CLOSTRIDIUM PERFRINGENS TYPE C ENTEROTOXEMIA OF THE NEWBORN PIG*

H. W. Moon† and M. E. Bergeland†

Clostridium perfringens type C is known to cause a necrotic, hemorrhagic enteritis in neonatal lambs, calves and pigs (1, 2, 3, 4, 8). It is also the cause of "struck" in adult sheep (5) and it has been reported to cause necrotic enteritis in fowl and enteritis necroticans in man (6, 10). The agent in man and fowl was originally designated as type F, but has since been classified as type C (7). It is apparent that *Cl. perfringens* type C is associated with diseases of the intestinal tract. The chief pathogenetic mechanism of *Cl. perfringens* is a necrotoxin, designated the beta toxin.

The disease in newborn pigs occurs in Great Britain, Europe, and North America. The first confirmed diagnosis of the disease in Minnesota swine was in 1962. Since that time it has been diagnosed several times annually on Minnesota and Iowa farms. It is likely that the disease occurred prior to that time, but was not recognized.

Cl. perfringens type C enterotoxemia affects pigs chiefly during the first week of life, but does affect pigs up to one month of age. In a typical enzootic, pigs are normal at birth and develop a diarrhea during the first or second day of life. The feces usually contain blood. Most affected pigs die within 12 to 48 hours after the onset of the bloody diarrhea. Some individuals rapidly become depressed and dehydrated, others remain active and alert until shortly before death. Usually almost all litters born during an enzootic are affected. Frequently only a portion of a particular litter develops the disease. The disease may recur on the same premise during subsequent farrowing seasons. This has been reported even after the introduction of new breeding stock from premises where the disease had not been known to occur (1).

At necropsy, there usually is staining of the perineum and tail with bloody feces (Figure 1). A segment of small intestine is thick walled, necrotic and usually hemorrhagic (Figure 2). Occasionally there is

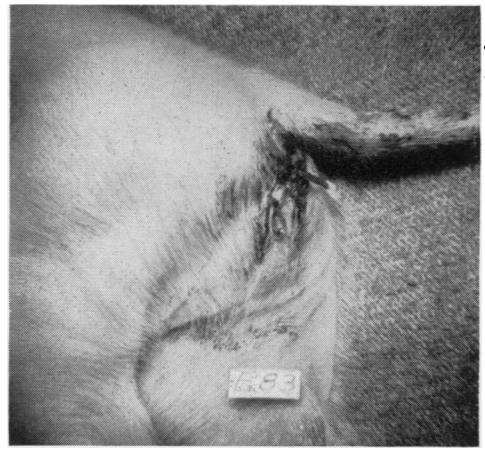

FIGURE 1. The rear quarters of a newborn pig with *Cl. perfringens* type C enterotoxemia. The perineum is soiled with bloody feces.

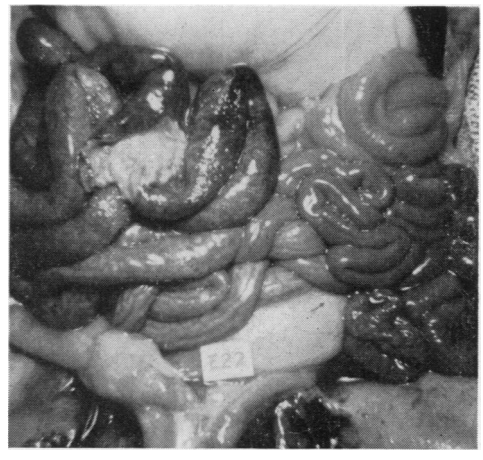

FIGURE 2. The intestinal tract of a newborn pig with enterotoxemia. A segment of the jejunum is necrotic, the mucosa is thickened, there is hemorrhage into the wall. The spiral colon is unaffected.

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†College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota.

crepitation of the affected wall due to gas trapped beneath the serosa. The mucosal surface of the intestine may be hemorrhagic or be thick, rather dry and yellow to white in color as the result of necrosis and diptheresis. In certain individuals segmental areas of intestinal mucosa are necrotic without marked hemorrhage into the wall (Figure 3). These lesions are frequently confined to the jejunum, although ileum, cecum and colon may on occasion be involved. In some cases, there is a serosanguinous or serofibrinous exudate in the peritoneal cavity.

FIGURE 3. The intestinal tract of a newborn pig with entertoxemia. A segment of the jejunum is necrotic, the mucosa is thickened, there is little or no hemorrhage.

