

Scientific Report

Hemorrhagic bowel syndrome in dairy cattle in Iran: a case report

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Summary

Hemorrhagic bowel syndrome is a highly fatal intestinal disease of adult dairy cattle with uncertain cause. In a dairy herd in Khorasan Razavi province, Northeast of Iran, two cows showed depression, anorexia, decrease in milk production, ruminal hypomotility, bruxism and dehydration. At necropsy, massive hemorrhage and clot formation was observed within the jejunum and bacterial culture of the intestinal ingesta and lesions showed the presence of a large number of *Clostridium perfringens*. Subacute ruminal acidosis was detected in fresh and mid-lactation cows. This report shows the possibility of diagnosis of other hemorrhagic bowel syndrome cases in dairy cows in Iran.

Key words: Hemorrhagic bowel syndrome, Dairy cow, Iran

Introduction

Hemorrhagic bowel syndrome (HBS, also known as jejunal hemorrhage syndrome, bloody gut or fatal jejunal hemorrhage) is a highly fatal intestinal disease and individual cases have occurred in Beef cow and in mature dairy cows (Abutarbush and Radostits, 2005).

Initially, HBS described in a dairy farm in the USA (Idaho) at 1991 and named "point source hemorrhage" (Anderson, 1991). Later clinical cases were reported in Canada, Germany and Italy (Rademacher *et al.*, 2002; Abutarbush and Radostits, 2005; Ceci *et al.*, 2006). Hemorrhagic bowel syndrome is sporadic and characterized by acute necrohemorrhagic enteritis of small intestine with progressive and occasionally massive development of clots within the intestine that create obstruction (Kirkpatrick and Timms, 2004; Ceci *et al.*, 2006).

At present, it is thought that HBS is a multi factorial disease and possible risk

factors of HBS include: stage of lactation, season, herd size, forage quality, ration composition and feeding management (Godden, 2003; Ewoldt and Anderson, 2005).

The case fatality rate ranging from 85 to 100% (Abutarbush and Radostits, 2005) and successful treatment of disease is rare, although successful treatment with intravenous fluids, calcium salts, anti-inflammatory drugs, antibiotics, and surgery was reported (Dennison *et al.*, 2002; Ceci *et al.*, 2006). Affected animals usually die within 24-48 h after the onset of clinical signs (Metre and Callan, 2005).

There is limited published information describing HBS in dairy cattle and little is known about the etiology, history, physical examination and the role of predisposing factors.

Case history

In October 2007, two cows were

examined because of anorexia in a herd of 800 Holstein dairy cows in Khorasan Razavi province, Northeast of Iran. The herd was fed total mixed rations (TMR).

A 4-year-old cow with 220 days in milk, 60 days gestation and 38 kg daily milk production showed depression, anorexia, decrease in milk production, ruminal hypomotility, bruxism and dehydration, tachycardia and normopnea. Body temperature was in normal range (38°C). Supportive treatment consisting of intravenous fluid therapy (saline dextrose 5%, 6 L) and antibiotic therapy (cotrimoxazole 24%, KELA Laboratoria N.V, Belgium, 24 mg/kg I.V.) was not successful and 20 h after the onset of clinical signs the cow was unconscious and unable to rise and euthanized.

Two days later, a 7-year-old dry pregnant cow (265 days gestation) showed anorexia, depression, bruxism and dehydration. However the body temperature was lower than normal range (37.5°C) and the rectum was empty. The cow euthanized 6 h after the onset of clinical signs due to lateral recumbency and unconsciousness.

Unfortunately, in none of the cases hematological and serum biochemical parameters were measured.

Immediately following euthanasia, complete gross necropsy was performed. At necropsy, in both cases, massive hemorrhage and clot formation were observed which created obstruction within the small intestine (jejunum) (Fig. 1). Enlargement of mesenteric lymph nodes and petechial hemorrhages of abomasal mucosa were evident. Samples of intestinal wall lesions and ingesta were obtained from the jejunum.

Bacterial culture of the tissue and ingesta samples showed the presence of a large number of *C. perfringens*.

Two groups of 12 cows were selected randomly. One group consisted of early lactation cows of the herd (3-20 days in milk) while the other consisted of mid-

lactation cows (60-150 days in milk). Ruminal fluid collection was carried out by means of rumenocentesis 4 to 6 h following TMR feeding (Nordlund *et al.*, 1995). Ruminal fluid pH was determined immediately with a portable pH-meter (Horiba, B-213, Kyoto, Japan). Cows with a rumen pH = 5.5 or less at the time of rumenocentesis, considered to be experiencing subacute ruminal acidosis (SARA) and a ruminal pH ≥ 5.8 considered as a non-affected cow. Rumen samples of fresh cows were obtained at the day of the first case observation and samples of mid-lactation cows were obtained at the day of the second case detection. Four cows in each group had rumen pH of 5.5 or less and both fresh and mid-lactation cows were considered to be experiencing SARA (Nordlund *et al.*, 1995; Garrett *et al.*, 1999).

