

# Rabbitfish (“Aras”): An Unusual Source of Ciguatera Poisoning

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## Abstract

**Background:** Ciguatera poisoning is the commonest fish-borne seafood intoxication. It is endemic to warm water tropical areas and is caused by consumption of bottom-dwelling shore reef fish, mostly during spring and summer. The causative agent, ciguatoxin, is a heat-stable ester complex that becomes concentrated in fish feeding on toxic dinoflagellates. The common clinical manifestations are a combination of gastrointestinal and neurologic symptoms. Severe poisoning may be associated with seizures and respiratory paralysis.

**Objective:** To describe a series of patients who sustained ciguatera poisoning in an uncommon region and from an unexpected source.

**Patients:** Two families complained of a sensation of “electrical currents,” tremors, muscle cramps, nightmares, hallucinations, agitation, anxiety and nausea of varying severity several hours after consuming rabbitfish (“aras”). These symptoms lasted between 12 and 30 hours and resolved completely. The temporal relationship to a summer fish meal, the typical clinical manifestations along with the known feeding pattern of the rabbitfish suggested ciguatera poisoning.

**Conclusions:** The Eastern Mediterranean basin is an unusual region and the rabbitfish an unusual source for ciguatera poisoning. There are no readily available and reliable means for detecting ciguatoxin in humans. A high index of suspicion is needed for diagnosis and a thorough differential diagnosis is essential to eliminate other poisonings, decompression sickness and encephalitis. Supportive therapy is the mainstay of treatment.

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Ciguatera poisoning is the commonest fish-borne seafood poisoning reported in the literature. It occurs mainly in the South Pacific, Caribbean and Indian oceans, particularly in the spring and summer months [1]. More than 400 species of fish in reef and bay areas may become carriers of ciguatoxin as a result of feeding on blue-green algae, protozoa and toxic dinoflagellates [2]. The latter are consumed by small herbivorous fish, which are the major food source of larger carnivorous fish. As a result, ciguatoxin becomes increasingly concentrated in the tissues of larger and larger fish. A common denominator for

ciguatera poisoning is therefore a large fish size [2]. The most common edible fishes implicated include barracuda, sea bass, parrot fish, red snapper, grouper, amber jack, kingfish and sturgeon. They may become poisonous within a matter of hours (with no damage incurred to them) and remain so for long periods, even after cooking [1,2].

The symptoms of ciguatera poisoning in humans include delayed and acute onset of diaphoresis, gastrointestinal symptoms (nausea, vomiting, abdominal pain, diarrhea), headache, dysesthesia, paraesthesia, reversal of temperature sensation, pruritus, bradycardia, dizziness, weakness and myalgia [1,3,4].

In this article we report ciguatera poisoning from an unusual region and an uncommon source – a meal consisting of the popular edible rabbitfish known in Israel as “aras” (*Siganus luridus*).

## Patients

In June 2000, two families consumed rabbitfish for their dinner. The fish had been caught in the Haifa bay area earlier that afternoon using a trammel net and was described as “fatter” than usual. The party consisted of four adults, one couple in their thirties and the other in their fifties, a 19 year old girl and two children aged 3 and 5.

Several hours after the meal they woke from their sleep and complained of a sensation of “electrical currents” in their limbs, tremor and muscle cramps. In the course of the night they were wakened several times by nightmares associated with agitation, anxiety and nausea. In the morning, about 12 hours after the fish meal, they sought medical attention although by that time they were asymptomatic. All patients were conscious, alert and coherent. Pulse, blood pressure, respiratory rate and temperature were within normal limits. Neurologic examination, including mental status, cranial nerves, muscle strength, tendon reflexes, superficial and deep sensation, coordination and gait, was unremarkable. The rest of the physical examination was normal. The parents told their physician that the two children who had eaten much smaller amounts of the fish had not developed any symptoms. The 19 year old girl presented the following day and described symptoms similar to the above, as well as hallucinations and nightmares that lasted for about 30 hours. Her vital signs, neurologic and physical examinations were also unremarkable.

The diagnosis of ciguatera poisoning was made on the basis of the typical clinical manifestations that were temporally related to the consumption of fish caught in a summer month and known to feed on benthic algae. Laboratory identification of ciguatoxin was not possible due to the lack of fish tissue. As spontaneous recovery ensued, no treatment was deemed necessary. Follow-up performed several days later revealed no recurrence of symptoms.

## Discussion

The word "ciguatera" was first used for poisoning caused by ingestion of a marine snail called "cigua" from the Caribbean. The poison is a combination of several toxins, the predominant one being ciguatoxin, with maitotoxin and scaritoxin in lesser amounts [5,6]. There are at least three ciguatoxins, CTX-1, 2, 3 with CTX-1 contributing 90% of the overall toxicity [7]. Ciguatoxin is a heat-stable, lipid-soluble, acid-stable, odorless and tasteless complex ester with a molecular weight of 1,100 D that is stored in the tissues of fish without causing them any harm [1].

