PEER REVIEWED

Acute Fluoride Toxicosis in Beef Cattle

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Abstract

Acute fluoride toxicosis, though less common than chronic fluorosis, occurs occasionally in cattle. Nine cows out of a herd of 15 became weak and ataxic, and three died on a pasture where utility poles had been treated with a fluoride-containing compound. After postmortem examination, two of the three dead animals were diagnosed with hemorrhagic gastroenteritis consistent with acute fluoride toxicosis. Diagnosis of fluoride toxicosis was based on history, clinical signs, gross lesions, and fluoride concentration in urine and rumen content.

Introduction

Fluoride is an ubiquitous element, though it is rarely found free in nature.[13] Exposure to fluoride has been documented to cause toxicosis in domestic animals, most frequently in cattle.[12] Chronic fluoride toxicosis in cattle has been reported more frequently than acute fluoride toxicosis.[9] Acute fluoride poisoning has been reported in humans.[2,6,8]

Chronic fluoride poisoning, also referred to as fluorosis, in cattle has been associated with natural and industrial contamination of forage and water, and with rock phosphate mineral supplements that have not been defluorinated.[1,4,12,13] Lesions of fluorosis occur in teeth and bones. Dental lesions, which are associated with exposure to high levels of fluoride during the first 30 to 36 months of life, include mottling and discoloration of incisors, enamel hypoplasia and rapid attrition.[3,4,9,12,13] Skeletal lesions include hyperostosis, osteoporosis, osteoschlerosis, osteophytosis, and osteomalacia, and usually involve the metatarsals,[2,9,12,13] but all bones in the body may be affected.[12] Significant dental or skeletal lesions are debilitating, causing decreased grazing, poor body condition, and general ill thrift.[4,9,12,13]

There are numerous reports of acute fluoride poisoning in humans associated with errors in water fluoridation and with excessive ingestion of fluoride supplements in children.[2,3,8] There are relatively few reports detailing acute fluoride toxicosis in cattle, but reported cases are usually associated with sodium fluoride.[4] Clinical signs reported in humans and cattle involve the gastrointestinal, nervous, and cardiovascular systems.[2,3,4,8,9] This paper documents a recent case of acute fluoride poisoning in a herd of beef cattle.

Case Study

A utility company in West Virginia treated utility poles in a pasture containing 15 head of mature Simmintal-Limousin cross cattle. One cow, in late gestation, was found dead soon after the poles were treated. Eight other animals showed some degree of generalized weakness and ataxia on the day of exposure. All affected cows were treated with activated charcoal and atropine. Two of the affected cows died within the next 48 hours, the remaining 6 recovered.

No significant lesions were found during necropsy of the first cow, however, the rumen content had a strong creosol-like odor, similar to that of the utility poles. The other two cows that died had congested lungs, inflamed abomasal and small intestinal mucosa, and hemorrhages on the gastrointestinal serosa. The rumen content recovered from these two animals also had a strong creosol-like odor.

According to the label, the compound used to treat the utility poles involved in this case contained 44% so-

dium fluoride, 45% creosote oil, and 3.2% sodium dichromate. Three urine samples, two liver samples, and samples of rumen content were collected from affected cows and submitted with samples of the compound used to treat the utility pole to the Oklahoma Animal Disease Diagnostic Laboratory for toxicologic analysis.

The submitted material was analyzed for fluoride using a modified fluoride electrode analysis procedure based on the technique published by Stahr.[14] Urine was diluted to a 50% concentration using TISAB IV® buffer. Utility pole material, liver, and rumen content were ashed in a muffle furnace, and 10 mg of ash was dissolved in 1.0 ml of 20% hydrochloric acid followed by 3.0 ml water and 5.0 ml TISAB IV® buffer. Pole material and rumen content were extracted with chloroform and analyzed on gas chromatography/mass spectrometry (GC/MS) using standard EPA acid and base neutral priority pollutants techniques for GC/MS.

Pole material consisted of a thick, black paste with a creosol-like odor. Analysis of this material revealed that it contained 5% fluoride. The major phenolic compound detected in the pole material was p-cresol, which was detected at a level of 0.28%. The two urine samples contained 166 and 129 ppm fluoride on a wet weight basis and both liver samples contained 0.04 ppm fluoride. Rumen content contained 475 and 47.5 ppm fluoride and 198.0 and 86.7 ppm p-cresol, respectively (Table 1). All results are reported on a wet weight basis.

Table 1. Fluoride ion and p-cresol content of biological and source material.

Source of Sample	Sample	Fluoride ion (ppm)	p-Cresol (ppm)
Cow # 93	Urine	166.00	
Cow #91	Urine	129.00	
	Liver	0.04	
	Rumen Content	475.00	198.00
Cow #391	Urine	Unsuitable	
	Liver	0.04	
	Rumen Content	47.50	86.70
Utility Pole Material	Oily substance	5000.00	0.28

Discussion

Diagnosis of acute fluoride toxicosis is based on history of exposure, clinical signs, pathologic findings, and elevated concentrations of fluoride in urine or tissues. The single acutely toxic dose of fluoride in cattle is estimated at between 50 and 70 mg/kg.[4] The quantity ingested by the cows in this study is not known.

