Long-term outcomes of percutaneous interventions for anastomotic portal vein stenosis after pediatric living donor liver transplantation

Poster No.: C-0328
Congress: ECR 2014
Type: Scientific Exhibit
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Keywords: Fluoroscopy, Ultrasound, Catheter arteriography, Liver, Vascular, Interventional vascular, Angioplasty, Stents, Transplantation
DOI: 10.1594/ecr2014/C-0328

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Aims and objectives

Introduction

Liver transplantation is an established treatment for end-stage liver disease [1]. Although deceased donor liver transplantation is considered a standard procedure, living donor liver transplantation (LDLT) has been widely performed owing to the shortage of donors [2, 3]. LDLT and split liver transplantation are technically demanding because of the use of short vascular pedicles, which are more likely to cause postoperative vascular complications. The rate of portal vein (PV) complications after deceased donor liver transplantation has been reported to be < 3% [4-7]. However, in patients with reduced-size liver transplantation or LDLT, the rate of PV complication can be higher (9-14%) than in patients with conventional deceased donor liver transplantation [8-11]. PV complications are divided mainly into anastomotic PV stenosis (PVS) and portal vein thrombosis (PVT) [12]. Anastomotic PVS can lead to graft failure if not properly treated. Treatment options for PVS after liver transplantation are surgical treatment and percutaneous interventions, including balloon angioplasty and stent placement [13]. However, surgical treatment of these complications has been limited owing to technical difficulties or invasiveness. Currently, the surgical treatment of PVS after liver transplantation has been replaced by percutaneous balloon angioplasty and stent placement, because of lower invasiveness and greater effectiveness [7, 14-19]. The purpose of our study was to retrospectively evaluate the long-term outcomes of balloon angioplasty with or without stent placement for anastomotic PVS in pediatric patients after LDLT.
Methods and materials

MATERIALS AND METHODS

Patients

Between October 1997 and August 2013, LDLT was performed in our surgery department in 527 pediatric patients (age < 18 years). PVS was clinically suspected with the following findings: (1) clinical symptoms of portal hypertension, such as ascites, splenomegaly, gastrointestinal tract bleeding from varices, and thrombocytopenia; and (2) ultrasound (US) findings, including greater than 50% stenosis (the diameter of stenosis/the diameter of a main PV on the mesenteric side) or no flow in the PV; or the presence of an acceleration of flow at the stenosis or a poststenotic jet flow or minimal flow in the intrahepatic PV on Doppler US [17]. Forty-seven patients were confirmed to have PVS using portography with or without manometry. Our inclusion criteria for PVS were: (1) greater than 50% stenosis (the diameter of the stenosis/the diameter of a PV on the distal side); or (2) > 5 mm Hg pressure gradient across the stenosis between the proximal and distal PV. All 47 patients underwent percutaneous interventions, including balloon angioplasty with or without stent placement. The age of the patients ranged from 7 months to 19 years (median, 2 years) at the first intervention.

Procedures

Four authors with 5, 29, 17, and 5 years of experience in interventional radiology performed the procedures. Percutaneous interventions were performed with general anesthesia in all patients. The approach to the intrahepatic PV was transhepatic in all patients at the first session of percutaneous intervention. Balloon angioplasty was performed following venography with a 7.0 Fr percutaneous transluminal angioplasty catheter (Powerflex Plus, Cordis, Warren, NJ) with a balloon diameter of 6-12 mm and a length of 40 mm. The diameter of the balloon was the same as the vein on the mesenteric side of the stenosis. The balloon was inflated three times for 60 s with an atmospheric pressure of 10 atm. Venography with or without manometry was then repeated to evaluate the effectiveness of the balloon angioplasty. For hemostasis, the sheath tract was embolized with collagen material (Avitene, Zeria Pharmaceuticals, Tokyo, Japan), when the sheath was removed.

