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Elapid snake envenomation in horses: 52 cases (2006–2016)

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Summary

Background: Snake envenomation is a cause of morbidity and mortality in domestic animals worldwide. The clinical features of crotalid snake (pit viper) envenomation are widely reported and well described in horses but elapid snake envenomation is poorly characterised.
Objectives: To describe the presentation, clinical and laboratory findings, treatment and outcome of horses with a diagnosis of elapid snake envenomation in Australia.
Study design: Retrospective case series.
Methods: Medical records of horses with a diagnosis of elapid snake envenomation (2006–2016) at several university and private veterinary practices were reviewed. Inclusion criteria comprised one or more of the following: (1) observed snakebite, (2) positive snake venom detection kit (SVDK) result, (3) appropriate clinical response to treatment with antivenom or (4) supportive post mortem findings.
Results: Fifty-two cases met the inclusion criteria. Most cases (94%) demonstrated clinical signs of neurotoxicity, characterised by generalised neuromuscular weakness. Associated neurologic signs included staggering gait, muscle fasciculations, recumbency, mydriasis, ptosis and tongue paresis. Concurrent clinically important conditions included rhabdomyolysis (50%) and haemolysis (19%). Of 18 urine samples evaluated with a SVDK, only three (17%) were positive. Overall survival was favourable (86%) among 49 horses that received antivenom. Eighteen surviving horses (43%) required more than one vial of antivenom.
Main limitations: Possible cases within the searchable database were not included if horses died acutely or responded to symptomatic treatment without receiving antivenom.
Conclusions: Elapid snake envenomation is primarily a syndrome of neuromuscular weakness. Supportive anamnesis or an obvious bite site are rarely encountered. In endemic areas, this diagnosis should be considered for horses with generalised neuromuscular weakness, altered mentation, rhabdomyolysis and/or haemolysis; especially during spring and summer months. Diagnostic suspicion is best confirmed by response to treatment with antivenom.

Introduction

Elapid snakes comprise many of the world's deadliest snake species, including cobras (*Naja* spp.) and mambas (*Dendroaspis* spp.), and are defined by their proteroglyphous (rostral grooved) fangs and long slender bodies [1-3]. This group is the predominant family in Australia where tiger snakes (*Notechis* spp.), brown snakes (*Pseudonaja* spp.) and black

snakes (*Pseudechis* spp.) are most commonly implicated in domestic animal envenomation [4,5]. Elapid venoms contain potent neurotoxins, cytotoxins and procoagulants that are responsible for the constellation of clinical signs documented in humans, dogs and cats [6,7]. Variations in venom composition between elapid snake species, the amount of venom injected and location of the bite will influence individual clinical presentations of envenomed animals [8]. Common clinical features include generalised neuromuscular weakness (progressive flaccid paralysis, ptosis, mydriasis and respiratory failure), rhabdomyolysis, haemolysis, acute kidney injury and venom-induced consumptive coagulopathy [9].

Many studies of snakebite in the horse originate from North America and describe envenomation by members of the crotalid sub-family (pit vipers, including rattlesnakes). Consistent with reports in other species [10], important clinical features described in horses bitten by rattlesnakes include marked local tissue swelling and necrosis, myocardial injury and cardiac arrhythmias, haemolytic anaemia and coagulopathy [11-14]. Although bites from elapid snakes are often speculated as a cause of sudden illness and unexpected death in endemic areas [15], there is a paucity of literature describing elapid snake envenomation in horses. Current reports are limited to single case descriptions [15,16] or small case series [17,18] in which evidence for envenomation was sometimes circumstantial. Diagnostic confirmation is often difficult and recommendations are either based on unquestioned anecdotal evidence, or extrapolated from clinical observations in dogs and cats or reports of crotalid envenomation. There are, however, important differences in the composition of crotalid and elapid venoms that should preclude direct comparisons of clinical data [19].

