**CASE REPORT**

**Phlebosclerotic Colitis: an Unusual Cause of Ischaemic Colitis in a 65-year-old Man**

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**ABSTRACT**

Ischaemic colitis is a well-known entity that yields a wide spectrum of pathological and clinical findings. Different precipitating factors have been incriminated. Phlebosclerosis involving venous drainage into the colon has been previously described in Japanese patients as a unique form of ischaemic colitis. This report presents the first known Chinese patient with a histologically proven case of phlebosclerotic colitis. Abdominal radiography showed threadlike branching calcification over the ascending colon along and perpendicular to the bowel wall. Computed tomography of the abdomen and pelvis demonstrated colonic distension and marked symmetrical colonic mural thickening with a target appearance predominantly in the ascending colon and caecum. Extensive calcification was noted in the entire wall (on both mesenteric and antimesenteric sides) of the affected colon, along its adjacent mesenteric vascular arcade, and in the wall of branches of superior mesenteric vein. Computed tomography angiography showed that the major branches of the superior mesenteric artery and vein were opacified. Volume-rendered images and maximal intensity projections showed that the calcifications were associated with veins and not with the arteries. The use of multidetector computed tomography is thus stressed as the imaging modality of choice.

**Key Words:** Calcinosis; Colitis, ischemic/etiology; Colon/blood supply; Sclerosis; Tomography, X-ray computed

**INTRODUCTION**

Ischaemic injury to the colon is a well-known entity that has a wide spectrum of pathological and clinical findings, ranging from mild self-limiting disease to bowel infarction and perforation. The term ‘ischaemic colitis’ is commonly used to describe a form of non-occlusive ischaemic disease that involves the arterial supply of the colon. Precipitating factors, such as atherosclerotic vascular disease, hypotensive episodes, arrhythmias, and colonic obstruction have been incriminated. Acute venous obstruction can also give rise to bowel ischaemia, whereas chronic venous insufficiency is rarely associated with ischaemic colitis.

We present a case of ischaemic colitis with clinical and pathological findings of phlebosclerotic colitis. The characteristic imaging findings are discussed, with emphasis on non-invasive means of establishing the diagnosis.

**CASE REPORT**

A 65-year-old Chinese man first presented to the Queen Mary Hospital in August 2002 with a 1-month history of non-specific abdominal pain, diarrhoea, and weight loss. He was a chronic smoker and alcohol drinker, but had been healthy before presentation. On physical examination, he had a generalised skin discoloration and was dehydrated but haemodynamically stable. The abdomen was non-tender, and no hepatosplenomegaly or mass detected. Signs of chronic liver disease were also absent. Initial blood tests showed a haemoglobin level of 45 g/L (normal range, 140-175 g/L), serum albumin level of 25 g/L (normal range, 35-50 g/L), plasma potassium level of 2.7 mmol/L (normal range, 3.5-5.0 mmol/L). Liver function was otherwise normal.

Abdominal radiography performed at the time of admission showed a dilated large intestine with a nodular mucosal outline and a thumbprint appearance. Thread-like branching calcification was evident over the region...
Phlebosclerotic Colitis: an Unusual Cause of Ischaemic Colitis

of the ascending colon (Figure 1). These calcifications ran both along and perpendicular to the bowel wall. Computed tomography (CT) of the abdomen and pelvis demonstrated colonic distension and marked symmetrical colonic mural thickening with a target appearance; these features predominantly affected the ascending colon and caecum. A short segment of the terminal ileum was also mildly thickened. The transverse colon and descending colon were affected but to a lesser extent. Extensive calcification was noted in the entire wall (on both mesenteric and antimesenteric sides) of the affected colon, along its adjacent mesenteric vascular arcade, and in the wall of branches of superior mesenteric vein (SMV). The inferior mesenteric vein branches were spared. Pericolic stranding and fluid at the bilateral paracolic gutters were present, but pneumatosis and portal venous gas were absent. The rest of the small intestine appeared unremarkable (Figures 2a and 2b). CT angiography showed that the major branches of the superior mesenteric artery (SMA) and SMV were opacified (Figures 3a and 3b). Volume-rendered images and maximal intensity projections showed that the calcifications were associated with veins and not with the arteries (Figure 3b).

