

# Bovine Rumensin Toxicity—A Case Study

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One hundred seventeen yearling and two year old cattle, in a winter feeding program of 1994, died within 42 days of being fed toxic levels of Monensin (Rumensin<sup>1</sup>) in a liquid protein supplement (CMS Loomix). Death loss commenced on the third day after the toxic feed was administered and followed clinic signs of anorexia, pica, diarrhea, depression, mild, posterior ataxia and dyspnea. Grossly observable lesions at necropsy of animals dying in the acute phase of the illness included hydrothorax, ascites, pulmonary edema with hemorrhages and myocardial hemorrhage, edema and necrosis. Animals dying at greater than ten days after the toxic feeding showed scarring of heart and skeletal muscle, generalized ventral edema, enlarged, firm, bluish discolored livers, and enlarged right heart.

Microscopic changes in animals dying in the acute phase consisted of pulmonary edema, congestion and hemorrhage, as well as localized areas of edema, hemorrhage and necrosis in cardiac and skeletal muscle. In cattle that died after ten days or more of illness, lymphocytic infiltrate, sarcolemmal nuclear proliferation and fibrosis of skeletal and cardiac muscle were remarkable.

Changes varied somewhat according to the area of heart or skeletal muscle that was affected. Active muscles such as those in the diaphragm were most severely affected.

## History

This cattle operation conducts an extensive and intensive breeding operation, performance testing of bulls, and a feeding program. Five groups of animals (Table 1) were involved with monensin toxicity. These five groups of animals were maintained in dry feedlots.

The feeding program has utilized monensin in the granular form as a feed additive for the past five years with satisfactory results. During the winter feeding period of 1980-81 a liquid protein supplement (CMS Loomix) was incorporated into the ration. The liquid supplement (CMS Loomix) contains vitamins, minerals, nonprotein nitrogen, and monensin calculated at 1200 grams per ton in a molasses base. The supplement was fed at the rate of 0.42 lbs per head

<sup>1</sup>Rumensin, Elanco Products, Division Eli Lilly and Co., Indianapolis, IN.

Table 1. Death loss by group.

Group	A	B	C	D	E
<b>Date</b>					
3/7	2				
3/8	19				
3/9	4	3			
3/10	3	1			
3/11	2			1	
3/12	9			3	
3/13	5			1	2 abortions
3/14	10	1			
3/15	6			1	
3/16	6				
3/17	4				
3/18	3				
3/19					
3/20	3			1	
3/21	3	1		1	
3/22	4				
3/23	2				
3/24	1	1		1	
3/25					
3/26	1			1	
3/27	2			1	
3/28					
3/29	1				
3/30	2				
4/1	2				
4/2				1	
4/7					2
4/9				1	
4/20				1	
Totals:	94	7		14	2
GRAND TOTAL:	117				

Group A: 1043, 650# 1 year old females.

Group B: 16, 600# 1 year old castrated males and 20, 1000# 8-10 year old females.

Group C: 285, 907# 1 year old males.

Group D: 500, 800# 2 year old female calves.

Group E: 130, 900# 2 year old females in late gestation.

to provide 252 mg. of monensin per animal per day.

A 5,000 gallon upright tank equipped with a recirculating pump was installed for storing the liquid supplement. This tank had a square base and the drainage outlet was near one side. The liquid supplement is recirculated for a minimum of 30 minutes prior to being sprinkled on top of the concentrates.

The rations for each group are listed in Table 2.

A harsh mobile mix feed truck is used in the feeding operation. The concentrates of each ration are placed in the

Table 2. Daily ration per head by group.

Group A - Yearling Females

3# grain  
3# chopped hay (alfalfa)  
1/2# cottonseed meal  
.42 lb Loomix (252 mg rumensin calculated)  
23# Silage

Group B - Steers and Cull Cows

Fed same rations as yearling females

Group C - Yearling Bulls

20# silage  
4# ground alfalfa  
10# grain  
1# cotton seed meal  
0.42# liquid supplement

Group D - 2 Year Old Heifers With Calves

5# chopped hay (alfalfa)  
5# grain  
.42# Loomix (252 mg rumensin calculated)  
.5# cottonseed meal  
35-38# ensilage

Group E - 2 Year Old Heifers, Late Gestation

7# grass hay chopped  
2# grain  
.42# Loomix (252 mg rumensin calculated)  
1/2# cottonseed meal  
Ensilage

truck and the total ration is continuously mixed during the loading and delivery to the feedlots.

The first load of liquid supplement was received 10-22-80 and fed completely before the second load was delivered on 1-23-81. Following the feeding of the last of this first tank of liquid supplement all five groups of animals went off feed for two days but returned to normal feeding in 2-3 days.

The second load arrived late at night and was not unloaded until the next morning. During this overnight stand, settling of materials occurred with 540 lbs of supplement remaining as sludge in the bottom of the delivering tanker.

All groups of animals received CSM Loomix supplement on March 4, 1981. The last of the second load of liquid supplement was fed on March 5, 1981. Two loads of feed received the "bottom of the tank" liquid supplement with the first load being fed to Group C yearling bulls and the final

load from the bottom of the tank being fed the Group A yearling heifers. Later in the day Group A (yearling heifers) received three more loads of feed containing no supplement. Clinical illness in the form of diarrhea was noticed in Group E (2 year old heifers without calves) on March 4, 1981. Diarrhea and anorexia was noticed in Group A (yearling heifers) and Group C (yearling bulls) on March 5, 1981. All feed bunks were cleaned of remaining feed and all groups received grass hay from March 6-11, 1981. Death loss commenced in Group A animals on March 7, 1981 and continued through April 20, 1981.

