

Beef Sessions

Moderator: Pat Burrage

A Review of Abomasal Ulcers in Beef Calves

Tessa Marshall, BVSc, MS, Diplomate ABVP

Assistant Teaching Professor, College of Veterinary Medicine, University of Missouri, Columbus, MO

Abstract

Abomasal ulcers are an economic concern for the calf raising industries. Many factors have been attributed to the development of ulcers, such as coarse feed, environmental stress, vitamin and mineral deficiencies, and bacterial infections. However, unlike human ulcers no one factor has been proven to have a significant relationship to abomasal ulcers in calves. Therefore, management of this disease and prevention of losses is limited to theoretical control of many known inciting causes.

Résumé

Les ulcères de l'abomasum occasionnent des pertes économiques à l'industrie de l'élevage des veaux de boucherie. Plusieurs facteurs contribueraient au développement de l'ulcère de l'abomasum, tels que les aliments à texture grossière, le stress environnemental, les carences en vitamines et en minéraux, et les infections bactériennes. Toutefois, contrairement aux ulcères des humains, aucun facteur unique ne s'est avéré avoir un lien significatif avec l'ulcère de l'abomasum chez le veau. Par conséquent, la gestion de cette maladie et des pertes économiques qu'elle entraîne se limitent au contrôle des nombreux facteurs qui y contribuent.

Introduction

Abomasal ulcers and erosions are an economic concern for calf raising systems. The prevalence of abomasal ulcers has been reported to be 0.2 to 5.7% in beef calves¹⁶, 32²⁶ to 76 % in healthy veal calves,²⁵ 1.0⁶ to 2.6% in healthy dairy cows,¹⁰ 1.8% in healthy beef cows¹⁴ and 1.6 % in feedlot cattle¹⁵. The difficulty in management of ulcers lies in determining underlying causes, many of which have been identified. Theories include dietary changes, in particular the addition of coarse roughage feeds causing trauma to the mucosa, as well as pica, secondary to enteritis, abomasal bezoars, environmental and physical stress, hyperacidity, vitamin E deficiency, lactic acidosis, mycotic infection and compromised

immune status associated with copper deficiency. Several microorganisms have been isolated from various cases of ulcers including *E. coli*, sarcina-like spp, *Clostridium perfringens* and more. However, unlike *Helicobacter pylori* in human gastric ulcers, no infectious component has been confirmed in cattle.

Both erosions and ulcers may be found in the abomasum. Erosions are discrete mucosal defects that do not penetrate the muscularis mucosae. They are usually multiple, circular and small, and appear as hyperemic indentations in the mucosa. In contrast, ulcers penetrate the entire thickness of the mucosa and may extend through the submucosa, muscularis externa and serosa. They may be single or multiple and vary in size, and usually appear as an irregular depression in the abomasal surface. The central crater of the ulcer is covered with fibrinonecrotic material and is surrounded by raised, rounded edges. There are four categories of abomasal ulcers described, however, an animal may have ulcers that simultaneously meet the criteria for more than one of these categories (i.e. ulcers that bleed and perforate).

- Type 1: non-perforating ulcer. The ulcer does not perforate the abomasal wall and intraluminal hemorrhage is minimal.
- Type 2: non-perforating ulcer with severe blood loss. The ulcer does not perforate the abomasal wall, but erodes a major vessel in the submucosa, resulting in severe intraluminal hemorrhage.
- Type 3: perforating ulcer with local peritonitis. The ulcer perforates the abomasal wall and abomasal contents leak into the peritoneal cavity or omental bursa. Peritonitis is localized by fibrin deposition and the abomasum becomes adhered to the peritoneum, omentum, or surrounding viscera.
- Type 4: perforating ulcer with diffuse peritonitis. The ulcer perforates the abomasal wall and abomasal contents quickly leak into and spread throughout the peritoneal cavity, resulting in diffuse peritonitis.²⁴

In veal calves, abomasal ulcers are typically found around the pylorus.^{8,10,22,25} The presence of ulcers in this region has been attributed to several things, including alkalization of the pyloric antrum by bile reflux from the duodenum.²¹ However, the ductus choledochus and ductus pancreaticus enter the small intestine at a greater distance from the pylorus in cattle than in humans, thus decreasing the likelihood of bile reflux,²¹ not to mention the presence of the torus pyloricus which may act as a ball valve preventing reflux. Mechanical trauma to the pyloric mucosa by coarse feedstuffs and trichobezoars has also been suggested.^{11,16,25}

