Awanui Veterinary

Pathology in focus

April 2025

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Zinc toxicity in cattle

Hania Klobukowska

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 however 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, and 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 e.g. excessive administration of zinc into in-line water feeding devices (Ackermann et al. 2012) or 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.

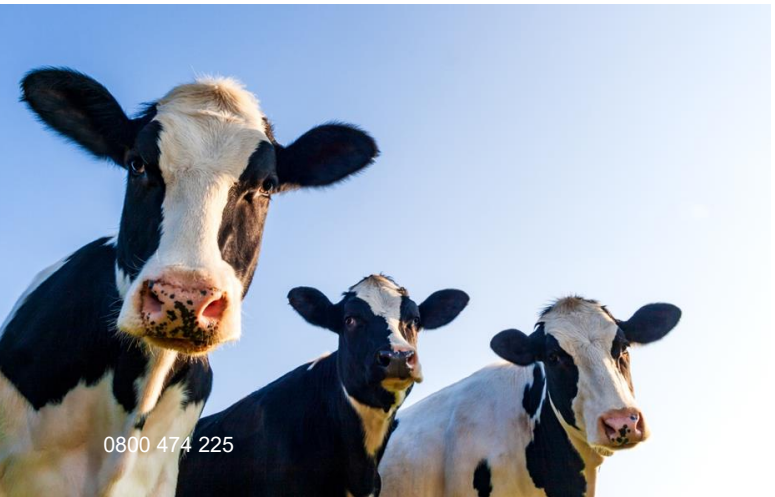
The following describes three cases of zinc toxicity as they presented and were diagnosed in our laboratories.

Case 1

Two Friesian cross R1 diary heifers presented with a severe anaemia (in-house PCV measurements were 11 and 14% (normal reference range 24 to 40%)). Although a full CBC 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, negative antibody titres to the following Leptospira serovars: pomona, hardjo bovis, copenhageni and tarrasovi, and within range GGT and serum copper levels. Zinc serum levels were found to be elevated at 208 and 146 µmol/L each (toxic level range 27-92 µmol/L). Upon further investigations it was found that these heifers were inadvertently given twice the recommended intra-ruminal zinc bolus dose.

Case 2

Two Friesian R1 dairy heifers were found dead. A post mortem was conducted on one of them where it was found to have 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 as well. 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 6294 µmol/kg (normal zinc liver range 380-1530 µmol/kg).



Zinc toxicity can present variably depending on the dosage and acuteness and chronicity of the 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 BVDV 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 to 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 175-250 kg weight range, despite the mob averaging 185 kg with large variance 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 were euthanised 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 preventative tool in mitigating the effects of facial eczema in New Zealand livestock. Zinc is primarily absorbed in the small intestine and although its 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 zinc through the gastrointestinal system (Parton K 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 however within 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, however, as mentioned previously, season, FE risk and known supplementation with zinc should raise suspicions, especially when many of the other 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 of missing chronic zinc toxicity is 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 a FE prophylactic is widespread amongst the North Island during FE season. As with other trace elements, the need for use must be weighed against 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 (e.g. dosing to the correct weight, correct interval etc.) as overzealous use can have grievous short and long-term consequences.

References

Ackermann S, Johnston H, Shelgren J. What was he zincing? Proceedings of the Society of Dairy Cattle Veterinarians of the NZVA, Annual Conference pp 2.16.1-4, 2012

Briston P and Pike C. The stink of zinc: The curious incident of the cows in the effluent paddock. Proceedings of the Society of Dairy Cattle Veterinarians of the NZVA, Annual Conference pp 209-18, 2015

Garland T. Zinc. In: Gupta RC Veterinary Toxicology: Basics and Clinical Principles. Academic Press, London pp 567-9, 2012

Jubb KVF and Stent AW. In: Jubb, Kennedy and Palmer's Pathology of Domestic Animals. Elsevier, Missouri Vol 2 pp 356-57, 2016

Parton K, Bruere N, Chambers P. Veterinary Clinical Toxicology. VetLearn, Massey University, Palmerston North pp108-12, 2006

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Pathologist spotlight

Hania Klobukowska has been part of our team since 2016. She graduated from Massey University in 2009 with a BVSc., then spent 2.5 years in small animal clinical practice in New Zealand and the UK. She then worked as a veterinarian for MPI for another 2.5 years before going on to complete a 3-year residency in anatomic pathology at Massey University. Hania passed her ACVP board examinations in 2017.

