Bites & stings

Could this be snakebite?

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Background. Australian snakes are among the most venomous in the world. Although usually obvious, the occurrence of snakebite is occasionally unrecognised by the patient and/or physician, resulting in delayed or inadequate treatment, or even death.

Objective. This article describes the historical, clinical and pathologic features associated with envenomation by various Australian venomous snakes, and discusses the investigation and management (including first aid) of suspected snakebites.

Discussion. A high index of suspicion should be maintained, particularly in rural areas and in patients unable to give a history. Investigations including creatine kinase, clotting profile and venom detection kit should be performed in cases of suspected snakebite. The choice of appropriate antivenom and its indications are discussed.

Suspecting snakebite

Given the size of the creatures and the sometimes dramatic circumstances surrounding snakebite, it might seem that the diagnosis of snakebite should be obvious. While this is often the case, snakebite is occasionally unrecognised by the patient and hence by his or her attending physician, leading to delayed or incorrect diagnosis and treatment.

The reasons for this are:

The bite itself may not be dramatic or painful

Australia's venomous snakes belong to the family of proteroglypyphs or front-fanged snakes. Most of their fangs are relatively small at ~3-6 mm in length (although the taipan's fangs may reach 13 mm), and this, combined with the fact that Australian snake venom generally causes little local pain or tissue destruction, may result in the bite being unrecognised or mistaken for a scratch or an insect bite or sting. This is particularly likely in the dark or in heavy scrub when the snake may not be seen. Examination of the bite site itself usually reveals small punctures or scratches, with little surrounding tissue reaction. The bite site is often difficult to see, and may be overlooked.

Patient is unable to give a clear history

Such patients include children, confused or comatose patients and those who are intoxicated. In addition, amateur herpetologists keeping venomous snakes illegally may be reluctant to present for medical help, may present late and may provide incomplete or spurious information.

A high index of suspicion is required in any case with features that are suggestive of envenomation by a snake. These may be gained from the history, signs and symptoms and results of laboratory investigations.

Indicators of envenomation

History

Location

Snakebites most commonly occur outdoors. Typical 'snake country' includes long grass, bushland, hay sheds (where the presence of rodents attracts snakes) or moist swampy areas, although snakebite may also occur in suburban areas and occasionally in the home. It has even occurred in hospital!

Children are at greater risk of snakebite due to their inquisitive and fearless nature and tendency to play outdoors. They are also more likely to sustain multiple bites when they encounter snakes. Combined with their lower body weight, this means that children may be more severely affected by snakebite.

Presentation

Symptoms may include:

➤ headache, nausea, vomiting, abdominal pain;

➤ collapse, unconsciousness (may be transient);

➤ painful muscles (myolysis);

➤ blurred vision, diplopia, difficulty swallowing or breathing, slurred speech, weakness, paresthesia (neurotoxicity).

Signs of snakebite may:

➤ include puncture marks (usually on limbs, *Figure 1*);

➤ be difficult to see;

➤ consist of single or double puncture marks or scratch marks;

➤ be bleeding/oozing;

➤ include multiple punctures consistent with severe envenomation;

➤ include regional tender lymphadenopathy (this may also be present after bites from non-venomous snakes, and is not itself an indication for antivenom).

Signs consistent with envenomation are:

➤ irritability, confusion, coma;

➤ bleeding from bite site, venepunctures, or occasionally other sites (care should be taken with puncture of arterial or central venous sites in the presence of potential coagulopathy);

➤ dark urine (myoglobinuria, haematuria);

➤ ptosis, dysarthria, weakness/paralysis, dyspnoea, respiratory failure (neurotoxicity);

➤ hypotension;

➤ cardiorespiratory arrest.

In managing the patient with suspected snakebite, it is necessary to ascertain whether significant envenomation has occurred and to attempt to identify the type of snake involved. It should be noted that a significant proportion of venomous snakebites do not result in envenomation, and the use of antivenom should be reserved for those cases with clinical or pathologic evidence of envenomation.

