

ORIGINAL ARTICLE

Death adders

TERENCE M. DAVIDSON, MD*¹ SUSAN F. SCHAFFER² and
BRIAN CAPELTO, MD³

¹Division of Otolaryngology – Head and Neck Surgery, University of California Medical Center, H-895, 225 Dickinson Street, San Diego, CA 92103, USA

²Department of Herpetology, Zoological Society of San Diego, USA

³Cerritos, California, USA

Introduction

Death adders inhabit Australia and New Guinea. The death adders belong to the genus *Acanthophis*. The three clinically important death adders responsible for the vast majority of human bites are *Acanthophis antarcticus antarcticus*, the common death adder, *Acanthophis antarcticus laevis*, the eastern death adder, and *Acanthophis pyrrhus*, the desert or central death adder.

Biology

Protruding from the leaf litter on the forest floor, a pale yellow worm wriggles vigorously. Nearby, a small bird foraging for a meal in the dim light of dusk is attracted by the motion of the worm and darts over to snatch it in its beak. At the same instant, the bird is stricken and seized by the predator's mouth. A potent venom, injected through a pair of 6 mm long fangs, quickly paralyzes the prey. Easing itself back into the loose soil and decaying leaves, the death adder leisurely consumes its meal.

Half-buried and cryptically colored to match the background, a death adder is virtually undetectable except for the grublike tip of its tail which lies motionless against the contrasting color of the snake's coiled body. It rarely searches for food when hungry, but rather, moves its tail into a fixed position in front of its mouth. When potential prey approaches, it wiggles the false larva temptingly and lures its next meal within striking distance.

Despite the common name, death adders belong to the cobra family, Elapidae. With viper-like appearance and ambushing behavior, most other cobras are slender and forage actively. Death adders are characterized by a stocky body, narrow neck, and broad triangular head. The end of the short tail narrows abruptly to form the lure, which may be light or dark, but usually contrasts with the color of the body [1, 2, 3].

As evidenced by elliptical pupils, death adders are nocturnal and particularly active on warm nights [4]. Feeding upon a variety of insectivores, including lizards, birds, and small marsupial mammals, they show an ontogenetic shift in diet, as the young prey primarily upon ectotherms, while the adults prey primarily upon endotherms [4,5]. They are inactive during the day, burrowing with side-to-side movements of the body into soft soil or leaf litter beneath trees or shrubs. When disturbed, they rarely flee. More likely,

*To whom correspondence should be addressed.

they flatten their bodies, hiss and strike with mouth agape [1,3,4]. They range in habitat from arid scrub and dry woodlands to coastal sand dunes and tropical forests [4]. All are ovoviviparous and give birth to 10–33 young between December and April on alternate years [1,6].

There are three taxonomically distinct species of death adder with exclusive ranges on mainland Australia, New Guinea and a number of neighboring islands. Although similar in appearance, they differ significantly in scale characters.

The common death adder, *Acanthophis antarcticus* (Fig. 1), is found throughout continental Australia except for the far north, the wetter parts of the southeast, and the central deserts [1]. Throughout much of its range, it inhabits scrublands and forests, preferring areas with greater rainfall and cooler temperatures than does its desert relative [4,7,8]. It varies in color, depending upon locality, from pale gray to reddish-brown with irregular light or dark contrasting cross-bands [1,4]. The tail tip is white or cream-colored. Specimens of 1 m in length have been recorded, although the average length is around 0.4–0.6 m [1,4,7].

The northern death adder, *A. praelongus*, resides in the extreme north of Australia, New Guinea, and several nearby islands, where it tolerates wetter situations in regions of rain forest [7,8]. It is the least studied of the death adders, although its habits are assumed to be similar to the common death adder. Moderately stout, it is intermediate between the common and desert forms in scale characteristics. Its color is darker than that of the common death adder, with a stronger dorsal pattern. It averages about 0.4 m in length, rarely reaching 0.7 m [1].

