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Characterization of Sulfur and Sulfate Toxicosis in Cattle

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Abstract

Sulfur and sulfate toxicoses are important causes of polioencephalomalacia (PEM) and feed refusal in cattle, resulting in decreased weight gain and increased death losses. The purpose of the study was to characterize the demographics of cattle with sulfur/sulfate toxicosis (ST), describe clinical signs and pathology associated with disease, and to identify potential sources of high sulfur/sulfate. Cattle submitted between August 2005 and April 2009 were separated into three groups: Group A had neurologic signs or histologic evidence of PEM, as well as confirmed exposure to elevated concentrations of sulfur/sulfate; Group B had neurologic signs and/or histologic evidence of PEM but no confirmed exposure to elevated concentrations of sulfur/sulfate; Group C had confirmed exposure to high sulfur/sulfate although no record of any pathology or clinical signs associated with ST. The following information was recorded when available: age, gender, production type, month of submission, clinical signs, pathology, and source of elevated concentrations of sulfur/sulfate. Statistical analysis was performed on data from Group A. Affected cattle were more likely to be females between three and 16 months of age (age, $P<0.001$; gender, $P=0.022$). A seasonal trend was not detected. Clinical signs in Groups A and B included neurologic signs and gastrointestinal problems (18/29); sudden death was reported in 11/29 cattle. Cattle in Groups A and B had PEM (16), meningoencephalitis (2), cerebral edema (1), gliosis (1), abomasitis (3), and/or cardiac or skeletal muscle degeneration, necrosis, and/or inflammation (5). Undifferentiated feeds were the most common source of sulfur/sulfate toxicosis.

Key words: brain, cattle, corn, distiller grain, polioencephalomalacia, sulfur, sulfate

Résumé

L'intoxication au soufre et au sulfate est une importante cause de polioencéphalomalacie (PEM) et de refus d'aliments entraînant une réduction du gain de poids et une hausse des pertes reliées à la mortalité. Le but de cette étude était de préciser les caractéristiques démographiques des bovins souffrants d'intoxication au soufre/sulfate, de décrire les signes cliniques et la pathologie de la maladie et d'identifier des sources potentielles de soufre et de sulfate élevé. Les bovins reçus entre les mois d'août 2005 et avril 2009 ont été divisés en trois groupes : le groupe A avait des signes neurologiques ou des caractéristiques histologiques reliés à la PEM de même qu'une exposition confirmée à des concentrations élevées de soufre/sulfate; le groupe B montrait des signes neurologiques et/ou des caractéristiques histologiques de la PEM sans confirmation d'une exposition à des concentrations élevées de soufre/sulfate; le groupe C avait une exposition confirmée à des concentrations élevées de soufre/sulfate sans documentation de pathologie ou de signes cliniques associés à l'intoxication. Lorsque disponible, les informations suivantes ont été recueillies : l'âge, le sexe, le type de production, le mois de réception des animaux, les signes cliniques, la pathologie et la source des concentrations élevées de soufre/sulfate. Une analyse statistique a été faite avec les données provenant du groupe A. Les bovins affligés avaient plus de chances d'être des femelles âgées entre trois et 16 mois (âge, $p<0.001$; sexe, $p=0.022$). Une tendance saisonnière n'a pas été détectée pour ce premier groupe. Les signes cliniques dans les groupes A et B incluaient des signes neurologiques et des problèmes gastro-intestinaux (18/39); la mort subite a été rapportée chez 11 bovins sur 29. La PEM (16), la méningo-encéphalite (2), l'œdème cérébral (1), la gliose (1), l'abomasite (3), et/ou une dégénération cardiaque ou des muscles squelettiques, la nécrose et/ou l'inflammation (5) ont été documentés dans les groupes A et B. Des aliments dont les ingrédients n'étaient pas spécifiés étaient la source la plus commune d'intoxication au soufre/sulfate.

