ABSTRACT

Purpose: To evaluate retrospectively the long-term outcome of percutaneous interventions for hepatic venous outflow obstruction (HVOO) after pediatric living donor liver transplantation (LDLT).

Materials and Methods: This study was approved by the Human Subjects Research Review Board at our institution. Between October 1997 and December 2012, 48 patients (24 boys, 24 girls; median age, 6 years) who had undergone LDLT were confirmed to have HVOO using percutaneous hepatic venography and manometry. All of the patients underwent percutaneous interventions, including balloon angioplasty with or without stent placement. Technical success, clinical success, patency rates, stent placement, major complications and over-all survival rate were evaluated.

Results: Technical success was achieved in 92 of 93 sessions (99.0%) and in 47 of 48 patients (97.9%), and clinical success was achieved in 41 of 48 patients (85.4%). During follow-up range, one to 182 months (median, 51.5 months), 28 patients were treated with a single session of balloon angioplasty and 20 who developed recurrent stenosis were treated with repeated percutaneous interventions. The rates of primary and primary-assisted patency at 1-, 3-, 5-, and 10-years after the balloon angioplasty were 64%, 57%, 57% and 52%, and 98%, 95%, 95% and 95% respectively. Of six patients

with stent placement, four had no recurrent HVOO after the stent placement, but two developed recurrent stenosis. The stent migrated to the right atrium in one patient.

Conclusion: Percutaneous interventions were effective treatments for HVOO after LDLT.

INTRODUCTION

Liver transplantation has been an effective treatment for the end stages of liver disease. [1, 2]. Recent advances in surgical techniques and immunosuppression have led to improved post-transplant outcomes, but vascular complications are still important causes of graft failure [3, 4]. Hepatic venous outflow obstruction (HVOO) is a rare complication, and the frequency of HVOO in orthotopic liver transplantation (OLT) is reported to be approximately 1.5 - 2.5% [5, 6]. Living donor liver transplantation has arisen as a means to address the continuing cadaveric donor shortage [2]; however, the rate of HVOO in LDLY has been reported to 2 - 9% [4, 7-11], higher than that with OLT owing to the shorter vascular pedicle, smaller anastomosis diameters, and the potential for a size mismatch in children [12]. Percutaneous interventions, including balloon angioplasty and stent placement, are accepted treatments in patients with HVOO because of the effectiveness and minimal invasiveness [12-16]. Balloon angioplasty is the accepted first therapeutic option for HVOO [10, 17], although there has been a report describing the effectiveness of primary stent placement for HVOO [18]. The purpose of our study was to retrospectively evaluate the long-term outcome of percutaneous interventions for HVOO after LDLT in pediatric patients in a single

institute.

MATERIALS and METHODS

The Human Subjects Research Review Board at our institution approved our protocol and retrospective study, and the board did not require informed consent. Informed consent regarding venography and interventional procedures was obtained from patients or the patient's parents.

Patient

Between October 1997 and December 2012, LDLT was performed in our department of surgery in 512 pediatric patients, (age < 18 years). Of the 512 patients 68 patients were suspected of having HVOO during postoperative follow-up and underwent venography and manometry. HVOO was clinically suspected with the following findings: (1) clinical symptoms, including ascites, pleural effusion and hepatomegaly; (2) Doppler US findings, including the disappearance of pulsatile hepatic venous flow, flattening of the hepatic venous wave, or weak Doppler-detectable blood flow (< 10cm/s); and (3) liver biopsy findings of congestion, hemorrhage, and necrosis around the central veins. Forty-eight of the 68 patients were confirmed to have HVOO using venography and

manometry. Our inclusion criteria for HVOO were: (1) greater than 50% stenosis (the diameter of stenosis / the diameter of a hepatic vein on the distal side) or (2) > 5mmHg pressure gradient across the stenosis between the distal hepatic vein and the right atrium [9, 12]. The characteristics of the 48 patients are shown in Table 1. We included 16 pediatric patients from our previous study [blinded reference]. All 48 patients underwent percutaneous interventions, including balloon angioplasty with or without stent placement. The age of the patients ranged from 2 months to 20 years (median, 6 years) at the first intervention. The interval between the liver transplantation and the first percutaneous intervention was 1 to 220 months (median, 18 months). One patient underwent the first percutaneous intervention in the acute period, 1 month after the LDLT; five patients received intervention in the subacute period, ranging 1 to 3 months after LDLT; and the other 42 patients received intervention from 4 to 220 months after LDLT.

Procedures

Four authors (blinded) with 5, 29, 5, and 17 years of experience in interventional radiology performed the procedures.

