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Postweaning Multisystemic Wasting Syndrome (PMWS) Surveillance Study

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Summary and Implications
PMWS is characterized by a clinical history of wasting or poor performance in weaned pigs and by severe lymphoid depletion and histiocytic replacement of follicles in lymphoid tissues. The detection of porcine circovirus type 2 (PCV2) antigen or nucleic acids within characteristic microscopic lesions is required for the diagnosis of PMWS.

Swine veterinarians submitted a specified set of samples from one hundred field cases that they felt fit the clinical definition of PMWS. All these cases were further analyzed for the presence or absence and scored for severity of the hallmark microscopic lesions (lymphoid depletion) of PMWS, the amount of PCV2 antigen associated with the lesions, and identification of concurrent bacterial and viral infections. Fifty-four of the 100 field cases were confirmed to be PMWS, whereas, no concurrent bacterial and viral infections. Fifty-four of the cases with PCV2-associated lesions were scored for the presence of the hallmark lesions of PCV2-associated PMWS: lymphoid depletion, histiocytic inflammation and replacement of follicles, and amount of PCV2-antigen associated with the lesions as determined by immunohistochemistry (IHC). All cases were scored for the presence of concurrent bacterial infection by routine bacterial culture and for the presence of PRRSV, SIV, and Mycoplasma hyopneumoniae (M. hyopneumoniae) by either IHC or PCR.

Results and Discussion
After microscopic evaluation, the cases were classified as PMWS, PCV2-associated lymphoid depletion, or as having no PCV2-association based on the lack of presence of PCV2 and PCV2-associated lesions. Fifty-four of the hundred field-cases had high amounts of PCV2-antigen associated with severe lymphoid depletion and inflammation and thus were confirmed to be PMWS. The majority of these PMWS cases (72.2% of the cases, 39/54) had concurrent PRRSV-infection, 33.3% (18/54) had concurrent M. hyopneumoniae infection, and 29.6% (16/54) had concurrent SIV-infection. Only one of the PMWS cases was singular PCV2-infection without detectible concurrent viral or bacterial co-infections. The most commonly isolated bacteria in PMWS cases were Pasteurella multocida in 29.6% of the cases (16/54), Streptococcus suis in 24% of the cases (13/54), and Salmonella sp. in 14.8% of the cases (8/54).

There were 46/100 case submissions that had no or only mild lesions in lymphoid tissues associated with PCV2-infection. Eighteen of these 46 cases were diagnosed as PCV2-associated lymphoid depletion since there was mild lymphoid depletion, mild-to-severe inflammation of lymphoid tissues and low-to-moderate amounts of PCV2-associated with these lesions in at least one of the lymphoid tissues examined. Fifty-six percent of these cases (10/18) had concurrent PRRSV-infection, and 27.8% (5/18) had concurrent M. hyopneumoniae- or SIV-infection. The most commonly isolated bacteria in cases with PCV2-associated lymphoid depletion were Streptococcus suis and Pasteurella multocida in 44.4% of the cases (8/18), and Arcanobacterium pyogenes in 27.8% of the cases (5/18). The incidence of confirmed bacterial confections was higher in this group of pigs compared to the groups classified as PMWS and those with no PCV2-associated lesions.

There was no association of lesions with PCV2 in 28/100 case submissions. This highlights the need for microscopic examination for confirmation of cases clinically suspected to be PMWS. The majority (14/28, 50%) of the non-PCV2-cases had concurrent PRRSV-infections, followed by SIV-infection in 39.3% (11/28) of the cases, and M. hyopneumoniae-infection in 35.7% (10/28) of the cases. In the cases with no PCV2-associated lesions, the most prevalent bacteria were Streptococcus suis in 28.6% (8/28) of the cases, Pasteurella multocida in 25% (7/28) of the cases, and Salmonella sp. in 17.8% (5/28) of the cases. Further genomic characterization of PCV2 isolates from cases with widely varying degree of PCV2-associated lesions is in progress to determine if this may explain differences in clinical manifestation of PCV2-associated diseases within and between herds.

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