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Case Report

Exotic snake bite: a challenge for the Scandinavian anesthesiologist?

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Background: Venomous snake bites are uncommon in the Scandinavian countries. Envenomation from exotic snakes do however occur, mostly amongst snake handlers. This case report documents the effects and treatment for envenomation from *Hoplocephalus bungaroides*, or the Broad-Headed snake, native to eastern and southern Australia. Snakes of the genus *Hoplocephalus* have previously been described as of 'lesser medical importance' because of their rarity.
Methods: This case report describes the signs, symptoms and management of systemic envenomation in a previously healthy man.
Results: The patient developed signs of severe coagulopathy less than an hour after envenomation. There was also biochemical evidence of rhabdomyolysis, and cardiotoxicity. At no time did the patient develop respiratory insufficiency, neurotoxicity or renal failure. The patient was initially managed with i.v. crystalloids, plasma, corticosteroids and antifibrinolytics and by observation in the intensive care unit (ICU). Coagulopathy resolved after causal treatment with monovalent Tiger snake antivenom.
Conclusion: The patient made good progress and was well on discharge from the ICU 26 h postenvenomation.

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Key words: Coagulopathy; envenomation; *Hoplocephalus bungaroides*; poisons; snake bite; toxicology.

VENOMOUS snake bites are uncommon in Scandinavia. The only known venomous snake in this region is the European or Common Adder (*Vipera berus*), whose bites are usually associated with local rather than systemic effects (1).

Systemic envenomation is well described for many exotic snake species (2–4), and although uncommon, it poses a problem also in the Nordic countries with bites occurring primarily amongst snake handlers. Denmark and Sweden have few restrictions regarding the import of exotic snakes of nonendangered species, as long as the owner is officially registered and creature's origin can be documented (6, 7, and personal communication, Dr Anna Landgren, Giftinformationscentralen, Stockholm, Sweden). Norway, on the other hand has banned the import of exotic animals that are to be kept in captivity of any form or as pets or domesticated animals (6).

The effects of Australia's many dangerous snake species have been well documented. Because of its rarity however, bites from the genus *Hoplocephalus* have not been well described and have been considered to be of 'lesser medical importance'. Here, we report a case of systemic envenomation from *Hoplocephalus bungaroides*, or the Broad-Headed snake in a previously healthy 33-year-old professional herpetologist.

Case report

A 33-year-old previously healthy male with no known allergies presented to the emergency department less than 1 h after being bitten by a Broad-Headed snake.

On hospital admission the patient was irritable, but alert and oriented. He was hemodynamically stable with a regular pulse rate of 130 beats per minute and blood pressure of 130/90 mmHg. There was spontaneous respiration at a rate of 12 min^{-1}, with a SaO2 of 95–97% with 5 l min^{-1} of oxygen via a face mask.

The patient complained of nausea and pain around his left hand, arm and thigh. A general examination revealed puncture marks to the left hand, in the web space between the fourth and fifth fingers, and

on the thenar eminence as well as to the left posterior thigh. The affected limbs were edematous and erythematous. There was a hematoma over the left eye, and scattered ecchymoses over the left arm and thigh. There was one episode of hematemesis (fresh blood) in the emergency room. Macroscopic hematuria and epistaxis were also noted. There were no abnormalities on auscultation of the heart and lungs. Palpation of the abdomen was unremarkable and the patient was neurologically intact.

Two large-bore i.v. lines were established. An arterial line for blood sampling and continuous monitoring of blood pressure as well as an urinary catheter were inserted. Peripheral oxygen saturation and a 5-lead ECG were continuously monitored. The patient received 1 l of Ringer's acetate, ranitidine 50 mg i.v., klemastin (Tavegyl) 2 mg i.v., hydrocortisone 100 mg i.v. and tetanus prophylaxis. He was admitted to the intensive care unit (ICU) 15 min after presentation to the emergency room. The Poisons Information Center was contacted for more information and to procure antivenom.

A 12-lead ECG revealed nodal bradycardia with multiple supraventricular and ventricular extrasystoles. The results of his biochemical tests on admission and for the next 55 h are shown in Table 1. Fluid resuscitation was begun with plasma and Ringer's acetate. Because of the risk of nephropathy resulting from rhabdomyolysis, copious i.v. fluids and furosemide were administered with the aim of forcing diuresis. Antifibrinolytic therapy with tranaxemic acid 1 g i.v. was given 4-hourly. Rantidinine and hydrocortisone i.v. were repeated 8-hourly.

