THE UNIVERSITY OF SYDNEY

Centre for Veterinary Education

Est. 1965

Critical Care in Internal Medicine & Surgery Conference

Proceedings 460

Critical Care in Internal Medicine & Surgery Conference

Notes

Proceedings No. 460

Published by:

Veterinary Science Conference Centre (VSCC)
Regimental Drive, The University of Sydney
NSW 2006
+61 2 9351 7979
cve.edu.au

Chapter 22

Less common snakebites and those of uncertain medical significance

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Introduction

Previous surveys of veterinarians in Australia have found that approximately 90% of all snakebites in animals are caused by either brown snakes (*Pseudonaja sp*), tiger snakes (*Notechis sp*), or red-bellied black snakes (*Pseudechis porphyriacus*) (Heller et al., 2005; Mirtschin et al., 1998). However, a number of other snake species are capable of inflicting significant morbidity and mortality upon dogs and cats. Incremental knowledge gains have occurred from published animal case reports, laboratory studies of milked venom and experience of veterinarians; not just reliance upon human reports. This section describes multiple snake species that can cause significant envenomation of domestic animals but are less commonly treated by veterinarians.

Death Adders

The death adders (*Acanthophis sp.*) are found in all states except Vic or Tas. There are multiple species of death adder present in Australia, found in geographically distinct regions including unique offshore island populations on Magnetic Island (Qld), Fraser Island (Qld), North Stradbroke Island (Qld) and Reevesby Island (SA). Death adders are also found in and around the Sydney area. A mailout survey of veterinarians in NSW found that 0.2% of all snakebite cases treated were death adder and 3.2% of clinics reported having treated cases (Heller et al., 2005). There is very limited published information on the envenomation syndrome in domestic animals although a series of four cases of envenomation responsive to death adder antivenom in WA and NSW is documented (Swindells et al., 2006). The death adder

is a copious venom producer. The venom contains primarily neurotoxins and has been well studied *in vivo* and *in vitro*. Recent *in vitro* studies have demonstrated the venom contains both pre-synaptic neurotoxins as well as the previously identified post-synaptic neurotoxins (Blacklow et al., 2010). The venom also has weak anticoagulant properties. Clinically, paralytic signs predominate, and limited evidence suggests may frequently be severe enough to require ventilatory support in animals. Local bite site swelling may occur, and antibiotics indicated. Specific diagnosis is possible using either the Snake Venom Detection Kit (SVDK) with distinctive reaction in the death adder well, or positive snake identification. The response to antivenom has been reported as variable in human cases. Previous studies in humans with death adder envenomation in the 1960s in PNG found a rapid reversal of paralysis following antivenom (Campbell, 1966). Recent studies in humans bitten in Australia have revealed continuing paralysis despite early antivenom, providing further evidence of irreversible pre-synaptic neurotoxins (Johnston et al., 2012). Specific death adder antivenom is indicated but supportive care including mechanical ventilation may also be required. Anticholinesterase compounds (eg neostigmine) have been advocated in the past although their effectiveness was of limited value in recent human cases (Johnston et al., 2012).

Copperheads

The copperhead snakes (*Austrelaps sp.*) are found in the cooler climates of SA, Vic, Tas and the highlands of NSW. Despite the common occurrence of this snake there is only one detailed case report of confirmed envenomation, although anecdotally veterinary practitioners in regions where copperheads are found describe treating occasional cases. The copperhead can easily be confused with a brown snake however the scales are much larger and typically 15 dorsal scales compared to browns with 17-19. Part of the reason for very few reported envenomation's in animals may be the short fangs and sluggish nature of the creature's strike. The venom has been studied *in vitro* and *in vivo* and is highly lethal to mice with LD50s of 0.3 to 1.3 mg/kg reported (Padula et al., 2018). The venom contains weak anticoagulant properties *in vitro* and exhibited *in vivo* due to phospholipase A2 enzymes named superbins (Subburaju and Kini, 1997). Both pre- and post-synaptic neurotoxins are also present and a direct acting haemolytic toxin. Studies conducted in monkeys injected

with copperhead venom revealed prolonged prothrombin time and markedly elevated serum creatine kinase (CK) at 3 hours (Sutherland et al., 1981). A detailed case report of a dog systemically envenomed that was successfully treated with antivenom, fluid therapy and mechanical ventilation (Wright and Indrawirawan, 2017). Tiger-brown snake antivenom is recommended for copperhead envenomation. Specific diagnosis can be made by careful snake identification (15 dorsal scales), clinicals signs of LMN paralysis and an SVDK reaction similar to tiger snake venom (Cox et al., 1992).

