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Sea snake envenomation

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ABSTRACT: Sea snakes are common venomous reptiles found in the warmer waters of the Pacific and Indian Oceans. The most common circumstance for sea snake bite is an accidental encounter by a fisherman while emptying a fishing net. The venom is extremely toxic and most significant on the nervous and musculoskeletal systems. Significant envenomation occurs in about 20% of bites, with death occurring in about 3% of bites. Death most often occurs from respiratory arrest secondary to neurotoxicity. Myotoxins may cause muscle pain and stiffness and lead to clinically significant rhabdomyolysis and possible acute renal failure. Sea snake bites should all be considered a medical emergency. Immobilization of the victim with compression of the envenomed area is important first aid, followed by transporting the victim to an emergency medical facility. Artificial ventilation and hydration with crystalloid may be important lifesaving maneuvers. Sea snake antivenom may be critical for survival in a significant envenomation.

KEYWORDS: antivenom, Hydrophiidae, myotoxin, neurotoxin, snake, snake bites.

Sea snakes are one of the most common (1–3) and venomous reptiles in the world. They are a family of 52 species of air breathing reptiles. Their distribution is limited to the warmer waters of the Indian Ocean and tropical Pacific Ocean (Fig. 1). Their habitat extends from as far south as Australia and New Zealand, north of the equator to the coast of southern California and the southern tip of Japan and the Persian Gulf. They are not found in the Atlantic Ocean or Caribbean, Mediterranean, or Red Seas.

Sea snakes are classified in the family Hydrophiidae and are further divided into subfamilies of Hydrophiidae (true sea snakes) and Lacticaudidae (sea kraits). The former spend all of their time in the ocean, while the latter may have a partial terrestrial habitat. Both subfamilies are related to the cobra (elapid) family of terrestrial snakes. Sea snakes range in size from an average of 1.2 m to as long as 3 m.

Sea snakes differ from land snakes in that they have vertically flattened tails (Fig. 2) to facilitate movement in the water and lack the ventral scales necessary for movement on land (1). They move backward and forward in the water with ease using their paddle-like tail, but they are very awkward on land. They may be mistaken for eels in an aquatic environment, but lack gills and fins. They also have nostrils, which are covered by an operculum when submerged in water. They have one to three pairs of short fangs (Fig. 3) anchored in the maxillary bone and associated with a venom gland on either side above the maxillary bone. Sea snakes tend to be bottom feeders, and can stay submerged for as long as 2 hours, although some, such as the yellow-bellied sea snake (*Pelamis platurus*), feed on the surface.

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Environment

Most sea snakes live near the shoreline or on reefs. They are often found in the crevices of rocks or around pilings or tree roots. Some, such as the yellow-bellied sea snake, spend their life on the open ocean. Other species may be found in the brackish waters of tidal rivers (1). Rare species are found in landlocked lakes such as Lake Bombon (or Lake Taal) in Luzon, the Philippines (4).

Most species of sea snakes are nonaggressive, but they may bite humans if handled roughly or encountered at the wrong time. The most common circumstance for a sea snake bite is when a

Fig. 1. Worldwide distribution of sea snakes is shown in hatched areas. (Adapted from ref. 1.)

commercial fisherman removes a sea snake from a fishing net (more than 50% of all bites) (5). Sea snake bites are also associated with snakes mating, ascending to the surface for air, and rough handling. Occasionally sea snakes may be stepped on while wading, swimming, or collecting seashells. Bites are usually single, but may be multiple, and commonly occur on feet, ankles, fingers, hands, and forearms (1).

Venom

Sea snake venom is one of the most powerful venoms of any creature. A single bite from an average size sea snake potentially contains enough venom to kill three average-size adults (6).

Fig. 2. *Lacticauda semifasciata* (courtesy of Dr. Anthony Tu). Sea snakes and kraits are easily distinguished from land snakes by their vertically flattened tails (left), which facilitate locomotion through the water. (Reprinted from Tu A, Sea snake bites. In: Clinics in dermatology, 1987:118–126, with permission from Elsevier Science.)

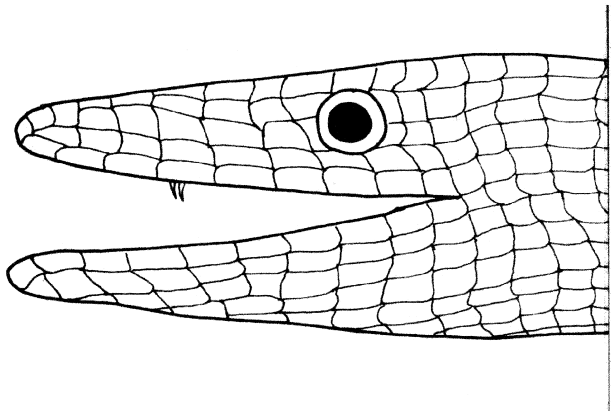

Fig. 3. Schematic drawing of a sea snake shows a single row of short fangs. The anterior-most fangs are associated with a venom gland. The round pupil of sea snakes can also help differentiate it from other poisonous terrestrial snakes.

