Spring poisoning hazards

Poisoning can be seasonal. Potential toxic hazards in the spring include adders, which emerge from hibernation as the weather warms. Adders may not be seen to bite a horse, but envenomation should be considered in a horse with localised limb swelling that spreads up the leg. There are several spring-flowering plants which contain toxic compounds such as cardiogenic glycosides (foxglove, lily of the valley, oleander), protoanemonin (Hellebore) and grayanotoxins (Pieris and Rhododendron). Saplings of sycamore are a spring source of hypoglycin A which causes atypical myopathy in horses. Treating plant poisoning in horses generally involves removing the plant, providing good quality food and giving supportive care. Managing the effects of adder bite is supportive, centring around administration of specific antivenom.

https://doi.org/10.12968/ukve.2021.5.2.76

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Key words: poisoning | toxicology | plant poisoning | adder bite | cardiac glycosides | atypical myopathy

As the weather warms in the spring, horses may be at risk of envenomation from adders emerging from hibernation or poisoning from grazing on the fresh spring growth of plants. Also, if the winter has been harsh and other forage is unavailable, horses may eat plants they would normally avoid. Plant poisoning can also occur if horses escape from their enclosure or if cuttings from spring-pruned plants or felled trees are left within their reach.

There is limited information on the incidence of poisoning in horses and it likely that cases are under-reported. In Europe, most cases of poisoning reported in horses involve plants and pesticides (Guitart et al, 2010; Bates, 2017; McFarland et al, 2017). If required, cases can be discussed with a poisons information service and can be reported retrospectively to the UK's Veterinary Poisons Information Service (VPIS) via its website (https://www.vpisglobalcom/report-a-case/).

Adder bite

European adders (*Vipera berus berus*) hibernate from around October to February. They emerge when the weather warms and bites typically occur in the spring and summer. Although relatively common in dogs, envenomation from an adder bite can also occur in horses and other large animals. 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 (*Figure 1*). Close examination might reveal puncture wounds, and horses can be bitten on a limb or the face (Arbuckle and Theakston, 1992; Anlén, 2008). Envenomation can cause significant morbidity but low mortality.

The venom of European adders is a complex mixture of compounds that causes release of pharmacologically active substances in the body, which can have a direct effect on organs. Local effects from an adder bite typically occur within a few hours with localised, progressive, painful swelling. Other signs reported include hypersalivation, depression, tachycardia, tachypnoea, pyrexia, generalised muscle spasms and reduced gut sounds with colic. There can 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 will likely require supportive care only and should be kept rested. The temperature, heart rate and blood pressure should be checked, if

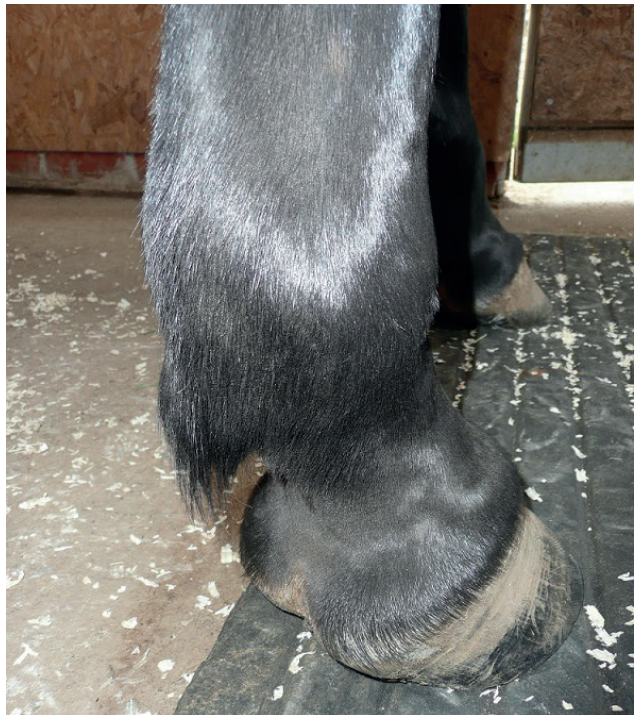

Figure 1. Limb swelling after an adder bite (Vipera berus berus) in a horse not given antivenom (day 3). Courtesy of Fiona Bolton.

possible. Animals should be monitored for signs of shock, myocardial damage and local tissue necrosis.