An improved understanding and awareness of the clinical syndromes associated with elapid snake envenomation in horses will aid veterinarians in making a prompt diagnosis, allowing the institution of appropriate and early treatment in these cases. The purpose of this multicentre study was to characterise the presentation, clinical and laboratory findings, treatment and outcome of horses with a diagnosis of elapid snake envenomation in Australia.

Materials and methods

Case records of horses with a diagnosis of elapid snake envenomation (2006 to 2016) were retrieved from the medical record databases of the University of Melbourne, Scone Equine Hospital, Murdoch University, the University of Queensland and the University of Adelaide. Private mixed-animal and equine-only veterinary practices in Victoria were contacted by

email or telephone seeking additional cases. Inclusion criteria comprised one or more of the following: (1) observed snakebite prior to the development of clinical signs, (2) positive snake venom detection kit (SVDK)[a] result, (3) appropriate clinical response to treatment with antivenom or (4) supportive post mortem findings.

Historical data collected included horse breed, age, and sex and owner observations (primary presenting complaint, whether a snakebite was witnessed, time elapsed since the horse was last seen to be clinically normal). The date of presentation and location of the horse were used to determine the maximum daily ambient temperature on the day of envenomation (Australian Bureau of Meteorology; www.bom.gov.au).

Clinical examination data at presentation comprised continuous variables including rectal temperature, heart rate and respiratory rate, and categorical variables including the presence or absence of pyrexia (rectal temperature >38.3°C), hypothermia (rectal temperature <37.0°C), tachycardia (heart rate >40 beats/min), tachypnoea (respiratory rate >20 breaths/min), dyspnoea, generalised neuromuscular weakness, muscle fasciculations, sweating, mydriasis, pupillary light response, colic signs, dysphagia, tongue paresis and discoloured urine. If present, signs of apparent abdominal pain (colic) were graded as mild, moderate or severe. Mentation was classified as normal, dull/obtunded or agitated/hyperresponsive. Any period of recumbency was classified as intermittent (voluntarily alternating between standing and recumbent positions during evaluation) or progressive (standing initially but progressing to sustained recumbency prior to the commencement of treatment). The suspected bite site, if apparent, was described by location and clinical appearance.

Clinical pathology data available for assessment from presentation or hospital admission varied, but typically included haematology, serum/plasma biochemistry analyses and venous blood gas analyses. Cardiac troponin I (cTnI) concentration and coagulation screening assay results were available for some cases. Categorical variables used to describe clinical pathology data comprised the presence or absence of haemolysis, leucocytosis, hyperfibrinogenaemia, hyperlactataemia, azotaemia, and evidence of rhabdomyolysis, myocardial injury and/or coagulopathy. Rhabdomyolysis was defined as plasma creatine kinase (CK) activity of >3,000 U/L to reduce the likelihood of recumbency contributing to mild increases above the reference interval. If a SVDK was used, the type of biological

sample (bite site swab, urine, plasma, whole blood) was recorded, and historical information was used to estimate the elapsed time between suspected envenomation and when the assay was performed. The venom immunotype of positive SVDK results were reported.

Data analysis

Descriptive analysis reported median (range) values for continuous data and proportions (percentage) for categorical data. When calculating percentages for incomplete data sets, the denominator was defined as the number of horses with data available for each variable.

Results

Animals

Fifty-two horses met the inclusion criteria. Cases were identified from the records of the University of Melbourne (16), Scone Equine Hospital (9), the University of Adelaide (2), Murdoch University (1), the University of Queensland (1) and private veterinary practices in Victoria, Australia. (23). Breeds included Thoroughbred (27), Standardbred (6), Quarter Horse (5), Warmblood (3), Arabian (2), Draught breed (2), Shetland Pony (2), Australian Riding Pony (2), Miniature Pony (2) and Australian Stock Horse (1). The median age was 7 years (range, 4 days to 23 years). Animals aged ≥1 year included 23 geldings, 18 mares and two stallions; animals aged <1 year included three colts and six fillies.

