Percutaneous Transsplenic Embolization of Gastric Varices in Left-sided Portal Hypertension

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─── **Abstract** ───

An 81-year-old man with previously diagnosed cancer of the pancreatic body presented with melena and anemia. Upper gastrointestinal endoscopy showed gastric varices with bleeding in the entire stomach. Contrast-enhanced computed tomography identified a splenic vein occlusion resulting from invasion by the pancreatic body cancer and dilated collateral pathways from the splenic hilum to the gastric fundus. The patient was diagnosed with gastric varices associated with left-sided portal hypertension caused by obstruction of the splenic vein and underwent percutaneous transsplenic embolization with *n*-butyl-2-cyanoacrylate mixed with lipiodol. Splenic subcapsular hematoma occurred and was treated conservatively. The patient died of advanced cancer 5 months after the procedure, without experiencing rebleeding. Percutaneous transsplenic embolization was effective in treating gastric variceal bleeding caused by left-sided portal hypertension.

Key words: Left-sided portal hypertension, Gastric varix, Percutaneous transsplenic embolization

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Introduction

Upper gastrointestinal bleeding occurs in 45-72% of patients with left-sided portal hypertension (LSPH) resulting from isolated obstruction of the splenic vein [1]. One of the causes of such bleeding is gastric varices, which develop as a consequence of dilation of gastric wall veins serving as collateral pathways for the occluded splenic vein [1]. When gastric variceal bleeding occurs, endoscopic sclerotherapy is usually selected to achieve hemostasis. In addition, splenectomy or partial splenic embolization (PSE) may be performed to reduce blood flow from the splenic vein to gastric varices [1-4]. Percutaneous transsplenic embolization is another method for decreasing splenic venous outflow to the varices. Few case reports of this technique have been published to date [5, 6].

Here, we present a case of LSPH-induced gastric variceal bleeding that was successfully treated with percutaneous transsplenic embolization.

Case Report

This retrospective case report was approved by our institutional review board, and the requirement to obtain informed consent from the patient for inclusion in this report was waived, although informed consent for all examinations and procedures was obtained from the patient in advance. The patient was an 81-year-old man with cancer of the pancreatic body who had undergone distal gastrectomy with Billroth-I reconstruction for gastric ulcer at age 64 years. He was undergoing chemotherapy for multiple liver metastases from the cancer. He complained of melena and had a low hemoglobin level of 5.9 g/dL. Upper gastrointestinal endoscopy showed enlarged nodular gastric varices in the submucosal layer throughout the stomach (**Fig. 1a**). Bleeding from these varices was also revealed. Contrast-enhanced computed tomography (CT) identified a splenic vein occlusion
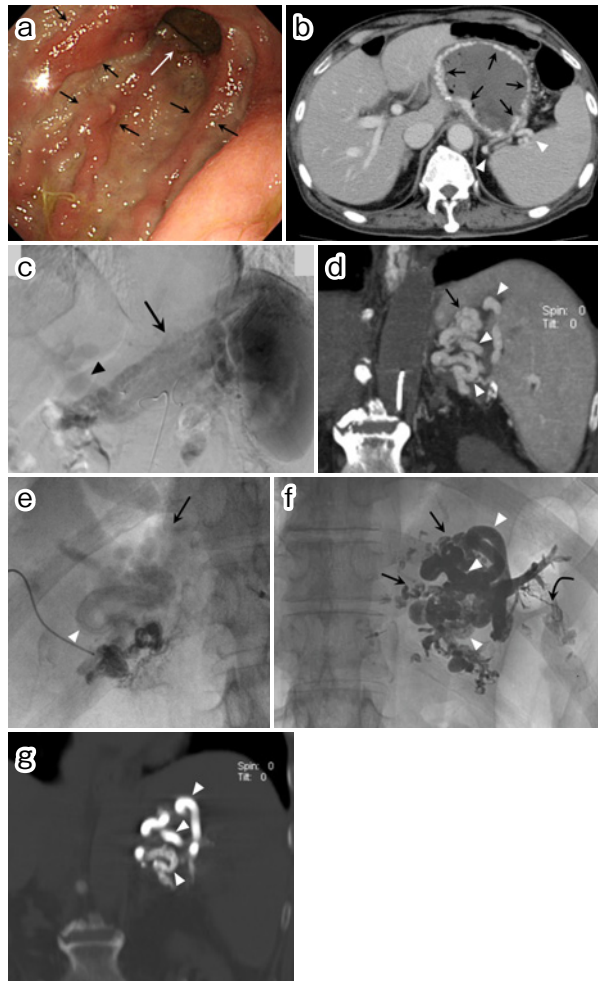
Figure 1. The patient was an 81-year-old man with cancer of the pancreatic body who had a history of distal gastrectomy. The splenic vein was obstructed as a result of cancer invasion. Gastric variceal bleeding caused by left-sided portal hypertension was diagnosed.

