

Notable Cases

Severe tiger snake envenomation in a wilderness environment

Antony Nocera, John Gallagher and Julian White

A 44-year old bushwalker was envenomed in an isolated area late in the afternoon, and a succession of difficulties with his rescue resulted in his not reaching hospital until nearly 24 hours later. This case highlights the problems of prolonged delays before treatment for envenomation, and points to the advantages of physician-staffed emergency helicopter services carrying snake antivenoms into the field. (MJA 1998; 168: 69-71)

The venoms of Australian snakes are uniquely toxic and, as shown in Box 1, envenomation without appropriate antivenom treatment results in significant mortality.¹ While there are no

1: Mortality after envenomation by some Australian snakes without antivenom treatment

Snake*	Mortality
Brown	8.6%
Tiger	45%
Taipan	80%
Death adder	50%
Mulga	30%

*List order reflects most common envenomations.

accurate statistics on incidence, it has been estimated that there are up to 3000 cases of suspected snakebite or envenomation in Australia annually.² Managing severe envenomation in remote areas may pose special problems. We describe a case of severe tiger snake (*Notechis scutatus*) envenomation in a wilderness environment which highlights some of these problems.

Case history

In March 1997, a previously well 44-year-old man was bushwalking alone in a canyon in the Blue Mountains National Park in New South Wales. Late in the afternoon he was bitten on the left ankle by an unseen animal while preparing to wade into a rock pool. His rescue is described in the panel on the following page.

On arrival at Westmead Hospital, nearly 24 hours after envenomation, the patient's temperature was 35.1°C, heart rate 95 beats per minute, and blood pressure 106/71 mmHg. He had frank myoglobinuria and a Venom Detection Kit (Commonwealth Serum Laboratories Ltd, Vic.) urine test was positive for tiger snake venom. Other laboratory findings on admission are shown in Box 2.

The patient's coagulation status (only slightly abnormal) was consistent with the coagulopathy of untreated tiger snake

2: Relevant laboratory findings (normal ranges) on admission

Potassium	4.5 mmol/L (3.4-5.5)
Urea	12.5 mmol/L (2.5-6.5)
Creatinine	200 µmol/L (60-120)
Calcium	1.78 mmol/L (2.13-2.63)
Albumin	24 g/L (35-53)
Phosphate	4.14 mmol/L (0.81-1.45)
Creatine kinase	287 200 IU/L (24-204)
pH	7.16 (7.35-7.45)
Partial pressure of carbon dioxide (PCO ₂)	44 mmHg (35-45)
Partial pressure of oxygen (PO ₂)	75 mmHg (75-100)
Bicarbonate	15 mmol/L (22-26)
Base excess	-13 (-3 to +3)
White cell count	19.7 × 10 ⁹ /L (4-11)
Activated partial thromboplastin time	38 s (25-36)
Prothrombin time	19 s (11-18)
International normalised ratio	1.4 (0.8-1.3)
D-dimer	Positive

envenomation, which typically resolves after 12-18 hours (JW, personal observation). The patient was initially treated with six ampoules of tiger snake antivenom (Commonwealth Serum Laboratories [CSL] Ltd, Vic.), without any clinical response. Two ampoules of polyvalent antivenom (CSL) and a further two of tiger snake antivenom were then given, after which we observed some movement from the patient, and a previously equivocal pupillary light reflex became normal.

Approximately six hours after admission, decompressive fasciotomies were performed down both forearms and hands for compartment syndrome. The development of local tissue oedema and injury sufficient to require fasciotomy is almost unheard of in Australian snakebite. Even for snakes known to cause local tissue injury, fasciotomy should be used as a treatment of last resort for uncontrollable intracompartmental pressures.

The patient was in the intensive care unit for 15 days, requiring intermittent positive pressure ventilation for 13 days, and was discharged to a high dependency unit on continuous positive airway pressure through a tracheostomy. He required haemodialysis for 32 days and was eventually discharged home after 34 days.