Abomasal ulcers are difficult to diagnose in the living animal because signs or symptoms are uncommon. Affected calves are often found dead, and usually are higher quality calves compared to the rest of the herd, and offspring of heavy-milking dams. Affected bottle-fed calves are usually vigorous nursers. This behavior may allow spillover of milk into the reticulorumen, allowing bacterial fermentation and colonization of the abomasum. However, ulcers in veal calves are a common incidental finding at slaughter. Severity scores²⁵ for ulcers showed calves raised in loose housing scored significantly higher than crated calves. Calves with higher scores (more severe lesions) also had the heaviest carcass weights, thus ulcers did not appear to have a significant effect on growth rates.²⁵ In one report, calves with abomasal lesions had similar feed efficiency as calves without ulcers.⁸

Age distribution of ulcers is fairly consistently divided into two groups. There is a reduction in cases around two months of age, creating two groups – one at the preruminant stage (<3 weeks) and at the transitional phase (3-8 weeks) when the abomasum is most susceptible to ulcer formation.¹² This age grouping has often been associated with seasonal occurrence rates, most likely due to having a suitable ‘susceptible’ group of calves present in the spring and early summer.

Outbreaks of abomasal ulcers and tympany often occur after periods of bad weather. During extreme weather calves often fail to nurse or nurse poorly, and may subsequently engorge themselves after the weather improves, which could result in proliferation of bacteria that cause fermentation.¹⁷

Housing has been found to play a small part in the incidence of abomasal ulcers. Increased stocking density or an increase in the availability of dirt or hair in the environment may play a role. In one report, calves on pasture had a significantly lower incidence of disease than those housed in pens or on stubble fields.¹⁶

Stress has been associated with ulcers in other species. Several factors discussed above (housing, group mixing and weather) are suspected to contribute to ulcer formation through the body’s response to stress. Cortisone and ACTH reduce gastric mucus secretion

which limits mucosal protection. Also, steroids decrease cell renewal in the gastric mucosa.¹⁷

Hair balls (trichobezoars) are thought to abrade the abomasal mucosa, which in turn may disrupt normal mucosal defensive barriers and allow autodigestion to occur. In one study¹³, calves less than one month of age and dying from an ulcer were almost four times more likely to have a hairball in their abomasum than calves dying of other causes. However, in older calves about 60% of calves had abomasal hairballs, regardless of cause of death. Thus, hairballs are not likely necessary for development of ulcers.

Mineral deficiencies are often associated with ulceration and poor performance, in particular copper deficiency. Low serum copper may result in derangement of elastin cross linkages, thus compromising the abomasal mucosa and its microvasculature. Copper deficiency may also cause decreased cytochrome oxidase activity of leukocytes, which contributes to decreased neutrophil function and increased susceptibility to infection.²³ The role played by copper deficiency in ulcer formation is questionable based on the methods of obtaining the research results currently available and their interpretations.

Cl. perfringens type A has been associated with many diseases in cattle. Experimental intraruminal inoculation of *Cl. perfringens* type A resulted in varying degrees of depression, diarrhea, abdominal bloat, abomasitis and abomasal ulceration.²³ However, the ulcers were frequently multiple, diffuse, never perforating and associated with ecchymotic and petechial hemorrhage and mucosal edema. In a study of 14 fatal ulcers, only six had a heavy growth of *Cl. perfringens* type A, suggesting this bacterium is not necessary for the formation of fatal ulcers.¹¹ The ulcerative lesions in beef cattle differ in that they are typically singular and localized to a discrete region of the abomasum.¹²

Cl. perfringens type D has been isolated from at least one case of abomasitis and ulceration. *Cl. perfringens* type D is a normal inhabitant of the small intestine of ruminants. Feed changes such as irregular feeding and hungry animals drinking excessive milk have been suggested as being responsible for promoting overgrowth of *Cl. perfringens* type D. Affected calves were six weeks of age and showed signs of decreased appetite and lethargy. At necropsy the abomasum contained dark fluid, and the mucosa was edematous and covered by many minute ulcers.⁵

