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Ileitis control: Still elusive?

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Ileitis continues to be the most significant enteric challenge that we face in North American swine in the finishing phase of swine production (50-270 lbs.) Ileitis is a common term to describe Porcine Proliferative Enteropathies (PPE), which is caused by *Lawsonia intracellularis (LI)*, an obligate, intracellular bacteria that has affinity for porcine intestinal epithelial cells. PPE is unique in the fact that is manifests itself in two distinct forms; a chronic form (PIA; Porcine Intestinal Adenomatosis) and a more acute form (PHE; Porcine Hemorrhagic Enteropathy). These two manifestations present themselves somewhat differently. PIA is usually associated with a gray, loose stool that may be associated with poor growth performance, but rarely any significant mortality. PHE is the more deadly form of Lawsonia infection and is what is seen when most producers describe what they are seeing as ileitis. PIA can be seen at any time during the finishing phase, but most commonly during the grower/early finisher phase. PHE is usually seen in late finishing pigs (near market weight) or in replacement gilts and is characterized by hemorrhagic diarrhea and acute death.[1]

Diagnostic approaches towards ileitis should always keep other pathogens that cause similar clinical signs in mind and not always assume that any loose stool in grow-finish pigs to be ileitis. Differentials should include *Brachyspira* infections (*B.hyodysenteriae*, *B pilosicoli)*, Salmonella spp., PCV2 enteritis, or even osmotic diarrhea caused by extremely hard water. *B.hyodysenteria,* which was once thought to be eliminated from modern 3-site production, has made a recent reappearance in herds across the US, particularly in the SE. PCV2 enteritis, I believe, was under-diagnosed up to the point where PCVAD became a significant clinical event a few years ago. All to often we had reports of loose stools in early-mid finishing that was unresponsive to antibiotics, that in hindsight, was PCV2 enteritis. Widespread application of commercial vaccines have reduced, but not eliminated, this phenomenon and must remain on a differential list. Gross necropsy lesions are variable, depending upon the form of PPE (PIA or PHE), but both center around a thickened ileum and proximal large intestine. The thickening results from the intracellular invasion of the organism in rapidly dividing cells, which in turn causes them to hyper-proliferate as immature enterocytes. Depending upon the thickness, the ridges and folds of the thickness can sometimes be observed, making it easy to elucidate that the ileum is indeed thickened. "Garden hose gut" is a term used to describe a case of severe thickening associated with chronic PE, but actually is not seen all that often. In more severe cases of PHE, black tarry stools are commonplace along with acute death of good looking finishing pigs or replacement gilts. Gross necropsy will reveal a thickened ileum and a blood clot in the lumen. This will help distinguish acute deaths from PHE from Hemorrhagic Bowel Syndrome (HBS) or intestinal torsions. The black, tarry material in the colon that is seen with PHE must be differentiated from a severe gastric ulcer, which can easily be elucidated from gross post-mortem examination of the stomach. Laboratory confirmation of ileitis on affected tissues is usually is done by IHC on affected tissues with the best sensitivity (87%) rather than H&E (37%) or Warthin-Starry Silver Stains (50%).[2] Ante mortem testing for LI infection has become a preferred method of screening for timing of infections. Shedding of LI can be directly detected in fecal material using PCR based assays or by indirect antibody assays (less common). Multiple reports have validated PCR assays with a specificity of near 100%, but sensitivity varies between 39-72%.[3] The other commonly used method of ante mortem diagnosis and screening is by IgG based serological assays. Indirect immunofluorescent antibody (IFA)and immunoperoxidase monolayer assay (IPMA) are serologic tests that detect LI specific serum IgG. Each has a sensitivity of approximately 90% and a specificity of approximately 100%.[4] Seroconversion on these assays occurs generally 2 weeks after infection and antibodies can persist for 3-13 weeks depending upon the type of infection (PHE or PIA) and the severity of the infection (presumed to be the log titer of *L.intracellularis*). Higher, long-lasting titers are detected in pigs affected with the PHE form of ileitis. Seropositivity in pigs with the PIA form is lower (1:30) and lasts for only a few weeks. Titers and windows of seropositivity for subclinically affected pigs may be even lower.[3] Serological surveys done in the US (in presumed primarily 3-site production models) would say that seroconversion peaks in pigs that are 18-24 weeks of age, but that could depend upon floor type, feed medication protocols and pig flow. A great demonstration of the population dynamics of LI infection was

