Summer poisoning hazards

There is a seasonal risk with some poisons and in the summer, when stinging insects and adders are more active, there is a risk of envenomation. Diagnosis of an adder bite may be missed in horses, as the biting event is rarely witnessed. Envenomation may result in local swelling and occasionally, systemic effects. Multiple bee stings are particularly hazardous as this can result in multi-organ involvement, which may be fatal. Grazing on some plants, such as ragwort or bracken, for a prolonged period can also result in poisoning as ragwort contains pyrrolizidine alkaloids which cause liver failure and bracken contains an enzyme that has an anti-thiamine effect, inducing thiamine deficiency. Photosensitisation following exposure to plants containing phototoxins and bright sunlight is also a risk in summer. Treatment of plant poisoning in horses is generally supportive and involves removing the plant, providing good quality food and supportive care. Thiamine supplementation may be required in severe cases of bracken poisoning. Animals should be protected from sunlight following exposure to a phototoxin, with supportive management of skin lesions. Management of adder bite is supportive with administration of adder antivenom. Multiple bee stings require aggressive supportive care.

https://doi.org/10.12968/ukve.2022.6.4.175

Nicola Bates BSc (Brunel) BSc (Open) MSc MA, Senior Information Scientist, Veterinary Poisons Information Service (VPIS), 2nd Floor, Godfree Court, 29-35 Long Lane, London, SE1 4PL. nicola.bates@vpisglobal.com

Key words: poisoning | toxicology | bracken poisoning | adder bite | bee sting | photosensitisation

Adders and stinging insects are more active in the warmer months, so the potential risk of horses being bitten or stung is increased. Bright sunlight increases the risk of photosensitisation following exposure to plants containing phototoxic chemicals, and grazing on plants that can be toxic with prolonged exposure may also occur over the summer months (and in the winter if the plants contaminate hay).

This article briefly discusses some of the seasonal summer poisoning hazards for horses. If more detailed information is required when managing a case, consult a veterinary poisons information service (*Box 1*).

Adder bite

European adders (*Vipera berus berus*, *Figure 1*) are most active in the summer. Although relatively common in dogs, envenomation from an adder bite can also occur in large animals including horses. Adders may not be seen to bite a horse, but envenomation should be part of the differential diagnosis in a horse with localised limb swelling that spreads up the leg. Close examination may reveal puncture wounds. Envenomation can cause significant morbidity but low mortality; and horses may be bitten on a limb or the face (Arbuckle and Theakston, 1992; Anlén, 2008).

Local effects from an adder bite typically occur within a few hours with localised, progressive, painful swelling. Other signs include depression, tachycardia, tachypnoea, pyrexia, generalised muscle spasms and reduced gut sounds with colic. There may be lameness after a bite on the leg and cellulitis may occur, as well as hypotension and shock. Complications reported in horses include prolonged exercise intolerance, tissue necrosis at the bite site, ventricular tachycardia (caused by venom-induced cardiac damage, which occurs days later), dyspnoea and dysphagia.

Horses with mild swelling after a suspected adder bite probably require supportive care only and should be kept rested. Temperature, heart rate and blood pressure should be checked, if possible. Animals should be monitored for signs of shock, myocardial damage and local tissue necrosis.

Analgesia should be given if required, but antibiotics are probably only required if infection occurs. There is no role for steroids in the treatment of adder bites (except in rare cases of anaphylactic reactions to antivenom) (Bates and Warrell, 2013). The initial swelling from an adder bite is not an inflammatory response but a result of the cytotoxic effect of the venom. Steroids can also slow and diminish the response to antivenom and increase the risk of infection, thus should not be given where antivenom is used.

Antivenom has been used in horses with adder envenomation (Anlén, 2008), and should be considered in any horse with significant swelling at the bite site or with any systemic signs such as coagulopathy, or evidence of myocardial damage or shock. The optimal dose in large animals has not been established, but the dose of antivenom is the same irrespective of the size of the victim, as the dose is designed to counteract the venom of one bite.

