Gillespie TG. Diagnosing endemic swine influenza virus in nursery pigs using cross-sectional serologic profiling. *Swine Health Prod.* 1999;7(2):81–83.

Diagnosing endemic swine influenza virus in nursery pigs using cross-sectional serologic profiling

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Summary

This case report describes how an outbreak of endemic swine influenza virus (SIV) in nursery pigs was diagnosed with the aid of a cross-sectional serologic profiling technique.

Keywords: swine, swine influenza virus (SIV), serology

Received: March 12, 1998
Accepted: October 5, 1998

Swine influenza virus (SIV) is a respiratory disease that affects pigs of all ages. The clinical signs include a deep-chested barking cough, sneezing, labored breathing, anorexia, lethargy, fever, a serous nasal discharge, and conjuctivitis.[1] Recovery is rapid. Reproductive problems, such as higher return-to-estrus rates, have been reported during severe outbreaks of SIV, especially when the clinical signs are at their most severe in the adult population.[1] Individual pigs that have secondary infections will show more severe signs.[1,2–5] It may take several days to weeks for the syndrome to spread and then resolve within a swine unit.

Because the period during which the virus can be isolated is quite short, SIV can be difficult to diagnose. Swine influenza virus is seldom detected on nasal swabs beyond 10 days postinfection, and can be isolated from lung tissue for an even more limited period. The best time to isolate SIV is when the animal is febrile, although this period is also short. Diagnosis is further complicated when concurrent pathogens, such as pseudorabies virus (PRV), porcine reproductive and respiratory syndrome virus (PRRSV), *Mycoplasma hyopneumoniae*, *Actinobacillus pleuropneumoniae*, *Haemophilus parasuis*, *Streptococcus suis*, and *Salmonella* spp, are present in the herd.[2–4] Concurrent infections with other viruses are associated with reduced ability to isolate SIV virus from tissue and nasal swabs, suggesting that "interference" between viruses is possible.[5,6]

Swine influenza virus is more difficult to diagnose when it is endemic in postweaned piglets due to maternal antibody interference, low exposure rates, and less severe clinical signs. The presence of maternal antibody does not prevent infection.[7] This report will describe how cross-sectional serologic profiling was used to help diagnose a case of endemic SIV in nursery pigs.

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Case herd

Nursery pigs in a 650-sow farrow-to-finish herd with no previous signs typical of SIV began to exhibit poor growth performance postweaning. The breeding herd was being vaccinated against PRRSV, *Leptospira* spp., parvovirus, erysipelas, *Escherichia coli*, and *Clostridium perfringens*. Quarterly serology was being performed on this herd, which was positive but serologically stable for PRRSV and *M. hyopneumoniae*.

This herd was housed on one site and located nearly 3.2 km (2 miles) from the nearest neighboring swine unit. Animals were moved all-in–all-out (AIAO) by room from farrowing to nursery, then pigs from two nursery rooms were consolidated into one finisher room. The average weaning age was 18.5 days.

The farrowing and nursery rooms were mechanically ventilated using negative pressure to pull tempered air from the adjoining hallway. The farrowing facility consisted of four rooms with a common hallway. The nursery facility had a total of eight rooms attached to a common hallway. A door that remained closed separated this hallway from the hallway adjoining the farrowing rooms. The ventilation system pulled fresh air from the hallway into each room. The fans were regulated by thermostatic controls located in the middle of each room.

