Ciguatera Fish Poisoning: First Reported Case in Sabah, Malaysia

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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^{0}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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