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【 CASE REPORT 】

Pipeline Esophagogastric Varices Secondary to Extrahepatic Portal Vein Obstruction Treated Endoscopically with the Assistance of Transileocolic Obliteration

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Abstract:

Endoscopic injection sclerotherapy (EIS) for esophagogastric varices (EGV) was attempted for a 29-year-old man with extrahepatic portal vein obstruction. However, pipeline varices characterized by a large blood flow volume were present, and the sclerosant did not accumulate sufficiently in them. Transileocolic obliteration (TIO) using coils was performed, but some EGVs and palisading veins remained. Thus, EIS was performed once again immediately after TIO. Since a reduction in the intravariceal blood flow was achieved by preceding TIO, effective injection of sclerosant into the vessels was possible. For pipeline varices difficult to treat endoscopically, combination therapy with TIO may be effective.

Key words: portal hypertension, interventional radiology, endoscopic injection sclerotherapy, endoscopic variceal ligation, endoscopic band ligation, pediatric

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Introduction

Extrahepatic portal vein obstruction (EHPVO) is a major cause of noncirrhotic portal hypertension, resulting in esophagogastric varices (EGVs). Most patients with EHPVO develop EGVs, and those due to EHPVO have a higher frequency of larger esophageal varices than those due to liver cirrhosis (1). While endoscopic procedures, including endoscopic injection sclerotherapy (EIS), are useful in the treatment of EGVs, they are often not sufficient to eradicate pipeline varices, i.e. those with a large blood flow that run straight up from the dilated gastric coronary vein to the esophageal wall (2).

Transileocolic obliteration (TIO) is a procedure with an antegrade catheter insertion to occlude the portosystemic shunt, thereby reducing the blood supply to the EGVs and achieving hemostasis. It is indicated in cases where EGVs are difficult to manage endoscopically (3). However, treatment with TIO alone carries a high risk of rebleeding, and the success rate of hemostasis can be increased when combined with endoscopic treatment (4).

We herein report a patient with EHPVO with pipeline EGVs in whom EIS was attempted, but the sclerosing agent leaked from the puncture site and did not accumulate in the vessel, making it difficult to complete the treatment by endoscopy alone. The EGVs were successfully treated with TIO by reducing the blood supply to the EGVs followed by subsequent endoscopy.

Case Report

A 29-year-old man was brought to our emergency room because of black stool. The patient had been diagnosed with EHPVO at three years old and had been attending the pediatric surgery outpatient clinic since then. He had been admitted to another hospital due to hematemesis and underwent EIS at four years old (details unknown). He underwent

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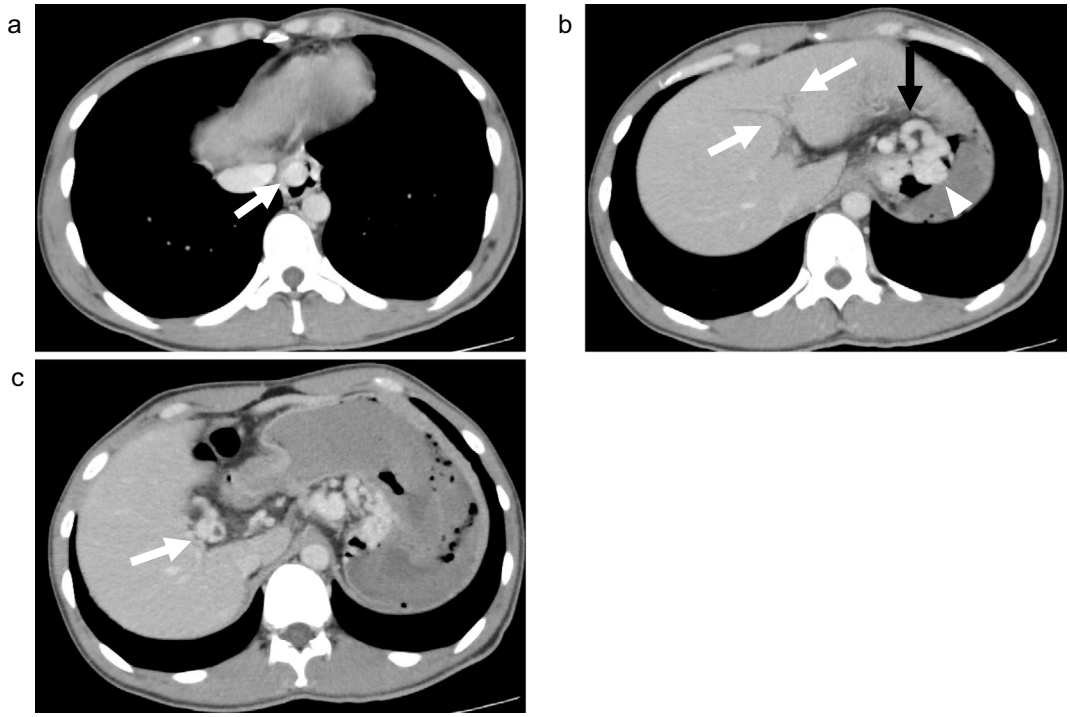