Box 1. Veterinary poisons information services

- Veterinary Poisons Information Service (VPIS) https://www.vpisglobal.com/
- American Society for the Prevention of Cruelty to Animals Animal Poison Control Center: https://www.aspca.org/pet-care/animal-poison-control
- Specialist veterinary poisons information services are available, but some human poisons centres also answer enquiries about animal poisoning. Check with your local centre.

*Figure 1. The European adder (*Vipera berus berus*) which can cause envenomation in horses during the spring and summer. If the biting event is not witnessed, the diagnosis may be missed.*

Figure 2. Multiple bee stings can cause multi-organ damage in victims.

Clinical improvement should be seen rapidly, particularly if given within a few hours of envenomation.

If there is no clinical improvement, within 2 hours of administration of the initial dose, then the regimen may be repeated. Antivenom should be given as soon as possible for maximum effect, and is indicated as long as there are signs of systemic envenoming (including shock, bleeding, cardiac effects and generalised oedema), even days after the bite. In animals with only local envenoming (such as swelling), there is no value in giving antivenom more than 24 hours after the bite and management would be supportive.

Bee stings

Bee stings may result in local effects, hypersensitivity reactions to a single sting (allergic reactions), or toxic systemic reactions from multiple stings (*Figure 2*). Africanised honeybees are more aggressive than other bees, but are only found in the Americas.

Local reactions include immediate pain, erythema, swelling, inflammation and pruritus. These effects are usually self-limiting and resolve within about 24 hours. In some cases, there may be a larger, more regional reaction which spreads out from the site of the sting with erythema, local oedema and cellulitis (infection is rare) (Fitzgerald, 2012).

In animals with multiple stings, local effects and multi-organ damage can be seen with associated depression, pyrexia, red-brown urine, rhabdomyolysis, acute renal failure, myoglobinuria, elevated liver enzymes, disseminated intravascular coagulation, pulmonary oedema and respiratory distress. Recovery may be slow because of multi-organ involvement. In one horse presenting 5 hours after multiple bee stings, recovery took 30 days (Fonteque et al, 2018). Deaths have been reported in horses with multiple bee stings (Anon, 1941; Staemplfi et al, 1993).

Multiple sting events in horses are most commonly reported in the veterinary literature (Staemplfi et al, 1993; Lewis and Racklyeft, 2014; Fonteque et al, 2018; Ribeiro et al, 2020; Veado et al, 2020), but a single sting in a sensitive individual could produce serious, potentially fatal, anaphylactic reactions. In addition, delayed reactions from insect stings may occur days later, although they are rare. Effects include rash, serum sickness, pyrexia, general malaise and lymphadenopathy (Fitzgerald, 2012).

Treatment of bee stings is supportive. Prompt treatment is required for animals with anaphylactic reactions. Any bee sting still embedded in the skin should be promptly removed. The emphasis is on speed of removal, without concern for the method used (Visscher et al, 1996). If multiple bee stings are suspected, the animal should be carefully examined to locate all the bees. Treatment of a local reaction from a sting is symptomatic, with antihistamines, steroids and analgesia if required. A cold compress may also help alleviate discomfort (Reisman, 1994).

Aggressive supportive care is required for any horse with multiple stings, with correction of hypovolaemia and maintenance of urine output. Monitoring should include vital signs (cardiac and respiratory), liver and renal function, haematology and coagulation parameters.

Pyrrolizidine alkaloids

Pyrrolizidine alkaloids are found in a number of plants families, but one of the most common plants associated with poisoning is ragwort (*Jacobaea vulgaris,* previously *Senecio jacobaea*, *Figure 3*). It is a common cause of poisoning in horses (Crews and Anderson, 2009; Vandenbroucke et al, 2010). Other plants containing pyrrolizidine alkaloids include *Cynoglossum officinale* (hound's tongue) and *Heliotropium* species (heliotrope).

