Porcine Polyserositis

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Introduction

Porcine fibrinous polyserositis is a gross pathological diagnosis with many etiologies possible. The most common bacterial agents associated with this disease include *Streptococcus suis*, *Haemophilus parasuis* (Glasser's disease), *Mycoplasma hyorhinis*, *Mycoplasma hypopneumoniae*, *Mycoplasma hyosynoviae*, *Actinobacillus sp*, and *Pasteurella multocida*.[1,3,7] The pathogens above may cause respiratory, neurological, and synovial infections and/or a combination of these clinical signs.[1,3,7] These pathogens are all of bacterial origin and are known to be a part of the porcine respiratory disease complex (PRDC).[1] Some of the more common viral components to PRDC include porcine reproductive and respiratory syndrome (PRRS), swine influenza virus (SIV), and porcine respiratory circovirus type 2 (PRC).[1] Swine farms often vaccinate against these viral components; however, as with many vaccines they are not one hundred percent effective and some of the viral pathogens mutate rapidly.[1] This becomes an issue when large populations are commonly commingled in small confinements. The sharing of feed, water troughs, bunking, rapid weather changes, poor ventilation, and improper rodent and/or insect control can allow for ambient conditions for disease spread.[1,3,7] As the name implies PRDC is often caused by more than one etiologic agent and can be a mixture of both viral and bacterial components.[1]

History and Clinical Signs

On March 28, 2018 three mature (~4-6 months old) white market hogs were presented to Mississippi State University College of Veterinary Medicine (MSU-CVM) Laboratory Services for necropsy after death on March 27, 2018. The owner of a 7200 head commercial pig barn (21 pigs per unit) had a recent outbreak of approximately 200 head of pigs dying spontaneously over a 1 month timeframe. The animals were noted to display some signs of runny stool and anorexia

prior to death. The vaccination history is unknown. The pigs were maintained on a corn based diet with a city water source.

Necropsy Findings

The abdominal cavity contained a moderate to severe amount of fibrinous clear yellow transudate. There were fibrinous adhesions noted on all visceral organs and were attached to the abdominal wall. The gastrointestinal serosa was hyperemic. Cranial cervical lymph node was enlarged, soft pale, and yellow on cut surface. The esophagus contained yellow mucoid material with a roughened and irregular mucosal surface that progressed distally. The stomach contained an irregular mucosal area near the cardia (gastric ulceration). There were petechial hemorrhages noted on the mucosal surface of the duodenum. The liver margins contained hypostatic congestion. The right lobe of the liver was paler with a distinct centrilobular pattern on cut surface with multiple distinct pale yellow/tan areas and floated in formalin.

There were severe fibrinous adhesions present on the lungs attaching to the thoracic wall and diaphragm. The dorsal portion of the lungs was white to tan with the remainder being pink to dark pink/purple. The cranioventral region was whiter in appearance with diffuse focal areas of white to pink mottling present throughout the lung fields. On cut surface the lung was shiny and oozed blood and clear yellow tinted material and was soft and edematous, floating in formalin. There were fibrinous adhesion on the pericardium and underlying heart. The gross diagnosis included serofibrinous pleuritis, pericarditis, peritonitis and gastric ulceration.

Diagnostics

Necropsy on one individual animal is not necessarily correlated to the total herd infection status; however, it is a good diagnostic approach to reduce herd infectivity and/or treatment

plans. The most common approach for herd testing in a disease outbreak is to provide samples from 2 acute and 1 chronic animal case.[4] This typically will provide the diagnostician with enough sample specimens to determine the etiologic agent and if there are any secondary causes. This is usually performed by selecting the individual with clinical signs of the disease out of the herd and sacrificing them for the "herd's sake," so to speak.[4] A variety of samples can be obtained to ensure proper diagnosis. Pooling of samples in formalin is a common diagnostic approach when herd outbreaks occur. Viral isolation, fluorescent antibody test (FAT), immunohistochemistry (IHC), and polymerase chain reaction (PCR) are the tests more commonly used for identifying the cause of disease in association with clinical signs and gross typical lesions, whereas serology is an indirect method.[4]

The diagnosis for this hog was severe acute gastric ulceration of the cardia and fibrinous polyserositis, with *Streptococcus*.

