Clinical Case Report

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Splenic artery pseudoaneurysm with splenic infarction induced by a benign gastric ulcer

A case report

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Abstract

Rationale: Splenic artery pseudoaneurysm induced by benign gastric ulcer is extremely rare and can lead to a fatal clinical situation. To the best of our knowledge, there are only five cases in the literature review. Therefore, it can be a diagnostic challenge for radiologists and clinicians because of rare incidence.

Patient concerns: The patient visited our hospital due to melena and dizziness.

Diagnoses: The patient showed a huge ulcer and an exposed vessel in the posterior wall of the gastric high body during endoscopy. Angiography revealed a pseudoaneurysm of the splenic artery and contrast extravasation into the gastric lumen.

Interventions: We performed coil embolization of splenic artery and gastrectomy.

Outcomes: Postprocedural course was uneventful and led to patient discharge on day 8.

Lessons: We suggest that the splenic artery pseudoaneurysm should be considered differential diagnosis in the patients with acute upper gastrointestinal bleeding, and the presence of a splenic infarction may provide a diagnostic clue.

Abbreviations: CT = computed tomography, GI = gastrointestinal, SA = splenic artery.

Keywords: embolization, endovascular treatment, pseudoaneurysm, splenic artery

1. Introduction

Acute gastrointestinal (GI) bleeding is a potentially life-threatening emergency that remains a common cause of hospitalization. In acute nonvariceal GI bleeding, peptic ulcers, erosions and esophagitis are the major causes; however, rupture of splenic artery (SA) pseudoaneurysm with visceral communication is a very rare cause. In terms of SA pseudoaneurysm, common causes are pancreatitis, trauma, iatrogenic and postoperative causes, and, rarely, peptic ulcer disease. To the best of our knowledge, there are only 5 cases of the SA pseudoaneurysms caused by gastric ulcer in the previous studies.[1–3] Herein, we present the clinical case of the gastric ulcer induced SA pseudoaneurysm which was successfully treated by coil embolization. We also provide a brief summary of this disease entity by a relevant literature review and highlight the importance of clinical suspicion in diagnosing the SA pseudoaneurysm in patients with upper GI bleeding.

2. Case report

Our institutional ethical committee approved this study and informed consent was waved. A 61-year-old male presented to our emergency room with chief complaints of melena and dizziness. He was a homeless person and an alcoholic. The patient was hemodynamically stable with anemia (hemoglobin of 2.7 g/dL). He underwent an upper GI endoscopy, which was nondiagnostic with poor visualization due to a large amount of blood in the stomach. Thus, upper GI bleeding suggested a possible cause of melena; however, the fundamental etiology was not clear. The patient subsequently underwent computed tomography (CT), which showed a hematoma-filled stomach and splenic infarction without the presence of gastric varix or pancreatitis (Fig. 1A). We failed to identify the focus of bleeding on CT, and performed diagnostic angiography. We conducted celiac and superior mesenteric angiography, and found no vascular anomalies, such as pseudoaneurysm, contrast extravasation or angiodysplasia, which were considered as definitive bleeding source at that time. Therefore, we performed "blind" embolization of the left gastric artery, which stabilized the patient and increased the hemoglobin level after transfusion. Two days later, the patient underwent endoscopy under the fasting state, which revealed a huge ulcer and an exposed vessel in the posterior wall of gastric high body during the endoscopy (Fig. 1B). Base on endoscopic findings, proton pump inhibitors applied to control

Editor: N/A.

The authors have declared that no competing interests exist.