Historical information

Owner reported primary presenting complaints are shown in Table 1. Two horses were observed to have been bitten by a snake prior to the development of clinical signs. The median time elapsed since each horse was last seen to be clinically normal was 8 h (range, 30 min to 120 h); the median time from recognition of clinical signs until examination by a veterinarian was 1 h (range, 30 min to 96 h). The month in which each case presented is shown in Figure 1. The median maximum ambient temperature on the day of suspected envenomation was 29.8°C (range, 24.4–41.2°C).

Clinical and laboratory findings

The clinical and laboratory findings reported on initial examination or admission to hospital are shown in Tables 2 and 3. Four horses (8%) were reported to demonstrate normal mentation, 30 horses (58%) were dull/obtunded, 17 horses (33%) were

agitated/hyperresponsive and one foal (2-month-old colt) presented in a comatose state. Thirteen horses (25%) remained standing throughout evaluation, 20 horses (38%) were intermittently recumbent and 18 horses (35%) were progressively recumbent; the comatose foal remained recumbent throughout evaluation. Dyspnoea was severe in three horses, while all cases with colic were graded as mild. A suspected bite site was identified in 14 cases (27%), with the location reported as the muzzle in 10 cases, jaw in one case and distal limb in three cases. Suspected bite sites were characterised by mild local swelling, erythema or wheal formation in all cases; fresh blood or a speculated pair of fang marks were occasionally observed in the centre of a lesion. Four horses (8%) presented with muzzle deviation due to unilateral facial nerve paralysis that was attributed to localised neurapraxia from an ipsilateral bite on the muzzle. One horse presented with generalised urticaria.

Activated clotting time was determined in four cases, three of which demonstrated prolonged clotting times. Prothrombin time and activated partial thromboplastin time were quantified in an additional four cases, all of which yielded normal results. The cTnI concentration was measured and markedly increased in four horses (Table 2), but no arrhythmias were detected with electrocardiography. For horses with rhabdomyolysis, median CK activity was 20,570 U/L (range, 4,996–356,960 U/L).

Eighteen cases underwent diagnostic evaluation using the SVDK. A urine sample was tested in all cases; 15 cases (83%) returned a negative result and three cases (17%) returned a positive result. One horse initially tested negative on a plasma sample, but subsequently tested positive when the assay was repeated using a urine sample within 1 h. Two positive results indicated the tiger snake immunotype and one positive result indicated the brown snake immunotype. The median time from suspected envenomation to performance of SVDK was 12 h (range, 8 to 36 h) for negative results and 12 h (range, 6 to 24 h) for positive results.

Treatment

Forty-nine horses were treated with at least one vial of polyvalent elapid snake antivenom (minimum 3000 IU tiger snake antivenom and 4000 IU brown snake antivenom per vial). All of these horses showed noticeable improvement in neuromuscular strength and/or mentation between 10 and 240 min after treatment commencement (median, 50 min). Thirty-one horses (63%) received one vial of antivenom, 11 horses (22%) received two vials, two horses (4%) received three vials, four horses (8%) received four vials and one horse (2%) received five

vials. For horses that were given more than one vial of antivenom, subsequent vials were administered when clinical deterioration (most commonly progressive neuromuscular weakness) occurred over varying periods of time. The most common treatment regimen was to administer one vial of antivenom diluted in 1 litre of an isotonic crystalloid solution (0.9% sodium chloride or Hartmann's solution) over 15 to 30 min; one horse received undiluted antivenom as a syringe bolus due to fractious demeanour. Three horses were not treated with antivenom, but met the inclusion criteria on the basis of supportive post mortem examination findings.

Premedications were administered in 44 of 49 cases (90%) that received antivenom. The type of premedication included: dexamethasone and chlorpheniramine (16), dexamethasone only (11), chlorpheniramine only (10), flunixin meglumine only (6), and flunixin meglumine and chlorpheniramine (1). Twenty-nine horses (59%) were administered antimicrobials for varying periods of time, including: procaine penicillin (11), procaine penicillin and gentamicin (6), trimethoprim/sulfadimidine (5), ceftiofur (3), procaine penicillin and enrofloxacin (2), trimethoprim/sulfadimidine and rifampicin (1) and ceftriaxone (1). Twenty-seven horses (55%) received non-steroidal anti-inflammatory drugs for varying periods of time, including: flunixin meglumine (16), phenylbutazone (10) and meloxicam (1). Other therapies that were administered comprised intravenous or enteral fluid therapy of varying regimens in 40 horses (82%), parenteral or enteral nutritional support in six inappetent horses (12%) and supplemental oxygen therapy in four dyspnoeic horses (8%).

