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Plants Causing Kidney Failure (12-Dec-2003)

A. P. Knight¹ and R. G. Walter²

¹Department of Clinical Sciences, College of Veterinary Medicine, Veterinary Teaching Hospital, Colorado State University, Fort Collins, CO, USA.

²Department of Biology, Colorado State University, Fort Collins, CO, USA.

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In North America a variety of indigenous and exotic plants have been associated with kidney disease in animals. Most notable are the devastating livestock losses in the western alkaline desert areas of North America where plants such as greasewood (*Sarcobatus vermiculatus*), and halogeton (*Halogeton glomeratus*) that contain oxalates, are abundant. Interestingly, the most toxic of the plants that have established themselves in these semiarid areas is halogeton (*Halogeton glomeratus*), a plant introduced from Asia and highly adaptable to dry alkaline soils. This plant alone has caused heavy death loss in sheep in particular because of its extremely high oxalate content [1-3]. In addition to oxalate containing plants, kidney failure is also commonly associated with the consumption of plants such as oak trees (*Quercus* spp.) that contain high levels of tannins.

Oxalate Poisoning

Oxalate poisoning in animals generally occurs when quantities of oxalate-containing plants are grazed by livestock that are not accustomed to eating the plants. The most important oxalate-containing plants in North America include halogeton (*H. glomeratus*), and greasewood (*S. vermiculatus*) [1,2]. In Australia and Africa oxalate poisoning has been associated with livestock grazing *Oxalis* spp. [4]. Other plants known to contain significant quantities of oxalate are listed in Table 7-1. Plants normally accumulate oxalates in the form of soluble potassium and sodium oxalates. The more toxic potassium acid oxalate predominates in plants with a very acid cell sap (pH 2) (*Oxalis* spp.); sodium oxalate occurs in plants with a cell sap pH of 6 (*Halogeton, Sarcobatus* spp.) [5,6].

Oxalate poisoning most often occurs when unadapted sheep or cattle are allowed to graze large amounts of *Halogeton* or *Sarcobatus* as they pass through or are pastured overnight on rangeland containing large stands of these plants. Under normal range conditions sheep are most frequently poisoned by oxalate-containing plants. Ruminants in general tolerate relatively more oxalate in their diet than other animals because they are able to detoxify oxalate in the rumen thereby preventing the absorption of the soluble oxalates. When large quantities of soluble potassium and sodium oxalates are eaten that overwhelm the rumen's ability to metabolize the oxalates, they are absorbed into the bloodstream and form insoluble calcium and magnesium oxalates. It is these insoluble salts that precipitate in the kidneys and cause kidney failure [2,7]. Factors that predispose an animal to oxalate intoxication include the amount and rate at which the oxalate plant is eaten and the quantity of other feed diluting the oxalate in the rumen. Prior adaptation of rumen microflora to oxalates allows the animal to consume

more oxalate because the increased number of oxalate-degrading bacteria in the rumen more effectively metabolize the oxalate [8]. Ruminants allowed to graze small quantities of oxalate-containing plants are able to increase their tolerance for oxalate 30 percent or more over a few days [2,8,9]. Once adapted to oxalate, sheep and cattle can make effective use of range forages containing oxalate that would otherwise be toxic.

Toxic Effects of Oxalates

Once absorbed from the gastrointestinal tract, soluble oxalates rapidly combine with serum calcium and magnesium, causing a sudden decrease in available serum calcium and magnesium [2,9,10]. In the acute phase of oxalate poisoning the sudden decrease in soluble serum calcium (hypocalcemia) impairs normal cell membrane function, causing animals to develop muscle tremors and weakness, leading to collapse and eventually death. Oxalates also interfere with cellular energy metabolism that contributes to the acute death of affected animals. In chronic oxalate poisoning, insoluble calcium oxalate filtered by the kidneys causes severe damage to the kidney tubules (oxalate nephrosis). If animals do not die from the acute effects of the low blood calcium levels and impaired cellular energy metabolism, death results from kidney failure.