At this stage the Poisons Information Center in Sweden and Denmark had been contacted, however, no antivenom was available. The patient's coagulation status continued to deteriorate over the next 8 h. The APTT was persistently over 200 s, INR reached a maximum of 1.6, 5 h after the bite, and there was evidence of active fibrinolysis. Clinically the patient continued to have macroscopic hematuria, epistaxis and hematemesis, but remained hemodynamically stable. There were no signs of cardiorespiratory failure, and the abnormal ECG findings resolved spontaneously after 8 h in the ICU. There were no detectable neurological deficits.

Appropriate antivenom was finally located at the Toxicology Institute in Munich, Germany. Causal treatment with 6000 units (two vials) of monovalent Tiger snake antivenom was given approximately 11 h after envenomation.

The patient's coagulation status improved markedly within 2 h of the antivenom administration. The APTT fell to 58 s and INR to 1.3. Six hours after antivenom administration, the coagulopathy had resolved and there were no longer any clinical signs

Table 1

Laboratory data 1–55 h after admission (reference range).

	Number of hours after the incident								
	1	2	5	9	13	17	21	25	55
Haemoglobin (131–163 g l^{-1})	152	149	123	109	116	105	107	102	106
Platelets (140–400 $\times 10^9$ l^{-1})	259		203	168	166	–	–	154	145
WCC (4.0–10.0 $\times 10^9$ l^{-1})	9.2							21.0	12.0
APTT (25–36 s)	>200	>200	>200	>200	58	36	31	30	25
INR (<1.2)	1	1.3	1.6	1.4	1.3	1.4	1.4	1.3	1.2
Fibrinogen (2.0–4.0 g l^{-1})		<0.5	<0.5	<0.5	<0.5	0.9	1.4	1.8	4.1
D-dimer (<0.3 mg l^{-1})		>20	>20	>20	>20	>20	>20	>20	1.6
ATIII (60–140%)		74	66	61	69	80	81	81	
CKMB (<5 μg l^{-1})	1.9								1.0
Troponin T (<0.06 μg l^{-1})	<0.05								
CK (<3.3 μkat l^{-1})			2.3			17.6		25.1	19.7
Creatinine (55–116 mmol l^{-1})	90							74	100
Urine myoglobin (arbitary units)							0		0
AST (<7 μkat l^{-1})	0.63							0.73	0.66
ALT (<7 μkat l^{-1})	0.50							0.37	0.34
GGT (<0.8 μkat l^{-1})	0.26							0.32	0.29
Bilirubin (<20 μmol l^{-1})	<2							22	4
CRP (<5 mg l^{-1})	<5							59	
Na (136–146 mmol l^{-1})	145	144	139	139	137	136	138	137	138
K (3.2–4.7 mmol l^{-1})	3.7	3.7	3.5	3.4	3.6	3.7	3.8	3.6	3.5
Lactate (0.7–2.5 mmol l^{-1})			2.5	1.6	1.4	1.7	1.6	1.5	

WCC, white cell count; APTT, activated partial thromboplastin time; INR, international normalised ratio; ATIII, antithrombin; CKMB, creatine kinase (mb fraction); CK, creatine kinase; AST, aspartate transaminase; ALT, alanine aminotransferase; GGT, gamma glutanyl transferase; CRP, C-reactive protein.

Table 2

Suggested treatment protocol.

First aid
ABC
Keep the limb as still as possible. Immobilize with splints and bandages.
A person that claims that he/she has been bitten should be observed for 12 h in hospital, bitemarks may not be visible
History and examination
Time, number and site of bites
Symptoms and signs of neurotoxicity, coagulopathy, cardiotoxicity, rhabdomyolysis, renal failure
Identify the snake
Contact the local Poisons Information Center for further information
Medical management
Two large bore i.v. lines
In-dwelling urinary catheter
Continuous ECG
Continuous SaO_2
Consider arterial line
Fluid resuscitation, including plasma, platelets as required, consider forced diuresis
Close observation and treatment in the ICU for:
• Neurotoxicity (ptosis, dyspnoea, weakness progressing to paralysis and respiratory failure)
• Coagulopathy (bleeding from injection sites, epistaxis, hematemesis, hematuria)
• Cardiotoxicity (bradycardia, any arrythmia)
• Rhabdomyolysis
• Renal failure
Causal treatment
Monovalent or polyvalent antivenom

