Editorial

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Ciguatera Poisoning and Neurological Effects

JH Lange[1] and Albert V Condello III[2]

1 2366 Golden Mile Highway, Pittsburgh, PA 15239, USA
2 LLC, 619A Waupelani Drive, State College, PA 16801, USA

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Corresponding Author: JH Lange

Jhlange1@hotmail.com

Envirosafe Training and Consultants, 2366 Golden Mile Highway, Pittsburgh, PA 15239, USA

Tel: 412 908-9274

Editorial

Ciguatera poisoning, a form of food poisoning, results from consuming "contaminated" marine herbivorous fish [1]. These fish bio-accumulate heat stable lipophilic toxins (Ciguatera toxins–CTX) belonging to a family of polyether compounds most commonly produced by the dinoflagellate Gambierdiscus [2]. This disease has been reported in all locations within the tropical marine environment, including the Caribbean, Indian, Atlantic and Pacific Oceans. It has been estimated that there may be 50,000–500,000 cases of ciguatera food poisoning (CFP) a year world-wide with an incidence rate in the United States of 5-70 cases per 10,000 people in endemic locations [3-5]. In Hawaii, Jack Fish, Grouper, Surgeon Fish and Snapper constitute the predominant fish species responsible for ciguatera cases, which are generally larger carnivorous fish [6]. The species of fish related to this disease does vary by region, with Barracuda, Grouper and Amberjack being primary contributors for cases in Florida [4]. A common characteristic of the contributing fish species to cases is that they are all "categorized" as reef fish.

Determination of Ciguatera food poisoning (CFP) is based on clinical characteristics of three syndromes: gastrointestinal, neurological, and cardiovascular, along with a history of ingesting fish likely contaminated with CTX [6]. Ideally, CFP is confirmed by laboratory testing of fish remains for the presence of CTX. Poisoning is not a result of improper handling of fish, but rather due to CTX bioaccumulation through the food chain [7].

Symptoms of CFP occur within a few minutes to 48 hours (approximate) upon ingestion of contaminated fish [6, 7]. It has been suggested that even <0.1 ppb of CTX in fish tissue constitutes a risk of poisoning with attack rates around 80-90% [5, 7]. In the course of CFP, gastrointestinal symptoms initially occur followed by neurological events. In rare cases cardiovascular occurrences can be observed [1]. Fatalities from this disease are rare, but has been reported to be in the range of <0.1 to 7 percent [8]. The most common gastrointestinal symptoms reported are abdominal cramps, nausea, vomiting and diarrhea with these events occurring early in the disease process [7, 9]. Neurologically, patients commonly experience paranesthesia of the hands and feet, perioral numbness, headache, pain, weakness, dysesthesia, itching, shakiness, fatigue, hot/cold reversal, and lack of muscle strength [3, 5, 8, 10-12]. Some of the neurological symptoms can persist for months to years [6, 8]. It has been reported neurological symptoms existed for more than a year in one-third of affected patients [6]. It is possible that such neurological symptoms become chronic in some individuals [9]. Cardiological symptoms can include bradycardia, junctional bradycardia, hypotension and arrhythmia [2, 10]. Other symptoms can also result from CTX which include muscular, neuropsychiatric and dermatological problems.

The mechanism of neurological symptoms is related to toxicity of sodium channels through hyperpolizaration [2]. This results in opening of channels during the resting potential, whereas they should normally be closed [13]. These findings are the basis of using mannitol in the treatment of CFP [11]. It has also been suggested that CTX can cause damage to muscle membranes [13].

With a growing demand for fresh fish and potential expansion of the geographic range of this disease due to climate change, a greater concern is need for CFP [12]. It is likely the actual number of cases is greatly underestimated warranting a greater awareness of symptoms and treatment of CFP [4].

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