Magnetic resonance imaging (MRI) and magnetic resonance arteriography of the abdomen was performed because of heavy vascular calcification, which may have obscured the assessment of luminal patency by CT. These examinations showed colonic mural thickening and oedema that were more marked on the right side of the colon than the left, which was in keeping with colitis (Figures 2c and 2d). Magnetic resonance angiography showed patent main trunks and branches of the SMA and SMV, including the ileocolic and right colic branches (Figures 4a and 4b). The overall radiological features were compatible with the previously described entity of phlebosclerotic colitis.

Colonoscopy showed a loss of normal haustration in the caecum and in the ascending and transverse colon, as well as diffuse pigmentation and ‘melanos’ (Figure 5a). Sigmoid and descending colons were similarly affected but to a lesser extent and with preserved haustration (Figure 5b). Two colonic polyps were also found, and these were removed by snare polypectomy. Histopathological findings after colonic biopsy confirmed the diagnosis of phlebosclerotic colitis — that is, prominent hyalinisation and fibrosis were present in the perivascular tissue and lamina propria, especially in the ascending colon. No evidence of amyloidosis, vasculitis, or viral inclusion bodies was detected. The 2 colonic polyps that had been removed were a villotubular adenoma and a tubular adenoma.

Owing to the abnormal skin pigmentation of this patient, various toxicology and biochemical screening tests were performed, including those to detect arsenic, uroporphyrin and coproporphyrin, adrenocorticotropic hormone, cadmium, manganese, copper, zinc, and lead levels. All levels were normal. Results of tests for autoimmune markers were also negative, as were serological test results for hepatitis B and hepatitis C. Ultrasonography of the liver showed no evidence of cirrhosis or portal hypertension. The spleen was also not enlarged. This patient was given conservative treatment. Diarrhoea gradually subsided and pigmentation improved after the cessation of alcohol drinking.

Follow-up CT was performed 1 year later and showed a reduction in the degree and extent of colonic mural thickening, as well as resolution of both pericolic fluid collection and inflammatory change. Vascular calcification remained similar.

DISCUSSION

Intra-abdominal visceral venous calcification is an uncommon phenomenon. Most of the vascular calcification identified on abdominal radiographs is arterial in nature. Extensive calcification in the portovenous
Figure 2. (a) and (b) Cross-sectional postcontrast axial computed tomograms showing colonic mural thickening and target appearance predominantly in the proximal transverse and ascending colon and the caecum (arrows), and characteristic calcification deep in the walls of the right-sided colon along the venous vascular arcades and draining into branches of superior mesenteric veins (arrowheads); the open arrow in (b) indicates pericolic stranding and free-fluid collection at the bilateral paracolic gutters. (c) and (d) Postcontrast axial magnetic resonance image of the abdomen at similar levels showing colonic mural thickening of the ascending colon and transverse colon (arrows); venous calcification was difficult to visualise.

Figure 3. Computed tomograms using a 16-slice multidetector scanner: (a) 3-dimensional reconstruction showing extensive, branching calcification along the ascending and transverse colon (arrows); (b) sagittal thin-slab maximal-intensity projection image showing patent arterial branches of the superior mesenteric artery, as well as venous calcification (arrowheads).
system has been described in patients with cirrhosis and portal hypertension. An incidence of 11% has been reported for patients with advanced cirrhosis. The calcification noted was located in the portal vein in as many as 88% of the patients, the splenic vein in 50%, and in the SMV in 50%; it was absent from the inferior mesenteric vein. In these published reports, no association with colitis was mentioned. The presence of calcification in the tributaries of the SMV in patients with colitis was first described in the Japanese literature by Koyama et al in 1991. The condition was named phlebosclerotic colitis. In the medical literature to date, only 19 cases have been reported — all of these among Japanese patients.

