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Campylobacter colitis: Rare cause of toxic megacolon

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ABSTRACT

INTRODUCTION: Campylobacter is the leading cause of bacterial diarrhoeal illness worldwide. Toxic megacolon is a rare but potentially devastating complication.
PRESENTATION OF CASE: A 55 year old female with liver cirrhosis, alcoholism and hepatitis C, presented with severe colitis and stool specimen positive for Campylobacter. She developed septic shock, multi-organ dysfunction syndrome and toxic megacolon unresponsive to medical therapy, and underwent a subtotal colectomy with end ileostomy. Despite initial improvement, the patient died on postoperative day 4.
DISCUSSION: Early surgical consultation is essential as toxic megacolon may be complicated by perforation or uncontrolled bleeding; progressive colonic dilatation with clinical deterioration is also an important indication for surgery.
CONCLUSION: Toxic megacolon should be considered in a patient with Campylobacter colitis who becomes critically unwell. Despite treatment, toxic megacolon is associated with a significant risk of mortality.

1. Introduction

Toxic megacolon is an uncommon but potentially devastating complication of colitis. It is characterised by total or segmental nonobstructive colonic dilatation, combined with evidence of systemic toxicity, and has an overall mortality of 19% [1]. Ulcerative colitis and pseudomembranous colitis are well-recognised precipitants and have been reported to account for 77% of cases [2], however almost any inflammatory condition affecting the colon may lead to this complication [1]. Campylobacter colitis rarely leads to toxic megacolon and there are only a limited number of published case reports [3–14]. This is remarkable, given Campylobacter is the leading cause of bacterial diarrhoeal illness worldwide [15]. We present a rare case of Campylobacter colitis complicated by septic shock, multi-organ dysfunction syndrome (MODS) and toxic megacolon.

2. Presentation of case

A 55 year old female with hepatitis C, intra-venous drug use and alcohol abuse complicated by Child-Pugh B liver cirrhosis presented with abdominal pain, diarrhoea, fevers and confusion. She had no history of colonic disease. Computed tomography (CT) of the abdomen demonstrated mildly dilated proximal large bowel without fat stranding or colonic wall thickening. Stool PCR was positive for campylobacter species. Treatment with azithromycin, ceftriaxone, metronidazole, intravenous fluids and electrolyte replacement was commenced.

She continued to deteriorate and subsequently had a long admission to the intensive care unit with aspiration pneumonitis, type 2 myocardial infarction, renal failure and metabolic acidosis, requiring intubation, vasopressors and dialysis.

Transthoracic echocardiography, lumbar puncture, blood cultures and CT scans of the head, chest, abdomen and pelvis did not demonstrate an alternative source of sepsis and antibiotics were escalated to piperacillin/tazobactam.

On day 16 of admission she developed bloody diarrhoea with ongoing refractory multi-organ dysfunction. CT abdomen demonstrated colonic wall thickening most prominent in the sigmoid and rectum, but there was no gross dilatation, intramural gas, free gas or obvious bleeding points. Upper endoscopy excluded varices and ulcers. At this point, the patient was transferred to our tertiary hospital for ongoing intensive care management and surgical review.

An emergent laparotomy was performed. Intraoperatively, viable mildly dilated colon and clear ascites were encountered. There was no evidence of ischaemia, necrosis or perforation. The liver was nodular and the remainder of the bowel was normal. Flexible sigmoidoscopy was performed on table and demonstrated discontinuous areas of non-bleeding ulcerated mucosa in the sig-

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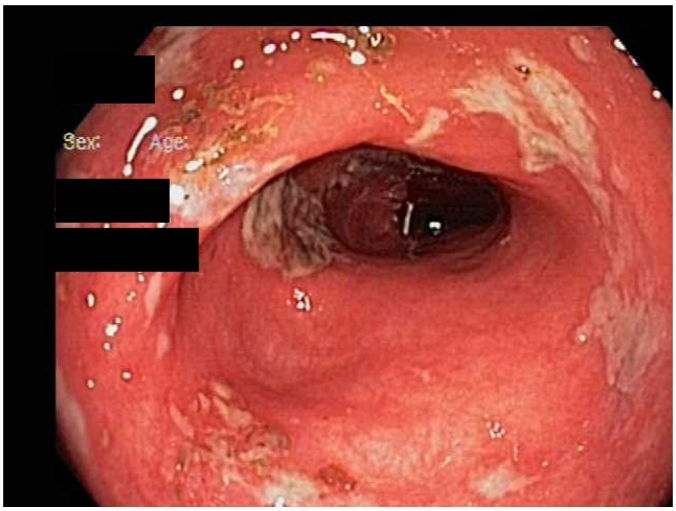

Fig. 1. Sigmoidoscopy demonstrated discontinuous areas of non-bleeding mucosal ulceration which can be consistent with infectious colitis.

moid colon (Fig. 1); biopsies were taken. The case was discussed with intensive care and infectious diseases teams and it was felt the findings were consistent with infective colitis with septic shock and MODS. The patient was returned to ICU for ongoing intensive care management and antibiotics were changed to daptomycin, ciprofloxacin, fluconazole and metronidazole.