Figure 1. Images of abdominal contrast-enhanced computed tomography at the time of the patient's visit to our emergency room (day 1). (a) Development of esophageal varices (white arrow). (b) Occluded intra-hepatic portal veins (white arrows) and dilatated gastric coronary veins (black arrow) with gastric varices (arrowhead). (c) Developed peribiliary plexus as hepatopetal vessels (white arrow).

proximal splenic-renal venous shunting and splenectomy for refractory EGVs at 11 years old, but spontaneous occlusion of the shunt was confirmed by regular follow-up computed tomography (CT) a year later. He had been hospitalized for gastrointestinal bleeding, suspected to be variceal bleeding at 12, 13, and 21 years old, all receiving conservative treatment all 3 times. Although inferior mesenteric vein-left renal vein shunting had been proposed, he and his family did not wish him to undergo surgery. He had dropped out of regular follow-up 6 years ago (23 years old) for personal reasons. Since then, he had not been taking any regular medications.

At the time of arrival (day 1), he was conscious, with blood pressure of 107/97 mmHg and heart rate of 125 bpm. The eyelid conjunctiva showed no evidence of anemia or jaundice. Blood tests showed total protein of 5.8 g/dL and albumin of 3.2 g/dL, indicating mild hypoproteinemia. The urea nitrogen level was elevated at 35.1 mg/dL, but the creatinine level was normal at 0.81 mg/dL. Total bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, and gamma-glutamyl transpeptidase were 0.7 mg/dL, 17 U/L, 12 U/L, 119 U/L, and 9 U/L, respectively. Mild anemia with hemoglobin of 10.0 g/dL was observed. The platelet count was $176\times10^3/\mu L$. The prothrombin time-international normalized ratio was 1.14. Contrast-enhanced CT showed dilated collateral vessels and large EGVs, while the intrahepatic portal branch and main portal vein were highly stenotic. The peribiliary plexus had developed as hepatopetal collaterals (Fig. 1).

Emergency esophagogastroduodenoscopy (EGD) was performed on the same day, and large esophageal varices [Ls F3 Cb RC3 (RWM); Fig. 2a] continuous with gastric varices (Lg-c F3 Cw RC0) were present. Erosion with a red plug was found on the surface of the cardiac varices (Fig. 2b). Based on the CT and EGD findings, the patient was determined to have large esophageal varices with direct blood inflow from the gastric coronary veins via cardiac varices, which were considered pipeline varices. Although there was no active bleeding, there was a large amount of fresh blood in the stomach. It was determined that the bleeding had occurred from cardiac varices, and EIS was subsequently performed with 67% n-butyl-2-cyanoacrylate (NBCA), mixed with ethyl ester of iodinated poppy-seed oil fatty acid. Using a 23-gauge Varixer needle (Top, Tokyo, Japan), 2.5 mL of the sclerosant was injected near the erosion. The procedure was completed after confirming the accumulation of sclerosant in the cardiac varices on abdominal X-ray (Fig. 2c) and no bleeding thereafter.

Follow-up EGD was performed on day 3, showing ulceration of the treatment site but no active bleeding. It was decided to perform intra-variceal EIS for the remaining EGVs. Ethanolamine oleate (EO) mixed with water-soluble contrast agent and adjusted to 5% was used as the sclerosant (EO-EIS). Puncture using a 25-gauge Varixer needle (Top) induced a large amount of backflow of blood from the side of the puncture hole, resulting in poor visibility with the scope. Furthermore, the sclerosing agent was washed out from the

