

Early and Late Removal of the Pressure Bandage in Brown Snake Envenomation: A Report of Two Cases

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ABSTRACT

Two cases of brown snake envenomation are presented where the duration of bandage application in one patient was prolonged compared with the other patient and was associated with a reduction in the total amount of antivenom required. One patient had the bandage removed 2 hours and twenty minutes after application and required 25 units of brown snake antivenom to neutralise the defibrination coagulopathy and manage an upper gastrointestinal haemorrhage. This patient also sustained an urticarial reaction during administration of the final 5 vials of antivenom. The other patient had the bandage released more than 22 hours after its application and only required a total of 6 units of brown snake antivenom to neutralise the defibrination coagulopathy. In the latter case, there was no reaction to any of the vials of antivenom

These cases suggest that bandage release could be delayed well beyond the usual recommended time to effect a reduction in peak and cumulative venom levels and antivenom requirements. (Critical Care and Resuscitation 2002; 4: 116-118)

Key words: Envenomation, pressure immobilisation, antivenom, coagulopathy

While Australian snakes usually cause only minor local tissue damage at the bite site, envenomation leading to coagulopathy and neuropathy can be life threatening. This has led to the recommendation of a pressure immobilisation technique to slow the centripetal movement of venom along lymphatics or low pressure vascular systems.^{1,2}

Two cases of brown snake envenomation are presented, where the duration of bandage application in one was prolonged (> 22 hr) and was associated with a reduction in the total amount of antivenom required when compared with the other case where the duration of bandage application was 2 hr 20 minutes. The cases suggest that bandage release could be delayed well beyond the usual recommended time,³ to effect a reduction in peak and cumulative venom levels and antivenom requirements.

CASE REPORTS

Patient 1

A 14 year old insulin-dependent diabetic high school student was admitted to the emergency department after being bitten on the big toe by a brown snake. Within fifteen minutes, a firm bandage was applied above the bite site and she was transferred to the local hospital where she vomited small quantities of bright blood.

On examination she was drowsy and weak. Her blood pressure was 100/80 mmHg, pulse 150 beats per minute and pulse oximetry revealed a saturation of 91% breathing air. A rash was visible around the bite site and up to the ankle.

A venom-detection kit at the peripheral hospital recorded a positive result for brown snake venom, so she received one vial of brown snake specific antivenom prior to transfer to our hospital. A second venom

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detection kit analysis confirmed brown snake venom at the bite site. During the administration of a second vial of brown snake antivenom the bandage was removed. This occurred 2 hours and twenty minutes after the envenomation. Intravenous hydrocortisone (100 mg 6-hourly) was administered with the intention of reducing the incidence of serum sickness.

At this stage a coagulopathy was documented, with the plasma fibrinogen level being unrecordable. The blood was also unclottable for 6 hours following the envenomation and for 4 hours after the bandage removal. After 25 vials of brown snake specific antivenom, 2 units of fresh frozen plasma and 8 units of cryoprecipitate, the plasma fibrinogen, APTT and INR levels were able to be measured (plasma fibrinogen level was 0.5 g/L and the APTT and INR were less than 200 s and 10, respectively). However, during the administration of the final 5 vials of antivenom, the patient experienced facial and distal upper limb swelling and a rash. She was given subcutaneous adrenaline (1 mg) and oral promethazine (25 mg) which over the next 24 hours settled the facial and limb oedema.

She returned home less than 48 hours after her severe envenomation, having experienced minimal local tissue injury, no rhabdomyolysis or renal dysfunction, and with a normal coagulation profile.

Patient 2

A 50 year old man with no past history of renal, cardiac or respiratory disease was bitten on his left middle toe while walking barefoot in his property. A "three foot long" brown snake was seen slithering away after he became aware of discomfort in his foot. He washed his foot before returning to his house where he collapsed after complaining of a headache and feeling "queer". His wife applied a firm bandage over his left lower leg within 15 minutes of being bitten.

He was admitted to our hospital oriented and alert. His blood pressure was 120/70 mmHg, pulse was 120 beats per minute and pulse oximetry revealed a saturation of 94% breathing air. There was local bruising at the bite site and a venom detection kit which assessed fluid at the bite site was strongly positive for brown snake venom.

While he had no clinically apparent bleeding, within two hours of the bite his fibrinogen had fallen to less than 1 gm/L, and, along with the APTT and INR, was unrecordable three hours later. His D-dimer recording at this stage was elevated to greater than 3.2 mg/L (normal range < 0.1 mg/L).

He was given hydrocortisone (100 mg i.v) and promethazine (25 mg orally) prior to the first dose of brown snake antivenom. To control the coagulopathy he

received 5 vials of antivenom, which was followed by 8 units of cryoprecipitate and 2 units of fresh frozen plasma.

