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Differential Diagnosis of Baby Pig Diarrhea

Leon A. Larson, BS, DVM*
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Colibacillosis, clostridial enterotoxemia, coccidiosis, transmissible gastroenteritis, and rotavirus are the most common causes of diarrhea in pigs from one day to three weeks of age. Diarrhea in neonatal pigs is one of the most important causes of economic loss for hog producers. In a 1985 National Hog Farmer survey, 51.8% of U.S. hog producers reported problems with baby pig scours, a 2.9% increase from 1970. In the same survey 22.5% of producers said neonatal diarrheas caused their greatest economic loss, twice that of any other problem named. 1

A 1984-85 survey by Iowa State University estimates that Iowa hog producers collectively lose $2.75 million a month in prevention, treatment, and death from neonatal scours. 2 Of this monthly cost, $887,000 lies in prevention costs alone.

Many disease conditions of neonatal diarrhea clinically appear very similar, and often multiple etiological agents are present. For example, a producer may be trying to prevent clostridial enteritis when the actual problem is coccidiosis. Or, he/she may be properly treating one disease, but because of the second infectious agent, no improvement results. The amount of money being spent to prevent neonatal diarrhea thus stresses the importance of a correct diagnosis.

A correct diagnosis begins with a good history and necropsy. A good history includes the number of affected litters, the number of pigs affected per litter, and the age of pigs affected. It also includes the status of the sows, gilts, and other animals on the premises. Age is very important because some agents can cause diarrhea a few hours after birth while others cannot initiate diarrhea before three or four days of age. It is also important to know if similar problems occurred in previous farrowings, and if so, the response to previous treatments.

The animal chosen for the necropsy is as important as the necropsy and the specimens collected. The best choice is an untreated, acutely affected pig that is euthanatized immediately before necropsy. More than one pig may be required for a proper diagnosis.

Before opening up the pig be sure to examine the skin and external orifices of the pig. Discoloration of the skin, fecal staining or a bloated abdomen can give important information for diagnosis. Also look at systems other than the G.I. tract, because other processes may be present along with the diarrhea.

Once the pig is open and the other systems are quickly examined, check the width of the gut wall. Thinning is often characteristic of viral enteritis, due to villous blunting. Thickening of the gut wall is characteristic of necrotic enteritis, such as coccidiosis, which produces a pseudomembrane. Also check for hemorrhage and areas of emphysema.

Next look at the mesentery and lacteals. If the pig has been nursing and is freshly dead, the lacteals should be filled with a milky white chyle. TGE and rotavirus infections cause villous destruction, causing a decrease in absorption. Necrotizing diseases like Clostridium perfringens type C may also destroy villi causing a decrease in chyle absorption. To better observe villous atrophy, remove a small section of affected small intestine and place it in a water-filled test tube. The convex surface of the tube will magnify the mucosal surface, and the hypotonic water will cause the villi to swell. To observe subtle changes, it may be necessary to compare the affected section with a normal section.

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Open several segments of bowel and examine the mucosa. Necrotic diseases like coccidiosis and clostridium often cause necrosis and sloughing of mucosa. In chronic cases a caseous pseudomembrane may be present.

Since most enteric diseases cannot be diagnosed by history and gross lesions alone, samples must be collected and preserved for laboratory testing. For histological examination, collect 1 cm sections of ileum, jejunum and duodenum, preferably from grossly affected areas, and preserve in 10% formalin. Sections for bacteriology can be taken at this time. Remove six to 12 inch segments of duodenum, jejunum and ileum and place in clear containers. Impression smears should also be taken before any more mucosal sloughing occurs. The impressions should be air dried and stained at a later time.

Feces or intestinal contents may be collected. Fecal pH varies with disease process present. A quick pH check with litmus paper can help distinguish viral enteritis (acidic pH) from colibacillosis (alkaline pH). Electron microscopy for TGE and ELISA test for rotavirus can also be attempted with feces.

