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Wound botulism in an injecting drug abuser

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Sir: *Clostridium botulinum* is an anaerobic, Gram-positive, spore-bearing organism. Wound botulism amongst injecting drug users (IDUs) is an increasing problem [1, 2, 3] and is associated with an anaerobic environment following intramuscular or subcutaneous injection [1]. We report a case of early identification and treatment of an IDU with wound botulism. The patient presented with a rapidly descending flaccid paralysis requiring urgent respiratory support. She made a full recovery and was discharged home after 40 days.

A 30-year-old female IDU presented with sudden onset dysarthria, dysphagia, visual disturbance, dyspnoea and urinary incontinence. Except for a deep venous thrombosis 2 months previously, past medical history was unremarkable. The patient was apyrexial, alert and oriented. Her respiratory rate was 18 bpm, and arterial saturation was 98% on air, with a clear chest to auscultation. There were weaknesses of the sixth (unilateral), seventh, ninth and tenth cranial nerves. Upper limb power was grade 4/5, shoulder abduction grade 3/5. Reflexes, sensation, coordination and lower limb power were normal. She had a discharging wound to the lower limb. Airway compromise necessitated intubation and ventilation with an oxygen concentration of 30% within 3 h of presentation. Full blood count, serum chemistry and cerebrospinal fluid analysis were unremarkable. Computed tomography of the head, brainstem magnetic resonance imaging and echocardiography were normal. Edrophonium testing was negative.

Botulism was diagnosed clinically on day 2 of admission, and intravenous antitoxin and benzyl penicillin were prescribed. The leg wound was debrided. Following tracheostomy sedation was weaned on day 5. This revealed worsening cranial nerve palsies, and proximal weakness in all limbs. On day 6 botulinum toxin A was identified. Electromyography demonstrated a pre-synaptic block with post-tetanic facilitation. Neuromuscular function recovered gradually. The tracheostomy was removal on day 36, and the patient returned home on day 40.

Wound botulism produces a rapidly descending bilateral flaccid paralysis, with cranial nerve, peripheral motor and autonomic effects. Dysarthria, dysphagia and diplopia are common presenting features. Clinical signs may be asymmetrical. Respiratory insufficiency may develop before peripheral weakness [4]. Sensation and coordination are unaffected; deep tendon reflexes may be reduced. Gastro-intestinal symptoms are typically absent, and patients remain afebrile. The differential diagnosis includes Guillain-Barré syndrome, particularly the Miller Fisher variant, myasthenia gravis, the Lambert-Eaton myasthenic syndrome, and intracranial or brainstem lesion. Guillain-Barré syndrome produces sensory symptoms, and CSF protein is raised. The Miller Fisher variant is confined to the cranial nerves, with prominent ataxia and generalized areflexia. Neither of the myasthenias has autonomic involvement; myasthenia gravis demonstrates decrement upon repetitive electromyographic stimulation. Botulinum toxin produces an irreversible presynaptic cholinergic block. Antitoxin is effective against *unbound* toxin only; early administration decreases both illness duration and mortality [5]. Recovery may take months, with minor autonomic and motor disturbances persisting for years.

Wound botulism amongst IDUs is an emerging problem in the United Kingdom. The initial diagnosis is based upon clinical suspicion and the exclusion of other differential diagnoses. Early supportive care and administration of antitoxin are vital to maximize the chance of recovery.

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