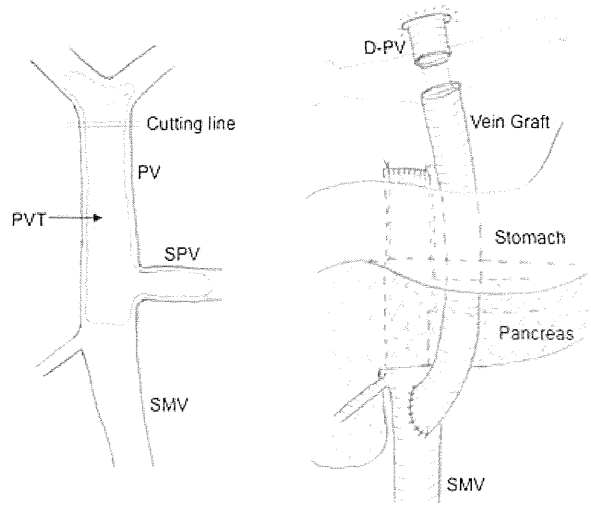

Fig. 2 Scheme of a jump graft. When irremovable portal vein thrombosis reaches the confluence of the splenic vein, a vein graft is interposed between the superior mesenteric vein and the donor portal vein through a route anterior to the pancreas and behind the stomach

vein. We performed a splenectomy simultaneously for all patients with hepatitis C virus-related disease to prevent thrombocytopenia induced by the use of interferon therapy against post-transplant recurrence of hepatitis C infection. We confirmed the front flow of the PV by clamping the collateral veins and flushing the vessel that was prepared to anastomose with the PV of the liver graft just before anastomosis. After reconstructing all the vessels, reperfusing the liver graft, and when the PV pressure read ≥15 mm Hg, a splenectomy was carried out to decrease PV pressure and prevent small-for-size syndrome. Doppler ultrasonography (US) after reperfusion of the graft liver estimated portal flow, arterial flow, and hepatic vein flow.

Anticoagulation therapy after LDLT

Our basic protocol after LDLT for the patients who underwent portal reconstruction for PVT had no anticoagulant therapy. Only patients with good coagulation (prothrombin time-international normalized ratio [PT-INR] < 1.5) or slow portal flow (velocity < 10 cm/s) were administered intravenous heparin at the dose of 5 $U \cdot kg^{-1} \cdot h^{-1}$ during the first week under careful observation for bleeding, and thereafter added oral warfarin sodium. Heparin was discontinued after their PT-INR reached the target range 1.5–2.0. Patients were weaned off warfarin sodium 12–24 months after the absence of rethrombosis was confirmed in outpatient clinic. We checked the blood flow of the PV, hepatic artery, and hepatic vein with Doppler US twice a day for the first 2 weeks and once a day for the next 2 weeks following LT. When any suspicious findings of portal rethrombosis or stenosis emerged, we made a precise diagnosis using enhanced CT.

The protocols described in this report were approved by the ethics committee of the Kyoto University Hospital, and were performed in accordance with the ethical standards of the Declaration of Helsinki established in 1975.

Results

Portal vein thrombosis was found in 48 (17%) of 282 consecutive patients who underwent adult LDLT. Patient