The clinical and necropsy findings in *Cl. perfringens* type C enterotoxemia are quite typical. One can make a tentative diagnosis without laboratory confirmation, although the lesions are not pathognomonic. On occasion, diarrheal disease of the newborn pig with segmental areas of necrosis in the small intestine is seen, from which the type C organism cannot be recovered. It is well to consider other known causes of diarrheal disease in the differential diagnosis. T.G.E., Hog Cholera, and "*E. coli* infection" do not usually result in the striking lesions seen in type C enterotoxemia. It is not unusual to recover *E. coli* from the heart, blood or liver of newborn pigs with enterotoxemia. This may lead to confusion. It is understandable however, that *E. coli* might penetrate the necrotic intestinal mucosa of a pig with type C enterotoxemia.

Gram stained smears prepared from the necrotic mucosa are a diagnostic aid. In such preparations from pigs with acute enterotoxemia large, Gram positive, nonsporulated rods typical of *Cl. perfringens* are the predominant flora. Laboratory confirmation of the diagnosis is dependent upon demonstrating the beta toxin, in intestinal contents or peritoneal fluid or by isolating the organism anaerobically and growing it in toxin producing media. The beta toxin is identified by neutralization tests with specific antisera. These tests are conducted in mice.

In view of the degree of intestinal necrosis and the acute course of the disease, affected pigs are usually not treated, and attempts to do so have been futile. The beta toxin however, as well as other pathogenic clostridial toxins, is a good antigen. Thus an effective preventive program is feasible.

Parenteral injection of *Cl. perfringens* type C antiserum, given shortly after birth before the pigs sicken has been successful in preventing the disease (9). This form of passive immunization is recommended when an enzootic is first diagnosed and there is not time for active immunization of dams. *Cl. perfringens* type C toxoid administered to dams prior to farrowing is an effective prophylactic. For best results, two separate injections are given two to three weeks apart. The latter injection should be about two weeks prior to farrowing. The active immunity of the dam is passively transferred to newborn pigs in the colostrum. A program of vaccination of dams reduced neonatal mortality from over 35% to less than 5% (9), in large Hungarian swine herds where the disease was enzootic.

In conclusion, there are clinical, pathological and bacteriological features of *Cl. perfringens* type C enterotoxemia of the newborn pig which permit an accurate diagnosis. The disease can be controlled on premises where it occurs.

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ABSTRACTS

Hoskins, F. H., and Hansard, S. L. (1964). Placental transfer of iron in swine as a function of gestation age.—Proc. Soc. exp. Biol., N.Y., 116, 7–11.

Twelve pregnant gilts were given a single i/v injection of Fe-59 citrate and killed 48 hours later at 35, 70 or 105 days of gestation. The proportion of the dose transferred to the foetuses increased sevenfold between 35 and 70, and five-fold between 70 and 105 days. Each foetus contained an average of 0.18 mg. of iron at 35 days, 4.25 mg. at 70 days and 14.9 mg. at 105 days. It was calculated that during pregnancy a sow would need an extra 26.7 mg. iron daily.

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Mitchell, D. & Robertson, A. (1964). Leptospirosis and Infertility in swine.—Vet. Rec. 76, 436.

Reference is made to a paper (V.B. 34, abst. 2083) in which leptospira were incriminated as the cause of infertility in a herd of pigs. The authors of this letter comment on a group of sows kept by them after abortion due to natural *L. pomona* infection which showed excellent reproductive ability subsequently, without therapy. They disagree with the conclusions reached by the authors of the earlier paper.

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Palludan, B. (1964). Studies on the vitamin A requirement of pigs. XI. Developmental anomalies in the eye caused by vitamin A deficiency. Aarsberetn. Inst. Sterilitetsforsk., Copenhagen pp. 59–76. (Da.e).

In piglets born to sows that had been depleted of the vitamin before pregnancy but had received the vitamin before the 17th day of gestation, no malformation of the eye was demonstrable, but administration of vitamin A to the sow on the 18th day was too late to prevent demonstrable eye lesions in the piglets. The development of the lesions during foetal life was studied histologically. The earliest changes were haemorrhages with proliferation of mesodermal tissue and defective closure of the palpebral fissure, while the lens and retina were still developing almost normally. At a later stage anomalies occured also in these tissues. The essential lesion is therefore considered to be damage to the endothelium of the small blood vessels.

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