1-13-89

Reynolds Electrical & Engineering Co., Inc.
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IN REPLY REFER TO:
500-02-95

JAN 13 1989

L. H. Dodgion, Administrator
Division of Environmental Protection
Nevada Department of Conservation
and Natural Resources
201 South Fall Street, Room 221
Carson City, Nevada 89710

Re: Urea Incident/Order of December 2, 1988

Dear Mr. Dodgion:

Enclosed please find the following packets of documents which we present in response to the referenced Division Order:

1. A narrative description of the events eading up to the incident of November 2, 1988; results of water samplings taken; a hydrogeological report; and information regarding human health consequences.

2. REECo plans and procedures for the proper handling of hazardous substances, as well as Company procedures for the proper handling of urea in the future.

As our representatives discussed with you at the meeting of December 20 in Carson City, REECo sincerely regrets that this unfortunate accident ever occurred, and is willing to work with the State to resolve the problem in an appropriate manner. To this end, you will be receiving under separate cover a REECo proposal which we hope the State will accept as alternative to pursuing further litigation in the matter.

Finally, as you have already discussed with Greg McKenna of our Legal Department, we are filing a Petition for Hearing with the State Environmental Commission on the matter. We have enclosed a copy of the Petition for your Division records. Again, please understand that by this action we do not intend to adopt an adversarial position with the State, but only to generally protect REECo's interests until the present situation is resolved.

JAN 13 1989

I look forward to meeting you on January 23 and discussing all of these mutual concerns in greater detail. Should you have any questions in the interim, do not hesitate to contact me or Arthur Williams, REECo General Counsel, at (702) 295-2225.

Very truly yours,

D. L. Fraser
General Manager

DLF:FGM:dm

Enclosures
As stated

cc: Terri Jay, w/encl.
Commission for the Preservation
of Wild Horses
Stewart Facility
Capitol Complex
Carson City, Nevada 89710

CLINICAL AND DIAGNOSTIC VETERINARY TOXICOLOGY

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DUBUQUE IOWA

UREA AND NONPROTEIN NITROGEN

Urea and other nonprotein nitrogen compounds are used as substitutes for natural protein in ruminant feeds. Although urea and related compounds are considered to be feed ingredients, if not used properly and under certain nutritional conditions, they can become toxicants that are highly lethal. This section will be concerned primarily with urea, since problems associated with its use have been the most prominent.

Source

The more common sources of nonprotein nitrogen (NPN) include urea, biuret, and diammonium phosphate. Ammonium chloride also is used to reduce the incidence of urolithiasis in cattle and sheep and as an expectorant for swine.

Urea is generally recommended in ruminant rations at a rate of approximately 3 percent of the grain ration or about 1 percent of the total ration. More recently, however, formulations increasing these levels have been recommended and appear to be in general use.

Biuret is recommended in ruminant rations at levels approximating 3 percent of the total ration. Ammonium chloride may be used at a rate of 0.75-1.5 ounces for cattle and 0.25 ounces for sheep/head/day to reduce urolithiasis. Diammonium phosphate may be used to furnish about one-third of the nitrogen requirements for ruminants.

Toxicity

The toxicity of urea and other NPN formulations is dependent upon their hydrolysis to ammonia. Cattle and other ruminants are the most susceptible because their rumen contains urease and is an ideal environment for hydrolysis of urea, releasing carbon dioxide and ammonia. Horses are mildly susceptible to urea but are more susceptible to ammonium salts. Monogastric animals are not susceptible to urea poisoning but are susceptible to poisoning by ammonium salts.

Circumstances which usually result in urea toxicosis often involve (1) improper mixing or formulation of NPN rations; (2) feeding urea to ruminants unaccustomed to NPN or animals which have been starved; (3) using high levels of urea in rations low in energy and protein and high in fiber; and (4) giving animals free access to a palatable source of urea concentrate.

In ruminants, urea usually is lethal at 1-1.5 gm/kg body weight; 0.3-0.5 gm/kg may be toxic. Urea phosphate usually is toxic at 1 gm/kg. Ammonium salts and diammonium phosphate may be lethal at 1-2 gm/kg (Singer, 1969).

In horses, urea is lethal at approximately 4 gm/kg body weight (Hintz *et al.*, 1970). Ammonium salts may be lethal at 1.5 gm/kg body weight.

In monogastric animals, urea and biuret have very low toxicity. Ammonium salts are toxic at approximately 1.5 gm/kg body weight (Bicknell, 1965).

