Porcine Epidemic Diarrhea, Diagnosis, and Elimination

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Etiologic agent
Porcine Epidemic Diarrhea virus (PEDV) is a member of the family *Coronaviridae*. It is a positive-sense, enveloped, single-stranded RNA virus. Different strains of PEDV exist with virulence dependent upon the spike (S) gene sequence.

Distribution
PEDV was first reported in the United States in 2013. It has also been reported in Hungary, Germany, China, Korea, and Japan. Severe outbreaks with high mortality are typically rare in Europe, but are recently reported to be common in Asia. China has seen a large increase in outbreaks since 2010 which has been attributed to the emerging of new strains. PEDV, to date, has not been detected in Central America or South America. There have not been recent studies on PEDV seroprevalence in North America or Europe.

Clinical Signs
Severity of disease is variable and dependent on the epidemiologic status of the herd. The primary, and often only, clinical signs are acute watery diarrhea and vomiting.

- Naïve herd: Vomiting, acute watery diarrhea, and loss of appetite in pigs of all ages within susceptible breeding herds/integrated premises. Morbidity approaches 100 percent.
    - Suckling pigs: watery diarrhea, dehydration, and metabolic acidosis with mortality typically between 50 and 80 percent.
    - Feeder and grower pigs: diarrhea, anorexia, depression with high morbidity, but low mortality (1-3 percent)
- Endemic herd: persistent diarrhea in recently weaned pigs.

Incubation Period
Experimentally, the incubation period has been demonstrated to be approximately 36 hours from inoculation until the appearance of clinical signs. When PEDV infected swine are introduced to a naïve premises, clinical signs typically appear within 4-5 days. Incubation period with PEDV is typically longer than with transmissible gastroenteritis. Virus is shed for 7-9 days.

Differential Diagnosis
Because of similar clinical presentation with transmissible gastroenteritis, laboratory testing is required for identification.

- Viral gastroenteritis – the PED virus is similar to, but antigenically distinct from Transmissible Gastroenteritis Virus (TGEV). Porcine rotavirus groups A and B are also major causes in viral enteric diseases of piglets with similar clinical presentation.
- Bacterial gastroenteritis – *Clostridium spp, E. coli, Salmonella spp, Brachyspira spp, Enterococcus durans, Lawsonia intracellularis*.
- Parasitic gastroenteritis – *Coccidia, Cryptosporidium, Nematodes*.

Diagnostic tools include PCR, immunohistochemistry (IHC) and histopathology. For diagnostic submissions fixed and fresh tissue samples are far better than fecal samples or swabs.

Transmission and Reservoir

PEDV is most commonly introduced via fecal-oral contact with infected swine, but may also be introduced to a naïve premises by contaminated equipment, fomites, or personnel. Infected pigs, manure, or any materials that carry manure may transfer the virus. Dirty boots, clothing, hands, equipment, or trucks can spread the disease. It is possible that PEDV can persist on a premises where consecutive litters are infected and do not have immunity after weaning.

Sanitation and biosecurity are the best means of prevention. Do not commingle sources or groups of pigs. Ensure facilities and transportation vehicles are thoroughly washed, disinfected, and dried before pigs enter. Do not take boots, clothing, or equipment between pig populations.

Pregnant females will require approximately three weeks to develop sufficient maternal antibodies to protect their litters from the PED virus. Piglets will need to ingest sufficient amounts of colostrum for the immunity to be protective. Neonatal piglet survival should begin to return to normal 3-4 weeks after the feedback and exposure process began. Vaccines have been tried but have historically been ineffective.

Elimination

Elimination of PED is expected to be similar to Transmissible Gastroenteritis Virus (TGEV). The key to success/failure of the elimination process is the quick and complete exposure of an entire population. The virus is easily spread using fecal material from clinically ill animals, intestinal tracts from infected neonatal pigs, or direct contact with infected/shedding animals. Elimination in a breeding herd generally uses immediate feedback of infected material with herd closure followed by sentinel testing. All replacement gilts necessary for a period of four to six months should be in the farm during the feedback exposure period. Sentinel animals may be used to determine if the virus has been eliminated prior to resumption of replacement flow (introduction of naïve animals).

The feedback material should consist of fecal material from infected/scouring animals and/or the intestinal tracts (viscera) from infected/scouring piglets. For maximum viral content, sacrifice the piglet within the first six hours of its clinical signs. Process the viscera through a garbage disposal to macerate thoroughly. Cold water may be used to extend/carry the feedback material. The virus is temperature sensitive – warm, hot, or chlorinated water should be avoided. Distribute the feedback material before or at the beginning of the feeding period. Freeze an amount of visceral material immediately so that re-exposure can occur if necessary.

