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Production Assistants
Steven Claas
Michael Klatt

Layout and CD-ROM
David Brown

Logo Design
Ruth Cronje, and Jan Swanson;
based on the original design by Dr. Robert Dunlop

Porcine circovirus-associated disease in Kansas

S.C. Henry, and, L.M. Tokach
Abilene Animal Hospital, P.A., Abilene, KS

Beginning in the fall of 2005, numerous swine producers in central and eastern Kansas noted a dramatic increase in mortality beginning 4 to 5 weeks after placing pigs in the finisher buildings. Pigs were 13 to 15 weeks old at the onset of mortality. In many cases mortality reached 10% to 20% within the month following onset in that group or lot of pigs. Losses then diminished after a 6-7 weeks course of disease, falling to expected levels and remained there until slaughter. Subsequent groups in these farms might or might not be affected; subsequent groups in the previously affected facility might or might not be affected. Survivor weight for age was not impacted at normal slaughter age. Clinical morbidity appeared to be equivalent to mortality in nearly all cases. Expression of disease seemed to be an individual pig phenomenon with pen and barn mates seemingly unaffected, a rather unique clinical disease presentation.

Clinical signs included anorexia, inactivity and apparent depression/listlessness in nearly all cases. Beyond these nearly universal signs, a diverse set of additional clinical signs were noted, sometimes as a multiplicity of signs in an individual yet, in others, as limited and discrete indications. Those clinically detectable signs included, roughly in order of frequency:

1. cessation of growth and dehydration with sharply reduced feed intake
2. lymphandenopathy with 3-5 fold enlargement of inguinal, popliteal and pharyngeal lymph nodes
3. pallor and oxygenation deficit based on the bluish discoloration in the distal extremities and margins of the ears
4. respiratory effort but without cough in most instances
5. dermal lesions with the most obvious and dramatic being those described for porcine dermatitis and nephropathy syndrome (PDNS) but less dramatic yet obvious "bulls-eye" skin lesions on flanks, rear limbs, ears and neck; approximately 30% of fatalities had dermal lesions with fibrinoid arteritis of a variety of tissues but not lung
6. nephropathy with necrosis
7. neurologic deficits especially noticeable in rear limbs ranging from the subtle signs of slack or weak semimembranosis muscles to an inability to stand on the rear limbs, resulting in dragging of the legs when attempting to move
8. prostration and rapid progression to death
9. exercise intolerance with distressed respiratory effort following even mild exercise resulted in pigs immediately dropping to sternal recumbency when pressure to move was halted
10. enteritis with pasty diarrhea containing undigested grain
11. ear tip necrosis incidence increased in affected and unaffected animals in some cases

The case description for the Kansas outbreaks matches very closely the description of F. Madec and N. Rose as the disease appeared in Europe (ISAH 2005, Warsaw, Poland).

The clinical diagnosis of PCVAD was confirmed by demonstration of histopathologic lesions of lymphoid depletion, interstitial pneumonia, and circovirus antigen associated with the lesions by means of IHC staining. Lymph node necrosis with vasculitis of arteries of the capsule and macrophage infiltration was common. Hemorrhagic lymph nodes and the presence of giant cells in some were also observed. Necrotic enteritis was associated with lymphocyte depletion in Peyer's patches. The requirements (Sorden 2000) for diagnosis were met in all herds affected. Some of the affected herds were PRRS negative, others PRRS positive. In some herds SIV was concurrently active, in others not.

Intervention methods have been, without exception, of no discernable value in our clinical observation. In-feed antibiotic metaphylaxis with chlortetracycline 400gm/T began at placement. Water delivered tetracycline at outset of clinical signs, early individual pig therapy with flunixin and a variety of antibiotics was attempted with a number of variations in affected pigs. Sorting of pigs into a hospital pen at first clinical signs for therapy was the initial practice but proved of no measurable benefit. Cessation of vaccination with mycoplasma vaccines containing amphigen was associated with less disease and loss in a number of groups subsequent to the initial outbreaks on

one farm. This was not a predictable effect to date but more observations are needed.

Presently, recommendations to our clients for managing PCVAD include:

- minimize stress of movement and handling on all pigs
- discontinue any injectable therapeutic interventions of affected pigs because of the clear lack of efficacy, the stress of handling pigs and the economic losses associated with drugs
- realize that the lymphoid depletion demonstrated in these animals makes recovery very unlikely and that signs are widely variable
- identify pigs early in the process and either euthanize them, if they are suffering and have a low likelihood of recovery, or move them to an observation pen to reevaluate; in most cases nearly all such animals die or must be euthanized
- focus on environmental comfort, hygiene and sanitation, especially in preparing a previously affected facility for the next group of animals

No clear physical or breeding stock association between all the affected herds adequately suggested any common source, focus or cause for these outbreaks.

Acknowledgements

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