



## **Jejunal Hemorrhage Syndrome**

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### **Introduction**

In the last several years, there have been an increasing number of reports of jejunal hemorrhage syndrome (JHS) in dairy cattle. The condition, also known as hemorrhagic bowel syndrome (HBS), bloody gut syndrome, dead gut and enterotoxemia, affects the small intestine, specifically the jejunum, of adult cows, first calf heifers and bulls. The first reports of JHS in the literature come from the early nineties in northeastern US but since that time there have been confirmed cases reported throughout the US and the world. The syndrome appears without warning signs and is frequently fatal. For most farms, the syndrome is sporadic but some experience recurring problems over a period of months or even years. Periods of concentrated problems are often interspersed by intervals free of apparent disease. Although the syndrome targets the jejunum of affected cattle, the most severely affected individuals have more extensive involvement of the small intestine.

### **Animals at Risk**

JHS is more common in mature cows than in first calf heifers and bulls, but many dairies report problems in cattle of all ages and stages of lactation. Initial reports of JHS showed a predilection for older cattle 100-110 days in milk but the disease can be seen at any stage of lactation, in dry cows and even in mature breeding bulls at stud. Brown Swiss appear to be at greater risk for the syndrome but it can be seen in any dairy breed. Most cattle appear to be in good health immediately prior to the development of JHS, although there may be, as yet poorly elucidated, herd risk factors such as sub-acute rumen acidosis on problem farms. There does not appear to be a consistently increased level of other production related diseases such as subclinical milk fever (hypocalcemia), mastitis or ketosis on dairies that experience JHS but there is undoubtedly much about this condition that we do not yet understand. An association between JHS and other diarrheal diseases such as Salmonellosis, Bovine Virus Diarrhea (BVD), Winter Dysentery, Johne's disease or parasites is not apparent on affected dairies.

## Clinical Signs

The typical cow with JHS becomes depressed, is suddenly off feed, and frequently has cold ears and extremities. Mild abdominal distension may develop, manure production is scant or absent and pain may be demonstrated by teeth grinding, kicking at the abdomen, or getting up and down frequently. The condition usually advances quite rapidly. Affected cattle usually have no fever but heart and respiratory rates are high due to pain and dehydration. Upon rectal examination, the veterinarian usually finds scant manure that contains fresh blood, blood clots and digested blood (melena) along with small intestine that is distended. The findings, though suggestive of JHS are not specific. Other causes of bloody diarrhea like Salmonella, BVD and Winter Dysentery are considered but those cows usually produce more manure and are not typically painful. JHS is also difficult to distinguish from other causes of intestinal blockage. Typically, cows with JHS die within the first 6-36 hours of showing clinical signs. Some progress more slowly over a couple of days and others appear to improve over the first 24 hours only to dwindle and do poorly over the following days. We have seen affected cattle that survive for up to 10 days from the initial episode of pain and abnormal manure. These individuals may survive longer because the intestine is not completely blocked but death results due to intestines that gradually lose their integrity and leak contents or bacteria that results in peritonitis.

## Cause

Because JHS cannot be experimentally reproduced in cattle, the definitive cause is as yet unknown. It appears that multiple factors are involved, with considerable attention being given to the role of the bacteria, *Clostridium perfringens* type A, the mold, *Aspergillus fumigatus* and nutritional factors. While all theories about cause have strong supporters, it is difficult to discern what truly initiates the syndrome from what happens as a result of the disease.

*Clostridium perfringens* bacteria are normal inhabitants of the intestinal tract of healthy cattle and are commonly found in the environment. *C. perfringens* type A, the type incriminated as playing a role in JHS, can also be found in haylage under some storage conditions. While the organism is ubiquitous and generally considered to be innocuous, it can become problematic with dietary stress, injury, changes in management, parasitism or other unusual circumstances that set up a favorable growth environment that allows the bacteria to proliferate and produce potent toxins. *Clostridium perfringens* type A has been associated with conditions similar to JHS of cattle in poultry (necrotic enteritis of fowl) and in people (pigbel), making further investigation of its role

either as a cause or a consequence of JHS necessary. As a group, these bacteria can proliferate after death and invade other tissues of the cow's body other than the intestine, creating problems in interpretation of a positive culture from post mortem specimens where there has been a delay between the time of death and sample collection and/or submission. In our Veterinary Hospital, we have obtained pure cultures of specific toxin producing *Clostridium perfringens* type A bacteria from fresh, appropriately handled post mortem specimens and from intestinal contents from JHS-affected cattle undergoing surgery to remove the blood clot or the abnormal portion of intestine. While the presence of toxins ( $\alpha$  and  $\beta$ -2) can help distinguish the potentially normal bacteria from disease producing strains, it is impossible in an affected cow to determine whether the toxins are a primary or secondary factor. Management practices may have an impact on the microflora of the small intestine, creating an environment that could enhance the growth of toxin producing *Clostridium perfringens* to disease-producing levels. Comparisons have been drawn to a condition known as pigbel, a disease seen in people in Papua New Guinea, where high protein diets (due mainly to sweet potatoes) containing abundant trypsin inhibitors allow overgrowth of *Clostridium perfringens* ingested in spoiled meat. In newborn calves, colostrally-derived trypsin inhibitors can have the same end result. Periods of low protein intake followed by sudden increases in protein feeding can trigger *Clostridium perfringens* enterotoxemia in other species, the low protein feeding resulting in reduced pancreatic protease production followed by sudden high protein feeds contaminated with *Clostridium perfringens* that overwhelm the down regulated defense mechanisms.

