Ruminal Acidosis: How to diagnose and how to treat

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BACKGROUND

Ruminal acidosis is a clinical disorder of cattle that can result in lameness, ruminal inflammation, liver abscesses, pneumonia, culling, and death. Of greater economic importance are losses that result from subclinical acidosis in dairy cattle, particularly those fed on pasture and grain, that may result in lameness, decreased intake of pasture and lower milk fat production. The risk of acidosis is present in all milk production systems, but especially when concentrates are fed. A large Australian study found that 10% of dairy cows less than 100 days in milk had acidosis when sampled (Bramley et al., 2008). Therefore, it is likely that many cows will experience some level of acidosis during lactation and indeed some may be affected many times. It can be estimated that if the prevalence of subacute acidosis is 10% and the duration of a case is 2 days, then there would be an incidence of approximately 1500 cases over a 300 day lactation in a 100 cow herd.

Ruminal acidosis is not simply one disorder, but rather a continuum of conditions that reflect the degree of generation and safe sequestration of hydrogen in the rumen. The degree of severity reflects the substrates available to cattle eg sugar and starch that predispose cattle to acidosis and the balance of the diet including fibre that reduces risk and protein that may have a bi-directional risk.

To simplify, however, we can consider acute acidosis that is usually present in low prevalence (or as an outbreak) or subacute acidosis that is very prevalent in Australian herds. It is important to recognize that lactic acid is usually only present in situations where sugars are present, whereas acidosis with high concentrations of volatile fatty acids, especially propionate and valerate is associated with excess starch ingestion (Golder, 2013).

DIAGNOSIS

Clinical signs

The clinical signs of ruminal acidosis usually reflect the severity of the case and are frequently not recognized or are subtle for milder cases of ruminal acidosis as their onset can occur after a time-lag from a predisposing event (Nordlund and Garrett, 1994). The occurrence of milder ruminal acidosis is often a herd problem (Enemark, 2008) and is difficult to diagnose in individuals. Many of the clinical signs associated with ruminal acidosis have many differential diagnoses (Britton and Stock., 1986); therefore, collective interpretation of all clinical signs observed is important.

Individual cattle

In individual cattle suspected of ruminal acidosis, a physical examination should be performed that includes measurements of heart rate, respiration rate, rate of rumen contractions, and body temperature. Locomotion, body condition, perineal staining, faecal consistency, and rumen fill should also be scored (Sprecher et al., 1997; Atkinson, 2009; Bramley et al., 2012). Further, demeanor, dehydration, and sites of pain should be assessed. Samples of rumen fluid should be taken; we prefer to use stomach tube methods.

Cattle with rumen perturbations consistent with subacute acidosis may present with a range of clinical and subclinical signs that include; diarrhoea, poor body condition, a dull and lethargic demeanor, dehydration, a lack of rumen fill, lameness, weak rumen contractions, depression in milk fat, and inappetance.

For acute acidosis: Ruminal distension, diarrhoea (often with grain in the faeces and a sickly, sweet smell), abdominal pain, tachycardia, tachypnea, staggering, recumbency, coma, a marked decline in milk yield, and death may occur (Oetzel, 2000; Krause and Oetzel, 2006).

The speed of progression of clinical signs may depend on the severity of rumen perturbation, but death can occur within 12 to 24 hours in peracute cases (Dirksen, 1970).

For chronic cases: Lameness with poverty lines, evidence of disseminated infections (especially hepatic and pulmonary), sporadic nosebleeds, and declines in milk production and weight loss may occur.

The key considerations in the diagnosis of acute cases of acidosis are the history of:

Access to sugars: Sources of sugars include forage beets, turnips, cereals that are frosted (eg oats, wheats), sugar, fruit and molasses.

Access to rapidly fermentable starch: Grains have the following order of risk of acidosis: wheat, triticale, barley, oats, maize, and sorghum (Opatpatanakit et al., 1994).

Adaptation: The less adapted to the substrate, the greater the risk to the cows. Most acute cases result from unlimited access to rapidly fermentable substrate and are usually obvious eg beef cattle breaking into a grain silo.

Diet structure: The lower the fibre in the diet, the greater the risk of acidosis– ie lush pastures with grain feeding. The risk is higher for dairy cows fed twice daily in the parlour than cows on partial mixed or total mixed rations.

Confirmation of diagnosis

Clinical signs as per the above. Sample of rumen fluids consistent with acute acidosis – low pH <5 and changes in the rumen fluid including a milky white appearance containing grain (if grain overload is the cause) and sickly sweet smell can be present.

Herd diagnosis: Subacute acidosis

While access to fermentable feeds is important to the diagnosis of subacute cases, the focus must be on the herd examination, as clinical signs of acidosis can be relatively subtle in the individual animal.

