Clinical Salmonellosis Related to Contaminated Feedstuffs in a Large Swine Production System

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Abstract

Thirty-seven groups of pigs of various weights (30–110 kg) in a commercial swine finishing system experienced clinical signs consistent with acute salmonellosis. Clinical signs included diarrhea, lethargy, pyrexia, and death. Gross lesions included hepatosplenomegaly, severe necrohemorrhagic enteritis, serosal hyperemia, and focal areas of colitis. High numbers of *Salmonella typhimurium var. Copenhagen* were isolated from small intestinal sections and feces. A combination of necropsies, diagnostic bacteriology, and a feed mill audit was subsequently used to help determine the nature of the relationship between the various sites included in the outbreak.

Presumptive evidence from diagnostic accessing and the feed mill investigation implicated feed as the most likely source of infection and all deliveries from the mill in question were immediately halted. Specific epidemiologic information about the clinical outbreak and its estimated costs are described.

Introduction

*Salmonella* spp. have long been known to occur in animal feedstuffs, particularly those ingredients derived from animal origin.1,2 Multiple serotypes have been documented, only rarely including types associated with disease in swine.3 For these reasons, while it is reasonable to expect feed contamination to be included as a risk factor in porcine salmonellosis, it is uncommon to find reports that document a clear relationship between the two.

In the described outbreak, diarrhea was identified concurrently in multiple swine confinement finishers belonging to a large commercial production company. All pigs included in these outbreaks were from sow herds and nurseries owned or managed by the same company but represented several different genotypes, multiple sexes, and farms of origin. At the initial outbreak, *Salmonella* enteritis with evidence of systemic involvement was confirmed through bacterial isolation and complementary histopathology. Additional finisher sites receiving feed from the supplier to the initial outbreak site also demonstrated similar clinical signs and were confirmed to have salmonellosis. Other pathogens including transmissible gastroenteritis virus, pseudorabies virus, and *Lawsonia intracellularis* were included as possible causative agents in the outbreaks but were not identified by the laboratory. An investigation was initiated to determine the nature, extent, and causal factors between the outbreaks. Historically, clinical salmonellosis had never been confirmed during previous routine disease investigations in the company’s different production phases.

Materials and Methods

A veterinarian investigated the first clinical episode within 24 hours of initial clinical signs. The site consisted of four, unconnected, 1200 pig-finishing buildings. Each building was fully confined, had concrete-slated floors, and contained 48 pens of approximately 25 pigs each. Pigs arrived in the facility weighing approximately 25 kg. Each barn was completely emptied, washed, and disinfected before being repopulated. Feed and water was delivered to each pen through a combination wet-dry feeder placed in the fence line between two adjacent pens. Pigs housed in a barn were within seven days of the same age; the mean pig age for each successive barn lagged the previous barn by about seven days. During the site visit, pig health was assessed and samples were collected for a diagnostic laboratory submission.

Four days after the initial outbreak, a second site, similar in design to the first, began to show similar clinical signs. Mortality of acutely affected pigs was much higher at this site than the first. During the next six weeks, seven additional sites exhibited similar symptoms and were visited by a veterinarian. Diagnostic samples were not submitted from all sites.

During the course of these continuing outbreaks, it became obvious that all sites were located in the same geographic area and were being supplied with pig feed from the same mill. Pigs on these sites were of various genotypes, gender, and had been born at several different farms but had been under ownership or management of the same company since birth. Building construction and animal management were similar across sites. The animal caretakers were different at most sites. The common link that was identified between the sites was the source of the pig feed. A single supplier was manufacturing the feed for all affected groups under a contractual agreement that limited their supply to only designated sites within the company’s production system. Feed produced at this plant was pelleted. Some of this pelleted feed, as well as some of the ingredients, was sampled during the outbreak period. When the link between feed source and clinical outbreaks was recognized, further deliveries from the plant were discontinued.
Results