Pyrrolizidine alkaloid toxicosis is characterised by liver damage, and signs of poisoning usually only occur after weeks, months or even years of exposure. Onset of signs may occur abruptly in horses, irrespective of the duration of exposure. Death usually occurs a few days after onset of clinical signs (Giles, 1983), but can also occur weeks or months after onset (Arzt and Mount, 1999), and months after removal from the toxic plants (Lessard et al, 1986; Craig et al, 1991; van Weeren et al, 1999).

The early signs of pyrrolizidine alkaloid exposure are non-specific with anorexia, weight loss and depression. There may also be


*Figure 3. Ragwort (*Jacobaea vulgaris*) is a common weed and contains pyrrolizidine alkaloids, which can cause liver damage.*

diarrhoea or constipation, tenesmus, photosensitisation, ascites, jaundice and signs suggestive of hepatic encephalopathy, such as head pressing, yawning, restlessness and aimless walking, apparent blindness and drowsiness.

In more chronic poisoning with ingestion of low levels of pyrrolizidine alkaloids over a prolonged period, animals may appear asymptomatic but ongoing liver damage may result in inflammation, fibrosis and ultimately, cirrhosis. Normal stresses such as pregnancy or lactation may result in clinical liver failure.

Mortality from pyrrolizidine alkaloid toxicosis is high, particularly in symptomatic animals, as advanced liver damage is irreversible. There is no specific treatment for pyrrolizidine alkaloid toxicosis. Animals should be removed from the source, followed by supportive management for liver disease and hepatic encephalopathy.

Bracken

Bracken (*Pteridium*) is a fern and is one of the most abundant plants in the world. It has long been recognised as a toxic plant and it contains several toxic compounds, but in horses it causes avitaminosis B1 (Evans, 1976). Thiamine is an essential vitamin involved in metabolism and maintaining the myelin of peripheral nerves. Bracken contains a type I thiaminase which does not destroy thiamine, but creates a thiamine analogue. This inhibits thiamine-requiring metabolic reactions and has an anti-thiamine effect which induces thiamine deficiency (Chick et al, 1985).

Poisoning only occurs as a result of ingesting bracken for several weeks (Vetter, 2009). Poisoning can also occur after ingestion of contaminated hay, following grazing on bracken when other food sources are unavailable because of adverse conditions or eating bracken used as bedding (Hadwen and Bruce, 1917).

Clinical signs of thiamine deficiency include depression, constipation and ataxia, and lack of coordination ('bracken staggers'). The appetite typically remains (Hadwen and Bruce, 1917). As the disease progresses (generally over 2–7 days), there is loss of condition, weakness, inability to stand, nervousness, severe muscle tremors, convulsions and opisthotonus. In addition, there may be haemolytic anaemia and haemoglobinuria (Roberts et al, 1949; Kelleway and Geovjian, 1978).

In mild cases of bracken poisoning, animals recover once removed from exposure. Treatment is supportive and administration of thiamine may be required in some cases. Good nursing care and high-quality feed should be provided. Affected animals should be monitored for signs of anaemia.

Photosensitivity

Photosensitivity is a syndrome that develops when an animal becomes abnormally reactive to sunlight owing to the presence of a phototoxin or photoallergen in or on the skin. This results in varying degrees of dermatitis and/or keratoconjunctivitis occurring in skin and/or eye membranes that are unprotected by melanin pigmentation, thick epidermis, hair or fabric covering. This subsequently results in inflammation, necrosis and sloughing. In severe cases, even pigmented skin is affected. In farm animals and horses, the cause is usually ingestion of or exposure to plants containing phototoxic compounds. In addition, secondary photosensitisation may occur with some plants that cause liver dysfunction. Phylloerythrin, a natural metabolite of the anaerobic fermentation of chlorophyll, cannot be metabolised by the damaged liver and intead accumulates in the skin, where it reacts with sunlight and causes severe cell damage. As sunlight is required for photosensitisation to develop, the risk is greater in summer when there is strong sunlight, the days are longer and more time is spent outside.

