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Acute Superior Mesenteric Arterial Occlusion Caused by a Thoracic Aortic Mural Thrombus in a Patient Without Coagulation Disorder or Aortic Disease: A Case Report

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Introduction: Aortic mural thrombus (AMT) formation in a patient without hypercoagulability and preexisting aortic disease is very rare, and acute superior mesenteric artery (SMA) occlusion resulting from AMT is clinically infrequent.

Case presentation: A 70-year-old woman was transferred to our institute with a diagnosis of acute SMA occlusion. Contrast-enhanced computed tomography (CECT) revealed thromboemboli in the SMA and a thoracic AMT. She immediately underwent angiography and subsequent endovascular aspiration and transcatheter infusion, which recanalized the SMA. However, CECT suggested intestinal ischemia; therefore, she underwent intestinal resection, and we administered anticoagulant therapy with heparin and warfarin postoperatively for the thoracic AMT. Although repeat thrombus formation developed in an ileal branch despite anticoagulant therapy, she had no abdominal symptoms; therefore, we continued the anticoagulant therapy. CECT 3 months later confirmed the absence of thoracic AMT and normal ileal findings.

Conclusion: We report a case of acute SMA occlusion and preceding splenic infarction that resulted from thoracic AMT in a patient without hypercoagulability and preexisting aortic disease. AMT should always be considered as a cause of acute SMA occlusion in patients without heart disease, including atrial fibrillation or severe arteriosclerosis.

Key words: Superior mesenteric artery – Mesentery vascular occlusion – Embolism – Thoracic arteries – Thrombosis

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Acute superior mesenteric artery (SMA) occlusion leads to intestinal and mesenteric ischemia and infarction, and is a life-threatening emergency. The causes of acute SMA occlusion are emboli or thrombi (embolus/thrombus ratio 1.4:1), and most result from heart disease (atrial fibrillation, valvular disorders, and myocardial infarction) and arteriosclerosis.[1] Aortic mural thrombus (AMT) is an infrequent cause of acute SMA occlusion, and patients without hypercoagulability or aortic disease are rarely affected with AMT.[2] We discuss a female patient without hypercoagulability or aortic disease who suffered acute SMA obstruction resulting from thrombosis of the thoracic aorta.

Case Report

A 70-year-old female complained of sudden abdominal pain and vomiting after breakfast and presented to her local hospital with melena. Contrast-enhanced computed tomography (CECT) findings showed superior mesenteric arterial occlusion; therefore, she was transferred to our institute. She had experienced cryptogenic splenic infarction 3 months earlier with several examinations, such as CECT, echocardiography, and anticoagulant tests, at that time. She had no known risk factors for atherosclerosis or coagulation abnormalities. At presentation at our institute, she had severe abdominal pain without peritoneal irritation signs, which had persisted for more than 6 hours. Laboratory data showed a white blood cell count of $2.34 \times 104/\mu L$, with elevated D-dimer (3.7 μg/mL) and lactate (36.0 mg/dL) levels. CECT revealed a thoracic aortic thrombus and ischemic changes in the small intestine and ascending colon, resulting from SMA occlusion (Fig. 1A and 1B). We diagnosed acute SMA obstruction without irreversible ischemic intestine and peritonitis and decided to perform emergency angiography first. Angiography findings showed complete SMA occlusion (distant to the right colic artery) and some of the jejunal arteries, and we were unable to visualize most of the small intestine and ascending colon (Fig. 2C). Immediate endovascular aspiration and transcatheter infusion of urokinase (480,000 IU) resulted in recanalization of the SMA and branch arteries (Fig. 2D), and good visualization of the intestine, so she did not undergo bowel resection on the first day. However, daily laboratory data revealed persistently high levels of inflammatory markers, and CECT was performed on the 6th day of admission. CECT findings revealed widespread ischemic changes in the terminal ileum (Fig. 2A); therefore, laparoscopic inspection was performed. Laparoscopic findings showed ischemic change and aperistalsis of the terminal ileum (Fig. 2B) and normal findings in the ascending colon. We also performed fluorescence imaging with indocyanine green to evaluate intestinal blood flow. Fluorescence images indicated a clear demarcation line between the ischemic and non-ischemic ileum (Fig. 2C); therefore, we decided to resect the ischemic ileum. We determined the extent of resection using a Doppler flow study and resected 90 cm of ischemic ileum. Operative time was 164 minutes and intraoperative blood loss was 40 g, and the resected specimen showed gross ischemic changes. Anticoagulation therapy for thoracic AMT was started with heparin on postoperative day (POD) 1 and with warfarin on POD 3. Despite anticoagulant therapy, CECT on POD 5 revealed a new thrombus in an ileal branch and localized ischemic changes in the remaining ileum (Fig. 3). However, the patient had no abdominal symptoms; therefore, we continued conservative management. She resumed oral intake on POD 7 and was discharged 20 days after the surgery. Follow-up CECT 3 months later confirmed the absence of thoracic AMT and normal ileal findings.

