Marine Bites and Stings

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Major box jellyfish (Chironex fleckeri) sting

The major box jellyfish, Chironex fleckeri, is the most dangerous jellyfish in the world. In Australia it has killed 67 people. The last 10 deaths in the NT have all been children in remote coastal communities, with the last Australian death being in a six-year-old child at Yarrabah, near Cairns. The death is quick, usually within 20 minutes of the sting, and the patient often succumbs on the beach. The mode of death is thought to be a cardiac death.

The major box jellyfish is found throughout tropical Australia, with cases being reported from Gladstone (Qld), through to Broome in WA. The greatest numbers of cases occur in the NT. In the NT, major box jellyfish stings have occurred in every month of the year, except one, where as in Qld the jellyfish season is said to be October to May. It is believed that these jellyfish breed in the estuaries then migrate to the ocean, being helped by the rainfall of the wet season. They have never been found in the offshore waters around coral reefs, only in coastal regions.

The stinging mechanisms, the nematocysts, are located on the tentacles of the jellyfish. They are like spring-loaded syringes, which explosively discharge toxic venom when activated, usually by contact but also by remote factors. The venom of the major box jellyfish has not yet been extensively characterized, although it is believed to have a cardiotoxic effect, dermatonecrotic effect (skin damage) and haemolytic effect, although this has not been demonstrated clinically.

Clinically, the patient screams with sudden severe pain and has welts on his or her skin. There have been reports of patients then running from the water and collapsing with a cardiac arrest. There are at least three reports of patients arresting on the beach but surviving with prompt CPR. In the latest case — near Cairns in 2002 — a child was pulseless after a major box jellyfish sting and survived with prompt CPR performed on the beach.

Although the literature has many cases of death reported, most cases of major box jellyfish sting are minor. In a 12 month prospective study looking at all cases of jellyfish sting presenting to the Royal Darwin Hospital there were no deaths, none required antivenom and only one case of the 28 with major box jellyfish sting required opiate analgesia. Most did well with symptomatic treatment.

The management of patients stung by the major box jellyfish is attention to airways, breathing, and circulation, ensuring it is safe for the first aiders. Vinegar should be applied for at least 30 seconds to the sting site.

As the patient may be in pain, ice packs to the sting site may help. If not, oral or injectable analgesics may need to be given. If the patient is in severe pain or is unstable then antivenom is recommended.

Antivenom to the major box jellyfish (C. fleckeri antivenom CSL), was first made available in 1970. From 1970–81 CSL reported 73 patients receiving antivenom. It is made from sheep serum

and seems to have a low rate of allergic reaction. The current NT indications for use of antivenom are:

a. severe pain unrelieved by opiate analgesia;
b. life threatening cardiac or respiratory decompensation, or cardiac arrhythmia.

The antivenom can be given IM (dose of three ampoules) but it is preferable to give intravenously. The intravenous dose is one ampoule diluted 1:10 in normal saline or Hartman's solution, given over 10 minutes. If the patient is in cardiac arrest then CPR should be continued, and IV antivenom given as a quick push. Up to six ampoules should be given.

Current experimental work from Qld, as well as a review of the literature, supports the NT current practice of not applying pressure immobilization bandages (PIB) for any jellyfish sting. There does not appear to be any evidence to support its use and laboratory work suggests that PIB increases the venom load delivered to the stung patient.

The sting should be treated as a burn to avoid secondary infection. Delayed reactions to major box jellyfish stings are common, with patients developing an itchy rash 7–14 days after a sting. This is thought to be a delayed hypersensitivity reaction and responds to topical steroid cream and oral antihistamines.

Irukandji syndrome

Much interest in Irukandji syndrome has occurred over the last few years, heightened in 2002 with the death of two tourists in north Queensland. Interestingly, both died from intracerebral haemorrhage, believed due to the initial hypertension seen with Irukandji syndrome.

