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Swine diagnostic pathology

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Case report: Front to back syndrome

History

A 350 sow farrow to finish operation. Farrowing in all-in, all-out groups of 70 every 42 days. All sows have a deliberate 21-day skip heat in this schedule. The breeding and gestation is a total crated facility. The sow herd has a very high total born litter size and excellent 21-day weaning weights, 7150 lb. Nursery and grow/finish are on-site and are all-in, all-out by building. Overall health status is very good. Replacement stock is in the form of grandparent stock, and replacements are made internally. The majority of matings are A.I. with on-farm collecting and processing of semen. Replacements are purebred animals from an SPF source.

Clinical signs

Occur only in females that are produced from grandparent stock, primarily gilts but occasionally p1 or p2's. Animals would eat but became very anxious after eating then would progress to continuous movement in the stall (front of stall to back of stall) and some self mutilation. Animals would not lay down. Body temperatures would be 105–106°F. Gilts and sows would abort if not treated.

Diagnosis

Interpreting PRRSV sequencing data

Jim Collins, DVM, PhD

The objective of this presentation is to remove some of the mystery surrounding PRRSV genetic analysis.

I. Basic information

A. PRRSV has 7 open reading frames (ORFs).

B. ORF 6 is the most conserved region of the PRRSV genome.

C. ORF 5 is among the most variable region of the PRRSV genome and codes for the viral attachment protein.

D. The Advanced Genetic Analysis Center (AGAC) of the College of Veterinary Medicine sequences ORF 5 and a portion of ORF 6 when characterizing PRRSV isolates.

II. Value of PRRSV sequencing

A. Epidemiological investigations

1. Study movement of PRRSV isolates between and within farms

B. Characterizing PRRSV as vaccine or field viruses

1. More sensitive than "predicted cut pattern" method

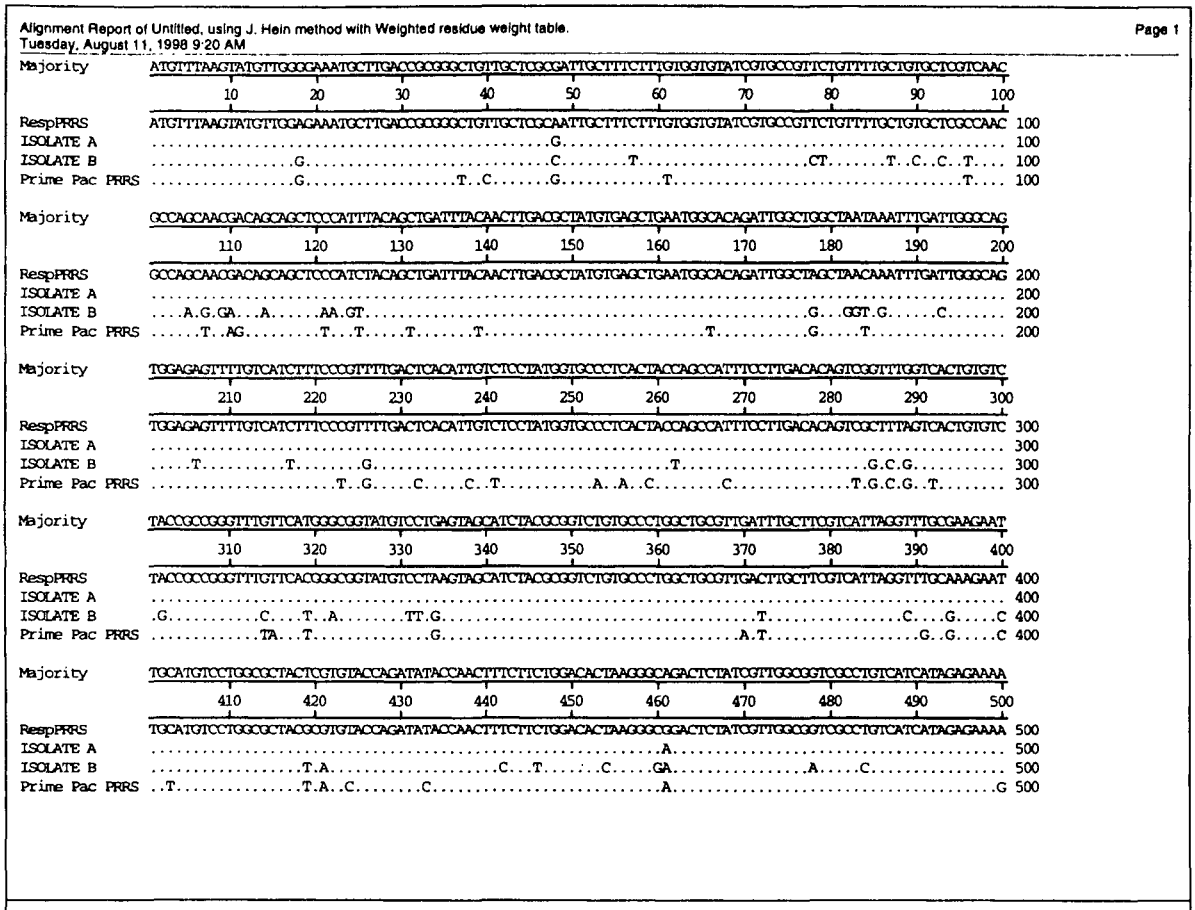
C. Study the evolution or mutation of PRRSV

