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Clinical and pathological findings of PMWS cases in Europe

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Introduction

In 1991, a new clinico-pathological entity characterized by growth retardation, pallor of the skin, and high lethality was described in nursery pigs in Saskatchewan (Canada). This disease was named postweaning multisystemic wasting syndrome (PMWS) (Harding et al., 1996), and since 1994, an increasing number of cases have been detected in Canada. In 1997, the presence of porcine circovirus (PCV) antigen was demonstrated in lesions of animals affected by PMWS (Clark, 1997). Furthermore, nucleotidic sequence analysis of the PCV associated with PMWS revealed important differences compared to PCV derived from PK-15 cells (ATCC CCL-33) (Hamel et al., 1998), and it was suggested that these viruses be named PCV type 1 (PCV-1) for the cell culture-derived virus and PCV type 2 (PCV-2) for the virus associated with the new disease (Allan et al., 1999).

PMWS has also been described in several countries of western Europe, including France, Spain, the United Kingdom, Germany, Denmark, Italy, and The Netherlands. The first recognition of PMWS in Europe was in spring of 1996 in Brittany (France) (named as *Maladie de l'Amaigrissement du Porcelet*, MAP), with similar clinical signs and lesions to those detected in the Canadian pigs. A year after, in spring of 1997, PMWS was detected in Spain. PCV was systematically detected in tissues of all French and Spanish cases of PMWS, and sequencing of those isolates were shown to be PCV-2 type.

Clinical picture in Spanish cases of PMWS

The main clinical signs of pigs affected by PMWS include wasting, dyspnea, and paleness of the skin (very occasionally also jaundice). However, in some particular cases, the major clinical picture includes diarrhea, dyspnea, or gastric ulceration of the pars oesophagea, with no apparent wasting disease.

Affected herds are mainly farrow-to-finish operations with continuous flow and fattening units of multiple pig sources. However, PMWS has been diagnosed in all types of farms, with range of size from very small (30 sows) to very large (6000 sows) operations.

PMWS most commonly affects pigs of 8 to 12 weeks of age (range of 7 to 15 week-old pigs). Morbidity and lethality is variable among farms and batches of animals, but the usual rates are 4–10% (ranging 1–60%) and 70–80% (ranging 50–100%), respectively. No reproductive disorders or disease in pigs of other ranges of age have been observed in farms experiencing PMWS. One of the most reported comments from swine practitioners is the lack of response to the antibiotic therapy in almost all cases of PMWS.

At necropsy, the most striking lesions are non-collapsed lungs and enlargement of lymph nodes (mainly the superficial inguinal, submandibular, mesenteric, and mediastinal ones). However, these lesions are not always present, and they cannot be used as the only marker of PMWS on a farm. The frequency of the aforementioned findings and others in a study of 148 PMWS affected pigs from Spain are summarized in **Table 1**.

How to establish a diagnosis of PMWS

As a general agreement, the final diagnosis of PMWS is made by clinical history (with a clinical picture compatible with PMWS), presence of characteristic histopathological lesions, and detection of PCV-2 in tissues of affected pigs. In the above-mentioned study using 148 PMWS-affected pigs from several Spanish provinces, the most frequent histopathological lesions are summarized in **Table 2**. In all those cases, PCV-2 was present in a variable amount in at least one tissue, always closely associated to the microscopic lesions.

In our experience, in order to make the definitive diagnosis of PMWS based on histopathology and detection of PCV-2, the most valuable tissues to examine include the following:

- Lymph nodes
- Tonsil
- Spleen
- Ileum (containing Peyer's patches)
- Lung
- Liver

Table 1: Frequency of macroscopic lesions observed in 148 pigs affected by PMWS

Macroscopic findings	Frequency (out of 148 pigs)	Percent
Non-collapsed, rubbery lungs	103	69.8 %
Enlargement of at least one lymph node	101	68.2 %
Pulmonary consolidation (bronchopneumonia)	79	53.4 %
Gastric ulceration of pars oesophagea	56	37.8 %
White-spotted kidneys	27	18.2 %
Jaundice	9	6.1 %
Hepatic atrophy	8	5.4 %