| Table 7-1. Plants Containing Oxalates | |
|--|-----------------------------|
| Scientific Name | Common Name |
| <i>Amaranthus</i> spp. | Red-rooted pigweed |
| <i>Bassia hyssopifolia</i> | Five hooked bassia |
| <i>Beta vulgaris</i> | Sugar beet |
| <i>Chenopodium</i> spp. | Lambs-Quarter |
| <i>Halogeton glomeratus</i> | Halogeton |
| <i>Kochia scoparia</i> | Kochia, summer cypress |
| <i>Oxalis</i> spp. | "Shamrock," soursob, sorrel |
| <i>Portulaca oleraceae</i> | Purslane |
| <i>Phytolacca americana</i> | Poke berry |
| <i>Rumex</i> spp. | Sorrel, dock |
| <i>Rheum rhaponticum</i> | Rhubarb |
| <i>Salsola</i> spp. | Russian thistle, tumbleweed |
| <i>Sarcobatus vermiculatus</i> | Greasewood |
| Grasses | |
| <i>Cenchrus ciliaris</i> | Buffel grass |
| <i>Panicum</i> spp. | Elephant grass |
| <i>Pennisetum clandestinum</i> | Kikuyu grass |
| <i>Setaria sphacelata</i> | Setaria grass |

| Table 7-1. Plants Containing Oxalates | |
|--|--|
| Scientific Name | Common Name |
| Common House and Garden Plants Containing Oxalates [35] | |
| <i>Arisaema</i> spp. | Jack in the pulpit |
| <i>Alocasia</i> spp. | Elephant's ear |
| <i>Anthurium</i> spp. | Anthurium, flamingo flower |
| <i>Arum</i> spp. | Lords and Ladies, Cuckoo-pint |
| <i>Calla palustris</i> | Wild calla, water arum |
| <i>Caladium</i> spp. | Caladium |
| <i>Dieffenbachia sequine</i> | Dumb cane |
| <i>Epiprenum</i> spp. | Pothos, variegated philodendron, taro vine |
| <i>Monstera</i> spp. | Monstera, cutleaf philodendron, bread fruit vine |
| <i>Philodendron</i> spp. | Philodendron |
| <i>Spathiphyllum</i> spp. | Peace lily |
| <i>Schefflera</i> spp. | Umbrella tree |
| <i>Zantedeschia aethiopica</i> | Calla lily |

Clinical Signs

Within a few hours of consuming toxic levels of oxalate, sheep and cattle develop muscle tremors, tetany, weakness, reluctance to move, depression, and recumbency resulting from hypocalcemia and hypomagnesemia [9-12]. Coma and death may result within 12 hours. Animals that survive the acute effects of oxalate poisoning frequently succumb to kidney failure. As animals become uremic (increased serum creatinine and urea nitrogen levels), they develop severe depression, stop eating, and after a few days become comatose and die.

Horses may over time develop chronic calcium deficiency while grazing certain tropical grasses containing soluble oxalates (Table 7-1). Oxalates in the grasses combine with calcium to form relatively insoluble calcium oxalate, thereby reducing calcium absorption and altering the calcium:phosphorus ratio. This causes mobilization of bone calcium through the action of parathyroid hormone to compensate for the low blood calcium levels. Over time the horse's bones lose sufficient calcium so that they become soft and misshapen (nutritional secondary hyperparathyroidism) [13-15]. Sheep and cattle can be similarly affected, but are better able to metabolize oxalates in the rumen thereby reducing their effect on dietary calcium [16].

Diagnosis

A diagnosis of oxalate poisoning can be made on the basis of the type of plants being eaten, the clinical signs, hypocalcemia, and the presence of oxalate crystals in the urine. However, oxalate crystals in the urine are usually not present in acute oxalate poisoning. Necropsy lesions seen in oxalate poisoning will depend on the severity of the poisoning. In acute poisoning, the kidneys may be edematous and dark red in color, whereas in chronic poisoning the kidneys may be pale and smaller than normal. Perirenal edema is a characteristic feature of oxalate poisoning in pigs and cattle consuming pigweed (*Amaranthus retroflexus*) [17-19]. The rumen may often become hemorrhagic due to the presence of large quantities of oxalate in the rumen epithelial lining. The demonstration of calcium oxalate crystals in the kidneys and rumen epithelium histologically is diagnostic of oxalate poisoning [10,11].

