International Pig Topics

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Swine influenza II

Pathology of influenza A in pigs – microscopic pathology

The key diagnostic lesions are those of a necropurulent bronchitis and bronchiolitis. The initial lesions can be seen as soon as 24 hours post infection and include vacuolar degeneration and necrosis of epithelial cells with a loss of apical cilia. This is occurring at the same time as abundant virus budding.

By 48 hours post infection, the sloughed epithelial cells are accumulating in the airway lumen together with neutrophils that have migrated across the epithelium to create the typical lesion of mucopurulent bronchiolitis. By 72 hours post infection the sloughing of necrotic epithelial cells is more prominent.

Between days four and five post infection the early signs of recovery appear. By days seven to 10 post infection varying degrees of interstitial pneumonia with perivascular and peribronchiolar lymphoid proliferations can be seen. By days 14 to 21 the damaged respiratory tissues show a full recovery at the microscopic level.

Gross pathology

The typical gross lesion of influenza A infection in pigs is that of a cranioventral bronchopneumonia. Sometimes gross lesions can be seen as soon as 48 hours post infection but by five days post infection the multifocal coalescing lobular pattern to pneumonic tissue has become clearly apparent. These lesions can be grossly indistinguishable from those of enzootic pneumonia caused by Mycoplasma hyopneumoniae.

Immunohistochemistry

Immunohistochemistry can be used to detect viral particles in infected tissues. The two antigens most frequently detected are nucleoprotein (NP) and haemagglutinin (HA) protein. Antibodies against NP protein can be used to detect all subtypes of influenza A virus, whereas antibodies against HA protein are subtype specific.

NP protein antigen is located in the nucleus and cytoplasm and HA protein antigen is located in the cytoplasm and along the cell's surface. The location of the antigen within the cell depends upon the location of viral replication. If infection is primarily confined to the bronchioles the immunoreactivity will be primarily in bronchiolar epithelial cells and within the neutrophils and necrotic debris in the bronchiolar lumens. If infection has spread to the alveoli of the lung tissue the immunoreactivity will be predominantly in type II pneumocytes.