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INTERVENTIONAL RADIOLOGY

ORIGINAL ARTICLE

Efficacy and safety of splenic artery embolization for intractable ascites using Amplatzer vascular plug versus coil after living donor liver transplantation

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PURPOSE
Intractable ascites (IA) is an uncommon but challenging complication after liver transplantation. Splenic artery embolization (SAE) modulates the splenic artery and regulates portal flow. This study aimed to evaluate the efficacy and safety of SAE using the Amplatzer vascular plug (AVP) versus coil embolization for post-living-donor liver transplantation (LDLT) IA.

METHODS
This retrospective study evaluated consecutive patients from 1 center who received LDLT (n = 1410) between March 2006 and August 2019. The inclusion criteria for SAE were splenomegaly with IA after LDLT.

RESULTS
Totally 15 patients underwent SAE for post-LDLT IA. Eleven patients who received AVP embolization (age, 51.2 ± 15.1 years; range, 8-63 years; 5 men and 6 women) were compared with 4 patients receiving coil embolization (age, 30.8 ± 30.8 years; range, 1.5-63 years; 2 men and 2 women). AVP and coil embolization both significantly reduced portal vein hyperflow (plug/coil; $P < .001/.006$) and decreased ascites volume (plug/coil; $P < .003/.042$). The benefits of AVP embolization included shorter procedure time ($P = .029$), significantly reduced splenic volume ($P = .012$), increased liver volume ($P = .012$), decreased spleen/liver ratio ($P = .012$), and improvement of pancytopenia ($P = .008$) due to secondary hypersplenism. No significant differences were found between the two groups in the length of hospital stay or complications such as splenic infarction, pancreatitis, or sepsis.

CONCLUSION
SAE using AVP and coil embolization provide effective and safe methods for managing patients with IA after LDLT. AVP embolization may be more efficient than coil embolization, providing more effective reduction of ascites volume and the advantages of shortened procedure time and improvement of hypersplenism.

Ascites is a common finding in liver cirrhosis with portal hypertension. After transplanting the liver and resolving the hemodynamic complications, persistent ascites is not expected but does occur in a few recipients. The risk factors for persistent ascites after liver transplantation are poor graft inflow and outflow, graft rejection, recurrent hepatitis, intraperitoneal infection, and cardiac or renal failure. However, the treatments of these known causes are sometimes refractory, which results in difficulty to eliminate ascites. Some researches attribute post-transplant intractable ascites (IA) to hypersplenism and persistent portal hypertension.[1-5] Liver graft-recipient-spleen-size ratio (GRSSR) is a measurable variable for determining small-for-size-syndrome. Low GRSSR (<0.6) may predict graft failure when severe splenomegaly and small liver graft lead to portal hyperinflow.[6] Post-transplant IA is a difficult problem because it may be associated with re-transplantation, high morbidity, or even leading to mortality.[4,5] Splenic artery embolization (SAE) was developed for modulating the splenic artery and regulating portal hypoperfusion in patients undergoing liver transplantation.[5,7,8] Coil is typically used for SAE but has several post-procedural complications, including splenic infarction, portal vein (PV) thrombosis, and splenic abscess.[9] Also, post-embolization

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syndrome including fever, left upper quadrant pain, small pleural effusion, and leukocytosis had been reported.[10] Migration is an intra-procedural complication of coil embolization and may cause bleeding or infraction of the spleen parenchyma.[11,12] A relatively new technique for embolization is the Amplatzer vascular plug (AVP) (AGA Medical Corp), which is currently being explored as an alternative method for SAE in patients undergoing liver transplantation.[7,8]

Several studies have investigated SAE as the treatment of IA after liver transplantation.[5,7,8,10,13] However, the choices of different embolic materials used in SAE still need to be discussed. Even though SAE using AVP and coils has been described,[12] the therapeutic efficacy and outcomes between AVP and coils for patients with IA after living-donor liver transplantation (LDLT) have not been established. This study aimed to compare the efficacy and safety of SAE of AVP versus coils as treatment for IA in patients undergoing LDLT.

Methods

Study design and ethical considerations

In this retrospective study, we compared the data of patients who received SAE using AVP with using coil embolization and determine the efficacy and safety of these 2 techniques for SAE in patients with IA after LDLT. This study was approved by the Institutional Review Board (IRB 202001734B0) and had been conducted in accordance with the Declaration of Helsinki. The signed informed consent of included patients was waived due to the retrospective nature of the present study.

Main points

- Intractable ascites is a challenging complication after liver transplantation. The splenic artery embolization modulates the splenic artery and regulates portal flow as a treatment of intractable ascites.
- Splenic artery embolization using the Amplatzer vascular plug embolization and the coil embolization both provide effective and safe methods for managing patients with intractable ascites after living-donor liver transplantation.
- The Amplatzer vascular plug embolization may be more efficient than the coil embolization, providing a faster reduction of ascites volume, shorter procedure time, and improvement of hypersplenism.