Percutaneous interventions were performed with general anesthesia in 32 patients and

with local anesthesia in 16 patients. The approach to the hepatic vein was transhepatic in 35 patients and transjugular in 13, and we adopted a transjugular approach in patients with ascites, coagulophathy, or those under 3 years. Balloon angioplasty was performed following venography and manometry with a 7.0-Fr percutaneous transluminal angioplasty catheter (Powerflex Plus; Cordis, Warren, NJ, U.S.A) with a balloon diameter of 6–10 mm and a length of 40 mm. The diameter of the balloon was the same as the vein on the hepatic side of the stenosis. The balloon was inflated 3 times for 60 seconds with an atmospheric pressure of 10 atm. Venography and manometry were then repeated to evaluate the effectiveness of the balloon angioplasty. Hemostasis was accomplished with manual compression, following sheath removal, in patients treated with the transhepatic approach without subsequent embolization of the transhepatic track.

Unlike in patients treated with the transhepatic approach, those treated with the transjugular approach were heparinized with 50 IU/kg heparin (Ajinomoto, Tokyo, Japan) intravenously. Heparin was used for 2–3 days following the procedure as a transition to warfarin (Eisai, Tokyo, Japan). Warfarin administration began the day after the procedure, and was usually continued for more than 6 months to maintain international normalized ratio at 1.5–2.0.

Follow-up evaluation

Laboratory data and Doppler US were evaluated bi-monthly on an outpatient basis and venography and manometry were performed, when recurrent HVOO was suspected.

Our current strategy for the treatment of HVOO is shown in Figure 1. Stent placement was recommended in patients who developed recurrent HVOO at the third intervention. We used self-expanding metallic stents, including ZA stents (Cook, Bloomington, IN, U.S.A.), SMART stents (Cordis, Warren, NJ, U.S.A.) and E-Luminexx stent (Bard Peripheral Vascular, Tempe, AZ, U.S.A), with a diameter 20% - 30% larger than that of the hepatic vein proximal to the stenosis and with sufficient length to cover the stricture. Early in our study, a small number of patients were treated with repeated multiple sessions of balloon angioplasty, because stent placement was not recommended in our hospital at that time.

Definitions

Two authors (blinded) reviewed the patient's medical records retrospectively, and evaluated technical success, clinical success, patency rate, stent placement, and major complications.

Technical success was defined as successful completion of percutaneous interventions with less than 20% stenosis at postoperative venography or less than 3 mmHg pressure gradient across the stenosis at postoperative manometry. Clinical success was defined as resolution or marked improvement of clinical symptoms, including ascites, Doppler US findings, or laboratory findings. Abnormal Doppler US findings were defined as a disappearance of pulsatile hepatic venous flow, flattening of the hepatic venous wave, or weak Doppler-detectable blood flow (< 10 cm/s) [19]. We also evaluated the serum levels of aspartate aminotransferase, alanine aminotransferase, total bilirubin and albumin. For patients presenting with marked ascites, abnormal Doppler US findings and abnormal laboratory data, clinical success was verified using clinical records and laboratory data describing marked improvement between just before the first balloon dilation and at the end of the follow-up period. With respect to patency rate, we evaluated primary patency and primary-assisted patency. Primary patency was defined as the interval between the initial balloon angioplasty and recurrent HVOO necessitating percutaneous intervention. Primary-assisted patency was defined as patency after the initial angioplasty until treatment with repeated percutaneous interventions was abandoned. The observational endpoints were the end of the follow-up period in our study and the patient’s death. We also evaluated non-responders,

defined as patients necessitating more than two sessions of percutaneous interventions, in terms of presence or absence of kinks, longer lesions, and the onset of HVOO. The timing of stent placement, additional interventions after stent placement and the patency of the stent were also evaluated. Complications related to the procedures were divided into major and minor categories according to Society of Interventional Radiology criteria [20]. Major complications were also evaluated.

Statistical analyses

Statistical analyses of clinical success and the difference in the onset of HVOO between responders and non-responders were performed using the Wilcoxon test and the Mann-Whitneys U test, respectively. P values < 0.05 were considered statistically significant. Statistical analysis of the patency rate was performed using the Kaplan-Meier method. Data processing and analysis were performed using software (SPSS for Windows version 21.0, SPSS, Chicago, IL, U.S.A).

RESULTS

Technical success

A total of 93 interventions were attempted in all 48 patients with HVOO. Ninety-two interventional sessions were successfully performed, but stent migration into the right atrium occurred in one patient. Technical success was achieved in 92 of 93 sessions (99.0%) and in 47 of 48 patients (97.9%).

