

Case Report

Toxic megacolon: a rare presentation of complicated diverticular disease

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ABSTRACT

Toxic megacolon is a life-threatening complication most commonly associated with inflammatory bowel disease (IBD) or *Clostridioides difficile* infection. Its occurrence secondary to complicated diverticular disease is exceedingly rare. We present the case of a 56-year-old woman who developed toxic megacolon in the context of sigmoid diverticular disease. She initially received intravenous antibiotics for Hinchey IB diverticulitis and was discharged on oral therapy. Four days later, she returned with abdominal distension, severe leukocytosis, and radiographic evidence of colonic dilatation and pneumatosis. Emergency surgery revealed a perforated stenotic sigmoid tumor with ischemic changes, and a subtotal colectomy was performed. Histopathological analysis confirmed ischemic colitis with features of toxic megacolon. The patient had an uneventful recovery and was discharged on postoperative day seven. Although toxic megacolon is classically linked to IBD and *C. difficile*, clinicians must be aware of rare etiologies such as complicated diverticular disease. Prompt recognition and early surgical intervention are crucial for favorable outcomes in atypical presentations.

Keywords: Toxic megacolon, Diverticular disease, Subtotal colectomy

INTRODUCTION

We report a case of toxic megacolon in a middle-aged woman secondary to complicated diverticular disease. Toxic megacolon most frequently occurs in patients with IBD or *Clostridioides difficile* infection.¹ In this patient, early surgical intervention-without a previous course of

conservative medical management-led to a favorable outcome.

CASE REPORT

A 56-year-old female with a history of irritable bowel syndrome and no history of drug abuse presented with a

9-day history of abdominal pain initially localized to the left iliac fossa, accompanied by fever and abdominal tenderness. Five days prior to admission, the pain intensified and radiated to the left hypochondrium. The patient was admitted and treated with intravenous antibiotics (meropenem, ceftriaxone) for five days due to Hinchey IB diverticular disease. She was subsequently discharged with oral ciprofloxacin.

Four days later, she returned with a two-day history of worsening abdominal pain, significant abdominal distension, and absence of bowel movements. Laboratory findings showed marked leukocytosis ($34,180/\mu\text{L}$). An abdominopelvic CT scan revealed a sigmoid colon mass, colonic dilation, and pneumatosis of the colonic wall. She was taken urgently to the operating room, where a dilated cecum and ischemic changes in the ascending colon were found. A stenotic, perforated tumor approximately 7×6 cm in size was identified in the sigmoid colon, with ischemic changes in the adjacent wall. A subtotal colectomy was performed (Figure 1 and 2). The patient was discharged on postoperative day 7.

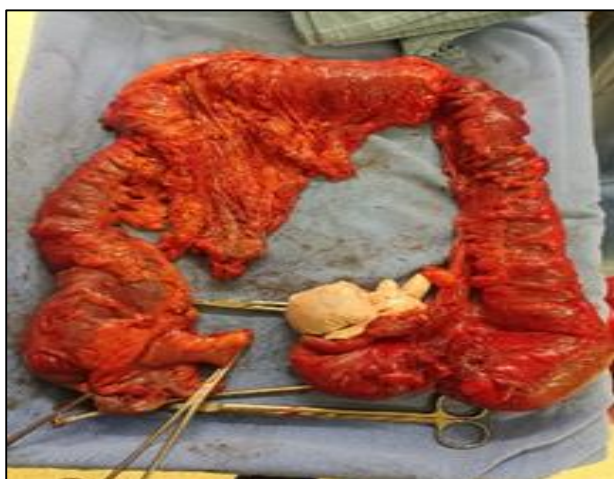


Figure 1: Subtotal colectomy surgical specimen.

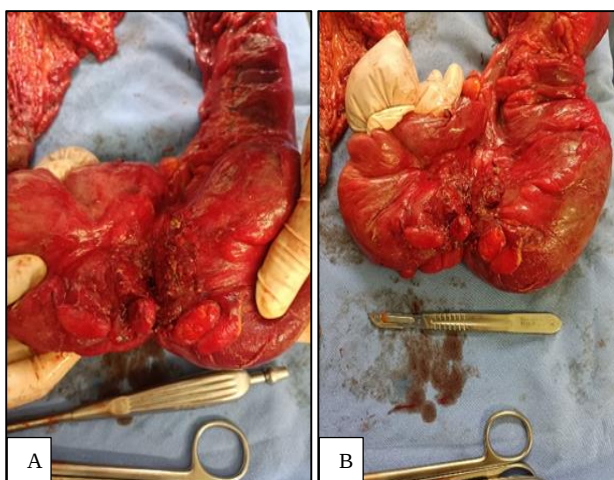


Figure 2 (A and B): Sigmoid colon tumor causing luminal stenosis.



Figure 3: Colonic dilation, and pneumatosis of the colonic wall.

Preoperative physical examination

The patient was somnolent with a heart rate of 130 bpm and a Glasgow coma scale score of 14. Lung fields were clear. The abdomen was distended, diffusely tympanic, and showed abolished peristalsis with superficial and deep tenderness.

