Post Weaning E. Coli Edema Disease

Introduction

Edema disease is an enteric disorder caused by E coli. Although considered an enteric disease, neurologic signs and multi-organ pathology are hallmarks of this disease. Disease is caused by pathogenic serogroups of E. coli that express F18 or F4 pilus types which are important for binding to F18 E. coli receptors on the intestinal epithelium. However, F18 E. coli intestinal receptors are not fully expressed in the pig until approximately 21 days of age and therefore E coli edema disease only occurs in post-weaned pigs. The source of infection in pigs is unclear but disease has occurred in new nurseries suggesting that farrowing room infections may be the source.

Clinical Signs and Lesions

The major clinical signs and appearance of lesions in E coli Edema Disease are highlighted in the table and figure (right). The onset of Edema Disease is usually 5 to 14 days post-weaning the clinical severity ranging from rapid death that usually in the largest and fastest growing pigs, to disease that lasts 7 to 14 days. The most common clinical signs include neurologic signs (loss of coordination, head tilt, convulsions, circling, and paddling), fluid accumulation or edema of peripheral tissues often seen as swelling or “puffiness” of the eyelids and forehead. Edema is also apparent in multiple tissues upon necropsy and is most pronounced in the greater curvature of the stomach and the colon mesentery (see Figure). Diarrhea is often accompanied by edema disease.

Pathogenesis

E coli bind to the intestinal epithelium and produce multiple enterotoxins that are responsible for clinical signs. Shiga-like toxin 2e (SLT2e) is the toxin responsible for edema and neurologic signs associated with this disease. SLT2e is absorbed across the gut, aided by a hyper-permeable intestine, and enters the circulation where it damages the lining of the blood vessels. Proteins and fluid leak out of the damaged blood vessels resulting in edema. Brain edema is the cause of neurologic signs. Clinical signs of diarrhea are dependent on the ability of specific F18 E coli to elaborate the enterotoxins Heat Stable toxins A (STa) and B (STb) and Heat Labile toxin (LT) which stimulate fluid secretion and impair absorption leading to clinical signs of diarrhea.

Diagnosis

Accurate diagnosis is based on proper tissue submission to a diagnostic lab. This includes live animal exhibiting clinical signs, or freshly collected ligated loops of small intestines submitted for culture being the specimens of choice. Submission of brain tissue is also important to rule out other neurologic diseases. Diagnostic tests generally include the isolation
of pure cultures of beta-hemolytic E. coli from small intestine, molecular detection of toxin and pilus genes, and histopa-
thology.

Table. Key features of E coli Edema Disease

- Occurs only in post-weaning period
- Major clinical signs:
  - Diarrhea
  - Neurologic signs
  - Rapid death
- Major post-mortem findings
  - Fluid accumulation (edema) in multiple tissues

Prevention/Treatment

Prevention is the most effective means of controlling this disease. Currently there are no vaccines or toxoids for E. coli edema disease in the U.S., however oral inoculation of weaned pigs with non-pathogenic strains of F18 E. coli (Stx2e-, LT-, Sta-, and STb- negative), to compete for binding sites with pathogenic strains, has shown to be effective. Once disease has occurred, treatment is often difficult and unsuccessful. Antimicrobial treatment can be effective at times however it can also be problematic with repetitive bouts of disease as antimicrobial resistance can develop. Other general enteric control strategies may include supplemental dietary Zn, water acidifiers (citric acid, organic acids, others), and Spray-Dried Porcine Plasma.

Summary

E coli edema disease continues to be a major post-weaning pig disease. Neurologic signs, peripheral edema, and diarrhea are the hallmarks for this disease. Proper sample submission to the diagnostic lab is necessary for definitive diagnosis of E coli edema disease and the subsequent design of control programs.

Suggested Readings