**Colibacillosis**

Colibacillosis, white scours, watery scours and baby pig scours are a few of the many names for *Escherichia coli* associated diarrhea. Colibacillosis is the most common cause of enteric disease in neonatal swine and one of the most common causes of morbidity and mortality of unweaned and post-weaning pigs.

Strains of *E. coli* producing enterotoxins cause intestinal hypersecretion. Villous atrophy is minimal and there is little decrease in absorption in the small intestine, hence the feces have an alkaline pH.

*E. coli* associated diarrhea can occur in pigs from two hours old up through the post-weaning period, but is most common in pigs one to four days old. Mortality is greatest in pigs less than four days old, with death commonly 12-24 hours after the onset of diarrhea. In pigs older than seven days, morbidity and mortality are much lower. All or part of a litter can be affected, and there is a slow sporadic spread through the farrowing house.

Grossly, the intestinal wall is of normal thickness, but may be distended with gas or fluid. The lacteals should contain chyle, and the mesenteric lymph nodes may be swollen. The mucosal surface appears normal with minimal villous atrophy and no areas of necrosis. An impression smear of the mucosa may show a large population of gram-negative rods.

A presumptive diagnosis is based on history, clinical signs, alkaline feces, normal villi and the presence of chyle in the lacteals. Isolation of a large population of *E. coli* from a rectal swab of an acutely affected live pig, or swabs taken from the jejunum or ileum of a freshly killed pig offers a definitive diagnosis.

Differentials for colibacillosis are TGE, rotavirus and starvation. Pigs that die of starvation are often diagnosed incorrectly as having colibacillosis. Starvation is usually associated with an empty stomach and thin, gas filled intestine. Meconium is present in the colon of starved pigs.

Since *E. coli* are part of the normal enteric flora, isolation alone cannot be used as basis for a definitive diagnosis of enteropathogenic *E. coli* scours. Final diagnosis is often an educated opinion based on clinical signs, purity and numbers of *E. coli* isolated, histopathology, response to specific antibiotic treatments, and lack of other demonstrable etiologic agents.

**Clostridium Perfringens** Type C

Clostridial enterotoxemia in its acute form is a highly fatal necrohemorrhagic diarrhea that is endemic in many hog operations of the Midwest. There are many forms of the disease, ranging from acute death to chronically infected, unthrifty pigs through the lactation period. The wide range of clinical presentations can make diagnosis of clostridial enteritis difficult.

Clostridial-associated diarrhea is seen in pigs 1-21 days of age, generally in pigs less than ten days old. It is often a persistent, recurring problem once established in a herd. Dose and virulence of the organism and the quantity and quality of maternal antibodies determines the severity of the disease. Consequently, each litter is affected differently and even littermates can be affected differently.

The peracute form of clostridial enteritis is often manifested as sudden death. Many times a healthy looking pig is found dead. If clinical signs are present, the affected pig will appear listless and a hemorrhagic diarrhea may be present. Affected pigs usually die within 12 hours after clinical signs. Necropsy reveals dark red and fluid filled sections of jejunum, perhaps with blood tinged peritoneal fluid. Impression smears demonstrate large populations of gram-positive rods.

In the acute form, affected pigs survive 2-3 days and have reddish brown scours that contain flecks.
of gray necrotic tissue. Affected jejunal sections are often well demarcated grossly by necrosis and emphysema. Hemorrhage is less noticeable and the lumen is filled with necrotic material often with a yellow/gray pseudomembrane present. Fibrinous adhesions may be present between necrotic segments and healthy segments of intestine. Villous atrophy may be detected if the mucosa is closely examined.