Eastern Small Eyed Snake

The eastern small eyed snake (*Cryptophis nigrescens*) is found all along the eastern seaboard regions of Australia. The snake has an appearance somewhat similar to a juvenile red-bellied black snake (RBBS). Due to its physical similarity to the RBBS it is possible cases could be inadvertently attributed to the RBBS. The snake is not well known, perhaps because of its nocturnal and shy nature. Nocturnal encounters between dogs and cats with this snake are possible and may account for some unexplained protracted illnesses. The action of the venom has been studied in dogs and reveals a profound myopathy and myoglobinuria (Winter and Pollitt, 1978). A recent detailed case report of a confirmed envenomation in a dog has expanded knowledge of the envenomation syndrome (Farquharson et al., 2011). The report describes a gradual onset of clinical signs in an envenomed Labrador over 1-2 days with profound depression, weakness, vomiting, myopathy, masticatory myositis, pigmenturia, elevated serum CK and AST levels. Complete recovery was reported by 14 days (Farquharson et al., 2011). Tiger-brown snake antivenom is recommended for cases diagnosed early in the course of the envenomation. A fatal human envenomation from acute renal complications has occurred in a snake handler (Sutherland and Tibballs, 2001).

White Lipped Snake

The white lipped snake (*Drysdalia coronoides*) is a small snake found in coastal NSW, eastern and southern Vic, southeast SA and Tas. The snake is common around Melbourne preferring forested areas. The white lipped snake is very cold tolerant and has been reported on Mt Kosciusko above the snow line. Typical length is 20-40 cm. The snake is identifiable by the characteristic white stripe above the upper lip.

The small size of the snake and minute venom delivery apparatus suggest that effective injection of venom would be difficult to achieve. Nevertheless, there is some convincing evidence that this snake contains potent toxins and has the capacity to deliver these. When a 36 cm white lipped specimen was allowed to bit a live mouse the mouse died in 27 minutes, and a guinea pig likewise in 2.5 hours (Kellaway, 1934). A human snakebite from this species was documented in 1994 which resulted in facial weakness and ptosis after 18 hours with serum CK of 57,300 and myoglobinuria (Sutherland and Tibballs, 2001). The patient was treated with tiger snake antivenom and recovered fully. A suspected white lipped snake envenomation in a cat in Melbourne presented with pigmenturia, weakness and elevated CK and fully recovered with tiger-brown antivenom and fluid therapy. The chewed-up snake was later positively identified by the museum as *Drysdalia coronoides*. Recent molecular studies of the venom transcriptome revealed the presence of proteins typically found in venomous snake families.

Black Whip Snakes

The black whip snakes (*Demansia sp.*) are found in northern regions of Australia. They are a fast-moving snake, black/brown/light brown in colour and are regarded as dangerous. Although venom yields are small, the venom has been shown to be lethal when injected into laboratory animals (Sutherland and Tibballs, 2001). The first detailed case report of a black whip snake envenomation describes a syndrome of bite site pain, tissue destruction and recovery without antivenom (Fawcett, 2014). The SVDK reaction was negative. Anecdotal reference was made in the report to a second lethally envenomed dog from a black whip snake. The yellow-faced whip snake (*Demansia psammophis*), which is found much further south in Australia, is also capable of inflicting a painful bite and the venom was lethal when injected into a guinea pig that died within 8 hours from respiratory paralysis (Kellaway, 1934).

