CASE REPORT

Possible Monensin Poisoning in a Group of Bulls

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Summary

Apparent monensin toxicity was diagnosed in a group of Maine Anjou bulls placed on a performance test in a commercial feedyard in east central Saskatchewan. The clinical, hematological and pathological findings were consistent with a diagnosis of congestive heart failure. The suspected dose ingested by the bulls was based on a mixing error evident from the feedlot's daily feed journal. When deaths in the group of bulls began to occur, the intoxicating feedstuff was no longer available for confirmation of the diagnosis.

Résumé

Intoxication probable à la monensine, au sein d'un groupe de taureaux

Les auteurs ont diagnostiqué une intoxication apparente à la monensine, au sein d'un groupe de taureaux Maine Anjou, soumis à un test de performance, dans un parc d'engraissement commercial du centre est de la Saskatchewan. Leurs observations cliniques, hématologiques et pathologiques se révélèrent compatibles avec une défaillance cardiaque congestive. La dose suspecte ingérée par les taureaux s'avéra imputable à une erreur de mélange évidente, comme le démontrèrent les dossiers quotidiens de l'alimentation. Lorsque la mortalité commença à sévir, il ne restait plus de moulée suspecte pour confirmer le diagnostic.

Introduction

Monensin is a feed additive which alters rumen fermentation and improves feed efficiency and rate of gain in cattle. Poisoning of cattle and horses with monensin has been reported recently (1,2,3,4). Because there is a wide margin of safety in cattle (5) it is thought that fatal cases of monensin toxicity in cattle are associated with the accidental consumption of lethal amounts of the drug. This may occur following errors in the mixing of feed in which monensin is added at levels considerably in excess of the amounts used as a feed additive.

This is a report of suspected monensin toxicity in a group of 86 Maine Anjou bulls on a performance test in a commercial feedlot in east central Saskatchewan.

History

Maine Anjou breeders in the east central area of Saskatchewan requested a commercial feedlot in the area to provide facilities and feed for a 140-day performance test similar to the one provided by the Central Record of Performance Beef Bull Test Station located at the University of Saskatchewan in Saskatoon. Bulls from 38 premises which were born between February 25 and May 15, 1979 were presented to the feedlot from October 14 to 16. They were on an adjustment diet from October 14 to November 13, after which they were fed the test diet which was 60% cereal silage, 31.8% oats and the remainder a commercial 32% beef cattle protein supplement on an "as is" weight basis. At no time were these bulls to receive any monensin in the ration.

For different reasons, from November 13 to December 22, four bulls were removed from the test pen. One died with undifferentiated bovine respiratory disease, another died from *Hemophilus* meningoencephalitis; one was returned due to lack of gain and one was sent home as a "buller".[1] A reduction in feed consumption between December 2 to 4 and December 22 and 23 was the only indication that something was abnormal. On December 30, bull no. 1 died, followed by no.'s 2 and 3 on December 31 and no.'s 4 and 5 on January 2. On January 2 three of the dead bulls were submitted to the Diagnostic Laboratory at the Western College of Veterinary Medicine. Necropsy examination of these bulls in various stages of decomposition revealed little more than terminal bloat and embolic pneumonia. At this time an error in feed processing or mixing was considered as a cause of the bloat and portions of the supplement in the test diet were withdrawn. On January 7 and 8, three additional bulls were obviously sick but not bloated. The referring veterinarian noted that the bulls were depressed, inactive and in mild respiratory distress. Two of these bulls (No.'s 7 and 8) were admitted to the Large Animal Clinic (WCVM) for detailed clinical and laboratory examination. The third affected bull (No. 6) collapsed and died while walking from the affected pen to the loading and handling area, a distance of approximately 100 m.

Clinical and Laboratory Findings

The clinical findings were similar in both bulls. There was moderate depression and the bulls were inactive and walked slowly. The rectal temperatures were 38.5-39°C, the heart rates

[1]"Buller" here refers to an animal that for unidentified reasons becomes sexually attractive within his group and is excessively handled and ridden by the more aggressive members in the pen.

100-120 per minute and the respiratory rates 48-60 per minute. The heart sounds were muffled but other abnormalities of the heart sounds were not audible. The jugular veins were markedly engorged and the jugular pulse was exaggerated. There was prominent subcutaneous edema of the brisket and slight edema of the intermandibular space. The respirations were slightly labored due to a prolonged expiration. Auscultation of the trachea revealed a mild expiratory grunt. Inspiration was slightly prolonged. The lung sounds were slightly louder than normal on both inspiration and expiration. There were no significant abnormal lung sounds. The rumens were about two-thirds full and static. The bulls would not eat when offered hay but drank small amounts of water. The feces were normal but scant. There was no dehydration.