The desert death adder, *A. pyrrhus*, is found throughout the desert regions of central Australia, extending into the arid parts of all the mainland states except Victoria. More slender than the other forms and of less predictable disposition, it is bright red with a black tail tip. The specific name, meaning 'first', refers to the snake's attractive coloring. Because it occurs in remote areas, it is little known, although it too is presumed to be



Fig. 1. *Acanthophis antarcticus*, the common death adder.

similar in habits to the common death adder. Nothing is known about its venom except that its physiological effects upon laboratory animals appear to be rapid in onset. It grows to 0.75 m [1,4].

Death adders are considered to be among the most dangerous snakes in Australia and the world. Taking into account venom toxicity, venom yield, fang length, temperament and frequency of bites, the common death adder, *A. antarcticus*, is considered as dangerous in Australia as the fierce snake, *Parademansia (Oxyuranus) microlepidota*, the taipan, *Oxyuranus scutellatus*, the brown king snake, *Pseudechis australis* [1,4], and the Australian tiger snake, *Notechis scutatus*. Due to its cryptic coloration and tendency to lie motionless, the death adder is often stepped on, and bites are fairly common [7,8].

Death adder populations have suffered a dramatic decline in Australia due to habitat destruction and the introduction of stock and feral animals. Foxes and cats prey upon the snake, as well as upon its natural food supply. Juvenile snakes are eaten by the giant cane toad, *Bufo marinus*, while adults are often killed after mouthing or ingesting the poisonous toad. Introduced into Australia as a biological control for a pestilent beetle in the sugar cane industry, the cane toad has had little effect upon the beetle, but a significant effect upon the native fauna. Relying upon virgin habitats for refuge and food, the death adder has shown little tolerance to changes in the environment, and its future remains uncertain [4,6].

In the wilderness, the snake appears relatively docile. Almost all bites occur when the animal is stepped upon or grabbed. The snake strikes low to the ground. Bites usually occur on the foot below the ankle. Campbell reported on 15 cases, indicating that the snake may bite and hold on [9].

Clinical considerations

Acanthophis is responsible for a significant number of venomous snake bites throughout its geographical distribution in Australia and New Guinea. The venom is principally neurotoxic with a curare-like action. Neurotoxins bind to post synaptic motor end plate membranes, blocking acetylcholine receptors [9]. Untreated patients develop respiratory failure due to paralysis of the muscles of respiration. Unassisted victims may die.

Bites may be accompanied by mild pain at the bite site associated with slight edema [9]. Ptosis is often the earliest sign of neurotoxic envenomation. This progresses to paralysis of other voluntary muscles, ultimately leading to decreased respiratory effort. Blood coagulation typically remains normal, but mild hemolytic and coagulopathic changes have been described [10]. Early reports of death adder envenomation suggest a mortality in untreated humans of up to 50% [9]. However, Currie estimated an overall mortality closer to 15% [11].

An interesting case from New Guinea was reported by Currie *et al* [11]. A 40-year-old male was bitten on the foot by a 30.5 cm death adder. He was treated with 'two tree bark tourniquets' and incisions over the bite site created with bamboo slivers. Two hours later, he first reached medical attention. At this time, he had tender inguinal adenopathy above the bitten extremity. The tourniquets were removed and a pressure dressing, as described later, was applied. The patient was transported to a central medical center. Eighteen hours post-transport, the victim remained well, so the pressure dressing was released. Over the ensuing two hours, ptosis, dysphasia and dysarthria became evident. Chest wall weakness with dyspnea was noted. Immunoassay confirmed envenomation by

a death adder. The patient was treated with 0.6 mg atropine IV, followed by 2.5 mg neostigmine infused over 2 min. Within 5 min, diminished diaphoresis and reversal of ptosis were noted. One ampule of antivenom was administered 29 min later. Neurotoxic symptoms did not return.

This case demonstrates the typical course of symptom progression. It also supports the value of pressure immobilization. In this case, it appears that the wrap impeded systemic venom uptake significantly. While there is no question that antivenom is the medical treatment of choice, neostigmine appears to have been useful. Neostigmine is a cholinesterase inhibitor, increasing the local concentration of acetylcholine in the area of the motor end plate. This feeds back on presynaptic receptors, thereby mobilizing additional acetylcholine for release by the neuromuscular junction. Neostigmine also stimulates receptors located at the first axon hillock, thereby causing repetitive firing of the nerve terminals [12]. Atropine blocks the muscarinic actions of neostigmine and is administered to prevent undesirable autonomic nervous system stimulation [13].