a) Upper gastrointestinal endoscopy before embolization. Enlarged varices can be seen in the submucosal layer of the stomach (black arrows). The white arrow indicates the anastomotic site of distal gastrectomy.

b) Equilibrium phase of contrast-enhanced computed tomography (CT). A dilated tortuous blood vessel is seen in the region extending from the splenic hilum to the gastric fundus (white arrowheads). Dilated blood vessels are also observed throughout the inside of the gastric wall (black arrows).

c) Venous-phase of splenic arteriography. No blood flow draining into the portal vein via the splenic vein can be detected. Contrast agent flows from the splenic hilum to varices (black arrow) in the stomach wall through the short gastric veins. Subsequently, the agent flows out of the varix and into the left gastric vein before draining into the portal vein (black arrowhead).

d) Coronal image of late-phase CT during splenic arteriography. The contrast agent flows from the splenic hilum to the gastric varices (black arrow) via the three short gastric veins (white arrowheads).

e) Splenic venogram using the micro-catheter with a percutaneous transsplenic approach, with the patient in the prone position. The short gastric vein (white arrowhead) and connected gastric varices (black arrow) are visualized.

f) Roentgenogram after embolization. The patient is placed in the supine position. Embolization is performed by injecting a liquid embolic material comprising *n*-butyl-2-cyanoacrylate and lipiodol mixed at a ratio of 1:5. Embolic material was delivered to the three short gastric veins (white arrowheads) and gastric varices (black arrows). The puncture route is filled with embolic material (black curved arrow).

g) Coronal non-contrast-enhanced CT after embolization. The image shows embolic material in three short gastric veins (white arrowheads).

resulting from invasion by the pancreatic body cancer. The examination also detected three dilated, tortuous short gastric veins in the area ranging from the splenic hilum to the gastric fundus. These veins extended into the gastric wall and functioned as collateral pathways for the occluded splenic vein (**Fig. 1b**).

To control bleeding from gastric varices, endoscopic therapies were carried out using argon plasma coagulation, clipping, sclerotherapy, and injection of hypertonic saline-epinephrine solution. However, the patient frequently experienced episodes of bleeding from gastric varices and ablation-induced ulcers. He received repeated blood transfusions and his hemoglobin levels fluctuated between 5.9 and 10.5 g/dL.

Angiography to assess hemodynamics was performed next. The superior mesenteric and portal veins appeared normal on superior mesenteric arterial portography. Conversely, venous-phase splenic arteriography revealed complete obstruction of the splenic vein caused by cancer invasion. The contrast agent that had reached the splenic vein at the splenic hilum flowed into venous vessels in the stomach wall through the short gastric veins. These vessels in the stomach wall were dilated, forming gastric varices. The contrast agent in these varices then flowed out to the left gastric vein and ultimately drained into the portal vein (**Fig. 1c**). Similarly, CT during splenic arteriography demonstrated that the splenic vein at the hilum of the spleen was connected to varices in the stomach wall via the three short gastric veins (**Fig. 1d**), and subsequently linked to the portal vein through the left gastric vein. Based on these observations, the patient was diagnosed with gastric varices associated with LSPH caused by the obstruction of the splenic vein.

Splenic angiography showed most splenic venous blood flowing into the portal vein through the short gastric veins. The esophageal vein and left gastroepiploic vein were also depicted but were thin and the contrast effects were poor. We, therefore, judged that percutaneous transsplenic embolization for the short gastric veins was possible and nontarget embolization could be avoided. The procedures were performed using an interventional radiology-CT angiography system (AXIOM Artis dTA; Siemens Medical Solutions, Erlangen, Germany). This procedure was performed with the patient under sedation using dexmedetomidine hydrochloride and local anesthesia using lidocaine. With the patient in the prone position, the splenic vein in the parenchyma of the spleen was percutaneously punctured under ultrasound guidance with a 22-gage, 20-cm Chiba needle (Chiba biopsy needle; Angiotech, Gainesville, FL, USA). A 0.014-inch micro-guidewire (Chevalier™ 14 Universal; FMD Co., Saitama, Japan) was inserted through the needle and advanced toward the splenic vein at the hilum. A 1.8-Fr micro-catheter (Prominent RAPTOR; Tokai Medical Products, Aichi, Japan) was then introduced into the splenic vein over the micro-guidewire. Angiography by micro-catheter visualized the three short gastric veins and connected gastric varices (**Fig. 1e**). Images of the short gastric veins were consistent with those veins on contrast-enhanced CT, venous-phase splenic arteriography, and CT during splenic arteriography before the procedure. Following angiography, embolization of gastric varices was performed using *n*-butyl-2-cyanoacrylate (NBCA) as the embolic material. NBCA was mixed with lipiodol in a ratio of 1:5. This ratio was determined based on angiographic images from the patient. The resulting NBCA mixture was expected to reach the gastric varices, but to polymerize before leaking into the portal vein. The end point of infusing NBCA was determined as the point at which NBCA filled the short gastric veins, as the inflow tracts for the gastric varices, and the splenic vein at the splenic hilum. However, if NBCA reached the left gastric vein before the end point, the infusion was to be terminated. The mixture was injected through the micro-catheter. The splenic vein at the splenic hilum and all three short gastric veins were embolized (**Fig. 1f**). Some embolic material reached the stomach wall, but did not flow into the portal vein. Finally, the puncture route was closed by infusing the same NBCA mixture as the micro-catheter was withdrawn. Overall, 11.6 mL of the NBCA mixture was administered. Post-embolization CT confirmed retention of the mixture in both the varices and the puncture route (**Fig. 1g**). Splenic subcapsular hematoma, classified as Grade 1 according to Common Terminology Criteria for Adverse Events version 5.0, was detected in the area surrounding the spleen, but did not expand over time. No other complications were encountered.

