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DISEASE OF FORESTOMACHS IN RUMENANT

Acute Carbohydrate Engorgement of ruminants: (Ruminal lactic acidosis, Rumen overload)

ETIOLOGY

Acute ruminal acidosis is most commonly caused by the sudden ingestion of toxic doses of carbohydrate-rich feed, such as grain. Less common causes include engorgement with apples, grapes, bread, baker's dough. **Subacute ruminal acidosis** (**SARA**) in dairy cattle is a disorder of ruminal fermentation in dairy cattle caused by the ingestion of large amounts of concentrates and inadequate amounts of fiber administered to increase milk production in early lactation lead to problem.

EPIDEMIOLOGY

All types of **ruminant** cattle, sheep and goat are susceptible to acute ruminal acidosis, but the disease is most common in feedlot cattle and it also occurs in lamb feedlots.

Because the type and level of ration consumed by a ruminant affects the numbers and species of bacteria and protozoa in the rumen. a change from one ration to another requires a period of microbial adaptation, which is a variable interval of time before stabilization occurs. This results in the rapid onset of abnormal fermentation.

In beef and lamb feedlots the rapid introduction of high-level grain diets is a major risk factor.

Outbreaks occur when animals gain access to a large quantity of grain. High mortality rate when large quantity of grain ingested.

Subacute ruminal acidosis is considered an important problem in dairy herds.

The morbidity will vary from 10% to 50%. The case–fatality rate may
be up to 90% in untreated cases, whereas in treated cases it still may be up to 30% to 40%.

All grains are more toxic when ground finely or even crushed or just cracked, which are processes that expose the starch component of the grain to the ruminal microflora and increase fermentation and increase problem.

PATHOGENESIS

1-Changes in Rumen Microflora:

The ingestion of excessive quantities of highly fermentable feeds by a ruminant is followed within 2 to 6 hours by a marked change in the microbial population in the rumen.

increase in the number of ***Streptococcus bovis***, which use the carbohydrate to produce large quantities of lactic acid.

In the presence of a sufficient amount of carbohydrate (a toxic or a lethal amount) the organism will continue to produce lactic acid, which decreases the rumen pH to 5 or less, which results in the destruction of the rumenal bacteria and protozoa.

2-Volatile Fatty Acids and Lactic Acid in the Rumen:

The concentration of volatile fatty acids increases initially, contributing to the **fall in ruminal pH**. The low pH allows **lactobacilli** to use the large quantities of carbohydrate in the rumen to produce excessive quantities of

lactic acid, resulting in **ruminal lactic acidosis**.

Both D and L forms of the acid are produced, which markedly **increases ruminal osmolality**, and **water is drawn** in from the systemic circulation, causing **hemoconcentration and dehydration**.

Increase rumen osmolality plays an important role in **decreasing appetite** and increasing dehydration.

Some of the **lactic acid is buffered by ruminal buffers**, but large amounts are **absorbed** by the rumen and some **moves** into and is absorbed further **down the intestinal tract and cause diarrhoae** which is similar to the effects of orally administered magnesium sulfate.

Lactate is a 10 times stronger acid than the volatile fatty acids, and accumulation of lactate eventually exceeds the buffering capacity of rumen fluid.

As the ruminal pH declines, the amplitude and frequency of the **rumen contractions are decreased**, and at about a pH of 5 there is **ruminal atony**.

3-Systemic Lactic Acidosis:

The absorption lactic acid from rumen to circulation, The L-lactate is rapidly metabolized to bicarbonate and increasing blood pH toward the normal range. D-lactate is very slowly metabolized and plasma concentrations decrease primarily by renal excretion, which is low in advanced cases of ruminal acidosis because of marked dehydration.