For Dairy: Check the latest herd test results. Milk fat to protein ratios less 1.02 to 1 for cows in the first 100 days in milk provide a weak, but useful, indication of acidosis. It is not true that all cows with a low test are likely to have acidosis, but cows with acidosis are very likely to have low fat test. The sensitivity and specificity for using a fat:protein ratio as a predictor of acidosis is 0.54 and 0.81, respectively.

The herd should be examined for the following:

Dung check: A good place is the dairy or feedyard (for beef cattle). If a high percentage of cattle are scouring (see photos), especially if the dung bubbles and contains grain – the risk of acidosis is high. Differential diagnoses (need to rule these out) ie very lush grass and parasites.

Lameness check: Only swelling of the coronary band occurs at the same time as ruminal acidosis BUT herds that have had acidosis causing other typical foot problems (see photos) that arise with aci

dosis often have active acidosis, especially if there has been no effort to control it. The changes in hooves that occur with acidosis of 'poverty lines' and paint brush haemorrhage indicate acidosis, but some time before examination.

Check the bulk vat: A low fat: protein test on a herd basis is similar to that in a cow. Again, it is only a rough guide, but a low herd fat:protein test is a cause to consider the possibility of acidosis.

History: Have cattle bled from the mouth (or nose) or have liver abscesses been reported for the farm? Both of these indicate that it is very, very likely the cows have had acidosis in the past. Some acidotic herds have history of increased respiratory disease, but there are many other things that cause respiratory disease apart from acidosis.

Ration: An essential step is to check the ration and feeding systems to see whether the following problems are present: Highly fermentable diets eg non-structural carbohydrates (**NSC**) >36% and Neutral Detergent Fibre (**NDF**) <32%. These need not be enough alone to provide a problem and problems can be present with less NSC and more NDF. Chemical analysis can be performed on individual feed components and residual TMR after feeding to obtain their percentage of dry matter, NDF, acid detergent fibre (**ADF**), crude protein (**CP**), starch, sugar, and NSC content (RAGFAR, 2007). This will allow estimation of the overall chemical composition of the cow's daily diet and for comparison with recommended requirements. This information, combined with the evaluation of the physical characteristics of the feed will indicate possible sub-optimum rumen function and ruminal acidosis (RAGFAR, 2007). It is often the way that the diet is fed eg cows can access extra grain in the milking parlour, very lush pastures or young grass, short chop or sorting in PMR or TMR herds or feedlots.

Feeding behaviour: Feeding behaviour of the herd including the following should be observed: percentage of cows cud chewing at rest should exceed 50%, sorting behaviour of a TMR, and DMI and whether cows are allowed to go straight to pasture after milking or are held to provide even access. Cows that have a low rumination time, are sorting their feed, have a cyclic feeding pattern, or low DMI may be at risk of ruminal acidosis (Britton et al., 1989; Maekawa et al., 2002). Cows that are low in the social order, which are frequently first lactation cows, often eat last and therefore can be exposed to feed with a different effective fibre content or chemical composition resulting from sorting from the previous cows and may increase their risk of ruminal acidosis (Kleen et al., 2003). All feed sources should be assessed for forage or chop length or particle size if applicable, and quality using relevant characteristics ie stage of maturity of pasture, type of pasture or forage.

Physical examination: Cattle should be checked for acidosis between 2 to 4 hours after feeding in the milk parlour or after receiving a PMR or TMR. These can be checked for ruminal pH – stomach tubing cattle is a quick and easy method for checking rumen contents. If more than 4 out of 10 cows have a low pH <6.5 on stomach tube or <6.0 on rumenocentesis, then, because these findings are present with other signs of acidosis, it is worth undertaking preventive steps to control acidosis. Within a herd, groups of cattle may be diagnosed with different ruminal conditions (Bramley et al., 2012). The sensitivity and specificity of using rumen pH values as a predictor of acidosis from rumen fluid collected using a stomach tube is 0.68 and 0.84, respectively, and from rumenocentesis is 0.74 and 0.79, respectively.

SECONDARY DISORDERS

Signs of one or more secondary disorders of ruminal acidosis may assist in the diagnosis of ruminal acidosis; however, many links between these disorders and ruminal acidosis have not been completely elucidated. At an individual cow level, clinical signs of secondary disorders of ruminal acidosis may have a differential diagnosis; however, a high prevalence of these signs at the herd level suggests that ruminal acidosis is also prevalent (Nordlund et al., 1995). There are several disorders believed to be secondary to ruminal acidosis including: laminitis (Nilsson, 1963), rumenitis (Enemark, 2008), epistaxis (Dirken, 1985), vena caval syndrome (Nordlund, 1995), cerebrocorticol necrosis (polioencephalomalacia) (Enemark et al., 2002), parakeratosis (Dirken, 1985), metabolic acidosis (Dunlop, 1972), and liver abcesses (Oetzel, 2000).

