

AN UPDATE ON CLOSTRIDIAL DISEASES/
ABOMASAL ULCERS

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Losses of calves in beef herds to Clostridial diseases continues to be a problem. In a large 1991 Colorado study¹ of 47 herds with 11,767 cows reported that two of the most common causes of calf mortality were enteric and sudden death diseases. Both of these entities are consistent with Clostridial disease. Unfortunately, less than 1% of the calves that died were ever presented to a diagnostic lab for confirmation of death and 99% was based on producer perception. A smaller study² conducted in 1992-93 of 15 beef herds in Colorado, Wyoming and Nebraska with 3,273 calves born attempted to at least get a better handle on the extent of Clostridial related disease. In this study, a total of 153 died (4.7%) with 93 calves dying between 48 hours and branding at about 2 months of age. All 93 calves were submitted for field necropsy and sample submission for confirmatory laboratory diagnosis. This was a cooperative effort involving 8 local veterinary practitioners who received uniform training and 2 state diagnostic laboratories. Causes of death were categorized into 1) abomasitis, 2) abomasal ulcer, 3) clostridial enteritis, 4) general enteritis, 5) respiratory/pneumonia, and 5) other causes of death. Samples were submitted to diagnostic labs in Colorado and Nebraska and the information assimilated into the same six categories reported by the practitioners. Table 1 reports the association between field necropsy and diagnostic lab results.

Table 1. Association of field necropsy and diagnostic lab results on calf losses between 48 hours and branding (about 2 months) in 15 beef herds in Colorado, Wyoming and Nebraska²

Category	Field DVM Dx	Lab Diagnosis	Agreement
Abomasitis	14 (15.1%)	3 (3.2%)	21.4%
Abomasal Ulcer	14 (15.1%)	4 (4.3%)	28.6%
Clostridial enteritis	14 (15.1%)	38 (40.8%)	50.0
General enteritis	22 (23.6%)	30 (32.3%)	50.0%
Resp/Pneumonia	8 (8.6%)	5 (5.4%)	50.0%
Other, injury	21 (22.5%)	13 (14.0%)	45.0%
Total	93 (100%)	93 (100%)	

Multiple theories have been presented in an effort to explain the development of Clostridial diseases. Commonly, these theories suggested feed changes, environmental or physical stress, and nutritional deficiencies in addition to the bacteriology as some of the disease factors. Many organisms have been isolated from affected abomasal tissues in affected neonatal calves. Still it remains an enigma for both veterinarians and producers. It is probable that multiple factors and multiple disease-producing agents may be involved. The objective of today's presentation is to update producers, in as much as possible, with the current understanding of this problem.

Is copper deficiency involved and if so what is its role?

The idea that ulcer formation may be related to a copper deficiency was first proposed by Lilly, et al. in 1985³. The association of copper deficiency with abomasal ulcers is summed up in a quote from that publication, "Our statistical data have shown a highly significant correlation between abomasal ulcers and copper deficiency. The exact role that copper deficiency plays in abomasal ulcers, however, is **purely speculative**". 1985:86). This statement pretty well sums up where we are with copper and its association with abomasal ulcers today. That copper deficiency is associated with increased disease incidence is not questioned but its association with specific diseases remains speculative. Although a small study², the herd with the lowest mean cow copper levels had the lowest IgG levels in calves, highest calf morbidity to disease (47%), and close to the highest calf mortality (7.7%) of all the herds. Possible impacts of copper deficiencies include (1) decreased immune function (decrease macrophage and cytochrome oxidase activities) and (2) compromised microvasculature of the abomasum leading to decreased motility and an impaired ability of the mucosa to protect itself from acid/pepsin digestion³. Results from other studies^{4,5} indicate copper concentrations (serum and tissue) are not related to the occurrence of abomasal ulcers. However, no numeric values were reported for either liver or serum copper concentrations in the first study⁴ and the second study⁵ only reported serum concentrations. One other consideration with regard to copper concentration is defining at what point do animals become deficient. Lilly, *et al.*³ reported hepatic copper values of between 45-48 ppm (dry weight) in their diseased animals, considering these to be significantly lower than their control group. Jelinski⁶ suggested, based on two separate sources (Personal communication with Dr. M. Smart, University of Saskatchewan; Puls, 1988:76), that a more reasonable cut-off point for defining hepatic copper deficiencies would be 35 ppm (dry weight) and 10 ppm (wet weight).