Clinical Signs

A thorough physical examination of suspected cases is essential because of the many differential diagnoses. Signs include lethargy, abdominal distension with tympany, colic, bruxism, fluid distension of the abomasum, diarrhea and death. The hematocrit is usually low with

Type 2 ulcers; a fecal occult blood test may be positive with both Types 1 and 2 ulcers. The Hematest is the best test currently available, however, a single negative test does not rule out gastrointestinal bleeding. The sensitivity of the test increases when testing multiple samples. Plasma fibrinogen concentration is often high with Types 3 and 4 ulcers, and a WBC count may show leukocytosis/neutrophilia. Abdominocentesis often indicates inflammation with Type 4, and sometimes Type 3 ulcers.

Exploratory laparotomy is the most definite way to diagnose ulcers antemortem. Laparotomy or necropsy are the only ways to definitively diagnose ulcers. Most ulcers are found on the greater curvature of the fundic region or in the pyloric region, while erosions are usually located on the edges (linear erosions) and sides (punctate erosions) of the abomasal folds.

Differential Diagnoses (incomplete list)

Bleeding ulcers: coccidiosis, salmonellosis, intussusception, duodenal ulcer, uterine artery rupture, abomasal volvulus, bovine viral diarrhea/mucosal disease, malignant catarrhal fever, winter dysentery, mycotoxicosis, heavy metal poisoning, coagulopathies.

Perforated ulcers: traumatic reticuloperitonitis, rupture of another abdominal organ (e.g., uterus), other septic conditions (metritis, mastitis).

Other differentials: ruminal tympany, uncomplicated LDA or RDA, indigestion, internal parasitism, dilatation/dislocation/torsion of the cecum and peritonitis of other origins.

Diagnostic Tests

One diagnostic test that has been investigated is pepsinogen concentration. Pepsinogen is an inactive form of pepsin, which is an important proteolytic enzyme. Bovine pepsinogen is activated when the abomasal pH is ≤ 5.0 . As the luminal pH increases (>3.0), the proteolytic activity of pepsinogen decreases. Increased activation of pepsinogen to pepsin by enhanced acidity can cause ulcers.²⁰ Pepsinogen is elevated when there is a gastric or duodenal ulcer, but the specific role of serum pepsinogen in detecting abomasal ulcers in cattle has not been confirmed. At least one study has shown that a serum pepsinogen concentration of >5.0 U/L coincided with severe ulceration of the abomasal mucosa. This test is non-specific as other diseases, such as ostertagiasis, will also cause an elevation in pepsinogen concentration.^{6,19,20}

Treatments and Control Recommendations

Normal preprandial abomasal pH in calves is between 1.0 and 2.0 units.¹ In adult cows this pH is between 2.0 and 2.5.

Suckling frequency has been mooted as a method for control of abomasal pH in calves. Ahmed *et al*² found that increasing the frequency of suckling increased the mean 24-hour abomasal luminal pH and the percentage of the 24-hour period that the pH was over 3.0. Beef calves usually suckle three to six meals a day. This study found that suckling eight times per day (q3h) increased the luminal pH to more than 3.0 for the greatest percentage of the 24-hour recording period.

In general, treatment is reserved for animals with bleeding ulcers or deep, non-perforated ulcers. Treatment of abomasal ulcers has historically included blood transfusions (4.5 to 9.1 ml/lb [10 to 20 ml/kg] blood) and antibiotic administration, changes in the diet and oral administration of antacid agents.^{7,9} Increasing the luminal pH with antacids is intended to provide a more favorable healing environment for ulcers.

Aluminum hydroxide $\text{Al}(\text{OH})_3$ and magnesium hydroxide $\text{Mg}(\text{OH})_2$ antacids neutralize acid in the abomasum, but require multiple doses (3X) per day. Antacids are an inexpensive option for treatment of cattle with ulcers and have a potential therapeutic advantage. $\text{Al}(\text{OH})_3$ directly absorbs pepsin, thus decreasing the proteolytic action of pepsin in the stomach.¹⁸ Also, $\text{Al}(\text{OH})_3$ and $\text{Mg}(\text{OH})_2$ bind bile acids, thus protecting against ulceration caused by bile reflux.¹⁸ Ahmed *et al*³ found that healthy calves given oral antacids had a transient increase in abomasal luminal pH. This effect appeared to be dose related and the extent of the acid neutralization was increased when not given with milk replacer.

