Halogeton (*H. glomeratus*) Poisoning in Cattle: Case Report

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Abstract

Historically, the most significant losses from halogeton poisoning have been reported in sheep, with multiple catastrophic deaths documented. While recorded death losses in cattle from halogeton poisoning are less common than in sheep, recent cases, including 2 reported here, and anecdotal reports from other ranchers suggest that the impact of halogeton losses in cattle herds in the western United States is much more widespread than originally thought. Halogeton may accumulate up to 30% oxalates; a small amount of the plant (300 g) is enough to cause death in sheep. Oxalates precipitate calcium from the blood, resulting in hypocalcemia, formation of calcium oxalate crystals, and uremia. In this report, 2 cases of halogeton poisoning in cattle are documented, including a history of halogeton grazing, supportive oxalate analysis of plants collected from the poisoning locations, and histological evidence of classic oxalate nephropathy and calcium oxalate crystal nephrosis.

Keywords: cattle, halogeton, oxalates

Introduction

Originally from Eurasia, halogeton (*Halogeton glomeratus*; figure 1) is an invasive, noxious weed in many parts of the world. In North America, it was first collected and identified in the United States in Wells, Nevada, in 1934 (Cook and Stoddart 1953). It is unclear how this weed was introduced, but it displaced native plants due to overgrazed and depleted desert range conditions, and within 40 years it infested 11.2 million acres throughout the intermountain west and Colorado Plateau (figure 2; James et al. 2005). Numerous cases of acute sheep death attributed to halogeton grazing were reported during the ensuing years, culminating in a very public Idaho case involving the death of nearly 1,300 head of sheep. In 1952, federal funds were allocated for eradication and control with the passage of a halogeton control bill (Young 1988).

The mechanism of halogeton toxicity was found to be soluble oxalates, sodium oxalate and potassium oxalate, which bind calcium, forming calcium oxalate monohydrate and insoluble calcium oxalate. Calcium oxalate monohydrate damages mitochondria, increases reactive oxygen species, and decreases tricarboxylic acid enzymes (succinate dehydrogenase, isocitrate dehydrogenase, malate dehydrogenase, and other respiratory enzymes), resulting in mitochondrial dysplasia and reduced oxidative phosphorylation (Chungang and McMartin 2005). Calcium oxalate may precipitate, forming crystals that mechanically injure renal tubules, interfere with rumen function, and affect specific metabolic pathways, including calcium homeostasis. Clinically, this is seen as renal failure with severe calcium oxalate crystalline nephropathy. Oxalate form, dose, and duration are important in disease progression, as certain acute poisonings can produce sudden death prior to the development of crystalline nephropathy.

Halogeton and greasewood *(Sarcobatus vermiculatus)* are the principal toxic oxalate-producing range plants found in North America. Halogeton oxalate content generally ranges between 15% and 18%, with reports as high as 36% dry weight. Acute toxicity has historically been

Figure 1. Halogeton with close-up of leaves (inset).

Figure 2. Halogeton in high desert pasture.

associated with sheep, but occasional cases have been reported in cattle (McMartin and Wallace 2005).

Case 1

Twenty-six mature Black Angus cows, in a herd of 350, were found dead in 2012 by a northwestern Utah producer. Cattle were in poor body condition (BCS 3.5/9) coming off of summer grazing and had been trucked 50 km to winter pastures. Death occurred within 1 week of moving. A necropsy was performed on 1 of the deceased animals, and the referring veterinarian submitted tissues. Histologic evaluation identified severe oxalate nephrosis, with intratubular material consistent with calcium oxalate crystals.

The producer submitted plant samples, and subsequent plant samples were obtained and submitted during a farm visit by the author. Submitted plants were identified as *H. glomeratus*.

A major significant growth area of halogeton with evidence of halogeton grazing was found in one of the pastures where a gate was left open. Halogeton plant growth seemed to be associated with disturbance along an old irrigation canal. Oxalate content of the plants collected at this site was 29%. Halogeton plants were identified and collected from a neighboring ranch 24 km away and found to contain 17% oxalates.

