Ciguatera fish poisoning

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Summary

Ciguatera fish poisoning is one of a variety of non-bacterial forms of human seafood poisoning. Consuming large predatory fish from tropical reef ecosystems may be hazardous. We describe a case that is typical of the disease, and illustrates the persistence of neurological symptoms that occur in some patients.

Keywords: ciguatera fish poisoning; ichthyosarcotoxaemia; poisoning; biotoxins

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Humans who eat large predatory reef fish in the tropics risk the acute and sometimes severe neurological and gastrointestinal symptoms associated with ciguatera fish poisoning. Acute management is symptomatic and supportive. Intravenous mannitol may be of use. Sequelae include chronic dysesthesia, which may be relieved by amitriptyline.

Ciguatoxin is a lipid-soluble polyether compound¹ that is probably synthesised by specific bacteria after phagocytosis by the benthic dinoflagellate *Gambierdiscus toxicus* (figure 1). The organism adheres to dead coral surfaces and bottom-associated algae. The toxin is then concentrated in the food web as the dinoflagellates are eaten by herbivorous fish species that graze on algae and detritus. These fish are in turn eaten by the larger marine carnivores, which may then be eaten by humans. As the toxin concentration is elevated along the food chain, larger fish are likely to contain

larger concentrations of toxin. We present a case illustrating the acute and chronic manifestations of ciguatera fish poisoning in humans.

Case report

A 42-year-old man presented 3 weeks after returning from Fiji complaining of insomnia, lethargy, dysesthesia of hands and feet, occasional cramps in the calves, light-headedness on standing, and a dry mouth. While in Fiji he had eaten barbecued fish, and fed the remains to a local cat. Within three hours of eating the fish, he developed abdominal pain, watery diarrhoea, and developed a burning sensation of the skin overnight that became very severe the next day while swimming. He then developed increasing shortness of breath and noticed that the cat that ate the fish remains had died. The patient summoned the help of a local who viewed the fish remains, and noted them to be of a species commonly associated with fish poisoning, and suggested he go immediately to hospital. On arrival he was in marked respiratory distress, weak, and experiencing cramps. He required a period of respiratory supportive therapy. The following day he was maintaining adequate oxygenation without support, and was discharged. He returned to his home country. A diagnosis of ichthyosarcotoxaemia, clinically consistent with ciguatera fish poisoning, was made. Dysesthesia of the hands and feet persisted for weeks, but resolved after 5 days on amitriptyline 100 mg daily.

Discussion

Ciguatera fish poisoning can occur in any region where humans eat carnivorous, and occasionally herbivorous, fish derived from tropical reef ecosystems. The toxin is unaffected by cooking, and no protective immunity is conferred by past exposure. In Fiji, *Lutjanus* spp, the Red Snapper or Red Bass (figure 2), and *Plectropomus* spp complex, the Coral Trout, are commonly implicated. The patient identified the Red Snapper as similar in colour and morphology to the fish he had eaten. Species which have been associated with ciguatera poisoning derive from the families Serranidae (groupers), Lutjanidae (snappers), Scaridae (parrot fish), Scombridae (mackerel), Muraeidae (moray eels), Sphyraenidae (barracudas), and Carangidae (jacks). Generally the disease occurs in a circumglobal belt between approximately 35° north and 35° south latitude, and is endemic in the tropical Indo-

Figure 1 Electron micrograph of the benthic dinoflagellate *Gambierdiscus toxicus*. Actual diameter 60 µm. Reproduced with permission of CSIRO, Australia from Hallegraeff¹

Figure 2 An example of *Lutjanus* spp, the Red Snapper or Red Bass. Reproduced with permission of HarperCollins Publishers, Australia, from Marshall¹²

Pacific Islands, and the Caribbean. Surveillance data suggests that incidence rates may be as high as 50 to 500 per 10 000 population per year² in the South Pacific region. The mortality rate is probably low with 0.1% being reported from a series from New Caledonia.³

The mechanism of ciguatera toxin-mediated disease in humans remains unclear. Suggested mechanisms include the 'opening' of sodium channels in cell membranes through the occupation of calcium sites, and both cholinergic and anticholinergic activity have been noted.⁴ The symptoms of ciguatera poisoning are well described,⁴ and include diarrhoea, vomiting, nausea, abdominal pain or cramping, bradycardia, hypotension, tachycardia, arthralgias, myalgias, paraesthesias of extremities, circumoral or facial paraesthesias, temperature reversal, asthenia, headache, pruritis, paresis, diaphoresis, dizziness or vertigo, ataxia, dyspnoea, and cough. As in our case, some symptoms may take weeks or months to resolve.⁵ The diagnosis is usually a clinical one, although it may now be supported by applying a rapid stick-enzyme immunoassay of horseradish and peroxidase-labelled sheep anti-ciguatera toxin antibody to the implicated fish.

Acute management is primarily supportive. Symptoms may be accentuated by the ingestion of alcohol. There is no available antitoxin. Anti-emetics, antidiarrhoeals, and intravenous rehydration are often necessary. Atropine has

Learning points

- ciguatera toxin is synthesised by bacteria phagocytosed by the benthic dinoflagellate *Gambierdiscus toxicus*
- ciguatera toxin concentrates in the food web, reaching particularly high levels in large predatory reef fish

been used for treatment of symptomatic bradycardia.⁶ Intravenous calcium gluconate may have some theoretic benefit in counteracting the inhibition of calcium uptake by excitable membranes.⁷ Pralidoxime chloride has been used as a cholinesterase reactivator based on laboratory data that suggests that ciguatera toxin acts as a cholinesterase inhibitor, although a controlled trial has not been carried out.⁷ Amitriptyline has been shown to provide variable relief from the neurological symptoms associated with ciguatera poisoning, perhaps by membrane stabilisation via sodium channel blockade, or through its anticholinergic activity.⁸ An uncontrolled study using intravenous mannitol 0.5 to 1.0 g/kg in a 20% solution given over 10 to 30 minutes produced immediate resolution of symptoms in most of 24 patients so treated.⁹ The mechanism of action is unclear, but may be related to membrane stabilisation.

Ciguatera fish poisoning is one of a variety of non-bacterial forms of seafood poisoning in humans. Others include paralytic, diarrhoeic, and amnesic shellfish poisoning, all of which are derived from dinoflagellate blooms, puffer fish poisoning due to tetrodotoxin, and clupeotoxism,¹⁰ which is also common in Fiji. Ciguatera fish poisoning may represent an under-reported and under-recognised cause of morbidity amongst travellers. Practitioners should consider including this health hazard amongst topics discussed with travellers prior to departure overseas. Tourists at greatest risk include those going on fishing tours, especially if they are not accompanied by an experienced local guide. Travellers should be warned of the risk and advised to avoid eating large predatory reef fish from tropical waters.

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