Small bowel perforation: an unusual cause

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Abstract

An 80-year-old lady with abdominal pain had a perforated terminal ileum at laparotomy 2 weeks after treatment for Campylobacter jejuni infective colitis. Histology of the excised bowel revealed transmucosal inflammation but no dysplasia or malignancy. Cases of bowel perforation secondary to Campylobacter are more common in children and HIV-positive individuals.

Keywords

Campylobacter; small bowel perforation.

Introduction

An 80-year-old lady presented with a 24-h history of severe generalised abdominal pain. She also complained of nausea and vomiting but had normal bowel action and no fever or urinary symptoms. Two weeks prior to this episode, she had been admitted to hospital with a 2-week history of diarrhoea, from which she had lost almost 4 kg in weight. Stool culture revealed growth of Campylobacter jejuni and a diagnosis of infective colitis was made. She had a course of oral erythromycin 500 mg twice daily, lasting for 7 days, fluid replacement and was discharged after 4 days.

She had a past medical history of ischaemic heart disease, atrial fibrillation, diverticular disease and a hysterectomy. She had been taking warfarin, frusemide, isosorbide mononitrate MR, nicorandil, atenolol, atorvastatin, AdCal D3 and weekly alendronic acid with no known drug allergies. She was a non-smoker and occasional alcohol drinker.

On examination, she was apyrexial, blood pressure 96/76 mmHg, heart rate 92/min, respiratory rate 18/min, oxygen saturations of 92% on 10L of oxygen. Heart and lung sounds were normal. The abdomen was soft but tender in the epigastric and suprapubic areas. No masses were felt and there was no organomegaly. Bowel sounds were present and digital rectal examination revealed no abnormalities.

Blood tests revealed amylase 110 U/L, white cell count 10.5 × 10⁹/L, C-reactive protein (CRP) 9 mg/L with normal liver and renal function otherwise. Blood gases were also within normal limits with no evidence of hypoxia and lactate levels were not raised. Initial radiologic films were unremarkable.

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Clinical deterioration in vital signs over subsequent hours prompted a repeat of blood tests and CRP was now 33 mg/L with white cell count $22.9 \times 10^9/L$. A chest radiograph revealed free air under both domes of the diaphragm (Fig. 1); an abdominal film was unremarkable (Fig. 2).

A preliminary diagnosis of bowel perforation secondary to diverticular disease was made. The patient was kept nil by mouth, given intravenous fluids and taken to theatre for laparotomy. At laparotomy, a discrete perforation was noted at the terminal ileum with adjacent inflammation and macroscopically normal bowel distal and proximal to the affected area. A wedge excision was performed at the perforation site and a primary anastomosis formed.

Histology showed a 2-mm perforation with the serosa covered in exudate. Inflammation was noted through the depth of the mucosa, with further exudate and ulceration. There were no signs of dysplasia, malignancy or diverticular disease.

Fig. 1. Chest radiograph showing free air under the diaphragm.

Fig. 2. Abdominal radiograph showing no abnormalities.
The patient was admitted to the intensive treatment unit for 4 days. Discharge from hospital was protracted due to prolonged small bowel ileus that required total parenteral nutrition. Episodes of diarrhoea followed this and a course of erythromycin was given. A further stool culture during admission showed no growth of organisms.

**Discussion**

*Campylobacter* species are amongst the most common bacterial causes of gastroenteritis\[1\]. Proportionally, more cases are seen in children and individuals positive for human immunodeficiency virus (HIV). In 80% of cases, infection results from ingestion of contaminated foodstuffs, such as poorly cooked chicken. Other sources include unpasteurized milk, unchlorinated swimming water and infected dogs. The most common species is *Campylobacter jejuni*, although others such as *C. coli* and *C. upsaliensis* are seen in individuals with HIV.

Gastroenteritis results from bacterial invasion of the intestinal epithelium and release of cytotoxins and inflammatory cytokines. Histologically, lesions may appear similar to that of granulomas or those in ulcerative colitis.

Patients present with crampy periumbilical pain and profuse watery diarrhoea that is bloody in 30% of cases. Physical findings are non-specific. Bacteraemia develops in 1% of cases. The main differential of this presentation is other infective species, such as *Shigella*, *Yersinia*, *Salmonella* and *Escherichia coli*; inflammatory bowel disease is also an important consideration.

As the condition is usually self-limiting, management is conservative, involving fluid replacement. In severe cases, or in the immunocompromised, a course of erythromycin or azithromycin, for 7 days is adequate.

Complications are rare, but include post-infective arthritis (1%) and Guillan–Barré syndrome (0.3%).

Occurrences of bowel perforation secondary to *Campylobacter infection* are rare and have been more commonly reported in children\[2\] and those with HIV\[3\]. Perforation has also been seen in those who have a toxic megacolon\[4\].

**Teaching point**

We believe that this is an uncommon case in an otherwise normal bowel in an HIV-negative adult. The reasons of this episode are unclear given the initial treatment of diarrhoea with antibiotics. It could represent inadequate treatment of the initial episode although symptoms had resolved and repeat stool cultures were negative for *Campylobacter jejuni*. This highlights the importance of a taking a thorough history to include recent episodes of infective gastroenteritis when exploring suspected bowel perforation.

**References**