Clinical success

Clinical success was achieved in 41 of 48 patients (85.4%). Of the 38 patients presenting with marked ascites, resolution or marked improvement was observed in all patients. The Doppler US findings normalized in all the 44 patients presenting with abnormal Doppler US findings. Decreased serum aspartate aminotransferase, alanine aminotransferase, total bilirubin or increased albumin levels were observed in 24 of the 31 patients presenting with abnormal laboratory values. In these 24 patients, the mean levels of serum aspartate aminotransferase were 43.6 ± 28.9 U/L before the procedure and 40.2 ± 22.7 U/L at the end of the follow-up period (P=0.55). The mean levels of serum alanine aminotransferase were 37.7 ± 24.9 U/L before the procedure and 29.1 ±

14.1 U/L at the end of the follow-up period (P=0.83). The mean levels of serum total bilirubin were 1.43 ± 1.88 mg/dl before the procedure and 0.94 ± 0.67 mg/dl at the end of the follow-up period (P=0.17). The mean levels of albumin were 3.46 ± 0.82 g/ml before the procedure and 3.74 ± 0.62 g/ml at the end of the follow-up period (P=0.095). None of the differences were statistically significant.

Patency rate

Follow-up periods in the 48 patients ranged from 1 to 182 months (median, 51.5 months), and the results are shown in Table 2 and Figure 2 showed our results. Twenty eight patients were treated with single balloon angioplasty (Figure 3), and did not develop recurrent stenosis. Eleven patients were treated with two sessions of percutaneous interventions. Two patients underwent more than five sessions of percutaneous interventions, including stent placement. One patient, who underwent seven sessions of balloon angioplasty and three sessions of stent placement, showed no recurrent HVOO. Another patient underwent nine sessions of balloon angioplasty and two sessions of stent placement, however the hepatic vein was thrombosed after the last balloon angioplasty, and re-transplantation was performed.

There were 10 non-responders and 38 responders, and no kinks and longer lesions

were seen in the ten non-responders. A significant difference in the onset of the initial HVOO was seen between the non-responders (12.1 ± 10.1 months) and the responders (39.9 ± 47.3 months) ($P < .05$) using the Mann-Whitney‘s U test.

During the follow-up periods (range, 1 to 182 months; median, 51.5 months), the rates of primary and primary-assisted patency (Fig. 4) at 1-, 3-, 5-, and 10-years after the initial balloon angioplasty were 64%, 57%, 57% and 52%, and 98%, 95%, 95% and 95% respectively.

Stent placement

Table 3 shows the outcomes in the patients treated with stent placement. Six patients with recurrent HVOO underwent initial stent placement at the second to forth interventional session. One patient underwent stenting at the second session, because of re-transplantation due to chronic rejection and early stent placement was recommended. Three patients underwent stenting at the third session and two patients at the forth session. Of the six patients, four had no recurrence of the HVOO 1 to 21 months (median, 9.5 months) after the stent placement (Fig. 5). One patient (Patient 3), who developed recurrent HVOO after the initial stent placement, was treated with an additional four sessions of balloon angioplasty and two sessions of stent placement. The

hepatic vein was patent for 84 months after the last stent placement in this patient. Another patient (Patient 1), who developed recurrent HVOO after the initial stent placement, was treated with additional six sessions of balloon angioplasty and one session of stent placement. However, the hepatic vein was occluded with thrombi, and the patient underwent re-transplantation.

Major complications

There was one major complication in a session of a 5-year-old boy with recurrent HVOO. At the third intervention, he underwent balloon dilatation followed by stent placement using the transhepatic approach and a SMART stent with 4cm in length and 10mm in diameter. After the stent was deployed, it shifted 1cm toward the right atrium. We then attempted placement of a second stent to cover the first, and inserted a catheter with the stent. However, the first stent migrated into the right atrium. Although we attempted to retrieve it with an Amplatz Gooseneck snare (Covidien, Plymouth, MN, U.S.A.), we were unsuccessful and a median sternotomy was performed surgically. The stent, which was lodged in the chordae tendinae of the tricuspid valve, was removed via a right atrial incision. The hepatic vein was thrombosed 1 month after the surgical procedure, and re-transplantation was performed. The patient showed no recurrent

HVOO 19 months after re-transplantation.

DISCUSSION

Without appropriate treatment for HVOO, hepatic congestion, massive ascites, portal hypertension, and finally liver cirrhosis and graft failure may occur. Therefore, it is important to diagnose and treat HVOO early based on clinical symptoms, laboratory data, and Doppler US.

There have been several reports describing the effectiveness of balloon angioplasty for HVOO and it has been accepted as a first-line treatment for HVOO [14-16]. Kubo et al. demonstrated the mid-term outcomes of percutaneous interventions in 20 patients who developed HVOO after LDLT [14]. In their study, 19 of 20 patients with HVOO were treated with balloon angioplasty alone and one was treated with balloon angioplasty and stent placement. Eleven patients showed no recurrent HVOO after the initial balloon angioplasty, and the primary patency rates at 3, 6, 12, and 60 months after balloon angioplasty were 80%, 65%, 60%, and 60% respectively. Ikeda et al. reported that 10 patients with HVOO after LDLT were treated with balloon angioplasty alone and 5 of 10 patients had no recurrent HVOO 5 - 73 months (median, 15 months) after the first session of balloon angioplasty [15]. The primary patency rates at 1, 3, 6, 12, and 60 months after balloon angioplasty were 78%, 67%, 67%, 56% and 56%, respectively.