Hania enjoys all aspects of diagnostic pathology and has special interests in small and large animal anatomic pathology, cytology and herd health investigations. Her broad set of skills allows her to approach cases in an integrative manner.

When not in the laboratory Hania enjoys spending time with her busy young family, watching sitcoms, and reading.

April long weekends

Our laboratories will be closed all of Easter weekend (Friday 18 April to Monday 21 April) and ANZAC weekend (Friday 25 April to Sunday 27 April). We will reopen for business on Tuesday 22 April and Monday 28 April respectively.

We do not recommend sending samples overnight on either Thursday 17 or 24 April, as they will not be delivered until after the weekend and will likely be unsuitable for testing (with the exception of formalin fixed tissues).

If you have any questions or concerns, please contact your local laboratory.

Testosterone testing no longer recommended

Kathryn Jenkins

There is currently no validated serum testosterone test for veterinary species available, and the referral test method in use has low sensitivity. As a result, serum testosterone is no longer recommended for use in veterinary species.

In many cases, this can be replaced by serum Anti-Müllerian hormone (AMH). AMH is produced by the follicles of a sexually mature ovary and Sertoli cells in a sexually mature testes. After complete castration or ovariectomy, levels of AMH decrease significantly.

AMH is tested at a medical referral laboratory, however this test has been validated for use in male horses (i.e. suspect cryptorchid or rig). AMH may also be a useful in determining the gonadal status of both dogs and cats (including cases of suspected cryptorchid or ovarian remnant).

Notes

- When evaluating for functional gonadal tissue it is recommended to wait at least 30 days after a de-sexing procedure, and animals should be over the age of 6-months.
- A negative result does not fully exclude ovarian remnant in some cases, and if clinical signs are supportive then further testing is recommended (e.g. vaginal cytology, serum progesterone, imaging).
- When evaluating ovarian remnant syndrome, testing both serum AMH and progesterone is recommended.
- The AMH test requires a minimum of 1 mL of serum (i.e. 2-3mL of blood in a red top tube), from a fasted patient, and should be transported to the laboratory within 24 hours of collection. Sending the sample at the beginning of the week is recommended.

References

Place, N. et al. Evaluation of combined assessments of serum anti-Müllerian hormone and progesterone concentrations for the diagnosis of ovarian remnant syndrome in dogs. JAVMA, 2019 Vol 254 (9)

Flock, U., et al. Anti-Müllerian hormone as a diagnostic tool to identify queens with ovarian remnant syndrome. JFMS 2022 Vol 24(8)

Walter, B. Anti-Müllerian hormone in dogs and cats reproduction. Reproduction in Domestic Animals, 2020;55 (Suppl.2)

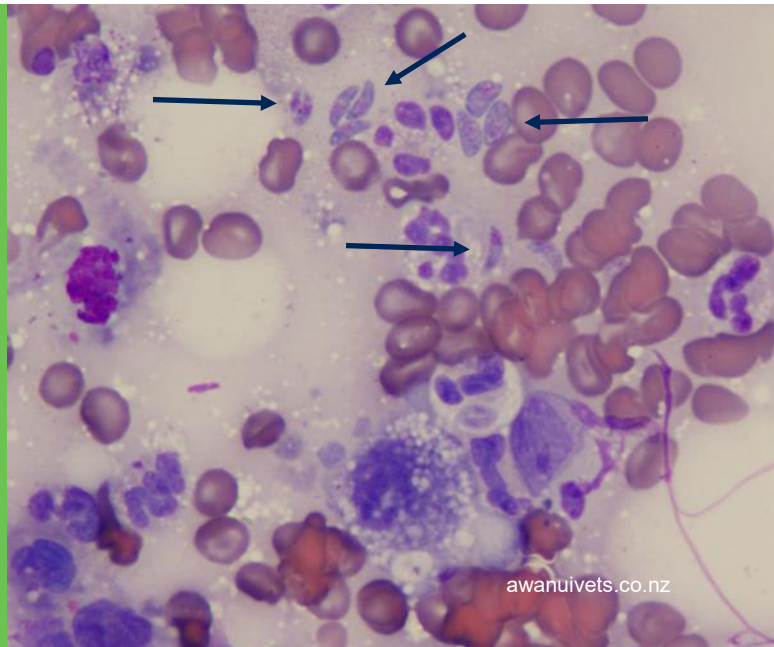
Claes AN, Ball BA. Biological Functions and Clinical Applications of Anti-Müllerian Hormone in Stallions and Mares. Vet Clin North Am Equine Pract. 2016 Vol 32.

