To our knowledge this is the first report of recurrent anaemia without frank gastrointestinal haemorrhage as a presentation of cholesterol embolism. This elderly male patient, although not hypertensive, had many risk factors for this disease: he was a diabetic, an ex-smoker and had evidence of generalized atherosclerosis. We cannot be certain in retrospect for how long cholesterol embolism in the superior mesenteric axis was the source of blood loss, but right hemicolectomy appeared to be curative in this case and no other cause for bleeding was found on close examination of the specimen. The interval of one month between

angiography and surgery makes it unlikely that the ischaemic changes observed were due to cholesterol embolism at aortic instrumentation. It is possible that the episode of small bowel obstruction was also a sequel of cholesterol embolism, since the healing process following an episode of extensive mucosal ischaemia can result in concentric fibrosis and narrowing of the bowel lumen.³

This case illustrates that cholesterol embolism should be considered as a possible cause of unexplained gastrointestinal blood loss in an elderly patient with atherosclerosis.

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Toxic megacolon with late perforation in *Campylobacter* colitis – a cautionary tale

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Summary: We report a case of *Campylobacter*-induced colitis complicated by toxic megacolon which led to colonic perforation requiring a total colectomy and ileostomy.

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Introduction

Campylobacter enteritis is now recognized as the commonest infectious cause of diarrhoea in the UK. The illness is usually self-limiting and life-threatening complications are rare. We describe a case of severe *Campylobacter* colitis presenting with toxic megacolon in a previously healthy young woman. This was complicated by late colonic perforation requiring a subtotal colectomy despite early treatment with antibiotics and intravenous steroids while the aetiology was unclear.

Case history

A previously fit 38 year old woman presented with a 4 day history of vague abdominal discomfort, fever, rigors and loose, watery diarrhoea. Her bowels opened up to 12 times per day with blood and pus in the stool. There had been no previous similar bowel disturbance nor had she received antibiotics just prior to this illness. On examination she was febrile (38°C), tachycardic (120 beats/minute, regular) and severely dehydrated (25 mmHg postural drop in systolic blood pressure). The abdomen was generally distended and tender, but there was no guarding.

Sigmoidoscopy showed an inflamed, friable rectal mucosa with overlying pus and contact bleeding. The abdominal radiograph showed a markedly dilated, empty colon with a maximum diameter of 6.7 cm in the transverse segment. There was widespread thickening of the large bowel wall and loss of the normal haustral pattern (Figure 1).

A provisional diagnosis of toxic megacolon secondary to infection or inflammatory bowel disease was made. Intravenous rehydration, hydrocortisone and oral ciprofloxacin were commenced. After 4 days, a rectal biopsy was reported as showing an abundance of acute inflammatory cells in the lamina propria, but no cryptitis, crypt abscesses, goblet cell depletion or granulomata were seen. *Campylobacter jejuni* was cultured from three stool samples but *Clostridium difficile* or its toxin were not present. Serial blood cultures remained sterile. The hydrocortisone was therefore discontinued.

Her general condition improved over 10 days with resolution of her hypotension, tachycardia, abdominal distension and tenderness. Bowel frequency decreased to three stools per day with occasional blood and pus. However, the haemoglobin concentration dropped from 13.2 g/l to 10.5 g/l. The serum albumin concentration fell from 36 g/l to 24 g/l, despite parenteral nutrition for the last 5 days.

On the eleventh day, she developed severe abdominal pain, tachycardia and increased epigas-

tric tenderness with guarding. Subhepatic free gas was present on the abdominal radiograph with a toxic megacolon (Figure 2).

Figure 1 Abdominal radiograph at presentation.

Figure 2 Abdominal radiograph the eleventh day showing gross colonic dilatation on subhepatic free air (arrow).

Emergency laparotomy confirmed a very dilated colon which was friable and oedematous, particularly in the transverse segment. The caecum and sigmoid colon were perforated with a fibrinous exudate over the transverse colon. A total colectomy was performed leaving an ileostomy and mucous fistula.

Histological examination of the excised colon, together with 4.5 cm of terminal ileum showed extensive mucosal ulceration and inflammation, but where the mucosa was preserved, there was minimal cryptitis or goblet cell depletion. Lymph nodes showed severe reactive hyperplasia. These findings were consistent with severe acute *Campylobacter* colitis.

Postoperatively, she was treated with intravenous cefuroxime, erythromycin and metronidazole and she made an unremarkable recovery. Her liver function tests returned to normal after 14 days. She remains well 8 months later and a reversal of ileostomy with ileo-rectal anastomosis is planned in the near future.

Discussion

Campylobacter enteritis is increasingly common with over 32,000 cases reported in England and Wales in 1989, a total which has quadrupled since 1979.¹ In the vast majority a relatively mild, self-limiting gastroenteritis is observed, hardly requiring any specific treatment. Atypical manifestations include meningitis, cholecystitis, urinary tract infection and Guillain-Barré syndrome. Rarely, a protracted and severe illness may be complicated by massive lower intestinal haemorrhage² or acute colitis mimicking a relapse of inflammatory bowel disease with toxic megacolon.³

Toxic megacolon due to *Campylobacter* colitis usually responds to appropriate antibiotics and supportive treatment,³ although it is possible that cases diagnosed after colonic resection may be

under-reported. We could find only one previous case of perforation following *Campylobacter*-induced toxic megacolon.³ In this, as in our case, intravenous corticosteroids and broad spectrum antibiotics were administered.

Parenteral hydrocortisone and oral ciprofloxacin, a 5-amino fluoroquinolone antibacterial agent, were employed empirically in our case until stool cultures and rectal histology became available. Whilst acknowledging the risk of exacerbating or masking an infectious diarrhoea by treatment with intravenous corticosteroids, we believe their use was justified in the initial management of toxic megacolon.

There is general acceptance to treat with antibiotics those patients with high fever, bloody diarrhoea of more than eight stools per day or persistent symptoms for more than one week. Most strains of *Campylobacter jejuni* are sensitive to ciprofloxacin or erythromycin.

Fluoroquinolone resistance has been reported in isolates from human stools in one series from the Netherlands in which 16 of 145 isolates (11%) were resistant (MIC 4 mg/l).⁴ The presence of erythromycin-resistant strains has varied up to 8% in Sweden⁵ and have been described in the UK.⁴ The isolates from our patient's stools, however, were sensitive to ciprofloxacin and erythromycin. The fact that this illness was accompanied by anaemia, hypoproteinaemia and a transient rise in liver enzymes suggested that the infection was severe and that complications might arise.

We conclude on a cautionary note in the management of an increasingly common condition which is usually self-limiting but which can produce life-threatening complications.

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