

## Boomslang envenomation in 2 dogs in KwaZulu-Natal, South Africa

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### ABSTRACT

Although snakebites are frequently seen in small animal practice in South Africa, boomslang (*Dispholidus typus*) bites are infrequent due to their shy habits. Boomslang venom is a potent procoagulant, causing a consumption coagulopathy and profuse haemorrhage. Boomslang monovalent antivenom is the most effective treatment. This case report describes and discusses 2 small dogs that were presented to a private practice after being bitten by the same boomslang. Boomslang monovalent antivenom administration to both resulted in cessation of bleeding within 45 minutes. One of the dogs developed severe adverse reactions to the antivenom, including vomiting, dyspnoea and nystagmus, which responded well to intravenous cortisone and symptomatic treatment.

**Key words:** antivenom, boomslang DIC, coagulopathy, dog, snakebite.

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### INTRODUCTION

Snakebites in dogs are frequently seen in small animal practice in South Africa<sup>14</sup>. Treatment, however, is usually symptomatic which includes large volumes of intravenous fluids<sup>13</sup>. In human and canine cases, the snake is often not identified, the patient presents a day or more after the event and the history is vague<sup>1,6,13,14</sup>. Boomslang (*Dispholidus typus*) bites, in dogs and humans, are infrequent as these snakes are arboreal, placid and shy<sup>6,13,14,15</sup>. Boomslang venom is a potent procoagulant that, in humans, activates prothrombin (factor II) and factor X resulting in disseminated intravascular coagulation (DIC), a consumptive coagulopathy, with consequent haemorrhage<sup>1,6,9,11</sup>. Thrombocytopenia is inconsistent and may be delayed in some cases<sup>1,3,9</sup>. In dogs and humans DIC may result in multiple organ failure, of which the kidneys and lungs are usually the most severely affected<sup>6,7,10,13</sup>. Small dogs are often the most severely affected due to the higher venom to mass ratio<sup>11,13</sup>.

This case report describes the clinical signs, treatment and outcome of 2 dogs from the same household in South Africa that were bitten by a boomslang.

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### CASE HISTORIES

#### Case 1: The Jack Russell Terrier

A 5-year old neutered male Jack Russell terrier was presented, together with the cranial half of an olive-brown snake, to a veterinary clinic near Durban early one morning. The snake was identified as a boomslang (*Dispholidus typus*) by the veterinarian and later confirmed by a KwaZulu-Natal Parks Board officer. The Jack Russell Terrier was alert and appeared clinically normal apart from bleeding profusely from 2 parallel punctures 4 mm long and 12 mm apart on the inner surface of the right ear pinna. According to the owner the bleeding had started immediately after the snakebite, 18 hours earlier, and did not respond to applied pressure and ice. A peripheral blood smear did not show any obvious abnormalities and the haematocrit was 35 %, determined by microcapillary tube centrifugation. A padded pressure bandage was applied to the venipuncture site and digital pressure was applied to the blood smear puncture site for 15 minutes to control bleeding. A diagnosis of boomslang envenomation was made and 2 vials of boomslang monovalent antivenom were ordered directly from the South African Institute of Medical Research (SAIMR)\* in Johannesburg to be couriered overnight, because the local human hospitals did not stock boomslang antivenom. The patient was treated with an intravenous crystalloid

infusion (Sabax Ringer-Lactate) at a flow rate of 1½ times maintenance (33 ml/hr) and intravenous ampicillin (Ranamp, Ranbaxy) at 20 mg/kg twice a day. The dog continued to bleed profusely in hospital, despite a padded pressure bandage, which started seeping blood within 1 hour of application. Owing to delay in the delivery of the antivenom, the uncontrollable haemorrhage, and because a normal haematocrit initially occurs following acute blood loss, a whole blood transfusion was given as interim treatment<sup>6,13,20</sup>. Five hundred ml of fresh whole blood, from an unmatched canine donor, was administered slowly (5 ml/kg/hour) intravenously. This had no clinical effect on the haemorrhage. The dog's appetite remained good during the day and night.

