Correspondence

Jellyfish responsible for Irukandji syndrome

Sir, 

Irukandji syndrome is a distressing array of symptoms following a jellyfish sting. Generally, symptoms develop 20–60 min after the sting, and include back pain, nausea, abdominal cramps, sweating, hypertension, tachycardia and a feeling of impending doom. The sting usually leaves only mild local signs. In a series of 116 cases presenting to Cairns Base Hospital in one year, 64% required hospital admission and there was one death. Patients suffer severe pain, as demonstrated by the adult patients in this series requiring a mean dose equivalent to 42 mg of morphine. There have been case reports of patients developing life-threatening cardiac failure requiring intubation and inotropic support. In Huynh’s series, 22% had evidence of myocardial injury, with an elevated troponin.

Reports of Irukandji syndrome have come from Australia, Hawaii, Florida, French West Indies, Bon Air, Caribbean, Timor Leste and Papua New Guinea. The syndrome may well occur in many other parts of the world, but not be recognized. Only two jellyfish have previously been definitively shown to cause Irukandji syndrome. In 1961, Barnes captured two small jellyfish (later named Carukai barnesi in his honour) in Palm Cove, Australia, and demonstrated this to be the casual agent by stinging the local lifeguard, his 9-year-old son and himself. All three developed Irukandji syndrome. Despite reports of Irukandji syndrome from many locations only one other, as yet unnamed, jellyfish has been identified as causing Irukandji syndrome. Very little is known about the venom or ecology of these jellyfish.

We now report five further cases of identified cubozoan jellyfish that we believe can cause Irukandji syndrome (Table 1), namely Alatina nr mordens, Carybdea alata, Malo maxima, Carybdea xaymacana and an as-yet unnamed ‘fire jelly’. Accurate identification of creatures responsible for human envenoming is essential. In Australia, the white-tailed spider, Lampona sp, was incorrectly identified as being responsible for necrosing ulcers. This led to much anxiety, misdiagnosis and incorrect treatment of patients, until a prospectively designed study demonstrated that this spider’s bite caused only minimal local symptoms. There have been very few data on jellyfish stings that correlated clinical syndromes with accurate identification of the offending jellyfish; published studies have often assumed that if a cubozoan jellyfish is found in a region, it is responsible for the envenoming syndrome, but with little data to support such assumptions. If research is to be performed on both the ecology and venom of the animal, it is essential the correct creature is identified.

We report five further cases of identified cubozoan jellyfish that we believe can cause Irukandji syndrome (Table 1), namely Alatina nr mordens, Carybdea alata, Malo maxima, Carybdea xaymacana and an as-yet unnamed ‘fire jelly’. Accurate identification of creatures responsible for human envenoming is essential. In Australia, the white-tailed spider, Lampona sp, was incorrectly identified as being responsible for necrosing ulcers. This led to much anxiety, misdiagnosis and incorrect treatment of patients, until a prospectively designed study demonstrated that this spider’s bite caused only minimal local symptoms. There have been very few data on jellyfish stings that correlated clinical syndromes with accurate identification of the offending jellyfish; published studies have often assumed that if a cubozoan jellyfish is found in a region, it is responsible for the envenoming syndrome, but with little data to support such assumptions. If research is to be performed on both the ecology and venom of the animal, it is essential the correct creature is identified.

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Table 1  Details of envenomings

<table>
<thead>
<tr>
<th>Date, patient details</th>
<th>Location</th>
<th>How jellyfish located</th>
<th>Symptoms</th>
<th>Patient outcome</th>
<th>Identified jellyfish</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oct 2002, 34M and 22F</td>
<td>Osprey Reef, near Cairns, Australia</td>
<td>Collected by envenomed patients when stung</td>
<td>Local pain, nausea and abdominal cramps</td>
<td>Symptoms settled within 12 h</td>
<td>Alatina nr mordens, bell height 100 mm, tentacles 1000 mm</td>
<td>Has 6 eyes, (not 2 or 4) and jellybean-shaped statoliths, suggesting these are not A. mordens⁶</td>
</tr>
<tr>
<td>Apr 2001, 28M</td>
<td>Roebuck Bay, Western Australia</td>
<td>Collected by envenomed patient when stung</td>
<td>Back and abdominal pain, nausea</td>
<td>Symptoms settled within 6 h</td>
<td>Malo maxima, bell height 50 mm</td>
<td></td>
</tr>
<tr>
<td>Oct 2000, 37M and 24F</td>
<td>3 nautical miles offshore from Waikiki Beach, Hawaii, USA</td>
<td>Collected by envenomed patients when stung</td>
<td>Abdominal, knee and ankle pain, nausea and sweating</td>
<td>Symptoms settled in 2 h</td>
<td>Carybdea alata, bell height 70 mm</td>
<td></td>
</tr>
<tr>
<td>Apr 2000, 19F</td>
<td>Briggs Reef nr Cairns, Australia</td>
<td>Captured at time of sting by dive buddy</td>
<td>Back and body pain, sweating, nausea, hypertension</td>
<td>Hospital admission for 2.5 days. Developed abnormal ECG and elevated troponin (5.4 mcg/l, N &lt; 0.7). Recovered Overnight admission, opiate analgesia. Recovered</td>
<td>Carybdea xaymacana, bell height 40 mm</td>
<td>Distinctive pink/purple nematocysts on bell, 4 strap like tentacles 50 mm length when contracted</td>
</tr>
<tr>
<td>Dec 1997, 5 children aged 4–10</td>
<td>Inside Palm Cove nets, Cairns, Australia</td>
<td>60+ jellyfish captured at time of stings at the north and south edge of an enclosed netted swimming area</td>
<td>Back pain, nausea sweating hypertension</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

⁶ A. mordens is a type of jellyfish that is known for its venomous stings. The comment suggests that the jellyfish identified in this case may not be A. mordens due to its unique characteristics.
References

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‘Mobile phone sign’ in early vitamin B12 deficiency

Sir,
I greatly enjoyed the letter from Kozak and Schattner regarding ‘Mobile Phone Sign’ in early vitamin B12 deficiency. I would, however, like to make one comment. I note that the patient reported in the letter was treated (successfully) with vitamin B12 injections, which he continues to receive monthly.

In patients such as this, where the aetiology is dietary deficiency of B12, rather than pernicious anaemia or other gastrointestinal pathology, the physiological mechanisms for absorption of B12 should remain intact. Thus oral, rather than parenteral, replacement of B12 may well suffice.

Obtaining suitable oral preparations is often difficult, but many years ago (as a Senior House Officer at St George’s Hospital in London) I was taught a trick that proved effective in a vegan patient who had presented with very severe macrocytic anaemia. We supplied the patient with ampoules of B12, gave instructions as to how to open the ampoules, and advised him to sprinkle the contents into a meal on a weekly basis. This was effective, despite obvious concerns as to ‘bio-availability’.

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Reference

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