Short reports

Paralytic Shellfish Poisoning in South Eastern Tasmania

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Introduction

Paralytic shellfish poisoning (PSP) is a public health risk worldwide, resulting from the consumption of seafood that has bioaccumulated toxins produced by microalgae.[1] Bivalve shellfish (such as mussels, clams, oysters and scallops) pose a particularly high risk as they feed by filtration, providing a method of toxin accumulation. Algae responsible for causing PSP include several species of the genus *Alexandrium, Pyrodinium bahamense,* and *Gymnodinium catenatum*. Paralytic shellfish toxins (PST) have been recorded in shellfish from Victoria, South Australia, New South Wales and Tasmania (data from state shellfish regulatory programs).

G. catenatum was introduced into Tasmanian waters around 1973.[2] Recurrent blooms form in the Huon River, D'Entrecasteaux Channel, and the Derwent River in autumn and occasionally spring, particularly after calm weather and when water temperatures are above 14°C. PST are commonly referred to as saxitoxins.[3] Saxitoxins bind with high affinity to sodium channels in mammals, blocking nerve and muscle cell membranes. Thus cell action potentials are slowed or suppressed.[1] The binding is reversible.

Diagnosis of PSP is generally based on the onset of neurological signs and symptoms after recent shellfish consumption. Symptoms begin anywhere from 15 minutes to 12 hours after consuming contaminated shellfish, although usually within 2 hours.[1,4] Symptoms begin with tingling and numbness around the mouth and face, progressing to the extremities. This is followed by dizziness, nausea, headache, vomiting, vertigo, a floating sensation, weakness, and muscular incoordination. In severe cases paralysis, difficulty in breathing leading to respiratory failure, and even death can occur.[1,4]

Clinical record

A male fish farm worker developed paraesthesia in his hands, feet, face and tongue approximately 20 minutes after ingesting 12 fresh cooked wild mussels, *Mytilus galloprovincialis,* on 6 April 2011. The mussels were collected from the side of commercial fish cages in Port Esperance, south eastern Tasmania. Informed consent has been obtained to publish this case data.

The patient presented to a regional health centre and was seen by a general practitioner within 3 hours of ingesting the suspect mussels. He was experiencing additional symptoms of clumsiness, limb muscle weakness and vertigo. He had not experienced any difficulties breathing and had an oxygen saturation of 97% on room air as measured by transcutaneous pulse oximetry. His speech was slurred. Haemodynamic observations and physical examination were otherwise normal. The patient was transferred to the Royal Hobart Hospital with a presumptive clinical diagnosis of PSP.

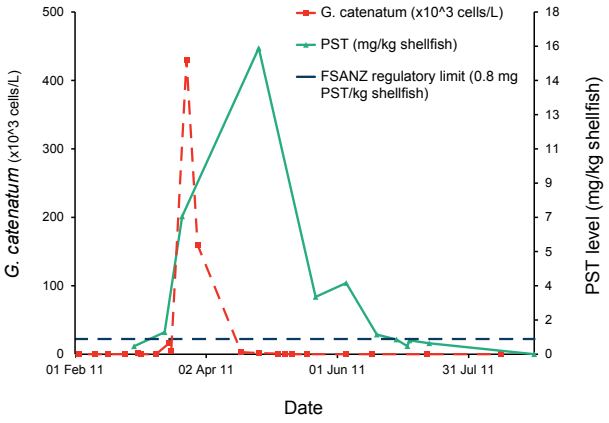
The patient was assessed in the Emergency Department 7½ hours after exposure. He was experiencing an additional symptom of a floating sensation in his limbs. There had been no further progression of his other symptoms. His haemodynamic observations remained normal. He had a peak expiratory flow rate of 210 L/min and his arterial oxygen saturation was 96% on room air. His examination findings included reduced lung air entry and wheeze bilaterally, diplopia with gaze to his left, mildly reduced power bilaterally in flexion of his elbows and fingers, and signs of cerebellar dysfunction (bilateral hypermetria and dysdiadochokinesis). Haematology and biochemistry testing showed no significant abnormality. His chest x-ray was reported as normal. A diagnosis was made of probable PSP but unfortunately no urinary sample was taken for saxitoxin testing. He was observed in the Emergency Department for 8 hours during which his symptoms improved. He was admitted to the medical ward for continued observation overnight and was discharged 31 hours after ingestion.

Discussion

At the time the patient presented, a toxic bloom of *G. catenatum* was present in the Huon Estuary, D'Entrecasteaux Channel and Port Esperance (Figure 1), resulting in high levels of PST in shellfish

Figure 1: Location of the *G. catenatum* bloom in south eastern Tasmania, March to April 2011

Figure 2: Cell counts of *G. catenatum* and corresponding levels of PST in Port Esperance, 1 February to 30 August 2011



from these areas. A public health warning against eating the shellfish due to the presence of PST had been issued on 25 March 2011.

G. catenatum was detected in Port Esperance in early March 2011 (Figure 2). Cell counts rapidly rose above 5 x 10^3 cells per litre (the harvest closure trigger) to 430 x 10^3 cells per litre on 29 March 2011, a week prior to the case presenting. Toxicity in the shellfish (analysed by mouse bioassay) followed the cell counts (Figure 2), exceeding the Australian maximum level[5] of 0.8 milligrams (mg) PST per kilogram (kg) of shellfish meat, by up to 20 times.

To confirm the diagnosis, the US Centers for Disease Control and Prevention recommend laboratory analysis for saxitoxins in either the urine or the seafood.[6] Individual sensitivity to PST varies considerably: the lowest reported doses causing mild symptoms of PSP are between 120 and 304 micrograms (μg) PST per person; and the lowest reported doses associated with severe intoxications or fatalities are between 456 and 1060 μg PST per person.[1,7] In the case presented in this paper, it is probable that the PST intake was between 1218 and 2688 μg (the 12 mussels consumed are estimated to be 168 grams of meat and PST concentrations around the time of illness ranged 7.25 to 16 mg/kg). The patient's symptoms were consistent with moderately severe poisoning.[3,8] Although confirmatory testing was not performed, toxin levels in the nearby commercial shellfish farm, microalgae in the water and a clinically compatible history support the diagnosis of a probable case of PSP.[6]

No previous PSP cases in Tasmania have been clinically confirmed. Only a few anecdotal cases were reported following the consumption of mussels during extensive blooms of *G. catenatum* in 1986 and 1993,[9,10] despite maximum levels of 340 mg PST per kg being detected in shellfish (over 400 times the Australian maximum level). It is possible that the local toxin profile in Tasmania is contributing to the lack of recorded illness. *G. catenatum* in Tasmania contains a high proportion of C-toxins, a group of analogues with considerably lower toxicity than saxitoxin.[11]

Whilst commercial fisheries are covered by shellfish quality assurance programs in each Australian state, recreational harvesting is an unmanaged public health risk. Campaigns to increase public health awareness during bloom periods need to be well considered and targeted. This case highlights that public health departments around Australia should be aware of the risks from recreational harvesting. Clinicians should be encouraged to perform urinary saxitoxin testing when patients present to the emergency department with clinical symptoms suggestive of PSP.

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