Bacteriology

The idea that *Clostridium perfringens* infections are associated with abomasal ulcers was started with two studies published by Kansas workers^{4,5}. The first study involved isolating *C. perfringens* from eight neonatal calves with suspected abomasal displacement or intestinal obstruction. Toxin neutralization tests in mice concluded seven of the 8 cases were *C. perfringens* type A. The second study involved inoculating eight calves intraruminally with toxigenic *C. perfringens* type A and then evaluating the calves for 10 days. All calves in the second study manifested signs of abdominal tympany or abomasitis to some degree, with two deaths being presumably due to inoculation with *C. perfringens* type A. However, a more

recent study⁶ has reported finding “no compelling evidence that *Clostridium perfringens* type A...[is] involved in ulcer formation”. This study evaluated histological sections and bacterial cultures for 30 unweaned calves (all <3 months of age) that were necropsied for fatal ulcers (14 cases) or unrelated deaths (16 controls). *Clostridium perfringens* as a group of organisms is divided into 5 types by its ability to produce one or more toxins. A number of diseases have been associated with these toxins (See Table 2)

Table 2. Diseases produced by toxigenic types of *Clostridium perfringens*⁷

Toxin Type	Major Toxin	Diseases
A	Alpha	Myonecrosis, food poisoning, necrotic enteritis in fowl, entertoxemia in cattle and lambs, necrotizing enterocolitis in piglets; possibly equine colitis, canine hemorrhagic gastroenteritis
B	Beta	Dysentery in newborn lambs, chronic enteritis in older lambs, hemorrhagic enteritis in neonatal calves and foals, hemorrhagic enterotoxemia in adult sheep
C	Alpha, Beta	Enteritis necroticans in humans, necrotic enteritis in fowl, hemorrhagic or necrotic enterotoxemia in calves , pigs, lambs, goats, foals, acute enterotoxemia in adult sheep
D	Alpha, Epsilon	Entertoxemia in calves , and lambs, enterocolitis in neonatal and adult goats, and possibly enterotoxemia in adult cattle
E	Alpha, Iota	Enterotoxemia likely in calves and lambs, enteritis in rabbits; host range and disease type unclear

Because Type A organisms are a part of the normal flora of the intestinal tract of virtually all warm-blooded animals, the veterinary community has been reluctant to accept a Type A etiology for enteritis and entertoxemia⁷. Type A *Clostridium perfringens* has been associated with gastrointestinal lesions⁸ and is the predominate isolate found in cattle with enterotoxemia in different studies^{9,10}. Further, unlike Types B, C, D, and E which are defined by what toxins they do produce (beta, epsilon, and iota), Type A is a loosely defined group of organisms that do not produce these toxins⁷. Thus, Type A is still a pool of organisms that do not fit the other types and therefore as yet undetermined groups within Type A may be associated with specific disease syndromes.

Vaccines

Protection against the diseases produced by the toxins is afforded by an immune response to the toxoid agent. Lack of association seen in some studies is most likely associated with failure to recognize the many facets of Clostridial diseases, misdiagnosis, and possibly improper vaccination protocols. Type C and D toxoids do not afford protection against Type A diseases. Autogenous vaccines have provided assistance in this area. Work is being done at a number of institutions in the development of a more complete vaccine.

Summary

Diagnostic techniques are currently available or becoming more available for typing out specific *Clostridium perfringens* isolates and toxins produced by those isolates. With this new technology, several studies are being conducted to better define the pathogenicity of the various syndromes associated with *Clostridium perfringens* Type A.

References

- ¹Salman, MD *et al.* Annual disease incidence in Colorado Cow-Calf Herds Participating in Rounds 2 and 3 of National Animal Health Monitoring System from 1986 to 1988. J Am Vet Med Assoc 198(6): 962-967, 1991.
- ²Tombs RE, *et al.* Postnatal Calf Losses in Beef Herds: Causes and Epidemiological Characteristics. Large Animal Practice Jul/Aug 1998: 19(4), 16-24.
- ³Lilley CW, *et al.* Linking Copper and bacteria with abomasal ulcers in beef calves. Veterinary Medicine, Oct 1985: 85-88.
- ⁴Roeder BL, *et al.* Isolation of *Clostridium perfringens* from neonatal calves with ruminal and abomasal typany, abomasitis, and abomasal ulceration. JAVMA 1987:1550.
- ⁵Roeder BL, *et al.* Experimental induction of abdominal typany, abomasitis, and abomasal ulceration by intraruminal inoculation of *Clostridium perfringens* type A in neonatal calves. Am J Vet Res 1988:201
- ⁶Jelinski MD, *et al.* 1995. The relationship between the presence of *Helicobacter pylori*, *Clostridium perfringens* type A, *Campylobacter* spp, or fungi and fatal abomasal ulcers in unweaned beef calves. Can Vet J 36:379-382.
- ⁷Songer JG. *Clostridium perfringens* Type A infection in Cattle. 1999 AABP Proceedings.
- ⁸Al-Mashat RR, Taylor DJ. 1983. Bacteria in enteric lesions in cattle. Vet Rec 112: 5-10.
- ⁹Daube G *et al.* 1989. Diarrhea associated with *Clostridium perfringens* type A enterotoxin in neonatal pigs. J Vet Diagn Invest 1:351-353.
- ¹⁰Yoo HS *et al.* 1997. Molecular typing and epidemiological survey of prevalence of *Clostridium perfringens* types by multiplex PCR. J Clin Microbiol 35: 228-232.