Most sea snake bites do not result in significant envenomation (about 20% cause envenomation) (4), but because of the toxicity of the venom, sea snake bites should be treated as a medical emergency until it is established that no significant envenomation has occurred. The case fatality rate for sea snake bite is estimated to be about 3%.

Sea snake venom has many components, including acetylcholinesterase, hyaluronidase, leucine aminopeptidase, 5′ nucleotidase, phosphomonoesterase, phosphodiesterase, and phospholipase A (7). Sea snake venom lacks a hemotoxin found in many snakebites (8), but nephrotoxicity and hepatoxicity have been reported (7). The most clinically significant toxins in the venom are neurotoxin and myotoxin.

Clinical manifestations of envenomation

The neurotoxin in sea snake venom contains both a presynaptic and postsynaptic component (4). The postsynaptic neurotoxin is the more important of the two. It binds to the postganglionic acetylcholine receptors of the peripheral nervous system. The initial clinical manifestation is weakness, but motor paralysis may result in respiratory failure and is the most common cause of death in sea snake envenomation. The signs and symptoms of sea snake venom neurotoxicity are listed in Table 1. Table 2 lists some of the common symptoms suggesting a more significant envenomation (1).

The myotoxin in sea snake venom, phospholipase A, leads to skeletal muscle necrosis (1).

Table 1. Neurotoxic manifestations of sea snake envenomation

Anxiety
Malaise
Slurred speech
Difficulty swallowing
Motor weakness
Muscular paralysis (flaccid or spastic)
Nausea and vomiting
Muscle twitching, spasms
Bulbar paralysis
Blurred vision
Jaw stiffness (trismus)
Drooling
Ptosis
Ocular paralysis
Respiratory distress

Clinically the patient experiences muscle stiffness or pain. A hallmark of sea snake envenomation is muscle pain with passive movement. Injury to skeletal muscle may lead to rhabdomyolysis, which can lead to renal failure through acute tubular necrosis.

Management of sea snake envenomation

First aid for presumed sea snake bites should begin immediately once a sea snake bite is suspected. Criteria for establishing a sea snake bite includes 1) opportunity for contact, 2) absence of pain at the site (sea urchin or fish stings more often hurt), 3) the presence of definite fang marks at the site of the bite (1–20 bite marks with 4 being most common), 4) positive identification of the snake, and 5) symptoms—painful muscle involvement, paralysis of legs, trismus, or ptosis (1). Initial symptoms of envenomation may occur anywhere from 5 minutes to up to 8 hours after the bite. First aid should be continued, including transportation to an emergency medical facility, until it has been clearly established that no significant envenomation has occurred. Venom detection kits are available, but they are of little use in sea snake bites because the venom is usually washed off by the seawater after the bite occurs.

Sea snake venom is rapidly absorbed. Some authorities feel there may be a limited role for suction of the wound if it is within the first few minutes after the bite (1). Beyond that time period there is clearly no role for suctioning the wound to remove venom and it may lead to other complications such as infection. The venom of the sea snake is stable over wide pH and temperature ranges (7) and thus there is no role for hot or cold packs or the application of vinegar to the sea snake bite.

The preferred technique is a compression-immobilization technique (9) to try and limit the spread of the venom. Most sea snake bites are on an extremity, and if possible, a compression dressing should be applied to the entire limb with enough pressure to occlude venous and lymphatic return (70 mmHg) without impairing arterial supply. A wet suit is often protective from the short fangs of a sea snake, but if the fangs do penetrate, the wet suit may provide a good compression dressing if left in place. The involved extremity should then be immobilized below the level of the heart. The victim should be discouraged from exerting him/herself. Maintenance of an airway and support of both breathing and circulation can be important early on if significant envenomation has occurred.

Antivenom for sea snake envenomation is available through the Commonwealth Serum Laboratories (http://www.csl.com.au/) and is made from the hyperimmunization of horses with the venom of the beaked sea snake (*Enhydrina schistosa*). It should only be used if there is clear evidence of sea snake envenomation, such as generalized muscle aches and pains following an established bite; moderate or severe pain on passive movement of arm, thigh, neck, or trunk muscles; tender lymphadenopathy (4); myoglobinuria 3–6 hours after the bite or a dusky yellow color to the urine with a positive protein and occult blood test; or rapid vascular collapse and shock after envenomation (6).

