CASE REPORT

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Multiple *Streptococcus* species implicated in lameness and central nervous system signs in piglets and sows

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Summary

β-hemolytic streptococcus (*Streptococcus equisimilis*) was isolated from swollen joints and meninges of suckling and nursery pigs in a 360-sow, farrow–to–finish herd. Incidence of clinical outbreaks of lameness and central nervous system dysfunction in neonates and weaned pigs had increased in the herd after introduction of new breeding stock in 1993. Isolation of *S. suis* from some pigs further complicated the clinical picture. Clinical signs abated after autogenous vaccines were administered to pigs, but reappeared in piglets and sows during periods of stress over the next several years. Streptococcal-related disease often occurs in sporadic outbreaks that are believed to be triggered by stress factors and/or the occurrence of subpopulations of naïve animals.

Keywords: *Streptococcus suis*, *Streptococcus equisimilis*, biosecurity, vaccination protocols

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Streptococci are common endemic pathogens in pigs raised in confinement pork production units.[1,2] *Streptococcus suis* (Lancefield Group D *Streptococcus*) has been associated with septicemia, meningitis, polyserositis, endocarditis, and pneumonia in young pigs.[3,4] In addition, suppurative arthritis associated with Lancefield Group C *Streptococcus* spp. (usually *S. equisimilis*) has also been reported (Collier. *Proc AVMA*.1951:169). Results tabulated from five diagnostic laboratories in the United States suggest that Lancefield Group C *Streptococcus* organisms have been isolated in 25% of the porcine cases submitted.[5]

The antibody protection produced when animals are exposed to streptococcal organisms is not believed to be cross protective among the different hemolytic groups or between different subtypes within each Lancefield group.[5]

This case report describes a herd in which clinical signs associated with both α and β hemolytic *Streptococcus* organisms were observed. The α-hemolytic streptococcal isolates were identified as *S. suis* or *Enterococcus* spp., and the β-hemolytic isolates were identified as *S. equisimilis*.

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Herd history

A single-site, 360-sow, farrow-to-finish herd in Missouri introduced grandparent-breeding stock from a high-health, commercial breeding stock company in April 1993. Isolation facilities consisted of an adjacent pasture lot located 46 m (50 yards) from the existing herd. The producer decided to repopulate this farm based on a need to decrease the backfat and increase the percent lean of market hogs. The gestation barn provided individual sow housing for 50% of the gestating sows. Sows were confirmed pregnant by day 35 of gestation and moved to an outdoor gestation lot for 5 weeks.

In November 1993, approximately 8% of preweaned piglets and 12% of nursery pigs were observed coughing, and had swollen joints and reduced weight gain. Etiologic agents considered included *S. suis*, other streptococci, *Haemophilus parasuis*, *Mycoplasma hyosynoviae,* and other *Mycoplasma* organisms. Seventeen affected pigs from the farrowing house and nursery were necropsied. Gross pathological findings narrowed the differential diagnosis to probable streptococcal septicemia. *Streptococcus equisimilis* septicemia was diagnosed when that organism was isolated from lung, liver, and spleen of nursery pigs (isolations performed by Oxford [Bayer] Laboratories, Worthington, Minnesota).

The number of weaned pigs per litter and the adjusted 21-day weaning weights were compared by a paired t-test(*Statistix for Windows User's Guide*, version 1.0. Statistix. 1996) and found to be significantly ($P<.05$) reduced from November 1993–January 1994 compared to the preceding 3-month interval (Figure 1).