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Medicine (2018) 97:29(e11589)

Received: 11 April 2018 / Accepted: 20 June 2018

http://dx.doi.org/10.1097/MD.0000000000011589

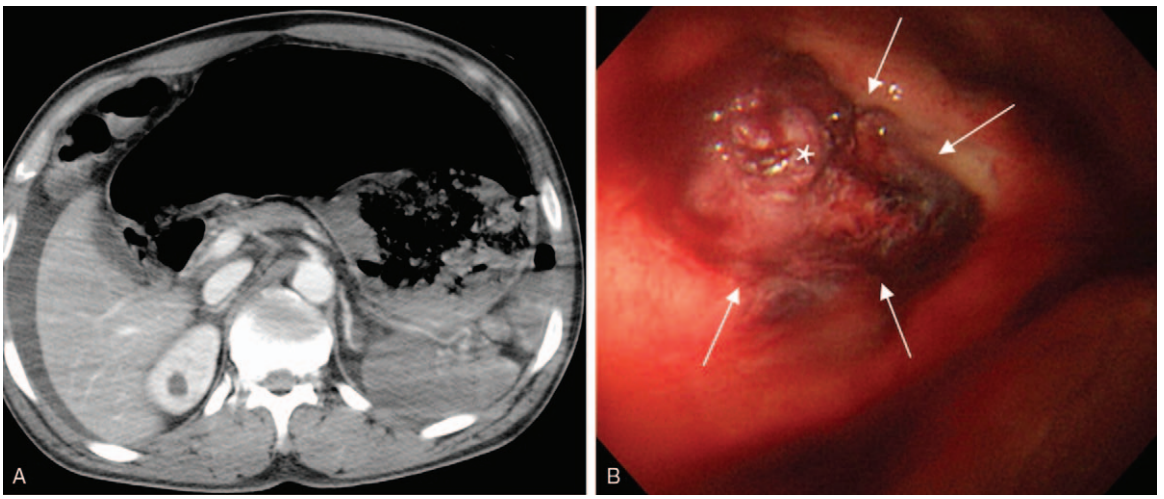

Figure 1. (A) Portal phase contrast-enhanced CT scan showing hematoma filled stomach and decreased enhancement of the spleen. (B) Upper GI endoscopy showing a huge ulcer (arrows) and an exposed vessel (asterisk) in the posterior wall of gastric high body. CT = computed tomography, GI = gastrointestinal.

the bleeding. However, on the same day evening, the patient showed changes in hemoglobin and hypotension, and underwent CT, which revealed a small pseunoaneurysm of the splenic artery (Fig. 2A). This finding did not detected in the previous CT scan. Upon review of the diagnostic angiography, we found an irregular part of the splenic artery at the same location (Fig. 2B). We performed urgent angiography, which revealed a pseudoaneurysm of the splenic artery and contrast extravasation into the gastric lumen (Fig. 2C). We performed a coil embolization of the SA to halt active bleeding using the sandwich method (upstream and downstream occlusion). A postprocedure angiography showed no further contrast extravasation (Fig. 2D). Due to the high risk of re-bleeding and gastric wall perforation, we performed a laparotomy the following day. We found a SA pseudoaneurysm densely adherent to the posterior wall of the gastric high body. Intraoperatively we observed a small defect (perforation) in the posterior wall of the stomach (Fig. 2E). We performed gastrectomy and the diagnosis confirmed as active gastritis with transmural ulceration on histopathologic examination. There was no malignant cell in the gastric tissue. Postoperative course was uneventful and led to patient discharge on day 8. We finally concluded that a benign gastric ulcer eroding into a SA might be a cause of bleeding as the patient fared satisfactorily after endovascular treatment and surgery. The accompanying splenic infarction occurred due to embolization by the splenic arterio-gastric fistula.

3. Discussion

SA aneurysms rarely occur, however, rupture of SA aneurysms can cause a fatal clinical situation. Of these, SA pseudoaneurysm is even more uncommon with a prevalence of about 5% of all visceral artery aneurysms.[1] It is well known that true aneurysms result from vessel wall abnormalities whereas pseudoaneurysms follow vascular injuries or erosions such as trauma or inflammation. The causes of SA pseudoaneurysm include pancreatitis (52%), trauma (29%) and postoperative causes (3%), and, rarely, peptic ulcer disease (2%).[2] The most common presentations of SA pseudoaneurysm are hematochezia or melena (26.2%), hemorrhage into the pancreatic duct (hemosuccus pancreaticus) (20.3%), and hematemesis (14.8%).[4]

