

# CASE REPORT

## CIGUATERA POISONING IN HONG KONG: A REPORT OF TWO CASES

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Ciguatera poisoning is endemic in the tropical and subtropical areas of the world where large amounts of reef fish are eaten (Bagnis *et al*, 1979; Gillespie *et al*, 1986; Morris *et al*, 1979). Classically, the clinical picture is dominated by a combination of gastrointestinal and neurological symptoms (Gillespie *et al*, 1986; Morris *et al*, 1982). Gastrointestinal symptoms (abdominal pain, vomiting and diarrhea) begin within three to six hours after eating a toxin-contaminated fish and usually persist for one to two days. Neurological symptoms (numbness and tingling of the tongue, lips and the extremities, pain and weakness in the extremities) may appear early in the course, or days after the gastrointestinal symptoms resolve and they may persist for weeks or even months. The toxin (or toxins) responsible for ciguatera is thought to be produced by a dinoflagellate, *Gambierdiscus toxicus* (Bagnis *et al*, 1980), and is concentrated up the foodchains from smaller to larger fish.

We report here an outbreak of ciguatera poisoning in Hong Kong associated with consumption of Mangrove Snapper (*Lutjanus argentimaculatus*) caught in the South China Sea.

### Case 1

A 15-year-old girl was admitted to our hospital in September 1991 with paresthesia and numbness around the lips and in the extremities, and pain and weakness in the extremities. She and other family members were eating a variety of seafood (red snapper, clams, mussels and oyster) in a restaurant on the day of admission. About four

hours after the meal, she had colicky abdominal pain, vomiting and diarrhea, which were soon followed by the neurological symptoms. She also complained of blurred vision and dry mouth. Eight family members, including **case 2**, who consumed the red snapper had similar complaints and 13 others who did not eat the fish were well.

When examined in hospital 12 hours after the meal, the patient looked exhausted with a blood pressure of 100/60 mmHg and pulse rate (sinus rhythm) of 80 per minute. She was alert and oriented. She was found to have mild (grade 4<sup>+</sup>/5) muscle weakness and the reflexes and sensation were otherwise normal. No cranial nerve deficit was found.

Full blood count, plasma electrolytes, calcium, renal and liver function tests and muscle enzymes were all normal except for a mild leukocytosis ( $11.5 \times 10^9/l$ ). Neither cardiac arrhythmia nor respiratory distress was observed during the subsequent illness. She was given intravenous fluids and simple analgesics. All her symptoms gradually subsided over the next two days and she was then discharged from hospital.

### Case 2

This 19-year-old girl had a bigger share of the red snapper than her younger sister (**case 1**) but their symptoms were very similar.

When admitted to hospital at the same time as her sister, she was mildly hypotensive (blood pressure 80/43 mmHg) with a heart rate (sinus rhythm) of 59 beats per minute. Examination of the neurological system revealed minimal (grade 5/5) muscle weakness. Her blood pressure became normalized and remained stable while on intravenous fluids. Complete blood counts, plasma electrolytes, renal and liver function tests and muscle

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enzymes were all normal. When discharged two days later, all her gastrointestinal symptoms had subsided but her neurological symptoms persisted.

She was readmitted three days after discharge because of persistent muscle pain and weakness in the extremities. Physical examination was unremarkable except for a minimal (grade 5/5 power) muscle weakness. Her neurological symptoms gradually improved and she was discharged three days later. When reviewed in the out-patient clinic a few weeks later, she was completely well.

Both patients had gastrointestinal and neurological features typical of ciguatera fish poisoning. **Case 2** who had a bigger share of the fish seemed to have more prolonged neurological symptoms.

Although there has only been one documented report of ciguatera poisoning in Hong Kong (Chan, unpublished observation), this condition is expected to be relatively common as much as the deep water fish consumed in Hong Kong come from the South China Sea. It is likely that most cases in the past were either unreported or misdiagnosed as food poisoning.

Several bioassays and immunoassays have been used to identify the ciguateric fish (Juranovic and Park, 1991), but they are not widely available. Therefore, the diagnosis of ciguatera poisoning is largely made on clinical grounds.

The therapy for ciguatera is essentially symptomatic and supportive measures are usually all that required (Gillespie *et al*, 1986; Morris *et al*, 1982; Russel and Egen, 1991). Gastric lavage should be performed if patient is seen soon after the poisoning. Analgesic, antiemetic and anti-diarrheal drugs can be used for symptomatic relief. Fluid and electrolytes should be replenished. In the presence of hypotension, intravenous crystalloid infusion and vasoactive agents may be required. Atropine sulphate for bradycardia and dopamine infusion for severe hypotension may be life-saving.

Simple advice on preventive measures should be offered to people visiting or living in affected areas (Juranovic and Park, 1991), especially for those with previous episodes of poisoning as subsequent exposure is likely to be increasingly severe. As the toxins are concentrated in the viscera, ingestion of these should be avoided. Large reef fishes such as groupers, barracudas, snappers and jacks should be avoided since they are more likely to be contaminated with the toxin. If ciguatera is suspected, the person should immediately call a physician for treatment.

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