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Jejunal hemorrhage syndrome in cattle: A newly emerging disease

Sameeh M. Abutarbush, B.V.Sc.

Jejunal hemorrhage syndrome (JHS) is a newly recognized syndrome that is also called:

- intraluminal intestinal hemorrhage syndrome
- bloody gut
- hemorrhagic bowel syndrome.¹

The syndrome affects both dairy and beef cattle.^{2,3} The etiology is unknown; however, *Clostridium perfringens* type A has been implicated. It has a peracute onset of clinical signs ranging from complete anorexia, abdominal distention and pain, to recumbency and sudden death. The frequency of the disease is increasing and it has a very high case-fatality rate; medical and surgical treatments seem to be unrewarding. The condition has been recognized in many parts of Canada³ and, as a result, it is very important for veterinarians to be aware of its highly fatal course so they can give accurate advice. Reporting the disease, as well as the different attempts at treatment and prevention, will help improve the approach to treating these cases.

Etiology

The exact cause of JHS is unknown; however, *Clostridium perfringens* type A (beta toxin positive) has been suggested as a possible cause of the syndrome.^{4,5} It has been isolated from the lesions of clinical cases and feed samples.⁵ Yet, inoculation of *C perfringens* type A into the abomasum and proximal jejunum of 12 non-lactating dairy cows failed to induce the syndrome.⁴ These findings, however, are not enough to rule-out the possibility that *C perfringens* type A causes JHS because it may be a multifactorial disease with other contributing factors (eg, stress, amount of feed intake, or pH of the gastrointestinal tract).⁴ In a case study of a 140-head herd of Brown Swiss cows with a high case incidence of JHS, Kirkpatrick *et al* found associations between increased milk production, increased intake of soluble carbohydrates, and increased risk of JHS.⁶



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Western College of Veterinary Medicine Department of Large Animal Clinical Sciences

52 Campus Drive
University of Saskatchewan
Saskatoon, Saskatchewan S7N 5B4

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Table 1: The frequency of clinical signs and the history of complaints in 22 cows with JHS. Observations collected from 15 to 22 cows.²

Clinical signs and complaint history	% of cows that showed the clinical sign
Depression	100%
Dehydration	86%
Tachycardia	62%
Abdominal distention	59%
Hypomotile rumen	59%
Pale mucous membranes	59%
Bloody feces	47%
Auscultable right-sided ping sound	41%
Acute reduction in milk production	38%
Dry or scant feces	24%
Recumbency	24%
Atrial fibrillation	24%
Muscle fasciculation	18%
Anorexia	10%
Ketosis	5%

Pathogenesis

There are two hypotheses for JHS pathogenesis.¹

- The first is similar to that for hemorrhagic enteritis caused by *C perfringens* type C in fast-growing suckling calves, lambs, or piglets. *C perfringens* type C multiplies rapidly and produces toxins under conditions of high carbohydrate and protein substrate availability. It is possible that this scenario arises in adult dairy cows in association with factors similar to those that lead to ruminal acidosis, (ie, the feeding of excess amounts of fermentable carbohydrates, insufficient effective fiber and/or inadequate fiber mat, or ration sorting by cows).¹

- A second hypothesis is that improperly fermented ensiled feeds (ie, poor silo or bunker management) may allow the accumulation of harmful molds, clostridia, or other harmful bacteria, as well as their potential toxins, that are then ingested by the cow.¹

History and clinical signs

JHS is a peracute to acute disease with a case fatality reaching 85% to 100%.⁶ The producer might not see prodromal signs and may simply find a dead mature cow or one that is recumbent and in a state of shock.⁶ Affected animals may show sudden, complete anorexia, a severe drop in milk production, abdominal distention, signs of colic and ileus, decreased fecal output, melena, diarrhea containing either frank blood or blood clots,¹ vocalization, bruxism, or sweating.⁶ Affected cows are usually not pyretic,¹ they have ruminal stasis, profound depression, pale mucous membranes, and may have atrial fibrillation or be dehydrated.² If the cow is standing, succussion of the lower-right abdomen can elicit a pronounced fluid slosh due to the back-up of fluid and gas behind blood clots that are obstructing the jejunum.⁶ In some cases, right-sided pings are heard (Table 1).² On rectal examination, distended loops of small intestine, a hard, large rumen, a dilated cecum, or a distended spiral colon can be felt in some cows.^{2,6} However, rectal palpation was normal in 3 of 14 cows in recent study.² The feces often contain melena or frank blood clots.

