

Northwest Veterinary Associates Newsletter

“That’s not Milk Fever”

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by Dr. Kokaram

During this month’s newsletter I thought I’d touch on a condition in the dairy cow that may be significantly under diagnosed here in the northeast; Hemorrhagic Bowel Syndrome (HBS). This disease, also known as “bloody gut” dead gut and enterotoxemia, affects the small intestine of adult dairy cows, first calf heifers and bulls. HBS was first reported in the northeast in the early nineties, and according to a 2005 study published by the American Veterinary Medical Association, HBS has since spread and has been implicated in roughly 5% of deaths of adult dairy animals nationally. This syndrome is frequently rapidly fatal and very sudden in its expression. For the majority of farms, incidence is often very sporadic with some experiencing periods of concentrated occurrences interrupted by long intervals of apparently no disease.

While HBS is most common in mature cows, some dairies have experienced disease across all age groups and stages of lactation. HBS was initially believed to occur primarily in cows at least 100 – 110 DIM; however, more recent research has shown that the disease may occur in animals much earlier in their lactations. Interestingly enough though, the individuals that have been diagnosed as dying from this syndrome have invariably been in apparently excellent health just prior and have had mean milk productions of almost 90 pounds. Thus, your best high producing cow has the greatest likelihood of succumbing to this syndrome. So what exactly do these cows look like?

Typically, these cows are suddenly very depressed, abruptly go off-feed, drop off significantly on milk production and invariably have cold ears and extremities. This condition is rapidly progressive and these cows will also display some mild abdominal distension, with significantly reduced to absent manure production, and are acutely painful. What little manure may be present may contain fresh blood, blood clots, or even dark, digested blood. As you can see, the overall picture is not very specific as initial signs may mimic a milk fever, or an acute RDA, while signs later on in the progression take on a very different picture, looking more suggestive of such diseases as Salmonella, Winter Dysentery, BVD or even intestinal blockage. Typically individuals die within 6-36 hours of presentation, although some cases have progressed much more slowly; even lasting as long as 10 days before finally succumbing to the syndrome. So where does this come from anyways?

Unfortunately, the exact causative agent in this syndrome has not successfully been identified. However, the two major partners that appear to have a significant role in this syndrome are the bacterium, *Clostridium perfringens* type A, and the mold, *Aspergillus*. *C. perfringens*, is a normal inhabitant of the bovine intestinal

tract, and may be found fairly commonly in the soil. *C. perfringens* type A has also been shown to proliferate in wet haylage and silages. This bacterium has a two-fold means of attack on the cow. Firstly, their growth in haylage results in elevated levels of butyric acid, which in turn serves to significantly reduce DMI, enhance ketone production by the bovine liver, and thus enhanced risk of DA's as well as reduced immune function. This bacterium also produces toxins of its own which themselves reduce immune function.

Aspergillus is also found in the soil and has been identified in both the feed bunks in the clumps of "spoilage" as well as in harvested grains. Infection with this mold may arise from feeding errors (feeding moldy feed), suppressed immune systems (as in the case of animals that are stressed), acidosis, or metabolic disturbances. Ingestion of spores in the feed allows for the mold to take hold in the intestines, leading to intestinal bleeding, and eventually enters the blood circulation allowing for systemic signs of disease to be demonstrated. Interestingly, both of these organisms require warmth, moisture and high pH in the silage to proliferate; conditions made perfect at this time of year where many are feeding the last vestiges of last year's silage.

Treatment of HBS is invariably unrewarding as this condition is frequently very rapidly progressive and often too acute to treat early or aggressively enough. As such, an ounce of prevention is indeed worth a pound of cure. Majority of the preventative measures involve reducing the risk of exposure and toxin load, as much as controlling subacute rumen acidosis and metabolic disease. Clostridia in general multiply rapidly following carbohydrate overload, high concentration of protein, and bouts of acidosis (ruminal or abomasal). Bunk management in terms of ensuring that the silage is appropriately and tightly packed, the top layer (3-8 inches) of infective silage is discarded, and the moisture content is carefully monitored are quite important, as is being careful not to feed silage high in butyric acids (associated with high *C. perfringens* load) to the pre- and post-fresh cows. In some cases, feed additives may be required to reduce the degree of exposure in the gut. Some additives act by binding *Aspergillus* spores in the moldy feed and enhance immune function of the cows, while others act to bind the toxins produced by the molds before they can have a detrimental effect.

Overall, HBS is an often under diagnosed syndrome in dairy cattle with a sad prognosis for recovery or response to therapy once clinical signs are evident. As such, prevention rather than prompt treatment is the hallmark of good management when dealing with such a condition as varied in its expression as this.