Pigs with the subacute form have a persistent non-hemorrhagic diarrhea which can last 5-6 days before the piglets die.10 Affected pigs appear alert and have a fair appetite, but they become emaciated. Feces are soft, yellow and often contain flecks of necrotic material. Affected intestine is thickened and friable, and often described as “ropey.” The serosal surface may have grayish-yellow longitudinal bands given the intestinal segments a “tiger-striped” appearance. A tightly adherent, gray pseudomembrane is present on the mucosal surface.3,6

Chronic clostridial infections may not cause diarrhea. Affected pigs just appear to waste away. If present, the diarrhea can be intermittent and last for more than one week. The scours can be yellow-gray to mucoid. The intestines may appear normal from the serosal surface, but when the mucosal surface is examined a tightly adherent gray, necrotic membrane is found. The entire small intestine may need to be examined because affected segments may only be 1-2 cm in length.5

Clinical signs and gross lesions allow accurate field diagnosis of the peracute and acute forms. The segmental necrohemorrhagic jejunitis is easily recognized and nearly pathognomonic. Impression smears provide additional evidence by demonstrating high populations of gram-positive rods. Diagnosis of subacute and chronic forms is more difficult because of similarity to the necrotic jejunitis of coccidiosis. Histological examination may reveal mucosal necrosis without coccidial organisms. Tests for TGE, colibacillosis and rotavirus infections may be necessary to rule out concurrent or multiple infections. Isolation of *Clostridium perfringens* on anaerobic BAP is significant, because the organism is rarely present in numbers high enough to be isolated without causing disease.6

If the chronic form is present, diagnosis is difficult. Isolation of the organism is unlikely because few organisms remain. Intestinal tissue is so badly damaged that histopathology reveals the nonspecific lesion of a necrotic enteritis. Diagnosis of clostridial infections in the chronic case is best approached by necropsy of a more acute-ly affected pig.

### Coccidiosis

*Isospora suis* infection has been described in veterinary journals for almost 50 years, but is just recently being recognized as a significant enteric problem in neonatal swine. Diagnosis is hampered because clinical signs are similar to clostridial enterotoxemia and because it is often seen with secondary colibacillosis or TGE.5,6,11,12

Coccidiosis is often a very predictable disease once established in a herd. Diarrhea and poor doing pigs begin to appear at approximately the same age every farrowing, usually at 7-10 days of age.

Coccidiosis is an enteric disease associated with an infection by the obligate parasite *Isospora suis*. The other coccidial agents in swine, the *Eimeria* spp., are not recognized as pathogens in neonatal pigs because of colostral protection. *Isospora suis* is not usually found in adult swine, therefore no colostral protection is transferred. Clinical disease results from the large infective doses of coccidial oocysts that accumulate in the neonatal pigs’ environment. Recent studies suggest that sows may not be the source of oocysts. Infected piglets from previous farrowings may be the source, as oocysts can survive up to 15 months in the environment of a farrowing house. Therefore, if the house is not properly cleaned between farrowings the oocyst population grows until enough oocysts are present that clinical signs can be seen in the pigs.11,13

Lesions of coccidiosis occur when the developmental stages of the coccidia destroy intestinal epithelial cells, with subsequent necrosis and villous atrophy. The first of these developmental stages are released between 3 and 5 days post infection. This means that even if the piglet ingests the oocysts right at birth, clinical signs of infection cannot be seen before 3 days of age and usually not before 5 days of age. Clinical disease is most commonly seen between 7 and 14 days of age, and may be seen in pigs up to 3 weeks old.

The disease occurs consistently, at 7-10 days of age as a chronic herd problem but does not consistently affect all litters or all pigs in a litter. Pigs appear listless with yellow watery scours progressing to yellow pasty scours over a 3-5 day period. The feces rarely contain blood, unlike coccidial infections in other species.12 Some pigs may not have diarrhea but just appear as poor doers with rough haircoats and poor appetites. If the villous damage is mild or the disease is uncomplicated, the diarrhea will run its course in 5-8 days.

*Iowa State University Veterinarian*
Gross lesions range from dull granular mucosa to a severe fibrinonecrotic pseudomembrane. Recent studies have indicated that only 10-20% of infected pigs have a fibrinonecrotic pseudomembrane. When the pseudomembrane is present, the intestines may feel ropey and the serosal surface may have a 'tiger-striped' appearance. The lesions can be segmental, with only portions of the lower jejunum and ileum being involved. In the affected areas, chyle absorption will be variable depending on the amount of villous destruction and the extent of the necrosis.

