PLANT POISONING IN HORSES

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Practice Notes

AT the time of writing, cases of atypical myopathy (AM) are peaking, so it seems appropriate to review plant poisoning as horses forage for food supplies as an alternative to grass.

About 1,500 known species of poisonous plants exist, so this article will only address some commonly encountered species and their toxic agents. AM is discussed as a separate entity. If poisoning is suspected, the plant should be identified before treatment begins. There are three treatment mainstays, regardless of cause – remove residual poison, provide antidotes if possible and supportive care. Activated charcoal (1g/kg to 3g/kg) can adsorb mycotoxins, plant alkaloids and bacterial toxins[1].

Atypical myopathy

AM, or seasonal pasture myopathy, appears to be due to the toxin hypoglycin A in sycamore seeds, although *Clostridium sordellii* and *Rhytisma acerinum* mycotoxins have been investigated. This theory is based on similar diseases; seasonal pasture disease of equines in the US and Canada, and a human disease called Jamaican vomiting sickness – both of which are attributed to hypoglycin A[2].

Although horses do not preferentially ingest sycamore seedlings, there is often considerable pasture contamination after autumnal winds and inadvertent ingestion occurs. A second peak of cases is commonly seen in the spring if there have been high numbers of autumnal cases. This highly fatal disease results in muscle weakness or stiffness, vague colic-like symptoms, laboured breathing, dark red-brown urine, recumbency and sudden death. It appears the type I oxidative fibres of the postural, respiratory and cardiac muscles are affected[2].

Biochemistry reveals markedly elevated creatine kinase (CK), aspartate aminotransferase, lactate dehydrogenase and lactate. These can be monitored and a decreasing CK is a positive prognostic indicator. It is common to see hypocalcaemia, hypochloraemia and mild hyponatraemia, although this is not definitive[3]. Horses often show an increased PCV; greater increases are linked with mortality. It is common to see respiratory alkalosis, although the ability to measure this is not always available. About 26 per cent of cases recover and therefore horses diagnosed early in the disease process should be treated aggressively if they have any chance of survival[4]. Positive prognostic indicators include remaining standing, normal temperature, normal mucus membranes and normal defaecation[4].

The mainstay of therapy is intravenous fluid therapy to restore circulating volume, increase myoglobin excretion and correct electrolyte derangements[4]. Calcium supplementation is not fully evaluated in cases of AM. Analgesia should be considered, although nephrotoxicity may occur with NSAID use in dehydrated individuals. Omeprazole may preserve appetite and reduce the formation of gastric ulceration. Muscle relaxants such as acepromazine may be beneficial, but, again, care with dehydrated cases is important[3]. Dantrolene should probably be avoided due to its inhibition on energy metabolism[3]. Other supportive muscle therapies, such as vitamins B and E supplementation, may be useful[3]. If a horse is in lateral recumbency, regular turning can improve ventilation and reduce the formation of pressure sores.

As muscle lipid metabolism is affected, nutrition must be carefully addressed – especially as many of these horses will maintain their appetite throughout the disease course. A diet of multiple small meals with a reduced lipid content and increased carbohydrate fraction is most appropriate[4]. Horses in lateral recumbency and with digestive dysfunction can be given parenteral nutrition, although anorexia is a poor prognostic indicator. Understandably, this disease is causing extreme concern among owners. As the pathogenesis has not been definitively identified, preventive advice is hard to give. Owners should limit grass turnout and give supplementary feeding in the field, although leaving out wet, rotten hay is inadvisable. They should also fence off areas heavily contaminated by sycamore seedlings and be aware high winds and flood waters can carry seeds to fields without sycamore trees[2].


Sparse grazing can result in inadvertent ingestion of seeds.

Pyrrolizidine alkaloid-containing plants

Senecio jacobaea (ragwort) poisoning is probably the most commonly encountered in horses. Ragwort is not palatable, although sufficient quantities can be ingested if there is limited feeding available or it is in dried fodder. The pyrrolizidine alkaloid metabolites are hepatoxic[1], causing non-specific symptoms including weight loss, mild jaundice, yawning, dysphagia, laryngeal dysfunction, disorientation and hepatic encephalopathy. After ingestion, it may be months before these symptoms are seen.

Clinical presentation, biochemistry and hepatic biopsy are usually diagnostic. Biochemistry will usually show increased gamma-glutamyl transferase, alkaline phosphatase and bile acids, together with hypoalbuminaemia. Greatly increased bile acids are linked to non-survival. Liver histopathology will show hepatic megalocytosis, fibrosis and biliary hyperplasia[1]. Treatment is usually only supportive, together with nutritional support.

Plants containing cardiac glycosides

Digitalis (foxglove)

Horses are less susceptible to *Digitalis* poisoning than ruminants and cases usually occur after hay is contaminated. Toxicity results in acute or subacute cardiac failure and arrhythmias, together with a tucked up appearance, dyspnoea and dehydration. In subacute cases, severe diarrhoea is

often seen. Treatment should involve activated charcoal as an adsorbent, together with fluid replacement therapy.

After electrocardiographic diagnosis, atropine (0.5mg/kg) may be used for arrhythmias and propranolol (5mg doses) for tachycardias[1]. In animals that die suddenly, stomach contents can be assayed for digitalis. Postmortem may show focal myocardial degeneration and pulmonary pathology, although this is usually only apparent if the animal has survived 12 hours from ingestion.

