

Zinc toxicity in cattle

Hania Klobukowska, of Gribbles Veterinary, contemplates one of the risks involved in zinc supplementation.

INTRODUCTION

Zinc toxicity is almost exclusively seen following the prophylactic use of zinc supplementation administered during the facial eczema (FE) season. Zinc toxicity is not new to the New Zealand scene, but it is important to consider it as a differential diagnosis under certain circumstances, especially when the farm is known to be supplementing the mineral. Although many zinc toxicity cases (including those described in this report) involve the inadvertent use of zinc boluses, it must be remembered that there are various methods of administering the product that can also result in toxicity – for example, excessive administration of zinc into in-line water feeding devices (Ackermann et al., 2012) and excess zinc in the soil (Briston and Pike, 2015). Therefore, when approaching suspected zinc toxicity cases, consideration should always be given to more widespread farm grazing and mineral management practices.

The following describes three cases of zinc toxicity as they presented and were diagnosed at Gribbles Veterinary, Palmerston North.

CASE 1

Two Friesian-cross R1 dairy heifers presented with severe anaemia. (The in-house packed-cell volume measurements were 11% and 14%, whereas the normal reference range is 24–40%.) Although a full complete blood count and biochemical profile were not performed, one of the heifers also had red urine, suggesting the anaemia was haemolytic in origin. Further investigations included a pooled *Theileria* PCR, which was negative; there were also negative antibody titres to the pomona,



hardjobovis, copenhageni and tarrasovi *Leptospira* serovars, and within-range gamma-glutamyl transferase (GGT) and serum copper levels. Zinc serum levels were found to be elevated at 208 $\mu\text{mol/L}$ and 146 $\mu\text{mol/L}$ each (toxic level range 27–92 $\mu\text{mol/L}$). Upon further investigation, it was found that these heifers had inadvertently been given twice the recommended intra-ruminal zinc bolus dose.

CASE 2

Two Friesian R1 dairy heifers were found dead. A postmortem conducted on one of them found it had severe abomasal pathology. Zinc toxicity was

suspected from the outset as these heifers, similarly to those in Case 1, were known to have been administered twice the recommended intra-ruminal zinc bolus dose. Furthermore, the water was being treated with an in-line zinc dispenser. Histopathology was performed and revealed a severe, acute necrohaemorrhagic abomasitis with submucosal vasculitis. There was also a submucosal vasculitis with associated oedema of the small intestinal wall. There was acute tubular necrosis and pigmenturia in the kidney. Zinc liver levels were found to be markedly elevated in the necropsied heifer at 6,294 $\mu\text{mol/kg}$ (normal zinc liver range: 380–1,530 $\mu\text{mol/kg}$).

ZINC TOXICITY CAN PRESENT VARIABLY DEPENDING ON THE DOSAGE AND ACUTENESS AND CHRONICITY OF TOXICOSIS.

CASE 3

A large proportion of R1 dairy heifers were found to have sub-optimal weight gains despite abundant feed. Some of them were also scouring. Initial laboratory results revealed within-range GGT and serum copper levels and a low bovine viral diarrhoea virus antibody pooled S/P ratio (0.08). Faecal testing revealed low numbers of strongyle eggs and minimal coccidial burdens and negative *Yersinia* cultures. Zinc serum levels were found to be elevated in all of the tested animals (range 44–110 µmol/L), raising suspicions this was the primary problem. Zinc supplementation was reviewed for these heifers, and it was found that they had received an initial zinc bolus targeted for the 175–250kg weight range, despite the mob averaging 185kg, with large variances between minimum and maximum weights. Subsequent zinc boluses were administered monthly despite poor weight gain, resulting in a cumulative overdose. By the end of the outbreak a small number of heifers had been euthanased due to poor condition. Histology of the pancreas revealed chronic atrophic and fibrotic lesions with attempts at regeneration, a classic histological finding with zinc toxicity (Jubb and Stent, 2016).

DISCUSSION

Zinc is an essential trace element for both humans and animals. It is found within all cells in the body and is involved in a multitude of biochemical processes. Not only is zinc required daily from a nutritional point of view, zinc supplementation has been utilised as an important preventive tool in mitigating

the effects of FE in New Zealand livestock. Zinc is primarily absorbed in the small intestine, and although it is distributed throughout the body, it tends to accumulate in the liver, pancreas, spleen and kidney (Garland, 2012). Zinc toxicity can present variably depending on the dosage and acuteness and chronicity of toxicosis. Furthermore, calves are thought to be more sensitive to the effects of zinc as they absorb larger quantities of it through the gastrointestinal system (Parton et al., 2006). Haematologic, gastrointestinal and pancreatic manifestations of zinc toxicity are well described, and animals can present with haemolytic anaemias, severe gastrointestinal upsets, diarrhoea and general ill-thrift. The pathogenesis of zinc toxicity as it relates to the systems involved is poorly understood.

The gastrointestinal manifestations of disease can mimic acute and chronic bacterial, viral and parasitic enteritides. However, consideration may also be given to less well known agents such as acorn and arsenic toxicity. These substances, along with zinc, can have a very corrosive effect on the gastrointestinal mucosa. Differential diagnoses for haemolytic anaemias are many, but in New Zealand considerations include copper toxicity, leptospirosis (specifically in calves), S-Methyl-L-Cysteine Sulfoxide (SMCO) toxicity, acute sporidesmin toxicity and theileriosis. Approach to ill-thrift can be an extensive operation, but, as mentioned previously, season, FE risk and known supplementation with zinc should raise suspicions, especially when many of the more common causes of ill-thrift have been ruled out.

Diagnosis of zinc toxicity involves zinc-level evaluations in blood or liver. Zinc levels may be within the toxic range in more acute to subacute cases. However, in chronic toxicities, zinc levels can fall into a more 'normal' range. This can be more challenging to interpret, and the risk is missing chronic zinc toxicity as the problem. If animals are dead or sacrificed then histopathology on a complete set of tissues is recommended, with a particular focus on the pancreas, liver, kidney and gastrointestinal tract. A full set of tissues is important as it will also help rule in/out other causes of death or possible co-morbidities. The pancreas is incredibly susceptible to the effects of excessive zinc, and there are not many other differential diagnoses that will result in the classic pancreatic histological lesions we see with zinc toxicity.

CONCLUSION

The use of zinc as an FE prophylactic is widespread in the North Island during FE season. As with other trace elements, the need for use must be weighed against the risk of disuse. It is generally accepted that zinc supplementation can be used as an effective tool for FE prophylaxis. However, careful management practices need to be instigated to prevent inadvertent toxicity (eg, dosing to the correct weight, correct interval, etc.), as overzealous use can have grievous short- and long-term consequences. ⁽⁸⁾

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