The bulls were hospitalized for further observation. Both bulls were treated with oxytetracycline because of the possibility of pneumonia. The hemograms were not remarkable but suggested a chronic infection (Table I). Bull no. 7 became much worse the following day and was euthanitized and a necropsy done immediately. Bull no. 8 became steadily worse, remaining inactive, anorexic and in sternal recumbency most of the time. Respiration became progressively more labored and the edema of the brisket became more prominent. It was euthanized on the fourth day of hospitalization and a necropsy done immediately.

TABLE I
HEMOGRAMS OF TWO BULLS WITH SUSPECTED MONENSIN POISONING

	Bull No.	
	8	7
WBC (10^9/L)	17.6	13.6
RBC (10^{12}/L)	10.4	6.9
Hgb (g/dL)	13.2	10.2
PCV (%)	35.0	27.7
Neutrophils (10^6/L)	6512	6120
Band neutrophils (10^6/L)	—	1360
Eosinophils (10^6/L)	1056	—
Basophils (10^6/L)	176	—
Lymphocytes (10^6/L)	9680	5304
Monocytes (10^6/L)	176	816
Protein (g/dL)	7.2	6.3

Gross Pathological Findings

The bulls were in good physical condition and each had gross evidence of circulatory failure. The ventricles of the heart were dilated, and tan areas could be seen in the myocardium. Subepicardial lymphatics and coronary vessels were prominent. The lungs edematous and congested, were firmer than normal when palpated. The bronchial and mediastinal lymph nodes swollen, edematous and congested, reflected the changes in the lungs. One bull had large volumes of edema fluid in the subcutaneous tissues, the abdominal fascia and the mesentery, and its pleural cavity contained approximately five liters of blood-stained transudate. Hemorrhages in the subcutaneous tissues and gastrointestinal tract were present in the second bull. Both animals had congested livers. An incidental finding in one case was mild purulent bronchopneumonia.

Microscopic Findings

Pathological changes were seen diffusely in the myocardium. The sarcoplasm of myocardial fibres was vacuolated and sometimes foamy. Affected fibres were often shrunken with hyperchromatic sarcoplasm and pyknotic nuclei. Muscle fibres were almost completely degenerated in many areas, leaving a few empty sarcolemmal sheaths, sometimes containing small fragments of sarcoplasm. Numerous endomysial cells and small capillaries present in these degenerated myocardial areas were supported by a loose meshwork of fine eosinophilic fibres which stained positively for collagen when stained by Masson's trichrome. In one myocardium, such areas contained more mature, coarse fibrous connective tissue. Lymphocytes and neutrophils were present in small numbers in the interstitum. Many of the myocardial fibres stained irregularly but strongly with basic fuschin when stained by hematoxylin-basic fuschin-picric acid (HBFP) indicating myocardial necrosis.

Alveolar septa throughout the lungs were thickened due to an increase in the number of septal cells, macrophages, and congested capillaries. The alveoli contained protein-rich fluid and numerous macrophages. Large cells with basophilic cytoplasm were aggregated into clumps in the alveoli or formed an epithelial layer around masses or fibrin, forming hyaline membranes. The nuclei of these cells were frequently in mitosis. Walls of medium-sized arteries had areas of severe fibrinoid degeneration and necrosis.

Edema was not evident in the lungs of one animal which was euthanitized, nor was arterial fibrinoid necrosis present, but alveolar septal thickening and congestion was marked. Pulmonary edema was severe in two animals which died, and in these cases the interlobular septa were widely dilated with edema and lymphatic thrombosis.

Investigation of Possible Source of Monensin

When it became apparent that monensin toxicity was a possibility, the log book of daily feed operations for the previous month was examined. It revealed that on December 21 the feedmill had diluted some monensin into a ground grain extender. The concentrated premix to be added into final feedlot rations for their commercial cattle at 3.25%, was moved from the mill to a storage bin via the mixer truck. Immediately after this, the same truck was used to mix the ration that was fed to the performance test bulls.

TABLE II
SEQUENCE OF DEATHS AND NECROPSY FINDINGS IN A GROUP OF BULLS WITH SUSPECTED MONENSIN POISONING

Date	Bull No.	Necropsy Finding
Dec 30	1	Bloat
Dec 31	2	Embolic and fibrinous pneumonia
Dec 31	3	Not examined
Jan 2	4	Bloat
Jan 2	5	Pulmonary edema and congestion
Jan 7	6	Diffuse myocardial degeneration and early fibrosis
Jan 8	8	Pulmonary edema and congestion
Jan 8	7	Widespread focal myocardial degeneration and fibrosis

The load mixed was only of 500 kg compared to the truck capacity of 5000 kg.

