

Jejunal Hemorrhage Syndrome in Adult Dairy Cows

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Introduction

Jejunal hemorrhage syndrome (JHS), also referred to as “hemorrhagic bowel syndrome” or “acute hemorrhagic enteritis of the small intestine”, has been reported with increasing frequency in adult dairy cows over the past three years by veterinary diagnostic laboratories in many dairy states including Minnesota, New York, Pennsylvania, Washington, Wisconsin, Colorado, Illinois, and Iowa. While the occurrence of JHS is often sporadic many cases have presented as part of a syndrome causing the death of significant numbers, or clusters, of adult dairy cows in affected herds.⁵ Hemorrhagic enteritis has been reported in other species and in calves, but published information describing JHS in adult dairy cows is limited.^{2,5,6,7,13,18} The purpose of this paper is to present a short review and a description of JHS cases presented to the University of Minnesota Veterinary Diagnostic Laboratory, and to discuss theories for causes, risk factors, and some suggested methods for control and prevention of the disease.

Clinical Syndrome and Treatment. Affected cows are often found dead (sudden death). Alternatively, they may be found recumbent and semi-conscious, or still standing, but very weak, shocky, and pale. Affected animals may exhibit sudden complete anorexia, a severe drop in milk production, signs of colic or abdominal pain, and abdominal distension. Cows may show decreased fecal output, bloody stool, or diarrhea containing either frank blood or blood clots. Affected cows usually do not have an elevated rectal temperature. Treating affected cows with antibiotics and supportive therapy (e.g. anti-inflammatories, fluids, dextrose) has generally been reported to be unsuccessful. Treatment with calcium has reportedly resulted in temporary improvement in the animal’s general condition, but this is short lived and death ensues shortly after.^{5, 18} There is an extremely high case fatality rate, with reports of 85 to 100% of affected animals dying within 24 to 36 hours of the onset of clinical signs.⁵ On exploratory surgery or necropsy, there are distinct sections of jejunum (a section of the small intestine) which are distended by a large amount of blood. Affected sections of intestine are sometimes three feet or longer. The intestine may contain either bloody or brown fluid or a large solid blood clot that obstructs the lumen, preventing any passage of ingesta or intestinal contents. Some veterinarians have reported limited success by surgically opening the intestine and removing the clot, or by massaging the clot through the small intestine. However, the prognosis is still grave. Spontaneous development of new clots, following removal of the original clot, has also been reported.⁵

Pathology. There are currently no studies published describing the cause of JHS in adult dairy cows. A review of 23 cases presented to the Veterinary Diagnostic Laboratory at the University of Minnesota between 1999 and early 2000, that showed typical gross lesions of either blood clots or bloody fluid in the jejunum, yielded the following diagnostic findings: 100 % of 22 cases tested were negative for *Salmonella* spp., 100% of 9 cases tested were negative for gastrointestinal parasites, and 100% of 18 cases tested were negative on virus isolation for bovine virus diarrhea virus (BVDV). Retesting using blood PCR, a DNA test, have found only three cases positive for BVD. While the following additional tests were performed on only a small subset of these cases, those tested have also consistently been negative for *Yersinia* sp., *Lawsonia intracellularis*, and Corona Virus (the cause of Winter Dysentery). One finding that has been common to the vast majority of these cases (86%, or 19 of 22 cases tested) is the isolation of *Clostridium perfringens* Type A. This has been recognized in cows submitted live that were then euthanized and fresh tissues collected, as well as in tissues submitted from animals that had died on the farm (i.e. tissues were collected by veterinarian several hours after death occurred).

Causes and Risk Factors for JHS? The importance of isolating *Clostridium perfringens* Type A in cases of JHS in adult dairy cows is unclear. Because *Clostridium* spp. are normal inhabitants of soil, live in the intestine of normal healthy animals, and are known to replicate rapidly in the intestinal tract after death, it is not clear if this bacteria is the primary cause of JHS, if it arises secondary to other predisposing changes in the intestine environment, or if it is simply an incidental finding.^{1,15} One theory as to the cause of JHS is a model similar to hemorrhagic enteritis caused by *Clostridium perfringens* Type C in fast-growing suckling calves, lambs, or piglets.^{1,7,8,11,12,15,19} *Clostridium perfringens* Type C will multiply rapidly and produce toxins under conditions of high carbohydrate and protein substrate availability in the small intestine. It is possible that this scenario could arise in the adult dairy cow in association with those same factors that also lead to ruminal acidosis (i.e. feeding excess amounts of rapidly fermentable carbohydrates, insufficient effective fiber and/or inadequate rumen fiber mat, or ration sorting by cows).⁷

