Wound Botulism Presenting as a Deep Neck Space Infection

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Otolaryngologists commonly evaluate patients with findings suspicious for deep space soft tissue infections of the neck. In this case, a woman with a history of injection drug use (IDU) presented with dysphagia, odynophagia, and neck pain. Multiple neck abscesses, too small to drain, were seen on imaging. Despite broad-spectrum intravenous antibiotics, she unexpectedly and rapidly developed respiratory failure requiring intubation. Further work-up diagnosed wound botulism (WB). To our knowledge, this is the first report of WB presenting as a deep neck space infection, and illustrates the importance of considering this deadly diagnosis in patients with IDU history and bulbar symptoms.

Key Words: Wound botulism, deep neck space infection, abscess.

INTRODUCTION

Wound botulism (WB) is a rare but deadly cause of soft-tissue infection. The number of cases has steadily increased with the proliferation of injection drug use, most notably the phenomenon of subcutaneously “skin popping” black tar heroin. Patients who inject intoxicants into their jugular veins commonly form neck abscesses. The early symptoms of dysphagia and dysarthria in a patient with WB can mislead the clinician into implicating the inflammatory and mass effects from a deep neck space infection rather than the true bulbar palsies due to botulinum toxin. Diagnosis and treatment with serum antitoxin and supportive care is necessary to decrease chances of serious sequelae. Herein, we present the case of a woman diagnosed with WB who presented with a deep neck space infection.

CASE REPORT

A 35-year-old woman with history of injection drug use (IDU) and multiple prior subcutaneous abscesses requiring incision and drainage presented to our emergency department (ED) with 3 to 4 days of general malaise and 2 days of dysphagia, odynophagia, and anterolateral neck pain. She confirmed intravenous neck injections in the past, but denied any in the past month. Vital signs showed no fever, hypoxemia, or hypotension. On physical examination, she was a drowsy woman with a weak voice, alert, oriented, and without respiratory distress. There were no deficits on initial exam of cranial nerves II-XII. Her neck was supple with bilateral-antrolateral tenderness to palpation. There was no induration or palpable fluctuance. Her skin exam revealed a 1-cm indurated area on the right arm and two similar areas of induration on her left upper extremity. Laboratory tests were notable for normal white blood cell count, renal function, and electrolytes. Contrast-enhanced computed tomography (CT) of the neck revealed bilateral subcentimeter, rim-enhancing, hypodense lesions at the level of the hyoid deep to the sternocleidomastoid muscles concerning for abscesses (Fig. 1). During her initial ED evaluation, she gradually developed increasing shortness of breath, muffled voice, and difficulty in swallowing her secretions. Flexible nasopharyngoscopy showed pooling of secretions, without any glottic or pharyngeal edema, normal vocal fold movement, and a widely patent airway. The patient was admitted for nonoperative management of her cervical infection and placed on vancomycin and ampicillin/sulbactam.

On hospital day 2, the patient was found apneic. She was emergently intubated for airway protection and mechanical ventilation. Given the history of opioid abuse, intravenous naloxone was administered, but had no effect. With the rapid and unexpected decline, we reevaluated our initial assessment and plan. We posited that the etiology of the presenting symptoms of dysphagia, voice weakness, and worsening dyspnea might be a neurologic disease affecting the corticobulbar tract rather than inflammation and mass effect from the abscesses. The differential diagnosis was expanded to...
WB was favored.

In-use and multiple abscesses, the working diagnosis of cerebrovascular event. With the patient’s history of hereditary syndromes, myasthenia gravis, acute poliomyelitis, and a history of drug injection, additional considerations included causes of bulbar palsies including Guillain-Barre syndrome, myasthenia gravis, acute poliomyelitis, and a history of drug injection. The patient’s history of heroin use and multiple abscesses, the working diagnosis of WB was favored.

Repeat physical examination revealed ptosis and ophthalmoplegia. A broader work-up was initiated, including lumbar puncture, CT angiography of the brain, CT angiography of the chest, echocardiogram, and blood and urine cultures. These were unrevealing for a cerebral vascular event, cardiogenic, or infectious etiology. To better characterize the palsies, surface-electrode electromyography (EMG) of the right peroneal, tibial, median, and ulnar motor nerves was performed, as well as needle-electrode EMG of the right lower limb. Results showed diffuse low amplitude baseline compound muscle action potentials, electric decrement at low frequency stimulation, and no facilitation with fast frequency stimulation. These findings are consistent with a systemic disease affecting neuromuscular signal propagation at the neuromuscular junction. Combined with this patient’s clinical presentation, WB was highly likely.

Botulinum antitoxin obtained from the Centers for Disease Control and Prevention was administered in coordination with the US Department of Public Health. In addition, her upper extremity abscesses were incised and drained, and cultures were sent.

The patient required ongoing mechanical ventilatory support for respiratory failure from severe diaphragmatic weakness. Early tracheotomy was performed. She had gradual improvement in cranial nerve palsies, as well as increasing proximal muscle weakness prior to transfer to a skilled nursing facility for continued ventilator management and rehabilitation. Mouse bioassay on the patient’s serum returned positive for botulinum toxin, definitively proving her diagnosis of WB.

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**DISCUSSION**

*Clostridium botulinum* is a ubiquitous spore-forming, Gram-positive, anaerobic bacillus in the soil. When growing, *C. botulinum* produces a toxin that is active at the neuromuscular junction presynaptic nerve terminal, where it causes essentially permanent dysfunction. Inability to release acetylcholine causes a characteristic flaccid paralysis. *C. botulinum* spores are resistant to heating of heroin mixtures typically done before injection, and thus can grow and produce toxin when in the anaerobic environment of subcutaneous and intramuscular tissue. Subcutaneous injection of black tar heroin is the leading risk factor for WB. The incidence is particularly high in California, but cases have been identified throughout the western United States.

Dysphagia and dysarthria are the two most common presenting symptoms of WB. Other symptoms and signs reflect a descending nerve paralysis involving the cranial nerves including ptosis, diplopia, dyspnea, dysphonia, and dry mouth. The disease will typically progress over several days to respiratory failure; the mortality rate is as high as 15% if not treated. The mainstays of treatment are early infusion of serum antitoxin and incision and drainage of infected wounds. Antibiotics targeting *Clostridium* are also given. Early tracheotomy is performed to decrease the risks of prolonged intubation given these patients’ long-term ventilatory requirement. Supportive care, including avoidance of infectious complications, is also necessary. Recovery ensues following regeneration of presynaptic nerve fibers over the course of months. For disease tracking, the US Department of Public Health should be alerted to any suspected case of WB.

**CONCLUSION**

In 2006, Preuss et al. reported a case of WB presenting as dysphagia in a patient with IDU history and a forearm abscess. In our case, the patient presented with similar symptoms and history, but was also found to have a deep neck space infection, which complicated the initial clinical assessment. This case illustrates the importance of considering WB in patients with a history of IDU presenting with dysphagia, dysphonia, visual changes, or proximal muscle weakness. Special attention should be given in the setting of cranial nerve neuropathies or bulbar symptoms out of proportion to clinical and radiologic findings.

**BIBLIOGRAPHY**