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Post weaning diarrhea in swine

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Post weaning diarrhea in swine is a common recurring health problem confronting veterinarians and producers. The cause(s) may be straightforward or complex. Housing, environment, nutrition and infectious agents are commonly interacting contributors to post weaning diarrhea. These factors may also complicate the diagnosis, treatment and prevention of post weaning diarrhea. Standard laboratory diagnostic investigations are predominantly oriented to look for infectious agents. It is the responsibility of the veterinarian and producer to apply the diagnostic test results to the clinical picture in order to help explain the problem. Communication with the diagnostician is important in difficult cases in order to expand the normal diarrhea examination.

In order to identify the causative agent(s) involved in the diarrhea, the correct pigs need to be identified and the proper samples submitted. Before sampling, it is important to characterize the entire post weaning diarrhea syndrome, as multiple agents may be involved concurrently or over different time periods. Some histories may be straight forward clinical episodes of diarrhea with obvious sampling requirements. Other histories may be chronic/recurring problems requiring sampling of multiple pigs of different ages or multiple pigs over different time periods. The clinical history and sampling requirements may be complicated because the nursery population may contain:

- pigs with varied disease status – both active and resolved
- different aged pigs
- pigs from gilts and sows
- pigs with different maternal antibody levels
- pigs exposed to different levels of endemic nursery pathogens

Also, consider the pig you are being asked to look at as the "problem pig" and decide if it is an acute presentation or actually a pig in the subacute or chronic stage of disease. Both types of pigs may be important for identifying the agents involved and their progression/interaction. Thin pigs in poor body condition with muscle atrophy are most likely subacute to chronic infections. These pigs may not be currently affected by diarrhea but rather are the end product of prior enteritis. Do not interpret pigs without diarrhea to be unaffected by intestinal disease.

For most diarrhea examinations, specifically look for acutely affected pigs that have not had antibiotic treatment. These are the best pigs for defining an acute clinical episode. Gross lesions in post weaning enteritis are commonly non-definitive. That is, the pigs have diarrhea and few if any gross lesions to suggest a causative agent. Pigs on feed should almost always have colon contents; pigs with diarrhea may have liquid colon contents or empty colons. Identification of infectious agents is dependent on laboratory testing. There are several important considerations to remember when collecting specimens for testing. Viral, coccidial and bacterial disease may be segmental in the small intestine. This is especially true for viral and coccidial disease. There are some generalities that help keep sample collection straightforward and valuable if they are consistently applied. Viral and coccidial enteritis have the most consistent lesions in the lower half of the small intestine. Viral and coccidial enteritis can be properly assessed by collecting a section of mid-jejunum, distal jejunum/upper ileum and distal ileum. Bacterial colonization of the small intestine is most consistent in the lower half of the small intestine. One section of fresh ileum/jejunum is sufficient for bacterial culture. Always collect sections of intestine with gross lesions for histopathology and culture, but do not limit submissions to these grossly affected areas. Sections of obvious necrotic enteritis may be devoid of infectious agents and filled only with cellular debris. Adjacent, grossly unaffected areas may still contain microscopic evidence of the infectious agent. It is imperative to get intestine sections fixed in formalin and fresh sections promptly chilled. Intestinal flora and structure rapidly change post mortem, more so than any other tissue. Post mortem decomposition of intestine will hinder microscopic interpretation of lesions and may delay or prevent bacterial cultivation and viral identification. Spiral colon and mesenteric lymph nodes in post weaning diarrhea are most important for assessing Salmonella infections. Formalin fixed and unfixed sections of each should be submitted. Fresh colon should be submitted in a bag separate from small intestine. Liver is a valuable specimen for submission. Some post weaning diseases, such as those caused by *Salmonella* spp. and *Escherichia coli* are characterized by a

bacteremia or septicemia. In some instances with decomposed small intestine, pathogens can still be identified from the liver. Submit brain with all cases of post weaning diarrhea. Not all cases of CNS disease are due to F18 *E. coli*.

Pigs with a post weaning failure to thrive syndrome are likely to have suffered from a prior enteritis. Pigs with viral or coccidial intestinal damage don't become instantly thin. It may take days or weeks for the intestinal damage to become clinically evident as weight loss or slow growth. Sampling the intestine as previously described is also important for this type of clinical problem as it can identify prior intestinal damage and a need to look for younger pigs with clinical diarrhea. Some pigs enter the nursery with a "failure-to-thrive" syndrome and no history of diarrhea. Intestine from these pigs should also be evaluated. Intestinal damage in farrowing may not become evident until pigs are switched from a milk diet to a solid diet.

Identification of representative pigs and submission of the appropriate samples are the most important part of diagnosing post-weaning diarrhea.

