ORIGINAL ARTICLE

Intra-operative management of low portal vein flow in pediatric living donor liver transplantation

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Keywords

inferior mesenteric vein cannulation, living donor liver transplantation, portal vein complication, stent.

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Conflicts of Interest

The authors have declared no conflicts of interest.

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Received: 25 November 2011 Revision requested: 15 January 2012 Accepted: 20 February 2012 Published online: 26 March 2012

doi:10.1111/j.1432-2277.2012.01464.x

Introduction

Liver transplantation has emerged as the definitive treatment for children with end-stage liver disease (ESLD). In regions where the deceased donor organs are limited, living donor liver transplantation (LDLT) has shown promising results in reducing the waiting list mortality with satisfactory long-term graft and patient survival [1]. Comparing with the whole-size deceased donor graft, LDLT for children presents a greater technical challenge.

Summary

For pediatric living donor liver transplantation, portal vein complications cause significant morbidity and graft failure. Routine intra-operative Doppler ultrasound is performed after graft reperfusion to evaluate the flow of portal vein. This retrospective study reviewed 65 children who had undergone living donor liver transplantation. Seven patients were detected with suboptimal portal vein flow velocity following vascular reconstruction and abdominal closure. They underwent immediate on-table interventions to improve the portal vein flow. Both surgical and endovascular modalities were employed, namely, graft re-positioning, collateral shunt ligation, thrombectomy, revision of anastomosis, inferior mesenteric vein cannulation, and endovascular stenting. The ultrasonographic follow-up assessment for all seven patients demonstrated patent portal vein and satisfactory flow. We reviewed our experience on the different modalities and proposed an approach for our future intra-operative management to improve portal vein flow at the time of liver transplantation.

> The shorter vascular pedicles and the mismatched sizes between the vessels of graft and recipient often potentiate the risk of vascular complications.

> Cirrhosis secondary to biliary atresia is the most common cause of pediatric ESLD [1]. As a result of previous surgery (Kasai procedure) and recurrent cholangitis, the portal vein (PV) becomes narrow and hypoplastic. The impaired quality of PV increases the risk of portal vein complication (PVC) with an incidence between 3.6% and 14% [2–5]. It commonly presents silently with absent clinical and biochemical

abnormality. Nevertheless, it is associated with significant risk of morbidity, graft failure and recipient mortality [3,5–8].

Many authors have described various techniques of PV reconstruction and treatments for PVCs [2,5,7,9–12]. In our earlier report, we have found that decreased PV flow velocity led to higher risk of early PVC [13,14]. However, there are only few reports concerning the intra-operative management when suboptimal PV flow is detected immediately after vascular reconstruction during transplantation [2,15]. This study reviewed our experience on this group of patients and described the various intra-operative techniques applied to optimize the PV flow, thereby, minimizing the risk of postoperative PVC.

Patients and methods

The hospital's Institutional Review Board's approval was obtained prior to commencement of the study. The approval number is 100-1607B and the protocol number is 1000520-01.

Between January 2005 and December 2009, 387 patients underwent liver transplantation at Kaohsiung Chang Gung Memorial Hospital. Sixty-five of them were pediatric patients with age below 18 years and received grafts from living related donors. Intra-operative Doppler ultrasonography (IODUS) was performed for all patients after vascular reconstruction and abdominal closure. Among the 65 patients, seven had suboptimal PV flow velocity of less than 10 cm/s and underwent on-table interventions. These seven patients were included in this study. Their clinical charts were reviewed. The peri-operative data were collated and analyzed. Follow-up is defined as the duration between the transplantation and the latest radiological and clinical assessment.

Operation

The techniques of LDLT are described in detail in our previous reports [1,16,17]. For all seven patients, the graft PV pedicle was anastomosed to the bifurcation of the right and left branches of the recipient's PV. The septum of the PV branches was split open to accommodate the larger graft PV (branch patch method). A growth factor of half to one times the diameter of the anastomosis was used. Saline flushing was performed for recipient PV to remove possible clot prior to reconstruction. No interposed vein graft was used. All abdominal closure was achieved without the use of prosthesis.

Intra-operative Doppler ultrasonography

Intra-operative Doppler ultrasonography was routinely performed by an accredited radiologist after vascular

Postoperative management Postoperative management Postoperative management For pediatric LDLT recipients wh graft was satisfactory, the routine lation required intravenous hepa

reconstruction and abdominal closure. When difficult PV anastomosis was encountered, IODUS would be arranged immediately after PV reconstruction. If reconstructed PV exhibited normal appearance after recanalization, IODUS would be done after completion of hepatic artery anastomosis with microsurgical technique. Following the standard hepatic vascular protocol, an Acuson 128 scanner (Acuson, Mountain View, CA, USA) with 7.0 or 4.0 MHz scanner was used. The flow velocity and cross-sectional area of PV were measured with Doppler mode. A PV flow velocity of less than 10 cm/s would be deemed as unacceptable and necessitated immediate measures to improve the flow velocity.

