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INTERVENTIONAL RADIOLOGY

ORIGINAL ARTICLE

Feasibility and mid- to long-term results of endovascular treatment for portal vein thrombosis after living-donor liver transplantation

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PURPOSE
We aimed to evaluate mid- to long-term results of endovascular treatment for portal vein thrombosis (PVT) after living-donor liver transplantation (LDLT).

METHODS
Thirty cases (14 males, 16 females; age range, 0.67–65 years) who underwent endovascular treatment including thrombolysis, angioplasty, stent placement, and/or collateral embolization for PVT after LDLT from 2001 to 2017 were retrospectively reviewed. Clinical and procedural data were collected and analyzed regarding the patency of the PVT site at the last follow-up date (PVT-free persistency) using Log-rank test. Results were considered statistically significant at $p < 0.05$.

RESULTS
Median follow-up was 120 months. The technical success rate was 80% (n=24). Patency rates at 1 week and 1, 3, 6, 12, 36, and 60 months were 73%, 59%, 55%, 51%, 51%, 51%, and 51% for primary patency and 80%, 70%, 66%, 66%, 66%, 61%, and 61% for assisted patency after secondary endovascular treatment. PVT-free persistency rates regarding the subgroups were as follows: children under 12 years vs. adults, 50% vs. 68% ($p = 0.42$); acute vs. nonacute, 76% vs. 46% ($p = 0.10$); localized vs. extensive, 90% vs. 50% ($p = 0.035$); transileocolic approach vs. percutaneous-transhepatic approach, 71% vs. 54% ($p = 0.39$); and thrombolysis-based treatment vs. non-thrombolysis-based treatment, 71% vs. 44% ($p = 0.12$), respectively. Among technically successful cases, PVT-free persistency rate was 94% for those with hepatopetal flow in the peripheral portal vein vs. 17% for those without hepatopetal flow ($p < 0.001$). The only major complication occurring was pleural hemorrhage (n=1). Minor complications (i.e., fever) occurred in 18 patients (60%).

CONCLUSION
In conclusion, mid- to long-term portal patency following endovascular treatment was approximately 50%–60% in PVT patients after LDLT. PVT site patency over three months after the first endovascular treatment, localized PVT, and hepatopetal flow in the peripheral portal vein were identified as key prognostic factors for mid- to long-term portal patency.

Portal vein thrombosis (PVT) is a vascular complication of living-donor liver transplantation (LDLT), with an estimated incidence of up to 4% (1, 2). The risk of vascular complications, including PVT, is higher in LDLT compared with conventional deceased-donor liver transplantation, because of the smaller vessels, insufficient vessel length for reconstruction, neointimal proliferation, and higher risk of twisting and kinking of the vascular pedicle (3) due to smaller graft size than in deceased-donor liver transplantation. PVT after LDLT can lead to graft failure and the need for retransplantation or death (2), making immediate treatment crucial.

Endovascular-based treatment is one option for treating PVT. The utility of target-focused thrombolysis, balloon angioplasty, and stent placement to restore portal flow has been reported previously (4–10). However, the efficacy of endovascular treatment after LDLT has only been presented in some case reports (11, 12) and the mid- to long-term outcomes remain unclear.

The purpose of this study was to evaluate the technical success, feasibility, and mid- to long-term results of endovascular treatment for PVT after LDLT in our institution.

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Methods

Patients

This study was approved by the institutional review board (decision/protocol No. R1225), and informed consent was waived based on the retrospective nature of the study. We retrospectively reviewed 982 consecutive cases who underwent LDLT at our institution from January 2001 to May 2017. We identified 30 cases (14 males, 16 females; mean age, 35 years; age range, 8 months to 65 years) of PVT that occurred post-LDLT and for which endovascular treatment was performed. Age distribution of patients was 8 infants/children <12 years (median, 1.8 years; range, 8 months to 10 years), and 22 adults ≥18 years old (median, 40 years; range, 19–65 years) (Table 1). No patients within the age range of 13–17 years were encountered. The median interval between LDLT and endovascular treatment was 712 days (range, 2–5711 days). The main causes of PVT were acute inflammatory changes in the hepatobiliary system after LDLT (n=4), decreased portal venous flow secondary to rejection, dehydration/bleeding, and collateral venous development (n=9), recurrence of the primary disease (n=6), and unknown (n=11). No organs from executed prisoners were used.

