PNEUMONIA IN CALVES ASSOCIATED WITH MIGRATING *ASCARIS SUUM* LARVAE

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Introduction

In addition to pigs, the infective larvae of *Ascaris suum* will establish in many animals (4, 6, 10, 15, 18) in which they migrate to the lungs, sometimes in large numbers, within a few days' time. In 1952 Taylor (17) suggested that fog-fever like signs may develop in calves infected with the larvated eggs of this nematode. After inoculation of *A. suum* eggs into calves, Kennedy (9) noted an increased temperature, a dry cough, as well as an increment in lung tones. Recently, Greenway and McCraw (7) were able to induce an atypical interstitial pneumonia in seven to 20-week-old calves which were given various dose levels of infective *A. suum* eggs. Naturally-occurring *A. suum* infections in calves have been reported also. In 1962, Allen (1) described an outbreak in which 15 yearling beef cattle displayed extreme respiratory distress ten days after being housed in pens where pigs had been raised. Later, Morrow (12) recorded a similar outbreak in 17 Holstein-Friesian heifers on a central New York dairy farm. Like the observations of Allen (1), acute signs of pneumonia occurred ten days after the heifers were exposed to ascarid ova. Both Allen (1) and Morrow (12) were able to identify *Ascaris lumbricoides* (*Ascaris suum*) larvae in lung sections or from bronchi.

This report is intended to correlate the clinical and pathological findings in a naturally-occurring ascarid infection of calves with previous experimental observations (7, 8, 11).

History and Clinical Signs

The owner of a small farm near Atwood, Ontario purchased on October 13, 1969, 20 calves (Hereford steers) aged nine to ten months and weighing approximately 350 lb each. These were housed in a pen which had been occupied previously by 34 pigs. The pigs had never been given anthelmintics and had been shipped to market one month earlier, apparently in good condition. Although an attempt had been made to remove the manure from the pen prior to placing the calves in it, the floor had not been washed or disinfected.

Seven or eight days after the calves were introduced into the pen, two developed respiratory signs characterized by dyspnea and an expiratory grunt which did not respond to penicillin-streptomycin[1] treatment given by the owner. Two days later (October 23) a veterinarian was called and all calves were given a sulfonamide-vitamin combination[2] in drinking water. In addition 12 were injected with a preparation containing dexamethasone, penicillin, streptomycin and chlorpheniramine[3]. At that time the rectal temperatures of the calves were normal or near-normal (two had a temperature of 103.5° F). The first calf died on October 24 and all were treated again with Azimycin. Late the following night a second calf died. The next day four calves were treated with a fluroprednisolone-neomycin preparation[4] in addition to a product containing phenylephrine and methapyrilene[5]. All the various antibiotic, corticosteroid and antihistamine treatments were evidently without effect, and on October 28 three more deaths occurred and a sixth calf died on November 6.

The farm was visited by us on October 27, about six days after clinical signs were first noticed; by this time most of the calves were in extreme respiratory difficulty and several were recumbent. Coughing, dyspnea as well as forced expiratory grunt were generally observed and two were unable to stand. Respiratory rates as high as 130 per min and a heart rate of up to 118 per min were recorded. Lung sounds were greatly increased.

Eight samples of bedding were collected from the pen and *A. suum* eggs were identified in six. The owner was advised to add considerably more clean bedding to the pen to cover

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[1]Fortimycin, Ayerst, McKenna and Harrison Limited, Montreal, Quebec, Canada.

[2]Extra-Sul, Diamond Laboratories, Downsview, Ontario, Canada.

[3]Azimycin, Schering Corporation Ltd., Pointe Claire, Quebec, Canada.

[4]Predef 2X, Tuco Products Co., Orangeville, Ontario, Canada.

[5]Pyrahistine with Phenylephrine, Pitman-Moore Ltd., Don Mills, Ontario, Canada.

the eggs and thereby minimize further infection.

Gross and Microscopic Findings

The first calf that died was submitted to the Ontario Veterinary College on October 24 for necropsy. The lungs were firm, light to dark red in colour with edema present in the interlobular septa of all lobes. Emphysema was widespread and prominent bullae were observed along the ventral borders of the lobes, especially the diaphragmatic lobes. The cut surface of the lung was bright red and presented an unusual meaty appearance. Larvae were seen by direct microscopic examination of froth expressed from bronchi. A 200-gram sample of lung was cut finely and placed in a Baermann apparatus containing 0.85% saline at 42° C. A calculated 21,000 third-stage *A. suum* were recovered from this sample indicating possibly as many as 5,700,000 larvae were present in the lungs. Many of these larvae were in the late third-stage of development (5).

