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Ruminal Acidosis in Milk-fed Calves

By Arcangelo Gentile, DVM, Dip. ECBHM

Ruminal acidosis in pre-ruminant calves is caused primarily by the inefficient function of the esophageal groove, producing an abnormal accumulation of fermentable liquid in the rumen. This issue of *Large Animal Veterinary Rounds* describes the most important factors that induce esophageal groove dysfunction and the etiological, pathogenic, and clinical aspects of ruminal acidosis in milk-fed calves. This issue will also focus on the role of lactic acid and disturbances in systemic acid-base metabolism, clinical findings associated with the acute and chronic forms of ruminal acidosis, characteristics of ruminal fluid, and guidelines for therapy.

Dysfunction of the esophageal groove reflex, with pooling of liquid in the forestomachs, is a possible complication of some neonatal calf diseases, especially diarrhea.[1] In the author's opinion, ruminal acidosis is an underestimated pathological condition. It is caused by fermentative disorders that occur when dysfunction of the esophageal (reticular) groove reflex allows milk to spill into the reticulo-rumen instead of being delivered directly into the abomasum.[2,3] In this situation, the calf is considered to be a "ruminal drinker."[4] In milk-fed calves, the main consequence of this "ruminal drinking" is acidification in the forestomachs that may provoke severe and sometimes lethal indigestion. Although scientific evidence is lacking, case reports and personal experience suggest that Simmental calves are predisposed to this condition. Also, in veal calves, stress factors and feeding practices may induce failure of esophageal groove closure; in this case, chronic disease usually develops.

Although pre-ruminant calves are considered to be functionally monogastric animals, the reticulo-rumen can undergo life-threatening pathological processes from the first days of life. Similar to adult cattle, there are 2 mechanisms that can lower rumen-pH in milk-fed calves:

- reflux of abomasal juice rich in hydrochloric acid
- fermentation, with accumulation of medium or strong organic acids.

In the case of abomaso-ruminal reflux, abomasal content flows back into the reticulum and the rumen as a consequence of disturbances in abomasal motility. This can occur in cases of right-side abomasal displacement, chronic pyloric ulceration with cicatricial narrowing, or when the abomasum is trapped in an umbilical hernia. In these instances, emptying of the abomasum is delayed due to mechanical obstruction. Abomasal inflammation, peritonitis, or omental bursitis delay emptying of the abomasum secondary to motility depression.[1] Reflux of milk may also be caused by overflow from the abomasum when its capacity is exceeded (eg, when >2 liters are fed to young calves during the first 2 weeks of life). In order to lower rumen pH, reflux of abomasal juice must be large. Clinical signs of this kind of ruminal acidosis are influenced by the primary problem and, therefore, signs of ruminal acidification may be overshadowed by those of the initiating disease.

Dysfunction of the esophageal groove

The esophageal groove extends from the cardia to the reticulo-omasal orifice. In milk-fed calves, its closure enables ingested fluid to pass directly to the abomasum. Sucking is an important part of the

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Table 1: Main causes of esophageal groove dysfunction

Pathological conditions	• neonatal diarrhea • "essential anorexia" • pain eg, painful cough, otitis, phlebitis of the jugular vein
Inadequate feeding techniques	• irregular feeding times • low-quality milk replacer • cold temperature of the milk • drinking from an open bucket
Forced feeding	• discomfort caused by the passage of the feeding tube inhibits the groove closure reflex
Stress factors	• long-distance transportation

stimulus for closure of the reticular groove; moreover, it also restricts the speed of fluid ingestion. Spillage from the esophageal groove may result from either a complete failure of groove closure or sequential opening and closure during drinking. Milk may also leak from the closed reticular groove when large volumes of fluid are swallowed which, due to excess pressure, force the groove lips apart. Dysfunction of the esophageal groove reflex can occur with (Table 1):

- diseases with systemic effects (eg, neonatal diarrhea,[3] "essential anorexia,"[5] and painful situations such as painful cough, otitis, or phlebitis of the jugular vein)
- inadequate feeding techniques (eg, irregular feeding times, poor quality milk replacer, cold temperature of the milk, drinking from an open bucket [calves suckling directly from their dam do not experience dysfunction from this cause])
- tube feeding (discomfort caused by passage of the feed inhibits the groove closure reflex)[6]
- stress factors such as long distance transportation.[4]

Consequences of dysfunction of the esophageal groove

In cases of dysfunction of the esophageal groove, lactose or other easily fermentable carbohydrates contained in the milk or nutrient solution (sometimes called "diet drinks") fall into the forestomach cavity and are fermented by the ruminal flora. Lactic acid[7] and short-chain fatty acids are produced,[7,8] but the most detrimental product is lactic acid.[9] Unlike eukaryotic cells that produce L-lactic acid almost exclusively, prokaryotic cells, such as lactobacilli, can produce both L- and D-lactic acid in substantial amounts. High quantities of both these isomers can therefore accumulate in the rumen.[9]