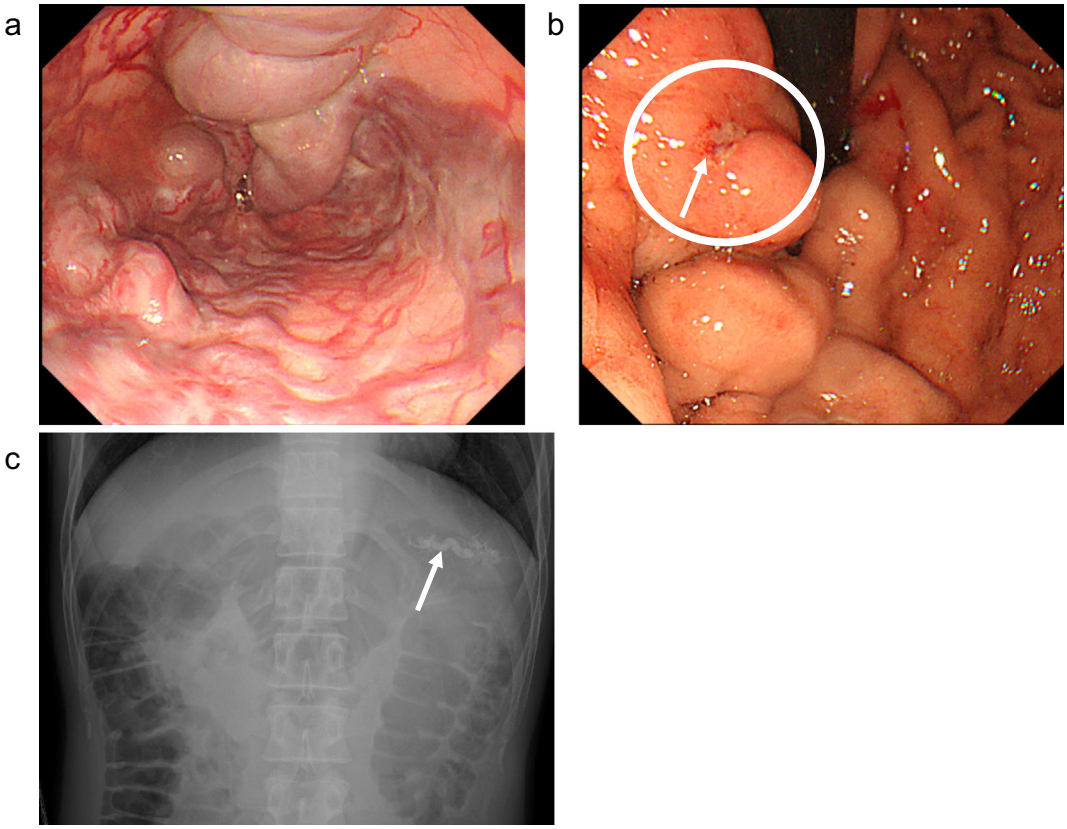

Figure 2. Images related to endoscopic injection sclerotherapy (EIS) using 67% n-butyl-2-cyanoacrylate (NBCA) as a sclerosant, for cardiac varices suspected to have bled (day 1). (a) Endoscopic image shows the presence of huge esophageal varices with red signs (Ls F3 Cb RC3 (RWM). (b) Endoscopic image shows cardiac varices (Lg-c F3 Cw RC0) with erosion (circle) with a red plug (white arrow) on the surface. (c) X-ray image shows the accumulation of sclerosants in cardiac varices post-procedure (white arrow).

side of the puncture hole and did not accumulate in the vessel. Since complete hemostasis was not achieved by subsequent balloon compression of the puncture site, endoscopic variceal ligation (EVL) was performed at the same place using a Pneumo-Activated Device (Sumitomo Bakelite, Tokyo, Japan). However, due to the large diameter of the esophageal varices, it was not possible to sufficiently suck the varix into the EVL device, and the ligation by the band was shallow, resulting in insufficient ligation. There was concern about massive bleeding due to early spontaneous detachment of the band. Since dynamic CT showed that the superior mesenteric vein supplied blood flow to the EGVs via the gastric coronary vein (Fig. 3), urgent TIO was considered to reduce the blood supply. There were no drainage vessels to consider the indication of balloon-occluded retrograde transvenous obliteration (B-RTO).

Under general anesthesia, a midline incision was made in the lower abdomen. After direct puncture of the ileocolic vein (Fig. 4a), a 5-French Destination Guiding Sheath (Terumo, Tokyo, Japan) was inserted from the ileocolic vein. A 4-French non-tapered GlideCath II catheter (Terumo) was advanced peripherally, and a 0.027-inch high-flow Masters catheter (Asahi Intecc, Seto, Japan) was inserted just proximal to the gastric varices via the dilated gastric coronary veins. When contrast medium was injected, blood flow from the collaterals toward the esophagus was observed (Fig. 4b). After advancing the catheter as far as possible into the peripheral gastric coronary vein, the vessel was embolized with Target XXL coils (Stryker, Kalamazoo, USA). Since these coils have been widely accepted for embolization of peripheral arteries and veins in interventional radiological procedures, we did not need to obtain specific approval. Multiple coils were loaded into the gastric coronary vein while the catheter was gradually withdrawn. After coil embolization, some of the dilated gastric coronary veins were occluded, but collaterals, including small ones, remained, and hepatofugal blood flow toward the esophagus was confirmed (Fig. 4c). Thus, it was decided to perform subsequent endoscopic treatment. EGD post-TIO showed reduced tenseness of the esophageal varices. A total of 15 mL of 5% EO was successfully injected intravascularly via two puncture sites into the large esophageal varices directly contiguous with the cardiac varices along the gastric lesser curvature. Finally, we injected the sclerosing agent into the remaining gastric coronary vein (Fig. 4d). A schematic illustration of the anatomy associated with the procedure is shown in Fig. 5.