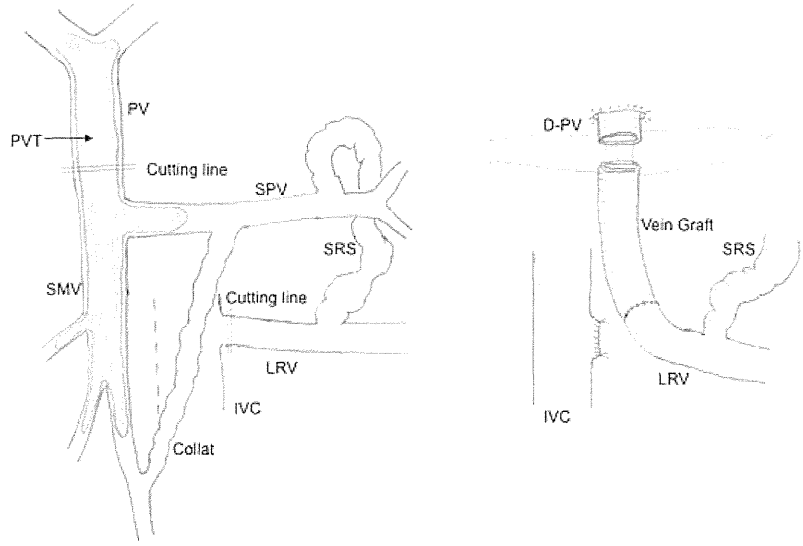

Fig. 3 Scheme of a reno-portal anastomosis. When a thrombosis obstructs the superior mesenteric vein as well as the portal vein trunk and all portal blood flows into the left renal vein via splenorenal shunts, we interpose a vein graft between a recipient's left renal vein and a donor portal vein. *Collat* collateral vein

demographics are shown in Table 1; none of the 21 patients with fulminant hepatic failure had PVT. Child–Pugh scores, model for end-stage liver disease (MELD) scores, ABO-compatibility, and graft recipient weight ratio (GRWR) were comparable between patients with and without PVT. Of the 48 PVT patients, 23 were men and 25 were women. Graft types were as follows: 48 LDLTs including 23 left lobe grafts and 25 right lobe grafts. PVT grades according to Yerdel's classification were as follows: 15 cases of grade I, 20 cases of grade II, 12 cases of grade III, and one case of grade IV. PV reconstructions were conducted as follows: thrombectomy/thromboendovenectomy in 30 cases, replaced graft in seven, jump graft between the recipient's SMV and the donor PV in seven, and interposed graft between the recipient's left renal vein and the donor PV in one; three cases did not require surgical intervention owing to minimal thrombosis (Table 2). Among 48 patients with PVT, four patients had a medical history of splenectomy prior to LT, and 29 patients underwent splenectomy simultaneously with LDLT, including 12 cases for portal pressure control and 17 for hepatitis C virus-related disease according to the algorithm mentioned above. The

Table 1 Recipient demographics, graft type and survival

			Total $n = 282$	w/o PVT $n = 234$	with PVT $n = 48$	P-value
Age (years)			51.0 ± 12.5	50.8 ± 12.4	52.1 ± 12.7	0.39[a]
Sex (n)		Male	138	115	23	
		Female	144	119	25	0.88[b]
Primary disease (n)		HBV-LC	15	12	3	
		HCV-LC	44	36	8	
		Non-B non-C-LC	26	19	7	
		HBV-HCC	25	18	7	
		HCV-HCC	52	44	8	
		Non-B non-C-HCC	4	4	0	
		PSC	7	7	0	
		PBC	30	25	5	
		AIH	7	6	1	
		PBC+AIH	3	2	1	
		IPH	3	1	2	
		Budd-Chiari	5	5	0	
		Biliary atresia	15	14	1	
		Metabolic disorder	7	6	1	
		Fulminant hepatic failure	21	21	0	
		re-liver transplant	16	12	4	
		others	2	2	0	0.22[b]
Child–Pugh score			10.1 ± 2.3	10.1 ± 2.3	10.4 ± 1.9	0.65[a]
MELD score			19.8 ± 9.5	19.8 ± 9.9	19.8 ± 7.7	0.66[a]
ABO compatibility (n)		Identical	162	132	30	
		Compatible	47	42	5	
		Incompatible	73	60	13	0.44[b]
Graft type (n)	LDLT	Lt. lobe	116	93	23	
		Rt. lobe	154	129	25	
		Rt. lateral sector	10	10	0	
	Domino	Whole	2	2	0	0.38[b]
GRWR (%)			0.93 ± 0.21	0.93 ± 0.21	0.91 ± 0.20	0.63[a]
Survival (%)		1Y	78	77	81	
		3Y	75	74	81	
		5Y	74	73	81	0.30[c]

Statistical analysis by
[a] Wilcoxon,
[b] Pearson and
[c] Log-rank test.