Diagnosis of PVT and portal occlusion

Serial Doppler ultrasonography (US) was performed in all cases every day after LDLT until hospital discharge and at all outpatient visits. When changes in the velocity of the portal flow or any abnormality was detected on US, such as weakening or lack of intrahepatic portal flow (13) or detecting defects in the portal vein, a precise diagnosis was obtained using contrast-enhanced computed tomography (CECT).

Since changes in the pathological features of the thrombus from acute to chronic finalize at 3–5 weeks and are accompanied by development of fibrotic changes, calcification, and vessel stenosis (14–17), we defined shorter and longer intervals between the last PVT-free date and endovascular treatment as acute PVT (aPVT) if it occurred ≤4 weeks post last PVT-free date and nonacute PVT (nPVT) if it occurred >4 weeks post last PVT-free date (aPVT, n=17; nPVT, n=13) (Table 1).

The location and extent of PVT was evaluated using CECT and catheter angiographic images. PVT was termed "localized" if confined to the portal trunk and "extensive" if it extended beyond the lobar branch (localized PVT, n=10; extensive PVT, n=20) (Table 1).

Interventional techniques

The endovascular procedure comprised the portal approach and treatment. The portal approach included percutaneous transhepatic (PH) or transileocolic venous (Ic) approaches, while treatments included thrombolysis, balloon angioplasty, stent placement, and collateral embolization, respectively (Fig. 1). Decisions on treatment methods were individualized by the interventional radiologists based on several parameters during the endovascular treatment: age, symptoms, PVT type, and location. Among 13 interventional radiologists with experience of 10–30 years in our institution, three or more operators participated in each endovascular treatment. Patients were heparinized with a first bolus injection of heparin sodium 100 IU/kg, followed by a bolus injection of 20 IU/kg every hour during the procedure.

Percutaneous transhepatic approach

The PH approach was performed in the angiography suite under local anesthesia (n=13). A peripheral intrahepatic portal branch was punctured with an 18- or 21-gauge puncture needle (Hanako Medical) under US guidance. A 7 F sheath (Brite Tip Sheath Introducer, Cardinal Health) was then placed using a stiff guidewire (Amplatz Support Wire Guide, Cook Medical or PTCD Kit, Sheenman) followed by intrahepatic-portography to evaluate thrombus extent and patency and flow in both the involved and downstream intrahepatic por-

Main points

- Endovascular treatment was effective for approximately half of the patients who developed portal vein thrombosis (PVT) after living-donor liver transplantation (LDLT) to achieve mid- to long-term portal venous patency.
- When obtaining mid- to long-term patency of the portal vein, key factors were post-therapeutic lesional patency, existence of hepatopetal flow in the peripheral portal vein (PV) after endovascular treatment, and maintaining them for more than three months after the endovascular treatment.
- Localized PVT was also suggested as another factor to achieve mid- to long-term portal venous patency.

Table 1. Patients' background and data regarding PVT-free persistency

	Total (n=30)	Achieved PVT-free persistency (n=19)	No PVT-free persistency (n=11)	*p*
Age group (age range)				
Infants/children	8 (8 mo to 10 yrs)	4 (1–10 yrs)	4 (8 mo to 7 yrs)	0.42
Adults	22 (19–65 yrs)	15 (19–64 yrs)	7 (24–65 yrs)	
Gender				
Male	14	10	4	0.39
Female	16	9	7	
PVT type				
Acute PVT	17	13	4	0.10
Nonacute PVT	13	6	7	
Location				
Localized PVT	10	9	1	0.035*
Extensive PVT	20	10	10	
Approach				
Ic only	17	12	5	0.39
PH only	13	7	6	
Treatment				
Thrombolysis-based	21	15	6	0.12
Non-thrombolysis-based	9	4	5	

PVT, portal vein thrombosis; Ic, transileocolic venous approach; PH, percutaneous transhepatic approach.
* $p < 0.05$.