Since EO was not injected into the esophageal varices

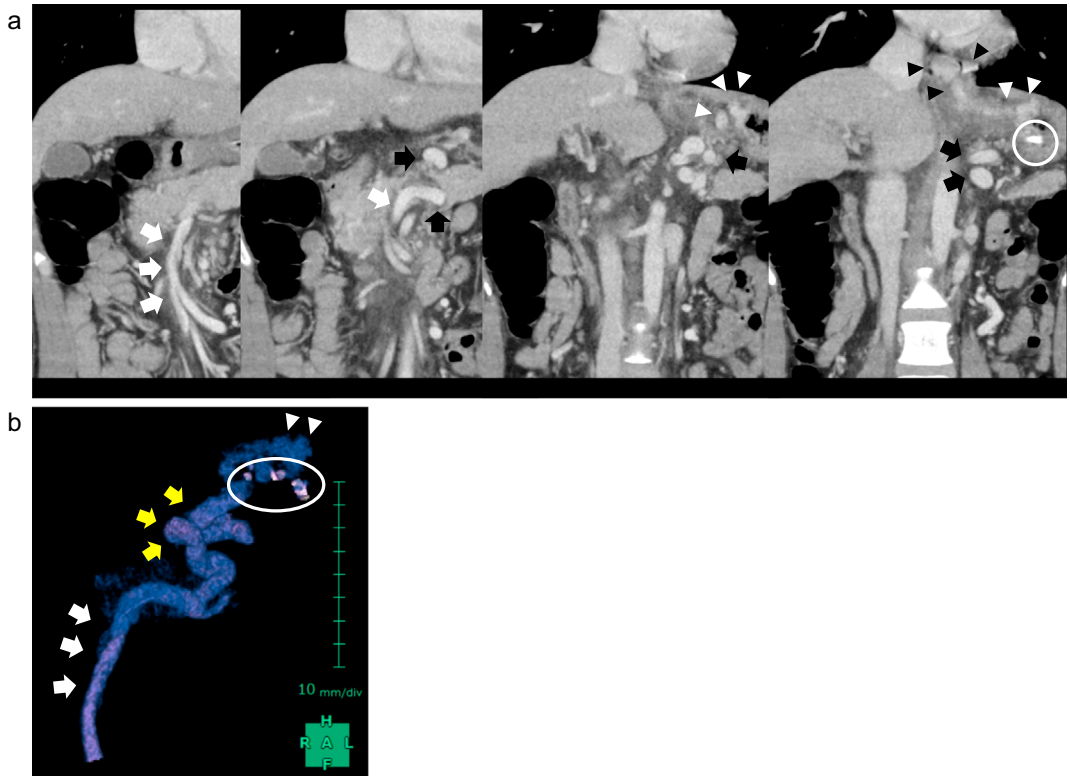

Figure 3. **Images of abdominal dynamic contrast-enhanced computed tomography (CT) after initial endoscopic injection sclerotherapy (EIS) using 67% n-butyl-2-cyanoacrylate (NBCA) and just before transileocolic obliteration accompanied with EIS using 5% ethanolamine oleate. (a) Coronary plane. The superior mesenteric vein ascended cranially (white arrows) and was continuous with the gastric coronary veins (black arrows). There was outflow from the gastric coronary vein to form cardiac varices (white arrowheads). NBCA was partially accumulated in cardiac varices (circle), but the remaining vessels were directly connected to large esophageal varices (black arrowheads). (b) Three-dimensional CT image showing cardiac varices (white arrowheads) formed from the superior mesenteric vein (white arrows) via the gastric coronary veins (yellow arrows). The oval shows the partial accumulation of NBCA in the cardiac varices.**

leading to the anterior wall and the greater curvature of the stomach, despite these remaining vessels not being large enough for ensured puncture, six shots of EVL were performed in the lower esophagus for these varices.