Table 2 Portal vein reconstruction according to portal vein thrombosis (PVT) grade

Grade (*n*)	Procedure (*n*)
I (15)	NT (3), TB (12)
II (20)	TB (15), RG (5)
III (12)	TB (3), RG (2), JG (7)
IV (1)	RP (1)

Grade: Yerdel's classification
JG jump graft, *NT* no treatment, *RG* replaced graft, *RP* renoportal anastomosis, *TB* thrombectomy

intraoperative PV pressure of these 53 patients was 21.0 ± 5.9 mm Hg immediately after laparotomy and 14.1 ± 3.5 mm Hg before finishing the operation. Following PV reconstruction for thrombosis, post transplant PV complications occurred in eight (17%) of 48 cases, including four cases each of portal rethrombosis and PV stenosis (Table 3). Among 33 cases after splenectomy, PV rethrombosis and stenosis occurred in three cases each. PV rethrombosis occurred by postoperative day 5 in all but one case, which occurred at postoperative month 7. We treated rethromboses immediately with PV re-reconstructions in two cases, a PV infusion with urokinase and heparin in one case, and balloon dilatation and stent placement in one late-onset case. On the other hand, PV stenoses occurred at a later stage, after 3 or more months following surgery, in all four cases. We treated stenosis with interventional radiology (IVR), which included ballooning in all four cases followed by stent placement in two cases because of re-stenosis. Anticoagulation therapy with warfarin sodium was administered to all eight patients for at least 6 months following recanalization of the PV, especially for over 20 months following stent placement. All eight patients with complications survived after proper surgical intervention or IVR (Table 4). Overall 1- and 5-year survival rates of 48 patients with PVT at the time of LT were comparable to 234 patients without PVT (1 year; 81% vs. 77%, 5 years; 81% vs. 73%) (Fig. 4).

Table 3 Incidence of post-transplant complications

PV reconstruction	Complications			
	Rethrombosis	Stenosis	None	Total
Thrombectomy	4	3	23	30
Replaced graft	0	0	7	7
Jump graft from SMV	0	1	6	7
Renoportal anastomosis	0	0	1	1
No treatment	0	0	3	3
	4	4	40	48

Data are number of patients.

Discussion

Portal vein thrombosis is a common complication in patients with end-stage liver cirrhosis. LT used to be contraindicated in PVT patients, even as a last resort potentially life-saving treatment option. Recent reports have shown how innovative surgical techniques have expanded the indication of LT in patients with PVT, and that 51/447 (11%) and 69/537 (13%) patients who underwent DDLT experienced PVT as a complication [1, 4]. Since adequate PV blood flow is essential for liver graft function after transplantation, many reports have described surgical procedures to introduce sufficient portal blood flow to liver grafts, such as thrombectomy, jump grafts, renoportal anastomosis, portocaval hemitransposition, and PV arterialization [4–8, 22, 23]. In the LDLT setting, there exist additional challenges in acquiring appropriate vein grafts, obtaining adequate portal flow, releasing portal hypertension, and preventing the development of small-for-size syndrome. Few papers have reported a systematic strategy and LDLT outcomes for patients with PVT [9, 15–17].

Table 4 Patients with post-transplant complication after portal vein reconstruction for portal vein thrombosis (PVT)

Case	Grade	Reconst.	Compli.	Time	Treatment	Prognosis
1	II	TB	Re-thr	POD 1	Re-anastomosis	1.5 years alive
2	II	TB	Re-thr	POD 3	Re-reconstruction with jump graft	6.4 years alive
3	II	TB	Re-thr	POD 5	Portal infusion with urokinase and heparin	5.9 years alive
4	II	TB	Sten	POM 3	Ballooning and placing stent	3.9 years alive
5	I	TB	Sten	POM 6	Ballooning	5.8 years alive
6	III	TB	Re-thr	POM 7	Ballooning and placing stent	3.8 years alive
7	III	JG	Sten	POM 12	Ballooning	5.4 years alive
8	III	TB	Sten	POM 13	Ballooning and placing stent	4.2 years alive

Grade: Yerdel classification, Reconst: PV reconstruction in liver transplantation (LT), Compli: complication after LT
JG jump graft, *POD* postoperative day, *POM* postoperative month, *Re-thr* rethrombosis, *Sten* stenosis, *TB* thrombectomy
Prognosis: prognosis after LT

