An Encephalomyelitis of Suckling Pigs in Ontario

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INTRODUCTION

Since November 1957, a disease affecting the nervous system of suckling pigs has been observed in Southern Ontario. Outbreaks involving 44 separate herds have been investigated by the Ontario Veterinary College, 33 of these having occurred since November 1958. In most outbreaks a clinical examination was made and 84 individual pigs (at least one from each outbreak) were necropsied.

The purpose of this paper is to record the occurrence of the disease, and the clinical findings in affected pigs, with a brief note on blood changes and necropsy findings. Details of the pathology and experimental data will be published later (1).

CLINICAL OBSERVATIONS

(1) *OCCURRENCE*: The outbreaks investigated seemed unrelated to breed, husbandry or feeding practices, but there was a marked age incidence. The disease was most common and severe in pigs under 2 weeks of age. Older suckling pigs were also affected, but less severely, and in at least 3 outbreaks sows suckling affected litters became mildly and transiently ill.

The morbidity amongst suckling pigs was variable both in the relative number of litters affected in a herd, and in the number of pigs affected within a litter. In many younger litters every pig was affected, but in some outbreaks in which the litters were older, only a few pigs in the entire herd showed clinical signs.

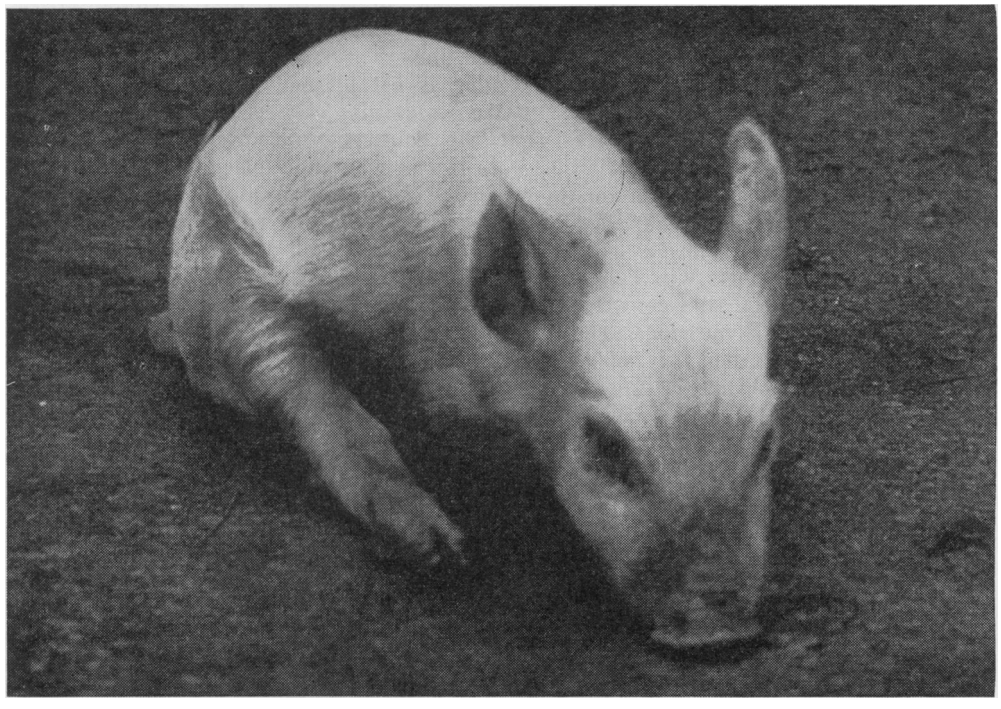

Fig. 1. An affected pig in a characteristically awkward posture.

The mortality rate was low in older litters (over 3 weeks of age) but within the very young litters it often approached 100%.

(2) *CLINICAL SIGNS*: In most cases the onset of clinical signs appeared in several litters simultaneously.

The first signs were usually complete or partial anorexia, constipation, staring coat and lethargy. The affected pigs huddled together in the straw. In many cases vomiting occurred, especially after feeding or dosing. It often continued intermittently for one or two days, but it was rarely severe and clinical signs of dehydration were not pronounced. A raised temperature was noted in some of the pigs, but in the majority the temperatures were normal. Terminally they were sub-normal.

As the disease progressed the nose and feet tended to become slightly cyanotic and a waxy brown discharge often accumulated at the inner canthus of the eye. Sometimes the abdomen became tense and appeared distended. At this, or at later stages of the disease, grinding of the teeth, accompanied by a bubbly salivation, commonly occurred. Occasionally a subdued sneezing, coughing, or upper respiratory embarrassment was observed, and some pigs were reported to root frantically in the straw prior to the onset of nervous signs.

The most consistent clinical signs were those typical of a severe encephalomyelitis. Sometimes these appeared at or near the onset of the illness, or, more commonly, after the illness had progressed for one to three days. The nervous signs varied both in severity and type, the variation depending to a certain extent on age. Older suckling pigs (4-6 weeks) usually suffered a mild transient illness in which posterior paresis was the most common sign. Their gait became drunken and swaying. In a few cases the paresis was accompanied by blindness.

The youngest suckling pigs (i.e. 2-10 days) were affected most severely, exhibiting various combinations of nervous signs. Hyperesthesia was a common finding and was manifest in an exaggerated reaction, such as rapid paddling and squealing, in response to sudden movement, noise or touch. The squealing varied in pitch and intensity. In some cases hyperesthesia was the only clinical sign of nervous involvement, the pigs appearing tense and anxious. Generalised muscle tremor was very common.

The gait of the younger pigs which were able to stand was usually stilted and jerky, sometimes with knuckling at the fetlocks. Paresis and ataxia occurred, and there was a tendency to walk backwards like a horse backing a load, often ending in the pig adopting a dog-sitting position or falling on its side.