Patient selection

Data of patients were from a single center who received LDLT (n = 1410) between March 2006 and August 2019. The inclusion criteria of patients who accepted SAE was splenomegaly with IA after LDLT. IA was defined as ascites that could not be treated satisfactorily with medical therapy within 4 weeks after LDLT. The patients with medical treatable ascites were excluded.

All patients underwent Doppler ultrasound or contrast computed tomography (CT) scans to evaluate the patency of hepatic vessels for identifying underlying causes of IA. Six patients (3 patients in AVP group and 3 patients in coil group) underwent liver biopsy to exclude graft rejection or hepatitis recurrence. All patients were investigated for the possible cardiac or renal causes of ascites. None of these patients accepted shunting procedures such as transjugular intrahepatic portosystemic shunt, peritoneal-venous shunt, or surgical portosystemic shunt.

Patients' demographic and clinical characteristics

The demographic and clinical characteristics of the 2 groups (plug and coil) are listed in Table 1. Fifteen patients (1.06%, 15/1410) underwent SAE for post-LDLT IA. Eleven patients underwent AVP embolization (age, 51.2 ± 15.1 years; range, 8-63 years; 5 men and 6 women), and 4 patients underwent coil embolization (age, 30.8 ± 30.8 years; range, 1.5-63 years; 2 men and 2 women). All SAE were performed within a mean interval of 68.5 days after liver transplantation. The plug group included 10 adults and one 8-year-old girl. The coil group included 2 adults and 2 boys aged 1.5 and 7.5 years. The graft weights were lower in the coil group because of inclusion of 2 children (graft weights, 316 and 403 g). No significant difference was found

Table 1. Demographic and clinical characteristics of patients

	Plug (n = 11)	Coil (n = 4)	P^a	P^b
Age (years)	55 (8-63)[c]	29.25 (1.5-63)[c]	.263	
Gender (men)	5 (45.45%)	2 (50.00%)		1.000
Graft type				
Right lobe liver	4 (36.36%)	0		.516
LLS+S4 liver	6 (54.55%)	2 (50.00%)		1.000
Extended LLS/LLS	1 (9.09%)	2 (50.00%)		.154
Indication for LDLT				
Alcoholic liver cirrhosis, HCC	2 (18.18%)	0		1.000
HBV-related liver cirrhosis	2 (18.18%)	0		1.000
HCV-related liver cirrhosis	5 (45.45%)	1 (25.00%)		.604
HBV, HCV-related liver cirrhosis	1 (9.09%)	0		1.000
Biliary atresia	1 (9.09%)	1 (25.00%)		.476
Congenital hepatic fibrosis	0	1 (25.00%)		.267
Autoimmune liver cirrhosis, HCC	0	1 (25.00%)		.267
Pre-LDLT parameters				
Body weight (kg)	57.1 (50.4-68.2)	33.45 (18.1-50.5)	.056	
Graft weight (g)	555 (512-629)	433 (381.3-501.3)	**.040***	
Spleen volume (mL)	1250 (799.5-1305.5)	813 (506.8-959.3)	.280	
GRWR	0.87 (0.85-1.18)	1.83 (0.99-2.30)	.073	
GRSSR	0.40 (0.38-0.55)	0.76 (0.55-0.91)	.343	
Duration from LDLT to SAE (days)	36 (30-95)	92 (29.8-123.3)	.661	

Categorical variables are expressed as n (%).
LLS, left lateral segments; LDLT, living-donor liver transplantation; HCC, hepatocellular carcinoma; HBV, hepatic b virus; HCV, hepatic c virus; GRWR, graft-to-recipient weight ratio; GRSSR, graft-to-recipient spleen size ratio; SAE, splenic artery embolization.
[a]Mann–Whitney U test; [b]Fisher exact test; Non-normally distributed variables expressed as median (interquartile range) and [c]Median (minimum-maximum).
*$P < 0.05$.

between the 2 groups in gender ($P = 1.000$), type of graft ($P = .168$), duration from LDLT to SAE ($P = .661$), pre-LDLT spleen volume ($P = .280$), pre-LDLT graft-to-recipient weight ratio (GRWR) ($P = .073$), and GRSSR ($P = .343$). Only 2 patients in the plug group and no patients in the coil group had low GRWR values (<0.8). Nine patients in the plug group and 1 patient in the coil group had low GRSSR values (<0.6) .

Procedure details

The delivery systems and devices of AVP and coil are listed in Table 2. Embolization via transfemoral artery approach was performed to access the celiac trunk. Initial angiograms demonstrated the hepatic and splenic circulations. The AVP and coil were placed at the distal splenic artery, bypassing the dorsal pancreatic artery and the great pancreatic artery. The AVP was oversized by 30%–50% relative to the diameter of desired occlusion site at splenic artery. The choice of coil was based on the estimated diameter of the target vessel.

The post-embolization angiogram for ascertaining splenic artery occlusion was performed 15-30 minutes after plug/coil deployment. If it failed to occlude the splenic artery, a second plug/coil would be placed proximal to the first device within another 15 minutes until post-embolization angiogram showed complete splenic artery occlusion. Procedure time was defined as the time between the diagnostic celiac or splenic angiogram and the first image showing complete splenic artery occlusion.