Histamine type-2 receptor antagonists reduce acid secretion of parietal cells by selective and competitive antagonism of histamine at H-2 receptors on the parietal cells. Cimetidine and ranitidine are synthetic H-2 antagonists that inhibit basal as well as pentagastrin- and cholinergic-stimulated gastric acid secretion. Daily oral administration of cimetidine at 4.5 mg/lb (10 mg/kg) for 30 days to veal calves was found to aid healing of abomasal ulcers.⁹ Oral administration of cimetidine (23 to 45 mg/lb; 50 to 100 mg/kg) and ranitidine (4.5 to 22.7 mg/lb; 10 to 50 mg/kg, q 8 h) caused a dose-dependent increase in abomasal luminal pH; and ranitidine had greater potency than cimetidine.¹

Proton-pump inhibitors, like omeprazole, act by preventing acid secretion. It has prolonged duration of action which provides for easy daily dosing regimes. In a study by Ahmed *et al*⁴ once daily oral administration of omeprazole to calves increased the mean 24-hour luminal pH by 1.3 pH units, however the pH tended to decrease over time with treatment. There appeared to be some compensation by the body to the elevated abomasal pH.

Surgical repair of ulcers can be performed on selected cases. Calves should be in an operable condition

and have isolated ulcers; surgery needs to be performed early in the disease process. Typically ulcers are diagnosed during laparotomy, and can be either resected or the serosa can be inverted with a mattress suture. One study reported a survival rate of 74% after surgical repair of ulcers (survival defined as calves living at least three months past surgery date or those dying due to another disease).¹⁶

Because the cause of ulcers is not really known and few animals in a herd show signs or die from ulcers, design of prevention strategies is difficult. Focus should be on assessing trace mineral supplementation, particularly copper, which may require assessment of sulfur and molybdenum concentrations in the diet and water. Liver biopsies for copper concentration from a percentage of the animals and feed/forage analysis may be necessary to determine correct supplementation rates.

Young calves should be vaccinated against clostridial diseases, including *Clostridium perfringens*. In herds where necropsies have been performed and *Cl. perfringens* type A has been diagnosed, it may be worthwhile to use an autogenous or generic type A vaccine. Reducing stressors, like mixing of groups of animals, providing appropriate housing and adequate feed may also be beneficial.

More research on abomasal ulcers in calves is needed to fill gaps in our knowledge so we can move from providing vague recommendations based on 'theory' to science-based prevention and treatment programs.

Conclusions

Many factors have been associated with ulcer development in calves. Ulcers are very common in veal calves, but rarely cause clinical disease. Perforating ulcers can cause acute sudden death in suckling beef calves on pasture. The etiology is uncertain, but is probably associated with prolonged inappetence and sustained low pH. Unfortunately ulcers appear to be a multifactorial disease requiring a management approach to minimize the risk to calves and reduce economic losses. More research is required to find economical prevention strategies for control of abomasal ulcers in calves.