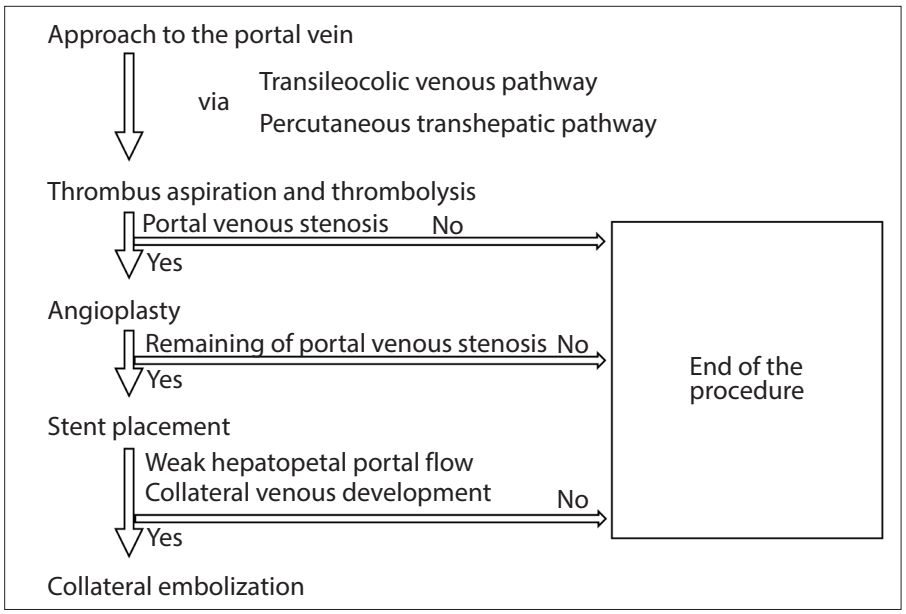

Figure 1. Flow chart showing the basic strategy for the procedures and techniques used in this study.

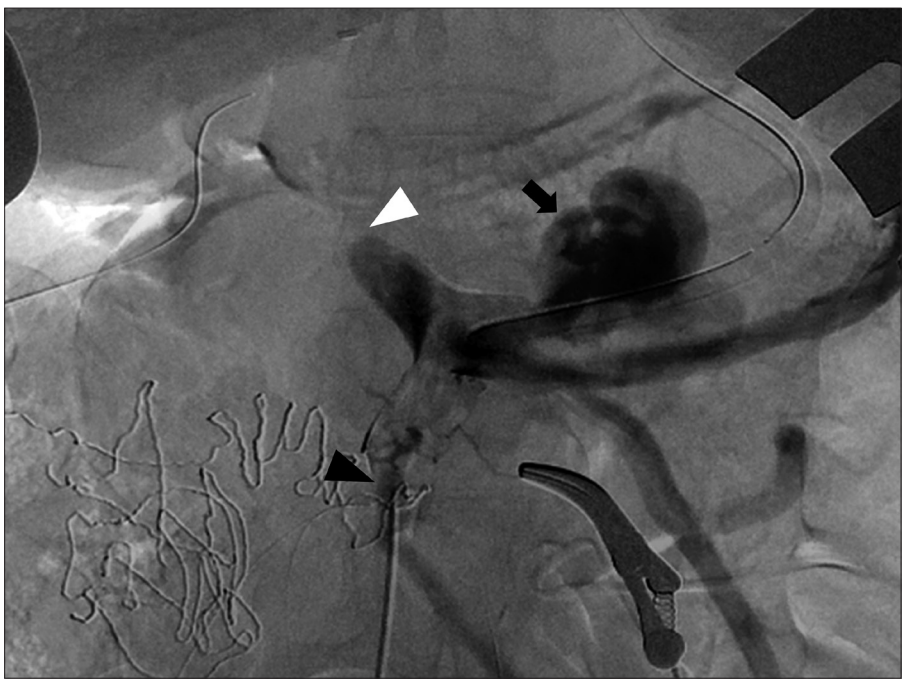

Figure 2. A 50-year-old woman with portal vein thrombosis on the second day after living-donor liver transplantation of the left lobe. Portography via the transileocolic approach demonstrates occlusion at the portal trunk *(white arrowhead)* with opacification of the superior mesenteric vein *(black arrowhead)* and a collateral vein *(black arrow)*.

tal vein (PV). Then, a 0.035-inch guidewire (Radifocus Guidewire M) was carefully advanced through the thrombus, followed by placement of a 4 F or 5 F catheter through the thrombus to perform portography for the examination of the thrombus extent and patency of the portal trunk.

After all the therapeutic procedures has been finished, including thrombolysis, balloon angioplasty, stent placement, and collateral embolization, we used microfibrillar collagen hemostat (Avitene, Zeria Pharmaceuticals) to seal the tract after the 7 F sheath removal.

Transileocolic approach

The Ic approach was performed in the operation room with a mobile C-arm or in the hybrid operating room with a fixed C-arm (n=17). Under general anesthesia, surgeons first performed laparotomy to place a 7 F sheath (Brite Tip Sheath Introducer, Cardinal Health) in an ileocolic venous branch, followed by portography to examine the thrombus extent, patency of the portal trunk, and intrahepatic portal flow (Fig. 2). A guidewire, usually a 0.035-inch (Radifocus Guidewire M, Terumo), was carefully advanced through the thrombus, followed by placing a 4 or 5 F catheter within or through the thrombus.