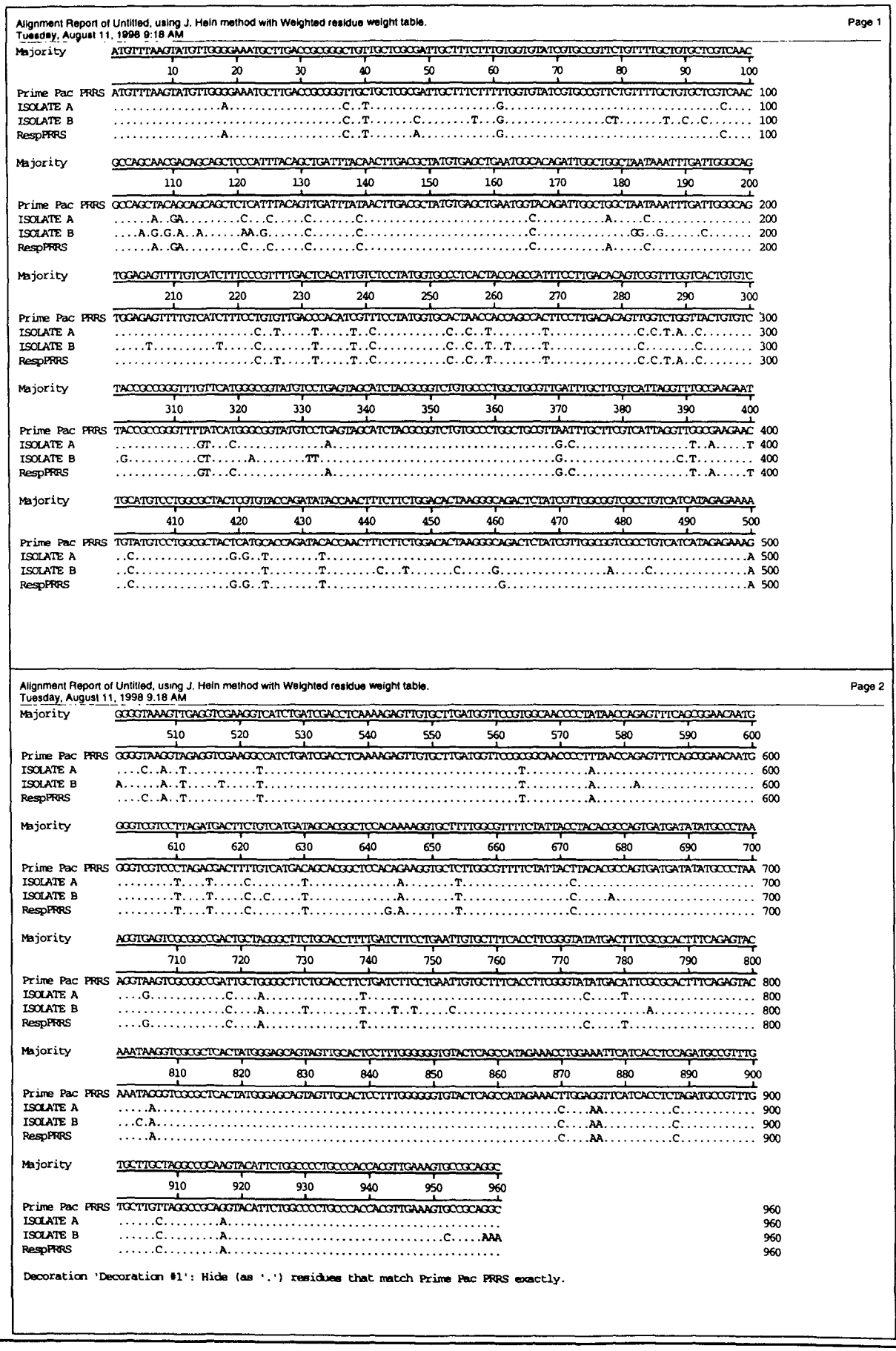
Note: Conclusions about vaccine efficacy or virulence cannot be made from ORF 5 and 6 sequencing data at this time.

