Accidental monensin toxicosis in horses in Mozambique

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ABSTRACT

Horses on several farms in Mozambique were inadvertently fed with a concentrate containing 69 ppm monensin. The horses developed acute signs of toxicity and several died. The animals were depressed, anorectic and paretic before death. Epistaxis was observed in 1 case. Petechial haemorrhages were present in the muscles, heart, lungs, gastrointestinal tract and spleen in 3 horses necropsied. No significant histopathological cardiac and skeletal muscle lesions were seen, except in 1 case, in which there was focal loss of myofibrils.

Key words: horses, ionophores, monensin, Mozambique, toxicosis.

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Monensin is an ionophore antibiotic derived from *Streptomyces cinnamonensis* and is used extensively as a feed additive to promote weight gain, to prevent bloat in cattle and as a coccidiostat in several animal species[1,5,8]. Ionophore antibiotics form lipid-soluble reversible complexes with cations (Ca^{++}, K^{+}, Mg^{++}), which facilitate specific ionic transport across biological membranes[5].

Monensin has been found safe and effective in target animals when administered at a recommended dose. However, toxic syndromes as a result of inappropriate use of this drug have been reported in several mammalian species and birds. Horses are far more sensitive to monensin than other species[6].

Clinical, clinicopathological and pathological alterations caused by monensin poisoning have been described in horses. Clinical signs include anorexia, depression, incoordination, muscle weakness, myoglobinuria, ataxia and diarrhoea[2,4,5]. Serum levels of aspartate aminotransferase (AST), lactic dehydrogenase (LD) and creatine phosphokinase (CPK) are increased[2,7]. Degeneration and necrosis of cardiac and skeletal muscles with variable inflammation are the predominant pathological changes[2,4,5].

The aim of this paper is to describe the clinical signs of monensin toxicosis and the pathological lesions that occurred in 3 horses after accidental feeding.

Clinical signs developed and mortality occurred in horses on 3 farms in Maputo city, 1 farm in the Manhica District, and on another farm in the Inhambane Province of Mozambique. Sudden death was the first indication of the problem. As the course of the disease progressed, many horses exhibited anorexia, depression, weakness and ataxia. Epistaxis was observed in 1 animal. Eight horses died. Owing to the acute onset of clinical signs, the feed was suspected. Consultation with the feed supplier revealed a mixing error and monensin had been added to the horse rations.

Analysis of the blood revealed elevated total serum protein (all 3 cases), and increased AST (2 cases), CPK (2 cases) and LD (all 3 cases) activity. Enzyme activity was measured spectrophotometrically at 37 °C using a 'COBAS' (Hoffman La Roche, Switzerland) auto-analyser. Ration samples from all implicated farms tested positive and the concentration of monensin in the sample from 1 of them was 69 parts per million. The colorimetric method for monensin was used for both the qualitative and quantitative assays[3].

Necropsy was performed on 3 horses from 1 of the farms in Maputo city. The most prominent gross lesions were petechial haemorrhages in the lungs (2 cases), gastrointestinal tract (2 cases), heart (2 cases) and spleen (1 case).

Samples of lung, heart, gastrointestinal tract, liver, spleen, kidney, gall bladder, skeletal muscles from the thigh, and brain were collected in 4 % buffered formaldehyde and routinely processed for histological examination. Multifocal degeneration with loss of myofibrils and light infiltration of mononuclear cells was observed in the heart and skeletal muscle of only 1 horse. Pulmonary oedema and emphysema (2 cases) and foci of slight catarrhal bronchiolitis and mononuclear peribronchiolitis (2 cases) were also observed. No significant changes were observed in the liver, kidney, spleen, gastrointestinal tract and gall bladder.

The horses in the present outbreak developed clinical and clinicopathological alterations compatible with monensin toxicosis in this species[5]. Epistaxis has not been reported previously, and in contrast to previous reports, diarrhoea was not observed. The sodium monensin detected in the rations fed to the horses confirmed the preliminary diagnosis of suspected toxicity. Similar outbreaks have been reported in horses involving feed containing <5 to 679 ppm of monensin[2,4,7].