After all the therapeutic procedures were finished, including thrombolysis, balloon angioplasty, stent placement, and collateral embolization, the sheath was removed with ligating the insertion site, and the abdomen was closed. When the interventional radiologists and surgeons decided to leave an infusion catheter for further intra-portal injection of urokinase, the 7 F sheath was replaced by a 4 or 5 F catheter using a 0.035-inch guidewire (Radifocus Guidewire M, Terumo) followed by surgical closure of the abdomen. This catheter was later removed surgically after completion of treatment.

Thrombolysis

There were 21 cases that underwent thrombolysis. Thrombolysis was performed first with a thrombus-aspiration catheter (Eliminate, Terumo or HYDROLYSER, Cardinal Health) or a multipurpose guiding catheter to remove the thrombus. A standard diagnostic angiographic catheter (standard catheter) or chemical spray catheter with multiple side holes (spray catheter, Fountain Infusion System, Sheenman) was then placed into the thrombus, followed by intracatheter urokinase injection (120 000–360 000 IU) (Fig. 3) in all cases.

When the patient was left with an infusion catheter inside PV after the endovascular treatment (n=15), 60 000–240 000 IU/day of urokinase was injected through the infusion catheter. The median period of infusion catheter placement was 15 days (range, 10–29 days).

Angioplasty

Angioplasty was performed with a non-compliant balloon catheter (NSE PTA, Goodman or POWERFLEX, Cardinal Health) to expand portal venous stenosis if present. The size and length of the balloon was decided based on the vascular diameter (size, 6–10 mm; length, 2–4 cm). Dilation was performed twice with balloon inflation at nominal atmospheric pressure in the stenotic segment lasting 60 seconds each time. A total of 13 cases underwent angio-

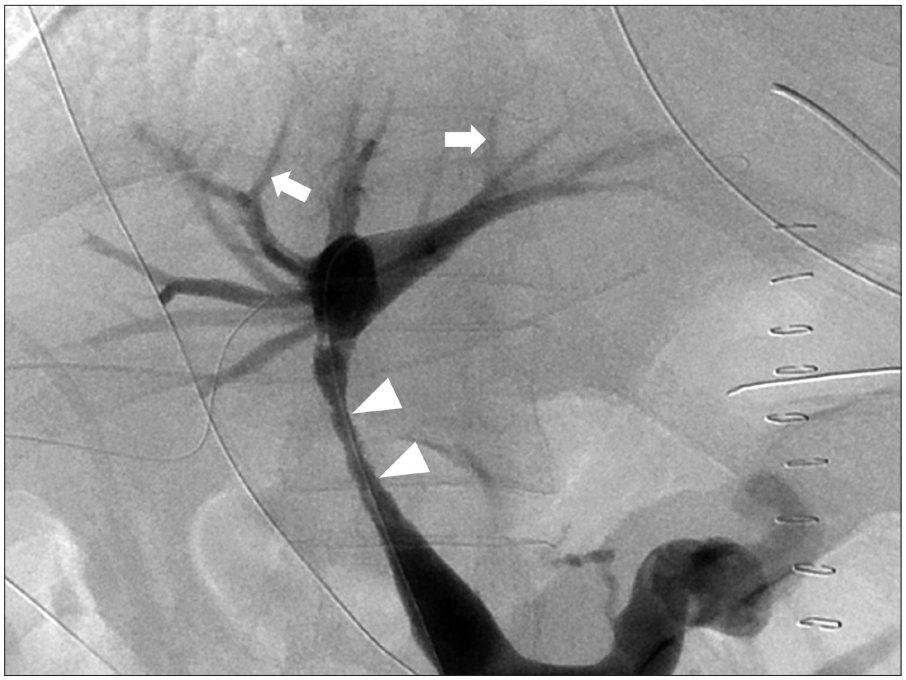

Figure 3. Portography after thrombolysis in the patient in Fig. 2 demonstrates the opacification of a tertiary branches of the portal vein *(arrows)* with moderate stenosis of the portal trunk *(arrowheads)*.