At the initial investigation, pigs in the most severely affected barn weighed approximately 115 kg. Forty to fifty percent of pigs had greenish-yellow colored diarrheic feces. Affected pigs showed varying degrees of lethargy, pyrexia, dehydration, recumbency, cyanosis, and dyspnea. Three affected pigs were euthanized and had gross lesions including hepatosplenomegaly, necrohemorrhagic enteritis, intestinal serosal hyperemia, focal colitis, and pneumonia. Tissue and fecal samples were collected and delivered to the veterinary diagnostic laboratory at Iowa State University, Ames, IA, USA for bacterial culture and histopathologic examination. Bacterial cultures from all three pigs yielded growth of Salmonella typhimurium var. Copenhagen. Histologic lesions included focal suppurative bronchopneumonia, diffuse congestive hepatitis, and necrosuppurative pseudomembranous enteritis. No Lawsonia or spirochaetal organisms were identified.

At the second site that was investigated, diagnostic evaluation of submitted tissues again revealed S. typhimurium var. Copenhagen with histopathology similar to pigs from the first site. A sample of complete feed was also submitted for Salmonella culture but was negative.

A total of nine sites were identified as having similar outbreaks to those described. Salmonella spp. were recovered from seven of the sites with only some of the isolates being serotyped. Only one serotype was recovered from these sites, S. typhimurium var. Copenhagen. At one site, S. typhimurium var. Copenhagen was recovered concurrently from affected pigs and from the feed (collected from the feed bin) they were consuming. When the link between affected groups and feed origin became evident, discussions with the feed mill management were initiated to further understand the potential for Salmonella contamination of the feed. An independent audit of mill operation was conducted in addition to bacteriologic testing of feed ingredients and manufacturing equipment. Full results of these procedures were not made available but it was learned that several Salmonella strains were isolated including S. canstatt (Group E), S. mbondaka, S. kentucky, and S. montevideo from meat and bone meal, fish meal, and from mill equipment. The mill audit identified inadequate operation of the pellet cooler, environmental moisture accumulation in several areas, cross-contamination between feed ingredients and manufactured feed, and areas of ingredient accumulation on the sides of storage bins as problems that could potentiate the risk of Salmonella contamination in feed manufactured in the mill.

This mill began feed deliveries to the described sites two weeks prior to the initial outbreak. In total, 56 groups of pigs were exposed to feed produced at the mill in question during the eight weeks of the outbreak. Thirty-seven of the 56 groups exhibited clinical signs consistent with salmonellosis. Fecal cultures were completed on seven of the 37 groups and all yielded growth of Salmonella spp. By comparison, only 10 of the 369 groups owned by the company during this time period, not receiving feed from this mill, exhibited any unexplained gastrointestinal disease. While these 10 groups were not pursued to specifically diagnose salmonellosis, their reduced morbidity and severity (compared to confirmed outbreaks), and lack of suggestive gross lesions made it unlikely that they were related to the documented outbreaks. A group of this company's pigs receiving feed manufactured at the mill in question were 24 times as likely to show clinical signs of diarrhea than those using feed from a different mill (relative risk = 24.38). If feed manufactured at this mill continued to be utilized, an additional 63% incidence of diarrhea would be expected in the population of pigs (attributable risk = 63.36%). (Table 1)

Table 1: Frequency Table of Feed Exposure and Clinical Signs (number of groups)

<table>
<thead>
<tr>
<th>Presence of clinical signs (diarrhea)</th>
<th>Y</th>
<th>N</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposed to feed from investigated mill</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Y</td>
<td>37</td>
<td>19</td>
<td>56</td>
</tr>
<tr>
<td>N</td>
<td>10</td>
<td>359</td>
<td>369</td>
</tr>
<tr>
<td>Totals</td>
<td>47</td>
<td>378</td>
<td>425</td>
</tr>
</tbody>
</table>

All Salmonella isolates recovered from outbreak groups were found to be sensitive to neomycin and cefiofur based on Kirby-Bauer in vitro antibiotic susceptibility testing. As a result, any groups of pigs in the risk group exhibiting diarrhea were treated with oral neomycin and intermittent use of parenteral cefiofur. These procedures resulted in satisfactory resolution of clinical signs but did not completely eliminate a persistent recurring diarrhea in 0.5 to 1.0% of the pigs in a barn.

