

A case of botulism in New Zealand

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ABSTRACT

We describe the first case of food-borne botulism seen in New Zealand for 30 years. Botulism is an important diagnosis to consider in a patient with rapidly progressive descending paralysis and normal sensorium. Early recognition, timely institution of intensive care support and administration of botulism antitoxin are the most important aspects of management.

Botulism is a rare, toxin-mediated disease with high mortality. The clinical presentation can mimic other acute progressive neurological disorders. We present the first case of food-borne botulism in New Zealand since 1984.

Case report

A 55-year-old male was admitted to Wellington Hospital two days after returning from Japan. On arrival in New Zealand, he consumed a 'wet risotto' packet meal despite it tasting 'rancid'. Within 24 hours, he developed vomiting, followed by dizziness, diplopia and dysarthria.

His provisional diagnosis on admission was of a posterior circulation stroke; however, on review by the neurology service six hours later, the findings were of ophthalmoplegia with right lateral rectus palsy, lower motor neuron facial weakness, dysarthria, dysphonia and glossal paresis. He had subtle distal weakness in both hands. Reflexes were retained. The patient remained alert and afebrile.

An MRI was unremarkable and cerebrospinal fluid (CSF) collection showed white cell count, protein and glucose within normal limits.

A rapid clinical deterioration followed such that within 10 hours he had developed severe bilateral ptosis, marked bilateral facial weakness, complete anarthria, moderate upper limb weakness, and all

reflexes were now absent. Forced vital capacity (FVC) was significantly reduced to 2.82 L (30 mL/kg). He was also tachypnoeic. Transfer to the Intensive Care Unit was facilitated due to continued respiratory deterioration and within hours he required intubation following a respiratory arrest.

Neurophysiology studies performed 48 hours after initial symptom onset showed markedly reduced compound muscle action potential (CMAP) amplitude with normal sensory studies. Of diagnostic significance, the abductor hallucis brevis CMAP amplitude increased 300% after tetanic stimulation.

While consideration was given to alternative diagnoses, such as Miller-Fisher variant of Guillain-Barre syndrome and acute neuromuscular junction disorders (myasthenia gravis), the speed of decline and combination of gastrointestinal symptoms followed by progressive cranial motor neuropathies, absent sensory features, normal sensorium and low CMAP amplitudes within hours of symptom onset all supported a probable diagnosis of botulism.

The patient was treated with botulinum antitoxin and required two weeks of intensive care support. Blood, stool and gastric washings were cultured, but no *Clostridium* species were isolated. *C. botulinum* toxin genes were not detected by PCR in gastric washings. Anti-GQ1B and acetylcholine receptor antibodies were

Table 1: Neurophysiology Studies

| Motor Studies | Within first 3 days of symptom onset | | | | 10 weeks post symptom onset |
|------------------------------|--|---------------------------|---|--|-----------------------------|
| | Distal Motor Latency (ms) | Conduction Velocity (m/s) | Pre tetanic stimulation CMAP Amplitude (mV) | Post tetanic stimulation CMAP Amplitude (mV) | CMAP Amplitude |
| Right AHB | 5.0 (3.96±1) | 43.8 (48.5±3.6) | 0.4 (5.8±2) | 1.2 (300% amplitude increase) | Not tested |
| Right APB | 2.96 (3.49±0.3) | 54.8 (58.7±5.1) | 0.1 (7±3) | 0.1 | 2.0 |
| Right ADM | 2.79 (2.95±0.4) | 59.2 (57.7±5) | 0.2 (5.7±2) | 0.2 | 6.8 |
| Sensory Studies (antidromic) | Sensory nerve action potential (SNAP) amplitude within first 3 days of symptom onset | | | 10 weeks post symptom onset | |
| Median (digit II) | 35.6 uV (38.5±15.6) | | | 50.6 uV | |
| Ulnar (digit V) | 20.8 uV (35.0±14.7) | | | 28.5 uV | |

AHB – Abductor hallucis brevis muscle, APB – Abductor pollicis brevis muscle, ADM – Abductor digiti minimi muscle

negative. His course was complicated with *Klebsiella pneumoniae* bacteraemia and ventilator associated pneumonia and acute kidney injury requiring temporary dialysis; however, after motor function began to return, he made a rapid and full recovery. Convalescent neurophysiology testing 10 weeks after the initial symptom onset confirmed the reestablishment of normal CMAP amplitudes. The neurophysiologic findings are shown in Table 1, with normal ranges given in brackets.

