

# *GI Puzzler Plagues*

## *High Producing Cows*

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A gastrointestinal problem known as ‘bloody gut’ has been recognized with increasing frequency in dairy cows over the last decade. The disease is also called jejunal hemorrhage syndrome or hemorrhagic bowel syndrome (HBS). These different names are quite descriptive of the way the problem appears in affected cows. Animals may be affected individually, or in a cluster of several affected on the same dairy.

HBS was first identified many years ago, characterized as a rare, sporadic disease. Recently dairies across the nation have experienced outbreaks of this problem for unknown reasons. The cause of HBS is also unknown, and no consistent predisposing factors have been identified. However, increased intensity of milk production, related to increased dietary energy and protein and decreased fiber in dairy rations have been proposed as risk factors. The purpose of this article is to familiarize you with the problem as we know about it at present.

***Disease signs*** - Many affected cows are found dead, without premonitory signs having been observed. Disease signs include severe depression, weakness leading to recumbency, hypothermia (low body temperature) and death following several hours later. Cows with a longer clinical course of disease show sudden decline in milk production, associated with depression, loss of appetite, diarrhea, plus abdominal pain (colic) and abdominal distention in many cases. These signs may be followed within 12 hours by dark tarry feces. More frequently the diarrhea includes blood clots. In almost all cases death occurs within 24 hours of the onset of recognizable disease.

Some cows may be considered for surgery, since colic can be a prominent sign. Additionally some cows will have recognizable pings in the right abdomen, and displaced abomasums can occur in conjunction with HBS. When the abdomen is explored at surgery or necropsy, the small bowel is distended and reddened. Some sections will be filled with fresh, usually clotted, blood. Bowel is usually affected diffusely in a manner that makes surgical removal impossible. In some cases the blood will extend into the lower intestine. Occasionally, a discrete section of bowel is affected and surgical removal is possible.

***Similar diseases*** - Diseases that cause similar signs should be excluded from this syndrome. Abomasal ulcers can bleed profusely, and in severe cases the blood will progress into the small intestine. This problem will closely mimic HBS. Other problems that can produce severe intestinal bleeding include BVD, salmonella infection, and coronavirus infection. Intestinal obstructions can provide almost identical signs as HBS.

Abomasal ulceration and HBS appear to be separate problems, but may be related. Some cases of abomasal ulceration appear to be caused by overgrowth and toxin release by *Clostridium perfringens*. In calves, *C. perfringens* type A has been identified as a cause of severe abomasitis, leading to perforation and often death. Similarly, the pathogen has been identified in adult cattle with similar problems.

***Possible causes*** - It is possible that HBS is another manifestation of clostridial enteritis. Although a specific causal factor has not been clearly identified, several reports have indicated a close association of the problem with *Clostridium perfringens* type A. This association has been based on the following observations: (1) affected cows have positive fecal cultures for this bacteria, (2) *C. perfringens* type A is isolated from blood clots in the intestine, and (3) there is microscopic evidence of intestinal necrosis associated with heavy growth of large, Gram-positive, rod-shaped bacteria presumed to be *Clostridium*.

*C. perfringens* exists almost everywhere in the environment and in the gastrointestinal tract of most

mammals. There are five defined types of *C. perfringens* (A, B, C, D and E), which are identified based on the lethal toxins that they produce: alpha, beta, iota, epsilon and/or enterotoxin. Type A usually produces alpha toxin, although isolates differ significantly in the amount of alpha toxin produced. Additionally, the recently discovered beta2 toxin may be produced by type A. *Clostridium perfringens* type A has been isolated in moderate or heavy growth from feces and/or intestinal blood clots in 93% of HBS cases seen at the VTH-CSU.

Veterinary laboratory diagnosticians in the United States have traditionally been reluctant to consider *Clostridium perfringens* type A as an important agent of intestinal disease because this organism has been proven to be a part of the normal flora of the intestine of livestock. Furthermore, this organism proliferates rapidly in the intestine after death, often making isolation from necropsy specimens of questionable diagnostic significance. Efforts to experimentally reproduce this disease have produced varied results. It has been proposed that the presence of the bacteria alone is not sufficient to produce disease, and other animal and environmental factors are needed for disease development.

***Factors in disease development*** - It is unclear at present whether overgrowth of *C. perfringens* type A occurs as part of the primary disease process or if this growth takes place secondary to some other disease process. There are several potential mechanisms that could promote bacterial overgrowth: 1.) Intestinal motility change or stasis of the bowel, caused by some other disease process or a feeding problem. 2.) Change in outflow of material from the rumen, especially if it includes high concentrations of soluble protein or sugar. 3.) Overabundant bacterial growth in the feed due to contamination. These first two conditions could be influenced by feed energy density and by fiber content and structure in the feed. These conditions could initiate explosive, secondary growth of *C. perfringens* that is already present in the intestine. There is evidence to support each of these mechanisms based on findings from herds with disease outbreaks. For some herds, changing the feeding program has been followed by sudden reduction in disease occurrence.

***Approach to disease outbreaks*** - Based on these findings, herds that develop HBS cases should consider several steps to investigate the problem. First, document by careful post mortem exam and sampling that the disease is really HBS, and not some other condition. Second, evaluate carefully the feeding program with special attention to energy density and fiber composition of the ration. Third, sample feed components for bacteriological culturing to determine if there are specific feedstuffs with overgrowth of Clostridium.

As mentioned above, there is insufficient knowledge about this problem to allow conclusive statements about cause. Further, we do not yet have effective treatments or preventive measures, although some general comments can be made. First, it is very important to not contaminate feed with dirt or manure, to help avoid this problem and others. Second, treatment success will be very unlikely unless it is implemented very early in the disease process. Such treatment should include high dose antibiotics, anti-inflammatory drugs, and Clostridium antitoxin. Currently, there is no vaccine approved for use in cattle in the United States that contains *C. perfringens* type A. Bacterin-toxoid vaccines for types C and D are currently available for use but appear to induce little cross-protection against HBS.

***Research at CSU*** – At CSU-ILM we have been monitoring cases, working with producers and veterinarians to accumulate herd information, and have been developing improved methods to identify Clostridium and its toxins.

Several other groups are also working to more clearly define the characteristics of affected cows and herds. These efforts will help determine whether HBS is one disease with a specific etiology, or rather a collection of several entities that are closely related. There are several groups working to improve vaccine efficacy, but this will depend both on vaccine characteristics as well as whether the disease is a primary clostridial problem. As further information is developed we will keep you informed.