Introduction

Polioencephalomalacia (PEM) is a neurologic dis-

ease characterized by acute cerebrocortical neuronal necrosis in cattle and other ruminant species.[6,14] Cattle with PEM often present with neurologic signs, including blindness, ataxia, recumbency, and seizures.[7,14] Cerebral neuronal necrosis can be associated with sulfur/sulfate toxicosis (ST), lead toxicosis, and salt toxicosis.[6,15,16,18,23] It is difficult to differentiate the possible causes of PEM histologically.[16] Ancillary testing on blood or liver, brain, water, and feed is required to confirm the exact etiology of the pathologic changes. Confirmation of ST requires measurement of the feed or water for sulfur or sulfate concentrations, respectively.

Sulfur/sulfate toxicosis is one of the most important causes of PEM in cattle, resulting in increased death losses.[13,23] Clinical signs can occur as early as two hours and can persist as long as one month after exposure to high levels of sulfur/sulfate.[13,14] Toxicosis is also associated with feed refusal, resulting in decreased weight gain.[1] Products consumed by cattle that contain elevated sulfur/sulfate concentrations include feed, water, and consumption of some plants within the *Brassicaceae* family.[10,11,14,22,23] Feed can be secondarily contaminated with sulfur through the application of pesticides that contain pure sulfur, sulfur dioxide, and sulfuric acid. These products are used as fungicides, insecticides, herbicides, fumigants, and fertilizers that are applied to feeds before and after harvest.[17,20] Ground water can be tainted with gypsum rock. Sulfur can also be introduced into the diet directly through medication and supplements.[12,23] With the expansion of the ethanol industry, low-cost distiller's grains and corn gluten with high concentrations of sulfur have flooded the cattle feed market. These feeds increase the risk of exposure to elevated sulfur concentrations, and can result in the development of PEM in cattle consuming rations containing high percentages of these feeds, such as high concentrate diets.[14] High levels of circulating sulfur can also affect circulating copper and selenium levels, resulting in deficiencies in these trace minerals.[7,9]

Concentrations of sulfur and sulfate in feed and water are highly variable and can range from 0 to 32,000 parts per million (ppm). The taste threshold for water in cattle is 2,000 ppm sulfate, and concentrations higher than this can result in feed refusal.[22] It is recommended that cattle consuming water with greater than 500 ppm sulfate have minimal sulfur in the ration.[22] The recommended minimal level of sulfate in the water is less than 500 ppm, and 1,000 ppm is the maximum safe level in cattle fed a diet low in sulfur.[24] Because concentrations of sulfate in wells increases as depth of water increases, cattle can be exposed to higher concentrations of sulfate during droughts.

Concentrations of 3,860 ppm sulfur in feeds resulted in brain lesions in eight-month-old calves.[21] In sheep, diets containing greater than 1,900 ppm sulfur resulted in clinical signs consistent with PEM.[25] However, sulfur and sulfate levels in different sources can have an additive effect, such that high normal concentrations in two separate sources can result in an overall toxic effect.

Given the increase in corn by-products in the cattle feed market, several aspects of ST may have changed, including the at-risk population, sources of elevated sulfur/sulfate concentrations, and the clinical presentation associated with toxicosis. The purpose of this study was to characterize the demographics of cattle with ST, document the clinical signs, describe the associated pathology, and identify the most common sources of elevated sulfur/sulfate concentrations in an effort to identify any changes in the pattern of disease within Oklahoma.

Materials and Methods

The computer database and toxicology files at the Oklahoma Animal Disease Diagnostic Laboratory (OADDL) were searched for any feed consumed by cattle or actual cattle submitted between August 2005 and April 2009 for toxicology or pathology testing. Records were reviewed and the age, gender, production type, month of submission, year of submission, clinical signs, pathologic findings, and sources of high sulfur/sulfate exposure were recorded. Cattle were grouped into three categories based on the following criteria:

Group A: Cattle with neurologic signs provided in the clinical history and/or pathologic changes consistent with PEM *and* documented exposure to high concentrations of sulfur/sulfate.

Group B: Cattle with neurologic signs provided in the clinical history and/or pathologic changes consistent with PEM, but exposure to high sulfur/sulfate concentrations was not documented in the history, or feed/water samples were not submitted for testing.

Group C: Cattle that were exposed to high concentrations of sulfur/sulfate, but had no reported neurologic signs or pathologic changes attributable to ST.

