CASE REPORT

Ciguatera poisoning in the Cook Islands

Stephanie Bailey, Tristan Withers

University College London,
London, UK

Correspondence to

Stephanie Bailey,
stephaniebailey1705@gmail.
com

Accepted 6 June 2014

SUMMARY

This case report presents two British medical students who contracted ciguatera poisoning while on elective in the Cook Islands. Thirty-six hours after consuming two reef fish they developed paraesthesia of the mouth, hands and feet, myalgia, pruritis and cold allodynia. Neurological examination was normal. Diagnosis of ciguatera poisoning was made on history of reef fish consumption and classical clinical presentation. Management was symptomatic (antihistamines) and both students made a full recovery within 10 weeks.

BACKGROUND

Ciguatera poisoning is the most common form of non-bacterial food-poisoning from fish worldwide.¹ It produces a wide range of neurological, cardiovascular and gastrointestinal symptoms. The estimated global incidence is between 10 000 and 50 000 per annum, although this is thought to be an underestimation.^{2–3} Worldwide data collection is ineffective and there is a generalised reticence to report disease. In non-epidemic areas the limited cases are seldom reported due to the lack of recognition of characteristic signs and symptoms. There is currently no clinical diagnostic test for ciguatera poisoning and diagnosis is based on presenting symptoms and recent history of reef fish consumption.

Ciguatera poisoning is caused by ingesting reef fish whose flesh is contaminated by ciguatoxins. Precursors of ciguatoxin are produced by dinoflagellates of the genus *Gambierdiscus* which live on algae in coral reefs. These precursors undergo bio-transformation and bioaccumulation during trophic transfer and metabolism in herbivorous and piscivorous fish and are converted to ciguatoxin.⁴ When ingested by humans the ciguatoxins cause neurological, cardiovascular and gastrointestinal symptoms. The hallmark symptoms are paraesthesia and reversal of thermal sensation. Symptoms can last for several days, weeks or even months and, although rarely fatal, the illness can be quite disabling.

Ciguatera endemics have been reported in many tropical and subtropical areas with the highest reported global incidence being on Rarotonga in the Cook Islands. However, given the current levels of fish imports and global travel, a differential of ciguatera poisoning should not be confined to these areas.

CASE PRESENTATION

Two medical students, a woman aged 23 and man aged 24, spent a 6-week elective placement in Rarotonga, Cook Islands. Both were previously

healthy and had no significant medical history. Twelve hours after eating a parrotfish and a trevally they both developed mild nausea with no associated vomiting or diarrhoea. These symptoms were followed by fatigue, lethargy and generalised weakness which lasted for 48 h. Thirty-six hours after eating the fish they developed a collection of neurological symptoms: extremity pruritis, paraesthesia of mouth, hands and feet, myalgia and cold allodynia. Cold fluids and objects were felt as a hot, burning sensation; hot fluid and objects were still felt as hot. Drinking cold fluids produced a tingling sensation in the mouth. The symptoms were unpredictable and fluctuating in nature, except for the cold allodynia which was triggered by touching cold objects and pruritis which was worse later in the day.

TREATMENT

Management was symptomatic: oral hydration and antihistamines.

OUTCOME AND FOLLOW-UP

The majority of the neurological symptoms had subsided by 4 weeks, but the cold allodynia persisted for 10 weeks. During this period, one student experienced several relapses of other symptoms on consumption of meat or alcohol. Both students are currently healthy with no long-term symptoms.

DISCUSSION

The global incidence of ciguatera poisoning is between 10 000 and 50 000 per year, although this is thought to be an underestimation. Ciguatera epidemics have been reported in California,⁵ Hawaii,⁶ the Caribbean⁷ and extensively in the South Pacific,⁸ which produces a significantly high number of cases. Rarotonga, the most populated island in the Cook Islands (pop. 13 700)⁹ reports the highest incidence of cases globally.¹⁰ Cases of ciguatera in the Cook Islands have been reported throughout history, and Captain James Cook himself described “scorching heat to the skin after eating toxic fish,”¹¹ although it was not until the 1990s that ciguatera poisoning became a major public health concern.¹² The number of cases of ciguatera poisoning in the Cook Islands per year from 1992 to 2013 is shown in [table 1](#).

