INTRODUCTION

Liver cirrhosis can be associated with PV hypoplasia and severe alterations of the liver hemodynamics. PV hypoplasia may lead to technical difficulties in PV reconstruction during LT due to size mismatch with the donor PV, elasticity, or the intimal thickening of the recipient PV, which increases the risk of posttransplant PV complications, and thus increases the risk of graft loss, especially in pediatric patients.\(^1\)

Several risk factors have been demonstrated regarding PV complications following pediatric LT, including recipient body weight <6.0 kg, sclerotic small caliber native PV (<4 mm), and the use of interposed vein grafts.\(^2-4\) The technical challenges associated with pediatric LT have led to the development of ingenious strategies for PV reconstruction and various innovative techniques have been reported to reduce posttransplant PV complications, including the venous patch technique, longitudinal venotomy, and ellipsoid anastomosis.\(^5-7\) PV reconstruction, however, remains a topic of debate due to the technical difficulty of anastomosis, especially in patients with PV hypoplasia/thrombi that extended to the SMV and the splenic venous junction (SMV-SpV junction).

We report our single-center experience with 10 pediatric living donor LT patients with PV hypoplasia/thrombus that extended to...
near the SMV-SpV junction, and the details of the anatomy and surgery of PV reconstruction using a “pullout technique.”

2 | PATIENTS AND METHODS

Between November 2005 and March 2018, 465 children underwent living donor LT at the National Center for Child Health and Development, Tokyo, Japan. The overall rates of 10-year patient and graft survival were 91.1% and 90.0%, respectively. During the same study period, 10 patients who showed PV hypoplasia/thrombus that extended to near the SMV-SpV junction, which necessitated the use of the pullout technique in PV reconstruction, were enrolled in the present study.

3 | OPERATION PROCEDURE

The standard LT procedure has been previously described. In cases with LGV development, the main PV is elongated down to the SMV-SpV junction, and then the LGV should be ligated. If the LGV drains to the SMV-SpV junction or the SpV, the LGV is ligated just above the SpV. All of the potential collateral vessels, including splenoretroperitoneal shunts, splenocolic shunts, mesentery collaterals and spleno-renal shunts are interrupted to obtain sufficient portal flow prior to implantation. Preoperative CT scan and intraoperative echography were used to determine the possible course/inflow site of the LGV and the SMV-SpV junction. In the present series, the PV hypoplasia/thrombus extended close to the SMV and SpV junction; thus, sufficient PV front flow could not be obtained through a branch patch of the PV bifurcation. We identified and meticulously dissected the posterior superior pancreatocoduodenal veins and the other branches from the pancreas to the PV. The SMV and SpV were then isolated and clamped individually, distal to the confluence. The posterior face of the pancreas was tunnelled along the PV (Figure 1). This made it possible to pull the PV out from the superior border of the pancreas to the inferior border of the pancreas. The hypoplastic PV/thrombus was completely removed thrombectomized with an eversion technique. The PV was returned to its original position with or without the use of an interposed vein graft.

4 | RESULTS

Ten (2.2%) LT procedures were performed using the pullout technique. The donor and recipient profiles are shown in Table 1. There were five male and five female patients. The median age was 9 months (range: 5 months-11 years). The median weight was 8.1 kg (range: 5.0-29.7 kg). The indications for LDLT included BA (n = 7), HBL (n = 2) and re-transplantation for chronic rejection with PV thrombosis (n = 1). The mean PELD score was 8.5 ± 6.9 (range: 0-20.9). A reduced LLS graft was used in two cases, and the LLS was used in 8. The median follow-up period was 48 months (range: 12-76 months).