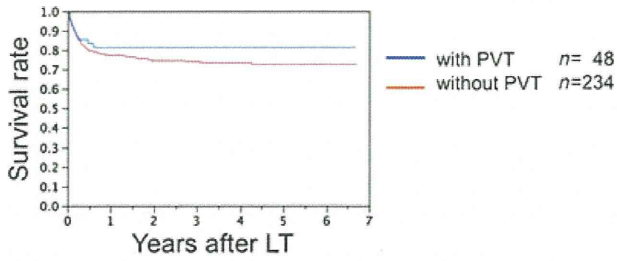

Fig. 4 Kaplan–Meier analysis of patients with or without portal vein thrombosis (PVT) after liver transplantation. The overall survival of patients with pre-existing PVT is comparable with patients without PVT, which is attributed to our treatment strategies for PVT in liver transplantation and postoperative management of these patients

We attempted to expand the indication of LDLT in patients with PVT followed by individual reconstruction of PVs according to thrombosis extent, hemodynamic modification, and diligent postsurgical follow-up. Between April 2006 and December 2011, PVT was found in 48 (17%) of 282 consecutive patients who underwent adult LDLTs at our institution. Child–Pugh scores and MELD scores of patients with PVT were unexpectedly comparable to those of patients without PVT. None of the 21 fulminant hepatic failure patients had PVT. In other words, PVT, which developed over a long period, did not always lead to deterioration in liver and renal function.

Precise PVT evaluations by imaging before LT were necessary to plan for PV reconstruction. Dynamic MD-CT was most useful in diagnosing PVT and collateral veins. However, patients with renal dysfunction or contraindications against the contrast medium underwent MRI [2]. Mural thin PVTs, which were not detected by imaging, were sometimes found in PV stumps during surgery. Although Moon et al. reported the efficacy of intraoperative cine-portograms to evaluate remnant PVT and portosystemic collaterals after thrombectomy [24], we had experienced poor images from portograms obtained with a conventional C-arm fluoroscope at our institution. A hybrid operating room equipped with a high-quality cine-angiograph must be useful for obtaining intraoperative portograms.

Grade I/II PVT limited to the PV trunk, which obstructed the PV either completely or incompletely, was usually removed using a previously reported method [9]. It should be attempted to use the original PV during reconstruction, unless remnant thrombosis, PV stenosis, or insufficient forward blood flow is present. We selected the PV system, such as the PV, the SMV, or even collateral vessels, for anastomoses with the PV of liver grafts, because portocaval hemitransposition and PV arterialization aim to supply nonanatomical blood to liver grafts without the influx of hepatotrophic factors from the intestines and the release of portal hypertension [11, 23, 25, 26]. We therefore rarely selected these procedures.

Whenever PVT or stenosis extended into the peripheral branch of the SMV, it was hard to determine appropriate portal blood flow. In rare cases, the left gastric vein dilated and flowed backwards as a collateral vein; thus, we anastomosed a vein graft to the left gastric vein in a side-to-end fashion. Subsequently, the vein graft was anastomosed to the PV of the graft liver (Fig. 5). This method was used as a last resort to save portal flow because the wall of the collateral vein was usually too thin and fragile to perform an anastomosis. One

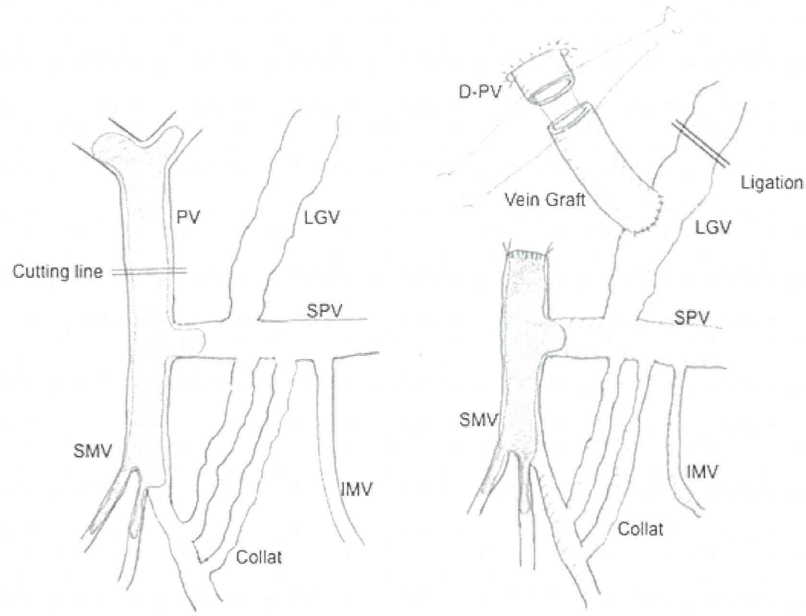

Fig. 5 Scheme of reconstruction using the left gastric vein. When a portal vein thrombosis extends into the peripheral branch of the superior mesenteric vein and we can only find appropriate portal flow in the left gastric vein, we interpose a vein graft between the left gastric vein and the donor portal vein. *IMV* inferior mesenteric vein, *LGV* left gastric vein