Lorenz et al. obtained a clinical success rates of 81% (13 of 16 patients) in their study [16]. The primary patency and primary-assisted patency at 3, 6, 12, 18 and 36 months after first dilation were 72.7%, 60%, 55.6%, 50% and 50%, respectively, and 90.9%, 90%, 88.9%, 87.5% and 83.3%, respectively. Our clinical success rate was 85.4% (41/48 patients). We saw marked improvement of the ascites and abnormal Doppler US findings in all patients. Our results were comparable to those of Lorenz et al [16], who reported that clinical failure was related to the presence of comorbidities, such as rejection, hepatitis, and primary graft failure. As for these authors, we also found that improved laboratory data did not always correlate with improvement of the HVOO.

Although balloon angioplasty is effective for managing HVOO, stent placement is required in some patients with recurrent HVOO refractory to the balloon angioplasty [9, 17]. Ko et al. described 108 patients who developed early-onset HVOO after LDLT and who were treated with stent placement. The overall 1-, 3-, and 5-year primary patency rates were 82.3 ± 0.3%, 75.0 ± 0.4%, and 72.4 ± 0.5%, respectively. The authors advocated primary stent placement for early post-transplant HVOO [17], but we believe that in pediatric patients after LDLT, stent placement should be avoided, if possible, because of the possibility of thrombosis, the long-term patency of metallic stents for decades is unknown, and the size of the graft hepatic vein may not match the size of the

inserted stent when the patients grow. Indeed, Ko et al. reported that only three patients younger than 15 years of age underwent stent placement in their 108 patients [17]. In our study, 28 patients treated with a single session of balloon angioplasty and 10 with two sessions of balloon angioplasty showed no recurrent HVOO during follow-up. We believe that primary stenting could be avoided for a number of pediatric patients with HVOO, because 79% (38/48) of our patients were successfully treated within two sessions of balloon angioplasty alone.

Stent placement maintains the patency of the hepatic vein better than balloon angioplasty and stent placement is needed in patients with HVOO refractory to balloon angioplasty. However, the appropriate timing of stent placement for refractory HVOO after balloon angioplasty remains unclear. Umehara et al. reported that stent placement should be considered in patients with chronic rejection who are refractory to several balloon angioplasties with early- or late-onset HVOO [21]. In our study, stent placement was performed at the third session of percutaneous intervention in 3 patients, at the fourth session of percutaneous intervention in two, and at the second session of percutaneous intervention in one (Table 3). Three patients with stent placement at the third session of percutaneous intervention showed no recurrent HVOO and additional percutaneous interventions were not needed after the stent placement. One patient

underwent stent placement at the second session of percutaneous intervention because he had undergone re-transplantation due to chronic rejection and we recommended early stent placement. The patient did not develop recurrent HVOO after the stent placement. Two patients who underwent stent placement at the fourth session of percutaneous intervention developed recurrent HVOO and underwent additional interventions, including balloon angioplasty and stent placement. Currently, we perform stent placement at the third percutaneous intervention. Although our results suggest that stent placement was effective in treating recurrent HVOO after repeated balloon angioplasty, there might be disadvantages. Stent migration occurs rarely, but it is a severe complication. Although stent migration may occur due to incorrect selection of the appropriate stent size, it can also sometimes be very difficult to place the stent safely between the hepatic vein and inferior vena cava, because of the discrepancy in the diameters of the vessels, or because of respiratory motion [22]. Also, metallic stents might reduce adequate surgical margins of the hepatic veins at re-transplantation.

To prevent stent occlusion, drug-eluting stents have been used clinically for coronary artery disease and peripheral artery disease [23, 24]. Although the long-term patency of these stents has not been established, they may be useful to prevent in-stent thrombosis in patients with HVOO.

Our study has some limitations, including the retrospective design. Also, early in our study, a defined treatment strategy for HVOO was not established. Because the number of patients in this study was small, a case control design was not possible before and after the establishment of our HVOO treatment strategy. Finally, the number of patients may have been too small to evaluate the complications of the percutaneous interventions.

In conclusion, balloon angioplasty was safe and effective for hepatic venous outflow obstruction after LDLT, and stent placement might be needed in patients with recurrent HVOO refractory to balloon angioplasty.

REFERENCES

1. Starzl TE, Demetris AJ, Van Thiel D. Liver transplantation (1). N Engl J Med 1989;321: 1014–1022.

2. Tanaka K, Uemoto S, Tokunaga Y, et al. Surgical techniques and innovations in living related liver transplantation. Ann Surg 1993; 217:82–91.