III. Evaluating a PRRSV sequence

A. Begin by examining the "Alignment Reports" below (**Figure 1**).

B. Examine the upper left hand corner of the report. (Note the long string of letters designating nucleotides: A=adenosine, T=thymidine, G=guanosine, C=cytidine.)


Alignment Report of Untitled, using J. Hein method with Weighted residue weight table.
Tuesday, August 11, 1998 9:20 AM
Page 1
Majority ATGTTTAAGTATGTTGGGGAAATGCTTGACCCCGGGCTGTTGCTCGCGATTGCTTTCTTTGTGGTGTATCGTGCCGTTCTGTTTTGCTGTGCTCGTCAAC
RespPRRS ATGTTTAAGTATGTTGGAGAAATGCTTGACCGCGGGCTGTTGCTCGCAATTGCTTTCTTTGTGGTGTATCGTGCCGTTCTGTTTTGCTGTGCTCGCCAAC 100
ISOLATE A 100
ISOLATE B 100
Prime Pac PRRS 100
Majority GCCAGCAACGACAGCAGCTCCCATTTACAGCTGATTTACAACTTGACGCTATGTGAGCTGAATGGCACAGATTGGCTGGCTAATAAATTTGATTGGGCAG
RespPRRS GCCAGCAACGACAGCAGCTCCCATCTACAGCTGATTTACAACTTGACGCTATGTGAGCTGAATGGCACAGATTGGCTAGCTAACAAATTTGATTGGGCAG 200
ISOLATE A 200
ISOLATE B 200
Prime Pac PRRS 200
Majority TGGAGAGTTTTGTCATCTTTCCCGTTTTGACTCACATTGTCTCCTATGGTGCCCTCACTACCAGCCATTTCCTTGACACAGTCGGTTTGGTCACTGTGTC
RespPRRS TGGAGAGTTTTGTCATCTTTCCCGTTTTGACTCACATTGTCTCCTATGGTGCCCTCACTACCAGCCATTTCCTTGACACAGTCGCTTTAGTCACTGTGTC 300
ISOLATE A 300
ISOLATE B 300
Prime Pac PRRS 300
Majority TACCGCCGGGTTTGTTCATGGGCGGTATGTCCTGAGTAGCATCTACGCGGTCTGTGCCCTGGCTGCGTTGATTTGCTTCGTCATTAGGTTTGCGAAGAAT
RespPRRS TACCCCCGGGTTTGTTCACGGCGGTATGTCCTAAGTAGCATCTACGCGGTCTGTGCCCTGGCTGCGTTGACTTGCTTCGTCATTAGGTTTGCAAAGAAT 400
ISOLATE A 400
ISOLATE B 400
Prime Pac PRRS 400
Majority TGCATGTCCTGGCGCTACTCGTGTACCAGATATACCAACTTTCTTCTGGACACTAAGGGCAGACTCTATCGTTGGCGGTCGCCTGTCATCATAGAGAAAA
RespPRRS TGCATGTCCTGGCGCTACGCGTGTACCAGATATACCAACTTTCTTCTGGACACTAAGGGCGGACTCTATCGTTGGCGGTCGCCTGTCATCATAGAGAAAA 500
ISOLATE A 500
ISOLATE B 500
Prime Pac PRRS 500
Alignment Report of Untitled, using J Hein method with Weighted residue weight table.
Tuesday, August 11, 1998 9:20 AM
Page 2
Majority GGGGTAAAGTTGAGGTCGAAGGTCATCTGATCGACCTCAAAAGAGTTGTGCTTGATGGTTCCGTGGCAACCCCTATAACCAGAGTTTCAGCGGAACAATG
RespPRRS GGGGCAAAGTTGAGGTCGAAGGTCATCTGATCGACCTCAAAAGAGTTGTGCTTGATGGTTCCGTGGCAACCCCTATAACCAGAGTTTCAGCGGAACAATG 600
ISOLATE A 600
ISOLATE B 600
Prime Pac PRRS 600
Majority GGGTCGTCCTTAGATGACTTCTGTCATGATAGCACGGCTCCACAAAAGGTGCTTTTGGCGTTTTCTATTACCTACACGCCAGTGATGATATATGCCCTAA
RespPRRS GGGTCGTCCTTAGATGACTTCTGTCATGATAGCACGGCTCCAGAAAAGGTGCTTTTGGCGTTTTCTATTACCTACACGCCAGTGATGATATATGCCCTAA 700
ISOLATE A 700
ISOLATE B 700
Prime Pac PRRS 700
Majority AGGTGAGTCGCGGCCGACTGCTAGGGCTTCTGCACCTTTTGATCTTCCTGAATTGTGCTTTCACCTTCGGGTATATGACTTTCGCGCACTTTCAGAGTAC
RespPRRS AGGTGAGTCGCGGCCGACTGCTAGGGCTTCTGCACCTTTTGATCTTCCTGAATTGTGCTTTCACCTTCGGGTACATGACTTTCGCGCACTTTCAGAGTAC 800
ISOLATE A 800
ISOLATE B 800
Prime Pac PRRS 800
Majority AAATAAGGTCGCGCTCACTATGGGAGCAGTAGTTGCACTCCTTTGGGGGGTGTACTCAGCCATAGAAACCTGGAAATTCATCACCTCCAGATGCCGTTTG
RespPRRS AAATAAGGTCGCGCTCACTATGGGAGCAGTAGTTGCACTCCTTTGGGGGGTGTACTCAGCCATAGAAACCTGGAAATTCATCACCTCCAGATGCCGTTTG 900
ISOLATE A 900
ISOLATE B 900
Prime Pac PRRS 900
Majority TGCTTGCTAGGCCGCAAGTACATTCTGGCCCCTGCCCACCACGTTGAAAGTGCCGCAGGC
RespPRRS TGCTTGCTAGGCCGCAAGTACATTCTGGCCCCTGCCCACCACGTTGAAAGTGCCGCAGGC 960
ISOLATE A 960
ISOLATE B 960
Prime Pac PRRS 960
Decoration 'Decoration #1': Hide (as '.') residues that match RespPRRS exactly.


