

ACADEMIC: CASE REPORT

Two cases of ciguatera fish poisoning following eel consumption

Steve Waqanivalagi, Michael Murphy, Peter Jones

Case

An 18-year-old man presented to the Emergency Department with generalised abdominal pain, nausea, loose bowel motions, haematochezia, and perioral paraesthesia starting thirty minutes after eating eel. He was tachycardic (108 bpm) and hypertensive (188/90 mm Hg), his abdomen was generally tender but soft, and he had bilateral perioral paraesthesia. His 45-year-old father presented with generalised abdominal pain, loose bowel motions, perioral paraesthesia, and bilateral foot paraesthesia starting one hour after exposure. His vital signs were normal and his abdomen was generally tender, but soft. Neither patient complained of chest discomfort, dyspnoea, visual disturbance, or muscle weakness.

Both patients were treated with two litres of 0.9% saline and monitored for bradycardia and hypotension. They remained haemodynamically stable after four hours of care and were then discharged home with return advice and routine general practitioner review.

The patients reported that the eel had been brought back from Tonga two days prior to being home-smoked for consumption. Three other family members had also eaten the eel, two of whom had reportedly developed similar symptoms and presented to another emergency department in Auckland.

Ciguatera fish poisoning (CFP) was considered clinically probable in these two cases given the close association between eel consumption and symptom onset, and the development of perioral paraesthesia, which is not typically seen in other foodborne illnesses.¹⁻⁶ Other toxin-related conditions may have been considered, had the source of the patients' illnesses been uncertain. For example, a history of shellfish ingestion would have prompted consideration of neurotoxic shellfish poisoning and paralytic shellfish poisoning, which are caused by the dinoflagellates *Gymnodinium breve* and those of the *Alexandrium* genus. The Japanese delicacy pufferfish, which produces tetrodotoxin, may also have been a relevant consideration, although pufferfish poisoning differs from CFP in that tetrodotoxin causes muscle weakness and paralysis. Other illnesses with similar features include organophosphate poisoning, foodborne botulism, Guillain-Barré Syndrome, multiple sclerosis, and eosinophilic meningitis.^{7,8}

The patients were followed up by telephone two weeks after discharge. They both reported resolution of their gastrointestinal symptoms but complained of ongoing perioral paraesthesia. The younger patient had also since developed perioral cold allodynia. Unlike gastrointestinal and cardiovascular complaints, which commonly improve within 48 hours and rarely extend beyond four days, neurological symptoms may persist for weeks to months.^{1,2,8} Although uncommon, they may even linger for many years depending upon toxin-related factors such as previous exposure and amount ingested.^{3,7}

Discussion

CFP is the commonest cause of toxin-related foodborne illnesses in tropical and subtropical regions.¹⁻⁴ It is caused by ciguatoxin-producing dinoflagellates such as those of the *Gambierdiscus* genus, which are a food source for herbivorous fish.^{1,3,4,6} Ciguatoxin climbs the food chain when affected fish are preyed upon by carnivorous species such as barracuda, moray eel, sea bass, and red snapper.¹ Thus, humans may contract CFP when contaminated fish are consumed.^{1-4,6}

Ciguatera toxicity is often under-reported and poorly-recognised,^{2,4} perhaps because it is infrequently seen in Australia and New Zealand.^{2,3} Nevertheless, CFP can have profound systemic effects compared to other foodborne illnesses, which necessitates its early recognition and management.¹⁻³ Fortunately, the cases we encountered were mild in that they required little intervention.

Ciguatoxin is difficult to detect because it is colourless, odourless, and tasteless. Attempts to mitigate CFP by cooking contaminated food are also futile, because ciguatoxin is heat-cold stable.¹⁻³ Thus, the only means of preventing CFP is by avoiding the consumption of reef fish.⁸ Ciguatoxin mediates its deleterious effects by binding to sodium (Na^+) channels, which increases Na^+ channel permeability and membrane excitability.^{1,5} Thus, in addition to milder symptoms such as paraesthesia and cold allodynia, patients may suffer from severe sequelae such as cardiotoxicity or a coma.²

The treatment of CFP is largely supportive.¹ Thus, fluid resuscitation is used to manage hypotension or to replace fluid losses, and neuromodulating agents such as gabapentin or pregabalin may be used to treat neurological symptoms.^{2,5} In severe cases, patients may require atropine to counter bradycardia, or vasopressors to treat persistent hypotension. Mechanical ventilation may even be required to manage hypoventilation.^{2,4,5}

Although infrequently seen in our department, the cases reported here highlight the importance of considering CFP in patients with gastrointestinal upset coupled with neurological symptoms after recently ingesting seafood.¹⁻⁴ The symptoms may be prolonged or require intensive intervention, which is why recognition must occur early.^{1,2} An increasing number of tourists are frequenting higher risk Pacific regions,² which has been associated with a rise in CFP in enquiries received by the National Poisons Centre.⁶ The cases we encountered were unique in that the contaminated food was brought ashore and then consumed by the patients. Given the growing Pacific population in Australia and New Zealand,² personally imported food may become an important feature of history taking in patients presenting with gastrointestinal upset coupled with neurological symptoms. Trends in CFP diagnoses are overseen by public health services.² Notification to a public health service is thus important for monitoring affected seafood and managing outbreaks.²⁻⁴

References

1. Traylor J, Singhal M. Ciguatera Toxicity [Internet]. Florida: StatPearls Publishing; 2019 [updated 2019 Apr 9; cited 2019 Jun 27]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK482511/>.
2. Armstrong P, Murray P, Nesdale A, Peckler B. Ciguatera fish poisoning. *N Z Med J*. 2016;129:113–6.
3. Edwards A, Zammit A, Farrell H. Four recent ciguatera fish poisoning incidents in New South Wales, Australia linked to imported fish. *Commun Dis Intell*. 2019;43.
4. Farrell H, Murray SA, Zammit A, Edwards AW. Management of ciguatoxin risk in eastern Australia. *Toxins*. 2017;9:367–77.
5. Brett J, Murnion B. Pregabalin to treat ciguatera fish poisoning. *Clin Toxicol*. 2015;53:588.
6. Schep LJ, Slaughter RJ, Temple WA, Beasley DM. Ciguatera poisoning: an increasing occurrence in New Zealand. *N Z Med J*. 2010;123:100–2.
7. Sobel J, Painter J. Illnesses caused by marine toxins. *Clin Infect Dis*. 2005;41:1290–6.
8. Friedman MA, Fleming LE, Fernandez M, Bienfang P, Schrank K, Dickey R, et al. Ciguatera fish poisoning: treatment, prevention and management. *Mar Drugs*. 2008;6(3):456–79.

About the authors

- › Dr Steve Waqanivalagi, MBChB, BMedSc(Hons). Adult Emergency Department at Auckland City Hospital, Auckland District Health Board, Auckland and Department of Medicine, University of Auckland, Auckland.
- › Dr Michael Murphy, MB BS, BPharm, BSc(Hons). Adult Emergency Department at Auckland City Hospital, Auckland District Health Board, Auckland.
- › Associate Professor Peter Jones, MBChB, MSc (Oxon), FACEM. Adult Emergency Department at Auckland City Hospital, Auckland District Health Board, Auckland and Department of Surgery, University of Auckland, Auckland

Correspondence

Dr Steve Waqanivalagi
SteveWaq@adhb.govt.nz
Adult Emergency Department at Auckland City Hospital
Auckland District Health Board
Grafton
Auckland