Data collection

Doppler ultrasound and abdominal CT scans were performed before and after SAE to evaluate intrahepatic blood flow and spleen and liver volume. The therapeutic effects were assessed using laboratory blood exams, body weight, ascites volume from drainage tube, and diuretic requirement. Possible complications were recorded, including splenic infarction, fever/infection, abdominal pain or vomiting, or pancreatitis.

Statistical analysis

Statistical significance was analyzed using the Statistical Package for Social Sciences (SPSS) software package (version 22, SPSS Inc.). The Shapiro–Wilk normality test was

Table 2. Delivery systems and devices of plug and coil

		Angiosheath	Angiocatheter	Microcatheter	Plug (pcs)	Coil (pcs)
Plug	1	6 Fr 25 cm	RH 4 Fr MPD 6 Fr	—	10 mm (2)	—
	2	5 Fr 25 cm	MPC 5 Fr	—	8 mm (2)	—
	3	6 Fr 25 cm	Mach 6 Fr	—	8 mm (3)	—
	4	6 Fr 25 cm	Mach 6 Fr	—	10 mm (2)	—
	5	6 Fr 25 cm	Mach 6 Fr	—	9 mm (2)	—
	6	5 Fr 10 cm	MPC 5 Fr	—	6 mm (1)	—
	7	6 Fr 25 cm	MPD 6 Fr	—	7 mm (1)	—
	8	6 Fr 25 cm	Mach 6 Fr	—	8 mm (2)	—
	9	6 Fr 10 cm	Mach 6 Fr	—	12 mm (2)	—
	10	6 Fr 10 cm	Mach 6 Fr	—	10 mm (2)	—
	11	6 Fr 25 cm	MPD 6 Fr	—	7 mm (2)	—
Coil	12	4 Fr 10 cm	IMA 4 Fr	SP	—	3 mm × 2.5 mm (3) 4 mm × 4 mm (2)
	13	4 Fr 10 cm	RC1 4 Fr	SP	—	2 mm × 3 cm (2)
	14	4 Fr 10 cm	RC1 4 Fr RH 4 Fr	SP	—	8 mm × 20 cm (1) 10 mm × 20 cm (1) 3 mm × 7 mm (3) 10 mm × 30 cm (1) 5 mm × 50 mm (2) 6 mm × 6.7 mm (2) 7 mm × 2.3 mm (1) 4 mm × 4.0 mm (5)
	15	6 Fr 25 cm	Mach 6 Fr MPC 5 Fr RH 4 Fr RC1 4 Fr	SP	—	6 mm × 6.5 mm (14) 5 mm × 5.5 mm (1)

pcs, pieces; RH 4 Fr, Terumo RH catheter 4 Fr; C1 4 Fr, Terumo RC1 catheter 4 Fr; IMA 4 Fr, Terumo IMA catheter 4 Fr; MPC 5 Fr, Codman Envoy Guiding Catheter MPC 5 F; MPD 6 Fr, Codman Envoy Guiding Catheter MPD 6 F; Mach 6 Fr, Boston scientific Model-6 F PV Mach1 CROSS 2; SP, Terumo Radifocus Micro Catheter 3.0-2.6 F.

Table 3. Techniques of splenic artery embolization and clinical outcomes

	Plug	Coil		
Number of plugs	2 (1-3)[c]			
Plug diameter (mm)	8 (6-12)[c]			
Number of coils		9.5 (4-16)[c]		
			P^a	P^b
Procedure time (minute)	49 (41.5-49.5)	58 (42.5-72.25)	**.029***	
Procedure cost (TWD)	71 295 (70 662-71 295)	66 617 (39 465-90 621)	1.000	
Hospital stay from SAE (days)	30 (30-63.5)	72.5 (50-88.5)	.483	
Adverse effects				
None	5 (45.45%)	3 (75.00%)		.569
Abdominal pain/vomiting	3 (27.27%)	0		.516
Pancreatitis	1 (9.09%)	0		1.000
Partial splenic infarction	2 (18.18%)	1 (25.00%)		1.000

Categorical variables expressed as n (%).
SAE, splenic artery embolization; TWD, New Taiwan Dollar.
[a]Mann–Whitney U test; [b]Fisher exact test; Non-normally distributed variables expressed as median (interquartile range) and [c]Median (minimum-maximum).
*$P < 0.05$.