Two days after embolization, contrast-enhanced CT identified embolic material in the splenic vein at the splenic hilum, short gastric veins, and gastric varices. No dilation of veins in the stomach wall was observed. Similarly, upper gastrointestinal endoscopy performed 3 days after embolization detected no dilation of veins in the submucosal layer of the stomach. The patient died of pancreatic body cancer 5 months after the procedure. However, until his death, he required no blood transfusions and experienced no new episodes of hematemesis or melena.

Discussion

Endoscopic sclerotherapy is the first choice in the prophylactic treatment of gastric varices associated with conventional portal hypertension [7]. In one published case, that therapy was also administered to a patient with gastric varices caused by LSPH [8]. However, the authors reported difficulties in controlling hemorrhage. Anatomically, LSPH-induced varices spread throughout the fundus of the stomach, so endoscopic sclerotherapy is unlikely to succeed. Transjugular intrahepatic portosystemic shunt and balloon-occluded retrograde transvenous obliteration are not indicated for patients with gastric varices caused by LSPH, such as our patient, because of the hemodynamic characteristics in patients with LSPH. Consequently, PSE and splenectomy have been used to reduce blood flow from the splenic vein to these varices [1]. Post-embolization syndrome becomes

severe when a large segment of the spleen is embolized in a single procedure. Certain patients may therefore require multiple embolization procedures targeting smaller areas. As our patient was undergoing chemotherapy for liver metastases from cancer of the pancreatic body, treatment within a short period of time would have been appropriate to avoid severe post-embolization syndrome. For similar reasons, the invasive surgical procedure of splenectomy was not chosen as a treatment option. In addition, in PSE for upper gastrointestinal bleeding complicated by LSPH, Wei et al. reported rebleeding in 11 of 23 patients (47.8%), and two case reports showed rebleeding [2-4].

Various studies have previously reviewed the safety of the percutaneous transsplenic approach. They reported success rates for this approach ranging from 91.6% to 96% [9, 10]. Based on these results, we decided to apply the percutaneous transsplenic approach to the embolization of gastric varices associated with LSPH caused by a malignant tumor. To date, few case reports have described the use of percutaneous transsplenic embolization to treat LSPH-induced gastric variceal bleeding [5, 6]. To manage the bleeding, they performed variceal embolization using NBCA mixed with lipiodol with a percutaneous transsplenic approach. After these procedures, patients experienced no further bleeding episodes during follow-up.

Bleeding complications under a percutaneous transsplenic approach are reportedly seen in 12.5-19.6% of patients, left upper abdominal pain in 6.5%, and left pleural effusion in 6.5% [9, 10]. To avoid bleeding complications, the decision was made to perform a percutaneous transsplenic approach in our patient using only a micro-catheter system without a sheath introducer, despite the risk of micro-catheter displacement due to respiratory motions. One of the reasons was that the patient already had anemia due to gastric bleeding from advanced cancer. As no sheath introducer was inserted, angiography using a micro-catheter was insufficient before embolization and impossible after injecting NBCA. Detailed diagnosis was, therefore, obtained from splenic arteriography and CT during splenic arteriography before embolization. The angiography showed that venous blood from the spleen flowed from the splenic vein at the hilum of the spleen into the stomach via the short gastric veins (**Fig. 1c**). This was the decisive factor for micro-catheter embolization with the end point that the short gastric veins and splenic vein at the hilum of the spleen were filled with NBCA.

In this case, ethanolamine oleate, ethanol, and gelatin sponge were not selected as embolic materials because multiple short gastric veins were acting as inflow pathways for gastric varices, and flow control was not possible. No recurrence of variceal bleeding was observed until the patient died of pancreatic body cancer 5 months after the procedure.

In our case, percutaneous transsplenic embolization was effective in treating gastric variceal bleeding caused by LSPH.

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