As a consequence of the accumulation of organic acids in the rumen, rumen pH drops from physiological levels (around 6.5 – 7.5), to very low levels (even below 4.0).[7,10] A single episode of ruminal drinking is responsible for only transitory acid production that does not produce noteworthy clinical

Table 2: Main consequences of ruminal acidification

Local (gastro-intestinal) repercussions	
Inflammation of the forestomach mucosa	• reticulitis – further dysfunction of the esophageal groove • rumenitis – further dysfunction of the esophageal groove • omasitis
Inflammation of the gastric mucosa	• abomasitis
Hyper/parakeratosis of the forestomach mucosa (in cases of protracted ruminal acidosis)	
Disturbances of ruminal motility	• chronic or recurrent ruminal bloat
Atrophy of the intestinal villi	• malabsorption and maldigestion of nutrients
Systemic repercussions	
Metabolic acidosis (acidemia expected in about 50% of cases)	
D-hyperlactatemia	
Negative energy and protein balance	
Impairment of non specific immunity	• reduction of serum lysozyme

repercussions. As soon as the fermentable substrate is emptied or removed, the rumen regains its physiological pH.[11] However, when fermentable carbohydrates are persistently present in the rumen (ie, in cases of delayed emptying due to ruminal atony or hypomotility, or in cases of recurrent arrival of substrate in the rumen), acidification persists and gradually causes pathological alterations at both local and systemic levels (Table 2).

Local effects

At the local level – the level of the gastrointestinal tract – different grades of inflammation of the forestomach mucosa (reticulitis, rumenitis, omasitis) and of the abomasums are observed.[12,13] Severe inflammation in the proximity of the esophageal groove can further compromise the proper functioning of the esophageal groove reflex, starting a vicious cycle.[14] If abnormal fermentation does not provoke a severe and, possibly, lethal alteration, and if the abnormal fermentation continues for 1-2 weeks, hyper- or parakeratosis of the forestomach mucosa can develop.[12] These alterations may induce disturbances in forestomach motility, causing chronic or recurrent ruminal bloat. Other consequences of chronic ruminal drinking are atrophy of the intestinal villi and a decrease in brush border enzyme activities.[15,16] These changes cause maldigestion and malabsorption of nutrients.

Systemic effects

Systemic repercussions are mainly due to the absorption of organic acids, chiefly lactic acid, from the intestinal tract

Table 3: Clinical forms of ruminal acidosis in milking calves

Acute ruminal acidosis

• Dysfunction of the esophageal groove as a consequence of other pathological conditions such as:
  • neonatal diarrhea
  • "essential anorexia"
  • pain (painful cough, otitis, phlebitis of the jugular vein)

• Secondary to impaired functioning of the esophageal groove or tube feeding
• Young calves, fed cow milk, are usually affected (1-3 weeks old)
• The onset is usually rapid and severe, the condition is sometimes fatal
Chronic ruminal acidosis

• Dysfunction of the esophageal groove is primary, may be precipitated by stressful situations:
  • Prolonged transport to an assembly centre and onwards to fattening unit
  • Grouping or mixing

• It should be considered a primary disease ("ruminal drinking syndrome")
• Veal calves fed milk replacers at the beginning of the fattening period are usually affected (3-8 weeks old)

and also likely from the rumen. The increased acid load in the bloodstream can lead to metabolic acidosis. It is uncertain to what extent low rumen pH is reflected in a systemic metabolic acidosis, but it appears that acidemia may occur in 50% of cases.[17] Among the organic acids, both the L- and D-isomers of lactic acid formed in the rumen might be responsible for the development of metabolic acidosis. However, L-lactic acid is cleared more rapidly by the calf and only D-lactate tends to accumulate in the blood.[9] The commonly claimed mechanism for this unilateral accumulation is that mammals lack specific enzymes for D-lactate metabolism, whereas they possess a very efficient enzyme for L-lactate metabolism – L-lactate dehydrogenase. There is a non-specific enzyme for D-lactate metabolism,[18] but it has only limited efficiency. This explains why in cases of ruminal acidosis, the calf is not able to metabolize all the absorbed D-lactic acid. As a consequence, this isomer tends to preferentially accumulate in the blood and interstitial space.[9]

It has been recently demonstrated that D-hyperlactatemia may explain some of the clinical findings observed in calves affected by ruminal acidosis, particularly depression, reluctance to move, ataxia, and impaired palpebral reflex.[19,20]

Clinical course

There are two different forms of fermentative ruminal acidosis in calves, the acute and a chronic form[13] (Table 3).