Antivenom should not be withheld in a case of clear envenomation because of concern for an allergic reaction. Halstead (1) recommends sensitivity testing with a 0.1 cc intracutaneous injection of 1:10 diluted antivenom. Alternatively one to two drops of a 1:10 dilution can be dropped onto the conjunctiva. A positive test is redness of the

Table 2. Signs and symptoms of sea snake envenomation with the poorest prognosis

Multiple tooth marks (suggest a heavy venom load)
Vomiting
Weakness of external eye muscles
Ptosis
Pupil dilation and sluggish reaction
Leukocytosis, >20,000 WBC/mm^3

conjunctiva within a few minutes after administration. It is recommended that in all patients receiving antivenom, preparations should be made for treating anaphylaxis. Another infusion setup through an infusion pump with an appropriate concentration of epinephrine should be available if symptoms or signs of anaphylaxis occur (10). Premedication with epinephrine should be considered in the presence of allergies or asthma and parenteral antihistamines should be considered. The minimum dosage is 1 ampule diluted 1:10 in a crystalloid solution and administered intravenously over a 30-minute period. A common mistake, however, is using too little antivenom. As many as 3–10 ampules of antivenom may be required for relief of symptoms. Antivenom is most effective if given in the first hour after envenomation, but may still be effective even if given more than 8 hours after the bite. If sea snake antivenom is not available, tiger snake antivenom may be used, but requires the administration of three times as much.

Complications of antivenom include immediate anaphylaxis if the patient is allergic to it. Serum sickness may develop for up to 2 weeks following the last dose of antivenom. This is characterized by urticaria, fever, arthralgia, myalgia, and lymphadenopathy. Management of allergic reactions may require epinephrine, bronchodilators, parenteral antihistamines, and corticosteroids (11).

Supportive measures may also be required following a sea snake bite and are largely dependent on the patient's clinical course. Intravenous fluid support is usually needed. Volume replacement may be the single most critical management issue for treating acute rhabdomyolysis from envenomation (12). Monitoring blood pH, calcium, and potassium levels may be critical in the presence of acute tubular necrosis from myoglobinuria. Blood or plasma transfusions may be required. Antibiotics and antitetanus precautions may be required, depending on the patients status. Oxygen and ventilatory support may be required as noted.

Management of sea snake envenomation may not fall under the guidance of a dermatologist, but rapid identification of sea snake envenomation, initiation of first aid, and transportation to an advanced medical facility may be lifesaving.

References

1. Halstead BW. Venomous sea snakes. In: Halstead BW, ed. Poisonous and venomous marine animals of the world. Princeton, NJ: Darwin Press, 1988:1036–1108.
2. Auerbach PS. Hazardous marine animals. Emerg Med Clin N Am 1984: **2:** 531–544.
3. Burke WA. Skin disease in fishermen. In: Langley RL, McLymore RL, Meggs WJ, Roberson GT, eds. Safety and health in agriculture, forestry and fisheries. Rockville, MD: Government Institutes, 1997:706–712.
4. Tu AT, Fulde G. Sea snake bites. Clin Dermatol 1987: **5:** 118–126.
5. Thomas C, Scott S. Sea snake bites. In: Thomas C, Scott S, eds. All stings considered. Honolulu: University of Hawaii Press, 1997:72–76.
6. Edmonds C. Sea snake. In: Cronin L, ed. Dangerous marine creatures. Singapore: Reed Books, 1989:102–106.
7. Tu AT. Biotoxicology of sea snake venoms. Ann Emerg Med 1987: **16:** 1023–1028.
8. Schmidt ME, Abdelbaki YZ, Tu AT. Nephrotoxic action of rattlesnake and sea snake venom: an electron microscopic study. J Pathol 1976: **118:** 75–81.
9. Acott C, Williamson J. Sea snakes. In: Williamson J, Fenner PJ, Burnett JW, Rifkin JF, eds. Venomous and poisonous marine animals: a medical and biological handbook. Sydney: University of New South Wales Press, 1996:396–402.
10. Department of Human Services. Women's and children's hospital CSL antivenom handbook: CSL sea snake antivenom. Accessed October 16, 2001, http://www.wch.sa.gov.au/paedm/clintox/cslavh_antivenom_seasnake.html#venom.
11. Soter NA. Urticaria and angioedema. In: Freedberg IM, Eisen AZ, Wolff K, et al., eds. Dermatology in general medicine. New York: McGraw-Hill, 1999:1409–1419.
12. Knochel JP. Rhabdomyolysis and acute renal failure. In: Glasscock RJ, ed. Current therapy in nephrology and hypertension. St. Louis: Mosby, 1998:262–265.