

[CASE REPORT]

The First Transileocolic Obliteration for Refractory Esophageal Varices: A Case Report and Review of the Literature

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Abstract:

Transileocolic obliteration (TIO) is a useful treatment for gastric, duodenal, or rectal varices. However, TIO for esophageal varices has not yet been reported. We herein report successful TIO performed for refractory esophageal varices with a large paraesophageal vein, with no subsequent recurrence of varices.

Key words: transileocolic obliteration, esophageal varices, paraesophageal vein

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Introduction

Transcatheter treatments, such as percutaneous transhepatic obliteration (PTO) (1) and transileocolic obliteration (TIO) (2), can be selected for cases of refractory varices after failure of endoscopic injection sclerotherapy (EIS) and/or endoscopic variceal ligation (EVL). In cases of severe liver atrophy, ascites, and neoplastic lesions of the liver, which are contraindications for PTO, or for cases in which a percutaneous transhepatic catheter operation is difficult, TIO can be a treatment option for varices. Although there have been reports of TIO for gastric, duodenal, and rectal varices, there have been no reports of TIO for esophageal varices according to a search of PubMed using the key words “transileocolic obliteration”.

We herein report the first use of TIO for esophageal varices with a large paraesophageal vein, along with a review of the literature.

Case Report

The patient gave his informed consent for the publication of the details of his case.

A 34-year-old man who achieved eradication of hepatitis C virus by peg-interferon and ribavirin treatment from February 201X to January 201X+1 developed autoimmune hepatitis in July 201X+1, and steroid treatment was initiated. In October 201X+1, abdominal ultrasonography and upper gastrointestinal endoscopy revealed liver cirrhosis and F1 esophageal varices, respectively. Upper gastrointestinal endoscopy in June 201X+6 showed enlargement of the esophageal varices to F2 along with new F1 gastric varices, and portal vein thrombosis appeared on abdominal computed tomography (CT).

Administration of antithrombin III resulted in a reduction in portal vein thrombosis. Following warfarin treatment, the portal vein thrombosis was resolved in April 201X+9. After the first EVL for esophageal varices in 201X+6, EIS and

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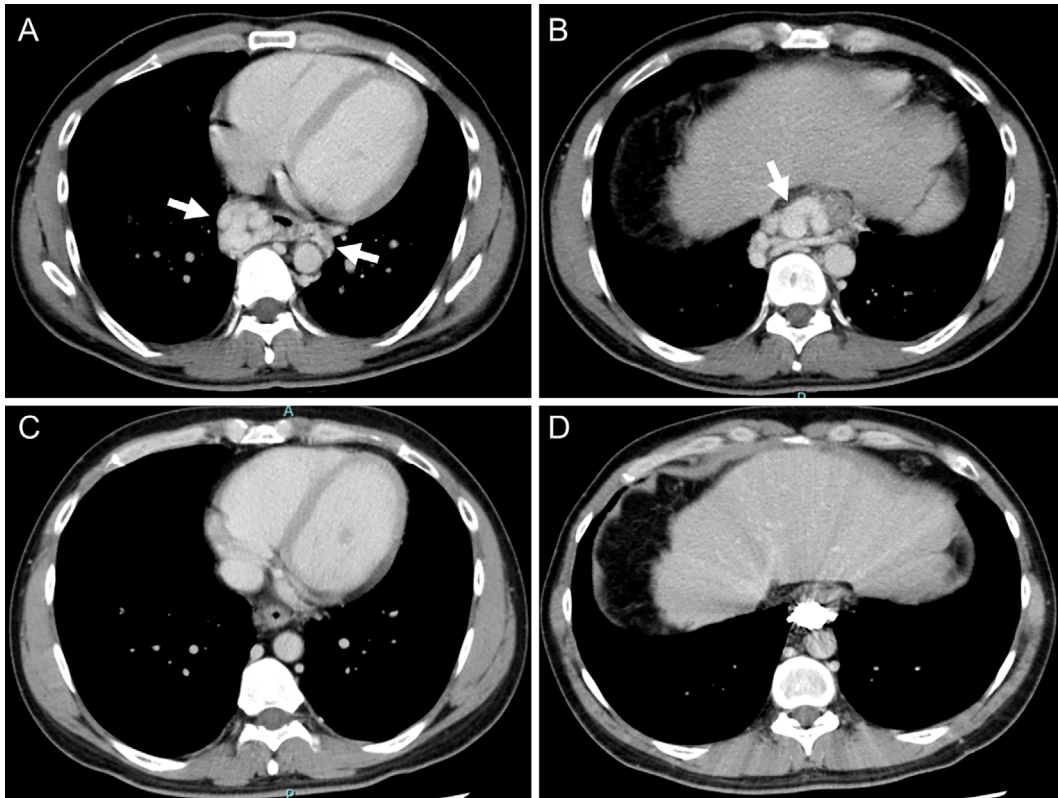