A second theory is that poorly fermented ensiled feeds, such as haylage (e.g. in the case of poor silo or bunker management), may accumulate harmful molds, clostridial or other harmful bacteria, and possibly their toxins. These bacteria or their toxins may then be fed directly to the cow. Kirkpatrick et al. sought to investigate the possible role of both ruminal acidosis and clostridial contamination of poorly fermented forages in a case study of a 140-head herd of Brown Swiss that had experienced multiple cases of JHS.⁵ Reported effects of the toxins produced by *Clostridium perfringens* type A in mammals have included illness, diarrhea, accumulation of fluid in intestinal loops, and death from shock.^{10,14} Additional effects of the toxins may possibly include reduced gut motility.¹⁷

Results of Surveys of Minnesota Veterinarians on the Occurrence of, and Potential Risk Factors for, Jejunal Hemorrhage Syndrome

Frequency of Diagnosis of Jejunal Hemorrhage Syndrome by Minnesota Dairy Vets. Given that dead cows are not routinely necropsied on many dairies, this disease may be underreported. Two small surveys were administered to two largely different groups of Minnesota bovine veterinarians in March 2000 and in February 2001. 50% to 59% of respondents indicated that they had diagnosed one or more cases of JHS in the previous 12 months (range 1 to 30 cases diagnosed per veterinarian), and that a median number of two client herds had been affected (range 1 to 8 client herds per veterinarian). Forty to fifty-six percent of respondents indicated that they had diagnosed more than one case on a single farm. When considering all respondents (those having and not having diagnosed JHS), the median number of cases of JHS was only one case diagnosed per veterinarian in the previous 12 month period (mean = 2.4, range = 0 to 30 cases per veterinarian).

Potential cow risk factors. In the first survey, vets were asked to describe the cow and herd-management for the most recent JHS case they had seen. Holstein cows accounted for 94% (n = 17) of JHS cases, with the remaining 6% (n = 1) being Jersey. As an aside, while none were reported in the mail survey, Brown Swiss cases have been submitted to the Veterinary Diagnostic Laboratory and have been reported by others.⁵ A total of 61% of JHS cases were reported to have occurred within the first 100 days of lactation, 22% occurred in mid-lactation (101-200 DIM) and 11 % occurred in late lactation (> 200 DIM). One case was reported during the close-up dry cow period. A significantly greater number of cases (94%) occurred in second lactation and older animals than did in first lactation heifers (6%). These findings are consistent with a case study of a single herd of 140 Brown Swiss cows that had experienced multiple cases of JHS, in which it was reported that the syndrome had affected only older cows.⁵ The authors of that study speculated that this apparent association could be due to higher milk production or higher dry matter intakes in the older animals.⁵ We suggest this could also relate to

different feeding behaviors (e.g. sorting, meal size and frequency). However all of these theories require investigation.

Potential season, region and herd management risk factors. While no statistically significant relationship existed, there was a trend for an increased rate of occurrence in the fall and winter months. JHS was reported in all dairy regions in Minnesota and in herds managed under a wide variety of systems typical of the Midwest: large (>500 cows) and small (<50 cows) herds, free stall and tie stall facilities, and in herds feeding typical Midwest forages (dry hay, corn silage, haylage) and using a variety of fermented forage storage systems (tower silo, bunker silo, or plastic bags). When compared to the average distribution of all Minnesota dairy herds by herd size, chi-square analysis showed a significantly higher risk for JHS in herds with ≥ 100 cows.^{9,20} Also, a significantly higher percentage (83%) of affected herds fed a total mixed ration (TMR), as compared to herds using component feeding.¹⁶ However, given that this was only a small preliminary survey and given the relatively small numbers of cases used in the analyses, readers should be cautious in their interpretation of these preliminary results since there is ample room for introduction of bias and confounding in the study. For example, it may be that larger herds are more likely to have adopted TMR feeding programs and also are more likely to routinely have the veterinarian necropsy all dead animals. Thus, they might simply find more of the disease because they are looking. Also, they have more cows to be at risk of developing the disease.

Strategies for control and prevention of Jejunal Hemorrhage Syndrome.

