Ciguatera fish poisoning: in Europe

Ciguatera fish poisoning is the commonest form of ichthyosarcoxicosis, 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 300,000, 2 and isolated outbreaks have been reported in temperate countries such as the United States and Canada. 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. 4–7 As simple treatment reduces the intensity and duration of symptoms, 8–10 recent cases of ciguatera diagnosed in Paris, and recommend that all neurologists be aware of the diagnostic features of this disease, even in Europe. 11

He was taken 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 discomfit. The same symptoms occurred, triggered by cold contact persisting 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.

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 fishmonger and ate it raw, cooked, or after rapid air delivery. The fish was still frozen upon 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 characterized 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 effect last no more than one or two days. 12 Within 12 to 24 hours of onset, the gastroenterological syndrome is followed by bothersome paraesthesia, and in most common presenting symptoms of ciguatera. 13 These paraesthesiaes are described as either numbness or tingling sensations in the distal extremities (89%) or paresthesia region (88–1%). 8–14 Patellar and Achilles reflexes are usually diminished. 15 They occur in 87% of cases, and Europeans seem to be more susceptible to them than other ethnic groups. 16 Hot stimuli do not generally generate this kind of paradoxic dysesthesia.

There may also be vertigo, ataxia, progressive muscular weakness, paralysis of the limbs and facial muscles, ophthalmoplegia, delirium, and, rarely, coma. 16,17 Headaches, tremor, and respiratory depression are usually normal, but patellar and Achilles reflexes may be diminished. 15 Death occurs in 0.1 to 1% of cases, reflecting individual susceptibility to toxins, increased sensitivity due to previous exposure, and consumption of viscera or larger fish with higher concentrations of toxin. 18 Neurological symptoms usually last about a week but may persist for months and even years. In two cases of polymyositis developed some years after the onset of ciguatera. 19 Victims of ciguatera tend to experience sensitization, and symptoms can recur after the ingestion of canned fish or other fish.

The clinical manifestations of ciguatera have been attributed to tiny quantities of an odorless, 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. 18 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 sciarotoxin, maitotoxin, and palytoxin. 20 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. 16 Ciguateric fish cannot be identified by simple inspection, and no simple, reliable test is available for screening purposes. 20

The paradoxic dysesthesiae are likely to be generated in cutaneous C polymodal nociceptor fibres. The intensity of the sensations depends on the discharge rate of these fibres. 13 At the molecular level, ciguatoxin has been shown to cause a prolonged and abnormal influx of Na+ through the voltage and depolarizing voltage dependent sodium channels at receptor site 5. 4

Manitol infusion has reversed the acute neurological manifestations of severe ciguateric fish poisoning. 21,22 However, 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. 20,22,23 Lidoceaine may also have a therapeutic effect via blocking the Na+ channels altered by ciguatoxin. 20 It is also recommended that fish and alcohol consumption are avoided to prevent a worsening or prolongation of neurological manifestations. 20,23 Clinicians familiar with ciguatera advise the use of steroids, opiates, and barbiturates during the acute phase of the disease.

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the right side when the patient intended to open his mouth but on closing his mouth, no activation of the masseter and temporal muscles could be detected (figure). On the left side, the innervation pattern was normal. Routine needle EMG analysis of the right temporal, masseter, and anterior digastric muscles showed a chronic neurogenic pattern. With the EMG needle in place we injected 75 u botulinum toxin (BOTOX®; Allergan) into the right masseter and temporal muscles. Three weeks after the injection the patient became progressively able to open his mouth, to eat, and to perform mouth care.

The pathogenesis of paradoxical activity of jaw muscles remains uncertain. For this patient we suggest that after a nuclear or axonal trigeminal lesion aberrant regeneration of trigeminal nerve fibres originally supplying jaw opening muscles led to false reinnervation of jaw closers. Thus, while intending to open the mouth, jaw closing muscles were falsely coactivated and prevented sufficient jaw opening. This innervation pattern was confirmed by EMG polygraphy. The interval of four months between infarction and the beginning of paradoxical activity of jaw muscles may reflect the time required for the perinuclear and intraneural regeneration process.

Paradoxical activity of jaw muscles has to be differentiated from other clinically similar disorders. These include hemimasticatory spasm, focal dystonia of jaw closing muscles, and diseases of the temporomandibular joint. Hemimasticatory spasm produces involuntary jaw closure due to unilateral contraction of jaw closing muscles, whereas paradoxical activity of jaw muscles occurs only during intended jaw opening. Jaw closure dystonia, which may occur unilaterally, is often linked to specific tasks or actions, may be accompanied by other dystonic movements, and lacks neurogenic change on needle EMG. Local tetanus can be identified by the absence of a silent period and its permanent muscle activity. Disorders of the temporomandibular joint would be obvious on radiological examination.

Botulinum toxin treatment is recommended as a simple, safe, and effective method in the management of paradoxical activity of jaw muscles.


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Treatment was started after two days and after a further two days she developed generalised, spontaneous, and provoked tonic muscle contractions leading to trismus, opisthotonus, risus sardonicus, and acute respiratory failure. We immediately started symptomatic treatment with mechanical ventilation and continuous midazolam and vecuronium. In addition to sedation and neuromuscular blockade antihypertensive treatment with urapidil and β-receptor blocking agents was necessary between days 2 and 7 and from day 19 until discharge from the intensive care unit. Specific intravenous treatment against causative Clostridium tetani consisted of penicillin G (20 million u/day) and human antitetanus immunoglobulin (10 000 U/day) on days 1 to 5. Antitetanus immunoglobulin was also given via the intrathecal route on days 15 and 16 (2000 U/day).

Complete resolution of the tonic muscle...
Botulinum toxin in the management of paradoxical activity of jaw muscles.

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