The indications for the pullout technique were PV hypoplasia in seven cases and PV thrombus in three cases. In the PV hypoplasia patients, the caliber of main PV trunk determined by preoperative ultrasonography ranges from 2.0~4.0 mm in diameter with insufficient flow (Table 1). The inflow sites of the enlarged LGV included the main PV trunk (n = 2), the SMV and SpV junction (n = 4) and the SpV (n = 4) by pre- and intraoperative findings. Attenuation of the PV was observed just above the termination site of the LGV and was removed in all cases (Figure 2). Interposed vein grafts were applied in nine patients (donor’s ovarian vein [n = 5], native left renal vein [n = 4], native left internal jugular vein [n = 2] and native external iliac vein in [n = 1]). An interposition vein graft was first anastomosed to the confluence of the SMV and the SpV after cutting the narrow/sclerotic native part of PV (Figure 3). The anastomosis was started at the posterior wall with continuous sutures, after which the anterior anastomosis was completed with interrupted 6-0 polydioxanone absorbable monofilaments. Direct PV anastomosis was possible in Case No. 8. The duration and blood loss of the recipient operation were 745.4 ± 227.4 minutes (range: 486-1108 minutes) and 212.4 ± 274.6 g/kg (range: 36.1-954.9 g/kg), respectively. A PV catheter was inserted in Case Nos. 2 and 7 for a week with a continuous infusion of heparin to ensure PV flow. The remaining eight patients received systemic heparinization for 1 week (5 U/kg/h; target activated partial thromboplastin time: approximately 40 seconds) and were switched to warfarin for 3 months after LDLT. Case No. 3 experienced PV stricture 5.7 years after LDLT, which was successfully managed with single balloon dilatation under radiological intervention. All patients are currently doing well with a patent PV.
### TABLE 1  Donor and recipient profiles

| Case | Original liver disease | Sex | Age | BW (kg) | Donor | Graft type | Indication of "pullout" | Preoperative PV caliber (mm) | Preoperative PV flow | Drainage site of LGV | Vein graft | Outcome (last follow-up) |
|------|------------------------|-----|-----|---------|-------|------------|-------------------------|----------------------------|----------------------|---------------------|------------|------------------------|
| 1    | BA                     | F   | 5 mo| 6.5     | Mother | Reduced LLS | Hypoplasia             | 2.5                        | To and fro          | SpV                 | Ovarian vein         | Alive (4 y 4 mo)      |
| 2    | BA                     | F   | 5 mo| 5.0     | Father | Reduced LLS | Hypoplasia             | 2.0                        | Hepatofugal          | SpV                 | Lt Renal vein         | Alive (4 y)           |
| 3    | BA                     | F   | 7 mo| 7.5     | Mother | LLS        | Hypoplasia             | 3.5                        | To and fro          | Confluence of SMV and SpV | Ovarian vein         | Alive (5 y 6 mo)       |
| 4    | BA                     | F   | 8 mo| 6.6     | Mother | LLS        | Hypoplasia             | 4.0                        | To and fro          | Main PV             | Ovarian + Lt Int Jugular vein | Alive (3 y 6 mo)       |
| 5    | BA                     | F   | 8 mo| 5.8     | Mother | LLS        | Hypoplasia             | 3.5                        | To and fro          | Confluence of SMV and SpV | Ovarian + Lt Renal vein | Alive (1 y)           |
| 6    | BA                     | M   | 10 mo| 9.2     | Father | LLS        | Hypoplasia             | 2.9                        | Hepatofugal          | Confluence of SMV and SpV | Lt Renal vein         | Alive (5 y 6 mo)       |
| 7    | BA                     | M   | 11 mo| 8.7     | Father | LLS        | Hypoplasia             | 2.0                        | Hepatofugal          | SpV                 | Lt Renal + Lt Int Jugular vein | Alive (1 y)           |
| 8    | HBL                    | M   | 2 y 7 mo| 12.4   | Mother | LLS        | Thrombus                | -                          | Not detected      | Confluence of SMV and SpV | -                     | Alive (6 y 4 mo)       |
| 9    | Re-transplantation      | M   | 2 y 9 mo| 11.5   | Mother | LLS        | Thrombus                | -                          | Not detected      | Main PV             | Ovarian vein         | Alive (1 y)           |
| 10   | HBL                    | M   | 11 y 10 mo| 29.7   | Father | LLS        | Thrombus                | -                          | Not detected      | SpV                 | External iliac vein   | Alive (1 y)           |