The most common clinical symptoms of phlebosclerotic colitis are abdominal pain and diarrhoea. The duration of illness is usually fairly long. In most cases, the right side of the colon is affected and the condition gradual extends to involve the distal colon. The cause of the symptoms is presumed to be chronic venous insufficiency and venous congestion. The characteristic radiological findings include the following: multiple threadlike calcifications along the right hemicolon on plain radiographs; thickening with calcifications along the colonic wall and around the veins near the SMV trunk on CT scans; disappearance of semilunar folds, luminal irregularities, rigidity, narrowing, and ‘thumbprinting’ in the right hemicolon on a barium enema examination; and narrowing of the marginal arteries, tortuosity of the vasa recta, and dilatation and tortuosity of the veins along the vasa recta on angiograms.

Characteristic clinical and histopathological findings include the presence in the right hemicolon, a dark purple mucosa, a marked thickening of the wall with an absence of the plicae semilunaris coli, and microscopic marked fibrous thickening of the venous wall with calcification, associated with marked submucosal fibrosis. No common underlying disease or predisposing factors exist, and the pathogenesis of the condition remains unclear. Speculations concerning its origin include chemical irritants and increased intraluminal pressure in the right side of the colon. In the Chinese population, as with the Japanese population, right-sided colonic diverticula are also more common than in 

Figure 4. (a) Contrast-enhanced magnetic resonance venogram showing that the main trunk of the superior mesenteric vein (arrows) and its major branches, including the ileocolic veins (arrowheads), are patent. (b) Contrast-enhanced magnetic resonance angiogram at the arterial phase showing the patent superior mesenteric artery (arrow) and its major branches (arrowheads).
western patients. Even though ours is the first case report of phlebosclerotic colitis in the Chinese population, a common aetiological agent could cause both conditions. Alcohol consumption may also play a role in the pathogenesis of the condition, as in our case, because cessation of alcohol drinking was associated with a reduction in disease severity. Reports of more cases in the future will probably unravel the mystery of this idiopathic condition.

Although very little is known about the exact aetiology, pathogenesis, and clinical course of phlebosclerotic colitis, all the cases reported so far have had unique radiological findings. In patients presenting with abdominal symptoms, plain abdominal radiographs are commonly taken. Awareness of the typical branching vascular calcification on a plain abdominal radiograph should prompt further investigation. Among various investigations, CT should be the imaging modality of choice. With the use of multislice CT, we are now able to produce 3-dimensional CT angiography and reformatted CT images. Both can demonstrate the vascular anatomy in high quality and can pinpoint the exact location of the calcification along the veins and in the colonic walls. The patency of the vessels can be demonstrated as a way of excluding thrombosis. We are also able to delineate the extent of disease involvement and to look for pericolic stranding and peritoneal fluid collection, which indicate the presence of active colitis.

Furthermore, with the use of CT, other causes of abdominal symptoms may be identified because phlebosclerotic colitis itself appears to be a long-standing condition associated with episodes of exacerbation, as demonstrated in our case. CT scans can also demonstrate possible complications in extreme cases, which include colonic infarction and perforation. Another advantage of using CT is the fact that the procedure is relatively non-invasive, and the information that can be gathered on the CT images is already adequate for a confident diagnosis of phlebosclerotic colitis to be made. This approach obviates the need for the more invasive procedure of abdominal angiography. MRI is probably not the first method of choice, because calcifications are usually not well visualised on MRI. However, the insensitivity to the blooming effect of calcium could allow a more confident diagnosis of vessel patency to be established with MRI than with CT.

In conclusion, phlebosclerotic colitis is a rare condition with an unknown cause. With its distinctive radiological features, a confident diagnosis can be made with
Phlebosclerotic Colitis: an Unusual Cause of Ischaemic Colitis

various radiological investigations. Awareness of this condition may allow more cases to be identified and shed more light on its cause.

REFERENCES


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