Case Report

A Case of Ischemic Colonic Stenosis of the Splenic Flexure

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Abstract : Ischemic colitis is characterized by lesions arising from colonic ischemia. The treatment of choice is surgery, and resection of the affected segment is often life saving. This study presents a case of segmental ischemic colonic stenosis of the splenic flexure. A 70-year-old woman was admitted to our hospital with abdominal pain and distension. Physical examination revealed mild tenderness of the left-upper abdomen but no peritoneal signs. A computed tomography scan demonstrated a thickening of the splenic flexure of the colon with active inflammation. A gastrografin enema revealed a 5-cm-long tight stricture at the left transverse colon, which suggested a subileus. Surgery for segmental ischemic colonic stenosis was performed because the stricture did not respond to treatment. Pathological examination revealed features typical of ischemic colitis, including ulceration and segmental colonic stenosis of the splenic flexure, but revealed no evidence of tumors, lymph node swelling, or vascular disorder.

Key words: stenosis, ischemic colitis, splenic flexure

Introduction

Ischemic colitis is the most common ischemic injury of the gastrointestinal tract. It is caused by the decrease or cessation of blood perfusion in the colon wall, which results in ischemic changes in the colon wall. Although it can occur at any age, approximately 90% of patients are over 60 years of age¹⁾. Ischemia can cause abdominal pain, diarrhea, hematochezia, stools with mucous and blood, and other uncharacteristic symptoms, making diagnosis at an early stage difficult²⁻⁵⁾. We present a case of segmental ischemic colonic stenosis of the splenic flexure, due to cryptogenic ischemic colitis.

Case Report

A 70-year-old woman without a medical history was admitted to our hospital for abdominal pain and abdominal distension. On the day of admission, her vital signs were normal except for her body temperature, which was 37.2°C. Physical examination revealed mild

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tenderness of the left upper abdomen but no peritoneal signs. Initial laboratory test results were abnormal as follows: white blood cell count (WBC), 4.800 cells / μ L (normal range: $4,000 \text{ cells / mm}^3 - 9,000 \text{ cells / } \mu\text{L}$; hemoglobin, 14.7 g / dL (normal range: 12.0 - 16.0 g /dL); platelet count, 195×10^3 cells / μ L (normal range: $150 \times 10^3 - 350 \times 10^3$ cells / μ L); and C-reactive protein (CRP), 0.2 mg/dL (normal range: < 0.2 mg/dL). The results for the electrolytes and coagulation tests were normal. The patient did not have a significant medical history. Abdominal radiography showed intestinal distension with some air-fluid levels in the central abdomen. Computed tomography (CT) scans demonstrated a thickening of the colon with active inflammation, and no evidence of lymph node swelling (Fig. 1). Colonoscopy findings demonstrated an edematous mucosa and cicatrization with contact bleeding and ulceration, revealing segmental stenosis of the mucosa extending at the splenic flexure (Fig. 2). The rectum and sigmoid colon appeared to be free from disease. Biopsy samples of colonoscopy revealed inflammatory cell infiltration and hemorrhage in the mucosa. The patient was diagnosed with subileus and ischemic colonic stenosis of the splenic flexure. The initial management was conservative with intravenous hyperalimentation and intravenous antibiotics. Colonoscopy performed 2 weeks after admission revealed no changes to the stenosis. A gastrografin enema revealed a 5-cm-long tight stricture at the left transverse colon (Fig. 3). As the initial treatment proved unsuccessful, a colectomy for segmental ischemic colonic stenosis of the splenic flexure was performed. Pathological examination of the lumen of the resected specimen revealed features typical of ischemic colitis, including ulceration and segmental ischemic colonic stenosis of the splenic flexure (Fig. 4), but revealed no evidence of vascular disorder or inflammatory bowel disease (IBD).

The origin of the ulceration and stenosis was due to cryptogenic ischemic colitis without vascular disorder or IBD. A conservative surgical option was preferred to avoid an extensive bowel resection, and a segmental resection was performed with a functional end-to-end anastomosis. The postoperative course was uneventful with clinical and biological parameters of inflammation returning to normal within 7 days. There was no recurrence 3 months after surgery.