Treatment

Treatment with intravenous calcium gluconate, although theoretically appropriate for correcting hypocalcemia, is not effective in reversing the effects of the oxalate on cellular energy metabolism. Irreversible oxalate nephrosis and the effects

of oxalates on cellular energy metabolism are more detrimental to the animal than hypocalcemia. A theoretical approach to treating acute oxalate poisoning would be to administer intravenous calcium gluconate, magnesium sulfate, glucose, and a balanced electrolyte solution to maintain kidney perfusion. Giving limewater (Ca[OH]₂) orally will help to prevent absorption of further soluble oxalate.

Prevention of Oxalate Poisoning

Livestock should not be grazed on rangeland on which oxalate-containing plants predominate without precaution, especially if the animals are hungry and have not been adapted to oxalate in their diet. Livestock should be introduced to oxalate plants for at least 4 days by incrementally increasing the time they are allowed to graze the plants. Overstocking and overgrazing will potentiate oxalate poisoning if there is not other vegetation for animals to eat. Cattle and sheep driven through or held overnight in pastures rich in oxalate-containing plants are prone to poisoning, and such circumstances should be avoided. Supplementary dicalcium phosphate in the diet before and during high-risk oxalate exposure is an effective means of reducing losses. High levels of dietary calcium bind oxalate in the rumen as insoluble, nonabsorbable calcium oxalate. Calcium may be provided to the animals in a salt mix (75 lb salt, 25 lb dicalcium phosphate) or in pelleted alfalfa at a 5 percent concentration and fed at the rate of 0.5 lb per sheep per day. Livestock diets can also be supplemented with hay to help reduce the total intake of oxalate-containing plants.

Five Hook Bassia, Smother Weed

Bassia hyssopifolia - Chenopodiaceae (Goosefoot family)

Habitat

Introduced from Eurasia, bassia has adapted well to the alkaline soils of western North America and is a weed of waste ground.

Description

Bassia is an erect (2 to 5 feet), annual, much-branched plant with alternate, sessile leaves ranging to 2 inches in length. The leaves and stem are hairy. The flowers are borne in panicles or short spikes and sometimes singly in leaf axils. The greenish flowers are small, without petals, and the five sepals are armed with a stout, spreading, hooked spines that distinguish it from the similar appearance of Kochina weed. The plant spreads by seed only.

Principal Toxin

Although not widespread in this country as yet, *Bassia* has the potential for accumulating greater quantities of oxalates than *H. glomeratus* [12].

Halogeton, Bavilla

Halogeton glomeratus - Chenopodiaceae (Goosefoot family)

Habitat

Introduced from Russia, halogeton prefers the arid alkaline soils or clays of western North America. The plant continues to spread covering large areas of rangeland especially in flood plains and along rivers and roadsides.



Habitat of Halogeton. *Halogeton glomeratus* - Chenopodiaceae (Goosefoot family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Halogeton is an annual, multibranched herb with branches spreading horizontally before curving upward to 2 feet in height (Fig. 7-1A). Seedlings are usually prostrate with four main branches in the form of a cross. Mature plants have red stems with fleshy, blue-green leaves terminating in a solitary hair (Fig. 7-1B). The small, inconspicuous flowers appear in the leaf axil. The fruits are bracted and are often mistaken for the flowers. The single seed is surrounded by five reddish to yellow-green

bracts.



Figure 7-1A. Halogeton (*Halogeton glomeratus*) - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 7-1B. Halogeton showing leaves with terminal hair (*H. glomeratus*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Soluble sodium oxalate may comprise 30 to 40 percent of the dry matter content of the plant. Poisoning in sheep occurs when 0.3 to 0.5 percent of the animal's body weight of plant is consumed over a short period. A lethal dose of halogeton for an adult sheep is about 1.5 lb of green plant. In animals deprived of their normal feed, the toxic dose of halogeton is about one-third to one-quarter of that needed to induce poisoning in normally fed animals. Halogeton remains toxic when dried and is found to be quite palatable by sheep and cattle that commonly utilize halogeton as a winter forage when grazing western rangelands. They are able to do this once they are adapted to eating halogeton, and there are some other forages available.

Greasewood, Chico

Sarcobatus vermiculatus - Chenopodiaceae (Goosefoot family)

Habitat

Greasewood often forms the dominant shrubby vegetation of the arid alkaline soils of western North America. It commonly grows in the same habitat with *Halogeton glomeratus*.