Alignment Report of Untitled, using J. Hein method with Weighted residue weight table.
Tuesday, August 11, 1998 9:18 AM
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Majority
Prime Pac PRRS
ISOLATE A
ISOLATE B
RespPRRS
Alignment Report of Untitled, using J. Hein method with Weighted residue weight table.
Tuesday, August 11, 1998 9:18 AM
Page 2
Decoration 'Decoration #1': Hide (as '.') residues that match Prime Pac PRRS exactly.

C. Note that each nucleotide is designated by a number (10,20,30,40) indicating its place in the genetic sequence.

D. A total of 960 nucleotides were sequenced. (Note the last number in the lower right hand corner of the report.)

E. Compare the two pages of the Alignment Report. Note that RespPRRS is listed first on one page and fourth on the other page.

1. RespPRRS
2. Isolate A
3. Isolate B
4. Prime Pac PRRS

F. The complete sequence of ORF 5 and a portion of ORF 6 of RespPRRS is given on the page where RespPRRS is listed first. The nucleotides of the other PRRSV isolates (Isolate A and Isolate B) are designated by a period where the nucleotides are the same as RespPRRS or are designated by the nucleotide letter (A, T, G, or C) if different from RespPRRS.

Note that at position 40, each of the 4 PRRSV isolates has the same nucleotide (T). This has no special significance, but if you can locate that information, you are well on your way to understanding the alignment report.

III. General guidelines for interpretation

A. If there are 10 or fewer differences in the genetic sequence, the PRRSV are considered to be closely related isolates.

B. Field strains of PRRSV usually have greater than 60 nucleotide differences when compared to vaccine strains, although there are exceptions.

IV. Interpreting the Alignment Report

A. Find the Alignment Report page where RespPRRS is listed.

B. For PRRSV isolate A, count the number of nucleotides that differ from those of RespPRRS.

1. The answer is 3 nucleotides. Thus, Isolate A is closely related to RespPRRS and is considered a vaccine strain.

C. Compare Isolate B to RespPRRS.

1. There are greater than 70 nucleotides that are different when Isolate B and RespPRRS are compared.

2. There are greater than 70 nucleotides that are different when Isolate B and Prime Pac are compared.

a. Conclusion: Isolate B is not closely related to Prime Pac or RespPRRS and is considered a field strain.