Clusters of as many as 15 sections of a third-stage. *A. suum* larvae (13) were readily located in the bronchioles (Figure 1). Neutrophils and mononuclear cells usually surrounded the larvae. The epithelium of some bronchioles was normal except for the presence of leucocytes that were occasionally found in large numbers in certain segments of the epithelium. In others, an acute bronchiolitis was evident. However, by far the most severe lesions were associated with the alveoli. Their walls were thickened and lined with fibrin. The lumina of the alveoli contained a proteinaceous fluid, and were packed with mononuclear cells many of which were typical "foamy macrophages" (Figure 2). These lesions were frequently replaced by areas of diffuse hemorrhage. Eosinophils were seldom observed in the alveoli. The interlobular septa were edematous and their lymphatics greatly inflated (Figure 3). Although plasma cells and eosinophils were present in the septa, they were not prominent elements. Many of the larger arteries were isolated from the lung parenchyma by wide spaces and the pleura was often well separated from the lung surface (Figure 3).

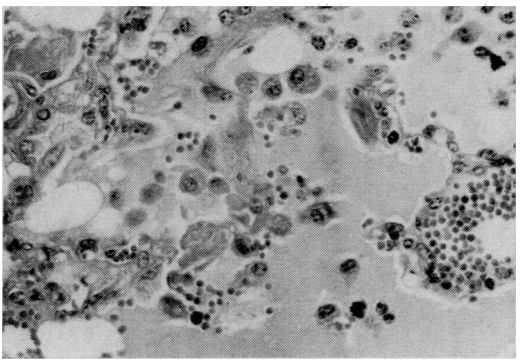

Figure 2. Severe disruption of alveolar architecture with the exudation of a proteinaceous fluid and mononuclear cells. ×230.

The severe congestion of the liver was reflected by its "nutmeg" pattern. Eight larvae were recovered from a 200-gm Baermann preparation.

A previously affected calf was examined at necropsy on December 30, about ten weeks after clinical signs first appeared. Except for atelectasis in various parts of the right cardiac and apical lobes, the lungs were essentially normal in appearance. The right diaphragmatic lobe was rubbery. Sections of the apical and diaphragmatic lobes revealed the following lesions: widespread muscular hyperplasia of the bronchioles, including the terminal bron-

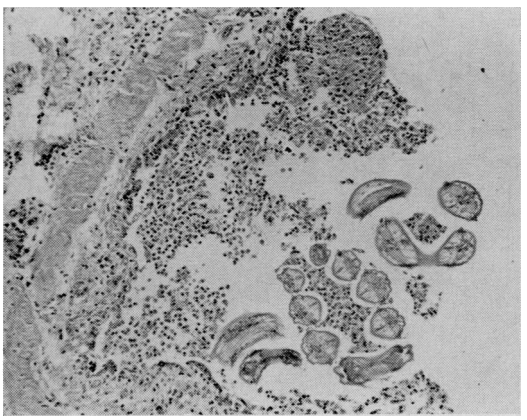

Figure 1. Clusters of third-stage *Ascaris suum* larvae in a bronchiole. ×60.

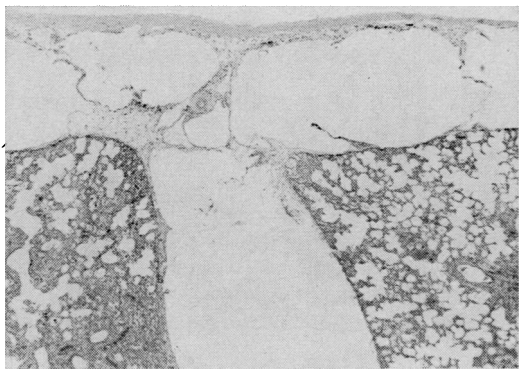

Figure 3. Subpleural, interlobular and alveolar emphysema of *Ascaris suum*-infected lung. ×25.

chioles, hyperplasia of the bronchiole epithelium, and atelectasis and thickening of the alveolar walls. These changes in the alveoli alternated with areas of normal alveolar architecture. No larvae were found.

Discussion

The clinical signs seen in the outbreak reported here were very similar to those induced in calves infected experimentally with larvated *A. suum* eggs (7). Greenway and McCraw (7) found that signs in calves attained maximum severity between the tenth and 13th days after infection, and consisted of dyspnea often with expiratory grunt, coughing and mouth breathing. The time at which signs appeared was about the same regardless of whether the experimental infection was primary or secondary. Emphysema was also generally present, and both respiration and heart rate increased following infection, the former reaching its height around the eight or ninth day and the latter between the tenth and 14th days post-infection. The clinical signs seen in severe *A. suum* infection in calves are consistent with those seen in atypical interstitial pneumonia (AIP), a term introduced by Blood (3) in 1962.