Intra-operative management of decreased portal vein flow

The intra-operative management of decreased PV flow aimed to identify the attributing factors and to correct them as soon as possible. Hence, if compromised PV flow was detected after abdominal closure, immediate re-laparotomy would be performed. Graft position adjustment, PV thrombectomy, collateral portosystemic shunt ligation and revision of anastomosis were surgical modalities attempted to improve PV flow.

Besides surgical techniques, endovascular interventions were also utilized. For patients with the risk of PV thrombosis, inferior mesenteric vein (IMV) cannulation would be performed with a Broviac catheter (Bard Access Systems, Salt Lake City, Utah, USA) after venotomy. The catheter needed to be secured to the distal part of IMV with silk tie whereas the proximal part of IMV would be ligated. It was important to ensure the precise tension of anchorage to prevent dislodgement without impeding its future removal. Heparinized saline infused via the IMV catheter during the early postoperative stage aimed to increase the PV flow and to lower the risk of thrombosis. If PV flow remains suboptimal or occluded despite above measures, a wall stent (Wallstent; Boston Scientific, Natick, MA, USA) would be placed to bypass the sites of narrowing. The technique of intra-operative vascular stenting for PV has been described in our earlier report [9].

For pediatric LDLT recipients whose vascular status of the graft was satisfactory, the routine postoperative anticoagulation required intravenous heparin infusion to achieve activated prothrombin time at 1.5–2 times the normal range for 5–7 days. However, in view of the increased risk of PVC, the seven patients in this series were anticoagulated with the same regime of intravenous heparin infusion followed by oral Dipyridamole (Persantin;

Boehringer Ingelheim, Ingelheim am Rhein, Germany) for 3 months. All patients received daily Doppler ultrasonography (DUS) surveillance during the first postoperative week. Thereafter, if the condition stabilized, the frequency of DUS monitoring would be tapered down till discharge.

Results

The mean age of the seven patients was 12.3 months (range 9–18 months). All of them had ESLD secondary to biliary atresia. All patients received left lateral segment graft. The recipient weight, graft weight, graft-to-recipient weight ratio, main PV diameter, and PV flow velocity on preoperative assessment are shown in Table 1.

To exclude outflow obstruction, when the low PV flow velocity was detected, the hepatic vein flow and central venous pressure were assessed. For all seven patients, the hepatic vein flow velocities were within normal range with biphasic or triphasic waveforms. Their central venous pressure ranged from 8 to 18 mmHg.

The intra-operative findings and interventions for decreased PV flow are shown in Table 2. Patient 1 had immediate re-laparotomy following the detection of low PV flow velocity after abdominal closure. The graft was re-positioned with Foley catheter which was later removed on postoperative day (POD) 14. An IMV catheter was also placed to reinforce PV flow. It was removed on POD 48. For patient 2, the IODUS after PV reconstruction showed absent PV flow. The PV flow was re-established after thrombectomy. However, patient developed HA thrombus and underwent HA thrombectomy on POD 4. Subsequently, she recovered well from these events. For patient 3, the IMV cannulation and endovascular PV stenting were performed simultaneously in view of her portovenography. A 6 Fr angiosheath was inserted

Table 1. The relevant preoperative demography.

Patients	Gender	Age at LT (months)	RW (kg)	GW (g)	GRWR (%)	Native liver weight (g)	Preop PV diameter on DUS (mm)	Preop PV diameter on CT (mm)	Preop PV hepatopedal flow (cm/s)
1	F	11	7.8	273	3.5	592	Not seen	3.7	Stasis
2	F	9	7.1	283	3.99	426	5.4	5.2	-5.5
3	F	9	6.8	270	3.97	448	5.3	3.6	7.9
4	F	11	8	253	3.16	431	Not seen	3	Stasis
5	F	16	6.7	232	3.46	441	4	2.5	Stasis
6	Μ	18	7.8	276	3.56	432	4.1	4	8
7	Μ	12	8	241	3.01	393	3.5	3	6

LT, liver transplantation; RW, recipient weight; GW, graft weight; GRWR, graft-to-recipient weight ratio; Preop, preoperative; PV, portal vein; DUS, Doppler ultrasonography; CT, computed tomography scan.