Clinical signs

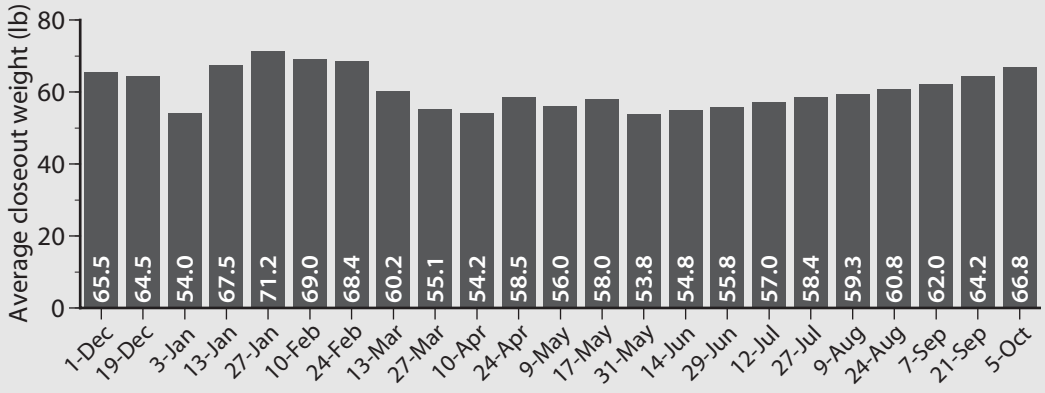
Clinical signs atypical of acute SIV, which persisted for 7–10 days, were first observed in nursery pigs about 5–7 days postweaning. By 2 weeks postweaning, pigs were gaunt, lethargic, sneezing, and occasionally coughing. Mucoid nasal discharge was evident. Rectal temperatures ranged from normal (38.4°C, 101.2°F) to elevated (41.4°C, 106.5°F). Pigs with high fevers were breathing rapidly. Approximately 2% of the pigs in the room had diarrhea.

In two of the eight nursery rooms, some pigs were also demonstrating clinical signs consistent with meningitis, including lateral recumbency, paddling, and seizures.

Mortality, which normally ranged between 0.9%–1.8% in this herd's nurseries, rapidly elevated to 3.5%. Weekly feed intake was estimated to be reduced by 15%–20% during the manifestation of the clinical signs. Closeout records indicated that average nursery move-out weights had dropped as much as 2.72 kg (6 lb) during the time of the outbreak (Figure 1).

Clinical signs diminished and the health of pigs improved by the fourth week postweaning. Pigs were clinically normal by week 5 postweaning.

Figure 1



Average closeout weights in nursery pigs during endemic swine influenza virus infection

This clinical disease cycle was repeated in each nursery room for several weeks.

Necropsy

Three pigs were selected to be submitted for necropsy. One pig was unable to rise and was exhibiting clinical signs of lateral recumbency, paddling, and seizures. The other two pigs were gaunt and had fevers of 41.1–41.7°C (106–107°F), rapid breathing, and nasal discharge.

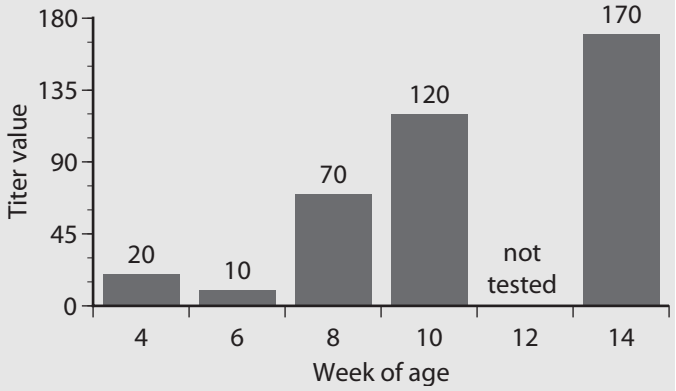
The pig with clinical signs of meningitis had a slight amount of cloudy cerebral spinal fluid, with congested meninges. The lungs of the pigs with respiratory signs were cranioventrally consolidated, and the dorsal sections were firm and noncollapsible. One of these pigs had scattered bilateral petechial hemorrhages in the lungs. The airways were filled with a frothy white foam.

Tissue samples of the lungs, trachea, lymph nodes, tonsils, brain, spleen, liver, and kidneys of the pigs were submitted to the Animal Disease Diagnostic Laboratory at Purdue University. The principal finding was a moderate, subacute, necrotizing bronchiolitis and bronchointerstitial pneumonia. Bronchioles were plugged with neutrophils and were lined by an attenuated or hyperplastic epithelium. Numerous bronchioles had focally denuded epithelium and were plugged with degenerate necrotic debris and neutrophils. The subjacent alveolar parenchyma was collapsed and septa were thickened by lymphocytes and macrophages. The alveolar lumina contained variable numbers of neutrophils and macrophages, suggesting bacterial infection (bacteriologists report, Purdue University-ADDL, March 1995).