Many plants have been implicated in causing photosensitisation, often on the basis of limited evidence (Collett, 2019). Well-recognised photosensitising plants include St John's wort (*Hypericum perforatum; Hypericum maculatum*, imperforate St John's wort), rue (*Ruta graveolens*) and wild parsnip (*Pastinaca sativa*).

KEY POINTS

- Some types of poisoning are seasonal.
- Summer hazards in horses include adder bites and bee stings, as these animals are more active in warm weather.
- Multiple bee stings in horses can cause multi-organ involvement and may be fatal.
- Repeated and prolonged ingestion of some plants, such as bracken and ragwort, can cause poisoning in horses.
- Exposure to a plant containing a phototoxin and bright sunlight may result in photosensitisation and dermal effects.

Photosensation has been reported in horses following exposure to wild parsnip (Stegelmeier et al, 2019), angelica (*Angelica archangelica*) and St John's wort (Modrá and Svobodová, 2009).

Treatment of photosensitivity is supportive with removal from sunlight and management of skin lesions. Management of liver dysfunction is more difficult, but liver biopsy may aid diagnosis (Stegelmeier et al, 2020).

Conclusions

There is a seasonal risk with some poisons. In the summer, stinging insects and adders are more active and are a risk to horses. The owner of any horse with sudden onset swelling should be questioned about possible adders in the area. Multiple bee stings are particularly hazardous and can cause multi-organ damage. Grazing on some plants, such as ragwort or bracken, for a prolonged period can result in poisoning. In addition, bright sunlight in the summer months may result in photosensitisation following exposure to plants containing phototoxins.

Careful questioning of owners may help the diagnosis of plant poisoning in some cases. Treatment of plant poisoning in horses is generally supportive with removal of the plant, providing good quality food and supportive care. Animals should be protected from sunlight following exposure to a phototoxin. Management of adder bite is supportive with administration of specific antivenom. Multiple bee stings require aggressive supportive care. EQ

Conflicts of interest

The author has no conflicts of interest to declare.

References

Anlén KG. Effects of bites by the European adder (*Vipera berus*) in seven Swedish horses. Vet Rec. 2008;162(20):652-6. https://doi.org/10.1136/vr.162.20.652

Anon. Death of horse due to bee-stinging. Can J Comp Med Vet Sci. 1941;5(9):270

Arbuckle JB, Theakston RD. Facial swelling in a pony attributable to an adder bite. Vet Rec. 1992;131(4):75–76

Arzt J, Mount ME. Hepatotoxicity associated with pyrrolizidine alkaloid (*Crotalaria* spp) ingestion in a horse on Easter Island. Vet Hum Toxicol. 1999;41(2):96–9

Bates NS, Warrell DA. Treatment of adder bites in dogs. Vet Rec. 2013;172(1):23–4 https://doi.org/10.1136/vr.e8620.

Chick BF, Quinn C, McCleary BV. Pteridophyte intoxication of livestock in Australia. In: Seawright AA, Hegarty MP, James LF, Keeler RF (eds). Plant Toxicology. Queensland Poisonous Plants Committee, Yeeronpilly, 1985; 453–464.

Collett MG. Photosensitisation diseases of animals: Classification and a weight of evidence approach to primary causes. Toxicon X. 2019;3:100012. https://doi.org/10.1016/j.toxcx.2019.100012

Craig AM, Pearson EG, Meyer C, Schmitz JA. Clinicopathologic studies of tansy ragwort toxicosis in ponies: sequential serum and histopathological changes. Equine Vet Sci. 1991;11(5):261–271

Crews C, Anderson WA. Detection of ragwort alkaloids in toxic hay by liquid chromatography/time-of-flight mass spectrometry. Vet Rec. 2009;165(19):568–9. https://doi.org/10.1136/vr.165.19.568

Evans WC. Bracken thiaminase-mediated neurotoxic syndromes. Botanical J Linnean Soc. 1976;73:113–131

Fitzgerald KT. Insects - Hymenoptera. In: Small Animal Toxicology, 3rd edition. Peterson ME, Talcott PA (eds). Elsevier Saunders, St Louis (MO): 2012; 573–588.