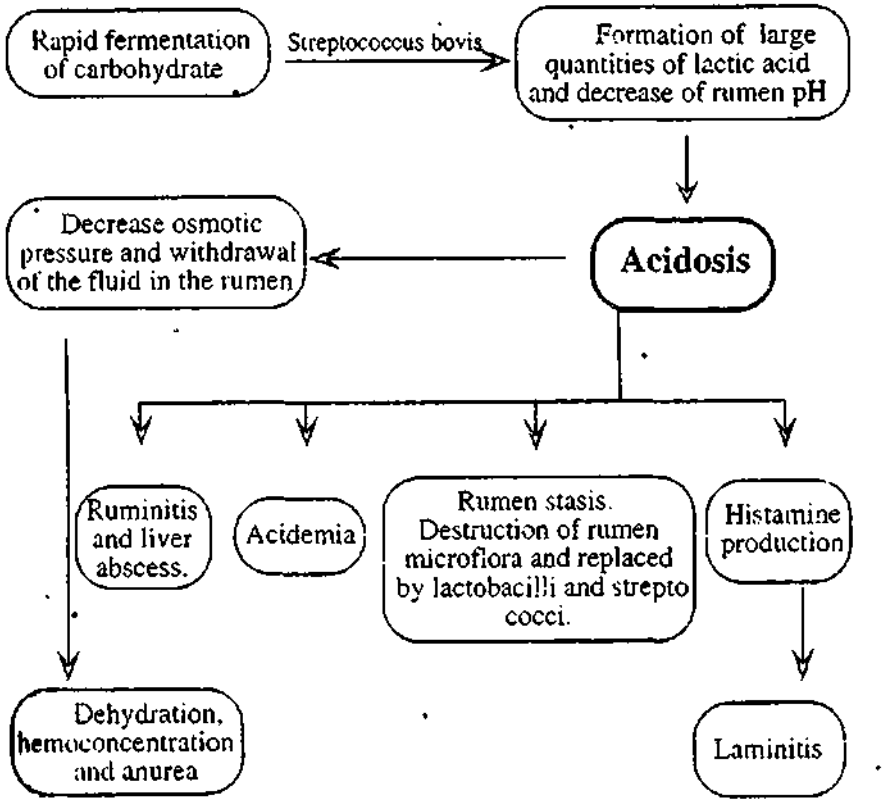
In animals with mild or moderate dehydration that survive the acute form of the disease, the rapid clearance of L-lactate and D-lactate and other compensatory mechanisms may overcompensate, resulting in alkalosis.

In severe cases of lactic acidosis the reserves of plasma bicarbonate are reduced, the blood pH declines steadily, and the blood pressure and renal blood flow decline, causing a decrease in perfusion pressure and oxygen supply to peripheral tissues. This results in a further increase in lactic acid from cellular respiration and decreased elimination of d-lactate in the urine.

4-Chemical and Mycotic Rumenitis:
The high concentration of lactic acid in the rumen causes chemical rumenitis, which is the precursor for mycotic rumenitis in those that survive; this occurs about 4 to 6 days later.
The low pH of the rumen favors the growth of *Mucor, Rhizopus,* and *Absidia* spp., which invade the ruminal vessels, causing thrombosis and infarction.
Severe bacterial rumenitis also occurs.
Widespread **necrosis** and **gangrene** may affect the entire ventral half of the ruminal walls and lead to the development of an **acute peritonitis**.
complete atony and this, together with the toxemia resulting from the gangrene, is usually sufficient to cause **death**.

5-Hepatic Abscesses:
In uncomplicated chemical rumenitis, the ruminal mucosa sloughs and heals with scar tissue and some mucosal regeneration.
Hepatic abscesses commonly occur as a complication as a result of a combination of rumenitis caused by lactic acidosis and allowing *Fusobacterium necrophorum* (formerly *Arcanobacterium* or *Corynebacterium pyogenes*) to enter directly into ruminal vessels and spread to the liver, which may have also undergone injury from the lactic acidosis. necrosis and hyperplasia of the bile duct epithelium and degeneration of renal tubules may also be present histologically.

6- Laminitis and Lameness:
Laminitis occurs in acute, subclinical, and chronic forms associated with varying degrees of severity of ruminal acidosis.
The association between acidosis and laminitis appears to be associated with altered hemodynamics of the peripheral microvasculature.
Vasoactive substances are released during the decline of rumen pH and the bacteriolysis and tissue degradation, These substances cause vasoconstriction and dilatation, which injure the microvasculature of the
Corium, Ischemia results, which causes a reduction in oxygen and nutrients reaching the extremities of digits.
Another causes of lameness may be duo to increase of histamine level in body cause peripheral vasodilation and alteration of osmotic pressure so intravascular fluid escape to extravascular space lead to pressure on the sensitive lamina in digits and cause pain, lameness appear on the affected animal.