Ration balance and feeding management. One theory suggests that JHS may occur in situations where an inadequate rumen fiber mat and/or high dietary levels of rapidly available carbohydrate result in an overflow of excessive quantities of carbohydrates into the small intestine. This could provide enough nutrients to allow for rapid multiplication and production of toxin by Clostridial organisms that are natural inhabitants in the gut. If this theory is true, then producers experiencing cases of JHS should work with their nutritionist to investigate ration fermentable carbohydrate and fiber levels, evenness of mixing, and shaker box analysis of particle size for both of fresh TMR and refusals (e.g. evaluate for sorting by the cows). Additional useful information may include evaluation of manure consistency, rumenocentesis to measure rumen pH, milk component data, and health data. Producers should strive to feed a ration balanced with adequate effective fiber to maintain good rumen health and integrity of the rumen mat. Even if we eventually learn that this theory has nothing to do with causing JHS, adequate dietary fiber and good rumen health are still something that we should strive for because they have other health and production benefits.

Forage Quality. A second theory for the cause of JHS is that poorly fermented ensiled feeds may accumulate harmful molds, clostridia or other harmful bacteria, and possibly their toxins that are then fed preformed to the cow. No scientific studies have confirmed this association. However testimonials from some producers state that the occurrence of JHS decreases or disappears if they stop feeding poor quality, poorly fermented forages. Producers experiencing JHS should work with their nutritionist to evaluate forages, including chemical analysis of nutrients, moisture, temperature and pH, as well as visual assessment for obvious spoilage or inadequate fermentation. Producers should discontinue feeding poorly fermented or spoiled forages to close-up and milking cows if another source of higher quality forages can be located. This will be most important for close-up, just fresh and high producing groups. Another strategy, if alternate forage sources are limited, may be to dilute the poorer forage out with higher quality forages. Again, even if we eventually learn that this theory has nothing to do with causing JHS, we all know that feeding high quality forages is the surest and cheapest way to get good production from our cows.

Vaccination. Before beginning this discussion, readers should be reminded that it is not conclusive whether *Clostridium perfringens* Type A is the causative organism, or, if it is, which specific toxin produced by this organism is responsible for JHS. This said, one approach some producers have tried is using an autogenous vaccine. However there are serious concerns in adopting this approach because of a lack of answers to important questions such as: What is the causative organism? What is the correct toxin? What is the yield of toxin needed for protection? What is the correct immunogen dose? What adjuvant should be used? How is enough adjuvant introduced to induce protection without inducing adverse reactions? How pure is the end product? What quality control and testing is done before release? How consistent are different batches? Is any animal testing done? Additionally, autogenous vaccines can be expensive and adverse reactions have been reported including tissue reactions, milk drop, abortion and premature calving. These risks must be weighed against the risk for a disease that is infrequent in nature.

There are no commercial vaccines currently approved for JHS, nor are there likely to be in the near future, until the cause of the disease is understood. Testimonials report no protection from commercial 7 or 8 way clostridial vaccines. This may be because they don't contain the correct bacterial strain or toxin type, or because they contain inadequate yields to be protective. The Type C and D vaccines may give some protection against Type B because the toxins produced by Types C and D organisms are the same as for Type B. Otherwise, there is no cross protection among the other identified *Clostridium perfringens* toxins. Anecdotally, one C and D type vaccine has been reported to give some short-term protection against JHS in some affected herds. The suggested explanation for short-term immunity (3 to 4 months) is that it might also contain some small amount of free alpha toxin from the pathogenic strain. Producers should keep in mind that these reports of protection are only testimonials and are not proof of efficacy. It is very possible that producers experiencing one or two cases of JHS may have responded by changing several factors at the same time in addition to vaccinating, such as discontinuing feeding bad feed and reviewing and tightening up the ration. If the occurrence of JHS seemed to stop for several months, it is difficult to know whether this was due to these other management changes, the sporadic nature of the disease, or whether the vaccine truly helped.

Summary

Jejun hemorrhage syndrome has been diagnosed with increasing frequency in the past few years. In two surveys of Minnesota bovine veterinarians it was reported to have been diagnosed by 50 to 59% of veterinary respondents, of whom 40 to 56% reported multiple cases in the same herds. While there is probably a low annual incidence rate of JHS for the entire population of adult dairy cows at risk, a large number of dairy veterinarians dealing with this disease in multiple client herds, and multiple cases occurring in many herds make this an economically important disease in affected herds. Possible risk factors for JHS that deserve investigation in future studies include parity, stage of lactation, season, herd size, forage quality, ration nutrient and fiber composition, and feeding management. While the exact cause of JHS is not yet known, producers may try to prevent it by working with a nutritionist to ensure that rations include only high quality well fermented forages and include adequate levels of effective fiber so as to ensure a healthy rumen mat and good rumen health. Feeding management should prevent slug feeding and sorting by cows. While there is no science yet to prove efficacy of vaccination against this disease, herds are encouraged to use commercial clostridial vaccines (e.g. 7-way or 8-way), as a matter of routine management, in the adult herd, to protect against other clostridial diseases.

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