patient underwent this method after enrolling in this study. When PVT extended into the peripheral branch of the SMV and no proper collateral flow was found, we determined that the patient was not a suitable candidate for LT.

The transplant surgeon has to make several preoperative plans, and consider available vein grafts from the recipient and/or the donor, because it is difficult to take vein grafts with suitable caliber and length from a living donor while maintaining donor safety in LDLT. The recipient's external iliac veins, internal jugular veins, umbilical portions of PVs, and left renal veins are possible candidates for vein grafts. It is relatively easy to harvest large and long vein grafts from a deceased donor in DDLT, such as common iliac veins and internal jugular veins. Although cryopreserved vein grafts are useful in long arrangements of PV reconstructions, it is difficult to prepare them because they are scarce and expensive, and their patency is controversial [14, 27, 28]. Multivisceral transplantations are possible in patients with diffuse portomesenteric thromboses [29]. However, this is difficult to accomplish in Japan because of the extreme shortage of deceased donors. An acceptable algorithm of PV reconstruction procedure according to PVT grade is shown in Table 5. A transplant surgeon, however, has to select a procedure from the viewpoints of available vein grafts and collateral veins as well as PVT grade in each patient.

We previously reported an algorithm for portal pressure control that targets ≤15 mm Hg at the end of transplantation [21]. In the case of small-for-size liver grafts, a splenectomy is performed to decrease PV pressure. This study showed that splenectomy would not be a risk factor of portal rethrombosis or stenosis after reconstruction of the PV, because these complications occurred in 6/33 and 2/15 patients with pre-existing PVT who underwent LDLT with and without splenectomy, respectively (P= 0.67 by Pearson analysis). Portosystemic shunts should be obstructed to prevent the "stealing" of blood flow after LDLT [18, 19] while monitoring PV flow and pressure.

Diligent follow-up and timely treatment of complications are crucial when reconstructing PVs. We performed Doppler US twice a day for 2 weeks after LT. Dynamic CT was performed when rethrombosis or stenosis was suspected on US. In some cases, rethrombosis that occurred in early post-transplantation period required immediate treatment, including re-reconstruction of the PV. In other cases, PV stenosis occurred in the later period after 3 months, and warranted treatment with balloon dilatations in all four cases followed by stent placement in two cases. These treatments for post-transplant complications were necessary to save both liver grafts and patient lives. Our strategies to treat PVT associated with LDLT led to survival rates in patients comparable to those in patients without PVT.

In conclusion, although LDLTs are no longer contraindicated in patients with PVT, transplant surgeons have to design precise technical surgical strategies based on the extent of PVT to acquire sufficient portal blood flow and proper portal pressure to the liver graft. Patent portal flow is the result of diligent follow-up and the timely management of postsurgical complications, which also leads to long-term patient survival. We propose our strategies as the standard methods for PV reconstruction in adult LDLT for patients with PVT.

Table 5 Algorithm of portal vein reconstruction according to portal vein thrombosis (PVT) grade

Procedure	PVT Grade			
Choice	I	II	III	IV
1st		TB		
2nd		RG		
3rd			JG	
4th				RP, LG
5th				MV, CI

CI contraindication, *JG* jumping vein graft, *LG* anastomosis with left gastric vein using vein graft, *MV* multivisceral transplantation, *PVT grade* Yerdel's classification, *RG* replaced vein graft, *RP* renoportal anastomosis using vein graft, *TB* thrombectomy

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Conflict of interest None declared.

Author contributions Study design: A Mori and S Uemoto. Acquisition of Data: A Mori, T Kaido, K Ogawa, Y Fujimoto, T Uemura, E Hatano and H Okajima. Analysis: A Mori, T Iida and J Iwasaki. Manuscript drafted by: A Mori.