Discussion

Ischemic colitis is a well-recognized clinical phenomenon, although its precise etiology remains unclear. It may manifest with a wide spectrum of severity, ranging from mild, transient mucosal erosion to fibrous scarring with stricture formation and even transmural infarction. Some cases are caused by acute macrovascular mesenteric occlusion due to surgical trauma⁶, thromboembolism⁷⁻⁹, or atherosclerosis¹⁰. Ischemic colitis typically develops spontaneously without signs of major vascular occlusion, and healthy intestine is present elsewhere in the tract. Isolated case reports have described the development of ischemic colitis in conjunction with mild allergy, hypertension, rectal prolapse, acute pancreatitis, sickle cell crisis, colon cancer, systemic lupus erythematosus, amyloidosis, anticardiolipin antibody syn-

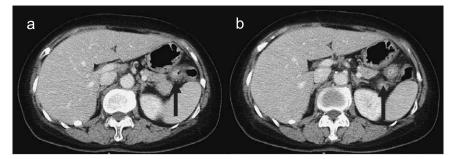


Fig. 1. Computed tomography

CT demonstrated a thickening of colon with an active inflammation (a) and no evidence of lymph node swelling (b).

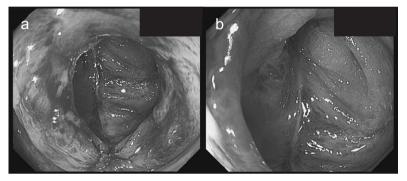


Fig. 2. Colonoscopy

Colonoscopic findings demonstrated an edematous mucosa and a cicatrization with contact bleeding and ulceration (a), which revealed a segmental stenosis of the mucosa extending at the splenic flexure (b).



Fig. 3. Gastrografin enema A gastrografin enema revealed a 5 cm tight stricture at left transverse colon (arrow).



Fig. 4. Resected specimen

A lumen of resected specimen showed a segmental ischemic colon stenosis in connection with ulceration of ischemic colitis.

drome, Buerger's disease, and Kawasaki syndrome¹¹⁻¹³⁾. Other case reports have described associations between the development of ischemic colitis and the use of some agents (such as progesterone, ergotamine derivatives, nonsteroidal anti-inflammatory drugs, and danazol)¹⁴⁾, intravenous vasopressin therapy¹⁵⁾, renal transplantation¹⁶⁾, chronic intermittent peritoneal dialysis¹⁷⁾, cocaine abuse, snake bite, and marathon running¹¹⁾. Clinical presentation is usually acute, with abrupt onset of cramping abdominal pain, abdominal distension, and bloody diarrhea. There may be local signs of peritoneal irritation over the affected segment, and if mucosal ulceration is present, bacterial invasion may also occur. However, manifestations vary widely, from severe pain with transmural infarction and early perforation to mild abdominal pain and only slight tenderness¹⁸⁾. Non-characteristic symptoms make it extremely difficult to distinguish between colitis, chronic ulcerative colitis, intestinal parasitosis, and even early colon tumors.

The diagnosis of ischemic colitis in our patient was established by colonoscopy and abdominal CT. This diagnosis was confirmed by colonoscopic examination and biopsy. As the local lumen was narrow, biopsies were performed to rule out malignant disease, other inflammatory diseases, and non-specific intestinal diseases. The results showed hyperplasia of the fibrous connective tissue in the biopsied sample. Therefore, it is believed that the narrowing of the lumen was closely related to hyperplasia of the fibrous connective tissue. Pathologically, hyperplasia of fibrous connective tissue reflects long-term ischemia. However, histological examination revealed no evidence of carcinoma.

Generally, major arterial or venous branches are easily detected by arterial or venous phase CT. Three-dimensional (3D) angiography based on multidetector-row computed tomography (3D-CT angiography) was able to sufficiently evaluate the anatomy of colonic arteries and communication between colonic marginal arteries. This information is useful in preoperative planning for vascular dissection and additional microvascular anastomosis¹⁹. Arterial and venous phase CT revealed that the patient in this study had a normal superior mesenteric artery (SMA), inferior mesenteric artery (IMA), splenic artery, proximal superior mesenteric vein (SMV), inferior mesenteric vein (IMV), and splenic vein. Ischemic colitis is induced by poor blood supply to the colon wall, which results in ischemic change.

Diagnosis of colonic ischemia is confirmed by colonoscopy and/or barium enema. One potent treatment is to vasodilate the surrounding blood vessels to recover the blood supply. This acts to resolve the ischemia of the colon wall by reducing clinical manifestations, restoring colon wall function, and preventing colon wall necrosis, which occurs when local ischemia develops into general ischemia⁴⁾. Nongangrenous ischemic colitis usually requires only conservative therapy, including repeated careful assessment, pain control, and fluid replacement, and it is associated with a good prognosis. It may lead to the sequelae of persistent segmental colitis or colonic strictures, occasionally requiring surgery. Urgent surgery and high morbidity and mortality rates are hallmarks of the gangrenous type of ischemic colitis²⁰⁾. Although the patient in this study is currently doing well after surgery,

we believe she requires a long-term follow-up period and prompt recognition of persistent disease.

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