

CASE REPORT



'Mass allergy': Acute scombroid poisoning in a deployed Australian Defence Force health facility

David Ian Ward

SO1 (Med), Headquarters 1st Division, Gallipoli Barracks, Enoggera, Queensland, Australia

Abstract

On the last night of disaster relief operations in Sumatra, Indonesia, a mass casualty event occurred that involved deployed Australian Defence Force personnel. Symptoms of acute urticaria, angioedema, wheeze and gastrointestinal upset were experienced to varying degrees by 16% of the deployed element. The present report describes a presumed scombroid poisoning cluster and demonstrates the difficulties of operating in a deployed environment, the confusion that might be associated with evolving non-kinetic mass casualties, and provides a learning opportunity for an unusual mass casualty incident.

Key words: *histamine, scombroid.*

Introduction

In November 2009, an Australian Defence Force medical team assisted the earthquake recovery efforts in Sumatra, Indonesia. This Health Support Element (HSE) was drawn mainly from the First Health Support Battalion, and was collocated with an engineering team at a remote village called Sungai Gerringging. It was tasked to provide primary health care to the local population while a temporary clinic was constructed.

The HSE was of necessity 'light' in personnel, stores and equipment, comprising primary health care, resuscitation, and a two-bed medium dependency unit for Australian Defence Force personnel. There was no imaging or pathology capability, and no holding capacity for civilian patients.

A mass casualty incident occurred on the last night of operations in Sungai Gerringging, involving seven of

the 42 deployed personnel (16%). It occurred after humanitarian aide operations had concluded and medical supplies packed for return to Australia.

The present report demonstrates the difficulties of operating in the deployed environment, the confusion that might be associated with evolving non-kinetic mass casualty incidents, and provides a learning opportunity for an unusual mass casualty event.

Case description

Over a period of 40 min, seven casualties presented with various signs and symptoms similar to a generalized allergic reaction (Table 1). All seven cases were fit and healthy soldiers between 20 and 35 years of age, without prior medical history or allergies. All were fully vaccinated and taking doxycycline for malaria prophylaxis.

Correspondence: Associate Professor David Ian Ward, 4 Barrett Place, Everton Park, Qld 4053, Australia. Email: david_ian_ward@hotmail.com

David Ian Ward, MBBS MPH & TM FACEM FACTM pjrcsp, Lieutenant Colonel.

Table 1. Patient presentations during this event

Patient number	Time of arrival	Symptoms and signs	Management
1	19.30 hours	Generalized urticaria, no respiratory or gastrointestinal tract symptoms	200 mcg adrenaline i.m.i. 25 mg promethazine p.o. 150 mg ranitidine p.o. 200 mg hydrocortisone i.v.i. 1000 mL 0.9% N/Saline i.v.
2	19.50 hours	Truncal urticaria, oral angioedema, hoarseness and dysphagia	200 mcg adrenaline i.m.i. 25 mg promethazine p.o. 150 mg ranitidine p.o. 200 mg hydrocortisone i.v.i. 1000 mL 0.9% N/Saline i.v.
3	19.50 hours	Urticaria and erythema, abdominal cramping and profuse watery diarrhoea	(required 200 mcg adrenaline i.m.i. 4 h later) 200 mcg adrenaline i.m.i. 25 mg promethazine p.o. 150 mg ranitidine p.o. 200 mg hydrocortisone i.v.i. 2000 mL 0.9% N/Saline i.v.
4	20.00 hours	Urticaria and erythema, wheeze (no stridor)	200 mcg adrenaline i.m.i. 25 mg promethazine p.o. 150 mg ranitidine p.o. 4 mg dexamethasone i.v.i. 1000 mL 0.9% N/Saline i.v. 1 mg nebulized adrenaline
5	20.00 hours	Urticaria and erythema, dysphagia, abdominal pain	(required 200 mcg adrenaline i.m.i. 6 h later) 200 mcg adrenaline i.m.i. 25 mg promethazine p.o. 150 mg ranitidine p.o. 200 mg hydrocortisone i.v.i. 2000 mL 0.9% N/Saline i.v.
6	20.00 hours	Urticaria and erythema, headache	200 mcg adrenaline i.m.i. 25 mg promethazine p.o. 150 mg ranitidine p.o. 4 mg dexamethasone i.v.i. 1000 mL 0.9% N/Saline i.v.
7	20.10 hours	Abdominal pain, nausea, vomiting and diarrhoea	200 mcg adrenaline i.m.i. 25 mg promethazine p.o. 150 mg ranitidine p.o. 200 mg hydrocortisone i.v.i. 1000 mL 0.9% N/Saline i.v.