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ELSEVIER

Organ Donation From Brain-Dead and Circulatory-Dead Donors: Single-Institution Experiences

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ABSTRACT

Although the number of organ donations is extremely small in Japan, organ donation from brain dead (DBD) donors is increasing since the revised Law for Organ Transplantation was enacted on July 17, 2010. In our institution, organ donations had so far been performed from 247 donors (DCD 242, DBD 5), which is the largest number in Japan. In this study, we analyzed the status of organ donation before and after the enforcement of the revised law. After the enforcement of the revised law, the option of organ donation was shown to the more families of potential donors by the doctors or donor coordinators. However, the final number of donors was almost the same. The frequency of DBD donors of all donors increased (33.3%) as compared to 9.1% before the enforcement of the revised law. Reasons for rejection of donation from donor families were mainly based on the lack of understanding of brain death. To increase organ donation, we should promote social recognition of brain death, having the Organ Donation Card, and discussion of organ donation in each family.

IN Japan, the Organ Transplantation Act was enforced in 1997. The number of the brain dead (DBD) donors, however, remained small, from 3 to 13 per year for over 10 years [1]. The number of DBD donors, however, has been increasing since the revised Law for Organ Transplantation was enforced on July 17, 2010 (Fig 1) [1]. This has contributed to the increased number of organ transplantations such as heart, lung, liver, pancreas, and small intestine. Although the number of kidney transplantations from DBD donors increased (6 to 7 times per year), as well as other organ transplantations, the total number of kidney transplantations did not increase because of the decreased number of DCD donors.

In our institution, organ donation and organ transplantation have been promoted for more than 30 years. Organ donations were performed from 247 donors (DCD donors 242, DBD donors 5) between January 1, 1979, and December 31, 2012, which is the largest number in Japan [2]. In the present study, the status of organ donation before and after the enforcement of revised Organ Transplantation Act in our institution was compared.

MATERIALS AND METHODS

Organ donations in Fujita Health University Hospital from approximately January 2008 to December 2012 were analyzed using the information from the patient case cards in the Neuro Care Unit (NCU) at our institution.

Patients were divided into 2 groups according to before or after the revision of the Organ Transplant Act: group 1 (N = 129), patients who died in the NCU between January 1, 2008, and July 16, 2010; group 2 (N = 123), patients who died in the NCU between July 17, 2010, and December 31, 2012. The number of deceased patients, potential donors, the potential donors whose families were shown the option of organ donation, and the donors were compared between the 2 groups.

Brain death was confirmed by the criteria for brain death in Japan: (1) coma, (2) apnea, (3) absence of brain stem reflux (pupil, ocular, corneal, pharyngeal, tracheal), (4) flat blain waves (electroencephalogram). In cases of organ donation, the apnea test must be added according to the criteria for legal brain death in Japan.

RESULTS

In group 1, 61 patients (47.3%) were considered to be the potential donors out of 129 deceased patients. The

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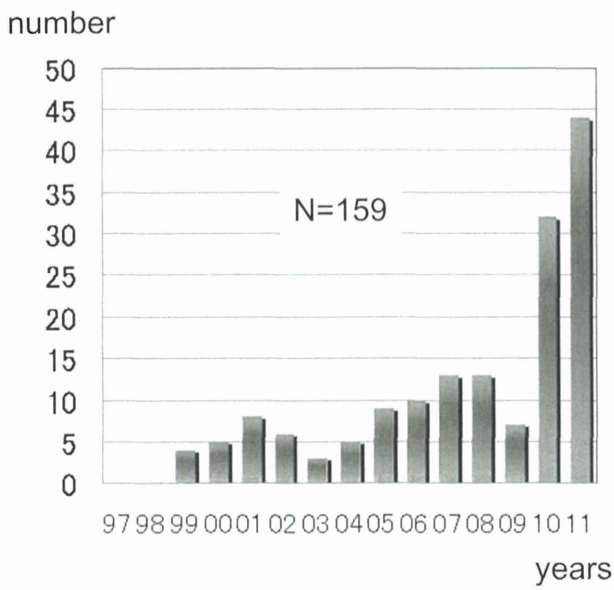

Fig 1. Changes in the number of brain dead donors in Japan (1997.10 ~ 2011.12).