More recently, the mold, *Aspergillus fumigatus*, has been associated with JHS. This organism, commonly found in soil, can be detected in feeds and forages and has been found in the blood and tissues of JHS-affected cattle. Although a causal relationship has not been established, some researchers believe that a primary insult like rumen acidosis, abnormal GI motility, metabolic disease, stress or an immune suppression, creates an environment for *Aspergillus* to gain entrance into the blood in high numbers. Alternatively, a heavy load of *Aspergillus fumigatus* gains entrance into the blood through an injured intestinal or abomasal lining. The mold inhibitor, Omni-Gen AF™, has been added to the feed on some farms to reduce the incidence of JHS.

### **Treatment**

The treatment of individual cattle with JHS is challenging. Our experience, like those of many veterinarians, is that the best chance of survival and recovery to expected production levels occurs with early diagnosis followed by aggressive medical and surgical therapy. Even

with intensive and aggressive treatment, the mortality rate for this condition still often exceeds 75%. Occasionally, very early recognition and aggressive medical treatment alleviates the need for surgery. Medical treatment usually includes fluids, pain-killers or anti-inflammatory drugs, antibiotics and *Clostridium perfringens* C and D antitoxin.

Unfortunately, there is no specific antitoxin for *Clostridium perfringens* type A, but there may be limited cross protection from the antitoxin of *Clostridium perfringens* type C and D.

Surgery for this condition is no small undertaking, especially in the field because, at the very least, the intestinal manipulation is extensive. Oftentimes, the intestine is opened to remove the blood clot or a segment must be removed with the necessity to reconnect adjacent unaffected segments. Generally, cows that have clots that can be massaged through the intestine do better than those that require either opening or removal of the intestine. Surgical intervention early in the course of the disease is superior. For all affected cows, treatment success is heralded by a return of normal intestinal movement and production of manure.

## **Prevention and Control**

Because the exact cause and/or contributing factors that lead to JHS are currently uncertain, precise preventive strategies are yet to be determined. The interventions that have seemed to decrease the prevalence of the disease on problem dairies are vaccination, feed additives and nutritional management. Currently there is no approved vaccine available for the use in cattle to protect against *Clostridium perfringens* type A enterotoxemia. There has been considerable interest amongst some herd owners and veterinarians in development of autogenous vaccines (bacterin-toxoids) to *Clostridium perfringens* type A isolated from affected herds. To be effective, products made under fermentation processes maximized for bacterial growth and toxin production using a genotypically appropriate *Clostridium perfringens* type A is necessary. At this point, testimonials are the best evidence we have for the efficacy of autogenous vaccines. Other herds have used *Clostridium perfringens* type C and D toxoid or 7- and 8-way *Clostridium* bacterin-toxoids. Some herds report a reduction in prevalence by vaccinating 2 to 3 times per year with these products.

Much of the preventative effort has been directed towards nutritional management on problem dairies. Control of subacute rumen acidosis, metabolic disease, diseases like Salmonellosis that affect gastrointestinal motility, and consistency of feed components, mixing, and delivery are logical targets when trouble-shooting a problem herd. Clostridial bacteria as a group tends to multiply

rapidly in response to specific opportunities such as carbohydrate overload, an overly acidic environment (either in the abomasum or rumen), high concentration protein or sugar overspill from the rumen into the small intestine or motility disturbances. Insuring a healthy rumen microflora, limiting oral medications and maintaining appropriate fiber length with high quality storage and fermentation of total mixed rations are also important. Inappropriately stored forages can be a source of pathogenic Clostridia and may also pose an added risk from contaminating molds like *Aspergillus fumigatus*. Feed additives have been used in problem herds. Omni-Gen AF™ claims to tie up *Aspergillus fumigatus* in moldy feed, while mannan oligosaccharides, fragments of yeast cell walls, are included in hopes of reducing toxin load, binding harmful bacteria like *Salmonella* and perhaps stimulating some immunity.

### **What Does The Future Hold For JHS?**

There are a number of research groups that are accumulating data on JHS problem herds with a view to better characterizing the condition, especially from the standpoint of epidemiology and risk factors. Considerable interest in dietary risk factors such as subacute rumen acidosis, fiber particle length, and other potentially inciting ration changes has been voiced but it is hard to find a modern dairy where frequent ration formulation changes do not occur. Several commercial laboratories are aggressively pursuing autogenous bacterins and bacterin-toxoids for commercial use against *C. perfringens* type A infections. There is continued interest in the presence of *Aspergillus fumigatus* in the blood and tissues of cattle suffering from JHS. But, until JHS can be reproduced experimentally, we can predict that significant numbers of cattle will receive autogenous bacterins/bacterin toxoids, be given feed additives and, better yet, be tightly managed from a feed delivery standpoint to remove those factors that are associated with susceptibility to JHS.

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