Follow-up evaluation
Laboratory data and Doppler US were evaluated bi-monthly on an outpatient basis and venography with or without manometry was performed when recurrent PVS was suspected. Stent placement was performed in patients who developed recurrent PVS. We used a self-expanding metallic stent (E-LUMINEXX, Bard Peripheral Vascular, Tempe, Arizona) with a diameter 20-30% larger than that of the PV 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**

Technical success was defined as successful completion of percutaneous intervention with less than 20% stenosis using postoperative portography or less than 3 mm Hg pressure gradient across the stenosis using postoperative manometry. With respect to the patency rate, we evaluated primary patency and primary-assisted patency. Primary patency was defined as the interval between the initial balloon angioplasty and recurrent PVS necessitating percutaneous intervention. Primary-assisted patency was defined as patency following the initial angioplasty until repeated percutaneous intervention therapy was discontinued.

The timing of stent placement, additional interventions after stent placement, and the patency of the stent were also evaluated.

We divided complications related to the procedures into major and minor categories according to the Society of Interventional Radiology criteria [20], evaluating major complication.

**Statistical analysis**

We used the Kaplan-Meier method for statistical analysis of the patency rate. We used SPSS for Windows, version 21.0 (SPSS, Chicago, IL) for all data processing and analysis.
Results

Technical success

A total of 78 percutaneous interventions were attempted in all 47 patients with PVS. Seventy-seven interventional sessions were successfully performed, but passing through the PVS with a guidewire and catheter failed in a one session in one patient. Technical success was achieved in 77 of 78 sessions (98.7%) and in 46 of 47 patients (97.8%).

Patency rate

Follow-up periods in the 47 patients ranged from 8 days to 166 months (median, 106 months). Thirty-three patients were treated with single balloon angioplasty (Fig. 1) with no recurrent stenosis. Six patients were treated with two sessions of balloon angioplasty. There were eight non-responders (17%) and 39 responders (83%). Kinking of the PV was suspected as a cause of PVS, and stent placement was performed in one of the eight non-responders. No significant difference in the onset of PVS was seen between the non-responders (14.5 ± 10.2 months) and the responders (23.0 ± 27.6 months) (p = .07) using the Mann-Whitney U test. During the follow-up periods (range, 8 days to 166 months; median, 109 months), at 1, 3, 5, and 10-years after the initial balloon angioplasty, the rates of primary patency were 80%, 76%, 73%, and 67%, respectively, and those of primary-assisted patency were 100%, 100%, 100%, and 96%, respectively (Fig. 2).

Stent placement

Three patients with recurrent PVS underwent stent placement. Of these, two had no recurrence of PVS 82 months (Fig. 3) and 68 months following stent placement. In the other patient, severe recurrent PVS due to PVT was seen 101 months after stent placement.

Major complications

There were two major complications in two sessions in two patients.

In a 1-year-old boy, a severe asthma attack occurred during recovery from general anesthesia. He was admitted to the intensive care unit (ICU), recovered and was discharged from the ICU the next day. The asthma attack may have been caused by an
allergic reaction to contrast media or collagen material for the tract embolization following the sheath removal.

In the other 1-year-old boy, PVT was seen the day following balloon angioplasty for PVS. Thrombectomy was performed by the transileocecal approach following laparotomy, and balloon angioplasty for the PV was repeated. Treatment followed with continuous intraportal infusion of urokinase for one week (20 000 unit/day), followed by continuous intraportal infusion of heparin for 9 days (1750 unit/day) via the catheter intraoperatively placed into the PV. No recurrent PVS was seen after the intraportal infusion.
Fig. 1: A 7-year-old girl with biliary atresia underwent left-lobe living donor liver transplantation (LDLT). Portal veinous stenosis (PVS) was suspected 5 years after LDLT, and portography was performed. (a) Pretreatment portogram showing an anastomotic stricture (arrow), collateral vessels (arrow head) and poor flow of intrahepatic portal vein. Manometry was not performed. (b) Fluoroscopic view during balloon angioplasty showing the notch of balloon at the stenosis. (c) Portgram after the balloon angioplasty showing improved blood flow to portal vein and disappearance of collateral vessels. PVS did not recur after the balloon angioplasty.