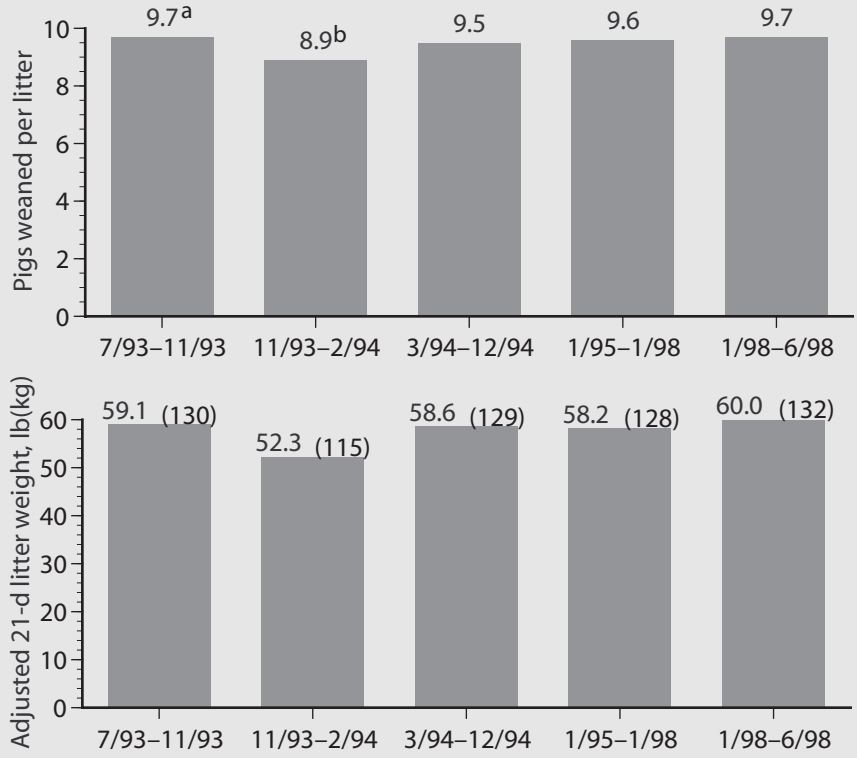
All pigs with swollen joints received 6600 IU per kg (3000 IU per lb) (1 mL per 10 lb) of procaine penicillin-G (PPG) intramuscularly (IM). Weaned pigs received water medicated with tiamulin (Denagard™, Fermenta [Novartis]) at the rate of 150 mg per head per day for 3–5 days postweaning to reduce bacterial colonization. A vaccination program was also initiated, in which breeding females were vaccinated 6 and 4 weeks prefarrowing with a 2-mL dose of a commercially prepared autogenous bacterin prepared from the specific on-farm isolate of *S. equisimilis*. Pigs also received 1 mL of the autogenous bacterin at weaning. Nursing pigs were vaccinated at 10 days of age and 3 days postweaning with a rhinitis and *S. suis* combination bacterin (CTSE, from Oxford [Bayer] Laboratories). Although this treatment program did not eliminate clinical signs, the incidence of joint lesions in the farrowing house and lameness and joint swelling of nursery pigs decreased to 1%.

There were no further clinical signs until January 1995, when *S. suis* was isolated from the brain, lung, and liver of four sows demonstrating head tilts and rear leg paralysis. It was determined that the gestating sows were being underfed. The clinical signs disappeared when proper nutrition was restored and affected sows were treated with 1500 IU of PPG per lb bodyweight once a day for 3 days.

Over the next 30 months, head tilts and periodic lameness were observed in piglets and nursery pigs during times of stress associated with seasonal change, improper feeding regimen, and labor turnover. Over this period, several different bacterins and treatment protocols were attempted with limited success. The management also implemented a complete herd health and biosecurity plan (Clark. *Proc Swine Dis Conf for Swine Pract*. Nov, 1995:69–73).

In February 1998, a complete slaughter check was conducted and tissues and swabs were obtained and submitted for histopathology and bacteriology. Eleven of 18 pigs showed pneumonic damage in 6%–10% of the lung tissue, and snout scores averaged between 1 and 2.[6] Alpha- and β-hemolytic streptococci were isolated consistently from snouts and acute pneumonic areas of lungs. Autogenous bacterins (MVP Laboratories, Nebraska) were manufactured using the herd-specific α– and β-hemolytic streptococci and administered to sows at 4 and 6 weeks before farrowing.