Habitat of Greasewood. *Sarcobatus vermiculatus* - Asteraceae (Sunflower family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Greasewood is an erect, deciduous, branched shrub often growing 4 to 6 feet (1 to 2 meters) in height (Fig. 7-2A). The stem and branches turn gray with maturity and have 1 to 3 inches (2.5 to 7.5 cm) woody spines. The leaves are alternate, bright green, fleshy, loosely round in cross section, and up to 1.25 inches (3 cm) long (Fig. 7-2B). The flowers are unisexual with the plants having both sexes of flower on the same plant. The female flowers are inconspicuous in the axils and the male flowers occur as terminal spikes (catkins). The fruits are winged and conical in shape.



Figure 7-2A. Greasewood (*Sarcobatus vermiculatus*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 7-2B. Greasewood leaves (*S. vermiculatus*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Sodium oxalate is the principal toxin and may comprise from 10 to 22 percent of the dry matter content of the plant. The leaves contain the highest concentration of oxalate. Toxicity occurs when 1.5 to 5.0 percent of an animal's weight of the plant is ingested over a short period of time.

Sheep and cattle will often browse greasewood in the winter months, and do well on it as long as they are adapted to eating oxalate containing plants.

Sorrel, Wood Sorrel, Soursob Creeping Oxalis, "Shamrock"

Oxalis spp. - Oxalidaceae (Wood sorrel family)

Habitat

Various species of sorrel are found throughout North America, growing at altitudes up to 9000 feet (2,743 meters). The plants are common in moist pastures, forests, along roadsides, and as weeds of cultivated soils.



Habitat of Sorrel. *Oxalis* spp. - Oxalidaceae (Wood sorrel family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Wood sorrels are herbaceous annuals or perennials that grow horizontally but occasionally erect with characteristic alternate or basal leaves that are palmately three- foliate and heart-shaped (Fig. 7-3). The flowers are usually yellow, with perfect stipules, and regular umbel-like or dichotomous cymes. The fruit is a capsule.



Figure 7-3. Oxalis, shamrock, wood sorrel (*Oxalis* spp.) - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxins

The principal toxins are soluble potassium and sodium oxalates. *Oxalis* spp. poisoning is rarely a problem in North America, but it has been associated with severe poisoning of livestock in Australia [3].

Pigweed, Amaranth

Amaranthus retroflexus - Amaranthaceae (Pigweed family)

Habitat

A variety of pigweeds grow throughout North America; the most common and typical of the genus is red-rooted pigweed (*A. retroflexus*). It is a common weed of most soils, found in cultivated and disturbed soils along roadsides and waste areas. It is common in and around corrals and animal enclosures.



Habitat of Pigweed. *Amaranthus retroflexus* - Amaranthaceae (Pigweed family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Other common species of pigweed include prostrate pigweed (*A. blitoides*), tumble pigweed (*A. albus*), and Palmer amaranth (*A. palmeri*). All have similar characteristics to the red-rooted pigweed.

Description

Plants are stout, erect, rapidly growing annuals, reaching 3 to 4 feet (1 to 1.2 meters) tall depending on the growing conditions. The stems are usually much branched and hairy and may be red to purple in color. The leaves are alternate, petiolate, ovate to lanceolate, and acute at apex. The flowers are monoecious in densely crowded spikes or panicles 3 to 8 inches (8 to 20 cm) long (Fig. 7-4A). The flowers are greenish with long, spine-tipped bracts. The taproot is usually bright red in color (red-rooted pigweed; Fig. 7-4B). Numerous, shiny black seeds are produced that ensure successful proliferation of the pigweed.



Figure 7-4A. Pigweed, amaranth flower spike (*Amaranthus retroflexus*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 7-4B. Red-rooted pigweed (*A. retroflexus*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Amaranthus spp. may accumulate significant quantities of oxalate that causes a syndrome in pigs and cattle characterized by

perirenal edema [17,18]. Renal tubular nephrosis probably causes death of the animal. However, renal nephrosis is often present without the presence of oxalate crystals, suggesting that *Amaranthus* spp. may contain other as yet undefined toxic substances.

As annual weeds, pigweeds also accumulate nitrates and can be a source of nitrate poisoning (see Chapter 1).