Table 4. Efficacy of splenic artery embolization methods in reducing intractable ascites, spleen volume, and biochemistry parameters

	Plug			Coil		
	Pre-SAE	Post-SAE 1 month	*P*	Pre-SAE	Post-SAE 1 month	*P*
Ascites (mL/day)	3040 (1877-3842)	74 (1-670)	.003*	2992 (1983-4176)	195 (3.75-966)	.042*
	Pre-SAE	Post-SAE 7 days	*P*	Pre-SAE	Post-SAE 7 days	*P*
WBC	2000 (1350-4350)	3900 (2950-5650)	.026*	3350 (2625-5325)	2575 (2062-8100)	.715
Platelet	53K (40K-69K)	62K (51.5K-112K)	.008*	40.5K (25.8K-61.8K)	65.5K (37.8K-90.3K)	.144
	Pre-SAE	Post-SAE 6 months	*P*	Pre-SAE	Post-SAE 6 months	*P*
Spleen volume (cm^3)	923 (898-1042)	702 (615-813)	.012*	1625 (916-1686)	659 (417-712)	.109
Liver volume (cm^3)	970 (853-1172)	1310 (1056-1355)	.012*	468 (435-1304)	470 (464-1268)	1.000
Spleen/liver ratio	1.10 (0.79-1.56)	0.54 (0.51-0.71)	.012*	0.76 (0.60-2.55)	0.37 (0.37-0.91)	.109

Non-normally distributed variables are expressed as median (interquartile range).
P, pre-SAE compared with post-SAE (Wilcoxon sign-rank test); SAE, splenic artery embolization; WBC, white blood cell.
*$P < 0.05$.

used to examine normal distribution of variables. Demographic data of patients in Table 1, SAE techniques and clinical outcomes in Table 3 were compared between 2 groups using the Mann–Whitney U test and Fisher exact test for non-normally distributed variables. The efficacy of SAE on decreasing ascites, biochemistry parameters, and liver/spleen volume before and after SAE were compared by the Wilcoxon signed-rank test for non-normally distributed variables (Table 4). The Doppler ultrasound results before and after SAE were compared by the paired sample t test for normally distributed variables (Table 5). The non-normally distributed variables were expressed as median (interquartile range), the normally distributed variables were expressed as mean ± standard deviation, and the categorical variables were expressed as n (%). Statistical significance was set at $P < .05$.

Results

Procedural data

The technique of SAE and patient outcomes are presented in Table 3. All patients underwent successful occlusion of the splenic artery after SAE. In the plug group, AVP diameter ranged from 6 to 12 mm. The number of AVP (range, 1-3 plugs) had no influence on successful complete splenic artery occlusion after 15-30 minutes. In the 2 groups, the final angiograms both demonstrated successful occlusion of the splenic artery and marked improvement of hepatic artery (HA) graft inflow compared to the initial angiogram (Figures 1 and 2). No events of misplaced AVP or coil, non-target embolization, or device migration occurred in either group. The total procedure time of AVP embolization was significantly shorter than that of coil embolization ($P = .029$). There was no significant difference between the procedural material charges in 2 groups ($P = 1.000$).

Efficacy of SAE for intractable ascites

A significantly decreased volume of ascites within 1 month after SAE was noted in the plug group and coil group (Figure 3 and Table 4), and the time required to remove the drainage tube of ascites was shorter in the plug group (30.9 ± 17.1 days) than in the coil group (125.3 ± 70.3 days) ($P = .022$). The result may indicate that the volume of ascites decreased more quickly and effectively in the plug group than in the coil group. No significant differences were found in body weight changes (plug/coil; $P = .694/0.071$) and diuretic doses (plug/coil; $P = .059/0.317$) before and after SAE in 2 groups. The overall body weight of the coil group was lower than that of the plug group because of inclusion of 2 children.

Table 5. Doppler ultrasound results pre- and post-splenic artery embolization

	Plug			Coil		
	Pre-SAE	Post-SAE 1-3 months	*P*	Pre-SAE	Post-SAE 1- 3 months	*P*
PV diameter (mm)	9.7 ± 0.6	8.9 ± 1.2	**.045***	9.1 ± 0.9	7.3 ± 0.9	.074
PV mean velocity (cm/sec)	34.4 ± 6.0	25.7 ± 6.9	**.002***	34.2 ± 3.8	19.8 ± 1.9	**.006***
PV volume (mL/min)	1548 ± 368	972 ± 395	**<.001***	1310 ± 130	619 ± 161	**.006***
HA peak systolic velocity (cm/sec)	38.3 ± 5.1	76.8 ± 17.8	**<.001***	48.8 ± 18.9	86.0 ± 16.5	**.002***
HA resistive index	0.84 ± 0.08	0.75 ± 0.07	**.005***	0.80 ± 0.08	0.64 ± 0.12	**.026***
HA pulsatility index	2.21 ± 0.47	1.25 ± 0.35	**<.001***	1.87 ± 0.53	1.12 ± 0.41	**.012***

Normally distributed variables are expressed as mean ± standard deviation.
P, pre-SAE compared with post-SAE (paired sample t test); SAE, splenic artery embolization; PV, portal vein; HA, hepatic artery.
*$P < 0.05$.

