

Tropical fish poisoning in temperate climates: food poisoning from ciguatera toxin presenting in Avonmouth

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ABSTRACT

Ciguatera toxin causes a range of gastrointestinal, cardiovascular and neurological symptoms that occur within 1–6 h of ingesting fish with the toxin and can last for days, months or years. It is a well-recognized problem in the tropics. Avon Health Protection Team investigated food poisoning on a ship at Avonmouth, which was thought by the crew to be related to a white snapper fish from the Caribbean. The symptoms were initially thought to be scombroid fish poisoning but were consistent with ciguatera fish poisoning. Cases of fish poisoning from fish imported from the Caribbean and Pacific or travellers returning from tropical countries may be ciguatera fish poisoning, but mistakenly diagnosed as scombroid fish poisoning.

Keywords ciguatera, ciguatera toxin, fish poisoning, scombroid fish poisoning

Introduction

Ciguatera toxin disease has long been known as a seafood-linked human disease¹ and is a well-recognized problem in the tropics. Ciguatera toxin, also known as ciguatoxin, is a class of polyether toxins that act on the sodium channels of cells causing changes in their electrical potential and permeability (Fig. 1).

The toxin comes initially from dinoflagellate algae, principally the coral reef species *Gambierodiscus toxicus*. The algae are eaten by herbivorous fishes that absorb the toxin without significant observable effect.³ The toxins remain in all parts of the fish but are concentrated in the viscera, liver and gonads. The concentrations increase higher up the food chain so that the fish with the highest quantity of toxins are those that are large predators such as sharks and barracouda. The act of digestion appears to potentiate the toxicity. The toxin is odourless and tasteless, and contaminated fish taste normal. The ciguatera toxins are heat stable and not destroyed by cooking, freezing or acid.

Ciguatoxins are amongst the most potent toxins known and start to exert their effects at their level of detection at ~0.1 parts per billion. There are three main forms of ciguatera toxin, each of which is found in a separate part of the tropics: Pacific Ocean, Red Sea, Indian Ocean and Caribbean Seas from 35°N to 35°S.

Ciguatera toxin causes a range of gastrointestinal, cardiovascular and neurological symptoms within 1–6 h of ingesting

fish with the toxin. The effects can last days, months or years. The epidemiology of ciguatera-toxin-related disease is given on the CDC website (<http://www.cdc.gov/nceh/ciguatera/default.htm>) and summarized in Table 1. At least 25 000 cases occur each year worldwide. It is the most frequently reported seafood-related disease in Australia, United States, the Caribbean and Papua New Guinea. This incidence is likely to increase with global warming, which is thought to predispose the death of coral reefs, thereby providing nutrient for the dinoflagellates.⁸

Increased overseas travel puts those travelling to tropical areas at risk, and there are increasing numbers of case reports of this disease in travellers returning to non-endemic countries from the tropics.^{9,10} Thus, cases of ciguatera fish poisoning are no longer confined to endemic areas. The increasing consumption of fish as part of a healthy heart diet together with an increase in international exports of large exotic fishes has extended the range of reported human poisonings to more temperate areas of the world. This makes awareness of this entity important.

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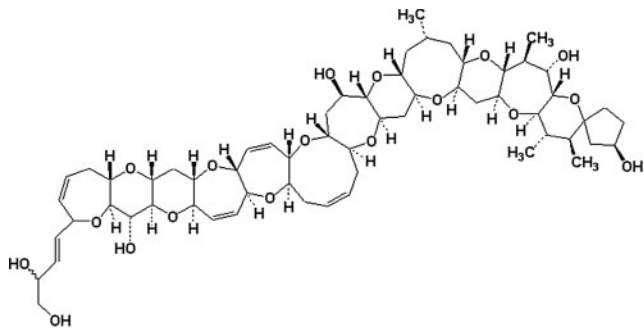


Fig. 1 Ciguatoxin.²

The transmission of symptoms to a baby via breast milk has been reported, as has transmission to a sexual partner via semen during sexual intercourse.¹¹ It has also been associated with transplacental transmission resulting in increased foetal movement but no other adverse effect on foetal development.^{11,12}

On 6 October 2004, Bristol City Council asked the Port Medical Officer (Consultant in Communicable Disease Control) from Avon Health Protection Team to see the crew of a Columbian ship at Avonmouth Port (South West England) who reported food poisoning. The crew thought the symptoms were related to a white snapper fish they had caught from the Caribbean island of St Eustatius on 25 September 2004 and eaten on 27 September 2004.

