Poisonous and venomous marine animals

Venomous marine animals may inflict injury by means of bites, stings or simply by direct contact. Poisonous marine animals refer to those causing systemic effects when ingested.

POISONOUS MARINE ANIMALS[1-3]

Red tides: toxic dinoflagellates

Dinoflagellate blooms ('red tides') are regular seasonal phenomena occurring on the west coast of South Africa. These blooms may discolour the sea to various shades of brown, orange, purple, yellow or red. These planctonic algae are food for filter-feeding bivalve shellfish (mussels, oysters, clams, etc.). The majority of dinoflagellates are non-toxic, but toxin-producing species, such as *Alexandrium catenella* and *Dinophysis acuminata*, occur locally. Mussels and other bivalve shellfish strain these from the water, digest them, and accumulate their poison. The shellfish do not seem to be harmed by the poison, but become toxic to humans when eaten. Three different types of shellfish poisoning occur locally.

Paralytic shellfish poisoning (PSP)

PSP is caused by the ingestion of mussels that have concentrated the poison, saxitoxin, produced by *A. catenella*. Saxitoxin blocks voltage-dependent sodium channels, causing inhibition of nerve impulse conduction and transmission, which may ultimately lead to neuromuscular paralysis. Saxitoxin is heat stable and will therefore remain unaltered by standard cooking or steaming.

Clinical features. Within 30 minutes to 2 hours after ingestion, a tingling sensation (paraesthesia) or numbness around the lips, gradually spreading to the face and neck, and a prickly sensation in fingertips and toes develop. Headache, dizziness, a floating or gliding sensation, nystagmus, vertigo, visual disturbances, dysphagia and dysarthria are also commonly experienced. There is general weakness of the legs and, if the patient is able to walk at all, he/she does so with an ataxic gait similar to that seen in alcohol intoxication. Although nausea and vomiting, abdominal pain and diarrhoea have been reported, these symptoms are not common.

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In severe cases, progressive muscular paralysis, with pronounced respiratory difficulty, develops. Tachycardia, T-wave changes on the ECG, and occasionally hypotension, have been reported. Death due to respiratory failure may occur within 2 - 24 hours after ingestion. The toxic effect of saxitoxin is completely reversible and symptoms and signs normally clear within 36 - 48 hours.

Management is symptomatic and supportive. Since respiratory depression can develop surreptitiously, extreme vigilance should be exercised to monitor and support patients, especially during the first 12 hours. The differential diagnosis includes other types of food poisoning (see 'Food-borne disease' on p. 479).

Diarrhetic shellfish poisoning (DSP)

D. acuminata is the causative dinoflagellate. It produces the poison okadeic acid, which is an inhibitor of protein phosphatases in mammalian cells causing excessive fluid accumulation in the intestines and resulting in diarrhoea.

Clinical features. Symptoms and signs usually develop within 4 hours (seldom more than 12 hours after a meal). The clinical picture is characterised by nausea, vomiting, diarrhoea, abdominal pain and rigors. Spontaneous recovery usually occurs within 3 days, irrespective of medical treatment.

Management is symptomatic and supportive, with particular attention to fluid replacement.

Neurotoxic shellfish poisoning (NSP)

Gymnodinium species are the causative dinoflagellates. They produce the poison brevetoxin (aerosol toxin) which activates the voltage-dependent sodium channel, causing repetitive firing. Some species are unique in that they produce a toxic aerosol that is irritating to human mucous membranes. Exposures in humans (usually in the surf close to the beach) cause irritation of the eye, nose and throat with coughing, sneezing and difficulty in breathing. It may precipitate asthma attacks in susceptible individuals. Management is symptomatic and supportive.

Cases of systemic poisoning have not been encountered locally.

Scombroid poisoning

Scombroid poisoning (also referred to as histamine food poisoning) is a form of ichthyosarcotoxism caused by the consumption of 'spoiled' fish which has undergone autolytic changes as a result of improper storage conditions. The term 'scombroid poisoning' originates from the fact that spoiled fish from the family *Scombridae* (e.g. tuna, mackerel and bonito) were originally implicated. It seems, however, that non-scombroid fish may also be involved, and on rare occasions even certain cheeses. The Cape yellowtail (*Seriola lalandii*) is involved in most local cases.

Clinical features. Scombroid poisoning resembles a histamine-like or acute allergic reaction. Most cases are mild and self-limiting, even without treatment. Serious complications are rarely encountered and no deaths have been reported recently. The diagnosis of scombroid poisoning is generally made clinically. The onset is rapid. Skin manifestations are the most prominent feature. They appear within minutes to an hour, and seldom last for more than 6 hours. Most patients experience a hot, blotchy flushing of the skin, resembling an ethanol flush or severe sunburn, with well-demarcated borders, especially of the face, neck and upper chest. Pruritus is an infrequent feature. Gastrointestinal symptoms and signs, such as diarrhoea, are also prominent. A diarrhoea, often watery, usually appears within 6 hours. Other clinical features, in order of frequency, include: palpitations, throbbing headache, and abdominal cramps with occasional vomiting. Paraesthesia, hypo-aesthesia, often described as a tingling sensation around the mouth, as well as in the tongue and legs, and a scratchy feeling in the throat, occur in 20 - 40% of patients. An unusual taste, frequently described as peppery, pungent or bitter, is sometimes experienced. Breathing difficulties/shortness of breath may occur, mainly in patients with a history of asthma or allergy.

