

12-1-2007

Clostridium perfringens Infections in Baby Calves

Russ Daly
South Dakota State University

Lori Rotert
South Dakota State University

Follow this and additional works at: http://openprairie.sdstate.edu/extension_extra

Recommended Citation

Daly, Russ and Rotert, Lori, "Clostridium perfringens Infections in Baby Calves" (2007). *Extension Extra*. Paper 397.
http://openprairie.sdstate.edu/extension_extra/397

This Other is brought to you for free and open access by the SDSU Extension at Open PRAIRIE: Open Public Research Access Institutional Repository and Information Exchange. It has been accepted for inclusion in Extension Extra by an authorized administrator of Open PRAIRIE: Open Public Research Access Institutional Repository and Information Exchange. For more information, please contact michael.biondo@sdstate.edu.



Clostridium perfringens Infections in Baby Calves

Russ Daly, DVM, Extension Veterinarian
Lori Rotert, Pre-Veterinary Student

Illnesses and death losses in baby calves are significant problems for producers raising calves in beef or dairy operations. Several of these issues, especially sudden deaths and certain enteric (intestinal) conditions, are potential effects of infections due to *Clostridium perfringens*. *Clostridium perfringens* are Gram-positive, spore-forming, anaerobic bacteria that are very commonly found in many environments, including soil, water, poorly preserved feeds, contaminated or improperly thawed colostrum or milk, calf-housing environments, and the normal bovine intestinal tract. In small amounts, these bacteria are generally harmless in the intestine, but under the right conditions they may grow and proliferate, resulting in **enterotoxemia**, a condition in which specific toxins produced by the bacteria in the small intestine result in both local damage and systemic (whole body) effects.

Pathogenesis: How *C. perfringens* causes disease

Because of the widespread nature of the organism, calves are readily exposed to *C. perfringens* in their environment and commonly ingest the bacteria in various quantities, after which it enters the stomach and intestine. Sometimes bacteria are ingested in sufficient quantities to cause disease, but oftentimes small quantities are ingested, followed by rapid proliferation in the intestine.

Enterotoxemia due to *C. perfringens* is more likely to affect baby calves (within the first two months of age) than mature cattle because the calves lack a fully functioning rumen. *C. perfringens* feeds on starches and sugars in the small intestine. In mature cattle, these

starches and sugars are predominantly digested in the rumen, so they are not available to the *C. perfringens* microbes for use. However, in baby calves, nearly all feed bypasses the rumen and is digested in the abomasum (true stomach) and the small intestine, so the starches are available for the microbes to feed on. This, coupled with a normal intestinal flora that has not yet developed, provides a suitable environment for the *C. perfringens* to proliferate. Several factors can contribute to this rapid proliferation. Primary among these are abrupt changes in feeding patterns (see "Prevention" below), physical or environmental stress, nutritional deficiencies, and conditions that impair movement of the intestine (such as diarrhea due to other causes).

As *C. perfringens* proliferates in the gut, the bacteria secrete toxins that have profound effects not only on the local intestinal environment (causing damage to the intestinal lining), but throughout the body as well. Death occurs when high levels of these bacterial toxins enter the bloodstream, leading to inflammation, shock, and cardiac arrest. *C. perfringens* is not spread from calf to calf, but it is not uncommon for several calves in a group to be affected at the same time, due to similar exposure and management practices.

Types of *C. perfringens*

There are five types of *C. perfringens* (see Table 1 below), designated A through E, which are identified based on the toxins they produce. It is the effect of these specific toxins that results in the clinical signs and syndrome attributable to each type.