Histologically, villous atrophy is present in the lower jejunum and ileum, and may be mild or severe depending on the number of oocysts ingested and the extent of secondary bacterial invasion. Numerous developmental forms may be found within the intestinal epithelial cells or free within the lumen. Coccidia are more likely to be found in the milder lesions.

Diagnosis of coccidiosis is based on clinical findings, gross lesions, histopathology, stained impression smears and aided by detection of oocysts in the feces. Clinical signs suggestive of coccidiosis include: 1) predictable outbreaks in previously healthy pigs at 5-14 days, 2) lack of response to treatment, 3) lack of response when sows are vaccinated prefarrow for E. coli scours, TGE and rotavirus, 4) and by variable incidence between litters or among the littermates.

Stained impression smears are very reliable for field diagnosis of coccidiosis. Examination of several pigs, if possible, is best because not all pigs will be positive. Since the disease can be segmental, several areas should be examined, especially areas showing mild gross lesions. To make the smear, open the intestinal segment and gently blot the mucosal surface with a paper towel to remove excess fluid and debris. Next press a glass slide firmly against the mucosa with a rotating movement and lift away. Allow the smears to air dry and then stain them with Wright's stain or modified Wright's, (Diff-Quick® Stain, Set-Harleco). Coccidia are recognized as crescent or comma-shaped organisms with magenta nuclei and dark blue cytoplasm. Larger, oblong forms with multiple circular nuclei can also be seen. Properly prepared, impression smears can be as reliable as histology for finding coccidial organisms.

Examination of feces for oocysts is unreliable. Only 50% of infected pigs shed oocysts and then only late in the course of the disease.

Because of the similarities in clinical signs and lesions, coccidial infections are often hard to differentiate from chronic Clostridium perfringens infections. [Clostridial enteritis usually affects the upper jejunum, while coccidiosis affects the lower jejunum and ileum. The pseudomembrane of Clostridium is often grayish while that of coccidiosis is a golden yellow.] Histology is often needed, and many times a number of pigs need to be examined before a definitive diagnosis can be made. The impression smear is also very helpful.

Transmissible Gastroenteritis, (TGE)

Transmissible gastroenteritis (TGE) is a highly contagious disease in pigs of all ages, with mortality near 100% in pigs less than two weeks old. Traditionally, it has been an acute disease that spreads rapidly through a herd, causing diarrhea and vomiting in hogs of all ages, then quickly disappears as herd immunity develops. With today's continuous farrowing operations, TGE is also recognized as an enzootic form that may affect pigs consistently in either farrowing house or nursery.

TGE causes villous epithelial destruction, which decreases surface area, intestinal enzymes, and the ability to absorb nutrients resulting in a malabsorption diarrhea. Unabsorbed lactose is converted to lactate by intestinal flora, causing gut contents to be acidic.

The clinical signs of TGE depend on the form of the disease. In the classical, epizootic form, the entire herd is susceptible and there is a rapid spreading of vomiting and diarrhea affecting all swine in the building or farm. Sows often are affected with diarrhea, vomiting and agalactia are common. The baby pigs may vomit and usually have foul-smelling yellow watery scours which often contains small curds of undigested milk. Piglets less than two weeks of age rapidly dehydrate and die. At necropsy the intestinal wall appears thin due to villous destruction. Due to villous atrophy in the lower jejunum and ileum the mucosa will have a dull granular appearance which is visible with the test tube test. The luminal contents are watery, have an acidic pH, and often contain curds of undigested milk and gas bubbles. The inability to absorb fat results in the lack of chyle in the mesenteric lacteals.

In the enzootic form the virus remains in the herd and sows are continually reexposed, so the sows are protected and show no clinical signs. The sows also pass colostral antibodies against the TGE virus to their nursing pigs, and the pigs do not scour until thecolostral protection wanes, which may range from 1 to 5 weeks. Since the
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*In all cases need fresh and formalin fixed sections of duodenum, jejunum, and ileum, plus cecal contents or feces

BI — Bacterial Isolation  IS — Impression Smear  HISTO — Histological Exam  FAT — Fluorescent Antibody Technique
VI — Virus Isolation  ELISA — Enzyme-linked Immunosorbent Assay  S.I. — Small Intestine  EM — Electron Microscopy
pigs do not scour until they are older, death loss from this form is less severe. Clinical signs and lesions in the baby pigs are similar but less severe than the epizootic form.