The geographic and seasonal occurrence of poisonings is explained by the sensitivity of dinoflagellates to temperature and salinity. Ciguatera outbreaks often follow coral reef disturbances by man-made or natural causes [8].

The mechanism of intoxication is believed to be increased sodium permeability that causes depolarization and stimulation of central or ganglionic cholinergic receptors [4,5,8]. In severe cases, edema of the adaxonal Schwann cell cytoplasm may be responsible for the neurologic manifestations [8]. Animal studies have shown indirect and direct positive inotropic actions on atria and papillary muscle at moderate levels, but negative effects at high concentrations [7]. Poisoning may occur after consuming fresh or frozen fish irrespective of the cooking method used.

The characteristic lag period between ingestion of the fish and appearance of symptoms varies between 2 and 30 hours, usually between 2 and 6 hours. Characteristic symptoms are acute onset of perspiration, abdominal pain, nausea, vomiting and diarrhea, as well as dramatic neurologic symptoms that include headaches, paresthesias and reversal of temperature discrimination. This perception disturbance is the result of exaggerated nerve depolarization in peripheral small A-delta myelinated fibers. Other common manifestations are myalgia (mainly in the legs), ataxia, arthralgia, weakness, and numbness of the tongue, lips, throat and perioral area. Bradycardia, orthostatic hypotension, vertigo, seizures and visual disturbances have been described. Dysuria, dyspareunia and pelvic discomfort may occur in women after sexual intercourse with men who are ciguatoxic. Ciguatoxin crosses the placenta and passes to breast milk. In cases of death, the cause was respiratory paralysis and seizures that were probably managed without adequate supportive therapy [1,3,4,8,9].

Poisonings at varying degrees of severity have been described. This is partly explained by fish size and the presence

of multiple ciguatoxins identified in the same fish. Differences in the clinical response after fish ingestion are also related to individual susceptibility and to the nutritional status of the species. Diarrhea, ataxia, bradycardia and hypotension were more frequently reported when the consumed fish was carnivorous [1,8,9].

Laboratory tests for ciguatera use enzyme-linked immunosorbent assay or high performance liquid chromatography. A rapid test for field use that will allow fish to be tested without processing of the toxin-containing tissues is under development [1,4].

Neuropathy may be evident as the slowing of sensory conduction velocity and prolongation of the absolute refractory, relative refractory and super-normal periods [10]. Normocalcemic latent tetany was reported in mildly symptomatic patients [11]. However, other authors reported normal electromyograph and nerve conduction studies [12].

It is of critical importance in the emergency department to make the differential diagnosis of organophosphate poisoning, carbon monoxide poisoning, botulism, decompression sickness and encephalitis, as well as tetrodon and shellfish poisonings. Diaphoresis is a common clinical finding that helps in the differential diagnosis of ciguatera [1].

The treatment of ciguatera poisoning should begin with the standard supportive care for a toxic ingestion. Spontaneous vomiting and diarrhea are thought to enhance elimination of the toxin. Some benefit may be derived from the administration of activated charcoal if given early enough. Intravenous fluids and electrolytes are essential in case of gastrointestinal fluid loss. Intravenous mannitol, 1 g/kg over 30–45 minutes, may alleviate the neurologic and muscular dysfunction, while gastrointestinal symptoms are less responsive. Mannitol should be used with caution as it may cause hypotension. Various treatments have been used, such as pralidoxime, nifedipine, corticosteroids, calcium gluconate, etc., without significant or consistent success [1,4,8]. Amitriptyline, a tricyclic antidepressant, was reported in several anecdotal cases and in a short series to have a partial, variable or temporary effect on the neurologic symptoms or no effect at all [13–17]. Amitriptyline is known to act in part by blocking sodium channels, and this, as well as possible anticholinergic activity, may be responsible for ameliorating ciguatera symptoms in some cases [18].

Gastrointestinal symptoms usually last for 24–48 hours. Neurologic and cardiovascular symptoms may last days to weeks depending on the amount of toxin ingested. Hiccups and protracted itching were described as delayed manifestations [1].

Countries affected by ciguatera monitor high risk periods by following the occurrence of toxic dinoflagellates. Numbers greater than 100 cells/gram of algae require caution, whereas cell density below 10 cells/g is unlikely to cause ciguatera poisoning [8].

This report is about two families who consumed rabbitfish ("aras") that had been caught several hours earlier and appeared fatter than usual. The temporal relationship to the fish meal, the typical clinical manifestations, together with the

known feeding pattern of the rabbitfish that was caught in a summer month suggested ciguatera poisoning.