The feed samples, which included pelleted feed and protein tubs, were analyzed for sulfur by ICP/Mass Spectrometry at the Michigan Diagnostic Center for Population and Animal Health. The water was analyzed for sulfate with the Hach Sulfate Test Kit Pocket Colorimeter™ II.[a] This sulfate analysis is based on 4500-SO_4^{2-} Turbidimetric Method.[4] Simple dilutions were made to bring the concentration of sulfate within the analytical range of the instrument. Exposure to high concentrations of sulfur/sulfate was defined as cattle provided with feed or water with sulfur or sulfate concentrations greater than or equal to 2,000 ppm in feed and/or 500 ppm in water.[13,21,22,24]

Information from Group A was used for statistical analysis. SPSS 17.0 for Windows™[,b] was used to perform chi-square analysis. To analyze the effect of gender,

cattle were grouped as female, bulls, or steers. Animals for which gender was not available were excluded from analysis. For analysis of age as a risk factor, cattle were grouped into the following ages: less than three months, four to 10 months, 11 to 15 months, greater than 16 months, and age not reported. These age-groupings were chosen based on beef cattle management practices that are most common in Oklahoma. To determine whether a seasonal trend existed, cattle were grouped based on month of submission as follows: Winter (December, January, February), Spring (March, April, May), Summer (June, July, August), and Fall (September, October, November). Significance was defined as $P<0.05$.

Results

Demographics

A total of 10,318 cattle carcasses or samples from cattle and 151 feed or water samples were submitted to OADDL during the study period. Of these, 40 cattle met the inclusion criteria; Group A = 13, Group B = 16, Group C = 11. The demographics of affected cattle are summarized in Table 1. Based on analysis of the data from Group A, female cattle between three and 16 months of age were the most likely group to be affected (compared to bulls, $P=0.022$; compared to other age groups, $P<0.001$). Neither seasonal nor annual trends were detected.

Clinical Signs

Neurologic signs were common in cattle with confirmed or suspected ST (A = 8/13, B = 11/16). Neurologic signs included blindness (A = 4, B = 4), ataxia (A = 3, B = 3), aggression (A = 1), paddling (A = 1), head pressing (A = 1, B = 1), and seizures (B = 2). A high proportion of cattle with confirmed or suspected ST died without observed clinical signs (A = 5/13, B = 6/16). Non-specific clinical signs in cattle with confirmed or suspected ST included anorexia (B = 1), teeth grinding (A = 2), diarrhea (A = 1), corneal opacity (A = 1), and bloat (A = 1, B = 1).

Pathology

The most common lesion in cattle with confirmed or suspected ST was PEM (A = 5/13, B = 11/16). Grossly, brains of affected cattle had variable fluorescence under a black light which concentrated along the superficial cerebral cortex. Histologically, areas of fluorescence observed grossly corresponded to regions of extensive laminar neuronal necrosis and mild to moderate edema (Figure 1). Other histologic lesions reported in the brain included mononuclear meningoencephalitis (A = 1, B = 1), gliosis (A = 1), and cerebral edema (A = 1).

Surprisingly, there were minimal lesions in the gastrointestinal tract, and lesions were restricted to abomasitis (A = 2, B = 1). Cardiac myonecrosis and/or myocarditis was seen in a small proportion of cattle (A = 1, B = 1). Myodegeneration and/or myositis of the

Table 1. Demographics of 40 cattle with confirmed sulfur/sulfate toxicosis (Group A), suspected sulfur/sulfate toxicosis (Group B), and exposure to high sulfur/sulfate concentrations with no associated clinical or pathologic lesions (Group C). Age, gender, and production type categories are mutually exclusive. Denominators in the final column are the total number of cattle or cattle tissues submitted to the laboratory within that category. Denominators were not available for production type.

	Group A	Group B	Group C	Total
Age				
Less than 3 months	0	0	0	0/538
4 to 10 months	6	6	1	13/1,101
11 to 15 months	2	3	5	10/983
Greater than 16 months	0	2	5	7/5,974
Not given	5	5	0	10/1,722
Gender				
Female	8	9	8	25/5,249
Bull	1	2	3	6/5,056*
Steer	1	5	0	6/5,056*
Not given	3	0	0	3/13
Production Type				
Beef on pasture	5	7	2	14
Beef in feedlot	2	1	1	4
Dairy	0	0	0	0
Not given	6	8	8	22

*The value 5056 includes all male cattle, including bulls and steers.

