Of 23 patients with infections due to non-capsulate *H influenzae*, 15 had bacteraemia; seven of these had extrapulmonary infections (meningitis, subdural empyema, periorbital cellulitis, dacrocystitis, urinary tract infection, neonatal septicaemia, and maternal bacteraemia). In the patients with subdural empyema and periorbital cellulitis *Streptococcus milleri* and viridans streptococci respectively were also isolated. Of eight patients with bacteraemia and pulmonary infection, six died; all had a serious underlying disease and five died within 24 hours after admission with overwhelming septicaemia. The mean duration of illness before admission in the group with bacteraemia was two days.

Non-capsulate *H influenzae* was isolated from pleural aspirate or abscess as the sole organism in five patients and in combination with *Str pneumoniae* in two patients and with *Str milleri* in one patient. All eight patients had underlying disease, and two, both of whom had carcinoma of the lung, died. The mean duration of illness before admission of these patients was four and a half weeks. The course of the illness in the surviving patients was protracted, and all six needed surgical drainage. The mean duration of stay in hospital for those who survived was 26 days.

Comment

Overall, 22 out of 26 cases of systemic *H influenzae* infections in children aged over 6 and in adults were due to non-capsulate strains; this is in striking contrast to the time honoured teaching that such strains are relatively non-pathogenic. Recent reports from the United States have also drawn attention to non-capsulate *H influenzae* as a cause of bacteraemic and non-bacteraemic pneumonia, meningitis, and various other infections in adults.[1-3] Non-capsulate strains may also cause maternal bacteraemia and lead to serious infection in the neonate.[4]

Non-capsulate *H influenzae* is part of the normal flora of the respiratory tract and may colonise the lungs in chronic bronchitis. Several factors may have contributed to the increased recognition of the organism in bacteraemia—namely, more accurate typing methods; the use of counterimmunoelectrophoresis; and improved blood culture methods, including the use of chocolate agar for subculture.[5]

Invasive infection due to non-capsulate *H influenzae* has been surveyed principally in the United States, and we are unaware of any other series documenting as many cases of empyema or abscess due to these strains as we have encountered in these five years. Many invasive infections due to non-capsulate *H influenzae* probably go unrecognised, and microbiologists should perhaps search more diligently for this pathogen.

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Adenocarcinoma of the ileum presenting as non-traumatic clostridial myonecrosis in cystic fibrosis

Recent reports of cholangiocarcinoma[1] and adenocarcinoma of the pancreas[2] in two young adults with cystic fibrosis led to the suggestion that cystic fibrosis may predispose to malignancy. We present a case of adenocarcinoma of the ileum in cystic fibrosis.

Case report

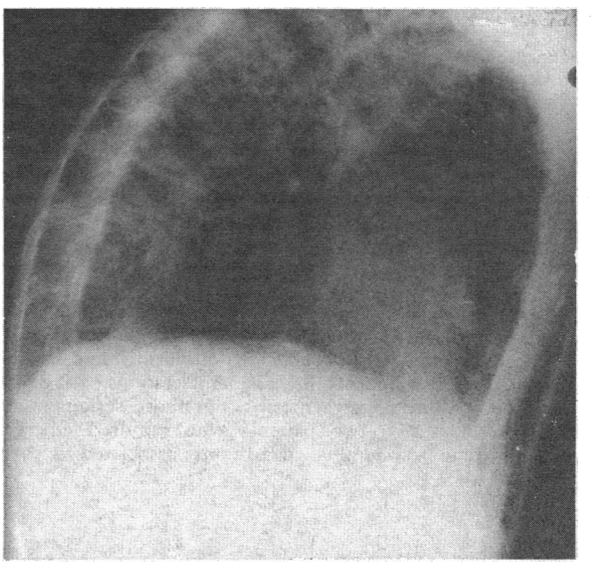
A 29 year old man was diagnosed as having cystic fibrosis at the age of 6 months (sweat sodium concentration 132 mmol(mEq)/l). Pancreatic supplements were started, and subsequent growth and development were normal, although symptoms of malabsorption persisted (average faecal fat excretion 14 g/day). *Pseudomonas aeruginosa* was consistently cultured from the sputum from the age of 12 years, and he required long term treatment with oral and nebulised antibiotics and frequent courses of intravenous antibiotics to maintain spirometric values at 40-60% of predicted values.

