CASE REPORT

Managing management-induced *Clostridium perfringens* type A infection in suckling pigs: A case study

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Summary: *An epidemic of transmissible gastroenteritis (TGE) in a 1650-sow herd prompted the initiation of a management protocol that included feeding back viscera from scouring piglets to the entire sow herd to hasten development of TGE antibodies. Several litters born the fourth week after the initiation of the protocol developed diarrhea at 3–5 days of age and, as was dictated by the protocol, were euthanized. When it became apparent, however, that the signs and effects on the pigs of this new outbreak of diarrhea differed from those seen with TGE, a diagnostic re-evaluation was begun. The diagnostic workup implicated* Clostridium perfringens *A. The sow herd was treated with BMD® and feedback procedures were revised.*

In February 1994, the manager of a totally confined 1650-sow herd in Nebraska discovered one litter of piglets in a 24-crate room of a total-confinement, weaning-to-breeding complex showing clinical signs of diarrhea. Within 3 days, 1- to 5-day-old piglets from 12 different litters in the room were showing clinical signs of severe diarrhea. More than 50% of their dams were also showing signs of inappetance, vomiting, and diarrhea. Further search in the building revealed a few sows in the crated gestation area also showing signs of diarrhea and anorexia. Affected piglets were severely dehydrated and mortality was 90% in litters that had been affected for > 48 hours. Physical exam, a review of the herd history, and necropsy resulted in a presumptive diagnosis of TGE.

Laboratory confirmation of TGE was completed by fluorescent antibody examination of samples of small intestine at the University of Nebraska Diagnostic Laboratory 6 days after the initial observation of diarrhea in the piglets. An epidemiologist[3] was contacted and the following protocol was developed:

- Sacrifice all pigs < 2 weeks of age that showed signs of diarrhea and collect the viscera. Combine the viscera with diarrheic sow feces, macerate, and administer as a slurry on the feed of gestating sows, continuing the feedback practice as long as there are affected specimens to harvest.
- Euthanize at birth all piglets born during the next 14 days (days 7–20).
- Euthanize all pigs in a litter born after day 20 in which even a single pig exhibited signs of diarrhea.
- Retain surviving piglets > 2 weeks old at the initiation of the outbreak in the crates of one farrowing room until they reached 30 lb.
- Discontinue all movement of pigs to the offsite nursery.
- Feed viscera to replacement gilts that are needed for the following 6 months to develop natural immunity.

Establishing immunity in the breeding herd in the presence of a TGE epidemic is readily accomplished by orally administering virus harvested from affected pigs.[1] In the unit concerned in this case, the entire breeding herd is held in crates, which aided in the assurance that all animals were equally exposed by the feedback protocol.

Litters farrowed to recovered dams on day 21 and after remained clinically normal and grew well until they were 3–5 days old. On day 26, however, several litters developed a white-to-yellow, pasty-to-watery diarrhea. Following the protocol, all pigs in affected litters were destroyed on days 26 and 27.

The unit manager examined the herd on day 28 and observed clinical signs that the syndrome, which by then was affecting 50% of litters as they reached day 3 of life, differed from that seen during the peak of the TGE epidemic. The affected pigs did not appear lethargic or chilled, did not become dehydrated, and did not develop watery diarrhea. Typical specimens were presented and appropriate tissues submitted to the University of Nebraska Diagnostic Laboratory. Bacterial culture and histopathology yielded a diagnosis of *Clostridium perfringens* type A. Treatment and control regimes for *C. perfringens* type A should be similar to that for type C.[4] Affected piglets were treated with orally administered bacitracin methylene disalicylate (BMD®) as a drench (Solu-Tracin 200, A. L. Pharma, One Executive Drive, Fort Lee, New Jersey 07024) and access to an electrolyte (Bluelite Swine For-

DPM: Swine Pro Associates, Swine Business and Health Center, 1106 19th Street East, PO Box 237, Columbus, Nebraska, 68602.

mula®, Techmix, Inc., 12915 Pioneer Trail, Eden Prairie, Minnesota 55347).

After treatment, clinical signs ameliorated and the pigs recovered, although the producer noted reduced weaning weights. The entire breeding herd was fed BMD® at 250 g per ton for 21 days. Litters born to sows during the first 8 days that the BMD® was administered experienced a 50% morbidity rate but less than a 5% mortality rate due to diarrhea. Litters born on day 9 and after of sow BMD® treatment exhibited little diarrhea. In the ensuing 6 months, only diarrhea diagnosed as coccidiosis by histopathology[2] has developed.

