Ciguatera fish poisoning
Patrick Armstrong, Peter Murray, Annette Nesdale, Brad Peckler

ABSTRACT
Ciguatera fish poisoning (CFP) is the most common cause of seafood-toxin poisoning in the world and is most prevalent in tropical and subtropical areas. It causes gastroenteritis but also myriad neurological and cardiovascular symptoms. We present a cluster of CFP that occurred in Wellington Hospital, New Zealand. It resulted in three patients with life threatening cardiotoxicity and a fourth case with severe gastrointestinal symptoms. The epidemiology, clinical manifestations, diagnosis, treatment and public health issues are discussed.

Ciguatera fish poisoning (CFP) is the most common cause of seafood-toxin poisoning in the world and is most prevalent in tropical and subtropical areas. It causes myriad neurological, gastrointestinal and cardiovascular symptoms, the latter of which can be life threatening. Though a well recognised condition in prevalent regions such as the Pacific Islands, CFP is likely to be under-reported or go unrecognised in non-prevalent countries like New Zealand. However, due to New Zealand's large Pacific population and the popularity of the Pacific Islands as a holiday destination, CFP is an important differential diagnosis for emergency physicians to be aware of.

We would like to report a cluster of CFP that occurred in Wellington Hospital, New Zealand. It resulted in three patients, presenting during a night shift in the emergency department (ED) with life threatening cardiotoxicity, and a fourth case with severe gastro-intestinal symptoms.

Case history
Case 1
At 1:00am on a busy night shift in the ED, a previously healthy 67 year-old lady was brought in by ambulance, with vomiting and dizziness. On arrival she was bradycardic (HR 32 bpm) and hypotensive (BP 72/35 mm Hg); her other vital signs were normal. She was lightheaded but alert; denied chest pain or shortness of breath and stated she was previously well before starting to vomit. Her electrocardiograph showed sinus bradycardia.

She was treated immediately with 600 mcg atropine and her vital signs normalised. A cardiological cause of her bradycardia was investigated and ruled out. Whilst in ED she developed paraesthesia of the lips, tongue and a metallic taste in her mouth. She reported she had eaten eel at 7:00pm with friends the previous evening and had started vomiting soon after.

Case 2
One hour later, a 67 year-old man self-presented with vomiting and diarrhoea. He collapsed in triage and was taken to a resuscitation bay. He was found to be profoundly bradycardic (HR 30 bpm) and hypotensive (BP 72/35 mm Hg). His vital signs improved with atropine. He stated that prior to developing symptoms he had eaten eel, alongside his wife, son and a family friend, and stated his wife was also unwell with vomiting at home.

Case 3
Forty minutes later the 41 year-old son of Case 2 who had registered to be seen for diarrhoea and vomiting, collapsed in the waiting room. He was also found to be bradycardic (HR 31 bpm) and hypotensive (BP 61/42 mm Hg), and was taken to the resuscitation bay. His vital signs immediately normalised with atropine. At this stage it was not known that cases 1, 2 and 3 had eaten together. Preparations were made in case of a further influx of symptomatic patients.

Case 4
The 58 year-old wife of Case 2 was contacted and advised to come to the ED for...
review as she had also consumed the same eel. She ate the eel earlier than Cases 1–3, and complained of diarrhoea and vomiting throughout the previous day. On arrival, her vitals and ECG were normal. However, she subsequently developed a fever of 39.5°C and her lab results showed an acute kidney injury.

Provisional diagnosis—CFP

It became clear from the cases’ respective histories that they had eaten the same eel and were displaying features suggestive of CFP. In light of this provisional diagnosis, the Regional Public Health was notified. Cases 1, 2 and 3 were admitted to the high dependency unit where they required 4–6 hourly atropine doses to maintain adequate heart rate and blood pressure. Their diarrhoea and vomiting settled in the first 24 hours, however, the cardiotoxicity persisted for 3–4 days. Case 4 was admitted to the medical ward, where she received IV rehydration and was discharged the following day.

Post discharge follow-up

All four cases were followed up two months after being discharged. All described lethargy and fatigue that persisted for a number of weeks. Cases 1–3 further described chronic symptoms of cold alldynia that developed two weeks following initial illness.

Public health management

Following the notification of a probable CFP outbreak, Health Protection Officers from Regional Public Health interviewed the ill people to ascertain a full food and risk factor history, the origin of the eel and to identify if other people were exposed and if any of the eel remained.

