4 CASE REPORTS

a Botulism

On 30 January 2015, a 44-year-old male was admitted to a private hospital in Johannesburg complaining of abdominal pain, difficulty in swallowing and breathing, constipation, and nausea. The following day his condition deteriorated rapidly; he developed respiratory failure and required intubation and ventilation. His condition continued to worsen and he developed acute flaccid paralysis. Due to the nature of the presentation (bulbar palsy and gastrointestinal symptoms), the disease progression (respiratory failure and acute flaccid paralysis), the patient's clinician considered botulism as a differential diagnosis.

Detailed history of food consumption in the two days prior to illness onset was obtained from the patient's wife, in order to identify possible foodborne sources of *Clostridium botulinum*. In summary:

- On 28 January 2015: the patient consumed food items purchased from a reputable commercial food store for lunch and supper, and also ate home-made pickled cucumbers (bought from an 'artisanal' producer).
- On 29 January 2015: the patient consumed food items purchased from a reputable commercial food store for lunch, and in the evening ate chicken curry with vegetables (potatoes, peas and carrots) at a restaurant in a shopping mall.

Laboratory investigation for botulism was performed at the NICD-NHLS using the mouse bioassay, which is the gold standard method for detection of botulinum neurotoxin (BoNT). This method entails an initial mouse toxicity assay where the mice are injected with untreated patient serum. In this case, a blood sample collected on day 2 of illness was used for the assay, and the mice died within 24 hrs post-inoculation. Subsequently, a BoNT mouse neutralisation assay was performed during which mice were injected with patient serum treated with anti-toxin A, B and E, respectively. Preliminary results indicate that the cause of illness is BoNT type E.

Although the patient required intensive care and mechanical ventilation, he is recovering. Unfortunately, it was not possible to identify the food item/s responsible for the disease.

Focus on botulism

Botulism is a paralytic illness caused in humans by neurotoxins produced by *Clostridium botulinum* (toxin types A, B, E, F, and H), and rarely, by botulinum-producing strains of *Clostridium butyricum* (type E toxin), *Clostridium baratii* (type F toxin) and *Clostridium argentinense* (type G toxin). There are five forms of botulism, characterised by the mode of acquisition: infant botulism, wound botulism, foodborne botulism, adult enteric infectious botulism, and inhalational botulism.

Although *C. botulinum* spores are ubiquitous in the environment, the growth and production of botulinum toxin in foods only occurs under particular conditions (anaerobic, low-salt, low-acid conditions). Canning and fermentation of foods both create anaerobic conditions that facilitate the germination of *C. botulinum* spores, and contaminated home- or commercially-canned foods (including a range of vegetables, meat, fish, and condiments) have been linked to outbreaks of botulism since the 19th century. However, a wide range of food items have been responsible for outbreaks, including: meat products (smoked ribs, sausages, ham); fish products (smoked, fermented, or salted); vacuum-packed ready-to-eat products (bean soup, meat-containing 'ready meals'); fermented vegetable products (tofu and other fermented bean products); and beverages (commercial carrot juice, illicit alcoholic beverages).

The onset of symptoms in foodborne botulism usually begins after an incubation period of 12-36 hours (range four hours to eight days) after ingestion of the preformed toxin. Prodromal symptoms are predominantly gastrointestinal (including nausea, vomiting, abdominal pain, diarrhoea) accompanied by dry mouth and sore throat. Clinical disease is characterised by cranial nerve palsies (symptoms may include blurred vision, diplopia, nystagmus, ptosis, dysphagia, dysarthria and facial weakness) followed by descending acute flaccid paralysis. Respiratory compromise requiring intubation and mechanical ventilation is common, caused by diaphragmatic paralysis and/or upper airway compromise. The disease course is variable, ranging from mild illness to rapidly progressive disease with death within 24 hours of symptom onset.

Management of foodborne botulism includes

intensive care support with mechanical ventilation if needed, and administration of equine antitoxin. Although the efficacy of antitoxin has only been evaluated in animal studies, observational studies suggest that antitoxin therapy is likely to benefit humans with botulism. Antitoxin should be administered as soon as possible if the clinical suspicion for botulism is high, and not be delayed pending the outcome of laboratory testing for botulism. Although the efficacy is likely to be greatest if administered early after onset of symptoms, given the protracted course of illness in severe cases and potentially fatal nature of the disease it is recommended to source and administer antitoxin regardless of delays in clinical diagnosis or antitoxin procurement. However, since equine antitoxin can cause sensitisation and anaphylaxis, it should only be given if the history and clinical presentation are highly suggestive of the disease. Antitoxin is not available in South Africa, but may be procured from international producers if warranted.

A clinical diagnosis of botulism is supported by detection of botulinum neurotoxin (BoNT) or *C. botulinum* in the stool or suspected food. It is important that clinical samples are collected as soon as possible after onset of illness (ideally within 3 days) to increase the likelihood of meaningful results.

Source: Division of Public Health Surveillance and Response and Centre of Emerging and Zoonotic Diseases, NICD-NHLS; Clinicians at Sunninghill Hospital