An Outbreak of "Inclusion-Body" Rhinitis in Pigs

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INTRODUCTION

THE PURPOSE of this paper is to describe a naturally occurring outbreak of so-called "Inclusion-Body" rhinitis in a herd of pedigreed Yorkshire pigs located in the Ottawa Valley. This condition was first described by Done (1) in 1955 in Great Britain.

Done et al (2) diagnosed 11 outbreaks of "Inclusion-Body" rhinitis in Great Britain from May to November 1954 and the senior author of this paper encountered two outbreaks in Scotland in 1955 (3). The need for histological confirmation of the diagnosis and the surprising equanimity with which pig breeders accept mortality in suckling pigs probably accounts for the fact that more reports on the conditions are not available especially in Canada and the United States where atrophic rhinitis is so widespread. As there has been no previous record of this particular entity occuring in North America it is our purpose to describe this outbreak and to briefly discuss its importance, principally from a clinical stand-point.

Clinical History

The outbreak occurred in September 1957. At this time 4 gilts, 2 sows and 1 boar made-up the entire breeding stock. All the females had been bred and raised on the premises, the boar was purchased in the spring of 1956 from an alleged rhinitis-free herd. No previous history of any condition resembling rhinitis was reported.

Three of the gilts had farrowed at the end of August within a few days of each other and had been permitted to run together. Their litters numbered 9, 10 and 14 pigs respectively. About one week after the owner noticed a number of the piglets scouring. These were examined and treated with parenteral iron and penicillin but in spite of this they grew progressively weaker and by the time the first visit was made 15 of the 33 piglets, which were now 3 weeks old, had died. Death was reported to occur about 4 days after onset of symptoms. In addition one of the sows had farrowed 5 piglets one week previously and her litter was also confine in the stable where indirect contact was possible.

Examination

The three affected litters with their dams were running together in one corner of the horse stable and were bedded down with liberal amounts of very dusty chopped oat straw. Even at rest it was observed that almost all the piglets were exhibiting dyspnoea and snuffling. On closer examination it was seen that there was a glary or mucopurulent nasal discharge present causing a variable degree of occlusion of the nares and consequent respiratory distress. Temperatures of affected pigs were slightly elevated (102.5 to 103.5° F.) and many showed evidence of diarrhoea. Anorexia was not apparent except in the worst affected pigs, two of which were taken on this day (Sept. 16) for post-mortem examination (Pigs 1 and 2). Inclusion bodies were demonstrated in the gland cells of the turbinate bones of these animals.

Treatment and Outcome

All the remaining piglets were given sulfamezathine (1½ grains per lb. body weight) orally and dihydrostreptomycin sulphate (0.2 grams per day) instramuscularily, for 3 successive days. In addition a solution containing Neo-

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Synephrine Hydrochloide*, 1 volume, and dihydrostreptomycin sulphate (1 gram per 10 cc.), 9 volumes, was prepared and 1 cc of this solution was instilled into each nostril daily for the next 5 days. No further losses occurred and when seen again on Oct. 9th snuffling was only evident in a few pigs — two of these were taken for post-mortem examination (Pigs 3 and 4).

On Nov. 14th, a piglet from the litter of the fourth gilt which was born on Nov. 4th, in a clean pen was sacrificed for examination. No gross abnormality was evident and histological study of the turbinates of this pig failed to reveal the presence of inclusion bodies. All the previously affected piglets were thriving on this date.

Further monthly visits were made but no evidence of clinical rhinitis was observed externally and in February 1958, i.e. at 5 months of age the first of the 23 survivors were marketed. The heads of 18 of these animals were obtained and examined for signs of pathological changes in the turbinate bones. No macroscopic abnormalities were observed.

Pathology

PIGS 1 AND 2

Gross Pathology — The nasal cavities of both pigs were filled with a copious quantity of yellowish creamy pus. No other significant lesions were noted.
Bacteriology — Examination of lung, liver, spleen, kidney, intestinal contents and nasal washings failed to reveal the presence of any recognized pathogens, only micrococci and coliforms organisms being isolated.

Histopathology — Histological examination was limited to the turbinates of the above two mentioned pigs. The pathology noted was essentially similar to that described by Done (1).

Paraffin sections stained with haematoxylin and eosin from both pigs were similar except that the destruction of surface epithelium and cellular infiltration were more severe in one. There was a definite tendency towards the formation of a squamous type epithelium. A heavy cellular inflitration of the propria and mixed glands was predominantly lymphocytic.

The most striking lesion noted was in the cells of the mixed glands and their ducts. The affected cells were greatly swollen, and located in the nuclei of these, were large deeply basophilic inclusion bodies. These inclusions, which appeared granular on high magnification and gave a "bird's eye" appearance to the nucleus, often contained one or more indentations with projections to the nuclear membrane. Within these indentations small eosinophilic hyaline-like bodies were often seen.

Glands so affected are destroyed and in many cases all that remains is a small pocket of pus.

PIGS 3 AND 4

Pig 3 — No abnormality was noticed grossly or histologically.