Down the ‘scope

A one-year-old Boxer dog presented with nodules throughout all lung lobes. She was febrile, lethargic and had marked elevations in AST, ALT and CK, plus elevated ALP and bilirubin. We received samples from lung lesions for cytology and culture.

Culture did not produce any significant growth but this is what we saw on cytology. What do you think the culprit is and what follow up testing would you recommend?

(Answer posted on last page)



Diagnosis and management

of canine and feline urinary tract infections

Karen Bailey

Urinary tract disease is a common reason for antibiotic use in cats and dogs. With increasing awareness of the need for appropriate antimicrobial stewardship, the International Society for Companion Animal Infectious Diseases (ISCAID) guidelines on UTI are a good reference. Whilst high level objective data is scant, the authors have useful advice, though these are guidelines not mandates and case by case decisions are still needed. In all cases, the primary objective is clinical cure with minimal risk of adverse effects, including the development of resistance. Microbiological cure is desirable, but may not be needed for clinical resolution.

Sporadic bacterial cystitis

Common in dogs and uncommon in cats, this results in inflammation with signs of pollakiuria, dysuria, stranguria or haematuria.

- Most cats with lower urinary tract signs don't have bacterial cystitis. Feline idiopathic cystitis (FIC) or urolithiasis are more common. Unnecessary antibiotic treatment should be avoided.
- Sporadic bacterial cystitis is rare in intact male dogs. If these present with lower urinary tract signs they should be investigated for bacterial prostatitis.
- Diagnosis should be based on clinical signs. Urinalysis and sediment examination should always be performed. Bacterial culture should be done in all cats and is preferable in dogs.
- Cystocentesis is preferred unless contraindicated. Voided urine samples have greater potential for inaccurate results. Refrigerate samples and culture within 24 hours.
- Clinical signs are due to inflammation. Analgesic treatment alone has been effective in humans and may be worth trying in animals (e.g. NSAIDS initially, adding antibiotics in 3-4 days if justified by clinical signs).
- Amoxicillin (or alternatively amoxicillin/clavulanic acid) is a good first choice antimicrobial in most cases. As high amoxicillin concentrations are achieved in urine, clavulanic acid may be unnecessary even for beta-lactamase producing bacteria. Trimethoprim-sulphonamide combinations are an alternative but should be avoided in dogs susceptible to hepatopathy, keratoconjunctivitis sicca or skin eruptions.
- Initial treatment should be for 3-5 days only.
- Fluoroquinolones or third generation cephalosporins should be used only when sensitivity testing shows that first line options are inappropriate.

- Lack of clinical response after 48 hours should prompt further investigation.
- If pre-treatment sample culture results indicate resistance to the initial antimicrobial, change treatment unless there is a clinical response. However, lack of clinical response should not necessarily prompt a treatment change, rather, further investigation is indicated.
- If clinical signs resolve, post treatment urinalysis or culture is not required or recommended.

Recurrent bacterial cystitis

- Sometimes there is an underlying risk factor. Identifying this is critical as repeated antibiotic treatment is unlikely to provide long term resolution in such cases and may risk adverse effects, antimicrobial resistance and unwarranted costs.
- Treatment is similar to that for sporadic bacterial cystitis. Therapy longer than 3-5 days may be warranted if complicating factors such as bladder wall invasion are suspected.
- Microbiological cure is not necessarily required for clinical resolution.
- Culture during treatment is not recommended for short duration therapy and benefits are unclear even for long duration therapy.
- Post treatment culture may add information but positive results should not necessarily prompt additional treatment.
- Efforts to identify and control underlying causes of recurrent infection should be made. Differentials include endocrinopathies, renal, prostatic or bladder disease, congenital or conformation abnormalities, urolithiasis, incontinence, obesity and immunosuppression.
- Prophylactic antimicrobial treatment is not recommended.
- There is currently insufficient evidence to recommend cranberry extract products, methenamine or other alternative therapies.