i.m.i., intra-muscular injection; i.v., intravenous; i.v.i., intra-venous injection; p.o., per oral.

The presentations covered a broad spectrum of histaminergic signs and symptoms – urticaria, facial and oropharyngeal oedema, hoarseness, wheeze and gastrointestinal upset (include crampy pain and watery diarrhoea). None of these fit young soldiers displayed significant hypotension or hypoxia.

Treatment for all patients included the use of intramuscular adrenaline (and nebulized for the patient with marked respiratory signs), oral promethazine and ran-

itidine, and i.v. steroids. Those patients with significant abdominal discomfort received titrated doses of morphine for analgesia, and Case Seven also received 4 mg i.v. ondansetron.

A generally rapid response was observed, with most symptoms and signs resolving over 15–30 min. Cases Two and Four required repeated doses of 200 mcg intramuscular adrenaline four and 6 h after the initial doses.

Military and logistic aspects of response

The arrival of the first casualty raised no alarms; however, Patient 2 informed the staff that 'some others' were also unwell. The index of suspicion for a mass casualty incident became high.

Concurrent clinical and administrative action was instigated, involving: (i) establishing assessment, treatment and holding areas within the treatment facility (unpacking stores and predrawing medications); (ii) an area search to locate other casualties and account for all deployed personnel; and (iii) early notification via the chain of command to generate increased resources and evacuation. Administrative and clinical personnel were alerted to the potential for large numbers of acute or critical casualties.

The initial six patients were treated one-on-one by medical staff, whereas the search for a causative agent was intense. All six had been playing with local children on a wet, muddy field the day before, and infective agents were considered. All six had moved that day into a lightly damaged accommodation building, and poisoning was considered a possibility (and the building evacuated). An ingested toxin was also considered; however, a common causative agent was unable to be identified at this time.

Whereas clinical management and causative investigations were being conducted, casualty evacuation was requested to the HMAS (Her Majesty's Australian Ship) Kanimbla which was at anchor offshore. Medical staff aboard were listening to the radio communications, and had conducted an internet search for possible causative agents. The possibility of scombroid toxicity was communicated to the HSE, and further questioning revealed that each patient had eaten three or more servings of the locally contracted fresh meal, which included a white-meat dish thought to be fish. (This dish was affectionately known as 'ficken' because of the inability to distinguish the type of white meat it contained). It was also found that about a dozen other personnel had mild nausea and abdominal cramping; however, no one else required treatment. A diagnosis of presumed scombroid toxicity was made.

Operational constraints precluded safe evacuation, and so the treatment area was converted into a holding ward. Given the limited holding and monitoring capacity of the HSE, a team of Navy doctors, nurses and medical technicians were deployed to assist with casualty management overnight. This team arrived around 3.5 h after the first casualty and brought additional

monitoring equipment and supplies of adrenaline and steroids, which by that time were almost completely exhausted.

The following morning, all seven patients were evacuated to HMAS Kanimbla for further observation, with several patients requiring additional antihistamine and steroid therapy. All patients made a full recovery.