Doppler ultrasound and abdominal CT results between pre- and post-SAE

Figure 4 and Table 5 present the results of Doppler ultrasound before and after SAE. Altogether, 7 patients in the plug group (63.64%) and 3 patients in the coil group (75.00%) had high PV flow > 250 mL/min/100 g graft liver weight before SAE (plug: 279.7 ± 85.4 mL/min/100 g; coil: 294.2 ± 112.8 mL/min/100 g). Regardless the patients underwent SAE using either AVP or coil, PV velocity and PV flow volume were reduced in both groups (plug/coil; $P = .002/.006$; $P < .001/P = .006$). Also, the ultrasound of 2 groups demonstrated improvement in HA perfusion with increased HA peak systolic velocity (plug/coil; $P < .001/P = .002$), decreased HA pulsatility index (plug/

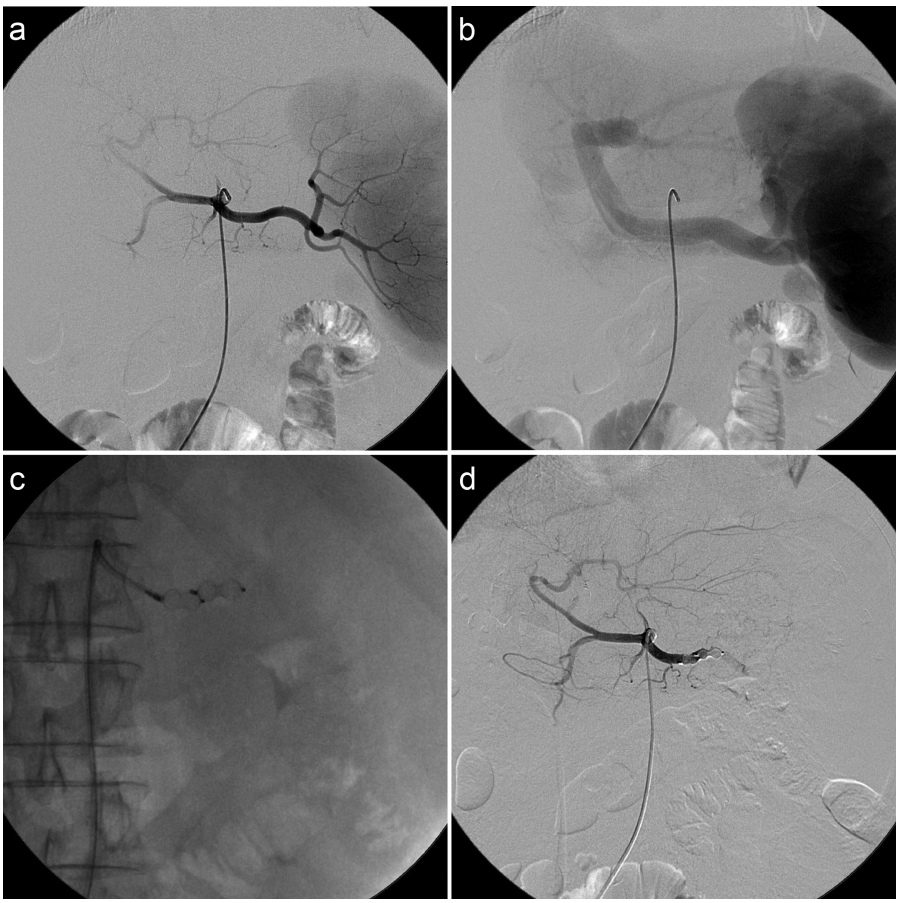

Figure 1. Angiogram of splenic artery embolization using Amplatzer vascular plugs. Pre-embolization angiogram demonstrated **(a)** splenomegaly, poor hepatic artery inflow, and **(b)** prominent portal vein. **(c)** Embolization of the splenic artery using 2 Amplatzer vascular plugs. **(d)** Post-embolization angiogram demonstrated successful occlusion of the splenic artery and improved hepatic artery inflow compared with the **(a)** initial angiogram.

coil; $P < .001$/$P = .012$) and decreased HA resistive index (plug/coil; $P = .005$/.026) in 1-3 months after SAE. However, PV diameter only decreased significantly in the plug group ($P = .045$). Compared to the abdominal CT before SAE with 6 months after SAE (Figure 3 and Table 4), significantly decreased splenic volume ($P = .012$), spleen/liver ratio ($P = .012$), and increased liver volume ($P = .012$) were only noted in the plug group. Only 1 patient in the coil group and 3 patients in the plug group did not undergo post-SAE abdominal CT due to individual reasons.

Outcomes and clinical follow-up

Five patients in the plug group and 3 quarters of all patients in the coil group were essentially well and asymptomatic after embolization (Table 3). In the plug group, 3 patients experienced tolerable abdominal pain or nausea/vomiting, and 1 patient had mild pancreatitis. However, the symptoms subsided in 3 days after SAE. Two patients in the plug group and 1 patient in the coil group had asymptomatic partial splenic infarction detected on follow-up CT scans, although no incidence of sepsis or splenic abscess occurred during 6 month follow-up. No significant differences were observed in hospital stays after SAE between the 2 groups ($P = .483$). Two patients in the plug group and no patients in the coil group died after SAE. One patient died from multiorgan failure within 30 days of the procedure, which was not SAE-related. One patient died from heart failure 1 year after the procedure. Platelet ($P = .008$) and white blood cell counts ($P = .026$) were elevated within 7 days after SAE in the plug group (Table 4). In the coil group, no significant differences between before and after SAE were found in hematologic parameters such as white blood cells and platelets (Table 4).

