Monensin Toxicosis in Swine

D. W. Miskimins and R. D. Neiger

J VET Diagn Invest 1996 8: 396
DOI: 10.1177/104063879600800324

13. Sutter MD, Rawson DC, McKeown JA, Haskell AR: 1958, Chronic copper toxicosis in sheep. Am J Vet Res 19890-892.
14. Thomburg LP, Beissenherz MD, Raisbeck MF: 1985, Histochemical demonstration of copper and copper-associated protein in the canine liver. Vet Pathol 22:327-332.
15. Tremblay RRM, Baird JD: 1991, Chronic copper poisoning in two Holstein cows. Cornell Vet 81:205-213.
16. Vinlove MP, Britt J, Comelium J: 1992, Copper toxicity in a rabbit. Lab Anim Sci 42:614-615.

J Vet Diagn Invest 8:396-397 (1996)

Monensin toxicosis in swine

D. W. Miskimins, R. D. Neiger

Monensin is an ionophorous antibiotic formed by Streptomyces cinnamonensis.[5] It was the first antibiotic made and sold exclusively for use as an anticoccidial in chickens.[4] Monensin is approved to be used in cattle feed in the United States to improve feed efficiency and for the prevention and control of coccidiosis in feedlot cattle. Monensin is not approved for swine. However, reports show it has been used for coccidiosis control in swine.[1] Because of the common use of monensin in the feed industry, swine may be accidently exposed to monensin and sometimes to toxic concentrations. Diagnosis of monensin toxicosis is established by observation of typical clinical signs and pathologic changes and documentation of toxic concentrations of monensin in the feed by chemical analysis.

Clinical, clinicopathologic, and pathologic alterations caused by monensin toxicosis have been characterized in swine under experimental conditions.[7] Clinical signs in acute poisoning include dyspnea, bluish skin, ataxia, diarrhea, reluctance to stand, knuckling of hind limbs, and recumbence. Pathologic changes are dominated by skeletal muscle necrosis. Tiamulin in the drinking water has been shown to potentiate monensin toxicosis in pigs.[6,8]

In this report, we document a significant field case of monensin toxicosis in swine and describe a feeding trial that utilized similar amounts of monensin to reproduce typical signs and lesions of monensin toxicosis.

Fifty of 150 18-32-kg pigs were discovered dead on a Monday morning by the farmer. The remaining live pigs appeared gaunt and were reluctant to consume the remaining feed. The history indicated a feed change on the previous Saturday evening.

One live, ill pig was submitted to the South Dakota Animal Disease Research and Diagnostic Laboratory for examination. Gross lesions included pale skeletal muscles in the rear legs. Microscopically, the affected skeletal muscle fibers in the diaphragm and hind leg had extensive hyaline necrosis with hypercontraction bands and clumps of disrupted contractile material scattered within the sarcolemma (Fig. 1). Low numbers of macrophages surrounded degenerate fibers. There were very mild multifocal coagulative necrosis and cell swelling involving ventricular cardiac myocytes.

Further history revealed that medicated sow feed had been given to grower pigs when their feeders became empty on the weekend. This farm-mixed feed was supposed to contain 90 g/ton monensin to assist in coccidiosis control. Analysis of a feed sample using the Minder calorimetric method revealed 658 g/ton monensin.[2,3] The pigs had received an estimated 34 mg monensin/kg body weight. Losses stopped soon after the feed was changed, but surviving pigs were gaunt for several weeks following the incident. The history and laboratory examinations supported a diagnosis of monensin toxicosis. A feeding trial was conducted to further study the effects of monensin toxicosis in pigs.

Six pigs weighing 19-25 kg were used in the feeding trial. Four pigs were fed a commercial ration to which 593 g/ton of monensin had been added. Two control pigs received only the commercial ration. One monensin-fed pig was euthanized at 48 hours after the start of the trial, 2 at 72 hours, and 1 at 96 hours. One control pig was euthanized at 48 hours and the 2nd at 72 hours. Skeletal muscle and heart specimens were placed into 10% neutral buffered formalin, processed by standard histologic methods, sectioned at 5 lm, and stained with haematoxylin and eosin (HE).

Twelve hours after the start of the feeding trial, the monensin-fed pigs were lethargic, reluctant to rise, and stiff. One pig had dark-brown urine, and another pig knuckled over in a hind foot. Temperatures were normal. Feed refusal became evident as the trial progressed. Control pigs appeared normal throughout the study.

Gross lesions in the monensin-fed pigs included pale skeletal muscles in all 4 limbs. At 48 hours, microscopic examination of the left atrium of the monensin-fed pig demonstrated moderate diffuse myocardial necrosis. Necrotic myocardial fibers stained intensely with eosin. There was diffuse infiltration of macrophages into the interstitium in affected areas and within necrotic myocytes. Skeletal muscle fibers from the diaphragm, triceps, and semitendinosis muscles had extensive hyaline necrosis and occasional hypercontraction bands at 48 hours. Clumps of disrupted contractile material were scattered throughout the sarcolemma. Macrophages surrounded necrotic myocytes. Mild to moderate myocyte necrosis was still present in skeletal muscle 72 hours following the start of the feeding trial, and clumps of

From the Veterinary Science Department, South Dakota State University, Brookings, SD 57007-1396.