V. Predicted Cut Pattern

A. Cut patterns have proven useful for classifying PRRS viruses (Wesley, 1998).

B. A computer program can be employed to determine the "predicted cut pattern" for each of the PRRSV isolates based on the genetic analysis.

C. Predicted Cut Patterns

1. RespPRRS 2–5–2
2. Prime Pac 1–4–4

Foreign animal disease review: African swine fever

Rod Frank, DVM, PhD

African swine fever (ASF) is a devastating viral disease caused by a large DNA virus (*Iridovirus*). ASF is one of the "big 3" exotic swine diseases and can be the most deadly. High fever, cutaneous hyperemia, abortions, edema, and hemorrhages in internal organs characterize ASF. It is a contagious, usually fatal virus disease of swine. The acute disease kills almost all pigs that become infected. A milder form of ASF has been increasingly reported since the 1960s which is more difficult to diagnose. Both acute and the milder form are often confused with hog cholera. ASF can also be confused with other swine diseases.

Sudden death of pigs without prior illness is often the first sign of ASF. Pigs affected acutely typically have an abrupt rise in body temperature to 105–108(F (40.5–42.2© which lasts for ~4 days. There is reddening on ears, snout, abdomen and hindquarters. Coughing and dyspnea are often seen. Abortion frequently occurs and may be seen at any stage of pregnancy.

Gross lesions of acute ASF reflect vascular injury and include marked splenomegaly, marked hemorrhages of visceral lymph nodes, and excessive straw-colored fluid in the pleural, peritoneal, and pericardial cavities. Lungs have hemorrhages and edema. Petechial and ecchymotic hemorrhages may be seen on the surface and throughout the lungs. Pulmonary edema may especially be seen in interlobular septa. Hemorrhages may also be seen in heart muscle, diaphragm, and kidney (pinpoint to diffuse). Important microscopic lesions include lymphoid necrosis, renal tubular degeneration, necrosis of periportal hepatocytes with infiltrating lymphocytes, and vessel wall lesions in various organs. Perivascular cuffs in the meninges (meningoencephalomyelitis) are associated with vessel injury in the brain.

Swine that survive the acute phase of ASF and swine affected by the less virulent strain may have extensive pulmonary consolidation, chronic pleuritis and pericarditis, enlarged spleen and lymph nodes, and lesions associated with other diseases.

Differential diagnoses for ASF include hog cholera and various toxicities such as those produced by rodenticides, mycotoxins, and toxic plants that injure blood vessels.

Porcine circovirus: Four cases and a historical review

Kurt D. Rossow, DVM, Ph.D.

Case 1

Clinical history

Death loss in nursery pigs has been 5–10% for approximately one year. Pigs were weaned at 18 days and one-week later, pigs were sneezing and had labored breathing. Pigs were all home raised. Sows were vaccinated with killed Porcine Reproductive and Respiratory Syndrome Virus (PRRSV), *Pasteurella multocida*, *Streptococcus suis*, *Haemophilus parasuis* and *Escherichia coli*. Modified-live PRRSV has not been used for over one year.

Gross lesions

Four live-pigs were submitted for examination. Diffusely, lungs of each pig were red/tan and firm. The nasal cavity of one pig was filled with pus. Randomly, multiple lymph nodes in each pig was enlarged. Colon contents in each pig was formed.

Histopathology

- Lung: Many alveoli contained necrotic cell debris, and septa were thickened by macrophages. Remaining alveoli and bronchioles contained neutrophils and macrophages. Lymphocytic peribronchitis and lymphoplasmacytic alveolitis also characterized microscopic lesions in one lung of one pig.
- Lymphoid tissue: Multiple lymph nodes had histiocytic inflammation centered on germinal centers. Some macrophages contained multiple basophilic cytoplasmic inclusion bodies. In other sections, lymphoid tissue was necrotic.
- Small intestine: Villi were mildly blunted and fused.
- Heart: Three pigs had lymphocytic myocarditis.
- Remaining tissues: No lesions were found.

Virology

Fluorescent antibody (FA) examination of 2/4 lungs were positive for PRRSV. PRRSV was isolated from pooled lung samples cultured in porcine alveolar macrophages. FA examination of each lung was negative for swine influenza virus (SIV).