By the time the boomslang antivenom arrived, 44 hours after the snakebite, the patient was depressed and clinically anaemic (pale mucous membranes, weak pulse, normal temperature and respiration, normal capillary refill time, lethargic but responded when called by name). The punctures on the dog's ear were still bleeding profusely. There were no signs of haemorrhage from anywhere else on the dog. The microhaematocrit had decreased to 11 % despite the blood transfusion. The dog was given 7 ml boomslang antivenom slowly intravenously and the bleeding stopped within 45 minutes after administration. The dog started vomiting 90 minutes after the antivenom administration and was treated with metaclopramide (Clopamon, Pharmcare) at 0.5 mg/kg intravenously. The dog also started showing very fast nystagmus (rapid phase to the left) and became dyspnoeic (inspiratory and expiratory) and tachypnoeic. On auscultation inspiratory crackles could be heard. The dog was treated with hydrocortisone sodium succinate (Solu-Cortef, Pharmacia) at 14 mg/kg intravenously and furosemide (Salix, Intervet) at 2 mg/kg intravenously for the adverse respiratory reactions to the antivenom. The nystagmus slowed down and the dog's respiration improved shortly thereafter. Three hours later, the adverse respiratory reaction had resolved, the dog's respiration had normalised and the nystagmus had

Table 1: Laboratory coagulation profile of the 2 dogs before and 4½ hours after antivenom was administered.

Blood test	Jack Russell terrier		Dachshund		Control dog	Reference range*
	Before	After	Before	After		
Partial thromboplastin time (PTT)	>180 s (failed to clot)	37 s	>180 s (failed to clot)	12 s	19 s	11–19 s
Prothrombin time (PT)	>180 s (failed to clot)	20 s	>180 s (failed to clot)	7 s	8 s	5–12 s

\*Bouwer & Partners, Durban.

stopped. An iron-containing tonic (Hep-tonic, Kyron) was given orally at 5 ml twice daily.

Blood coagulation profiles were run before the antivenom was administered and 4½ hours thereafter. Although the results of the latter sample were still out of the normal range, the improvement in response to the antivenom was marked and the uncontrollable bleeding had stopped. (Table 1.)

On the 3rd day (24 hours after antivenom was administered) the dog's habitus and colour had improved, although the dog vomited once in the morning. This was treated with metaclopramide as well as ondansetron (Zofran, GlaxoSmithKline) at 0.1 mg/kg intravenously. The dog started eating small amounts of Hills a/d in the afternoon and was discharged on the 4th day with amoxicillin (Moxymax, MDI) at 20 mg/kg twice daily for another 4 days and an iron containing tonic. Although the dog was still mildly subdued, the microhaematocrit had increased to 27 %.

### Case 2: The Dachshund

A 5-year old neutered male Dachshund from the same owner was presented, at the same time as the Jack Russell Terrier, to the same veterinary clinic. The Dachshund was bleeding very slowly from 2, 4 mm parallel punctures, 12 mm apart, adjacent to the manubrium of the sternum. The bleeding had started immediately after the snakebite 18 hours earlier and had slowly continued. The dog was otherwise clinically normal. A peripheral blood smear did not show any obvious abnormalities. Prolonged digital pressure was required to control bleeding from the blood smear puncture site. A diagnosis of boomslang envenomation was made. The dog's habitus was good and remained so during the entire stay in the hospital. The area around the wounds was shaved and cleaned. The dog was treated with intravenous crystalloid infusions (Sabax Ringer-Lactate) at a flow rate of 1½ times maintenance (40 ml/hr) and was given ampicillin (Ranamp, Ranbaxy) at 20 mg/kg twice a day intravenously. The overnight monitoring was unremarkable

and no additional treatment was given until the antivenom arrived. The dog was treated with 3 ml boomslang monovalent antivenom slowly intravenously and the bleeding stopped within 45 minutes.

The Dachshund vomited once, 3 hours after the antivenom administration. This was treated with metaclopramide (Clopamon, Pharmicare) at 0.5 mg/kg intravenously. The dog soon started eating again and was discharged the same day with amoxicillin (Moxymax, MDI) at 20 mg/kg twice daily for another 4 days.

Blood coagulation profiles were run before the antivenom was administered and 4½ hours thereafter. The latter results were within the normal range and lower than the control sample. (Table 1.)

### DISCUSSION

The only specific treatment for boomslang envenomation is boomslang monovalent antivenom<sup>1,6,11,20</sup>. A unit of antivenom (10 ml) neutralises a fixed quantity of venom and therefore is not given as a dose per kilogram<sup>13</sup>. In these 2 dogs the symptoms were assumed to be indicative of the volume of venom received. The Jack Russell showed more severe clinical signs than the Dachshund and was therefore given a larger quantity of the antivenom. A 2nd vial was available had the bleeding not stopped within 1 hour. A small test dose is no longer required by the SAIMR in their package insert and was therefore unnecessary<sup>13,20</sup>.

