PROBLEM WEEDS IN HAY AND FORAGES FOR LIVESTOCK

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ABSTRACT

Although they may only make up a small portion of the cases presented to veterinarians, poisoning cases often require special effort. When food animals are exposed to poisonous weeds, a devastating case with large morbidity and mortality may ensue. The concern is for the herd and economics as much as treatment of the individual animal. In addition, producers and veterinarians must address food safety concerns and handle additional emotion, publicity, and medico-legal issues.

Livestock poisoning associated with the ingestion of weeds in hay and forages can cause sudden death as seen with oleander or nitrate accumulating plants or a chronic disease as observed with the ingestion of pyrrolizidine alkaloid containing plants. Other problems associated with alfalfa include photosensitization and mechanical injury.

Key Words: photosensitization, mechanical irritation, oleander, nitrate, pyrrolizidine alkaloids

INTRODUCTION

Alfalfa is one of the most important forage crops in the United States, but alfalfa itself can lead to disease in animals. The disease problems associated with alfalfa pasture include bloat and an acute respiratory distress syndrome that can be prevented by pasture management and grazing control. Other disease problems associated with alfalfa are rare and include photosensitization and estrogenic effects. Most often, contamination of alfalfa hay with poisonous plants is the cause for disease in livestock. Even though animals are selective about what they eat, there are instances (for example, herbicide applications) that may change palatability or increase toxicity in some plants. If weeds are embedded in alfalfa cubes, animals may not be able to avoid ingestion of the weeds.

Poisonous weeds that have been found in alfalfa include oleander, nitrate accumulator plants, foxtail, bristly oxtongue and pyrrolizdine alkaloid containing plants. Undesirable effects may result from ingestion of large amounts but may also be associated with the ingestion of minute amounts of highly toxic plants present in alfalfa. Awareness of poisonous plants growing in a certain geographical region and their associated clinical signs are instrumental in making a diagnosis and initiate treatment. Most importantly, recognition of poisonous plants in hay or forage may help prevent plant poisonings in animals.

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Photosensitization. Photosensitization associated with consumption of alfalfa or other Medicago species appears to be uncommon, at least in California. The photoactive substance associated with Medicago species is unknown and it is not known why or how it is formed. Photosensitization occurs when phototoxic or photoactive substances accumulate in the skin and interact with sunlight to produce a sometimes severe, crusting, itching or painful dermatitis. The extent of the skin damage is typically limited to the unpigmented skin on an animal that is exposed to sunlight and does not occur on darkly pigmented areas. Local swelling and skin damage can be mild to severe and the affected skin may weep serum and slough or peel. There are several different types of photosensitization related to plant exposures. Primary photosensitization is a result of the direct absorption of photosensitizing agents into the blood and distribution to the skin. The following plants have been associated with this type of photosensitization: *Hypericum perforatum* (St. Johnswort), *Fagopyrum esculentum* (Buckwheat), *Ammi majus* (Bishop's weed), *Cymopterus* spp. (spring parsley), *Heracleum* spp. (cow parsnip), *Lomatium* spp. (wild parsley), *Apium* spp. (celery), *Pimpinella major* (burnet saxifrage), *Ambrosia* spp. (ragweed) and *Medicago* spp. (Burrows et al., 2001). Secondary photosensitization occurs when the liver is damaged by toxins and can no longer excrete phylloerythrin, a photoactive substance produced from ingested chlorophyll. Phylloerythrin accumulates in the blood and tissues and leads to photosensitivity in unpigmented areas that are exposed to sunlight. In affected animals, hepatic disease can be diagnosed by evaluating liver function. Examples of plants leading to secondary photosensitization are: *Agave lecheguilla* (Agave), *Panicum coloratum* (Kleingrass), and *Tribulus terrestris* (puncture vine, McDonough, SP et al., 1994). The ingestion of moldy alfalfa hay has caused secondary photosensitization in cattle (Scruggs et al., 1994). The nature of the hepatic changes and the association of the disease with water-damaged alfalfa hay were implicated with a yet unidentified mycotoxin. Horses grazing clover pastures that were infested with a fungus (*Cymodethea trifolii*) developed edema, erythema, vesiculation and necrosis of the light pigment areas. *Cymodothea trifolii* is a fastidious fungus that causes black blotch or sooty blotch disease of clover and alfalfa. Optimum conditions for growth are greater than 80% humidity and 20 - 25 C. The horses had liver dysfunction leading to photosensitization and the clover pasture infested with *Cymodothea trifolii* was considered a likely cause. Alfalfa (*Medicago sativa*) and related *Medicago* spp. are listed among the many plants having photosensitizing potential.

