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Brief Report

Partial splenic infarction after endoscopic hemostasis: a case report

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Introduction

Splenic infarction is an uncommon clinical condition, most frequently associated with thromboembolic disease and hematologic disorders [1]. It has rarely been reported as a complication of endoscopic therapy. Gastric fundal varices typically develop in the setting of portal hypertension; however, they may also arise secondary to splenic venous obstruction, leading to localized or segmental portal hypertension [2].

Endoscopic sclerotherapy is an effective hemostatic treatment for gastric variceal bleeding. Nonetheless, altered postoperative venous anatomy may predispose patients to unexpected complications. To the best of our knowledge, this is the first reported case of partial splenic infarction following endoscopic sclerotherapy with lauromacrogol. In this case, the sclerosant was injected into gastric fundal varices that developed secondary to splenic vein stenosis after laparoscopic distal gastrectomy (TLDG).

Case report

A 52-year-old man was admitted with melena and hematemesis for 2 days. He had undergone TLDG for gastric cancer 1 month earlier. He had no history of liver cirrhosis, portal hypertension, hematologic disease, or coagulation disorders. Initial treatment with proton pump inhibitors and somatostatin failed to control the bleeding. Emergency esophagogastroduodenoscopy (EGD) identified an erosive lesion with active bleeding in the upper gastric body (Fig. 1A). Initial hemostasis was achieved using hemoclips, but hematemesis recurred 3 days later. Computed tomography angiography (CTA) revealed varicose vessels in the gastric fundus with an abnormal venous communication between the stomach and spleen, involving enlarged inferior lateral and superior branches of the splenic vein (Fig. 1B). Compared with images taken before TLDG, the proximal splenic vein was narrowed (Fig. 1C), suggesting impaired splenic venous outflow and the development of secondary fundal varices. Given the recurrent bleeding, repeat endoscopic treatment was performed. A 1% lauromacrogol solution was used, with a total volume of 11 mL administered intravaricealy on the posterior wall of the upper gastric body. Multiple injection sites were employed, each receiving approximately 0.5 mL of sclerosant, injected slowly at 0.5–1.0 mL/min under low pressure. No fluoroscopic or ultrasound guidance was used due to the emergency bedside setting. No compression or adjunctive hemostatic methods were applied. Hemostasis was achieved, and the patient tolerated the procedure well. Follow-up CTA on the first postoperative day demonstrated marked regression of the gastric fundal varices but revealed newly developed partial infarction of the upper pole of the spleen. The patient remained hemodynamically stable, with no evidence of splenic rupture or infection. Conservative management with close monitoring was adopted. The patient was discharged after a 24-day hospital stay. During follow-up, serial CT examinations showed a gradual reduction of the splenic infarction, with significant improvement at 2 months and complete resolution by the seventh month. No recurrent gastrointestinal bleeding occurred during follow-up (Fig. 1D).

Discussion

Left-sided portal hypertension (LSPH) is a rare cause of upper gastrointestinal hemorrhage. It results from impaired splenic venous outflow, which diverts blood through collateral pathways, such as the short gastric veins, leading to the formation of gastric fundal varices. LSPH is most commonly associated with pancreatitis, pancreatic malignancy, or splenic vein thrombosis. Patients with LSPH typically have preserved liver function, making the diagnosis more challenging and easily overlooked. In our patient, the absence of cirrhosis or portal hypertension history and the atypical endoscopic findings initially obscured this diagnosis. Based on these clinical findings, we considered a non-variceal vascular lesion and selected lauromacrogol for chemical hemostasis. This agent has demonstrated efficacy and safety in managing non-variceal

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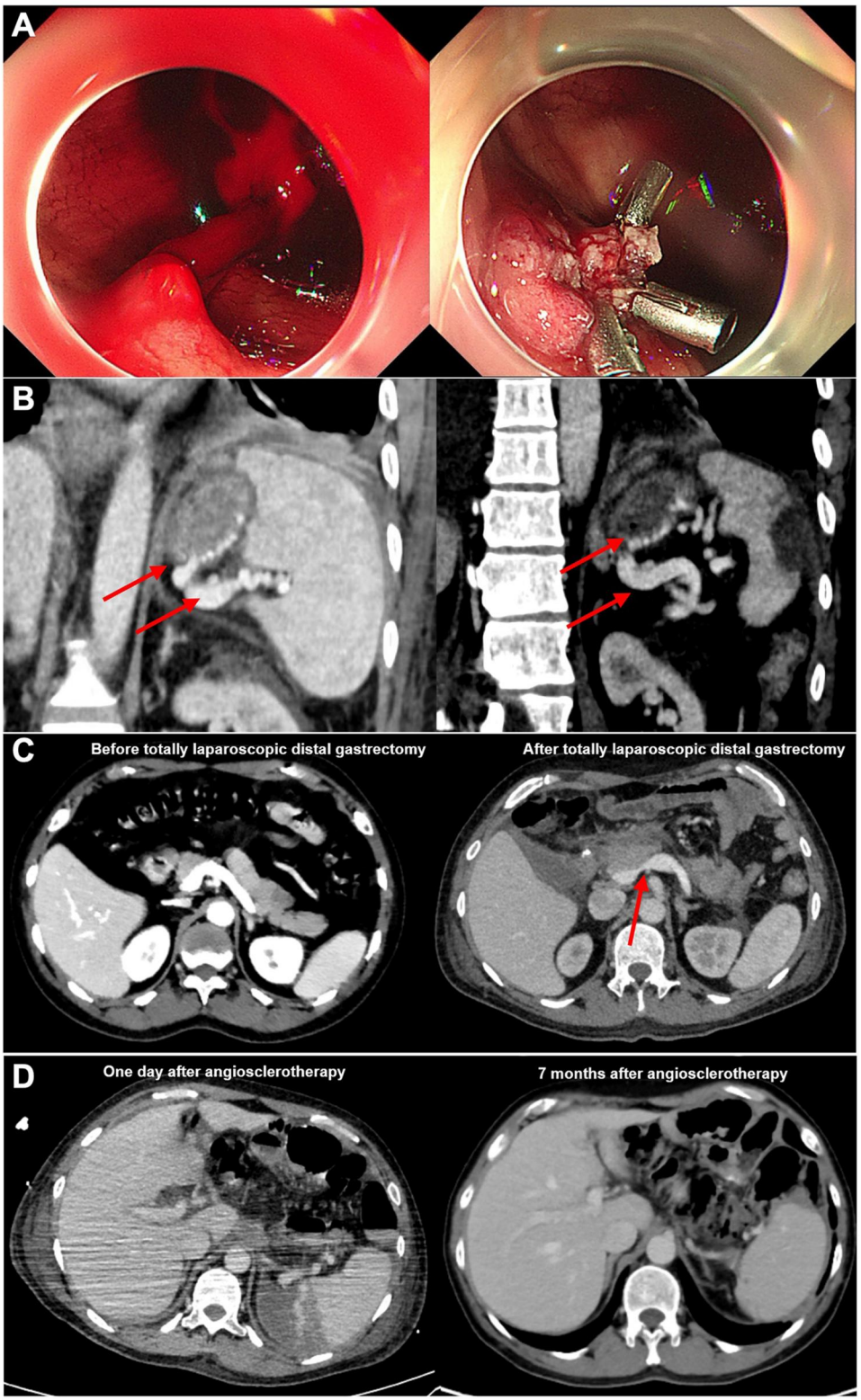

