WOUND BOTULISM PRESENTING AS A DEEP SPACE NECK INFECTION

Christopher Gouveia1,2, Somnath Mookherjee3, Matthew Russell4

1Department of Otolaryngology- Head and Neck Surgery, Northwestern University, 2School of Medicine, University of California, San Francisco, 3Department of Medicine, University of California, San Francisco, 4Department of Otolaryngology- Head and Neck Surgery, University of California, San Francisco

ABSTRACT

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 space neck infection, and illustrates the importance of considering this deadly diagnosis in patients with IDU history and bulbar symptoms.

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 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 attributing the inflammatory and mass effects from a deep-space neck 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 space neck infection.

CASE REPORT

A thirty-five year old woman with history of IDU presented to UCSF emergency department with 3-4 days of general malaise and two days of dysphagia, odynophagia, and anterolateral neck pain. Vital signs were normal. On physical examination, she was a drowsy woman with weak voice, alert, oriented, and without respiratory distress. Cranial nerves were normal. Her neck was supple with bilateral-anteralateral tenderness to palpation, no induration or palpable fluctuance. Her skin exam revealed a 1 centimeter indurated area on the right arm and two similar areas of induration on her left upper extremity. Her labs showed normal white blood cell count, renal function, and electrolytes. Imaging was performed (Figure 1). Flexible nasopharyngoscopy showed pooling of secretions, without any glistening or pharyngeal edema, normal vocal fold movement, and a widely patent airway. The patient was admitted for non-operative management of her cervical infection and placed on vancomycin and ampicillin/subactam.

On hospital day 2, the patient was found apneic. She was emergently intubated for airway protection and mechanical ventilation. With the rapid and unexpected decline, the differential diagnosis was expanded to include causes of bulbar palsies including Guillain-Barre syndrome and a cerebrovascular event; with the patient’s history of heroin use and multiple abscesses, the working diagnosis of wound botulism (WB) was favored.

Repeat physical examination revealed ptosis and ophthalmoparesis. Lumbar puncture, CT angiography of the brain, CT angiography of the chest, echocardiogram, and blood and urine cultures were unrevealing for a cerebrovascular, cardiogenic, or infectious etiology. EMG 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 disease at the neuromuscular junction. Combined with this patient’s clinical presentation, wound botulism was highly likely.

Botulinum antitoxin obtained from the CDC was administered in coordination with the Department of Public Health (DPH). 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 strength 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.

DISCUSSION

*Clostridium botulinum* is a ubiquitous spore-forming, gram-positive, anaerobic bacillus in the soil. It produces toxin that is active at the neuromuscular junction presynaptic nerve terminal; inability to release acetylcholine causes a characteristic flaccid paralysis. *C. botulinum* spores are resistant to heating of heroin mixtures typically done before injection drug use, and thus can grow and produce toxin when in the anaerobic environment of subcutaneous and intramuscular tissue [1]. Subcutaneous injection of black tar heroin is the leading risk factor for WB [2]. The incidence is particularly high in California, but cases have been identified throughout the Western United States [3].

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 [4]. 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 ID & D 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. Recovery ensues following regeneration of presynaptic nerve fibers over the course of months [5]. For disease tracking, the Department of Public Health should be alerted to any suspected case of wound botulism.

REFERENCES


CONCLUSION

In 2006, Preuss et al reported a case of wound botulism presenting as dysphagia in a patient with IDU history and a forearm abscess [6]. In our case, the patient presented with similar symptoms and history, but was also found to have a deep space neck infection, which complicated the initial clinical assessment. This case illustrates the importance of considering WB in patients with 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.