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CASE REPORT
Other

A case report of cardiac toxicity from barracuda ingestion in Mexico

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Background Ciguatera toxicity is a fish-borne illness that initially manifests with gastrointestinal symptoms, followed by bizarre neurological symptoms including heat-cold sensation alteration, peculiar feeling of loose teeth, and peripheral neuropathy. However, cardiac manifestations are rare and underreported in the literature.

Case summary A 73-year-old man presented with symptomatic bradycardia and hypotension after ingestion of barracuda fish in Mexico. He received atropine and dopamine with subsequent improvement in his symptoms, but continued to experience peripheral neuropathic and other odd sensations. Four of his family members ingested the same fish and had similar symptoms. He was managed conservatively and did not require temporary or permanent pacing. Within 1 week from toxin exposure, bradycardia had improved. Heart rate was 40–50 b.p.m. at rest, and he was discharged with an ambulatory monitor. Heart rate had increased to 77 b.p.m. at 1-month follow-up on repeat electrocardiogram (ECG).

Discussion Although the predominant manifestations of ciguatera toxicity are neurological, cardiac complications tend to be more acute and require attention. Unlike neurological symptoms, bradycardia and hypotension are short-lived, often resolving within a week. Treatment continues to be largely supportive, and patients may require temporary treatment with positive chronotropic agents such as atropine or dopamine.

Keywords Ciguatera • Poisoning • Bradycardia • Case report

Learning points

- Cardiac manifestations from ciguatera toxicity are rare, affecting 10% of cases.
- Bradycardia and hypotension from ciguatera toxicity are managed with atropine and dopamine as needed, and should resolve within 1 week from exposure.
- IV mannitol can help reduce neurologic but not cardiac effects.

Introduction

> 'Without the least suspicion of its being of a poisonous quality, we had ordered it for supper'—Captain Cook

Toxicity from fish ingestion is a common cause of food poisoning both in the USA and worldwide.[1] In 1772, Captain James Cook, a British explorer in the British Royal Navy, described a constellation of gastrointestinal and curious neurologic symptoms following fish ingestion during a voyage across the South Pacific.[2] He spoke in great

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detail about pain, heat, and numbness in his crewmembers' limbs, their extreme weakness, nausea, and a peculiar feeling of loose teeth. Nowadays, we suspect Captain Cook's crew was actually afflicted by two different fish toxins: ciguatera toxin and tetrodoxin.[3] However, lacking from his accounts is the rare cardiac toxicity that also stems from these fish toxins. This report describes a case of a 73-year-old man who suffered from symptomatic bradycardia and hypotension following ingestion of raw barracuda fish in Mexico, requiring the administration of atropine and dopamine. The patient was clinically diagnosed with ciguatera toxicity. Informed consent for publication of this case was obtained per COPE guidelines by the authors who retain the original copy.

Timeline

Time 0	Ingestion of raw barracuda fish.
4–6 h after ingestion	Patient and his four family members develop nausea and diarrhoea.
24–48 h after ingestion	Patient and his family experience paresthesia, cold/hot sensation alteration, and hallucinations.
24 h after ingestion	Patient is admitted to a hospital in Mexico for dehydration and acute kidney injury, and found to have bradycardia with a heart rate of 29. Patient receives IV mannitol with some improvement in neurological symptoms.
2–4 days after ingestion	Patient receives boluses of atropine and dopamine drip for bradycardia and hypotension.
7 days after ingestion	Patient's heart rate is improved to 40–50 b.p.m. at rest, augmenting to 60 with exertion. He is discharged from the hospital with ongoing neuropathy.
1 month after ingestion	Patient is seen in follow-up clinic visit where his heart rate has now increased to 77 b.p.m. at rest.