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The scombrotoxins (including histamine) are heat stable and scombroid poisoning occurs after ingestion of cooked, smoked or canned fish.

Patients with scombroid poisoning are often misdiagnosed as having a food allergy and inappropriately instructed to refrain from eating seafood. Factors which support the diagnosis of scombroid poisoning are the following: the high number of individuals afflicted in a group outbreak; the consumption of a common species of fish by everybody involved in an incident, and also a common species implicated in different outbreaks; the detection of high levels of histamine in the toxic fish; and the lack of a history of allergies. The differential diagnoses include food allergy, bacterial food poisoning, disulfiram reaction, Chinese restaurant syndrome, ciguatoxin poisoning, carcinoid syndrome, phaeochromocytoma and Zollinger-Ellison syndrome (see 'Food-borne disease' on p. 479).

Scombroid poisoning is a relatively mild, self-limiting condition, but it can pose a serious risk to patients suffering from allergic conditions, the elderly, those suffering from cardiac disease, and also patients on isoniazid therapy.

Management. The treatment of the condition is symptomatic and supportive. The majority of patients respond well to antihistamines. The classic antihistamines (H_1-receptor blockers, e.g. promethazine or diphenhydramine) are the drugs of choice. Patients may also show rapid relief of symptoms with the intravenous administration of cimetidine (H_2-receptor blocker). In the atopic patient, where bronchospasm or other severe histamine reactions may occur, the use of η-adrenergic agonists, theophylline and even corticosteroids, should be considered.

VENOMOUS MARINE ANIMALS[4-8]

Venomous marine animals may inflict injury by means of bites,

stings or simply by direct contact. Secondary infection is a common complication.

Coelenterata

Coelenterata are responsible for most marine envenomations. These include blue bottles (the Portuguese man-of-war or *Physalia*), jellyfish (Scyphozoa), sea-anemones (Actinaria), fire corals (*Millepora*) and hydroids (sea firs). All these cnidarians have specialised stinging cells (nematocysts), situated mainly on tentacles. Several million cells may fire during contact. Human reactions to venom result in a local and, rarely, a systemic response. Classically a sting results in a local, linear (whip-like), painful, urticarial eruption at the areas of tentacle contact. Local lesions usually resolve within a day or two. Blistering, regional lymphadenopathy and vesicular dermatitis with ulceration may follow. Ocular manifestations of stings include intense burning pain, lacrimation, conjunctival injection and corneal ulceration. Systemic toxic reactions, such as cardiovascular collapse, may occur but are not common. Allergic reactions include contact dermatitis and anaphylaxis.

Management. Stings must be irrigated with copious amounts of seawater. Fresh water or rubbing may cause nematocysts to discharge. The use of vinegar (5% acetic acid) or a 20% aluminium sulphate solution (Stingose) may help to inactivate the toxin. Physical removal of the nematocysts may be achieved by shaving or scraping of the exposed area with the edge of a sharp knife, razor or even a plastic card. A hot bath for 30 minutes may alleviate symptoms (the venom is heat labile). Application of topical steroids may be indicated.

Echinodermata

These spiny creatures include sea urchins (Echinoidea), starfish (Asteroidea) and sea cucumbers (Holothuroidea). The brittle spines of most sea urchins are non-venomous, but easily penetrate the skin, causing intense burning pain, swelling, and at times profuse bleeding. The flower urchin (*Toxopneustes pileolus*) is the most venomous of all our sea urchins, whereas *Diadema*, the long-spined needle urchin, causes the most serious injuries. Spine impalement of deeper structures is common in the hand. Several months after the initial injury, persistent nodular, sarcoid-like granulomatous lesions may develop as shown in Fig. 1.

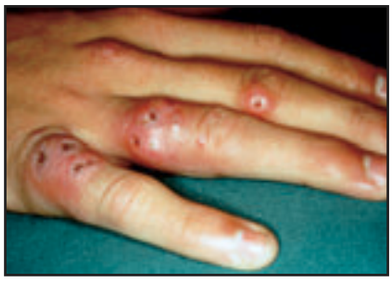

*Fig. 1. Multiple inflamed nodules or granulomata 3 months after common sea urchin (*Parechinus angulosus*) injury.*

Immersion in very hot water is the recommended treatment for all echinoderm injuries. Local anaesthetics and topical calamine lotion can be used for contact dermatitis. If urchin spines do not dissolve, local exploration of the wound and surgical removal of foreign material should be considered.