Be aggressive and relentless with the feedback exposure. Clinical signs will appear 12 – 36 hours post-exposure. One should not assume every animal has received an infective dose. Examine individual sows at least once a day (twice a day is preferred) to identify those that are clinically ill. Diarrhea and/or vomiting will be the most obvious signs. For questionable animals, consider feed intake and take rectal temperatures. It is helpful to mark every sow that exhibits clinical signs to ascertain exposure. Any animal detected scouring, vomiting, or off feed is marked. For

the sow herd, mark the individual sow cards so caretakers can easily identify those animals which did NOT become ill. A bright colored marker is easily visible and highly recommended. Similarly, closely observe and record signs in each nursery, grower, or finisher pen.

Expect to record clinical signs in 90+ percent of the population or evaluate feedback and exposure. Collect serum from a statistical sampling of the non-clinical population two weeks post exposure and have the lab conduct ELISA for antibodies when the assay is available.

If some animals fail to show clinical signs within the first few days, repeat the exposure/feedback process. At some point, infective material may run short, so target those animals that have not already shown clinical signs.

In the nursery, grower or finisher, feedback (described above) may be used, but nose-to-nose contact and fecal/oral spread can quickly contaminate entire pens, rooms, and buildings. To expedite the process, mix pigs within pens or switch entire pens to place heavily shedding animals next to those clinically unaffected.

It may be important to change the flow of pigs post-weaning to ensure they are not re-contaminated after maternal immunity has declined below a protective level.

Elimination Procedure Summary and Timeline

1. Laboratory confirmation of diagnosis.
2. Day 1: Close herd and procure replacement gilts for the minimum of the next three months
3. Day 1 – 21: Expose the entire herd including replacements to intestinal contents from acutely scouring pigs with PED. Feedback should begin immediately and continue until either clinical signs are observed or no intestinal material from affected piglets are available.
4. After clinical signs have subsided begin a strict all in/all out movement of animals for farrowing. Clean, disinfect, and dry the rooms in between groups.
5. Thirty days after cessation of clinical signs place sentinel pigs from a negative source and preferably from the same source as your replacement gilts. Allow these animals to enter after a period of isolation. Collect blood for serum assay for antibodies to PED when the assay is available.
6. Observe sentinel animals daily for clinical signs of diarrhea and if diarrhea occurs euthanize and necropsy acutely affected pigs and submit tissues to the diagnostic laboratory. If the samples are PED positive review the management practices and reconsider additional exposure. Collect blood from sentinels monthly and assay for antibodies for PED.
7. If no clinical signs are observed in the sentinels for 30 days and serology indicates sentinels have not been infected, it is safe to assume the virus has been eliminated.

All of these steps may not be necessary but with this virus it is prudent to be conservative and to implement the discipline necessary after cessation of clinical signs virus is properly eliminated from the environment by mechanical removal and sanitation, and thus it is necessary for the all in/all out procedure for farrowing and growing pigs.

Precautions
Elimination will succeed with a disciplined program under the direction of a veterinarian. Feedback is not without risk. It is possible that additional pathogens could be transmitted via the process. Careful discussion in herds that are PRRS active need to be conducted before feedback is initiated.

Vaccines
There are no commercial PED vaccines approved for use in the United States. Current commercial PED vaccines produced in Southeast Asia do not induce effective lactogenic immunity to the Chinese like PEDV. Immunity can be induced via feedback of intestinal contents and feces to sows and replacement gilts; however, immunity is not lifelong.

References

Becton, Lisa. TGE: hard lessons learned. AASV 2001. 365-367.

Carpenter, Jane; Templeton, Catherine. Evaluation of a transmissible gastroenteritis virus eradication program in a breeding stock supply herd. Swine Health and Production, 1996, Volume 4, Number 5.

Geiger, Jerome O., Christianson, William T. Whole Herd Exposure – A Review of Techniques for TGE and PRRS Control.

Harris DL, et al. Eradication of Transmissible Gastroenteritis virus without depopulation. Proc AASP: 1987. 18:557-561

Montenegro, MM, personal communications,
Song D. and Park B., Porcine Epidemic Diarrhea Virus, a comprehensive review of molecular epidemiology, diagnosis and vaccines, Virus Genes, (2012) 44:167-175

TGE Recovery/Elimination Procedures

USDA. Case Definition for Porcine Epidemic Diarrhea. May 21, 2013.

Wiseman BS, Harris DL, Curran BJ, Elimination of transmissible gastroenteritis virus from a herd affected with the enzootic form of the disease - Proc AASP, 1988, 19:145-149.

Zimmerman, J., editor, Diseases of Swine, 10th edition, pp 514-517.