In California, several severe outbreaks of photosensitization in horses occurred in the summer of 2004. An estimated 70 horses were affected and all horses were fed newly purchased alfalfa hay beginning several days prior to the onset of clinical signs. Hay samples were thoroughly inspected for contamination with potentially toxic weeds and analyzed for a variety of toxins, but none were found. A phototoxicity assay was performed on three hay samples, but was negative. A feeding study with three primarily white horses was conducted. Two horses developed skin lesions in the face at 14 days after being fed the suspect alfalfa hay. A biopsy was taken and confirmed photosensitivity. Based on the results of the feeding study, it was concluded that the alfalfa hay contained a phototoxic compound that caused the outbreak of photosensitization. The phototoxic compound is yet to be determined.

PROBLEM WEEDS

Mechanical Irritation: Plants with sharp and barbed bristles or spines have been associated with disease in animals. The resulting mechanical injury is a particularly serious problem in horses. Lesions are often seen in the mouth and they will cause pain and result in reluctance to eat. Deep ulcerations of the tongue, gums, and cheeks, which may lead to abscesses, may also be noticed. Recently, bristly oxtongue (*Picris echioides*) was identified in alfalfa hay and resulted in disease in horses. *Picris echioides* is in the Asteraceae family and resembles sowthistle (*Sonchus* spp.) in general appearance. Bristly oxtongue is a broadleaved biennial weed that is distributed throughout California. Mature plants are 2 to 3 feet (60 - 90 cm) tall with angled stems and covered with harsh, barbed, stiff hairs and yellow flower heads. Although there are no published reports of toxicity associated with the consumption of bristly oxtongue, the spiny character of the plant was considered the cause for mechanical irritation in the affected horses. Over the past few years, exposure of horses to alfalfa hay contaminated with *Setaria* spp. has resulted in mechanical irritation. Horses developed blisters and lesions in the mouth and ulcerations on the tongue and lips. The alfalfa hay contained large amounts of two different species of bristlegrass: *Setaria viridis* (green foxtail) and *Setaria glauca* (yellow foxtail, yellow bristlegrass, bristly foxtail). Both species have sharp and barbed bristles, particularly *S. glauca*. The bristles are capable of penetrating the mucous membranes and causing serious erosions of the mouth. Problems usually occur when the grass is cut late and a substantial number of panicles are present. Although mainly a problem in horses, the mechanical injury has also been reported in cattle (Fava et al., 2000). The bristles may be present in the wounds and need to be removed, if possible. If alfalfa hay is contaminated with bristlegrass, it should not be fed to livestock animals. It may be necessary to have a veterinarian evaluate all animals and initiate treatment, if necessary.

Oleander: In California, the potential for exposure of animals to oleander is high. Recently, oleander exposure resulted in the acute death of six dairy cattle while there was concern that other cows were exposed to the toxic plant but did not die. While oleander poisoning can be quickly diagnosed, there are concerns regarding the transfer of oleandrin into milk and meat. There is only limited data available regarding the distribution of oleander toxins and research is underway to address this important issue. Oleander (*Nerium oleander*) is an ornamental, evergreen shrub that is very drought tolerant and can survive unattended in pasture lands where livestock graze. However, ingestion from clippings or dried leaves is the most common cause of oleander poisoning in animals. Modern agricultural practices, like crimping of hay can create a greater risk for oleander contamination from leaves blowing into the fields before harvest. In the most recent case in dairy cows, it was assumed that the hay added to the total mixed ration (TMR) was contaminated with oleander leaves.

The toxicity of oleander results from cardiac glycosides, with oleandrin being the most prominent one. The toxins are present in all parts of oleander, and toxicity is retained with drying. The leaves and flowers have the highest concentrations of toxic cardenolides. 10 –20 medium-sized leaves can be lethal to a horse, while as few as 8 leaves can cause death in a cow (Galey et al., 1998). Clinical signs of oleander poisoning include diarrhea, excess salivation, depression, and anorexia. As the disease progresses, the animals develop a variety of cardiac signs, including bradycardia and arrhythmias. At this stage of the disease, the animals may also

show tremors and difficulty breathing. However, often the disease progresses so rapidly that the animal is found dead and clinical signs are not observed. Diagnosis of oleander poisoning has improved significantly since the development of several specific analytical methods (Tor et al., 2005). Suspect contaminated plant material can also be analyzed for oleandrin to prevent fatal oleander poisoning in livestock.

Nitrate accumulators: Nitrate poisoning is an acute disease in ruminants, especially cattle. Clinical signs of acute nitrate poisoning include depression, respiratory distress, tremors, ataxia, rapid heartbeat, and terminal convulsions. Death may occur within 6-24 hours of ingestion. Diagnosis is based on appropriate clinical signs and laboratory analysis of nitrate and nitrite in serum, blood, ocular fluid, rumen contents, and forage. Toxic nitrate concentrations can be found in common crop and pasture plants as well as weeds. The most common nitrate accumulating weeds identified in alfalfa hay are pigweed (*Amaranthus retroflexus*) and lamb's quarters (*Chenopodium* spp.). Among crop plants, especially oat hay and sorghum have been incriminated with nitrate toxicosis, but alfalfa itself may contain potentially toxic nitrate concentrations. Nitrate accumulates in the vegetative tissue, particularly in stems with less in the leaves (Bedwell et al., 1995). Heavy fertilization of pastures, herbicide treatment, drought, cloudy weather, and decreased temperature may increase the nitrate concentrations in plants. Forage nitrate levels of 0.3% and above are potentially dangerous, with acute poisoning likely to occur if the nitrate level exceeds 1%. Forage management techniques can affect the concentration of nitrate and can reduce the risk. Careful use of nitrogen fertilizers, harvest under appropriate conditions, supplementation of ration with corn, ensiling, and testing hay and forage for nitrate content are approaches to minimize the risk of nitrate poisoning in animals.