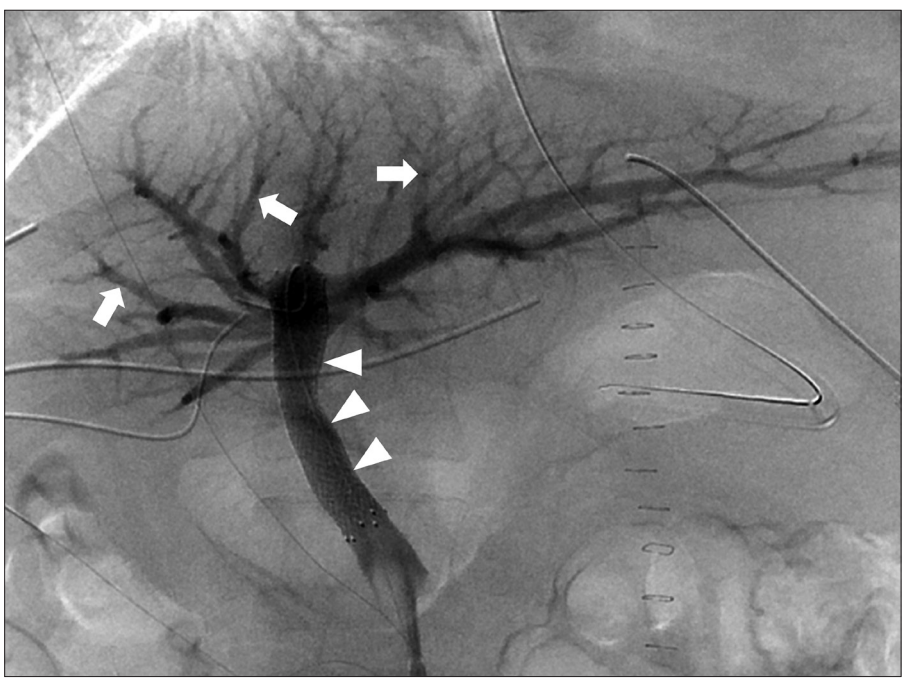

Figure 4. Portography after stent placement for residual portal stenosis in the patient in Fig. 2 demonstrates improvement in the portal trunk diameter with the stent *(arrowheads)* and good opacification of tertiary *(arrows)* and smaller portal venous branches.

plasty alone, combined with thrombolysis in 7 cases.

Stent placement

Stent placement was first performed by placing a self-expanding nitinol vascular stent (SMART, Cardinal Health) (Fig. 4). A balloon catheter was then used to bond the stent to the vessel wall. The size and length of the stent and the balloon were decided based on the vascular diameter (size, 8–14 mm; length, 4–6 cm). Stent placement was performed in 6 cases, all with the combination of thrombolysis and angioplasty.

Collateral embolization

Embolization of portosystemic collaterals using coils (10–16 mm × 40–60 cm Penumbra Smart Coil, Boston Scientific Corporation) or balloons (Attendant, Terumo) with ethanolamine oleate (Oldamine, Fuji Chemical) was performed if a case with portosystemic collaterals demonstrated poor improvement in hepatopetal flow of the portal trunk after thrombolysis, angioplasty, or stent placement.

Postintervention follow-up

All cases underwent follow-up Doppler US until hospital discharge and at scheduled outpatient visits. When any findings suggestive of PVT emerged, a precise diagnosis was obtained with CECT. Dynamic CECT of the liver was also performed regularly at 3- to 6-month intervals. Antithrombotics/anticoagulants (warfarin potassium 1.0–3.0 mg or edoxaban tosilate hydrate 30 mg) were administered indefinitely. The dose was adjusted by surgeons to maintain an international normalized ratio for prothrombin time of approximately 1.5–2.0 based on each patients' clinical condition (13). Until the successful adjustment of the antithrombotics/anticoagulants, patients received continuous heparin perfusions of 200 IU/kg/day, after the intervention.

Secondary intervention was performed when recurrence or enlargement of PVT occurred. Secondary treatment was performed in 8 cases (aPVT, n=5; nPVT, n=3) with a median interval of 22 days (range, 3–120 days) from the first endovascular treatment.

Data analysis

Technical success was defined as completion of all procedures performed and visual improvement in the vessel lumen diameter and hepatopetal flow at the PVT site between pre- and post-therapeutic portography. Patency was defined as confirmation of the contrast opacification of vessel lumen at the PVT site on portography or CECT. Assisted patency includes the patency after secondary treatment when performed. PVT-free persistency (PfP) was defined as the patency at the last follow-up date, and it was classified as "yes" if the PVT site was patent at the last follow-up date. If the PVT site demonstrated no patency at the last follow-up date, PfP was classified as "no". Peripheral portal opacification was defined as the visualization of tertiary PV branch by hepatopetal flow on post-therapeutic portography.