Laboratory and imaging findings

First hospitalization (11.04.2024)

CBC: Hb-11.1 g/dl, Hct-34.4%, platelets- $672\times 10^9/\text{l}$, WBC- $22.19\times 10^9/\text{l}$ (Neutrophils-84%, lymphocytes-7.7%)
CMP: Cl-102 mmol/l, BUN-7 mg/dl, urea-14.5 mg/dl, Cr-0.46 mg/dl, Na-125 mmol/l, K-3.8 mmol/l and Cl-90 mmol/l

At discharge from first hospitalization

Hb-12.1 g/dl, Hct-36.6%, platelets- $905\times 10^9/\text{l}$, WBC- $14.93\times 10^9/\text{l}$ (Neutrophils- $12.54\times 10^9/\text{l}$), PT-13.5 sec, INR-1.16 and aPTT-30.3 sec

Second hospitalization

RBC- $4.77\times 10^{12}/\text{l}$, Hb-13.2 g/dl, Hct-39.9%, platelets- $855\times 10^9/\text{l}$, WBC- $37.03\times 10^9/\text{l}$ (Neutrophils- $34.18\times 10^9/\text{l}$, 92.3%), PT-16.8 sec, aPTT-26.5 sec, INR-1.45, glucose-141 mg/dl, BUN-12 mg/dl, urea-25 mg/dl, Cr-0.56 mg/dl, Na-131 mmol/l, K-3.6 mmol/l, Cl-88 mmol/l, Ca-9.3 mg/dl, P-3.88 mg/dl and Mg-2.3 mg/dl.

Histopathology

Findings were consistent with marked ischemic colitis and toxic megacolon. Focal ischemic necrosis of the intestinal wall with small vessel thrombosis was observed. No malignancy was identified.

DISCUSSION

Toxic megacolon is classically defined as non-obstructive colonic dilatation.¹ A more precise definition includes colonic dilation >6 cm with clinical signs of acute colitis and systemic toxicity.^{2,3} It affects men and women equally, although its incidence in the general population remains unclear.³ While it is most commonly associated with IBD, infectious etiologies such as *C. difficile*, *Salmonella*, *Shigella*, *Campylobacter*, and *Cytomegalovirus* (especially in HIV-positive patients) have also been documented.⁴⁻⁶ Contributing factors include the use of anticholinergic or narcotic medications, electrolyte imbalances, and diagnostic procedures like barium enemas or colonoscopy.⁷

The pathophysiology remains incompletely understood. However, altered colonic responses to cellular mediators resulting in decreased motility have been proposed. A predominant nitric oxide-mediated response causes smooth muscle relaxation.⁸ Additionally, involvement of the enteric nervous system-particularly the non-adrenergic, non-cholinergic inhibitory neurons stimulated by substance P-has been noted, especially in ulcerative colitis. An increase in neurons responsive to substance P and serotonin has also been observed.⁹

The association between inflammation and reduced colonic motility is supported by studies in guinea pig models, showing that infectious processes can impair neuromuscular transmission.¹⁰

In this case, we hypothesize that the colonic obstruction caused by the tumor led to increased intraluminal pressure and compromised perfusion, facilitating bacterial translocation and subsequent infection. This likely contributed to a severe motility disorder culminating in toxic megacolon. The patient had previously received antibiotics-her only known infectious risk factor. *C. difficile* infection has been associated with antibiotics like clindamycin, cephalosporins, and fluoroquinolones.¹¹⁻¹³ Although the patient did not exhibit diarrhea, *C. difficile* infection cannot be ruled out, as atypical presentations without diarrhea are documented.^{13,14}

Dumitru et al reported a similar case of toxic megacolon in a patient hospitalized for a colonic tumor complicated by *C. difficile* infection.¹⁵

Diagnostic criteria for toxic megacolon include radiographic evidence of colonic dilation (>5.5-6 cm) and at least three of the following: fever >38.6°C, heart rate >120 bpm, leukocytosis >10.5×10⁹/L, or anemia, plus one of the following: dehydration, altered mental status, or electrolyte abnormalities.¹⁶⁻¹⁸ Radiographic findings include colonic dilation (100%), abnormal haustration (96%), pericolic fat stranding (58%), and ascites (57%).¹⁷

Initial management includes fluid resuscitation, antibiotics, and-in the case of IBD-immunosuppressants, typically high-dose corticosteroids. Medical therapy should be initiated promptly to avoid colectomy. Surgical intervention is recommended if no improvement occurs within 2-3 days.^{12,13,19} In our patient, due to sigmoid stenosis and pneumatosis, urgent surgery was performed with satisfactory outcomes.

Mortality following subtotal colectomy for toxic megacolon varies based on the underlying condition, ranging from 57-80%.^{13,14} Dallal et al identified advanced age and vasopressor use as predictors of preoperative mortality.¹³

Histopathological findings in our patient revealed ischemic colitis and toxic megacolon. Only two other cases with similar histopathological findings have been reported-by Markoglou in 1993 and Torres in 1996.^{20,21}

CONCLUSION

Toxic megacolon is a severe complication arising from inflammatory processes affecting the colonic wall, often sharing a common pathophysiological mechanism. While IBD is the most common cause, various infectious and mechanical etiologies should be considered, as illustrated in this case of complicated diverticular disease.

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