The anticholinesterase treatment is important, particularly in New Guinea, for two reasons: (1) the specific antivenom is not readily available throughout New Guinea; and (2) the antivenom is relatively expensive, and in that country, less costly therapy is a consideration. Anticholinesterase therapy is not necessary in Australia where antivenom is more available, and the expense is of seemingly less concern.

FIRST AID

In the event of an actual or probable bite from a death adder, execute the following first aid measures.

1. Make sure that the responsible snake has been appropriately and safely contained and cannot inflict another bite.
2. Immediately arrange to transport the victim to an appropriate medical facility.
3. Keep the victim calm, supine or prone, with as little movement as possible. Rest the bitten limb at a level lower than the victim's heart.
4. Immediately wrap a large crepe bandage snugly around the bitten limb. Start at the bite site and wrap proximally, the full length of the limb if possible. The bandage should be as tight as one might bind a sprained ankle. Distal arterial pulsations should not be impeded. Fig. 2 shows the steps redrawn from the Commonwealth Serum Laboratories (CSL) first aid recommendations of S.K. Sutherland [14].
5. Secure a splint to the bandaged limb to prohibit motion. Avoid bending or moving the limb excessively while applying the splint. Hold the splint in place with a second bandage.
6. **DO NOT REMOVE** the splint or bandage until the victim has reached the hospital and is receiving antivenom.
7. If available, have death adder antivenom (10 vials; CSL) ready to carry with the victim to the hospital. Polyvalent Antivenom (CSL) is also effective and can be used if death adder specific antivenom is unavailable or if the snake's identity is uncertain. If antivenom is not available, contact a regional poison control center in the US or the Commonwealth Serum Laboratories, (45 Poplar Road, Parkville, Victoria 3052, Australia. Telephone # 61-3-887443).
8. **DO NOT** incise the bite site.
9. **DO NOT** apply ice to the bite site.

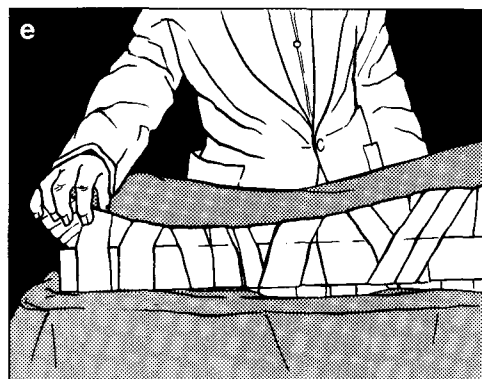
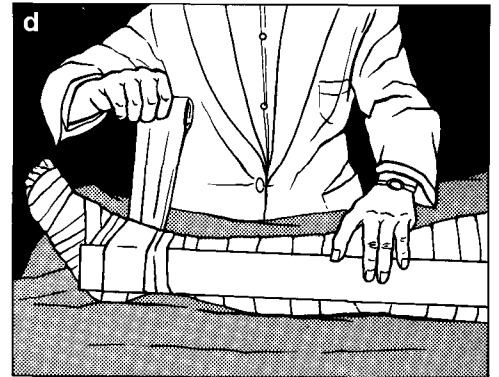
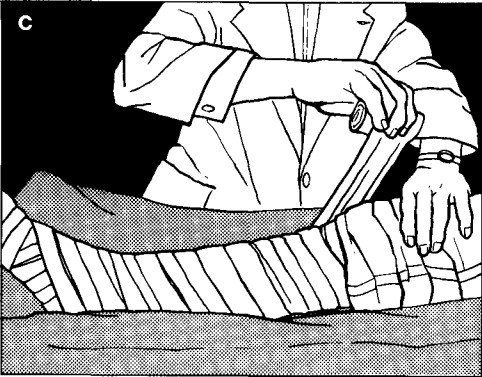
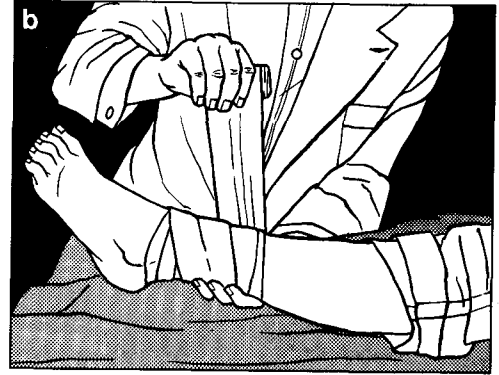
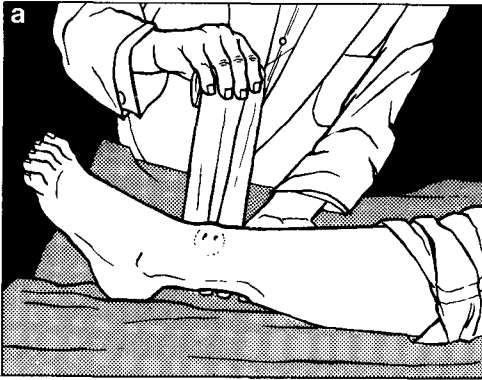