Pyelonephritis

Pyelonephritis may be suspected when urine culture is positive and there are systemic signs such as lethargy, pyrexia or azotaemia, but signs may be vague and definitive diagnosis is difficult. Lower urinary tract signs or evidence of bacteraemia may be absent. Dilation of the renal pelvis is not specific for pyelonephritis. Pyelocentesis may be considered if culture of a cystocentesis sample is negative or not possible. Blood culture is

recommended in febrile or immunosuppressed animals and leptospirosis should be included in the differentials.

- Treatment targeting Enterobacteriaceae should begin immediately whilst awaiting culture. Amoxicillin (or amoxicillin/clavulanic acid) is a reasonable first choice. Oral treatment is recommended unless the patient is unwell enough to need intravenous antimicrobials.
- If sensitivity results indicate resistance to current treatment, a change of treatment may not be necessary if clinical response is good.
- If sensitivity results indicate the treatment should be effective but there has been no clinical response within 72 hours the diagnosis should be re-evaluated.
- Although prolonged (4-6 weeks) treatment has been previously recommended, human studies have shown no additional benefit over 7-14 days of treatment and in the absence of specific veterinary data, a 10-14 day treatment period is recommended.
- A recheck 1-2 weeks after cessation of treatment is recommended but a positive urine culture need not prompt additional treatment if there has been clinical resolution.

Bacterial prostatitis

The blood prostate barrier poses treatment challenges, especially in chronic prostatitis. Antimicrobials known to reach effective concentrations in the prostate should be used.

- Intact male dogs with bacteriuria should always be investigated for bacterial prostatitis, including rectal palpation and ultrasound evaluation if possible.
- If possible, cytology and culture should be performed on prostatic aspirates, third fraction of ejaculate, or prostatic fluid from urethral catheterisation or prostatic massage. Ultrasound guided aspirates or biopsies are preferred as diagnostic yield is higher and contamination less likely. Culture results on urine and prostate samples may be discordant.
- Any prostatic abscesses should be drained as medical treatment alone rarely resolves these.
- Empirical treatment should target Enterobacteriaceae. The blood prostate barrier limits the usefulness of penicillins, cephalosporins, aminoglycosides and tetracyclines. First choice options may be a fluoroquinolone (e.g. enrofloxacin) or trimethoprim-sulphonamide.
- There is limited data to guide treatment duration but 4 weeks is typically recommended for acute cases and 4-6 weeks for chronic disease. Poor response should prompt reassessment.
- Castration is recommended in dogs not required for breeding and should be performed as soon as possible.
- Prostate size and shape should be monitored post treatment.

Uroliths

- Urine culture should be performed in all cases of urolithiasis.
- Most struvite calculi in dogs are infection-induced but feline uroliths are usually sterile.
- If urine culture is negative, culture of canine struvite uroliths is recommended as only sterile uroliths need dietary management.
- Treatment should be based on culture results. If a non-urease producing organism (e.g. *Escherichia coli*) is isolated, antimicrobial treatment is not usually justified, unless cystitis is present.
- If dissolution fails to progress, urine culture should be repeated.

Subclinical bacteriuria

This is defined as positive bacterial culture from a properly collected urine specimen in the absence of clinical evidence of infectious urinary tract disease. It is not uncommon, 2-12% of healthy dogs, higher in diabetic, paralysed or other at risk groups. Prevalence in cats may be lower but rates of 1-13% in healthy cats have been reported. In humans there is ample evidence that treatment is not needed for asymptomatic bacteriuria, even in compromised patients, unless they are undergoing urological procedures. Clinical signs may be occult in animals that are paralysed or have unobservant owners but observation of e.g. odiferous urine or pyuria do not necessarily justify treatment.