In the present case, we failed to identify the focus of bleeding at first and performed empiric embolization of the left gastric artery. Then, the diagnosis of SA pseudoaneurysm was based on gastric ulcer on repeated endoscopy after recurrent UGI bleeding, irregularity of the SA and splenic infarction on additional CT. We performed coil embolization to control the bleeding, and the patient also underwent gastrectomy due to ulcer perforation. As this clinical case, SA pseudoaneurysms trigger a life-threatening condition resulting in rapid death with range of 10% to 40% of cases.[5,6] Therefore, it is imperative to diagnose and treat SA pseudoaneurysms for preventing fatal complications. Because a complication is not always related to the size of the pseudoaneurysm, treatment should be planned immediately upon diagnosis.[7]

Current therapeutic options of SA pseudoaneurysm have been reported as embolization (37%), splenectomy and distal pancreatectomy ((26%), splenectomy alone (11%), ligation alone (10%), endovascular stenting (4%), ligation and splenectomy (3%), distal pancreatectomy alone (3%), and gastrectomy and splenectomy (1%).[1,8] Endovascular approaches to manage the SA aneurysm offer an alternative to conventional surgery and are associated with high success rate and low procedure-related morbidity and mortality.[9] Conservative treatment leads to patient stabilization if the SA pseudoaneurysm is caused by pancreatitis or postoperative causes, successfully treated with endovascular therapy. However, the choice of treatment option can vary depending on the individual conditions such as the cause and location of the SA pseudoaneurysm, general condition of the patient, and the risk of organ ischemia following the intervention. In this case, SA pseudoaneurysm induced by gastric ulcer perforation required additional surgery to remove the bleeding cause as shown in the previous reports.[2,8] In addition, the endovascular therapy played an effective role to facilitate preoperative hemodynamic stabilization and to secure the visual field during operation.

The SA branches from the celiac artery, and passes predominantly behind the stomach, along the upper border of the pancreas; suprapancreatic course (74.1%).[10] It shows tortuous appearance through its course and the arterial tortuosity increases with the age.[11] Near the tail of the pancreas it enters the lienorenal ligament and then divides into 5 or more terminal branches that