Discussion

In the present study, all patients underwent successful occlusion of the splenic artery after SAE, either by AVP or coil embolization techniques. AVP and coil embolization both significantly reduced PV flow, improved HA inflow, HA resistive index, and HA pulsatility index, and decreased ascites volume. The benefits of AVP embolization included shorter procedure time and improvement of pancytopenia due to secondary hypersplenism. No significant differences were found between the 2 groups in the length of hospital stay or complications such as splenic infarction, pancreatitis, or sepsis.

Limited clinical data are available in the literature regarding SAE for IA using AVP versus coil after LDLT. SAE was reported previously to be a safe and effective treatment for IA after liver transplantation.[5,7-10] Embolization has been described using a variety of embolic agents, such as coils, plugs, liquid embolic agents, and sclerosants. However, the choice for each patient is based on operator preference, and no clear consensus exists on the therapeutic efficacy and outcomes of SAE between various techniques for treating IA in LDLT patients. To the best of our knowledge, the present study is the first to report the efficacy of SAE using AVP and coil in IA treatment after LDLT. The present results showed that ascites decreased more quickly and effectively in the plug group than in the coil group and also significantly reduced spleen volume and improved pancytopenia in the plug group.

Ascites is a common finding in the cirrhotic liver with portal hypertension and splenomegaly. After transplantation, portal hypertension should be relieved by replacing the cirrhotic liver with a normal liver. However, splenic circulation is not regulated immediately.[1,2] Because the splenic circulation may contribute as high as 60% of the portal flow,[16] portal hypertension from splenomegaly with hyperdynamic splenic circulation after transplantation becomes the main risk factor for IA. Persistent ascites after liver transplantation may cause several complications such as subsequent graft failure, renal failure, peritonitis, and prolonged hospital stay.[2]

In the present study, both groups had a high prevalence of post-transplant portal hyperperfusion (63.64% in the plug group and 75.00% in the coil group). A previous review article reported that SAE was performed to prevent sequelae in patients with portal hypertension and treat splenic artery steal syndrome in liver transplant

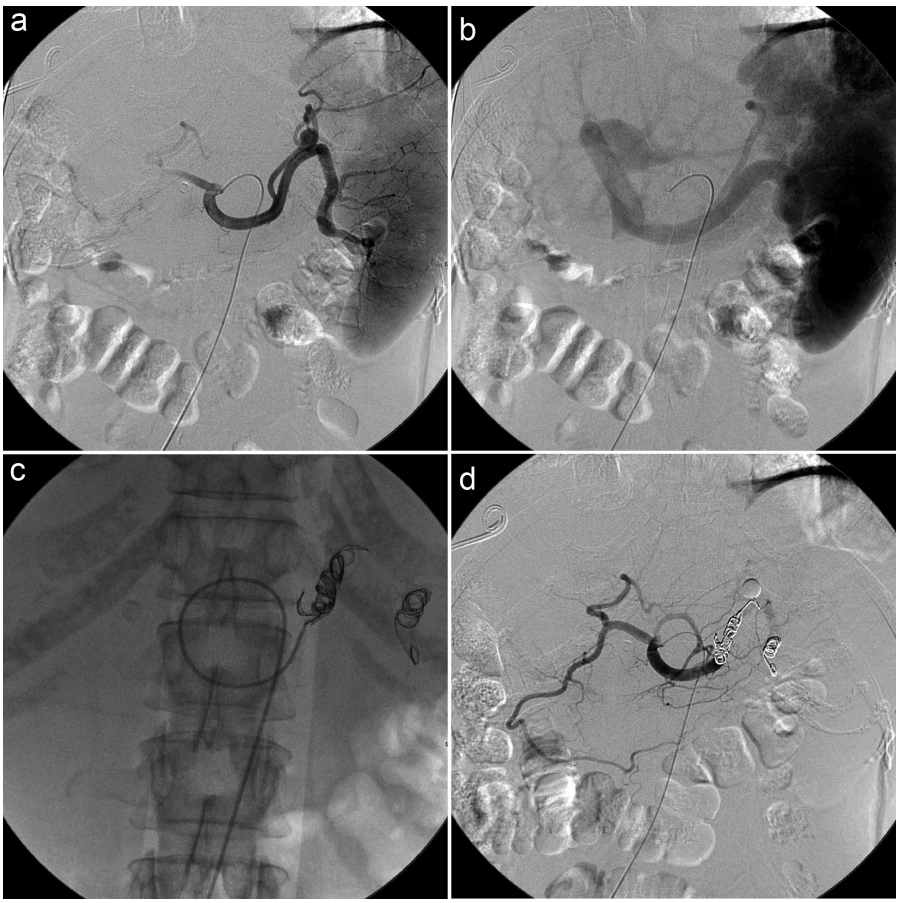

Figure 2. Angiogram of splenic artery embolization using coils. Pre-embolization angiogram demonstrated (**a**) splenomegaly, poor hepatic artery inflow, and (**b**) prominent portal vein. (**c**) Embolization of the splenic artery using 2 Amplatzer vascular plugs. (**d**) Post-embolization angiogram demonstrated successful occlusion of the splenic artery and improved hepatic artery inflow compared with the (**a**) initial angiogram.