Outcome

Forty-two of 49 horses (86%) treated with antivenom survived to discharge from hospital or the conclusion of on-farm veterinary management. Eight of nine (89%) foals survived. The median duration of hospitalisation or on-farm veterinary management was 3 days (range, 1 to 14 days). Seven horses required hospitalisation for >7 days; the reasons for prolonged hospitalisation included: severe rhabdomyolysis associated with acute kidney injury (2), severe rhabdomyolysis not associated with acute kidney injury (2), further monitoring at the owner's request (2) and prolonged anorexia requiring nutritional support (1). Three of four horses with facial nerve paralysis survived, with muzzle deviation reported to have resolved at follow-up times of 2, 5 and 10 months, respectively. One mare was 30 days pregnant at the time of envenomation and survived to deliver a healthy full-term foal.

Ten non-surviving horses were subjected to euthanasia on financial grounds; three of which did not receive antivenom. The remaining seven horses initially responded positively to the administration of antivenom but did not receive further treatment after clinical deterioration. Reported post mortem findings in five horses included multifocal endothelial injury leading to haemorrhage from small vessels in multiple organs and tissues, microvascular thrombosis, acute renal tubular necrosis and generalised hyaline degeneration of cardiac and skeletal muscle. These gross and histopathological findings were considered supportive of a diagnosis of elapid snake envenomation [20].

Discussion

Elapid snake envenomation in horses can present a diagnostic and therapeutic challenge for veterinarians. Common clinical features included tachycardia, generalised neuromuscular weakness, altered mentation and rhabdomyolysis. Although haemolysis was demonstrated in a small number of cases, venom-induced consumptive coagulopathy was not a major manifestation of envenomation in this population of horses. Findings from the current report illustrate important differences between crotalid and elapid snake envenomation in horses, particularly with regard to the clinical manifestations of disease.

Consistent with reports from humans, dogs and cats [5-7], neurotoxicity was the principle manifestation of disease for the majority of horses in this series. Progressive generalised neuromuscular weakness was characterised by staggering gait, muscle fasciculations, recumbency, mydriasis with delayed/absent pupillary light response, ptosis, dyspnoea, dysphagia and/or tongue paresis. Elapid venom contains a cocktail of potent neurotoxins that act at the neuromuscular junction to disrupt nerve function and thus incapacitate intended prey [21]. Neurotoxicity is therefore a key feature of elapid snake envenomation. Examples of elapid neurotoxins include pre-synaptic phospholipase A_2 toxins, e.g. notexin (tiger snakes), textilotoxin (brown snakes) and pseudexin (black snakes), and post-synaptic α-neurotoxins, e.g. notechis III (tiger snakes) and pseudonajatoxin-b (brown snakes) [22].

Myotoxicity was detected in half of the horses studied, and appears to be a relatively common feature of envenomation by elapid snake species. In addition to previously noted neurotoxic effects, some phospholipase A_2 toxins possess myolytic activity [22,23]. Myotoxicity is a reliable feature of tiger snake envenomation in humans, dogs and cats, with creatine kinase activity often used to aid in diagnostic confirmation [7,20,24,25]. Generalised acute and

hyaline degeneration of skeletal muscle is a consistent post mortem finding in dogs and cats [26,27]. However, there are notable differences in myolytic activity between the venoms of elapid snake species. Black snakes possess only a weak myolysin [28] and brown snake venom does not possess any myolytic activity [29]. Secondary nephrotoxic effects of severe rhabdomyolysis resulted in prolonged hospitalisation in two horses.