He developed oliguric renal dysfunction (plasma creatinine peaked at 119 umol/L) and pulmonary oedema. The latter required CPAP for 4 hours and was most likely due to excess intravenous fluid. He had no evidence of rhabdomyolysis (plasma creatinine kinase peaked at 119 U/L) or ECG changes of myocardial ischaemia. The renal dysfunction was attributed to a combination of dehydration, vasodilatation and direct nephrotoxicity of brown snake envenomation.

The bandage was left in place for a total of 23 hours after the bite, and was removed during the infusion of the sixth vial of brown snake antivenom. He suffered no return of his symptoms of envenomation and was discharged home the following morning after a normal coagulation profile had been recorded.

DISCUSSION

The divergent clinical course and antivenom requirements (Figure 1) of these two patients appear to be influenced by the timing of the bandage release, altering the rate at which venom reached the circulatory system to affect coagulation. The time taken to apply the bandage after the bite was approximately 15 minutes in both cases, whereas the removal of the bandage was early (i.e. 2.3 hours) in the first case and late (i.e. 23 hours) in the second case.

It is generally accepted that if application of a broad-based firm bandage is effective in reducing peak venom levels,^{2,4} then it should be left on until the antivenom is immediately available.³ However, it would seem logical to leave the low pressure bandage on for a sustained period, as there may be an advantage in having a slow "lymphatic seep" rather than rapid release of the venom into the circulation.

Australia is unique in having a high proportion of venomous snakes that do not produce significant local tissue damage that may be exacerbated by a pressure bandage that causes venous congestion or increased local concentration of venom. Any problems of prolonged bandage application then amount to inconvenience rather than potential local tissue injury.

Furthermore, the cumulative amount and not just the rate of envenomation may be reduced by prolonged bandage application. It is not clear how much of the venom becomes denatured by temperature and proteolytic enzymes while residing in human tissues with a bandage impeding its centripetal progress, but there is a demonstrable reduction in coagulopathy-inducing proteins.⁵

An argument can be made that the tourniquet should

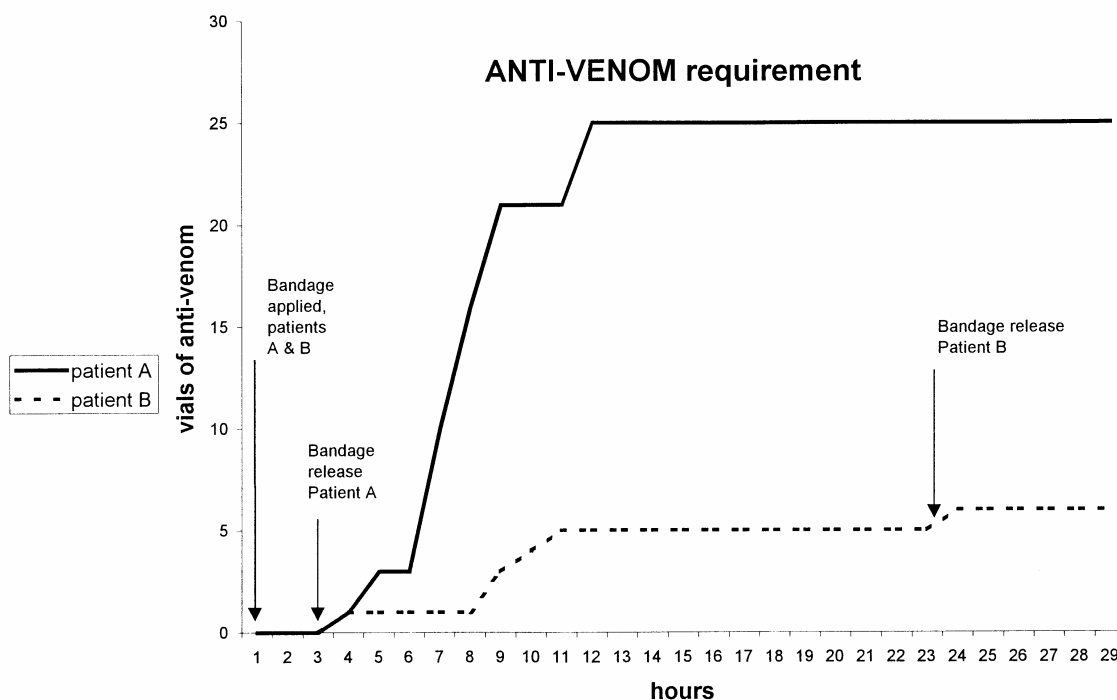


Figure 1. The antivenom requirements and time of administration following the envenomation

remain in place much longer than is the usual practice, with the hope that denaturation of the toxic components will occur to a greater degree, and that the coagulopathic and neurotoxic effects can thus be attenuated without the administration of an unnecessarily large amount of horse serum-based antivenom, with its attendant risks.

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