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Notable Cases

The rescue operation and prehospital treatment

Approximately 5 pm: Patient bitten on the left ankle by an unseen animal. Applies no first aid measures, attempts to walk back. Is overcome by blurred vision, ptosis, progressive weakness and shortness of breath.

Approximately 9 am the following morning: Accommodation lodge custodian finds the patient, who complains of weakness, shortness of breath and a swollen tongue, stating he has been bitten by either a snake or spider. Lodge custodian notes that the patient has blue lips and tongue. Leaves to raise the alarm.

10:45 am: Police and Ambulance are activated.

12:19 pm: Police Rescue and Ambulance personnel reach the patient, noting cyanosis, temperature of 34.9°C per axilla, heart rate of 104 beats per minute, systolic blood pressure of 80 mmHg, blood glucose level of 6.6 mmol/L, and a 3-cm bruise below the left medial malleolus. Ambulance officers apply a compressive bandage, place the patient in the left lateral position, insert a nasopharyngeal airway and begin giving supplemental oxygen through a face mask.

It is estimated that carrying the patient to the nearest vehicle access point would require 25 people and approximately three hours, plus about a 20-minute drive to the nearest hospital. Radio communications are not possible because of the height of the canyon walls, so a member of the NSW Police Rescue walks back to call for helicopter assistance and to organise additional personnel for a possible "carry out".

12:53 pm: NRMA CareFlight helicopter is dispatched.

1:28 pm: Helicopter arrives over the scene.

2 pm: A NSW Ambulance Service Special Casualty Access Team paramedic and an emergency medicine registrar are winched 150 feet down to the ground party. Patient has a temperature of 35.4°C, heart rate of 105 beats per minute with a normal electrocardiogram monitor trace, systolic blood pressure of 80 mmHg, a Glasgow Coma Score of 12, and a respiratory rate of 30 breaths per minute, is unable to open his eyes, has bilaterally dilated pupils non-reactive to light and cannot raise his arms against gravity.

Patient is centrally and profoundly peripherally cyanosed despite receiving oxygen (8 L/min) by face mask. His tongue is protruding and swollen without any associated airway obstruction, and his speech is laboured and dysarthric. His respirations are almost purely abdominal, with minimal chest wall movement, and air entry is globally poor, with inspiratory and expiratory wheezes and coarse crepitations at both lung bases.

Treatment includes splinting of patient's left leg and application of a compressive crêpe bandage, insertion of two intravenous lines, and giving 1.5 L polygeline. There is no change in his cardiovascular parameters. The ambulance C-size oxygen cylinder expires, increasing the patient's respiratory distress.

Anaphylaxis after snakebite is known,³⁻⁵ but it is not possible to determine whether such a reaction is contributing in this patient. Without oxygen or suction, and with the potential for hyperkalaemia, acute renal failure and coagulopathy, it is decided to give adrenaline to optimise the patient's condition before attempting endotracheal intubation.

Patient is given adrenaline (1 mg by subcutaneous injection and three intravenous injections of 0.1 mg). His dysarthria partially resolves, and his symptoms abate somewhat.

2:30 pm: Helicopter returns, but evacuation of the patient is postponed to allow his airway to be secured before moving him to a suitable winch extraction point. The aircraft C-size oxygen cylinder and additional intravenous fluids are winched to the ground and a second rendezvous arranged for one hour later. Patient is given an additional 1.5 L of polygeline; his systolic blood pressure improves to 100 mmHg. The last 500 mL of polygeline is used to facilitate a rapid sequence induction of anaesthesia with suxamethonium to allow intubation. Oral endotracheal intubation is achieved with the aid of a nylon introducer. Patient is sedated and ventilated; his oxygen saturation (previously registering between 50% and 80%) rapidly improves to 97% on ambient air. A nasogastric tube is passed for gastric decompression.

