TOXIC WEEDS IN HAY AND THE EFFECTS ON LIVESTOCK

Birgit Puschner

ABSTRACT

Eliminating toxins from the hay begins in the field. However, toxic weeds frequently end up in hay and forage and pose a serious risk to livestock. The effects of poisonous plants on animals depend on the amount ingested and the individual weed present in hay. Many indications for poisoning exist. Obvious cases involve sudden onset of disease in a number of animals. Finding of common feed or environmental conditions will further support a suspicion of poisoning. A toxicosis is also suggested in the animal that is found "suddenly dead". Some toxic weeds may result in acute poisoning while others have to be ingested over a period of weeks to months before clinical signs of illness develop.

Most plant poisonings have no specific treatments and thus, are best prevented by avoiding exposure to toxic plants in the first place. Awareness of poisonous plants growing in a certain geographical region and their associated clinical signs are instrumental in making a diagnosis, and initiating treatment. More importantly, the recognition of poisonous plants in hay or forage may help prevent plant poisonings in animals. It is often prudent to consult with a veterinary toxicologist at the earliest stage of a case, especially if one is available in your proximate area. Many potential poisons are unique to a geographic location, or a specific time of year. The veterinary toxicologist is uniquely qualified to discuss differential diagnoses, sampling techniques, and potential treatment options.

Alfalfa hay has been contaminated with poisonous plants and has resulted in illness and death of animals. The presence of nitrate accumulators, pyrrolizidine alkaloid-containing plants, summer pheasant's eye, foxtail, and oleander are discussed as these weeds are most commonly associated with alfalfa hay. In addition, spoiled forage or the presence of decomposed animal carcasses in hay can result in botulism and blister beetles have been found in alfalfa hay resulting in disease in cattle.

Key Words: alfalfa, nitrate, pyrrolizidine alkaloids, summer pheasant's eye, bristlegrass, oleander, botulism, blister beetles, toxicosis

INTRODUCTION

Plant poisonings can have a tremendous impact on profitability of livestock production. Hay growers, producers and farm managers, along with veterinarians and diagnosticians play important roles in recognizing plant poisonings and in preventing reoccurrences. Despite our knowledge of the high nutritional quality of alfalfa hay as animal feed, it may contain

B. Puschner, DVM, Ph.D., Diplomate of the American Board of Veterinary Toxicology; California Animal Health and Food Safety Laboratory System, University of California, West Health Sciences Dr., Davis, CA 95616; Email: bpuschner@ucdavis.edu. **In:** Proceedings, National Alfalfa Symposium, 13-15 December, 2004, San Diego, CA, UC Cooperative Extension, University of California, Davis 95616. (See http://alfalfa.ucdavis.edu for this and other proceedings).

compounds that may inadvertently affect animals. Even though alfalfa hay is primarily used as animal feed for dairy cows, it is also included in rations for beef cattle, sheep, goats, chickens, turkeys, horses, and other farm animals.

Nitrate accumulators: Ruminants, and especially cattle are most susceptible to nitrate poisoning because of the reduction of nitrate to nitrite by ruminal microorganisms. There are a number of common crop and pasture plants, and weeds that can accumulate toxic nitrate concentrations. Among weeds, pigweed (*Amaranthus* spp.), lamb's quarters (*Chenopodium* spp.) and nightshades (*Solanum* spp.) have been found to contain nitrate at a potentially toxic concentrations (Casteel et al., 2004). Among crop plants, especially oat hay, corn and sorghum have been associated with nitrate toxicosis, but alfalfa itself may contain potentially toxic nitrate concentrations. Conditions such as heavy fertilization of pastures, herbicide application, drought, cloudy weather, and decreased temperature result in increased concentrations of nitrate in the plant. In addition, nitrate accumulates in the vegetative tissue resulting in highest concentrations in lower stalks and stems. Leaf material and grain or seeds generally do not contain toxic nitrate levels. Forage nitrate levels of 0.3% (3,000 ppm) and above are potentially dangerous, with acute poisoning likely to occur if the nitrate level exceeds 1% (10,000 ppm). Clinical signs of acute nitrate poisoning in ruminants are a result of the formation of the nitrite ion, which oxidizes hemoglobin to methemoglobin and ultimately results in anoxia (Osweiler et al., 1985). Methemoglobinemia is apparent as dark brown or chocolate-colored blood and is a common feature of nitrate/nitrite poisoning. Clinically affected animals become depressed, have difficulty breathing, and develop tremors, ataxia, a 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. Forage management techniques can affect the concentration of nitrate and can reduce the risk. While drying does not alter the nitrate concentration, ensiling of forages results in a large reduction of nitrate within 1 to 2 months. Careful use of nitrogen fertilizers, harvest under appropriate conditions, supplementation with high-carbohydrate feed, testing hay and forage for nitrate content and providing a feed additive are additional approaches to minimize the risk of nitrate poisoning in animals (Casteel et al., 2004).