Laboratory findings

The complete blood count is variable. It may show an elevated packed cell volume and total protein.⁶ Some cows have neutropenia and left shift, others leukocytosis, neutrophilia with left shift, and hyperfibrinogenemia.^{2,6} The serum chemistry may reveal hypocalcemia, hypochloremia, metabolic alkalosis, hyponatremia, hypokalemia, and hyperglycemia.^{2,6} Hypermagnesemia, hypercalcemia, high anion gap, hyperphosphatemia, hypophosphatemia, and azotemia have also been reported.² Serum enzyme activities, including creatine kinase (CK), γ -glutamyl transferase (GGT), sorbitol dehydrogenase (SDH), aspartate aminotransferase (AST), were high in several affected cows.²

Transabdominal ultrasonography

Ultrasonography was performed over the right paralumbar fossa in 12 of 22 cows in a recent study.² It showed dilated loops of small intestine filled with homogeneous echogenic material consistent with clotted blood (in 4 cows) or hypo-echoic material consistent with fluid ingesta (in 3 cows). There were no abnormalities in 5 cows.

Figure 1: Melena of a dairy cow affected with JHS



Courtesy of Dr. Radostits, WCVM

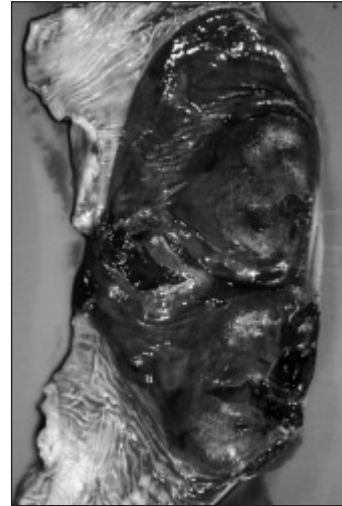
Diagnosis and differential diagnosis

JHS should be suspected in cows with a history and clinical signs of peracute onset of anorexia, a drop in milk production, distended abdomen, abdominal pain, and passage of melena or feces containing frank blood (Figure 1). Palpation of distended loops of small intestine rectally and the presence of hypochloremia, hypokalemia, and metabolic alkalosis are diagnostically helpful findings. In some cases, a definitive diagnosis can be ascertained only with an exploratory laparotomy or a postmortem examination. Due to the ubiquitous nature of *C. perfringens* type A, a positive fecal culture for this organism is not diagnostic.⁶ Differential diagnoses for JHS include salmonellosis, winter dysentery, abomasal ulceration and hemorrhage, right abomasal displacement and volvulus, intestinal volvulus, intussusception, and acute peritonitis.⁶

Gross pathology and histopathology

Postmortem examination reveals segmental lesions localized to the jejunum containing clotted blood.⁶ There is severe necrohemorrhagic enteritis or jejunitis with intraluminal hemorrhage or blood clots (Figures 2 and 3).² Some cases develop intussusception immediately adjacent to the area of segmental hemorrhage and clotting⁶ and some affected cows have fibrinous peritonitis.² In a recent study, the most prominent histologic findings in surgically collected biopsy specimens were severe, segmental, submucosal hemorrhage and edema of the small intestine. This was often accompanied by a

Figure 2: Intramural hemorrhage of the jejunum of a dairy cow with JHS



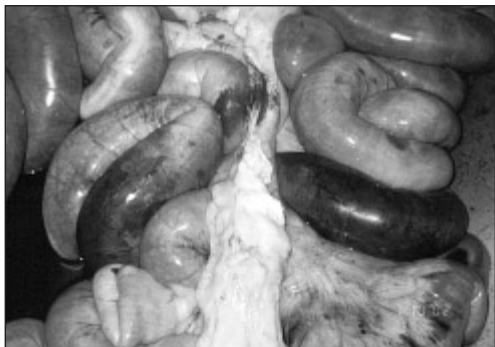
Courtesy of Dr. Philibert, WCVM

mixed inflammatory or cellular infiltrate. Gram-positive rods or cocci were evident in biopsy specimens in some affected cows.²

Medical treatment

Due to the peracute nature of the syndrome, it should be considered an emergency.⁶ There is no exact protocol for the treatment of JHS. Different medications have been tried with variable results. The use of intravenous calcium was beneficial in some cases. Flunixin meglumine (1.1 mg/kg IV) or isoflupredone acetate (20 mg/kg IM) have been used for pain and shock control.⁶ Attempts have been made to flush the blood clot from the lumen of the intestine by using oral fluids and mineral oil with variable results.⁶ In a recent study of 22 affected dairy cows, 7 of 8 cows receiving a wide range of medical treatments died. The therapies included: antibiotics (procaine penicillin G 22,000 U/kg IM, ceftiofur sodium 2.2 mg/kg IM or IV, oxytetracycline, erythromycin), intravenous fluid therapy, flunixin meglumine and 3% lidocaine.² Calcium salts, *Clostridium perfringens* types C and D antitoxin, nalbuphine, metachlopropamide, dexamethasone, morphine, magnesium hydroxide and sulfate, neostigmine, transfaunation, hypertonic saline solution, poloxalene, and neutral-buffered 10% formaline were also used.²