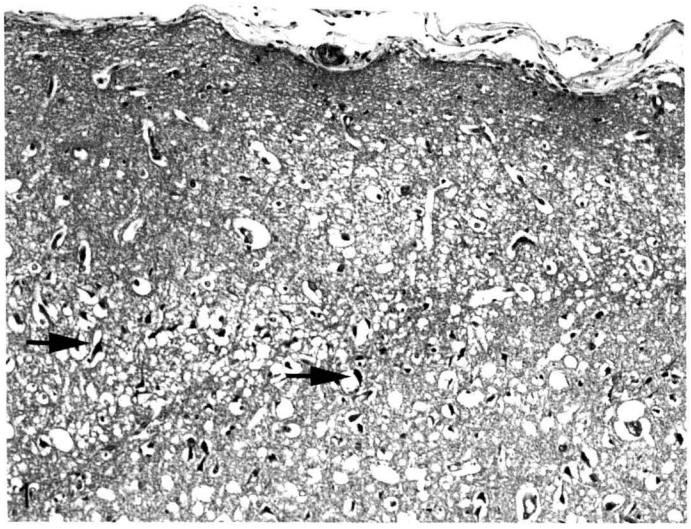

Figure 1. Brain of cow. There is necrosis of the neurons (arrows) within the superficial cerebral cortex with locally extensive edema of the surrounding neuropil.

skeletal muscle were also present in a small fraction of cattle (A = 1, B = 1). Renal tubular necrosis was seen in a single case (A). Bronchopneumonia was also rarely reported in affected cattle (A = 1, B = 1). Mild to moderate hepatic lipidosis was seen in two cattle (A).

Sources of Elevated Sulfur / Sulfate

The sources of elevated sulfur/sulfate concentrations are summarized in Figures 2 and 3. Undifferentiated feeds (i.e. feeds not further specified) were the most common source of high sulfur/sulfate concentrations for both Groups A and C. Interestingly, corn by-products, water, and gypsum that contain high sulfur/sulfate concentrations were all associated with disease (Group A), but protein tubs, liquid feeds, and forage with high concentrates of sulfur were not associated with disease (Group C). These data were surprising because field experience with clients has associated protein tubs and liquid feeds with sulfur toxicosis (Dr. Sandra Morgan, personal communication). Of the feed and water samples submitted, the highest percentage of total submissions with elevated sulfur levels were in the categories "feed other than forage" and "other diet additives or contaminants", including supplements and gypsum.

Discussion

All of the cattle with confirmed or suspected ST in this study were beef cattle either on range or in a feedlot. Another report indicated that cattle housed in feedlots and barns were more predisposed to ST than pasture cattle.[16] In the present study, affected cattle were more likely to be females between three and 16

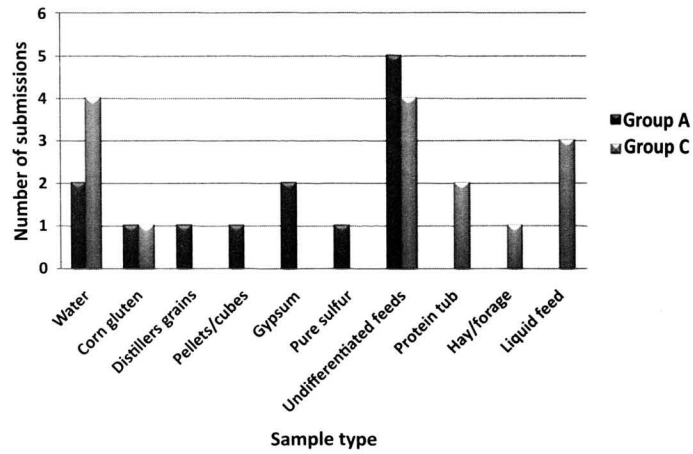

Figure 2. Graph demonstrating the number of submissions with high sulfur/sulfate concentrations by sample type for Groups A and C. (Group A: Cattle with neurologic signs provided in the clinical history and/or pathologic changes consistent with PEM *and* documented exposure to high concentrations of sulfur/sulfate. Group C: Cattle that were exposed to high concentrations of sulfur/sulfate, but had no reported neurologic signs or pathologic changes attributable to ST.)