Analgesia should be given if required but antibiotics are 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, so they should not be given where antivenom is used.

There is no information on the incidence of adverse effects to adder antivenom in animals. Most antivenoms, particularly the older products, are produced in horses but some are also derived from sheep (Lamb et al, 2017). In an analysis of 2400 human cases treated with various antivenoms, the incidence of adverse effects was 1.5% (Lamb et al, 2017).

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, 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. 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 administering 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 envenomation (shock, bleeding, cardiac effects, generalised oedema), even days after the bite occurred. In animals with only local envenomation (swelling), there is no value in giving antivenom more than 24 hours after the bite and management decisions will be based on supporting the horse.

Cardiac glycoside-containing plants

Cardiac glycosides are found in numerous plants. Most cases of cardiac glycoside poisoning in horses involve oleander (*Nerium oleander*) (*Figure 2a*) (Anon, 1971; Hughes et al, 2002; Bazargani et al, 2008; Durie et al, 2008; Renier et al, 2013; Butler et al, 2016), but foxglove (*Digitalis purpurea*) poisoning (*Figure 2b*) has also been reported (Wijnberg et al, 1999). Other plants that contain cardiac glycosides are lily of the valley (*Convallaria majalis*) (*Figure 2c*), bluebells (*Hyacinthoides nonscripta*) and related species like Spanish bluebells (*Hyacinthoides hispanica*) and Italian bluebells (*Hyacinthoides italica*). Bluebell poisoning is occasionally reported in horses (Forsyth, 1979).

Some plants such as foxgloves are bitter and so may not be eaten readily, although deer, for example, have been observed to choose to graze foxgloves with subsequent toxic effects (Corrigall et al, 1978). Therefore, a plant's bitter taste may not always deter animals and provide protection. Dried oleander is reported to be more palatable than fresh (Butler et al, 2016). Cardiac glycosides cause electrolye distrubance resulting in changes in the electrical conductivity of the heart. Although therapeutic doses of cardiac glycosides (such as the drug digoxin) have an anti-arrhythmic effect, larger amounts are pro-arrhythmic.

Figure 2. Plants containing cardiac glycosides include (a) Nerium oleander *(oleander), (b)* Digitalis purpurea *(foxglove) and (c)* Convallaria majalis *(lily of the valley).*

In an experimental study, donkeys given 10 or 20 mg/kg of powdered oleander leaves developed arrhythmias but survived, whereas a dose of 30 mg/kg was fatal (Rezakhani and Maham, 1994). A dose of 10–12 leaves of oleander is potentially lethal to an adult horse (Renier et al, 2013).

Sudden death after ingesting plant material containing cardiac glycosides is reported commonly. Signs reported in horses include abdominal discomfort and colic, depression, diarrhoea, ataxia, weakness, bradycardia or tachycardia, cold extremities, signs of ileus or gastrointestinal hypomotility, signs of decreased tissue perfusion (including weak pulse and slow capillary refill time), renal impairment, arrhythmias and cardiovascular collapse. Death can occur 12–36 hours after ingestion (Hughes et al, 2002). In one equine case, death occurred 72 hours after the onset of clinical signs but had possibly been delayed by aggressive supportive care (Butler et al, 2016).

Laboratory findings in animals poisoned with cardiac glycosides include haemoconcentration, hyperkalaemia, hyperglycaemia, acidosis, raised creatine kinase, lactate, liver enzymes, urea and creatinine, and increased leucocytosis.