Common causes of post weaning enteritis

Post-weaning E.coli

The *E. coli* bacteria most commonly associated with post weaning diarrhea are the enterotoxigenic *E. coli* with F4(K88) or F18 fimbriae. Recovery of *E. coli* from pigs post weaning is usually associated with episodes of diarrhea; however, the level of bacterial shedding may vary. There are also some instances when diarrhea may not correlate with bacterial shedding. F4(K88) *E. coli* infections are most likely to occur early in weaning. F18 *E. coli* infections are more common 10-14 days post weaning. Mixed infections of F4(K88) and F18 infections can occur within groups of pigs. F18 *E. coli* infections may be due to F18ab (Edema disease associated, shiga-like IIE toxin gene) and /or F18ac (shiga-like IIE toxin gene negative) *E. coli*. F18ac *E. coli* infections are clinically similar to K88 *E. coli*. Gross lesions of *E. coli* diarrhea may include liquid yellow to red intestinal and colon contents, empty colons and slightly enlarged mesenteric lymph nodes. Edema disease lesions may be limited to slightly swollen eyelids and pigs with a head tilt, paddling and circling. Classic lesions of peri-colonic and gastric edema are uncommon anymore in edema disease infections. Many pigs may not have diarrhea at the onset of CNS disease associated with edema disease. Chronic edema disease may start days or weeks after an intestinal infection with pigs characterized by poor growth, muscle atrophy and head tilt. Diagnosis of chronic Edema disease depends on identification of brain stem lesions and / or vascular lesions in the brain and intestine. Most post weaning *E.coli* infections can be diagnosed by bacterial culture of intestine or rectal swabs. Because more than one pilus type of *E.coli* infection can occur in a group of pigs, more than one isolate should be characterized by PCR after isolation.

Rotavirus

Post weaning rotavirus enteritis is a widespread and common cause of post weaning diarrhea as well as neonatal diarrhea. Approximately 20% of post weaning diarrheas involve rotavirus. Rota viruses are subdivided into different groups. Group A, B and C rotaviruses are associated with post weaning diarrhea in pigs. In one report, 38% of post weaning rotavirus infections were caused by group A, 24% by group C and 19% by group B. Pigs are infected with rotavirus by fecal oral transmission. Rotavirus can survive in dry feces, dust and manure.

Most intestinal damage occurs 1 to 3 days post infection. Rotavirus damages intestinal villi as well as having toxin-like like effects that decrease small intestinal digestion and absorption. Rotavirus infections also favor colonization by post weaning *E.coli*. Replacement or repair of damaged intestine may take 2 to 3 weeks. Severe villous damage may never be completely resolved resulting in slow growing or fall back pigs.

Gross lesions of rotavirus infection are characterized by thin walled intestine and liquid, gritty (undigested feed) colon contents. Rotavirus infection is diagnosed by histopathology, electrom microscopy, fecal ELISA or immunohistochemistry. Electron microscopy can identify but not differentiate all rotavirus groups. Fecal ELISA detects group A rotavirus. Immunohistochemical detection is dependent on the antibody type(s) used in the test.

Salmonella

Post weaning enterocolitis caused by *Salmonella* spp. affects pigs from weaning to 4-months-old. Infection may be acute and chronic. Affected pigs usually have watery yellow diarrhea with or without blood. *Salmonella* spp. infection can spread rapidly with in a pen in a matter of days. The initial diarrhea lasts 3-7 days and may recur 2 or 3 times. Most pigs recover and some may remain carriers for up to 5 months. *Salmonella typhimurium* and *S. cholerasuis* have the most significant clinical implications for morbidity, mortality and persistence. Non-*Salmonella typhimurium* or *S. cholerasuis* species are clinically important for the affected pigs but are clinically less severe. *Salmonella typhimurium* is characterized primarily by small and large intestinal lesions of necrotic enteritis, enlarged mesenteric lymph nodes and subsequent septicemia. Some *Salmonella typhimurium* infections can be characterized by a rapid onset of septicemia. *Salmonella cholerasuis* is typically characterized by an acute onset of septicemia with enteric lesions developing in pigs surviving the septicemia. *Salmonella* spp. can be isolated

from small and large intestine, mesenteric lymph nodes and liver. *Salmonella* spp. infections in a group of pigs are usually due to one type. Pigs with necrotic colitis should also be cultured for *Brachyspira* spp.

TGE

TGE has become a less common cause of post weaning diarrhea but still exists. TGE enteritis is more commonly seen in nursery pigs rather than in neonates. TGE infection in naïve pigs is usually characterized by watery diarrhea with high morbidity and low mortality. Endemic TGE may be characterized by only transient, mild diarrhea in pigs 3 weeks post weaning. TGE infection generally causes more severe intestinal damage compared to rotavirus. TGE fecal shedding may last approximately 2 weeks. TGE infection is most commonly diagnosed by immunohistochemistry or PCR. TGE infection in endemic herds can be very segmental and difficult to diagnose. TGE infections in naïve pigs result in extensive intestinal infection and are more easily diagnosed. PCR diagnosis of TGE infection has the advantage of antemortem sampling and potentially improved sensitivity compared to immunohistochemistry. TGE infection can be diagnosed by serologic testing with the TGE/PRCV differential ELISA; however, the test requires waiting 28 days post infection for antibody maturation.

Coccidia

Coccidiosis isn't limited to 7 to12-day-old pigs and has become a common cause of post weaning diarrhea. Diarrhea begins approximately 5 days after onset. Infections are limited to the small intestine and are diagnosed by histopathology or direct microscopic examination of intestinal contents. Gross lesions range from non-descript diarrhea to necrotic enteritis. Severe necrotic enteritis is a more common lesion in farrowing pigs. A distinction between post weaning coccidia infection and pigs coming into the nursery with resolving farrowing infections should be attempted for appropriate timing of treatment.

Post weaning diarrhea can be a very complex, multifactorial disease. Characterization of the clinical problem and the group of pigs affected prior to sampling will help minimize the time and effort required for diagnosis and effective treatment.