Cardiotoxicity has been described in horses with crotalid envenomation [14] and degeneration of cardiac muscle has been reported at post mortem examination in dogs and cats with tiger snake envenomation [26,27]. Clinicopathologic evidence of myocardial injury was identified in all four cases in which serum cTnI concentrations were measured, although symptomatic cardiac disease was not appreciated in these horses. It remains unclear how commonly myocardial injury occurs in animals following elapid snake envenomation [15]. Whether cardiotoxicity is a clinically important aspect of elapid envenomation in horses requires further investigation.

Venom-induced consumptive coagulopathy is present in a very high proportion of humans bitten by elapid snakes and, to a lesser extent, envenomed dogs and cats [30,31]. Eight horses in the present study had coagulation testing performed, with three demonstrating prolonged clotting times; however, clinical evidence of a haemorrhagic diathesis was not detected in any case. Haemolysis was present in a small number of horses included in this study. Haemolytic cytotoxicity is due to cytotoxic actions of certain phospholipase A_2 toxins and is a noted feature of black snake envenomation, but occurs to a lesser extent with tiger snake envenomation [32]. Comparisons between species are obviously difficult [33], and further studies are required to elucidate whether coagulopathy and haemolysis are clinically important features of disease in horses, especially in cases that die acutely following envenomation.

Depending on the constellation of clinical signs present, differential diagnoses for elapid snake envenomation in Australian horses could include: viral encephalitides; tick (*Ixodes holocyclus*) paralysis; botulism; tetanus; plant toxicoses such as Darling pea (*Swainsona greyana*), dune onion weed (*Trachyandra divaricate*) or bracken fern (*Pteridium* spp.); metabolic disturbances; myopathies; and neurological trauma. Tick paralysis is an important differential for horses exhibiting neuromuscular weakness in high rainfall areas along the east coast of Australia; although a tick infestation is usually obvious and adult (larger) horses are

uncommonly affected [34]. The presence of tachycardia, tachypnoea and pyrexia in horses with neuromuscular weakness, altered mentation or dyspnoea warrants particular mention given the overlap with clinical signs of Hendra virus infection [35]. Three horses in the present study were subject to Hendra virus exclusion testing.

The majority of horses in this study were evaluated without definitive anamnesis, as a witnessed snakebite occurred in only two cases prior to the onset of clinical signs. The identification of a suspected bite site was considered helpful to the diagnostic process, but was present in only 27% of cases. Most suspected bite sites occurred on the muzzle and, importantly, swelling was often subtle. This finding is in stark contrast with the clinical manifestation of crotalid bites, where marked swelling and tissue necrosis often necessitate an emergency tracheotomy to maintain airway patency in horses bitten around the head [12,13]. Another key finding of this series is that the absence of an obvious bite site should not exclude the possibility of elapid snake envenomation in the horse, as has been noted for small animal species [7,20].

The detection of venom using a commercially available multivalent SVDK can be useful to confirm a diagnosis or to aid in selecting an appropriate monovalent antivenom to use (if available). In human studies, a bite site swab is considered to provide the most valuable result [36], but was not performed for any horse in the present study. *In vitro* studies have validated the SVDK for equine urine and plasma samples [37], although test performance has not been widely evaluated in clinical cases. Low test sensitivity (17%) for detecting venom in equine urine was demonstrated in the present study. False-negative results, also reported in human studies [25], suggest that venom was either below the limit of detection or not present in urine at the time of collection. A study of cats confirmed the detection of venom in urine for up to 24 h post-envenomation [38], but information regarding the kinetics of elapid venom excretion in equids is not available. Due to the small number of horses that tested positive, statistical evaluation for the poor sensitivity was not possible; however, the time post envenomation at which the SVDK was performed was similar between positive and negative groups. It is important to recognise that a negative SVDK result does not rule out envenomation, nor should it preclude treatment with antivenom in suspected cases.

All horses that were treated with elapid snake antivenom received a polyvalent product due to its common availability. No adverse effects to the administration of antivenom were noted.

