CLINICAL VIGNETTE

Skin-Popping, Breath Stopping

Vivian Wang, M.D., and Mana Khafaf, M.D.

Introduction

Botulism is a very rare but potentially life-threatening infection due to the neurotoxin of the bacterium *Clostridium botulinum*. Botulism can occur after various modes of infection, such as ingestion of the botulinum toxin, which can cause foodborne botulism; ingestion of the clostridial spores, which subsequently produce toxin in the GI tract resulting in infant and adult botulism; and infection of a wound by the bacteria itself with subsequent production of the neurotoxin. "Skin-popping" or the act of injecting black tar heroin intramuscularly or subcutaneously rather than intravenously is a major risk factor for the development of wound botulism (WB), especially in the western United States where this drug is commonly used.[1]

Case

A 31-year-old male with a past medical history of asthma and polysubstance abuse presented to the emergency department with difficulty speaking. He also had the sensation of a foreign object in his throat, the feeling that his tongue was paralyzed, and shortness of breath. His symptoms started on the morning of presentation and increased in severity throughout the day. His initial vital signs, limited exam, and lab studies were unremarkable. CT neck showed symmetrical medial deviation of the bilateral vocal cords suggestive of vocal cord paralysis. He was scheduled for admission to the surgical intensive care unit for emergent surgical tracheostomy, but he refused admission and left against medical advice from the emergency department. He returned 3 hours later with worsening dyspnea and agreed to admission. His physical examination was notable for new bilateral ptosis, mydriasis, worsened speech, and bilateral upper extremity weakness without fatigability. He required emergent endotracheal intubation as he developed increasing oxygen requirements and continued to have worsening respiratory effort. As he was being positioned for intubation, a suspicious syringe was found underneath the covers by his left thigh. The syringe was sent for analysis, and its contents were found to be consistent with black tar heroin. A more thorough physical exam revealed large bilateral buttock wounds from intramuscular drug injection which required surgical debridement. Infectious disease advised treatment with penicillin G and antitoxin due to high suspicion for wound botulism. Type A botulinum toxin was later confirmed by the CDC lab. Electromyogram revealed pre-synaptic neuromuscular junction pathology. He underwent tracheostomy on hospital day 5. His generalized weakness and vocal cord paralysis improved steadily over the next 21 days, monitored by serial electromyograms and laryngoscopy. His tracheostomy was decannulated, and he was discharged home on hospital day 42. Seven months after discharge, he presented to the emergency department for shortness of breath thought due to his asthma and was discharged on inhaled albuterol. Unfortunately, he has no subsequent follow-up.

Discussion

Black tar heroin (BTH) was first introduced to the U.S. in the 1970s and gained popularity due to its low costs. BTH is frequently contaminated as it is diluted and "cut" with fillers such as paper, black shoe polish, and dirt to increase profit.[1] The majority of wound botulism (WB) in California since 1988 has been associated with drug injection, especially with BTH via the subcutaneous or intramuscular route.[1,2] California's WB cases account for about 75% of the cases in the U.S. Studies have found that early administration of antitoxin can potentially decrease the length of intensive unit stay and duration of mechanical ventilation. Thus, early recognition is crucial.[3]

Botulism classically presents with acute bilateral cranial neuropathies with symmetric descending weakness.[4] Other features include blurred vision, symmetric neurologic deficits, a consistently responsive patient, normal or slow heart rate with normal blood pressure, and absence of fever. The classic "5 Ds" of botulism include diplopia, dysphonia, dysarthria, dysphagia, and descending paralysis, which starts with facial muscles and results in progressive respiratory and extremity muscle weakness.[5]

Diagnostic lab studies in the U.S. are only available through the CDC and about 20 government public health laboratories. Lab testing includes identifying botulinum toxin from the patient's serum, stool, gastric contents, and wound. Serum samples must be obtained before treatment with antitoxin. There are 7 types of botulinum toxin designated with letters A through G.

Common misdiagnoses including Guillain-Barre syndrome, Myasthenia gravis, and Lambert-Eaton syndrome can be differentiated by electromyogram in addition to a thorough history. Of note, cerebrospinal fluid is unchanged in botulism but can be abnormal in many central nervous diseases. Botulism differs from the other flaccid paralyses in its lack of sensory damage as well as its significant symmetric cranial nerve palsies, which are out of proportion to milder weakness and hypotonia below the neck.[6]

Therapy for botulism includes passive immunization with botulism antitoxin, supportive care, monitoring of respiratory status in the intensive care unit, and debridement of any wounds. Early recognition will decrease further nerve damage but will not reverse existent paralysis. Patients with neurologic signs concerning for botulism should be given the antitoxin as soon as possible. Treatment should not be delayed for laboratory testing. In the U.S., botulism antitoxin is available through the CDC via local government health departments. The trivalent antitoxin contains antibodies that neutralize toxins A, B, and E, which are the most common types found in humans.[6]

Although antibiotics do not directly have an effect on botulinum toxin, Penicillin G, or Metronidazole in patients with penicillin allergy are recommended for wound botulism after antitoxin has been given.[7] Patients with WB are at risk for soft tissue infections and these secondary infections frequently require antibiotics. Aminoglycosides and clindamycin are contraindicated due to their potential to worsen neuromuscular blockade.[6]

Conclusion

WB is very rare and can be difficult to diagnose. It is important to take a thorough history and physical exam, which would be notable for symmetric descending paralysis starting with facial muscles and with gradual respiratory and extremity weakness, as well as weakness without fatigability. Prompt administration of botulinum antitoxin is crucial as it will decrease further nerve damage.

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