Methods

Twelve crew members with food poisoning were seen by the Consultant in Communicable Disease Control on 6 October 2004 and were prescribed cetirizine hydrochloride (10 mg). The presenting symptoms were thought to be scombroid fish poisoning, which is a common form of fish poisoning in the United Kingdom.¹³ This was discussed with the London Chemical Hazards and Poisons Division (CHaPD). The advice from CHaPD was that the symptoms should resolve within 8–12 h if untreated. However, the crew's symptoms had persisted for 10 days. This did not accord with the symptoms of scombroid fish poisoning, and snapper was not identified in the CHaPD information about scambrotoxin.¹⁴

A Google web search for white snapper and food poisoning suggested ciguatera poisoning as a possibility.

A fact sheet was written for the crew members, and a questionnaire was given to record the time of onset and duration of symptoms. The ship departed before the questionnaires could be collected. However, the crew members completed and posted the questionnaires.

Results

Ciguatera poisoning was discussed with CHaPD, and their information sheet confirmed that the symptoms, origin of the fish and type of fish were consistent with ciguatera poisoning.

Table 1 What is ciguatera poisoning?⁴

Key points

Origin: Ciguatera poisoning results from ingestion of fish that are endemic to tropical reefs and contain a toxin known as ciguatoxin. Fish most commonly implicated include the groupers, barracudas, snappers, jacks, mackerel and triggerfish

Response: There is wide variation in individuals' response to ciguatera

Onset: Usually 1–6 h, but up to 30 h

Duration: Gastrointestinal effects usually resolve in 1–3 days, and neurological and cardiac effects can last for weeks or months or years in severe cases.

Recurrent attacks of illness may occur for years after the initial poisoning following the ingestion of alcohol or non-toxic fish. These recurrences have been noted as similar to those of myalgic encephalomyelitis (ME).¹ Anecdotal reports of recurrence of neurological symptoms (weakness and muscle aches) have followed the eating of pork and chicken reared on fish meal

Symptoms: Gastrointestinal, cardiovascular and neurological signs and symptoms. Initially, watery diarrhoea, vomiting and abdominal cramps, then neurological symptoms including paraesthesia, headache, dysaesthesia (distortion of sense)—especially touch and hot–cold temperature reversal. Many other effects have been reported including weakness (usually of lower extremities), myalgia, pruritus, arthralgia, malaise, hypersalivation, blurred vision, dysphagia, tremor, ataxia, headache, toothache, metallic taste, chills, sweating, dysuria, dizziness and erythema. Hypotension, bradycardia (rarely tachycardia) and reversible T-wave changes may occur. In severe cases, there may be respiratory depression, muscle paralysis, shock and convulsions. Fatality is uncommon, with a rate judged to be 1.5% (range 0.1–12%).^{5,6} The toxin is transferred to breast milk and can cause symptoms in breast-fed infants⁷

Diagnosis: Based entirely on symptoms and recent dietary history

Treatment: Mannitol is thought by some to reduce the severity and duration of neurological symptoms, if administered within 48 h after symptoms appear⁶

Half of the crew (14 people) had eaten the fish and completed the questionnaire. The crew reported a range of 18 symptoms (Fig. 2). All those who had eaten the fish had diarrhoea and vomiting, and the majority also had painful joints (93%) and a headache (79%). Additional neurological symptoms were reported—e.g. 50% had a tingling sensation in the tongue. The time of onset and the duration of symptoms are shown in Figs 2 and 3. Fifteen symptoms persisted for 5–10 days after eating the fish. A metallic taste and pain in urinating persisted 11 days or more after eating the fish.

Discussion

Main findings

Fish poisoning among crew who had eaten white snapper fish in the Caribbean was initially thought to be scombroid fish poisoning but was found to be ciguatera fish poisoning.

What is already known

Ciguatera fish poisoning is a common form of fish poisoning in the Caribbean and the Pacific but rare in Europe; therefore,