demonstrated by Lasley et al (2008) where they established that there was significant differences in both timing and incidence of LI infection when looking at traditional 3 site production compared to wean-finish sites in a large system. Also interesting to note in that study was that they saw no difference in flooring type (full vs. partial slats) as previous reports had stated that there were differences.[5] One could presume that the stress of shipment and re-socialization upon arrival into the finishing barn was enough to exacerbate the shedding and infection with LI. We have seen outbreaks of PIA/PHE occur after stressful events such as topping or grading pigs out of a group at marketing time as well as feed outage events.

Treatment and control measures center around timed mass medication pulses (feed or water), vaccination with the commercially available attenuated *Lawsonia intracellularis* vaccine (Enterisol® Ileitis, Boehringer Ingleheim) or a combination of the two. Treatment of clinical cases of ileitis seemed to respond best to a combination of injectable antibiotics such as Tylan or Lincomycin to clinically affected animals in combination with a mass treatment of water soluble antibiotics to reduce the impact of infection in the rest of the population. The most common water soluble antibiotics to treat ileitis have been Tylan (250mg/gal or 1 jar: 3 gallons stock solution), Lincomycin (3.8 mg/lb body weight) or Tiamulin (30-60 ppm). We have seen some cases of PHE that became refractory to one of these choices if that drug had been used extensively in the past (feed medication), necessitating a switch to one of the other, lesser used options with good success. That is in no way to imply that resistance exists for LI to antibiotics used today, but it does reaffirm the need to continue to investigate that possibility. Feed grade pulses timed around LI exposure seem to do a consistent job in controlling clinical signs, but constant monitoring of the timing has to be observed. Many practitioners and production systems have utilized feeding carbadox (Mecadox®, Phibro Animal Health) in the late nursery/early finishing period with success in keeping pigs free from clinical signs of infection.

Enterisol Ileitis hit the market and for the first 18 months was a "light switch" product. Application of this vaccine, when done properly, eliminated PIA and PHE in pig flows where their clinical appearance was historically repeatable in every group of pigs. Anecdotal reports from practitioners and producers utilizing reduced dosing indicated success regardless of the final dose. Most of these reports were based upon the presumption that the vaccine strain was shed from vaccinated pigs and was viable when these pigs were introduced to negative controls and the controls were subsequently challenged without seeing clinical signs of PPE. This allowed for a reduction of all finishing feed medication, resulting in significant cost savings initially. As the manufacturer refined and improved the consistency of their vaccine production process, we started to observe clinical signs of PIA and PHE in some groups of pigs, but performance was still better than non-vaccinated, non-medicated controls. Even these clinical episodes, were usually devoid of any significant mortality and were rapidly resolved with water soluble treatment of antibiotics. The vaccine (at the recommended dose) continues to be a effective control measure against LI. The epidemiological tools described above can be used to establish timing of vaccination necessary to optimize clinical protection. It is generally accepted that vaccination must occur 7-8 weeks prior to seroconversion to be effective.

References

1. McOrist S, Gebhart CJ. Porcine proliferative enteropathies. In: Straw BE, D'Allaire S, Mengeling W, Taylor DJ, eds. *Diseases of Swine*. 8th ed. Ames,Iowa: Iowa State University Press; 1999:521–534.

2. Guedes RMC, Gebhart CJ, Winkelman NA, Mackie-Nuss RAC, Marsteller TA, Deen J. Comparison of different methods for diagnosis of porcine proliferative enteropathy. *Can J Vet Res.*2002;66:99–107.

3. Gebhart CJ. ***Lawsonia intracellularis***: Diagnostic update. *Proc AASV* San Diego, CA. 2008;39:493–498

4. Guedes R. Update on epidemiology and diagnosis of porcine proliferative enteropathy. *J Swine Health Prod.* 2004;12(3):134–138.

5. Lasley P, Main R, Pelger G. Effect of flooring-type and post-weaning production method on the timing of seroconversion to *Lawsonia intracellularis* in a multi-site production system. *Proc AASV* San Diego, CA. 2008;39:87–90