Pyrrolizidine alkaloids: Plants containing pyrrolizidine alkaloids (PAs) are found throughout the world. PA poisoning is of great economic importance as a cause of progressive liver disease in livestock, wildlife and humans. The disease has been reported from most areas of North America and is mostly caused by plants from the genus *Senecio,* but other plant genera such as *Amsinckia, Cynoglossum, Echium and Heliotropium* also contain the toxic alkaloids. Within the *Senecio* genus, *Senecio vulgaris* (common groundsel) and *Senecio jacobaea* (tansy ragwort) are common weeds in hayfields in California and are also widely distributed along the East Coast and Canada. PA-containing plants are usually unpalatable and animals avoid them. However, animals are often not able to differentiate the toxic weeds in hay and ingest the toxic alkaloids. PAs are hepatotoxic, causing irreversible liver damage (Cheeke, 1998). Horses and cattle are the major livestock species poisoned by 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). PA poisoning is usually a chronic disease and clinical signs may not appear for 2-8 months after the first ingestion of the toxic plants. Affected animals lose condition, and develop icterus. Cattle may also develop photosensitization. Neurological signs are commonly seen in horses, and the

condition is called "walking disease". After the onset of clinical signs, the prognosis is poor. The presumptive diagnosis is based on clinical signs, and gross as well as histologic lesions (Hopper, 1978). Identification of PA-containing weeds in alfalfa and detection of PAs in forage are important to establish an accurate diagnosis. However, due to the prolonged delay in onset of clinical signs the affected hay that was eaten weeks or months before may no longer be available for evaluation. Prevention is the best control measure. Careful inspection of feed is important and contaminated feed should be discarded or fed to less susceptible species. 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).

Summer Pheasant's eye: Recently, horses have died as a result of eating grass hay contaminated with *Adonis aestivalis* also known as summer pheasant's eye or summer Adonis (Woods et al., 2004). Summer Adonis has long been utilized in European folk medicine for its cardiac-enhancing effects. Summer pheasant's eye was introduced into North America as a horticultural plant, escaped cultivation and is now naturalized in the western United States (Burrows et al., 2001). *Adonis* spp. are members of the Ranunculaceae family, and are considered unpalatable which is most likely the reason why reports of poisoning are rare. However, adonis poisoning has not only been reported in horses, but also in pigs (Davies et al., 1989) and sheep (Hurst, 1942). *Adonis* spp. contain a series of cardiotoxic compounds, similar to the toxins found in oleander. Leaves and flowers have the highest concentrations of toxins. Horses poisoned with summer pheasant's eye developed colic, hemorrhagic enteritis, diarrhea and decreased gut motility. In addition, heart arrhythmias are common clinical findings that can result in death. Poisoning of livestock with summer Adonis is becoming more likely through contamination of fields harvested for animal feed. The plant may become better established in the western regions of North America and may be more commonly found in hay. Recognition of summer Adonis in hay is instrumental in preventing poisoning. In addition, diagnostic procedures of a suspect Adonis poisoning case in livestock include pathological and toxicological examination. Susceptibility of cattle to Adonis is unknown, but cases involving cardiotoxicity need to be carefully evaluated for the presence of Adonis in hay or tissues (Filigenzi et al., 2003).