Purslane, Pusley

Portulaca oleraceae - Portulacaceae (Purslane family)

Habitat

Purslane is a very common weed of gardens, cultivated ground and waste places.



Habitat of Purslane. *Portulaca oleraceae* - Portulacaceae (Purslane family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Purslane is an annual, succulent plant with prostrate stems, that are pink to red in color (Fig. 7-5A). Leaves are fleshy and flat, obovate in shape, and either alternate or clustered on the stem. Flowers are yellow, solitary, and open for only a short time (Fig. 7-5B). Fruit is a circumscissile capsule.



Figure 7-5A. Purslane showing prostrate stems (*Portulacca oleracea*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 7-5B. Purslane flowers. - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Oxalates are the principal toxins and account for the sour taste to the plant. As much as 9 percent dry matter of the plant may consist of oxalate. Few cases of poisoning in livestock have been reported.

Purslane is eaten by some people as a vegetable, and does not pose a toxicity problem because, in cooking the plant, the oxalates are leached out by the steaming or boiling process.

Dock, Curly Leaf Dock, Sorrel

Rumex crispus - Polygonaceae (Buckwheat family)

Habitat

Dock is a common plant of gravelly soils of pastures, plains, and roadsides throughout North America. There are a variety of *Rumex* spp. with similar characteristics and habitats.



Habitat of Dock. *Rumex crispus* - Polygonaceae (Buckwheat family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Dock is a perennial weed from a stout tap root. Flower stems may reach 6 to 7 feet (2 meters) in height without axillary branches. Leaves are 4 to 12 inches (10 to 30 cm) long, oblong to linear-lanceolate with crisped, wavy margins (Fig. 7-6A). An ocrea is present at the base of each petiole. The flower is a compound raceme or panicle having many small perfect or unisexual flowers. The perianth is six-parted with the inner three segments becoming the wings of the fruit. The inflorescence turns a dark brown when dry (Fig. 7-6B).



Figure 7-6A. Dock, sorrel (*Rumex crispus*). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 7-6B. Dock with curly leaves and ripe seed head (*Rumex crispus*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Oxalates are the primary toxins in *Rumex* spp. Although not a common source of oxalate poisoning, *Rumex* spp. will cause poisoning if eaten in excess by livestock [11,18,19].

Rhubarb

Rheum rhabarbarum (*R. rhabarbarum*) - Polygonaceae (Buckwheat family)

Habitat

Rhubarb is a domestic plant found throughout North America, although it may escape and be found great distances from any dwelling. Moist, fertile soils are preferred habitats.

Description

Rhubarb is a stout herbaceous, perennial plant with large, fleshy roots and large leaves that are heart shaped, with entire margins and long reddish petioles. Flowering stems, 1 to 2 meters long, support a many-flowered panicle of greenish or whitish flowers. The fruit is a winged achene that turns a dark brown color when mature.

Principal Toxin

The leaves contain large amounts of soluble oxalates. The red leaf stalk (petiole) contains much less oxalate than the leaves. Feeding the leaves to livestock may induce severe oxalate nephrosis. Human poisoning is almost always associated with eating rhubarb leaves used as a vegetable or salad. Human poisoning from eating rhubarb stems is very unlikely as they contain minimal amounts of oxalate, and a person would have to eat literally several pounds of rhubarb to be affected. Cooking them with some calcium carbonate to produce insoluble calcium oxalate can further reduce the toxicity of the rhubarb stems.

Anthraquinone compounds in the leaves that have cathartic properties are the likely cause of the abdominal pain accompanied with vomiting encountered in people who have eaten rhubarb leaves

Oak

Quercus spp. - Fagaceae (Oak family)

Habitat

Some 60 species of oak grow in North America in a wide variety of habitats ranging from moist, rich soils of hard wood forests to drier mountainous areas.

Description

Ranging from large trees to shrubs, oaks have alternate, simple, toothed, or lobed dark green glossy leaves. The leaves may be deciduous or persistent depending on the species of oak. The plants are monoecious with the staminate flowers occurring in long catkins and the pistillate flowers occurring singly or in small clusters. The fruit, an acorn, is a nut partially enveloped by an involucre of scales (Fig. 7-7). Two common species of oak growing in western North America commonly associated with livestock poisoning are scrub oak and shinnery oak.