No additional laboratory tests were done on these 2 dogs to confirm the presence of DIC such as fibrinogen, fibrinogen degradation products (FDP), d-dimer or a full blood count<sup>3,10</sup>. In humans, boomslang venom causes DIC, although thrombocytopaenia is not always initially evident<sup>1,3,6,9,17</sup>. It has been extrapolated from the human literature that boomslang envenomation can cause DIC in dogs<sup>13,17,20</sup>. DIC was presumed in these 2 dogs based on the history and clinical examination, despite the normal appearing blood smear. Peripheral blood smears are not always a true reflection of central venous cell counts<sup>19</sup>.

Although heparin has been recommended in the past as a treatment for DIC and specifically venom induced DIC, it is

controversial<sup>1,6,7,10,20</sup>. More recently it has been said to be contraindicated as boomslang venom-induced DIC is heparin resistant<sup>11,13,21</sup>.

The anticoagulant effect of heparin is dependent on adequate antithrombin III (AT III) levels, which are depleted in venom-induced canine DIC (AT III levels may also be depleted in other forms of canine DIC)<sup>8,10,13</sup>. Fibrin-stabilising and thrombolytic drugs are also contra-indicated. Fibrin-stabilising drugs prolong the lifespan of micro-thrombi and aggravate end-organ ischaemia and hypoxia which may be fatal. Physiological fibrinolysis is necessary to clear the micro-thrombi. It is the coagulation, not the haemorrhage, which has the greatest impact on morbidity and mortality<sup>3,10</sup>. Intravenous fluids help to maintain circulation and reduce the possibility of multiple organ failure<sup>7,10</sup>.

The fresh whole blood transfusion of the Jack Russell failed to prevent continued bleeding as the transfused clotting factors were probably consumed by the venom activity<sup>1</sup>. This may have aggravated the DIC by 'feeding the fires' of the coagulopathy<sup>6</sup>. Fresh or fresh frozen plasma could also have been used as a source of clotting factors, as the haematocrit was still relatively high<sup>20</sup>. The red blood cells may have been life saving as the microhaematocrit decreased from 35 % to 11% despite the transfusion (part of the decrease in haematocrit would have been due to the dilutional effect of the intravenous fluids). Had the blood not been administered, the microhaematocrit may have been lower. More blood transfusions may have maintained a higher haematocrit, but may also have aggravated the DIC<sup>6</sup>.

Another blood transfusion after the antivenom administration, which was the definitive turning point, may have resulted in a more rapid recovery.

The vomiting experienced by both patients was a typical adverse reaction to the antivenom. The adverse respiratory reaction and nystagmus were probably also reactions to the antivenom, but may also have been due to the DIC. The timing of these reactions (1½ and 3 hours after antivenom administration) is more indic-

ative of a delayed antivenom reaction. Short-acting corticosteroids are indicated for delayed antivenom reactions and diuretics for the adverse respiratory reaction and possible cerebral oedema<sup>2,6,16</sup>. Adrenalin is the drug of choice for acute anaphalactic reactions<sup>2,16</sup>. In humans, adverse antivenom reactions occur in 2–56 % of cases and late serum sickness-like reactions in more than 50% of patients<sup>2,12,18</sup>. Serum sickness (characterised by pruritic skin rash, pyrexia and arthralgia about 10 days after treatment) is seldom seen in dogs<sup>13</sup>. The iron containing tonic was given to provide essential nutrients that were lost during the prolonged bleeding episode, to support new red blood cell production. Owing to the critical condition of the dogs, antibiotics were administered to prevent secondary infections. Snake mouths may carry various bacteria and the open bite wounds may become secondarily infected<sup>5,13,20</sup>. However, snake venom has antibacterial properties and sepsis in human snakebite victims is usually associated with haematoma or necrosis at the bite site<sup>4</sup>.

Although the Dachshund continued to bleed after admission to hospital, it was mild, and the dog was never in a critical or life threatening condition. This dog may have recovered with symptomatic and supportive therapy alone, including fresh or fresh frozen plasma<sup>17,20</sup>. However, the antivenom administration was again the definitive turning point. Antivenom is readily available and can be couriered to most parts of South Africa overnight\*.

