CASE REPORT

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Unusual *Streptococcus suis* type 2 disease on two farms feeding incorrectly formulated nursery feed

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Summary

Pigs on two separate farms, ranging in age from 28–35 days, suffered an outbreak of *Streptococcus suis* serotype 2 disease. Aerobic bacterial culture from necropsied pigs yielded pure growth of *S. suis* serotype 2 from the lumen of the small intestine. Pigs on both farms were fed the same commercially milled and mixed ground corn/soy stage-2 postweaning feed, which contained a flavor-enhanced vitamin premix included at 58.5 g per kg of feed instead of the normal 3.5 g per kg. In addition, the salt normally added to the ration was inadvertently omitted. Affected pigs were found dead in their pens with no promontory central nervous system signs observed. The predominant clinical sign noted on both farms was a mild diarrhea in a small percentage of pigs. Necropsied pigs revealed gross intestinal lesions consistent with coliform enteritis. Despite treatment with spectinomycin in the drinking water at 6.7 mg per mL, sporadic deaths continued for several days until the owner of one farm discovered the feed formulation error. Misformulated feed was then removed from all farms and *S. suis* deaths abated.

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Modern feed manufacturing practices ensure quality products and services, and errors in formulating, balancing, grinding, or mixing rations that result in clinical disease are uncommon. When unintentional alterations in formulation occur, they can cause perplexing health problems that are difficult to diagnose. Animals from several farms using the same feed service may experience the same syndrome.

Case description

Farm One

Six nursery pigs were found dead without premonitory clinical signs. All pigs were about 35 days old and had been weaned for approximately 10 days. The dead pigs had recently been switched to a second-stage postweaning ration—a ground corn/soy ration that had been milled and delivered by a local feed mill. Approximately 10% of penmates displayed mild diarrhea. No other signs of disease were observed. Necropsies on all six pigs revealed congested intestines, swollen mesenteric lymph nodes, congested lungs, slightly enlarged spleens, and serosanguinous fluid and fibrin in the abdomen. Intestinal contents were clear and serous with the serosa notably congested.

A tentative diagnosis of coliform enteritis was made based on the enlarged mesenteric lymph nodes and congested intestines. Samples taken for bacterial culture included spleen, lung, liver, and swabs from the lumen of the intestine. Feed in the affected pens was removed from the feeders and these pens were placed on rolled oats. Spectinomycin water medication was instituted for the entire nursery at 6.7 mg per mL of drinking water. Aerobic in-house bacterial culture yielded pure growth of α-hemolytic streptococci from all organs. Bacterial cultures were sent to Iowa State University diagnostic laboratory for identification and serotyping.

Two days later, three more pigs from previously unaffected pens died; Pigs in these pens had been switched to the second-stage nursery ration 3 days earlier. Necropsies on these three pigs revealed similar postmortem lesions as the first pigs that died. The same culture samples, in addition to brain swabs, rendered the same pure streptococci colonies from the intestine and all other tissue samples. Intestinal sections were fixed in 10% formalin and sent to Oxford Diagnostic Laboratory for pathological examination.

Farm Two

The feed usage, history, and case presentation were similar to Farm One. Farm Two recorded acute deaths in which *S. suis* type 2 was recovered in pure culture from the intestinal lumen. As with Farm One, gross intestinal lesions that were consistent with coliform enteritis were observed, and deaths ceased after the feed was removed from affected pens. Unlike Farm One, however, previous diagnostic work on Farm Two indicated that the nursery pigs were positive for PRRSV.

Feed

Several days after the onset of *S. suis* deaths on Farm One, the owner of this farm noticed that the second-stage feed smelled and tasted sweet. Investigation at the mill revealed that the second-stage feed had been prepared with a flavor-enhanced vitamin premix at a concentration of 58.5 g per kg instead of the normal 3.5 g per kg. Upon this discovery, the misformulated feed was removed from all farms that had received this feed and was replaced with properly formulated feed. No other pigs from Farms One or Two died after switching to the correct feed formulation.

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Vitamin A and invert sugar levels were assayed in the suspect feed. The vitamin A level was 209,000 IU per kg and the invert sugars were 7.4%. Though the vitamin A and all other vitamin concentrations were high, they were not considered toxic.[1] Histopathological examination of the fixed intestinal samples revealed no major pathological changes. Some mild villus blunting was noted. There was no evidence of bacterial colonization or parasitism.[2]

Neither Farm One nor Two utilized computerized records. However, both nurseries were managed using all-in–all-out technology, making it simple to calculate quick estimates of mortality. Owner estimates of nursery mortality on Farm One was 5%, while mortality on Farm Two approached 10%. No objective measurements are available for changes in average daily gain or feed efficiency. However, both owners report a subjective impression that pigs improved feed consumption and growth rates once the misformulated feed was replaced with properly formulated feed.

Discussion

Outbreaks of *S. suis* in young pigs have been recognized as early as 1951 and were manifested as septicemia, arthritis, and meningitis.[3] Later reports link *S. suis* serotype 2 with meningitis, arthritis, polyserositis, valvular endocarditis, and pneumonia.[4] Additionally *S. suis* has been associated with rhinitis, vaginitis, and abortions.[5]

As many as 29 serotypes of *S. suis* exist, with even more serotypes that are as-yet untypeable.[6] Serotypes 2, 4, 3, and 7, in the order of prevalence, are the most common in midwest neurological cases.[7]

Recent evidence has demonstrated that *S. suis* is carried into the brain within infected monocytes. This explains the predominance of clinical signs associated with meningitis.[8] Other virulence factors that contribute to pathogenicity are high molecular weight muramidase-released proteins (MRPs) from the cell wall. MRPs have been associated with virulent strains of *S. suis* type 2 and are absent from nonpathogenic strains.[9]

The literature describes *S. suis* involvement in many organs. However, there have been few reports of *S. suis* being recovered from the intestine. We speculate that the unusual recovery of pure *S. suis* from the intestine was associated with the inadvertent feed formulation error. *Streptococcus suis* is harbored in the tonsil and this close association with the digestive tract might have allowed for the rapid growth of this organism in the intestine, given the unusually high sugar concentrations and abnormally low salt concentrations.

Implications

- This unusual case reports an uncommon presentation of a familiar pathogen.
- Abnormal circumstances may produce aberrant disease presentations.
- Thorough diagnostic investigations (including feed analysis) are necessary when unusual findings present themselves.
- Our observations in this case may suggest a heretofore-unrecognized aspect of *Streptococcus suis* pathogenicity.

References

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