INCLUSION BODY RHINITIS

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Recognition of a new disease syndrome in swine may occur because of one of two reasons. The first is occasioned by the recognition of an existing but previously overlooked specific disease agent and its attendent clinical syndrome while the second is due to the introduction of a hitherto absent pathogen into an area, region, or country. In general, it requires some interval of time to be reasonably certain which of these two situations is applicable to a newly recognized clinical problem.

A case of inclusion body rhinitis has recently been diagnosed in Iowa swine. This communication will attempt to outline the clinical nature of this disease and to summarize the state of our present knowledge about the etiological agent and the general group of viruses to which it belongs.

This disease was first described in England by Done in 1955 (2). It is caused by a virus that is a member of the salivary gland group of viruses of man and animals (3). This is the first member of this group of viruses with a strong affinity for the nasal cavity. These are moderate-sized viruses that produce a distinctive, large inclusion body. The great size that may be attained by affected cells (25 to 40 microns) and by the intranuclear inclusions (8 to 10 microns) are outstanding features of this group of viruses (6), (10). Known members of this group of viruses include salivary gland viruses of mice, rats, voles, hampsters, chimpanzees, and man in addition to the inclusion body rhinitis of swine. The only one of this group that is frequently associated with clinical disease, other than inclusion body rhinitis, is the salivary gland virus of humans. This agent may produce a disease resembling that produced by several hemolytic anemias of newborn infants. In addition there may be cerebral involvement resembling congenital toxoplasmosis. The incidence of chronic or latent infection in humans is high, reaching an incidence of 70 percent in some surveys.

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Information about inclusion body rhinitis of pigs is still very meager, but some general information has emerged. The virus infection is transmitted from chronically infected carriers to young swine and there may be occasional in-utero transmission since at least one case has been diagnosed in a one-day-old pig (3,5,8). Older swine appear to be much more resistant to the clinical manifestation of the disease than young pigs. It appears that the most intense clinical manifestation occurs in pigs from one to three weeks of age. In pigs of this age there may be an acute rhinitis that reaches such intensity that the nasal cavity is blocked by exudate and the pig suffocates. This syndrome may terminate fatally in three to four days. The initial nasal discharge is muco-purulent but rapidly becomes purulent plugging the nasal cavity and external nares.

The typical inclusions have been observed in several different tissues of the pig but the nasal cavity is the only site where they are consistently present or abundant in numbers (3). There have been reports that the virus is capable of producing a chronic proliferative pneumonia in pigs (1,4,9). These findings need additional verification but are in general agreement with the findings in some other species infected with the salivary gland virus.

The virus of inclusion body rhinitis is believed to have been recently grown in cell cultures (3) but details of the procedure have not yet become available. Such an advancement in our knowledge may allow serological detection of this viral infection. It appears, however, that the production of antibodies does not bring about the rapid elimination of the viral infection. When pigs are exposed at an early age, what effect the transmission of antibodies to the pig has on the clinical manifestation of this disease is not known, but it seems likely that this would markedly reduce the clinical severity of the disease. It is reported that in England infections are fairly common and the clinical manifestations associated with this infection ranges from mild to severe. In some areas as many as 50% of the herds are estimated to be infected. In this country there has been only one published report of the occurrence of this disease, (7) this occurred in Maryland.

In the study of the causes of turbinate atrophy in Iowa swine it has been necessary to examine a considerable number of swine nasal turbinates over a period of several years. In the course of this work, the typical dramatic inclusion bodies were not observed. This leads to the conclusion that if inclusion body rhinitis had been present in a very high percentage of the swine population, it would have been observed before. This fails to confirm the fact that other undiagnosed cases have not occurred in the state, but it does suggest that the disease still has a low incidence in Iowa.