Figure 1 Endoscopic and CT findings in a post-gastrectomy patient with gastric bleeding and subsequent splenic infarction after lauromacrogol sclerotherapy. (A) Esophagogastroduodenoscopy revealed an erosive lesion with active bleeding in the upper gastric body, which was successfully treated with hemoclips. (B) CTA demonstrated an abnormal venous communication between the stomach and spleen, characterized by a gastric fundal varix connected to a beaded inferior lateral branch and the superior branch of the splenic vein (arrows show). (C) Pre-angiosclerotherapy CTA images showed the proximal splenic vein was narrowed compared with that observed in the CTA image taken before totally laparoscopic distal gastrectomy (arrows show). (D) CTA images showed splenic infarction and faded fundus varices after endoscopic injection of lauromacrogol. Subsequent CTA images revealed that the splenic infarction resolved following treatment.

vascular lesions, such as small bowel hemangiomas and radiation-induced rectal bleeding [3, 4]. However, retrospective review of contrast-enhanced CTA revealed serpiginous vessels in the gastric fundus and concurrent splenic vein stenosis, indicating that the bleeding source was actually localized gastric varices due to LSPH. Previous reviews have also identified splenic venous obstruction-related venous hypertension as a key mechanism underlying gastric fundal varices and bleeding, with imaging playing a central role in diagnosis [5].

Management of bleeding in LSPH differs from that of cirrhosis-related portal hypertension and remains controversial. Systematic reviews and meta-analyses suggest that splenectomy may be more effective than endoscopic therapy or local vascular embolization in reducing rebleeding rates, suggesting that endoscopic hemostasis alone may be insufficient in this setting [6]. Accordingly, interventional radiologic approaches, such as partial splenic artery embolization and surgical interventions, are often considered to address the underlying hemodynamic abnormality [7]. In previously published LSPH cases, treatment strategies have focused mainly on reducing splenic venous pressure, while splenic infarction as a complication and its potential mechanism have seldom been discussed [8]. In our case, endoscopic sclerotherapy with lauromacrogol achieved initial hemostasis, and although partial splenic infarction occurred subsequently, the patient recovered well during follow-up, with resolution of the infarct and no recurrence of gastric varices.

Although splenic infarction has been reported following endoscopic injection sclerotherapy, most such cases involve cyanoacrylate or ethanolamine oleate, agents known to carry a higher risk of systemic embolization [9, 10]. There is also a report of lauromacrogol-associated thrombosis during treatment of lower limb varicose veins [11], suggesting that this agent has thrombotic potential. However, to date, no previous cases of splenic infarction following lauromacrogol injection have been reported.

In the present case, splenic vein stenosis developed after distal gastrectomy. Considering the anatomical proximity of the splenic vein to the posterior pancreas, several intraoperative factors could explain this finding: (1) unintended thermal injury or mechanical traction on the splenic vein during pancreatic capsule stripping; (2) direct or indirect trauma during lymphadenectomy along the superior or posterior margin of the pancreas, where the splenic vein is exposed; (3) subsequent postoperative inflammation or fibrosis in the operative field leading to progressive stenosis.

This case illustrates a rare postoperative complication in which LSPH-associated gastric fundal varices developed secondary to splenic vein stenosis after distal gastrectomy. Endoscopic sclerotherapy successfully achieved hemostasis, but partial splenic infarction likely occurred due to retrograde migration of sclerosant through abnormal vessels. This case highlights the need for careful evaluation of venous anatomy in patients with prior upper gastrointestinal surgery and raises awareness of potential complications, even with sclerosants considered to have a low risk of embolization.

Authors' contributions

Q.L., X.X., and J.L. designed the study and drafted the manuscript, and edited the images. X.X. critically revised the manuscript for important intellectual content and obtained funding.

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Conflicts of interest

The authors have declared no competing interests.

Data availability

No datasets were generated or analyzed in this study; therefore, data sharing is not applicable. Written informed consent was obtained from the patient.

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