

Investigation into the Severe Acute PRRS Outbreaks

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Summary and Implications

The clinical, diagnostic, and preliminary experimental evidence that we have to date suggests that severe acute PRRS/SAMS/atypical PRRS is due to a severe manifestation of PRRSV. The fact that the outbreaks are so severe, and that they have occurred in well-vaccinated herds is reason for serious concern. The unique hepatitis lesions in some of the cases suggest that there could be additional pathogens involved in this syndrome or that strains of PRRSV have developed tissue tropism for the liver.

Introduction

Management practices such as replacement stock acclimatization, nursery depopulation, all-in-all-out pig flow, and vaccination have generally been successful in minimizing losses associated with PRRS. Since the last quarter of 1996, unusually severe disease outbreaks described as "atypical PRRS" or "severe acute PRRS" or "Swine Abortion and Mortality Syndrome" (SAMS) were reported and investigated by practitioners, diagnosticians, and researchers^{1,2}. Although these cases were relatively few in number, several things called particular attention to the episodes.

- incidence of abortions reached 10 to 50% in a 1 to 5 week period
- abortions occurred at all stages of gestation
- incidence of sow mortality reached 5 to 10% of the breeding herd inventory during the episode
- most of the herds involved were vaccinated multiple times with PRRSV vaccine

Most of the herds also experienced markedly increased preweaning mortality and decreased nursery pig performance primarily due to respiratory disease.

Materials and Methods

Several cases of severe acute PRRS were investigated through the Iowa State University Veterinary Diagnostic Laboratory to determine the etiology of the outbreaks. Three of those cases will be described in this report.

Case #1. A 950-sow herd had experienced an outbreak of abortions and respiratory disease typical of PRRS in

1995. Since that time the herd had been vaccinating at 7-days-postfarrowing with a modified live PRRSV vaccine. A 60-day gilt acclimatization program where the gilts received two doses of live, attenuated PRRSV vaccine also was implemented in 1995. In March of 1997, a large group of acclimated gilts was introduced into the herd. In April-May of 1997 the herd experienced 110 abortions, 20 dead sows, and a 20% nursery mortality.

Multiple fetuses, weakborn pigs, and neonatal pigs with respiratory disease were submitted. Several of the neonatal pigs had mottled tan lungs that failed to collapse and enlarged tan lymph nodes. Microscopic examination revealed mild interstitial pneumonia and moderate necrotizing umbilical arteritis in fetuses. Nonsuppurative encephalitis and interstitial pneumonia was observed in neonatal pigs. Sows had nonsuppurative meningoencephalitis, mild nonsuppurative hepatitis, multifocal necrosis of corpus lutea, and moderate nonsuppurative endometritis and myometritis.

Immunohistochemical (IHC) examination was positive for PRRSV. PRRSV was isolated from neonatal pigs. The isolates were tested by Restriction Fragment Length Polymorphism (RFLP) analysis and the pattern did not match that of RespPRRS/VR2332 and the isolates reacted with monoclonal antibody SDOW-17 suggesting that they were not vaccine viruses.

Case #2. A 1,200 sow herd had experienced an annual abortion rate of over 15% for the last 2-year period. Cycles of increased abortions and sow mortality occurred every 2 to 3 months. Sows were acutely anorexic, had high fevers (105 to 107 °F), and sometimes had cyanotic extremities. Females in the herd were being vaccinated at 60 days of gestation and 6 days post farrowing with a live, attenuated PRRSV vaccine. Multiple submissions of aborted fetuses were diagnostically unrewarding. Three live sows were then submitted to the diagnostic laboratory.

One sow had a bloody vaginal discharge and 15 dead fetuses in the uterus. The second sow had nine dead fetuses, and the third sow had aborted before arriving at the lab. All three sows had moderate lymphoplasmacytic and histiocytic hepatitis with mild random foci of hepatocellular necrosis, 3 of 3 sows had moderate lymphoplasmacytic endometritis and myometritis, 1 of 3 sows had moderate multifocal necrosis of the corpus lutea, 2 of 3 sows had mild-to-moderate interstitial pneumonia. A few of the fetuses had mild multifocal proliferative interstitial pneumonia.

Immunohistochemical examination was positive for PRRSV antigen in sow lung, tonsil, and ovary. PRRSV was isolated from sow tissues. The

RFLP pattern of the PRRSV isolates matched that of RespPRRS/VR2332.

The PRRSV isolates from this case were determined to be vaccine-like based on RFLP analysis. Inoculation of CDCD pigs with liver tissue filtrates from the sows in this herd resulted in isolation of a PRRSV isolate from the experimentally-inoculated pigs that was a wild type isolate. These results suggest that cell culture techniques used in diagnostic laboratories may select for cell culture-adapted vaccine strains. In this case we believe that the sows were infected with both a wild type strain and the vaccine strain. The vaccine failed to provide heterologous protection.

Case #3. A 1,200 sow multiplier herd was experiencing about 85 to 100 abortions per month for a 3 month period and had 15% annual sow mortality, 20% preweaning mortality, and 15% nursery mortality. The herd was being vaccinated at 50 days of gestation and 5 days post farrowing with live, attenuated PRRSV vaccine. Twenty live pigs, two sows, and multiple fetuses were submitted.

Fetuses had subcutaneous edema, edema of the mesocolon, and a few had segments of friable and hemorrhagic umbilical cords. Pigs from 2 days to 5 weeks old had mottled-tan lungs that failed to collapse, enlarged tan lymph nodes, pale yellow fatty livers, and variable degrees of arthritis and polyserositis in the older pigs. Sows had increased amounts of yellow pericardial and abdominal fluid, uteruses were flaccid and edematous with hyperemic mucosa and patches of pus, and necrotic debris adhered to the endometrium.

Microscopic examination revealed that a few of the fetuses and essentially all of the pigs had proliferative interstitial pneumonia typical of PRRS. Several of the pigs and the sows had nonsuppurative meningoencephalitis with perivascular cuffing, gliosis, and vasculitis. Both sows had moderate interstitial pneumonia and mild-to-severe multifocal lymphoplasmacytic hepatitis with random focal hepatocellular necrosis. Both sows also had moderate nonsuppurative endometritis.

Immunohistochemical stains were positive for PRRSV antigens in lungs and tonsils of pigs and sows. Several PRRSV isolates were recovered. RFLP analysis confirmed the presence of isolates with patterns that matched that of the vaccine and also the presence of wild type strains.

PRRSV isolates and liver filtrates from sows from this farm have been experimentally inoculated into CDCD pigs. Lesions typical of PRRSV (interstitial pneumonia, encephalitis, lymphadenopathy) have been reproduced. The duration and severity of the PRRSV-like lesions were remarkable. Hepatitis lesions not previously observed in PRRS cases also have been reproduced and the etiology of the hepatitis continues to be of interest.

References

1. Epperson B., L. Holler. 1997. An abortion storm and sow mortality syndrome. In Proceedings of the American Association of Swine Practitioners, Quebec, Canada, p479-484.
2. Halbur P. G., E. Bush. 1997. Update on abortion storms and sow mortality. Swine Health and Production 5:(2)73.