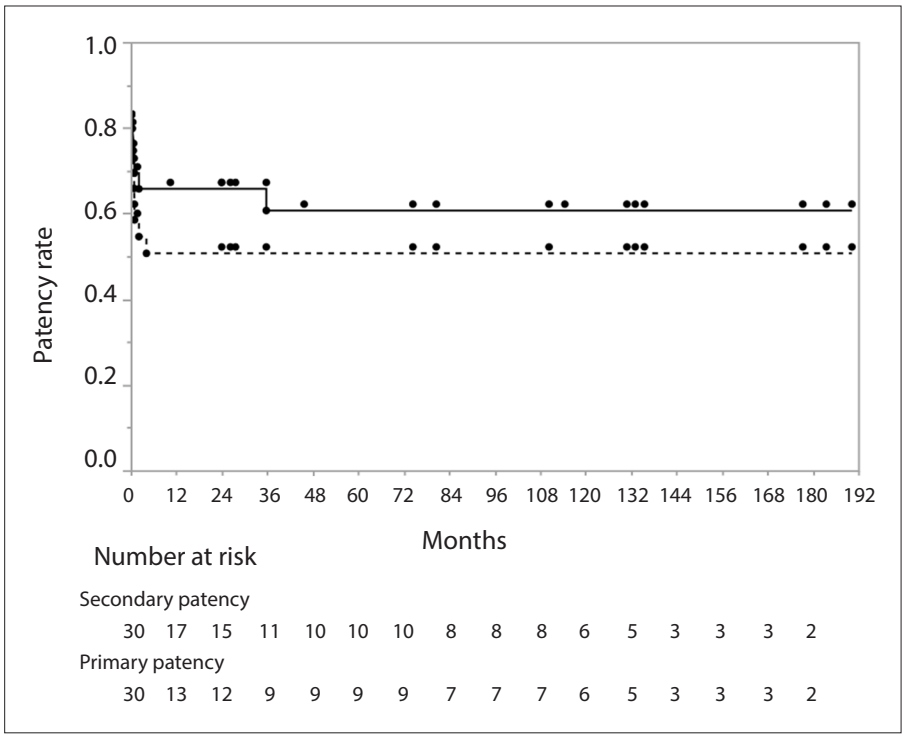

Figure 5. Kaplan-Meier plot of primary patency and assisted patency: patency rate *(dotted line)*, assisted patency rate *(solid line)*, and censored cases *(small dots)*.

Table 2. Opacification of tertiary portal branches and PVT-free persistency in patients with technical success

	Total (n=24)	Achieved PVT-free persistency (n=18)	No PVT-free persistency (n=6)	*p*
Opacification of tertiary portal branches				
Yes	18	17	1	< 0.001*
No	6	1	5	

PVT, portal vein thrombosis.
*$p < 0.05$

Primary patency rates and assisted patency rates at 1 week and 1, 3, 6, 12, 36, and 60 months after treatment were analyzed with Kaplan-Meier plots using JMP pro 13® software (SAS Institute). The number and frequency of cases who achieved PfP were aggregated and expressed in fractional form and percentage among subcategories including age (infants/children or adults), PVT type (aPVT or nPVT), location (localized or extensive), approaching pathway (PH only or Ic only), therapeutic technique (thrombolysis-based or non-thrombolysis-based), and peripheral portal opacification (yes or no). Therapeutic technique was divided into two groups, thrombolysis-based or not, based on performance of thrombolysis. Non-thrombolysis-based treatment included angioplasty only and angioplasty with collateral embolization. PfP regarding peripheral portal opacification was analyzed among technically successful cases, while other subcategories were analyzed among all 30 cases with Log-rank test using JMP pro 13® software (SAS Institute). Results were considered statistically significant at $p < 0.05$.

All complications were recorded based on previously established criteria (18).

Results

Median clinical follow-up was 120 months (interquartile range, 36–151 months). Technical success was achieved in 24 cases (80%). Primary patency rates at 1 week and 1, 3, 6, 12, 36, and 60 months after the treatment were 73%, 59%, 55%, 51%, 51%, 51%, and 51%, respectively. Secondary treatment was performed in 8 cases (age range, 8 months to 57 years); assisted patency rates at 1 week and 1, 3, 6, 12, 36, and 60 months after the first treatment were 80%, 70%, 66%, 66%, 66%, 61%, and 61%, respectively (Fig. 5).