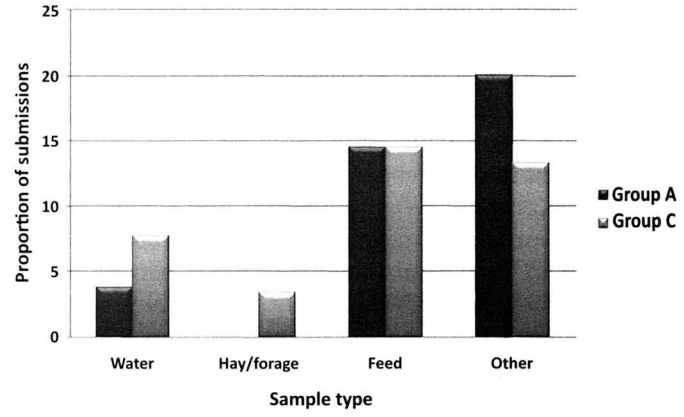

Figure 3. Graph demonstrating the proportion of submissions with high sulfur/sulfate concentrations out of the total number of feed or water samples tested for Groups A and C. (Group A: Cattle with neurologic signs provided in the clinical history and/or pathologic changes consistent with PEM *and* documented exposure to high concentrations of sulfur/sulfate. Group C: Cattle that were exposed to high concentrations of sulfur/sulfate, but had no reported neurologic signs or pathologic changes attributable to ST.)

months of age. This is consistent with another report where cattle less than one to two years of age, primarily stocker calves between the ages of three and eight months, were most susceptible.[16] Another study found

that heifers approximately six months of age consuming water with elevated sulfate concentrations developed PEM, while older cows remained clinically unaffected.[2] In that study, the authors speculated that the concentrations of sulfate in water was in dietary excess for the smaller weight heifers compared to adult cattle.[2] The cause of the age and gender bias in the present study is not known, and was beyond the scope of the project. It is possible that management factors related to young, female beef cattle play a role in the higher incidence of disease in this population. It is also possible that age-related rumen adaptation, including changes in rumen microbial populations, may also play a role.

Gould[7] reported that PEM associated with sulfur/sulfate toxicosis occurs primarily in the summer and fall in range cattle and during the winter in feedlot cattle. Rainfall is thought to promote the accumulation of sulfur in some plants.[19] A seasonal trend did not exist in the present study, most likely because dietary sources of high sulfur/sulfate concentrations did not include sulfur-accumulating plants. Differences in feeding practices and variability in supplementation with products high in sulfur/sulfate may have also played a role.

Previous studies have reported clinical signs including blindness, ataxia, recumbency, head pressing, seizures, and paddling,[2,16,19] which are attributable to massive cerebral cortical damage within the brain associated with PEM. In the present study, similar clinical signs were noted in affected cattle, along with aggression. Other neurologic signs previously reported, but not observed in the present study, include opisthotonos, nystagmus, circling, hypersalivation, and hypersensitivity.[11,14,16] A surprising number of cattle with confirmed or suspected ST died prior to observation of clinical signs. Apparent sudden death was likely because clinical signs in the animal were not observed rather than a true lack of clinical signs prior to death.

Other non-specific signs rarely seen in cattle in the present study included diarrhea, bloat, and teeth grinding. Clinical signs related to the gastrointestinal system, including feed refusal and diarrhea, have been reported with ST in other studies.[13] Once in the rumen, microbes convert inorganic sulfate to hydrogen sulfide.[26] Hydrogen sulfide is then rapidly absorbed into the circulation through the rumen wall.[26] Gastrointestinal disease, such as depressed rumen motility, is thought to be related to toxic effects of free hydrogen sulfide or sulfur dioxide gases, hydrogen sulfide in solution, and/or pentathionic acid.[8,13,15,26] It is possible that the bloat and diarrhea observed in affected cattle in the present study was related to ingestion of high concentrations of sulfur/sulfate. Teeth grinding is a non-specific sign typically associated with pain, which could be related to gastrointestinal upset in cattle with ST. Underlying neurologic disease may also be associated with teeth grinding.