Rongo and van Woessik¹³ found that both reef disturbance and density of herbivorous fish were good predictors of ciguatera poisoning. Both of these factors increased following major cyclones, thus they suggested that high numbers of cyclones affecting Rarotonga are responsible for the high

CrossMark

To cite: Bailey S, Withers T. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-204847

Table 1 Cases of ciguatera poisoning in the Cook Islands (1992–2013)

Year	Number of cases seen	Number of admissions to Rarotonga hospital		
		Male	Female	Total
1992	148	8	11	19
1993	55	12	4	16
1994	216	12	7	19
1995	281	14	15	29
1996	304	25	14	39
1997	161	27	20	47
1998	215	23	34	57
1999	209	35	18	53
2000	162	18	6	24
2001	173	21	19	40
2002	169	24	16	40
2003	227	23	17	40
2004	469	33	15	48
2005	421	39	24	63
2006	258	28	15	43
2007	245	20	17	37
2008	223	15	20	35
2009	129	22	13	35
2010	78	19	7	26
2011	102	18	5	23
2012	90	12	4	16
2013	90	7	2	9
Total	4425	455	303	758

Courtesy of Mr T Lorangi, medical records manager and statistician, Rarotonga 2014.

number of ciguatera cases. Furthermore, they suggested that the decrease in frequency of cyclones is responsible for the decrease in cases of ciguatera poisoning.

The clinical features of ciguatera poisoning are due to ciguater toxins in the flesh and viscera of contaminated fish. Precursors of ciguatoxin are produced by dinoflagellates of the genus *Gambierdiscus*. The dinoflagellates adhere to coral and algae which are eaten by herbivorous fish and in turn carnivorous fish and humans. The precursors of the ciguatoxin undergo bio-transformation and bioaccumulation during trophic transfer and metabolism in herbivorous and piscivorous fish and are converted to ciguatoxin.⁴ The fish species prone to toxicity in the South Pacific include: heavybeak parrotfish, humphead wrasse, great barracuda, giant moray, chinaman fish and spotted coral grouper.¹⁴ The Pacific ciguatoxin is distinct from the Caribbean toxin, but both bind to site five on the voltage-gated sodium channel.¹⁵ The toxin activates the sodium channel, causing an influx of ions followed by a cascade of events which result in the characteristic symptoms.

The clinical features of ciguatera can be split into three categories: gastrointestinal, neurological and cardiovascular. The gastrointestinal symptoms first occur between 2 and 12 h after ingestion of fish and include nausea, vomiting and watery diarrhoea. They normally diminish within 72 h. The cardiovascular symptoms of hypotension and bradycardia also occur early and can last for up to a week. The onset of neurological symptoms is variable, but normally later than gastrointestinal and cardiovascular symptoms. The neurological symptoms characterise ciguatera, and the range is broad. Sensory symptoms include: paraesthesia, paradoxical dysesthesia or cold allodynia (which is

almost pathopneumonic),¹⁶ asthenia, myalgia and pruritis. Motor symptoms include ataxia, spasticity, weakness, seizures and rarely respiratory arrest. Other symptoms include confusion, delirium and coma. Ciguatera is rarely fatal and the symptoms usually resolve after several weeks or months.

Ciguatera poisoning is a clinical diagnosis based on a history of reef fish ingestion, presenting symptoms and exclusion of other causes. There is currently no standard laboratory test to confirm ciguatera poisoning, however, an assay¹⁷ exists that can qualitatively measure ciguatoxin in fish fluid; and a Japanese group¹⁸ has developed a monoclonal antibody immunoassay which can detect the toxin in human fluids. Likewise, there is no standard treatment regime for ciguatera poisoning. This is likely due to the infrequency of cases and the lack of resources to conduct clinical trials in the places cases occur more frequently. Therefore, the mainstay of treatment is supportive. The most investigated therapeutic treatment for ciguatera poisoning is intravenous mannitol. Although some beneficial effects have been recorded as case studies,^{19 20} a randomised control trial conducted in Rarotonga hospital in the Cook Islands found the effects of mannitol no more effective than normal saline, and it produces more side effects.²¹

Patient's perspective

- ▶ As two medical students on our elective, we were somewhat surprised to find ourselves afflicted with a rare condition that few students or doctors have even heard of. What's more, at the time we developed ciguatera poisoning, we were undertaking an audit looking at the prevalence of peripheral neuropathy in the Cook Islands. While we wondered whether we would see any rare causes of neuropathy, it never occurred to us that we might experience an unusual neurological disease first-hand. Before experiencing several of our symptoms, it was difficult to imagine what they might actually be like. Fatigue for example, we had always just imagined as the feeling you get when you have not had enough sleep or have been on a long walk. However, the reality of feeling too exhausted to walk to the sink and get a glass of water was far worse than either of us had previously realised. Both of us feel that this has informed our future practice, and are very grateful that the symptoms resolved!