Bacterial isolation recovered *Streptococcus suis* type 2 from the brain stem of the pig with meningitis. *Pasteurella multocida* type A was isolated from the lungs of the pigs with the respiratory signs. The diagnosis was SIV with secondary bacterial infection.

Virus isolation was performed using fluorescent antibody (FA) tests for SIV and PRV. However, no virus, including PRRSV, SIV, and PRV, was isolated from the lung tissue.

Figure 2



Geometric mean antibody titers to swine influenza virus from cross-sectional serologic profile

Serologic cross-sectional profiling

A serologic cross-sectional profile was taken from pigs that were 4, 6, 8, 10, and 14 weeks of age. Ten pigs in each age group were sampled.

Hemagglutination inhibition (HI) test was performed to assess antibody response. The S:P ratios for PRRSV ranged from 0.4–1.8 with an average S:P ratio of 1.23. Titers for PRV and coronavirus were negative for all ages sampled. The SIV titers suggested exposure (Figure 2). There was a slight decrease in geometric mean titers (GMT) for SIV in 6-week-old pigs, perhaps due to declining maternal antibody before the immunologic response to SIV infection.

Treatment

Treatment consisted of vaccinating sows at 5 weeks and 2 weeks prior to farrowing using a commercially licensed vaccine (Maxivac Flu™, Schering Plough Animal Health, Omaha, Nebraska). Piglets born to unvaccinated sows received a 1-cc dose intramuscularly (IM) at 5–7 days of age. A second 2-cc IM dose was given at weaning (approximately 18 days of age). The offspring's vaccination program was discontinued in piglets born to vaccinated sows. A maintenance vaccination was administered (one 2-cc dose) to sows 2–3 weeks prior to farrowing subsequent pregnancies. Replacement animals received a 2-

cc dose during their isolation and acclimation period.

The pigs exhibiting *S. suis* type 2 meningitis were injected intramuscularly with 10 mg per lb of an antibiotic and corticosteroid combination (Polyflex, Fort Dodge Animal Health, Fort Dodge, IA) once clinical signs were seen. There was no noticeable improvement in clinical signs (fever and respiratory signs) after pigs were treated with water, feed, and injectable antibiotics.

Discussion

The serological profile indicated that concentrations of maternal antibodies at 4 weeks were low (Figure 2). The definite rise in titer levels up to 14 weeks of age were interpreted as due to exposure to SIV.

The inability to isolate the SIV virus from tissue samples may be due to how samples were collected and handled. Taking nasal swabs from febrile pigs should improve a practitioner's ability to make a definitive diagnosis of SIV. Newly available immunohistochemistry (IHC) tests also should improve a practitioner's ability to detect the presence of SIV antigen when there are low numbers of virus particles.

There have been no previous reports of SIV infection reducing performance in nursery-aged pigs. Average daily gain and feed:gain, as measured by PigCHAMP® were improved in the nursery when the producer began to vaccinate pigs against SIV, but the greatest improvements were seen when unvaccinated offspring from vaccinated females were weaned into the nursery rooms. This response may illustrate the number of vaccinated animals present on the single site unit by this time. It is possible that prefarrowing vaccination, with resulting maternal antibody production, will limit clinical signs in young piglets. Maternal antibody titers can persist for several weeks in offspring.[1,7] It has been shown that weaned piglets with maternal antibody may be infected and may shed virus.[1,7]

The variation in the degree of immunity in gestating sows may influence the ability of the virus to infect litters. The commingling of several litters to fill a nursery room would exacerbate virus activity due to variability in the immunity of the nursery population.

Implications

- Swine influenza virus can be difficult to diagnose in postweaned pigs because the duration of the period during which it can be isolated from tissues or nasal swabs is very brief.
- The presence of secondary pathogens, such as the *S. suis* meningitis, in this case herd complicated the clinical picture and increased treatment costs as well as increased the mortality rate.
- A cross-sectional approach to profiling serologically was beneficial in supporting the diagnostic findings.
- Vaccination of piglets and dams was used to control postweaning swine influenza infections.

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