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G85-747 Enteric Diseases (Scours) of Swine

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This NebGuide describes four major enteric diseases of swine, and discusses the cause, clinical signs, differential diagnosis, treatment, and control of each.

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Four of the most important infectious enteric diseases of swine are reviewed in this publication. These include colibacillosis, transmissible gastroenteritis (TGE), rotavirus, and enterotoxemia (clostridial enteritis). A fifth disease, swine dysentery, is discussed in NebGuide G85-748.

Colibacillosis

Cause

Colibacillosis is caused by enterotoxigenic (toxin producing) *Escherichia coli* (ETEC) strains. In addition to producing toxins, these ETEC strains have the ability to adhere to the wall of the small intestine by means of "hair-like" structures known as pili on the surface of the bacteria. Several types of pili have been identified in ETEC strains producing colibacillosis in swine; the three major ones have been designated as K88, K99, and 987P. The combined presence of pili and enterotoxins enables these pathogenic *E. coli* to adhere and multiply in very large numbers on the surface of the small intestine, and to secrete the enterotoxins that cause severe digestive alterations leading to clinical diarrhea, dehydration and high mortality rates.

Clinical Signs

Colibacillosis can strike three age groups of pigs: 1- to 4-day-old, 3-week-old, and newly weaned pigs around 4 to 5 weeks of age. In 1- to 4-day old piglets, the disease has a very high incidence and mortality. There is a rapid onset of signs that include listlessness and diarrhea (scours), followed by dehydration and emaciation, and a rough haircoat. The tail and the skin around the anus becomes wet and pasted with fecal material. The base of the tail may become reddened. Death often occurs 12 to 24

hours after the onset of diarrhea. Mortality can reach 70 percent in affected litters, but usually not all of the litters in a farrowing group are affected. Litters from gilts are more frequently affected than litters from sows. Colibacillosis in 3-week-old piglets is similar to that in very young pigs, but of less severity both in incidence and mortality. Post-weaning colibacillosis in swine is the least severe form and death loss is greatly reduced.

Differential Diagnosis

A definite differential diagnosis of colibacillosis requires laboratory confirmation of the presence of ETEC strains in the feces or intestine of diarrheic piglets. In many respects, colibacillosis and transmissible gastroenteritis (TGE) are similar. However, there are some disease patterns that can help in the clinical differentiation of these two diseases. Colibacillosis often has a shorter incubation period (12 to 24 hours) than TGE (24 to 48 hours). It spreads slowly and does not affect all litters, while TGE tends to spread very rapidly, affecting most of the litters in a short time. The mortality in colibacillosis varies from 5 to 70 percent, while in TGE it approaches 100 percent in piglets less than 7 days old. Sows or older pigs are not affected in cases of colibacillosis, but they are in acute outbreaks of TGE. A presumptive diagnosis of colibacillosis can be made based on the pH of the diarrheic feces. For this, moisten a strip of pH test paper with feces at the anus (not in postmortem intestinal contents). A pH of 8.0 or higher is highly suggestive of colibacillosis. In viral and protozoal diarrheas the pH is 7.0 or lower.

Treatment

1. Injectable or oral antibiotics or sulfonamides.
2. Injectable fluids to replace the water, electrolytes and other nutrients lost as a result of diarrhea.
3. Injections of iron dextran to prevent anemia and increase the pig's resistance.
4. Oral dosage of cultures of *Lactobacillus acidophilus* milk.
5. Increase the farrowing house temperature.

Control and Prevention

Control of colibacillosis is best accomplished by management practices designed to reduce the problem. The following practices are suggested:

1. Try to get the newborn pig from the birth canal to the sow's teat for an early feeding of colostrum while reducing its exposure to disease-producing *E. coli* bacteria. This requires good sanitation, a clean sow, and a clean and disinfected farrowing house.
2. Keep pigs warm, clean, and dry.
3. Prevent anemia and increase the pig's resistance by injecting iron dextran or other injectable iron products.
4. Vaccines containing pili from ETEC strains are now available to immunize pregnant gilts and sows. These gilts' and sows' colostrum and milk will contain antibodies to protect their nursing piglets from ETEC.

Transmissible Gastroenteritis (TGE)

Cause

TGE is caused by a virus classified in the coronavirus group. The disease was first described in 1946. Since then, this disease has been reported in most of the major swine-producing countries in the world.