3. Patkowski W, Nyckowski P, Zieniewicz K, et al. Biliary tract complications following liver transplantation. Transplant Proc 2003; 35: 2316–2317.

4. Buell JF, Funaki B, Cronin DC, et al. Long-term venous complications after full-size and segmental pediatric liver transplantation. Ann Surg 2002; 236:658–666.

5. Navarro F, Le Moine MC, Fabre JM, et al. Specific vascular complications of orthotropic transplantation with preservation of the retrohepatic vena cava: review of 1361 cases. Transplantation 1999; 68:646–650.

6. Parrilla P, Sanchez-Bueno F, Figueras J, et al. Analysis of the complications of the piggy-back technique in 1,112 liver transplants. Transplantation 1999; 67: 1214–1217.

7. Emond JC, Heffron TG, Whitington PF, Broelsch CE. Reconstruction of the hepatic vein in reduced size hepatic transplantation. Surg Gynecol Obstet 1993;176:11–17.

8. Chen HL, Concejero AM, Huang TL, et al. Diagnosis and interventional radiological treatment of vascular and biliary complications after liver transplantation in children with biliary atresia. Transplant Proc. 2008; 40: 2534-2536

9. Ko GY, Sung KB, Yoon HK, et al. Endovascular treatment of hepatic venous outflow obstruction after living-donor liver transplantation. J Vasc Interv Radiol 2002;13: 591–599.

10. Egawa H, Inomata Y, Uemoto S, et al. Hepatic vein reconstruction in 152 living-related donor liver transplantation patients. Surgery 1997;121:250–257.

11. Broelsch CE, Emond JC, Whitington PF, Thistlethwaite JR, Backer AL, Lichtor JL.

Application of reduced-size liver transplants as split grafts, auxiliary orthotopic grafts, and living related segmental transplants. Ann Surg 1990;212:368–75.

12. Carnevale FC, Machado AT, Moreira AM, et al. Midterm and Long-term Results of Percutaneous Endovascular Treatment of Venous Outflow Obstruction after Pediatric Liver Transplantation. J Vasc Interv Radiol 2008; 19:1439–1448

13. Akun E, Yaprak O, Killi R, Balci NC, Tokat Y, Yuzer Y. Vascular complications in hepatic transplantation: single-center experience in 14 years. Transplant Proc. 2012; 44: 1368-72

14. Kubo T, Shibata T, Itoh K, et al. Outcome of percutaneous transhepatic venoplasty for hepatic outflow obstruction after living donor liver transplantation. Radiology 2006;239:285-290.

15. Ikeda O, Tamura Y, Nakasone Y, et al. Percutaneous transluminal venoplasty after venous pressure measurement in patients with hepatic venous outflow obstruction after living donor liver transplantation. Jpn J Radiol 2010; 28: 520-526

16. Lorenz JM, Van Ha T, Funaki B, et al. Percutaneous treatment of venous outflow obstruction in pediatric liver transplants. J Vasc Interv Radiol 2006; 17:1753-61.

17. Ko GY, Sung KB, Yoon HK, et al. Early posttransplant Hepatic Venous Outflow Obstruction: Long-term Efficacy of Primary Stent Placement. Liver Transpl 2008; 14: 1505-1511

18. Sakamoto S, Egawa H, Kanawaza H et al. Hepatic venous outflow obstruction in pediatric living donor liver transplantation using left-sided lobe grafts: Kyoto University Experience. Liver Transpl 2010; 16:1207-1214.

19. Hwang HJ, Kim KW, Jeong WK, et al. Hepatic outflow obstruction at middle hepatic vein tributaries or inferior right hepatic veins after living donor liver transplantation with modified right lobe graft: comparison of CT and Doppler ultrasound. AJR Am J Roentgenol. 2009; 193:745-51.

20. Omary RA, Bettmann MA, Cardella JF, Bakal CW, Schwartzberg MS, et al. Quality

improvement guidelines for the reporting and archiving of interventional radiology procedures. J Vasc Interv Radiol 2003;14(pt 2): S293–S295

21. Umehara M, Narumi S, Sugai Y et al. Hepatic venous outflow obstruction in living donor liver transplantation: Balloon angioplasty or stent placement? Transplant Proc. 2012; 44: 769-771

22. Dancy MD. Management of venous outflow complications after liver transplantation. Tech Vasc Interv Radiol. 10(3): 240-5, 2007.

23. Di Lorenzo E, Sauro R, Varricchio A, et al. Long-Term outcome of drug-eluting stents compared with bare metal stents in ST-segment elevation myocardial infarction: results of the paclitaxel- or sirolimus-eluting stent versus bare metal stent in Primary Angioplasty (PASEO) Randomized Trial. Circulation 2009; 120:964-72.