In the subgroup analysis, the number of cases who achieved PfP were: 4 children (50%) vs. 15 adults (68%) ($p = 0.42$); 13 aPVT (76%) vs. 6 nPVT (46%) ($p = 0.10$); 9 localized PVT (90%) vs. 10 extensive PVT (50%) ($p = 0.035$); 12 Ic only approach (71%) vs. 7 PH only approach (54%) ($p = 0.39$); and 15 thrombolysis-based treatment (71%) vs. 4 non-thrombolysis-based treatment (44%) ($p = 0.12$) (Table 1).

Among 24 technically successful cases, peripheral portal opacification was achieved in 18, and PfP was achieved in 17 of 18 cases (94%) (Table 2), while PfP was available in only one of six cases with no peripheral portal opacification (17%) ($p < 0.001$).

Secondary treatment was performed in 8 cases (aPVT, n=5; nPVT, n=3) with a median interval of 22 days (interquartile range, 11–56 days). These cases underwent secondary thrombolysis-based treatment for PVT enlargement using the same approach as used with the first treatment (PH, n=5; Ic, n=3). PfP was achieved in six cases (PH, n=3; Ic, n=3).

Collateral embolization was performed in two cases: one-stage coil embolization in one patient, and two-stage balloon-occluded retrograde transvenous obliteration in the other.

Complications occurred in 19 cases, as fever (grade 1, n=8), elevation of hepatobiliary enzymes (grade 1–2, n=8), nausea (grade 1, n=2), vomiting (grade 1, n=2), abdominal distension (grade 1–2, n=2), abdominal pain (grade 2, n=1), and grade 3 pleural hemorrhage (n=1) necessitating ligation of the bleeding vessel (Table 1).

Five patients died of causes unrelated to intervention; retransplantation for recurrence of the primary disease was seen in two cases; and surgical reanastomosis of the PV was seen in one case. Three cases were lost to follow-up, due to transfer to another hospital for patient-specific circumstances.

Discussion

PVT after LDLT may result in graft failure, need for retransplantation, or death (2). Prompt treatment is vital. In our study, mid- to long-term patency was obtained in approximately 50%–60% of cases during

the first 5 years of follow-up. Obtaining and maintaining the patency of the PVT site and hepatopetal flow in the peripheral PV were considered as the two key factors to achieve mid- to long-term patency in this study. A decrease in hepatopetal flow in the peripheral PV causes stagnation and turbulence of portal venous flow, resulting in PVT, which corresponds to one of the Virchow's triad (19). In this study, PfP was achieved in 94% of cases with hepatopetal flow in the peripheral PV, while PfP was available in only one case with no hepatopetal flow in the peripheral PV. Even if a case developed PVT recurrence, mid- to long-term patency was obtained when hepatopetal flow in the peripheral PV could be secured by the second treatment. There were some cases to support this point; one patient, who underwent a collateral portosystemic shunt embolization to decrease collateral flow and increase hepatopetal portal flow in the peripheral PV, achieved mid- to long-term patency. In other cases, even if hepatopetal flow in the peripheral PV was achieved, it was lost later due to hepatic parenchymal portal hypertension caused by recurrence of the primary disease or rejection, and mid- to long-term patency was not achievable. PVT recurrence occurred and primary patency rates decreased within three months, mostly within the first month, after endovascular treatment. Furthermore, primary patency rates were unchanged after three months post treatment. This suggests that preserving the hepatopetal PV flow during the first three months after endovascular treatment is paramount to achieve mid- to long-term PV patency. Moreover, since assisted patency rates were higher than primary patency rates, performing additional endovascular treatment to preserve PV patency and its hepatopetal flow in cases of PVT recurrence should be considered to achieve mid- to long-term PV patency.

Localized PVT was another factor associated with improved PfP. By definition, localized PVT has two elements: shorter extent and proximal location. Higher efficacy for shorter thrombi agrees with previous reports of middle cerebral artery occlusion (20). Moreover, proximal location is considered to facilitate access to the PVT and greater hepatopetal portal flow from the larger space behind the PVT, once the occlusion is reversed.

