AN OUTBREAK OF MENINGITIS IN SWINE CAUSED BY HAEMOPHILUS SPECIES OF BACTERIUM

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THE PURPOSE of this paper is to present the history, clinical, pathological and bacteriological findings in an outbreak of a disease in a group of feeder pigs characterized primarily by meningitis and rapid death apparently due to a member of the *Haemophilus* species.

HISTORY

In November, 1962, the owner of a moderately large swine farm became alarmed when he found two dead and many sick pigs in a pen containing four-month-old feeder pigs, each weighing about 150 lb. The owner assumed that his pigs were poisoned because just three days earlier he had mixed a new batch of feed. The feed mixture consisted of home-grown grains (oats and barley) on which the pigs had been fed previously and a very recent shipment of hog concentrate. The owner stated that the pigs were depressed, would not eat, moved about the pen slowly and died in six to twelve hours. On arrival at this clinic with a typically affected pig, still alive, the owner stated the disease had been present about 24 hours and nine pigs had died. One pig was reported to be acutely lame, the others were not.

The owner also related that a group of 30 recently weaned pigs in an adjacent pen and a group of six gilts and two boars in another part of the barn were all receiving the same feed, but were apparently unaffected.

CLINICAL FINDINGS

The pig presented for necropsy was examined clinically before euthanasia. It was in lateral recumbency, made struggling attempts to rise but could stand only when lifted and supported by the tail. While supported, it walked very slowly with obvious incoordination of the hind legs. When the tail was released, the pig collapsed into lateral recumbency and slight superficial muscular tremors were noted. The skin colour was normal over the entire body. The temperature was 106.5° F., heart rate 160 per minute, and a clearly audible pericardial friction rub was heard over the right cardiac area. The respirations were normal and the lungs were normal on ausculation. The feces were scant and firm. The hock joints were slightly swollen, were painful to touch and an increased warmth could be appreciated.

A necropsy was carried out on this pig (designated number 1), the results of which are given below under pathology. The disease in this pig was clearly infectious but the agent could not be immediately specified and since this was the only pig in the group with signs of polyarthritis there arose some doubt as to whether or not it exemplified the disease in the whole group. Therefore,

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it was decided to make a visit to the farm the next day to make further observations and obtain more material for diagnostic purposes.

On arrival at the farm, the owner stated that five more pigs had died in the last 24 hours (a total of 14 pigs in 48 hours) and an additional 15 pigs were affected and expected to die in a few hours. The owner appeared to be a good herdsman, farm conditions were good, and the barn and yards were exceptionally well cared for.

The affected pen contained about 40 pigs, approximately four months old, and at a glance it was obvious that they were doing very well as feeder pigs since they were in good bodily condition. In the alleyway adjacent to this pen, there were five pigs, still alive, in lateral recumbency, and which had been removed earlier from the affected group. These five made no attempt to rise when approached and tended to huddle very close to each other. Clinical examination of the most severely affected pig which was in an almost moribund state revealed the following: paddling of the limbs was marked, muscular tremors of the head and neck regions occurred intermittently lasting only a few seconds, blindness was evidenced by the absence of the eye preservation reflex to an approaching finger thrust, the pupils were widely dilated and reacted poorly to a strong light, a small amount of tenacious serous ocular discharge had accumulated at the medial canthus of the eye and "matted" the adjacent hairs, the temperature was 100.2° F., the heart rate 140 per minute, and the lungs were normal on auscultation. The feces were firm and scant. The skin colour was normal. The pig made no attempt to stand and could not stand even when assisted. The remaining four pigs were lying on their sides, did not respond to external stimuli, and could not stand when assisted. Three were apparently blind, muscular tremors of the head and neck occurred every few seconds, and their temperatures ranged from 105° and 107° F. The heart rates ranged from 100 to 140 per minute. The respirations were slower than normal and ranged from eight to fifteen per minute.

