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A rare cause of upper gastrointestinal hemorrhage: chronic thrombosis of the splenic artery

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ABSTRACT
Upper gastrointestinal bleeding (UGIB) represents one of the most serious conditions of the gastrointestinal tract with a mortality rate of 10%. The main cause of UGIB is peptic ulcer, accounting for 28%–59% of cases. A rare cause of UGIB is submucosal arterial collaterals, which develop after splenic artery thrombosis. UGIB secondary to gastric submucosal collateral arteries should be considered in patients with endoscopic appearance of varicose veins in the absence of portal hypertension. Computed tomography angiography (CTA) is the only fast and noninvasive definitive imaging technique for such patients.
Keywords: Gastrointestinal hemorrhage, splenic artery, thrombosis, computed tomography

INTRODUCTION

Gastrointestinal system (GIS) bleeding is classified according to the localization of the bleeding site. Bleeding from proximal to the Treitz ligament is identified as upper gastrointestinal bleeding (UGIB). UGIB represents one of the most serious conditions of the GI tract, with a mortality rate of 10% (1). The main cause of UGIB is peptic ulcer, accounting for 28%–59% of cases (2).

In this study, we present computed tomography angiography (CTA) findings of a patient with UGIB that occurred because of arterial collaterals in the gastric wall, which resulted secondary to chronic thrombosis of the splenic artery.

CASE PRESENTATION

A 52-year-old man with a history of hypertension, cerebrovascular disease, and multiple sclerosis was admitted to our hospital with hematemesis and cold sweats. Hypotension and decreased hemoglobin level were detected on physical examination and blood test, respectively. After administering two units of erythrocyte suspension, hemodynamic stability was achieved, and upper GIS endoscopy was performed. Fresh blood in the antrum, pylorus, duodenal bulb, and the second portion of the duodenum was visualized on endoscopy; however, the source of bleeding could not be found in these regions. Gastric corpus could not be evaluated for the source of bleeding due to large amount of clotted blood that could not be removed after washing and aspiration. Endoscopic findings discussed in the case were suggestive of bleeding because of gastric varices. There was no history of chronic liver disease, alcohol consumption, and UGIB. Physical examination of the patient revealed no finding regarding portal hypertension or chronic liver disease. CTA examination with un-

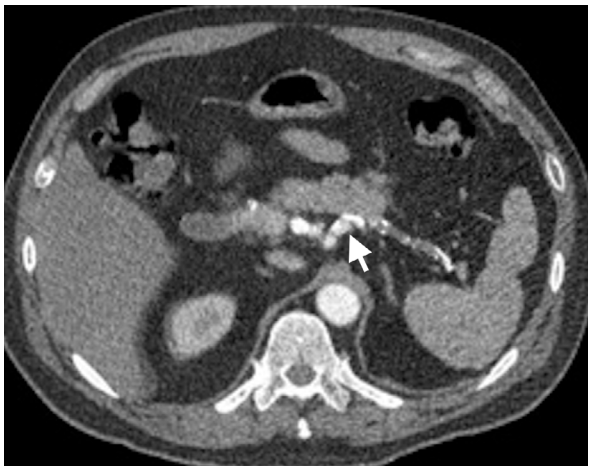

Figure 1. Splenic artery occlusion. Axial contrast-enhanced CT demonstrates chronic occlusion of the splenic artery secondary to calcified atherosclerotic plaques (arrow)