After treatment of the varices, weight gain (2.2 kg) due to ascites was observed, so oral diuretics (furosemide 20 mg/day plus spironolactone 25 mg/day) were started (day 7), after which the weight gradually decreased to the baseline level. EGD on the 9th day showed that esophageal varices had become bronze-colored due to intravascular injection of EO, and the size had decreased to F2. Red signs had improved to RC1, and there were multiple post-EVL ulcers. Since there were only slight red signs, and the patient desired early discharge, additional treatment was not carried out, and he was put on careful follow-up. The patient was discharged from the hospital on the 12th day with an overall good course.

Dynamic CT on the 19th day showed that the gastric coronary veins, lower esophageal varices, and cardiac varices were thrombosed and reduced in size. There was no significant change in the peribiliary venous plexus. Continuous thrombosis was found from the superior mesenteric vein to its branches, where catheter insertion was implemented. There was no evidence of intestinal ischemia, and no ascites remained.

On the 187th day, the patient had esophageal varices of Ls F3 Cb RC3 and gastric varices of Lg-c F2 Cw RC0, and it was decided to perform additional intravascular EO-EIS. A total of 11.3 mL of 5% EO was injected into the varices of the lower esophagus. The injection of 5% EO resulted in bronze-colored changes of the varices in about half of the esophageal wall. Fluoroscopic images showed that the sclerosing agent had been injected into some of the remaining supplying vessels. Subsequently, EVL was performed at seven locations of the esophageal mucosa. No specific adverse events occurred after this treatment.

EGD on the 297th day showed marked improvement of the esophageal varices with Lm F1 Cb RC0 S+ and gastric varices with Lg-c F1 Cw RC0, and there was no need for additional treatment (Fig. 6). The patient is in good condition and is currently being followed at the outpatient clinic.