The specific case encountered in Iowa originated in the south central portion of the state in a farm herd consisting of seventeen brood sows and their litters. This case was referred for diagnosis by the attending veterinarian, Dr. Keith Cogley, upon recognition of the extraordinary nature of the case. The sows were raised on the farm and had been a relatively trouble-free group of sows having farrowed large litters of good-sized pigs. The husbandry was well above average. The first few litters did not have any respiratory ailment although they developed a diarrhea when a few days of age. This diarrhea is at the present time believed to be an incidental, unrelated syndrome. The subsequent litters developed normally until about one week or slightly older in age at which time they manifested severe sneezing associated with a moderate mucopurrulent nasal discharge. In a few days the nasal discharge had become purulent and had encrusted the external nares. This blocked the nasal cavity and the pigs suffocated, usually within three to five days after the onset of symptoms. Almost all subsequent litters farrowed developed the disease. The final mortality figure was not available but it was estimated that over 50 percent of the pigs died, primarily from suffocation. The surviving pigs were stunted and appeared anemic even though they had received injections of iron. Tissue sections prepared from one of the pigs

that had survived the initial outbreak revealed evidence of anemia and an extraordinary amount of hemopoietic activity of the spleen.

Tissue sections prepared from the nasal turbinates revealed large numbers of the very dramatic and characteristic inclusion bodies located primarily in the collection ducts of the tubulo-alveolar glands. These inclusion bodies resembled in all essential details those observed by other workers and they form the basis for the diagnosis of this case as inclusion body rhinitis.

The initial diagnosis of a disease within a state or geographical area poses difficult problems for disease control and eradication agencies and officials. If it is actually a new disease just gaining entrance into an area, then a rapid quarantine and eradication program is, in most cases, highly desirable. On the other hand, if it is a well established disease that has only recently been recognized, quarantine attempts only work a hardship on the herds to which they are applied without giving control of the disease. The regulatory officials must make their initial decisions before all these questions can be answered. The best information available at the present time indicates that inclusion body rhinitis is still uncommon in Iowa swine and that this initial outbreak manifested itself as an acute, severe and highly fatal rhinitis. No comparable clinical syndrome

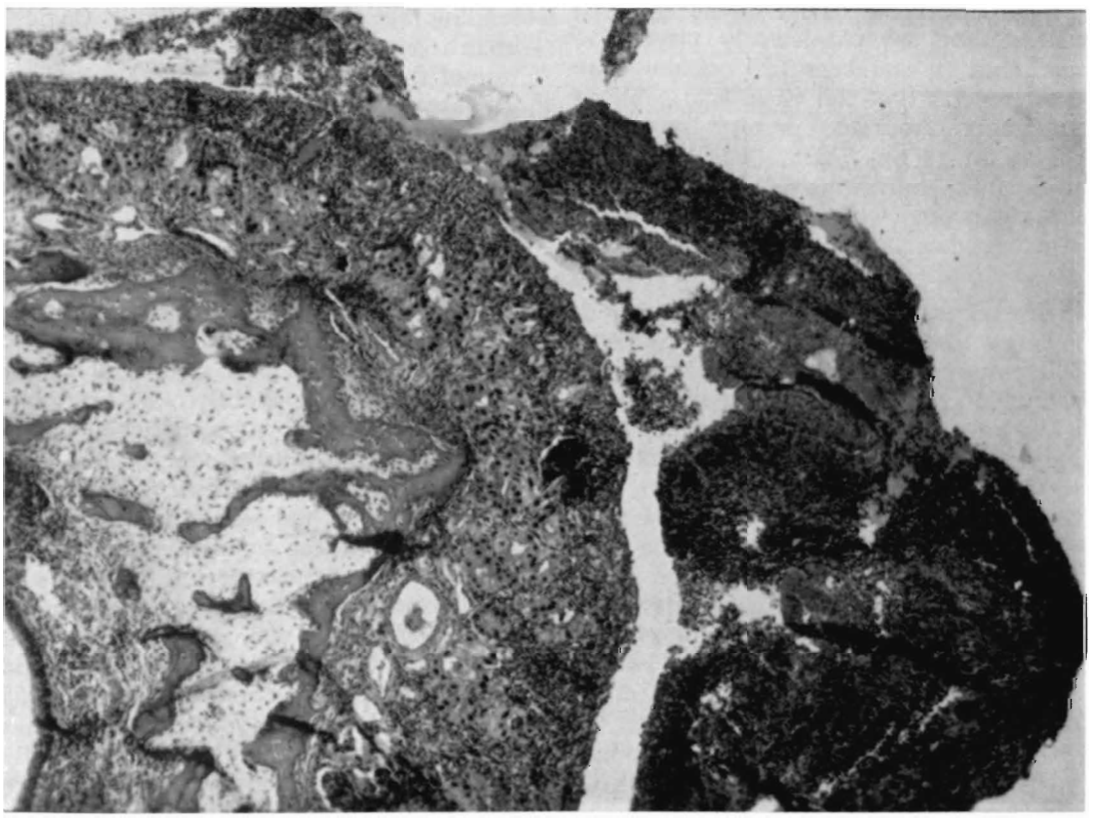

Fig. 1 Porcine turbinate. Note the increased number of enlarged cells in the tubules of the tubulo-alveolar glands in the submucosa and the mass of exudate on the surface of the turbinate. H&E. X40