Received for publication August 15, 1995.

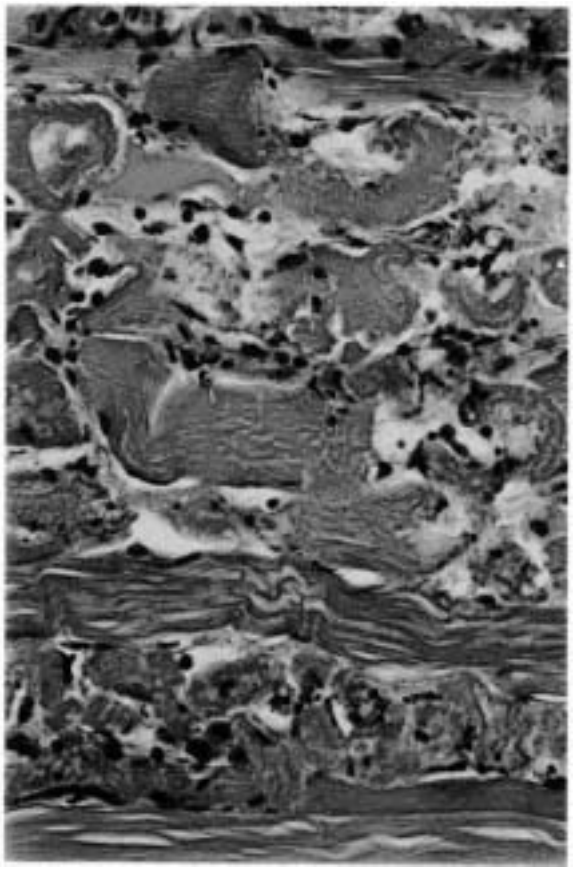

Figure 1. Skeletal muscle; pig with monensin toxicosis. There is hyaline necrosis, and low numbers of macrophages surround degenerate fibers. HE. 250 x .

disrupted contractile material were still present. Macrophages surrounded the necrotic myocytes. At 96 hours, the skeletal muscles had less intense infiltrations of macrophages. Necrotic fibers were being replaced by small, pale, basophilic regenerating fibers. No lesions were observed in the hearts or skeletal muscles of the control pigs.

The field case of monensin toxicosis described in this report demonstrates the devastating effects of overdosing pigs with monensin. A mixing error was responsible for the loss of 50 of 150 animals. The muscle lesions observed in this case were similar to lesions described in an experimental study.[7] The LD_{50} for monensin in swine is 16.7 mg/kg body weight, and the toxic level in feed is 500 g/ton[5] The amount of monensin consumed in this case was approximately 34 mg/kg body weight, which exceeds the amount required to cause toxicosis. The experimental ration contained 593 g/ton monensin. The gross and microscopic skeletal muscle and heart lesions observed were similar to lesions observed in an experimental study.[7] The fact that no experimental monensin-fed pigs died during the trial was attributed to increased feed refusal as the trial progressed.

The high death loss in this field case might be explained by heavy feed consumption following fasting.

References

1. Dore M, Morin M: 1987, Porcine neonatal coccidiosis: evaluation of monensin as preventive therapy. Can Vet J 28:663-666.
2. Golab T, Barton SJ, Scroggs RE: 1973, Colorimetric method for monensin. J Assoc Off Anal Chem 56:171-173.
3. LaPointe MR, Cohen H: 1988, High-speed liquid chromatographic determination-of monensin, narasin, and salinomycin in feeds, using post-column derivatization. J Assoc Off Anal Chem 71:480-484.
4. McDougald LR, Roberson EL: 1988, Antiprotozoan drugs. In: Veterinary pharmacology and therapeutics, ed. Booth NH, McDonald LE, 6th ed., pp. 950-968. Iowa State University Press, Ames, IA.
5. Osweiler GD, Carson TL, Buck WB, VanGelder GA: 1985, Monensin. In: Clinical and diagnostic veterinary toxicology, 3rd ed., pp. 152-159. Kendall/Hunt, Dubuque, IA.
6. Pott JM, Skov B: 1981, Monensin-tiamulin interactions in pigs. Vet Rec 109:545.
7. VanVleet JF, Amstutz HE, Weirich WE, et al.: 1983, Clinical, clinicopathologic, and pathologic alterations of monensin toxicosis in swine. Am J Vet Res 44:1469-1475.
8. VanVleet JF, Runnels LJ, Cook JR, Scheidt AB: 1987, Monensin toxicosis in swine: potentiation by tiamulin administration and ameliorative effect of treatment with selenium and/or vitamin E. Am J Vet Res 48:1520-1524.