Bacteriology

Haemophilus parasuis was isolated from the lung of each pig. *Streptococcus suis,* serotype 2, was isolated from two of the lungs. Indirect FA examination of lung from one pig was suspect for *Mycoplasma hyopneumoniae*. *Pasteurella multocida* was isolated from nasal turbinate swabs.

Serology

- PRRSV S/P ratios: 1.07, 1.38, 0.97, 1.97
- SIV HI: 3 negative, one 1:20
- TGE SN: 1:16, 1:64, 1:8, <1:8
- Mycoplasma ELISA: all negative

Diagnosis

- Broncho and interstitial pneumonia—PRRSV, *Haemophilus parasuis*, *Streptococcus suis* serotype 2
- Myocarditis, lymphocytic
- Small intestine: villus atrophy, mild
- Lymphadenitis, histiocytic with inclusion bodies
- Rhinitis—*Pasteurella multocida*

Comments

Lung lesions in one pig were suggestive of *Mycoplasma hyopneumoniae*. The heart lesion was compatible with PRRSV infection.

Case 2

Clinical history

Ten percent of the pigs were coughing and "falling behind" as they went to into the grower. Tissues from 12–14 week-old pigs were submitted.

Gross lesions

The practitioner described the lungs as gray and edematous.

Histopathology

- Lung: Alveoli contained necrotic cell debris and septa were thickened by macrophages.
- Small intestine: Diffusely, villi were moderately blunted and fused.
- Liver: Random, small foci of lymphocytes and plasma cells.
- Colic lymph node: A few macrophages contained inclusion bodies.

TABLE 1: Case 4, seriology results

Test	9-weeks-old	6-weeks-old	4-weeks-old
PRRS Elisa S/P ratio	0.98	0.86	0.15
Mycoplasma ELISA	0.56 (s)	0.42	0.27
SIV	neg	neg	neg
TGE SN	1:16	1:64	1:64

Virology
FA examination of lung was positive for PRRSV. PRRSV was isolated from lung cultured in porcine alveolar macrophages. FA examination of small intestine was positive for TGE virus. Electron microscopic examination of feces was negative for virus particles.

Bacteriology
No pathogens were isolated from lung, liver, or intestine. Indirect FA examination of lung was negative for *Mycoplasma hyopneumoniae*. Darkfield examination of the colon was negative for spirochetes.

Clinical pathology
Fecal floatation was negative.

Diagnosis
- Interstitial pneumonia—PRRSV
- Atrophic enteritis—TGE virus
- Colic lymph node—macrophages with basophilic inclusion bodies

Case 3

Clinical history
Pigs were weaned at 14–18 days and one week later they quit eating and "faded away." Affected pigs also had clinical signs of pneumonia. Thirty-three percent of 500 pigs were affected and five pigs died every day. All of the pigs were home raised. Sows were vaccinated with PrimePac®.

Gross lesions
Three dead pigs were submitted and lungs of each pig were diffusely red/tan and firm. Multiple lymph nodes in each pig were enlarged. One pig had severe pericarditis and the nasal cavity of each pig was filled with pus. Colon contents in each pig was well formed.

Histopathology
- Lung: Alveoli contained necrotic cell debris and alveolar septa were thickened by macrophages. Remaining alveoli and bronchioles were filled with neutrophils and macrophages. Neutrophils and macrophages thickened pleura.
- Brain: Scattered blood vessels were cuffed by lymphocytes and plasma cells and there were a few foci of gliosis.
- Lymphoid tissues: Multifocally, histiocytes replaced normal tissues and there were clusters of macrophages with basophilic cytoplasmic inclusion bodies.
- Nasal turbinates: Lumens were filled with neutrophils.
- Liver: Random small foci of lymphocytes and plasma cells.
- Heart: Inflammatory cells thickened the epicardium.

Virology
FA examination of lung from each pig was FA positive for PRRSV and negative for SIV. Virus isolation for PRRSV was negative.

Bacteriology
Streptococcus suis was isolated from one lung. *Pasteurella multocida* was isolated from each nasal turbinate and was toxin positive by polymerase chain reaction (PCR). Indirect examination of each lung was negative for *Mycoplasma hyopneumoniae.*

Diagnosis 1
- Broncho and interstitial pneumonia—PRRSV and *Streptococcus suis*
- Rhinitis—toxigenic *Pasteurella multocida*
- Lymphadenitis—histiocytic with inclusion bodies
- Epicarditis—subacute
- Encephalitis—subacute

Comments
The brain lesion was compatible with viral inflammation and may be related to PRRSV infection. The epicarditis was compatible with bacterial inflammation; however, a causative agent was not identified.

