Diamonds and Pearl

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Introduction

Swine erysipelas is a multisystemic infectious disease commonly referred to as “diamond skin disease”. *Erysipelothrix rhusiopathiae* is a significant bacterial pathogen of swine, which has been recognized as zoonotic though human infection is rarely reported.[6] Susceptibility to erysipelas is not well understood and many apparently healthy pigs harbor *E. rhusiopathiae*.[5,6] Passive acquired immunity provides protection for 3 months of age. Subclinical exposure generally has a protective effect on pigs older than 3 years of age, therefore pigs in this age range rarely present with erysipelas.[5,7] Swine erysipelas results in septicemia, dermal vasculitis, endocarditis and polyarthritis; causing substantial economic loss in the swine industry worldwide when uncontrolled.[1,6] All stages of pork production may be affected. Infection with *E. rhusiopathiae* has a tremendous impact on growing and finisher pigs from sudden death due to acute septicemia or going lameness and arthritis for those that survive, resulting in poor growth and slaughterhouse condemnation.[7] Other swine dermatologic disease differentials such as Porcine Dermatitis Neuropathy Syndrome, acute Hog cholera, acute septicemia of *Salmonella chloerasuis*, Streptococcal infection and *Actinobacillus suis* must be considered.[5,6,7]

History and Presentation

Pearl was an approximately 6-year-old female Vietnamese Potbellied pig, who presented to Mississippi State University College of Veterinary Medicine (MSU-CVM) Food Animal Service on December 10, 2018. Prior to her arrival at MCU-CVM, on December 8, 2018 the rDVM, noted multiple to locally extensive areas of erythema and small areas of crusts along her ventrum and hind quarters. At the time she was hyperthermic with a temperature of 106℉. Pearl received enemas, for a previously diagnosed megacolon, and was started on the broad-spectrum

antibiotic Trimethoprim Sulfa (TMS). Due to progressions of the ulcerative skin lesions she was switched to Penicillin and referred to MSU-CVM for further evaluation. At MSU-CVM Pearl presented with a three-day history of fever, lethargy and anorexia. Upon presentation, Pearl had a red-purple rash along her entire ventrum and limbs, with ventral and facial edema. Pearl was hypothermic, with a temperature of 98.7 ℉. During attempts to place an intravenous catheter in the marginal ear vein, Pearl stopped breathing and died. Pearl then presented to MSU-CVM Laboratory Services for necropsy examination.

Pearl had a body condition score of 5/5. She was housed indoors, slept in the bed with the owner and there were two other pigs in the household. Pearl had been diagnosed in July 2017 with atresia ani and a recto-vaginal fistula resulting in ongoing constipation issues. There was no history of vaccination or deworming. She was being fed ½ cup twice a day of Mazuri elder formula feed.

Necropsy Findings

On necropsy examination there were multifocal red, small, rounded, ulcers present in the oral cavity. On the skin, along the ventrum were multifocal and widely disseminated, round, irregular red-black papules, with ulcerative crusts (20x35mm). The distribution of the skin lesions was along the inner thighs of the hindlimbs, ventral abdomen, inner area of the forelimbs and along the ventral neck. The interpretation of these lesions is a dermal vasculitis resulting in infarction and tissue necrosis.

Within the stomach mucosa are locally extensive bright red to light black and blue regions (1 x 2.5cm), and an out pouching in the cardia region of the stomach. Dark black ingesta (melena) was found throughout the small intestinal tract. The colon was markedly enlarged and

distended (megacolon 36cm in diameter). Hard-formed feces were present in the spiral colon and descending colon. The bladder wall was diffusely thickened with a wall thickness of 1cm. A normal uterus was not present and where the uterus should be located was a luminal, thick walled pouch that extended into the vaginal cavity. There was no external anal opening with the termination of the colon into the vaginal opening with marked narrowing. The lungs were diffusely firm, dark red, and had a mottled appearance, consistent with an interstitial pneumonia

Diagnostics – Culture Results

Aerobic culture of the mucoid bladder fluid grew *Psuedomonas aeruginosa, Enterobacter cloacae* and *Enterococcus faecalis*. Light growth of *Erysipelothrix spp.* Was isolated from samples of skin, confirming a diagnosis of *Erysipelothrix rhusiopathiae*. In cases of erysipelas additional culture sites include blood, joints, lung and liver.

For cases in which culture is not rewarding or an expedited result is desired, polymerase chain reaction (PCR) and immunohistochemistry assay (IHC) are available.[4]

Diagnostics - Histopathology

This histopathology of the skin was characterized by locally extensive epidermal necrosis. In areas of necrosis thee was loss of nuclear detail and hyalinization with lesser affected areas of epidermis contained large numbers of apoptotic cells and edema. Vessels in the underlying dermis and subcutaneous tissue were congested, lined by plump reactive endothelial cells and occluded by fibrin thrombin. In scattered vessels was expansion of the vessel wall with fibrin, necrotic debris, hemorrhage and small numbers or neutrophils. The skin histology

confirmed a dermal fibrinoid vasculitis resulting in infraction with epidermal necrosis. This change is consistent with *E. rhusiopathiae.*

Pathophysiology

Eryispelothrix rhusiopathiae is a ubiquitous gram-positive aerobic bacteria that is both commensal and pathogenic. The bacteria is introduced by ingestion of contaminated feed or water.[3,5,6] Environmental contamination occurs through excretion in the urine, feces, oronasal, or bodily secretions from carrier pigs.[6] The organism likely enters through the tonsils or gastrointestinal associated lymphoid tissue.[6] Approximately 30-50% of apparently healthy pigs, harbor E. *rhusiopathiae* in of their tonsillar tissue and potentially serve as a source of environmental contamination.[1,6] Passive acquired immunity provides protection for pigs 3 months of age. Subclinical exposure generally has a protective effect on pigs older than 3 years of age, therefore pigs in this age range rarely present with erysipelas.[7]

