Abomasitis and abomasal bloat

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Abomasitis (abomasal bloat) is a sporadic disorder of neonatal to weanling calves, lambs, and goat kids. It is characterized by diffuse, hemorrhagic to necrotizing inflammation of the abomasal mucosa, frequently involving the deeper layers of the abomasal wall in severe or chronic cases. Abomasal ulceration and perforation may occur in a subset of affected animals. Emphysema and edema of the abomasal wall may be present.

Clinical signs include lethargy, abdominal distension with tympany, colic (abdominal pain), bruxism (teeth grinding), fluid distension of the stomach, diarrhea, and death. Although the number of case studies on abomasitis is few, upon review of the available literature, the case fatality rate appears to be high (75 to 100 percent).

Cause
A variety of proposed causes for this disease exist, including primary bacterial or fungal infection, immunosuppression, pica, trauma from coarse feed or trichobezoars (hairballs), and vitamin/mineral deficiencies. In 1987, investigators at Kansas State University detected Clostridium perfringens types A and E in stomach contents of affected calves, and the following year reproduced the disease experimentally by intraruminal inoculation of C. perfringens type A in calves. Belgian investigators have also detected C. perfringens type A in the abomasums of affected calves.

The ability of this organism to produce gas is considered to contribute to gastric dilation and intramural emphysema evident in affected animals. More recently, Salmonella typhimurium DT104 was isolated from the abomasal wall of Midwestern veal calves with abomasitis. Another bacteria, Sarcina ventriculi, has been associated with abomasal bloat in calves, lambs, and goat kids.

Treatment
Treatment of abomasitis/abomasal bloat is often unrewarding. Antitoxin for Clostridium perfringens types C and D has unknown efficacy in treatment of cases involving C. perfringens type A. Intravenous fluid therapy, parenteral antibiotic therapy, and stopping milk feeding for 12 to 24 hours are often included in initial medical management. Orogastric tube passage and fluid/gas decompression of the stomach may be helpful.

Elevation of the calf’s forequarters while the tube is placed may (continued on next page)
facilitate the release of gas. Dropping the end of the tube to a level below the calf’s stomach may aid in siphoning off retained ingesta from the stomach. Oral antibiotics such as penicillin or tetracycline may be helpful in reducing the rate of intraluminal gas production.

Decompression of the abomasum via percutaneous ventral abomasocentesis has been described, and intraluminal injection of antibiotics could be performed after decompression. Laxatives appear to be of limited benefit in such cases, and large doses of magnesium oxide/hydroxide laxatives are likely contraindicated, as they may worsen metabolic alkalosis, induce hypermagnesemia, and simply pull more fluid into the gut lumen.

A large, right-sided tympanic resonance in an ill calf may actually be a case of abomasal or cecal volvulus, and surgical exploration is indicated if initial medical management does not result in resolution of tympany. Similarly, a left-sided tympanic resonance may reflect left displacement of the abomasum (LDA), and given the apparent high rate of ulceration of the abomasum associated with LDA in calves, surgical exploration is warranted in cases that do not respond to medical management.

Abomasotomy may be indicated for refractory cases of abomasal tympany. Abomasotomy allows for removal of luminal foreign bodies such as hairballs and removal of putrefying milk, both of which may prevent a satisfactory response to medical management.

**Prevention**

Although authors of earlier case reports associated copper deficiency with abomasitis and abomasal ulcers in beef calves, Roeder and colleagues from KSU demonstrated that abomasitis could occur spontaneously and be induced experimentally in the absence of copper deficiency. Thus, although copper deficiency may act as a contributory factor for abomasitis and enteric disease of calves, it does not appear to be a requisite factor for either condition.

Cases of this disease in neonatal beef calves have been associated temporally with management practices that cause delays in regular nursing patterns (e.g. calf separation at branding) or changes in environment that interrupt normal nursing patterns (e.g. winter storms).

In dairy calves, poor milk hygiene, intermittent feeding of large volumes of milk, and feeding cold milk or milk replacer have been empirically incriminated as potential contributory factors for abomasal tympany, ulceration, and abomasitis. Epidemiologists at The Ohio State University are currently conducting a practitioner survey study on abomasitis and abomasal tympany, and further insights into potential preventive strategies for this disease syndrome are forthcoming.

Anecdotal reports indicate that increasing the frequency of milk or milk replacer feeding and decreasing the volume fed at each feeding, as well as maintaining milk or replacer at body temperature until it is fed, may reduce the incidence and severity of this condition. Investigating which calves are affected, and their order in the feeding pattern, may reveal a greater incidence of disease in calves that receive cooled milk or milk replacer.

A vaccine that induces high antibody titers against alpha toxin, the primary virulence factor of *C. perfringens* type A, has recently been released into the U.S. market for prevention of diseases in cattle caused by this organism. As a dry cow vaccine, this product may increase colostral titers against alpha toxin, but the efficacy of this product in reducing calfhood diseases caused by *C. perfringens* type A is currently undetermined.

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