BRIEF COMMUNICATION

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Monensin intoxication in broiler chicks: Is it really so easy to identify?

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Monensin, a monocarboxylic acid ionophore antibiotic, is widely used in cattle, swine and poultry feeds. In poultry, this agent is used for its effective anticoccidial properties, despite a low margin of safety (1). In recent years, concern has arisen over the use of monensin because of several instances of acute poisoning observed in chicken and turkey flocks (2,3). These poisonings were largely the result of either drug incompatibilities or improperly mixed feeds, leading to drug overdose (4-6).

Monensin is readily absorbed from the gastrointestinal tract of most animals, however, residue concentrations do not reach high levels in body tissues, even with severe intoxication (7). Also, monensin residues in frozen poultry tissues degrade over time (8). These two facts prohibit diagnosis of a flock poisoning on the basis of tissue analysis.

Several references describe in detail the clinical signs and lesions which occur with monensin poisoning [see Langstom (9) for an excellent review]. Throughout a series of investigations at the University of Saskatchewan, we did not observe gross or histological lesions in broiler chicks fed any of several concentrations of monensin.

We conducted five studies. Specific details of each study are contained in other papers as referenced below, or submitted for publication. One study involved feeding an elevated level of sodium selenite followed by a lethal dosage of monensin (350 mg/kg) (10). The high sodium selenite diets ranged from 0.5 to 15.0 mg/kg and were fed over a period of four weeks to 192 birds. A second study involved the simultaneous feeding of monensin (at either 50 or 250 mg/kg) and sodium selenite (at either 0 or 10 mg/kg) over a four week period to 96 birds. A third study involved the simultaneous feeding of monensin (from 50 to 200 mg/kg) and arsanilic acid (from 0–500 mg/kg) for a period of four weeks to 264 birds (11). The last two experiments involved kinetic studies in which the birds were given lethal concentrations of monensin as a single dose.

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In the first kinetic study, 30 chickens in five groupings were given a range of monensin concentrations, 200–800 mg/kg, in order to establish the signs of monensin intoxication and the LD50. In the second kinetic study, 12 chickens were given a range of monensin concentrations, 100–1,000 mg/kg, in order to determine the approximate drug withdrawal period and in which tissues monensin residues concentrated. The latter part of this study is published (8). Thus, in the group of five studies, concentrations of monensin given to the birds ranged from 50–250 mg/kg in the feeding trials to 100–1,000 mg/kg in the kinetic studies, with the appropriate controls in each study.

All birds were raised from day-old chicks and the experimental periods ran from three to seven weeks of age. All birds were wing-banded and vaccinated for Marek's disease at one day of age. All experimental diets were starter crumble rations formulated to provide 13.0 mJ/kg of metabolizable energy, 23.9% protein, 1.0% calcium, 0.18% sodium and 0.5% available phosphorus. Whether kept in wire-batteries or floor pens, the birds were housed in rooms maintained at 23–25°C (with supplemental heat available), 40% humidity, and under continuous incandescent light set at five Lux, with feed and water available *ad libitum*. Clinical signs of intoxication were recorded for all birds (n = 652); necropsies were completed on 126 birds, with cardiac muscle, skeletal muscle (taken from the right quadriceps), liver and kidney taken for histological examination; monensin residue analyses were also completed on these four tissues.

All of the birds that died displayed three or more characteristic clinical signs of monensin intoxication, which included feed refusal, growth depression, cream-colored diarrhea, dyspnea, muscular stiffness and/or weakness, and sternal recumbency

Clinical signs of intoxication were readily observable. All of the birds that died displayed three or more characteristic clinical signs of monensin intoxication, which included feed refusal, growth depression, cream-colored diarrhea, dyspnea, muscular stiffness and/or weakness, and sternal recumbency. As well, a small

percentage of surviving birds in the feeding trials displayed characteristic clinical signs of intoxication.

Gross pathological changes were not observed in any of the birds studied. This confirms the work of Chalmers (12), who reported that gross pathological changes are seldom found in acutely intoxicated birds. Isolated cases of bile duct proliferation were observed in a few chickens, but this was attributable to their young age (C. Riddell, personal communication). Histological lesions attributable to monensin intoxication could not be found. Residues of monensin in even the most severely intoxicated birds remained low, and were similar to the results obtained by Donoho (4).

In conclusion, monensin intoxication in broiler chicks, indicated by flock history, may not be easy to confirm based solely on either analysis for residues or postmortem examination. A detailed description of the clinical signs observed in the birds of a flock is essential for proper diagnosis. Based on clinical observation alone, a strong case can be made for monensin poisoning, provided three or more of the "characteristic" signs are observed.

We believe that, because of the lack of postmortem findings and residue analysis information, the prevalence of monensin poisoning in poultry flocks may be higher than currently thought. Under-diagnosis of severe cases of poisoning may occur due to the absence of tissue residues and lesions. With mild poisoning, expressed solely as growth depression, intoxication may go completely unnoticed. Thus, great care must be taken when investigating flocks in which either large numbers of birds have died, or poor growth rates are exhibited. Detailed clinical observation appears to be the key to proper diagnosis of monensin poisoning in poultry.

References

1. Beck BE, Harries WM. The diagnosis of monensin toxicosis: A report on outbreaks in horses, cattle, and chickens. Annu Proc Am Assoc Vet Lab Diagnost 1979: 269–282.
2. Bartov I, Jensen LS. Effect of dietary ingredients on monensin toxicity in chicks. Poult Sci 1980; 59: 1818–1823.
3. Hanrahan LA, Corrier DE, Naqi SA. Monensin toxicosis in broiler chickens. Vet Pathol 1981; 18: 665–671.
4. Howell J, Hanson J, Onderka D, Harries WN. Monensin toxicity in chickens. Avian Dis 1980; 24: 1050–1053.
5. Kemp J. Monensin poisoning in turkeys. Vet Rec 1978; 102: 467.
6. Stuart JC. An outbreak of monensin poisoning in adult turkeys. Vet Rec 1978; 102: 303–304.
7. Donoho AL. Biochemical studies on the fate of monensin in animals and in the environment. J Anim Sci 1984; 58: 1528–1539.
8. VanderKop PA, MacNeil JD, Patterson JR. Tissue dependent degradation of monensin residues in chicken tissues with prolonged freezer storage. J Vet Diagnost Invest 1989; 1: 176–177.
9. Langstom VC, Galey H, Lovell R, Buck WB. Toxicity and therapeutics of monensin: A review. Vet Med 1985; 10: 75–84.
10. VanderKop PA, MacNeil JD. Protection provided by sodium selenite against an oral toxic dose of monensin in broiler chicks. Can J Anim Sci 1989; 69: 477–482.
11. VanderKop PA, MacNeil JD. Effects of arsanilic acid and monensin when both are given simultaneously in the diet of broiler chicks. Vet Hum Toxicol 1989; 31: 209–213.
12. Chalmers GA. Monensin toxicity in broiler chickens. Can Vet J 1981; 22: 21–22.