Chronic copper poisoning in sheep grazing pastures fertilized with swine manure

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A flock of 47 crossbred ewes, ranging in age from 2 to 10 years old, had been pastured from July to March on a 12-acre grass lot consisting mainly of fescue. A ram had been placed with the ewes in late summer for planned January lambing. Eight ewes died over a 4-week period from mid-December to mid-January.

Initial examination in December revealed 3 ewes with icterus, anorexia, and signs of depression. All died one day after examination. As other ewes were affected, they became weak, recumbent, and began shaking. One of the 3 ewes had a subnormal rectal temperature (37.2°C), and high heart and respiratory rates (140 beats/min and 35 breaths/min, respectively). Dark red urine dribbled from this ewe’s vulva. Five ewes with no clinical signs of illness were found dead. The 8 dead ewes were scheduled to lamb within the next 2 to 3 weeks. Most died after ambient temperature decreased to approximately 10°C.

A blood sample obtained from one ewe prior to its death revealed hemoglobinemia. Analysis of the serum revealed a high concentration of creatinine (6.2 mg/dl, normal, 0.4 to 1.1 mg/dl) and high activities of alkaline phosphatase (8,400 U/L, normal, 12 to 72 U/L) and aspartate transaminase (2,040 U/L; normal, 0 to 100 U/L). Because of the extreme hemolysis, most of the routine hematologic values could not be ascertained; however, the concentration of hemoglobin was 7.6 g/dl, the PCV was 8%, and numerous Heinz bodies were observed. The concentration of copper in the blood was 194 μg/100 ml.

Necropsy of the 8 ewes revealed generalized icterus, which was so severe in 2 ewes that the brain was stained yellow, and they had splenomegaly, with purplish reddish pulp, large yellowish livers, swollen dark-red kidneys, reddish brown urine in the urinary bladders, and various amounts of fluid in body cavities.

Histologic findings were available for 3 ewes. The major changes were congested spleens with erythrocytosis and hemoglobinuric nephropathy. In one ewe, changes in the liver included scattered centrilobular and paracentral necrosis (a frequent sequela of anemia), characterized by numerous lymphocytes, proliferating bile ducts, and variable sized lobules. One 10-year-old ewe had advanced portal fibrosis with heavy bands of fibrous tissue joining adjacent portal areas and regenerative nodules. Presumably this lesion was the result of insults of excessive copper to the liver over a period of 7 years.

Analysis by an atomic absorption spectrophotometer revealed high concentrations of copper in the livers of 7 ewes ranging from 1,473 mg/kg of body weight to 3,025 mg/kg and high concentrations of copper in kidneys ranging from 62 mg/kg to 630 mg/kg. Copper values from the other dead sheep were not ascertained. Copper concentrations in 2 sheep pastured in the same field, but dying from reasons unrelated to the cause of death in the ewes of this report, were 1,062 and 930 mg/kg in their livers and 54 and 58 mg/kg in their kidneys. Although their liver and kidney copper concentrations were high, necropsy did not reveal these sheep to have lesions resembling copper poisoning. Two sheep on an adjacent farm had copper concentrations of 405 and 460 mg/kg in their livers and 35 and 42 mg/kg in their kidneys.

Because of the high liver, kidney, and blood copper concentrations and the characteristic postmortem findings, a diagnosis of copper intoxication was made. Following the death of the second ewe, the remaining 45 sheep were given ammonium molybdate (300 mg/ewe, PO) and sodium thiosulfate (500 mg/ewe, PO), daily for 4 weeks. The 6 other ewes died within the first 2 weeks of this treatment regimen.

The source of the copper was not readily apparent. The pregnant ewes were being given ground corn concentrate and orchard grass hay in addition to the pasture grass. The corn and hay each contained < 1 mg of copper/kg. The only water source was from the city supply, which contained < 1 μg/L.

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aAllied Instrumental Laboratory, Model No. 8570, Waltham, Mass.
bAll copper and molybdenum concentrations were computed on a dry-weight basis.
of copper/ml. Salt not containing copper was available to all sheep. The sheep owner indicated that the pasture where the sheep were grazing had been treated with swine manure slurry each summer for the preceding 7 years. An analysis of a recent slurry that hadn't yet been spread revealed 85 mg of copper/kg. Sudden deaths in a few sheep had occurred each winter, but the cause of the deaths was not investigated. Four randomly obtained soil samples from the affected pasture were analyzed for copper and molybdenum. The mean copper concentration was 26.35 mg/kg (SD, 4.7). The mean molybdenum concentration was 0.65 mg/kg (SD, 0.11). Four randomly collected forage samples from this same pasture also were analyzed and contained a mean copper concentration of 13.25 mg/kg (SD, 2.85), and a mean molybdenum concentration of 0.45 mg/kg (SD, 0.18). Four randomly obtained soil samples from an adjacent pasture where swine manure had not been applied contained a mean copper concentration of 7.25 mg/kg (SD, 1.5), and a mean molybdenum concentration of 0.85 mg/kg (SD, 0.14).