Figure 3: Intestines filled with blood in a cow with JHS



Surgical treatment

Affected cattle are poor candidates for surgical intervention and may not even survive transportation.⁶ Surgical procedures that have been tried include intestinal resection and anastomosis or alternatively, manual massage of the affected area to break down the offending clot.⁶ In one study, 9 of 13 cows that underwent surgery died or were euthanized.² The most commonly reported surgical observations in this study were hemorrhagic enteritis, devitalization and ischemia of the proximal portion of the jejunum, distended loops of bowel, dark red to purple discoloration of the serosal surface of the bowel, and intraluminal blood clots tightly adherent to the mucosa. In another study, 7 of 13 cows that underwent surgery were discharged.⁷ The surgical procedures that were performed on the discharged cows were intestinal resection 1/3 (number survived/number of times procedure performed), enterotomy 2/6, and massage 4/4.

Case discussion

Case 1

A 3-year-old Holstein-Friesian cow presented with a history of anorexia, abdominal distension, and blood in the feces. On physical examination, the cow appeared depressed and uncomfortable. The heart rate was irregular and elevated to 96 beats/minute (normal range 60-80 beats/minute). Temperature and respiratory rate were normal. The mucous membranes were pale and the gastrointesti-

nal tract was static. On auscultation and percussion, a pinging sound was heard over the upper abdomen. Fluid sloshing sounds were audible on succussion of the right lower flank. Rectal palpation revealed a full and doughy rumen, distended loops of small intestine in the right abdomen, and a reduced amount of dark brown feces in the rectum.

Laboratory findings

Venous blood gas analysis showed hypochloremia (73 mmol/L), hypokalemia, and metabolic alkalosis. A hemogram showed evidence of inflammation and anemia. Urine analysis was within normal limits. The fecal occult blood test was positive. Abdominocentesis revealed evidence of enterocentesis.

The differential diagnosis and the plan

The differential diagnoses for this cow include JHS, winter dysentery, bleeding and perforating abomasal ulcers, acute salmonellosis, right abomasal displacement and volvulus, intestinal intussusception and volvulus, and acute peritonitis. Winter dysentery is a herd problem. It is not usually limited to 1 cow or characterized by distended loops of small intestine on rectal examination. Abomasal ulcer disease would not explain the presence of severe hypochloremia, metabolic alkalosis, or distended loops of small intestine. Acute salmonellosis and peritonitis are usually associated with fever; also, they are not associated with severe hypochloremic metabolic alkalosis. Right abomasal displacement does not cause rapid systemic deterioration or distention of the small intestine. In case of right abomasal volvulus, the abomasum is usually palpable per rectum and there is no distention of the small intestine. Intestinal intussusception and intestinal volvulus are very difficult to differentiate from JHS; however, melena is usually not seen and the feces are scant and more mucoid.⁸ Based on these considerations, a tentative diagnosis of JHS was made and the cow was treated with intravenous lactated Ringers solution and oxytetracycline at a dose of 10 mg/kg BW for 4 days. The cow did not improve and had to be euthanized. JHS was confirmed on the post-

mortem examination and *C. perfringens* type A was isolated from the intestinal contents.

Summary

JHS is a newly recognized, fatal syndrome with unknown etiology. It affects both dairy and beef cattle. JHS should be suspected when there is a sudden death or when signs of abdominal pain and distention, bloody feces, and pale mucous membranes are reported. Necropsy findings usually reveal hemorrhagic and necrotic inflammation of the jejunum, with intraluminal blood clots. Diagnosis can be made from clinical signs, history, surgery, and necropsy findings. Surgical treatment of the syndrome seems to be more beneficial than medical treatment, but even so, the mortality is high. Bovine practitioners should be aware of the clinical signs and necropsy findings in order to report affected cattle and assist in a better understanding of the syndrome. It is hoped that with greater awareness and better recognition of this new syndrome, methods will be found to treat and prevent this condition.

