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Field experiences with *Clostridium perfringens A* diarrhea and options for treatment and prevention

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Introduction

Clostridium perfringens type A disease of swine has been a frustrating experience in the field. It contrasts with *Clostridium perfringens* type C, where vaccines have been available for many years and are highly effective. Since the identification of *Clostridium perfringens* type A as a cause of neonatal scours in piglets to its solidification as the number one agent associated with neonatal piglet diarrhea, it has continued to elude effective prevention, treatment and control strategies in the field.

Clinical signs

Clostridium perfringens type A (CpA) is a normal inhabitant of the porcine gastrointestinal tract. Like other Clostridial organisms, they are large, gram positive, spore forming, anaerobic rods. Characteristics of diarrhea associated with CpA infection are a pasty to creamy diarrhea and typically last about five days. Feces can become mucoid and pinkish in appearance.

Pathogenesis

The pathogenesis of *Clostridium perfringens* type A enteritis is not clear. Presumably, piglets are infected shortly after birth by spores shed by the sow in her feces. Disease can be seen as early as 12 hours after birth, but is most common in three day old piglets. Most infections are resolved by seven to ten days of age. Mild cases may result in no additional pre-weaning mortality, while severe cases can have a significant effect on pre-weaning mortality. Additionally, weaning weights and pig quality suffer when piglets are affected by *Clostridium perfringens* type A. Stunting of growth can extend well beyond weaning. The clinical signs associated with CpA infection are due to intestinal damage and toxin production. It is thought that two toxins are responsible for the secretory diarrhea by CpA infection: enterotoxin and beta2 toxin.

Diagnosis

Diagnosis of *Clostridium perfringens* type A is made through a combination of clinical signs and isolation of large numbers of organisms from affected portions of the small intestine. In some cases, large numbers of rods can be seen in histologic sections of the small intestine lumen. Genotyping can be done to confirm the organism's ability to produce enterotoxin and beta2 toxins. Co-infections with *Clostridium dificile* and/or rotavirus can occur, so it is important to rule these agents out. *C. dificile* co-infection is perhaps the most difficult to rule out, since culture for this agent is not routinely done, and the toxin used to confirm *C. dificile* is easily degraded in poorly-handled diagnostic samples.

Prevention and treatment

There are no known "silver bullets" for CpA prevention or treatment. Many things have been tried, but to my knowledge, none have been consistently effective. These include:

- Increase hygiene
- Control of concurrent disease
- Feedback
- Antibiotics
- Electrolytes
- Anti-toxins/anti-sera
- Autogenous biologics
- Conditionally licensed biologics
- Herbal extracts
- Direct fed microbials
- Parity segregation

Increase hygiene

For farms that are dealing with continuous CpA problems, a review of sanitation practices in the farrowing house is in order. Review practices to ensure that bio-films that can harbor spores are being removed in the wash process. Sanitation can be increased through the use of hot water, physical scrubbing in combination with an effective detergent or cleaner, and use of a disinfectant with known sporicidal activity. However, because the etiology of CpA has not been worked out, do not be surprised if an increase in sanitation practices is not wholly effective in controlling the disease.

Control of concurrent disease
Diagnostics to confirm the role of CpA in neonatal scour cases are also useful in determining if there are other agents present. When rotavirus is involved effective feedback and/or vaccination can improve the degree of clinical severity due to mixed infections. Be sure to submit frozen colon contents from euthanized piglets for *C. dificile* toxin identification. Watch for the colonic edema that is often present in *C. dificile* affected piglets. From field cases, clinical PRRS infection of the sow herd can increase the number and severity litters affected with CpA.

Feedback
Fecal feedback from CpA scours has been commonly tried with limited success. In fact, there are reports that this practice can exacerbate CpA cases.

Antibiotic treatment
Pre-farrow and farrowing feed medication with BMD seems to help. Due to the way this product is labeled, accurately dosing sows requires a tremendous commitment by farm staff. It is not clear if the failures of this product are due to efficacy of compliance limitations. Administration of ceftiofur containing products can increase the number of scour cases that are treated by farm staff. From field cases, three day oral treatment of piglets with BMD soluble or injectible treatment with Tylan appear to be the most effective treatments for affected piglets.

Electrolytes
Since piglets become dehydrated due to the secretory diarrhea produced by CpA infection, rehydration therapy can assist in controlling the mortality and number of poor thrift piglets.

Anti-toxin/anti-sera
There are commercially available preparations of anti-toxin/anti-sera that have been produced for use in other species. These have been used periodically for many years for CpA infections in piglets. In my experience, they are largely not effective.

Autogenous biologics
Autogenous vaccines have been used for several years. Anecdotal reports are mixed from no effect to very positive. It is not clear why there is such a dramatic range of experiences with autogenous products.

Conditionally licensed biologics
There is a conditionally licensed biologic for CpA that recently became available. Results from a controlled field study has not demonstrated value in using this vaccine in addition to traditional pre-farrow scour vaccination programs containing *E. coli*, *Clostridium perfringens* type C and rotavirus antigens.

Herbal extracts
Herbal extracts have been shown to have therapeutic value in addressing CpA infections in piglets. However, in field applications, there is not definitive evidence confirming the benefits from the use of these type of products.

Direct fed microbials
Many direct fed microbial products have been tried for prevention and/or treatment of CpA infections. There is not convincing evidence to date that these products add value in CpA infections.

Parity segregation
In mixed parity herds, disease due to CpA can be seen in pigs from all parities. This contrasts to parity segregated flows where the vast majority of CpA cases are confined to the P1 farm.

Conclusion
We continue to struggle to better understand this disease and how to better control and prevent it.

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