Discussion

Transmissible gastroenteritis virus is known to be shed by ill and recovering pigs in massive amounts and can be recovered from both intestinal and respiratory tissues up to 104 days after recovery.[1] Natural infection provides the highest and longest-lasting level of immunity to infection. To avoid endemic TGE, one must accomplish equal and concurrent infection of susceptible animals.[1] Transmitting maternal antibodies via colostrum and subsequently weaning the pigs at less than 21 days of age to an offsite nursery can eliminate the disease from the offspring.[5]

Clostridium perfringens type A occur as normal flora in the intestinal tracts of all swine.[6] Reports of clinical disease due to the organism have been made only since 1976[6] and have initiated further investigation and recommendations for diagnosis and treatment.[7] *Clostridium perfringens* types C and A both gain access only by ingestion. In the case of the piglet, maternal feces are a common source, as are contaminated environments once the bacteria contaminate a premises. In the case of this herd, the only oral-fecal contact possible was via the feedback protocol, which was important in the development of the secondary *C. perfringens* type-A infection. Management and sanitation are very good in this herd and clinical disease of any type is almost nonexistent. Transmissible gastroenteritis virus had never entered the herd in the 10 years since its inception, nor had Clostridial enteritis been diagnosed. The TGE epidemic demanded rapid response to minimize the duration of the clinical signs.

It appears more than coincidental that clinical *C. perfringens* type A appeared as an epidemic after the entire breeding herd had been exposed to massive amounts of intestinal viscera containing TGE virus and other potentially pathogenic organisms concurrently existing therein. The management for TGE appears to have initiated the *C. perfringens* type-A infection. High doses of organisms routed to the piglets through their dams probably resulted in the clinical appearance of *C. perfringens* type-A infection. Prior to initiating the feedback protocol, the facility design and management practices in this herd had limited the access of the sows to fecal organisms.

Feedback of potentially contaminated materials to the swine breeding herd is a common practice to establish immunity against parvovirus, other enteroviruses, *Escherichia coli*, and TGE. This case illustrates the disadvantage of this practice: it encourages the establishment of opportunistic secondary infections in the herd. This is particularly true with enteric disease in sows housed with minimal oral access to infective organisms. If it had been *C. perfringens* type C that had become established in the herd, it would have had more disastrous results and would have involved more extensive and expensive control measures.[6] Porcine reproductive and respiratory syndrome (PRRS) can be spread within a herd by feedback;[7] other pathogens, such as *Erysipelothrix insidisia, Serpulina hyodysenteriae*, and *Salmonella*[8] present a threat when exposure is intentionally accelerated. In this herd, adequate immune response would probably have been achieved by feeding back thoracic viscera alone, given the affinity of the TGE virus for pulmonic tissue.[1]

Implications

- *Clostridium perfringens* type-A infection may appear in situations where the fecal-oral route of exposure to a source of the organisms is accelerated.
- Failure to consider the presence of nontarget pathogens in feedback materials can result in unexpected clinical disease.
- BMD® appears to be useful in the control of clinical *C. perfringens* type-A infection.

References

1. Saif LJ, Wesley RD. Transmissible gastroenteritis. In: Leman AD, Straw BE, Mengeling WL, D'Allaire S, Taylor DJ, eds. *Diseases of Swine*. 7th ed. Ames, Iowa: Iowa State University Press;1992:362–386.

2. Interdepartmental laboratory report. University of Nebraska Diagnostic Laboratory. 1994.

3. Torrison JL, Personal communication. 1994.

4. Schultz RA, Personal communication. 1994.

5. Harris DL. at: Executive Veterinary Program, University of Illinois, Module 11, May 12–14, 1993.

6. Taylor DJ, Bergeland ME. Clostridial infections. In: Leman AD, Straw BE, Mengeling WL, D'Allaire S, Taylor DJ, eds. *Diseases of Swine*. 7th Ed. Ames, Iowa: Iowa State University Press;1992: 454–460.

7. Christianson W, et al. Experimental reproduction of SIRS in pregnant sows. *American Association of Swine Practitioners Newsletter*. 1992; 4(4):24.

8. Alexander TJL, Harris DL. Methods of disease control. In: Leman AD, Straw BE, Mengeling WL, D'Allaire S, Taylor DJ, eds. *Diseases of Swine*. 7th Ed. Ames, Iowa: Iowa State University Press;1992:813–14.