Clinical Signs

TGE is generally first observed in growing and finishing pigs on the farm with some vomiting and diarrhea, but the disease generally appears to be fairly mild. Following infection of older pigs, TGE next appears explosively in the farrowing house, with most of the baby pigs becoming ill within a 24- to 48-hour period. First clinical signs of TGE in the baby pigs are a roughening of the haircoat, shivering, vomiting, refusal to nurse, and extreme thirst. The baby pigs attempt to drink water from any source, and squeal weakly when handled. This is followed closely by a severe, watery diarrhea that has a very putrid, characteristic odor. These pigs become dehydrated, weak, and die within 2 to 5 days. In acute TGE, the mortality rate for pigs under 7 days old will approach 100 percent. In older nursing piglets, the mortality rate is lower, but can be influenced by stress such as chilling, dampness, or secondary bacterial infections. In acute cases of TGE, when the lactating sows are susceptible, they too become infected and may vomit, develop severe diarrhea, go off feed, and cease lactating.

In weaned pigs, the number of pigs infected approaches 100 percent, and the pigs will show all the clinical signs of TGE that are evident in the nursing pigs. However, the mortality rate will be greatly reduced as the pigs are more mature, have more stored energy, and generally recover. Stress factors again may influence the morbidity and mortality. Older growing and finishing pigs, gilts and gestating sows, and other swine in the breeding herd can become infected and frequently will show inappetence (not eating) or only a few clinical signs of illness. There is no evidence that infection with TGE virus during the gestation period affects the pigs before birth.

TGE occurs more frequently during the winter months of December to April. However, in continuous farrowing operations TGE can occur at any season, and can affect each subsequent farrowing for several months. TGE virus has been found in the feces of pigs eight weeks after apparent recovery from the infection. TGE virus has been recovered from the lungs of market weight swine and from both the lungs and intestines of experimentally infected pigs up to 104 days post-inoculation. TGE virus has been found in the feces of starlings up to 32 hours after the starlings were fed the virus.

Differential Diagnosis

Diagnosis of TGE is by history, clinical signs, and postmortem examination. On postmortem the stomach is full of curdled milk that may be dry and firm. The villi (small finger-like projections) in the lower two-thirds of the small intestine become shortened and shrunken.

TGE must be differentiated from colibacillosis as described in the previous section. A diagnostic laboratory can determine the presence of TGE virus in the intestine of piglets killed at the onset of diarrhea. Samples taken after 24 hours of diarrhea may yield negative results.

Chronic or Enzootic TGE

Enzootic (endemic) TGE is a persistent form of the disease in which carrier pigs within the herd shed virus and infect susceptible pigs. Enzootic TGE is found in those herds with multiple or continuous farrowing so that susceptible pigs are available at all times. In these herds TGE has been reported to occur in pigs as early as 10 days of age, but 10 days old to weaning appears to be the most common age of infection.

Enzootic TGE has many of the clinical signs seen in acute TGE, but they are less severe in the older pigs. Consequently, it is much more difficult to differentiate between enzootic TGE, rotaviral and clostridial infections. Mild villous atrophy can be found in both rotaviral and enzootic TGE infections,

but can be distinguished with proper specimens by examining intestinal tissue for villous atrophy or by the FA (florescent antibody) technique. In some cases, dual infections with both TGE and rotavirus have been reported.

Treatment

When lactating sows are affected with TGE, their milk production is greatly reduced. Their young pigs also become infected and the resulting dehydration is compounded by the lack of fluid intake caused by the sow's reduced milk production. It is, therefore, recommended that all litters over one week of age be weaned and fed one of the following:

1. A gruel made from water and a prestarter that contains dried milk as one of the ingredients.
2. A good quality sow's milk replacer.
3. Lamb's milk replacer that contains 20 percent fat.

Research workers at Purdue University have suggested a replacement fluid therapy that, in their experience, has been about 80 percent successful in laboratory pigs. The fluid consists of sodium bicarbonate and dextrose, and is injected subcutaneously in 60 to 80 milliliter amounts twice daily for three or more days. Antibiotics and vitamin B-complex can also be added to the fluids. Sow's milk or milk replacer and drinking water should also be available. Temperature of the floor should be maintained at 90 to 95 degrees Fahrenheit.

Best results were obtained when treatment was started about 24 hours after the onset of diarrhea. If treatment is started too early, the pig's blood may become too alkaline from the sodium bicarbonate. The experimental pigs were three days old when treatment was started.

Control

Avoid exposure to TGE virus by rigid isolation of the swine herd from dogs, foxes, and starlings. Do not bring feeder pigs that may be carriers to the farm just before or during the farrowing season.

All swine producers should practice some of the rules used for the specific-pathogen-free (SPF) program. These include: 1) visitors are not allowed into the swine facility without clean boots and outer garments; 2) trucks and truckers are not allowed near the main breeding herd; 3) new additions to the swine herd should first be isolated for 60 days and then added to the herd before the breeding season starts; and 4) measures to control the movement of dogs, wild animals, and birds should be attempted.