CLINICAL FINDINGS

The speed of onset of the illness varies with the nature of the feed. The severity increases with the amount of feed eaten. If cattle are examined clinically within a few hours after engorgement, the only abnormalities that may be detectable are a **distended rumen** and **abdomen**, and occasionally some abdominal discomfort, evidenced by kicking at the belly. In the **mild form**; affected cattle are **anorexic** and still fairly bright and alert, and the **feces may be softer than normal**, Rumen **movements** are **reduced** but not entirely absent, Affected cattle do **not ruminate** for a few days but usually begin to eat on the third or fourth day without any specific treatment.

In **outbreaks of the severe form**, within 24 to 48 hours some animals will be **recumbent**, some **staggering**, and others standing quietly alone. most affected cattle are **anorexic** and **depressed**, **teeth grinding** may occur in about 25% of affected sheep and goats. Once they are ill they usually do not drink water, but cattle may engorge themselves on water if it is readily available immediately after consuming large quantities of dry grain. In an outbreak, inspection of the feces on the ground will usually reveal many spots of soft to watery feces, **depression**, **dehydration**, **inactivity**, **weakness**, **abdominal distension**, **diarrhea,** and **anorexia are typical**. The temperature is usually **below** normal, 36.5°C to 38.5°C but animals exposed to the sun may have temperatures up to 41°C (106°F). In sheep and goats, the rectal temperatures may be slightly higher than normal. The **heart rate** in cattle is usually increased and continues to increase with the severity of the acidosis and **circulatory failure**. Generally, the prognosis is better in those with heart rates below 100 beats/min than those with rates up to 120 to 140 beats/min. In sheep and goats, the heart rate may

be higher than 100 beats/min. The **respirations** are usually shallow and increased up to 60 to 90 beats/ min. A mucopurulent discharge is common because animals fail to lick their nares.
absence of feces is considered by some veterinarians as a grave prognostic sign, but diarrhea is much more common.
The **dehydration is severe and progressive.** In mild cases, the dehydration is about 4% to 6% BW with severe involvement up to 10% to 12% BW. **Anuria** is a common finding in acute cases, and diuresis following fluid therapy is a good prognostic sign.
Careful examination of the rumen is important. The rumen contents palpated through the left paralumbar fossa may feel firm and doughy in cattle that were previously on a roughage diet and have consume large amount of grain.
Acute laminitis may be present and is most common in cases that are not severely affected and appear to be good treatment risks. Affected animals are lame in all four feet, they walk slowly, and may be reluctant to stand. The **lameness** commonly resolves if the animal recovers from the acute acidosis. Evidence of chronic laminitis may develop several weeks later.
Recumbency usually follows after about 48 hours but may occur earlier. Affected animals lie quietly, often with their **heads turned into the flank**, and their response to any stimulus is much decreased so that they resemble **parturient paresis**.

Table: clinical forms of carbohydrate engorgement in rumenant.

Degree of illness	Mental state and muscular strength	Degree of dehydration (% of BW)	Abdominal distension	Heart rate (min)	Body temp. (°C)	State of rumen; fullness, consistency of contents, movements and pH
Percute	Severely depressed, weak, in lateral recumbency, unable to stand, apparent blindness, sunken eye ball and slow response	8–12	Prominent	110–130	35.5–38.0	Distended with fluid and soft rumen contents, complete stasis, pH below 5 and usually about 4 No protozoa
Acute	Depressed, still able to walk but ataxic, complete anorexia, may want to drink water, sunken eye ball and slow response	8–10	Moderate	90–100	38.5–39.5	Distended with fluid, complete stasis, sweet–sour smelling fluid contents Rumen pH between 5 and 6 No protozoa
Sub acute	Fairly bright and alert, able to walk, no ataxia, may eat, usually wants to drink, eye ball normal	4–6 (Just barely detectable clinically)	Mild Or none	72–84	38.5–39.0	Moderate distension with fluid, some doughy ruminal ingesta palpable, some weak ruminal contractions, rumen pH between 5.5 and 6.5 Some protozoa alive
Mild	Bright and alert, able to walk, no ataxia, eats and drinks normally	Not detectable clinically	Not significant	Normal	Normal 38.5–39.0	No detectable distension, ruminal contents palpable, ruminal contractions still present but not as strong as normal, rumen pH 6.5–7 Almost normal protozoal activity

CLINICAL PATHOLOGY

The severity of the disease can usually be determined by clinical examination, but field and laboratory tests are of some additional value, particularly in diagnosing SARA in lactating dairy cattle.