The common source of the CFP outbreak was identified as moray eel that had been purchased in Samoa and brought back into New Zealand by one of the cases. The eel was cooked prior to consumption. A sample of the implicated eel was eventually obtained and sent for ciguatoxin testing, which was initially negative. However, subsequent testing was strongly positive for ciguatoxin-1B (CTX-1B), thereby confirming the diagnosis of CFP. All the cases were provided with information about CFP including ways to prevent symptom recurrence and future exposure.

Ciguatera fish poisoning

CFP is a common fish-related food borne illness in tropical and sub-tropical regions. It results from the consumption of certain tropical reef fish species (eg moray eel, barracuda and amberjack) that bioaccumulate toxins of the naturally occurring Gambierdiscus spp. The toxins produced by these microalgae—ciguatoxins—are heat-stable, so are not inactivated by cooking or freezing. New Zealand fish species are not known to cause CFP.

Epidemiology—New Zealand

CFP cases and outbreaks are notifiable if they cause acute gastroenteritis. Accordingly, cases without gastrointestinal features or those with delayed presentations (ie after consuming affected fish whilst in the Pacific Islands) may not be notified.

Surveillance of CFP in New Zealand occurs through the EpiSurv notification database or hospitalisation records. From 2006–2014, there were 17 notifications and 54 hospitalisations of CFP (Table 1). The disparity between hospitalised and notified cases highlights the under-reporting of CFP to public health units. Over this period there have been four CFP outbreaks recorded, with each involving two to six cases. Some of these outbreaks have been linked to tropical reef fish being brought into New Zealand by travelers returning from the Pacific.

Clinical manifestations

The clinical features of CFP usually develop within 6–12 hours of ingestion of fish contaminated with ciguatoxins. Gastrointestinal features such as nausea, vomiting, abdominal pain and diarrhoea are common and occur soon following ingestion. However, not all patients will present with gastrointestinal features.

Neurological symptoms present as paraesthesia, pruritus, myalgia and classically, reversal of temperature perception (“hot/cold” reversal, cold alldynia) where cold sensation is experienced as painful burning. Neuropsychiatric conditions also can occur with anxiety, depression, memoryless, delirium, ataxia and coma all being reported. These symptoms can last for weeks to months and rarely years. Symptoms can also recur when eating any
type of fish, alcohol, nuts, caffeine, chicken, pork or physical exertion.1,2 These features are more common in Pacific cases.

Cardiac toxicity from ciguatoxins, hypotension and bradycardia are the most severe and potentially life-threatening consequence of CFP.1 These features can present soon after ingestion of contaminated fish and require immediate medical attention.1,15

Following the acute illness, the clinical manifestations of CFP can reoccur when triggered by certain precipitants, such as eating any type of fish, alcohol, nuts, caffeine, chicken, pork or on physical exertion.1

**Diagnosis**

Diagnosis of CFP is primarily clinical, as no biomarkers are currently available to confirm exposure in patients.1,16 A history of fish ingestion in a patient with cardiovascular, gastrointestinal or neurological symptoms should prompt suspicion of CFP.1,16 CFP is confirmed when ciguatoxins are detected in the implicated fish.1

**Treatment and management**

Treatment of CFP is primarily supportive.1 Symptomatic bradycardia can be treated with atropine or temporary cardiac pacing.15 Hypotension can be managed with intravenous hydration. Very rarely patients develop respiratory failure and assisted ventilation may be required.15,17

IV mannitol was previously recommended in CFP treatment, given within 48–72 hours of ingestion to reduce acute—and prevent chronic—neurological symptoms.1,4 It was thought to act by reducing neuronal oedema and scavenge free radicals created by the CTX molecule. However, a double-blind randomised controlled trial found no benefit compared with placebo.18 Gabapentin and amitriptyline were used to treat pain, and paraesthesias and fluoxetine for neuropsychiatric conditions such as anxiety and chronic fatigue following ciguatera poisoning.19,20

Notification of suspected CFP to the local public health unit is important and is a legal requirement under the Health Act 1956.5 Early notification can facilitate case finding, acquisition of implicated fish samples, testing of samples, and also provide education on ciguatera, how to prevent future exposure and reduce recurrent symptoms.1

**Conclusion**

This case report highlights the importance of recognising CFP in the ED. It shows even small outbreaks can put significant strain on resources both in the ED and in the inpatient setting. This case was unusual, given the predominance of cardiotoxicity in three of the cases. It also demonstrates that early public health notification can facilitate the acquisition and testing of implicated seafood. Given the large Pacific population in New Zealand and the large number of tourists frequenting the Pacific islands, it is important to consider and notify CFP in patients presenting with clinically compatible symptoms.

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Table 1: New Zealand CFP notifications and hospitalisation by year.6–14
Competing interests: 
Nil.

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REFERENCES: