Poisonous Plants in Harvested Feeds

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There are hundreds of plants with potential toxicity to cattle. This presentation is limited to a few that are likely to cause problems in harvested feed. Many livestock producers now buy feed from those who specialize in crop production, thus they may have minimal control over the quality of the feed.

Modern methods of feed preparation (grinding, pelleting, and cubing) may make it impossible to ascertain the presence of poisonous plants unless quality control is rigidly practiced from field to animal.

Nitrite Poisoning

High levels of ingested nitrates of plant origin may be reduced in the rumen and be absorbed as nitrites. Nitrites convert hemoglobin to methemoglobin which renders the molecule nonfunctional for oxygen transport.

Nitrite poisoning, both acute and chronic, may be the most economically important plant poison found in harvested feed. This is especially true in areas where forage crops are pushed for maximum production by heavy fertilization. In California, sudan grass *(Sorghum vulgare var sudanensis)* may be involved with both cyanide and nitrite poisoning. Actually, nitrite poisoning is the more likely to occur because of the nitrogen applied to get maximum yields. Sudan grass is fed as green chop or as hay. In either case accumulated nitrates may cause poisoning if the right set of circumstances prevail.

A number of conditions affect the levels of nitrate in the plant. First, the actual content of nitrate in the soil: all nitrogen sources applied to the soil ultimately are converted to nitrate. This becomes the utilizable nitrogen ion available to the plant; nitrate is absorbed through the root system. Secondly, adequate moisture is necessary to promote soil nitrate formation and subsequent plant absorption. Thirdly, under certain circumstances drought conditions favor an accumulation of nitrate. This may be occurring at very specific growth stages of a plant. Fourthly, plant species vary considerably in their ability to accumulate nitrates.

Plants vary in ability to absorb nitrates at different growth stages. The preblooming stage is a time of high accumulation. With maturity, nitrate absorption usually diminishes.

The stems usually have more nitrate than the leaves, and the lower parts of the plant have more also. This is a natural consequence of the movement of nitrate through the root system, up the stem and into the leaves.

During the process of transporting nitrate to the leaves, the nitrates are acted upon by an enzyme, nitrate reductase (N.R.), which converts the nitrate to nitrite to ammonia to amino acids and protein in the plant. If conditions are not favorable to the conversion of the nitrate to ammonia there will be an abnormal accumulation of nitrate in the plant and this will primarily be within the stem.

Factors which tend to lower the level of nitrate reductase favor the accumulation of nitrate. Various nutrient deficiencies (molybdenum, sulphur and phosphorus) will contribute to diminished nitrate reductase activity. Certain climatic conditions, such as low temperature, inhibit enzyme action. A certain amount of light is required to maintain enzymatic action. Thus, continued cold and/or persistent shade may favor the accumulation of nitrate.

Herbicides recently applied to an area to be harvested may favor the accumulation of nitrates, because the plant ceases to grow and go through its normal metabolic processes, although it is still absorbing nitrates.

Ruminant species are more susceptible to nitrite poisoning because ruminal microorganisms favor nitrate reduction. Any condition which upsets the normal rumen microflora and fauna may facilitate reduction of nitrite, hence poisoning. These conditions might include worming, long-term antibiotic therapy or indigestion. These then might be contributory to the development of nitrite poisoning on an otherwise satisfactory diet.

It is important to recognize that on a good plane of nutrition, eating feed containing high levels of carbohydrates and good quality proteins, cattle can tolerate much higher levels of nitrate in the diet. Animals on full feed could consume a diet with 2 to 5% nitrate in the diet as long as they are on a good production or fattening ration. If that same forage was fed to stocker animals on a marginal diet, toxicity could result.

Besides sudan grass, other weeds may contaminate hay. I've seen baled hay containing 50-70% lambsquarter *(Chenopodium album)* with a KNO_3 content of 6%.

Truck-garden trimmings (lettuce, celery, brocolli) may also be high in nitrates. Many forage crops and weeds are capable of accumulating nitrates under special circumstances.

I needn't dwell on the clinical syndromes associated with nitrite poisoning, nor the therapy which are standard clinical procedures. There may, however, be differential diagnostic problems.

Consider a situation in which sudan grass is being fed as green chop to cattle. How does one differentiate between cyanide and nitrite poisoning? A venous blood sample should provide a quick, accurate test. The venous blood from a cyanide case is bright red while that of a nitrite case is dark or even brownish.

Differential diagnosis between nitrite and cyanide poisoning becomes crucial when treatment is considered. If one uses the standard cyanide therapy (Na nitrite and Na thiosulfate) and the animal is actually suffering from nitrite poisoning, the result is obvious.

Nitrite blood may appear like that from an animal near death from pneumonia or other anoxic conditions. The difference is that anoxic venous blood will absorb atmospheric oxygen and become red with five minutes' exposure. Nitrite blood will not change color.

The question most frequently asked is "What levels of nitrate in the plant will cause poisoning?" There is no pat answer. A critter on a marginal diet may be affected by levels as low as 0.5% KNO_3. A dairy cow on a high carbohydrate intake may ingest levels above 2% KNO_3 without harm.

Cyanide Poisoning

Classical cyanide poisoning is known to every cattle practitioner. There are two situations in which cattle might be poisoned with cyanide in harvested feeds. The first is in green chop. If sudan or sorghum is used in the fall of the year after the first frost or early in the season before frosts cease one could get a change in the glycoside composition in that harvested feed.

Arrow grass *(Triglochin maritima)* grows in wet meadows that may be harvested for hay. The glycoside in this plant is not detoxified during the drying process as occurs when sudan grass is harvested as hay.

Clinical signs of cyanide and nitrite poisoning are similar.

It should be remembered that the effect of cyanide on the tissue is that of inhibition of the cytochrome oxidase system at the cellular level. This interference occurs in all tissues, including the brain. There is localized cerebral anoxia resulting in CNS signs such as convulsions and tetanic spasms.

Pyrrolizidine Alkaloid Poisoning

Next to nitrite poisoning pyrrolizidine alkaloid poisoning is California's most important plant problem, especially in hay.

There are over 50 pyrrolizidine alkaloids which have been identified chemically. All are potentially dangerous, but only a few are of clinical significance.

Pyrrolizidine alkaloids are produced by five or six plant species but only two species are important for our consideration in harvested feeds: fiddleneck or fireweed *(Amsinckia intermedia)* and common groundsel *(Senecio vulgaris)*. Both of these plants are common weeds of hayfields in California.

New plantings of alfalfa may be overgrown with the weeds. The first cutting could be 10-50% weeds. Stands of alfalfa that are heavily infested with weevil may allow weeds to take over. Oat hay may be contaminated with these weeds also.

There may be a marked variation in the alkaloid content of plants from season to season and during different growth stages.

Pathogenesis of the lesion: pyrrolizidine alkaloids damage hepatocytes by preventing normal cell division. With the failure of mitosis, the cell continues to grow up to 4-10 times its normal size. Terms used to describe the lesions are hepatocytomeglia or karyomeglia.

Once the cell is affected it continues to grow until it outgrows its nutrition and ultimately dies. Most of the cells in the liver may be affected by one exposure to the alkaloid. When sufficient cells are necrosed, the liver ceases to function and a typical hepatic insufficiency syndrome will develop.

Clinical Signs: The clinical signs manifested in pyrrolizidine alkaloid poisoning cannot be differentiated from infectious hepatitis or other hepatopathies caused by other plants. The clinical signs in cattle may be more subtle than in horses because the CNS component so typical of the horse with hepatopathy may be subdued. Cattle tend to the hepatic coma form. If the animal attempts to walk it is usually incoordinated. Abnormal stances may be assumed as it seeks to balance itself or as it walks. Under certain circumstances cattle push their heads into a corner or up against a solid wall. Other signs less commonly seen include tenesmus and hepatic coma. The animal may become more bright and alert and may even stand again after this coma period has passed.

There are some important facts that must be borne in mind when considering pyrrolizidine alkaloid poisoning.

1. The clinical effects are not usually seen until two to eight months following an exposure. This makes it extremely difficult to obtain a proper history and piece the puzzle together.
2. Young, rapidly developing animals are more susceptible to the effects of pyrrolizidine alkaloids than more mature animals.
3. Pyrrolizidine alkaloids cross the placental barrier and can affect the developing fetus.
4. Pyrrolizidine alkaloids are secreted in the milk of cows that are actively consuming plant material containing the alkaloids.

This then makes it possible for human involvement. I'm aware of one instance in the eastern United States where pyrrolizidine alkaloid poisoning was identified in a small home dairy. The family had been drinking the milk from this group of cows for a long period of time. We are not yet aware of the clinical incidence in the humans but it is too early to tell for sure as just a few months have passed.

Pathology: Pyrrolizidine alkaloids produce characteristic lesions. The hepatocytomeglia, bile duct proliferation and fibrosis are almost pathognomonic.

The extent of each lesion is dependent upon the degree of exposure both in terms of dose and repeated intake.

The only other hepatopathy that approximates the lesions of pyrrolizidine alkaloid poisoning is aflatoxicosis. There may be hepatocytomeglia to some degree, but pathologists tell me that the two lesions are easily differentiated.

Many clinicians have the impression that pyrrolizidine alkaloid poisoning always produces marked fibrosis resulting in a cirrhotic liver. Not necessarily so. Some livers will be atrophic due to loss of the parenchyma. The degree of fibrosis is dependent on the rate of necrosis of hepatocytes. A single massive dose of the alkaloid will produce more fibrosis than a low, prolonged exposure.

Clinical pathology: Hemograms and serum chemistries are similar to those of any hepatopathy. However, serum enzymes may not be elevated. B.S.P. clearance should be increased. Liver biopsy is the most precise diagnostic tool and could be used to screen a group of cattle which has been exposed.

In a differential diagnosis one must consider the other central nervous system diseases such as encephalitis, rabies, hemorrhages of the brain, tumors, and abscesses. Aflatoxicosis must also be considered.

Treatment of pyrrolizidine alkaloid poisoning is non-existent. Once the clinical signs have developed, the liver is already damaged beyond repair and the animal will succumb to the effects of the disorder.

Mycotoxicosis

Table 1 lists a group of mycotoxins that may affect cattle.

The two major problems are ergotism and aflatoxicosis. The clinical manifestations and lesions are well known to cattle practitioners. Perhaps the important point is to recognize that new syndromes and their toxins are being identified. Our knowledge of mycotoxins in medicine is in infancy. Be alert to the possibilities.

Oleander Poisoning

Oleander poisoning is caused by the ingestion of any part of the ornamental shrub called *Nerium oleander*. It is found commonly in the warmer or subtropical climates of the United States. It may be grown as a houseplant ornamental throughout the United States.

This is an extremely hazardous shrub. The leaves can be found in harvested feeds if the shrub is grown as a hedgerow and the leaves gain access to areas that are raked into hay windrows. The poisoning is more likely to occur when cuttings are placed in an area where cattle customarily feed.

The poisonous principle found in oleander is a cardiac glycoside. This glycoside is similar in effects to digitalis, but the effects are more lasting and generally more toxic. The clinical syndrome associated with cardio-active glycoside is rather characteristic but the signs may lead one to false conclusions because the first sign that will be noticed clinically is diarrhea, which may be hemorrhagic. There may be vomiting. The cattle may become dyspneic from circulatory difficulties. There may be weakness and ataxia, associated with anoxia and cyanosis.

If the heart is auscultated carefully, over a period of time almost every type of cardiac conduction irregularity will be noted; heart blocks, dropped beats, tachycardia, bradycardia, cardiac stoppage or ventricular fibrilations.

The extremities may be cold and the general body temperature may be either normal or low. The pulse may be rapid and weak or it may be slow. The toxic effects from oleander would not likely occur until a number of hours had passed subsequent to the ingestion of the leaf.

Table 1: Mycotoxicoses of Cattle
(Modified from Buck, et al.: Clinical and Diagnostic Veterinary Toxicology)

Fungus	Common Substrate	Disease Produced	Toxins
Aspergillus flavus	Cottonseed, corn, peanuts, sorghum	Aflatoxicosis; hepatotoxicosis and hepatic carcinogenesis; cholangiohepatitis, hemorrhage, slow growth	Aflatoxins B_1, B_2, G_1 G_2, M_1
Claviceps purpurea	Ovary of rye and cereal grains	Dry gangrene of extremities; hypogalactia in lactating sows; may be convulsions, ataxia, tremors	Ergotamine and related alkaloids
Claviceps paspali	Dallis grass	Paspalum staggers; ataxia, tremors, nervousness, convulsions; recovery in 5-10 days	Ergotamine and lysergic acid
Fusarium nivale	Fescue hay	Fescue foot; gangrene of the extremities	Butenolide
Fusarium tricinctum	Corn	Diarrhea, milk reduction, weight loss; possible dermonecrosis, gangrene	Diacetoxyscirpenol
Rhizoctonia leguminicola	Clover hay and pasture	Slobber factor; histamine-like disease, excessive salivation, lacrimation, diarrhea, bloat	Slaframine
Penicillium cyclopium	Corn, silage, feed	Tremors, convulsions, ataxia; possible smooth muscle stimulation also	Tremorigen
Penicillium rubrum	Corn	Hepatotoxicity, generalized hemorrhage	Rubratoxin

The lethal dose for oleander is 50 mg per pound of body weight of either the green or the dry leaf. This is an extremely lethal plant, 50 gms of leaf (less than a handful), is sufficient material to kill a thousand-pound cow. The presence of one or two leaves in the rumen contents would be sufficient grounds for diagnosis of oleander toxicity.

Treatment of oleander toxicity is not very satisfactory. There are essentially no drugs that will counteract the effects, at least in cattle. There are substances that have been used in man for digitalis poisoning, but their clinical usefulness in cattle has not been demonstrated. If oleander poisoning is diagnosed in valuable animals, I would recommend that a rumenotomy be done. Completely wash out the rumen and re-inoculate the rumen with contents from a normal animal. I cannot stress too strongly the fact that just a few leaves are capable of killing cattle from oleander toxicity.

Milkweed poisoning

There are numerous species of plants which exude a milky juice when the stem is cut. Not all of these are poisonous. However, members of the genus asclepias do contain toxic resins and glycosides. These plants may be found in harvested hays and are toxic in the dry state. Milkweeds are cosmopolitan in distribution throughout the United States. Different species have different habitats but all must be considered to be potentially poisonous.

The clinical syndrome associated with milkweed poisoning may take either a gastroenteric or a nervous form.

The nervous form is characterized by depression and anorexia, the animal is apathetic, there is usually weakness with loss of muscular control as evidenced by knuckling at the fetlock and falling. There may also be dilatation of the pupil and respiratory paralysis. In certain instances the animal may show convulsions. This is difficult to differentiate from anoxic struggling that may occur in the terminal stages of other conditions.

The gastroenteric form consists of initial intestinal stasis. The fermentation products will emanate from the mouth with foul odors present, and the animals will ultimately develop a fetid diarrhea. Bloat will develop along with salivation. The animal may succumb within one hour or die several hours later.

There are no pathognomonic lesions of milkweed poisoning. This must be differentiated from all the other infectious diseases that might cause similar gastroenteritis. As in all cases of poisoning, the most important factor is to get a proper history for the inclusion of the poisonous plant in the harvested feed. One must be very meticulous in evaluating a hay sample or a grain sample. You must recognize that poisonous plants may only be present in certain bales or certain portions of the bale and that you may or may not actually get the proper history.

Mechanically injurious plants

One usually thinks of mechanically injurious plants as causing problems by being caught in the hair, causing superficial injuries. Many of these plants may be found in hay and can cause problems in the mouth or in rare instances further along in the digestive tract.

The prime problems with mechanically injurious plants are the grass awns that penetrate the surfaces of the mouth. The classical plant is the foxtail *(Hordeum spp)* problem as seen in the small animal practice. The awns of this plant may become impacted in various areas in the mouth.

Animals with normal chewing action will usually keep grass awns out of the mouth with the tongue. The awns may cause erosion of the mucous membrane with subsequent invasion of the subcutis. The awns have retrograde barbs on the shafts which aid in the penetration and progression of the awn through the tissue.

Another grass awn that causes an unique lesion in cattle is yellow bristle grass, *Setaria lutescens.* This is a common weed of moist areas. If hay harvested from these areas comprises a rather significant portion of the hay, stomatitis can develop. I've seen cases in dairy herds and in beef cattle that have been fed on poor quality hay.

The clinical syndrome of yellow bristle grass disease is that of an ulcerative stomatitis. The lesion may be anywhere from 3 mm to 2 cm in diameter. Characteristically the surface of the ulcer contains many sharp, broken off awns of grass. The lesions may be found on the margins of the lips, on the tongue, on the buccal surfaces of the cheeks and the gingiva.

The treatment for yellow bristle grass injury is to remove the offending hay. The erosions must be curreted to remove the awns. Once this is accomplished one can treat the ulcer with any topical medication.

In each one of our practices we become acquainted with the more common clinical syndromes and recognize these quite easily. When a strange syndrome is observed it sometimes taxes our diagnostic accumen. Such a situation developed for a colleague and subsequently myself. The condition turned out to be lathyrism which is an ancient disease of man but has rarely been diagnosed in the U.S.

Lathyrus spp. are legumes. Included in the genus are ornamental sweetpeas, caley pea and rough pea. Lathyrus is closely related to vetch *(Vircia spp.)* and is easily confused with it in baled hay.

Lathyrism comes in two forms: osteolathyrism and neurolathyrism. Osteolathyrism is an experimental model used to study collagen synthesis. Neurolathyrism is produced by dipeptides in the plant that cause posterior spinal cord neuronal degeneration.

We experienced an outbreak of poisoning involving a field planted with what was thought to be vetch and harvested as a vetch hay. However, it turned out to be lathyrus. Clinical syndromes developed in both cattle

and horses when fed this hay. I could describe the condition as approximating the "tying-up" syndrome in horses. Animals had a stilted gait. The center of gravity was pushed forward over the front legs, giving the appearance of the horse balancing on the front legs and using the hind legs to move forward in short mincing steps.

Specific dipeptides produce neurodegeneration of the terminal segment of the spinal cord. There may be a functional upset in mild cases, but permanent damage is done if exposure is prolonged.

This outbreak of lathyrism reminded me that new problems can and do show up. Agriculture is still trying to improve production with new plant species. Different cultural practices may cause the development of new plant problems. We need to keep an open mind as we see syndromes that don't fit the usual pattern.

The scientific sessions and even the business meeting were all well attended.