# Clostridial Abomasitis and Enteritis in Ruminants

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**KEYWORDS**

• *Clostridium perfringens* • *Clostridium difficile* • Abomasitis • Enteritis • Enterotoxemia • Ruminant

**KEY POINTS**

- Clostridial abomasitis and enteritis are characterized by necrosis of the abomasal or intestinal mucosa caused by exotoxins produced by *Clostridium perfringens* or *Clostridium difficile* in the lumen of the gastrointestinal tract.
- *C perfringens* types A, B, C, D, and E can cause enteric disease in all species of domestic ruminants. The 5 genotypes are identified by the presence of the genes for the lethal exotoxins alpha, beta, epsilon, and iota.
- Multiplex polymerase chain reaction is used diagnostically to identify *C perfringens* genotypes from anaerobic culture of samples.
- Proliferation of *C perfringens* in the ruminant gastrointestinal tract is associated with a combination of increased availability of carbohydrate or protein, and alteration in gastrointestinal motility.
- Treatment of abomasitis and enteritis caused by *C perfringens* should focus on 6 goals: relief of abdominal distention, systemic fluid support, prevention of *C perfringens* proliferation, decreasing or preventing exotoxin production, restoration of normal gastrointestinal flora, and providing pain management as needed.

## INTRODUCTION

Clostridial diseases affecting the gastrointestinal tract are common in ruminant livestock; however, their classification can be confusing because of the varied nomenclature of the disease conditions.[1–4] Described diseases have included hemorrhagic enterocolitis, enterotoxemia, pulpy kidney disease, overeating disease, braxy, bradsot, struck, lamb dysentery, enterotoxemic jaundice, yellow lamb disease, clostridial

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abomasitis, and clostridial enteritis. Other than braxy (bradsot) of sheep, which is caused by *Clostridium septicum*, these diseases are all caused by different subtypes of *Clostridium perfringens*. *C perfringens* type A can also cause gangrenous mastitis and may be present in high numbers in spoiled milk.

Clinical disease is associated with rapid bacterial overgrowth within the gastrointestinal tract and subsequent exotoxin release. Although limited tissue invasion by *C perfringens* does occur, most local and systemic lesions result from the effects of potent exotoxins produced by certain genotypes of these bacteria. *C perfringens* is a large, gram-positive, anaerobic bacillus that exists ubiquitously in the environment and in the gastrointestinal tract of most mammals.[3,5–8] There are 5 defined types, or genotypes, of *C perfringens*: A, B, C, D, and E (**Table 1**). These genotypes are identified based on the lethal toxins that they produce: *C perfringens* alpha (CPA), *C perfringens* beta (CPB), epsilon (ETX), and *C perfringens* iota (CPI).[7–10] The alpha toxin gene (*plc*) is present on the chromosome of all *C perfringens* isolates.[7,8] All genotypes produce alpha toxin, although isolates differ significantly in the amount of alpha toxin produced.[11,12] The other lethal toxins, CPB (*cpb* gene), ETX (*etx* gene), and CPI (*iap/ibp* genes) are contained on transferrable plasmids.[7,8]

Two other toxins, enterotoxin (*C perfringens* enterotoxin [CPE], *cpe* gene) and the beta-2 toxin (CPB2, *cpb2* gene), are also carried on transferable plasmids in livestock isolates. Enterotoxin can be expressed by any of the subtypes if the plasmid containing this gene (*cpe*) is present, but it is not required for pathogenicity. Enterotoxin is not released by vegetative bacteria but only in sporulating *C perfringens* cells during lysis of the vegetative cell.[7] Thus, the toxin may not be present in intestinal contents of animals with *C perfringens* enteritis unless sporulation is occurring. The beta-2 toxin may be produced by type A as well as by some isolates of types B, C, and E.[13] Strains of *C perfringens* that carry the beta-2 toxin gene have been isolated from a variety of species of domestic animals, including horses, camelids, cattle, and swine.[13,14]

The genotype, and thus the specific subtype of *C perfringens*, can be determined by a multiplex polymerase chain reaction (mPCR) that detects the specific toxin genes carried by an individual isolate. Because the clinical and pathologic presentations of diseases caused by *C perfringens* types are not always distinct, anaerobic culture and polymerase chain reaction (PCR) genotyping of the isolates can be instrumental in determining the subtype involved and can help identify specific control measures. Each of the subtypes of *C perfringens* is associated with specific disease syndromes that are directly or indirectly related to the toxins they produce. The term enterotoxemia is often loosely used to describe enteric or systemic disease caused by any of the *C perfringens* toxinotypes. The term enterotoxemia refers to systemic disease caused by absorption of a toxin from the intestine. Clostridial abomasitis and enteritis does not require absorption of the toxins. The term enterotoxemia is best reserved for cases in

**Table 1**
**Toxins, gene, genetic location, and toxin action expressed by different *Clostridium perfringens* toxinotypes (genotypes)**

| | *C perfringens* Type | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Toxin** | **A** | **B** | **C** | **D** | **E** | **Gene** | **Gene Location** | **Toxin Action** |
| Alpha (CPA) | + | + | + | + | + | *plc* | Chromosome | Phospholipase |
| Beta (CPB) | — | + | + | — | — | *cpb* | Plasmid | Pore formation |
| Epsilon (ETX) | — | + | — | + | — | *etx* | Plasmid | Pore formation |
| Iota (CPI) | — | — | — | — | + | *Iap/ibp* | Plasmid | Cytoskeleton disruption |