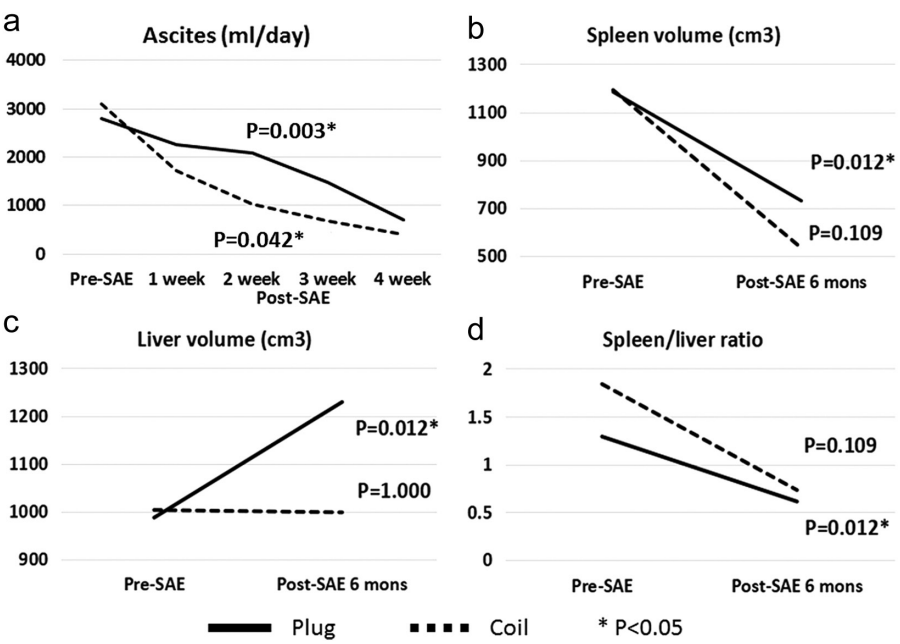

Figure 3. Effectiveness of splenic artery embolization in (**a**) reducing intractable ascites, (**b**) spleen volume, (**d**) spleen/liver ratio, and (**c**) increasing liver volume.

recipients.[16] The hepatic arterial buffer response balances the HA flow and PV flow to maintain a stable total blood flow to the liver. Increased PV flow mediates HA vasoconstriction through the hepatic arterial buffer response.[17] The HA hypoperfusion caused by portal hypertension may be due to increased splenic outflow. Thus, occluding the splenic artery may decrease portal flow and improve liver hypoperfusion.[18,19] Results of the present study reveal that patients who underwent the AVP and coil embolization both had significantly decreased ascites volume, reduced PV hyperflow, and improved HA inflow. However, significantly increased HA resistive index and HA pulsatility index within 1 week (pre-SAE compared with post-SAE 1 week, $P = .004$) were only noted in the plug group. The improved HA inflow which indicated decreased portal pressure is faster in the plug group than in the coil group. It may be the main pathophysiology for earlier decrease of ascites with AVP embolization. Furthermore, the significantly decreased PV diameter, splenic volume, spleen/liver ratio, and increased liver volume after SAE was only seen in the plug group. This suggests that the AVP and coil both can be used as embolization devices for SAE, but AVP embolization may be more efficient compared with coil embolization. Furthermore, SAE is used to improve hematologic parameters (i.e., pancytopenia, thrombocytopenia, leukopenia, or anemia) in patients with hypersplenism.[16] Improvement of pancytopenia was an additional benefit of AVP embolization in the present study.

An important attribute of favorable embolic agents is that they can lead to significantly reduced procedure time and, subsequently, decreased radiation dose. In the present study, the average procedure time of AVP embolization was significantly shorter than that of coil embolization. Occluding a large caliber and high-flow splenic artery with multiple coils is a particularly time-consuming procedure. Although a single plug is often costlier than a single coil, fewer AVPs are usually needed to embolize the splenic artery. Even though the cost analysis was not performed in the present study, there was no significant difference between the procedural material charges of AVP or coil embolization.

Large AVPs (≥10 mm) require the use of at least a 5 Fr angiocatheter and a 5 Fr angiosheath. The intraoperative limitations