Discussion

Acute SMA occlusion is a life-threatening emergency requiring immediate diagnosis and treatment. The overall incidence of acute SMA occlusion is approximately 8.7/100,000 person-years.[3] The most important examination for early diagnosis is CECT with rapid image reconstruction in the sagittal, coronal, and transverse planes.[4] There is no specific plasma marker for early diagnosis. CECT findings reveal the cause and location of the occlusion in the middle or distal part of the SMA, with or without intestinal ischemia, and patients with intestinal ischemia require laparotomy and resection of necrotic intestine. Muneoka and colleagues indicated that the time limit for irreversible intestinal ischemia is within 5 hours in patients with occlusion in the middle SMA (proximal to the right colic artery) and within 12 hours in patients with occlusion in the distal SMA.[5] In *in vivo* experiments, mucosal change was detected 1 hour after SMA ligation and all layers of the intestinal wall became necrotic 8 hours after SMA ligation.[6]

As to treatment strategy for acute SMA occlusion, Acosta and his colleagues proposed a surgical algorithm.[3] Patients with peritonitis should undergo

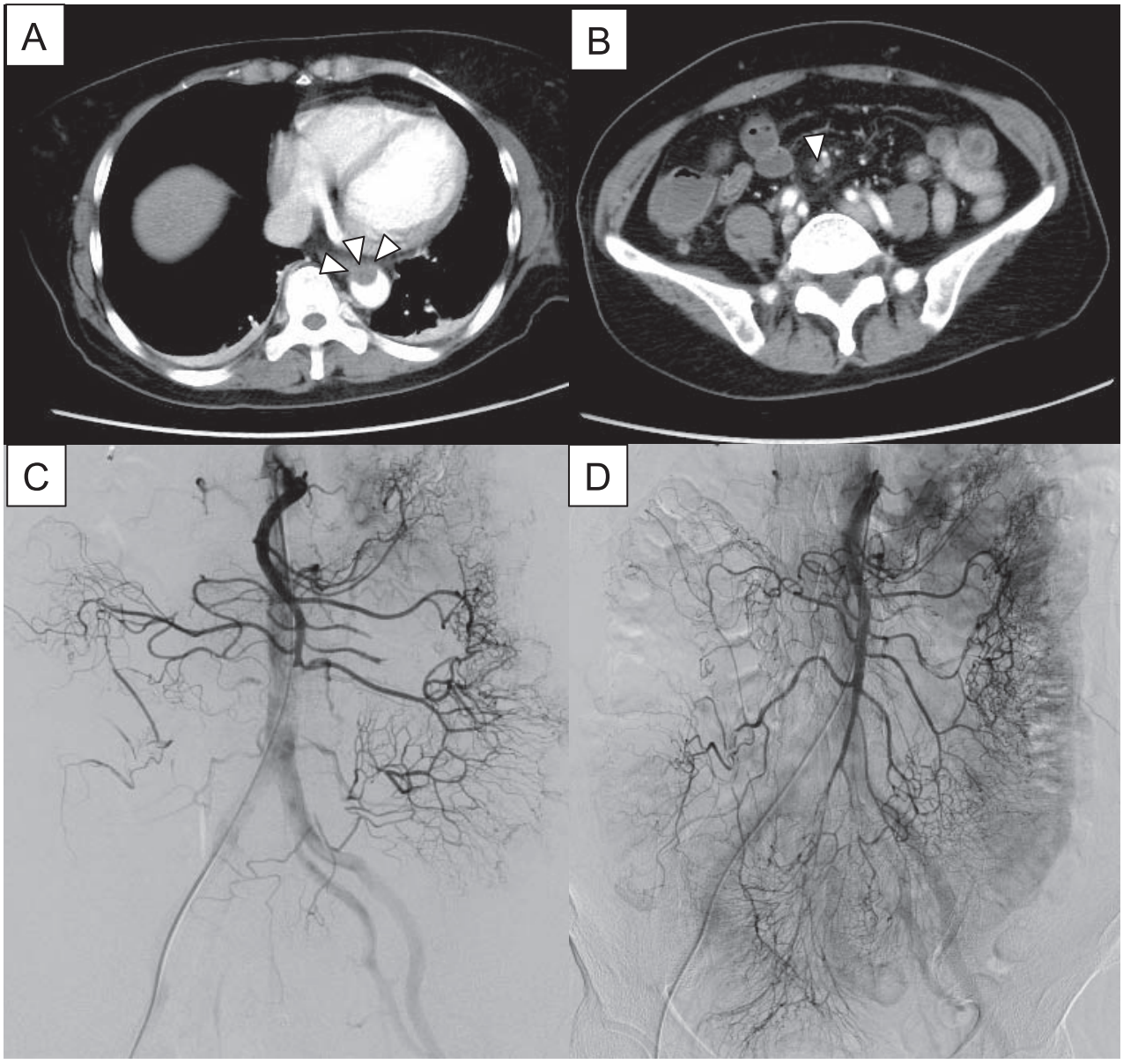

Fig. 1 Image findings in a case of acute superior mesenteric artery (SMA) occlusion resulting from thoracic aortic thrombus. CECT showing a thoracic aortic thrombus (arrowheads; A) and ischemic changes in the small intestine that resulted from the SMA occlusion (arrowhead; B). The thrombus ranged from origin of the descending aorta to 10 cm in length and the greatest dimension was 14 × 10 mm. Superior mesenteric arteriography showing occlusion of the SMA (distant to the right colic artery) and some of the jejunal arteries (C). The arteries were recanalized after infusion of urokinase (480,000 IU; D).

exploratory laparotomy and open embolectomy; however, patients without indications for emergency surgery may undergo endovascular therapies[3] that include aspiration embolectomy, pharmacologic thrombolysis, mechanical embolectomy, and SMA stenting. Recent success rate of the transcatheter aspiration with adjunctive therapy, such as thrombolysis, ranged from 73.3% to 100%.[7] Adjunctive thrombolysis could reduce remaining peripheral emboli after aspiration. Moreover, subsequent surgery in case of worsening of symptoms of acute abdomen was associated with the reduction of in-hospital mortality. This combined treatment resulted in a mortality rate of 27.0% compared with 50% with traditional operative therapy.[7,8]

