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Inclusion Body Rhinitis of Swine

by

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and  
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Inclusion Body Rhinitis (IBR) is a viral disease of swine characterized by large basophilic intranuclear inclusion bodies in epithelial and reticular cells. It has been found in pigs in North America, Europe, Japan, Australia and Russia since it was first described in England by Done (1954). The causative agent is a member of the Herpes family of viruses and is classified as a cytomegalovirus. Similar cytomegalic viruses have been described for mice, man and guinea pigs. In these species infection may occur at all ages, although the young generally develop clinical illness, while adults have inapparent infection.

Inclusion body rhinitis is a relatively new disease to Iowa swine. The first reported outbreak in Iowa was by Switzer (1963). A survey by Duncan et al. (1964) reported IBR lesions in 25 of 211 (12%) Iowa herds. Currently, diagnosed incidence is low in Iowa swine, although infected animals are presented each year to the diagnostic laboratory. Clinical disease may be severe with high baby pig mortality or very mild with some inclusion bodies found as an incidental finding on postmortem examination of the nasal mucosa.

Clinical signs in piglets appear to be age associated. In a recent report, Plowright et al. (1976), noted that a piglet infected with IBR virus during the first week of life developed both epithelial and reticular cell involvement, while older animals developed only epithelial cell involvement. In man and mice, the greater pathogenicity to the young has been associated with a generalized infection involving the reticular cell system. Infection of epithelial cells alone resulted in subclinical problems. Changes which occur in reticular cells of the 3-4 week old pig make those cells less susceptible than neonatal reticular cells.

Piglets infected during the first days of life develop a severe rhinitis, dyspnea, pallor and anorexia about 10 days after infection. Rhinitis, the most prominent sign, begins as a mucopurulent exudate which progresses to severe purulent exudate in several days. The nares become encrusted and plugged, blocking the nasal air passages. The piglets may refuse to nurse, being anorexic and unable to feed and breathe simultaneously. Suffocation and starvation are the major consequences, with mortality approaching 50 percent in some herds. Usually, morbidity approaches 100% with average mortality about 10%. Survivors may be anemic and stunted despite good nutrition and management. In some cases, stunting and severe anemia are the major causes for admission to the diagnostic lab. Many swine surviving the initial disease develop atrophic rhinitis. Although IBR has not been shown to produce turbinate atrophy, the inflammation and epithelial reactions it produces may allow Bordetella bronchiseptica to become more intensely established and thereby increase the severity of atrophic rhinitis.

Postmortem examination of experimentally infected pigs reveals edema of the lungs, pericardial sac, subcutis and occasionally effusions of the pleural and peritoneal cavities. Petechiation is seen in conjunction with the edema. In many animals subcapsular renal petachiae will

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occur to a variable degree. These lesions follow the weakening of the blood vessel walls by viral infection of the endothelial cells. Characteristic inclusion bodies can be found in macrophages and capillary endothelium in many organs of the body during a systemic infection. Inclusions are also found in the sinusoides of the liver, lymph nodes, adrenals and bone marrow. Inclusions in renal tissue are found in the cortical capillaries and glomerular cells. The central nervous system may contain capillary inclusions in the choroid plexus and other areas outside the cortex. Focal areas of encephalitis with lymphocyte accumulations have been occasionally observed. These lesions do not generally contain inclusion bodies.

The nasal mucous glands have been the primary area for identification of IBR inclusions. Inclusion bodies in the nasal mucosa generally precede signs of rhinitis by several days and persist for about 14 days after rhinitis develops. This short life may explain why inclusions are found on an irregular basis and may not be seen in slaughter weight animals. When a generalized infection occurs, capillary endothelial cells of the nose and other tissues may be involved.

Older pigs (over 2 to 3 weeks of age) may have a mild or inapparent infection. In some pigs, mild petechiation of the testicles as well as kidneys has been observed. Inclusions can be found in the nasal mucous glands and tubular epithelium of the kidney but cause little functional problem. These lesions may be found incidently during a complete postmortem examination.

Treatment for IBR infected animals is usually not rewarding. Antibacterials generally will not influence the course of the disease.

Prevention of this problem is difficult because the epidemiology of the disease is unclear. Transmission is reported to be primarily by the aerosol route, however, in one case a one-day old pig was found to have typical inclusion bodies. This would suggest possible intrauterine infection as a source of infection. The presence of carrier sows and transmission from sow to litter, then horizontal transfer between litters is a likely method of spread.

At this time, carrier animals cannot be easily identified, nor can past exposure history be developed because of the inapparent infection of adult swine.

REFERENCES