Ciguatera fish poisoning: also in Europe

Ciguatera fish poisoning is the commonest form of ichthyosarcotoxicism, which results from ingestion of a wide variety of weak fish. 1,2 It is endemic in the tropics and subtropics, where its annual incidence varies from 100 to 300 100 000,3 and isolated outbreaks have been reported in temperate countries where the handling of fish from tropical and temperate areas has led to ciguatera.4 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.1,3 As simple treatment reduces the intensity and duration of symptoms and subtype recent cases of ciguatera diagnosed in Paris, and recommend that all neurologists be aware of the diagnostic features of this disease, even in Europe.

On his way back to France from a vacation in the West Indies, a 31 year old woman developed nausea and diarrhea eight hours after eating coral trout. Within 12 hours she developed numbness in the extremities, generalized myalgia, and weakness. Thinking she was tired because of the journey, she took a cold shower and experienced an intense burning sensation and tingling discomfort. The same symptoms were triggered by cold contact persisting 48 hours after onset. Neurological examination was normal. Laboratory studies and ECG were normal. Vitamin B6 was 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 store. His blood was typed by rapid cold agglutinins. He was then sent in an isotherm 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 watery diarrhea 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 that last no more than one to two days. Within 12 to 24 hours of onset, the gastroenterological syndrome is followed by aseptic meningitis, and in most cases presenting symptoms of ciguatera.1 These paraesthesiae are described as either numbness or tingling sensations in the distal extremities (89%) or perioral region (81-4%).3 The disease (reversing of temperature perception), which manifests as painful tingling or burning sensations specifically triggered by cold stimuli, are considered almost pathognomonic for ciguatera toxin poisoning.4 Indeed, they occur in more than 87% of cases, and Europeans seem to be more susceptible to them than other ethnic groups.5 Hot stimuli do not generally generate this kind of paradoxical dysaesthesia. There may also be vertigo, ataxia, progressive muscular weakness, paralysis of the limbs and facial muscles, ophthalmoplegia, delirium and, rarely, coma.1,2 Paraesthesiae of the extremities and body temperature are usually normal, but patellar and Achilles reflexes may be diminished.3 Death occurs in 0-1% of cases, reflecting individual susceptibility to toxins, increased sensitivity to toxins due to prior exposure, and consumption of viscera or larger fish with higher concentrations of toxin.4 Neurological symptoms usually last about a week but may persist for months and even years. Reports of two cases of polymyositis developed some years after the onset of ciguatera.4 Victims of ciguatera tend to experience sensitisation, and symptoms may recur after ingestion of canned fish or a full course of antibiotics.5,6

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.1,4 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 scia toxin, maatoxins, and paolytoxins.3 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.7 Ciguateric fish cannot be identified by simple inspection, and no simple, reliable test is available for screening purposes.8

The paradoxical dysaesthesias are likely to be generated in cutaneous C polyomodal nociceptor fibres. The intensity of the sensations depends on the discharge rate of these fibres.9 At the molecular level, ciguatoxin has been shown to cause a prolongation and abnormal influx of Na+ through excitatory amino acid-activating voltage dependent sodium channels at receptor site 5,9

Manitol infusion has reversed the acute neurological manifestations of severe ciguatera. The fish that causes this effect of manitol is unknown, but possibil-