The fact that two calves developed signs within seven or eight days after their introduction into the pen indicates that the bedding was heavily contaminated resulting in the calves becoming infected very quickly. Experimentally, signs first appeared on the sixth or seventh day after infection (7). An important aspect of the spread and development of ascarid pneumonia is that the egg production of *A. suum* is high; estimates of the daily output of a female worm range from 100,000 to nearly 2,000,000 eggs (14, 16). Moreover, the eggs of *Ascaris* are believed to survive as long as six years (2). Thus, large numbers of eggs may accumulate and remain viable in areas where pigs have been confined.

Nearly all the gross and microscopic lesions seen in the lungs of calves dying as a result of experimental infections (11) were also observed in this outbreak. Fewer eosinophils were found in sections of lungs than in those of the experimental calves that died, possibly because the reaction to larvae was so acute that eosinophil reserves were temporarily exhausted. Unfortunately, in this outbreak, total circulating eosinophil counts were not made prior to death, but previously (8) when a calf died following experimental *A. suum* infection there was very little increase in these cells. On the day of its death one experimental calf had a count of only 493 per cu mm (8). However, animals which survived often had very high eosinophil counts 11 to 14 days after infection. The recovery of many *A. suum* larvae from the lungs as well as their abundance in sections corresponds with earlier findings. Our experience has been that large numbers of larvae may be recovered from calves that die following infection with this nematode, in sharp contrast to the failure to find larvae from animals that survive and are examined later at necropsy (11).

Evidently the lungs of calves infected with large numbers of *A. suum* larvae eventually undergo considerable repair. However, the extent of repair is variable and possibly depends on the number of eggs ingested as well as the length of time during which the calves are exposed to infection. More extensive lesions were present in the naturally-infected calf examined at necropsy ten weeks after the onset of clinical signs than in a calf examined 27 days after the last experimental inoculation (three doses totalling 1,000,000 eggs) with infective *A. suum* eggs (11). The surviving calves from the present outbreak were reported to be doing well eight months after becoming ill.

Summary

An outbreak of pneumonia in calves naturally infected with large numbers of *Ascaris suum* larvae is described. Within ten days of being placed in a pen previously occupied by pigs, the calves developed severe respiratory signs consisting of dyspnea, expiratory grunt and increased respiratory rate. At necropsy the lungs were firm and bright red, and there was severe edema and emphysema. Many third stage *A. suum* larvae were observed in the bronchioles and alveoli. The walls of the alveoli were thickened and their lumina contained a proteinaceous fluid, mononuclear cells and a few eosinophils. These changes were accompanied by areas of diffuse hemorrhage into the alveoli. Six of 20 calves died.

Résumé

Les auteurs font part d'une manifestation subite de pneumonie chez des veaux victimes d'une infestation naturelle massive par des larves d'*Ascaris suum*. En moins de 10 jours après avoir été placés dans un enclos préalablement occupé par des porcs, ces veaux manifestèrent des troubles respiratoires graves consistant en dyspnée, expiration bruyante et respiration accélérée. A la nécropsie, les poumons s'avérèrent fermes et de couleur rouge

brillant, fortement œdémateux et emphysémateux. Les bronchioles et les alvéoles contenaient plusieurs larves du troisième stade d'*A. suum*. Les parois des alvéoles se montraient épaissies et la lumière alvéolaire renfermait un fluide protéinique, des monocytes et quelques éosinophiles; des foyers hémorragiques alvéolaires diffus accompagnaient ces changements. Six des 20 veaux sont morts.

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ABSTRACT

Stern, J. S., Antoniades, H. N., Baile, C. A. & Mayer, J. (1969). Insulin-like activity in goat serum.—Endocrinology 85, 976–981 (Dep. Nutrition, Harvard Sch. Public Hlth, Boston, Massachusetts 02115).

Studies indicated that goat serum contains at least two forms of insulin-like activity. One form can be immunoassayed, has a molecular wt. of 12,000, and its level is increased by i/v administration of glucose to goats: it seems to be similar to pancreatic insulin. The second form is undetectable by radioimmunoassay in vitro, has a molecular wt. of 30,000–50,000, and tends to decrease transiently after i/v administration of glucose. When given i/p to rats, the latter form promotes uptake of labelled glucose into the diaphragm and epididymal fat and glucogen. This activity is inhibited by anti-insulin antiserum. The reason why there is no reaction in the radioimmunoassay is not known.

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