Examination of the pigs in the affected pen revealed most unusual findings. The pigs could be divided into three groups clinically. About one-third of the group was normal. Another third of the pigs moved slowly, were obviously depressed, responded poorly to external stimuli, were empty in the abdomen, had temperatures ranging from 105° to 107° F. and heart rates up to 140 per minute. The third group, probably more advanced, were reluctant to stand, preferred to lie in sternal recumbency, were indifferent to external stimuli, were apparently blind, appeared relatively empty in the abdomen, had temperatures of 104° to 106.5° F. and heart rates of 120 to 140 per minute. These pigs stood only with a great deal of persuasion and made very little attempt to walk. While standing, two of them collapsed into lateral recumbency while the examiner was some distance away. At this time, a slight paddling of the limbs and muscular tremors were noted. The paddling occurred in periods lasting up to a minute and between these periods, the animals lay quietly. Their temperatures were 106° F. and heart rates 130 to 140 per minute.

In summary, the major clinical features were high temperature, rapid heart rate, and inactivity, which was the most noticeable initial sign, was followed very quickly by recumbency, paddling, muscular tremors, blindness and death.

Three of the dead pigs were necropsied on the farm (pathology Nos. 2, 3, 4) and their heads were removed and returned to the Ontario Veterinary College. In addition, three severely ill pigs were returned to the College; two of these died soon after arrival (pathology Nos. 5 and 6) and the third pig, still able to walk and with a temperature of 106° F. was treated with two million units of procaine penicillin G and 2.5 gm. of dihydrostreptomycin intramuscularly. Twelve hours later this third pig appeared normal and remained so.

Pathology

Gross pathological findings on number 1 were different from those in the remaining five pigs. In this first pig, there was severe fibrino-haemorrhagic pericarditis with scant fluid exudation, and severe fibrino-purulent polyarticular synovitis. The lungs were erect and uniformly hyperaemic, cyanotic, and oedematous with patchy alveolar haemorrhages. The brain was examined microscopically and showed no sign of injury. Pericardial fluid, joint fluid and spleen were submitted for culture with special attention given to the possible presence of *Haemophilus*.

Pigs number 2 to 6 showed a uniform pathologic picture. Serous and synovial membranes were normal, grossly and microscopically. The lungs did not collapse, were hyperaemic, intensely oedematous with distention of interlobular septa, and contained subpleural and parenchymal haemorrhages. There was an extensive accumulation of fibrinopurulent exudate in the leptomeninges of brain (Fig. 1) and cord, and the ventricular fluid was turbid.

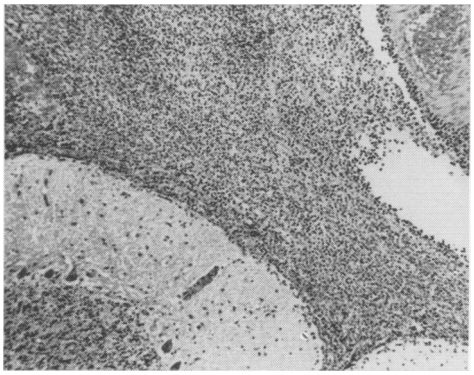

Figure 1. Fibrinopurulent meningitis of the cerebellum.

Microscopically, there were intense focal accumulations of neutrophils in intertubular capillaries of the renal cortices with acute necrosis of adjacent tubules, hypercellularity of the glomeruli and occasional thrombi in interlobular veins. The lungs were uniformly hyperaemic with extravasated red cells in most alveoli and in distended lymphatics. There was a diffuse sequestration of leucocytes in alveolar capillaries. The meningitis was copiously fibrinopurulent and a small amount of exudate was present in the ventricle. Direct invasion by neutrophils across the pia mater and into the superficial cerebral

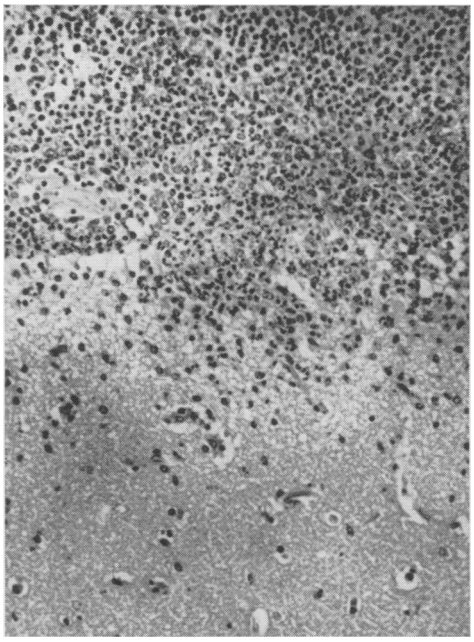

FIGURE 2. Fibrinopurulent exudate and invasion by neutrophils across the pia mater into the superficial cerebral parenchyma.