of bleeding. He received a total of 12 units of plasma and 81 of crystalloids. There was no evidence of nephrotoxicity, cardiotoxicity and neurotoxicity. The patient remained in the ICU for a total of 26 h before being discharged to the medical ward for a further observation period of 2 days. The affected limbs were checked daily for evidence of compartment syndrome, of which there was none. He was finally discharged to the care of his general practitioner 3 days after the incident.

Discussion

Venomous snake bites are uncommon in the Scandinavian countries. In fact, only one venomous snake exists, *V. berus*, more commonly known as the European or Common Adder, whose bites are usually associated with local, rather than systemic effects. Envenomation from exotic snakes do however occur, mostly amongst herpetologists/collectors in private settings (7). The Poisons Information Center in Sweden handles 20–30 cases of exotic snakebites per year (personal communication, Anne Landgren, Giftinformationscentralen, Stockholm, Sweden).

Although Australia experiences approximately 3000 snakebites per year, fatalities are uncommon. Moreover, most deaths are a result of the more commonly found brown snakes (genus *Pseudonaja*) (3). Bites from *H. bungaroides* are rare. Even in Australia, bites from this species are extremely uncommon, with only three cases described (personal communication, Drs G. Hawdon and K. Winkel, Australian Venom Research Unit, Melbourne, Australia). Therefore, there is little documentation in humans as to the effects of envenomation. This case report documents the effects and treatment as a result of envenomation from *H. bungaroides*, or the Broad-Headed snake, native to eastern and southern Australia. Snakes of the genus *Hoplocephalus* have previously been described as of 'lesser medical importance' because of its rarity, however, this case report provides evidence that this snake may be more dangerous than previously thought.

The patient in this case was bitten several times in the hand and thigh, suggesting that a reasonably large quantity of venom was injected. As with most other venomous snakes in Australia, a distinguishing feature of envenomation from *H. bungaroides* in this case was severe defibrination coagulopathy developing less than an hour after the bite. There was evidence of rhabdomyolysis, with CK levels of 17.6 $\mu kat\,l^{-1}$ (normal range $<3.3\,\mu kat\,l^{-1}$) in a blood sample taken 17 h after envenomation. This remained elevated for 3 days after the bite. As envenomation from *Hoplocephalus* are rare, no specific antivenom against *H. bungaroides* exists. The recommended antidote is the monovalent Tiger snake antivenom (8, and personal communication Drs G. Hawdon, Australian Venom Research Unit, Melbourne, Australia and

A. Landgren, Giftinformationscentralen, Stockholm, Sweden). In this case the patient received a single dose of 6000 units (two vials) i.v. with resolution of coagulopathy occurring shortly (approximately 2 h) after administration. Interestingly, no signs of neurotoxicity were seen in this case, although this has previously been described (9).

As a general rule (Table 2), the patient should be kept still and pressure immobilization bandages applied (3, 4). In-hospital management of the patient should consist of close observation initially with bandages for at least 4 h, and if symptoms fail to develop, continued close observation for a further 12–24 h after slowly removing the bandages. Clinicians should be mindful of the coagulopathic, myopathic, cardiotoxic and neurotoxic effects of systemic envenomation. In addition, renal failure may occur as a result of myolysis, the development of microthrombi or direct toxicity. ECG changes have also been described with ST segment changes and bradycardia. Treatment (10–12) includes respiratory and circulatory support, and may include artificial ventilation and the administration of plasma in more severe cases. Definitive treatment usually requires the administration of antivenom (10, 13), preferably directed specifically against the genus (monovalent), as polyvalent antivenom is associated with a higher incidence of anaphylaxis. The use of Venom Detection Kits (10) is helpful in this regard. In the Nordic countries however, most owners are aware of the genus and species of their snakes, and often even know where to obtain antivenom so that Venom Detection Kits are often not needed.

Expert help should be sought early, and local Poisons Information Centres should be helpful in this regard.

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