Copper is a biologically essential element for all animals, because many enzyme systems depend on its presence. Because it is considered essential, this element is routinely added to commercial feeds and trace element preparations. Copper generally is recognized as safe and necessary as a livestock feed ingredient by the FDA. A delicate biological relationship exists between copper, molybdenum, and sulfate, particularly in sheep; however, the addition of molybdenum to livestock feeds and minerals is prohibited because the FDA does not recognize molybdenum as being a safe and essential element. Cattle can tolerate high concentrations of copper and low concentrations of molybdenum in their diets. Sheep, on the other hand, may accumulate large amounts of copper in their livers if the ratio of dietary copper to molybdenum is > 10:1 over a prolonged period. Large amounts of copper can accumulate in sheep livers over several weeks or months without any apparent problems; however, a rapid release of copper from the liver precipitates an intravascular hemolytic crisis. Stress such as strenuous exercise, handling, hauling, or weather changes predispose to the release of the hepatic copper into the blood.

Most cases of copper poisoning in sheep in North America are the result of giving feeder lambs grain containing copper but no molybdenum or using mineral mixes designed for cattle. These supplements often contain copper but no molybdenum. Other less common sources of copper poisoning in sheep are pond water containing copper algicides, ingestion of copper containing forages around mining sites, improper dosing of injectable copper preparations, and ingestion of chicken litter containing excessive copper. Dietary copper may actually be concentrated 2 to 3 times in animal waste material. Diagnosis of copper poisoning is made on the basis of clinical signs consisting of an acute onset of icterus, hemoglobinuria, and recumbency, leading to death, necropsy findings suggesting a hemolytic crisis, and high concentrations of copper in blood, liver, and kidney. Other possible diagnoses include leptospirosis, bacillary hemoglobinuria, babesiosis, and poisoning by rape, onion, and phenothiazine. Treatment of sheep with hemolytic crises from copper toxicosis is unrewarding. Preventing other sheep exposed to excessive copper intakes from developing a hemolytic crisis may be possible. Treatment with ammonium molybdate at the rate of 50 to 500 mg/day and with sodium thiosulfate at 300 to 1,000 mg/day for 3 weeks has had beneficial results. It has been found that within 4 days of initiating this treatment, there is an increase in fecal copper concentration and a reduction in hepatic copper concentration as well as a decrease in the death rate. Penicillamine, a chelating agent that reduces the copper concentration in tissues and increases urinary excretion of copper, is too expensive for practical use in sheep.

Stress has been shown to be a major factor in the induction of hemolytic crises associated with excess hepatic storage for copper in sheep. The 8 ewes that died were all scheduled to lamb, which along with the weather changes, were stressful enough to induce the release of copper into the blood. Administration of ammonium molybdate and sodium thiosulfate to other ewes appeared to prevent additional losses. The other 39 ewes remained clinically normal and lambed without problems.

Copper sulfate is often added to swine feeds at concentrations up to 250 mg/kg for a growth promotant effect. The sheep owners were aware that high concentrations of copper in feed could predispose to problems; however, the owners were unaware of some of the subtle sources of copper such as swine manure.

In this episode of copper toxicosis, the high hepatic concentration of copper was the result of 2 factors—the 7 years of fertilizing the pasture with swine manure rich in copper and the unfavorable ratio of copper to molybdenum in the pasture plants. The copper concentration of the pasture was probably higher earlier in the year, because, at the time the ewes became ill, several months had elapsed since the manure slurry had been applied.

Diminution of aflatoxin toxicity to growing lambs by dietary supplementation with hydrated sodium calcium aluminosilicate

Hydrated sodium calcium aluminosilicate (HSCAS), an anticaking agent for mixed feed, was added to the diets of growing wethers (mean body weight, 34.0 kg) and was evaluated for its ability to diminish the clinical signs of aflatoxicosis. The experimental design consisted of 4 treatment groups of 5 wethers each, consuming concentrations of 0 g of HSCAS and 0 g of aflatoxin (AF)/kg of feed (control; group 1); 20 g of HSCAS/kg (2.0% group 2), 2.6 mg of AF/kg (group 3); or 20 g of HSCAS (2.0%) plus 2.6 mg of AF/kg (group 4). Wethers were maintained in indoor pens, with feed and water available ad libitum for 42 days. Lambs were observed twice daily and weighed weekly, and blood samples were obtained every 2 weeks for hematologic and serum biochemical analyses, and for measurement of mitogen-induced lymphocyte-stimulation index. At the termination of the study, wethers were euthanatized and necropsied. Body weight gain was diminished significantly (P < 0.05) by consumption of 2.6 mg of AF/kg of feed, whereas body weight of lambs consuming HSCAS plus AF did not differ from that of control wethers. The AF-alone treatment increased serum aspartate transaminase and γ-glutamyltransferase activities, prothrombin time, and cholesterol, uric acid, and triglyceride values and decreased albumin, glucose, and urea nitrogen values, and urea:creatinine ratio. A 27% decrease in lymphocyte stimulation index, increased spleen weight (as a percentage of body weight), and decreased liver weight were induced by AF-alone treatment. Results indicate that HSCAS may be a high-affinity sorbent for AF, that 2.6 mg of AF/kg of feed induces signs of aflatoxicosis in growing wethers, that lambs may not be as resistant to the effects of AF as previously thought, that 2.0% HSCAS can substantially reduce the toxic effects of 2.6 mg of AF/kg, and that sorbent compounds may offer a novel approach to the preventive management of aflatoxicosis in livestock.—Roger B. Harvey, Leon F. Kubena, Timothy D. Phillips, et al in Am J Vet Res 52 (January 1991).