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Ciguatera Toxicity

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Introduction

Ciguatera toxicity is a food-borne illness caused by eating fish contaminated with Ciguatoxin. This toxin is a potent neurotoxin and it is produced by Dinoflagellates such as Gambierdiscus toxicus. They adhere to algae, coral and seaweed where herbivorous fish eat them. Larger carnivorous fish will suffer a toxin build-up from consuming these herbivores. Ciguatera poisoning is endemic to tropical and subtropical regions mainly in the South Pacific and Caribbean. However, it can be found anywhere fish is consumed. It affects other parts of the world where it is not indigenous through the import of contaminated fish. The fishes that most commonly cause Ciguatera toxicity are Barracuda, Grouper, Moray Eel, Amberjack, Sea Bass, Sturgeon, Parrotfish, Surgeonfish and Red Snapper. [1][2][3][4]

Etiology

Gambierdiscus Toxicus is a Dinoflagellate responsible for the production of various toxins that can cause Ciguatera toxicity. These include Ciguatoxin, Maitotoxin, Palytoxin, Scaritoxin, and Palytoxin. Predator species in tropical waters are most likely to cause Ciguatera toxicity. Barracudas, groupers, moray eels, snapper, and amberjacks are commonly implicated, but it is also found in over 400 species of Reef fish. Ciguatoxin is tasteless, odorless, lipid soluble, and heat-resistant, so normal cooking cannot detoxify Ciguatoxin-laden fish. [1][5][4][2]

Epidemiology

Ciguatera toxicity is the most common worldwide fish poisoning with up to 50,000 cases occur globally every year. This number is felt to be under-reported because most physicians do not realize that it is a reportable disease. While thought to be endemic to the South Pacific and Caribbean, it was recently isolated in the Red Sea and the Atlantic Ocean. It affects 3% of travelers to the endemic regions. It is the most common fish-related foodborne illness in the United States. Ciguatera toxicity is most commonly caused by eating Barracuda, Grouper, Moray Eel, Amberjack, Sea Bass, Sturgeon, Parrotfish, Surgeonfish and Red Snapper. [3][6][7][1][2][8]

Pathophysiology

Ciguatoxin decreases the threshold for opening voltage-gated sodium channels in synapses of the nervous system. Opening a sodium channel causes depolarization, which may cause muscle paralysis, cardiac dysfunction and altered sensation of heat and cold. Cold allodynia is a unique symptom of ciguatera. Cooking or freezing the fish does not prevent Ciguatera toxicity as these methods do not kill the Ciguatoxin. It has no odor. Researchers are looking at the possibility of Maitotoxins playing a larger role in Ciguatera fish poisoning. When introduced to mice by intraperitoneal injection and oral consumption, it caused toxicity in the research trials. Further research is being done on Ciguatoxins by introducing the toxin to human brain-derived cell lines to evaluate its toxicity. [7][1][2][8]

History and Physical

Clinical symptoms of Ciguatera toxicity include gastrointestinal and neurological effects. Gastrointestinal symptoms

include nausea, vomiting, gastric upset, belching and diarrhea, whereas neurological symptoms include headaches, muscle aches, perioral paresthesia, numbness, vertigo, metallic taste in the mouth, blurred vision, ataxia, pruritus and hallucinations. Severe Ciguatera toxicity can cause cold allodynia, which is a perception of burning sensation on coming in contact with cold object. Persistent Ciguatera toxicity may be misdiagnosed as Multiple Sclerosis due to its symptomatology. Dyspareunia has been reported following sexual intercourse suggesting the toxin may be sexually transmitted. Breastfeeding mothers have reported diarrhea and facial rashes in their infants. This supports the theory that Ciguatera toxins are secreted into breast milk. Cardiovascular effects can include bradycardia and hypotension. Cardiac symptoms are only present in the early stages of the toxicity. Alcohol consumption during toxin ingestion has been found to increase the risk of developing bradycardia, hypotension and altered skin sensation. Signs and symptoms can last from weeks to years and usually most recover with an occasional relapse. Relapse may be triggered by consumption of alcohol, nuts, seeds, fish, chicken, and eggs. [9][2][10][4]

Evaluation

Multiple tests are available to detect Ciguatoxin including liquid chromatography-mass spectrometry (LCMS), cytotoxicity assays and receptor binding immunoassays. These tests are not readily available at the time of patient presentation in the emergency room. Routine laboratory testing is often non-specific and rarely helpful. Treatment is based on clinical findings including history and physical examination, as well as disease progression. [3][2][4][11]

Treatment / Management

The treatment of ciguatera poisoning is supportive care. There is no specific antidote for the toxin. If the patient's nausea and vomiting are not severe, activated charcoal may be used in the first few hours of toxicity to prevent further absorption of the ciguatoxin. Antihistamines can be used for pruritus. Symptomatic relief of nausea and vomiting should also be provided. Dehydration can occur due to nausea and vomiting and should be treated with intravenous fluids. If intravenous (IV) fluid resuscitation is not sufficient, then IV vasopressor infusion may be added. There is evidence that calcium channel blockers such as nifedipine and verapamil are useful in treating some symptoms like headache. In rare cases, patients may experience respiratory failure and should be managed by traditional rapid sequence intubation. Symptomatic bradycardia is treated with intravenous atropine. Medications such as amitriptyline may reduce some symptoms, such as paresthesia and fatigue. Steroids and vitamin supplements support recovery but do not reduce toxic effects. Mannitol role is controversial in Ciguatera poisoning as a clinical trial found no difference between mannitol and normal saline. But at the same time many trials have demonstrated improvement of neurologic symptoms after administrating mannitol. A mid approach can be followed by giving only one dose of Mannitol in addition to Normal Saline. [2][4][12][13]

Differential Diagnosis

Diagnosis is often difficult, as physicians do not recognize symptoms and their hesitation to report. Ciguatera has similar symptoms to scombroid and other enteroviruses. Ciguatera can be mistaken for organophosphate toxicity, botulism, multiple sclerosis, Guillain-Barre syndrome and a wide range of other food poisonings. [2][4]

Prognosis

The toxicity of ciguatera is generally self-limiting with symptoms generally only lasting a few days. However, a patient has to be counseled to avoid caffeine, fish, alcohol, and nuts within six months of poisoning as it may trigger a recurrence of symptoms. [2][4]

Enhancing Healthcare Team Outcomes

Patients with ciguatera poisoning initially present to the emergency department. However, because of the lack of a rapid diagnostic test, clinical acumen is required to make the diagnosis. The poisoning is best managed by a team that includes the emergency department physician, infectious disease expert, internist, primary care provider and nurse practitioner.

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After recovery the patient must be warned not to eat fish or related seafood as the symptoms may recur. The outlook for most patients is excellent. [2][4][12][13]

Questions

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