

Raw salmon or red herring: ascending paralysis with suspected seafood poisoning

Naren Gunja, Robert P Dowsett and Karl Ng

A 16-year-old boy presented with rapidly progressive ascending paralysis 1 hour after eating raw salmon.

Seafood poisoning was initially considered. Although salmon is not a common cause of toxic seafood poisoning, cases have been reported in the Pacific region. The patient rapidly developed acute left heart and respiratory failure, and investigations revealed a rare tracking intramedullary haematoma of the spinal cord.

Structural abnormalities of the central nervous system may present with acute paralysis and spinal shock, mimicking toxicological syndromes. (MJA 2007; 187: 468-469)

Clinical record

A 16-year-old boy presented to the emergency department with severe epigastric pain, headache and ascending lower limb weakness 1 hour after eating raw salmon. The weakness ascended rapidly over the next hour to involve the upper limbs. In hospital, he was alert, but was soft-voiced and in obvious respiratory distress. He had tachycardia (heart rate, 110 beats per min), hypertension (blood pressure, 205/128 mmHg), tachypnoea (22 breaths per min) and oxygen saturation of 89% (using a non-rebreathing oxygen mask). Although his sensation and mental state appeared normal, he had profound quadriparesis. Pupils were 3 mm in diameter, equal and sluggishly reactive to light. Because of a rapid deterioration in his respiratory status, the patient was intubated, ventilated, and sedated with a propofol infusion before a more complete neurological examination could be completed.

The patient's medical history included thalassaemia minor and a short viral illness 2 weeks previously. He denied using any regular medications or substance misuse.

Initial chest radiography revealed clear lung fields, but, 10 minutes after intubation, pinkish frothy sputum was noted from the endotracheal tube, and there were widespread crackles. An urgent echocardiogram showed severe global hypokinesis with a left ventricular ejection fraction estimated as less than 30%. Laboratory tests revealed neutrophilia, but all other results of a full blood count, serum electrolyte concentrations, renal function, liver function tests and coagulation screen were normal. Serum creatine kinase and creatine kinase-MB concentrations were raised, and peaked the following day at 2716 U/L (reference range [RR], 30–135 U/L) and 10 µg/L (RR, <0.7 µg/L), respectively, suggesting myocardial injury. An electrocardiogram showed sinus rhythm with voltage criteria for left ventricular hypertrophy, but no acute ischaemic changes.

Provisional diagnosis at this stage was a neurological or toxicological aetiology. The fact that the patient's mother also ate the salmon without becoming unwell counted against seafood poison-

1 Magnetic resonance image of the cervical spinal cord



A sagittal T2-weighted image showed a high signal (arrow) within the cervical cord caused by blood breakdown products (intracellular methaemoglobin).

ing but did not exclude it (eg, in puffer-fish poisoning, a specific part of the fish is most poisonous).

To exclude a cervical lesion, magnetic resonance imaging (MRI) of the spine and brain was performed urgently, with the intention of proceeding to nerve conduction studies if MRI results were normal. The MRI showed an extensive intramedullary haemorrhage within the spinal cord, originating at the T7/T8 vertebral level, and extending from the conus caudally to the cervicomedullary junction rostrally (Box 1).

The patient was managed supportively in the intensive care unit, with complete resolution of pulmonary oedema and normalisation of cardiac function by Day 3. A spinal angiogram confirmed the presence of an arteriovenous malformation arising from the anterior spinal cord at the T9 vertebral level. The malformation was excised the following week, and histopathological examination of the resection specimen confirmed the radiological diagnosis (Box 2).

After 6 months of intensive rehabilitation, the patient had restored power in one

arm, but remained paralysed below the T9 cord level.

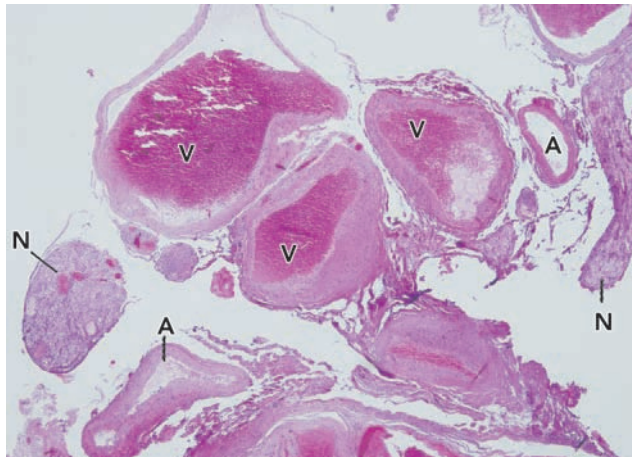
Discussion

Initially, this case represented a diagnostic challenge, with acute onset, rapidly progressive ascending paralysis associated with left ventricular failure, and a history of seafood ingestion. MRI gave the correct diagnosis, obviating the need for peripheral neurophysiological investigations. Useful clues to the diagnosis of myelopathy, such as sphincter dysfunction and the level of sensory loss, were difficult to assess because of the patient's sudden cardiorespiratory deterioration. The initial differential diagnosis is summarised in Box 3.

