Case report

Botulism in a heroin addict

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Introduction

Botulism most commonly follows the ingestion of contaminated canned, smoked or fermented food or food products. In the past 100 years, the United Kingdom has had three outbreaks: Loch Maree (1922) with 8 deaths, Birmingham (1978) with 4 deaths, and an outbreak in north-west England in 1989 with 27 cases [*1*]. Botulism is much more common in the United States of America where about 100 cases are reported each year [*2*]. Contamination of wounds with *Clostridium* spp. occasionally leads to the illness, but wound botulism is comparatively rare. We report what we believe is the first case of wound botulism in Jordan, following attempted intravenous injection of heroin that was contaminated with clostridial organisms (*C. novyi*) [*3*].

Case history

A 27-year-old male intravenous heroin user was admitted to the surgical ward with painful erythematous swelling in the right inguinal region. There was no other medical history. He was pyrexial (38 °C) with a polymorphonucleocytosis. Ultrasound examination showed no abscess formation. Intravenous cefuroxime and metronidazole were administered, changing to oral cephalexin and rectal metronidazole after 24 hours. His symptoms improved and he was discharged after 4 days.

After 3 days, he presented again with progressive dysphagia, dyspepsia, generalized weakness and fatigue. He denied further drug abuse. He was mildly hypothermic (35 °C). His tonsils were enlarged and infected. Neurological examination results were normal. His white blood cell count was marginally raised. Intravenous amoxycillin was prescribed for the tonsillitis. The following morning, his condition deteriorated suddenly. He became cyanosed and unresponsive. Respiratory arrest followed. He was intubated, ventilated and transferred to the intensive care unit where he regained consciousness. Neurological assessment showed normal or raised muscle tone in the limbs, generalized motor weakness affecting the bulbar and proximal muscles more than the distal ones. Pupil reactions were normal. There was no ophthalmoplegia. Tendon reflexes were brisk and plantar reflexes normal. Sensation was intact and the groin swelling had disappeared. Diagnosis at this point was not clear.

Over the next 24 hours, the patient remained partially ventilator-dependent but had reasonable limb movements. Computerized tomography of the brain undertaken prior to lumbar puncture was normal. Lumbar puncture revealed marginally elevated protein. Magnetic resonance imaging of the brain and the spinal cord performed 3 days later was normal. A tensilon test was inconclusive. The Miller–Fisher variant of

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Guillain-Barré Syndrome was considered to be a possible diagnosis and a course of immunoglobulin treatment was begun. His neurological function continued to deteriorate, however, leading to full ventilator dependency and a further decrease in all motor power. Percutaneous tracheostomy was performed.

Eleven days after admission to intensive care, electromyography (EMG) was performed and suggested acute toxic myopathy, possibly secondary to botulism. This was subsequently confirmed by serology. Intravenous penicillin and metronidazole were administered together with two doses of botulism antitoxin. There was no evidence of the previous groin infection and therefore no attempt was made to explore the previously inflamed area.

Over the next 4 weeks, the patient's peripheral motor weakness improved. At present, he has motor power sufficient to stand and walk unaided, despite still needing ventilatory support. Facial weakness remains. Swallowing has improved but the cough reflex remains impaired.

Discussion

Botulism is an acute, symmetrical, descending paralysis of the cranial and autonomic nerves produced by the exotoxin of *C. botulinum*. There are at least 8 different toxin-producing strains (A, B, C1, C2, D, E, F, G). Humans are usually affected by strains A, B and E.

Wound botulism is caused by the growth of *C. botulinum* in contaminated wounds accompanied by *in vivo* toxin production. Neurological findings are indistinguishable from those produced by food-borne botulism, but gastrointestinal symptoms do not occur. The wounds may not be obvious or grossly infected. Between 1943 and 1985, 33 cases of wound botulism were reported in the United States [*4*]. Most patients had traumatic injuries including fractures and deep contaminated wounds. The first reports of wound botulism and drug addiction appeared after 1980 when an asso- ciation between botulism, needle puncture sites and nasal sinus lesions (due to cocaine snorting) was reported [*5*]. In the 10 years up to 1996, 78 cases were reported in the United States, mostly linked to black tar heroin. The average age of the affected individuals was 38 years. Most were male. All but 12 cases were due to type A organism [*4*].

The clinical syndrome results from the toxin-induced blockade of the voluntary motor and autonomic cholinergic junctions. All toxins produce an identical clinical pattern. Dryness of the mouth, blurred vision and diplopia are the earliest neurological symptoms. In mild disease, the symptoms resolve and pass unremarked. In severe cases, the initial symptoms are followed by dysphonia, dysarthria, dysphagia and peripheral muscle weakness. Respiratory weakness leading to respiratory failure occurs in the most severely affected patients. Full neuromuscular regeneration may take up to 7 months, but most regeneration occurs within 2–8 weeks [*4*]. Mortality is mainly a result of a failure to recognize the severe form of the disease and respiratory complications.

The differential diagnosis includes Guillain-Barré syndrome and its variants, myasthenia gravis, tick paralysis, chemical intoxication (carbon monoxide, methyl alcohol, methyl chloride and organophosphorus compounds), mushroom poisoning (cholinergic symptoms), poliomyelitis, diphtheria, antibiotic associated muscle weakness (aminoglycosides), periodic paralysis, and psychiatric illness [*4*].

Cerebrospinal fluid examination helps to differentiate botulism from Guillain–Barré

syndrome. However, a slightly elevated cerebrospinal fluid protein level is occasionally seen in botulism while the protein level may initially be normal in Guillain–Barré syndrome [*6*]. A tensilon test helps to differentiate between myasthenic syndromes and botulism. Brain and cord imaging may rule out stroke, cord injury and tumour [*7,8*]. EMG may be helpful as a characteristic EMG pattern is present in botulism [*9–11*]. Toxicity testing of serum specimens and culture of tissues debrided from wounds are currently the gold standard but may not be applicable [*4*]. In the case reported here, there was a slight elevation of CSF protein, tensilon test was equivocal, EMG indicated acute myopathy and the serum was positive for type A toxin. As there was no obvious wound, we could not isolate the organism.

Treatment consists of administration of trivalent antitoxin, although delayed use may be pointless, and antibiotics (penicillin) together with appropriate symptomatic support, including mechanical ventilation [*4*].

The diagnosis of botulism is usually clinical, with a history indicating that a group of people have developed a similar illness. Wound botulism however shows an isolated occurrence. Although extremely rare, botulism is one of the few differential diagnoses for acute motor neuropathies. The predilection for the bulbar area is a further indication.

We believe our patient contracted the infecting organism from a contaminated batch of heroin. Unusual symptom complexes in heroin addicts should indicate the possibility of unusual anaerobic infections.

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