Pathophysiology

Gastric Ulceration

Gastric ulcers are most commonly seen during necropsy or slaughter and found to be located at either the pars esophagea or cardiac mucosa.[7] The process of gastric ulceration begins with parakeratosis, erosions, and finally ulceration of the gastric mucosa. Some form of this condition is noted to occur in upwards of 90% of swine production systems depending on husbandry and feeding practices.[7] Most often hogs are noted to have acute death and further herd observation may include pale hogs within the herd.[7]

Gastric ulcers can be caused by stress/heat stress, non-steroidal anti-inflammatory drugs (NSAIDs), off feed events, improper feed particle size, blocked feeders/waterers, genetic

predilection, and presence of vomitoxin in feed.[7] Animals in high stress may release high levels of cortisol as a result may block the protective effect prostaglandins play on the glandular portion of the stomach via inhibiting the production of arachidonic acid. Although this is part of the inflammatory cascade research has shown ulceration of the pars esophagea is not mediated by glucocorticoids.[7] However, NSAIDs also inhibit the protective effect of prostaglandins on the stomach via directly blocking cyclooxygenase (COX). As such, use of NSAIDs during respiratory outbreaks to decrease inflammation and pyrexia may lead to increased risk for gastric ulceration; as well as, off feed events or inadequate feed present within the trough. All of which may cause a reduction in the pH present within the stomach. Low pH levels in association with increased histamine levels causing gastric acid secretion may lead to mucosal damage to the stomach and possible ulceration.[7]

Gastric ulceration in grower/finisher pigs can be caused by poor quality pellets (coefficient of variation of particle size causing increased feed variation), allowing out of feed events, and lack of aggressive feeder adjustment.[5,7] When you combine all three components can result in gastric ulceration.[5]

Swine with severe gastric ulceration may have black tarry feces, pale in appearance, or show signs of abdominal pain including arching back and grinding of teeth (bruxism).[7] If the ulceration is severe enough they may have complete pars esophagea destruction and stenosis of esophagus and will regurgitate after eating while continuing to eat shortly after the regurgitation event.[7]

Streptococcus suis

Streptococcus suis is a Gram positive cocci bacteria with approximately 35 different capsular serotypes and is considered a zoonotic agent.[2,3,7] The most common serotypes present within the United States are serotype 2, 3, 4, and 7.[2,3,7] The most common worldwide is *S. suis* serotype 2.[3] The prevalence varies on farm and worldwide.[3] Many farms may have one or more capsular serotypes present. *S. suis* is considered normal flora in the upper respiratory tract of swine.[2,3,7] In the event of an immunocompromised host or when environmental and/or managerial stressors occur the respiratory epithelium can become compromised.[3] This will allow for *S. suis* to invade the crypts of the palatine tonsils and penetrate the lymphatics to progress to the mandibular lymph nodes where they may reside or cause a septicemia.[3] Some of the organisms may live within phagocytes and travel to the CSF, brain, meninges, lungs, and joints.[3] The organism is capable of causing meningoencephalitis, pneumonia, arthritis, and/or polyserositis.[3] Clinical signs associated with *S. suis* are often associated with the age of the animal. Younger nursery pigs show short, acute clinical signs of septicemia including lateral recumbency and paddling associated with CNS disease. In older pigs such as grower/finishers the clinical signs may include CNS: ataxia, opisthotonus, incoordination, tremors, convulsions, blindness, deafness.[3] Other signs include polyarthritis, swollen joints, and lameness. *S. suis* is typically a secondary invader of the lungs and when pneumonia is diagnosed other etiological agents must be considered, as well.[3,7]

Prevention/Treatment

Gastric Ulceration

Prevention and control of gastric ulceration events may include ensuring well maintained cooling and ventilation systems. Seasonal changes and infectious processes will increase the prevalence of gastric ulceration within swine production systems. Swine are susceptible to overheating/heat stress and will decrease feed intake leading to less gastric fill. There is an association between high fluid content within the stomach being more associated with gastric ulceration versus firm stomach content and increased stomach fill to help prevent stomach ulceration.[7] During feed processing the feed particle size is important regarding how the grain is milled. If the procedure involves grinding of the grain (as with wheat or pelleting feeds) it will produce a finer particle size increasing ulceration risk and severity.[7] As such, barley and rolling oats seem to have a protective effect on the gastric mucosa.[7] Preventing out of feed events and aggressive feeder adjustment will decrease the prevalence of gastric ulceration.[5,7] Some suggested gastroprotectants in swine industry include increasing the levels of antioxidants such as, Vitamin E and Selenium beyond the NRC requirements has not been shown to be useful; however, it has been shown sunflower hulls will decrease the amount of gastric lesions noted.[7]

