulism should be also differentiated from metabolic encephalopathy, brainstem encephalitis, sepsis, spinal muscular atrophy type 1, neonatal myasthenia gravis, and dehydration.

Diagnosis of Botulism

Clinical suspicion is important, as early administration of antitoxin therapy is lifesaving while waiting for culture results and confirmatory tests. Detection of the botulinum toxin in the serum of suspected individuals is diagnostic.

The toxin can also be isolated in stool, vomit, food remnants. Repetitive nerve stimulation and electromyography can be used to differentiate between myasthenia gravis, LEMS, and botulism especially wound botulism. Cerebrospinal fluid is usually normal.

Infant botulism: the spores and the toxin can be isolated from the stool of infected infants but not in the serum. The diagnosis usually takes a few days until the results of the culture are positive or the toxin is detected in stool samples.

Management of Botulism

Patients with suspected botulism should be monitored closely in the hospital or even the intensive care unit to ensure adequate respiration and oxygenation. Many patients will need intubation and mechanical ventilation to prevent respiratory failure and airway compromise. Adequate feeding should be continued for prolonged respiratory support with a nasogastric tube or even parenteral nutrition.

Antitoxin therapy should be administered to all patients based on clinical suspicion of botulism without waiting for confirmatory tests. This therapy is proven to decrease mortality rates for patients diagnosed with botulism even after a long period of symptoms. Patients presenting with a febrile descending paralysis and cranial neuropathies should also be evaluated for botulism.

There are two types of antitoxin therapy: equine serum heptavalent botulism antitoxin and human-derived botulism immunoglobulin. Equine serum heptavalent botulism antitoxin therapy includes antibodies against 7 types of botulinum toxins. It is mainly administered to adults and children older than 1 year of age. A hypersensitivity reaction with anaphylaxis and serum sickness can result from the equine serum antitoxin; thus, a skin test, desensitization, and proper dosing are important prior to therapy. Other adverse effects include rash, nausea, fever, headache, and chills.

Human-derived botulinum immunoglobulin therapy is used for infants less than 12 months of age.

Antibiotics can be given for patients with wound botulism due to infection, but they are contraindicated in patients with infant botulism or foodborne botulism. Antibiotic-mediated lysis of the *Clostridium* bacteria can increase the toxin amount and its absorption in the circulation.

Prevention of Botulism

Prevention of foodborne botulism depends mainly on the proper canning of food. Boiling of food before consumption is sufficient to denature the heat-labile toxin. For infant botulism, prevention is mainly achieved through the avoidance of honey in infants younger than the age of 1. Early treatment is mandatory in infants with no history of honey consumption.

Avoidance of exposure to dust in areas that have high toxin content in the soil has also been shown to reduce the occurrence of the disease.

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