Case 2

In 2006, a rancher in southeastern Idaho had 6 cows die over a 2-day period, followed by 3 more deaths in the subsequent 2 days. Two of the cows were submitted for necropsy evaluation at the Utah Veterinary Diagnostic Laboratory. Cows were in average body condition (BCS 5/9). Upon opening the rumen, it was noted that the rumen was full of plant material unfamiliar to the pathologist. Inspection by the toxicologist identified the plant material as *H. glomeratus*. Histologic evaluation found oxalate nephrosis with intratubular crystalline material consistent with calcium oxalate crystals in both cows.

Subsequent investigation with the rancher revealed that the cattle had been moved from a high mountain range 24 hours prior to the first death. The cattle had been unloaded and were being trailed to a new pasture. Inspection of the site where the cows were bedded the first night revealed a large stand of halogeton that had been extensively grazed. The rancher described the cows as actively going to those plants. Further discussions with the rancher found that the cows had not been receiving any salt or mineral supplements for an extended period prior to the halogeton exposure.

In total, 9 cows out of 85 that were bedded next to the halogeton stand died over a period of 4 days. Oxalate nephrosis with intratubular oxalate crystals supported the diagnosis of *H. glomeratus* poisoning.

Discussion

Deaths were attributed to either acute nephrosis and renal crisis or severe hypocalcemia secondary to grazing halogeton. Because the cattle in these 2 cases were in poor to average body condition and perhaps dehydrated from being trucked some distance, the toxic effects of oxalic acid ingestion (sodium and potassium) from the halogeton were exacerbated. Food and water deprivation was previously reported as a contributor to the severity of oxalate toxicosis (Lincoln and Black 1980). The binding of calcium in the formation of calcium oxalate and crystallizing in the kidney during excretion caused renal tubular epithelial degeneration and necrosis. While hypocalcemia was not determined in these cases, it has been reported as a common consequence of oxalate formation, as the binding of calcium depletes serum calcium levels, and it likely played a role in these cases (James

1968, Sebastian et al. 2007). In ruminants, hypocalcemia is a major contributor to rumen stasis, paresis, and death (Ruckebusch 1983, Cheng et al. 1998, Van Winden and Kuiper 2003, Russell and Roussel 2007).

Halogeton growth in Case 1 seemed to be associated with previously disturbed land, with the majority of growth near an old ditch bank. On the neighboring ranch, halogeton was growing in the corner of a pasture used for cultivation. In Case 2, the halogeton growth was also associated with disturbed areas, a livestock trail in this case. This is consistent with previously reported investigations of halogeton toxicities (Stoddart et al. 1951). The variation in oxalate levels seen here between neighboring ranches validates previous reports of large variations in plant oxalate content (Stoddart et al. 1951, James 1972, Lincoln 1980, James et al. 2005).

The following are recommendations for avoiding oxalate toxicity:

1. Avoid introducing hungry, thirsty, or salt-deprived animals into halogeton-infested areas.
2. Ensure that plenty of fresh water, adequate forage, and calcium-enriched trace mineral salt are available.
3. Provide supplements with calcium carbonate, calcium chloride, or dicalcium phosphate, e.g., 83% alfalfa pellet with 15% calcium carbonate and 2% molasses (Cook and Stoddart 1953).
4. Treat affected animals with parenteral calcium (intravenous calcium borogluconate); this treatment is reported to be more effective in cattle than sheep.
5. Halogeton may be controlled with 2,4-D herbicide. Seeding with perennial grasses, such as crested wheatgrass, will compete with the halogeton and help prevent reinvasion.
6. Avoid overgrazing, and practice good range-management techniques to slow halogeton invasion. Soil disturbance may result in a rapid spread of halogeton.

The cases reported here document that halogeton continues to be a problem for livestock ranchers in the intermountain United States region. Furthermore, while cases have historically been associated with grazing sheep, halogeton toxicoses in cattle are possible and should be considered when cattle are grazing in areas where halogeton is endemic. Precautions should be taken accordingly.

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