When lying in sternal recumbency affected pigs tended to adopt awkward postures with the legs in unnatural positions, somewhat in the manner of pigs suffering from bowel edema (Fig. 1).

In later stages, pigs became prostate, lying on their sides and paddling with their legs; this was sometimes accompanied by blindness and nystagmus. Opisthotonus was uncommon, but dyspnea often occurred. In many cases coma preceded death.

Between the mild disease, typical of older pigs, and the severe disease, typical of the very young pigs, all degrees of severity occurred.

(3) *SEQUEL*: The duration of most outbreaks was brief, rarely longer than two weeks. The majority of pigs which survived the first three to five days of illness recovered in seven to ten days. The recovery of some pigs showing nervous signs was remarkable for its speed and completeness.

Litters that were not affected during the first week of the outbreaks, and litters born two or three weeks after the outbreaks began, usually remained healthy. No recurrences were reported in herds where outbreaks had occurred, and the subsequent litters of sows whose litters had been involved in an outbreak were usually healthy.

CLINICAL PATHOLOGY

Total white cell counts were made on blood samples from 25 affected pigs. Two were below 5,000/cmm, (subnormal) (2) (3), one was 22,000/cmm and all the rest were between 5,000/cmm and 15,000/cmm.

Urea nitrogen estimations were carried out on blood samples from 36 affected pigs. Six were between 20-30 mgs % (within the normal range) (4) (5) eleven were between 30-60 mgs % (slightly high), nine were between 60-100 mgs %, and ten were over 100 mgs %, one being as high as 240 mgs %. No estimations were made of hemoconcentration.

NECROSPSY FINDINGS

At gross post-mortem examination no significant lesions were observed, but in every pig necropsied, a histological examination of the central nervous system revealed a non-suppurative encephallomyelitis. This was characterized by perivascular cuffing with mononuclear cells, neuronal degeneration and microglial cell nodules. Both grey and white matter were affected, but the former was the more severely involved. The lesions were most extensive in the brain stem and spinal cord. Inclusion bodies were not observed.

BACTERIOLOGICAL EXAMINATION

Bacteriological cultures were made from the spleen, small intestine and brain of piglets killed immediately prior to necropsy. No bacterial species was consistently found, and in most cases, the spleens and brains yielded no growth on blood agar and MacConkey's medium. The brains from six affected piglets were cultured anaerobically and a special search was also made in these brains for *Listeria monocytogenes*, with negative results.

TREATMENT AND CONTROL

Various therapeutic agents, including most of the common antibiotics, have been used with no apparent alteration in the course of the disease.

Farmers have been advised to isolate their susceptible unaffected pigs and pregnant sows which are close to farrowing, and to disinfect the pens where affected pigs have been. Because of the danger of the spread of the disease, and because no recurrences have been recorded following the disappearance of the disease from a farm, farmers have also been advised against selling sows whose litters have been affected.

DISCUSSION

The cause of the disease described in this paper is unknown, but the histopathological lesions suggest that it is a virus.

The clinical syndrome in some outbreaks of Aujesky's disease in piglets (6) (7) closely resembles the disease described here. However, neither the convulsions nor the inclusion bodies observed in cases of Aujesky's disease have been observed here.

The clinical syndrome in Talfan disease (8) is also similar. The virus causing Talfan disease has been shown to be closely related to the viruses causing Teschen disease and poliomyelitis suum (9), although these latter two diseases differ somewhat from Talfan disease in their clinical syndrome and age incidence (10) (11).

Rabies and hog cholera cause signs of encephalomyelitis in swine, but affect a wider age group and produce clinical signs not observed in this disease. Hog cholera is not endemic in Ontario.

Transmissible gastroenteritis (TGE) has a high morbidity and mortality rate in pigs under 10 days of age, but in TGE the most consistent clinical sign is scouring, and no encephalomyelitis has been reported (12) (13). TGE is not known to occur in Ontario.

Bacteria have been tentatively ruled out as the cause of this disease because of the histopathological and bacteriological findings. However, where only one litter is affected, it may be extremely

difficult for the practitioner in the field to distinguish clinically between this disease and bacterial meningoencephalitis caused by bacteria such as Listeria, streptococci, or *Escherichia coli*. Meningoencephalitis due to bacteria is often accompanied by other lesions and clinical signs (e.g. arthritis caused by streptococci, or scouring caused by *E.coli*). The bacterial conditions may show some response to antibiotic therapy.

Hypoglycemia may cause a similar syndrome in very young pigs (14) (15). Nervous signs are sometimes present, but often the only clinical signs is "sleepiness". Pigs suffering from hypoglycemia usually respond to injections of glucose.

At the Ontario Veterinary College, a condition which has become known as "vomiting and wasting disease" has been tentatively differentiated from the disease described herein, although there is no proof that the two are separate entities. In "vomiting and wasting disease" the vomiting is typically more severe, lasting often for a week or more, and resulting in extreme dehydration and emaciation. "Vomiting and wasting disease" pursues a more chronic course and few, if any affected pigs, recover. Nervous signs or lesions have not been reported.

In 1944, Madsen *et al* (4) described a uremic condition of baby pigs, and uremia has been observed in TGE (5) and "vomiting and wasting disease" (16). The high levels of blood urea recorded in the disease described here have not been explained.

SUMMARY

Forty-four outbreaks of an encephalomyelitis of suckling pigs have been investigated by the Ontario Veterinary College. The disease appeared to affect suckling pigs only. It was characterized by a high morbidity and mortality which decreased rapidly with age, and by pronounced clinical signs of a C.N.S. derangement. A detailed description of the clinical observations, and brief notes on the clinical pathology and necropsy findings, as well as a discussion on the differential diagnosis and possible cause, have been given. The cause is unknown.

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