Discussion – scombroid toxicity

Scombroid poisoning is caused by the ingestion of histamine and other biogenic amines in spoiled fish.^{1,2} Initially thought to be related to ingestion of fish from the Scombridae and Scomberesocidae families (tuna, mackerel, bonito), it has now been described in many other non-scombroid fish (mahi-mahi, bluefish, sardines and others). It has also been described after ingestion of some cheeses (e.g. Swiss).³ The fish is often described as having a peppery or metallic flavour, although in this incident it was well hidden by the Indonesian-style preparation.⁴ Sporadic single and multiple cases of scombroid poisoning have been reported in the medical literature throughout the world, including Australia.^{5–11}

The mechanism for the formation of these biogenic amines is not certain. It is known that bacterial histidine decarboxylase can act on endogenous histidine in fish flesh to form histamine, and storage of caught fish at <16°C can reduce this bacterial activity.¹² Causative organisms include *Proteus* sp., *Clostridium* sp., *Escherichia* sp., *Salmonella* sp. and *Shigella* sp.¹³ Neither histamine nor histidine decarboxylase activity is reduced by freezing or cooking.^{2,12,14} The levels of histamine in contaminated fish corresponds to the severity of the syndrome, and although the concentrations of histamine and other amines in the flesh might be variable, consumption of larger amounts of scombrotoxic fish produces more symptoms.^{9,14} It appears that the histamine in ingested spoiled fish is more toxic than equivalent doses of ingested 'pure' histamine, possibly reflecting the role of the other biogenic amines.¹⁵

Scombroid toxicity is generally mild and self-limiting; however, life-threatening toxicity (including death) has been described.^{16–18} The patients in this outbreak tended towards the more severe end of the spectrum, consistent with ingestion of large amounts of biogenic amines.

Symptoms of scombroid generally occur within 1 h of the ingestion, and commonly include erythema, pruritus, urticaria, gingival burning, nausea, vomiting, abdominal cramps, diarrhoea, headache, tachycardia

and palpitations. More rarely patients can develop angioedema, bronchospasm, dysphagia, transient visual loss and hypotension.^{4,18} Although it might be misdiagnosed as anaphylaxis or fish allergy, a cluster of cases (as in this instance) virtually rules out these alternatives.

Discussion – military and logistic aspects

The remote location of these events affected patient management. The threshold for aggressive treatment (including intramuscular adrenaline) was somewhat lowered, demonstrated by the early use of adrenaline as part of the treatment for Case one (urticaria). His excellent response probably influenced the treatment of the other cases, although those with more generalized allergic symptoms and signs would likely have received adrenaline anyway. In this population of predominantly healthy, fit, young men, the risk-benefit ratio is heavily skewed in favour of its use. Adrenaline is an effective antidote, whatever the source of the histamine!

The planned capacity of the HSE was exceeded by this event. Stores of adrenaline and steroids were rapidly depleted. Only two patient monitors were available, allocated to those patients with the most significant symptoms and signs, whereas the rest were nursed one-on-one under constant observation. The requirement to re-establish resuscitation bays and then expand a two-bed medium-dependency unit into a seven-bed high-dependency unit, followed by the loss of one of the senior nurses to scombroid, severely tested the capability of the deployed medical element. The positive outcome for all patients was a credit to those involved in their management.

The timing of the event further challenged the HSE and chain of command. Most of the stores and equipment had been packed, with two medical packs available for first aid. These packs alone were insufficient for this event; however, the staff were able to rapidly regenerate a critical care capability and manage the crisis.

The management of mass casualty events in the deployed environment involves concurrent clinical and administrative activities. Casualty identification, regulation and tracking, early reporting and constant high-level communications are the key to successful patient outcomes. The ability to rapidly regenerate capability and reinforcement is one of the key benefits of serving in the military on these types of operations. In this

instance, with 16% of the deployed force affected by scombroid, managing the event was as important as managing the patients. In combination with early, aggressive clinical treatment, it contributed to the positive outcome for all concerned.

Conclusion

Scombroid poisoning is a cause of histaminergic signs and symptoms following ingestion of contaminated fish. A high index of suspicion needs to be maintained if multiple casualties present with typical features, as it can be life-threatening. Early and aggressive treatment might be the key to positive outcomes, especially in an isolated and remote environment.