A submission of live pigs from this herd was requested to better characterize bacterial involvement and facilitate PRRSV isolation. Three live pigs were submitted and 3/3 had PRRSV pneumonia (FA and virus isolation positive). *Haemophilus parasuis* was isolated from 2/3 lungs. *Bordetella bronchiseptica* was isolated from 2/3 nasal

turbinates. One pig had Mycoplasma arthritis. Lymphoid lesions were characterized only by necrosis.

Diagnosis 2

- Broncho and interstitial pneumonia—PRRSV and *Haemophilus parasuis*
- Arthritis—*Mycoplasma sp.*
- Rhinitis—*Bordetella bronchiseptica*

Case 4

Clinical history

Pigs of different ages had pneumonia. Pigs were home raised and had been vaccinated for *Streptococcus suis*, *Pasteurella* and Rhinitis. Pigs were weaned at 14 days.

Gross lesions

One each of 4, 5, 6 and 9-week-old pigs were submitted for examination. The 5-week-old pig was submitted dead; the remaining pigs were submitted alive. In the 4 and 6-week-old pigs, approximately 10% of AV lung lobes were purple and firm. Approximately 40% of AV lung lobes in the 9-week-old pig were purple and firm. Lung from the 5-week-old pig did not have lesions. Multiple lymph nodes in the 4, 6 and 9-week-old pigs were enlarged. Colon contents in the 5 and 6-week-old pig were soft and not formed.

Histopathology

9-week-old pig

- Lung: Alveoli were filled with necrotic cell debris and septa were thickened by macrophages.
- Lymphoid tissue: Multifocal histiocytic inflammation and macrophages with basophilic cytoplasmic inclusion bodies.
- Small intestine: Villi are mildly blunted and fused.

6-week-old pig

- Lung: As described for the 9-week-old pig.
- Lymphoid tissue: Increased numbers of germinal centers filled with blast-lymphocytes. Multifocally, lymphoid tissue is necrotic and there are scattered polykaryocytes.
- Small intestine: Villi were blunted and fused. Multifocally, rod-shaped bacteria line enterocytes.

5-week-old pig:

- Tissues did not have lesions.

4-week-old pig

- Lung: Alveoli contained neutrophils and macrophages. Other tissues did not have lesions.

Virology

9- and 6-week-old pigs

FA examination of lung was positive for PRRSV and negative for swine influenza virus. PRRSV was isolated from each lung. Lung was cultured in porcine alveolar macrophages. FA examination of small intestine from the 6-week-old pig was negative for rotavirus and TGEV. EM examination of a fecal sample was negative for virus particles.

5-week-old pig

EM examination of feces was positive for rotavirus. FA examination of small intestine was negative for rotavirus and TGEV.

4-week-old pig

FA examination of lung was negative for PRRSV and SIV. PRRSV was not isolated from lung. FA examination of small intestine was negative for rotavirus and TGEV.

Bacteriology

9-week-old pig

Haemophilus parasuis was isolated from the lung. Indirect examination of lung was negative for *Mycoplasma hyopneumoniae*.

6-week-old pig

Many K88 and 2134P *E. coli* were isolated from the small intestine. No pathogens were isolated from the lung. Indirect FA examination of lung was negative for *Mycoplasma hyopneumoniae*. Darkfield examination of colon was negative for spirochetes.

5-week-old pig

No pathogens were isolated.

4-week-old pig

Pasteurella multocida and Streptococcus suis were isolated from the lung. Indirect FA examination of lung was negative for *Mycoplasma hyopneumoniae*.

Serology

See **Table 1**.

Diagnosis

9-week-old pig

- Interstitial pneumonia—PRRSV
- Lung—*H. parasuis* isolation
- Small intestine—villus atrophy, mild
- Lymphadentis, histiocytic with inclusion bodies

6-week-old pig

- Interstitial pneumonia—PRRSV

- Atrophic enteritis—K88 and 2134P *E. coli*

5-week-old pig

- Feces—rotavirus

4-week-old pig

- Bronchopneumonia—Pasteurella multocida and Streptococcus suis

Comments

Villus atrophy described in the small intestine is suggestive of a prior viral or coccidial enteritis.