Over the next 12 h the patient deteriorated with worsening haemodynamic instability, requiring increasing amounts of noradrenaline and vasopressin. A re-look laparotomy was performed, which demonstrated grossly dilated colon, without perforation or full thickness ischemia. A subtotal colectomy was performed and an end ileostomy and distal sigmoid mucous fistula were fashioned.

Immediate improvement was noted postoperatively; vasopressin was ceased and her noradrenaline infusion was halved. Until post-operative day 3 she continued to improve however she again developed escalating vasopressor requirements, acidosis, liver failure and respiratory failure. She died on postoperative day 4.

Histology of the pathological specimens showed features of infectious colitis and ischemia. Macroscopically, multiple discrete areas of mucosal ulceration were noted in the ascending, transverse and descending colon. There was no overtly infarcted bowel or pseudomembranes. Microscopically, the ulcerated mucosa showed congestion, neutrophilic infiltration and fibrin thombi. There was no architectural distortion and cryptitis and crypt abscesses were not prominent features. Features typical for inflammatory bowel disease were absent.

3. Discussion

Toxic megacolon is a serious condition with an overall mortality up to 19% and up to 41% mortality when perforation occurs [1]. Campylobacter colitis complicated by toxic megacolon is rare and to our knowledge, there are only twelve case reports in the literature, and none published since 2000 [3–14].

In previous case reports, the diagnosis of toxic megacolon was typically preceded by a prolonged diarrhoeal illness of at least 10 days and the majority occurred in healthy young patients, in contrast to our patient. Five cases reported total or subtotal colectomies performed for actual colonic perforation, and one case for intraoperative findings suggestive of imminent perforation [3,5,8,11–13]. All of these patients recovered uneventfully postoperatively, but none of these patients were reported to have any significant comorbidities. The importance of differentiating inflammatory bowel disease from infective colitis has been highlighted, since toxic megacolon of infective aetiology does not often require operative management and may be worsened by corticosteroids [3,5,10,13].

The diagnosis of toxic megacolon was established clinically. A commonly used set of diagnostic criteria requires radiographic evidence of colonic dilatation and at least three of the following: Fever >38° Celsius, heart rate >120 beats per minute, white cell count >10500/microL or anaemia [16]. Furthermore, at least one of the following is also required: dehydration, altered mental status, electrolyte disturbance or hypotension [16]. The clinical signs of systemic toxicity differentiate toxic megacolon from other pathological processes which can also lead to colonic dilatation, such as Ogilvie syndrome, volvulus or Hirschprung's disase [17]. In the case presented, evidence of gross colonic dilatation was apparent intraoperatively in addition to the profound MODS evident in the intensive care unit.

The diagnosis of toxic megacolon is not an absolute indication for surgery [17]. The underlying cause of colitis should be treated to restore colonic motility and prevent perforation [17]. Fluid and electrolyte disturbances need to be corrected, signs of toxaemia need to be addressed and anti-motility agents need to be withdrawn [17,18]. Although toxic megacolon precipitated by infectious colitis often does not require surgery, early surgical consultation is essential and operative management should be considered in those not improving [3,13,18].

Indications for surgery include perforation, uncontrolled bleeding, and progressive colonic dilatation with clinical deterioration [1,2,16]. The operation of choice is a subtotal colectomy with end ileostomy and Hartmann's pouch or sigmoid mucous fistula [2]. Compared with emergent total proctocolectomy, emergent subtotal colectomy is associated with a lower mortality and morbidity and also offers an opportunity for subsequent reconstruction of alimentary continuity [1]. The resected specimen can be sent for definitive diagnosis of the colitis. The procedure removes the septic focus and prevents further bacterial translocation, which can result in rapid clinical improvement.

The decision to perform a colectomy in the previously published cases was relatively straightforward as they were relatively young and healthy. In this patient, the decision was difficult and initial surgery was delayed due to her co-morbidities: the mortality in Child-Pugh B cirrhosis patients undergoing emergency surgery is 38% [19]. For these reasons, at the initial laparotomy where the colon was dilated but relatively normal in appearance, it was felt that performing colectomy at the time would increase morbidity and mortality risk. However, following the initial laparotomy, worsening systemic toxicity despite maximal medical management meant death was likely imminent. Therefore, a second laparotomy and a subtotal colectomy was performed, and an immediate initial clinical improvement was observed. Ultimately, the patient's demise indicates her clinical state was likely irretrievable at the time of surgery.

4. Conclusion

Toxic megacolon needs to be considered in patients with Campylobacter colitis who become critically unwell. Early surgical consultation is essential, as the disease can be complicated by perforation and uncontrolled bleeding: progressive colonic dilatation with worsening systemic toxicity is also an important indication for surgery. Despite treatment, toxic megacolon is associated with a significant risk of mortality, especially in patients with multiple unfavourable comorbidities.

Conflicts of interest

None.

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Authors contributions

MK and AM wrote the article and cared for the patient. JB and CL reviewed and revised the paper, cared for the patient and were part of the team that performed the operations. All authors approved the content of the final manuscript.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Ethics approval

Not applicable. This is a case report based on the clinical notes of an individual patient where written consent for publication has been obtained from the next of kin.

Guarantor

Michael Kwok and Andrew Maurice.

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