Acidosis in Ruminants

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Noninfectious diseases of ruminants are more common than infectious diseases. The digestive system is more vulnerable to disorder than any other system. Disorders ranging from stomatitis, choke, bloat, indigestion, intoxications, and displacements are observed in ruminants almost daily. It is doubtful that any animal ever reaches maturity without suffering at some time or another from some kind of digestive dysfunction. Changes in feed are among the most common causes of digestive dysfunction in ruminants.

A sudden change of feed components as occurs in feedlot animals, over-feeding during production competition, or preparing animals for competition showing are examples of causes of feeding troubles resulting in acidosis.

Rumen Bacteriology

The rumen is an efficient incubator where multiplying organisms convert feed into nutrients. Protozoa are factors in this mechanical breakdown of food for they feed on bacteria and are probably utilized by the host as food. Bacteria constitute an estimated 10% of rumen volume content. In each cubic centimeter of rumen contents there are 100 billion bacteria. Cellulose is converted into soluble compounds through the action of microorganisms in the digestive tract.

The adaptation of ruminants to high grain rations involves changes in the rumen microbial population. The type of ration, its physical form, and rate of feeding can influence numbers and kinds of rumen microorganisms. Lactic acid is a product of carbohydrate fermenting bacteria. Also present in the rumen however are bacteria which utilize lactic acid. These bacteria utilize lactic acid for energy and produce some of the rumen fermentation products. Lactic acid may be considered an intermediate step in the production of the three major volatile fatty acids: acetic, propionic and butyric. If the rate of lactic acid production exceeds the rate of utilization, the rumen concentration of lactic acid increases.

Pathogenesis of Acidosis

According to Dr. Tom Huber, associate professor of physiology and pharmacology, College of Veterinary Medicine, University of Georgia, acidosis of ruminants develops in this sequence.

Two of the lactic acid producing organisms, streptococci and lactobacilli, are considered to be present in relatively low numbers in the rumen of cattle on low levels of concentrate. When the ration is rapidly changed from roughage to high concentrate, streptococci and lactobacilli increase in number at a more rapid rate than do the lactic acid utilizing bacteria and lactic acid accumulates in the rumen.

Streptococci outgrow the lactobacilli at first but are soon inhibited by the high acidity which they themselves produce but lactobacilli continue to grow. Rumen pH values of 4 or below are not uncommon. The low pH is below the range at which most rumen bacteria and protozoa can function. In severe cases of lactic acidosis the normal organisms may be destroyed. The lactic acid producing bacteria, especially the lactobacilli, thrives at low pH and lactic acid accumulates in the abscence of adequate numbers of lactic acid utilizers.

Adapting ruminants to high grain rations of graded increases in grain over a period of 14-21 days allows lactic acid utilizers to maintain a similar rate of growth with lactic acid producers. Short-term starvation may also precipitate excessive rumen lactic acid accumulation. Studies have shown that when the starvation period extends 48 hours or so, many types of bacteria and protozoa die. The microbial loss appears to be differential since streptococci remain in fairly large numbers. Thus, animals off feed for two to three days because of shipment or disease unrelated to feeding may need to be treated as unadapted animals during refeeding.

In the rumen most of the lactic acid dissociates into lactate ion and hydrogen ion. If not buffered, the hydrogen ions cause a decrease in the pH of the rumen contents. Sodium bicarbonate, which is present in the saliva, provides protection against such changes. When excessive lactic acid is produced in the rumen, hydrogen ions deplete the bicarbonate and the pH of the rumen contents decrease to extremely low values.

Results of Acidosis

When the rumen pH reaches 4.0 or below, rumen motility is completely inhibited. Many rumen organisms are destroyed, especially the gram-negative. The lactate anion increases the osmotic pressure of rumen contents and water is drawn from the body fluid compartments into the rumen resulting in tissue dehydration. Profuse diarrhea is common and total body water losses of 10% of the body weight have been reported. Sodium, potassium, and chloride appear to be lost from plasma in approximately isotonic proportions. Loss of plasma water is reflected by an increased concentration of red blood cells.

Lactic acidosis decreases blood flow to the kidneys with resulting renal failure. Toxic substances have been incriminated as playing a role in the pathogenesis of lactic acidosis. These include endotoxins, histamine, tyramine, and alcohol. Depending on the severity of the acidosis the animal may exhibit signs ranging from loss of appetite to coma and death.

Treatment of Acidosis

- 1. Mild Cases. Increase percent of crude fiber in the ration in the form of hav or straw.
- 2. Severe Cases. Treatment depends on whether the rumen is overloaded or not and to which stage the acidosis has proceeded. Rumen overload (first phase). a.
 - - (1)Depending upon the value of the animal and the severity, rumenotomy may be the first consideration. a Replacement of contents from healthy, fistulated or slaughtered animal into the evacuated rumen is important.
 - (2) Sometimes the rumen contents can be evacuated by lavage.
 - Ancillary treatment requires repeated oral applications (3) of 5-10 gm of chlortetracycline or other effective antibiotic plus up to 5 liters water in several drenches over 24 hours.
 - b. Rumen overload (second phase). When rumen contents have become liquid.
 - (1)Remove fluid with hose.
 - Rumen lavage. (2)
 - (3) Oral antibiotics.
 - Brewers yeast with plenty of water. (4)
 - (5) Rumen inoculations.
 - (6) Oral administration of alkalizing agents.
 - intoxication, dehydration, and loss To combat c. of electrolytes.
 - (1) Use of antihistamines.
 - (2) Thiamine
 - (3) Ringers lactate.