Acidosis

Lactic acidosis, Carbohydrate engorgement, Rumenitis,Founder

- Grain overload is an acute disease of ruminants that is characterized by rumen hypomotility to atony, dehydration, acidemia, diarrhea, depression, incoordination, collapse, and in severe cases, death.

CAUSES

- Accidentally gain access to large quantities of readily digestible carbohydrates, particularly grain.
- Wheat, barley, and corn are the most readily digestible grains.
- Ingestion of toxic amounts of highly fermentable carbohydrates is followed within 2–6 hr by a change in the microbial population in the rumen.
- The number of gram-positive bacteria (such as *Streptococcus bovis*) increases markedly, which results in the production of large quantities of lactic acid.
- The rumen pH falls to ≤5, which destroys protozoa, cellulolytic organisms, and lactate-utilizing organisms, and impairs rumen motility.

- The low pH allows the lactobacilli to utilize the carbohydrate and to produce excessive quantities of lactic acid.
- The superimposition of lactic acid and its salts, L-lactate and D-lactate, on the existing solutes in the rumen liquid causes osmotic pressure to rise substantially,
- which results in the movement of excessive quantities of fluid into the rumen, causing fluid ruminal contents and dehydration.
- The low ruminal pH causes a chemical rumenitis, and the absorption of lactate, particularly D-lactate, results in lactic acidosis and acidemia.
- In addition to metabolic (strong ion) acidosis and dehydration, the pathophysiologic consequences are
- Hemoconcentration,
- Cardiovascular collapse,
- Rrenal failure,
- Muscular weakness,
- Shock, and death.

- Animals that survive may develop mycotic rumenitis in several days and hepatic abscesses several weeks or months later.
- They may have evidence of ruminal epithelial damage at slaughter.
- The relationship between grain overload and chronic laminitis in cattle is unclear.

Clinical Findings

- simple indigestion to a rapidly fatal acidemia and strong ion (metabolic) acidosis.
- The interval between overeating and onset of signs is shorter with ground feed than with whole grain, and severity increases with the amount eaten.
- Abdominal pain (manifest by belly kicking or treading of the hindlimbs).
- In the mild form, the rumen movements are reduced but not entirely absent, the cattle are anorectic but bright and alert, and diarrhea is common.
- The animals usually begin eating again 3–4 days later without any specific treatment.
- Body temperature is usually below normal, 98°–101°F (36.5°–38.5°C).
- Respirations tend to be shallow and rapid, up to 60–90/min.

- The heart rate usually is increased in accordance with severity of the acidemia; the prognosis is poor for cattle with heart rates >120 bpm.
- Diarrhea is common and usually profuse and malodorous.
- The feces are soft to liquid, yellow or tan, and have an obvious sweet-sour odor.
- The feces frequently contain undigested kernels of the feed that has induced the overload.
- In mild cases, dehydration equals 4%–6% body wt, but losses may reach 10%–12% in severe cases.
- Acute laminitis may be present and is most common in those animals not severely affected; chronic laminitis may develop weeks or months later.
- Anuria is a common finding in acute cases, and diuresis after fluid therapy is a good prognostic sign.

DIAGNOSIS

- The diagnosis can be confirmed by the clinical findings, a low ruminal pH (<5.5 in cattle unaccustomed to a high grain diet),
- Examination of the microflora of the rumen for presence of live protozoa.
- Clinical signs—a static rumen with gurgling fluid sounds, diarrhea, ataxia, and a normal temperature—are characteristic.
- Rumen fluid analysis in these animals is required to confirm the diagnosis of grain overload.
- Although parturient paresis may resemble rumen overload, diarrhea and dehydration are not typical, the intensity of heart sounds is reduced, and the response to calcium injection is usually dramatic.
- Peracute coliform mastitis and acute diffuse peritonitis may also resemble overload, but usually a careful examination will reveal the cause of the toxemia.

TREATMENT

- Restricting water intake for the first 18–24 hr is helpful, although this has not been proved.
- Removal of rumen contents and replacement with ingesta taken from healthy animals is necessary.
- In animals still standing, rumenotomy is preferred to rumen lavage, because animals may aspirate during the lavage procedure and only rumenotomy ensures that all ingested grain has been removed.
- Rumen lavage may be accomplished with a large stomach tube if sufficient water is available.
- A large-bore tube (2.5 cm inside diameter, 3 m long) should be used, and enough water added to distend the left paralumbar fossa; gravity flow is then allowed to empty out.
- Repeating this 15–20 times achieves the same results (and requires about as much time) as using rumenotomy to empty and wash out the rumen with a siphon.

- Rigorous fluid therapy to correct the metabolic acidosis and dehydration and to restore renal function.
- Initially, over a period of ~30 min, 5% sodium bicarbonate solution should be given IV (5 L/450 kg). During the next 6–12 hr, a balanced electrolyte solution, or a 1.3% solution of sodium bicarbonate in saline, may be given IV, up to as much as 60 L/450 kg body wt.
- Urination should resume during this period.
- Usually, it is unnecessary and even undesirable to also administer antacids PO (or intraruminally), particularly if IV sodium bicarbonate has been administered.
- Procaine penicillin G (22,000 U/kg/day) should be administered IM to all affected animals for at least 5 days to minimize development of bacterial rumenitis and liver abscesses.
- Thiamine should also be administered IM to facilitate metabolism of L-lactate via pyruvate and oxidative phosphorylation; animals with grain overload also have low concentrations of thiamine in rumen fluid because of increased production of thiaminase by ruminal bacteria.