PORCINE EPIDEMIC DIARRHOEA

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Porcine Epidemic Diarrhoea (PED) is an acute viral disease of the intestine of the pig. The disease was first diagnosed in Great Britain in 1971 and has been reported in most European countries and in Asia. The first reported occurrence of PED in this hemisphere was for confirmed cases in USA on May 16th, 2013. Since then the virus has spread to 30 states of the USA and to Canada, Colombia, Mexico and in the Caribbean, in the Dominican Republic. In early 2014, another related virus, Porcine Delta Coronavirus (PDCoV), was discovered in the USA.

PED is normally divided into 2 forms. Type 1 causes diarrhoea in pigs from weaning onwards only, while Type 2 causes diarrhoea in neonatal /nursing piglets as well. Type 2 can present in a very similar manner to Transmissible Gastroenteritis (TGE) but without the mortality characteristic of the latter disease other than losses in nursing pigs, either through dehydration or loss of milk from an affected mother.

PED is of great economic importance and can result in major production losses and rapid spread in a naïve pig population. Although it has not been seen in Jamaica the proximity of the outbreaks and the similarity to TGE (which is reportable) make this disease noteworthy for veterinarians and the pig industry alike.

Aetiology and Epidemiology

The porcine epidemic diarrhoea virus (PEDV) is not related to any other member of the Coronaviridae which includes TGE virus, Porcine Respiratory Coronavirus (PRCV) and Porcine Haemagglutinating Encephalomyelitis (PHEV) and thus will not offer cross-protection against the diseases caused by these agents. Pigs are the only known susceptible species, but thus far antibodies to the virus have not been found in wild pigs.

After introduction into a susceptible breeding herd, disease is followed by a strong immunity within two to three weeks. Colostral immunity then protects the subsequently-born piglets. The virus is eliminated spontaneously from small breeding herds but tends to be maintained in finishing farms due to the repeated introduction and subsequent infection of susceptible pigs. Outbreaks in the USA caused 60%–100% mortality in suckling pigs and mild to severe diarrhoea in all pigs of all ages.

The pathogenesis and immune mechanisms of PED are similar to those reported for TGE. Oral infection results in viral replication in the epithelial cells of the small intestinal villi. Cells on colonic villi also become infected. No other tissue tropisms have been shown.

The virus is shed in the faeces for up to three to four weeks. Transmission is thus faecal-oral. Contamination routes are mainly directly through infected pigs and indirectly through virus-contaminated fomites including feed and via transport trucks.

Clinical Findings

Diarrhoea is the only direct virus-induced clinical sign, although vomiting occurs frequently. Acute disease occurs where the virus is introduced into a susceptible population for the first time and resembles a TGE outbreak. In such cases up to 100% of sows may be affected, showing a mild to very watery diarrhoea often greenish in colour. Two clinical pictures are recognized: PED Type 1 only affects growing pigs where as PED Type 2 affects all ages including sucking pigs and mature sows. The incubation period is approximately 2 days and the disease episode lasts for 7 to 14 days. In suckling pigs the disease can be mild or severe with mortalities up to 100%. Mortality of ~90% in pigs 8-21 days of age was noted in the USA. A markedly increased number of acute deaths may be seen, particularly in pigs infected toward the end of the finishing period and in stress-sensitive breeds. Death may even occur during the incubation period. Generally sows and older growing pigs show various signs which tend to be milder than those in nursery pigs.

For endemic disease in large breeding herds, particularly if kept extensively, not all the females may become infected the first time round so there may be recrudescence. This will only occur in piglets suckling from sows with no maternal antibodies and is therefore sporadic.

Older pigs are more lethargic and depressed with PED than with TGE with sick pigs appearing to have colic.

Lesions

Macroscopic lesions are confined to the small intestine, with villous shortening as the main characteristic. These lesions closely resemble those seen with TGE. No lesions have been described in the colon. A consistent finding is acute necrosis of back muscle.

Diagnosis

Diagnosis is based on history, clinical signs and examination of faecal samples for evidence of PEDV by ELISA tests or electron microscopy. Post-mortem examination of dead pigs and laboratory tests on the small intestine may be necessary to confirm the diagnosis.

Clinical differentiation from TGE is difficult and as such, laboratory tests are required. TGE in its typical epidemic form causes rapidly spreading diarrhoea in animals of all ages, with high mortality in neonates. With PED, the diarrhoea spreads at a slower rate, and although diarrhoea is seen in most of the litters, some litters may remain healthy even in the absence of immunity. Morbidity is 100% in older pigs, and they are severely sick. Acute deaths in adults and finishing pigs due to muscle necrosis and that occurs during an outbreak of diarrhoea are typical of PED and are not seen with any other infectious diarrhoea.

