SIMPLE INDIGESTION, RUMEN ACIDOSIS AND NON-INFECTIOUS DIARRHEA IN ADULT CATTLE

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INTRODUCTION

Simple indigestion is considered the most common cause of diarrhea in adult cattle. Though there is little support for this claim in the literature, it is widely accepted in veterinary texts. Postulated causes of simple indigestion include subacute rumen acidosis (SARA), ingestion of moldy feed, ingestion of damaged (cold or heat) feed, abrupt feed changes, and ingestion of poor digestible roughage. There is unfortunately little direct evidence to explain how these feed changes lead to diarrhea in adult cattle.

Diarrhea is caused by one of four major mechanisms—secretion, increased osmotic load, inflammation, or hypermotility of intestines. Increased secretion of fluid beyond the absorptive ability of the intestine is best exemplified by the diarrhea seen in neonates infected with enterotoxigenic E. coli. Osmotic diarrhea can occur when an osmotically active compound is ingested such as magnesium sulfate or when there is a poorly digested and absorbed nutrient such as the case in lactose intolerance. Salmonellosis causes significant damage to absorptive epithelium and leads to an inflammatory diarrhea. Hypermotility occurs with many causes of diarrhea and is rarely a solo mechanism. For example, inflammatory conditions also commonly cause increased intestinal motility. It is not clear how each of these mechanisms applies to simple indigestion, yet in this paper and presentation we will discuss potential links between the well-researched fields of rumen physiology and microbiology and diarrheal mechanisms.

SECRETION

Of the listed mechanisms of diarrhea, secretion is probably the least likely to contribute to diarrhea seen in clinical cases of simple indigestion. As previously mentioned, secretory diarrheas are almost always associated with an infectious agent producing a toxin. Yet in cases of rumen acidosis with high concentrations of rumen degradable protein, significant quantities of histamine can be produced (Garner et al., 2004; Pilachai et al., 2012). Histamine has been associated with cases of watery diarrhea that are believed to be secretory in nature (Baum, 1989; Bredfeldt et al., 1980). It is unclear if the mechanism of this hypersecretion is similar to that seen in enterotoxigenic causes or not.
OSMOTIC DIARRHEA

Contrary to secretion, osmotic causes of diarrhea are likely the easiest to associate with causes of simple indigestion. With SARA, there is a significant production of volatile fatty acids and lactic acid. Further the lactate-metabolizing protozoa are lost at a lower pH (Newbold, 1987). As these compounds normally are absorbed across the rumen epithelium, the presence of them in the small intestine and colon can contribute to an osmotic pull of water through the paracellular spaces and into the intestinal lumen. This movement of water is similar to what occurs within the rumen in more severe cases of rumen acidosis.

The balance of the microbial flora and fauna is finely tuned to the substrates to which they are accustomed, such that byproducts of one fermentative process are rapidly utilized by another organism. This prevents the accumulation of fermentative metabolites other than methane and volatile fatty acids. Abrupt feed changes, SARA, or ingestion of moldy, damaged, or indigestable feeds can easily disrupt this balance, allowing compounds that were previously completely metabolized to now accumulate. This could be due to an increase in production of the compound or a decrease in consumption. These un-metabolized compounds could contribute to an osmotic diarrhea as they pass through the small intestine and colon.

Decreased retention of feed material due to decreased particle size can lead to passage of incompletely digested carbohydrate into the small intestine and colon (Pearce, 1964). This can contribute to an osmotic diarrhea, and when it reaches the colon, secondary fermentation and bacterial overgrowth can occur. This exacerbates the osmotic diarrhea as these bacteria produce a larger concentration of volatile fatty acids than is typically found.

INFLAMMATION

Inflammatory diarrheas are commonly associated with damage to the intestinal epithelium. Once this absorptive epithelium is damaged, a malabsorptive and osmotic diarrhea can occur. Inflammation can also be associated with increased secretion and hypermotility through activation of the enteric nervous system. In simple indigestion, ethanol produced in the rumen (Nagaraja, 2007) and could potentially reach the intestines and damage the epithelium after being converted into acetaldehyde (Jokelainen, 1994). Further, bacterial overgrowth mentioned above can acidify the colon leading to mucosal inflammation and damage (Plazier, 2009). Finally, mycotoxins at high enough concentrations can lead to inflammation and ulceration in the gastrointestinal tract contributing to the diarrhea seen when cattle ingest moldy feeds (Radostits, 1999).
HYPERMOTILITY

Hypermotility can contribute to diarrhea in inflammatory conditions through activation of the enteric nervous system, and has been associated with histamine as mentioned earlier. A primary cause of hypermotility may be production of endotoxin from the killing of bacteria in cases of SARA (Bruins, 2003).

CONCLUSIONS

There does not appear to be one clear mechanism by which simple indigestion and SARA contribute to diarrhea in adult cattle. The most likely mechanisms include the osmotic draw of unabsorbed products of abnormal fermentation and hypermotility associated with endotoxin release. Inflammation and secretion can also contribute in cases of production of toxic metabolites including histamine and ethanol or ingestion of mycotoxins. Fortunately, treatment for all of these conditions is focused on restoring the rumen environment by removing the offending feedstuff if possible, allowing the cow to become accustomed to the new diet, and transfaunation in severe cases. Rarely do these cases need specific medical therapy.

REFERENCES

Radostits OM, Gay CC, Blood DC, Hinchcliff KW. Veterinary Medicine, 9th ed. 1999; 1686.