Rhododendron

Rhododendrons are often ingested when animals escape into landscaped gardens. The highest level of cardiac glycosides is found in the leaves. Toxicity will affect the gastrointestinal tract and salivation, colic, diarrhoea and tremors will usually be seen within six hours[5]. This is commonly followed by cardiac irregularities and, occasionally, convulsions. Some animals will recover without treatment, although the remainder become comatose and die. Treatment may not be possible due to the rapidity of death, although activated charcoal, fluids and cardiac rhythm protective drugs can be used.

Taxine-containing plants

Yew trees are often found in landscaped gardens and contain taxine, which is a mixture of alkaloids. The whole plant is toxic, with ingestion of 0.35g/kg to 0.7g/kg bodyweight viewed as poisonous[1]. Most animals exhibit sudden death; rarely, animals will take up to 48 hours to become symptomatic.

If clinical signs are seen, they will include trembling, bradycardia, dyspnoea and gastroenteritis, rapidly followed by death. At postmortem, gastrointestinal inflammation or haemorrhagic lesions of the heart may be identified, although there are often no gross pathological findings[6]. If animals are found after ingestion, before clinical affectation, treatment may be attempted with nasogastric intubation of activated charcoal or magnesium sulphate, followed by fluid therapy to stabilise cardiovascular function. Atropine or norepinephrine have been used for reduction of arrhythmia formation, although atropine may reduce gastrointestinal clearance of the toxin[6].

Plants containing phenols

It is believed tannins and gallotannins within oak, and particularly acorns, are toxic. Toxicosis is seen in spring, associated with buds, while autumnal cases are associated with acorns, although high levels of ingestion are required for poisoning. Poisoning can result in anorexia, colic, tachycardia, diarrhoea, constipation, polyuria and death. Biochemistry may reveal high blood urea nitrogen and urinalysis may show low specific gravity and proteinuria[1]. At postmortem, gastrointestinal oedema can be seen, together with mesenteric oedema, ulcerative enterocolitis and nephrosis. Treatment consists of supportive fluid therapy for days to weeks after ingestion.

Plants containing piperidine alkaloids

Poison hemlocks look like parsnips and cases of poisoning are mostly seen in spring when the plant is more palatable. Poisoning is due to volatile alkaloids, including coniine, contained within them. Plant toxicity increases with maturity and decreases on drying. Approximately 0.2 per cent to 0.8 per cent of bodyweight ingestion of poison hemlock is toxic[1].

Symptoms are usually seen within two hours of ingestion and an initial excitement phase, including tremors, incoordination and salivation, is followed by a depressed phase. Bradycardia, reduced respiratory rate and hypothermia are seen in this second phase. Some animals are found dead without symptoms. Animals either perish within five to 10 hours, or exhibit symptoms for hours to days then recover[5]. Treatment includes sedation and nasogastric intubation with activated charcoal if possible, although this may be unsafe in seizuring horses.

Thiaminase-containing plants

Thiaminases in bracken ferns are a cumulative toxin, requiring high intakes before poisoning occurs. Most cases are associated with young ferns contaminating hay, as the mature ferns are unpalatable. Clinical symptoms are seen weeks after ingestion and include incoordination, muscle tremors, cardiac irregularities and bradycardia. Diagnosis is based on low blood thiamin levels, which can then be successfully supplemented[1].

Tropane alkaloid-containing plants

Tropane alkaloids are substances such as atropine and hyoscine, and are found in plants like deadly nightshade and thornapple[1]. Toxicity affects the autonomic nervous system, resulting in dilated pupils, tremors, restlessness, convulsions and recumbency. Fortunately, poisoning is relatively rare due to the plants' unpalatability.

Steroidal alkaloid-containing plants

Nightshades

Despite their unpalatability, poisoning from the nightshade family (*Solanum* species) of plants is occasionally seen. They result in gastrointestinal irritation and clinical signs include reduced appetite, colic and diarrhoea.

There may also be central nervous symptoms such as depression, dyspnoea, incoordination, collapse, convulsions and death, although these are more commonly associated with subacute poisoning[1].

Potatoes

Potatoes are toxic once they are green and start sprouting, although an intake of more than 50 per cent of the diet is required for toxicity, so this is unlikely in horses[1]. Clinical cases will show depression and recumbency, with hyperaemic gastrointestinal mucosae postmortem.

Hypericin-containing plants

St John's wort is used in human medicine due to its wide range of beneficial effects, although it is toxic to livestock. It dramatically loses toxicity after drying[5], so is less problematic in dried feedstuffs. Horses are less susceptible to this plant than ruminants, although cases of poisoning are seen. It results in photosensitivity, especially of the non-pigmented skin, and treatment involves supportive therapy and avoiding exposure to sunlight. Occasionally, only the eyes will be affected. The photosensitivity may be due to a primary reaction or secondary to hepatic damage.

Protoanemonin

This low-toxicity compound is found in buttercups and cowslips, and results in almost immediate oral and gastrointestinal irritation, so most horses will avoid ingesting the plants. Affected horses require symptomatic, supportive treatment until the irritation has passed[5].

- Some drugs in this article are used under the cascade.

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