

Hemorrhagic Bowel Syndrome: Update and Observations

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Introduction

Hemorrhagic bowel syndrome, characterized medically as Jejunal Hemorrhage Syndrome (JHS) but also known as bloody gut syndrome, is an important, acute enterotoxemic disorder of adult dairy cattle. Sporadic outbreaks of the condition are reported with increasing frequency since 1991. A definitive cause of JHS has not been established and the condition cannot be experimentally reproduced but two agents, the bacteria, *Clostridium perfringens* type A and a common mold, *Aspergillus fumigatus* have been incriminated as having some role in this condition. Characterized as an acute, often fatal condition of high producing dairy cattle that are second lactation or greater, in the first 100 days in milk, consuming a high energy total mixed ration (TMR) and using bovine somatotropin, the condition is reported all over the world. Although the incidence of JHS in most herds is less than 10%, the economic impact is significant as the target is typically a highly productive dairy cow at peak performance and a disease outcome that is frequently death.

Clinical Signs

The clinical findings most commonly associated with JHS are listed below.

- Depression
- Decreased rumen motility
- Decreased feed intake
- Decreased milk production
- Succussible fluid with ballottement of the right abdomen
- Reduced to scant fecal production
- Colic
- Right-sided abdominal ping during simultaneous percussion and auscultation
- Dehydration
- Elevated heart rate
- Dark, tarry feces (melena)
- Clotted blood in feces

As individual clinical signs, none of these are specific for JHS but, taken together as a cluster of signs, the diagnosis is more conclusive. With the progression of intestinal injury, hemorrhage, peritonitis and toxemia, more severe clinical signs of cold extremities, hypothermia, muscle fasciculations and recumbency are seen. Conclusive tests such as diagnostic ultrasonography to find dilated small intestine (jejunal portion) with thickened walls and echoic luminal contents suggestive of blood, exploratory surgery and/or post mortem examination are needed to confirm the diagnosis of JHS in an

Presented at the Four States Nutrition Meeting (June 2014)

individual cow. Severe intestinal distension and segmental dark red to purple discoloration of the serosal surface are characteristic findings. Luminal contents contain blood, blood clots, fibrin and/or casts. Gross lesions are associated with the microscopic findings of segmental hemorrhage, edema, ulceration and necrosis. Without the definitive findings discussed here, there is a danger of over diagnosis in some herds.

Diagnosis of a JHS herd problem is more complex and relies on careful assessment of herd records and accurate case identification to elucidate targeted animals or groups of cattle, seasonality, lactational incidence, nutritional factors, health or other relevant risk factors. Individual cow exams and diagnostic tests such as fecal screening, rumen pH determination, serum ionized calcium and potassium concentrations may be helpful. Bulk tank MUN data is essential. In individual animals and herds, abomasal ulcers, other causes of enteritis (*Salmonella*, Bovine Virus Diarrhea and Corona virus), indigestion, and poor intestinal motility should be ruled out.

Treatment of JHS

Without surgery, the JHS mortality is extremely high (77-100%). Surgical options include manual massage of the intestine to break down the blood clot, opening the intestine (enterotomy) to remove the blood clot or resection of the abnormal segment of intestine (enterectomy). A 60% survival rate is reported in JHS cattle that underwent surgery. Early diagnosis, followed by surgery with manual massage of the blood clot carries the best prognosis but survivors are at risk of recurrence, especially within the first 12-months of the initial episode. Alone or in combination with surgery, medical treatment must be instituted early and aggressively to enhance intestinal motility. Fluid therapy provided intravenously, orally or in combination should be high volume (40 L or more) and contain essential electrolytes like calcium, potassium and magnesium. Non-steroidal anti-inflammatory drugs are provided to control pain and to minimize the effects of the inflammatory mediators released when *C. perfringens* type A alpha toxin activates the arachidonic acid cascade. Penicillin and *Clostridium perfringens* type C and D antitoxin are frequently added to the treatment protocol for JHS cases.

Prevention of JHS

Without knowledge of a specific cause of JHS, preventive strategies are based on managing known risk factors that can be controlled. Considering that JHS may be the result of an agent like *Clostridium perfringens* type A taking advantage of an opportunity like abomasal or intestinal motility disturbances to utilize appropriate substrate for rapid proliferation and toxin production, prevention strategies should consider the agent, management factors that enhance abomasal and intestinal motility and ration formulation that minimizes the delivery of favorable substrate.

The bacteria most commonly associated with JHS, *Clostridium perfringens* type A, is ubiquitous in the environment and part of the normal intestinal flora of cattle. Experimental infusion of *Clostridium perfringens* type A cultured from clinical cases into the jejunum of non-lactating cows, however, did not reproduce JHS. Yet, alpha and beta 2-toxin producing *Clostridium perfringens* type A are isolated from feces, intestines and tissues and intestinal lumen toxins are found in JHS cases at a higher rate than from

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unaffected cattle. Vaccines directed against *Clostridium perfringens* type C and D, which do not provide protection against alpha-toxin but which may provide some cross protection through the beta 2-toxoid component, are widely used in dairy herds but vaccinated animals have developed JHS and new cases continue to develop in the face of vaccination. *Clostridium perfringens* type A toxoid has been incorporated into the vaccination protocol of many dairy herds concerned with JHS but controlled studies are not published to evaluate its efficacy. The requirement that *Clostridium perfringens* type A have bioavailable zinc in the intestinal tract for multiplication and for stability, destructive properties and disease induction from its alpha toxin provides additional insight into the pathophysiology of JHS. While a dietary limit on zinc is neither appropriate nor advocated, control of excessive dietary zinc may be indicated in herds with JHS risk.

The potential role for the common mold, *Aspergillus fumigatus* (AF), in JHS is strengthened by knowledge that it can produce a similar enteric hemorrhagic disease in people and that AF DNA has been demonstrated in blood and intestines of JHS cows but not in controls. *A. fumigatus* may act directly or through other toxins, like gliotoxin, to decrease host defenses and cause immune suppression. The mold inhibitor, Omnigen AF (Prince Agri Products, Inc., Quincy, IL) has been included in the diet of many dairy herds with concern for or experience with JHS cases but controlled studies are not published to validate efficacy as a preventive measure.

Maintenance of normal abomasal and intestinal motility should minimize JHS risk. Dietary consistency with regard to components, amount, moisture content, digestibility, access, quality and availability of minerals and buffers are especially important in the high feed intake groups that are most at risk for JHS. High energy total mixed rations (TMR) have been associated with JHS risk but whether this is due to starch overflow to the small intestine, a reduced fiber mat, high volatile fatty acid (VFA) concentrations, pH change, increased osmolality of abomasal contents, or elevated insulin levels is unknown. Change in the quantity, quality or source of dietary protein may also increase the risk of JHS by enhancing *C. perfringens* type A growth and gas production or altering abomasal motility. Limit stress by minimizing group changes and insuring quiet handling of cattle for timed breeding or bST injections, especially in the high feed intake groups that are at most risk for JHS.

Lingering Questions

- What is the relationship between JHS, other abomasal conditions (ulcers, impaction, functional abomasal outflow obstruction, displaced abomasum), intestinal ileus or indigestion? Are these conditions a continuum of an underlying motility disturbance, fermentation disorder, ingredient overflow, luminal content aberration, or metabolic condition?
- To what degree is JHS an infection, a nutritional issue that has its basis in starch or protein amount, quality or source or a metabolic/motility issue?
- Do breed or genetic factors play a role in JHS?
- Are there tools that enhance early detection of JHS?

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