Irukandji syndrome was first described by Flecker, a Cairns radiologist, in 1952. He described that after a short (up to 60 minutes) interval after a seemingly innocuous sting patients became unwell, began vomiting, and suffered significant back, loin, limb and chest pain and sweating. Patients have also been noted to be hypertensive and tachycardic. In 1961 Barnes captured two jellyfish, now called Carukia barnesi, and demonstrated that these caused the Irukandji syndrome by stinging himself, the duty lifeguard and his nine-year-old son. All three developed Irukandji syndrome and were taken to Cairns Base Hospital for treatment. Since 1987 there have been reports in the literature of patients with Irukandji syndrome developing pulmonary oedema, with some patients requiring ventilation and inotrope support. It is now believed that more than one jellyfish is responsible for Irukandji syndrome. Cases have occurred across tropical Australia, although the majority of cases are reported from the Cairns region.

Nothing is known about the venom, and there is no antivenom. There are no significant data about the biology of the C. barnesi or any other jellyfish that may be responsible for the Irukandji syndrome.

In a one-year review of 62 Irukandji syndrome patients presenting to EDs in Cairns, 56% presented within a three week period in December. The majority (76%) were stung at coastal locations, and 61% required parenteral analgesia to relieve the pain. Other than two patients who developed pulmonary oedema and needed critical care admission, all patients were discharged home pain-free by 24 hours after admission.

It appears that a significant percentage of patients with ongoing pain from Irukandji syndrome develop evidence of cardiac injury. In the summer 2001–02 Cairns Base Hospital treated 116 patients with Irukandji syndrome. Of these 22% developed abnormal troponin results (a marker for cardiac injury). This group of patients also had abnormal ECGs, and eight had abnormal cardiac echocardiographs showing reduced cardiac function. Reassuringly, 36% of all patients

presenting to ED with Irukandji syndrome were sent home that day, with another 47% being discharged the next morning. Only 17% needed ongoing care in hospital, and all of these had ongoing analgesia requirement.

First aid management involves the use of vinegar to the sting site. This prevents any further nematocysts (the jellyfish stinging cells) releasing more venom. Pressure immobilization bandages (PIB) are not recommended as research suggests that PIB may increase the amount of venom introduced into the body.

These patients will be in severe pain and will need regular doses of opiod. There is debate about which one to use. There are theoretical grounds not to use pethidine, however morphine or fentanyl have been used. The patients who seem to develop cardiac dysfunction have ongoing pain, and will need to be transferred to a centre where further investigations can be performed. Pulmonary oedema seems to present within 10 hours of the sting, however there are reports of patients developing this within three hours of a sting. These patients may require ventilation and adrenalin as an inotrope. Clearly these cases are rare, and expert opinion should be sought.

Stonefish sting

The Synanaceia genus is found throughout tropical Australia, as well as in the Indo-Pacific region. It is a particularly unattractive fish that is well camouflaged in coral or as a piece of rock. Usually the unwary person either stands on the stonefish, or picks it up.

The venom apparatus of the stonefish are the dorsal spines, with each spine having two lateral venom glands. This system is for defense of the stonefish and has nothing to do with capture of prey. When the stonefish is threatened the spines become erect. If an object, such as a foot, is pressed down on the dorsal spine then the venom gland has the venom forced into the object. The venom has been demonstrated to have neurotoxic, myotoxic, cardiotoxic and cytotoxic effects on experimental animals, however the major effect on humans is one of pain, probably as a result of enzymes in the venom.

There have been no reported deaths from a stonefish sting in the Northern Territory, although deaths have been reported in the Indo-Pacific region (e.g. the Seychelles), and there is a report of a death in 1915 at Thursday Island, Queensland.

In a one-year period (July 1989 to June 1990) there were 25 cases of stonefish antivenom usage reported to CSL. Four of these cases were from the NT, with the majority being from Queensland

Patients who have been stung by a stonefish know so almost immediately. They present with severe local pain, often with the entry site of the spines visible. The pain is often worsened by the application of a pressure immobilization bandage or tourniquet, and therefore is not recommended.

The initial management should involve placing the affected limb in hot water, ensuring that the water does not scald the limb. Often the pain is severe. Other options to reduce the pain include local anaesthesia, either injected into and around the sting site or a regional block, such as a foot block. The patients often will need an opiod injection, such as morphine or pethidine, and repeated doses.