Oleander: Oleander (*Nerium oleander*) is extremely toxic to all animals. It is an ornamental, evergreen shrub that is very drought tolerant and often able to survive unattended in pasture lands where livestock graze. Oleander is also cultivated widely in the southern United States and ingestion of clippings or dried leaves from the plant is a common cause of poisoning (Galey et al., 1996). The dried plant is less bitter and more palatable to animals. Modern agricultural practices, like crimping of hay can create a greater risk for oleander contamination from leaves blowing into the fields before harvest. The toxicity of oleander results from cardiac glycosides, with oleandrin being the most prominent one. Cardiac glycosides result in cardiac arrhythmias and death is typically due to ventricular fibrillation. All parts of the plant are toxic and toxicity is retained with drying. The leaves and flowers have the highest concentrations of toxic cardenolides. As few as 7 average-sized leaves can be lethal to a horse, while as few as 12 leaves can cause death in cattle (Galey et al., 1998). Animals exposed to oleander are often found dead. If found alive, the animals may have colic, excessive salivation, depression, and anorexia. The poisoning progresses rapidly as affected 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. Oleander leaves may be present in the gastrointestinal contents, but identification is not always possible. Every effort should be made in a suspect oleander poisoning case to reach an accurate diagnosis so that adequate therapeutic measures can be initiated. In addition, the source of oleander has to be identified and removed from animals. Diagnosis of oleander poisoning has improved significantly since the development of several specific analytical methods (Tor et al., 1996; Puschner et al., 2001). Confirmation of the toxic glycoside oleandrin in biological samples, such as serum, urine and gastrointestinal contents provides invaluable information to the clinician in directing the clinical course and initiating adequate treatment in confirmed, non-fatal poisoning cases. Suspect contaminated plant material can also be analyzed for oleandrin to prevent fatal oleander poisoning in livestock. Animals should be denied access to areas that contain oleander, especially if dried leaves or clippings are present.

Bristlegrass: Exposure to *Setaria* spp (bristlegrass) is commonly seen in horses. Affected horses develop blisters and lesions in the mouth and ulcerations on the tongue and lips. In many of the diagnosed cases, alfalfa hay was contaminated with large amounts of *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 is present. The mechanical injury is a particularly serious problem in horses, but has also been reported in cattle (Fava et al., 2000). The lesions in the mouth will cause pain, resulting in a reluctance to eat and a loss of weight. The bristles can cause deep ulcerations of the tongue, gums and cheeks, which may result in abscesses. The bristles may be present in the wounds and need to be removed, if possible. Careful inspection of alfalfa hay is recommended and if bristlegrass is identified, the hay should not be fed to livestock animals.

Blister beetles: The disease caused by blister beetles is a result of exposure of animals to cantharidin, a strong irritant that can result in a severe digestive and urinary tract disease. Clinical signs include colic-like signs, salivation, oral ulceration, acute downers, polyuria and unexplained death. Cantharidin toxicosis is most commonly seen in the southern United States, but can occur anywhere because of the transport of alfalfa hay. Cantharidin is heat-stable and thus processed alfalfa, such as pellets or cubes can also contain the toxic compound. Cantharidin can be present in hay after the bettles have been crushed. Studies have demonstrated that blister beetles often contain between 1 - 5 % of cantharidin (on a dry weight basis). Beetles from the genus *Epicauta* are most frequently associated with cantharidin toxicosis and are especially common in Texas and Oklahoma (Stair et al., 2004). However, in California a different genus of beetles was recently identified in alfalfa hay, called *Tegrodera*. Three species of *Tegrodera* are known and have been found in the Owens Valley, in the San Bernadino area, along the Colorado River and in the Imperial Valley. Alfalfa hay from Inyo County contained *Tegrodera latecincta* and contained potentially toxic cantharidin concentrations. Even though cantharidin poisoning is more common in horses, cattle, sheep and goats are occasionally poisoned. Blister beetle poisoning in cattle can result in significant economic losses because of a substantial drop in milk production, animal illness and death. Blister beetles concentrate in alfalfa fields during blooms to feed on pollen and plant nectar. Modern agricultural practices such as cutting and crimping

create a greater risk for toxicosis preventing escape from beetles. First-cutting hay generally is least risky and later cutting of alfalfa hay should be harvested in the vegetative stage.

Botulism: Ingestion of preformed toxin associated with carcasses or decayed organic matter is most common in cattle and horses. Thus, exposure to poorly ensiled small grain haylages, carcasses, poultry litter, or bones from dead animals may result in exogenous botulism poisoning (Rings, 1987). If alfalfa hay or hay cubes are contaminated with a carcass, botulism toxin may be present. The classical clinical syndrome of botulism is progressive flaccid paralysis (Galey, 2001). In cases in which large numbers of animals are affected, the feed source is usually contaminated. Botulism is usually a clinical diagnosis and supported by a thorough investigation to rule out the many other causes that result in clinical signs similar to botulism. It is very difficult to establish a definite diagnosis of botulism as laboratory tests are often not sensitive enough. As with many other toxicoses, prevention is the best way to deal with botulism. Forage should be examined for evidence of decaying carcasses or other foreign matter. A rodent control program reduces the number of rats and mice in stored feeds.

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