PRDC

There are many components involved in both prevention and treatment of PRDC. Ventilation is a key factor in all enclosed animal populations regardless of species. As such improper ventilation can lead to elevated ammonia levels which as a result can cause mechanical damage to the epithelium of the respiratory tract leading to secondary infections.[4] Overcrowding of housed animals can lead to poor ventilation along with increased stress, dust levels, and overheating which may lead to respiratory compromise.[1,3,7]

Introducing naïve animals into the herd via mixing of swine populations from multiple sources and age groups along with not utilizing all in all out approach method increases the risk

for PRDC. It is also important to mention herds with a specific negative disease status such as PRRS or Mycoplasma are more susceptible to infection if introduced into a group of disease exposed serology positive pigs.[4,7] As such, PRRS is the most common virus isolated in PRDC.

Vaccination against common PRDC pathogens is highly recommended however, does not guarantee prevention of disease or infection.[1] Some swine industry standards or protocols include Mycoplasma/Circovirus vaccine or implementing *Mycoplasma* free herds.[1,3] *Streptococcus* vaccines are available however due to the various amounts of *Streptococcal* capsular serotypes present within the United States it may be more efficacious to have an autogenous strain produced to control the most common strains within the producers region or in the case of a herd outbreak.[1,2,3,7] It is also important to note PRRS and/or SIV infected, positive, and/or exposed swine herds are more susceptible to *Streptococcus* and *Histophilus* infection.[3]

Treatment of *Streptococcal* infections is often tailored to herd treatment with antibiotics (such as, ampicillin) pulsed into the watering system for a few days and treating individual pigs that are showing severe clinical signs with injectable antibiotics (such as, enrofloxacin).[1,] Anti-inflammatory medications such as aspirin/ibuprofen are also placed into the watering system to help with the lung inflammation and treatment with suspect SIV.[1] Broad spectrum antibiotics are utilized to control secondary infections and/or unknown disease causes when the clinical signs suggest multiple etiologies are likely.[1,3,4,7]

Different methods are used to control diseases including: ensuring biosecurity, all in all out methods, same age group at initial barn loading, disease free herds, and proper disinfection between barn loadings is key to aid in prevention.[1] Some disinfectants that are commonly used and are effective against *Streptococcus* include phenols, quaternary ammoniums, formaldehyde, hypochlorite, chlorhexidine, and 3% iodine.[3] Other considerations including but not limited to

the pork quality assurance guidelines include proper rodent and insect control.[3] Flies are noted to carry *Streptococcus* along with other pathogens for a minimum of 5 days.[3,7] With various factors influencing the pig host, *Streptococcal* infections are challenging to prevent.

Expected Outcome and Prognosis

Severity of gastric ulceration will greatly affect the prognosis. Complete gastric perforation of the pars esophagea is not sustainable with life; however, other gastric ulcer lesions may only decrease overall production performance of the hog.

Streptococcus suis infection has a variable morbidity and mortality based on the serotype virulence and the herd exposed to the pathogen.[2,3] Herds with prior exposure to the *S. suis* will have protection against it.[2] If the herd is vaccinated and not protected from the strain present on the farm those pigs that are considered immunocompromised or PRRS, SIV positive will likely experience a greater morbidity and mortality.[2,3] Recognizing clinical signs and treatment in a timely manner may help in reducing the severity of disease.[1] Antibiotic sensitivity testing should be considered in a herd outbreak due to *Streptococcal* antibiotic resistance occurring, especially with tetracyclines.[1,4]

Conclusion

Multiple etiologic agents are responsible for causing fibrinous polyserositis.[1,3,7] It is important to recognize clinical signs associated with the disease including CNS, polyarthritis, and pneumonia.[1,3,7] Individual injectable treatment is most ideal for those showing clinical signs.[1] Proper ventilation, management practices, biosecurity, vaccination, and early recognition of clinical signs will assist in preventing excessive herd outbreaks.[1,3,4,7] Electively sacrificing

animals with acute and chronic clinical signs to perform necropsies and diagnostics including antimicrobial sensitivity is the most effective method for treatment and control.[1,4,6]

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