Journal of Infection (2006) **52**, e103–e106

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CASE REPORT

Toxic megacolon complicating *Escherichia coli O157* infection

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Accepted 29 July 2005
Available online 29 August 2005

KEYWORDS
E. coli O157;
HUS;
Toxic megacolon;
Antibiotic therapy

Summary Toxic megacolon is a well known complication in inflammatory bowel disease such as ulcerative colitis or Crohn's disease. The development of toxic megacolon as a complication of infectious colitis is rare. However it is recognised as a complication of enteric infections caused by *Clostridium difficile*, *Campylobacter jejuni*, *Shigella*, *Salmonella* species, Cytomegalovirus and amoebae. We describe a case of necrotising haemorrhagic ileo-colitis in a previously fit and healthy young adult female caused by *Escherichia coli O157* where toxic megacolon developed as a complication along with hemolytic uremic syndrome (HUS).

Clinical presentation

A 39-year-old white female was admitted to hospital with a 4 day history of bloody diarrhoea (10–12 episodes/day), severe lower abdominal pain, nausea and generalised myalgia. There was no significant past medical history. She was tachycardiac, hypotensive with a blood pressure of 80/60 mmHg, cold peripheries and low jugular venous pressure. Abdominal examination revealed right and left lower quadrant tenderness with sluggish bowel sounds. On per rectal examination liquid stool mixed with blood was found. The rest of the systemic examination was normal. A preliminary diagnosis of infective colitis/inflammatory bowel disease was made.

The full blood count on admission showed low haemoglobin (111 g/l) high white cell count with predominant neutrophilia (26.9×10^9/l) and very low platelets (30×10^9/l). She was in acute renal failure with a serum urea of 25.3 mmol/l and serum creatinine of 483 μmol/l. The abdominal X-ray revealed dilated loops of small bowel.

In the absence of peritonitis, the patient was managed conservatively with supportive therapy and intravenous cefuroxime and metronidazole. At this stage the possibility of hemolytic uraemic

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0163-4453/$30.00
doi:10.1016/j.jinf.2005.07.029

syndrome was suggested based on a blood film result reporting neutrophilia with left shift, schistocytes without polychromasia and thrombocytopenia with large platelets consistent with peripheral destruction. There was also evidence of microangiopathic haemolytic anaemia.

Overnight, the patient deteriorated, became anuric and developed abdominal distention. An urgent CT abdomen revealed grossly oedematous and dilated colon (>6 cm) with severely affected transverse colon, small bowel dilatation and ascites. There was no evidence of perforation. Antibiotic therapy was revised to intravenous ciprofloxacin and metronidazole. Hydrocortisone was also added. An emergency laparotomy was performed revealing haemorrhagic and necrotic colitis from caecum to lower sigmoid colon sparing of the rectum, compatible with toxic megacolon. A putrid fluid was found in the abdominal cavity with no evidence of perforation of the bowel. She required a total colectomy with ileostomy.

Histological examination of the bowel tissue showed large areas of mucosal ulceration and destruction and underlying oedema with congestion of the serosa. There were no thrombi, granulomas or ischaemia and no evidence of Crohn's or ulcerative colitis was found. There were also no features of pseudomembranous colitis. The conclusion was that the colitis was of infective origin.

Postoperatively, the patient was transferred to intensive care unit (ITU), where she had a very stormy postoperative course, requiring ventilatory support, daily dialysis, antibiotic therapy and a tracheostomy. Finally, after nearly 3 weeks in ITU and a week on a surgical ward, the patient recovered well and was discharged home with normal renal function, normal blood pressure and a well-functioning ileostomy. A follow-up at 8 weeks was satisfactory with normal renal function and normal blood pressure.

Microbiological investigations included repeat stool and blood cultures. An initial stool sample was cultured for *Salmonella* spp., *Shigella* spp., *Campylobacter* spp., *Yersinia* spp. and *Escherichia coli O157*, using routine selective media. This sample had been taken after the patient had already received antibiotics (cefuroxime and ciprofloxacin) and was negative on culture for all enteric pathogens. Repeat stool and blood cultures were negative. Intraoperative peritoneal fluid and pus specimens grew a mixture of *E. coli*, *Bacteroides* sp. and *Clostridium* sp. *E. coli O157* was not isolated from these specimens after culturing on selective media. However, a serum sample taken 10 days after admission was positive for IgM antibodies to lipopolysaccharide of *E. coli O157* by Western blot technique (Laboratory of Enteric Pathogens, Health Protection Agency, Colindale, London). She was negative for antibodies to *Campylobacter jejuni*. As part of the epidemiological investigations it was found that all members of her family had experienced mild diarrhoea and gastrointestinal discomfort. The entire family had eaten a Chinese takeaway meal 48 h prior to onset of the patient's symptoms. All members were screened by performing stool cultures for all enteric pathogens using appropriate selective media. Only a stool sample from her husband grew *E. coli O157* positive for shiga-toxin genes (serotype-O157, phagetype 2, genotype 2). Therefore, these findings were strongly suggestive of *E. coli O157* infection in the family and, although not ultimately proven, of acute *E. coli O157* colitis in our patient.

On the basis of radiological, surgical and microbiological findings, and the triad of thrombocytopenia, haemolytic anemia and renal failure, a diagnosis of haemolyic uraemic syndrome complicated by toxic megacolon secondary to infection with *E. coli O157* was made.

Discussion

Three aspects of this patient's clinical presentation and management are remarkable. First, an unexpected and to date unrecognised aetiology of toxic megacolon was observed.

This previously fit and well patient with no significant past medical history presented with an initial diagnosis of infective colitis/inflammatory bowel disease. She rapidly deteriorated requiring an emergency subtotal colectomy with an intraoperative finding of toxic megacolon. Toxic megacolon is a complication that usually presents in inflammatory bowel disease such as ulcerative colitis or Crohn's disease.[1] The frequency is 1.6–21.4% among patients with ulcerative colitis and 0.3–2% in those with Crohn's disease.[2] Toxic megacolon as a complication of infectious colitis is a rare but well recognised complication[3] attributed to toxogenic *Clostridium difficile*, *Campylobacter jejuni*, *Shigella* spp. *Salmonella* spp., *Cytomegalovirus* and amoebae.[1,3–5] To date only one group[6] reported a severely oedematous, haemorrhagic colon as part of their detailed histopathologic investigations of three cases of *E. coli O157* associated haemolytic uremic syndrome without progression to toxic megacolon. There is only a recommendation for surgical exploration for toxic megacolon in children with haemolytic uremic syndrome[7] as part of