Fonteque JH, Mendes RP, Souza AF, Granella MCS, Schade J, Casa MS, Yonezawa LA, Volpato J. Systemic toxic reaction due to multiple honeybee stings in equine: Case report. Arq Bras Med Vet Zootec. 2018;70(3):767–772. https://doi.org/10.1590/1678-4162-10012

Giles CJ. Outbreak of ragwort (*Senecio jacobea*) poisoning in horses. Equine Vet J. 1983;15(3):248-50

Hadwen S, Bruce EA. The poisoning of horse by the common bracken (*Pteris aquilina* L). Dept of Agriculture, Health of Animals Branch, Scientific Series Bulletin No. 26, 1917

Kelleway RA, Geovjian L. Acute bracken fern poisoning in a 14-month-old horse. Vet Med Small Anim Clin. 1978;73(3):295–296

Lessard P, Wilson WD, Olander HJ, Rogers QR, Mendel VE. Clinicopathologic study of horses surviving pyrrolizidine alkaloid (*Senecio jacobaea*) toxicosis. Am J Vet Res. 1986;47(8):1776–1780

Lewis N, Racklyeft DJ. Mass envenomation of a mare and foal by bees. Aust Vet J. 2014;92(5):141–8. https://doi.org/10.1111/avj.12177

Modrá H, Svobodová Z. Incidence of animal poisoning cases in the Czech Republic: current situation. Interdiscip Toxicol. 2009;2(2):48-51. https://doi.org/10.2478/v10102-009-0009-z

Reisman RE. Insect stings. N Engl J Med. 1994;331(8):523-7.

Ribeiro PR, Bianchi MV, Henker LC, Gonzales F, Pavarini SP. Acute renal failure in a horse following bee sting toxicity. Ciência Rural, Santa Maria. 2020;50(5):e20190940. https://doi.org/10.1590/0103-8478cr20190940

Roberts HE, Evans ET, Evans WC. The production of "bracken staggers" in the horse and its treatment by Vitamin B1 therapy. Vet Rec. 1949;61(35):549–550

Staemplfi HR, Wollenberg G, Jewell G, McCutcheon LJ. Acute fatal reaction to bee stings in a mare. Equine Vet Edu. 1993;5(5):250–252

Stegelmeier BL, Colegate SM, Knoppel EL, Rood KA, Collett MG. Wild parsnip (Pastinaca sativa)-induced photosensitization. Toxicon. 2019;167:60–66. https://doi.org/10.1016/j.toxicon.2019.06.007

Stegelmeier BL, Davis TZ, Clayton MJ. Plant-induced photosensitivity and dermatitis in livestock. Vet Clin North Am Food Anim Pract. 2020;36(3):725–733. https://doi.org/10.1016/j.cvfa.2020.08.008

van Weeren PR, Morales JA, Rodríguez LL, Cedeño H, Villalobos J, Poveda LJ. Mortality supposedly due to intoxication by pyrrolizidine alkaloids from *Heliotropium indicum* in a horse population in Costa Rica: a case report. Vet Q. 1999;21(2):59–62

Vandenbroucke V, Van Pelt H, De Backer P, Croubels S. Animal poisonings in Belgium: a review of the past decade. Vlaams Diergeneeskundig Tijdschrift. 2010;79:251–268

Veado HC, Conceição RS, Nogueira K et al. Massive Africanized honeybee stings in two hair sheep and a mare. Toxicon. 2020;177:35–40. https://doi.org/10.1016/j.toxicon.2020.01.015

Vetter J. A biological hazard of our age: bracken fern [*Pteridium aquilinum* (L) L Kuhn] - a review. Acta Veterina Hungarica. 2009;57(1):183–196. http://doi.org/10.1556/AVet.57.2009.1.18

Visscher PK, Vetter RS, Camazine S. Removing bee stings. Lancet. 1996;348(9023):301–2