24. Scheinert D, Katsanos K, Zeller T, et al. A prospective randomized multicenter comparison of balloon angioplasty and infrapopliteal stenting with the sirolimus-eluting stent in patients with ischemic peripheral arterial disease: 1-year results from the

ACHILLES trial. J Am Coll Cardiol 2012; 60:2290-5.

Table 1. Patients' Characteristics

Patient Profiles	
Characteristic	Data
Sex	
Male	24
Female	24
Age at the initial percutaneous intervention	
Range	2 months to 20 years
Median	6 years
Mode of transplantation	
Lateral segment	31
Left lobe	16
Right lobe	1
Original disease	
Biliary atresia	38
Fulminant hepatitis	2
Wilson disease	2
Alegre syndrome	1
Citrullinemia	1
Fulminant hepatic failure	1
Liver cirrhosis	1
Ornithine transcarbamylase deficiency	1
Primary sclerosing cholangitis	1

Table 2. Number of Interventional Sessions

1 session of BA	28 patients
2 sessions of intervention	11 patients
2 sessions of BA	10 patients
1 session of BA and 1 stenting session	1 patient
3 sessions of intervention	6 patients
3 sessions of BA	2 patients
2 sessions of BA and 1 stenting session	4 patients*
4 sessions of BA	1 patients
More than 5 sessions of intervention	2 patients
7 sessions of BA and 3 stenting session	1 patient
9 sessions of BA and 2 stenting session	1 patient

Total 93 sessions in 48 patients

BA = balloon Angioplasty

* One patient who experienced stent migration was included.

Table 3. Summary of Percutaneous Interventions in the Patients who Underwent Stent Placement

No.	Total Procedures	Procedure Description	Result
1	11	Procedure 1-3, 5-6, 8-11: balloon angioplasty Procedure 4,7: stent placement	Occlusion →re-transplantation
2	2	Procedure 1: balloon angioplasty Procedure 2: stent placement	No recurrence
3	10	Procedure 1-3, 5-6, 8-9: balloon angioplasty Procedure 4,7, 10: stent placement	Patent
4	3	Procedure 1-2: balloon angioplasty Procedure 3: stent placement	No recurrence
5	3	Procedure 1-2: balloon angioplasty Procedure 3: stent placement	No recurrence
6	3	Procedure 1-2: balloon angioplasty Procedure 3: stent placement	No reccurence