Competing interests

None.

Received 23 August 2001; revision received 1 October 2001; accepted 24 October 2001

References

1. Morrow JD, Margolies GR, Rowland J, Roberts LJ 2nd. Evidence that histamine is the causative toxin of scombroid-fish poisoning. *N. Engl. J. Med.* 1991; **324**: 716–20.
2. Staruszkiewicz WF, Barnett JD, Rogers PL, Benner RA Jr, Wong LL, Cook J. Effects of on-board and dockside handling on the formation of biogenic amines in mahi-mahi (*Coryphaena hippurus*), skipjack tuna (*Katsuwonus pelamis*), and yellowfin tuna (*Thunnus albacares*). *J. Food Protect.* 2004; **67**: 134–41.
3. Taylor SL, Stratton JE, Nordlee JA. Histamine poisoning (scombroid fish poisoning): an allergy-like intoxication. *J. Toxicol. Clin. Toxicol.* 1989; **27** (4–5): 225–40.
4. Aurbach PS, Robert NL. *Disorders Caused by Reptile Bites and Marine Animal Exposures*. Harrison's Online, Access Medicine from McGraw-Hill. [Cited 15 Dec 2009.] Available from URL: <https://www-accessmedicine-com.cknservices.dotsec.com/content.aspx?aID=2898087&searchStr=scombroid+fish+poisoning#2898087>
5. Brown C. Scombroid poisoning. *Med. J. Aust.* 1993; **158**: 435–6.
6. Smart DR. Scombroid poisoning. A report of seven cases involving the Western Australian salmon, *Arripis truttaceus*. *Med. J. Aust.* 1992; **157**: 748–51.
7. Hall M. Something Fishy: six patients with an unusual cause of food poisoning. *Emerg. Med.* 2003; **15**: 293–5.
8. Guly HR, Grant IC. Case of the month: lesson of the week: don't forget scombroid. *Emerg. Med. J.* 2006; **23**: 955–6.

9. Feldman KA, Werner SB, Cronan S *et al.* A large outbreak of scombroid fish poisoning associated with eating escolar fish (*Lepidocybium flavobrunneum*). *Epidemiol. Infect.* 2005; **133**: 29–33.
10. Eckstein M, Serna M, Delacruz P, Mallon WK. Out-of-hospital and emergency department management of epidemic scombroid poisoning. *Acad. Emerg. Med.* 1999; **6**: 916–20.
11. Muller GJ, Lamprecht JH, Barnes JM, De Villiers RV, Honeth BR, Hoffman BA. Scombroid poisoning. Case series of 10 incidents involving 22 patients. *S. Afr. Med. J.* 1992; **81**: 427–30.
12. Lehane L, Olley J. Histamine fish poisoning revisited. *Int. J. Food Microbiol.* 2000; **58** (1–2): 1–37.
13. Russell FE, Maretic Z. Scombroid poisoning: mini-review with case histories. *Toxicon* 1986; **24**: 967–73.
14. Patrick DJ. eMedicine from webMD. [updated 08 Apr 2010][Cited 15 Dec 2009]. Available from URL: <http://emedicine.medscape.com/article/818338-overview>
15. Taylor SL. Histamine food poisoning: toxicology and clinical aspects. *Crit. Rev. Toxicol.* 1986; **17**: 91–128.
16. Borade PS, Ballary CC, Lee DKC. A fishy cause of sudden near fatal hypotension. *Resuscitation* 2007; **72**: 158–60.
17. Iannuzzi M, D'Ignazio N, Bressy L, De Sio A. Severe scombroid fish poisoning syndrome requiring aggressive fluid resuscitation in the emergency department: two case reports. *Minerva Anestesiol.* 2007; **73**: 481–3.
18. Burke WA, Tester PA. Skin problems related to non-infectious coastal microorganisms. *Dermatol. Ther.* 2002; **15**: 10–17.