ENTERIC DISEASE IN SUCKING AND WEANED PIGS INITIATED BY AND ASSOCIATED WITH CLOSTRIDIUM PERFRINGENS TYPE A.

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Introduction

C. perfringens Type A is known to cause food poisoning in man (Hobbs 1965), necrotic enteritis in chickens (Al Sheikhly and Truscott 1977) and has been considered to cause diarrhoea in lambs (Hauschild 1967) and calves (Niilo and Dorward 1971). Disease associated with C. perfringens in pigs is usually considered to be associated with Type C and to occur in piglets (Barnes and Moon 1964) although Type A has been isolated from pig faeces and intestinal contents on many occasions (Amtsberg et al. 1976). This paper describes the isolation of C. perfringens Type A from inflammatory lesions in the intestinal tracts of piglets dying from a number of disease conditions and experimental studies carried out to test the pathogenicity of one isolate of the organism for hysterectomy-derived, colostrum-deprived piglets and for conventional weaned pigs.

Materials and Methods

116 piglets aged 3 days to 3 weeks were obtained from 3 farms for post mortem examination. Most were dead but a few were chronically or severely ill and were killed. Gross and microscopical changes were recorded and bacteria present in the intestinal mucosa were isolated and recorded.

An isolate of C. perfringens Type A obtained from inflammatory small intestinal lesions in a 3-day old diarrhoeic piglet was used to infect 3 HDCD piglets in a controlled experiment and 10 conventional weaned pigs in 2 controlled experiments. In a further study, the development of the changes following infection was examined by killing 6 inoculated weaned pigs at daily intervals with appropriate controls. Inoculum was prepared from a low passage freeze dried culture of the isolate and given orally to pigs after overnight fasting. Approximately 10^9 organisms were fed on each occasion. Clinical signs and daily liveweight gain were recorded in the two weaned pig studies and the presence of C. perfringens Type A in the faeces was monitored by culture on reinforced clostridial medium. A thorough post mortem examination was carried out on animals at the end of the study.

Results

C. perfringens was isolated from 12/116 piglets from 3 farms in the survey. It was isolated from the jejunum, ileum, caecum and colon, particularly from piglets which had died from diarrhoea within 10 days of birth. Lesions at sites from which it was isolated included congestion of the mucosa and areas of focal haemorrhage and necrosis. The gut contents were in most cases fluid, often creamy in consistency and sometimes contained flecks of blood. Agents also identified in piglets from these farms included coccidia, cryptosporidia, coronavirus of the epidemic diarrhoea type, rotavirus and Campylobacter coli. Entero pathogenic E. coli were rare.

When inoculated into HDCD piglets a transient rise in temperature to 40°C occurred and a profuse, creamy diarrhoea containing flecks of blood developed. C. perfringens resembling the inocular strain was isolated in profuse culture only from the inoculated animals. The inoculated animals died or were killed in extremis within 72 hours of inoculation and were found to be in poor bodily condition with sunken eyes and evidence of dehydration. The thoracic pericardial and abdominal cavities contained varying amounts of fluid. The liver was pale, but the most marked changes were seen in the small and large intestines. The serosal surface was congested and the intestine was flaccid with fluid or pasty contents. In the small intestine the contents were fluid and contained specks of blood and small pieces of necrotic debris. In the large intestine the contents were creamy in colour, pasty and contained flecks of blood. The mucosa of the small intestine was congested with pinpoint haemorrhages and small areas of necrosis. There was villous atrophy. Localised areas of inflammation were seen in the large intestinal mucosa. The histological changes were those of congestion, destruction of the mucosal architecture and necrosis. C. perfringens Type A was isolated from the jejunum, ileum, caecum and colon of these inoculated piglets but not from the controls.

In the first two studies carried out with weaned pigs the clinical signs were restricted to a variable rise in rectal temperature (to 40.1°C), depression, dullness and in one case transient incoordination. Loose faeces with varying amounts of mucus, some of which contained blood, was passed from days 3 to 9 post inoculation. Feed conversion efficiency was depressed in one study. C. perfringens Type A was isolated only from the faeces of infected animals. At slaughter 21 days after inoculation changes were restricted to the small intestine which was flaccid with mucoid contents and congested, mildly necrotic mucosa. C. perfringens Type A was isolated from these areas in every case.

When pigs were killed at daily intervals the most prominent changes were seen in the jejunum between days 2 and 4 post inoculation. The small intestine was flaccid, congested with brownish fluid contents and the mucosa was pale on day 2 but became progressively more congested. The histological changes were principally those of villous atrophy with apical villous damage and oedema of the submucosa. C. perfringens Type A was isolated from both the intestinal contents and the mucosa.

Discussion

These studies suggest that C. perfringens Type A can be found in diarrhoeic syndromes in sucking piglets but, because of the number of other agents which may have been present their significance was difficult to assess. In experimental infections, creamy faeces flecked with blood are passed by non-immune piglets and blood and mucus may occur in the faeces of weaners. The clinical signs are less severe in older pigs. The main site of the infection appears to be the small intestine and inflammatory and necrotic changes appear to result from infection. Mortality may occur in non-immune piglets and it is possible that productivity may be affected in older pigs.

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