

Fatal envenomation by jellyfish causing Irukandji syndrome

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TO THE EDITOR: Interpretation of the report describing the first death attributed to the Irukandji syndrome should be tempered by the fact that significant unstated assumptions have been made in attributing the cause of death to a jellyfish.¹ While envenomation by a jellyfish remains the likely diagnostic possibility, no evidence is presented that unequivocally confirms a jellyfish as the lethal agent.

Several methods could have been used to support or confirm the diagnosis of jellyfish envenomation, including sampling of nematocysts from the victim's skin (before or after death), jellyfish capture, or reports of other similar, but less severe, stings from the same beach around the time the victim was stung.

In severe jellyfish envenomation, attempts are often made to harvest nematocysts from patients' skin, most commonly by skin scraping or by sticky tape sampling.² Recovered nematocysts may help to identify the species, and confirm the diagnosis.³ Although successful nematocyst recovery is uncommon in Irukandji syndrome, it is disappointing that "no attempt was made to sample nematocysts"¹ given the relative simplicity of the procedure and the importance of this case. The authors state that "no sting site was clearly delineated",¹ but then go on to say that there were, in fact, areas of "skin flushing and intermittent diaphoresis"¹ over a significant period of

time. Sticky tape sampling of these areas may have yielded nematocysts, allowing positive species identification.

Postmortem skin sections have also been employed in *Chironex fleckeri* fatalities, and have shown nematocyst barbs on the victim's skin.⁴ Postmortem examination may also have revealed other contributing factors.

I am particularly interested in the assertion that almost every Irukandji syndrome patient in the Whitsundays develops a "rise in cardiac troponin levels".¹ In fact, the cited article makes no mention of troponin, simply stating that CK-MB (creatin kinase isoenzyme) levels "can be abnormal",⁵ and that "some severe cases [of Irukandji syndrome] may have a CK-MB [level] well above the normal range".⁵

Many aspects of the diagnosis and treatment of jellyfish envenoming remain controversial. Accurate reporting of unusual cases is thus of the utmost importance.

1. Fenner P, Hadok JC. Fatal envenomation by jellyfish causing Irukandji syndrome. *Med J Aust* 2002; 177: 362-363.
2. Currie BJ, Wood YK. Identification of *Chironex fleckeri* envenomation by nematocyst recovery from skin. *Med J Aust* 1995; 162: 478-480.
3. Taylor McD D, Pereira P, Seymour J, Winkel KD. A sting from an unknown jellyfish species associated with persistent symptoms and raised troponin I levels. *Emerg Med (Fremantle, WA)*. 2002; 14: 175-180.
4. Little M. Is there a role for the use of pressure immobilisation bandages in the treatment of jellyfish envenomation in Australia. *Emerg Med (Fremantle, WA)*. 2002; 14: 171-174.
5. Fenner P, Carney I. The Irukandji syndrome. A devastating syndrome caused by a north Australian jellyfish. *Aust Fam Physician* 1999; 28: 1131-1137. □

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TO THE EDITOR: In their Notable Case on jellyfish envenomation causing Irukandji syndrome,¹ Fenner and Hadok suggest that similar deaths may have

occurred in the past, with the relationship to Irukandji syndrome not being recognised. However, their call for urgent research into developing an antivenom needs to be based on a clear understanding about the risk of death. Unfortunately, they have not presented data that clearly establish causation or support their conclusions about treatment and the need for further research.

While they reported a history that supports envenomation, there was no confirmation by detection of nematocysts or autopsy to examine for other causes of death. An alternative explanation could be that the patient was overcoagulated and died from complications of an intracerebral haemorrhage.

While it is clear that blood pressure must be monitored, to suggest that it must be treated with phentolamine is not supported by this case report. Clearly, supportive management and, in particular, the optimal treatment of cardiovascular complications needs to be defined and may obviate the need for antivenom.

1. Fenner PJ, Hadok JC. Fatal envenomation by jellyfish causing Irukandji syndrome. *Med J Aust* 2002; 177: 362-363. □

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IN REPLY: While overcoagulation causing intracerebral haemorrhage could have caused the death of the patient we described,¹ he was normotensive until developing signs and symptoms of Irukandji syndrome some 20 minutes after being stung. The Irukandji syndrome is, and always has been, a clinical diagnosis only. Biochemical and pathological test results become abnormal later, but are not diagnostic — actual cause and effect have been described only once, with the experiment unlikely to be repeated.²

Nematocyst studies, while established for *Chironex fleckeri*,^{3,4} have never identified species associated with Irukandji syndrome, except *Carukia barnesi*, which appears to occur in the Cairns area only. Other species probably cause the more severe syndrome seen in the

Correspondents

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There should be no more than 5 references. The reference list should not include anything that has not been published or accepted for publication. Reference details must be complete, including: names and initials for up to 4 authors, or 3 authors et al if there are more than 4 (see mja.com.au/public/information/uniform.html#refs for how to cite references other than journal articles).