When he was 20 diabetes mellitus was diagnosed and hepatosplenomegaly noted associated with raised activities of alkaline phosphatase and γ-glutamyltransferase.

In December 1983 he was admitted with constipation, abdominal pain, and a mass in the right iliac fossa. Meconium ileus equivalent was diagnosed and treated with acetylcysteine by mouth and enema. The symptoms and the mass resolved. Abdominal ultrasonography and computed tomography showed hepatosplenomegaly and dilatation of the hepatic portal vein compatible with portal hypertension.

In September 1984 he was admitted with a short history of severe left inframammary pain after coughing. Examination showed an erythematous area below the left nipple roughly 5 cm in diameter with crepitus and tenderness on deep palpation. His temperature was normal, pulse 90/minute, blood pressure 130/80 mm Hg, and respiratory rate 22/minute. Scattered crackles throughout both lung fields and hepatosplenomegaly were unchanged from his previous admissions. No other masses were palpable in the abdomen. A posteroanterior chest radiograph showed only the pulmonary changes of advanced cystic fibrosis. A small amount of subcutaneous gas was visible in a lateral view.

Two hours later the erythema and crepitation had extended to most of the chest and abdominal wall. A large amount of subcutaneous gas was now visible in a lateral chest radiograph (figure). He was feverish (38·5°C) and



Lateral chest radiograph showing extensive subcutaneous emphysema two hours after admission.

mildly jaundiced with pulse 130/minute and blood pressure 90/60 mm Hg. Haemoglobin concentration was 14·1 g/dl, white cell count $18·8 \times 10^9$/l (82% neutrophils), platelet count 121×10^9/l, random blood glucose concentration 28·2 mmol/l (508 mg/100 ml), bilirubin 34 μmol/l (2·0 mg/100 ml), alkaline phosphatase 1570 IU/l, γ-glutamyltransferase 143 IU/l, and creatinine phosphokinase 435 IU/l. Results of other liver function tests and urea and electrolyte concentrations were normal. Necrotising fasciitis was diagnosed provisionally and treatment with intravenous benzylpenicillin, metronidazole, and gentamicin started. Despite this profound systemic toxicity and cardiovascular collapse rapidly developed. About 12 hours after admission and 24 hours after the onset of pain he died.

Clostridium septicum was later isolated from blood, stool, and abdominal muscle aspirate. Limited postmortem examination confirmed widespread myonecrosis of the chest and abdominal wall. An unsuspected ulcerating lesion of the terminal ileum was found about 1 cm from the ileocaecal valve. Histological examination showed this to be a moderately well differentiated adenocarcinoma. The lungs, liver, and pancreas showed changes entirely compatible with cystic fibrosis.

Comment

Non-traumatic clostridial myonecrosis is rare and often fatal despite early treatment. Six of 10 patients reviewed by Jendrzejewski

et al had occult adenocarcinomas of the large bowel.[3] An association with neoplasm of the small bowel has not been described. In this case portasystemic shunting and diabetes mellitus may have been additional predisposing factors.

Adenocarcinoma of the ileum is rare in this age group.[4] Its coexistence with cystic fibrosis may have been a chance happening, but we speculate that a combination of slowed small bowel transit, raised faecal bile acid concentrations, altered bowel flora, and low plasma selenium and vitamin E concentrations, all previously reported in cystic fibrosis, may predispose to the development of gastrointestinal malignancy. Any such predisposition would become more obvious as life expectancy in cystic fibrosis improves.

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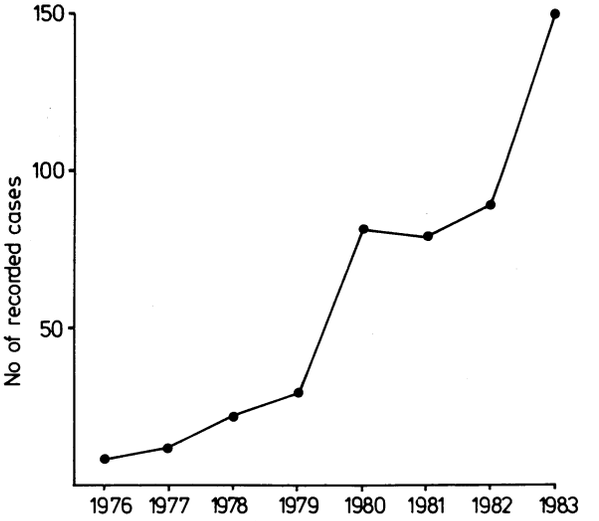
Intestinal perforation associated with Yersinia enterocolitica infection

