Diagnosis of *Clostridium perfringens* type A enteritis in piglets

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History

A 120-sow, continuously farrowing swine unit from Minnesota experienced an epidemic of diarrhea in 1- to 3-day-old pigs. Clinical illness, which usually began shortly after birth, was characterized by high morbidity (nearly 100%) and negligible mortality (< 1%). Diarrheic pigs remained vigorous, continued to nurse, and recovered spontaneously in 4–5 days. Diarrhea was clinically unresponsive to treatment with gentamicin or trimethoprim-sulfadimethoxine but responded well to spectinomycin therapy. The illness did not adversely affect preweaning mortality rates, which remained low (< 10%) despite the persistent diarrhea in this herd throughout a 4-month period. Routine vaccination of sows against transmissible gastroenteritis (TGE), rotavirus, *Clostridium perfringens* type C, and *Escherichia coli* pilus types K88, K99, 987P, and F41 did not prevent or reduce the morbidity.

Laboratory examinations

Three live, unmedicated, acutely diarrheic, 1- to 2-day-old pigs were presented for necropsy. The pigs were alert, active, robust, and well hydrated and were excreting copious amounts of foamy, fluid, yellow feces. Grossly, lacteals throughout the length of the small intestine contained chyle, and colonic mesenteries were slightly edematous. Microscopically, villi in the small intestine were long, slender, and lined by epithelial cells distended by colostral protein. A few neutrophils were in the lamina propria. Many spore-bearing Gram-positive bacilli were in the lumen and occasionally in apposition with epithelial cells at the tips of villi. Immunofluorescence testing for TGE virus and rotavirus were negative. Viruses were not seen by electron microscopic examination of negatively stained feces. Aerobic culture of small intestine from each pig yielded a mixed growth of coliforms. Anaerobic culture of small intestine of each pig yielded a dense growth of *C. perfringens.*

Discussion

Clostridium perfringens type A enterotoxicosis, a sporadic cause of diarrhea in piglets, should be suspected in herds experiencing diarrhea in nursing piglets if the following conditions are present:

- diarrhea usually begins from 0–3 days of age;
- morbidity is high (> 90%) and mortality is low (< 1%);
- affected piglets are robust, active, well-fed, and have liquid to creamy colonic contents;
- small intestine is normal in thickness and lacteals contain chyle;
- other known enteropathogens are absent; and
- the disease often responds to antimicrobial therapy.

To establish a diagnosis of *Clostridium perfringens* type A enterotoxemia, specimens should first be examined for the more common enteric pathogens. Specimens required for optimal success in achieving an enteric disease diagnosis are listed in Table 1.

If etiologic agents are not identified using conventional diagnostic methods, additional testing can be done to detect evidence of enterotoxigenic *C. perfringens.* A diagnosis can usually be made based on history, absence of other enteropathogens, the presence of typical small intestinal lesions, and by culturing a dense growth of *C. perfringens* from the jejunum. Microscopically, small intestinal villi are of normal length. There may be increased numbers of neutrophils in the lamina propria and large, Gram-positive, spore-bearing bacilli in the small intestinal lumen. (The microscopic lesions are in marked contrast to the severe, highly fatal necrosis caused by *C. perfringens* type C.) Anaerobic culture is useful if specimens are obtained from live pigs. Swabs should be collected from jejunum because *C. perfringens* is part of the normal flora of the large intestine but is absent or found in low numbers in the jejunum. Obtaining a dense growth of *C. perfringens* from the jejunum of live, untreated pigs provides further evidence of type A enterotoxemia. Detecting type A enterotoxin may provide additional supportive evidence, but this is not routinely done and is not widely available to veterinarians in the United States.

The pathogenesis of *C. perfringens* type A diarrhea in piglets is unclear. In humans, *C. perfringens* type A causes food poisoning by elaborating a 34,000-MW protein enterotoxin which acts on the mucosa of the intestinal tract, causing fluid and

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Table 1

Specimen selection from diarrheic nursing and weanling pigs*

Histopathology

- Segments 3 cm long from duodenum, upper and lower jejunum, ileum, and colon. Flush the intestinal lumen with 10% buffered formalin, then immerse in formalin. Do not ligate because formalin penetrates poorly through the serosa.

Bacteriology

- Refrigerated jejunum and ileum and/or jejunal and ileal swabs in transport medium.
- Four air-dried impression smears of ileal mucosa may be submitted for *E. coli* pilus antigen detection by immunofluorescence.

Virology

- **Immunofluorescence**—10-cm-long segments of jejunum and ileum, placed in a plastic bag and refrigerated. Preferably do not freeze.
- **Electron microscopy**—2 to 5 ml of cecal or colonic fluid in a leak-proof container and refrigerate.

Parasitology

- Feces submitted for electron microscopy can be used for oocyst examination. Unfixed, refrigerated ileum collected for viral immunofluorescence can be used to make Giemsa-stained ileal smears for the detection of *Isospora suis* merozoites.

*Specimens should be from live, untreated pigs early in the course of the disease. Specimens should be shipped in styrofoam containers containing adequate numbers of frozen ice packs. Overnight mail should be used when needed to ensure that specimens arrive to the laboratory within 24 hours of collection.

electrolyte secretion in the ileum. The enterotoxin also induces secretion in the ileum of piglets. There are conflicting reports on the importance of enterotoxin in field cases of type A enterotoxemia. Although some authors suggest that *C. perfringens* causes wasting, it has been our experience that piglets seldom die from the effects of *C. perfringens* type A and growth rate is only mildly affected unless complicated by other enteric pathogens. Nonetheless, the disease is problematic because the diarrhea creates an unsanitary environment and because producers often request veterinary intervention, it increases treatment costs.

Treatment and control

Specific therapy and control procedures for *C. perfringens* type A have not been determined. Procedures commonly used for *C. perfringens* type C have been implemented for type A.

- Sanitation procedures such as all-in-all-out pig flow and washing and disinfecting between each farrowing group reduce exposure. Washing the sow prior to bringing her into the farrowing house also helps reduce piglet contamination, as does routine scraping of farrowing house sow manure prior to farrowing and during the first week of life.
- Colostrum uptake (timing, quality, and quantity) is very important to get maximum benefit from the sow's natural immunity.
- Heat-lamp management is critical to avoid stress and immunosuppression caused by chilling.
- Feed-grade antibiotics such as BMD™ at 250 g per ton have had some benefit when fed from 2 weeks prefarrowing through lactation.
- Individual piglet treatment (oral or injectable routes) has been successful in most cases, although evaluation of such treatment is questionable because many piglets recover if left untreated. Injectable gentamicin, penicillin, spectinomycin, and neomycin have been used successfully to reduce clinical signs of piglet diarrhea.
- Back feeding has been practiced with some success, utilizing manure from sows in the farrowing house and piglet diarrhea. The material is fed to gestating animals one to three times during the period 2-5 weeks prefarrowing.
- Success of autogenous vaccines is difficult to evaluate because autogenous vaccination is usually just one of many treatment or control procedures employed simultaneously.

More research and clinical trials must be conducted to determine the effects of *C. perfringens* type A on preweaning mortality and weaning weights.

References

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