option of organ donation was presented to the families of 27 patients (44.3%) by the doctors or donor coordinators. Of these families of 27 patients, 11 families (40.7%) consented to organ donation from the patients (Fig 2). In group 2, 48 patients (39.0%) were potential donors out of 123 deceased patients. The option of organ donation was presented to the families of 34 patients (70.8%). Out of the families of 34 patients, 12 families (35.2%) consented to organ donation from the patients (Fig 2). After the revision of the Organ Transplant Act, the option for organ donation was presented to more families of potential donors (71%) as compared to before the revision of the Organ Transplant Act (44%). The final number of donors, however, showed no difference between groups 1 and 2.

In group 1, only 1 of 11 donors (9.1%) was a DBD donor, and the other 10 were DCD donors. In contrast, 4 donors (33.3%) were DBD donors in group 2. These results were comparable to the data of organ donation in Japan. The outcomes of organ transplantations from 5 DBD donors are shown in Table 1. Twenty-three organs were transplanted into 20 patients. Although 2 patients who underwent liver transplantation died, the other 18 patients were alive with functioning grafts.

The reason for donor family agreement to donate was "contribution for other patients" in 5 cases, "donor's will (donor card)" in 4 cases, "no wish for the donor to survive after brain death" in 2 cases, "organs will live in other patients" in 1 case. The reason for donor family refusing to donate was "no wish of the family for more surgery" in 6 cases, "unknown donor's will" in 4 cases, "emotional reject" in 3 cases, "need for the time of parting" in 3 cases, "no consensus among the family" in 2 cases, "hope of more resuscitation" in 2 cases and others in 4 cases.

DISCUSSION

In Japan, under the Organ Transplantation Act, which has been in place since 1997, an extremely small number of organ transplantations from DBD donors was performed. Under the revised Law for Organ Transplantation, which was finally enacted on July 17, 2010, the number of organ transplantations increased 7- to 8-fold because only the agreement from donor families was legally needed for organ donation.

In the NCU in our institution, discussing organ donation with donor families increased from 44.3% to 70.8% after

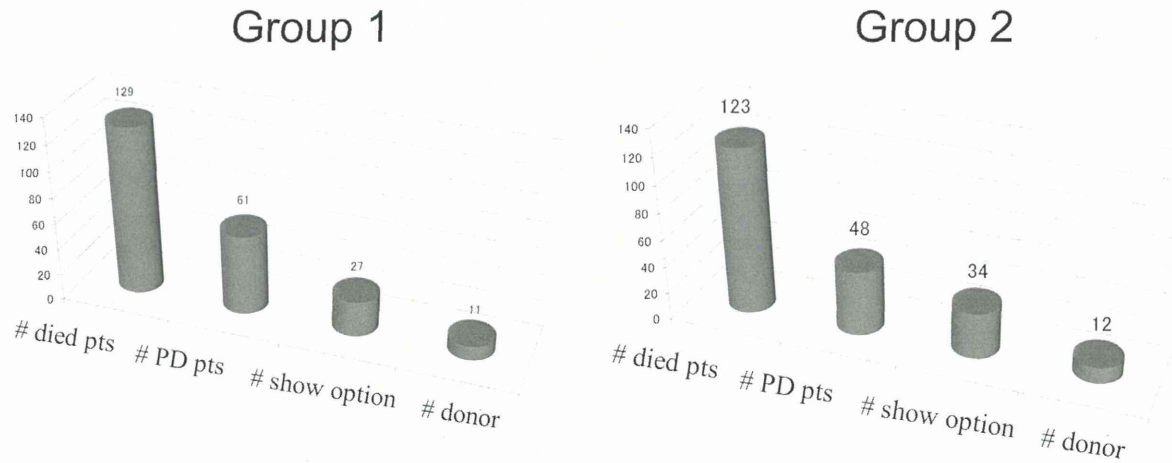

Fig 2. Number of died patients, the potential donors (PD pts), the potential donors whose families were shown the option of organ donation (show option) and the donors in Group 1 (from January 1, 2008 to July 16, 2010) and Group 2 (from July 17, 2010 to December 31, 2012) – Fujita Health University Hospital.