Figure 1. (A, B) Dynamic contrast-enhanced computed tomography (CT) showing a large paraesophageal vein (arrows). (C, D) Follow-up CT after 12 months showing the diminished paraesophageal vein.

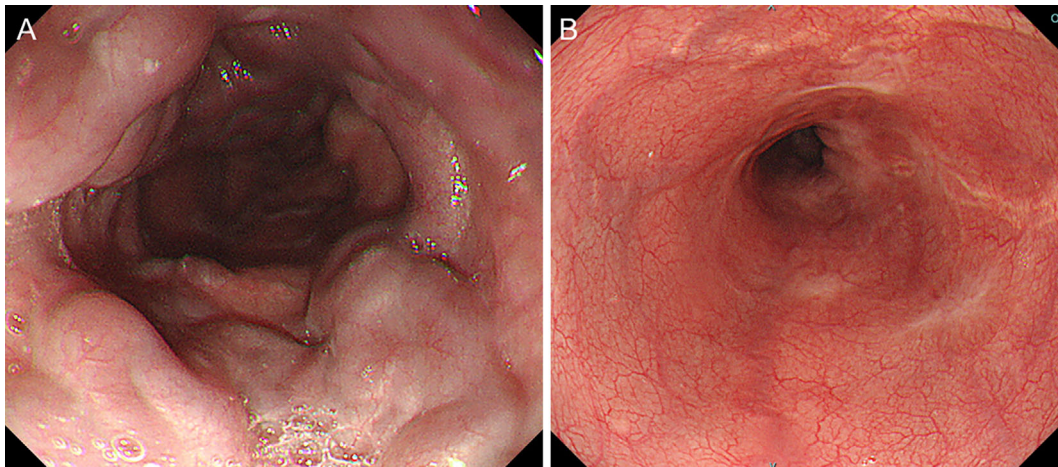


Figure 2. (A) Esophagogastroduodenoscopy showing esophageal varices before the transileocolic obliteration (TIO) procedure. (B) Esophagogastroduodenoscopy six months after TIO showing the shrunken esophageal varices.

EVL were repeated for recurrence of esophageal varices; EIS was performed in 201X+7 and 201X+8, and EVL was performed twice in 201X+8. Because abdominal CT in August 201X+6 showed not only submucosal venous dilation but also traffic with dilated paraesophageal veins through penetrating blood vessels (Fig. 1A, B), endoscopic treatment was considered an ineffective approach to managing the blood supply from the paraesophageal veins.

In 201X+8, the patient was referred to our hospital for

the treatment of paraesophageal varices. An attempt at PTO was unsuccessful due to the tortuosity of the feeding blood vessel tract. Because the size of the esophageal varices had worsened to F2 (Fig. 2A), the patient was hospitalized for TIO treatment of paraesophageal varices. He was administered ursodeoxycholic acid, rebamipide, prednisolone, famotidine, a potassium preparation, and bisphosphonate. Warfarin had been discontinued one week before the TIO procedure. Laboratory studies showed elevated transaminase lev-

Table. Laboratory Data.

WBC	3.50×10 ³ /μL	Na	143 mEq/L	Type 4 collagen 7s	10.0 ng/mL
RBC	4.64×10 ⁶ /μL	K	3.5 mEq/L	Hyaluronic acid	185.0 ng/mL
Hb	14.5 g/dL	Cl	111 mEq/L	M2BPGi	1.94
Ht	41.1 %	BUN	15 mg/dL	IgG	1,148 mg/dL
Plt	5.5×10 ⁴ /μL	Cr	0.73 mg/dL	IgM	103 mg/dL
PT	59 %	eGFR	99.5 mL/min/1.73m ²	ANA	40
PT-INR	1.29	TP	6.2 g/dL	HBsAg	N.D.
AST	42 U/L	Alb	3.7 g/dL	Anti-HBs	N.D.
ALT	41 U/L	T-Cho	158 mg/dL	Anti-HBc	N.D.
γ-GTP	17 U/L	Glucose	100 mg/dL	Anti-HCV	Positive
ALP	195 U/L	HbA1c	4.9 %	HCV RNA	N.D.
LDH	196 U/L	NH ₃	54 μg/dL	FIB4-index	4.29
T-Bil	1.9 mg/dL	Zn	64 μg/dL	ALBI score	-2.15
D-Bil	0.6 mg/dL	AFP	1 ng/mL	Modified ALBI grade	2a
CRP	0.04 mg/dL				