Historical review

Porcine circovirus

First identified in Germany in 1974 as a contaminant of a pig kidney cell line obtained from the American Type Culture Collection, porcine circovirus (PCV) has been proposed by Canadian researchers as the cause of a new disease of swine called postweaning multisystemic wasting syndrome (PMWS). Circoviruses are small DNA viruses that have also been described as the causative agent of psittacine beak and feather disease and infectious anemia of chickens.

Limited surveys have demonstrated porcine circovirus antibodies in 13 of 13 tested herds from Georgia (11 herds), Iowa (1 herd) and North Carolina (1 herd) with 53% of tested animals seropositive (indirect fluorescent antibody technique). One Canadian survey found PCV antibodies in slaughter swine and cull sows but not in a university specific pathogen free swine herd. In Germany, the seroprevalence of PCV antibodies was 77–95% in slaughter swine, 83% in 2–3 year-old pigs and antibodies were also detected in wild boars. Although a prevalence rate was not reported, researchers from Northern Ireland used colostrum deprived pigs due to an inability to locate a PCV seronegative herd. A recent study from Germany described antibodies in the serum of mice, humans, and cattle that cross-react with PCV. These results indicate that PCV is a common virus in the swine population.

PCV has been described to infect macrophages and monocytes in-vivo and in-vitro. Twenty-four hours after experimental infection of one-day-old colostrum deprived pigs, PCV was detected in buffy coat, nasal mucosa, and thymus. The viremia lasted two days while PCV was commonly detected in lung, lymphoid tissue and liver from 2 to 11 days after infection. PCV was isolated from nasal swabs 3–6 days after infection and from feces 13 and 14 days after infection. Other than intracellular antigen, no microscopic lesions were described. There were no clinical signs of infection in one PCV study and no description of clinical signs in a second PCV pathogenesis study. One hundred sixty fetal sera from field abortion cases were negative for PCV antibodies and PCV was only isolated from two fetal sera samples and one fetal spleen.

Experimental infections of porcine alveolar macrophages with PCV identified a transient reduction in the ability of the macrophages to interact with B and T lymphocytes and stimulate their proliferation. PCV infection of alveolar macrophages had no effect on phagocytosis or the expression of complement or Fc receptors.

In two studies, PCV infection did not cause gross or microscopic lesions and was transiently shed in nasal secretions and feces. The authors of one study concluded that while transplacental PCV infection occurs, it was not commonly associated as a cause of reproductive failure of swine in Northern Ireland. It was suggested the cell adapted PCV used in their study may be attenuated resulting in less influence on macrophage function.

Postweaning Multisystemic Wasting Syndrome

PMWS has been described by Canadian researchers as a new disease of swine affecting weaned pigs from small or large, high health herds. The syndrome is most commonly affects 5–6 week-old pigs and is characterized by wasting, dyspnea and enlarged lymph nodes. Less commonly, PMWS causes diarrhea, pallor (absence of skin color) and jaundice. In PCV naïve herds, pig mortality may reach 10% while endemically infected herds have lower morbidity and mortality. Factors associated with increased severity of the syndrome include overcrowding, poor air quality and commingling different age pigs. The difference in virulence of the Canadian PCV isolate linked to PMWS compared to the cell culture adapted PCV has been attributed to genomic differences.

Gross lesions associated with PMWS are poor body condition, enlarged lymph nodes, pallor or icterus, firm mottled red/gray lungs, liver atrophy, enlarged and "waxy" kidneys, white foci beneath the kidney capsule, splenomegaly and a fluid filled intestine. All of the gross lesions are not usually found and 3–10 pigs may need to be examined to demonstrate all of the gross lesions.

Diagnosis of PMWS associated with PCV is dependent on demonstration of typical tissue lesions and macrophages with cyotplasmic inclusion bodies, an appropriate clinical syndrome and ruling out other causes of failure-to-thrive syndromes in pigs. Tissues to submit for PMWS diagnosis include multiple enlarged lymph nodes (sternal, mesenteric, inguinal and portal), tonsil, lung, kidney, spleen, ileum and liver. Immunohistochemistry and in-situ hybridization for the detection of PCV have been reported but are not widely available. PCV isolation from infected tissues is hindered by the widespread contamination of cell culture lines with the virus.

PMWS has not been experimentally reproduced with PCV and tissues/samples for the diagnosis of other swine dis-

ease should be included. In one survey, PRRSV was demonstrated in approximately 50% of PCV infected tissues.

Clinical recommendations for treatment and control of PMWS have not been published to date.

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