Table 1. Outcome of Organ Transplantations From 5 DBD Donors, Fujita Health University Hospital

Number	Organs	Primary Disease	Patient Survival	Graft Survival
64	Liver	Fulminant hepatitis	○	○
	Kidney	CGN	○	○
	Kidney	CGN	○	○
89	Heart	Dilated cardiomyopathy	○	○
	Lung	Pulmonary hypertension	○	○
	Liver	HB cirrhosis	X	X
	Pancreas and kidney	type 1 DM + ESRD	○	○
	Kidney	CGN	○	○
114	Heart	Dilated cardiomyopathy	○	○
	Lung	Cystic fibrosis	○	○
	Liver	Hepatic failure	X	X
	Pancreas and kidney	Type 1 DM + ESRD	○	○
	Kidney	CGN	○	○
133	Lung	Idiopathic interstitial pneumonitis	○	○
	Pancreas and kidney	Type 1 DM + ESRD	○	○
	Kidney	CGN	○	○
196	Liver	Wilson's disease	○	○
	Pancreas	Type 1 DM	○	○
	Kidney	FGS	○	○
	Kidney	IgAN	○	○

the revision of the law. The understanding of DBD donors proceeded, and the importance of presenting the option to donor families was well recognized by medical staff, including doctors, nurses, and coordinators. However, the consent rate for donation slightly decreased from 40.7% to 35.2%, and thus the final number of organ donations was almost equal between the 2 groups.

The reasons for refusal of donation by the donor families seemed to be based on a lack of understanding of brain death. Japanese people have been used to cardiac death for a long time. The first heart transplantation from a DBD donor in 1968 in Japan caused a social problem, and brain death became taboo for about 30 years. A lack of discussion about brain death with each family was another major factor for the rejection of organ donation by the family. This also may be because of a lack of understanding of brain death. In order to increase the number of organ donations, we should promote the social recognition of brain death and discussion of organ donation with each family.

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Long-Term Outcomes of Clinical Transplantation of Pancreatic Islets With Uncontrolled Donors After Cardiac Death: A Multicenter Experience in Japan

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ABSTRACT

Background. Pancreatic islet transplantation has emerged as an effective treatment for type 1 diabetes mellitus, but its use is limited due to an insufficient supply of cadaveric pancreata. In Japan, uncontrolled donors after cardiac death (DCD) are not deemed to be suitable for whole-organ pancreatic transplantation, and can provide a source of pancreas for islet transplantation. However, the long-term outcomes and utility of uncontrolled DCD in the clinical setting remain controversial. Here, we summarize the long-term outcomes of islet transplantation employing uncontrolled DCD as reported to the Japan Islet Transplantation Registry.

Methods. Sixty-four isolations and 34 transplantations of pancreatic islets were conducted in 18 subjects with type 1 diabetes mellitus under the cover of immunosuppression with basiliximab, sirolimus, and tacrolimus. All donors were uncontrolled DCD at the time of harvesting. The mean follow-up time was 76 months.

Results. Of the 18 recipients, 8, 4, and 6 recipients received 1, 2, and 3 islet infusions, respectively. Overall graft survivals (defined as a C-peptide level $\geq$0.3 ng/mL) were 72.2%, 44.4%, and 22.2% at 1, 2, and 5 years, respectively, whereas the corresponding graft survivals after multiple infusions were 90.0%, 70.0%, and 30.0%, respectively. Three of these recipients achieved insulin independence in 14, 79, and 215 days. Hb_{A1c} levels and the requirement of exogenous insulin were improved before loss of graft function. All recipients became free of severe hypoglycemia unawareness, however, at least 5 of 14 patients who had graft failure experienced recurrence of severe hypoglycemia after the loss of graft function.

Conclusions. Islet transplantation from DCD can relieve glucose instability and problems with hypoglycemia when the graft is functioning. However, islets from uncontrolled DCD may be associated with reduced long-term graft survival. Further improvements in the clinical outcome by modification of islet isolation/transplantation protocols are necessary to establish islet transplantation using DCD.

PANCREATIC islet transplantation has emerged as an effective treatment for type 1 diabetes mellitus (T1DM), but its use is limited due to an insufficient supply of cadaveric pancreata. Pancreatic islets are obtained from donors after brain death (DBD) all over the world, but access to DBD in Japan is quite rare. Also, pancreatic islet

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