1- Ruminal Fluid pH:

The pH of the ruminal fluid obtained by specially designed stomach tubes or by rumenocentesis through the left ventral abdominal region can be measured in the field using wide-range pH (2–12) indicator paper. The ruminal fluid must be examined immediately because the pH will increase on exposure to air.

Cattle that have been fed a roughage diet will have a ruminal pH of 6 to 7; for those on a grain diet it will be 5.5 to 6.
A ruminal pH of 5 to 6 in roughage-fed cattle suggests a moderate degree of abnormality,
but a pH of less than 5 suggests severe grain overload and the need for aggressive treatment.

2- **Ruminal Protozoa**:

Microscopic examination of a few drops of ruminal fluid on a glass slide (with a coverslip) at low power will reveal the absence of ruminal protozoa, particularly medium- and large-sized protozoa, which is a reliable indicator of an abnormal state of the rumen that is usually acidosis.

3- Serum Biochemistry

The degree of hemoconcentration, as indicated by hematocrit, increases with the amount of fluid withdrawn from the extracellular fluid space into the rumen and probably provides the best single indicator of clinical severity in ruminal acidosis.

4- Urine pH

The urine pH falls to 4.5 to 5.0 in advanced cases of acute ruminal acidosis and becomes progressively more concentrated as the animal becomes more dehydrated; terminally there is anuria.

NECROPSY FINDINGS

In acute cases in which the animal dies in 24 to 48 hours, the contents of the rumen and reticulum are thin and porridge-like and have a typical odor suggestive of fermentation.
The cornified epithelium may be mushy and easily wiped off, leaving a dark, hemorrhagic surface beneath. This change may be patchy, caused probably by the production of excess lactic acid in pockets in which the grain collects, but is generally restricted to the ventral half of the sacs. Abomasitis and enteritis are also evident in many cases. The abomasum may contain large quantities of grain. There is a pronounced thickening and darkening of the blood, and the visceral veins stand out prominently. In cases that have persisted for 3 to 4 days the wall of the reticulum and rumen may be gangrenous. This change is again patchy but may be widespread.
In affected areas the wall may be three or four times the normal thickness,
show a soft black mucosal surface raised above surrounding normal areas, and have a dark red appearance visible through the serous surface. The thickened area is very friable and on cutting has a gelatinous appearance.
If the examination takes place less than an hour after death, estimation of ruminal pH may be of value in confirming the diagnosis, but after 1 hour the pH of the rumen contents begins to increase and its measurement may not be reliable. A secondary enteritis is common in animals that have been ill for several days.
Hepatic abscesses spread on the liver surface in sub acute cases may noted with PM changes.

Differential Diagnosis:

When outbreaks of the disease with an appropriate history are encountered, the diagnosis is usually readily obvious and confirmed by the **clinical findings** and

examination of the ruminal fluid for pH and **rumen protozoa**.
When the disease occurs in a single animal without a history of engorgement, the diagnosis may not be readily obvious. The **anorexia**, **depression**, **ruminal stasis** with **gurgling fluid sounds from the rumen**, **diarrhea**, and a **staggery gait** with a **normal temperature** are characteristics of rumen overload.
Acute and subacute carbohydrate engorgement must be differentiated from the following:
• **Simple indigestion**: (there are history of change in diet)
The consumption of large quantities of palatable feed, such as ensiled green feed offered to cattle for the first time, may cause simple indigestion, which may resemble grain overload. The rumen is full, the movements are reduced in frequency and amplitude, and there may be mild abdominal pain from the distension, but the ruminal pH and protozoan numbers and activity are normal.
• **Parturient paresis**:
Severe cases that are recumbent may resemble parturient paresis, but in the latter the feces are usually firm and dry, marked dehydration does not occur, the absolute intensity of the heart sounds is reduced, and the response to calcium injection is favorable.
• **Toxemias**:
Common toxemias of cattle that may resemble ruminal overload include peracute coliform mastitis and acute diffuse peritonitis, but clinical examination will usually reveal the cause of the toxemia.
• **Subacute ruminal acidosis:**
must be differentiated from diseases of dairy cows in early lactation in which there is reduced appetite and milk production. These include simple indigestion, left-side displacement of the abomasum, and ketosis, as well as other causes of suboptimal milk production in dairy cows in early lactation. Feeding management problems such as poor-quality forage or poor feeding bad management are common causes of suboptimal performance in lactating dairy cows that are not affected with SARA.