Toxicological considerations

Poisoning from ingested seafood is a global and increasing problem that should be considered in the emergency department. Seafood associated with medically important poisoning include puffer fish (fugu), ciguateric fish, several types of shellfish and

2 Lesion excised from the spinal cord



Histopathological examination of the resection specimen showed abnormally clustered vessels of venous (V) and arterial (A) type intermingled with spinal nerve bundles (N). This vascular configuration is typical of an arteriovenous malformation. (Haematoxylin and eosin stain; original magnification, $\times 40$.)

mussels, as well as fish from the Scombridae family (eg, mackerel and tuna). Salmon is not known as a major cause of seafood toxin poisoning, although scombroid and ciguatera have been described after salmon ingestion.¹⁻³

Tetrodotoxin from fugu fish causes a rapid descending paralysis and cardiovascular collapse in severe cases.⁴ Paralysis is uncommon in ciguatera, which is caused by a toxin produced by marine dinoflagellates.⁵ Paralytic shellfish poisoning is clinically similar to tetrodotoxin poisoning and typically causes a descending paralysis. Autonomic disturbance can also be a feature of marine neurotoxin poisoning.

Other toxins that can cause paralysis include botulinum, diphtheria and tick paralysis toxin, but these have slower onset of action and produce prodromal symptoms. Organophosphate poisoning is also worth considering: paralysis and copious bronchial secretions consistent with cholinergic toxicity are seen.

Neurological considerations

The hyperacute onset of our patient's symptoms suggested an acute inflammatory demyelinating polyneuropathy, especially in

3 Toxicological and neurological causes of acute paralysis

Ascending paralysis

Toxicological: tick paralysis (*Ixodes* spp.)

Neurological (peripheral): acute inflammatory demyelinating polyradiculoneuropathy (Guillain-Barré syndrome)

Descending paralysis

Toxicological: puffer-fish (tetrodotoxin) poisoning, paralytic shellfish poisoning, snake bite, botulism, diphtheria

Neurological (peripheral): myasthenia gravis

Other

Toxicological: ciguatera, neurotoxic shellfish poisoning, organophosphate poisoning

Neurological (peripheral): myopathies (including myositis and periodic paralysis), acute poliomyelitis and toxic neuropathies

view of the preceding infective illness. Even so, the presentation and progression of the weakness were unusually rapid. Similarly, autoimmune neuromuscular junction disorders, such as myasthenia gravis and Lambert-Eaton myasthenic syndrome, run a more subacute, relapsing course. Periodic paralysis can cause sudden weakness, but there is often a history of recurrent attacks. Toxic and inflammatory myopathies and neuropathic heavy metal poisoning have a more chronic and progressive course.

The cause in our patient was an intradural intramedullary arteriovenous malformation, the most common form of spinal cord vascular lesion in childhood and adolescence.⁶ These lesions can haemorrhage and cause catastrophic autonomic dysregulation accompanied by spinal shock. Complications include neurogenic pulmonary oedema, with the sympathetic storm contributing to transient myocardial impairment via direct neurogenic and humoral mechanisms.

Conclusion

This patient illustrates an uncommon scenario of rapidly progressive flaccid paralysis presenting to the emergency department. Emergency physicians need to consider a broad neurological and toxicological differential diagnosis in the patient with flaccid paralysis. Structural abnormalities of the central nervous system may present with acute paralysis and spinal shock, mimicking toxicological syndromes.

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Competing interests

None identified.

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References

- Dowsett RP. Seafood toxins. In: Wolfson AB, Hendey GW, Hendry PL, et al, editors. Harwood-Nuss' clinical practice of emergency medicine. 4th ed. Philadelphia: Lippincott Williams and Wilkins; 2005: 1710-1714.
- Smart DR. Scombroid poisoning: a report of seven cases involving the Western Australian salmon, *Arripis truttaceus*. *Med J Aust* 1992; 157: 748-751.
- Ebesu J, Nagai H, Hokama Y. The first reported case of human ciguatera possibly due to a farm-cultured salmon. *Toxicon* 1994; 32: 1282-1286.
- Isbister GK, Kiernan MC. Neurotoxic marine poisoning. *Lancet Neurol* 2005; 4: 219-228.
- Swift AE, Swift TR. Ciguatera. *J Toxicol Clin Toxicol* 1993; 31: 1-29.
- Krings T, Mull M, Gilsbach JM, Thron A. Spinal vascular malformations. *Eur Radiol* 2005; 15: 267-278.

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