WBC: white blood cells, RBC: red blood cells, Hb: hemoglobin, Ht: hematocrit, Plt: platelets, PT: prothrombin time, PT-INR: prothrombin time international normalized ratio, AST: aspartate aminotransferase, ALT: alanine aminotransferase, γGTP: γ-glutamyl transpeptidase, ALP: alkaline phosphatase, LDH: lactic acid dehydrogenase, T-Bil: total bilirubin, D-Bil: direct bilirubin, CRP: C-reactive protein, BUN: blood urea nitrogen, Cr: creatinine, eGFR: estimated glomerular filtration rate, TP: total protein, Alb: albumin, T-Cho: total cholesterol, AFP: α-fetoprotein, M2BPGi: mac-2-binding protein glycosylation isomer, IgG: immunoglobulin G, ANA: antinuclear antibody, HBsAg: hepatitis B surface antigen, Anti-HBs: antibody to hepatitis B surface antigen, Anti-HBc: antibody to hepatitis B core antigen, Anti-HCV: antibody to hepatitis C virus, ALBI: albumin bilirubin, N.D.: not detected

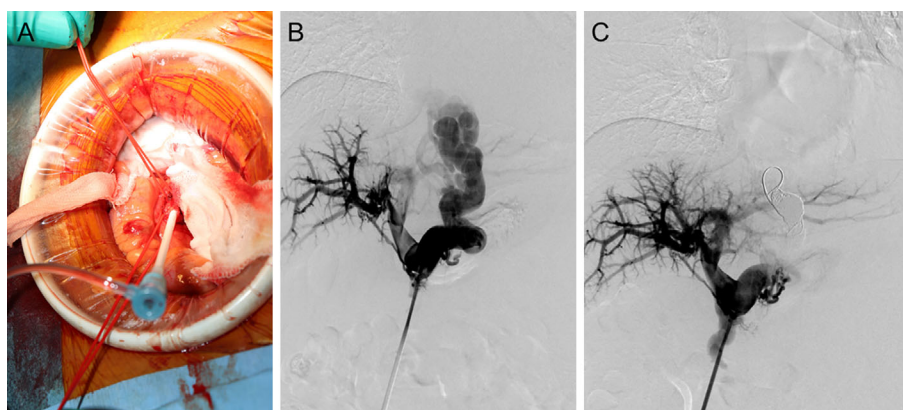


Figure 3. (A) Catheterization of the ileocolic vein through the catheter introducer under laparotomy. (B) Portography showing a large paraesophageal vein before embolization. (C) Portography showing an improved portal vein blood flow in the left lobe of the liver after embolization.

els, with a Child-Pugh score of 5 (Table). The albumin bilirubin (ALBI) score was -2.15, and the modified ALBI grade was 2a.

TIO was performed as follows: under general anesthesia, an incision was made in the right lower rectus abdominis muscle, and the ileocolic vein was exposed. An 18-gauge indwelling needle puncture was performed under direct vision (Fig. 3A), a guide wire was advanced toward the intrahepatic portal vein, and an 8-Fr sheath (PINNACLE; Terumo, Tokyo, Japan) was inserted. Portography at the superior mesenteric vein depicted a large paraesophageal vein as a collateral vessel from the coronary vein arising from the superior mesenteric vein (Fig. 3B). Temporary blood flow stasis in the paraesophageal vein was achieved using a 2-cm

balloon catheter (Selecon MP; Terumo, Tokyo, Japan) in the proximal portion of the coronary vein. In addition, 20 mL of 5% ethanolamine oleate with iopamidol (EOI) was injected, and the proximal portion of the coronary vein was immediately embolized with 7 metallic coils (28 mm×60 cm, 20 mm×60 cm, 14 mm×60 cm, Packing Coil; Penumbra, Alameda, USA). Because the paraesophageal vein was still visualized through a small branch on the proximal side, 4 mL of 5% EOI and a small amount of N-butyl-2-cyanoacrylate and lipiodol mixed at a ratio of 1:2 were additionally injected. After embolization, the paraesophageal vein was no longer visualized (Fig. 3C), and the intrahepatic portal vein blood flow in the left lobe of the liver significantly improved.

