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CASE REPORT

Conservative treatment of idiopathic mesenteric phlebosclerosis

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Summary Ischemic bowel disease is caused by occlusion or stenosis either of the mesenteric artery or, less frequently, of the vein. Phlebosclerotic colitis is a rare chronic and gradually worsening ischemic colitis caused by mesenteric phlebosclerosis, and its symptoms are the gradual onset of right-side abdominal pain with or without diarrhea, and recurrent ileus. The most commonly affected site is the region from the terminal ileum to the sigmoid colon, particularly the cecum and ascending colon. The characteristic radiologic findings of mesenteric phlebosclerosis are luminal narrowing, wall thickening of the affected colon (typically the proximal colon), and calcification of the mesenteric veins. The diagnosis may be made on the basis of imaging findings. We report on a 59-year-old man with periumbilical abdominal pain for 1 week. The pain was dull and persistent. A physical examination revealed a soft, tender abdomen with normoactive bowel sounds and mild rebounding tenderness in the right upper abdomen. The abdominal plain X-ray and abdominal computed-tomography findings were compatible with idiopathic mesenteric phlebosclerosis. The symptoms resolved after only conservative treatment, and the disease did not recur during 5 years of follow-up. Idiopathic mesenteric phlebosclerosis is a rare disease, and its pathogenesis is not yet clearly understood; however, it differs from other rare causes of nonthrombotic mesenteric venous occlusion. Idiopathic mesenteric phlebosclerosis can be diagnosed on the basis of imaging findings, and most symptoms resolve only after conservative treatment.

Conflicts of interest: All authors declare that they have no conflicts of interest.

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1. Introduction

Ischemic bowel disease varies from full-thickness necrosis or gangrene to transient inflammation. The disease is caused by occlusion or stenosis either of the mesenteric artery or, less frequently, the vein. Some cases are idiopathic, whereas others may be due to decreased perfusion in the absence of a vascular lesion.[1] Phlebosclerotic colitis is a rare chronic and gradually worsening ischemic colitis caused by phlebosclerosis of the mesenteric vein and its tributaries.[1] Its symptoms are the gradual onset of right-side abdominal pain with or without diarrhea, and recurrent ileus. The most commonly affected site is the region from the terminal ileum to the sigmoid colon, particularly the cecum and ascending colon.[1,2]

The characteristic radiologic findings of mesenteric phlebosclerosis are luminal narrowing, wall thickening of the affected colon (typically the proximal colon), and calcification of the mesenteric veins. Plain radiographs and computed tomography can easily detect the characteristic linear calcification of numerous branches of the mesenteric vein at the affected site.[2–5]

An endoscopic examination reveals a dark-purple, edematous mucosa, particularly in the ascending colon. Occasionally, it may be accompanied by various types of ulcer.[2] Endoscopic biopsies of the lesion site may reveal characteristic mucosal changes in ischemic colitis.[1] However, a limited endoscopic examination of the less involved segment or a biopsy of nonspecific ulcers or congested mucosa may result in misdiagnosis;[6] therefore, the diagnosis may be made on the basis of imaging findings alone.[1,3,7]

We present a rare case of idiopathic mesenteric phlebosclerosis successfully managed conservatively.

2. Case Report

A 59-year-old man who received an appendectomy 20 years previously presented with dull and persistent periumbilical abdominal pain, which persisted for 1 week. He denied having a past medical history of hypertension, cerebral vascular disease, chronic hepatitis, diabetes, and hyperlipidemia, as well as long-term Chinese herb usage. A physical examination revealed a soft, tender abdomen with normoactive bowel sounds, and mild rebounding tenderness in the right upper abdomen. Laboratory tests revealed a leukocyte count of 9900/mm^3 with 84% being neutrophils, and a C-reactive protein level of 11.3 mg/dL. The other laboratory data were normal. A plain abdominal radiograph revealed diffuse treelike calcified plaques in the superior mesentery vein and its branches (Fig. 1). Moreover, an abdominal computed tomography revealed diffuse arteriosclerotic calcified plaques in the superior mesentery vein, inferior mesentery vein, and their branches, and a diffusely edematous and mildly enhanced thickened bowel wall, particularly in the right side of the colon (Fig. 2). These findings were compatible with idiopathic mesenteric phlebosclerosis. In addition, an angiography revealed no definite arterial occlusion or stenosis, and the venous return of the ascending colon was delayed and poor (Fig. 3). The patient received conservative treatment with fasting, parenteral first-generation cephalosporin, and aminoglycoside after admission. No notable peritoneal sign was observed, and the symptoms improved the day following admission. He began sipping water and ate a soft diet on the 3rd day. The symptoms subsided and he was discharged on the 5th day. The symptoms did not recur during 5 years of follow-up. A follow-up plain abdominal radiograph did not reveal any significant changes, and the laboratory data were normal; however, he has been lost to follow-up since then.

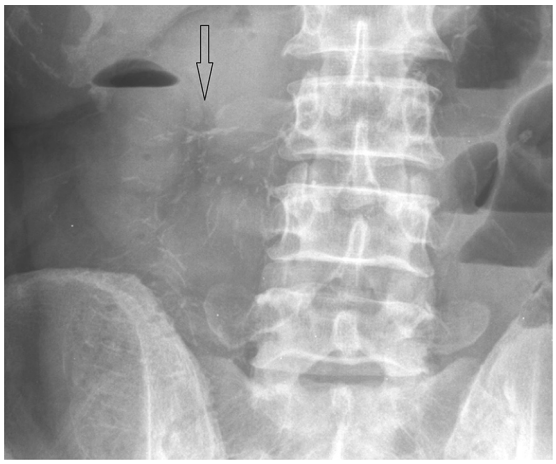

Figure 1 A plain abdominal radiograph depicting diffuse treelike calcified plaques in the superior mesentery vein and its branches (arrow).

3. Discussion

Idiopathic mesenteric phlebosclerosis is a rare disease. It is diagnosed only according to specific imaging findings,[1,3,7] and cannot be diagnosed according to laboratory tests. The pathogenesis is not yet clearly understood; however, it differs from other rare causes of nonthrombotic mesenteric venous occlusion.[6] A histological examination has shown marked fibrous thickening of the venous wall with calcification of small mesenteric veins and their intramural branches. Moreover, submucosal fibrosis and deposition of collagen around the mucosal vein without thrombi in the affected vessels have been reported.[1,2,6]

Mesenteric venous abnormalities may cause ischemic colitis, resulting in progressive fibrosis, calcification, and obstruction of the colonic and mesenteric veins. Subsequently, the affected colon wall becomes thickened, which interferes with motility and may lead to subserosal calcifications and luminal stenosis.[3] In addition, myointimal hyperplasia may be a common secondary reactive change in an insult to blood vessels, and myointimal hyperplasia may accelerate vascular occlusion.[6] The change starts as a slow and long-term direct hypoxic injury to the venous muscular layer, which leads to gradual mummification and subsequent sclerosis and calcification. The damaged veins typically have reactive myointimal hyperplasia and repeated mummification injury, and calcification results in gradual lumen occlusion.[6]

Calcification of thickened venous walls is rarely observed in the portal vein and its tributaries; however, it