Investigation

Venom detection kit

This is a rapid two step enzyme immunoassay (EIA) test used to select the most appropriate antivenom. Swabs from the bite site provide the best sample for use in the CSL Snake Venom Detection Kit *(Figure 2)*. The patient's clothing may also be sampled for venom. Samples of blood or urine may also be used in the venom detection kit, although the results may be less reliable than those obtained from the bite site, especially if the urine is collected soon after envenomation has occurred. The presence of venom at the bite site is not in itself an indication that systemic envenomation has occurred, nor can its absence be used to exclude envenomation.

Blood studies

➤ **Clotting studies:** international normalised ratio/prothrombine (INR/Pt), activated partial thromboplastin time (APTT), anti-coagulant therapy (ACT), D-dimer, X-fibrin degradation products (X-FDP), fibrinogen. In remote areas where sophisticated clotting tests are unavailable, a sample of the patient's blood in a plain tube should clot within a few minutes in the absence of coagulopathy.

➤ **Creatine kinase** for myolysis.

➤ **Renal function** may be impaired secondary to myoglobinuria, disseminated intravascular coagulopathy or other mechanisms.

➤ **White cell count** is usually only mildly elevated. A significantly raised white cell count may indicate other pathology.

Urine study

➤ **Urinalysis** for haemoglobin, myoglobin.

Differential diagnosis

The combination of neurological disturbance and evidence of coagulopathy in a patient with an appropriate history is strongly suggestive of severe envenomation, but there are other differential diagnoses that should be considered *(Table 1)*.

Management of snakebite

Effective management of snakebite relies upon:

➤ good first aid treatment;

➤ recognition of significant envenomation; and

➤ judicious use of appropriate antivenom and supportive care.

The symptoms and signs of envenomation, and the time course they follow, vary enormously between individual patients. They are influenced by such factors as:

➤ body weight;

➤ amount of venom injected;

➤ age and state of health of the patient;

➤ time elapsed since the bite; and

➤ site of the bite.

Variation between snakes is also important. The size and maturity of the snake and the time since it last injected venom will influence the severity of the envenomation. Some features of envenomation are more prominent in bites from certain species of snakes. Myolysis, for example, is particularly prominent in sea snake and tiger snake envenomations, while death adder venom is predominantly neurotoxic in action. Myolysis may lead to renal failure, which probably accounted for the delayed deaths at 2-7 days described in the past. Both tiger snake and taipan venoms contain post and presynaptic neurotoxins, the latter being difficult to reverse if the patient is not treated promptly. Coagulation disturbance, usually secondary to defibrination, is common after bites from most Australian venomous snakes, although severe haemorrhage is rarely seen. There have been seven published fatal cases of intracerebral haemorrhage.

Identification

Identification of the offending snake aids the choice of the appropriate antivenom and alerts clinicians to look for the envenomation characteristics of that particular snake.

In cases of snakebite involving zoo staff, herpetologists or other experienced snake handlers, the snake's identity may be known (although this cannot always be relied upon). Identification of snakes by the general public or by hospital staff is frequently unreliable, as scale

appearance and colour are variable within species and many species may be confused on superficial inspection. If there is any doubt about the identification of the snake, the bite should be treated as if the snake were unidentified, that is, with a venom detection kit and if necessary with polyvalent antivenom or combined tiger/brown snake antivenom in Victoria. In Tasmania, the only venomous snakes of clinical importance are the tiger snake and the copperhead, both of whose bites may be successfully treated with tiger snake antivenom. For all other areas of Australia, as well as Papua New Guinea, polyvalent antivenom should be used if the type of snake cannot be identified.

First aid

It is salutary to note that in the most recent published survey of deaths from snakebite in Australia 12 deaths were reported over a 3 year period (1992-1994), and effective first aid was not employed in any of these cases.

The **pressure immobilisation method** of first aid has been well described and its efficacy in Australian snake envenomation has been shown both clinically and in the laboratory. It involves firm bandaging of the entire bitten limb from the tip proximally (as tightly as for a sprained ankle) and immobilising both the limb (with splints or slings) and the patient. The aim is to slow the movement of venom from the bite site into the circulation until an appropriate medical facility has been reached and definitive management of the patient can be undertaken. Transportation should be brought to the patient if possible to minimise limb movement.