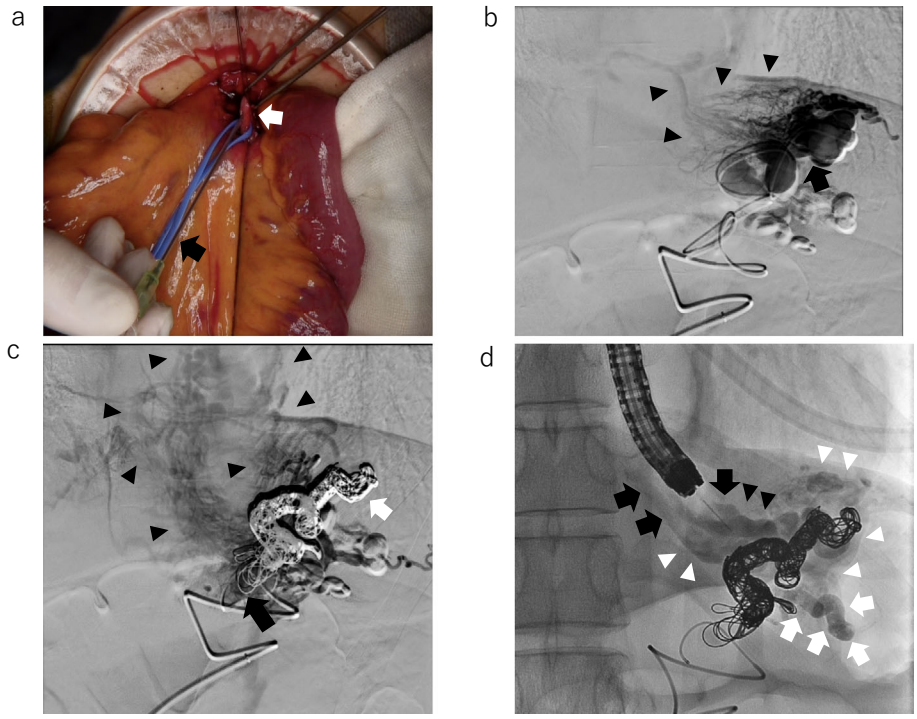

Figure 4. **Representative images during transileocolic obliteration (TIO) and subsequent endoscopic treatment (day 3). (a) After a small abdominal incision, the ileocolic vein (white arrow) was identified, and an 18-gauge puncture needle (black arrow) was then inserted. (b) Digital subtraction venography with contrast injection. The tip of the catheter is advanced peripherally in the dilatated gastric coronary veins and reaches just proximal to the gastric varices (white arrow). Gastric coronary veins (black arrow) and collateral vessels toward the esophagus (arrowheads) are confirmed. (c) Digital subtraction venography with contrast injection after coil embolization. The mainly dilated gastric coronary veins are occluded by multiple coils (Target XXL coils; Stryker, Kalamazoo, USA) (white arrow). The tip of the catheter is placed in the gastric coronary veins proximal to the embolization coils (black arrow). Collateral vessels including palisading veins remain, and hepatofugal blood flow toward the esophageal varices is found (arrowheads). (d) Venography during endoscopic injection sclerotherapy just after TIO. A total of 15 mL of 5% ethanolamine oleate (EO) was injected intravascularly at the lower esophagus (black arrows). The contrast-enhancing agent mixed with EO shows direct thick communicating varices from the esophagus to the stomach, considered pipeline varices (black arrowheads). Successful injection of the sclerosing agent from the esophageal varices to the remaining gastric coronary veins is confirmed (white arrowhead). The white arrows show retention of 67% n-butyl-2-cyanoacrylate, mixed with liposoluble contrast media in the cardiac varices, injected on day 1.**

Discussion

A case of EHPVO with pipeline EGVs that was difficult to treat with intravariceal EIS alone due to large intravariceal blood flow volume was described. TIO was performed to reduce the blood flow by blocking the supplying vessels, and EO-EIS accompanied by EVL was performed again immediately afterwards, resulting in effective treatment.

EHPVO is defined as "a vascular disorder of the liver characterized by occlusion of the extrahepatic portal vein" (5). Initial occlusion in the portal vein is often unrecognized. Multiple hepatopetal collaterals subsequently develop around the portal vein and progress into a cavernoma (6), followed by dilatation of hepatofugal vessels, such as EGVs and portosystemic collaterals. Compared to liver cirrhosis, patients with portal hypertension due to EHPVO have a higher frequency of larger esophageal varices and varices involving the gastric cardia (1, 7).

The Baveno VI consensus workshop stated that endoscopic treatment of EGVs following EHPVO as well as cirrhotic EGVs is useful (8). In the present case, esophageal varices that were continuous with cardiac varices developed. A good indication for intravariceal EIS is the presence of esophageal varices continuous with cardiac varices (9). However, during the initial EO-EIS for pipeline varices, the blood flow was too high to allow the sclerosant to remain in the varices in the present case.

Pipeline varices, including those in the present case, are those with direct blood flow into the esophagus from the

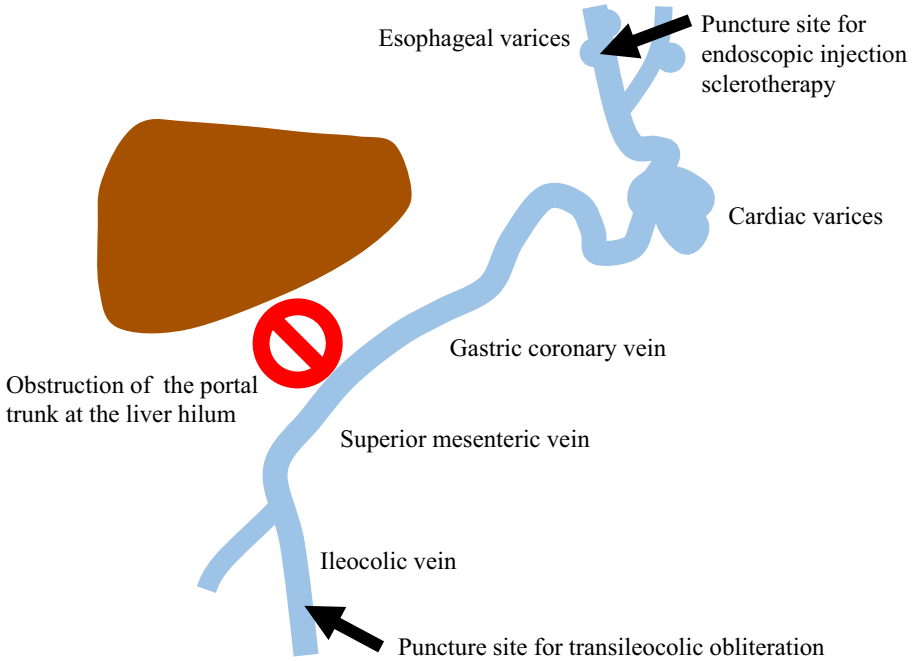

Figure 5. **Schematic illustration of the portal venous system at the time of TIO with endoscopic treatment. Due to occlusion of the trunk of the portal vein in the hilar region, blood flow from the superior mesenteric vein, especially to the gastric coronary vein, was increased. This resulted in the development of cardiac and esophageal varices.**