Discussion

The described series of Salmonella enteritis outbreaks provides good evidence that pelleted feed produced by a commercial feed manufacturer can be a significant risk factor in the occurrence of salmonellosis in large scale pig production. Five events outline the relationship in this case:

1. There was no evidence of clinical salmonellosis in the production system prior to the receipt of feed from the suspect feed mill.
2. Multiple, confirmed, concurrent outbreaks of salmonellosis were identified at sites utilizing feed from this mill.
3. Concurrent isolation of identical serotypes from feed and pigs was documented.
4. There was no other significant risk factors (age, pig source, genotype, sex, animal caretaker) common to the sites that could explain the outbreaks.

5. No new outbreaks of salmonellosis were identified after the feed source was discontinued.

It is difficult to establish the exact cost of this outbreak but certain costs can be estimated. Most of the costs were associated with antibiotic treatment, reduced average daily gain, and an increase in the number of substandard pigs. Mortality was not a significant factor with the exception of two groups, which added approximately 3% to their total death loss. Additional costs included those associated with quarantines the company placed on the affected sites (restrictions on people and truck movements on and off the farm, more extensive cleaning and disinfection procedures after a barn was emptied). Table 2 outlines some of these cost estimates.

The economic cost of this outbreak was estimated to range from $2.15 to $3.35 (depending on mortality) per head at each affected site. The total cost of the outbreak including mortality attributed to the outbreak was estimated to be $91,575. This total includes the costs associated with all 37 affected sites (41,921 pigs).

When clinical outbreaks began occurring, it took several weeks to establish a clear link to the feed source. It is disappointing that this could not have been identified more quickly but a common challenge in large production systems is differentiating a true change in health status from the inevitable coincidental "outbreaks" that frequently occur. The clinical presentation of this Salmonella outbreak is unique in the fact that the serotypes that were isolated are not typically associated with systemic disease; they are generally regarded as simple enteric pathogens. The evidence of septicemia and possibly bacteremia that was described in the pathology reports suggested that the pigs involved in the outbreak must have been exclusively sensitive to Salmonella infections. This could be related to their naivete to Salmonella spp in general (as evidenced by the lack of previous isolations) or a combination of unknown genetic, environmental, or nutritional factors.

The veterinary practitioner (and pig producer) typically approaches disease diagnosis from the perspective of the pig and the impact of the pig's health on profitability of the production unit. That is to say, "ill" pigs are assumed to grow slower, convert feed into meat less efficiently, and die at a higher rate than "healthy" pigs. This approach lends itself to the development of a differential diagnosis list for a particular set of clinical signs that includes all the relevant animal pathogens. What this approach does not lend itself to is the inclusion of pathogens that have limited relevance to animal disease but have significant implications in foodborne diseases. A mentality must be developed that forces those in animal agriculture to include food pathogens on the list of differential diagnoses so that the impact of pig disease on human health is considered when developing a diagnostic or therapeutic plan.

In the long term, a system for producing Salmonella-free feed will be required by producers and consumers of pork. In the mean time, reducing the use of animal proteins, certifying good manufacturing processes of feed and ingredient suppliers, and establishing internal routine health monitoring programs will result in faster reaction to problems.

References


<table>
<thead>
<tr>
<th>Parameter</th>
<th>Effect</th>
<th>Cost per head in affected groups ($U.S.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average daily gain (grams)</td>
<td>Reduced by 45</td>
<td>0.60</td>
</tr>
<tr>
<td>Antibiotic therapy</td>
<td>Combination of mass medication and individual treatments</td>
<td>0.30</td>
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<tr>
<td>Substandard pigs</td>
<td>Increased by 2%</td>
<td>1.00</td>
</tr>
<tr>
<td>Mortality (when applicable)</td>
<td>Increased by 3%</td>
<td>up to 1.20</td>
</tr>
<tr>
<td>Quarantine and biosecurity costs</td>
<td>Various</td>
<td>0.25</td>
</tr>
<tr>
<td>TOTAL COST PER HEAD ON AFFECTED SITES (depending on mortality)</td>
<td></td>
<td>2.15 to 3.35</td>
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