Previous reports detail clinical signs associated with acute fluoride poisoning in dairy cattle.[4] Clinical signs include a rapid onset, weakness and central nervous depression, gastroenteritis, decreased rumen motility, bloat, and diarrhea.[9,13] The ingestion of excessive amounts of fluoride by cattle has been reported to cause clonic convulsions and cardiac failure is frequently believed to be the cause of death.[9,13] Dogs infused with intravenous sodium fluoride die from ventricular fibrillation.[3] The only clinical signs reported in this case were associated with central nervous depression and ataxia.

Diagnosis of gastroenteritis was made based on gross pathology and histopathologic evaluation of the gastrointestinal tract of two of the affected animals. Pathologic findings were consistent with those previously reported in cases of acute fluoride intoxication in cattle and humans.[2,9] Less commonly reported lesions of pulmonary congestion, edema, and myocardial necrosis were also found in two of the cows in this case.[4]

No information was found concerning urinary fluoride concentrations associated with acute fluoride poisoning. Fluoride is rapidly excreted in the urine.[8] Urine fluoride levels in cattle represent recent intake, but fluctuate erratically and may be influenced by the total amount of fluoride consumed, rate of absorption, rate of skeletal uptake, time of day, and urine specific gravity.[9,11] Normal, background urinary fluoride levels range from 1 to 6 ppm. A fluoride concentration of 25 ppm in cow urine is associated with the chronic ingestion of dietary fluoride levels of 40 ppm or more.[4]

Liver fluoride levels greater than 20 ppm have been considered diagnostic for fluoride toxicosis in cattle.[4] The two livers in this case contained 0.04 ppm. It is unclear why the liver fluoride levels in these cows were low. Fluoride is rapidly absorbed from the gastrointestinal tract.[8,13] Soft tissue distribution and accumulation of fluoride tends to be minimal.[13] Fluoride does not readily cross cell membranes.[3] It is rapidly removed from circulation by sequestration in the bone and renal excretion.[8,12] It is possible that at the time these samples were taken, most of the absorbed fluoride had already been sequestered in bone or excreted, and gastrointestinal absorption was slowed due to decreased rumen motility. Rumen content fluoride levels associated with intoxication have not been reported. The levels reported in these cases, especially in cow #91, appear to be elevated and suggest that substantial quantities of fluoride were ingested.

Though sodium and chromate were known ingredients in the wood preservative used to treat the poles, they were not present in sufficient quantities to cause death in these cows. The chronic toxic dose for zinc chromate in cattle is 30 mg/kg.[10] Another component of the

wood preservative was p-cresol. However, at the low level of p-cresol detected in the pole material by GC/MS (0.28 ppm), it is unlikely to be the cause of the toxicosis. The toxic dose of coal tar, which contains 20000 to 80000 ppm (2 to 8%) light oils (phenols, cresols, and napthalene), is 15 g/day over several days.[9] Clinical signs and lesions seen in the cows were more consistent with those reported for fluoride toxicosis. Based on the history of exposure, clinical signs, pathologic findings and elevated fluoride levels in urine and rumen content, a diagnosis of acute fluoride toxicosis was made.

Treatment of acute fluoride toxicosis in humans has not been effective. Likewise, success with treatment of cattle has been limited. If treatment is attempted, the veterinarian should focus on decreasing fluoride absorption and on supportive care. The most effective way to decrease absorption is removal of the fluoride from the rumen via rumen lavage.[4] Fluoride binds readily to cations.[3,4] Aluminum, calcium, or magnesium based binding agents are indicated.[2,8,13] Aluminum-based antacids have been given to fluoride intoxicated humans.[2] Administering calcium compounds, such as fluids containing calcium or milk, may be beneficial.[13] Magnesium sulfate is indicated to provide magnesium ions and for cathartic action.[8]

Heart rate, serum pH, and serum electrolyte balance, especially calcium, magnesium, and potassium must be monitored.[2,8] Sodium bicarbonate can be administered to induce alkalosis and increase the rate of fluoride excretion, however, calcium balance must be carefully monitored.[3] Fluoride precipitates cations and intoxicated individuals rapidly become hypocalcemic.[2,3,8] Hypocalcemia may be the cause of some of the nervous signs reported, including weakness.[2] Some calcium is also moved to the intracellular compartment, which is believed to open calcium-dependent potassium channels, resulting in hyperkalemia.[2,3,7] Hyperkalemia may be delayed for several hours and may be correlated with cardiac arythmia.[3,7] Attempts to reverse hyperkalemia and associated cardiac complications with glucose, insulin, bicarbonate, and lidocaine have not been successful.[7] Quinidine, which blocks calcium-dependent potassium channels, has increased survival in experimental dogs when given simultaneously with high intravenous doses of sodium fluoride.[7]

Conclusions

Acute fluoride toxicosis should be considered in cattle with known exposure to wood treatment compounds. Fluoride intoxicated cattle have a rapid onset of central nervous depression and gastroenteritis.[9,13] When fluoride toxicosis is suspected, samples from cows, including urine antemortem and urine and rumen content postmortem, and material from possible sources of exposure should be submitted to the toxicology laboratory for analysis.

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