**Clostridium perfringens** type C causing necrotising enteritis

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**SUMMARY** A rapidly fatal case of enteritis necroticans in a 24 year old man with diabetes was caused by *Clostridium perfringens* type C. The role of beta toxin in the disease is discussed. This type has not been previously described as a causative agent in necrotising bowel disease of man outside endemic areas.

Outbreaks of acute necrotising enteritis have been recorded in New Guinea\(^2\) and in post-war Germany.\(^3\)\(^4\) Good evidence was obtained that the syndrome may be caused by the beta toxin of *Clostridium perfringens* type C.\(^5\) We report an isolated fatal case of acute necrotising enterocolitis associated with this organism.

**Case report**

A 24 year old male nurse was admitted to his own ward with a one day history of bloody diarrhoea. The morning of the same day he had worked normally. Examination showed a painful, slightly distended abdomen with vivid peristalsis and without muscular resistance. He had had insulin dependent juvenile diabetes since the age of 11, which he managed himself; his blood sugar on admission was 9.7 mmol/l. He was treated with an infusion of normal saline, and for his diabetes short acting insulin was given.

Twelve hours after admission his condition rapidly deteriorated, with increasing abdominal distension, loss of peristalsis, profuse bloody diarrhoea, and shock. Results of x ray examination showed distended ileal loops with gas in the enteric wall, the mesenterium, and the intrahepatic branches of the portal vein. He vomited a bloody and bile stained fluid. Enteritis necroticans was suspected. Treatment with blood and plasma transfusions, intravenous penicillin \(2 \times 10^6\) U every 2 h plus intravenous metronidazol 500 mg three times daily, prednisolone and dopamine was started. Gas gangrene antitoxic horse serum was given at a dose of 75 000 U intravenously. Gastric suction drainage was applied, and within a few hours the patient became anuric and he died 24 h after admission.

Microscopical examination of the faecal specimens taken 12 h after admission showed Gram positive rods in abundance, some of which contained subterminal spores. Biochemical tests and gas-liquid chromatography carried out on cultures of the organism by Luton Public Health Laboratory (England) confirmed the identity as *Clostridium perfringens*. Guinea pig skin neutralisation tests carried out according to Sterne and Batty\(^6\) using cultures grown for 7 h in veal cooked meat broth with added starch (1%) identified the organism as *C perfringens* type C (Figure).

**PATHOLOGY**

Necropsy was performed 17 h after death. The body appeared to be in good nutritional condition. On opening the abdomen 2050 ml of serosanguineous fluid was found. The bowels were considerably distended and showed a dull red serosal surface with many adhesions between intestinal loops. The lumen contained large amounts of haemorrhagic fluid, while the mucosa was dark red and mildly swollen; the mucosal folds were still visible. No ulcerations were found. The other layers of the intestinal wall were also oedematous. From about 15 cm distal to the Treitz ligament the entire small intestine was affected and large parts of the colon were also involved, but to a lesser extent. There was pronounced oedema of the mesentery and retroperitoneal fatty tissue, and several small gas filled blebs were present. Microscopical examination of the small and large intestine showed extensive mucosal necrosis with many rod like microorganisms in the tissue. They could not be identified.
Discussion

Necrotising bowel disease due to *Clostridium perfringens* beta toxin in man is characterised by its sudden onset with acute inflammation and pronounced necrosis of intestinal mucosa giving rise to bloody diarrhoea, abdominal cramps, and shock. This disease was first recognised in Lübeck, Germany, where it was called “Darmbrand,” meaning “fire bowels.” Its appearance was limited to the early years after World War II; the highest incidence was in 1948, after which the disease disappeared. The overall mortality was about 40%. *C. perfringens* type F, later shown to be a heat resistant variant of type C, was isolated from many cases and the disease was thought to be caused by the beta toxin produced by types B and C but not type A of this organism.

Excessive eating of rich food by a malnourished population is thought to be an important causative factor. Pig-Bel is a form of acute segmental necrotising enteritis which occurs mainly in children in the Highlands of Papua New Guinea. The disease is characterised by abdominal cramps, shock, and bloody diarrhoea. Mortality is high, about 60% in 1961; this declined to 33% after the introduction of combined medical and surgical treatment.

Necrosis of large parts of the entire small intestine, often with perforation, is seen. It occurs in association with the widespread practice of butcher-
ing and eating many hundreds of pigs on special festive occasions. All parts of the animal, including the entrails, are consumed. Unsanitary preparing conditions, undercooking, and saving meat for future use give the C perfringens type C, which may normally be present in the pig intestine, ample opportunity to multiply and produce beta toxin. Low levels of digestive proteases in the intestinal lumen, associated with the low protein diet of New Guinea Highlanders may enable the beta toxin to enter the upper small intestine without being inactivated.

Arguments for the C perfringens type C aetiology include the following:

1. This type is not a normal inhabitant of the human bowel outside endemic areas but is often isolated from resected bowel of patients with Pig-Bel.

2. Rising titres of beta antitoxin were found in paired sera from patients following Pig-Bel disease.

3. Active immunisation with toxoid prepared from type C resulted in a significant reduction of the incidence of Pig-Bel.

Acute segmental ischaemic enteritis occurred in the Chiang Mai area of Thailand and enteritis necroticans in southern Ghana. Both had clinical features resembling Pig-Bel but differed in epidemiological aspects; the cause of these conditions is not known. Sporadic cases of necrotising bowel disease due to C perfringens have been reported from other parts of the world, including England, the United States, and Canada, but none have been proved to be caused by C perfringens type C.

In our patient there was no history of malnutrition. His diabetes was not adequately controlled, and it is well known that diabetic patients have a lowered resistance to infections. It is therefore tempting to speculate that this may have been a contributing factor. The day before admission he had consumed roast pork at a party, but none of the other guests became ill. The disease was not present among patients in his ward.

Treatment of necrotising enteritis includes administration of large amounts of fluid parenterally and careful supportive care. Resection of affected bowel must be considered if there is a rapid increase in signs of toxaemia, localised or diffuse peritonitis, or persistence of paralytic ileus. Anti-gas-gangrene antitoxin is not effective; beta antitoxin containing type C antiserum should be given but is not available outside endemic areas. In view of the extensive involvement of the intestine and the very rapid course of the disease no beneficial effects of surgical intervention could be expected in our patient.

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References


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