HPS ranks as leading cause of death in pigs

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A 2,000-head, wean-to-finish site single stocked with 15-week-old pigs in southern Minnesota had a recent history of increased cough, and 16 pigs had died suddenly within the general population over a span of two days.

A barn walk revealed that clinically, 20% of pigs were coughing. Many of these pigs had increased respiratory effort, and 2% had stiff and swollen joints and decreased activity throughout the barn.

A necropsy of several of these pigs revealed lesions that included the presence of yellow-colored fluid in multiple joints, and 30-50% had pneumonia, with lungs that were heavy and fluid filled. A rough-appearing, yellow-to-tan material also covered the surface of the lungs and heart.

Initially ruled out for this case were porcine reproductive and respiratory syndrome (PRRS), swine influenza virus (SIV), *Haemophilus parasuis* (HPS), *Streptococcus suis*, *Actinobacillus suis* and porcine circovirus. Diagnostic testing, however, isolated the bacterial agent HPS as well as SIV.

HPS is a bacterium that, even among healthy pigs, is a normal colonizer of the upper respiratory tract. Given the opportunity, it can invade the host and cause respiratory disease and death in pigs as early as three weeks of age.

HPS is frequently recognized as a secondary agent in pigs that are suffering from common viral diseases, including SIV, PRRS and porcine circovirus. These viruses interfere with the normal function of the immune system and allow HPS to thrive.

Stressors such as weaning, transport or a suboptimal environment can also increase susceptibility to HPS-related disease.

In the case described above, HPS was "the killer" but was likely set up by the presence of SIV. Once HPS establishes an infection in the respiratory tract, it can be disseminated through the bloodstream of the pig, causing lesions in several other organs, including the central nervous system and joints.

Due to the fact that HPS can be a multi-systemic disease, the clinical signs associated with infections often vary. Common signs include: swollen joints, cough, increased respiratory effort, sudden death and central nervous system signs such as paddling, lack of coordination and recumbence.

Confirming the presence of HPS is commonly done through routine culturing or through the use of a polymerase chain reaction assay.

The polymerase chain reaction has increased the sensitivity of HPS detection and has allowed us to isolate this bacteria in cases where culturing may have yielded negative results.

Once it is established that HPS is present in a case, linking its involvement in the observed disease can be difficult. Because of the ubiquitous nature of this bacterium, it can be isolated from cases in which it was...
not a factor in disease or death loss.

In our practice, we have found that HPS that is isolated from joint and/or central nervous system tissues carries more disease significance than HPS that is isolated from respiratory tissues alone.

Strain or genetic differences in HPS isolates may also play a role in the severity of disease.

Bacterial genotyping -- which is used to characterize HPS based on genetic similarity or genomic fingerprints -- has become an area of increased interest among veterinarians and diagnosticians in an attempt to find associations between specific genotype and disease characteristics. Additionally, genotyping has allowed farms to distinguish strain differences among their farms or flows. That has been beneficial in designing vaccination and treatment programs.

Genotyping has also allowed us to investigate the immune response generated by pigs to HPS and how a protective response to one serotype may not necessarily elicit protection against other serotypes -- an important consideration when discussing vaccination options for HPS (Oliveira et al., 2004; Smart et al., 1993).

Working to prevent HPS-related disease using vaccination has been a primary goal of ours using either commercial or autogenous products. Because vaccine-induced immunity against HPS can be serotype specific, autogenous vaccines have allowed for the inclusion of the specific HPS strain(s) causing problems in a farm or flow that is important for protection.

Timing of vaccine administration is important. Many bacterial vaccines require multiple doses to generate a protective response, and when given only at weaning or shortly after weaning, there is often not enough time for the development of adequate immunity in the pig. The best results tend to be achieved when the vaccine is administered at 5-10 days of age with a booster at weaning or soon thereafter.

HPS treatment involves the use of injectable, feed and/or water antibiotics.

The best way to determine which antibiotic to use is to submit live pigs or multiple tissues from recently affected pigs to a diagnostic lab for examination. This will allow the pathologist to grow the bacteria in culture and conduct antibiotic sensitivity testing.

An antibiotic sensitivity provides direction on which antibiotic to choose to combat the specific HPS on that farm.

Preventative antibiotic programs in problem flows or in groups that are under an active viral challenge have also been successful in reducing death losses related to HPS.

HPS continues to be a recurring problem for many farms. The best treatment for HPS has been through preventative health management, including updating farm biosecurity measures to reduce viral entry into a farm, the use of vaccines and administering effective antibiotics when necessary.

Current research on HPS genetic profiles and their relationship with severity of disease, the immune response to infection and vaccine development will all help reduce the effect HPS has on growing pigs.

References