Figure1. Schema of our Strategy for HVOO Treatment

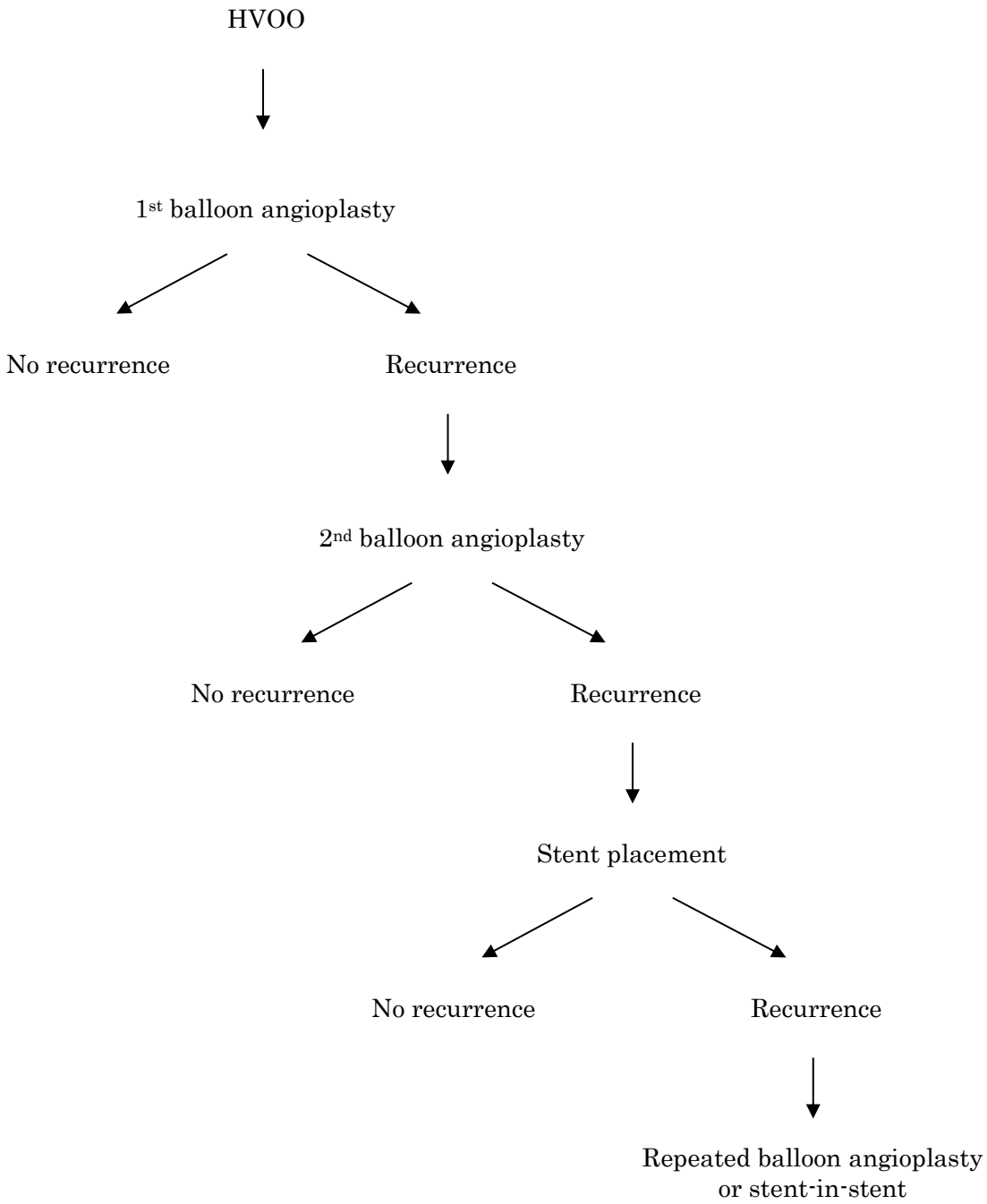


Figure 2. Outcomes of the Interventions in the 48 Patients

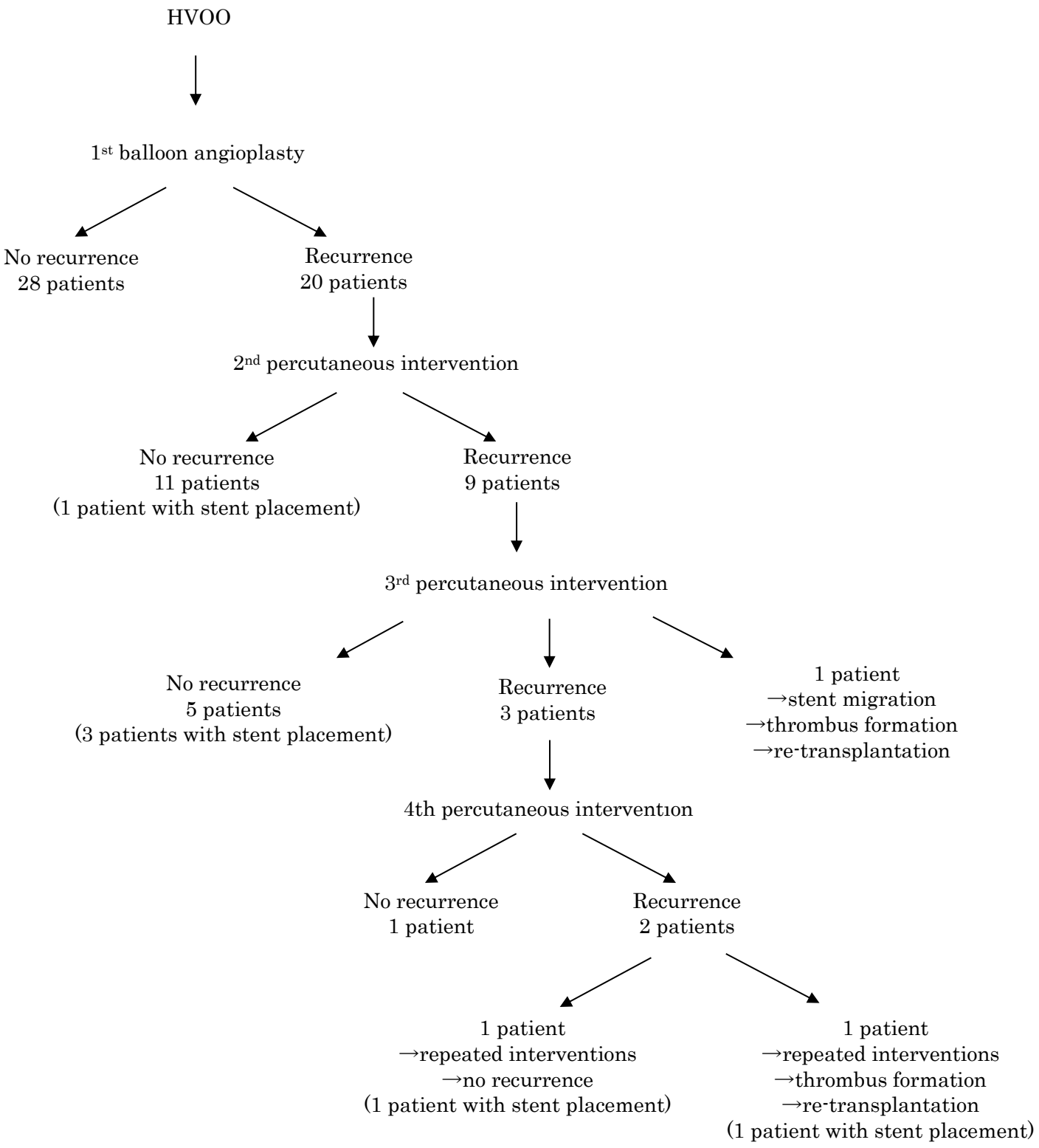


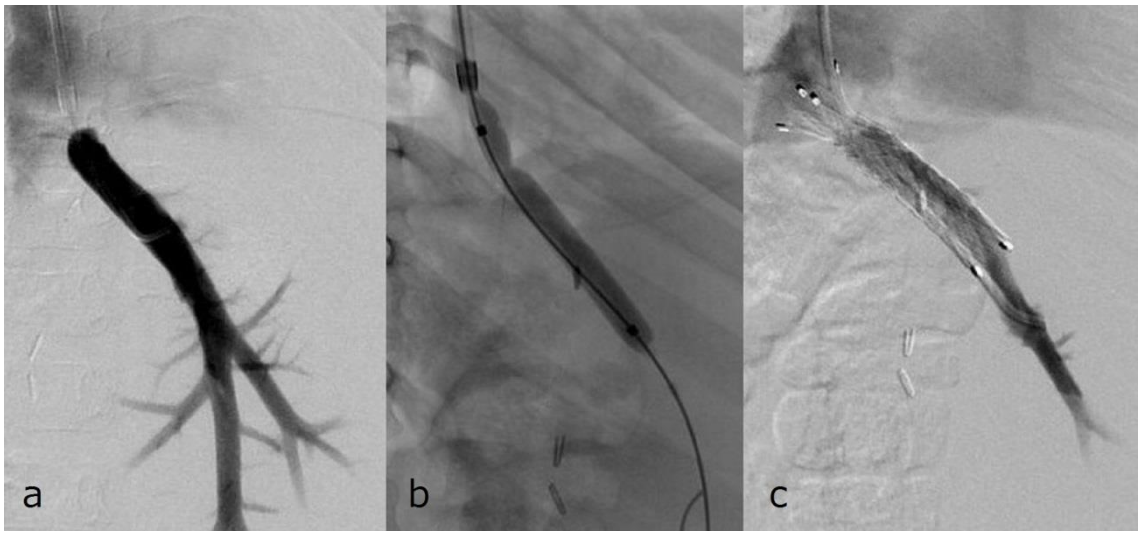
Figure 3

A 4-year-old girl with citrullinemia underwent left-lobe living donor liver transplantation (LDLT). Hepatic venous outflow obstruction (HVOO) was suspected 3 months after LDLT, and venography and manometry were performed. (a) Pretreatment hepatic venogram showing an anastomotic stricture between the left hepatic vein and the inferior vena cava. The pressure gradient across the stenosis between the distal hepatic vein and the right atrium was 17 mmHg. (b) Fluoroscopic view during balloon angioplasty showing full expansion of the balloon. (c) Hepatic venogram after the balloon angioplasty showing improved blood flow into the right atrium. The pressure gradient decreased to 2 mmHg. HVOO did not recur after the balloon angioplasty.

Figure 4. The Primary Patency and Primary-Assisted Patency Rates

