1960

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Chronic Copper Poisoning and Pancreatitis in Sheep

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During the fall of 1958 a prominent sheep breeder in central Iowa began to lose some sheep. On October 12, 1958, a ram showed anorexia, and appeared as if he were in pain. Upon palpation, tenderness was noted on the right side of the abdomen, and central nervous system disturbances appeared prior to his death. When he became recumbent, he showed running movements and foamed at the mouth. A necropsy examination was not made.

On December 15, 1958, a nine year old ewe died suddenly, apparently from bloat. Necropsy examination revealed a torsion of the intestine, and forestomachs. The abomasum was impacted with feed and distended with gas.

On April 17, 1959, a yearling ewe was observed in this flock of 210 head which showed symptoms similar to those exhibited by the ram. Necropsy examination revealed an acute diffuse necrotic pancreatitis, and intense icterus, anemia, swelling of the spleen, brown pigmentation of the kidneys, and hemoglobinuria. No additional losses occurred until May 5, 1959, when another ewe showed similar symptoms and died. Necropsy examination revealed an acute diffuse necrotic pancreatitis, icterus, anemia, swelling of the spleen, brown pigmentation of the kidneys, and hemoglobinuria. The pigmentation of the kidneys suggested copper poisoning but excessive amounts of copper were apparently not being fed.

Then on May 12, 1959, a ram was presented for necropsy examination. It had been ill for several days and had shown anorexia, depression, a blood tinged diarrhea, tenderness on the right side of the abdomen, icterus, and hemoglobinuria. Necropsy examination revealed an acute diffuse necrotic pancreatitis, intense icterus, hemorrhagic enteritis, edema and congestion of the lungs, severe fatty degeneration of the liver, focal areas of myocardial necrosis, anemia, and swollen kidneys which had a chocolate-brown color.

Later that same day a ewe died that had shown symptoms similar to those observed in the ram. Necropsy examination revealed an acute diffuse necrotic pancreatitis, intense icterus, slight myocardial degeneration, severe fatty degeneration of the liver, hemoglobinuria, large swollen kidneys which had a dark brown color, and a hemorrhagic enteritis. It was noted that a crust of salt was present on the muzzle of this animal. Necropsy findings, except for the necrotic pancreatitis, were typical of copper poisoning. Histological

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examination of the kidney showed extensive necrosis of the tubular epithelium, tubules distended with a protein material, and an increased cellularity of the glomerular tufts. Microscopic examination of the liver revealed fatty degeneration of the hepatic cells, excessive amounts of blood pigments in the hepatic cells and bile canaliculi, and a moderate interstitial lymphocytic hepatitis. Chemical analysis of the liver revealed 32.24 milligrams of copper per 100 grams of liver.

At this time another ram was observed to be critically ill, showed anorexia, icterus, a blood-tinged diarrhea, hemoglobinuria, and tenderness when the right side of the abdomen was palpated. It was observed that the sick ram was eating large amounts of the mineralized salt. He would eat salt, then drink water, then go back and eat more salt, and then drink more water, but he was not observed to consume any grain or hay. Several other adult animals in the flock were a little slow in movement, but other than that showed no other indication of illness. None of the lambs were or had been ill. In an attempt to determine if leptospirosis was present, the flock was bled. No significant titers were found.

On May 18, 1959, the ram, observed to be ill on May 12, died. A necrotic pancreatitis was not present. There was a moderate hemorrhagic enteritis. The liver showed a moderate fatty degeneration, cirrhosis, and hepatic cell degeneration. There was an acute edema and congestion of the lungs, the animal was icteric, and the kidneys were swollen and dark brown in color. Considerable renal tubular necrosis was present. Chemical analysis of the liver revealed 40.7 milligrams of copper per 100 grams of liver. The excessive amount of copper in the liver indicated chronic copper poisoning was the cause of the icterus, hemoglobinuria, and necrotic pancreatitis in this flock.

An attempt was made to determine the source of the copper. This area of Iowa does not have excessive amounts of copper in the soil, and the feed did not contain more than the normal amount. A new well had been drilled on the farm and an analysis of the water revealed only a trace of copper. The only source of copper in the ration was a mineralized salt mixture which was flavored with anise to increase salt consumption. The analysis on the label indicated the copper content was 0.048 per cent. Analysis of the salt revealed this was the true content.

The work by Boughton and Hardy and others has shown that the feeding of one gram of copper sulfate per day will produce chronic copper poisoning in sheep. They produced copper poisoning when 0.645 grams of copper were fed daily, but they gave no minimum figures as to how little copper sulfate was actually required to produce poisoning.