TREATMENT

The following are principles of treatment:
1-Correct the ruminal and systemic acidosis and prevent further production of lactic acid.
2-Restore fluid and electrolyte losses and maintain circulating blood volumes.
3-Restore forestomach and intestinal motility to normal.

In per acute cases:

- Rumenotomy

- Sodium bicarbonate 5 L (5%) IV in 30 min (for 450 kg BW) followed by isotonic balanced fluids and electrolytes at 150 mL/kg BW for 6–12 h

In acute cases:

- Consider immediate slaughter
- Rumen lavage or rumenotomy
- Sodium bicarbonate and fluids IV as in peracute case
- Feed hay

In sub acute cases:

- Magnesium hydroxide 500 g/450 kg BW into rumen
- Fluids if indicated
- Feed hay
- Should begin eating in 24–36 h

In mild cases:

- Feed hay and observe for 48 h
- Watch for anorexia.

When cattle are found engorging themselves, the following procedures are recommended:

• Prevent further access to feed.
• Monitor water intake and prevent the rapid intake of excessive quantities of water.
• Offer a supply of good-quality palatable hay equal to one-half of the daily allowance per head.
• Exercise all animals every hour for 12 to 24 hours to encourage movement of the ingesta through the digestive tract.

Rumenotomy:

In severe cases, in which there is recumbency, severe depression, hypothermia, prominent ruminal distension with fluid, a heart rate of 110 to 130 beats/min and a rumen pH of 5 or below, a rumenotomy access to appropriate facilities, particularly when many animals are involved.

Intravenous Sodium Bicarbonate and Fluid Therapy:

The systemic acidosis and the dehydration are treated with intravenous solutions of 5% sodium bicarbonate at the rate of 5 L for a 450-kg animal given initially over a period of about 30 minutes. This will usually correct
the systemic acidosis. This is followed by isotonic sodium bicarbonate (1.3%) at 150 mL/ kg BW intravenously over the next 6 to 12 hours.

Rumen Lavage:

In less severe cases, in which affected cattle are still standing but are depressed, their heart rate is 90 to 100 beats/min, there is moderate ruminal distension, and the rumen pH is between 5 and 6, an alternative to a rumenotomy is rumen lavage if the necessary facilities are available. A large 25- to 28-mm inside-diameter rubber tube is passed into the rumen and warm water is pumped in until there is an obvious distension of the left paralumbar fossa; the rumen is then allowed to empty by gravity flow after

creating a siphon. The rumen can be almost completely emptied by 10 to 15 irrigations, but there is the risk of aspiration pneumonia and cattle may become recumbent during the procedure.

Intraruminal Alkalinizing Agents:

In moderately affected cases, the use of 500 g of magnesium hydroxide per 450 kg BW or magnesium oxide in 10 L of warm water pumped into the rumen, followed by kneading of the rumen to promote mixing, will usually suffice.

Ruminal Transfaunation:

transfer at least 5 L of fresh rumen fluid from a healthy animal19 to adult cattle with acute ruminal acidosis, but only after rumen lavage or rumenotomy has been performed, excessive amounts of grain have been removed, and ruminal pH is within the normal range of 6.0 to 7.0

Ancillary Therapy:

Ancillary treatment has included antihistamines for laminitis, NSAIDs for their anti-inflammatory and analgesic effects, thiamin or brewer's yeast to promote the metabolism of lactic acid, and parasympathomimetics to stimulate gut motility. Their efficacy has been difficult to evaluate, and it is unlikely that any of them would be of much value. Calcium borogluconate is used widely because there is a mild hypocalcemia and a beneficial but temporary response does occur, but it is of doubtful value.

Orally administered antimicrobials, including penicillin and the tetracyclines, have been used to control growth of the bacteria that produce lactic acid, but they appear to be of limited value.