Brown Snake Envenomation Complicating Near Drowning and Amphetamine Overdose

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ABSTRACT

A case is described of a near drowning and amphetamine overdose in a patient who developed rapidly progressive coagulopathy, thrombocytopaenia and anuria. As the coagulation abnormality was not perceived to be in keeping with the amphetamine toxicity, a septic and toxological screen were performed which revealed a positive urine result for Pseudonaja texilis envenomation. Despite resuscitation using coagulation factors and monovalent antivenom, the patient died. This case highlights the importance of early testing for envenomation when an atypical coagulopathy is present. (**Critical Care and Resuscitation 1999; 1: 360-361**)

Key words: Pseudonaja textilis, coagulopathy, near drowning, amphetamine overdose

The infrequency of reports of fatal envenomation in Australia[1-3] may delay inclusion of the common (or eastern) brown snake *(Pseudonaja textilis)* bite in the differential diagnosis of shocked and unconscious patients with rapidly progressive coagulopathy, thrombocytopaenia and anuria, particularly when the history is atypical.

CASE REPORT

A 49-year-old man was admitted to the hospital emergency department at 0500 hours after he had been found by his partner face down in an outside 'spa' on a warm summer night. The duration of immersion was believed to be no greater than fifteen minutes. The patient was a known intravenous drug user and was hepatitis C positive; otherwise he was previously healthy with no recent infections, malignancy or liver disease. Immediately before this incident he was known to have injected himself intravenously with an unknown amount of amphetamines.

The patient was intubated at the scene by paramedics. His Glasgow Coma Score was 3, pupils were equal and reactive, a gag reflex was present and he was making some spontaneous respiratory effort. Naloxone 1.6mg in divided doses was given parenterally without effect. On admission, his blood pressure was 71/50 mmHg, pulse 175 beats per minute, respiratory rate 48 breaths per minute and temperature 36.9°C. A total of 5mg of metaraminol and 1.5 litres of Haemaccel® were administered intravenously in the emergency department. The patient was transferred to the intensive care unit at 0715 hours, with a blood pressure of 82/47 mmHg, pulse 153 beats per minute and respiratory rate 16 breaths per minute.

Synchronised intermittent mandatory ventilation was initiated at a rate of 16 breaths per minute with pressure support 10 cmH_2O, positive end expiratory pressure 5 cmH_2O and a F_IO_2 of 1.0. The tidal volumes were 750mL, with peak airway pressures of 25 cmH_2O. A central venous catheter, radial arterial line, urinary catheter and nasogastric tube were inserted and cardiorespiratory monitoring instituted.

The patient remained anuric with a BP100/50 mmHg after 1 litre of 4% albumin in 0.9% saline solution and an adrenaline infusion at 4.7μg/minute were administered. The initial coagulation profile was within normal limits with a prothrombin time (PT) of 12 seconds, activated partial thromboplastin time (APTT) 28 seconds (normal < 32 sec) and fibrin degradation products (FDPs) of 1.5μg/mL (normal < 10 μg/mL). The haemoglobin was 140 g/L, white cell count 9.6 x 10^9/L and platelet count 260 x 10^9/L. Arterial blood gases (ABGs) with an inspired F_IO_2 1.0 revealed a pH 7.34, carbon dioxide tension ($PaCO_2$) 29 mmHg, oxygen tension (PaO_2) 211 mmHg, bicarbonate (HCO_3^-) 15.1mmol/L and a base excess (BE) of -10.7. Plasma

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toxicology revealed ethanol < 2.2 mmol/L and absence of paracetamol. By 1000 hours the patient's F_IO_2 was 0.4. The ABG at this time revealed a pH 7.31, $PaCO_2$ 33mmHg, PaO_2 155mmHg, HCO_3^- 16mmol/L and BE -10.1. The chest X-ray was clear.

However, excessive bleeding was noticed from his central line insertion site and 60 mL/h of fresh blood was aspirated from the nasogastric tube. The coagulation profile was repeated two hours after the initial sample, which showed a PT 57 secs, APTT > 300 secs, fibrinogen 0.2 g/L and fibrin degradation products > 12 μg/mL.

While ingestion of 3,4 methylene dioxymet-amphetamine (MDMA, 'Ecstasy') has been reported to produce coagulopathy and renal failure, it is usually in association with hyperthermia[4] which was not present in this case. Other tests were performed to detect unusual causes of coagulopathy, one of which was a urine test for the common brown snake *(Pseudonaja textilis)* envenomation. The rapid sandwich enzyme immunoassay method was used which has a high sensitivity and specificity.[5] It returned a positive result for *Pseudonaja textilis.* Monovalent antivenom was commenced and 8,000 units given parenterally. At 2200 hours the haemoglobin was 91 g/L and platelet count was 43 x 10^9/L, and by 2338 hours, had fallen to 84 g/L and 37 x 10^9/L respectively. This was despite transfusion of 2 units of packed cells, 4 units of fresh frozen plasma, 10 units of cryoprecipitate and 4 units of platelets. Although the adrenaline infusion was increased up to 60μg/min and a noradrenaline infusion started and increased to 60μg/min, the patient was unable to maintain an adequate circulation and died early the following morning, 18 hours after presentation. There was no evidence of haemolysis or rhabdomyolysis (creatinine kinase was 114 U/L). An autopsy revealed no cardiac abnormality, no obvious 'bite' marks or occult cause of disseminated intravascular coagulation or sepsis. Subsequent questioning of the patient's associates revealed that the patient kept snakes.

DISCUSSION

The common brown snake *(Pseudonaja textilis)* possesses the second most toxic venom in the world, containing presynaptic neurotoxins, procoagulants, cardiotoxins and nephrotoxins. The procoagulant has a prothrombin activator, which stimulates disseminated intravascular coagulation with active secondary fibrinolysis, which may be total within 30 minutes of the painless, difficult to detect bite.[6] A total of 4.69 + 0.85 mg venom may be injected subcutaneously, or into deeper tissues, with a first bite.[7] Common brown snake envenomation refractory to administration of appropriate antivenom and inotropic support is unusual,[8-10] as is a related coagulopathy accompanied by thrombocytopaenia and a falling haemoglobin level.[11] This presentation was complicated further by the amphetamine overdosage and near drowning, hindering a rapid diagnosis and exacerbating the lethal nature of this disorder.

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