King Brown Snake

The king brown snake (*Pseudechis australis*), sometimes referred to as the Mulga snake, is a not a brown snake but rather a member of the black snake family. The king brown is a large snake and massive venom producer, with milked specimens typically yielding 180 mg to over

1200 mg (Sutherland and Tibballs, 2001). The king brown could be mistaken for a large brown snake. The king brown is widely distributed across the central regions of Australia and is present in all states, except the south-west corner of WA, Vic and Tas. Despite the widespread distribution there are almost no published accounts of cases in animals, although they do occasionally occur. In a survey of veterinarians in NSW only 1.3 % of all cases were attributed to king brown and 6.7% of clinics reported a case (Heller et al., 2005). The venom is only moderately toxic compared to tiger and brown snakes, but the large quantity is what makes it potentially lethal to humans and animals. The major venom components are a myotoxin, neurotoxin and haemolytic toxins. Widespread muscle damage, mildly prolonged bleeding time and potential paralytic neurotoxicity may occur. In humans, local bite tissue tissue necrosis is commonly seen and when bites occur on extremities finger or even limb amputation is a potential complication. Specific diagnosis can be made from identification of the snake or use of the SVDK with strong reaction in the 'black' snake immunotype well. The recommended antivenom has been tiger (or tiger-brown) but a more specific black snake antivenom of larger volume and made using *P. australis* as the immunising antigen is available.

Spotted (Blue-Bellied) Black Snake

The spotted black snake (*Pseudechis guttatus*), also called the blue-bellied black snake (BBBS) accounted for 0.5% of all animal snakebite cases in NSW with 3.2% of clinics reported treating cases (Heller et al., 2005). The BBBS occurs from the Hunter River District of New South Wales into south-eastern Queensland. More specifically, it is found inland of the coast, except around Queensland's Tuan State Forest, and extends north in Queensland to around the Junee State Forest and south in New South Wales to around Young. The Western limit is to about the longitude that extends through Hebel-Dirranbandi in Queensland (Mirtschin et al., 2017). The belly of the BBBS is blue-black in colour sometimes with light yellow spots and typically 1.2 to 2 m in length. The dorsum may be black with cream spots to a more predominately cream colour with black scales. When upset the BBBS has a characteristic deep resonant hiss and flattens out its body. The venom is similar to the red-bellied black snake (*Pseudechis porphyriacus*) with haemolysins, neurotoxins, and a coagulant. Myotoxic activity is present but *in vitro* studies and two recent human case reports suggest

muscle damage is significantly less likely to occur than other black snakes (Jansen et al., 2007; Ramasamy et al., 2004). Animal cases do occur but there are no detailed published reports in the literature. The recommended antivenom is tiger-brown, similar to RBBS.

Taipan

The coastal taipan (*Oxyuranus scutellatus*) is large, fast moving, highly venomous snake that has caused many human fatalities. Before the introduction of a specific taipan antivenom in 1956 all human patients almost invariably died. The taipan is found in northern Australian regions, typically along the wetter coastal regions of north-west WA, NT and into south-east Qld. The taipan is a copious producer of a highly lethal venom. It is estimated that a large adult taipan contains enough venom to kill 50 adult humans. The venom contains potent pre-synaptic neurotoxins, procoagulant (similar to brown snake), and myotoxins. Specific diagnosis can be made using the SVDK, particularly useful in areas of Australia where both the brown snake and taipan distributions overlap. Only a single case report from a taipan envenomed dog has been published (Judge, 2015) although anecdotal evidence suggests veterinarians in northern Australia (eg Cairns, Townsville) do treat occasional cases. Rapid onset clinical signs and death has been anecdotally reported for envenomed dogs. A survey of veterinarians revealed 2.1% of all snakebites in animals in Qld were attributed to the taipan (Mirtschin et al., 1998). The clinical signs described in a dog envenomed by a juvenile taipan were progressive LMN paralysis, coagulopathy within 30 mins (ACT > 300s), and pigmenturia (SVDK positive for taipan) (Judge, 2015). The dog was administered taipan antivenom (12,000 units), mechanically ventilated for 4 days and was successfully discharged from hospital after 6 days (Judge, 2015). Specific taipan antivenom is required as brown snake antivenom is reported as ineffective clinically (Trinca, 1969). Interestingly, laboratory studies in taipan envenomed mice have demonstrated approximately 20% cross protection against lethality for tiger snake antivenom (Morgan, 1956).

Others

Other potentially dangerous snakes include the Brown Tree Snake (*Boiga irregularis*), False King Brown Snake (*Pailsus pailsei),* De Vis' Banded Snake (*Denisonia devisii*), Ornamental snake (*Denisonia maculata*),

Marsh Snake, Swamp Snake (*Hemiapsis signata*), Common Keelback (*Tropidionphis mairii*), Golden Crown Snake (*Cacophis squamulosus*)and Brown Headed Snake (*Furina tristis*).

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