Table 2. ⊤	he	intra-operative	PV	events	and	interventions.
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Patients	Time of detection	PV flow before intervention (cm/s)	Interventions	PV flow after intervention (cm/s)	Outcome	Follow-up duration (months)
1	AC	6.4	Graft reposition with Foley catheter IMV cannulation	11.6	Uneventful	17
2	PVR	0	PV thrombectomy	19	HA thrombectomy on POD 4	21
3	HAR	0	IMV cannulation PV stent	14	Uneventful	23
4	AC	6	PV thrombectomy IMV cannulation	13.2	PV stent on POD 1	21
5	HAR	0	PV thrombectomy	12.2	Recurrent PV occlusion HA thrombosis	1
6	AC	0	PV thrombectomy IMV cannulation	15	Recurrent PV occlusion HA thrombosis	1
7	HAR	0	PV anastomsis revision IMV cannulation	12	Uneventful	72

PV, portal vein; AC, abdominal closure; IMV, inferior mesenteric vein; PVR, portal vein reconstruction; HA, hepatic artery; HAR, hepatic artery reconstruction; POD, postoperative day.

into left PV of the implanted graft via the stump of segment 4 PV branch. A 7 mm × 5 cm wallstent was placed from the intrahepatic PV to main PV near the splenomesenteric junction. Her IMV catheter was removed on POD 51. Patient 4 underwent immediate re-laparotomy, PV thrombectomy, and IMV cannulation when IODUS showed absent PV flow after abdominal closure. On POD 1, as follow-up DUS indicated absent PV flow, portovenograpphy was performed via the IMV catheter and demonstrated a long segment narrowing of PV from the splenomesenteric junction to the anastomotic site. A 7 mm × 4 cm wallstent was introduced into the graft intrahepatic PV whereas another 7 mm × 5 cm wallstent was placed from umbilical portion to splenomesenteric junction. Thereafter, adequate PV flow was re-established. Her IMV catheter was eventually removed on POD 42. For patient 5, although her PV flow was restored after thrombectomy and ligation of collateral shunts, she developed recurrent PV thrombus on POD 1. The subsequent DUS after PV thrombectomy and anastomosis revision demonstrated satisfactory PV flow. However, HA thrombosis was noted on POD 7 and 14. Despite multiple attempts of HA thrombectomy and redo-anstomosis, she died of graft failure secondary to HA thrombosis on POD 21. Patient 6 had PV thrombectomy and IMV cannulation during transplantation. His postoperative course was complicated by the dislodgement of IMV catheter, intraabdominal bleeding and HA thrombosis. He was treated with IMV catheter replacement, revision of PV anastomosis, ligation of collateral vessels, and HA thrombectomy. His follow-up DUS indicated adequate PV flow. Unfortunately, he died of multi-organ failure on POD 23. For patient 7, his PV flow improved and maintained after successful revision of PV anastomosis coupled with IMV cannulation.

The five surviving patients were followed up clinically and with regular DUS surveillance for 17–72 months (mean 30.8 months, median 21 months). Their latest DUS studies demonstrated patent PV with good flow. On comparison, for the 58 pediatric LDLT patients who had presented with satisfactory PV flow during the intra-operative and early postoperative stages, five patients developed late PVC at 1–3 years after transplantation. The overall incidence of PVC for pediatric LDLT patients was 12% (8/65).

Discussion

In this series, different intra-operative, on-table methods were employed to improve PV flow. They consisted mainly of surgical techniques, such as graft re-positioning, PV thrombectomy, ligation of collateral vessels, and anastomosis revision. These remedies can be augmented with endovascular interventions, such as IMV cannulation and intraluminal PV stent.

The characteristics of our patients are consistent with the reported risk factors for PVC, namely, young age, biliary atresia as primary disease, small PV size and slow or hepatofugal PV flow [3,4,6,13,14]. Previous investigators have described various techniques for PV reconstruction, including the use of vascular graft. Although the cryopreserved vascular grafts have been frequently utilized for outflow reconstruction in our center, we do not apply this technique for PV reconstruction because of the higher pressure present within the portal system. This is echoed by the finding of Buell et al. which reported an elevated risk of PVC associated with cryopreserved venous conduit [18]. More specifically, in a study looking at LDLT patients with late-onset PVCs, Kyoden et al. found a higher usage rate of cryopreserved vein graft [19]. Although fresh vascular graft provides a preferable alternative, its availability is limited by the scarcity of deceased donors. The "branch-patch" reconstruction, in a study by Saad et al., has been shown to associate with lower postoperative PV flow because of inferior quality of recipient PV [2]. We, however, have not encountered this finding. In our experience, it is still the method of choice for PV reconstruction because it negotiates well between the two size-mismatched ends of anastomosis.

The intra-operative management for suboptimal PV flow should begin with the IODUS after vascular reconstruction. DUS is a noninvasive, rapid, and real-time assessment of the vascular patency of liver graft [15]. Although a recent report has questioned the reliability of using intra-operative PV peak velocity as a predictor for postoperative PVC [20], we still recommend PV flow velocity of less than 10 cm/s as an indication for immediate intervention [6]. The importance of early detection of PVC cannot be overemphasized. The loss of graft and mortality related to PVC can be avoided if early detection and timely treatment are instituted [8,21,22]. Therefore, when suboptimal PV flow is shown in IODUS, the immediate salvage procedure should be guided by the intra-operative finding of the possible attributing factors.

