Monensin toxicity in calves

Geoff Orbell, Registered Specialist in Veterinary Anatomic Pathology, of NZVP/IDEXX, Palmerston North, reports two fatal cases of monensin toxicity in weaned dairy calves.

RECENTLY, NZVP/IDEXX HAS had submissions from two outbreaks of sudden death in weaned dairy calves where post-mortem histology has led to the identification of monensin as the underlying cause.

CASE 1

A veterinarian was called to visit a farm where four three-month-old mixed sex dairy calves out of 150 had died over a two-day period. Other calves appeared clinically normal.

Post-mortems were performed on two calves. The most significant post-mortem findings were effusions in the abdominal and pleural cavities. The fluid was colourless and watery and did not clot on exposure to air. There were no fibrin tags identified on the serosal surfaces. Grossly, the liver appeared swollen with rounded edges.

The veterinarian called the laboratory about the case to discuss differential diagnoses. These included septicaemic pasteurellosis, histophilosis, sporadic bovine encephalomyelitis (SBE), coliform septicaemia, galegine (goat's rue) toxicity, monensin toxicity and white muscle disease. Histology revealed congested lungs and liver, multifocal myodegeneration, and necrosis of cardiomyocytes in the myocardium associated with infiltrates of macrophages, lymphocytes and neutrophils. No fibrin was identified adherent to the serosa of any organs examined. No lesions suggestive of SBE were identified in the brain.

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STAFF REVEALED THAT LEFTOVER FEED WITH PREMIXED MONENSIN FOR THE MILKING HERD HAD BEEN GIVEN TO THE CALVES IN THE PREVIOUS FEW WEEKS.

Based on the myocardial changes, nensin toxicity was suspected, with ıe bicavitary effusion secondary to congestive heart failure rather than an infectious process.

On further questioning, farm staff revealed that leftover feed with premixed monensin for the milking herd had been given to the calves in the previous few weeks.

CASE 2

A veterinarian was called to visit a farm where five four-month-old dairy calves out of 100 had died over a two-week period. Other calves appeared clinically normal.

A post-mortem was performed on one calf that revealed pleural and peritoneal effusions without fibrin exudation. Grossly, the small intestinal mucosa appeared reddened and the liver swollen, and the lungs were diffusely reddened.

The veterinarian suspected salmonellosis clinically, and took representative tissue samples for histology and fresh faeces for *Salmonella* culture, which was negative.

Histology revealed congested lungs and liver, with multifocal foci of necrosis associated with few neutrophils suggestive of a septicaemia. There was also multifocal myodegeneration and necrosis of cardiomyocytes similar to the previous case.

In the report, monensin toxicity was suggested, although it didn't explain the random multifocal liver necrosis. On receipt of the report, the veterinarian phoned the lab and stated that the calves recently had a coccidia issue and that monensin was being mixed into the feed. On further investigation into the amount of premix used, the calves were getting approximately 300mg/calf/day, rather than the recommended 100mg/calf/day.

Monensin is an ionophore, so named as it interferes with cation movement across biological membranes – namely sodium, potassium and calcium (Hall, 2000).

It is a fermentation by-product of *Streptomyces* bacteria (usually *S. cinnamonensis*), and is used to improve growth rates in livestock by altering ruminal microflora to improve feed conversion efficiency and prevent ruminal acidosis (Novilla, 1992).

It is also used as a coccidiostat in the ruminant and poultry industries (Potter et al., 1984).

Ionophore toxicosis results from changes in primary ion concentrations across cell membranes releasing sequestered intracellular ions, altering the intracellular pH and causing damage to the cell membrane or intracellular organelles (Novilla, 1992).

Clinical signs are dose-dependent, with sudden death frequently the initial clinical presentation in cattle. Less-intoxicated cattle can be anorexic, tachycardic, dyspneic with diarrhoea, dehydration, ruminal atony, muscle tremors, weakness and incoordination. Jugular pulses will sometimes be identified due to right-sided congestive heart failure (Hall, 2000).

Ante-mortem diagnosis can be assisted with CK and AST, but some studies have shown these are only elevated within the 24 hours prior to death. Biochemistry may reveal dehydration (elevated albumin and prerenal azotemia), decreased sodium, potassium and calcium (Hall, 2000).

Post-mortem examination is often unremarkable, with gross lesions rarely identified within the cardiac or skeletal muscle. In some cases there will be evidence of congestive heart failure, with hepatic congestion and nonfibrinous pleural and peritoneal effusions.

Cases of monensin toxicity have also been reported in dogs and horses in New Zealand, with the latter even more sensitive to ionophores than ruminants (Divers et al., 2009; *Surveillance*, 2011, 2013, 2014).

In cattle and most domestic species, cardiac and skeletal muscle are most susceptible, and intercostal muscles have been suggested as the skeletal muscle of choice for histology (Australian Society of Veterinary Pathology Annual Conference, 2013), along with the left ventricle of the heart. However, a full range of post-mortem tissues, including brain, should always be submitted fixed for histology in cases of sudden death. VS

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