Pyrrolizidine alkaloids: Pyrrolizidine alkaloid (PA) poisoning can result in chronic, irreversible liver disease in animals (Cheeke, 1998). Cattle and horses are most susceptible to the toxic effects of PAs. Sheep, goats and small herbivores (e.g. rabbits, guinea pigs, hamsters) are resistant to PA toxicity due to detoxification processes in the liver (Cheeke, 1994). Clinical signs of chronic PA poisoning may not appear for 2-8 months after the first ingestion of PA containing plants. Affected animals lose condition, and develop icterus. Cattle may also develop photosensitization. In horses, neurological signs are commonly observed (Schmitz, 1998). In the US, PA poisoning is mostly associated with the exposure to *Senecio* spp. (groundsels and ragworts), but other plant genera such as *Amsinckia* and *Cynoglossum* spp. can also result in toxicosis. *Senecio vulgaris* (common groundsel) is commonly found in alfalfa fields in California, but is also widely distributed along the East Coast and Canada. New plantings of alfalfa and alfalfa weakened by heavy weevil infestations are more susceptible to competition by common groundsel. Identification of PA-containing weeds in alfalfa and detection of PAs in forage are important to prevent poisonings. While there is some degradation of PAs in silage, the PA content of hay remains constant over many months. Silage contaminated with more than 5% *Senecio* spp. is considered unsafe for cattle or horse feeding (Candrian et al., 1984).

SUMMARY

Livestock animals are at the mercy of toxic plants when these plants are present in feeds or hay. Hay and feed should be carefully inspected for contamination with potentially toxic weeds before the first feeding and owners should establish excellent working relationships with

suppliers and growers. Producers and farm managers, along with veterinarians and diagnosticians play important roles, and all contribute information that may be important to a poisoning case. Once all the information is available, all evidence is collected, and proper sampling of specimens has occurred, a summary of findings can be provided and will be instrumental in preventing reoccurrences.

REFERENCES

Bedwell CL, Hamar DW, Hoesterey ML et al.: 1995, Comparison of four methods for forage nitrate analysis. J Vet Diagn Invest 7:527-530.

Burrows GE, Tyrl RJ: 2001, Apiaceae. **In:** Toxic Plants of North America. 1st ed., pp. 43-47. Iowa State University Press, Ames, IA.

Candrian U, Luthy J, Schmid P, et al.: 1984, Stability of pyrrolizidine alkaloids in hay and silage. J Agric Food Chem 39:930-933.

Cheeke PR: 1998, Natural toxicants in feeds, forages, and poisonous plants. 2nd ed., pp.338-352. Interstate Publishers, Inc., Danville, IL.

Cheeke PR: 1994, A review of the functional and evolutionary roles of the liver in the detoxification of poisonous plants, with special reference to pyrrolizidine alkaloids. Vet Hum Toxicol 36:240-247.

Fava E, Rossi F, Speranzini G et al.: 2000, Enzootic ulcer in the back of the tongue in cattle after ingestion of hay containing flower clusters of yellow bristle-grass. Dtsch Tierarztl Wochenschr 107:351-354.

Galey FD, Holstege DM, Johnson BJ et al.: 1998, Toxicity and diagnosis of oleander (*Nerium oleander)* poisoning in livestock. **In:** Toxic Plants and other Natural Toxicants. Garland T, Barr AC (eds), pp. 215-219. CAB International, New York, NY.

McDonough SP, Woodbury AH, Galey FG et al: 1994, Hepatogenous photosensizitation of sheep in California associated with ingestion of *Tribulus terrestris* (puncture vine). J Vet Diagn Invest 6:392-395.

Schmitz DG: 1998, Toxicological problems. **In:** Reed SM, Bayly WM (eds). Equine Internal Medicine, pp. 981-1042. WB Saunders, Philadelphia, PA.

Scruggs DW, Blue GK: 1994, Toxic hepatopathy and photosensitization in cattle fed moldy alfalfa hay. J Vet Med Assoc 204:264-266.

Tor ER, Filigenzi MS, Puschner B: Determination of oleandrin in tissues and biological fluid by liquid chromatography-electrospray tandem mass spectrometry. J Agric Food Chem 2005; 53:4322-4325.