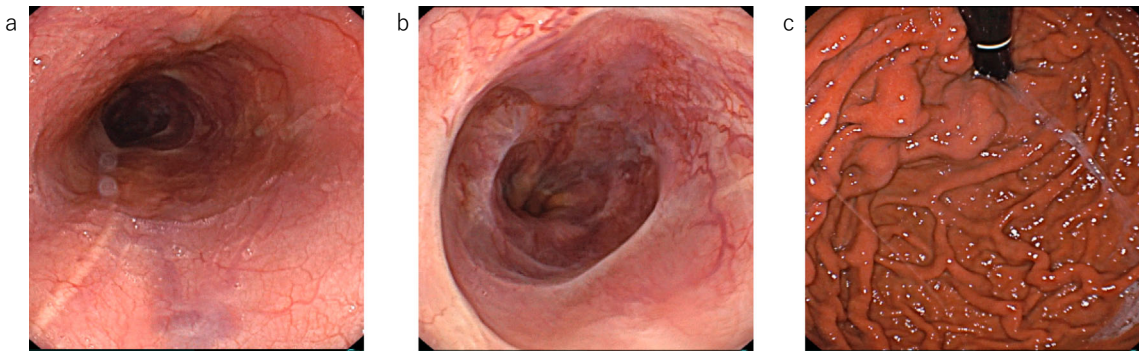

Figure 6. **Endoscopic images of esophagogastroduodenoscopy on day 297. (a, b) Improvement of esophageal varices with Lm F1 Cb RC0 S+. (c) Improvement of gastric varices with Lg-c F1 Cw RC0.**

blood-supplying vessels. Compared to palisading-type varices, a higher intravariceal blood pressure and a lower pressure reduction rate towards downstream are characteristics of pipeline varices (10). Thus, pipeline varices are often refractory to endoscopic treatment. In patients with EGVs refractory to EIS, the percentage of pipeline varices was 100%, and regarding adverse events associated with EIS, bleeding requiring blood transfusion occurred in 38% (2).

Several treatment options for acute variceal bleeding that are difficult to treat endoscopically are suggested in the Baveno guidelines (8), including transjugular intrahepatic portosystemic shunt (TIPS) and balloon tamponade. However, in the present case, the intrahepatic portal vein was highly stenotic, so TIPS was not applicable. Balloon tamponade was also not selected in the present case because it was not expected to be effective in the long term.

The use of self-expandable metal stents (SEMSs) is another way to compress the bleeding site on the esophageal wall to achieve hemostasis (8). A randomized control study showed that SEMSs have greater efficacy with less-severe adverse events than balloon tamponade in the control of esophageal variceal hemorrhaging (11). Although the use of SEMSs for refractory variceal bleeding has shown good immediate bleeding control with an acceptable technical success rate, the mortality and rebleeding rates were reported to be higher than those of TIPS in a meta-analysis (12). Thus, according to the European Society of Gastrointestinal Endoscopy (ESGE) Guideline, the use of SEMSs is considered as a temporal bridge therapy to TIPS (13), and we did not consider the use of SEMSs to be a positive option.

Other choices include TIO and percutaneous transhepatic obliteration (PTO). In both procedures, blood flow in the supplying vessels is reduced to achieve hemostasis of the bleeding varices. PTO requires percutaneous transhepatic in-

Table. Previous Reports of Transileocolic Obliteration to Treat Varices in Patients with Portal Hypertension.

Reference	Age	Sex	Etiology	Location of varices	Prior treatment to TIO	Obliterating agents	Combined treatment with TIO	Treatment effect	Adverse events
(4)	57	Female	Cirrhosis	Gastric cardia	None	Coils	EIS	Eradication after additional EIS	Early rebleeding
	53	Female	Cirrhosis	Gastric cardia	Not described	Coils	EIS	Eradication after additional EIS	Early rebleeding
	55	Female	Cirrhosis, HCC	Esophagus, gastric cardia	Not described	Coils	EIS	Eradication after additional EIS	Not described
	59	Female	Cirrhosis, HCC	Gastric cardia	Refractory to EIS	Coils	EIS	Shrinkage after additional EIS	Not described
(15)	65	Female	HCV	Duodenum	Refractory to EIS	EO, coils	B-RTO	Eradication	None
(16)	60	Male	Alcohol	Gastric fornix	None	Coils, 50% glucose	Left gastric arterial embolization and PSE	Eradication after additional EIS and EVL	None
(17)	46	Male	HBV	Esophagus, gastric fornix	None	EO	B-RTO	Eradication	Mild hematuria
(18)	72	Male	EHPVO post choledochojejunostomy for CBD stenosis	Anastomotic varices	None	Coils	None	Eradication	SMV thrombosis
(19)	78	Female	EHPVO post PD	Bile duct	Refractory to APC and HSE injection	NBCA	None	Eradication	None
(20)	70	Male	Alcohol	Rectum	None	EO, coils	None	Eradication	None
(21)	69	Male	EHPVO post left hepatic trisegmentectomy	Jejunal varices	None	Coils	None	Hemostasis	None
(22)	86	Male	EHPVO post PD	Anastomotic varices	None	Coils	PV stenting	Eradication	None
(23)	72	Female	NASH	Duodenum	Incomplete retrograde obliteration	coils, 50% glucose	PSE	Eradication	Bacteremia (*Klebsiella aerogenes*)
(3)	34	Male	HCV, AIH	Esophagus	Refractory to EIS, EVL and TIO	EO, coils, NBCA	None	Eradication	PV thrombosis
Our case	29	Male	EHPVO	Esophagus and gastric cardia	Incomplete hemostasis with endoscopy	Coils	EIS, EVL	Eradication after additional EIS and EVL	Mild ascites