Lt, left.
DISCUSSION

The importance of the LGV is increasingly recognized through the treatment of esophagogastric varices, and in the surgical management of gastric cancer as a major drainage vein of the stomach. The anatomical variation of the LGV, also known as the gastric coronary vein, was first described by Hochstetter in 1886. Recent advances in laparoscopic gastrectomy have allowed for a better understanding of LGV variations to ensure sufficient lymph node dissection for cancer and to prevent unnecessary bleeding. Several studies have demonstrated the precise determination and classification of the branching pattern of the LGV. Wu et al. reported that the LGV flowed into the PV trunk in 45%, the SMV and SpV junction in 21% and the SpV in 33% based on MD-CT imaging findings in 825 adults. Due to the retrograde flow of the LGV in patients with severe portal hypertension, spontaneous hypoplasia of the PV might have originated from immediately above the termination site of the LGV. The LGV should be ligated in every pediatric LT recipient with liver cirrhosis in order to obtain sufficient front flow to the main PV because there is less PV overperfusion (small-for-size) syndrome in pediatric LT in comparison with adult LT. Our recent study showed that the PV pressure could be increased to ≥25 mm Hg after potential collateral interruption in pediatric LDLT, which resulted in a lower incidence of PV complications.

In cases where the LGV terminates at the SMV and SpV junction or the SpV, which may account for 54% of the general population, the native PV pedicle can be shortened for safe anastomosis and a vein graft should be placed, especially in LDLT when the graft of the left PV has a short stump. We recommend the use of an interposed vein graft for PV reconstruction in LDLT because it is necessary to remove the sclerotic native PV and to ensure sufficient PV front flow with or without the initiation of pullout technique. During the same study period, 38 patients (8.2%), whose PV caliber less than 4 mm, received interposed vein graft without pullout technique. The patient survival rates with interposed vein graft at 5 years was 97.4%, and corresponding cumulative interposed vein graft patency rates was 92.1%, respectively. Recent studies have shown the effectiveness of intraoperative PV stenting in sustaining PV flow, and this alternative procedure may be considered as a final option if all other types of surgical intervention have failed. Anticoagulation therapy has not been applied in our LDLT program; however, routine use of systemic heparinization for 1 week and switched to warfarin for 3 months is routinely adopted in these particular patients. This is the first series of patients to be successfully treated with a novel PV reconstruction procedure using a pullout technique. The pullout technique will be useful if the native PV showed sclerotic small caliber (<4.0 mm) and/or PV thrombus extended to the SMV and SpV junction in preoperative imaging study. The hypoplastic PV/PV thrombus could be completely removed by pulling out the PV from the superior border to the inferior border of the pancreas.
This surgical method allows for the anatomic reconstruction of the PV without a jump graft in patients with attenuated PV/PV thrombus involving the SMV and SpV junction, and the redundancy of PV anastomosis could be avoided using various vein grafts.

In conclusion, the pullout technique provided a good operative field, which allowed for the complete removal of the hypoplastic PV or thrombectomy and the safe placement of various interposed vein grafts.

CONFLICT OF INTEREST
The authors declare no conflict of interest in association with the present study.

AUTHORS’ CONTRIBUTIONS
All authors contributed significantly to the concept/design the study and critical revision of the manuscript and approved the final version. M.K.: study design, writing of the paper; S.S.: study design, critical revision of the article for clinical content; K.S., H.U., M.T., Y.H., A.F.: collection of the data.

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How to cite this article: Kasahara M, Sasaki K, Uchida H, et al. Novel technique for pediatric living donor liver transplantation in patients with portal vein obstruction: The “pullout technique”. Pediatr Transplant. 2018;22:e13297. https://doi.org/10.1111/petr.13297