Case presentation

A 73-year-old male with a past medical history of type 2 diabetes mellitus, hypertension, and stage III chronic kidney disease was vacationing in Mexico when he and his four family members became ill with nausea, vomiting, and diarrhoea several hours after ingesting grilled and raw barracuda fish. Over the following 2 days, they began experiencing a constellation of peculiar neurological symptoms including the sensation of a metallic taste in their mouth, tingling and burning in the extremities, as well as a feeling of intense heat when touching cool objects and an ice cold sensation with warm objects. They presented to a nearby hospital in Mexico where our patient was initially admitted due to an acute kidney injury and uncontrolled blood sugars. On presentation he had a blood pressure of 80/60 mmHg, heart rate of 58, and temperature of 36.3°C. His cardiovascular exam was notable for absence of jugular venous distention, a slow but regular heart rate, normal heart and breath sounds, and no lower extremity oedema. He had a normal neurological and musculoskeletal evaluation. An electrocardiogram (ECG) was then performed (*Figure 1*) which revealed sinus bradycardia with a heart rate of 29 and no atrioventricular delays or block. He had no electrolyte derangements at the time of his bradycardia. He became intermittently lightheaded with pre-syncopal symptoms and also reported shortness of breath. He received intravenous (IV) mannitol therapy, followed by IV atropine for his symptomatic bradycardia. A dopamine drip was initiated for ongoing bradycardia and hypotension. His daily home medications notable for pravastatin 20 mg, metformin ER 1000 mg, losartan 12.5 mg, and aspirin 81 mg were initially held. Two days later, he was air lifted back to the USA and admitted to the cardiac intensive care unit for monitoring. At that time no further cardiac studies were pursued including cardiac enzymes or echocardiogram. No head imaging was performed. During his hospitalization, he had an episode of hypotension with a blood pressure of 83/46 mmHg, which improved to 110/55 mmHg with fluid resuscitation. He otherwise remained free of pre-syncopal episodes and did not require further chronotropic agents. By the time of discharge, the patient noted some ongoing peripheral neuropathy and altered heat and cold sensation. However, his heart rate had improved, now ranging from 40 to 50 b.p.m. at rest, and augmenting to 60 with exertion. He was discharged with an ambulatory monitor. At follow-up 1 month after his discharge, it was noted that his heart rate had increased to 77 b.p.m. at rest (*Figure 2*) and he had remained free of pre-syncopal symptoms or shortness of breath.

Discussion

Our patient presented with sinus bradycardia, hypotension, gastrointestinal symptoms, and altered sensory feeling after ingesting barracuda fish in Mexico. Many marine illnesses resulting from toxic fish present with similar symptoms, but as discussed below, our patient's presentation was most typical for a case of ciguatera toxicity, a diagnosis that must be made clinically. Indeed remnants of the consumed fish can be sent for laboratory testing but there is currently no conclusive diagnostic analysis to confirm ciguatera toxicity in humans.

Ciguatera toxin is the most common fish-borne illness in the world and accounts for the most common non-bacterial type of food poisoning in the USA.[4] While the illness is the result of fish ingestion, the actual toxin is produced by a microalgae called *Gambierdiscus toxicus* found in coral reefs, classically around Hawaii, Florida, and the Caribbean.[1,4] The toxin accumulates along the fish food chain in greater concentrations in the viscera of larger fish, with common culprits being barracuda, snapper, and grouper species.[5] In peripheral nerves, the toxin carries out its effect via competitive inhibition of calcium on voltage-gated sodium channels, which forces sodium channels to remain open and ultimately leads to nerve blockade.[6] Animal studies on guinea pig atria and papillary muscles showed that moderate toxin concentrations had positive inotropic effects, while higher concentrations had direct negative inotropic action.[7] Further studies on human atrial trabeculae suggested that the cardiotoxic effects of ciguatera might actually come from the nerves innervating the heart rather than the cardiac cells themselves.[8]

Classic symptoms of ciguatera toxicity are mainly gastrointestinal and neurologic. Patients typically present several hours (up to 12 h)

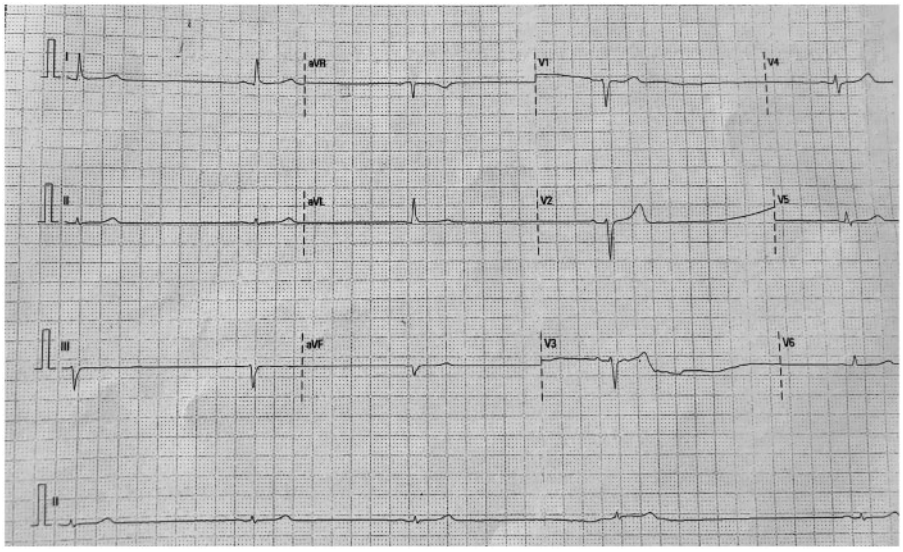

Figure 1 Electrocardiogram performed on patient's initial presentation. Pertinent findings include sinus bradycardia with a rate of 29, no atrioventricular delays or block.

