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SOME HAEMATOLOGICAL PARAMETERS IN POSTWEANING MULTISYSTEMIC WASTING SYNDROME-AFFECTED PIGS

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Summary

Postweaning multisystemic wasting syndrome (PMWS) is a recently described disease that affects late nursery and fattening pigs (Harding and Clark 1997, Segales et al., 1997). In Romania PMWS affected pigs have been described for the first time in western region of Transylvania in 2003 (Cadar et al. ,2006, 2007). Pigs clinically affected by PMWS show poor body condition, paleness of the skin, dyspnoea and, occasionally, jaundice. Although, gross pathology is not conclusive, the presence of enlarged lymph nodes, non-collapsed and surface-mottled lungs, a yellowish-orange liver and gastric ulcers of the pars oesophagea are of value in the diagnosis of PMWS (Harding and Clark 1997). The aim of this study was to evaluate some haematological parameters, such as total number of leukocytes, lymphocytes, monocytes, eosinophiles and basophiles of PMWS-affected pigs and pigs without clinical signs of PMWS, respectively. An increase in mean white blood cells count was observed for the pigs with naturally occurring PMWS infected pigs compared with healthy pigs. This may be related to the viral infection and/or secondary viral/bacterial infections which are often recorded in pigs suffering from PMWS.

Postweaning multisystemic wasting syndrome (PMWS) was first recognized in Canada in 1996 as a new emerging disease which caused wasting in post weaned pigs. Since then, PMWS has been recognized in pigs in many countries. The syndrome is caused by a DNA virus referred to as porcine circovirus 2 (PCV2), which is classified in the family Circoviridae. Microscopic lesions and the detection of PCV-2 are nowadays considered the key points for PMWS diagnosis. PMWS has been experimentally reproduced using PCV-2 together with porcine parvovirus (PPV) as inoculum (Allan et al., 1999), while the use of only PCV-2 led to a mild form of PMWS (Balasch et al., 1999). Paleness of the skin is commonly described as a clinical sign of PMWS, but haematological investigations have not been undertaken.

Materials and methods

Blood samples were collected in commercial tubes containing tripotassium ethylenediamine tetra-acetic acid anticoagulant by puncturing the jugular vena. The pigs were classified into two groups: 100 pigs with naturally occurring PMWS (these pigs showed growth retardation and dyspnoea, and half of them showed some degree of skin paleness; PCV2 was confirmed by PCR) and 100 pigs without

PMWS clinical signs. The leukocytes differential count was performed, using light microscopy, by identification of 100 cells on a blood smear for each sample. A Student's t test for independent samples was used to compare the naturally affected pigs and pigs without PMWS clinical signs.

Results and Discussions

Mean haematological values of the two different groups of pigs are summarised in table 1. Statistically significant differences were observed in white blood cell count and lymphocytes.

Table 1.

Mean values for the haematological parameters from pigs with naturally occurring PMWS and healthy pigs.

Parameters	PMWS affected pigs	Healthy pigs
White blood cell count (x 10^3/ mm^3)	80-110	140-200
Lymphocytes (%)	30-39	50-68
Monocytes (%)	4-6	2-3
Neutrophils (%)	50-60	30-35
Eosinophils (%)	1-3	1-2
Basophiles (%)	0.1	0

An increase in mean white blood cells count was observed for the pigs with naturally occurring PMWS infected pigs compared with healthy pigs. This may be related to the viral infection and/or secondary viral/bacterial infections which are often recorded in pigs suffering from PMWS. Alternatively, the mean of the lymphocyte percentages was reduced, while the means of the monocyte and neutrophil percentages were increased in the naturally diseased animals when compared with healthy pigs. These findings indicated changes of the neutrophil: lymphocyte ratio in naturally affected pigs compared with healthy pigs. However, the mean of monocyte percentages were increased in naturally PCV-2 infected pigs compared with healthy pigs, which suggest an increase or proliferation of monocyte/macrophage lineage cells, possibly viral induced. This fact could be related to the increase of macrophages infiltrating target tissues that is observed in cases of PCV-2 infection (Rosell et al., 1999).

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