Table 2: Frequency of microscopic lesions observed in 148 pigs affected by PMWS

Microscopic findings	Frequency (out of 148 pigs)	Percent
<i>Lymphoid tissues</i>		
Lymphocyte depletion	129	87.2
Histiocytic inflammatory infiltration	114	77.0
Intracytoplasmic inclusion bodies	67	45.3
Syncytial (multinucleate) cells	54	36.5
Multifocal coagulative necrosis	18	12.2
<i>Lung</i>		
Interstitial pneumonia	130	87.8
<i>Liver</i>		
Slight to moderate hepatitis	82	55.4
Intense hepatitis and destruction of parenchyma	11	7.4
<i>Kidney</i>		
Interstitial nephritis	67	45.3

- Kidney

PMWS and other infections: The Spanish situation

Other infections may be found in farms experiencing PMWS, but a direct relationship with the syndrome is difficult to establish. Most conventional farms in Spain are seropositive against porcine reproductive and respiratory syndrome (PRRS) virus; in other words, it is very difficult to find a PMWS affected herd seronegative against the PRRS virus. In the routine diagnostic, we tested immunohistochemistry (using SDOW 17 monoclonal antibody as the first antiserum) of over 300 lung tissue samples from pigs diagnosed as PMWS cases; PRRS virus was detected in 72 lungs (24%). However, it could be that the use of a more sensitive technique such as polymerase chain reaction (PCR) would have increased the percentage of dually infected animals. At the individual pig level, in a serological study made on 60 pigs with PMWS, 40 pigs were seropositive against PRRS virus, but 20 were seronegative (no information on the viremia of those pigs at the time of death was available). Also, on some farms, we have seen seroconversion against PRRS virus before, during, and after an acute outbreak of PMWS.

These data suggest that PRRS may be involved in the clinical problem on some farms but not on others.

Other diseases that have been concomitantly detected with PMWS are Aujeszky's (Pseudorabies) disease, Glässer's disease, streptococcal meningitis, salmonellosis, *Escherichia coli* gastroenteritis, unspecific diarrhea, dietetic hepatosis, and suppurative bronchopneumonia (with *Pasteurella multocida*, *Bordetella bronchiseptica*, and *Streptococcus suis* being the major bacteria involved).

Control of PMWS

Presently, management is the only strategy that can be applied to control PMWS. This includes the following measures:

- Strictly managing all in - all out
- Maintaining the adequate density of pigs per pen
- Strictly applying general hygienic measures
- Providing adequate ventilation systems
- Avoiding mixing animals (of different age or origin)
- Controlling other concomitant diseases that are also present in the farm (via vaccination, antibiotics)

- Avoiding sudden increases of the breeding population

Very few experiences are recorded in Spain in order to control PMWS. Some swine farms applied a partial depopulation system (nursery depopulation), and, after several months, it seemed that production indexes of nursery and fattening pigs have returned to the ones recorded before to the PMWS outbreak. However, it is still too early to fully evaluate the usefulness of this control strategy. On the other hand, it has been observed that PMWS may show a cyclic clinical presentation, and in some farms it may disappear spontaneously after several months.

The most important thing to remember before starting a control program is to establish a very detailed diagnosis of the problem. Not all wasting pigs are PMWS or PRRS.

Diagnostic profile in a farm with PMWS

In order to test how PCV-2 infection progresses in a PMWS affected herd, we randomly chose a 220-sow, farrow-to-finish operation which experienced a marked number of pigs with growth retardation and dyspnea since January 1999. Mortality rate in fattening pigs of this particular farm were variable among batches but ranged from 15–30% during five months (productive data of fattening pigs returned to previous levels in May-June 1999). No reproductive problems were detected on this farm during the previous year (June 1998 - June 1999). We made the following observations:

January 1999

- Acute diseased 3 month-old pigs (n=4); complete necropsy

March 1999

- Chronically diseased 3–4 month-old pigs (n=11); complete necropsy
- Healthy 3–4 month-old pigs (n=8); complete necropsy
- These pigs were from different batches of the previous pigs.

