**Acute Carbohydrate Engorgement Of Ruminants (Ruminal Lactic Acidosis, Rumen Overload)** Etiology

1. The sudden ingestion of toxic doses of carbohydrate-rich feed, such as grain, is the most common cause of the acute form.
2. engorgement with apples, grapes, bread, baker's dough, sugar beet, mangels.
3. sour wet brewers' grain that was incompletely fermented in the brewery.
4. concentrated sucrose solutions used in apiculture.

EPIDEMIOLOGY:

1. All types of ruminant cattle and sheep are susceptible
2. Most commonly in feedlot cattle and dairy cattle fed on high- level grain diets.
3. a change from one ration to
4. another requires a period of microbial adaptation.
5. Animals being fed a low-energy ration are most susceptible to a rapid change to a high energy ration because satisfactory adaptation cannot occur quickly enough
6. Accidental consumption of excess carbohydrates
7. The adaptation of the ruminal microflora and papillae from a requires a gradual change during a period of 3-5 weeks.
8. Morbidity will vary from 10-50%.
9. case fatality rate may be up to 90% in untreated cases, while in treated cases it still may be up to 30-40 %.
10. All grains are more toxic when ground finely or even crushed or just cracked - processes that expose the starch component of the grain to the ruminal microflora.
11. Amounts of feed that are lethal range from 50-60 g of crushed wheat/kg BW in undernourished sheep
12. 75-80 g/kg BW in well-nourished sheep.
13. in cattle doses ranging from 25-62 g/kg BW of ground cereal grain or corn produced severe acidosis.

PATHOGENESIS:

1. The ingestion of excessive quantities of highly fermentable feeds by a ruminant
2. followed within 2-6 hours by a marked change in the microbial population in the rumen.
3. increase in the number of Streptococcus bovis
4. utilize the carbohydrate to produce large quantities of lactic acid.
5. In the presence of a sufficient amount of carbohydrate the organism will continue to produce lactic acid
6. decreases the rumen pH to 5 or less
7. destruction of the cellulolytic bacteria and protozoa.
8. The concentration of volatile fatty acids increases initially, contributing to the fall in ruminal pH.
9. 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.
10. increases ruminal osmolality, and water is drawn in from the systemic circulation, causing hemoconcentration and dehydration.
11. As the ruminal pH declines, the amplitude and frequency of the rumen contractions are decreased
12. The absorbed lactic acid is buffered by the plasma bicarbonate buffering system.
13. With nontoxic amounts of lactic acid, the acid-base balance is maintained by utilization of bicarbonate and elimination of carbon dioxide by increased respirations
14. the blood pressure declines, causing a decrease in perfusion pressure and oxygen supply to peripheral tissues and resulting in a further increase in lactic acid from cellular respiration.

Sequels of ruminal acidosis:

* 1. Chemical and mycotic rumenitis

The low pH of the rumen favors the growth of *Mucor*, *Rhizopus* and *Absidia* spp. which invade the ruminal vessels, causing thrombosis and infarction

* 1. Hepatic abscesses

*Fusobacterium necrophorum* and *Arcanobacter (Corynebacterium) pyogenes* enter directly into ruminal vessels as a result of a combination of rumenitis

* 1. Laminitis

Vasoactive substances (histamine and endotoxins) are released during the decline of rumen pH and the bacteriolysis and tissue degradation. These substances cause vasoconstriction and dilation, which injure the microvasculature of the corium. Ischemia causes physical degradation of junctures between tissues that are structurally critical for locomotion.

* 1. Mild hypocalcemia due to temporary malabsorption.

CLINICAL FINDINGS:

1. Cattle suffered from distended rumen and abdomen
2. abdominal discomfort, evidenced by kicking at the belly
3. anorexic and still fairly bright and alert, and the feces may be softer than normal
4. Rumen movements are reduced but not entirely absent.
5. Affected cattle do not ruminate for a few days but usually begin to eat on the third or fourth day without any specific treatment.
6. In severe form, within 24-48 hours some animals will be recumbent,
7. some staggering and others standing quietly alone.
8. Teeth grinding may occur in about 25 % of affected sheep and goats.
9. Once they are ill they usually do not drink water
10. Cattle may engorge themselves on water if it is readily available immediately after consuming large quantities of dry grain.
11. inspection of the feces on the ground will usually reveal Depression, dehydration, inactivity,
12. The temperature is usually below normal, 36.5-38SC (98-101°F), but animals exposed to the sun may have temperatures up to 41°C (106°F).
13. In sheep and goats, the rectal temperatures may be slightly higher than normal.
14. The heart rate in cattle is usually increased and continues to increase with the severity of the acidosis and circulatory failure.
15. In general, the prognosis is better in those with heart rates below 100/min than those with rates up to 120-140/min.
16. In sheep and goats, the heart rate may be higher than 100/min.
17. The respirations are usually shallow and increased up to 18) 60-90/min.
18. A mucopurulent discharge is common because animals fail to lick their nares.
19. dehydration is severe and progressive.
20. The rumen contents palpated through the left paralumbar fossa may feel firm and doughy
21. pH of the rumen fluid is usually below 5.
22. Severely affected animals have a staggery, drunken gait and their eye sight is impaired.