Treatment with antivenom is standard practice in human cases of elapid snake envenomation [6]; however, the number of vials of antivenom to be administered has been a subject of debate [25,39]. In people, there is a documented risk of acute and delayed hypersensitivity reactions following antivenom treatment [40], and evidence that a single vial of antivenom can bind all circulating venom in most cases [6,29,41]. Antivenom is reported to not only neutralise circulating venom before it binds to nerve terminals, but may also facilitate the dissociation of toxin from the acetylcholine receptor at post synaptic sites and accelerate recovery from neuromuscular blockade [21]. The titration of multiple vials of antivenom to effect is not uncommon in small animal veterinary practice. Although most horses received only a single vial, over one-third of survivors received multiple vials of antivenom due to recurrent generalised neuromuscular weakness. The cost of antivenom can be substantial, but it is the authors' opinion that multiple vials of antivenom should be considered in horses with significant neuromuscular weakness, especially as recumbency should be avoided in large animals where possible.

The use of prophylactic antibiotics for bite site infections was common in this study, but has been suggested to be unnecessary due to the low incidence of secondary bacterial infections observed in studies of crotalid bites [10,13]. The routine use of corticosteroids or antihistamines as a premedicant to the administration of antivenom has also been questioned and is no longer recommended in human medicine [6]. Administration of antibiotics, corticosteroids and antihistamines are therefore unlikely to be necessary in the majority of elapid envenomations. Described supportive treatments including fluid, nutritional and oxygen therapies are an essential adjunct to the administration of antivenom in critical cases, and should be tailored to the individual animal.

The survival rate of horses that received antivenom was favourable (86%), although caution should be used when applying these results to a wider population due to an inherent degree of selection bias. It should be noted that most horses met the inclusion criteria on the basis of their response to treatment with antivenom, which although strongly supportive, may not be sufficiently robust to exclude every differential diagnosis. Horses within the searchable database that died acutely or were euthanased without a diagnosis, and horses with mild clinical signs that recovered without receiving antivenom, were not included. The difficulty in confirming a diagnosis in horses that did not receive antivenom precluded a useful

statistical analysis of factors influencing survival. Another limitation was an inability to separate descriptions of envenomation by different elapid snake species.

The current study provides the most comprehensive overview of elapid snake envenomation in horses to date. The relevance of these findings resides in the characterisation of naturally occurring clinical cases in which treatment with elapid snake antivenom contributed to a successful outcome.

Authors' declaration of interests

No competing interests have been declared.

Ethical animal research

Research ethics committee oversight not required by this journal: retrospective study of clinical records. Explicit owner informed consent for inclusion of animals in this study was not stated.

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Authorship

N.J. Bamford, S.B. Sprinkle and B.S. Tennent-Brown conceived and executed the study, analysed the data and drafted the manuscript. L.A. Cudmore, A.M. Cullimore, A.W. van Eps and E.J.M.M. Verdegaal contributed to study execution and revised the manuscript. All authors approved the final manuscript.

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401 **Tables**

402 **Table 1:** Owner reported primary presenting complaint for 52 horses diagnosed with elapid
403 snake envenomation.

Primary presenting complaint	Number (%)
Weakness/unsteady gait*	33 (63)
Dull/inappetent*	10 (19)
Recumbency/reluctance to stand	4 (8)
Facial swelling	2 (4)
Stiff gait	1 (2)
Colic	1 (2)
Agitated mentation	1 (2)

404

405 *One horse in each of these groups was observed to have been bitten by a snake prior to the
406 development of clinical signs.

407 **Table 2:** Continuous data for clinical examination and clinical pathology variables recorded
408 on admission in 52 horses diagnosed with elapid snake envenomation.