Table 1. *C. perfringens* types, major toxins, and associated syndromes

TOXIN TYPE	MAJOR TOXIN	SYNDROMES
A	Alpha	<ol style="list-style-type: none"> 1. Abomastitis (stomach inflammation) in calves 2. Bloating in calves 3. Mild diarrhea in calves 4. May be associated with hemorrhagic bowel syndrome (HBS) in dairy cattle
B	Beta, Alpha, Epsilon	<ol style="list-style-type: none"> 1. Hemorrhagic enteritis in newborn calves (NOT FOUND IN U.S.)
C	Beta, Alpha	<ol style="list-style-type: none"> 1. Sudden death in very young calves 2. Hemorrhagic enteritis (bloody scours)
D	Epsilon, Alpha	<ol style="list-style-type: none"> 1. Enterotoxemia resulting in sudden death, usually in calves on feed (finishing cattle)
E	Iota, Alpha	<ol style="list-style-type: none"> 1. Fatal enterotoxemia in newborn calves

Type C is one of the more commonly encountered types of *C. perfringens*. It is especially virulent in calves less than 10 days old (and often less than five days old). Enterotoxemia due to *C. perfringens* Type C may result in severe bloody diarrhea, although often-times calves die before diarrhea develops.

Clostridium perfringens **Type A** has been increasingly identified as a cause of abomasal inflammation, which may result in abdominal distension (bloating) or abomasal ulcers in young calves. Varying degrees of diarrhea, and occasionally sudden onset of weakness and coma, have also been associated with Type A, which generally affects a slightly older (2-4 weeks old) calf. It is also occasionally associated with wound contamination and gas gangrene. Diagnosis of Type A enterotoxemia can be difficult due to the fact that it is a very common inhabitant of the normal intestinal tract; therefore, culture results need to be matched to clinical signs and lesions in the tissues (see “Diagnosis” below).

Enterotoxemia due to *C. perfringens* **Type D** is sometimes associated with sudden death in finishing cattle because it proliferates when there are high amounts of carbohydrates available in the diet, which is often the case in cattle being fed high amounts of grain in finishing diets.

Type E enterotoxemia has been diagnosed, although infrequently, in South Dakota calves. It causes a severe local intestinal necrosis and systemic toxemia similar to the syndrome described with Type C.

Type B is almost non-existent and has not been found in North America.

Diagnosis of *C. perfringens*

Necropsy of the calf, in cases of Types C and E, will sometimes reveal severe necrosis (tissue damage) and hemorrhage in the small intestine. Calves affected with Type A will often show inflammation, ulceration, and hemorrhage of the lining of the rumen and abomasum. Because *C. perfringens* is often found in the intestine of normal calves, a simple culture of the organism from the calf is not sufficient by itself to confirm a diagnosis of disease due to *C. perfringens*. Culture results are matched with clinical signs, lesions in the tissues, and, in some cases, toxin identification, to obtain a true diagnosis. Tissue samples from calves suspected of having clostridial enterotoxemia should be collected soon after death and kept well-preserved (after the death of the calf, normal populations of clostridial organisms can overgrow and confuse diagnosis).

TREATMENT

The treatment of syndromes caused by *C. perfringens* is frequently unsuccessful. Because clinical signs are usually a result of the toxin, not the bacteria itself, treatment with antibiotics (which act solely on the bacteria and not the toxins) is often less than rewarding. In addition, with many of these syndromes, death or severe illness occurs before treatment can even be attempted; therefore, early detection becomes essential if treatment is to be successful.

Typical treatments for calves with milder clinical signs consist largely of antibiotics (especially penicillin) and the use of *C. perfringens* antitoxin products. Several injectable antitoxin preparations that contain specific antibodies directed against toxins produced by *C. perfringens* are currently available. While these antitoxin products are developed expressly for use against toxins produced by Types C and D (beta and epsilon toxins), there may be some effect against alpha toxin (Type A) as well. Supportive care with oral or IV fluids and anti-inflammatories may also be indicated. Any treatment plan needs to be developed in close consultation with the herd veterinarian.

PREVENTION

Prevention of enterotoxemia due to *C. perfringens* infection focuses on three areas: 1) minimizing exposure, 2) enhancing immunity in the young calf, and 3) managing feeding practices to discourage the proliferation of *C. perfringens* in the gut.

1) **Minimizing exposure** Despite the fact that clostridial organisms are ubiquitous in the calf's environment, the more organisms a newborn calf is exposed to the more likely it is to succumb to disease. For that reason, careful attention must be paid to sanitation of the calving area, whether it be a maternity pen in a dairy or a calving pen in a beef operation. Buildup of all enteric pathogens occurs when the same calving area is used throughout the calving season, as these organisms are generally very hardy in the environment.