Approximately 3:30 pm: Weather conditions have deteriorated, preventing the helicopter from returning.

Approximately 3:45 pm: A member of the NSW Police Rescue is dispatched to initiate plans to carry the patient out.

Approximately 3:50 pm: The second C-size oxygen cylinder expires.

4:05 pm: A break in the cloud cover allows the helicopter to return and the patient and physician attendant are winched up. The winch freezes at 70 feet off the ground after the stretcher becomes snared in trees. Crewman manually frees the stretcher.

4:52 pm: Helicopter carrying patient lands at Westmead Hospital.

Discussion

While the role of delayed antivenom administration in treating envenomation is not clear, early administration significantly reduces morbidity and mortality. This is particularly important for envenomation by snakes whose venom causes presynaptic neurotoxicity, such as tiger snakes, taipans and rough-scaled snakes. The principal tiger snake neurotoxin, notexin, is both directly myotoxic and neurotoxic. Its presynaptic neurotoxic action ultimately irreversibly inhibits the release of acetylcholine by destroying the terminal bulbs of neurones, resulting in prolonged paralysis refractory to antivenom therapy.^{6,7}

The period between snakebite and the onset of muscular paralysis may be prolonged.⁶⁻⁸ Muscles vary considerably in their susceptibility to snake neurotoxins, with diaphragmatic movement being preserved even after complete intercostal paralysis.^{7,8} Once basic first aid measures have been applied, snakebite victims should be positioned sitting up (if they can tolerate this) with their backs supported. If back support is not available, victims should be placed in the left lateral position.

Positioning snakebite victims to maximise diaphragmatic function⁹ may be important in a wilderness environment where they may have to be left alone while help is sought.

Lymphatic flow rates from a limb in simulated peripheral envenomation are significantly reduced by applying a compressive bandage to the entire limb, immobilising the limb and strictly preventing the casualty from walking.¹⁰ In a wilderness environment this mandates carrying all potential snakebite victims to the nearest vehicle access point. Helicopter evacuation of suspected snakebite victims promptly transports these patients directly to an appropriate medical facility with minimal movement.

Untreated severe envenomation carries the risk of death and therefore warrants an aggressive medical response. Snakebite should be suspected with field reports of collapse, fitting, weakness, bleeding, dark urine or swelling of the lips and tongue. In the case described here, if the helicopter emergency medical service had been dispatched with first notification, the patient would have reached definitive medical care approximately two hours sooner.

Compared with the estimated time it would have taken to deploy enough people to carry this patient out and transport him by road to the nearest hospital facility, antivenom treatment could have been commenced six hours earlier had it been available on the helicopter. Given the potential for unpredictable delays in extracting patients from wilderness environments, antivenom should be available to begin treatment in the field. Any decision to administer antivenom in the field requires a careful assessment of the relative risks of delaying antivenom therapy versus the problems of anaphylaxis outside a hospital environment.

Air transport of antivenom to a snakebite victim at an isolated Western Australian community health centre with no antivenom supplies has been reported.¹¹ Providing physician-staffed helicopter emergency medical services with appropriately stored supplies of antivenom ensures a ready antivenom reserve for cases of severe envenomation. Helicopter emergency medical services can deploy a critical care doctor with additional antivenom supplies to hospitals in a 200-km radius of Sydney within one hour. When combined with the use of NSW Ambulance Service fixed-wing aircraft, this service could be extended to hospitals in a 450-km radius of Sydney within two hours.

Conclusion

Early antivenom therapy reduces morbidity and the costs of subsequent hospitalisation of individuals with significant envenomation. Thus, this incident highlights the need to dispatch physician-staffed helicopter emergency medical services equipped with antivenom in the initial response to suspected snakebite victims in wilderness environments where logistical and environmental factors may prolong pre-hospital times. Supplying helicopter services with stocks of antivenoms would also have the advantage of providing hospitals with timely access to additional supplies in cases of severe envenomation.

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