Whitsundays and on the Great Barrier Reef, where these deaths occurred.⁵ One of us (PJF) is possibly the only person to have captured specimens likely responsible for causing Irukandji syndrome from the Whitsundays, and the species remain unidentified, as they are a new species and not described to date. Also, when the moribund patient was admitted, no obvious sting site was visible, and a negative skin scraping would not rule out a jellyfish sting.

Phentolamine has previously proved effective for relieving distressing autonomic symptoms,⁶ and not just for cardiovascular complications, although it appeared ineffective at the lower doses used in our patient. However, nothing appears to prevent toxic cardiac dilatation occasionally occurring later in the syndrome.⁷ Further research is currently under way.

Antivenom development may prevent some (possibly all) major symptoms of Irukandji syndrome. However, production is impossible until sufficient specimens of all species (some six to 10) causing the syndrome are caught and their venom assessed. Such advances are many years away and may never be achieved with current poor levels of funding.

Cardiac markers for jellyfish envenomation have previously been identified.^{5,8} Since 1999 troponin level has replaced creatine kinase isoenzyme (CK-MB) level, and both are invariably raised in patients stung by the Whitsunday jellyfish. Thus, the words "cardiac markers" should have been used in the article and for not doing so I apologise.

Despite *C. barnesi* stings being common at north Cairns beaches, it has taken six years of dragging the beaches, with nets to catch jellyfish of this species. The thought of trying to catch a 12 mm jellyfish that makes erratic and irregular appearances in several hundred square kilometres of ocean around the Whitsunday Islands is totally daunting, but the possibility is being assessed. Such a venture will depend on funding becoming available.

Other stings were reported in the area at the time of our patient's death and are well known at the resorts where people who have been stung in surrounding areas are taken for treatment. However, stings remain erratic; they have no pre-

dictable patterns of appearance, and unfortunately prophecy is currently impossible.

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Chemical-biological-radiological (CBR) response: a template for hospital emergency departments

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TO THE EDITOR: The article by Tan and Fitzgerald¹ raises numerous concerns. The authors report that their recommended personal protective equipment (PPE) conforms to standards "in a hospital environment where the chemical vapour concentration will not be high". At the same time, the authors acknowledge data indicating most patients from a disaster will present to the local hospital by private transport (ie, without triage, decontamination, or prehospital care). These two considerations are incompatible and further ignore the possibility of the hospital as a direct terrorist target. The authors' assertion that their three decontamination lines "allow mass casualties, as well as trolleys and equipment, to be decontaminated quickly, efficiently, and in an orderly fashion" is simply not evidence based.

Of greater concern, the authors report "major considerations were policies and plans [referring to the hospital External Disaster Committee] and the emergency department response". Although this bottom-up approach to disaster planning is typical, it pays inad-

equately attention to interdisciplinary issues of proper hazard identification and management, environmental health, syndromic surveillance, and field outbreak investigation. Readers seeking robust emergency department templates are better referred to other sources for guidance.^{2,3}

Of greatest concern, the authors report "our recommendations are similar to systems in the US and Israel, but much less intensive, as the threat of a terrorist attack here is perceived to be much lower". The three references cited for that statement date back to 1994, with none more recent than 1999. Moreover, the logic of the unreferenced threat assertion confuses hazard and risk. Although the absolute probability of a given hazard may be low, the risk attending that hazard encompasses vulnerability of the exposed population. With weapons of mass destruction, the conditional probability of catastrophic public health consequences is high — one event is the only number you will ever need.

The current public health context of chemical-biological-radiological (CBR) incident management in Victoria is one of limited experience, performance improvement indicators, and budgetary support from public health authorities. Public health is at risk when authorities report that "faced with dozens of requests each day to attend sites to assess white powder, the stretch capacity did not exist and nor should it".⁴ As a result, the leading trauma centre in Australia extracts \$20 000 from its existing operations budget to discharge its CBR responsibilities. This is not good enough. Nevertheless, the authors deserve credit for their initiative. Until cross-trained and disaster-experienced healthcare authorities reprioritise, this article shows the reader an excellent way to play a very weak hand.

1. Tan GA, Fitzgerald MCB. Chemical-biological-radiological (CBR) response: a template for hospital emergency departments. *Med J Aust* 2002; 177: 196-199.
2. Association for Professionals in Infection Control and Epidemiology, Inc, and Center for the Study of Bioterrorism and Emerging Infections. Mass casualty disaster plan checklist: a template for health care facilities. October 2001. Available at <http://www.apic.org/bioterror/checklist.doc>
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