Urea poisoning in cattle

Introduction

Urea poisoning is a common cause of accidental toxicity resulting in cattle deaths in the Northern Territory. Urea is routinely included as a source of non-protein nitrogen (NPN) in feed supplements. Once consumed, the urea converts to ammonia in the rumen, where it is used by rumen microflora (bacteria and protozoa) to synthesise protein. This protein then becomes available to the animal through the normal processes of digestion and absorption.

However, if urea consumption exceeds the rate at which rumen organisms can metabolise it, excess ammonia is absorbed from the rumen into the blood instead. Absorbed ammonia converts back to urea in the liver, before excretion through the kidneys. This pathway can easily be overwhelmed if excess ammonia and urea circulate in the blood, causing poisoning. Poisoning can occur rapidly, from a few minutes to four hours after consumption, where cattle present with signs of nervous system dysfunction, bloat and sudden death.

Causes of urea poisoning

Causes include:

- excess consumption of urea by greedy feeders or as a result of over-supply
- sudden, rather than gradual introduction to high quantities of urea
- irregular consumption of urea
- wet supplement containing urea – higher consumption owing to dissolved chemical in fluids
- urea separating out from the supplement after transport, remixing prior to feeding is advised.

Signs of urea poisoning

Signs of poisoning can include twitching of ears and facial muscles, grinding teeth, frothy salivation, bloat, abdominal pain, frequent urination, rapid and laboured breathing, weakness, staggering, violent struggling and bellowing, and terminal spasms. It is common to find dead cattle near the source of the urea supplement and poisoning may affect multiple animals.

Diagnosis of urea poisoning

The most useful diagnostic indicators are a history of access to urea and the clinical signs shown by live, affected animals. There are a limited number of ways to confirm urea poisoning.

History of access to urea

Recent feeding history is important. Cattle become accustomed to metabolising urea, but if feeding is inconsistent with bursts of intermittent access, or a large amount suddenly fed to cattle that have not been receiving supplement, poisoning may occur. Urea is soluble and dissolves rapidly into puddles of water that form on blocks or in lick troughs after rain. Cattle that lick up these puddles may consume excess urea.

Recommended feeding quantities vary, according to what other feed is available, and whether the cattle are accustomed to urea. Tolerance decreases with starvation, and when animals otherwise only consume a low protein, high fibre diet, such as native vegetation from the end of the wet season and through the dry, without access to hay or silage. About 35g of urea per day is sufficient for a 400kg cow (approximately 0.1g/kg body weight). Urea should provide no more than 3% of the concentrate ration, or 1% of the total feed intake, and no more than one third of the total nitrogen intake should be NPN.

In cattle, 0.3-0.5g/kg/day (120 to 200g for a 400kg cow) is toxic and 1-1.5g/kg/day (400 to 600g for a 400kg cow) may be fatal.

Laboratory testing

It is possible to measure blood ammonia, but this is only useful in live, sick animals. Protein molecules circulating in the blood break down rapidly after death and produce ammonia; therefore, testing blood from dead animals is rarely of value. Handling and storage of blood after collection is very important. Collect blood into lithium heparin or EDTA tubes (green or pink top), chill immediately on ice and separate plasma within 30 minutes of collection. Plasma may be stored for two hours at 4°C before testing, or frozen immediately and kept frozen until ready to test. These restrictions on measuring blood ammonia make it impractical as a diagnostic test in most field situations.

If it is important and feasible to measure blood ammonia, collect blood from animals that appear unaffected as well as from sick animals, and treat all samples the same way. If all samples record elevated ammonia levels, then it is likely to be a non-specific finding owing to storage. To measure ammonia levels in rumen fluid straight after death, keep samples frozen until tested.

The most useful diagnostic sample is aqueous humour. This is fluid collected from the front of the eyeball of a recently deceased, affected animal, and stored frozen in a small container (approximately 1ml volume) after collection.

Post mortem and histology findings

Animals decompose rapidly after death from urea poisoning, with no specific changes seen in body tissues at post-mortem. Non-specific findings may include bloat and ammonia odour when opening the rumen. If pH strips are on hand, the rumen acid-base content may measure pH 7.5-8.0. Normal rumen pH is 5.7-7.3, with lower pH seen in cows that consume a high grain diet. There may be a large pool of rumen fluid on the ground at the nose of the beast. If the animal suffers severe bloat, the build-up of gases forces the rumen fluid out through the mouth when the animal dies.

Other findings may include excess fluid in the pericardial sac (around the heart), pulmonary oedema (fluid swelling in the lungs) with stable white foam in the large airways and haemorrhages (bleeding) on the heart wall.

There is very little in the literature on histopathological signs, which are those seen in the tissue cells under a microscope. Berrimah Veterinary Laboratories in the Territory have seen inflammatory changes in the rumen, particularly in animals that survived initial poisoning but died or were euthanised a day or 2 later. Submission of formalin-fixed sections of rumen and reticulum (the forestomaches) from animals that die from suspected urea poisoning may assist diagnosis.

Treatment of urea poisoning

Treatment is rarely effective. Passing a stomach tube can relieve bloat, followed by drenching the animal with a large volume of cold water. Use 45 litres for an adult cow, followed by 2 to 6L of 5% acetic acid or vinegar. This dilutes rumen contents, reduces rumen temperature and increases rumen acidity, which all help to slow down production of ammonia. Treatment may need repeating within 24 hours and relapses are common. Rumenotomy (surgical opening of the rumen by a veterinarian) and removal of rumen contents is a 'heroic' treatment approach for valuable animals.

Summary of best practice

- When introducing urea in supplement, start with pure salt and gradually introduce urea supplement, increasing it slowly to about 0.1g/kg body weight/day. This equates to 35 to 40g/day for a 400kg cow.
- Ensure that cattle get daily access to supplement after commencing supply.
- If cattle unavoidably miss urea supplementation for a couple of days, restart at a lower intake level.
- Prevent over-consumption of supplement mix or blocks by using salt to regulate intake.
- Feed supplement mixes or blocks under a roof to prevent urea getting wet and dissolving.
- Re-mix loose lick prior to feeding, in case urea has settled out during transport.
- Suspect urea poisoning if cattle die close to the supplement.

Differential diagnoses

It is important to establish the cause of death in outbreaks of urea poisoning. Collect and submit a full range of tissue samples from recently deceased cattle. Collect blood samples from unwell, live animals if possible, paying meticulous attention to blood sampling and storage requirements.

The clinical signs of urea poisoning in cattle are very similar to the signs of:

- lead poisoning
- salt poisoning
- liver failure
- polioencephalomalacia
- exotic or emergency animal diseases including rabies, Aujeszky's disease, and Australian bat lyssavirus.

A thorough investigation will rule-out infectious causes. Contact your regional field veterinarian or livestock biosecurity officer for further advice.

Livestock Biosecurity Branch contact details

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