Fortunately, there is a horse-derived antivenom, which is used as an intramuscular injection. One ampoule (2000 units) is said to neutralize 20 mg of venom, and each ampoule is recommended for each two spine puncture sites visible. The antivenom is safe, and is recommended for nearly all cases. As it is a horse-derived antivenom there is a small risk of anaphylaxis.

With any marine wound it is important to ensure the patient has adequate tetanus cover, and watch for any evidence of retained spine or infection.

Stingray barb injuries

The stingray is the largest of the venomous fish, however it is the barb that is located on its tail that produces the major injury to humans.

There is not a lot known about the venom of the stingray. Evidence from human stings suggests that the venom associated with the barb produces tissue necrosis.

The most common mechanism of injury is when a human stands on the stingray in the shallows. The tail of the stingray flips up and penetrates the lower limb of the person. Other injuries occur when a stingray has been netted whilst fishing and the person is injured whilst trying to release the stingray.

Most of the injuries produced by stingrays are minor. Fenner reported over 100 cases of stingray injury from Queensland. All patients complained of pain and a wound, with most being treated with symptomatic care. This included placing the limb in hot water, and/or infiltrating the wound with local anaesthetic. As there have been reports of infections developing and the barb remaining in the wound, it is important that the wound is examined for a retained barb, and washed or scrubbed. Some patients have required antibiotic therapy.

Tragically, there have been two deaths reported in Australia due to stingray barbs. In both cases the barb penetrated the left side of the chest and caused an injury to the myocardium. In one case a 12-year-old child died six days after a barb struck the left side of his chest. It is believed that the toxin on the barb caused a chemical myocardial necrosis resulting in myocardial rupture.

Therefore, it is prudent that any person with a stingray barb injury to the chest or abdomen will need a high index of suspicion for a significant internal injury, and should probably have their care monitored by a surgical service.

Management of a stingray barb injury to the chest is to ensure no immediate threat to life. Treat the wound ensuring bleeding and pain is controlled. The wound needs to be explored, ensuring there is no retained barb, and scrubbed. An X-ray may be required to confirm the presence or absence of a barb. Tetanus status should be checked and antibiotics may be required. If the wound appears contaminated, it is safer to leave the wound open and review the wound in 24–48 hours.

Ciguatera poisoning

Ciguatera is an illness that develops from eating larger tropical fish. The toxin, ciguatoxin, is produced by a dinoflagellate (algae), Ganbierdiscus toxicus, and the toxin accumulates in larger fish as they eat the smaller fish. The toxin is tasteless and is not destroyed by heat. Symptoms of ciguatera have occurred in tropical Australia, as well on tropical island communities where fish is a very important part of the diet. From 1964–86 more than 19,000 cases were reported in French Polynesia. With the ever-increasing export of fish throughout the world there have been reports of ciguatera poisonings in non-tropical parts of the world.

There have been discovered a number of ciguatoxins, and the structure of these toxins is known. Ciguatoxin is known to bind to sodium channels, and by this induces membrane depolarization.

Clinically a person with ciguatera poisoning presents initially with a gastrointestinal illness within 1–2 days of ingesting the offending fish. Usually the symptoms of vomiting, abdominal pain and diarrhoea occur within 12 hours of ingesting the fish. Patients then develop parasthesia around the

mouth, hands and feet, myalgias, arthralgias, ataxia, and itching. The patients can also develop temperature sensation reversal (hot things feel cold, cold things feel hot). Symptoms are worsened with alcohol consumption. Hypotension and bradycardia is rare, and the quoted fatality rate is less than 1%.

There is no specific treatment or antidote for this poisoning. If possible gastrointestinal decontamination should be attempted using activated charcoal, but this may be difficult if the patient is vomiting. Numerous drugs have been tried with limited success. Intravenous mannitol has been described as the most effective treatment for ciguatera poisoning, especially acute poisoning. Usually this is given in the dose of up to 1 g/kg over one hour, with some good anecdotal success; however there has not been any randomized trials to confirm it effectiveness.
|Symptoms can last for days, and there have been cases with symptoms lasting for months.

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