	Data available (n)	Median (range)	Reference interval
Clinical examination (adults)			
Rectal temperature (°C)	41	38.5 (34.4–41.5)	37.0–38.3
Heart rate (beats/min)	42	62 (32–120)	20–40
Respiratory rate (breaths/min)	41	28 (12–80)	10–20
Clinical examination (foals)			
Rectal temperature (°C)	7	38.8 (36.6–41.0)	37.2–38.9
Heart rate (beats/min)	8	120 (60–140)	60–80
Respiratory rate (breaths/min)	8	32 (24–120)	20–30
Clinical pathology			
Packed cell volume (%)	38	34 (17–78)	25–45
Total solids (g/L)	28	68 (36–100)	58–76
White blood cell count ($x10^9$/L)	37	10.7 (5.1–24.3)	6.0–12.0
Lactate (mmol/L)	25	3.6 (0.5–24)	<1.5
Urea (mmol/L)	34	7.5 (0.9–47.1)	3.6–8.9
Creatinine (mmol/L)	37	0.13 (0.06–0.77)	0.08–0.15
Creatine kinase (U/L)	40	2,870 (67–356,960)	50–400
Aspartate aminotransferase (U/L)	31	689 (202–12,400)	150–400
Cardiac troponin I (μg/L)	4	0.80 (0.41–2.22)	≤0.03

409 **Table 3:** Dichotomous data for clinical examination and clinical pathology variables recorded
410 on admission in 52 horses diagnosed with elapid snake envenomation.

	Data available (n)	Present (n [%])
Clinical examination		
Tachycardia	50	48 (96)
Neuromuscular weakness	52	49 (94)
Altered mentation	52	48 (92)
Muscle fasciculations	52	44 (85)
Recumbency	52	39 (75)
Absent/reduced PLR	40	30 (75)
Mydriasis	47	34 (72)
Tachypnoea	49	33 (67)
Sweating	48	26 (54)
Pyrexia	48	23 (48)
Tongue paresis	30	13 (43)
Dyspnoea	50	20 (40)
Dysphagia	15	6 (40)
Pigmenturia	49	15 (31)
Colic signs	52	11 (21)
Hypothermia	48	5 (10)
Clinical pathology		
Hyperlactataemia	25	20 (80)
Leucocytosis	37	22 (59)
Rhabdomyolysis*	40	20 (50)
Hyperfibrinogenaemia	25	6 (24)
Azotaemia	37	9 (24)
Haemolysis	43	8 (19)

411

412 *Defined as plasma creatine kinase >3000 U/L. PLR, pupillary light response.

413 **Figure legends**

414 **Fig 1:** Month in which 52 horses with a diagnosis of elapid snake envenomation were

415 presented to veterinarians in Australia. No cases presented between May and August,

416 inclusive.

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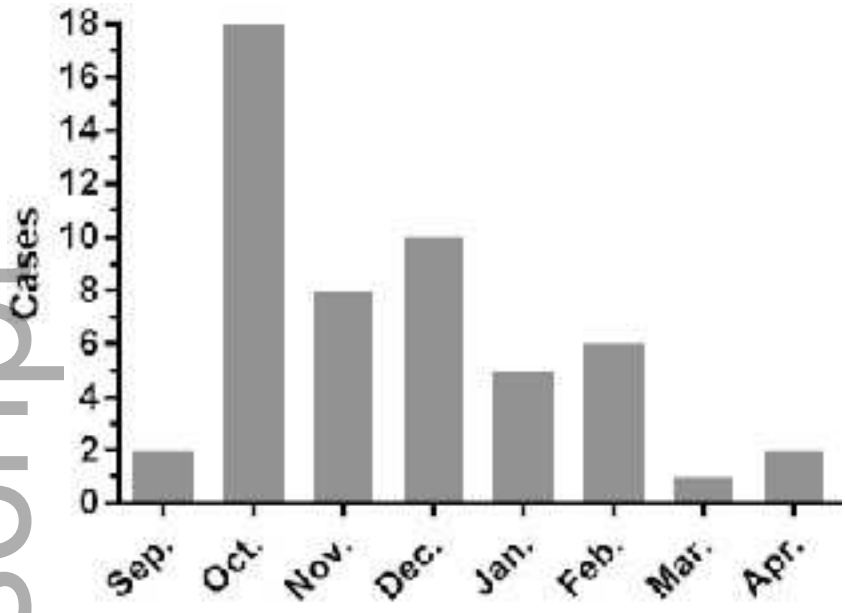

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