Rabbitfish are caught in small quantities in trammel nets in Israel, Lebanon and Tunisia. It feeds on seaweed close to the sea bottom [19]. The only report of poisoning following consumption of this common edible fish was published more than 20 years ago [20]. In 1988, Raikhlin-Eisenkraft et al. [21] described a similar clinical picture after consumption of the saupe (*Sarpia salpa*) caught in the eastern Mediterranean. Interestingly, in that report the saupe had been fried together with a mullet and a rabbitfish, but no symptoms were exhibited by the person who ate rabbitfish. This observation supports the above-discussed variability in the occurrence of symptoms.

Summing up, it is important to address the issue of fish-borne food poisoning with a high index of suspicion. To date there are no readily available and reliable means for detecting ciguatoxins in humans. A thorough differential diagnosis is essential for distinguishing between ciguatera poisoning and intoxications for which antidotes are available. Supportive treatment is the mainstay of therapy.

## References

1. Tunik MG, Goldfrank LR. Food poisoning. In: Goldfrank LR, Flomenbaum NE, Lewin NA, Weisman RS, Howland MA, Hoffman RS, eds. *Goldfrank's Toxicologic Emergencies*. 6th ed. Stamford, CN: Appleton & Lange, 1998:1159–76.
2. Marine animals poisonous to eat: vertebrates. In: Halstead BW, ed. *Dangerous Marine Animals*. Centreville, MD: Cornell Maritime Press, 1980:161–83.
3. Nantel AJ. Food-borne poisonings. In: Descotes J, ed. *Human Toxicology*. Amsterdam: Elsevier, 1996:35:703–10.
4. Food poisoning. In: Ellenhorn MJ, ed. *Ellenhorn's Medical Toxicology: Diagnosis and Treatment of Human Poisoning*. 2nd ed. Baltimore: Williams & Wilkins, 1997:1036–77.
5. Russell FE. Toxic effects of animal toxins. In: Klassen CD, ed. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 5th ed. New York: McGraw-Hill, 1996:801–39.
6. Legrand AM, Galonnier M, Bagnis R. Studies on the mode of action of ciguateric toxins. *Toxicon* 1982;20:311–15.
7. Lewis RJ, Wong Joy AW, McGiffin DC. Action of ciguatoxin on human atrial trabeculae. *Toxicon* 1992;30:907–14.
8. Glaziou P, Chinain M, Legrand AM. Clinical toxicology of ciguatera poisoning. In: Meier J, White J, eds. *Handbook of Clinical Toxicology of Animal Venoms and Poisons*. Boca Raton, FL: CRC Press, 1995:59–74.
9. Bagnis R, Kuberski T, Laugier S. Clinical observations on 3,009 cases of ciguatera (fish poisoning) in the South Pacific. *Am J Trop Med Hyg* 1979;28:1067–73.
10. Cameron J, Flowers AE, Capra MF. Electrophysiological studies on ciguatera poisoning in man (Part II). *J Neurol Sci* 1991;101:93–7.
11. Butera R, Prockop LD, Buonocore M, Locatelli C, Gandini C, Manzo L. Mild ciguatera poisoning: case reports with neurophysiological evaluations. *Muscle Nerve* 2000;10:1598–603.
12. Casanova MF, Sanchez R, Marco R. Ciguatera poisoning [Letter]. *Arch Neurol* 1982;39:387.
13. Lange WR, Snyder FR, Fudala PJ. Travel and ciguatera poisoning. *Arch Intern Med* 1992;152:2049–53.
14. Hampton MT, Hampton AA. Ciguatera fish poisoning. *J Am Acad Dermatol* 1989;20:510–11.
15. Davis RT, Villar LA. Symptomatic improvement with amitriptyline in ciguatera fish poisoning [Letter]. *N Engl J Med* 1986;315:65.
16. Calvert GM, Hryhorczuk DO, Leikin JB. Treatment of ciguatera fish poisoning with amitriptyline and nifedipine. *Clin Toxicol* 1987;25:423–8.
17. Lipkin KM. Ciguatera poisoning presenting as a psychiatric disorder. *Arch Gen Psychiatry* 1989;46:384–5.
18. Swift AEB, Swift TR. Ciguatera. *J Toxicol Clin Toxicol* 1993;31:1–29.
19. Ben Tuvia A. Siganidae. In: Whitehead PJP, Bauchot ML, Hureau JC, Nielsen JG, Tortonese E, eds. *Fishes of the Northeastern Atlantic and the Mediterranean*. Vol 3. Paris: UNESCO, 1986:964–6.
20. Herzberg A. Toxicity of *Siganus luridus* (Rueppell) on the Mediterranean coast of Israel. *Aquaculture* 1973;2:89–91.
21. Raikhlin-Eisenkraft B, Finkelstein Y, Spanier E. Ciguatera-like poisoning in the Mediterranean. *Vet Human Toxicol* 1988;30:582.

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