In acute ruminal acidosis, dysfunction of the esophageal groove usually occurs secondary to another disturbance. In our experience, this acute and sometimes severe form is typical of young calves fed cow's milk during their first weeks of life. It is characterized by severe clinical signs and often has a lethal outcome. Force-feeding sick calves is also a risk factor for acute ruminal acidosis.[21,22] Force-feeding usually indicates that the calf is not drinking because it is already inappetent due to another disease. As a consequence of a combination of poor functioning of the groove secondary to the primary disease, reduced stimulus for groove closure, and discomfort from passage of the tube (or simply bypassing the groove with a long tube), the feeding solution is likely to be deposited in the rumen.

In the chronic form, dysfunction of the esophageal groove occurs primarily in initially healthy calves, the result of stressful situations, such as prolonged transportation to assembly centres and onwards to fattening units or new groupings.[23] In this case, ruminal acidification is not superimposed on another pre-existent pathological condition. The chronic form mainly affects calves purchased for veal production. During the first weeks in the fattening unit, affected calves experience recurring dysfunction of the esophageal groove. Clinical signs are initially mild and only begin after some weeks.[24] Due to the different compositions of commercially-available milk replacers and the consequent different kinds of fermentation processes, the milk replacer can play an important role in the clinical evolution of the disease.[25]

Clinical findings

Calves affected by acute ruminal acidosis[13,26] appear to be in pain, demonstrating weakness, depression, prolonged recumbency, reluctance to move, a stance with an arched back, inappetence, dehydration, cardio-circulatory collapse, and they may eventually die. Grinding the teeth can also be a sign of rumenitis. The abdomen may be slightly to moderately distended and ballotment of the left flank usually produces loud splashing sounds that can be heard without a stethoscope. Finger percussion may produce a ping over the left flank. Auscultating a ruminal drinker in the left flank during drinking reveals the typical plunging sound of the milk flowing into the fluid-filled rumen. In contrast, in calves that are drinking normally, a soft gurgling is heard, which originates from the passage of the milk through the closed reticular groove. When the course of the disease is prolonged, calves may have patches of alopecia that sometimes can be widely distributed. When a tube is inserted into the forestomach, rumen content usually flows out spontaneously.

The chronic form is mainly characterized by general malaise and depression, poor sucking reflex, poor or retarded growth, dry and scaly haircoat, extensive hair loss, refusal of milk, recurrent tympany, ventral abdominal distension, weakness, and passing of feces with a putty-like appearance (sticky clay-like feces that are solid and white in colour).[27]

Figure 1: A calf affected by "ruminal drinking." Ruminal fluid flows spontaneously out through a foal nasogastric tube introduced into the mouth. Ruminal fluid has a white colour and contains milk coagulum.

Ruminal fluid analysis

Ruminal fluid analysis is the most important diagnostic tool.[13,26] It may be necessary to aspirate the liquid with a vacuum pump but, usually, liquid flows out spontaneously, particularly in cases of ruminal drinking (Figure 1). A foal nasogastric tube with a weighted endpiece, can be useful in obtaining a sample. The tube should be introduced into the mouth, taking care that it is not bitten by the teeth. To avoid this, the fingers of the examiner should keep the tube pushed against the median raphe of the hard palate.

Besides pH, the colour, smell, consistency, and presence of casein coagula or other particles should be evaluated. Normal ruminal juice is a light beige colour, musty or stale smelling, has a watery consistency, and a pH between 6.5 and 7.5.[28] In cases of ruminal drinking, the rumen fluid usually has a milk-like appearance and its pH may fall to values below 4.0. It usually has a pungent, sour smell and may be rich in milk coagulum (Figure 1). Occasionally, the ruminal fluid looks like fermented milk (yogurt).The colour depends on the kind of liquid that has been ingested. Milk gives a white colour. However, if the liquid is an energy-containing solution, the colour will depend on the components of the solution and the type of fibers, vegetable gum, energy sources, vegetable fats, cocoa, or gruel that it contains.

Treatment

If there are severe metabolic alterations, such as dehydration and metabolic acidosis, initial treatment should focus on replacement of fluids and restoring acid-base balance. Since acute ruminal acidosis often occurs as a secondary process, the next goal is to treat the primary disease that provoked dysfunction of the esophageal groove.[26] This is of the utmost importance; eliminating the cause of the groove dysfunction will prevent other fermentable substrate from falling into the rumen.