Figure 1



Production performance during *Streptococcus* outbreaks
[ab] values with different superscripts are significantly different ($P < .05$)

Discussion

Septicemic disease in swine associated with β-hemolytic streptococcus (*S. equisimilis*) usually appears within the first 2–3 weeks of life, and the clinical signs typically observed include increased temperatures, lameness, and joint swelling.[4] Infections result from contamination with vaginal secretions and milk of postparturient sows.[7] Previous investigators have observed macroscopic lesions in joints of non-vaccinated, challenged pigs, consisting of edema of periarticular tissues; swelling, edema, and hyperemia of synovial membranes; and increased amounts of seropurulent to fibrinopurulent synovial effusions.[8] Roberts, et al., concluded that septicemia is necessary for establishing infection in synovial and osseous tissues.[9] Insufficient consumption of colostrum or milk or inadequate levels of antibodies (as seen in gilts) may predispose to this disease.[10]

At the University of Missouri Veterinary Medical Diagnostic Laboratory, α-hemolytic streptococcus organisms (*S. suis*) were isolated from 78% of all cases submitted between January 1996–December 1997 when clinical signs suggested streptococcal problems. Twenty-two percent of the isolates in these cases were β-hemolytic streptococcus (n = 479) (Fales. *Proc Pork Academy*. NPPC. 1998:67–93).

On the case farm, the increased incidence of observed clinical signs associated with streptococcal septicemia corresponds with the introduction of new gilts into the breeding herd as well as environmental stresses. It is possible that the gilts may have introduced the organism into the herd, as β-hemolytic *Streptococcus* can be found in the vaginal tracts of gilts and sows.[1] It is unlikely that the organism was in the pasture, because β-hemolytic *Streptococcus* tends to die outside the body. However, the newly arrived gilts were probably exposed to cull sows being held in the pasture lots.

The vaccination program using autogenous bacterins made from herd-specific isolates was associated with marked reduction in clinical signs after the initial outbreak of streptococcal septicemia in 1993. Woods and Ross (1977) reported that pigs vaccinated with *S. equisimilis* bacterins were protected against the experimentally induced disease.[7] Field studies by Hare, et al., and Helms provided some evidence that Group C streptococcal infections in young pigs can be controlled by the use of autogenous bacterins (Helms. *Fort Dodge Biochem Rev.* 1962;31:8–27).[8] The use of antibiotics in this herd may also account for reducing the clinical signs of the disease.

The apparent success of the bacterin for the first year was encouraging, but after this initial success clinical signs again began to be expressed. It is possible at this time that a new strain of *Streptococcus suis* was introduced or environmental conditions allowed the organism to propagate.

Given that streptococcal species are ubiquitous in swine herds, and notorious for appearing clinically during times of stress and then disappearing whether or not therapeutic, bacterin, or management interventions are initiated, it is not possible to determine whether any of the interventions undertaken in this herd were responsible for the improvement in the clinical situation.

Implications

- A proper understanding of the nomenclature and taxonomy of streptococcal organisms can help decipher diagnostic reports.
- Both α and β streptococcal organisms are common endemic pathogens on pig farms. These organisms can and will occasionally cause outbreak of disease resulting in reduced productivity.
- In this case the best treatment regimens included a recognition of streptococcal organisms as an endemic pathogen and making efforts to maximize herd immunity while at the same time securing the herd from the spread of the known endemic pathogens.
- Controlling disease outbreaks in this case required an increase in overall herd immunity, reducing environmental stresses, and providing proper nutrition to sows.
- Cost of intervention must be evaluated and compared to expected financial benefits in an effort to avoid questionable vaccination programs that can be more costly than the disease.
- The lack of treatment controls in this case does not allow us to assess the efficacy of the treatments implemented.

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