Urea toxicity varies with the age of ruminant animals. The very young ruminant has very low susceptibility because its rumen flora have not developed. After rumen development, the younger animals appear to be more susceptible than older animals. Cattle and sheep adapt to the feeding of urea rather quickly but also quickly lose their adaptation. By slowly increasing the amount of urea fed, the ruminant can tolerate as much as 1 gram urea/kg body weight daily. In practice, however, the feeding of very high levels of urea may be dangerous because animals may go off feed during adverse weather conditions or digestive upsets and quickly lose their tolerance for urea. Then after coming back on full feed, they may be poisoned by the high levels of urea.

Some predisposing factors for urea toxicosis in ruminants include (1) fasting, (2) high roughage diets, (3) lack of conditioning to high NPN diets, (4) high ruminal pH, (5) high rumen and body temperature, and (6) dehydration or low-water intake.

It is often imperative to be able to calculate the concentration of urea in a feed. Pure urea equals 292 percent protein equivalent; however, commercial urea equals 262-280 percent protein equivalent. Thus, 1 pound of pure urea is equivalent to 2.92 pounds of NPN protein. If a label indicates 64 percent protein from a urea or NPN source, the concentration of urea is 64/292 equals 22 percent urea. If a feed contains 10 percent urea, the NPN protein is $10 \times 2.92 = 29.2$ percent.

Mechanism of Action

Toxicosis from NPN formulations results from the absorption of NH_3 from the gastrointestinal tract. Ammonia from urea and ammonium salts is absorbed into the bloodstream more rapidly when rumino-reticulum pH is high (8.0 or above). An alkaline reaction also enhances urease-ureolysis to NH_3 and CO_2. At a pH of 7.0 or below, the ammonia is in the form of ammonium ion (NH_4^+) and, thus, would not readily be absorbed through the gastrointestinal wall nor would it be available in the gaseous form for eructation.

Although alkalosis of the rumen occurs during urea toxicosis, a systemic alkalosis does not occur. In fact, a metabolic acidosis develops but is not the probable cause of death. Lloyd (1970) has shown that blood pH drops from 7.4 to 7.0 at the time of urea-induced death. There is an apparent inhibition of the citrate cycle with resulting compensatory anaerobic glycolysis. Highly significant increases in packed cell volume, blood ammonia, blood glucose, blood urea nitrogen, serum potassium and phosphorus, blood lactate, SGOT, SGPT, and rumen pH occur during urea toxicosis in cattle and sheep. There are concomitant decreases in blood pH and urine excretion. Death probably is due to hyperkalemic cardiac blockage and cessation of respiration (Lloyd, 1970).

Some workers have theorized that since urea is hydrolyzed into ammonia in the rumen, it follows that it would be eructated by the ruminant and aspirated into the respiratory tract, causing irritation and increased susceptibility to respiratory infections. While it is true that urea is hydrolyzed into ammonia under normal conditions of digestion, over 99 percent of the released ammonia is in the form of NH_4OH which is nongaseous. Also, that NH_3 which is present is soluble in the liquid portion of the rumen contents and would also be nongaseous. Therefore, unless the rumen pH was elevated to 8-9, no gaseous NH_3 would be available for eructation. If the rumen pH becomes this high, the animal will show overt signs of ammonia toxicosis. It is plausible to assume that if ammonia were present in the gaseous form in the rumen, it could be aspirated into the respiratory tract, since Dougherty and Cook (1962) have shown that a major percentage of the ruminal gases are aspirated into the respiratory system at the time of eructation.

Clinical Signs

Toxic manifestations usually occur in ruminants when the concentrations of ammonia reach 80 mg/100 ml in rumen fluid and 2 mg/100 ml in serum or whole blood. The clinical course of urea toxicosis usually is rapid and acute, from 10 minutes to as much as 4 hours after consumption. Clinical signs, indicating abdominal pain, include frothy salivation, grinding of the teeth, and kicking at the abdomen. There usually is polyuria, muscle tremors, incoordination, weakness, forced rapid breathing, bloat, violent struggling and bellowing, and terminal tetanic spasms. There usually is a marked jugular pulse. Respiration often is forced and rapid. Toward the terminal stages, vomiting is especially common in sheep. Hyperthermia and anuria usually are evident just prior to death.

Physiopathology

Upon opening the rumen of an animal which has recently died of urea toxicosis, one occasionally can detect the odor of ammonia. There are no characteristic lesions of urea-NPN poisoning. Pulmonary edema, congestion, and petechial hemorrhages are fairly common findings. There may be a mild bronchitis, and commonly rumen ingesta is found in the trachea and bronchi, especially in sheep. There may be a catarrhal gastroenteritis.