Laboratory diagnosis in neonates is made by PCR and/or direct immunofluorescence on cryostat sections of small intestine or colon. ELISA to detect viral antigens in faeces or intestinal contents is more useful for older pigs. Antibodies can be detected in paired serum samples through ELISA-blocking.

Sampling

Sow herd: collect faeces from piglets or sows acutely affected with diarrhoea

Minimum: 3 pools of faeces for PCR. Each faecal pool is composed of faeces from 5 diarrheic pigs from different litters or locations. Optional pig tissues can be submitted, along with pools of faeces to the laboratory. Serum from at least 10 dams is highly recommended and live pigs can also be submitted.

For weaned (nursery, grower, finisher) pigs at least 2 oral fluid samples AND 3 pools of 5 loose/diarrheic faeces each should be submitted. Optionally representative pigs or pig tissues can be submitted to a diagnostic laboratory and serum from 10-15 pigs for serology is highly recommended.

Treatment

There is no specific treatment for the virus but often secondary bacterial infections may complicate the picture and these can be treated using broad spectrum antibiotics. Measures taken during an outbreak are of a general nature. Pigs with diarrhoea should isolated in a clean, dry, draft free environment with access to high quality drinking water (electrolytes may be beneficial). Finishing pigs should have feed withheld for 1–2 days.

Prevention and Control

Good biosecurity is a key to reduce the risk of introduction and reduce spread both within the primary production facility (farm) and externally. Cross contamination with any suspected pig's faeces has to be limited. A "Line of Separation" which marks the separation between your facility, transport vehicles or the outside /inside of production site. Veterinarians can help in assessing a farm's risk for disease and providing guidance in developing the farm's good agricultural practices including biosecurity, sanitation and waste disposal.

Biosecurity of transportation vehicles is important in PED. Vehicles should be clean, disinfected and dry. Several disinfectants have been demonstrated to effectively inactivate PEDV, such as phenolic, chlorine or iodine based disinfectants or peroxide, formalin, sodium carbonate, lipid solvents, and strong iodophors in phosphoric acid.

The disease may occasionally become endemic in finishing units as new weaners are introduced onto the farm. Under such circumstances it is necessary to break the cycle by stopping purchasing for three weeks or utilizing segregated disease control methods.

All-in all-out procedures with disinfection will often break the cycle.

PED virus can be eliminated from herds without total depopulation by:

- Maximizing immunity with planned infection of the sow herd – i.e. feeding sows faeces from infected pigs to produce an infection from which the sows will recover with strong immunity which is passed to their piglets passively.
- An "all-in/all-out" management of farrowing, nursery, and grower rooms; and good sanitation.

Because PED virus is easily spread during an epidemic by people, animals, and fomites, special care should be taken to prevent spread to unexposed groups of pigs and to neighbouring herds. Prompt removal and sanitary disposal of waste reduces the chance of exposure.

An Asian PED vaccine has been produced, however studies are in progress and several pharmaceutical companies are also reported to be working on a vaccine.

References:

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3. The Merck Veterinary Manual: accessed at http://www.merckmanuals.com/vet/digestive_system/intestinal_diseases_in_pigs/porcine_epidemic_diarrhea.html?qt=Porcine Epidemic Diarrhea&alt=sh
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6. USDA-APHIS Swine Enteric Coronavirus Diseases (SECD), including Porcine Epidemic Diarrhoea virus (PEDv)- http://www.aphis.usda.gov/wps/portal/banner/help?1dmy&urile=wcm%3apath%3a%2Faphis_content_library%2Fsa_our_focus%2Fsa_animal_health%2Fsa_animal_disease_information%2Fsa_swine_health%2Fct_ped_info

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1. Almanzar, Digega, 9th Meeting of the CaribVET Steering Committee, PED Situation in Dominican Republic

Links

1. CaribVET- Porcine Epidemic Diarrhoea (PED) first reported in the Caribbean! Link http://www.caribvet.net/en/breakingnews/alerts/porcine-epidemic-diarrhea-ped-first-reported-in-the-caribbean
2. CaribVET-link USDA- Technical Note – Porcine Epidemic Diarrhoea (PED) http://www.caribvet.net/en/system/files/ped_tech_note_usda.pdf