References

- Ahmed AF, Constable PD, Misk NA: Effect of orally administered cimetidine and ranitidine on abomasal luminal pH in clinically normal milk-fed calves. *Am J Vet Res* 62:10:1531-1538, 2001.
- Ahmed AF, Constable PD, Misk NA: Effect of feeding frequency and route of administration on abomasal luminal pH in dairy calves fed milk replacer. *J Dairy Sci* 85:1502-1508, 2002.
- Ahmed AF, Constable PD, Misk NA: Effect of an orally administered antacid agent containing aluminum hydroxide and magnesium hydroxide on abomasal luminal pH in clinically normal milk-fed calves. *J Am Vet Med Assoc* 220(1):74-79, 2002.
- Ahmed AF, Constable PD, Misk NA: Effect of orally administered omeprazole on abomasal luminal pH in dairy calves fed milk replacer. *J Vet Med A* 52:238-243, 2005.
- Assis RA, Lobato FCF, Facury Filho EJ, Uzal FA, Santana FJF, Dias LD, Parreiras PM: Isolation of *Clostridium perfringens* type D from a suckling calve with ulcerative abomasitis. *Arch Med Vet XXXIV* 2:287-292, 2002.
- Aukema JJ, Breukink HJ: Abomasal ulcer in adult cattle with fatal hemorrhage. *Cornell Vet* 64:303-317, 1974.
- Braun U, Bretscher R, Gerber D: Bleeding abomasal ulcers in dairy cows. *Vet Rec* 129:279-284, 1991.
- Breukink HJ, Wensing T, van Dijk S, Mevius D: The effect of clenbuterol on the incidence of abomasal ulcers in veal calves. *Vet Rec* 125:109-111, 1989.
- Dirksen GU: Ulceration, dilatation and incarceration of the abomasum in calves: clinical investigations and experiences. *Bov Pract* 28: 127-135, 1994.
- Hemmingsen I: Erosiones et ulcera bomasi bovis: *Nord Vet Med* 18:354-365, 1966.
- Jelinski MD, Ribble CS, Chirino-Trejo M, Clark EG, Janzen ED: The relationship between the presence of *Helicobacter pylori*, *Clostridium perfringens* type A, *Campylobacter* spp, or fungi and abomasal ulcers in unweaned beef calves. *Can Vet J* 36:379-382, 1995.
- Jelinski MD, Ribble CS, Campbell JR, Janzen ED: Descriptive epidemiology of fatal abomasal ulcers in Canadian beef calves. *Prev Vet Med* 26:9-15, 1996.
- Jelinski MD, Ribble CS, Campbell JR, Janzen ED: Investigating the relationship between abomasal hairballs and perforating abomasal ulcers in unweaned beef calves. *Can Vet J* 37:23-26, 1996.
- Jensen R, Deane HM, Cooper LJ, Miller VA, Graham WR: The rumenitis-liver abscess complex in beef cattle. *Am J Vet Res* 15:202-216, 1954.
- Jensen R, Pierson RE, Braddy PM, Saari DA, Benitez A, Lauerman LH, Horton DP, McChesney AE: Fatal abomasal ulcers in yearling feedlot cattle. *J Am Vet Med Assoc* 169:524-526, 1976.
- Katchuk R: Abomasal disease in young beef calves: Surgical findings and management factors. *Can Vet J* 33:459-461, 1992.
- Lilley CW, Hamar DW, Gerlach M, Johnson JL: Linking copper and bacteria with abomasal ulcers in beef calves. *Vet Med* 80:85-88, 1985.
- Maton PN, Burton ME: Antacids revisited. A review of their clinical pharmacology and recommended therapeutic use. *Drugs* 57(6):855-870, 1999.
- Mesaric M, Zadnik T, Klinkon M: Comparison of serum pepsinogen activity between enzootic bovine leucosis (EBL) positive beef cattle and cows with abomasitis. *Slov Vet Res* 39(3/4):227-232, 2002.
- Mesaric M: Role of serum pepsinogen in detecting cows with abomasal ulcer. *Vet Arhiv* 75:2:111-118, 2005.
- Ooms L, Oyaert W: Electromyographic study of the abomasal antrum and proximal duodenum in cattle. *Zentralbl Veterinarmed [A]* 25:464-473, 1978.
- Pearson GR, Welchman D de B, Wells M: Mucosal changes associated with abomasal ulceration in veal calves. *Vet Rec* 120:557-559, 1989.
- Roeder BL, Chengapa MM, Nagaraja TG, Avery TB, Kennedy GA: Experimental induction of abdominal tympany, abomasitis, and abomasal ulceration by intraruminal inoculation of *Clostridium perfringens* type A in neonatal calves. *Am J Vet Res* 49:201-207, 1988.
- Smith DF, Munson L, Erb HN: Abomasal ulcer disease in adult dairy cattle. *Cornell Vet* 73:213-224, 1983.
- Welchman D deB, Baust GN: A survey of abomasal ulceration in veal calves. *Vet Rec* 121:586-590, 1987.
- Wensing T, Breukink HJ, Van Dijk S: The effect of feeding pellets of different types of roughage on the incidence of lesions in the abomasum of veal calves. *Vet Res Comm* 10:195-202, 1986.