This finding allowed the possibility that any monensin premix remaining in the truck was incorporated in the bull performance ration at a concentration of at least ten times more than had the truck been completely filled. In addition, this remaining premix left in the bottom of the truck may not have been included in the mixing action of the truck and therefore was flushed out in a high concentration when delivery of the feed to the bulls began. By the time monensin toxicity was suspected the feed containing the possible toxic concentration of the drug was no longer available for sampling.

Clinical Examination of Remaining Bulls

The remaining test bulls were examined one month after initial bulls died to determine if any of the animals had clinical evidence of congestive heart failure and respiratory insufficiency. Although several bulls had temperatures above 40°C, only one bull was thought to have poor exercise tolerance and another bull was felt to have engorged jugular veins. On March 31, approximately three months after the deaths, when the bulls were once again run through a chute, low exercise tolerance in one bull, and jugular engorgement in the other were not evident.

Discussion

Monensin poisoning was considered a possibility when the clinical findings of congestive heart failure was present in two and possibly more bulls at the same time. The third bull which collapsed and died without clinical signs of bloat suggested that it also may have had congestive heart failure and a reduced exercise tolerance.

The possible causes of congestive heart failure occurring in several bulls over a period of a few days were limited. Chronic atypical interstitial pneumonia with subsequent cor pulmonale was a possibility. In chronic atypical interstitial pneumonia the lung sounds may be muffled, normal, or slightly louder than normal and the characteristic histopathological findings were not seen in these cases. High altitude disease causes congestive heart failure but was not considered plausible because of the geographical location of the bulls.

Monensin is used in feedlot rations at a rate varying from 11 to 33 mg per kg of feed depending on the stage of feeding. This represents a daily intake of from 0.3 to 1.0 mg per kg body weight. The LD_{50} for monensin in cattle is estimated at 26.6 ± 3.9 mg per kg body weight (5).

Monensin poisoning may have occurred in the bulls if they had accidentally received a feed mix containing 25 to 30 times the recommended feed additive level for one feeding. The unexplained sudden drop in feed consumption followed by a quick recovery which occurred in the bulls is circumstantial evidence that the feed may have contained a toxic dose of monensin. Feed refusal is one of the earliest clinical findings in cattle which have consumed feed containing toxic levels of monensin within the previous day (5).

Acknowledgments

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References

1. COLLINS, E.A. and C.T. McCREA. Monensin sodium toxicity in cattle. Vet. Rec. 103: 386. 1979.
2. VAN DER KERK, R. Monensin poisoning in horses. Tijdschr. Diergeneesk. 103: 699-700. 1978.
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4. ORDIDGE, R.M. Death of horses after accidental feeding of monensin. Vet. Rec. 104: 375. 1979.
5. POTTER, E.L. and R.D. MULLER. Rumensin for growing cattle on pasture. Proceedings of Monensin Symposium, September 26-27. Eagle River, Ontario. pp. 76-111. Scarborough, Ontario: Eli Lilly and Company (Canada) Ltd. 1979.

CANADIAN VETERINARY MEDICAL ASSOCIATION

1981 Annual Meeting

Notice of Meeting

In conformity with by-law 33 of the Canadian Veterinary Medical Association notice is hereby given that the 1981 Annual Meeting of the Association will be held on Tuesday, July 7 in Winnipeg, Manitoba at the Winnipeg Convention Centre. The meeting will convene at 3:15 p.m.

D.A. LANDRY, D.M.V.
Secretary Treasurer

Ottawa, Ontario, April 1, 1981

ASSOCIATION CANADIENNE DES VÉTÉRINAIRES

Assemblée annuelle 1981

Avis de convocation

Conformément au règlement 33 de l'Association canadienne des vétérinaires, avis est par la présente donné que l'Assemblée annuelle de l'Association en l'an 1981 sera tenue mardi le 7 juillet à Winnipeg, Manitoba au centre de convention de Winnipeg. La réunion débutera à 3:15 p.m.

D.A. LANDRY, D.M.V.
Secrétaire trésorier

Ottawa, Ontario, le 1 avril 1981

ERRATA

Can. vet. J. 22: 31-33. 1981.

Growth Rates at the Extremities of Limb Bones in Young Horses. H.O. Goyal *et al.*

Page 33, left hand column, line 20 should read, "In the radius, however, the distal extremity grows faster than the proximal."

Page 33, left hand column, second paragraph, line 19, the references should read "(18,32,37)."