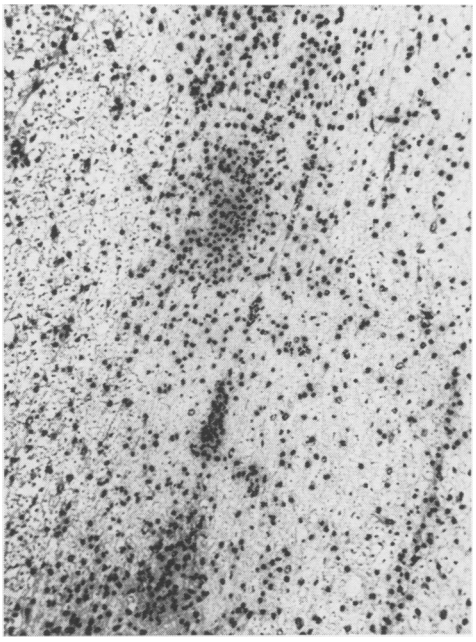

FIGURE 3. Perivascular invasion of spinal cord leading to early focal softening.

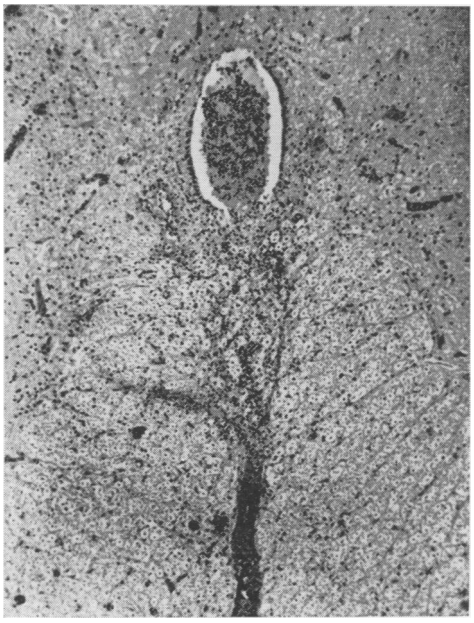

FIGURE 4. Infiltration of the ventral sulcus by neutrophils and ependymitis caused by extension of the inflammatory process into the central canal.

parenchyma (Fig. 2) was more extensive than is usually seen in meningitis but there was scant invasion via Virchow-Robin spaces. Parenchymal invasion was

even more prominent in the spinal cord where perivascular invasion led to early focal softening (Fig. 3) and ependymitis developed in several places by extension of the inflammatory process from the ventral sulcus (Fig. 4).

Bacteriology

Pericardial fluid, joint fluid and spleen of pig number 1 and brain, liver, lung, kidney and spleen of pigs number 5 and 6 were presented for bacterial examination. Direct smears were made from the pericardial fluid, joint fluid and spleen of pig number 1, and from the meninges of pigs number 5 and 6. All smears were stained by Gram's method. Small Gram-negative rods were observed in the spleen of number 1, and in the meningeal smears of the other two pigs.

Tissues were cultured on 5.0 per cent citrated sheep blood agar plates with a staphylococcus streak and incubated at 37° C. for 48 hours. Tiny dewdrop colonies with a diameter of 0.5 to 1.0 mm. were observed adjacent to the staphylococcus streak on the culture plates from the spleen and joint fluid of pig number 1 and from the brain of pigs 5 and 6. Growth was not obtained from the other organs. A smear of these colonies revealed small Gram-negative rods with many long, tangled filaments. This organism was maintained by subculturing at 48-hour intervals on sheep blood agar with a staphylococcus streak.