Occasionally, it may be useful to carry out a rumen lavage. Sour ruminal content is siphoned off through a stomach tube and the rumen cavity is repeatedly flushed with 1-2 liters of warm tap water until the contents have lost their sour smell. Rumen lavage is not necessary in all cases. It is indicated especially when the primary disease cannot be accurately diagnosed or when it cannot be treated effectively by other means. It may be particularly useful if the calf has been repeatedly force-fed or the ruminal juice contains many clots of casein.[26,29]

Calves that fail to improve their esophageal groove reflex despite these measures may be deprived of their milk ration.[30] In this case, the mucosa can "rest" without being assaulted by the irritating action of strong acids. In our experience, withholding feed is indicated if the pH of the ruminal fluid remains acidic following ruminal lavage on 2 consecutive days. We suggest withholding milk for 1-2 days. During this fasting period, calves should receive parenteral nutrition with 2 liters of physiological saline and 2 liters of hypertonic glucose solution (20%) every 24 hours.[26] Drinking water should always be available. Due to the insufficient energy supply of this kind of nutrition, this approach is not recommended for cachectic calves.

Another important measure is to train the calf to drink correctly. This can be achieved by allowing the calf to suck on a finger before offering the nipple. Appetite, sucking reflex, and function of the esophageal groove can be stimulated by offering the daily milk ration in 3-4 portions. Calves always need tenderness and care, especially if they are timid, fearful, or weak.

If, despite treatment, the calf does not improve, it should be weaned abruptly and given hay and concentrates. Calves may be convinced to begin eating by patiently introducing some hay directly into their mouths.[5]

Short bowel syndrome in human medicine

The pathogenesis of ruminal acidosis in cattle (both adult and milk-fed calves) has some interesting overlaps with the "short bowel syndrome" in human medicine."[31] Short bowel syndrome can affect people who undergo a massive small bowel resection because of different circumstances (eg, necrotizing enterocolitis, malrotation with volvulus, vascular accidents of mesenteric circulation). Beside gastroenteric abnormalities, the disease is characterized by neurological symptoms (eg, confusion, loss of memory, slurred speech, and inappropriate behaviour).[32]

In this syndrome, intestinal absorption is greatly reduced and, therefore, patients experience malabsorption. As a result, a massive amount of fermentable carbohydrates reaches the proximal bowel lumen. The physical and microbial conditions in the human colon can be compared with those in the calf rumen. Quantitatively and qualitatively, the flora, pH, and redox potential are similar. If the small intestine is resected or bypassed, a large quantity of unabsorbed starch can reach the large bowel and be fermented. The consequent production of short-chain fatty acids and lactic acid induces a drop in the pH of the bowel. The D- and L-lactic acids absorbed under these conditions result in metabolic acidosis. L-lactate can be metabolized, but because humans lack D-lactate dehydrogenase, this isomer accumulates in the blood and concentrations increase to >10 mmol/L (normal values 0.1-0.3 mmol/L).[33] It is not known if the neurological symptoms and signs are proportionate to the degree of D-lactate intoxication, but it is believed that clinical findings can appear with blood concentrations >3 mmol/L. It is also unclear if the D-lactate or some other metabolite or toxin produced in parallel with it causes the syndrome of intoxication.[32-34]

Conclusion

Ruminal acidosis should be considered a possibility every time a milk-fed calf suffers neonatal diarrhea or other diseases that compromise the correct functioning of the esophageal groove reflex. The consequences of such ruminal alterations may be very detrimental for the calf and include both local and systemic repercussions, with reticulo-ruminitis and metabolic acidosis, respectively, being the most important. The cause of metabolic alterations is absorption of both isomers of lactic acid formed in the rumen. However, due to the different metabolic pathways, only D-lactate tends to accumulate in the blood. This isomer directly causes some of the signs of depression that are part of the clinical picture of the disease.

The course of the disease can be acute or chronic. Passing a tube facilitates both diagnosis via ruminal juice analysis and therapy using ruminal lavage. Other important steps include correction of any underlying primary conditions and restoring the fluid and acid-base balance. Temporary deprivation of the milk ration may be tried when dysfunction of the esophageal groove persists.

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Abstract of Interest

Investigations on the influence of serum D-lactate levels on clinical signs in calves with metabolic acidosis.

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Correlations between the degree of acidosis and clinical signs (changes in posture, behaviour, intensity of suckling reflex) in neonatal diarrhoeic calves have been described in various studies. However, base excess values varied widely in calves exhibiting similar clinical symptoms. The objective of this study was to elucidate whether the clinical picture of acidotic calves with neonatal diarrhoea is influenced more by D-lactate concentration than by degree of acidosis. Eighty calves up to three weeks old that were admitted to the II Medical Animal Clinic with acute diarrhoea and base excess values -10 and -25 mmol/L were included in the prospective study. Posture, behaviour, suckling and palpebral reflexes, and position of the eyeballs were scored during the initial examination. Base excess and serum D-lactate and urea concentrations were determined in venous blood. In order to quantify the influences of base excess and D-lactate on the clinical parameters, groups of different clinical categories were compared. The results show that variations in behaviour, and in posture can be better explained by elevations of serum D-lactate concentrations than by decreases in base excess. Disturbances of the palpebral reflex appear to be almost completely caused by high levels of D-lactate.

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