**Rotavirus**

Field diagnosis of epizootic TGE is based on clinical signs, history, and gross villous atrophy. For a definitive diagnosis, laboratory confirmation is needed. Samples required for submission include fresh and formalin-fixed fixed sections of the ileum, jejunum and duodenum. Histology on fixed samples is helpful when typical villous atrophy is present, but virus identification is needed for confirmation of TGE.

Many methods are used to identify the virus. Iowa State University Veterinary Diagnostic Laboratory, and most other laboratories, use a fluorescent antibody technique on fresh pieces of the jejunum and ileum. Virus isolation is also performed in a few laboratories. The virus can be isolated from infected epithelial cells or feces of infected pigs. Electron microscopy of intestinal contents, fresh or frozen, is being done by some laboratories.

Rotavirus-associated enteritis is a poorly defined disease. The virus is very prevalent, but its significance is not well understood. Pigs are commonly infected, but clinical signs are often absent in uncomplicated infections. It is usually an endemic problem. Sows usually pass maternal protection to their pigs, making it more of a post-weaning than neonatal problem.

Rotavirus infections are usually seen in pigs one to six weeks old, and are most significant at the time of weaning, irrespective of age. Effects on baby pigs are similar to TGE, but less severe. The diarrhea is watery to pasty and may contain flecks of undigested milk curd.

Rotavirus infections can be segmental, involving only short sections of intestine, so much of the gut may appear normal. Affected areas appear thin walled and chyle is absent in the lacteals. Villous atrophy is present, but to a lesser degree than with TGE, and may not be apparent grossly.

Diagnosis of rotavirus begins with history of diarrhea in pigs older than seven days. Histology shows a mild villous atrophy and a minimum amount of necrosis. For a definitive diagnosis, electron microscopy of feces or intestinal sections may demonstrate the virus, or immunofluorescence may be done on frozen sections or smears. Because of the segmental nature of the rotavirus infection the ISU Veterinary Diagnostic Laboratory uses an ELISA test on feces or lower gut contents.

In summary, for proper diagnosis of baby pig scour, fixed samples of duodenum, jejunum and ileum should be sent to the laboratory. Sections should be from affected areas, and fresh sections of these areas should also be submitted for bacteriology and virology along with feces or lower gut contents for ELISA tests.

Other less common causes of neonatal diarrheas have been reported. Of these, *Strongyloides ransomi* is a problem in the southern and southeastern United States. Pigs can be infected in utero or via colostrum, therefore they can be infected at or near birth. Clinically the pigs have an acute, creamy white diarrhea with rapid emaciation. Grossly there may be erythematous pustules and petechial hemorrhages on the sow or pigs’ skin from larval penetration, and larval migration through the lungs may produce local hemorrhage. Diagnosis can be made by identification of eggs in a fecal float. Impression smears or scrapings demonstrate the adult worm.

Other bacteria and viruses have been rare causes of baby pig scour. Swine dysentery (*Treponema hyodysenteriae*) has been diagnosed as a cause of diarrhea in pigs one week old.

Salmonellosis may be a causative agent on rare occasions. Enteroviruses, adenoviruses and coronaviruses other than TGE can cause neonatal scour, but their significance is not known at this time. Toxins and nutritional problems must also be considered in the cases that can not be explained by the five or six major neonatal diarrheal agents.

The intent of this paper was to differentiate the causative agent in most cases of neonatal scour. Clinical signs, necropsy findings, diagnostic techniques and samples needed for laboratory confirmation were briefly outlined for each of the diseases, and are outlined in Table 1. Procedures for collecting and preserving the necessary samples were also outlined. This paper does not discuss all causes of baby pig scour, but is a quick review of how to diagnose the five or six major causes.

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