



Case report

Severe acute pulmonary haemorrhage and haemoptysis in ten dogs following eastern brown snake (*Pseudonaja textilis*) envenomation: Clinical signs, treatment and outcomes



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ABSTRACT

This report describes a series of ten cases of fulminant pulmonary haemorrhage in dogs following envenomation by the eastern brown snake (*Pseudonaja textilis*) in south eastern Queensland, Australia. All cases were presented for veterinary treatment during 2011–2018 at a specialist veterinary emergency centre. Each case received prompt antivenom treatment and supportive care. Pulmonary haemorrhage was diagnosed based on clinical examination; overt haemoptysis; thoracic radiographic demonstration of a diffuse alveolar pattern; and, the presence of venom induced consumptive coagulopathy. The median elapsed time from hospital admission to onset of haemoptysis was 2 h (range 0–18 h). In 80% (8/10) of cases endotracheal intubation was required, whilst 20% (2/10) were successfully treated with mask oxygen supplementation alone, and 40% (4/10) received mechanical ventilation; but only 25% (1/4) of these survived to hospital discharge. Fresh frozen canine plasma was administered to 70% (7/10) of cases and 43% (3/7) of these survived. Of the total number of cases presented for treatment, 30% (3/10) survived to hospital discharge, 60% (6/10) were euthanised due to poor prognosis and 10% (1/10) died from cardiac arrest. Initial serum brown snake venom antigen levels were retrospectively measured from frozen serum samples by venom specific sandwich ELISA in two dogs at 154 ng/mL (survived) and 3607 ng/mL (euthanised); no free venom was detected post-antivenom. Dogs that survived were discharged from hospital without apparent complications. Pulmonary haemorrhage is an uncommon event following envenomation by *P. textilis* in dogs and has not been described in similarly envenomed humans. This case series highlights the potential for fulminant and fatal pulmonary haemorrhage in dogs following eastern brown snake envenomation.

1. Introduction

The Australian eastern brown snake (*Pseudonaja textilis*) is a frequent cause of potentially fatal snakebite in dogs and cats in eastern Australia (Padula and Leister, 2017). Envenomed dogs commonly present with clinical signs relating to neurotoxicity such as lower motor neuron dysfunction and potentially fatal respiratory paralysis. Other physiological disturbances such as prolonged blood clotting time, and infrequently intravascular haemolysis and haemoglobinuria also occur. Neurotoxicity is arguably the most significant clinical sign in dogs following *P. textilis* envenomation, requiring antivenom, supportive care and mechanical ventilation for survival (Padula et al., 2016). However, some envenomed cases present with minimal lower motor neuron signs and subsequently experience haemoptysis, progressing to fulminant,

catastrophic, and fatal pulmonary haemorrhage. The syndrome of pulmonary haemorrhage is poorly described in *P. textilis* envenomed dogs and has not been reported in similarly envenomed humans despite much knowledge of the toxicity of the venom and its components (Padula and Leister, 2017).

Despite coagulation parameters being prolonged pulmonary haemorrhage was not reported in a case series of 149 definite brown snake envenomed humans (Allen et al., 2012). Major haemorrhage was documented in five cases though, with three gastrointestinal bleeds and two intracranial bleeds associated with hypertension (Allen et al., 2012). Intracranial haemorrhage (ICH) in humans was reported from retrospective analysis of hospital records with 2% (5/248) brown snake cases resulting in ICH (Berling et al., 2015). There are only limited reports available in dogs of clinically significant haemorrhage following

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Table 1
Summary of clinical cases with pulmonary haemorrhage. FFP = Fresh frozen plasma. OMM = Oral mucous membranes.

Case No.	Signalment	Presenting Clinical Signs	Initial ACT	Onset Time ^a	Treatment	Outcome
1	Two-year-old 34 kg desexed female Maremma Sheepdog	Skin bleeding from bite sites	No clot	On Presentation	FFP Mask Oxygen 2 vials of Antivenom C	Survived
2	Five-year-old 51 kg desexed female Great Dane	Hypoxaemia	No clot	3.5 h	FFP Mask Oxygen 2 vials of Antivenom A	Survived
3	Three-year-old 30 kg desexed female German Short Haired Pointer	Skin bleeding from bite sites Soft cough Otic/bite haemorrhage Bradycardia Shock	No clot	On Presentation	FFP Intubation 3 vials of Antivenom A	Euthanised
4	Two-year-old 20 kg desexed male Staffordshire Bull Terrier	Bleeding laceration on ventral eyelid	No clot	3 h	Intubation CPR 1 vial of antivenom B	Died from cardiac arrest
5	Seven-year-old 20 kg desexed female Border Collie	Tetraparesis Obtundation Pyralism Mild respiratory effort	No clot	2 h	FFP Mech Vent 2 vials of Antivenom C	Survived
6	Two-year-old 56 kg desexed female Great Dane	Haemoptysis	No clot	On Presentation	FFP Intubated 2 vials of Antivenom C	Euthanised
7	15-month-old 30 kg desexed female Labrador	Pale mucous membranes Pyralism	No clot	2 h	Intubated 1 vial of Antivenom C	Euthanised
8	Four-year-old 21 kg non-desexed female American Staffordshire Terrier	Bleeding neck bite sites Mild weakness Mucoid diarrhoea	No clot	2 h	FFP pRBC Intubated Mech Vent 5 vials of Antivenom C	Euthanised
9	Five-year-old 5.5 kg desexed female Miniature Fox Terrier	Bleeding bite sites on thorax	No clot	18 h	FFP Mask OxygenMech Vent 5 vials of Antivenom C	Euthanised
10	Two-year-old 46 kg desexed male Rhodesian Ridgeback cross	Haemoptysis Collapse Respiratory distress Shock	No clot	On presentation	Mask Oxygen Mech Vent 1 vial of Antivenom C	Euthanised