CLINICAL PATHOLOGY:

1. Ruminal fluid pH
   1. A ruminal pH of 5-6 in roughage fed cattle suggests a moderate degree of abnormality
   2. a pH of less than 5 suggests severe grain overload and the need for energetic treatment.
   3. Feedlot cattle that have been on grain for several days or weeks and are affected with grain overload usually have a pH below 5.
2. Ruminal protozoa
3. Serum biochemistry

1- Blood lactate and inorganic phosphate levels rise 2- blood pH and bicarbonate fall markedly

3- mild hypocalcemia

1. Urine pH
   1. The urine pH falls to about 5 and becomes progressively more concentrated; terminally there is anuria.

NECROPSY FINDINGS:

1. the contents of the rumen and reticulum are thin and porridge-like and have a typical odor suggestive of fermentation.
2. The cornified epithelium may be mushy and easily wiped off, leaving a dark, hemorrhagic surface beneath

DIFFERENTIAL DIAGNOSIS:

1. Simple indigestion
2. Parturient paresis
3. Toxemias TREATMENT

The principles of treatment are:

1- Correct the ruminal and systemic acidosis 2- prevent further production of lactic acid

3- Restore fluid and electrolyte losses 4- maintain circulating blood volumes

5- Restore forestomach and intestinal motility to normal 6- Prevent further access to feed

1. Do not provide any water for 12-24 hours
2. Offer a supply of good-quality palatable hay equal to one- half of the daily allowance per head
3. Exercise all animals every hour for 12-24 hours to encourage movement of the ingesta through the digestive tract
4. Rumenotomy in severe cases
5. Intravenous sodium bicarbonate and fluid therapy
   1. 5% sodium bicarbonate at the rate of 5 L for a 450 kg animal given initially over a period of about 30 minutes
   2. followed by isotonic sodium bicarbonate (1.3%) at 150 mL/kg BW intravenously over the next 6-12 hours.
6. Rumen lavage in less severe cases
7. Intraruminal alkalinizing agents in moderately affected cases, the use of 500 g of magnesium hydroxide per 450 kg/BW
8. NSAIDs for shock therapy
9. thiamin or brewer's yeast to promote the metabolism of lactic acid
10. parasympathomimetics to stimulate gut motility.
11. Calcium borogluconate

RUMINAL TYMPANY (BLOAT)

Ruminal tympany is abnormal distension of the rumen and reticulum caused by excessive retention of the gases of fermentation, either in the form of a persistent foam mixed with the rumen contents or as free gas separated from the ingesta. Normally, gas bubbles produced in the rumen coalesce, separate from the rumen contents t o form pockets o f free gas above the level of the contents and finally are eliminated by eructation.

ETIOLOGY

1. Primary ruminal tympany (frothy bloat)
   1. Leguminous, is due to the foaming qualities of the soluble leaf proteins
   2. cattle that graze pastures consisting of bloating forages (alfalfa, clover)
   3. lush pasture.
   4. Spring and autumn are the most dangerous seasons, when the pastures are lush and young
   5. the leaves of the plants contain a high concentration of soluble proteins
2. Secondary ruminal tympany (free-gas bloat):

Physical obstruction to eructation occurs in esophageal obstruction caused by a foreign body, by stenosis of the esophagus, by pressure from enlargements outside the esophagus

PATHOGENESIS:

1. Normally, gas bubbles produced in the rumen fluid coalesce, separate from the rumen contents to form pockets of free gas above the level of the contents, and are finally eliminated by eructation
2. In frothy bloat, the gas bubbles remain dispersed throughout the rumen contents
3. Most of the gas is mixed with the solid and fluid ruminal contents to form a dense, stable froth
4. producing an abnormal increase in the volume of the ruminoreticular contents and, consequently, inhibiting eructation.
5. In free-gas bloat the gas bubbles coalesce and separate from the rumen fluid but the animals cannot eructate

the pockets of free gas because of abnormalities of the reticulorumen or esophagus.

CLINICAL FINDINGS:

1. Bloat is a common cause of sudden death (or found dead) in cattle.
2. Obvious distension of the rumen occurs quickly, sometimes as soon as 15 minutes after going on to bloat-producing pasture
3. the animal stops grazing.
4. The distension is usually more obvious in the upper left paralumbar fossa but the entire abdomen is enlarged.
5. There is discomfort and the animal may stand and lie down frequently, kick at its abdomen and even roll.
6. Frequent defecation and urination are common.
7. Dyspnea is marked
8. mouth breathing, protrusion of the tongue, salivation and extension of the head.
9. Ruminal contractions are usually increased in strength and frequency in the early stages
10. Trocarization or the passage of a stomach tube, only small amounts of gas are released before froth blocks the cannula or tube.
11. In Secondary bloat Passage of a stomach tube or Trocarization results in the release of large quantities of gas and subsidence of the ruminal distension

DIFFERENTIAL DIAGNOSIS:

1. vagus indigestion
2. Carcinoma and papillomata of the esophageal groove and reticulum and actinobacillosis of the reticulum
3. Animals found dead (Blackleg, lightning strike, anthrax and snakebite)

TREATM ENT:

* 1. Emergency Rumenotomy
  2. Trocar and cannula
  3. Promote salivation (tie a stick in the mouth)
  4. Stomach tube
  5. Antifoaming agents
     1. Poloxalene synthetic surfactants at a dose of 25-50 g
     2. Any nontoxic oil, doses of up to 500 mL