Colonic Perforation Secondary to Idiopathic Intramural Hemorrhage

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INTRODUCTION

Intramural colonic hemorrhage is rare and found in only 2% of patients with acute lower abdominal pain [1]. Most cases involve rectal hemorrhage and/or trauma or anticoagulation. Several reports of spontaneous intramural hemorrhage in the ascending colon due to a hematoma have been published. In this case report, we describe a patient with an ascending colonic perforation secondary to spontaneous intramural hemorrhage.

CASE REPORT

A 35-year-old male with a history of untreated hypertension presented to an outside facility with acute onset of abdominal pain. The patient was afebrile, and his vital signs were normal. Physical examination revealed localized abdominal tenderness in the right upper quadrant. Laboratory studies showed a white blood cell count of 19,200/μL and a serum creatinine of 1.27 mg/dL. Blood loss anemia was evident (hemoglobin decreased from 14.7 g/dL to 7.0 g/dL). An abdominal computed tomography scan (Fig. 1A) showed an area of increased density in the hepatic flexure and ascending colon. At this point, diagnostic considerations included ischemic colitis and diverticulitis.

Based on the patient's overall situation, the diagnosis of diverticulitis was made. The patient was admitted, and nonoperative management, including no oral intake and administration of cefazolin, was begun. On the fifth hospital day, the patient’s pain persisted, and laboratory tests showed anemia (hemoglobin decreased from 14.7 g/dL to 7.0 g/dL). A contrast-enhanced computed tomography scan revealed free air. An emergent right hemicolectomy was performed. Intramural hematoma and ischemia with perforation, with no obvious etiology, were found. The patient was discharged on the 14th postoperative day.

Keywords: Intramural hemorrhage; Colon; Perforation
Computed tomography scan was performed, which showed enlargement of the high-density area (Fig. 1B). The patient was then transferred to our hospital with the diagnosis of a large hematoma around the ascending colon. At the time of transfer, vital signs included a temperature of 38.3°C, a blood pressure of 179/128 mmHg, a pulse of 103 beats/min, and a respiratory rate of 16 breaths/min. Physical examination showed mild tenderness in the right upper quadrant. Nonoperative management was continued, with no oral intake and no antibiotic administration. Because the systolic blood pressure had been greater than 170 mmHg, nifedipine was given to limit additional bleeding due to high blood pressure. On the eighth hospital day, although the white blood cell count remained elevated at 12,700/μL, the serum hemoglobin was increased to 7.7 g/dL, and abdominal symptoms and physical findings were resolving, so oral intake was started.

Six hours after eating, the patient complained of recurrent and more severe abdominal pain. Physical exam revealed significant abdominal tenderness in the right upper quadrant. A computed tomography scan (Fig. 1C) showed free air in the abdominal cavity, so an emergent laparotomy was performed with a presumptive diagnosis of colonic perforation. Intraoperatively, a massive hematoma was found posterior to the ascending colon. The odor of feces was present, but no spillage of stool was noted. A right hemi-colectomy was performed.

Intraoperative examination of the specimen showed an intramural hematoma with an ischemic mucosa. At the center of the area of ischemia, a pinhole perforation was seen (Fig. 2). Histological findings showed hemorrhage spreading from the submucosal layer to the subserosal layer. In the subserosal layer, neutrophils, monocytes, proliferations of capillary vessels, and proliferations of fibroblast cells were seen, suggesting an inflammatory granulation response. These findings support the hypothesis that the subserosal findings were older than the perforation. The hemorrhage preceded the ischemia and perforation (Fig. 3). Based on this concept, we believe that spontaneous intramural bleeding resulted in ischemia followed by perforation. The patient was discharged 14 days postoperatively after an uneventful postoperative course.

**DISCUSSION**

Intramural hematomas in the alimentary tract are rare. When
they do occur, blunt trauma and anticoagulants are the typical etiologies. The majority of intramural hematomas have been reported in the duodenum and jejunum. Thirty-six percent of these patients were receiving anticoagulant therapy, and 40% were secondary to trauma [3]. Many patients taking anticoagulant medications have prolonged prothrombin times above the therapeutic range [3]. Colonic intramural hematomas are extremely rare, accounting for less than 5% of all intramural hematomas [4]. Most patients with intramural hematomas present with intestinal obstruction. Nonoperative therapy is usually sufficient because the hematomas will resorb [2].

Idiopathic colonic intramural hematomas are particularly rare [3]. One report has been published on a patient with a transverse colonic intramural hematoma that was managed nonoperatively; the patient was undergoing antiplatelet therapy [5]. An idiopathic hematoma in the transverse colon without a history of trauma or anticoagulants that was managed nonoperatively was reported by Umeda et al. [4], and an idiopathic hematoma in the transverse colon that was managed surgically was reported by Nozu [6]. However, to the best of our knowledge, no reports of idiopathic intramural hematomas of the colon with perforation have been published. The present patient did not have a history of trauma or hematologic disorders and was not receiving anticoagulant therapy. Based on clinical findings, the perforation occurred 8 days after the hematoma.

The normal mammalian response to injury occurs in 3 stages: inflammation, new tissue formation, and remodeling. The first stage occurs immediately after tissue damage. Neutrophils are recruited to the wound, and in the first few days, monocytes appear in the wound. The second stage of wound healing occurs from 2 to 10 days after injury and is characterized by cellular proliferation and migration of various cells. New blood vessels form, and the sprouts of capillaries associated with fibroblasts and macrophages (monocytes) replace the fibrin matrix with granulation tissue [7]. In this patient, the subserosal layer contained neutrophils, monocytes, proliferations of capillaries, and proliferations of fibroblasts, suggesting an inflammatory granulation response. These histological findings suggest that the hemorrhage preceded ischemia and perforation by more than 2 days (Fig. 3). We believe that spontaneous intramural bleeding led to ischemia, followed by perforation.

The findings in this patient provide indirect evidence of a colonic perforation secondary to an idiopathic intramural hematoma. Another possible explanation is a minor perforation in the area of diverticulitis with bleeding that sealed and then reperforated due to ischemia caused by the hematoma. The imaging studies and pathologic evaluation of the specimen did not show evidence of diverticular disease suggesting an idiopathic etiology, but this can also explain the clinical course of this patient. In conclusion, colonic perforation secondary to an idiopathic intramural hematoma is very rare. In our patient, enlargement of the hematoma likely led to ischemia and perforation. Thus, a patient with an intramural hematoma in the gastrointestinal tract should be monitored closely, and surgical treatment should be considered if the hematoma is progressively enlarging.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.
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