Yersinia enterocolitica was first recognised as a pathogen in man in 1939.[1] It is a Gram negative coccobacillus of worldwide distribution and produces a wide range of clinical manifestations.[2 3] The commonest is a self limiting gastroenteritis, but more serious variants may occur. We describe two patients who required emergency operations for acute abdominal conditions associated with this infection.

Case reports

Case 1—A 7 year old Japanese boy presented with a two day history of abdominal pain, fever, and diarrhoea. On examination he had a fever (38 C) and was dehydrated, and there was tenderness in the left iliac fossa. Gastroenteritis was diagnosed provisionally, and treatment comprised intravenous rehydration. The symptoms continued for seven days, and when blood cultures yielded *Bacteroides fragilis* intravenous gentamicin and metronidazole were started. There was little clinical response, and a laparotomy four days later showed a pelvic abscess secondary to a perforated appendix. After appendicectomy and drainage he made an uneventful recovery. Subsequently an antibody titre of 1/320 against *Y enterocolitica* type O3 was detected in blood obtained on the 10th day of the illness.

Case 2—A 14 year old English girl presented in a critically ill condition. She had been unwell for three weeks with diarrhoea, vomiting, abdominal pain, fever, and weight loss (9·5 kg). Two days before her admission the diarrhoea had suddenly stopped. On examination she had a fever (38 C), was dehydrated, malnourished (plasma albumin concentration 17 g/l), hypotensive and had a tachycardia. The abdomen was massively distended, and an *x* ray film showed intestinal obstruction. After resuscitation laparotomy showed mechanical obstruction of the small bowel due to an inflammatory mass in the pelvis associated with numerous perforating ulcers in the ileum. A similar mass in the upper abdomen was related to jejunal ulceration. Two resections with primary anastomoses were performed to relieve the obstruction and restore intestinal continuity. Postoperatively, despite total parenteral nutrition and intravenous penicillin, gentamicin, and metronidazole she was critically ill for eight days with persistent fever and ileus. An antibody titre of 1/640 against *Y enterocolitica* type O3 was detected and chloramphenicol substituted for the previous antibiotics. She remained seriously ill for a further 11 days and improved only after the dosage was increased. Eventually she recovered completely and was discharged home 32 days after admission. Throughout the illness all blood cultures were sterile, rectal swabs grew no pathogens, and computed tomography of the abdomen postoperatively showed no localised collections. She received total parenteral nutrition for 26 days and chloramphenicol for 18 days (seven days at high dosage).



Incidence of positive diagnoses of *Yersinia enterocolitica* infection over past decade in United Kingdom. Diagnosis was made by serological examination or isolation from blood, stools, or lymph nodes, or both. (Courtesy of Dr C J Mitchell, Public Health Laboratory Service, Leicester.)

Comment

The antibody titres against *Y enterocolitica*, although measured in only single blood samples, were considered to be diagnostic by the Public Health Laboratory Service in Leicester (the British reference centre for this organism). During 1976-83 only 469 such infections were recorded by the Public Health Laboratory Service, and thus such infection appears to be either rare or underdiagnosed. The mean number of cases recorded each year increased from 18 (SD 9·5) in 1976-9 to 100 (34) in 1980-3 (figure), but whether this reflects an increased incidence or greater awareness of the infection is uncertain. Yersinia is more prevalent abroad,[2 3] but our patients must have contracted the infection within the United Kingdom as neither had been abroad recently.

The risks of yersinia infection are considerable; deaths have been recorded,[4] and life threatening complications include acute appendicitis,[2] septicaemia,[2 3] peritonitis,[4] and gangrene of the small bowel.[5] We suggest that yersinia may be a more important cause of acute abdominal conditions than is currently appreciated, and if antibody titres were obtained whenever acute intestinal problems were being investigated many more positive diagnoses would probably be made.

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