Fig. 2a. Apply a broad pressure bandage over the bite site as soon as possible. Don't take off clothes, as this movement enhances venom absorption. Keep the bitten extremity still.

Fig. 2b. The bandage should be applied as tightly as would be an ace wrap to a sprained ankle.

Fig. 2c. Extend the bandage wrap as high (proximal) as possible.

Fig. 2d. Apply a splint to the leg.

Fig. 2e. Bind the splint firmly to the leg, immobilizing the extremity.

MEDICAL MANAGEMENT

1. A crepe bandage and splint have been applied to retard the absorption of venom. **DO NOT REMOVE** these until the patient has arrived at the hospital and is receiving antivenom.
2. Make sure that at least 10 vials of death adder antivenom are available. If you need assistance locating appropriate antivenom, call the regional poison control center, the San Diego Regional Poison Control Center (Telephone # 619-543-6000), or the Commonwealth Serum Laboratories, Parkville, Victoria, Australia.
3. Envenomation is diagnosed by the presence of characteristic signs and symptoms. Blurred vision and ptosis are often the first indications of neurotoxicity; generalized paralysis of voluntary musculature may be variable and delayed. Common neurologic manifestations are listed in Table 1.

Hematologic and vascular symptoms are minimal and may include bleeding from the bite site and mild coagulopathy. Hypotension is seen with severe bites. Albuminuria has been noted.

General signs and symptoms (Table 2) typically develop within one hour of the bite. Fang marks may be present as one or more well defined punctures, as a series of small lacerations or scratches, or may be absent. Absence of fang marks does not preclude the possibility of a bite, especially if a juvenile snake is involved. Conversely, the presence of fang marks does not always confirm envenomation.

4. Admit the patient to an intensive care setting. Begin a peripheral IV infusion of lactated Ringer's solution at a rate sufficient to maintain a brisk urine output. Children and individuals with compromised cardiovascular or renal function may not tolerate a fluid challenge without invasive monitoring to allow judicious administration of osmotic or loop diuretics.
5. Draw blood samples from the contralateral arm to obtain the laboratory tests listed in Table 3. It may be necessary to repeat some tests during the hospital course to monitor the effects of antivenom therapy.
6. Observe the patient closely for signs and symptoms of envenomation. If no sign or symptom is noted after 2 h, it is possible that the patient received a 'dry' bite (no venom injected). Very slowly, remove the splint(s) and begin to unwrap the pressure bandages. Observe carefully for any change in the patient's status. If a change occurs, assume the patient has been envenomed and prepare to give anti-

Table 1. Neurological signs and symptoms of death adder envenomation.

Ptosis
Blurred or diminished vision
Dysarthria
Dysphasia
Dyspnea
Respiratory paralysis
Headache
Drowsiness
Loss of consciousness
Flaccid paralysis
Ataxia

Table 2. General signs and symptoms of death adder envenomation.

Pain at the bite site (usually mild)
Regional lymph node tenderness (pain can be severe)
Regional lymphadenopathy
Vomiting
Cough
Profuse sweating
Local edema (tends to be slight around the bite site)

Table 3. Laboratory tests required in the management of death adder envenomation.