Bites to the limbs constitute more than 95% of all snakebite cases. If possible, localised firm pressure should be applied to bites on the trunk, head or neck, but breathing should not be restricted. Pressure bandages may be cut away from the bite site to allow swabs to be taken for venom detection and new bandages quickly applied over the site. Once the patient has reached hospital and the appropriate antivenom and other drugs have been assembled, first aid measures may be removed. Bandages and splints should not be left in place for a prolonged period once the patient has reached adequate medical facilities. If, on removal of first aid measures, the patient's condition deteriorates, the bandages can be reapplied while antivenom is administered. It is preferable that such treatment takes place within a critical care area of the hospital where complications arising from envenomation or reactions to antivenom can be managed.

Hospital management of snakebite

Initial management

Once the patient reaches hospital, initial management consists of obtaining intravenous access and resuscitation if required. If possible, a careful history and examination should be undertaken with reference to the features described above, as well as previous envenomations and allergies to antivenom or to horse serum. This will assist in diagnosis and aid decision making with respect to definitive treatment. Samples for venom detection and for pathology should be obtained, and an attempt made to identify the genus of snake, if possible (from clinical and

geographic features, venom detection kit results and from the snake itself, if it has been brought with the patient to hospital). First aid measures may be removed when an intravenous line is in situ and antivenom and resuscitation facilities, including adrenaline, are assembled. If the patient has not developed any symptoms or signs of envenomation, nor any indication of coagulopathy or myolysis on blood taken 4 hours after the removal of first aid (or after the bite if no first aid was used) then the patient has probably not sustained a significant envenomation.

Premedication

If there is evidence of systemic envenomation, then the appropriate antivenom should be chosen and administered after premedication with parenteral antihistamine and **subcutaneous adrenaline** (0.2-0.3 mg, or 0.005-0.01 mg/kg for children). This is particularly desirable for patients previously exposed to antivenom or equine protein. Skin testing for allergic reactions is not recommended, as it is time consuming and the results are unreliable.

The use of adrenaline as a premedication is controversial. There is evidence to suggest that premedication with subcutaneous adrenaline may decrease or prevent allergic reactions to snake antivenoms (CSL Australian snake antivenoms consist of approximately 17% equine IgG protein). However, it has been suggested that adrenaline may cause hypertension and thus contribute to intracerebral haemorrhage in the presence of coagulopathy. There is no definitive evidence to support this view. None the less, it should be stressed that the intravenous route is not recommended for premedication with adrenaline. If the patient shows no evidence of an adverse reaction to the initial dose of antivenom, then premedication need not be repeated before further doses. For patients with a history of previous exposure to antivenom (or other equine protein) or those into whom a large volume of antivenom has been infused, a prophylactic course of prednisolone should be considered to prevent the development of serum sickness.

Antivenom

Before the availability of antivenom, approximately 45% of tiger snake envenomations and more than 90% of taipan envenomations resulted in the victim's death. The decision to use antivenom should be based on the patient's history, examination and pathologic findings, and the type of antivenom used will depend on geographic, clinical and pathologic factors as described above. Antivenom should be diluted in at least 100 mL of normal saline, 5% dextrose or Hartmann's solution, immediately before administration. It should initially be administered slowly while the patient is observed for signs of allergic reaction. If no reaction is observed, then the rate of infusion may be increased. If the patient reacts to the antivenom, the rate may be slowed or ceased temporarily. If the reaction is more severe, then treatment with adrenaline, plasma volume expanders and beta agonists should be undertaken as required. The decision to recommence antivenom should be based on the clinical state of the patient. Although severe reactions are uncommon with Australian antivenoms, physicians should be ready to deal with such an eventuality. In the case of the patient with a known allergy to antivenom or to horse serum, the decision to withhold antivenom should be clinically based, bearing in mind the resuscitation facilities available.