TREATMENT (Modified from RAGFAR, 2007)

Treatment of clinical acidosis can be difficult and chances of success depend on the severity of the case. Individual cattle can be treated successfully, however if a significant proportion of the herd is involved a triage approach is critical. The logistical challenge of providing treatment is substantial and prioritisation of cases to be treated based on severity, labour availability and expertise and value of the cattle is critical.

In a herd outbreak clinicians should spend some time identifying individuals of the highest value and assessing these first. Treatment of severe cases (dehydration >8%, collapsed and subnormal temperature, static rumen and evidence of diarrhoea) can be unrewarding, time consuming and expensive. These cases should only be treated when economic value determines that the attempt is warranted.

The following are our treatment protocols for acute cases – note that these are the high end options for treatment and economics and time will determine what you can do in the field. Good systems for administering and extracting fluids via stomach tube make a big difference to the speed with which cases can be treated.

TREATMENT PROTOCOL 1

- 1 g/kg bodyweight of sodium bicarbonate dissolved in 10 litres of water given via stomach tube followed by 0.5 g/kg bodyweight of sodium bicarbonate dissolved in 5 litres of water given by stomach tube every 6 hours for a further 2 to 3 treatments
- 0.5 g/kg bodyweight of magnesium hydroxide suspended in 5 litres of water given via stomach tube once only, after the first sodium bicarbonate
- 1mg/kg bodyweight of flunixin meglumine given by intravenous injection every 8 hours for a maximum of three treatments (other alternate anti-inflammatories are fine – eg ketoprofen)
- 10 mg/kg bodyweight of thiamine given by intramuscular injection every 24 hours for three treatments
- 12 mg/kg kg bodyweight of procaine penicillin G given by intramuscular injection twice daily for 3 days
- 1 ml/50kg bodyweight of chlorpheniramine maleate given by intramuscular injection every 8 hours for four treatments
- Calcium borogluconate 250 mls (either straight or as 4 in 1) subcutaneously
- Offer clover or legume and oat hay
- Monitor and re-introduce grain

TREATMENT PROTOCOL 2 (Severe)

- Instigate and maintain treatments outline in treatment protocol 1
- Inject 7500 mg of procaine penicillin G into the rumen via the left paralumbar fossa
- Administer hypertonic saline 2L slowly IV
- Administer 5 to 7 litres of 5% sodium bicarbonate solution intravenously over approximately 30 minutes
- Administer a further 150 ml/kg bodyweight of isotonic (1.3%) sodium bicarbonate solution intravenously over the next 12 hours

- Perform a rumenotomy and evacuate the rumen as per standard surgical technique described in Techniques in Large Animal Surgery, Second Edition. Turner AS and McIlwraith CW. Lea and Febiger, Philadelphia. 1989.
- Replace rumen contents with at least 12L of contents obtained by stomach tube from one or several other cows.
- Offer clover or legume and oat hay
- Monitor and hopefully re-introduce grain

TREATMENT AND PREVENTION OF SUBACUTE ACIDOSIS

- Reduce the amount of concentrate fed – it is not being used effectively (bring back to 36% NFC). Consider, if cows are high producing, using fats to keep the energy content of the diet up.
- Provide effective fibre – long hay or silage (minimum chop length >2.5 cm) to bring the ration up to 30 to 32% NDF and 20 to 22% effective NDF (or if grass is lush and the NDF is up to 32% – provide around 1 kg of good cereal or pasture hay or 0.5 kg straw). Ensure even access and check that it is effective by checking whether ~ 50% of cows are chewing their cud at rest).
- Provide even access to concentrates and forages. The formulated diet is one thing, what the cows eat is another. In particular, check that heifers get equal access to pasture (release all cattle to pasture at the same time or provide a break for later milked cows). In TMR/ PMR, ensure that stanchions are not over full or troughing is sufficient – consider a heifer string.
- Consider use of feed additives to reduce the risk of acidosis eg buffers, ionophores. These have some efficacy in controlling acidosis and act by different means.
- In herds where access continues to be uneven and control is difficult, consider using rumen modifiers, including antibiotics, to control fermentation. The only registered product for acidosis is virginiamycin and where herds are at high risk should be considered. The combination of monensin and tylosin is effective in reducing the risk of liver abcessation (an outcome of acidosis) and can be considered as an option. These products are available to veterinarians as pre-formulated pellets that can be mixed with grain or other feeds to reduce the risk of poor distribution and producer errors and occupational health and safety with powders containing ionophores and antibiotics (see RAGFAR 2007 for prudent use advice).
- It is apparent that some herds have a high inherent risk of acidosis, possibly reflecting large differences in responses of cattle to fermentable carbohydrates (Golder 2013).

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