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Short communication

Muco-cutaneous candidiasis in two pigs with postweaning multisystemic wasting syndrome

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Abstract

In two distinct commercial swine herds, poor weight gain and an increased number of animals showing wasting were observed among nursery and growing pigs. Cases of postweaning multisystemic wasting syndrome (PMWS) and infection with *Haemophilus parasuis* had been previously diagnosed in these herds. One growing wasted pig from each herd was necropsied and showed enlarged lymph nodes. Pseudomembranous material adhered to the dorsum of the tongue, soft and hard palate in case 1, and in case 2, fibrinous material was seen as whitish plaques on the oesophageal surface with hyperkeratosis of the non-glandular stomach.

The main histological lesions in both cases were found in lymphoid tissues with a multifocal accentuated lymphohistiocytic infiltrate, areas of lymphoid depletion and intracytoplasmic inclusions in histiocytic cells in lymph nodes and Payer's patches. Focally, extensive ulceration was found in the stratified pavement epithelium of the tongue with necrosis and necrosuppurative infiltrate in case 1; in case 2, there was ulceration in the stomach with lymphohistiocytic infiltrate in the submucosa and ulceration in the mucosa of the oesophagus associated with yeast cells and pseudo-hyphae. *Candida albicans* was isolated from the oral cavity lesions. Immunohistochemistry of the lymph nodes was positive for porcine circovirus 2 (PCV2). The association between PMWS and mucocutaneous candidiasis reported here supports the potential immunosuppressive state of PMWS infected pigs.

Keywords: Swine; PMWS; Immunosuppression; Mucocutaneous candidiasis

Postweaning multisystemic wasting syndrome (PMWS) was first recognized in 1996 in Canada as a new emerging disease causing wasting in postweaning pigs. Since then, PMWS has been described in pigs in Asia, North and South America and Europe (Chae, 2004). The causative agent is porcine circovirus 2 (PCV2), a small non-enveloped DNA virus containing a unique single-strand circular genome (Ellis and Allan, 2000). Many studies have indicated that diseases or syndromes associated with PCV2 affect pig herds worldwide (Ellis and Allan, 2000) and the most frequent clinical signs are wasting or a failure to thrive, enlarged lymph nodes, dyspnoea, pallor, jaundice and diarrhoea (Harding, 2004). Enlargement of lymph nodes (mainly inguinal, mesenteric and mediastinal) is the main macroscopic finding at necropsy. Other gross lesions have included non-collapsed, tan–mottled lungs, kidneys with multiple pale foci of

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variable diameter, reduction or increase in liver size, with orange yellow discolorations (Segalés et al., 2004). The principal microscopic lesions are found in lymphoid tissues and include a variable degree of lymphocytic depletion with loss of follicular architecture combined with a multifocal to diffuse, slight to very intense lymphohistiocytic multinucleated giant cell infiltration (Chianini et al., 2003; Segalés et al., 2004).

It has been suggested that PMWS may be related to immunosuppression in pigs (Segalés et al., 2001). Studies performed in conventional pigs affected with PMWS have described a decrease in circulating B-cells, loss of lymphocytes in B-cells areas, and decreases in CD4+ and/or CD8+ T lymphocytes (Segalés et al., 2001). The lack of response to antibiotic therapy, the existence of a litter effect and the occurrence of other unusual disease syndromes or serious infections with ordinarily non-pathogenic secondary organisms may reflect the immunosuppressive features of PMWS (Segalés and Clark, 2002). This hypothesis is supported by typical microscopic lymphoid damage in PMWS affected tissues and the association of the disease with opportunistic pathogens commonly associated with immunosuppression, such as *Aspergillus* spp. (Segalés et al., 2003), *Chlamydia* spp. (Carrasco et al., 2000), *Pneumocystis carinii* (Clark, 1997) and *Cryptosporidium parvum* (Núñez et al., 2003).