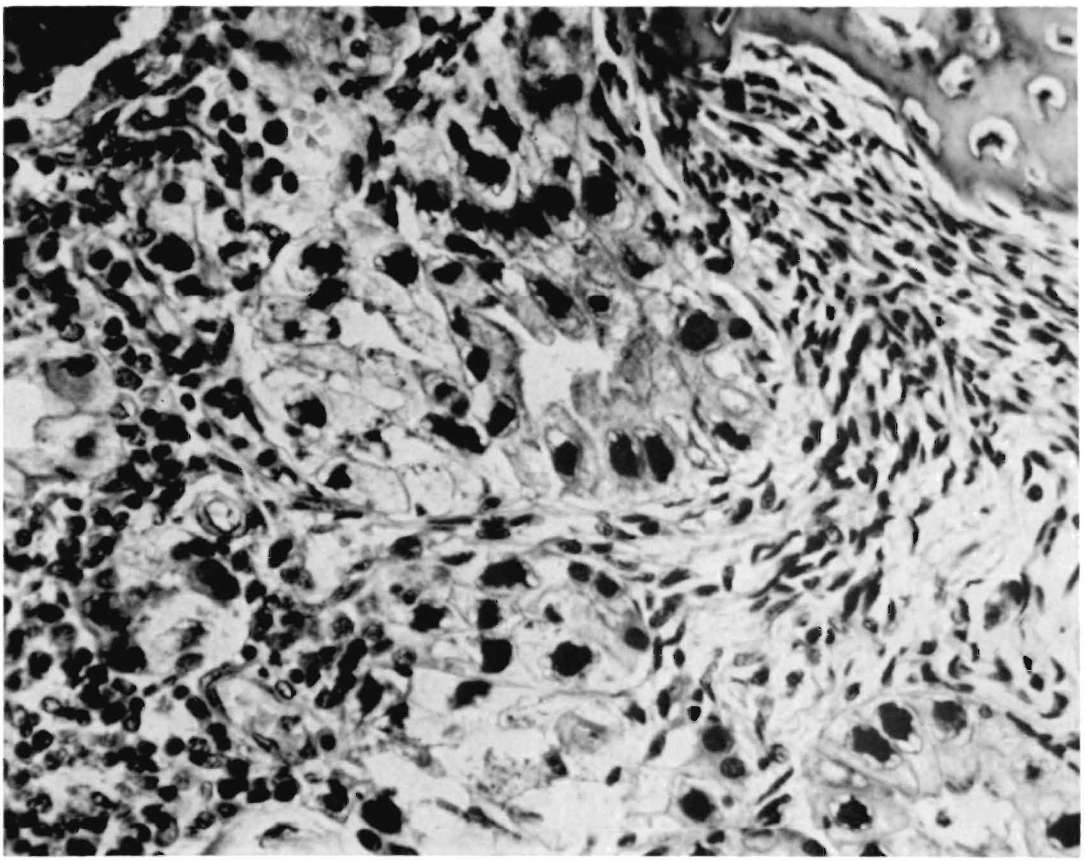

Fig. 2 Same section as figure one. The large nuclear inclusions are plainly visible. The nuclear membrane is distended beyond the inclusion. H&E. X420

had ever been observed by the local veterinarian. Therefore, it seems quite logical at this time to quarantine the affected herd and await further information on the occurrence and incidence of this disease in Iowa swine.

There is urgent need for an intensive study of this disease. If further work should prove that it has been recognized before and has become widespread in our swine population, then a good detection system would be extremely helpful in eradication efforts. As always, it becomes a problem of adequate facilities and support to attract the hands and minds to be trained so that they can adequately assist the practitioner and thereby the swine industry in eradication, prevention and control of swine diseases.

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