Learning points

- ▶ Ciguatera is the commonest form of non-bacterial food-poisoning from fish worldwide.
- ▶ Given current levels of fish imports and global travel, ciguatera poisoning should be considered as a differential worldwide and not just in areas of epidemics.
- ▶ There is no clinical diagnostic test and diagnosis is based on presenting symptoms and history of reef fish consumption.
- ▶ There is currently no standardised treatment regime, although intravenous mannitol has had some success.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Friedman A, Fleming E, Fernandez M, *et al*. Ciguatera fish poisoning: treatment, prevention and management. *Mar Drugs* 2008;6:456–79.
- 2 Barbier HM, Diaz JH. Prevention and treatment of toxic seafood-borne disease in travellers. *J Travel Med* 2003;10:29–37.
- 3 Ting JY, Brown AF, Paern JH. Ciguatera poisoning: an example of public health challenge. *Aust N Z J Public Health* 1998;22:140–2.
- 4 Dickey RW, Plakas SM. Ciguatera: a public health perspective. *Toxicon* 2009;56:123–36.
- 5 Barton ED, Tanner PA, Turchen SG, *et al*. Ciguatera fish poisoning. A southern California epidemic. *West J Med* 1995;163:31–5.
- 6 Gallop JH, Po EW. Ciguatera: a review. *Hawaii Med J* 1992;51:91–9.
- 7 Pottier I, Vernoux JP, Lewis RJ. Ciguatera fish poisoning in the Caribbean islands and Western Atlantic. *Rev Environ Contam Toxicol* 2001;168:99–141.
- 8 Russel FE, Egen NB. Ciguatera fishes, ciguatoxin and ciguatera poisoning. *J Toxicol Toxin Rev* 1991;10:67–76.
- 9 The Central Intelligence Agency. World Factbook. <https://www.cia.gov/library/publications/the-world-factbook/geos/cw.html> (accessed 5 Feb 2014).
- 10 Rango T, van Woesik R. Socioeconomic consequences of ciguatera poisoning in Rarotonga, southern Cook Islands. *Harmful Algae* 2012;20:92–100.
- 11 Broadbent G. Ciguatera. *Aust Fam Physician* 1987;16:127–8.
- 12 Rongo T, Van Woesik R. Ciguatera poisoning in Rarotonga, southern Cook Islands. *Harmful Algae* 2011;10:345–55.
- 13 Rongo T, van Woesik R. The effects of natural disturbances, reef state, and herbivorous fish densities on ciguatera poisoning in Rarotonga, southern Cook Islands. *Toxicon* 2013;64:87–95.
- 14 Farstad DJ, Chow T. A brief case report and review of ciguatera poisoning. *Wilderness Environ Med* 2001;12:263–9.
- 15 Swift A, Swift T. Ciguatera. *J Toxicol Clin Toxicol* 1993;31:777–81.
- 16 Caravati EM, McGuigan MA, *et al*. Marine envenomation and poisoning. In: Dart RD. ed *Medical toxicology*. 3rd edn. Lippincott Williams and Wilkins, 2004:1635–41.
- 17 Baden DG, Melinek R, Sechet V, *et al*. Modified immunoassays for polyether toxins: implications of biological matrixes, metabolic states, and epitope recognition. *J AOAC Int* 1995;78:499–508.
- 18 Tsumuraya T, Fujii I, Hiramama M. Production of monoclonal antibodies for sandwich immunoassay detection of pacific ciguatoxins. *Toxicon* 2010;56:797–803.
- 19 Friedman MA, Fleming LE, Fernandez M, *et al*. Ciguatera poisoning successfully treated with delayed mannitol. *Mar Drugs* 2008;6:456–79.
- 20 Mitchell G. Treatment of a mild chronic case of ciguatera fish poisoning with intravenous mannitol, a case study. *Pacific Health Dialo* 2005;2:155–7.
- 21 Schnorf H, Taurarii M, Cundy T. Ciguatera fish poisoning: a double-blind randomized trial of mannitol therapy. *Neurology* 2002;58:873–80.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow