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CASE REPORT

Percutaneous Covered Stenting in Splenic Vein for Left-sided Portal Hypertension Caused by Chronic Splenic Vein Obstruction

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Abstract:
Left-sided portal hypertension (LSPH), an uncommon manifestation of portal hypertension, is characterized by conditions such as isolated gastric varices and splenomegaly, which result from impeded splenic venous drainage in the presence of pancreatic disease. We employed a percutaneous transhepatic technique to achieve regression of isolated gastric varices by implanting a covered stent within a blocked splenic vein and by embolizing the posterior gastric vein and varices using N-butyl-2-cyanoacrylate. We report the successful treatment of stenting for LSPH by the covered stent placement.

Keywords:
chronic pancreatitis, gastric varices, left-sided portal hypertension, portal hypertension, splenic vein occlusion

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Introduction

Obstruction of the splenic vein (SV) in conjunction with chronic pancreatitis is observed in approximately 13% of patients within 8.2 years of diagnosis. A large proportion of patients are suspected to have left-sided portal hypertension (LSPH) [1, 2], which is characterized by the diversion of splenic venous blood into the portal tract through alternate pathways due to stenosis or blockage of the SV. Several recent reports have described the usefulness of splenic vein stenting (SVS) for LSPH for various reasons, one of which is that SVS is recognized as an approach that adheres to the original anatomical treatment [3]. The patient described in this case report consented to the publication of this manuscript. Approval from the institutional review board was not necessary.

Case Report

For nearly two decades, a 60-year-old man had endured obstructive SV symptoms caused by alcohol-induced pancreatitis. Endoscopic examination showed the presence of isolated intragastric varices (type 1 as classified by the Sarin system) at the cardia and upper portion of the stomach, exhibiting gradual expansion throughout the observation period [4]. Contrast-enhanced computed tomography (CT) scanning showed that the pancreas had undergone considerable atrophy, with pancreatic stones and pseudocysts near the tail of the pancreas (**Fig. 1a and 1b**). The patient's SV was obstructed, which caused redirection of the venous flow from the splenic hilum to the coronary veins via the short gastric and posterior gastric veins and to the gastrocolic trunk via the gastroepiploic vein as collateral hepatopetal veins. A splenorenal shunt was confirmed, which also indicated drainage to the systemic vein. We diagnosed LSPH with the progression of gastric varices and judged the patient to be at an increased risk of variceal rupture. Preoperative contrast-enhanced CT scan revealed a minimal contrast effect on the portal vein (PV) side of the SV, prompting us to opt for reconstruction of the SV. The results of the blood tests assessing liver and renal function were within the normal ranges.

After percutaneous puncture of the right anterior PV under local anesthesia, an introducer sheath (7.2F, 30 cm, Super Sheath; Medikit Co. Ltd., Tokyo, Japan) was inserted. After introducing a multipurpose catheter (4F, 80 cm, HeartCath; Terumo Corp., Tokyo, Japan) into the PV, portography

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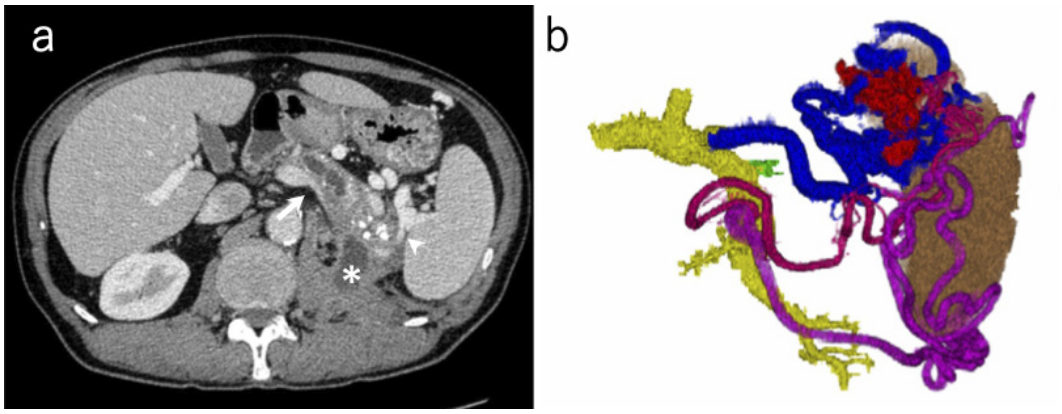

Figure 1.
a: In this contrast-enhanced CT image, the portal side of the occluded SV is marked with a white arrow, and the splenic hilum side is indicated by a white arrowhead. White asterisk denotes the presence of a pancreatic pseudocyst.
b: This volume rendering view depicts the PV, with the superior mesenteric vein and main trunk of the PV shown as yellow. The short gastric vein and left gastroepiploic vein are marked in red, whereas the posterior gastric vein is shown as blue. The splenic vein is shown in pink. The terminal part of the occluded SV is highlighted in green.

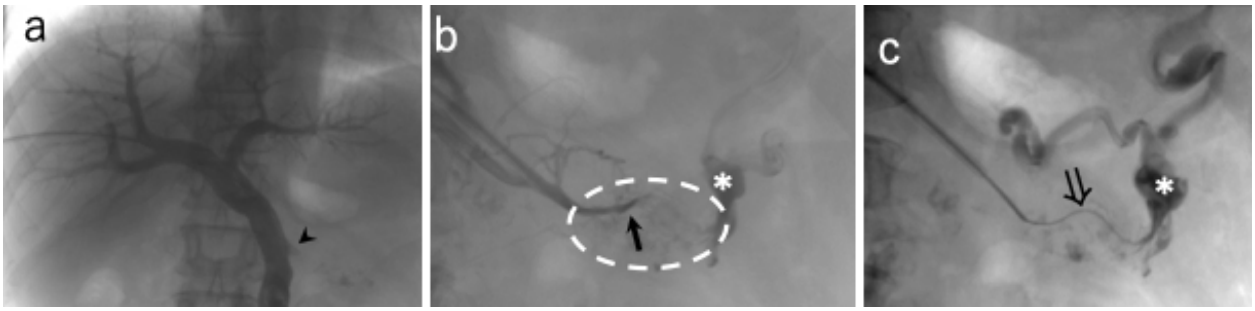

Figure 2. Portal venography from the superior mesenteric vein.
Image (a) shows portal venography from the SMV; image (b), the tip injection from the occluded SV; and image (c), splenic venography from a regionally crossed site of an occluded vein. The stamp of the occluded SV is indicated by the black arrowhead. A 5F catheter is represented by the black arrow. A microchannel in an occluded SV is surrounded by the white dotted circle. A microcatheter is indicated by the black dashed arrow. The short gastric vein is shown by a white asterisk.

was performed (**Fig. 2a**). A blood flow-residual lumen at a margin of the obstructed SV was shown to be on an early phase of an earlier CT scan (**Fig. 2b**). From the margin of the obstructed SV, a microcatheter (1.7F, 110 cm, Progreat λ; Terumo Corp., Tokyo, Japan) with a guidewire (0.014 in, 175 cm, Treasure; Asahi Intecc Co., Ltd., Aichi, Japan) successfully traversed the region (**Fig. 2c**). The pre-angioplasty pressure gradient was 7 mmHg, with 4-mmHg central PV pressure and 11-mmHg distal SV pressure. Even after dilatation of the occluded SV using a 4 × 20 mm balloon catheter (Sterling; Boston Scientific Corp., Boston, MA), advancing a 7F guiding sheath (Ansel Flexor; Cook Medical, USA) was difficult. Consequently, the guiding sheath was successfully inserted to the splenic hilum of the SV using the balloon-assisted tracking technique (**Fig. 3a and 3b**). Through a 5F 70-cm catheter (disposable catheter; Hanaco Medical Co., Ltd., Saitama, Japan) in the guiding sheath, the microcatheter was advanced into the dilated short gastric vein. The posterior gastric vein and gastric varices were embolized using a 20% n-butyl-cyanoacrylate (NBCA)-lipiodol mixture (**Fig. 4**).

A 0.035-in stiff guidewire (Amplatz Super Stiff guidewire; Boston Scientific Corp., Boston, MA) was advanced to perform predilation of the occluded SV using an 8 × 20 mm balloon catheter (Mustang; Boston Scientific Corp., Boston, MA). Subsequently, the guiding sheath was removed with the guidewire in place within the SV. Then, 8 × 40 mm and 8 × 60 mm covered stents (8F Fluency; BD Bard Inc., Karlsruhe, Germany) were deployed from the distal side of the SV to cover the occlusion length. Splenic venography confirmed blood flow resumption within the SV (**Fig. 5a**). Minimal gradient pressure between the central PV (5 mmHg) and distal SV (6 mmHg) was observed. Heparin was not used during the procedure. After the procedure, a regimen of acetylsalicylic acid 100 mg and clopidogrel hydrogen sulfate 75 mg was prescribed for 6 months. Contrast-enhanced CT conducted 2 years postprocedure revealed no reocclusion of the covered stents (**Fig. 5b**). Gastric varices

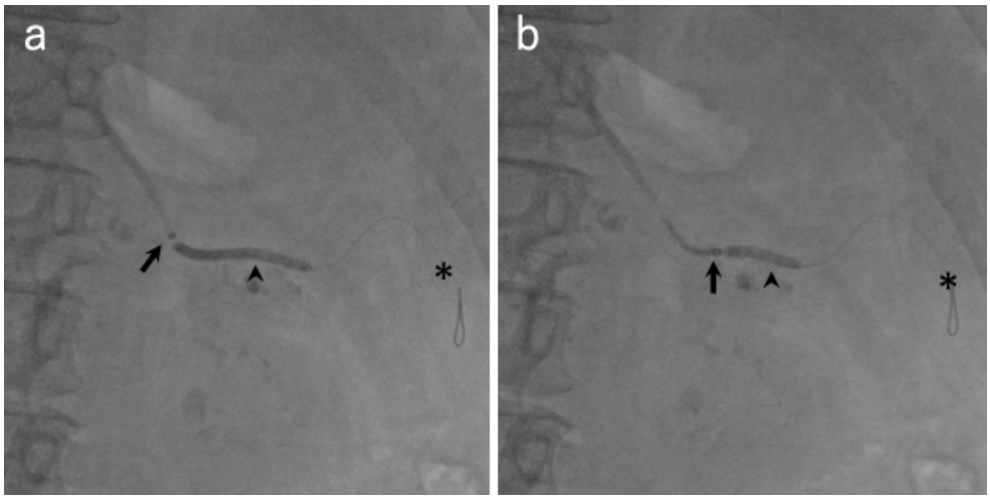

Figure 3. Balloon-assisted tracking technique.
Image (a) depicts the pushing of a guiding catheter against a balloon. Image (b) shows the guiding catheter being advanced by slow deflation and retraction of the balloon. The guiding catheter tip is indicated by the black arrow. The 4 × 2 cm balloon is indicated by the black arrowhead. A 0.014-in guidewire is denoted by a black asterisk.

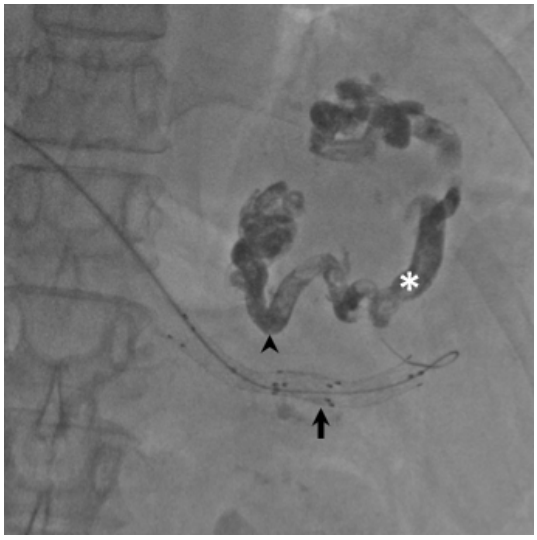

Figure 4.
View of occlusion of the short and posterior gastric veins.
The black arrow in the image indicates the deployed covered stent graft, whereas the black arrowhead indicates the site at which 20% n-butyl-cyanoacrylate (NBCA)-lipiodol glue was injected into the posterior gastric vein. In addition, the white asterisk marks the location at which 20% NBCA-lipiodol glue was injected into the short gastric vein.

remained reduced at 3 and 12 months after treatment, as observed *via* endoscopy.

Discussion

Gastric varices, which might cause severe gastric intestinal bleeding [5], were observed in 45%-72% of patients with LSPH [1]. Following the exclusion of cirrhosis, the diagnosis of LSPH is made through a comprehensive assessment, including blood test and imaging. Although gastric varicose veins from the fundus of the stomach secondary to cirrhosis has hepatofugal blood flow into the systemic vein, most of the gastric varicose veins in LSPH are distributed throughout the stomach wall as hepatopetal blood flow from the spleen to the PV. This phenomenon contributes to the difficulty in treating LSPH cases using procedures such as endoscopic sclerotherapy, balloon-occluded retrograde transvenous obliteration, and percutaneous transhepatic obliteration [6].

A simpler method, splenic artery embolization (SAE), has the same purpose as splenectomy: shrinking the varices by cutting off blood flow to the spleen. Several case reports have described the usefulness of SAE for LSPH; however, there are few systematic reviews in the literature [7]. Wei et al. retrospectively analyzed the outcomes of SAE and SVS in 37 patients diagnosed with LSPH [3]. Their study demonstrated a significantly higher rate of rebleeding among those who underwent SAE (47.8%) than among those who underwent SVS (7.1%).

The effectiveness of splenectomy, which has the potential to significantly decrease blood supply to gastric varicose veins, is controversial [3, 6, 8]. The efficacy rate of splenectomy for preventing hemorrhage has been reported to range from 65% to 100% [8-10]. For retrospective analysis, Liu et al. divided 33 patients with LSPH into 3 groups: SVS group (n = 9), splenectomy group (n = 12), and conservative treatment group (n = 12). The results indicated that rebleeding did not occur in either the SVS or splenectomy group, but bleeding occurred in three patients (25%) in the conservative treatment group [9]. The study results suggest that SVS is a more effective treatment option for preventing gastrointestinal bleeding than conventional methods such as SAE.

The application of SVS might be limited by the low success rate [3]. In reference to recanalization for PV occlusion, Klinger et al. reported a success rate of 76.5% for recanaliz-

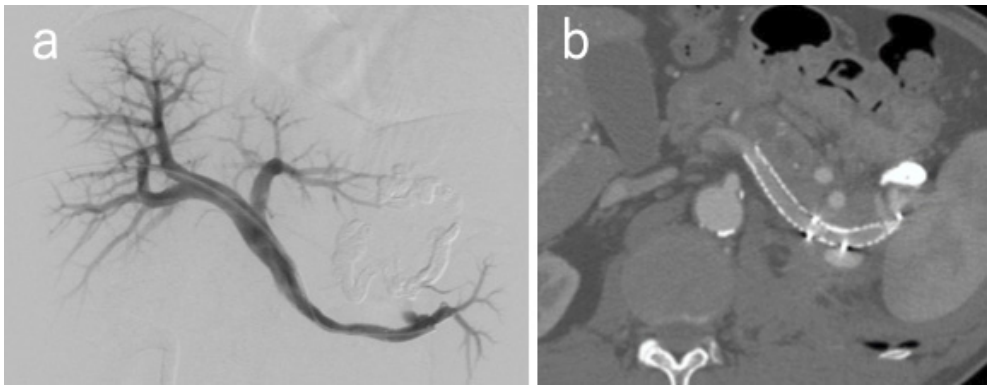

Figure 5.
a: Splenic venography image shows the absence of gastric varices and optimal stent patency.
b: CT scan image shows sustained stent patency 2 years after implantation.

Table 1. Treatment Outcomes for LSPH.

Authors	Treatment	Case of numbers	Technical success rate	Free of rebleeding rate
Köklü et al. [1]	Splenectomy	3	100%	100.00%
Wei et al. [3]	SAE	11	100%	45.50%
	SVS-SAE	12	-	58.30%
	SVS	14	53.80%	92.90%
Yalin et al. [8]	Splenectomy	43	100%	65.10%
Liu et al. [9]	SVS	9	100%	100.00%
	Splenectomy	12	100%	100.00%
Fernandes et al. [10]	SAE	5	100%	60.00%
	Splenectomy	5	100%	100.00%
Stein et al. [11]	SVS	21	85.70%	90.50%

SAE, splenic artery embolization; SVS, splenic vein stenting; SVS-SAE, splenic vein stenting and splenic artery embolization

ing PV occlusion for 17 non-cirrhotic patients, with a sustained recanalization rate of roughly 70% at 2 years [11]. Liu et al. conducted a study for which they performed stenting for SV obstruction in nine patients, achieving a 100% success rate in both procedural success and stent patency at 6 months postprocedure [9]. However, it is noteworthy that the technical success rates of SVS have been reported as 53.8%-85.7% [3, 11]. It is important to consider that the overall technical success of SVS procedures is not consistently very high (**Table 1**).

We used a transhepatic approach for treatment. However, a transsplenic approach exists as an alternative strategy for performing SVS. In patients with LSPH, the transsplenic approach is characterized by i) splenomegaly; ii) thin walls of intrasplenic vein branches; iii) risk of intraabdominal hemorrhage caused by splenic hilum puncture; iv) unfavorable angulation between splenic tributaries at the splenic hilum and occluded SV, negatively affecting the maneuverability of catheters and guidewires; and v) obstructed sections up to the splenic hilum in many patients, leading to poor operability [3]. Consequently, the transsplenic approach is regarded as more challenging and entailing a higher risk of hemorrhagic complications compared with the transhepatic approach.

No report has described the effectiveness of embolization of collateral vessels to increase blood flow through the stent in LSPH cases. It is important to maintain blood flow in the stent to prevent stent occlusion. One way to achieve blood flow maintenance is by preembolizing the short gastric vein, similar to the technique used for the transjugular intrahepatic portosystemic shunt (TIPS) procedure. Chen et al. reported that the implementation of TIPS with coronary vein embolization (n = 54) was linked to a greater primary shunt patency rate than that for TIPS without embolization (n = 52) over a 6-month period (96.2% vs. 82.0%, P = .019) [12]. Therefore, in LSPH, embolization of the short gastric vein simultaneously with SVS can be expected to increase blood flow through the stent and prevent stent occlusion.

According to Wei et al., the selection between bare and covered stents is extremely important for SVS. A covered stent should be selected to achieve three important tasks: first, preventing SV rupture during times of high balloon inflation pressure; second, preventing intraabdominal hemorrhage when slight contrast medium leakage is observed after balloon dilation or after bare stent insertion; and third, supporting a dilated SV when an impression is left on the bare stent after it has been inserted alone [3]. The endpoints of SVS were confirmation of adequate stent placement within the occluded SV, patency of the SV, and a decrease in the number of collateral vessels by venography. Few data are

available to guide the choice of stent type, particularly for bare metal versus covered stents [13].

Conclusion

Treatment of SVS for the occluded SV and embolization of the collateral vessels might prove to be efficacious for treating refractory gastric varices in LSPH cases.

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Conflict of Interest: None

Author Contribution: All authors (Y.M., H.S., H.T., K.H., O.H.) meet the International Committee of Medical Journal Editors (ICMJE) criteria for authorship and take responsibility for the content of the article.

IRB: All procedures for studies involving human participants were performed in accordance with the ethical standards of the institutional and national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

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