Ciguatera poisoning: an increasing occurrence in New Zealand

We wish to draw the attention of healthcare professionals to the occasional cases of ciguatera poisoning in New Zealand citizens. In recent years, the National Poisons Centre (NPC) has received an increasing number of inquiries regarding patients contracting this marine poison following vacations in tropical regions, notably the Pacific islands and the northern regions of Australia. Tourists have become ill following the ingestion of contaminated fish that have been caught around tropical reefs. They may display symptoms while still on holiday or not develop clinical effects until they return to New Zealand; the onset of symptoms can be quite variable ranging from less than 1 hour to up to 48 hours following exposure. Affected people may present to New Zealand medical facilities without any knowledge of the cause of their illness. Their symptoms can be severe and persist for one to two weeks in mild cases, but up to months or even years in some cases. If there is a suspected case of ciguatera, appropriate advice can be sought from the NPC (calling 0800 POISON). The local Health Protection Unit, as agent for the NZ Food Safety Authority (NZFSA), should also be notified.

Ciguatera is a human poisoning caused by the consumption of fish from tropical and sub-tropical reefs. These fish contain heat-stable marine biotoxins called ciguatoxins; they are produced by benthic dinoflagellates, predominantly of the genus *Gambierdiscus*, that are believed to live on dead coral, thriving in a medium rich in algae, fungi, yeast and bacteria. Events such as storms, heavy rains, earthquakes, tidal waves or human activities can cause increased destruction of coral, leading to an increase in ciguatera outbreaks. The toxins and their metabolites are transferred up the food chain as the algae are consumed by herbivorous fish, which are consumed by larger carnivorous fish, which in turn are consumed by humans at the top of the food chain. Ciguatoxins tend to accumulate higher up in the food chain, thereby rendering larger predatory fish more toxic. Cooking will not destroy these toxins and they are resistant to gastric acid.

The hallmark of ciguatera poisoning are gastrointestinal and neurological symptoms; often they occur in combination, but one may be more prominent than the other, depending on the geographical location of the contaminated fish. The initial symptoms typically consist of moderate to severe gastrointestinal effects including diarrhoea, abdominal pain and vomiting. Neurological symptoms commonly include cold allodynia (dysaesthesia when touching cold water or objects), generalised paraesthesia with numbness around the lips and tongue and tingling in the limbs. In addition, muscle weakness, myalgia, arthralgia, extreme fatigue, headaches, ataxia, and dizziness are also reported.

Cold allodynia is almost pathognomonic of ciguatera poisoning but is also reported following exposure to brevetoxin. Other less common effects include dysuria, pruritis, sweating, nonspecific, often macular skin rashes, and hypotension and bradycardia. Although deaths have been reported, ciguatera appears to be rarely
life-threatening; however, symptoms are often quite prolonged and debilitating. Diagnostically, the consistent presence of paraesthesia differentiates ciguatera from most other forms of food poisoning and gastroenteritis.

The various neurological effects are attributed to prolonged activation of the neuronal fast sodium channels, causing excess sodium influx across excitable nerve cell membranes, with prolongation of refractory periods and slowing of nerve conduction velocities in both myelinated and unmyelinated fibres. Associated influx of water may also play a role; axonal oedema and nodal swelling are reported.

The major treatment requirement is typically symptomatic and supportive care, though this is not always highly effective. Intravenous mannitol has been considered an antidote, but a controlled, double-blind, randomised trial has shown no significant difference between administration of saline and mannitol treatment. In the majority of cases, the outcome is good provided the patient receives appropriate supportive care.

Medications such as amitriptyline, fluoxetine, tocainide, gabapentin, nifedipine and corticosteroids have been used with varying degrees of success. Some of these, including amitriptyline, have fast sodium channel blocking activities, which may be useful for persistent neurological effects such as paresthesia, pruritis and dysesthesias. A beneficial effect may not, however, occur in every case. Uncommonly, aggressive fluid replacement and vasopressors are required for severe cases involving cardiovascular collapse.

Summary and recommendations—A diagnosis of ciguatera poisoning should be considered in patients returning from tropical holidays with persistent neurological symptoms, and a history of consuming fish that may have been obtained from reefs. This illness can be debilitating and there is no specific antidote or well proven treatment regimen. Therefore, a greater focus on prevention is required. Practitioners, for example, should consider including a discussion of the hazards of ciguatera prior to travel. Until there is a reliable, widely used method of detecting ciguatoxins in fish, people should be warned to avoid ingestion of large piscivorous fish taken from reef waters in tropical or subtropical regions of the world.

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References: