Shell Fish Neurological Poisoning

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Citation

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Abstract

Four patients who had consumed shell fish and presented with a distinctive neurological syndrome are presented. They all improved with supportive therapy. Oman has a large fish eating population and adequate health education is essential to inform the public on this rare disease.

INTRODUCTION

Bivalve mollusks (mussels, clams, oysters and scallops)are the usual groups associated with shell fish related poisoning. These shell fish are filter feeders and accumulate toxins produced by microscopic algae in the form of diatoms and dinoflagellates. Four distinctive neurologic syndromes paralytic shell fish poisoning (psp), neurologic shell fish poisoning (nsp), diarroheal shell fish poisoning (dsp) and amnesic shell fish poisoning (asp) have been identified. water soluble, Heat and acid stabile toxins not inactivated by ordinary cooking methods called saxitoxin (psp), brevetoxin (nps), okadaic acid (dsp) and domoic acid (asp) have been identified. Sporadic outbreaks are reported in Europe, Asia, Africa and pacific islands. Red tide and resultant massive kills of various bird and marine animals have become an enormous concern. In addition, infectious agents like hepatitis a, Norwalk virus, vibrio parahemolyticus and vibrio vulnificus can also be transmitted through shell fish ingestion.

Mortality rates in psp and asp vary from 1-12%,nsp and dsp have none. Clinical features start within 15 minutes to 18 hours after ingestion. PSP presents with distal and facial paraesthesias followed by varying degrees of paralysis, ataxia and cranial nerve dysfunction. Occasionally diarrhea is associated. Death is usually due to respiratory failure in the first 12 hours. PSP usually lasts 3 days although paralysis may persist for several weeks.

NSP is milder with more prominent sensory symptoms – paraesthesias of face, trunk, limbs, reversal of hot and cold sensations, myalgias, ataxia, tremors but with less paralysis.allergic manifestations like urticaria,bronchospasm may predominate. DSP presents with short duration diarrheal illness. Only one outbreak of asp in 1987 has been reported. They present with short term memory loss with rare permanent defects although in severe cases ophthalmoplegia, seizures and coma with death in 3% have been reported.

Diagnosis of these cases is based predominantly on clinical features with a temporal relation to shell fish ingestion. enzyme linked immunosorbent assay and liquid chromatography of saxi and brevotoxins can de undertaken in some advanced laboratories. Therapy is supportive with activated charcoal, monitoring of respiratory functions, oxygen and ventilation if needed.

CASE REPORT

A 35 year old Indian working as a poultry farmhand presented with a history of having consumed shellfish (about 200gms)along with six of his colleagues 10 hours prior. 3 hours later he developed severe vomiting associated with dizziness, heaviness of speech, weakness and instability of the body. He found that he could not sit or stand without any support.

There was no h/o headache, loss of consciousness, cranial nerve or sensory symptoms. There was no h/o fever, diarrhea, rashes or dyspnea. No prior such episodes were noted. There were no significant systemic illnesses or addictions. On examination he was conscious, oriented dysarthric with bilateral gross cerebellar signs. Th fundi were normal. No other deficits were noted.

Routine blood and biochemistry and ct brain was normal.

Arterial blood gas showed ph 7.4 , po2 78 mm hg, pco2 41.1 mm hg, hco3 25 mmol/l, base excess 0.2, oxygen saturation 96%. An hour later, three of his colleagues who shared the

meal presented with the same symptoms, One in the same intensity with gross bilateral cerebellar signs, abg showing ph 7.41, po2 81.1 mm hg, pco2 40.8 mm hg, hco3 25.3 mmol/l, base excess 0.7, oxygen saturation 96%. and two milder with minimal ataxic signs, abg showing ph 7.41, po2 103.9 mm hg, pco2 41.3 mm hg, hco3 25.7 mmol/l, base excess 1, oxygen saturation 98% and ph 7.38, po2 110.4 mm hg, pco2 40 mm hg, hco3 23 mmol/l,base excess -1.6, oxygen saturation 98%.

They were treated with iv dextrose saline, 24% nasal oxygen and activated charcoal 50 gm 8 hourly. Arterial blood gases normalized by the second day. Clinically, they made remarkable improvement and by third day all were discharged with no residual dysfunction.

DISCUSSION

These four patients with no major illnesses in the past presented with an acute neurological syndrome –ataxic in type 3 hours after ingestion of shell fish. They responded well to supportive care. The local public health authority in Sur where they come from have been informed to set up a surveillance and follow up in the area.

This syndrome although rare should be kept in mind when a cluster of cases occur with temporal ingestion of shell fish. They have to be managed in a facility where ventilation if needed is feasible. Shellfish is commonly consumed by marine diet loving people residing in Oman and health education is needed regarding its possible deleterious effects.

Adequate health education regarding possible poisoning during shell fish ingestion should be disseminated.

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