Kaplan-Meier curve shows primary patency rate and primary-assisted patency. Solid and dotted lines indicate primary patency and primary-assisted patency, respectively. Vertical lines on both lines indicate censored observations. The primary patency and primary-assisted patency rates at 1-, 3-, 5-, and 10-years after the first balloon angioplasty was 64%, 57%, 57% and 52%, and 98%, 95%, 95% and 95% respectively.

Figure 5.



A 1-year-old girl with biliary atresia had undergone left-lobe LDLT and two sessions of balloon angioplasty for HVOO. Because recurrent HVOO was suspected due to ascites, venography and manometry were performed. (a) Pretreatment hepatic venogram showing a severe anastomotic stricture between the left hepatic vein and the inferior vena cava. The pressure gradient across the stenosis between the distal hepatic vein and the right atrium was 14 mmHg. (b) Fluoroscopic view during balloon angioplasty before stent placement showing the notch of the balloon at the site of the anastomotic stricture. (c) Hepatic venogram after stent placement showing improved blood flow into the right atrium. The pressure gradient decreased to 2 mmHg and HVOO did not recur after stent placement.

出典

平成２５年１１月発行 Journal of Vascular and Interventional Radiology

第２４巻 第１１号 １６７３頁～１６８１頁掲載

doi: 10.1016/j.jvir.2013.07.010.