

Ciguatera fish poisoning: first reported case in Sabah, Malaysia

Heng Gee Lee, MRCP¹, Chui Pin Leaw, PhD², Po Teen Lim, PhD (Fisheries Science)², Steffiana J Jipanin, B.Sc (Aquaculture)³

¹Infectious Diseases Unit, Queen Elizabeth Hospital, Kota Kinabalu, Sabah, Malaysia, ²Institute of Ocean and Earth Sciences, University of Malaya, Kuala Lumpur, Malaysia, ³Likas Fisheries Research Center, Likas, Sabah, Malaysia

SUMMARY

Ciguatera fish poisoning (CFP) is the most common natural marine toxin poisoning worldwide and yet under recognised in Malaysia. We report the first confirmed case of CFP in Sabah with severe neurological, cardiovascular and gastrointestinal manifestations after consumption of emperor snapper. Early recognition of CFP is important because it will result in improved patient care and public health intervention.

INTRODUCTION

Ciguatera fish poisoning (CFP) is the most frequently encountered natural marine toxin poisoning worldwide.¹ Humans acquire CFP by consuming reef fishes contaminated with ciguatoxins (CTXs). Large predatory fishes (snappers, barracuda, groupers, Spanish mackerels, and moral eel) account for majority of cases.² CTXs are produced by microscopic algae called dinoflagellates, in the genus *Gambierdiscus* which are found attached to seaweeds, corals and surfaces in shallow tropical and subtropical waters. The CTXs are tasteless, odourless, colourless, heat stable and stable at commercial freezing temperatures which makes it difficult to detect and prevent. In Malaysia, CFP was first reported in 2010.³ All cases were caused by red snapper consumption. Here, we report the first confirmed case of CFP in Kota Kinabalu, Sabah, Malaysia.

CASE REPORT

A healthy 38-year-old woman experienced generalised pruritus and reported symptoms of temperature inversion in which her hands felt burning hot when touching cold water or cold objects for past three days. She had perioral numbness and burning sensation in her mouth and nose when drinking cold water. Besides, she had postural giddiness and weakness of both lower limbs. Two days prior to that, she bought a large red snapper from the local fish market and consumed it daily with her family. The fish was identified as emperor snapper (*Lutjanus sebae*). On the day of admission, she had malaise, lethargy, nausea, epigastric pain and diarrhoea for five times per day, eight hours after ingesting the fish with her family. Her husband who consumed smaller amount of it, experienced milder symptom with burning sensation over his hands and mouth.

On examination, she was alert, her blood pressure was 84/45mmHg, her pulse was 60 beats per minute, the temperature 37.2°C and the respiratory rate 18 breaths per minute. She had proximal muscle weakness of both lower limbs. Other neurological, cardiovascular, respiratory and abdominal examination were normal. Electrocardiogram (ECG) showed sinus bradycardia. Her white cell count, serum electrolytes, liver function test, cardiac enzymes, and amylase were normal. A diagnosis of ciguatera fish poisoning was made, and she was admitted.

Over the next three days, she had recurrent hypotension and sinus bradycardia to 48 beats per minute necessitating boluses of fluid. She experienced severe headache and pruritic rash over the upper limbs and chest. On day three of admission, she had oliguria, transaminitis, bilateral pleural effusion, ascites and bilateral lower limbs oedema. On day five of admission, the hypotension and sinus bradycardia resolved. Thereafter, signs of fluid overload gradually resolved. She continued to experience generalized pruritus and numbness over the distal extremities upon discharged on day eight. She was advised to avoid fish, nuts, caffeine and alcohol consumption.

A month after symptom onset, she appeared well, had regular pulse and normal blood pressure. She continued to experience intermittent generalized pruritus and burning sensations over the extremities. Findings on neurological examination, nerve conduction study and electromyography were normal. At six months, her symptoms of pruritus and temperature reversal were almost completely resolved, and patient began to reintroduce fish into her diet. Selected ion monitoring (SIM)-Liquid chromatograph mass spectrometry (LCMS) confirmed the presence of CTX in the fish.

DISCUSSION

The diagnosis of CFP should be suspected in a patient with recent fish-eating history especially reef fishes and who has compatible clinical presentation. CFP is characterised by neurological, cardiovascular and gastrointestinal symptoms. The severity of illness reflects the amount, types of CTX ingested, and ingestion of CTX-rich fish parts (head, viscera, roe and skin).²

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Corresponding Author: Dr. Heng Gee Lee

Email: henggee77@gmail.com

Neurological symptoms which occur within first two days of illness include paraesthesia in the hands, feet or mouth, metallic taste, generalised pruritus, headache and dizziness. A distinctive pathognomonic symptom is cold allodynia or “hot-cold reversal”, an alteration of temperature perception in which touching cold surfaces or substances produces a burning sensation.⁴ Neuropsychological symptoms, which occur in the days or weeks after the acute illness, include confusion, reduced memory, difficulty concentrating, depression and anxiety.

The diagnosis of CFP can be challenging. In our patient, the presence of the pathognomonic finding of temperature reversal, together with history of ingestion of emperor snapper and the presence of a similar illness in the husband who ingested the same fish are suggestive of CFP. Poisoning from other marine toxins from different seafood such as paralytic shellfish poisoning, neurotoxic shellfish poisoning, and puffer fish poisoning are excluded with a thorough exposure history of fish ingestion. In addition, these marine poisonings typically present within minutes to few hours of ingestion in comparison to a more delayed onset of few hours to twelve hours on average in CFP (in some cases 24 to 48 hours after ingestion). The absence of flushing and the presence of severe bradycardia make scombroid fish poisoning unlikely in our patient. Illnesses caused by non marine toxins such as organophosphate poisoning and foodborne botulism are excluded by a thorough exposure history.

There is no antidote for ciguatera fish poisoning and treatment is mainly supportive.¹ Patients presented within first few hours after ingestion of fish may benefit from activated charcoal to prevent further toxin absorption. Cardiovascular complications including hypotension and bradycardia can be severe, prolonged and life threatening. Prompt treatment with intravenous atropine, intravenous fluid replacement and inotropic support should be initiated. Occasionally, temporary cardiac pacing and assisted ventilation are required. Intravenous mannitol was previously recommended in CFP management, but a double-blind randomised controlled trial found no benefit compared to placebo.⁵ Amitriptyline and gabapentin are used to treat paraesthesia and pain. Fluoxetine is used to treat neuropsychiatric complications such as anxiety and chronic fatigue.

CTXs are divided based on the regions, example in the Pacific (P-CTX), Caribbean (C-CTX) and Indian Ocean (I-CTX), and based on their chemical structures, with the toxin potential in decreasing order of Pacific-CTX > Indian Ocean-CTX > Caribbean-CTX. Despite no regulatory limits for CTX, US Food and Drug Administration (USFDA) has set the guidance level at 0.01 ppb CTX-1B as the guidance level equivalent.

Globally, annual ciguatera incidence is estimated between 20,000 to 50,000 cases, and many are believed to go unreported or misdiagnosed. In Sabah, which is a popular tourist’s destination for its seafood delights, CFP is still under recognised. Delays in diagnosis will lead to unnecessary investigations and management, including delayed public health intervention. To reduce risk of CFP, the public should avoid eating large coral reef fish especially the head, viscera, roe and skin. However, some small reef fishes can be very toxic based on our current understanding. These pose a challenge in the management of CFP especially in Pacific island countries that depend on fishes as their main source of protein.

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