**Pathology**

Deaths losses occurred over a thirty day period and post mortem examinations were conducted on animals dying or euthanized on days four, five, six, nine, fourteen, twenty and twenty-six after the last feed containing monensin was consumed.

Even though outside temperatures were cool, post mortem decomposition was made to examine animals as quickly after death as possible.

Both gross and histopathologic lesions changed with increasing time between the last exposure and death. In early cases there were scattered ecchymotic hemorrhages in the subcutis and fascia around the neck and front limbs. Excess clear yellow fluid was in the thorax. Lungs were heavy and wet and failed to completely collapse. Interlobular septae were mildly congested early, with more marked congestion on days six and nine. Froth and fluid was variably present in the trachea and bronchi.

Pericardial fluid was mildly increased and reddened, and localized hemorrhages were frequently seen in myocardium and around the main pulmonary trunk. Myocardium was mottled and pale yellow-pink to red. Abomasal mucosa was reddened and patchy to segmental areas of diffuse hemorrhage were in the small intestine.

On day nine there were definite pale areas in myocardium and skeletal muscles such as sublumbar, caudal thighs, and diaphragm. Lungs were similar to earlier cases but were slightly increased in consistency and lobules in ventral portions were accentuated by hemorrhage and congestion. Intestinal changes were similar, and thoracic fluid was increased.

During the remainder of the losses intestinal lesions were not seen. Pale areas and streaks as well as localized hemorrhages were seen in cardiac muscle, abdominal and thigh muscles, and in the diaphragm.

Lungs were increasingly more reddened and increased in consistency in ventral portions. This was extreme in one animal in which the left ventricular chamber was markedly dilated and interventricular and left ventricular myocardium was thinned. In other animals mild to moderate dilatation of the right ventral was present, and these animals were also characterized by accumulation of edema in the

subcutis along the ventral midline, in mesentery, and subserosally. The livers of these animals were variably characterized as pale, swollen, reticulated and increased in consistency as is typically seen in chronic passive congestion.

Microscopically, changes in early cases were minimal. Gross impressions were confirmed in lung and GI tract. In heart, individual myocardial fibers were spread, suggesting edema. Sarcoplasm was granular and mildly vacuolated. There was an overall mild increase of mononuclear and polymorphonuclear inflammatory cells between fibers. In a few small foci, fibers were disrupted and focal aggregates of principally mononuclear inflammatory cells and myonuclear proliferation were present. These changes became more prominent and had a patchy distribution by day six. Myorrhesis, loss of cross striations, and nuclear pyknosis were evident in these foci. By day nine these changes were widespread in all ventricular myocardium. There was inflammatory infiltrate comprised of lymphocytes, macrophages, neutrophils, and eosinophils present as well. In a few areas fibroplasia was beginning. Similar but less severe changes were seen in the skeletal muscles in which gross abnormalities were detected.

In time these foci were characterized by rarefaction of fibers, fibroplasia, mild lymphocytic infiltration and an overall decrease in size of myocardial fibers. In the last animal examined lesions in myocardial and skeletal muscle were difficult to detect, but lesions associated with chronic right heart failure were widespread in other organs.

Lesions in other tissues were variable. In a few early cases these were vacuolation of hepatocytes and mild centrilobular necrosis. No significant lesion was seen in rumen, kidney, brain, pituitary glands, adrenal glands, thyroid glands, spleen or lymph nodes in animals in which these tissues were examined.

### Laboratory Findings

Normal enteric organisms were isolated from hemorrhagic lesions in the intestine. No organisms were isolated from liver, muscle or lung with the exception of one animal which had inhaled rumen content agonally.

Virus isolation on lung, heart and liver of several animals

was negative, and EM scan of feces from animals with enteric lesions was also negative.

Liver and kidney of several animals were negative for lead, mercury and arsenic. Copper levels in tissue and serum were mildly depressed and molybdenum was mildly increased, but not beyond the normal ranges for healthy animals in that area.

A third load of monensin was delivered to the ranch and placed in the 5,000 gallon holding tank. This was not fed to any group of animals. On March 17, 1981 representatives from the Food and Drug Administration from the Regional Office in Denver collected samples for analysis. As part of the ranch operation deals with the sale of performance tested bulls, cardiac soundness is of extreme importance. Two hundred and four bulls were examined with electrocardiogram. All were normal except for three animals which showed some indication of heart abnormalities, not necessarily associated with monensin toxicity and heart muscle damage. Ten bulls were selected for tissue study. These were slaughtered in a local slaughtering establishment and histology was conducted on the heart and lung tissues. These tissues were normal. Blood enzyme tests were also conducted on a random sample of ten bulls, all of which were normal.

**In reviewing the epidemiological findings on this case of monensin toxicity, it is postulated that the reason for the increased number of deaths occurring in the yearling heifer group, is a dose/weight relationship. The other factor involved is that this group of animals received the very last portion of liquid supplement at the bottom of the tank. The deaths in the yearling heifers were primarily in the larger, more aggressive heifers. This, of course, puts some question on the dose relationship but when viewing the total amount of feedbunk availability, the smaller heifers were not allowed to the first feeding which contained the toxic level of monensin. The other groups where death losses occurred were in the two year old heifers with calves at side and the yearling steers. The yearling steers and cull cows running together again indicates a dose/weight relationship. Although the two year old heifers with calves at side were heavier, it is postulated there may be some relationship due to stress on this particular group of animals.**