Compound muscle action potential (CMAP) is a summation of all underlying individual muscle fibre action potentials.¹ The table shows extremely low amplitude CMAPs on initial testing, which then normalised when retested 10 weeks later. Distal motor latencies and conduction velocities were essentially normal. The large increase in CMAP with tetanic (repetitive) stimulation of abductor hallucis brevis helped to localise the problem to the presynaptic part of the neuromuscular junction, and differentiated it from other neuromuscular junction disorders such as myasthenia gravis. The sensory nerve studies were normal at disease onset and during follow-up studies.

Public Health Investigation

Regional Public Health staff interviewed the patient's travel companion (via interpreter) and family. There was no history of intravenous drug use or therapeutic or cosmetic botulinum toxin application. His companion reported that he had eaten something 'off' at a relative's house, and had been the only person to do so. His family reported that it was a packet risotto product. Further investigation revealed that the risotto was a wet, chilled product purchased by a family friend. It had not been refrigerated and would have been several months past its best-before date. Packaging was not available for testing. When later shown photos of packaging of the presumed brand, the patient felt very confident that this was the product he had eaten. He confirmed that the risotto had a 'blue cheese' taste to it, and was also 'very bitter'. Of concern was that instructions to keep chilled were in very small font on the back of the package, 'Ready to eat' was written on the front despite the need for thorough reheating, and there was a best-before as opposed to a use-by date.

Discussion

This case illustrates that while botulism is rare, it must be included in the differential diagnosis of a patient with rapidly progressive descending paralysis and normal sensorium. Normal CSF protein and low amplitude CMAPs help determine the diagnosis. Mortality is greatly reduced by timely institution of intensive care support and administration of botulinum antitoxin.² Effectiveness of antitoxin is greatest when given early and should be given on clinical suspicion without waiting for results of diagnostic testing.³

Botulism is a toxin-mediated disease with high mortality. Botulinum toxins act presynaptically, preventing acetylcholine release at the neuromuscular junction, resulting in flaccid paralysis. These are the most potent toxins known, with an estimated oral lethal dose of 70 mcg in a 70 kg man.⁴ *Clostridium botulinum*, *C. butyricum* and *C. baratii* may produce botulinum neurotoxins (BoNT), of which BoNT A, B, E, and F cause human botulism.⁵ In adults, the commonest exposure to botulinum neurotoxins is through ingestion of pre-formed toxin in food which has been incorrectly preserved or stored, allowing growth of toxin-producing clostridia species. Gastrointestinal symptoms often develop within hours of ingesting the contaminated meal, with neurological symptoms evolving rapidly over subsequent hours to days.³ Shorter incubation periods are associated with greater toxin doses, a more rapid

progression of symptoms and more severe illness.⁶

Laboratory confirmation of botulism is difficult, particularly from samples with high levels of competitive bacterial flora or proteinases which may degrade the toxin.⁵ The gold standard test is the mouse lethality assay which is not available in New Zealand. Detection of *C. botulinum* toxin genes in gastric washings was attempted by PCR at the Animal Health Laboratory, Ministry of Primary Industries. This assay does not detect all possible types of BoNT.

The last reported cases of botulism in New Zealand were two sisters who became ill after ingesting home-preserved watercress and mussels.⁷

There have been other cases from similar wet, chilled products. In France in 2008, two people developed severe botulism requiring ventilation after eating pre-cooked chicken enchiladas, which had been kept at room temperature for two weeks. French authorities recalled the product, requested the manufacturer improve packaging to make storage instructions more visible, and issued a reminder about respecting storage conditions.⁸ Inappropriate storage also featured in two outbreaks in California in 1994, of vacuum packed clam chowder and black bean dip, both kept at room temperature for 1 month and 3 weeks respectively.⁹ This reinforces the need for adequate labelling around storage and shelf-life and consumer adherence to these recommendations in New Zealand.

Competing interests:

During the time of the investigation, Regional Public Health (the employer of Dr Deverall and Dr Nesdale) was providing 'Food Safety and Suitability Services' under contract to the Ministry of Primary Industries.

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