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Sameeh M. Abutarbush, B.V.Sc. graduated from the Jordan University of Science and Technology in 1999. After graduation, he worked for one year in a large animal practice in Jordan and then spent one year in the large animal clinic at Purdue University, Indiana, USA. He completed an internship in large animal medicine at the WCVI. Currently, he is in the first year of a 3-year large animal internal medicine residency.

Abstracts of Interest

Hemorrhagic bowel syndrome in dairy cattle: 22 cases (1997-2000).

DENNISON AC, VANMETRE DC, CALLAN RJ, DINSMORE P, MASON GL, ELLIS R.P. COLORADO USA.

OBJECTIVE: To determine signalment, history, and clinical, necropsy, and microbiologic findings in dairy cows with hemorrhagic bowel syndrome.

DESIGN: Retrospective study.

ANIMALS: 22 adult dairy cows from a single farm in Colorado.

PROCEDURE: Medical records were reviewed for information on signalment, medical and reproductive history, the owner's chief complaints, results of physical examinations and ancillary diagnostic tests, treatment and response to treatment, results of microbiologic testing, and, if applicable, post-mortem findings.

RESULTS: Common clinical signs were acute signs of profound depression, decreased milk production, tachycardia, ruminal stasis, abdominal distention, and dark clotted blood in the feces. Rectal examination revealed distended loops of small intestine in 7 of 14 cows. Transabdominal ultrasonography revealed small intestinal ileus and distention in 12 of 12 cows and homogeneous echogenic intraluminal material compatible with intraluminal hemorrhage and clot formation in 4. Seven of 8 cows treated medically died; 9 of 13 cows that underwent surgery died or were euthanized. *Clostridium perfringens* was isolated from fecal samples from 17 of 20 cows. The most common morphologic diagnosis at necropsy was severe necrohemorrhagic enteritis or jejunitis with intraluminal hemorrhage or blood clots. The most prominent histologic finding was severe, segmental submucosal hemorrhage and edema of the small intestine.

CONCLUSIONS AND CLINICAL RELEVANCE: Results confirm that in adult cattle, hemorrhagic bowel syndrome is a sporadic acute intestinal disorder characterized

by intraluminal hemorrhage and obstruction of the small intestine. *Clostridium perfringens* was consistently isolated from the feces of affected cows. The prognosis for affected cows was grave. *J Am Vet Med Assoc* 2002;221(5):686-9.

Case Report – Jejunal Hemorrhage Syndrome of Dairy Cattle

KIRKPATRICK MA, TIMMS LL, KERSTING KW, KINYON JM.

In the last three years veterinary practitioners from Iowa, Minnesota and Wisconsin have reported, with increased frequency, a peracute, segmental hemorrhagic enteritis in mature dairy cattle. Based on these reports, clinicians at Iowa State University have begun to suspect Jejunal Hemorrhage Syndrome (JHS) as a new emerging disease syndrome.

The morbidity rate for this disease has been sporadic, and mortality approaches 85-100% due to the peracute nature and severity of this disease. There are frequently no prodromal signs and the mature cow is found dead, or an individual cow is found down and in systemic collapse. Clinical signs include sternal recumbency, diaphoresis, enophthalmia and signs of shock due to occlusion of the small intestine. Ballotment of the standing cow in the lower right abdominal area can elicit a pronounced fluid slosh, due to backup of ingesta and fluid behind the occlusive lesion. Signs of abdominal pain include bruxism, vocalization, treading and kicking at the abdomen. At necropsy, segmental lesions localized to the jejunum are observed. These areas consist of frank hemorrhage and immediate clotting, forming a functional occlusion of the small intestinal lumen. Necrosis of the lumen may or may not be apparent.

In April 1999, the Veterinary Diagnostic and Production Animal Medicine Department at the Iowa state University College of Veterinary Medicine was asked by a northeastern Iowa veterinarian to investigate recurring sporadic peracute death losses. Examination of affected cattle, production records, rations and postmortem results led investigators to conclude that a variant of *Clostridium perfringens*, specifically type A, should be considered further as a possible cause of this disease syndrome.

Recommendations for investigation of a problem herd are discussed. Specific items of increased risk in this particular herd included an association between increased milk production and death loss, an increased incidence rate associated with higher soluble carbohydrate feeding rates, and disease following re-introduction of culture-positive alfalfa haylage. There appear to be gut motility aberrations, evidenced by intussusceptions in tandem with jejunal hemorrhage syndrome lesions.

Bovine Practitioner 2001;35:104-116.

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Fax: (306) 651-4179
Email: butlerd@sk.sympatico.ca
www.cattle.ca/wcabp/

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