AIH: autoimmune hepatitis, APC: argon plasma coagulation, B-RTO: balloon-occluded retrograde transvenous obliteration, CBD: common bile duct, EHPVO: extrahepatic portal vein obstruction, EIS: endoscopic injection sclerotherapy, EO: ethanolamine oleate, EVL: endoscopic variceal ligation, HBV: hepatitis B positive, HCC: hepatocellular carcinoma, HCV: hepatitis C positive, HSE: hypertonic saline-epinephrine, NASH: nonalcoholic steatohepatitis, NBCA: n-butyl-2-cyanoacrylate, PD: pancreaticoduodenectomy, PSE: partial splenic embolization, PV: portal vein, TIO: transileocolic obliteration

sertion of a catheter into the gastric coronary vein (14), whereas TIO begins with a minor abdominal incision, followed by insertion of a catheter through the ileocolic vein to embolize the blood-supplying vessels (3). As TIO is the more invasive of the two, it is indicated for patients with severe hepatic atrophy, ascites, or other conditions that make it difficult to puncture the intrahepatic portal vein. Since the intrahepatic portal vein was highly stenotic in the present case, TIO was selected.

Table shows the characteristics of the present case and

cases of TIO that could be found in PubMed using the key words of "transileocolic" or "ileocolic vein" and "varices" (3, 4, 15-23). The locations of varices that could be approached via the portal venous system varied. There were 5 EHPVO patients among the 15 cases of TIO (33%). Eradication or hemostasis of varices by TIO monotherapy was achieved in five cases. Ten patients required combination therapy other than TIO, including five cases of endoscopic treatment (including the present case), two cases of B-RTO, two cases of partial splenic embolism (PSE), and one case of portal vein stenting. Six patients, including the present patient, required further endoscopic treatment until eradication of the varices. Although there have been several case reports on TIO, none have reported a case series with a large population, making it difficult to satisfactorily assess the risk of variceal recurrence or rebleeding and the time to these events. For reference, a report on PTO, in which embolization was performed by approaching the collateral vessels via the portal system, like TIO, states that PTO has a high recurrence rate of varices, as 65% of patients rebled within a mean of 4.6 months (24). Notably, Yamamoto et al. reported that two of the four patients who underwent TIO using metallic coils rebled within a few days while awaiting EIS (4). We believe this is because it is difficult to embolize all collateral vessels with TIO or PTO alone, and there remains a potential risk of variceal recurrence and rebleeding.

In fact, in the present case, blood flow in esophageal varices and some collateral vessels remained after TIO monotherapy using coils, suggesting a high risk of early recurrence and rebleeding due to vessel recanalization and neovascularization. Thus, additional endoscopic treatment immediately after TIO was considered reasonable in terms of reducing the risk of early rebleeding from the remaining varices. There have been no previous reports of cases in which EGVs were treated with endoscopic techniques immediately after TIO.

Since EGV recurrence is a concern even after TIO combined with EIS, we believe that continuous endoscopic follow-up is mandatory, and additional EGV treatment should be carried out if necessary. In the present case, the peribiliary plexus developed as hepatopetal collaterals, and there was a risk of exacerbation and rupture of these vessels due to increased portal pressure. However, CT showed no evidence of worsening peribiliary plexus after EGV treatment. Regarding other treatment-related adverse events, there was an increase in ascites after TIO with EIS, which was manageable only with the use of short-term diuretics.

In conclusion, for EGVs that are difficult to manage by endoscopic treatment alone due to fast intravariceal blood flow, TIO may be an option for reducing the flow to the collaterals in order to allow for successful endoscopic treatment.

The authors state that they have no Conflict of Interest (COI).

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