NATURAL AND EXPERIMENTAL INFECTION OF PIGS WITH CAMPYLOBACTER COLI.

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Introduction

The possibility that Campylobacter coli could cause enteric disease in pigs was examined by Taylor and Olubunmi (1981) who had found the organism in the inflamed small intestinal mucosa of piglets with enteritis and in inflammatory large intestinal lesions of pigs incompletely treated for swine dysentery. Their studies, coupled with recent results of the experimental infection of calves with the closely-related C. jejuni (Al-Mashat and Taylor 1980) suggested that, in non-immune pigs C. coli might cause a mild, mucoid diarrhoea and that some of the small intestinal changes noted in piglets in their survey might have been entirely due to infection by C. coli.

In order to test this hypothesis, pure cultures of C. coli were fed to pigs in a number of controlled experiments. They were fed to milk-fed hysterectomy-derived, colostrum-deprived (HDCD) piglets and to weaned HDCD pigs, to conventional sucking piglets and to weaned conventional piglets of the same stock.

Materials and Methods

The pigs used were all from a minimal disease herd and were a commercial hybrid. As the herd carried C. coli, hysterectomy was carried out on 2 sows and the piglets reared until required to produce HDCD pigs for Experiments 1 and 2. Experimental HDCD piglets were housed in isolators. Sucking piglets were housed with the sow in conventional accommodation and weaned pigs on straw or shavings and fed on a standard weaner ration which contained no non-nutrient additives. One study each was carried out with HDCD piglets, HDCD weaned pigs, conventional piglets and conventional weaned pigs.

The inoculum was prepared by harvesting the surface growth from 48 hour blood agar plate cultures of C. coli and suspending it in saline. The isolate used had been obtained from the small intestine of a 7-day old piglet which had died from diarrhoea. It had been cloned twice and was stored freeze-dried. The density of organisms present was counted and each pig received 2-4 x 10^{10} organisms on a single occasion after feed had been withheld overnight.

Clinical and bacteriological observations were carried out daily and post mortem examinations were carried out at the end of each study. C. coli was isolated using blood agar containing campylobacter supplement SR69 (Oxoid).

The presence of agglutinating antibody to C. coli of the inocular strain was determined using the test described by Butzler and Skirrow (1979) on sera taken at the beginning and end of each study.

Results

1. Infections in HDCD piglets

Inoculation of 4-day old piglets was followed by a rise in rectal temperature to 41.1°C within 3 days and this was maintained for the remaining 10 days of the study. Yellowish diarrhoea developed on day 2 post inoculation and from day 3 this was accompanied by mucus which sometimes contained blood. C. coli was isolated daily from day 2 onwards. No diarrhoea or C. coli were recorded in the faeces of the controls. At post-mortem examination, the infected piglets were in poor bodily condition with thickening of the ileum and enlargement of the mesenteric lymph nodes. The jejunal contents were yellowish, contained excess clear mucus and the mucosa was hyperaemic in patches. In the ileum these changes were accompanied by thickening. The caecal and colonic contents were pasty and adherent. Inflammatory changes were present at all levels of the intestine and lymphoid proliferation was found to be prominent in the ileum. C. coli was isolated from the jejunum, ileum, caecum and colon of all infected piglets and antibody was present at titres of 1:160 in their sera. No evidence of C. coli infection was found in the controls.

2. Weaned HDCD pigs

When two six-week old, weaned pigs were inoculated with the same organism a similar rise in rectal temperature was seen. There were few faecal changes except for occasional looseness and the presence of C. coli and excess mucus on the faeces. Affected pigs were dull for 3-4 days post infection. Lesions resembling those described in the piglets were present in the intestinal tract of infected pigs. Antibody was present at slaughter at titres of 1:640. None of these changes were seen in the controls.

3. Conventional piglets

Pasty faeces were passed by these piglets within 2-6 days post infection and C. coli was isolated from their faeces from 2 days post infection. Lesions resembling those seen in the HDCD piglets were found in their intestinal tracts at post-mortem examination and C. coli was isolated from the same sites in the HDCD piglets. Antibody levels of up to 1:640 were present.

Infection with C. coli did not develop until day 9 in litters farrowed normally in the farm of origin.

4. Conventional weaned pigs

As with the weaned HDCD pigs, few clinical signs developed other than mild fever and the presence of clear mucus on the surface of formed motions. C. coli was isolated daily from all pigs of the infected group and occasionally from the faeces of the controls. The infected group all showed thickening of the terminal ileum and enlargement of the mesenteric lymph nodes at post-mortem examination and all had serum antibody to the inocular strain at levels of 1:320 - 1:640 compared with 1:20 and 1:40 pre-inoculation.

Discussion

There seems to be little doubt that C. coli can initiate a mucoid diarrhoea which may contain blood in non-immune piglets, and, in animals of all ages, can infect the jejunum, ileum, caecum and colon to cause inflammatory change and lymphoid hyperplasia. The studies described here did not suggest that C. coli caused death even in piglets but its presence may contribute blood, mucus and some diarrhoea to enteric syndromes and some inflammatory changes in the jejunal, ileal, caecal and colonic mucosa to the pathology of some enteric diseases. The effects of infection on productivity still need to be studied in more detail.

References

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