Figure 7-7. Oak leaves and acorn (*Quercus* spp.). - To view this image in full size go to the IVIS website at www.ivis.org . -

Q. gambelii (Gambels oak, scrub oak) is a shrub or small tree reaching heights of 15 to 20 feet (4.5 to 6 meters). It grows in dense stands in the dry foothills and mountain slopes up to altitudes of 9000 feet (2,743 meters).

Q. havardii (*Shinnery oak*) is a shrub that seldom attains heights over 4 feet (1.2 meters). It is confined more to the lower elevations and sandy soils of southwestern North America.

Principal Toxin

The principal toxin is gallotannin, a polyhydroxyphenolic combination of tannic and gallic acid [20-22]. The tannins found in the leaves, bark, and acorns of oaks produce poisoning through their effect on the intestinal tract and kidneys [21-23]. Gallotannins are hydrolyzed in the rumen to smaller molecular weight compounds including gallic acid, pyrogallol, and resorcinol [24]. These compounds react with cell proteins to denature them, with resulting cell death. Most severe lesions occur in the kidneys, liver, and digestive tract. In small quantities the rumen microflora detoxify the tannins, and only when large amounts of tannic acid are eaten and bypass the rumen does poisoning occur [25]. Goats and wild ruminants are apparently better able to detoxify tannic acid than other livestock because they have a tannin-binding protein in their saliva that neutralizes tannic acid [26,27]. Goats have been used effectively to browse on oaks thereby reducing the spread of the oak and increasing the grazing capacity of the range. Oaks at any stage of growth are poisonous, but they are particularly toxic when the leaf and flower buds are just opening in the spring. Consumption of oak buds can be markedly increased in a heavy, late spring snowstorm, when cattle browse the oak that protrudes above the snow. As the leaves mature they become less toxic. Ripe acorns are less toxic than when green. Cattle, sheep, horses, and pigs are susceptible to oak poisoning [21,28-30].

Clinical Signs

Signs of oak poisoning will vary according to the quantity of oak consumed. Initially affected animals stop eating, become

depressed, and develop intestinal stasis [22-24]. Excessive thirst and frequent urination may be observed. The feces are hard and dark initially, but a black tarry diarrhea often occurs later in the course of poisoning. Teeth grinding and a hunched back are often indicative of abdominal pain. Severe liver and kidney damage is detectable by marked elevations in serum liver enzymes, creatinine, and urea nitrogen [30]. Icterus, red-colored urine, and dehydration are further signs encountered in oak poisoning. Animals may live for 5 to 7 days after the onset of clinical signs.

Necropsy

A mucoid, hemorrhagic gastroenteritis is a common finding in oak poisoning [23,29,31]. Hemorrhages on various organs and excessive amounts of fluid in the peritoneal and pleural spaces are often present. The kidneys are usually pale swollen and covered with small hemorrhages. Histologically kidney tubular necrosis, and liver necrosis are characteristic of oak poisoning [23,29,31].

Treatment

Animals should be removed from the oak and given supportive care in the form of fresh water and good quality hay. Oral administration of a calcium hydroxide solution is helpful in neutralizing residual tannic acid in the rumen [32]. Intravenous fluids should be given to rehydrate severely affected animals and maintain kidney function. Animals that continue to eat have a much better prognosis. Cattle that survive oak poisoning appear to have compensatory weight gains and appear to do well [33].

Grain or pelleted rations containing 10 to 15 percent calcium hydroxide are beneficial in preventing oak poisoning if cattle have to graze pastures overgrown with oak brush [32]. Goats are effective biological controls and may be used to browse oak for range management purposes because they are unaffected by the tannins in the oak [34].

Acorn Calf Syndrome

The acorn calf syndrome is not related to oak poisoning attributed to gallotannins but is encountered in calves born to cows on a low plain of nutrition and which have consumed quantities of acorns [36]. Acorn calves are born with laxity of the joints, shortened legs (dwarfism), deformed hooves, and either a domed skull or long narrow head. Compared to normal calves, the acorn calves are stunted and grow poorly. The acorn calf syndrome has also been reported in cows that graze heavily on lupine during mid pregnancy [37]. The toxic principal responsible for this congenital syndrome has not been determined. Protein malnutrition and the presence of a teratogen may be involved in the development of the acorn calf syndrome.

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