The recommended initial doses of antivenom are included in *Table 2,* and are based on the average amount of venom injected by each of the snakes concerned. There is evidence, however, that these doses may be insufficient to reverse coagulopathy associated with the bites of several Australian venomous snakes, notably the brown snake and the taipan. Larger initial doses should be considered if there is evidence of severe envenomation (multiple bites, rapidly progressive symptoms, large snakes). The dose of antivenom for children should not be reduced according to their weight, since the amount of venom injected by the snake is independent of the victim's size.

Supportive care

The severely envenomed patient may in addition to antivenom, require other treatment measures and supportive care. This should include admission to hospital and observation for a period of at least 24 hours. Regular (ie, at least hourly) neurological observations and repeated pathology studies should be performed to monitor the progression of the illness.

The frequency at which clotting studies should be repeated is uncertain. After circulating antivenom has been neutralised, it may be several hours (4-6 hours) before clotting times return to normal. The lack of improvement in clotting times may represent insufficient antivenom or time before re-testing. Improvement in clotting times may represent the efficacy of antivenom or the natural history of the disease. Worsening of coagulopathy, however, is an indication that circulating procoagulants remain unneutralised, and that more antivenom is required.

Other treatments such as analgesia (avoid sedating agents such as morphine if possible), plasma volume expanders and fresh frozen plasma may be required. Supplemental oxygen and sometimes mechanical ventilation are indicated for severe envenomations in which there is respiratory compromise. Incipient renal failure may be treated with adequate volume replacement and diuretics, but haemodialysis may be required, particularly in cases where treatment has been delayed. Hyperkalaemia secondary to rhabdomyolysis may be treated with calcium, insulin and glucose and resonium. All patients should receive appropriate tetanus prophylaxis and consideration should be given to antibiotic prophylaxis if the bite wound is contaminated. Rarely, the snake's fangs may break and become embedded in the wound, acting as a foreign body and a nidus for infection.

Epidemiology of snakebite

The most recent published survey of deaths from snakebite in Australia found a death rate of around 3.2 deaths per year, although it is likely that the actual number is somewhat higher than this, due to unrecognised snakebite deaths. Before the development of specific antivenom therapy and improved supportive care, snakebite was associated with a high case fatality rate. Most bites occur during the warmer months, when snakes are more active and people are more likely to venture outdoors and into the bush. Recent Australian studies of snakebite incidence demonstrate a preponderance of males among the victims, possibly related to risk taking behaviour or to occupational exposure. It is also of note that several cases of snakebite death

have been associated with alcohol intoxication. Half (six) of the deaths reported in the 1992-1994 survey were due to bites from brown snakes, including several sudden or unexpected deaths (within 1 hour). Tiger snake was the second most common cause of death, involved in four fatalities.

Summary of Important Points

➤ Maintain a high index of suspicion, particularly if the history or presentation is suggestive ('Snake country', rural setting, patient unable to give history, children).

➤ Features to look for:

Clinical

    ➤ puncture marks or scratches; may be oozing blood or serum

    ➤ headache, nausea and vomiting, abdominal pain

    ➤ neurologic disturbance: ptosis, slurred speech, dysphagia, weakness, respiratory difficulty

    ➤ muscle pain and tenderness, dark urine

Pathology

    ➤ coagulopathy, decreased fibrinogen

    ➤ increased creatine kinase

    ➤ myoglobinuria

    ➤ venom detection kit

➤ Not all snakebites result in envenomation. Antivenom is indicated only if clinical or pathologic evidence of envenomation is present.

Poisonous Australian snakes

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Brown snakes

Common or eastern brown snake *(Pseudonaja textilis)*

Snakes of the genus Pseudonaja, which also contains the dugite *(P affinis)* and the gwardar *(P nuchalis),* are found throughout Australia, and are now responsible for the majority of snakebite deaths in this country. Coagulation disturbance is common in brown snake bites, as is neurotoxicity. Myolysis is not a feature of brown snake envenomation, although renal failure may ensue possibly as a result of direct nephrotoxicity or disseminated intravascular coagulation. There is also a suggestion of a possible cardiotoxin present in brown snake venom.