When attempting to optimize the PV flow, graft position adjustment should be the first consideration. The liver grafts for pediatric patients are smaller comparing with their adult counterpart. The free movement of the graft predisposes it to the risk of vessel kinking and twisting. Previous authors have reported maneuvers, such as fixation of round ligament, placement of bowel, and additional side-to-side cavo-cavostomy to stabilize the graft [23–25]. The use of tissue expander was first reported by Inomata *et al.* [23]. We have modified this technique with a Foley catheter [26]. The placement of prosthesis device provides support for the graft to stay in the desired position. In this series, it was performed in patient 1 with satisfactory outcome. On POD 14, the Foley catheter was gradually deflated and removed as graft had been fixated by adhesion.

When PV flow improved after graft repositioning, IMV cannulation can be considered as a supplementary measure to minimize the risk of PVC. Although the high graft-to-recipient weight ratio and pathological PV quality hinder the flow and result in PV thrombosis, the heparin infusion via the IMV catheter plays a role in augmenting the PV flow. In this series, IMV cannulation was performed in five patients. Four of them had good outcome with no late PVC. Unfortunately, one patient had hemoperitoneum following IMV catheter dislodgement. The beneficial effect of IMV infusion may still be controversial as some investigators have suggested that the limited flow volume of infusion would not have significant impact on the outcome. Nevertheless, we believe that in pediatric patients, this remains to be an effective intervention to selectively increase the PV flow velocity. In addition, this catheter also provides access for intra-operative portovenography to ascertain the cause of suboptimal PV flow. During the postoperative course, the IMV catheter also facilitates angiographic assessment of PV patency as well as administration of therapeutic thrombolytic agent if PV thrombosis is present.

If the reconstructed PV is noted to be redundant or angulated, the PV anastomosis needs to be revised to establish a smooth course of flow. The narrowing at the anastomotic sites only becomes apparent after reperfusion. It decreases the PV flow and further leads to thrombosis and stenosis. Revision of anastomosis is therefore necessary. When IODUS or direct examination of PV indicates possible thrombus, thrombectomy needs to be performed. Depending on the sites of PV thrombus, it should be removed via portal venotomy at either graft or recipient side. If thrombus extends beyond the anastomotic site, anterior wall of anastomosis can be opened up for thrombectomy.

In pediatric patients with biliary atresia, the long standing hepatobiliary disease predisposed them to neoformation of portosystemic collateral circulations and gives rise to poor or even heptofugal flow. At preoperative assessment, dilated coronary vein and recanalized umbilical vein are often noted. At intra-operative portovenography, these can also be appreciated. If these findings are present in patients with decreased PV flow after graft reperfusion, collateral shunts must be ligated to improve PV flow.

The role of intra-operative intraluminal PV stenting at the time of transplantation has been reported in numerous studies [9,11,27,28]. It showed good success rate by supporting the sclerotic and hypoplastic PV and ensuring its lumen patency. However, endovascular stenting should

remain as the final remedy to be considered only when all other interventions have failed to improve PV flow. This is because although it addresses the primary issue of weakened PV wall, it is irreversible. Its long-term impact on children who still have significant potential for growth of organs and vessels still remains unknown. In this series, one patient had stent placed at the time of transplantation whereas the other had it placed when PV develop total occlusion on POD 1. Their subsequent DUS surveillance has shown patent PV with satisfactory flow. A notable finding for patient 3 was that a recent DUS performed 2.5 years after transplant has indicated a growth in diameter of the stented PV from the initial 5.8 mm during the early post-transplant period to 7.1 mm on follow-up. Currently in our center, the interventional radiology team would be at standby at the time of transplantation when pretransplant assessments demonstrate significantly narrow PV lumen or abnormal PV flow.

In conclusion, when suboptimal PV flow velocity is detected on routine IODUS after vascular reconstruction or abdominal closure, the priority of management should be on the identification and the correction of attributing causes. It may require one or several remedies. The approach should begin with conservative measures, such as graft re-positioning, followed by more invasive options such as anastomosis revision or endovascular stenting. With this strategy, we have been able to re-establish satisfactory PV flow at the time of transplantation. Nevertheless, stringent postoperative monitoring of PV status ought to be performed for these high-risk patients.

Authorship

CCW, CLC and YFJ: designed the study. SHW, YWL and TSL: collected data. CCL, WFL and BJ: analyzed data. CCY and TYC: performed the study; TLL, LWC and AMC: performed the study and co-write the manuscript.

Funding

This article has no funding.

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