Erysipelas produces a neuraminidase, an enzyme that cleaves siliac acid from glycoproteins, glycolipids and polysaccharides in host cell walls which may mediate the widespread vascular damage. [5,7] Bacterial adherence to endothelial cells in the absence of platelet-activating factor, allows for vascular damage, leading to thrombosis and interference with microcirculation in capillaries and venules.[7] Endothelial damages results in thrombus formation and infarction.[5,7] Widespread ecchymotic hemorrhages are a result of microthrombi.[7]

Acute cases commonly present with sudden death, ill thrift, severely elevated temperature, painful joints, reluctance or inability to rise, and abortion in pregnant sows. Of acute cases present with classic cutaneous rhomboid urticaria coalescing over the ventrum, rump, back shoulders.[5,7] In acute forms pigs can present with significant respiratory distress due to

interstitial pneumonia.[7] Subacute forms presents with mild fever, infertility, mild skin lesions, lower mortality.[7] Chronic cases often have endocarditis and chronic joint infections characterized by , marked synovial villous proliferation and thickening of the joint capsule with possible progression to ankylosis[5,6,7] Pigs that have valvular lesions may be apparently healthy, until they are physically exerted and have signs ofrespiratory distress, lethargy, and cyanosis.[6]

Differentials

Important differentials to consider with a similar clinical presentations and cutaneous lesions are: *Actinobacillus suis*, acute Hog Cholera, acute septicemia from *Salmonella chloerasuis*, Porcine dermatitis neuropathy syndrome (PDNS) and Streptococcal infection.[5,7] Diagnosis of chronic cases are often challenging due to unsuccessful culture attempts.[5] *Actinobacillus suis* may cause septicemia, pneumonia, and sudden death with almost identical lesions to those of *Erysipelothrix rhusiopathiae*. *Salmonella choleraesuis* is also an important cause of septicemia and similar dermal lesions as those seen in erysipelothrix infection.[7] PDNS, which is associated with porcine circovirus type 2 infection, results in vasculitis secondary to a type III hypersensitivity reaction.[6,7]

Treatment and Management

Erysipelothrix rhusiopathiae is sensitive to penicillin, which is the treatment of choice, typically resulting in an adequate response within 24-36 hours. Twelve-hour treatment intervals, for a minimum of 3 days are ideal, however longer therapy duration may be required to resolve severe infections.[5,7] The organism is sensitive to the beta-lactam antimicrobial therapy such as, penicillin, ampicillin, or cephalosporins like ceftiofur, and tetracyclines.[6,7]

Penicillin is the best choice for antibiotic therapy from an economic standpoint, but ampicillin and ceftiofur may yield satisfactory results in acute cases.[6,7] Tetracyclines in feed and water, may be more practical in circumstances, where it is not functional to inject a large number of pigs.[5,6,7] Erysipelas antiserum is not commonly accessible in an acute outbreak situation but can be an effective adjunct to antibiotic therapy, until three days post-clinical resolution.[5,6] Non-steroidal anti-inflammatories, such as flunixin meglumine or aspirin in the water, may be of beneficial use in acutely febrile cases. Treatment of chronic infections is ineffective and not cost efficient.[5,6,7]

A diagnosis of acute erysipelas can be made through responsiveness to penicillin therapy.[6] Unfortunately Pearl was on TMS for a couple days, prior to being switched to penicillin, at which time severe septicemia resulted in a critical state.

Pre-breeding vaccination is advisable in sows, gilts and boars as it creates some maternal immunity. It is not recommended to vaccinate at the time of weaning due to maternal interference. It is advantageous to wait to until the pigs are into the nursery for vaccination protection to carry through to finisher pigs.

Prevention is most efficiently achieved through a combination of good sanitation protocol, vaccination programs, stress management, proper elimination and quarantine of carriers with lesions and new stock for best control.[5,6,7] Current vaccines of *E. rhusiopathiae* serotypes 1 or 2 are inactivated bacterins for intramuscular injection or attenuated vaccines for whole herds.[5,6,7] Vaccination programs are advisable on premises of previous outbreaks. Duration of immunity is 26 weeks, so two doses of most bacterins usually protect growers to market weight with boosters at the end of grower/finisher stage, if there is a history of repeated exposure to the organism.[5,7] Therefore breeding stock should receive boosters once or twice annually.[3,5]

There are food safety implications as *E. rhusiopathiae* can survive for several months in animal tissues, byproducts or dried blood, whether frozen, chilled, cured or smoked pork.[3] Erysipleas is resistant to alcohols, aldehydes, oxidizing agents and phenol disinfectants; and requires hypochlorites, caustic soda, or sodium hydroxide to be destroyed. [3,5,7]

Conclusion

Erysipelothrix rhusiopathiae is harbored by many apparently healthy pigs.[6,7] The reason why the bacteria become pathogenic is largely unknown, but host immune response likely plays a role.[5] Although Pearl was kept indoors with two other pigs, she had not been vaccinated and was in had an excessive body condition score of 5/5. Pearl's exposure to the *E. rhusiopathiae* is unknown but may have been introduced via a number of different environmental sources. The average lifespan for an overly conditioned pig, does not typically surpass Pearl's age. The challenge in Pearl's case was that she was treated initially with TMS, and not a penicillin. The rectovaginal fistula, ongoing cystitis and ineffective antimicrobial selection likely played a role in decreasing the host immune system. Pearl died due to the systemic effects of sepsis and presented with the classic skin lesions.

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