A blood test on the second day after the TIO procedure showed an increase in the D-dimer level, and contrasted CT revealed thrombosis of the ileocolic vein; therefore, heparin treatment was initiated, and warfarin was restarted on the fifth day after the TIO procedure. Contrast-enhanced abdominal CT performed seven days after the TIO procedure showed no significant change in the ileocolic vein thrombosis, but a new thrombosis was found in the left gastric vein extending to the main portal vein. Esophagogastroduodenoscopy performed 29 days after the TIO procedure showed a marked reduction in the size of the esophageal varices, and contrast-enhanced abdominal CT performed three months after the TIO procedure revealed disappearance of the portal vein thrombosis. Subsequent contrast-enhanced abdominal CT performed 10 months after the TIO procedure showed regression of the paraesophageal vein (Fig. 1C-D), and the esophageal and gastric varices had not worsened for 12 months (Fig. 2B). The Child-Pugh score 12 months after the TIO procedure was 6 (prothrombin time and international normalized ratio, 1.97 under warfarin administration), the ALBI score was -2.67 (albumin, 4.1 mg/dL; total bilirubin, 1.0 mg/dL), and the modified ALBI grade was 1, indicating an improvement in the liver function.

Discussion

Esophageal variceal bleeding is potentially fatal and affects the prognosis of patients with portal hypertension. For esophageal varices, first-choice endoscopic treatments include EIS and EVL (3). However, in refractory cases, interventional radiology (IR) procedures can be performed. The IR procedures for portal hypertension include two methods. One is embolization of the collateral circulation, such as PTO (1) and TIO, and the other is decompression of portal pressure, such as partial splenic embolization (4) and transjugular intrahepatic portosystemic shunt (TIPS) (5). In 1979, Ueda et al. first performed TIO, in which the ileocolic vein was exposed under laparotomy, and a catheter was inserted into the portal vein to embolize the varices (6). TIO is indicated for cases in which puncture is not possible due to severe liver atrophy, ascites, and neoplastic lesions in the liver, or for cases in which a percutaneous transhepatic catheter operation is difficult. The inclusion criteria for TIO at esophageal varices include the following: cases of intractable recurrence of esophageal varices which are difficult to treat by endoscopic treatment, and cases that cannot be treated with PTO. In the present case, because of the recurrence of esophageal varices immediately after endoscopic treatment due to the large paraesophageal vein, we attempted PTO; however, the meandering nature of the blood supply tract prevented a successful catheter procedure.

There have been various reports on the relationship between paraesophageal veins and esophageal varices (7, 8). The left gastric vein, which is the feeding vessel for the esophageal varices, divides into anterior and posterior branches. The anterior branch supplies blood to the esopha-

geal varices via the blind blood vessels, and the posterior branch donates to the esophageal varices from the paraesophageal vein via the penetrating branches. While endoscopic treatment reduces submucosal vasodilation and penetrating branches, dilation of the posterior branch of the left gastric vein and the paraesophageal vein may progress. Highly developed paraesophageal veins and large penetrating blood vessels are reportedly difficult to control using EIS alone (7, 9). In addition, it has been reported that varices are significantly more likely to recur when the paraesophageal vein contacts the esophagus (7), as in our case.

Since TIO causes an increase in portal vein pressure, the appearance of new collateral circulation may lead to the development of esophageal varices. Cases of esophageal varices appearing after TIO for gastric varices have been reported, and it is useful to combine other embolic therapies to prevent recurrence of esophageal varices (7, 10). In our case, the esophageal varices were significantly improved, but close follow-up of the esophageal varices and paraesophageal veins was required. Furthermore, although there have been few reports of complications due to TIO, portal vein thrombosis and ascites were observed in our case. Therefore, clinicians should monitor patients for complications after TIO. Furthermore, the ALBI score was improved with visualization of the portal vein blood flow after embolization of the paraesophageal vein, which was consistent with the findings of a previous report on the improvement of the liver function after balloon-occluded transfemoral obliteration (11).

In conclusion, to our knowledge, this is the first report of TIO for esophageal varices. TIO is useful for treating refractory esophageal varices with a large paraesophageal vein.

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

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