LETTERS TO THE EDITOR

Ciguatera fish poisoning: also in Europe

Ciguatera fish poisoning is the commonest form of ichthyosarcotoxiasm, which results from the ingestion of a wide variety of tropical fish.1 It is endemic in the tropics and sub-tropics, where its annual incidence varies from 100 to 300/100 000,1 and isolated outbreaks have been reported in temperate countries in which the inhabitants eat cold-stored fish.2 4 The current trend towards more adventurous eating and increased seafood consumption might favour importation of fish capable of causing ciguatera in western societies. 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, we describe recent cases of ciguatera diagnosed in Paris, and recommend that all neurologists be aware of the diagnostic features of this disease, even in Europe.

A 54 year old woman was brought to France from a vacation in the West Indies, a 31 year old woman developed nausea and diarrhoea 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 occurred eight hours later, and cold stimulation diminished the unpleasant sensation and discomfort felt when she washed with hot water or handled hot objects. Body temperature was normal. The paraesthesia specifically triggered by cold contact persisted for 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.

Ciguatera is characterised by the onset of gastrointestinal symptoms including nausea (43-5%), abdominal pain (42-5%), and vomiting (36-8%), that usually occur within 12 hours of eating fish and affect 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. These paraesthesiae are described as either numbness or tingling sensations in the distal extremities (89%) or perioral region (20-5%);1 4 but also involve the face (10% of patients), and, in some cases, the trunk and limbs (less frequent).2 4 The patient may experience cold temperature awareness, erythromelalgia, and deep burning pain.2 4 Muscle wasting and weakness of the limbs2 4 are also common; indeed, they occur in more than 80% of cases, and Europeans seem to be more susceptible to them than other ethnic groups.1 Hot stimuli do not generally generate this kind of paradoxical paraesthesia. There may also be vertigo, ataxia, progressive muscular weakness, paralysis of the limbs and facial muscles, ophthalmoplegia, delirium and, rarely, coma.1 4 The same symptoms appear abruptly and body temperature 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 toxicity, increased intake, and the toxicosis due to previous exposure, and consumption of viscera or larger fish with higher concentrations of toxin.1 4 Neurological symptoms usually last about a week but may persist for months and even years, and two cases of polymyositis developed some years after the onset of ciguatera.2 4 Victims of ciguatera tend to experience sensitisation, and symptoms can recur after the ingestion of canned fish or other fish.2 4

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. Ciguatotoxin 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 scartoxin, mahtoxin, and palytoxin.2 4 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 4 Ciguateric fish cannot be identified by simple inspection, and no simple, reliable test is available for screening purposes.2 4

The paradoxical paraesthesiae are likely to be generated in cutaneous C polyomodal nociceptor fibres. The intensity of the sensations depends on the charge rate of these fibres.4 At the molecular level, ciguatoxin has been shown to cause a prolonged and abnormal influx of Na+ through excitable membranes, by sensitising voltage dependent sodium channels at receptor site 5 6. Manitol infusion has reversed the acute neurological manifestations of severe ciguatera.4 Fish and plant toxicity and the mechanism of action of manitol is unknown, but possibilities include competitive inhibition of Na+ at the cellular membrane, diuretic effect eliminating the toxin, and direct chemical detoxification.1 4 Lidocone may also have a therapeutic effect when hooking the Na+ channels altered by ciguatoxin.3 It is also recommended that fish and alcohol consumption are avoided to prevent a worsening or prolongation of neurological manifestations.1 2 Clinicians familiar with ciguatera advise against the use of steroids, opiates, and barbiturates during the acute phase of the disease.1 4

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Botulinum toxin in the management of paradoxical activity of jaw muscles

Paradoxical activity of jaw muscles is a rare jaw movement disorder that occurs after lesions of the trigeminal motor nucleus or the trigeminal nerve.2 4 The disorder includes a progressive jaw opening disability due to unilateral or bilateral distonic jaw muscles. No adequate treatment has been reported.

A 62 year old man with longstanding hypertension was admitted to our intensive care unit with acute dysarthria, diplopia, right sided cerebellar ataxia, and severe left sided hemiparesis. Cranial CT showed an ischaemic brain infarction of the right parietal and lower cerebral hemisphere. Cranial Doppler sonography documented occlusion of the basilar artery. Four months later he became progressively unable to open his mouth. He became unable to eat, speak, or brush his teeth and was confined to tube feeding.

Simultaneous EMG recordings were obtained from the temporal, masseter (jaw closers), and digastric muscles (mouth openers) on both sides. All of these jaw muscles were synchronously activated on
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