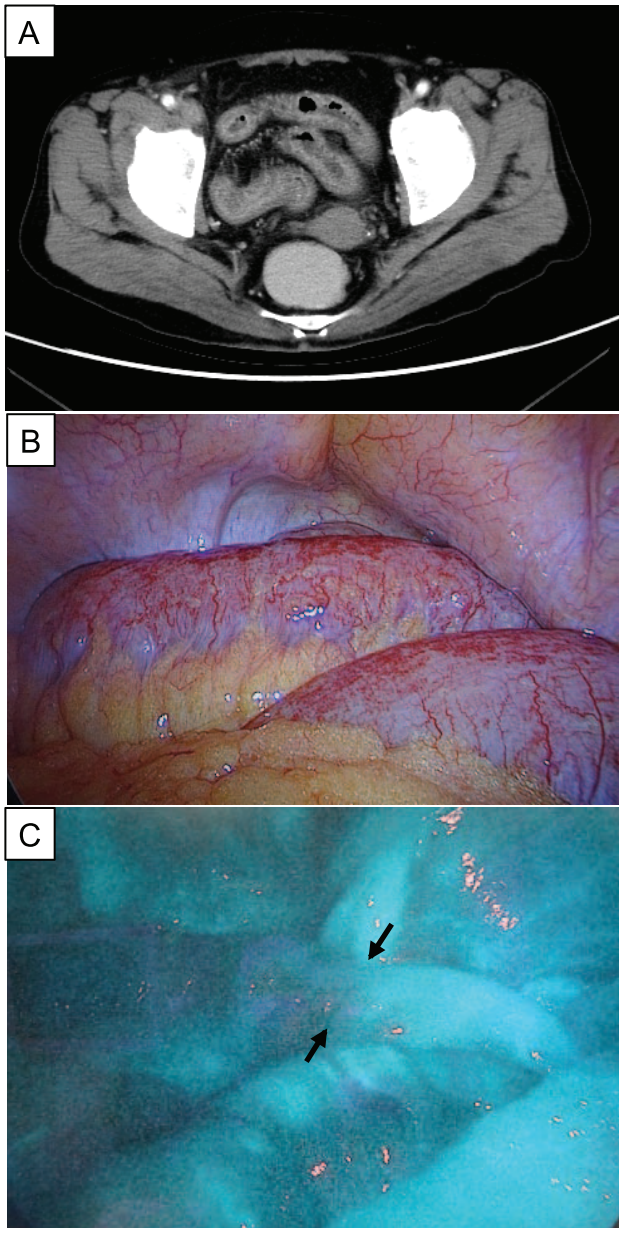

Fig. 2 CT findings on the 5th day after admission and intraoperative findings. CECT findings showing persistent massive ischemic colitis of the ileum (A). Laparoscopic findings showing ischemic changes and aperistalsis of the ileum (B). Fluorescence images with indocyanine green showing a clear demarcation line (arrow) between the ischemic and non-ischemic ileum (C).

AMT in the absence of hypercoagulability and preexisting aortic disease is clinically rare; therefore, the pathophysiologic mechanism, natural history, and prognosis remain poorly defined.[9] Recent developments in CECT, magnetic resonance imaging, and transesophageal echocardiography have increased clinicians' ability to recognize AMT. Machleder *et al* reported an incidence rate of AMT of 0.45% and an incidence of 0.028% in cases with major thromboembolic occlusion undergoing autopsy.[10] Therapeutic options for managing AMT include anticoagulant therapy, surgery, thrombolysis, and endovascular exclusion with stent grafts.[9,11] Fayed *et al* concluded in their meta-analysis that surgical management was favored in patients with a high risk of recurrence (thrombus located in the ascending aorta or arch, mild atherosclerosis