Ciguatera fish poisoning: also in Europe

Ciguatera fish poisoning is the commonest form of ichthyosarcotoxinism, which results from the ingestion of a wide variety of tropical fish.1 It is endemic in the tropics and subtropics, where its annual incidence varies from 100 to 300/100,000,2 and isolated outbreaks have been reported in temperate countries in which the handled fish are from endemic areas.3 The current trend towards more adventurous eating and increased seafood consumption might favour importation of fish capable of causing ciguatera in western countries. The disease is usually self limiting, but 0-1% to 1% of patients die as a result of heart failure or cerebral oedema.3-10 As simple treatment reduces the intensity and duration of symptoms,11-13 recent cases of ciguatera diagnosed in Paris, and recommend that all neurologists be aware of the diagnostic features of this disease, even in Europe.4

Ciguatera is a highly toxic form of poisoning acquired by the ingestion of fish. The toxins, which are not destroyed by normal storage conditions and cooking, are highly resistant to heat and acid stable. Vitamin B6 is prescribed and the symptoms disappeared in less than a week.

A 45 year old West Indian man who had been living in Paris for more than 10 years received a reef fish purchased from a local street vendor and sent in an isothermal bag by rapid air delivery. The fish was still frozen on arrival (less than 12 hours after mailing) and “looked OK.” It was cooked in the usual way on the same day. All family members became ill within two hours of the meal, with nausea and abdominal cramp. The wife, daughter, and stepmother had mild symptoms that rapidly subsided. The patient, who had consumed a large portion, developed severe diarrhoea and complained of distal paraesthesia and leg weakness. He also experienced “electric shock” and a burning sensation when he touched cold objects. His temperature was 37°C. Paraesthesia triggered by cold contact lasted more than a day. Physical examination, ECG, and laboratory studies were normal. The burning sensation disappeared two weeks later without treatment.

Ciguatera was diagnosed in both cases on the basis of ingestion of tropical fish from endemic regions, the presence of characteristic neurological features, and spontaneous recovery.

Ciguatera is characterised by the onset of gastrointestinal symptoms including nausea (43-5%), abdominal pain (42-5%), and vomiting (38-6%), that usually occur within 12 hours of eating fish and effect last no more than one or two days. Within 12 to 24 hours of onset, the gastroenterological syndrome is followed by bothersome paraesthesia, which is the most common presenting symptoms of ciguatera.4 These paraesthesias are described as either numbness or tingling sensations in the distal extremities (89%) or perioral region (88-1%);10-13) 88-1%) and cold stimuli, which are usually normal, but patellar and Achilles reflexes may be diminished.1 Death occurs in 0-1 to 1% of cases, reflecting individual susceptibility to toxins, increased sensitivity due to prior exposure, and consumption of viscera or larger fish with higher concentrations of toxin.14 Neurological symptoms usually last about a week but may persist for months and even years.15 In two cases of polymyositis developing some years after the onset of ciguatera,1 victims of ciguatera tend to experience sensitisation, and symptoms can recur after the ingestion of canned fish or a raw fish.

The clinical manifestations of ciguatera have been attributed to tiny quantities of an odourless, tasteless, heat, and acid stable toxin, which is unaffected by normal storage conditions and cooking. Ciguatoxin is produced by the photosynthetic benthic dinoflagellate Gambierdiscus toxicus found on macroalgae in the coral reef environment.16 Other dinoflagellates may also play a part in the aetiology of ciguatera, but G. toxicus is by far the most toxic. Other toxins implicated in ciguatera include scartoxin, maatoxin, and palytoxin.17 The toxins are accumulated through the food chain. Herbivorous fish eat G. toxicus in algae, and the toxin concentrates in their viscera and flesh. These fish are then consumed by larger carnivorous fish, which further concentrate the toxins. Humans ingest the toxins by eating the flesh or viscera of carnivorous fish.2 Ciguateric fish cannot be identified by simple inspection, and no simple, reliable test is available for screening purposes.

The paradoxical dysaesthesias are likely to be generated in cutaneous C. polyomodal nociceptor fibres. The intensity of the sensations depends on the charge rate of the spikes, which is a function of the membrane potential and external sodium channels at receptor site 5.4

Manitol infusion has reversed the acute neurological manifestations of severe ciguateric fish poisoning, but the mechanism of action of manitol is unknown, but possibil-
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