June 1999

- Healthy 6.5 month-old pigs (n=12); samples of lymphoid tissue (tonsil and submandibular lymph node) and sera taken at the slaughterhouse

The test battery performed on these pigs included the following:

- Histopathology
- In situ hybridization to detect PCV-2
- Immunohistochemistry to detect PRRS virus and Aujeszky's disease virus (ADV)

- Serology against PCV-1 (IPMA), PCV-2 (IPMA), PRRS virus (ELISA), ADV (ELISA gE), and porcine parvovirus (PPV; ELISA)
- PCR for PRRS virus in serum

Some conclusions may be derived from this study, but we do not know if all of them are applicable to all PMWS affected farms:

Characteristic microscopic PMWS lymphoid lesions are seen mainly in the acute phase of the disease. Lesions such as presence of lymphocyte depletion, histiocytic infiltration, formation of syncytial cells are fully expressed in acutely affected pigs. Chronically affected pigs had also lesions but with significantly lower intensity. Therefore, to establish a diagnosis of PMWS, samples from pigs in the initial phase of disease should be submitted to the laboratory.

Healthy pigs in a PMWS affected farm are also infected by PCV-2. The eight, healthy, randomly selected pigs in contact with diseased animals also showed PCV-2 in tissues. Virus was found in several organs, mainly lymphoid tissues, associated with mild lesions of the same type as described for PMWS affected pigs. This finding suggests that when PCV-2 is introduced on a farm, probably most pigs—if not all—become infected, but just a proportion of them become ill. It is not yet known which other factors are involved in the outcome of the infection.

Healthy pigs from a PMWS affected batch (which experienced disease at 3–4 months of age) are not infected by PCV-2 at slaughter. Lymphoid organs collected at slaughter from these 11 pigs showed no histopathological lesions, and PCV-2 nucleic acid was not detected in their tissues by in situ hybridization. These data suggest that PCV-2 infection has disappeared by the time of slaughter.

Serology is not a good tool for diagnosing PMWS. Serological techniques are still under development. Our group has been applying an immunoperoxidase monolayer assay (IPMA) using ATCC CCL-33 (PK-15 continuous cell line) and a porcine kidney cell line infected with PCV-2 in order to detect antibodies against PCV-1 and PCV-2, respectively. All pigs of this study yielded seropositivity with very high titers against PCV-2. These results were expected, based on the presence of PCV-2 nucleic acid in almost all pigs.

Other viral infections may have a role in the clinical problem on this farm. Serological tests for PRRSV, ADV, and PPV showed that all three viral infections occurred in the farm during the study period. PRRSV and ADV antibodies were found in all investigated groups, at different proportions. PPV antibodies were found in 11 out of 12 slaughtered pigs; by contrast, only one of the 22 pigs necropsied (a chronically diseased, growth-retarded pig)

had also PPV antibodies. These results suggest the following:

- Pigs were infected by PRRSV at the same time as with PCV-2.
- ADV circulated in the farm during the PMWS outbreak and reached serological saturation at slaughter.
- PPV circulated in the late phase of the fattening period, unrelated to PMWS.

References

Allan GM, McNeilly F, Mechan BM, Kennedy S, Mackie DP, Ellis JA, Clark EG, España E, Saubi N, Riera P, Botner A,

Charreyre CE (1999) Isolation and characterisation of circoviruses from pigs with wasting syndromes in Spain, Denmark and Northern Ireland. *Vet Microbiol.* 66: 115–123.

Clark E (1997) Post-weaning multisystemic wasting syndrome. In: *Proceedings of the American Association of Swine Practitioners*, 499–501.

Hamel A, Lin L, Nayar G (1998) Nucleotide sequence of porcine circovirus associated with postweaning multisystemic wasting syndrome in pigs. *J Virol.* 72: 5262–5267.

Harding JC (1996) Post-weaning multisystemic wasting syndrome: preliminary epidemiology and clinical findings. In: *Proceedings of the Western Canadian Association of Swine Practitioners*, 21.