Animals dead of urea toxicosis often are extremely bloated, and the carcass appears to decompose quite rapidly. Body tissues taken for diagnostic purposes should be obtained from an animal which has recently died. As a carcass is allowed to decompose, the breakdown of the natural protein in the tissues and stomach contents will result in a buildup of ammonia, which would tend to lead one diagnostically astray.

Diagnosis

Diagnosis of urea or NPN poisoning is based on the history of acute illness following consumption of feed containing urea or other NPN formulations. It usually is very important that the feed be analyzed for urea content and calculations be made to determine the possibility of poisoning under the conditions of consumption. If the clinical signs and necropsy findings are compatible with urea toxicosis, the diagnosis may be confirmed by analysis for ammonia in whole blood, serum, ruminal fluid, and urine. The specimens should be frozen immediately and thawed only at the time of analysis *or* preserved by the addition of saturated mercury chloride to stop enzymatic action on the natural protein. If the specimens are not frozen or preserved, proteolytic enzymes will break down the amino acids and tissue proteins, releasing ammonia.

Thus, the longer a cadaver decomposes, the higher will be the ammonia content.

In most cases of urea poisoning, the rumen ammonia content will be greater than 80 mg/100 ml and may be as high as 2,000 mg/100 ml. It is important to take samples from several areas in the rumen and reticulum for ammonia analysis because it is possible that an animal may die before the urea that has been consumed has a chance to evenly distribute throughout the rumen and reticulum. Blood or serum ammonia nitrogen concentrations between 2-4 mg/100 ml or greater would be compatible with a diagnosis of NPN (ammonia) poisoning.

Conditions that may be confused with urea toxicosis include acute encephalitic diseases such as thromboembolic meningoencephalitis and polioencephalomalacia, grain engorgement, acute nitrate and cyanide poisoning, organophosphorous and chlorinated hydrocarbon insecticide poisoning, and enterotoxemia.

Treatment

The best treatment for urea poisoning is to give several gallons of cold water orally. As much as 5-10 gallons should be given to an adult cow. If 5 percent acetic acid or vinegar is available, 1 gallon should be given along with the cold water. The rationale for this treatment is that the cold water will reduce the temperature of the rumen and lower the pH, thereby slowing down hydrolysis of the urea to ammonia. In addition, the water tends to dilute the ammonia already present, lessening the concentration available for absorption into the bloodstream. The water also acts as a diuretic. Lloyd (1970) has shown that as long as an animal had adequate urine output, death did not occur during urea toxicosis.

If bloat accompanies urea toxicosis, it should be relieved; and Lloyd (1970) has suggested the administration of normal saline with magnesium and calcium solutions intravenously.

Case History

An Iowa farmer lost eight black Angus stock cows 8-10 hours after giving them a supplement containing urea. The herd of registered Angus cows consisted of 61 adults and 5 calves. They had been pastured in a cornstalk field for at least two months, and the owner decided they needed some pelleted supplement since it was midwinter. He inquired with the local feed dealer as to what supplement he should use. He was given a pelleted supplement called "Beef 64," which the label indicated contained 61 percent protein from a nonprotein nitrogen source. The instructions on the label recommended that it not be fed by itself but that it be mixed thoroughly with the grain ration before feeding. It also recommended that no more than 1 lb/head/day be fed. A warning on the label suggested that the supplement was designed for fattening cattle on full feed of grain. However, contrary to the recommendations on the label, the feed representative told the farmer that he could safely feed these pellets to the cattle by pouring the pellets on the ground in the cornfield. On January 15, the owner fed 60 pounds of these pellets to 60 cows and 5 calves. No problems were noted. The next day he did not feed the cows. On January 17, he fed the same amount as on January 15. Eight to ten hours later, eight animals had died. A veterinarian was called and noted the following signs: staggering, unstable walk, foaming at the mouth, rapid breathing, and hyperirritability. Those animals which died went down on their side in tetanic convulsive seizures. The following day, all surviving animals appeared normal. Samples of the pelleted feed were submitted for chemical analysis and found to contain 24 percent urea. Rumen fluid specimens taken from an animal which had recently died contained an average of 120 mg ammonia nitrogen per 100 ml. Whole blood from an affected animal was found to contain 4.5 mg ammonia nitrogen per 100 ml.

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