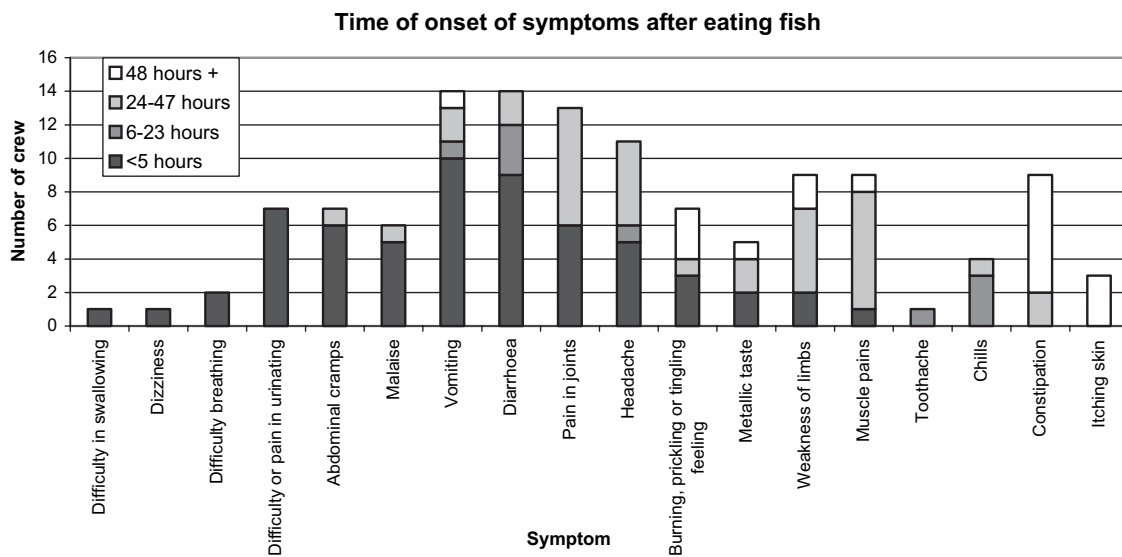


Fig. 2 Graph: time of onset of symptoms after eating the fish.

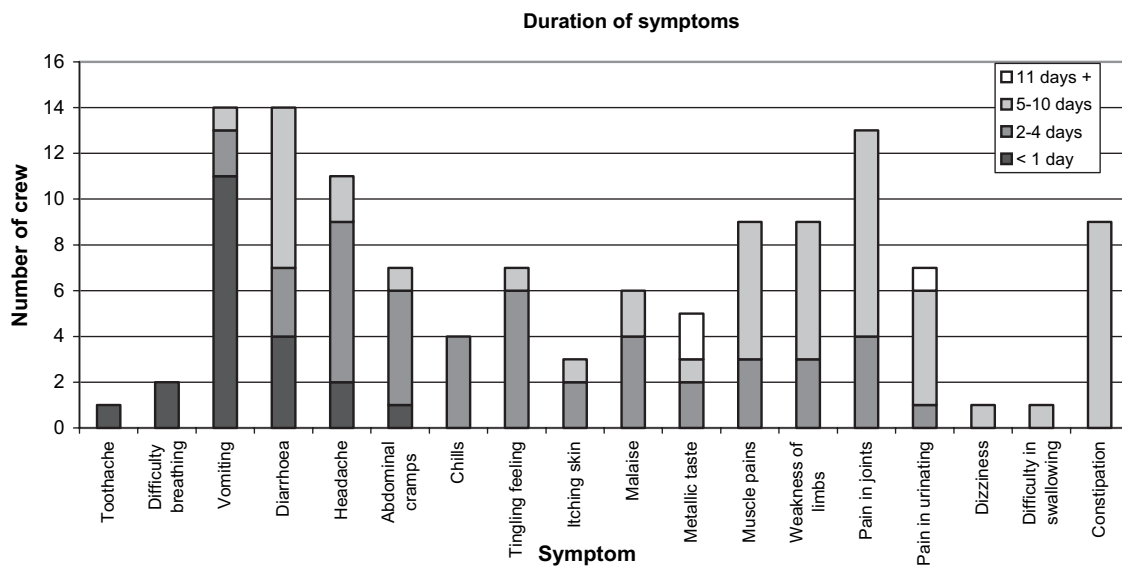


Fig. 3 Graph: duration of symptoms.

it is easy for it to be overlooked as a possible cause of fish poisoning.¹² It is possible that there is under-ascertainment of ciguatera as a cause of fish poisoning. There are many case reports of isolated cases that have undergone extensive neurological tests before the history of a fish meal in the tropics/subtropics was sought and the diagnosis deduced. Toxin assays are being developed but are not sufficiently reliable for general use.

What this study adds

Cases of fish poisoning from fish imported from the Caribbean and Pacific or travellers returning from tropical countries may be ciguatera poisoning, but mistakenly diagnosed as scombroid poisoning, particularly if they present early.

Limitations

The crew completed the questionnaire about the time of onset and the duration of symptoms 11 days after eating the fish. It was not possible to follow the crew to determine how long the symptoms persisted.

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