^a Time from hospital admission to onset of first respiratory distress and/or haemoptysis.

brown snake envenomation. An extradural haematoma was diagnosed and successfully treated by decompressive spinal surgery in a dog following brown snake (*Pseudonaja* sp.) envenomation in Western Australia (Ong et al., 2009). The physiological response of dogs to *P. textilis* venom shares both similarities and differences with human envenomation. Dogs frequently develop neurotoxicity (Padula and Leister, 2017), whilst this is rare in humans; however, both dogs and humans develop venom induced consumptive coagulopathy (VICC) (Padula and Leister, 2017). VICC is characterized by a coagulopathy in a patient after envenomation where there are low or undetectable fibrinogen levels which results from the activation of clotting pathway by procoagulant toxins in venom (Isbister et al., 2010). Other coagulation abnormalities manifest as a prolongation of prothrombin time (PT) and/or activated partial thromboplastin time (aPTT) as well as activated clotting time (ACT) (Padula and Leister, 2017). In these patients, VICC is a result of the activation of the coagulation pathway by the prothrombin activator and *P. textilis* contain group C prothrombin activators (Isbister et al., 2010).

The following report retrospectively describes the clinical signs, treatment and outcomes of ten cases (see Table 1) of severe acute pulmonary haemorrhage in dogs in south eastern Queensland following brown snake envenomation. This is the first report of a series of pulmonary haemorrhage cases in dogs following *P. textilis* envenomation and confirms the potential for VICC to result in fatal complications in dogs.

2. Case reports

2.1. Case 1

A two-year-old 34 kg desexed female Maremma Sheepdog presented to the veterinary hospital after the owner found the dog playing with a brown snake in its yard 6 h earlier. At initial examination, the dog had subcutaneous haemorrhage from a presumed bite site on its left ear, was tetraparetic, exhibited haemoptysis and was hypoxaemic, saturating at 93% (normal 96–100) despite oxygen flow by. A blood sample was collected from a peripheral vein and an activated clotting time test performed (ACT) no clot formed. The packed cell volume (PCV) and total serum protein (TP) levels were 42% and 60 g/L respectively. Two vials of antivenom C were administered by slow intravenous infusion. Three hours post-admission the PCV, TP and ACT were 32%, 56 g/L and no clot respectively. Two units of canine fresh frozen plasma (FFP) was administered due to the concern for an ongoing pulmonary haemorrhage as demonstrated in the dog's drop in PCV and TP. After the transfusion the prothrombin time (PT) and activated partial thromboplastin time (aPTT) (Coag Dx™ Analyser, IDEXX Laboratories, Rydalmere, Australia) were measured at 16 s (normal 11–17 s) and 133 s (normal 72–102 s) respectively. At 22 h post-admission, the dog's respiration further deteriorated. Its PCV and TP were 24% and 58 g/L respectively. Thoracic radiographs were taken which revealed a marked alveolar pattern in the cranial and caudal lung fields. It was then placed on mask oxygen supplementation as well as butorphanol at 0.2 mg/kg/hr (Butorgesic, Ilium Veterinary Products, Australia) intravenously as an anxiolytic, which improved its clinical status. A further one unit of canine FFP was infused to improve circulating clotting factor levels and control bleeding. The dog's thoracic radiographs were repeated 60 h post-admission and showed a marked resolution of alveolar infiltrates. It was subsequently successfully weaned off mask oxygen support at 65 h post-admission and the dog started ambulating and eating the same night. The dog was discharged from the hospital 80 h after admission and appeared fully recovered (see Fig. 1).

2.2. Case 2

A five-year-old 51 kg desexed female Great Dane dog presented to the veterinary hospital in the evening shortly after the owner found a

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