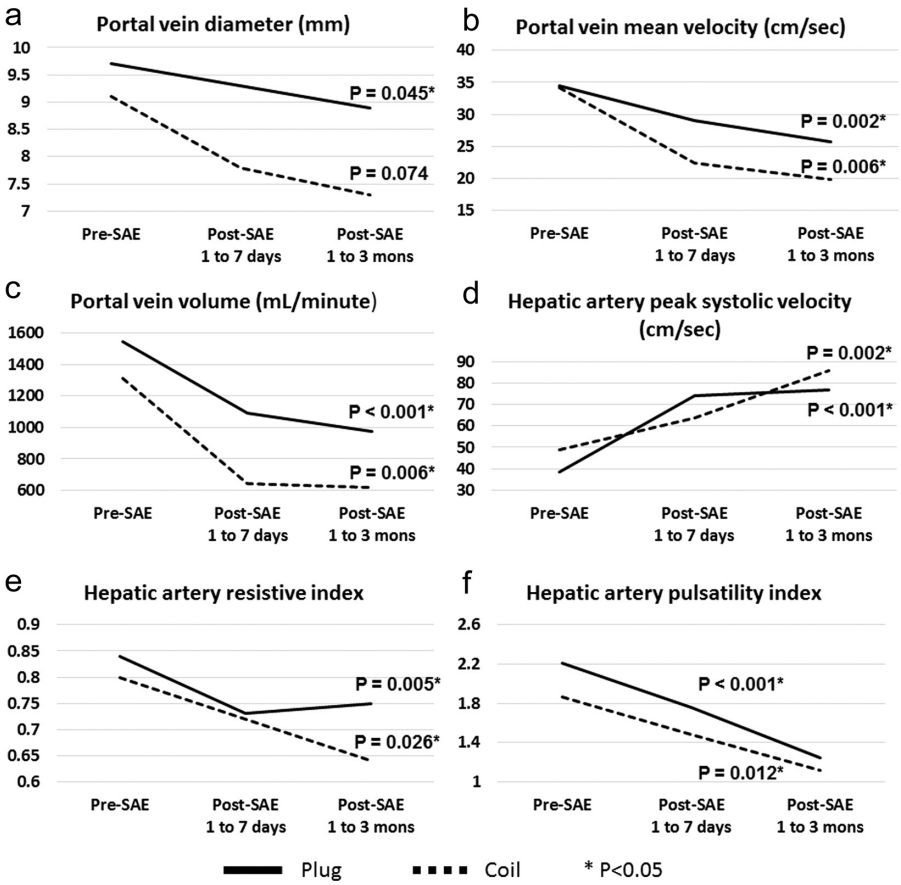

Figure 4. Effectiveness of splenic artery embolization in **(a)** reducing portal vein diameter, **(b)** portal vein mean velocity, **(c)** portal vein volume, **(e)** hepatic artery resistive index, **(f)** hepatic artery pulsatility index, and **(d)** increasing hepatic artery peak systolic velocity.

of AVP embolization include difficulty placing the device at the tortuous vessel and requiring the use of a 5-6 Fr angiocatheter rather than the use of a 4 Fr angiocatheter required in coil embolization.[20] Establishing access with larger angiocatheters in small caliber and marked tortuous vessels can be technically challenging and may occasionally be unsuccessful. Additionally, the use of a larger sheath to deliver the embolization device may result in an increased rate of vascular access-site complications. But there was no vascular access-site complication such as femoral arterial pseudoaneurysm, arteriovenous fistulas, and arterial dissection in the present study that occurred in the 2 groups. In a previous study,[21] placement of the AVP had a high success rate and a low failure rate.

The advantage of coil embolization is that microcatheters can be used if a 4 Fr angiocatheter cannot be used to establish access in a vessel with a small caliber.[22] The main disadvantage of coil embolization is accuracy of the coil placement. Coil migration may extend into the intrasplenic arterial branches, which may increase the potential for further splenic infarction.[22,23] The ability to reposition and resheath the AVP before release makes it possible to achieve precise deployment once the delivery sheath is placed. This is particularly important in high-flow vessels where there is risk of distal migration of coils.[13,21] A previous systematic review and meta-analysis[23] revealed that distal embolization at small arterial branches within the splenic parenchyma may lead to splenic infarction or increase the risk of splenic abscess formation. Proximal embolization at the main splenic artery may decrease the risk of infarction because the rich network of collateral circulation from the left gastric, gastroepiploic arteries, pancreatic, and omental branches enters the spleen. Thus, the accuracy of device placement is important to reduce the incidence of distal migration of the device and bypass the dorsal pancreatic artery and the great pancreatic artery. However, no misplaced AVP or coil, non-target embolization, or device migration occurred in the present study. Also, no significant differences were found between the 2 groups in length of hospital stay or in complications such as sepsis, splenic infarction, splenic abscess, or pancreatitis.

GRSSR is a predictive factor for measuring the contribution of splenic circulation to portal flow. Low GRSSR (<0.6) may predict the development of post-transplant graft hyperperfusion,[5,6] and a low GRWR (<0.8) may be a risk factor for post-transplant complications that cause ascites such as small-for-size syndrome.[5] In the present study, 2 patients in the plug group (18.18%) and no patients in the coil group had low GRWR values. However, 9 patients in the plug group (81.82%) and 1 patient in the coil group (25.00%) had low GRSSR values (<0.6). These results may suggest that patients with a low GRSSR are more likely to develop portal hypertension, as noted previously,[5] and may determine the need for modulation of portal flow. Thus, GRSSR can be a valuable predictive factor for post-LDLT IA and can help determine whether modulation of portal flow is required. SAE using AVP and coil provide effective and safe methods for managing patients with IA after LDLT. AVP embolization may be more efficient than coil embolization, including quicker and more effective reductions in ascites volume and the advantages of shortened procedure time and improvement of pancytopenia.

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Conflict of interest disclosure

The authors declared no conflicts of interest.

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