Corneal opacity was observed in one of the cattle with ST in the present study. Corneal opacity in cattle is typically associated with infectious processes, such as infection with *Moraxella bovis* or bovine viral diarrhea virus.[3,27] Exposure to anhydrous ammonia can also cause corneal opacity in cattle.[5] The cause of the corneal opacity in the single case in the present study was not pursued further by the pathologist, and the cause is not known. However, the authors suspect the corneal opacity was unrelated to ST.

Hydrogen sulfide in the gas form can be eructated and inhaled.[13] Because of this, respiratory distress has been noted in cattle with ST.[13] Respiratory distress was not reported for any of the cattle in the present study, including the two animals that had gross and/or histologic evidence of bronchopneumonia.

In cattle with confirmed or suspected ST, PEM was the most common pathologic finding. Polioencephalomalacia associated with ST is well documented.[6,16] The pathogenesis of PEM has been widely speculated, but has not been definitively proven.[16] Earlier reports speculated that gaseous hydrogen sulfide is eructated, breathed in through the lungs, and absorbed into systemic circulation.[13,18] Once there, the hydrogen sulfide reacts with the nerve centers in the brain, resulting in neuronal necrosis.[26] In the present study, additional pathologic findings in the brain included edema, gliosis, and mononuclear meningoencephalitis. Gliosis associated with ST has been previously reported in cattle exposed to high sulfate levels in water.[11]

Lesions within the gastrointestinal tract, including inflammation and congestion of the forestomachs, have been reported with ST in cattle.[26] Sulfurous acid produced by the reaction of sulfur dioxide with water is thought to directly injure the mucosa of the forestomachs.[26] In this study, gastrointestinal lesions were rare and were restricted to a mild to moderate abomasitis; lesions within the rumen and a strong smell of sulfur were not described in any of the cases.

Myocardial degeneration has also been reported in cases of ST.[28] Interestingly, pregnant cattle exposed to sulfur dioxide were at risk to have fetuses, stillborn calves, and calves less than one week of age with degenerative lesions within the heart and skeletal muscle.[28] In the present study, lesions of myodegeneration, necrosis, and inflammation were observed in some of the cases, all of which were over three months of age. The pathogenesis of these changes is not known. These changes may be a result of a direct toxic effect of sulfur or possibly indirectly related through a mechanism of secondary selenium deficiency and subsequent white muscle disease. The changes may also be secondary to hypoxia or other circulatory abnormality. Unfortunately, selenium levels were not available for the animals in the present study.

Data presented in this study suggest that cattle are more likely to be exposed to toxic concentrations of sulfur in feed supplements rather than forage or water. These findings are similar to those previously reported.[16] Given the increased availability of distiller's grains and corn gluten, which might contain high concentrations of sulfur, the authors suspected that ST resulting from consumption of these products would be on the rise. Surprisingly, only three of 28 samples with high sulfur/sulfate concentrations were distiller's grains or corn gluten. One of the 28 samples with elevated concentrations of sulfur was pelleted/cubed feed. Nine of the 28 samples were designated as "undifferentiated feed", which may have also contained corn by-products. These feed products can contain distiller's grains or corn gluten. However, of these three cases where cattle were offered feed that contained high concentrations of sulfur, all were associated with clinical signs and death in the cattle consuming the feed.

The findings described in this paper suggest that feed other than forage and feed supplements are more likely to have elevated sulfur/sulfate concentrations compared to water and forage. However, practitioners should be aware that sulfur toxicosis has an additive effect. For example, a combination of consumption of water with high-normal concentrations of sulfate and feed with high-normal concentrations of sulfur can result in sulfur/sulfate toxicosis and PEM.

Conclusions

Results of this study suggests that female beef cattle between the ages of three and 16 months are the most likely to die from ST. Neurologic signs and gastrointestinal disease are the most common clinical evidence of disease. Histologically, PEM predominates as the defining lesion. Corn by-products, water, and gypsum rock are more likely to contain high concentrations of sulfur/sulfate that cause clinical signs and death rather than protein tubs, mineral blocks, or forage.

Endnotes

[a]Hach Sulfate Test Kit Pocket Colorimeter™ II, Catalog number 58700-29, Loveland, CO
[b]SPSS 17.0 for Windows™, SPSS Inc., Chicago, IL

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