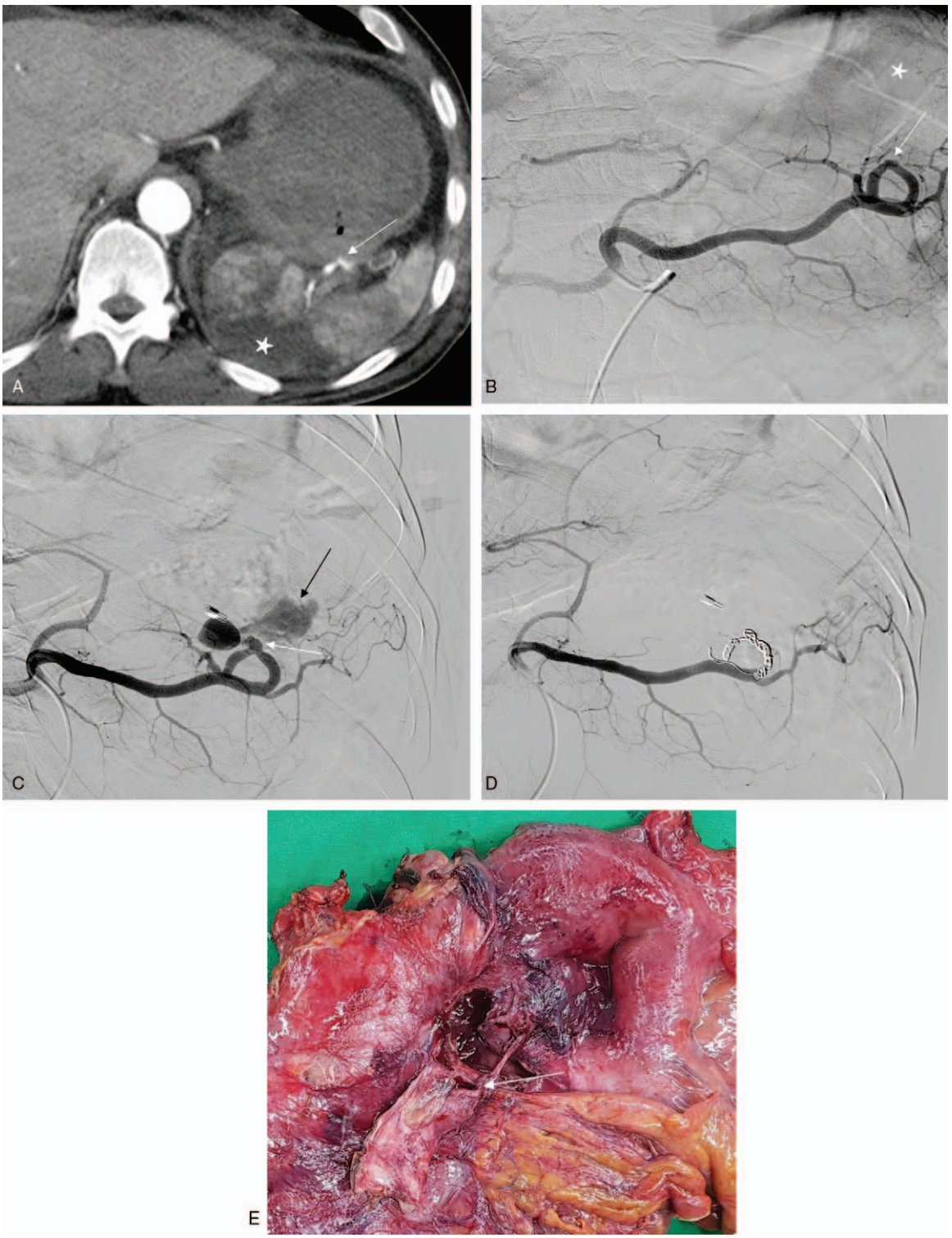

Figure 2. (A) Decreased hemoglobin level and blood pressure and repeated CT scan showing tiny SA pseudoaneurysm (arrow) and splenic infarction (asterisk). (B) Diagnostic angiography of embolization of the left gastric artery indicates irregular part of the splenic artery (arrow) (missed once) and splenic infarction (asterisk). (C) Angiographic image showing SA pseudoaneurysm (white arrow) and contrast extravasation into the gastric lumen (black arrow). (D) Final angiogram after coil occlusion of pseudoaneurysm, upstream and downstream of the lesion using sandwich method; residual segment of SA flow allowed persistent enhancement of the spleen parenchyma. (E) Pathologic specimen showing gastro-splenic artery fistula (arrow). CT = computed tomography, SA = splenic artery.

enter the hilum of the spleen.[12] In our case the SA showed the suprapancreatic course and tortuous appearance. These anatomical characteristics may be prone to close contact with SA and posterior gastric body wall at the hilum level of the spleen.

The short gastric arteries arise from the end of the SA, and occasionally cause GI bleeding due to anastomosis with branches of the left gastric and left gastroepiploic arteries. However, it is not accompanied by splenic infarction because the short gastric

artery is the terminal branch of the splenic artery. In our case, the splenic infarction was associated with splenic arteriogastric fistula in the proximal SA. A splenic infarction alone may present without other clues indicating the cause of the GI bleeding.

To the best of our knowledge, this is the only pathologically proven clinical case of the SA pseudoaneurysm induced by a benign gastric ulcer with coexisting splenic infarction. Diagnosis may be difficult because of its rarity, but the splenic infarction may provide a diagnostic clue as in our case. In daily clinical practice, the presence of SA pseudoaneurysm with GI bleeding can be helpful for prompt diagnosis and suggest the need of angiography with transarterial embolization.

In conclusion, we present the clinical case of the gastric ulcer induced SA pseudoaneurysm with successful coil embolization, presenting as acute GI bleeding. Through this report, we suggest that the SA pseudoaneurysm should be considered for a differential diagnosis in the patients with acute upper GI bleeding, and the presence of a splenic infarction on the abdominal CT scan can facilitates the diagnosis and prompt treatment.

Acknowledgments

The authors would like to thank "Harrisco Language Editing Service" for the English language review and editing; http://www.harrisco.net.

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