of the aortic wall, and stroke presentation) because of a higher likelihood of recurrence or persistence of the thrombus (26.4% versus 5.7%) and a trend toward a higher incidence of complications (27% versus 17%) in the anticoagulation group.[11] Also, Boufi *et al* suggested the use of stent grafts because this is a less invasive procedure than surgical embolectomy.[9]

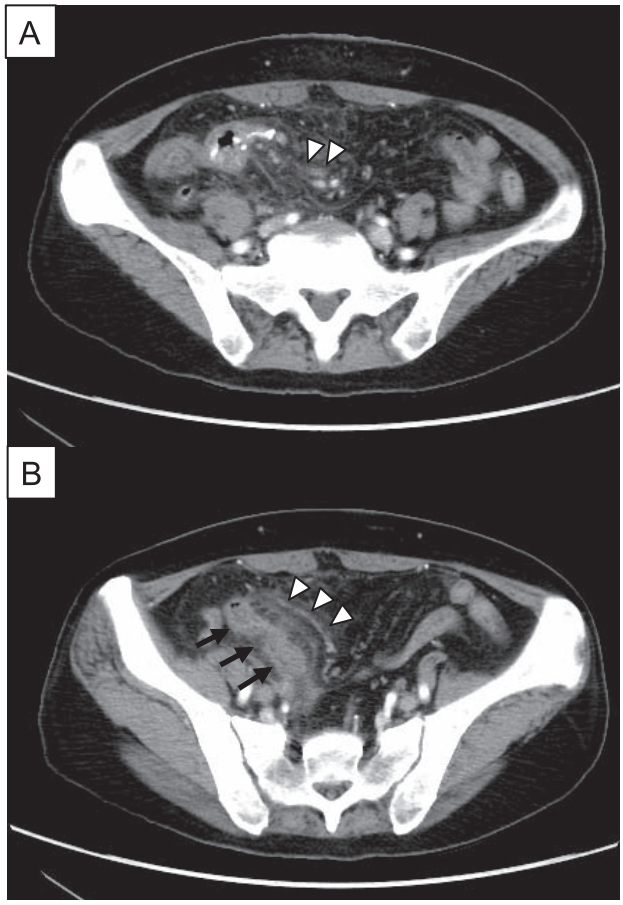

Fig. 3 CECT findings on postoperative day 5 showing a newly formed thrombus in an ileal branch (arrowheads) and localized ischemic changes in the remaining ileum (A, B).

However, anticoagulant therapy should be chosen as the primary therapy in patients without a high risk of recurrence because over 70% of all AMT patients had treatment success.

In conclusion, we report a case of acute SMA occlusion and preceding splenic infarction that resulted from thoracic AMT in a patient without hypercoagulability and preexisting aortic disease. AMT should always be considered as a cause of acute SMA occlusion in patients without heart disease, including atrial fibrillation or severe arteriosclerosis.

Acknowledgments

The authors declare that they have no conflicts of interest.

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