- Treatment of subclinical bacteriuria is rarely justified and urine culture should not be performed when signs of urinary tract disease are absent if a positive result would not mandate treatment.
- Bacterial counts cannot differentiate subclinical bacteriuria from bacterial cystitis. Higher counts do not correlate to greater risk of disease.
- If it is unclear if clinical signs are related to cystitis, a 3-5 day treatment course may be considered but treatment should be stopped if there is no response as an infectious process is unlikely.
- Treatment of pyuria is not justified if clinical signs are absent.
- Isolation of a multi-resistant species is not a reason to treat subclinical bacteriuria.
- In patients unable to display signs (e.g. paralysed) clinical judgement is needed – systemic signs such as fever may justify treatment.

Urinary catheterisation

Catheterisation increases the risk of bacteriuria. Most cases will be subclinical but cystitis may occur. Aseptic catheter placement and maintenance are important. Routine catheter replacement to prevent bacteriuria is not recommended but duration of catheterisation should be as short as possible. Intermittent catheterisation could be considered in some patients. Routine cytology, culture or prophylactic antibiotics are not recommended for catheterised patients. Systemic signs such as fever, bacteraemia, or change in urine character should prompt investigation and cystocentesis is preferred. In those uncommon cases where treatment is needed, the recommendations are as for sporadic bacterial cystitis.

Urological surgery/invasive procedures:

In humans prophylactic antibiotics are recommended if preoperative cultures are positive and this approach may be justified for some veterinary procedures such as cystoscopy or urological surgery. Treatment for 3-5 days immediately before the procedure is suggested. Perioperative intravenous antibiotics could be considered in some cases.

Reference:

Weese JS, Blondeau J, Boothe D, et al. International Society for Companion Animal Infectious Diseases (ISCAID) guidelines for the diagnosis and management of bacterial urinary tract infections in dogs and cats. *The Veterinary Journal* 247: 8-25, 2019.

(Article previously published in VetScript, November 2019)

In brief

- After several recent client enquiries we have amended our invoices so our **bank account name** now appears alongside the other account details. This will help ensure you have the correct name to comply with new banking regulations.
  - > Account name: APHG NZ Investments Ltd t/a Awanui.
  - > Account No: 01-0102-0291961 00
  - > Swift Code: ANZBNZ22
- Just a reminder that **PMSG testing** is currently unavailable due to a faulty kit. We recommend performing an Oestrone Sulphate test at >100 days instead.

From page 5: ***Down the 'scope answer*** *- Moderate to marked pyogranulomatous inflammation with protozoal infection. The protozoal organisms were morphologically consistent with tachyzoites/zoites. Given their morphology, toxoplasmosis or neosporosis were considered, but Sarcocystis sp. organisms could not be excluded, while this was less likely. Serology testing for toxoplasmosis and neosporosis were subsequently performed. The toxoplasma titre was negative, and Neospora was 1/400.*

Contact us

- contacting Awanui Veterinary couldn't be easier.

EMAIL

auckland-vetlab@awanuigroup.co.nz
palmerston.vetlab@awanuigroup.co.nz
christchurch.vetlab@awanuigroup.co.nz
dunedin.vetlab@awanuigroup.co.nz

PHONE

0800 474 225

WEBSITE

www.awanuivets.co.nz

FACEBOOK

www.facebook.com/AwanuiVets

CATEGORY MANAGERS

- Rachel Howie - Rachel.howie@awanuigroup.co.nz - 027 604 8690
- Paul Fitzmaurice - Paul.fitzmaurice@awanuigroup.co.nz - 027 644 6892

LABORATORY MANAGERS

- Auckland - Trish Snegirev - Trish.Snegirev@awanuigroup.co.nz - 021 229 7979
- Palmerston North - Tara Gowland - Tara.gowland@awanuigroup.co.nz - 06 350 2944
- Christchurch - Daniel Westlake - Daniel.westlake@awanuigroup.co.nz - 03 363 6717
- Dunedin - Denise Carian-Smith - Denise.carian-smith@awanuigroup.co.nz - 03 489 2632

WEBSITE ADMINISTRATOR

- Karen Cooper - Karen.cooper@awanuigroup.co.nz - 027 290 8778

Awanui Veterinary