Subculture was made as soon as possible after isolation to the following test media: Tryptose agar plates with staphylococcus streak, trypticase soy broth[1] trypticase soy broth with 5.0 ug/ml. coenzyme I[2] added. The organism grew adjacent to the staphylococcus on tryptose agar; and the trypticase soy broth with added coenzyme I supported growth. Growth did not occur on sheep blood agar or tryptose agar without the addition of staphylococcus, or in trypticase soy broth without coenzyme I.

Fermentation reactions were determined in phenol red broth base[3] with 0.5 per cent of the carbohydrate to be tested. Coenzyme I (5.0 ug/ml.) was added to each tube. Results were read after five days incubation at 37° C. Acid but no gas, was produced in dextrose, dextrin, levulose, mannitol, mannose, maltose, sucrose and xylose. The organism failed to ferment arabinose, dulcitol, galactose, inositol, inulin, lactose, raffinose, rhamnose, salicin, sorbitol or trehalose.

This organism did not require the "X" factor; growth was obtained on tryptose agar with the staphylococcus providing the "V" factor, and in trypticase soy broth with only coenzyme I added. Satellitism was exhibited in association with staphylococcus. This organism was probably a member of the *Haemophilus parainfluenzae* group of bacteria.

Subsequent History

When it was decided that a meningo-encephalitis was present as evidenced by pathological findings and the bacteriological results indicated a *Haemophilus* species bacterium, all pigs on the farm were treated with a high therapeutic

[1]Baltimore Biological Laboratory, Inc., Baltimore 18, Maryland.
[2]Cozymase, Nutritional Biochemicals Corporation, Cleveland 28, Ohio.
[3]Baltimore Biological Laboratory, Inc., Baltimore 18, Maryland.

dose of a combination of procaine penicillin G and dihydrostreptomycin. All affected pigs were back to normal in 24 hours, no deaths were reported and no further cases developed.

An accompanying paper describes a disease in young pigs attributed to a similar organism characterized by a bacteremia (1).

Summary

An outbreak of meningo-encephalitis in swine of high mortality and morbidity is described. Clinically, the disease was characterized by a high fever, inactivity and the rapid development of weakness and recumbency, death occurring in a few hours. A uniform pathologic picture was seen and consisted of a fibrino-purulent meningitis with extension into the cerebral parenchyma. Bacteriological cultures revealed a *Haemophilus* species bacterium designated as probably belonging to the *Haemophilus parainfluenza* group.

Affected pigs treated with penicillin and streptomycin recovered in 12 to 24 hours and no further cases occurred.

Résumé

L'auteur décrit une éruption de méningo-encéphalites à forte morbidité et mortalité dans un élevage de porcs. Cliniquement, la maladie se manifeste au début par des températures élevées, suivie rapidement de faiblesse progressive, avec la mort survenant en quelques heures. Les lésions observées étaient uniformes dans tous les cas et consistent en une méningite fibrino-purulente avec atteinte au parenchyme cérébral par continuité. Un organisme du type *Hémophilus*, probablement du groupe *Hémophilus parainfluenza* a été isolé.

Les porcs malades, traités avec la combinaison pénicilline-streptomycine, ne montraient plus de symptômes après 12 à 24 heures.

Reference

1. Thomson, R. G., and Ruhnke, H. Louise. Haemophilus septicemia in piglets. Can. Vet. Jour. 4: 271. 1963.

ABSTRACT

"Studies of Proteus Organisms of Canine Origin". G. Fraser. J. Comp. Pathology. 73: 9. 1963.

This paper describes the characters of a number of strains of Proteus isolated from dogs and reports on a method of determining their antigenic structure which is useful in tracing the spread of infection. Antibiotic sensitivity was unsatisfactory because of the characteristic swarming type of surface growth. Proteus bacilli were not recovered from the external ears, the middle ears or the anterior nares of healthy dogs but 40 per cent harboured the organism in their rectum. However, the incidence of *Proteus Mirabilis* in the rectum is higher in dogs harbouring the organism in the ears and is usually associated with a chronic otitis. The infection with *Proteus Mirabilis* is probably due to contamination from an external source or to auto-infection from the intestines; there is no evidence that ascending infections occur in adult dogs. *H.C.R.*