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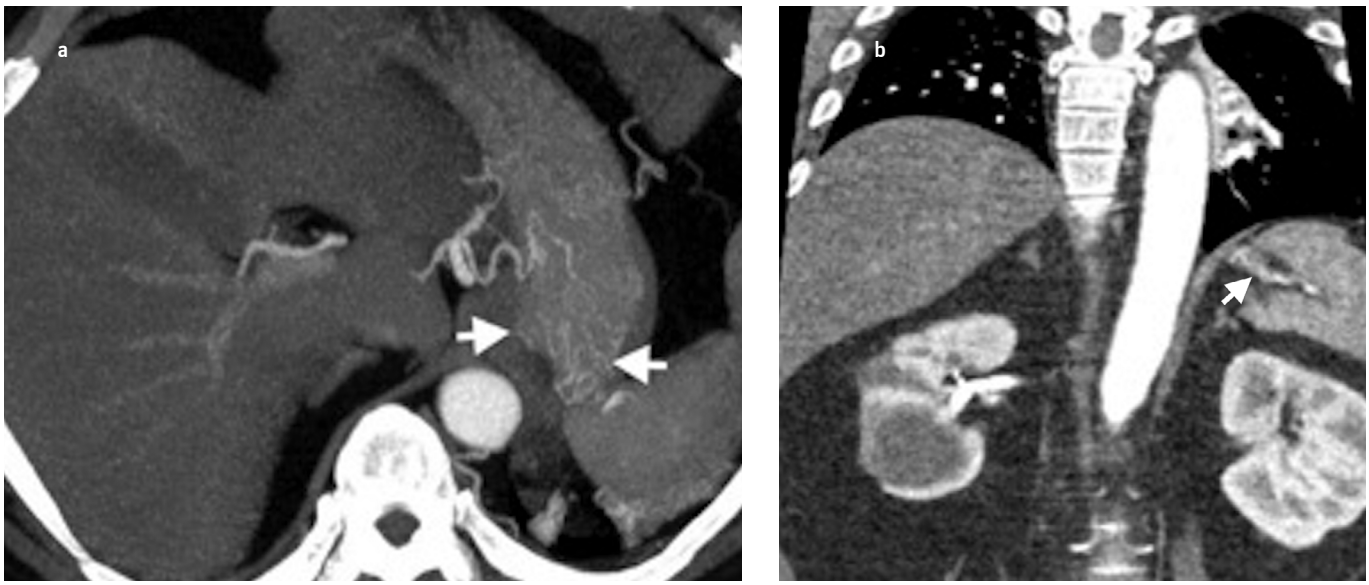

Figure 2. a, b. Collateral vessels in the gastric wall (a) Maximum intensity projection (MIP) image of CTA at arterial phase reveals tortuous vascular structures (arrows) in the gastric fundus. (b) Coronal CTA shows an extension of vascular structures in the gastric fundus to splenic hilum (arrow)

enhanced, arterial and venous phase scanning was performed to clarify the etiology of the hemorrhage. CTA revealed chronic occlusion of the splenic artery (Figure 1), chronic infarct area at the lower pole of the spleen, collateral tortuous vascular structures at the level of the gastric fundus, and collateral vessels from the left gastric artery to the spleen (Figure 2). There were chronic atherosclerotic changes in the abdominal aorta and abdominal visceral arteries. No signs of active bleeding were manifested on CTA. In the follow-up period, the patient died secondary to recurrent bleeding.

DISCUSSION

Isolated gastric varices may occur secondary to splenic vein obstruction and accompanying left-sided portal hypertension. Splenic vein obstruction can result from pancreatitis, pancreatic neoplasm, splenic arterial aneurysm, pancreas surgery, and coagulopathies (3). In our case, left-sided portal hypertension was not primarily considered as a cause of UGIB because no evidence of chronic pancreatitis, coagulopathy, or pancreatic neoplasm, as well as no history of abdominal trauma that caused splenic artery thrombosis, was observed.

Computed tomography angiography of our patient revealed splenic artery occlusion, tortuous vascular structures in the gastric fundus, and extension of these vascular structures to the splenic hilum. In our case, UGIB was considered to result from collateral arteries. In addition, in our case, significant atherosclerotic changes in major abdominal arteries and a history of cerebrovascular diseases and hypertension led us to assume that chronic atherosclerotic changes were the cause of splenic artery occlusion.

The main complication of splenic artery thrombosis is UGIB secondary to submucosal arterial collaterals that feed the spleen. In the literature, UGIB was reported in two patients with congenital absence of splenic artery and in three patients with splenic artery thrombosis (4-8). Suggested collateral pathways in cases of splenic artery thrombosis include anastomosis between the right gastroepiploic artery (from the gastroduodenal artery) and left gastroepiploic artery (from the splenic artery) on the greater curvature of the stomach and gastric branches of the left gastric artery and short gastric arteries (9).

In summary, UGIB secondary to gastric submucosal collateral arteries should be considered in patients with an endoscopic appearance of varicose veins in the absence of portal hypertension. CTA is the only fast and noninvasive definitive imaging technique for such patients.

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