Arrhythmias are typically ventricular in origin and often include ventricular premature contractions and ventricular tachycardia but atrioventricular (AV) block, AV dissociation, sinoatrial (SA) block, bundle branch block, prolonged PR interval, ST deviation and ventricular fibrillation have also been reported (Wijn-

Figure 3. Plants containing grayanotoxins include (a) Rhododendron *species and (b)* Pieris *species.*

berg et al, 1999; Hughes et al, 2002; Durie et al, 2008; Renier et al, 2013). In a review of 30 cases of oleander poisoning in equids, the mortality was 50% (10 horses died, 5 were euthanased). Survival was lower for animals with arrhythmias and greater for those who remained hospitalised for longer (Renier et al, 2013).

Treatment of cardiac glycoside toxicosis is supportive. Activated charcoal (1–3 g/kg) can be given if ingestion was recent but should not be given in animals with reduced bowel movements or constipation. A good quality diet should be provided with rehydration, if required. Analgesia may be required for abdominal discomfort. If possible, the electrolytes, particularly potassium should be checked. The echocardiogram should be monitored, if possible. Rehydration may be required, avoiding calcium-containing fluids as elevated serum calcium can increase the effect of cardiac glycosides on the myocardium. Potassium should only be given if hyperkalaemia is absent. Atropine can be used for atrioventricular block or bradycardia. However, there is the risk of gastrointestinal stasis with atropine in horses, but this is small. Lidocaine has been used in the management of ventricular arrhythmias (Durie et al, 2008; Renier et al, 2013; Butler et al, 2016) and phenytoin (10–2 mg/kg orally every 12 hours) has been used for cardiac glycoside-induced arrhythmias (Wijnberg et al, 1999; Smith et al, 2003; Durie et al, 2008).

Digoxin antibody Fab fragments (DigiFab®, Digibind®) are an option, but these drugs are expensive. In veterinary medicine it is recommended that 1 or 2 vials of either product are given initially and the effects observed (Gwaltney-Brant and Rumbeiha, 2002). More vials can be given if there is some clinical improvement, but cost may be prohibitive. Digoxin antibody Fab fragments have been used in a horse with oleander poisoning reported to the VPIS. The horse died but this may have been for several reasons, including a suboptimal dose of antibodies. Digoxin antibody Fab fragments have been used successully in a cat (Galton et al, 2020) and dogs with cardiac glycoside poisoning (Mukai et al, 2009; Pao-Franco et al, 2017).

Plants containing grayanotoxin

Grayanotoxins are found in the spring flowering plants of the *Rhododendron* species (which includes azaleas) (*Figure 3a*) and *Pieris* species (*Figure 3b*). These plants contain several grayanotoxins in the nectar, flowers, leaves and stems. Grayanotoxins act on cell membrane sodium channels and cells remain activated. The main effects occur in nerve and muscle cells. Although grayanotoxin poisoning is common in sheep and goats it has been reported occasionally in horses and donkeys (Forsyth, 1979; Thiermann, 1991).

Clinical signs of grayanotoxin poisoning generally occur within 6 hours of ingestion (Puschner et al, 2001). Initial signs are gastrointestinal with severe hypersalivation and abdominal discomfort. Neurological signs (progressive restlessness, ataxia, depression, generalised muscle tremors, dilated pupils) and cardio-respiratory effects (dyspnoea with variation in the pace, intensity and frequency of respiratory movements, episodes of apnoea, bradycardia with cardiac arrhythmia) can also occur. There may also be bruxism, hypotension, pyrexia and vocalisation. In the final stages there is increased muscle weakness, flaccid paralysis, coma and convulsions. Aspiration pneumonia (secondary to regurgitation of stomach contents) is a potential risk.

Elevated creatine kinase and alanine aminotransferase (caused by muscle damage) has been reported in equines. Findings reported in other species are non-specific, and include leucocytosis, anaemia, metabolic acidosis and evidence of dehydration (Plumlee et al, 1992; Pearson et al, 1996).