Polychaeta

Marine bristle worms, particularly the fire worm, have tiny setae (bristles) that cause fierce skin irritation and dermatitis. These are easily removed from the skin with adhesive tape. Immersion in hot water offers symptomatic relief.

Porifera

Sponges (Porifera) very commonly produce a contact dermatitis persisting for several weeks. Systemic toxic effects have not been reported locally. Treatment is symptomatic.

Mollusca

The only molluscs of real concern in South Africa are the conus species of sea snails. Bites from octopi and squids are rare in our waters. Conidae (sea snails) can inject a potent curare-like neurotoxin into the skin. Envenomation causes local ischaemia, burning pain and paraesthesia or numbness. Serious envenomation may cause progressive muscle paralysis, leading to respiratory failure. Treatment is symptomatic and supportive. Local serious envenomations are not common.

Acanthotoxic fish

These fish produce their venoms by means of glandular structures and are usually equipped with an envenoming device (such as spines) to deliver their venoms. This group includes stingrays (Rajiformes), scorpion fish (Scorpaenidae), toadfish (Batrachoididae), rabbit fish (Siganidae), stargazers (Uranoscopidae) and catfish (Tachysuridae). Most of these well- camouflaged fish are usually sedentary bottom dwellers and injury occurs when they are stepped on. Venom is introduced when the victim is injured by their sharp spines or fins. Only the zebrafish or devilfish (*Pterosis*) is known to be aggressive when threatened. Scorpion fish, and in particular the stonefish (*Synanceja*), are the most venomous in our region.

The venom apparatus of most of the venomous fish is comprised of a bony spine, covered by an integument sheath, with or without

grooves in it. Poison cells/glands are embedded in the grooves and/or the tissues surrounding the spine. During the stabbing action, the integument sheath is stripped off, exposing the poison glands. Venom and even fragments of the integument sheath may be retained in the wound. Sometimes fragments of the spine are also left in the wound. The effects that ensue are therefore often a combination of the action of the toxin as well as a response to the foreign material. The venom apparatus of the stonefish, however, is more sophisticated. The poison glands are more sack-like and arranged around the mid-portion of the spines. The venom is injected through the tips of the spines via distinct poison ducts. The stonefish is widely distributed and common on reefs north of Zululand. Injuries may be fatal.

In general, the venoms of the abovementioned venomous fish are basically cytotoxic, causing severe local pain and oedema for several hours. Frequently the wound, ischaemic and/or cyanotic, bleeds profusely. Oedema and erythema in the area of the penetrated skin may simulate bacterial cellulitis. There may be residual cutaneous granulomata, particularly in the wake of secondary infection. Local necrosis is also common. Systemic reactions include nausea, gastrointestinal symptoms, conjunctivitis, diaphoresis, cardiac arrhythmias, hypotension, muscular weakness or paralysis, seizures and even coma, but these are uncommon.

In the event of injury immediately irrigate the wound with salt water until the affected part can be immersed in hot water (45 - 50°C) for 30 - 90 minutes, or until pain is relieved. The venom is heat labile. Pain control may be augmented by infiltrating locally with lidocaine, without adrenaline. Wound exploration and debridement may be necessary. The wounds, notoriously slow in healing, often become infected. Patients with systemic toxic effects should be hospitalised and monitored closely. A stonefish antivenom exists, but is not available locally. Antibiotics are appropriate where indicated.

Hydrophidae

Pelamis platurus, the yellow-bellied pelagic sea snake, is the only sea snake reaching our shores. It produces a heat labile neurotoxic venom purportedly 2 - 10 times more poisonous than cobra venom. It blocks neuromuscular transmission and may cause renal tubular damage. The victim will require hospitalisation, for at least 24 hours, to monitor and support respiratory and renal function. No actual cases of envenomation have been documented locally.

References available on request.

IN A NUTSHELL

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SINGLE SUTURE

Epilepsy drugs may lead to misdiagnoses

Misinterpretation of anticonvulsant therapy as evidence of a diagnosis of epilepsy leads to misdiagnoses. Many typical epileptic drugs are used for conditions other than epilepsy, e.g. for neuralgias. It is important that patients who are taking these drugs for conditions other than epilepsy are aware of their drug indication. In two case reports patients receiving epileptic treatment for neuropathic pain and leg spasms, respectively, were inappropriately diagnosed as epileptic after subsequent blackouts. The non-epileptic seizures subsequently took over from the original complaint and became the main complaint. It is also important that an assumed diagnosis of epilepsy is not made for all patients who have a seizure. Of all people referred to neurology clinics up to 20% have psychogenic non-epileptic seizures. Guidelines published by the American Academy of Neurology and Child Neurology Society indicate that the risks associated with anticonvulsant treatment in children could outweigh the benefits of preventing seizures. Before anticonvulsants are prescribed, the treating doctor must be certain that a convulsion is indeed an epileptic seizure.

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