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Fig. 2: 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 were 80%, 76%, 73% and 67%, and 100%, 100%, 100% and 96% respectively.

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Fig. 3: A 2-year-old girl with biliary atresia had undergone left-lobe LDLT and seven sessions of balloon angioplasty for portal vein stenosis (PVS). Because recurrent PVS was suspected, portography was performed. (a) Pretreatment portogram showing a severe anastomotic stricture and no flow into intrahepatic portal vein. Manometry before stent placement was not performed. (b) Portogram after stent placement showing improved blood flow into portal vein. The pressure gradient between the proximal and distal side of the stent was not noted, and PVS did not recur after stent placement.

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Conclusion

Discussion

Similar to previous articles [11, 14, 19, 20], we completely agree with the concept that manometry was not indispensable. This is because gradient pressure does not necessarily reflect the severity of PVS by the presence of collateral vessels and because it might be impossible to pass through the occlusive site again with guidewire following manometry in patients with portal venous occlusion.

Balloon angioplasty for PVS after LT was first published by Raby in 1991 [21], and some authors reported the efficacy and effectiveness of balloon angioplasty for PVS in patients after LT [13, 15, 20]. Funaki et al showed that 7 of 14 patients who underwent initial balloon angioplasty had required no further intervention with PV patency being maintained for 9-56 months (mean, 36.7 months ) in their recent article [13]. Shibata et al [15] described the effectiveness of balloon angioplasty in 35 patients who underwent balloon angioplasty after adult and pediatric LDLT. In their study, 71.4% (25 of 35) patients with PVS were successfully treated with a single session of balloon angioplasty, 9 of the remaining 10 patients were treated with repeated balloon angioplasty. In the other patient, PV was thrombosed after two sessions of balloon angioplasty. Park et al [20] reported that all of 6 patients with PVS after adult and pediatric LT were treated with balloon angioplasty alone. Four of 6 patients had no recurrent PVS 42 months (median, 18.5 months) after the first session of balloon angioplasty, and the other 2 patients were treated with two sessions of balloon angioplasty. Our result was not inferior to their results.

Stent placement has been used widely to treat PVS [11-14, 22]. Funaki et al reported their excellent result of percutaneous treatment of PVS in adult and pediatric LT [12, 13, 22]. In their recent reports, Stent placement was performed in 7 patients with recurrent PVS after initial balloon angioplasty and 5 patients with elastic stenosis after balloon angioplasty, and no recurrent PVS after stent placement was seen in 12 patients for 5-61 months (mean, 47 months) [13]. Primary patency of stent at 3-year was 100%. Carnevale et al described that stent placement was performed in 4 patients after failed balloon angioplasty and with recurrent PVS after initial balloon angioplasty [11]. After stent placement, recurrent PVS was not seen in all. Ko et al reported the efficacy and safety of primary stent placement for patients with PVS after LDLT at early posttransplantation period (less than 1 month) [14]. No recurrent PVS was not noted in 7 of 9 patients who underwent primary stent placement. They preferred to perform primary stent placement rather than balloon angioplasty in patients with PVS at early posttransplantation period for 2 reasons. First, there was the possibility of anastomotic rupture by balloon angioplasty at early posttransplantation period. Second, they assumed that PVS caused by discrepancy of PV size between donors and recipients, tension or twisting of PV, or extrinsic
compression of the PV by hematoma and reactive edema could not be corrected by angioplasty alone.

Our study has some limitations. First, this was retrospective study. Second, therapeutic strategy for PVS was not unified in our study. In particular, the timing of stent placement was not decided in early period of our study. Third, it was impossible to design a case-control study before and after the established of our therapeutic strategy for PVS, because the number of patients in this study was small. Fourth, the patients with PVS due to PVT were not included in our study. Finally, the number of patients who underwent stent placement or with complication was too small to evaluate the effectiveness of stent placement or complications of percutaneous interventions for PVS.

In conclusion, percutaneous interventions, such as balloon angioplasty and stent placement, were effective in patients with PVS after pediatric LDLT. Stent placement might be needed in patient with refractory PVS to balloon angioplasty.
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References