Thrombolysis assumes a central role in the endovascular treatment of venous thrombosis. Its advantages over systemic lysis are well known, including direct delivery of thrombolytic agents into the thrombus at higher concentrations which is expected to yield higher efficacy, easier lysis of more peripheral thrombotic debris, reduced dose and duration of thrombolytic agents, and possible reductions in the risk of bleeding complications (21–23). After thrombus formation, organization of the thrombus and narrowing of the relevant vessel occur as the fibrous component increases (14, 24–28). Despite the presence of fibrous components in chronic PVT, thrombolysis plays an important role in shrinking the thrombus debris after peripheral travel and in preventing thrombus enlargement. Despite the importance of thrombolysis in the therapeutic strategy for PVT, especially in the acute phase, PfP rate was not significantly related with thrombolysis-based treatment or aPVT in this study. This lack of association depended on whether patients obtained sufficient peripheral portal flow. Obtaining enough peripheral portal flow is considered as an important factor in achieving successful thrombolysis. To support this point, we saw a statistically significant association between high PfP and obtaining sufficient peripheral portal flow. In this study, treatment for PVT using a transjugular intrahepatic portosystemic shunt was not performed; however, this approach may be more useful because it could improve hepatopetal portal vein flow (29).

Acute-to-chronic conversion will build up over time, making treatment more difficult and requiring further options during endovascular treatments such as angioplasty and stent placement. Angioplasty and stent placement would be effective when the PVT is induced by anastomotic portal venous stenosis. A risk of suture dehiscence during angioplasty in the early postoperative period has been suggested (30), although we encountered no patients who developed suture dehiscence despite a minimum interval from LDLT to endovascular treatment of 4 days. Some cases of portal venous stenosis induced by chronic PVT are not resolved with balloon angioplasty, because the wall flexibility induces easy expansion and reversion of the PV wall by inflation and deflation of the balloon (4). Stent placement benefits these cases.

Generally, some technical difficulties are secondary to body size in infants and children, which limits the devices that can be used and the operator's ability when using small vascular access systems. Other differences between adults and children relate to preprocedure preparation, dose of contrast media, or angiography injection parameters (31, 32). Although PfP in infants and children was lower than in adults and not statistically significant, in our study, the *P* value was generally larger, suggesting a smaller influence of the age group on PfP than other factors, such as PVT type or location. New devices and tools have been developed recently in interventional radiology, and thus, differences in the success rate related to the operator's skill were smaller in our study, compared with previous devices.

Regardless of the risks of general anesthesia, difficulty in exposing vessels in the postoperative anatomy, and higher invasiveness, the Ic approach has advantages in terms of the certainty of portal catheterization (6, 7, 28, 33), and the avoidance of risks associated with the PH approach, which can easily damage the liver graft. The only severe complication in our study was pleural hemorrhage induced by the PH approach, which required surgical hemostasis. Treatment approach was selected according to the peripheral portal patency and PVT type; PH approach was usually performed in chronic PVT cases when the peripheral portal vein was patent, while Ic approach was used in all other cases. Installation of a hybrid operating room in our institution was a positive reason for selecting the Ic approach. Imaging devices in the hybrid operating room provide better image quality than the traditional C-arm alone, and angiography is easier to perform; therefore, we tended to select the Ic approach, regardless of the PVT type.

This study had some limitations. First, the sample size was small. Second, the study was not prospective in design. Third, heterogeneity was seen in the PVT conditions and details of treatment. Because the smallest number of patients in any group was one patient, separating the patients into different groups regarding their background conditions and techniques was difficult. Fourth, nPVT may also include aPVT, although we confirmed that all cases of nPVT represented chronic PVT by reviewing CT and US images. Fifth, we analyzed data for juvenile and adult cases together because of the small sample sizes and similarity in the endovascular therapeutic procedures and techniques between adult and pediatric patients, despite the differences in patients' body sizes and contrast media. Sixth, this study had a possibility of bias

because treatments for each case were set according to the individual condition. Seventh, although based on consensus of all interventional radiologists who participated in each patient's treatment, evaluation and comparison of portography between pre- and post-procedure was subjective, since there was no objective evaluation method available. Eighth, the follow-up period in some cases was short and might not be eligible to be classified as "long-term" because of the patient's death or discontinued follow-up from causes that were not related to the procedure. Ninth, some parameters including the PV pressure at pre- and post-endovascular treatment are lacking, since they were not available in the medical records of some patients.

In conclusion, we obtained mid- to long-term patency of the PV using endovascular treatment in approximately 50%–60% of patients with PVT after LDLT. Achieving patency and hepatopetal flow in the peripheral PV for localized PVT, and maintaining patency for more than three months after the endovascular treatment, may be key factors for achieving mid- to long-term patency of the PV.

Conflict of interest disclosure

The authors declared no conflicts of interest.

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