2) **Enhancing immunity** Colostral (passive) immunity is of utmost importance in the resistance of calves to *C. perfringens* enterotoxemia, since these calves are generally too young (and clinical signs occur too early) to respond to active vaccination. Antibodies against *C. perfringens* and its toxins are passed from dam to calf through colostrum. Calves should consume an adequate amount (4 quarts) of high-quality colostrum within the first 18-24 hours of life to gain maximum protection.

A strategy for maximizing colostrum antibody levels is vaccinating the dam before she calves. Vaccines against clostridial diseases are typically "bacterin-toxoids," meaning that the vaccine is meant to stimulate a response against both the bacteria and the toxin produced by the bacteria. Several pre-calving vaccines ("scour shots") contain *C. perfringens* Type C (and

occasionally D) bacterin-toxoids. A separate vaccine against Type A antigens has been conditionally licensed and is available. Since vaccines for Type C (against the beta toxin) do not cross-protect against other types (other toxins), and while not expressly labeled for this use, this vaccine has been used in cows pre-calving to try to maximize colostrum protection against Type A enterotoxemia (alpha toxin).

Along with colostrum antibodies, antitoxin preparations, as mentioned above, are available. In certain situations of excessive exposure, these products may be administered to calves at birth in an attempt to prevent clinical disease.

Active vaccination of young calves with *C. perfringens* vaccines (such as 7-ways or 2-ways containing Types C and D, or the Type A vaccine) is frequently attempted, but little is known about how these vaccines perform in very young (<1 month) calves. Again, these vaccines do not offer protection against the other types (other toxins) not included in the vaccine.

3) **Managing feeding practices** While cow-calf producers have very little control over their calves' feeding practices, those raising calves (e.g., dairy calves in hutches) have complete control over their calves' dietary intake. In those operations, it is extremely important to follow strict calf-feeding guidelines. *C. perfringens* overgrowth in the intestine is aided by any of the following:

- Milk replacer is improperly mixed (too much or too little water added).
- Milk replacer is incompletely mixed (clumps of powder still present at feeding).
- Additives such as electrolytes are added to the milk replacer, creating excessive osmolarity or sodium concentrations.
- The temperature of milk replacer is too high or low when the calf is fed. Milk replacer temperature should be close to the calf's body temperature (~101° F).
- Feeding schedules are erratic. Regular, consistent schedules and delivery methods (bucket vs. bottle) are important in avoiding clostridial overgrowth.
- Changes in diets are made suddenly.
- Calves drink too quickly (e.g., nipple opening is too large).
- Equipment (buckets, bottles, nipples, etc.) becomes contaminated due to lack of hygiene.

Clostridial infections have been and will continue to be significant challenges to beef and dairy producers. Attention must be paid to all aspects of the disease, including the newborn calf's environment, immunity,

and feeding practices. With these aspects in mind, and with close consultation with a veterinarian, it is possible to manage and minimize the effect of these syndromes in our operations today.

REFERENCES

- McGuirk, S. M. Solving Calf Morbidity and Mortality Problems, Preconvention Seminar 7: Dairy Herd Problem Investigation Strategies, American Association of Bovine Practitioners 36th Annual Conference, September 15-17, 2003 - Columbus, OH
- Mortimer, R.G. et al. 2001. An Update on Clostridial Diseases/Abomasal Ulcers. Range Beef Cow Symposium XVII Proceedings: 32-35.
- Songer, J. Glenn. 1999. Clostridial Enterotoxemia. Current Veterinary Therapy Food Animal Practice. 4th ed: 388-390.
- Songer, J. Glenn and Dale Miskimins. 2005. Clostridial Abomasitis in Calves: Case Report and Review of the Literature. Anaerobe 11:290-294.
- Songer, J. Glenn and Dale Miskimins. 2004. Clostridium perfringens Type E Enteritis in Calves: Two Cases and a Brief Review of the Literature. Anaerobe 10:239-242.