Effects of grayanotoxicosis can be short-lived, often lasting no more than 24 hours because of the rapid elimination of grayanotoxins (Hikino et al 1979). In more severe cases, however, recovery may take up to a week and is usually complete. In fatal cases, death is usually caused by respiratory failure and may occur within a few hours of ingestion.

Treatment of grayanotoxin poisoning is supportive and there is no antidote. Activated charcoal (1–3 g/kg) may be given if ingestion was recent.

Plants containing toxalbumin

Horses appear to be particularly susceptible to the toxic effects of *Robinia pseudoacacia* (black locust or false acacia) (*Figure 4*) (Landolt et al, 1997; Hopper, 1999; Thursby-Pelham, 1999; Metzger et al, 2006; Uhlig et al, 2007; Vanschandevijl et al, 2010). The specific toxin has not been identified and numerous compounds have been described, although the naming of these is subject to confusion. The main toxic principle may be the toxalbumin robin, but the plant also contains robitin (a glycoside) and robinine (an alkaloid).

Onset of signs following ingestion of *Robinia pseudoacacia* can be rapid (often within 1–2 hours but can be up to 12 hours). Although small doses cause only gastrointestinal signs, a large quantity can result in neurological effects (Uhlig et al, 2007).

Gastrointestinal effects are common with colic, which can be severe, and diarrhoea. Other signs include weakness, depression, dilated pupils, tachycardia, reduced or absent bowel sounds, hyper- or hypothermia, sweating, tremor, muscle spasms and elevated liver enzymes. In severe cases there may be absent menace response, nystagmus, head pressing and ataxia (Landolt et al, 1997; Metzger et al, 2006). There may be periods of excitement alternating with periods of depression (Uhlig et al, 2007). This may progress to dyspnoea, recumbency, coma, increased muscle tone, spastic limbs, trismus and convulsions (Metzger et al, 2006). Arrhythmias have also been described (Vanschandevijl et al, 2010).

Treatment of *Robinia pseudoacacia* poisoning is supportive with analgesia, rehydration, sedation and oxygen, if required. Fatal cases are rare (Kingsbury, 1964) but have been reported in horses (Metzger et al, 2006; Vanschandevijl et al, 2010).

Plants containing hypoglycin A

Atypical myopathy (or seasonal pasture myopathy as it is known in North America) is a muscle disease of horses with a high rate of mortality. It is associated with chronic ingestion of seeds or seedlings of some *Acer* species, including (*Acer negundo* or box elder/ashleaf maple). These plants contain hypoglycin A (McKenzie et al, 2016), which disrupts oxidation of fatty acids, resulting in a severe energy deficiency and muscle destruction. Signs are brought on by acute degeneration in postural and respiratory muscles and sometimes the myocardium (Votion et al, 2014).

Cases of poisoning occur in the spring from ingestion of sycamore (*Acer pseudoplatanus*) seedlings and in the autumn from ingestion of sycamore seeds (samaras) (Baise et al, 2016). Based on the concentrations of hypoglycin A and the toxic dose (Valberg, 2014), the estimated toxic dose for a 500 kg horse would be 50–200 juvenile sycamore seedlings or 85–196 single samaras (Aboling et al, 2020).

Initial signs of hypoglycin A toxicity include a reluctance to move and a quiet demeanour. Signs then progress to pronounced muscle weakness (with stiffness, dysphagia, recumbency and respiratory distress) and pain (sweating, depression, gastrointestinal impaction). Tachycardia may result from cardiomyopathy and/or pain. There is hyperlipidaemia and hyperglycaemia, and pigmenturia showing as a dark red/brown urine occurs in the majority of cases (Rendle, 2016).

Secondary complications are common with hyoglycin A poisoning and include head oedema, pressure sores, buccal ulceration, gastric ulceration, choke, diarrhoea, renal dysfunction, paraphimosis and corneal ulceration. Death can occur from cardiac, or more commonly, respiratory failure (Rendle, 2016).