Tiger snake

Tiger snake *(Notechis scutatus)*

Tiger snakes are found in the temperate areas of southern Australia, including Tasmania, where they are particularly large and venomous. Identification of tiger snakes by the presence of stripes is unreliable, since this varies with the seasons and the maturity of the snake, and there is also an unstriped black species *(N ater).* Several other venomous and non-venomous Australian snakes may also be striped. Features of tiger snake envenomation include neurotoxicity (caused by pre-synaptic and post-synaptic neurotoxins), coagulopathy and rhabdomyolysis.

Taipan

Taipan *(Oxyuranus scutellatus)*

This aggressive and highly venomous snake is found along the coast of northern Australia from Brisbane to Darwin. It has the largest fangs and is the longest venomous Australian snake. The clinical syndrome include neurotoxicity, coagulopathy and rhabdomyolysis. Before the development of an antivenom in 1955, an effective taipan bite was invariably fatal.

Black snakes

Red-bellied black snake *(Pseudechis porphyriacus)*

Large Mulga (King Brown) snake *(Pseudechis australis)*

This genus includes the large Mulga or King Brown snake *(Pseudechis australis)* and the red-bellied black snake *(P porphyriacus),* as well as Collett's snake *(P colletti),* the blue-bellied black snake *(P guttatus)* and the Papuan black snake *(P papuanis).* The Mulga snake has the

largest recorded venom output of any snake. It is found throughout Australia, except in Victoria, Tasmania and the most southern parts of Western Australia. The name 'King Brown' snake may lead to confusion and to the incorrect use of brown snake antivenom, and is therefore best avoided. Mulga snake venom contains neurotoxins, myotoxins and procoagulants. Black snake antivenom should be used for envenomation by the Mulga or the Papuan black snake.

The red bellied black snake, while still dangerous, is somewhat less venomous than many other Australian snakes. Its bite may cause coagulopathy, neurotoxicity and myolysis, but no deaths have been confirmed. Its range covers eastern Australia, but not Tasmania. Black snake or tiger snake antivenom may be used, and the latter is preferred due to the smaller volume of protein solution required.

Fierce snake

Fierce snake *(Oxyuranus microlepidotus)*

Also known as the Western or inland taipan or the small-scaled snake, this snake produces the most toxic venom of any snake worldwide. Although its distribution is supposedly limited to a small area of western Queensland, there have been reports of the snake well outside this area, and the true current range is unknown.

Copperhead

Copperhead *(Austreleps superbus)*

This snake is limited to Victoria, Tasmania and the western plains of New South Wales. It is the only venomous snake found above the snowline. It produces copious amounts of venom with neurotoxic, procoagulant and myolytic activity, but rarely causes fatalities. Its bite may be effectively treated with tiger snake antivenom.

Rough scaled snake

Rough scaled snake *(Tropidechis carinatus)*

Also called the Clarence River snake, this bad tempered snake is found only in isolated pockets along the coast of Queensland and northern New South Wales. Its venom contains myolytic, coagulant and neurotoxic components and is neutralised by tiger snake antivenom.

Death adder

Death adder *(Acanthophis antarcticus or praelongus)*

The death adder, which is found throughout Australia with the exception of Victoria and Tasmania, is readily identified by its short squat appearance. Unlike most snakes, the death adder

will not necessarily retreat from humans and may therefore be more easily trodden upon or disturbed by the unwary. Its venom contains a post synaptic neurotoxin, with negligible coagulant or myolytic activity.

Sea snake

Sea snake *(Family Hydrophiidae)*

At least 32 species of sea snake have been recorded in northern Australian waters and some species are also found in the southern waters off Victoria, Tasmania and South Australia. All sea snakes are venomous, and rhabdomyolysis is a major feature of sea snake envenomation, resulting in muscle pain and sometimes trismus. Myoglobinuria develops after 3-6 hours. The bite itself is not particularly painful, and may go unnoticed, distinguishing it from envenomation by stinging fishes or jellyfish, both of which usually cause immediate and often excruciating pain. Envenomation should be treated with sea snake or tiger snake antivenom if sea snake antivenom is unavailable. In the case of the latter, two ampoules should be given initially.