\*Antiserum may be acquired from the SAIMR: Tel: 011 386 6016; Fax: 011 386 6016; PO Box 28999, Sandringham, 2131 South Africa.

## REFERENCES

1. Aitchison J M 1990 Boomslang bite – diagnosis and management. A report of 2 cases. *South African Medical Journal* 78: 39–42
2. Anonymous 2006 Evidence behind the WHO guidelines: hospital care for children: should s/c adrenaline, hydrocortisone or antihistamines be used as premedication for snake antivenom? *Journal of Tropical Pediatrics* 52: 155–157
3. Bick R L, Arun B, Frenkel E P 1999 Disseminated intravascular coagulation, clinical and pathophysiological mechanisms and manifestation. *Haemostasis* 29(2–3): 111–134
4. Blaylock R S 2000 Antibacterial properties of KwaZulu Natal snake venoms. *Toxicol* 38: 1529–1534
5. Blaylock R S 2001 Normal oral bacterial flora from some southern African snakes. *Onderstepoort Journal of Veterinary Research* 68(3): 175–182
6. du Toit D M 1980 Boomslang (*Dispholidus typus*) bite. A case report and a review of diagnosis and management. *South African Medical Journal* 57: 507–510
7. DuFort R M, Matros L 2005 Acquired coagulopathies. In Ettinger SJ, Feldman EC (eds) *Textbook of veterinary internal medicine, 6th edn, Vol. 2*. Elsevier Saunders, Missouri: 1933–1937
8. French J K, Edmond J J, Gao E, White H D, Eikelboom J W 2004 Adjunctive use of direct thrombin inhibitors in patient receiving fibrinolytic therapy for acute myocardial infarction. *American Journal of Cardiovascular Drugs* 4(2): 107–115
9. Gomberts E D, Demetriou D 1977 Laboratory studies and clinical features in a case of boomslang envenomation. *South African Medical Journal* 51: 173–175
10. Guillermo Couto C 2002 Disseminated intravascular coagulation. *Proceedings of the 27th World Small Animal Veterinary Association Congress, Granada, Spain, 3–6 October 2002*. <http://www.vin.com/proceedings>
11. Hutton R A, Warrell 1993 Action of snake venom components on the haemostatic system. *Blood Reviews* 7: 176–189
12. Jurkovich G J, Luteran A, McCullar K, Ramenofsky M L, Curreri P W 1988 Complications of crotalidae antivenom therapy. *Journal of Trauma* 28(7): 1032–1037
13. Leisewitz A L, Blaylock R S, Kettner F, Goodhead A, Goddard A, Schoeman J P 2004 The diagnosis and management of snakebite in dogs – a southern African perspective. *Journal of the South African Veterinary Association* 75(1): 7–13
14. Lobetti R G, Joubert K 2004 Retrospective study of snake envenomation in 155 dogs from the Onderstepoort area of South Africa. *Journal of the South African Veterinary Association* 75(4): 169–172
15. Marais J 1986 In *Snakes versus man: a guide to dangerous and common harmless snakes of Southern Africa*. Macmillan, South Africa: 52–57
16. Otten E J, McKimm D 1983 Venomous snakebite in a patient allergic to horse serum. *Annals of Emergency Medicine* 12(10): 624–627
17. Otto J, Blaylock R 2003 Vine snake (*Thelotornis capensis*) bite in a dog. *Journal of the South African Veterinary Association* 74(1): 27–28
18. Smalligan R, Cole J, Brito N, Laing G D, Mertz B L, Manock S, Maudlin J, Quist B, Holland G, Nelson S, Lalloo D G, Rivadeneira G, Barragan M E, Dolley D, Eddleston M, Warrell D A, Theakston R D G 2004 Crotaline snake bite in the Ecuadorian Amazon: randomised double blind comparative trial of three South American polyspecific antivenoms. *British Medical Journal* 329: 1129–1135
19. Topper M J, Welles E G 2003 Hemostasis. In Latimer K S, Mahaffey E A, Prasse K W (eds) *Duncan & Prasse's veterinary laboratory medicine 4th Edn*. Blackwell Publishing, Iowa: 99–135
20. Vaughn-Scott T, Lobetti R G 1995 Boomslang envenomation in a dog. *Journal of the South African Veterinary Association* 66(4): 265–267
21. Warrel D A 1999 WHO/SERA guidelines for the clinical management of snakebites in south east Asian region. *Southeast Asian Journal of Tropical Medicine and Public Health* 30: 1–85