

Case Report. A Complex, Catastrophic Toxicology Case in a Herd of Beef Cows

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Introduction

A catastrophic toxicology case involving a well managed herd of 168 beef cows is reported. During the course of this case several rule-outs were considered, and many diagnostic tests were run.

Materials and Methods

One-hundred thirty cattle were moved to a new pasture on August 20, 2005. Thirty-eight more cattle were added on September 2, 2005. Fertilizer was applied to the pasture on August 25, 2005 at a rate of 100 lb (45 kg) of ammonium sulfate and 50 lb (23 kg) of urea applied per acre. There was no appreciable rainfall for the six weeks prior to fertilizer application but 0.3 inches (0.76 cm) rain was received after fertilizer application. In addition, the cattle were without water a portion of the weekend of September 3 and 4. Nineteen cattle died by noon of September 5. At least one cow appeared to have been dead since late Saturday, September 3 or early Sunday, September 4. Ten more cattle died late in the day on Monday. Cattle were moved to a different field late on Monday. Fifteen more cattle died on Tuesday, and an additional 11 cattle died over the next two days (Wednesday and Thursday). A total of 55 cows died and about 20 aborted calves.

Results

Clinical signs were depression, diarrhea, weakness and recumbency. Over 90% of the cattle were affected to some extent. Approximately 10-20 cows were belligerent on the first day. The initial diagnosis was nitrate toxicity; therefore, initial treatments consisted of calcium supplements and methylene blue intravenously. Some downer cows initially responded to calcium treatment. Before blood results were available, this was mistakenly believed to be a response to the methylene blue therapy, and 25 cows were treated in this manner. Clinical pathology and toxicology tests were done on a number of samples. Some, but not all, serum samples were elevated in sodium, indicating possible water deprivation. For the next two days, sodium toxicity/water

deprivation was the working diagnosis because of the elevated serum sodium levels. Over the course of those two days more results became available. Clinical pathology changes were most consistent with a renal problem, including hypoalbuminemia, hyperphosphatemia, hypocalcemia, elevated BUN and elevated creatinine. Many animals had an elevated anion gap consistent with a metabolic acidosis. Lesions were found in the kidney on histopathological exam. Urinalysis revealed isosthenuria (S.G. 1.015) and losses of protein, ketones and red blood cells in the urine. The urine pH was very acidic (pH 5).

Toxicology testing on serum, eyeball fluid and kidneys ruled out nitrates, lead and arsenic toxicity. Brain cholinesterase activity was normal, ruling out OP insecticides and also most probably carbamate insecticides. Brains from two cows and their unborn calves were also analyzed for sodium. One cow had sodium levels consistent with water deprivation; the others had a high normal to marginally high sodium concentrations. Mineral fed during this time was analyzed for calcium, phosphorus and sodium. All were present at the appropriate levels of minerals as indicated on the tag. Soil samples were also taken from the field and neighboring fields that had not been fertilized. Soil samples were indicative of fertilization with ammonium sulfate and urea.

Known toxic causes of renal damage in cattle are relatively limited. Most common is acorn exposure causing perirenal edema and assorted GI problems. No evidence of acorn consumption or perirenal edema was found. Pigweed consumption causes similar lesions to acorns. Although the pasture had some spiny pigweed, there was no evidence of excessive consumption, and perirenal edema and some other clinical features and clinical pathologic changes commonly noted with pigweed were not present. Lead and arsenic are fairly common toxicoses in cattle and can cause renal lesions, but they were not detected in kidney tissue. This left other less common causes of kidney problems for ruleouts.

Because of recent ammonium sulfate fertilization, we did a literature search on its possible toxic manifestations. While not commonly reported as a cause of kidney problems in veterinary toxicology textbooks,

high doses of ammonium salts are known to cause a CNS excitation syndrome similar to urea. Belligerency is one rather key clinical sign associated with the syndrome. Ammonium salts typically do not cause an increase in rumen pH similar to urea, but rather the pH is typically in the 6.5-7 range, similar to that found in this case. Several articles in the 1970s reported dosing of sheep with ammonium salts. In these papers, kidney lesions were similar to those seen in these cattle. Clinical pathology changes included albumin and red blood cells in urine, as well as acidic urine (pH around 5). Sheep that survived a high toxic dose and showed initial CNS excitement had pink colored urine because of RBCs leaking into the urine the next day.

Ammonium sulfate is somewhat acidic when ingested and can cause a metabolic acidosis and an acid urine. It is also capable of causing renal problems. Most likely the grass absorbed the ammonium following the light rain, but did not convert it into plant protein because of drought stress. The cattle then consumed ammonium each day during grazing of the pasture. It has been proposed that lower doses of ammonium sulfate consumed over several days can cause renal problems without causing CNS excitement characteristic of a large one-time dose of ammonium salts. The belligerency seen in these cattle is more consistent with a higher dose of ammonium salt poisoning. The combination of history of exposure, rumen pH, belligerency, metabolic acidosis, renal damage and urine changes are consistent with a diagnosis of ammonium sulfate toxicosis. Unfortunately, a definitive diagnosis of ammonium sulfate toxicity was not possible because the kidney lesions were similar to other renal toxicants. Testing for the toxicant is hindered because both ammonia and sulfur are

normal constituents of the body, and ammonia does not persist in the plasma, making analysis impossible the next day. Rumen bacteria continue breaking down protein in rumen samples, forming additional ammonia. Rumen samples not frozen immediately are worthless for analysis for ammonia.

The role of water deprivation in this outbreak is uncertain. One cow had brain sodium values consistent with water deprivation, but the other one did not and no brain lesions consistent with water deprivation were noted in the one brain examined. The timeline for sodium toxicity/water deprivation did not fit this case either. We concluded that water deprivation was not the primary toxicity, but it likely contributed to the severity of the toxicosis. The fact that one cow probably died on Saturday night or early Sunday also reduces the likelihood that water deprivation was the primary cause of death.

Significance

This report gives an example of a severe toxicosis that can occur in a well managed beef herd that is following best management practices under adverse environmental conditions. Ammonium toxicity has only been described in the literature in experimental studies, and one case report from England in the late 1970s and early 1980s. Because ammonium is a weak acid, rumen pH is not helpful as a diagnostic tool, unlike cases of urea and ammonia toxicity. This case indicates that, under drought conditions, care needs to be taken when making recommendations to fertilize pastures cattle are grazing.