Colon perforation due to collagenous colitis: a case report

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Abstract

Collagenous colitis (CC) is microscopic colitis characterized by watery diarrhea without bloody stools. The main treatment for CC is discontinuing the causative drugs and smoking cessation. Very few patients are refractory to drug treatment and require surgical management.

Background:

Collagenous colitis (CC) is microscopic colitis characterized by watery diarrhea without bloody stools. The main treatment for CC is discontinuing the causative drugs and smoking cessation. Very few patients are refractory to drug treatment and require surgical management.

Case presentation: A 58-year-old female presented to the hospital with the chief complaint of lower abdominal pain and was diagnosed with colonic perforation by computed tomography. The patient then was transferred to our hospital for surgery. Emergency surgery revealed perforation in the descending colon,
and partial colon resection and ileostomy were performed. Macroscopic examination revealed a longitudinal ulcer and no diverticulum. Histopathological examination revealed a collagenous colitis. The patient had a history of smoking, and was prescribed lansoprazole. She subsequently smoking, and lansoprazole was changed to famotidine. The patient had an uneventful post-procedural course without any complications and was discharged on day 10. Ileostomy closure was performed 3 months after discharge.

Conclusion:

Here, we report a case of colonic perforation caused by a CC. It is important for surgeons to cite CC as a differential diagnosis for colonic perforation of unknown origin.

Key words: collagenous colitis, colonic perforation, microscopic colitis

Background

Collagenous colitis (CC) is a microscopic colitis characterized by watery diarrhea without bloody stool [1]. CC often occurs in middle-aged females. Smoking and the use of medication of proton pump inhibitors (PPI) and nonsteroidal anti-inflammatory drugs (NSAIDs) are known risk factors [2]. The main treatment for CC is the discontinuing of medication and smoking cessation. Medication therapies such as glucocorticoids, budesonide and prednisone are added to the active state of the disease [3]. Very few patients are refractory to drug treatment and require surgical treatment [4].

Herein, we report the case of a 58-year-old female who underwent emergency surgery for colonic perforation due to CC.

Case Presentation:

A 58-year-old female, complaining of constant abdominal pain presented to the hospital. There were no symptoms of diarrhea. Her past medical history was hypertension, insomnia, constipation, and a 38-year smoking history. Her medications at presentation were lansoprazole, irbesartan, amlodipine besilate, and etizolam. She has been on these medication for several year. As computed tomography (CT) detected colon perforation, she was transferred to our hospital for surgery and intensive care. Her vital signs on arrival were as follows: blood pressure, 114/60 mmHg; pulse rate, 60/min, oxygen saturation, 99% on room air, and body temperature 37.2 degree in Celsius. Physical examination revealed left lower abdominal tenderness and rebound tenderness. The laboratory tests results were as follows: white blood cell counts of 11,500/μL; C-reactive protein, 1.95 mg/dl. An abdominal CT scan showed a thickened descending colon, accompanied by free air (Fig. 1). An emergency laparotomy was performed. The was a few purulent ascites in the abdomen. Although no obvious perforation site was identified, the descending colon was thickened and suspected to be the cause of perforation. Thus, partial colon resection and covering ileostomy were performed. The anastomosis was performed by functional end-to-end anastomosis. Three 19Fr drains were placed in the abdomen, and the operation was completed. Macroscopic examination of the resected colon revealed a longitudinal ulcer and no diverticulum (Fig. 2). The length of ulcer was 7.5 centimeters. Histopathological examination revealed a colonic subepithelial collagen band in the superficial epithelium on hematoxylin and eosin (HE) staining, which showed an edematous appearance of the submucosa and a generalized neutrophilic infiltrate, and CC was diagnosed (Fig. 3). Antibiotic therapy was continued for five postoperatively. Drains were removed on postoperative day 5. The patient was discharged without complications on postoperative day 9. There was no diverticulum or inflammatory bowel disease on histopathological examination. PPI therapy was thought cause of CC, and lansoprazole was changed to famotidine. After discharge from the hospital, she had no recurrence of abdominal pain of new-onset watery diarrhea. Ileostomy closure was performed 3 months after discharge.

Discussion:

Colonic perforation is a rare complication of CC. The leading cause is iatrogenicity, such as colonoscopy, and most of these perforations occur in the right-side colon [5]. Linear ulcerations and deep mucosal tears, observed mainly on the right side of the colon, are considered to be a risk of the perforation [6, 7]. Furthermore, spontaneous perforation in the present case is rare. Mori et al. reported seven cases of spontaneous colonic perforation associated with collagenous colitis [8]. All the patients underwent colon resection and recovered
well. Interestingly, these cases and the present case occurred in the descending colon, while spontaneous colon perforation unrelated to collagenous colitis commonly occurs in the sigmoid colon [9]. Therefore, it might be useful to consider the possibility of collagenous colitis if a descending colon perforation of unknown origin is observed.

Watery diarrhea is the main symptom of CC, but almost 10% of patients with CC have no typical symptoms [10]. The present case also had no history of diarrhea. The mechanism of diarrhea is associated with surface injury of the mucosal membrane due to inflammatory mediators in the luminal propria [11]. Although the present did not complain of diarrhea, histopathological examination showed an edematous appearance of the submucosa and a generalized neutrophilic infiltrate. This can lead to spontaneous colonic perforation of the colon.

The criteria for the histological diagnosis of CC are thickened subepithelial collagenous band\[?\]10\(\mu\)m [12]. In the present case, a thickened sub-epithelial band was also found around the perforation site. There were no clinical findings indicating diverticulitis, inflammatory bowel disease, or tumors. Furthermore, the present case took PPIs regularly. Therefore, we concluded that CC caused colon perforation in the present case.

**Conclusion:**

We encountered a rare complication of CC. In the case of colon perforation in an uncommon site, without diverticulum or tumor lesion, and when no obvious cause of perforation can be identified from the specimen, it is important to examine the patient’s background, particularly medication history, and symptoms in detail to identify the etiology and consider CC as a potential cause.

**Declarations**

**List of abbreviations:** CC: Collagenous colitis; PPI: proton pump inhibitors; NSAIDs nonsteroidal anti-inflammatory drugs; CT: computed tomography; HE staining: hematoxylin and eosin stain;

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**References**


Figure 3

(a)  

(b)