Some animals in this group of Iowa sheep had a distinct craving for the anise flavored salt mixture. It was observed that the sick sheep would eat salt, then drink water, eat more salt, and drink more water. The animals would not eat roughage and concentrates available to them. It was not determined whether they ate 50, 100, or 200 grams of salt a day. The flock, as a whole, had consumed an unusually large amount of salt, and it is quite probable that some animals in the flock consumed a great deal more than did others.

If the copper content in the medicated salt is converted to hydrated copper sulfate equivalent, the amount of copper in this mineral salt mixture would represent 0.277 grams of copper sulfate per 100 grams of mineralized salt mixture. If one animal consumed 200 grams of this mineralized salt mixture, 0.555 grams of copper sulfate would have been consumed or nearly as much copper sulfate as was used by Boughton and Hardy when they consistently produced copper poisoning in sheep with 0.645 grams of copper sulfate.

The copper content of the livers of sheep which died from copper poisoning, as reported by Boughton and Hardy, ranged from 14.7 to 81.2 milligrams per 100 grams of liver. The copper content of the livers of normal sheep ranged from 0.48 to 4.19 milligrams of copper per 100 grams of liver. The copper content of the
liver of a ewe, necropsied on May 12, was 32.24 milligrams per 100 grams of liver. The analysis of the liver of the ram, necropsied on May 18, revealed 40.7 milligrams of copper per 100 grams of liver. Both of these analyses fall within the range of the figures given by Boughton and Hardy in fatal cases of copper poisoning, and is considerably greater than the amount of copper normally found in the liver. This indicates the mortality observed in this flock of sheep was due to chronic copper poisoning. When the mineralized salt mixture was discontinued, no additional deaths occurred.

The association of acute diffuse necrotic pancreatitis with copper poisoning is of extreme interest because this lesion was not reported by Boughton and Hardy and others in their descriptions of copper poisoning. This observation is also of importance because the necrotic pancreatitis and peritonitis was so severe that it was believed at first that the pancreatitis was the cause of the icterus and deaths in this flock. The role of copper poisoning, although suspected, was not considered seriously until a number of animals had died. The pathogenesis of the pancreatitis was not determined.

Many flocks of sheep in Iowa experience a few deaths each year. Some of these animals show icterus, anemia, hemoglobinuria, dark brown kidneys, and a swollen spleen. Most of these animals are receiving a mineralized salt mixture which contains copper. Occasionally animals are observed which have a necrotic pancreatitis. It is suspected that some of these, perhaps all, are chronic copper poisoning.

In Iowa, sporadic deaths occur in cattle in which the principle lesions are anemia, icterus, swelling of the spleen, dark brown kidneys, and hemoglobinuria. These cattle are also receiving mineral mixtures in which copper is present. Although copper poisoning in cattle is usually not recognized, Schaper and Luetje report losses in both cattle and sheep exposed to copper in Bordeaux mixture. It is quite possible that some cattle will consume excessive amounts of mineralized salt containing copper, and that copper poisoning will result just as it does in sheep.

**CONCLUSION**

1. Mineralized salt, containing traces of copper, may cause chronic copper poisoning if excessive amounts of salt are consumed.

2. The use of flavoring agents, such as anise, to increase salt consumption should be discouraged because some animals consume excessive amounts of the medicated salt.

3. Necrotic pancreatitis in sheep can be caused by chronic copper poisoning.

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**Arthritic Specimens Wanted**

A National Institutes of Health grant for the study of arthritis in domestic animals has been received by the Veterinary Medical Research Institute, College of Veterinary Medicine, Iowa State University. This project is under the supervision of W. P. Switzer and will encompass attempts to isolate bacteria, pleuropneumonia-like organisms and viruses from joint specimens. Dr. R. F. Ross is assisting this research. Material from all species of domestic animals will be examined, but special emphasis will be placed on the arthritis complex of swine, sheep and cattle.

Practitioners who encounter arthritis problems in their practice are invited to submit specimens for examination. The procedure for doing this is to first contact Dr. Switzer or Dr. Ross, fill out a case history and then submit a fresh specimen for examination. The cooperation of all Iowa practitioners is urged and all results secured will be made available to the practitioner submitting the specimen.

Your cooperation in this project will not only furnish additional information about the arthritis problems in your practice but will aid in increasing our knowledge about the overall problems of arthritis in animals.