Degeneration and necrosis of cardiac and skeletal muscles are the most prominent microscopic changes in monensin poisoning. Two of the horses examined in this study lacked significant gross and microscopic lesions. One horse exhibited skeletal and cardiac muscle degeneration with loss of myofibrils. The severity of these lesions may depend upon dose and duration of exposure to the drug[5,9]. Animals that die soon after exposure to ionophores may have no significant lesions, or the lesions may be subtle[5]. The high level of sodium monensin detected in the ration may also explain the lack of significant lesions, as described in other outbreaks in horses[2,7] and adult turkeys[10]. This study demonstrates that death due to monensin poisoning may result in a non-specific or negative necropsy. If monensin poisoning is suspected, a comprehensive history, clinical examination and feed analysis are important to confirm the diagnosis.

REFERENCES

1. Bergstrom R C, Maki L R 1976 Coccidiostatic action of monensin fed to lambs: body weight and feed conversion efficacy. *American Journal of Veterinary Research* 37: 79–81
2. Bezerra P S, Driemeier D, Loretti A P, Riet-Correa F, Kamphues J, de Barros C S 1999 Monensin poisoning in Brazilian horses. *Veterinary and Human Toxicology* 41: 383–385

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3. Golab T, Barton S J, Scroggs R E 1973 Colorimetric method for monensin. *Journal of the Official Association of Analytical Chemists* 56: 171–173
4. Kamphues J, Meyer H, Liebler E M, Johannsen A 1990 Animal nutrition for veterinarians: recent cases of clinical disorders after intake of ionophore-containing feed. *Deutsche Tierärztliche Wochenschrift* 97: 537–539
5. Novilla M N 1992 The veterinary importance of the toxic syndrome induced by ionophores. *Veterinary and Human Toxicology* 34: 66–70
6. Oehme F W, Pickrell J A 1999 An analysis of chronic oral toxicity of polyether ionophore antibiotics in animals. *Veterinary and Human Toxicology* 41:251–257
7. Ordidge R M, Schubert F K, Stoker J W 1979 Death of horses after accidental feeding of monensin. *The Veterinary Record* 104: 375
8. Ruff M D, Reid W M, Rahn A P 1976 Efficacy of different feeding levels of monensin in the control of coccidiosis in broilers. merican Journal of Veterinary Research 37: 963–967
9. Sales M S, Barros C S L, Barros S S 1999 Ionophore (narasin) poisoning in rabbits. *Veterinary Human Toxicology* 36: 437–444
10. Stuart J C 1978 An outbreak of monensin poisoning in turkeys. *The Veterinary Record* 102: 303–304

Book review — Boekresensie

Diagnostic Manual for Aquatic Animal Diseases

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Rapid developments in aquaculture have necessitated revision of the International Aquatic Animal Health Code and its companion work, the Diagnostic Manual for Aquatic Animal Diseases, by the Fish Diseases Commission of the Office International des Épizooties. The 2000 edition of these volumes appeared recently and perpetuates the standard of excellence set by the previous 2 editions[1].

Those familiar with the Code and Manual will find the greatest changes in the sections on crustacean diseases. The increased emphasis on crustacean diseases is largely due to massive losses suffered worldwide in cultured prawn species, with previously undescribed diseases emerging under the crowded and stressful conditions of intensive prawn farming.

The Diagnostic Manual for Aquatic Animal Diseases contains technical information on diagnostic methods for notifiable and significant diseases. Sections on quality management in veterinary diagnostic laboratories and principles for validation of diagnostic assays have been added.

Considering the increasing emphasis on quality management in diagnostics, these sections are extremely valuable. The Manual is divided into various sections that deal with the diseases of fish, molluscs and crustaceans. Each section contains a background discussion of aspects relevant to diagnostic methods for the group. As in the previous editions, the chapters devoted to specific diseases are detailed and complete. The chapter summaries are extremely useful for quick reference.

Two new diseases of fish are included under significant diseases and the sections on crustacean diseases have been extensively revised. The Manual is required reading for anyone involved in aquatic animal diagnostics and is an essential source of information for those in a regulatory capacity.

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[1]As this volume went to press, the 2001 edition of the Code was received and will be reviewed in the December issue of the journal.