Before rumenocentesis, TMR sampling was performed from the feed bunk at the time of feed delivery. TMR sampling was carried out from ten places across the bunk at random intervals (Behnke, 1996). Particle size analysis of TMR samples was performed using the modified Penn State Particle Separator (Kononoff *et al.*, 2003). Dry matter, Kjeldahl nitrogen (Buchi 339, Buchi Corp., Switzerland), fat (Soxhlet method, Buchi 810, Buchi Corp., Switzerland), neutral detergent fiber (NDF, Tecator 1010 fiber digester, FOSS Corp., Denmark) and ash percentage of TMR samples were measured.

Physically effective neutral detergent fiber (peNDF) of rations were estimated by as a proportion of dry matter retained by 19, 8 and 1.18-mm screens of Penn State Particle Separator multiplied by dietary NDF content. The ration of mid-lactation cows had the minimum recommended peNDF (Shaver, 2005).

Non-fiber carbohydrate (NFC) was calculated as: 100 - (%NDF + %CP + %fat + %ash). Non-fiber carbohydrate of both rations was in recommended range (Table 1)

Table 1: Overview of some measured parameters in the rations of early and mid-lactation cows

	NDF (%)	NFC (%)	peNDF (%)
Fresh cows	32.35	39.37	25.5
Mid-lactation cows	30	42.6	22.2
Recommended range	25-28	35-50	20-22
	(NRC, 2001)	(Krause and Oetzel, 2006)	(Yang <i>et al.</i> , 2001; Shaver, 2005)



Fig. 1: Massive hemorrhage and clots formation in the jejunum of dry pregnant cow (A and B). Massive hemorrhage of the wall of the jejunum and bloody intestinal contents (C)

(Krause and Oetzel, 2006).

The forage and feed stuffs (corn silage and hay) were sampled and analyzed for dry matter and NDF. Visual evaluation and comparison between the measured NDF and dry matter values and NRC values showed good quality of forage and feed stuffs.

Discussion

Typical gross lesions at necropsy and bacteriological results supported the diagnosis of HBS in both cases (Dennison *et al.*, 2002; Ceci *et al.*, 2006; Muskens *et al.*, 2007), although there was not histopathologic, hematologic and biochemical data.

Clostridium perfringens was isolated from the intestinal contents or faeces of the majority of HBS cases and is suspected as a cause or contributing factor in HBS, although the role of this organism in the pathogenesis of HBS is uncertain. *Clostridium* spp. live in the intestine of normal healthy cows (Godden, 2003) and rapid overgrowth of bacteria following death can occur, therefore, faecal culture of *C. perfringens* can not be considered as a diagnostic test. In this study, sampling of the lesion sites immediately after euthanasia, made postmortem overgrowth of *C. perfringens* unlikely. At present, it is unclear whether proliferation of *C. perfringens* in HBS affected cows occurs as a part of the primary disease process, or is secondary to another disease process or triggering factor. However, the toxins release during rapid growth of *C. perfringens* could contribute to the intestinal wall lesions that are characteristic of HBS (Metre and Callan,

2005).

A hypothesis for the cause of *C. perfringens* overgrowth is the overflow of finely ground carbohydrates from forestomachs (Ewoldt and Anderson, 2005). This situation arises in association with the same factors which lead to SARA (i.e. feeding excess amounts of rapidly fermentable carbohydrates or insufficient effective fiber) and some authors have discussed a possible association between the occurrence HBS and SARA (Godden, 2003). In a dairy herd two groups of cows are more susceptible to SARA, fresh cows and mid-lactation cows (Nordlund *et al.*, 1995). Therefore, we assessed the occurrence of SARA in these groups. This is the first time that contemporary occurrence of SARA and HBS has been approved.

The NFC of both rations was in the recommended range, but in addition to the level of NFC, the type of NFC in the ration affects the carbohydrates degradation at rumen and is essential for SARA occurrence (Krause and Oetzel, 2006). Also, the type of NFC may affect the overgrowth of *C. perfringens*. We did not determine the type of ration carbohydrates which seems important in HBS cases.

Another hypothesis for the etiology of HBS is poorly fermented ensiled feeds which may lead to accumulate the harmful mold or bacteria and their toxins. The occurrence of HBS with feeding poor quality silage has been reported by Kirkpatrick and Timms (2004). However, forage and feed stuffs of good quality were used in this study.

Some authors believe that the occurrence of HBS increase in the fall and winter

months and clinical cases mostly observed in Holstein cows (Godden, 2003; Abutarbush and Radostits, 2005), but others reported the most cases in spring and summer in Brown Swiss cattle (Ceci *et al.*, 2006). A total of 61% of HBS cases were reported within the first 100 days of lactation and 22% occurred in mid-lactation (101-200 days in milk). Also, 94% of cases occurred in the second lactation and older cows (Godden, 2003; Kirkpatrick and Timms, 2004). Higher milk production of these cows is in association with higher dry matter intake and carbohydrate consumption, both of them are considered as possible risk factors for HBS and SARA. Therefore, HBS is most commonly seen in early to peak lactation cows and the occurrence in dry period is less usual (Metre and Callan, 2005). There is higher risk for HBS in herds with ≥ 100 cows and TMR feeding (Godden, 2003).

Without any correction, no new case of HBS was diagnosed in this herd. In the past few years, the prevalence of HBS has increased (Godden, 2003). Although, the cause of HBS is not yet known, identifying and controlling all proposed causes or risk factors may be the best strategy to prevent HBS cases in dairy herds.

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