Treatment of hypoglycin A poisoning is supportive and should be started as soon at the disease is recognised. Renal and respiratory function, triglycerides and glucose should be monitored. An echocardiogram and troponin concentrations should be monitored for evidence of myocardial dysfunction. A low-fat diet is recommended and food should be given little and often to prevent large fluctuations in glucose (Rendle, 2016). Vitamins and antioxidants (such as vitamin E, selenium, vitamin C) may be helpful (van Galen et al, 2012). Oxygen may be required in animals with respiratory distress and the head should be elevated to prevent head oedema. Lidocaine or magnesium sulphate can be given for ventricular tachycardia. Negative inotrope drugs should be avoided, such as beta blockers and calcium channel blockers (Rendle, 2016). Massaging affected muscles may be helpful to ease pain and stimulate blood flow (Rendle, 2016).

Mortality rates in horses with hypoglycin A toxicity varies from 3–57% (Rendle, 2016). In survivors there is usually deterioration over 48–72 hours before improvement. Animals that survive beyond 4–5 days have a better prognosis and there don't appear to show long-term sequelae (Valberg, 2014).

Not all *Acer* species are associated with hypoglycin A poisoning. Hypoglycin A is not found in field maple (*Acer campestre*) or Norway maple (*Acer platanoides*). *Acer rubrum* (red maple) causes haemolysis and methaemoglobinaemia (Corriher et al, 1999) and poisoning has been reported in horses, ponies (George et al, 1982; Tennant et al, 1991; Alward et al, 2006) and zebras (Weber and Miller, 1997). It is most commonly reported in North America rather than Europe.

Plants containing protoanemonin

Protoanemonin is found in hellebores (*Helleborus* species) (*Figure 5*), *Ranunculus* species (which includes buttercups) and other plants. Protoanemonin is a volatile yellow oil and a potent irritant that is formed from the glycoside ranunculin when the plant is

Figure 4. Robinia pseudoacacia *(false acacia, black locust) causes gastrointestinal signs but a large quantity can result in neurological effects.*

KEY POINTS

- Some types of poisoning are seasonal.
- Spring hazards in horses include adder bite and common spring flowering plants.
- The saplings of other plants such as sycamore are also a source of poisoning in horses.
- Clinical signs will depend on the type of toxic compounds involved.
- In most cases treatment is supportive with removal of the plant, rehydration, activated charcoal and providing good nutrition.

crushed (Hill and Van Heyningen, 1951). Protoanemonin-containing plants are irritant and have a bitter taste. Protoanemonin is unstable and spontaneously breaks down to inactive anemonin if plant material is dried or heated. Therefore, such material (such as in hay) is less irritant.

Buttercups have long been recognised to cause adverse effects in horses (Gerrard, 1874; Carruthers, 1899). Most cases of poisoning occur after grazing on pasture containing high concentrations of buttercups (Piekarz, 1981; Griess, 1997) or in horses fed fresh cut grass containing buttercups. Some animals seem to acquire a taste for buttercups and seek them out (Forsyth, 1979).

Ingestion of plant material containing protoanemonin can result in hypersalivation, inflammation of the mouth, gastroenteritis and colic. In severe cases there may be ulceration of the mouth and gastrointestinal tract, diarrhoea, ataxia and convulsions. However, severe cases are uncommon as the plants tend to have a bitter taste, which discourages ingestion. Buttercups vary in their toxicity and do not cause severe toxicity under most circumstances.

Treatment is supportive with gastrointestinal protectants and analgesia.

Figure 5. The main toxin compound in Helleborous *species (hellebore) is protoanemonin.*

Conclusion

Potential toxic hazards in the spring include adders as they emerge from hibernation, and spring-flowering plants such as hellebores, foxgloves, lily of the valley, oleander, pieris and rhododendron. In addition, the saplings of sycamore are a spring hazard for horses, just as the seeds are in the autumn. Treatment of plant poisoning in horses is generally supportive with removal of the plant, providing good quality food and supportive care. Management of adder bite is supportive and includes administering specific antivenom. EQ

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