In herds where acute TGE infection has resulted in the loss of pigs, the sows should be re-bred, as the following litter from these immune sows might be protected from acute TGE. When TGE infection occurs in a farrowing house during the farrowing season, feed pregnant sows that have 3 weeks or more prior to farrowing minced intestines from infected baby pigs that have just started having clinical signs (diarrhea). Do not use intestinal tissue with TGE virus from another farm as other infectious agents could be brought onto your farm. Sows infected with TGE three weeks or longer before farrowing are able to protect their pigs through antibodies in the milk. Pigs become susceptible a few hours after weaning.

Sows that are to farrow in less than 2 weeks should be farrowed in individual isolation houses or sold to market. A planned infection of this type is one means by which a large producer in a multiple farrowing unit can control a TGE infection with a minimum loss of time and expense.

Modified live virus TGE vaccines are available commercially. Consult with your practicing veterinarian about the advisability of starting a TGE vaccination program.

Rotavirus Diarrhea

Cause

Rotavirus is an important cause of diarrhea in swine, as well as in most young animals including humans. However, each animal species is affected by different rotaviruses.

Clinical Signs

Rotaviral infections in piglets have an incubation period between 2 and 4 days, depending on the virulence (ability to cause disease) of the virus strain, the age of piglets, the immune status of the sow, and the environmental and management conditions. In field outbreaks diarrhea can be observed in new-born piglets but is found more commonly in 2- to 6-week-old animals, toward either the end of the lactation period or a few days after weaning. Infected piglets lose their appetite and become depressed a few hours prior to the onset of diarrhea. Vomiting has been observed, but it does not appear to be as prominent a clinical sign as with TGE. Adult animals generally do not show any clinical signs of infection, although diarrhea has been observed in gilts.

Diarrhea can be quite severe, starting with watery to creamy feces and changing rapidly to profuse yellow to green liquid feces. Diarrhea can last for up to 10 days. Return to normal feces is gradual and may take 1 or 2 additional weeks after recovery from severe clinical signs. Dehydration is more evident in younger piglets, and in those with prolonged diarrheic periods. The number of pigs having rotavirus infections in a typical outbreak is generally higher than 80 percent, affecting the entire herd within a few days. Mortality can reach 20 percent of the infected animals, although it is often higher in younger piglets.

Differential Diagnosis

The diagnosis of rotaviral infections in swine presents some problems. Clinical signs of rotavirus infections and of TGE virus infections of piglets are very similar, especially in cases of enzootic TGE. In general, the villous atrophy produced by TGE is quite severe, but in enzootic TGE the mild degree of villous atrophy very closely resembles that of rotavirus infections, making it difficult to separate these two conditions on that basis alone. At present, the best tool available to diagnostic laboratories for detecting porcine rotavirus is the examination of fecal samples. Fecal samples must be collected as soon as diarrhea is first observed and examined by electron microscope for rotavirus particles.

Treatment

Fluid therapy, antibiotics, and vitamins as indicated for TGE are recommended.

Control

Good sanitation practices can control a great deal of the risk of infections with rotaviruses, as well as with the other viruses or bacteria. The immunity to rotavirus in swine follows the same pattern as that of TGE virus infections. Vaccines are available commercially for the immunization of pregnant gilts, sows, and nursing pigs.

Clostridium perfringens, Type C Enteritis

Clostridial enteritis is a form of scours that affects young pigs and has been reported worldwide. Other names for clostridial enteritis are enterotoxemia, hemorrhagic enteritis, and bloody scours. The disease usually affects piglets during the first week of life, but nursing pigs up to a month of age can sometimes be affected.

Cause

A bacterium, *Clostridium perfringens*, Type C.

Clinical Signs

Most cases occur during the first week of life. Diarrhea usually begins as a watery, yellow scours that may contain traces of blood. After a few hours the feces become bloody, and the pigs may die within a few hours to two days.

Diagnosis

Diagnosis is determined by history, clinical signs, postmortem examination and laboratory assistance in which the causative bacteria are identified.

Treatment

There is no effective treatment after clinical signs of clostridial enteritis appear.

Prevention

Injections of Type C antitoxin, given to the newborn pigs as soon after birth as possible.

This disease can be prevented, or the severity reduced in future farrowings, by giving the sows injections of Type C toxoid 10 weeks before farrowing and repeating the injection three weeks before farrowing.

It has been reported by veterinarians that vaccinating sows with *Clostridium perfringens*, Type C toxoid reduces losses from non-specific scours that are not characteristic of clostridial enteritis in that the feces are not bloody. This may indicate that there is a low virulence type of clostridial scours that also responds to preventive vaccination.

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