We describe here two cases of mucocutaneous candidiasis in PMWS affected pigs. The cases occurred in two distinct industrial farms in the state of Rio Grande do Sul, Brazil. The symptoms and clinical history were similar, namely poor weight gain and increased wasting in nursery and growing pigs. Cases of PMWS and infection with *Haemophilus parasuis* and *Pasteurella multocida* type D had been previously diagnosed in these herds. Because of the bacterial infection the animals received therapeutic medication with 200 ppm amoxicillin in the food for 15 days. One growing pig from each herd, with poor body condition was euthanased for diagnostic purpose.

Necropsy was performed just after euthanasia and several tissues were collected, fixed in 10% neutral buffered formalin solution, sectioned at 5 μm thick and stained with haematoxylin/eosin for histopathological examination. To look for yeast cells and pseudohyphae, histological slides of the tongue (case 1), oesophagus and stomach (case 2) were stained with Grocott and PAS (periodic acid Schiff). Fragments of nasal turbinates and tongue (case 1) were collected for bacteriological and mycological culture, respectively. Samples collected from the tongue were cultivated on Sabouraud's dextrose agar with 0.5 g/L chloramphenicol at 37 °C for five days. For species identification we used filamentation in equine serum and production of chlamydospores in corn meal agar (Sidrim and Moreira, 1999). Bacteriological examination was by routine culture of the material collected from the lesions (blood agar and MacConkey agar), incubated at 37 °C aerobically and anaerobically with evaluation at 24 and 48 h.

Immunohistochemical procedures were performed on formalin fixed, paraffin-embedded sections of lymph nodes which were digested in 0.005 g/10 mL protease XIV for 15 min at 37 °C. Polyclonal rabbit antibody to PCV2 (Sorden et al., 1999) at 1/1000 dilution was incubated for 1 h and then stained by the streptavidin-biotin immunoperoxidase technique using diaminobenzidine as chromogen.

The main gross findings in both animals were enlargement of mesenteric and inguinal lymph nodes, and consolidation of the apical and middle lobes of the lungs. Cyanosis of the ears was noted as was a white pseudomembrane adhering to the dorsum of the tongue, soft and hard palate and accentuated atrophy of the nasal turbinates (case 1). Fibrinous material arranged as whitish plaques on the oesophagus surface (Fig. 1) and marked hyperkeratosis of the non-glandular mucosa of the stomach were also noted (case 2).

Microscopically, in both cases, lymphoid tissues showed multifocal accentuated lymphohistiocytic infiltrates with areas of moderate lymphoid depletion and sharply, demarcated spherical, intracytoplasmic inclusions in histiocytic cells in lymph nodes and Payer's patches. In the lungs, hyperplasia of the lymphoid tissue around the bronchi and bronchioles with mononuclear and polymorphonuclear cells inside, and thickening of the alveolar septa were also present. Mild lymphohistiocytic inflammatory infiltration was found in the portal zones of the liver and submucosal multifocal lymphohistiocytic inflammatory infiltrates were in the colon.

In the stratified pavement epithelium of the tongue, focally extensive ulceration, necrosis and necrosuppurative infiltrate associated with yeast cells and pseudohyphae could be observed. In the nasal turbinates atrophia of the spongiform bone with lymphohistiocytic

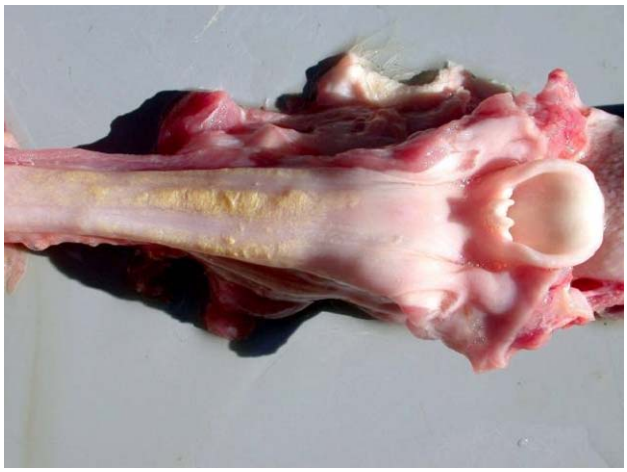

Fig. 1. Case 2: oesophagus. Fibrinous material longitudinally corrugated in the mucosal surface.