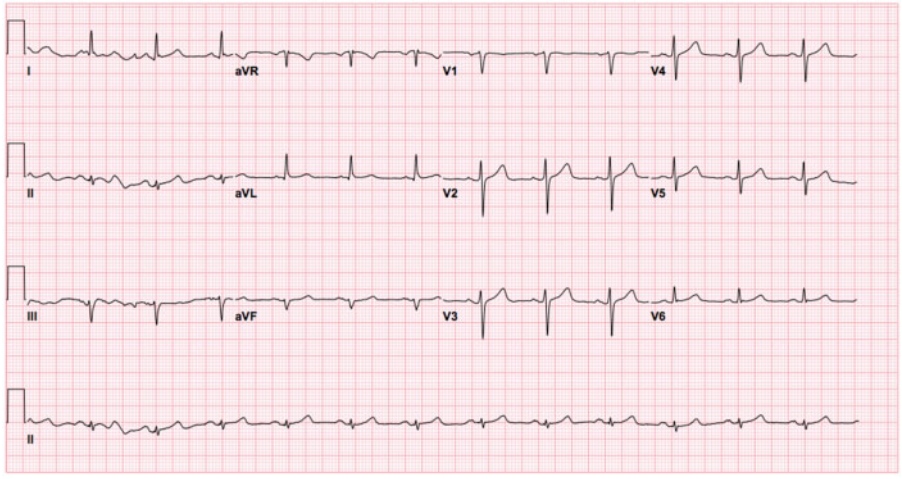

Figure 2 Electrocardiogram performed at 1 month after hospital discharge. Pertinent findings include sinus rhythm with a rate of 77.

after ingestion of the fish with watery diarrhoea, followed by unique sensory symptoms, which vary based on geographical location but include paresthesias, dysthesias, myalgia, arthralgia, pruritis, and/or ataxia (typically within 48 h from saltwater fish ingestion). The sensation of temperature reversal reported by our patient and his family is often pathognomonic for ciguatera.[5]

Our patient's case was of particular interest to us due to his hemodynamic instability and symptomatic bradycardia. The cardiac effects of ciguatera toxin are more rare, occurring in only about 10% of cases, but can be fatal and include bradycardia, hypotension, and arrhythmias.[4] One prospective study of an outbreak in Hawaii[9] found that bradycardia was associated with more severe presentations and correlated with higher amounts of toxic fish ingestion. Patients with repeat exposures of the toxin were also more likely to develop cardiac toxicity[9] though this was not noted in our patient's history. Low body temperatures have been associated with high frequency of cardiac symptoms[10] but again our patient was noted to have a normal temperature on admission. Increased age and body weight have also been correlated to bradycardia,[9] which may have put our patient at increased risk for developing cardiovascular instability.

There are a number of ciguatera toxin mimics, but these often do not present with cardiac side effects. For instance brevitoxin, or 'neurotoxin shellfish poisoning', causes similar paresthesias including the classic temperature reversal and diarrhoea, but is more self-

limiting and patients are not at risk of cardiovascular and respiratory compromise.[11] Severe cases of large amounts of tetrodoxin can lead to hypotension and bradycardia, but can be distinguished from ciguatera by the presence of whole body motor paralysis, fixed and dilated pupils, and the patient's history, which would reveal ingestion of puffer fish rather than large predatory fish.[12]

Treatment for the cardiac toxicity remains supportive. Limited data have demonstrated the benefit of IV mannitol in reducing the length of neurologic symptoms for ciguatera; however, bradycardia does not appear to improve with this therapy.[13] This was further demonstrated by in vitro studies where mannitol had no significant effect on the action of the toxin on human atrial trabeculae.[8] Interestingly, our patient's neurologic symptoms persisted, but they had lessened over several days compared to his family members who only received a single dose of mannitol. Atropine and dopamine are currently used for the management of symptomatic bradycardia and hypotension, as was the case in our patient. While neurologic symptoms can take months to resolve,[5] and indeed persisted in our patient, the cardiac effects appear to be more short-lived and had already improved within 5 days from toxin ingestion.

Conclusion

Cardiac toxicity is a rare but potentially dangerous effect of ciguatera toxicity. Clinicians should familiarize themselves with this clinical picture including its classic gastrointestinal and neurologic features, and travellers should be made aware of the risks. While IV mannitol is recommended for diminishing neurologic side effects, it is not effective in managing cardiac toxicity. Atropine and dopamine should be used for the management of bradycardia and hypotension in these patients, and cardiac toxicity will classically resolve within 1 week from ingestion.

Lead author biography

Alice Haouzi, MD, graduated from Tufts University as Summa Cum Laude for her B.S. in biochemistry and philosophy, where she also completed a thesis in the field of genetics with highest honors. She formed an interest in cardiology at Emory University School of Medicine where she received her MD in 2019. Currently, she is undergoing her residency in Internal Medicine at Beth Israel Deaconess Medical Center, with plan to pursue a fellowship in cardiology.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

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