Pig 4 — *Gross Pathology* — Severe rhinitis with atrophy of the turbi-

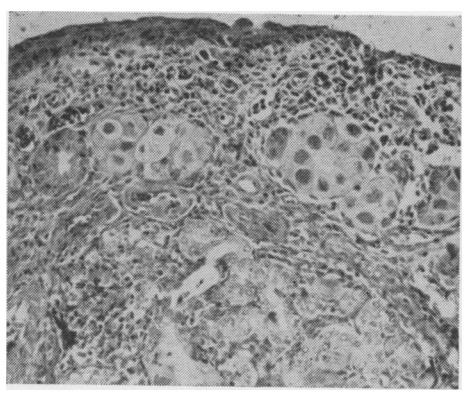

Figure 1 — Portion of nasal epithelium showing affected glands and the large intranuclear inclusions. H and E x 168.

*Tradename — Winthrop, contains 0.25% phenylephrine hydrochloride.

nates was present, the nasal passages being almost completely blocked by thick cheesy pus. Areas of marked inflammation were evident on the surface of the nasal septum and turbinate bones. No other significant lesions were seen in this carcase.

Bacteriology — Culture of nasal material yielded only coliform bacilli and micrococci.

Histopathology — The turbinate bones showed an extensive acute cellular infiltration and haemorrhage into the mucous membrane resulting in destruction of the surface epithelium. No inclusion bodies were seen.

Experimental Transmission

Unfortunately no material from Pigs 1 and 2 was made available for transmission into susceptible pigs. However, nasal material from pigs 3 and 4 was used to infect 6 day-old pigs. One of the experimental pigs was killed at 11 days and no atrophy of the turbinates was evident, however at 60 days when the others were killed they had developed atrophic rhinitis. No inclusion bodies were seen in either the experimental or normal control pigs.

Nasal material from the pig killed at 11 days was instilled into the nostrils of four day old pigs. One was killed 10 days later and showed no atrophy or inclusion bodies. The remaining three were killed at 60 days, one was normal, two showed an excess of mucus in the nasal passages and one of these showed some atrophv of the turbinates. No inclusion bodies were seen in any of these animals.

DISCUSSION

The most obvious point of interest is the relationship of this "Inclusion-Body" rhinitis to true atrophic rhinitis. Done (1) reported that he and his co-workers have been able to demonstrate typical inclusion-bodies in the early stages of naturally occurring and experimentally induced atrophic rhinitis. We have not yet been in a position to confirm this, although as has been described, atrophic rhinitis was produced in experimental pigs treated with material from pigs in the group where inclusion-bodies had been previously demonstrated. We too have been unable to demonstrate inclusion bodies in pigs over 4 weeks of age. Done (4) has stated that atrophic rhinitis can be considered an entity only from the point of view of morbid anatomy and it may well be that several etiological agents are involved of which "Inclusion-Body" rhinitis may or may not be one. He still holds that this entity is important as a breaching agent in atrophic rhinitis and that it is probably the principal one involved in cases of atrophic rhinitis in Great Britain. Whether the same conditions pertain in Canada is open to question, but there is little doubt in the authors' minds that there are other factors involved which precipitate the onset of clinical symptoms.

The following clinical observations were made during the course of the disease:-

(1) Conditions of poor hygiene and management were usually apparent, piglets being housed in damp, cold and dusty pens. Dusty bedding causing irritation of the nasal mucosa was thought to be a complicating factor in the case described.

(2) A concurrent or super-imposed disease condition such as enteritis or anaemia which could lower resistance appeared to precipitate the onset of clinical symptoms of "Inclusion-Body" rhinitis.

(3) Morbidity was high, approaching 100% and mortality varied according to the severity of the associated disease conditions and the rapidity with which corrective measures were initiated. In this case mortality was close to 50% but no further losses occurred following treatment. The efficacy of nasal instillation of a de-congestant-antibiotic mixture is open to question but it ap-

pears that death in some cases may be due to blockage of the nares and consequent asphyxiation; therefore attempts to relieve the nasal congestion appear to be justified.

(4) Inclusion bodies have not been demonstrated in pigs over 4 weeks of age and symptoms develop principally in animals from 2 to 4 weeks old.

The degree of infectivity and the viability of the causal agent cannot be accurately assessed but it would appear that it is only spread when litters of susceptible age are in fairly direct contact. In each of the two outbreaks seen in Scotland, both in large piggeries, only one litter was affected and in the present case only the 4 litters in the horse stable were affected. A litter born 2 months later on the same premises under improved management showed no evidence of the condition. Examination of the turbinate bones of the survivors of this outbreak revealed no abnormality and the time taken to reach market weight was not markedly prolonged.

SUMMARY

This paper describes the clinical picture encountered in an outbreak of "Inclusion-Body" rhinitis in a herd of Yorkshire pigs in the Ottawa Valley. Limited transmission experiments were inconclusive although atrophic rhinitis was produced with material from piglets whose litter mates had previously been shown to be affected with "Inclusion-Body" rhinitis. The histopathological picture is described. The clinical aspects and the etiology of the condition especially with regard to its relationship to true atrophic rhinitis are briefly discussed.

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REFERENCES

1. DONE, J.T. An "Inclusion-Body" rhinitis of Pigs (Preliminary Report) Vet. Rec., 67:525-527. 1955.
2. DONE, J. T., MUNRO, R. B. T. and HOLROYD, H. Unpublished work. 1954.
3. MITCHELL, D., GELLATLY, T. B. M. Unpublished work. 1955.
4. DONE, J.T. Personal communication. 1957.

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