Complete blood count with differential and platelet count.
Coagulation parameters:
a. prothrombin time
b. partial thromboplastin time
c. fibrinogen level
d. fibrin degradation products
Serum electrolytes, blood urea nitrogen/creatinine, calcium, phosphorus, lactate dehydrogenase (with isoenzyme analysis), urinalysis.
Electrocardiogram

venom immediately. If signs and symptoms still fail to manifest, continue close observation for an additional 24 h.

7. If any sign or symptom becomes apparent, begin antivenom therapy. Dilute the contents of one vial (6000 units) of death adder antivenom in lactated Ringer's solution to a total volume of 60 ml. If the adder-specific antivenom is unavailable, use Polyvalent Antivenom (CSL). Instructions for dilution, administration and titration are the same as for adder-specific antivenom. Administer the diluted antivenom intravenously over 30 min at a rate of 2 ml min⁻¹ (1 vial per 30 min or 200 units per min). Anticipate using (including the initial dose) two to three vials for a minor bite with signs of envenomation. Three to six or more vials may be necessary to treat moderate or severe bites.
8. Should any sign of allergy/anaphylaxis (e.g. cough, dyspnea, urticaria, itching, increased oral secretions, etc.) develop, immediately discontinue the administration of antivenom and treat the victim with epinephrine, steroids and antihistamines. As soon as the patient is stabilized, continue the antivenom infusion at a slower rate. Simultaneous administration of an epinephrine infusion may be necessary.
9. After 15 min of antivenom administration, the splint and bandages may be removed. This should be done over a period of 5 min to minimize a bolus release of venom. If the patient's condition worsens, reapply the crepe bandage and wait 10 min. Release the bandage again, over 10 min, while antivenom administration continues.
10. If breathing becomes impaired, provide respiratory assistance. Copious secretions may necessitate suctioning and, possibly, endotracheal intubation. Supplemental

oxygen is advised. Disseminated intravascular coagulopathy is not an anticipated complication. Death adder venom also lacks myolytic or nephrotoxic actions.

11. It is important to keep venom neutralization continuous. Keep a close watch on the patient's status. If the victim's condition worsens, additional antivenom should be administered in doses of 1 vial (6000 units).

General considerations

The onset of dangerous neurotoxic symptoms can be rapid and subtle. They are more rapidly reversed in the early stages than when fully developed. It may be necessary to wake the patient and perform a brief neurological examination every hour to assure that breathing and other vital functions are not impaired. Respiratory obstruction and failure are the greatest immediate concerns. Make sure that adequate suction equipment is available and operative at the bedside. If any sign of oropharyngeal paralysis or impaired swallowing exists, nothing should be given by mouth, and the patient kept on his side, head down. Narcotics are contraindicated, for they will suppress respiration. Diazepam may be given, but not in large doses. Where circulatory shock is uncorrected by antivenom therapy, plasma volume expanders and/or vasopressor agents may be given with appropriate considerations. Tetanus prophylaxis should be current. Prophylactic antibiotics are not recommended.

Special considerations

It is possible for a death adder to deliver more than one bite in an attack. If there is evidence that this occurred, twice the initial dose of antivenom should be given: two vials (12000 units) diluted in lactated Ringer's solution to a total volume of 120 ml, delivered over 30 min at a rate of 4 ml per min (two vials per 30 min or 400 units per min). If the patient shows severe signs of envenomation, particularly soon after the bite, the initial dose of antivenom may be doubled or tripled. Dilute the antivenom 1:10 in lactated Ringer's solution and deliver it over a period of 30 min. If the patient is fluid overloaded, antivenom may be delivered in higher concentration until the patient is in appropriate fluid balance.

It is not advisable to utilize subcutaneous or intradermal testing for sensitivity to equine products, in that such testing may be unreliable and unnecessarily delay antivenom therapy. If there is reason to believe that the patient may be sensitive to equine protein products (e.g. previous snake bite treated with antivenom), administer 1 g of methylprednisolone intravenously 15 min prior to antivenom therapy. Administer antivenom at the rate tolerated by the patient, beginning at a rate of 1.5 ml min^{-2} ($150 \text{ units min}^{-1}$). Monitor pulse and blood pressure carefully. Be prepared to treat anaphylaxis with epinephrine and other appropriate medications.

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