Pleuropneumonia of pigs - a new disease

Pleuropneumonia of pigs is a severe respiratory disease caused by infection with Actinobacillus pleuropneumoniae *(formerly* Haemophilus pleuropneumoniae*). It was first observed in England in 1957 and has been reported worldwide since. It has now been diagnosed in New Zealand.*

The owner of a fattening unit in the Waikato was concerned with the number of pigs showing respiratory disease and dying. Over a 2 month period mortality rate rose from less than 1% to 2.5%.

A typical diseased 12-week-old porker with a deep cough and laboured abdominal breathing was submitted to the Ruakura Animal Health Laboratory for necropsy. Grossly, the lungs were focally attached to the chest wall. The diseased lung tissue was hard, red and raised. Several joints contained inflammatory exudate. Microscopically the lesions were of a subacute fibrinous pleuropneumonia consistent with *A. pleuropneumoniae* infection. The diagnosis was confirmed by bacteriological isolation. Isolates of the bacterium were sent to the Animal Research Institute, Yeerongpilly, Queensland, Australia for serotyping. They were identified as *A. pleuropneumoniae* serovar 7.

Within a month another case was diagnosed in a line of slaughtered "poor doers" from another herd in the Waikato.

Pleuropneumonia attributed to *A. pleuropneumoniae* is widely distributed in pig populations throughout the world. Transmission of the disease is by the airborne route and is facilitated by close contact. All ages are susceptible, but the disease most commonly affects pigs aged 6 weeks to 6 months. The attack rate and mortality can vary from less than 10% to 100%.

A slaughterhouse survey of 77 porkers from the first infected herd showed lesions characteristic of chronic *A. pleuropneumoniae* infection in the lungs of 17% of pigs.

A. pleuropneumoniae is highly pathogenic and appears capable of invading and proliferating rapidly in all parts of the lung. The course of the disease is manifest in peracute, acute, subacute and chronic forms. Deaths in the peracute form occur after a short period of depression, fever and, maybe, haemorrhage from the nose. A typical lesion is a fibrinonecrotic and haemorrhagic pneumonia with fibrinous pleurisy. Many pigs which recover show loss of appetite and decreased body weight gain.

The pneumonia caused by *A. pleuropneumoniae* is mostly bilateral, with involvement of the diaphragmatic lobes, which helps to differentiate it from enzootic pneumonia caused by *Mycoplasma hyopneumoniae* and pneumonias caused by *Pasteurella multocida* and *Bordetella bronchiseptica.*

There are at least five serotypes of *A. pleuropneumoniae*, the virulence of which varies. Circulating antibodies can be detected as early as 10 days after exposure. Complement fixation titres reach a maximum by 3 to 4 weeks and persist for many months.

The extent of the disease within New Zealand is unknown. Neither is it known how long it has been here. Epidemiological studies are to be undertaken soon.

Although relatively little pathological material from the lungs of pigs is submitted to animal health laboratories in New Zealand, and although special cultural procedures are necessary to identify this pathogen, it is unlikely that *A. pleuropneumonia* has been simmering in New Zealand pig herds for very long. In 1989 at least 30 lung tissues from pigs were checked out for *A. pleuropneumoniae* with negative cultural results before the first isolation was made. No pathologist at Ruakura has personally seen or read a case history describing gross lesions characteristic of *A. pleuropneumoniae* infection. Two pathologists have had working experience of the disease in North America.

The ideal diagnostic samples are portions of fresh and fixed lung tissues. Pneumonia due to *A. pleuropneumoniae* should be suspected when pneumonic lesions are present in the diaphragmatic lobes. The complement fixation test is of no diagnostic value in acute outbreaks, but is an important tool in identifying chronically infected herds.

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