involves surgery in the area surrounding the portal vein, the pancreas, and the spleen. Pancreatic fistula causes hemorrhage, abscess, etc., and may result in the death of the patient[6-8]. Only 1 previous study has reported 2 cases of leakage of pancreatic fluid after liver transplantation. In those cases, leakage of a mixture of pancreatic fluid and bile was observed at the anastomosis site of the bile duct after DDLT[9]. However, there is no clear consensus on the management of pancreatic fistula after liver transplantation. Generally, conservative therapy is the first line of treatment, and surgery is performed only when the patient does not respond to conservative therapy[6-8]. Recently, however, endoscopic treatment has attracted more attention because it is less invasive than surgical treatment. We present 2 cases in which endoscopic treatment was effective against refractory pancreatic fistulas that developed after LDLT.

CASE REPORTS

Case 1

A 61-year-old woman underwent LDLT for primary biliary cirrhosis; the graft was obtained from the left liver lobe of her son. Abdominal computed tomography (CT) performed on postoperative day (POD) 7 revealed a portal

5

vein thrombus; therefore, urgent exploratory laparotomy was performed. The thrombus was believed to have been induced by reduced portal blood flow, which was caused by splenorenal steal by an artificial shunt. After removing the thrombus, portal vein reconstruction was performed by using the right external iliac vein, and this procedure was followed by splenectomy. To increase the portal blood flow, a splenorenal shunt was ligated. The main pancreatic duct on the dorsal side of the pancreas was injured at the time of hemostatic manipulation; however, this injury was not identified immediately. CT performed on POD 13 revealed a hematoma at the lower edge of the pancreas. CT on POD 21 revealed that the hematoma under the pancreas had decreased in size (fig 1). The amylase level of the drainage fluid was 22690 IU/l; therefore, the hematoma at the inferior edge of the pancreas was considered to have ruptured because of a pancreatic leak. Another CT examination revealed fluid collection in the mesentery on the ventral side of the upper pole of the left kidney; therefore, open drainage was performed. To this end, a drain was placed at the tail of the pancreas and administration of octreotide was started. However, the leakage of pancreatic fluid from the drain did not stop. After several days, the patient's general status stabilized, but surgical treatment for

6

pancreatic fistula was still unsafe because of inadequate liver function. Therefore, the patient was discharged on POD 128 with the drain in place and was followed up. The patient was in a stable state at discharge. However, on POD 318, she was readmitted to our department because of fever. Examinations revealed that the drain at the tail of the pancreas had deviated and that the patient had developed liver necrosis, supposedly because of contact with the drain. On POD 320, we repositioned the drain by using a fluoroscope. A contrast test performed at that time revealed that the main pancreatic duct was completely disrupted (fig 2). The patient was diagnosed with refractory pancreatic fistula, and an endoscopic naso-pancreatic drainage (ENPD) tube was inserted to the proximal side of the leakage on POD 331 (fig 3); this procedure resulted in a remarkable decrease in drain output (fig 4) (fig 5). The ENPD tube was removed on POD 368, and the drains at the tail of the pancreas were removed on POD 371. The patient was discharged on POD 375 without abnormal fluid collection around the pancreas (fig 6). The patient is well without the recurrence of pancreatic fistula up to this time.

Case 2

7

A 58-year-old man underwent LDLT for cirrhosis C; the graft was obtained from the right liver lobe of his daughter. Because the portal vein was occluded by a thrombus, the portal and splenic veins were stripped off from the surrounding tissue and were exposed in order to remove the thrombus. However, the upper edge of the pancreatic head was injured during this process. The amylase level measured at the upper edge of the pancreatic drain was high on POD 1; therefore, the patient received octreotide on POD 2. On POD 5, the patient showed high fever and acute peritonitis. Therefore, an emergency exploratory laparotomy was performed. Because fluid collection was observed around the pancreas, drains were placed at both the right and left edges of the pancreas. On POD 21, the total output from the drain was 460 ml/day, and the amylase level of the drainage fluid was 166700 IU/l. The patient was diagnosed with high-output pancreatic fistula, and ENPD was performed on POD 22 (fig 7). The drain output decreased very rapidly (fig 8); therefore, the patient was allowed to consume solid foods on POD 49, and octreotide administration was stopped on POD 50. The ENPD tube was removed on POD 65, and the drains placed at the right and left edges of the pancreas were removed on POD 68 and POD 70, respectively. The patient was

8

discharged on POD 100 without abnormal fluid collection around the pancreas (fig 9). The patient is well and receiving a regular out-patient treatment showing no recurrence of pancreatic fistula.

DISCUSSION

Pancreatic fistula is primarily treated by conservative therapy, which includes rapid total infusion or enteral nutrition along with administration of octreotide. The recovery rate after conservative therapy ranges from 44% to 85% [6-8]; thus, a number of cases are not resolved by conservative treatment. Surgical treatment has been performed in such cases. However, surgical treatment is highly invasive and may lead to various complications. Further, surgical treatment is associated with high mortality rates, with the mortality rate being as high as 23%-67% in the cases showing early peritonitis after the operation[9]. Endoscopic drainage of the main pancreatic duct via the ampulla of Vater, which was first reported in 1991[10], has drawn considerable attention. Boerma et al. (2006) reported an excellent recovery rate (87%) after endoscopic treatment of 15 cases of pancreatic fistula[11]. In addition, other studies have reported recovery rates of about 58%-100% in the cases of pancreatic fistulas that do not

9

respond to conservative therapy and involve endoscopic treatment[12-20]. To date, only 1 death caused by acute pancreatitis has been reported. However, since this death may also have been caused by inadequate drainage, a direct relationship between the death and endoscopic treatment could not be confirmed[13]. Unlike LDLT, endoscopic treatment for pancreatic fistula allows greater accessibility to the ampulla of Vater. Further, endoscopic treatment is less invasive than surgical treatment; therefore, it can easily replace conservative therapy if sufficient drainage is achieved. Thus, patients who undergo endoscopic treatment for pancreatic fistula can be expected to make an early recovery. Irrespective of their merits and demerits, both ENPD and endoscopic pancreatic stenting (EPS) have been referred to in the reports. ENPD causes a sense of discomfort in the pharynx; however, this technique enables easy diagnosis of occlusion and dropout because it allows monitoring of the pancreatic fluid. In contrast, in EPS, the diagnosis of occlusion and dropout is difficult; however, this technique causes no sense of discomfort in the pharynx. We selected ENPD to enable safe monitoring of 2 channels of drainage: the endoscopic retrograde pancreatic drain as well as the interperitoneal drain. In case 1, the drain tube had to be replaced because of the fever caused by

10

occlusion; therefore, the choice of ENPD was considered to be reasonable. The patient in case 1 could have recovered earlier if the endoscopic treatment for pancreatic fistula had been initiated earlier. In each case, the patient recovered within approximately 40 days after ENPD. Further, the treatment had no influence on the patients' general status. Endoscopic treatment is considered to be safe for treating pancreatic fistulas that develop after LDLT. New endoscopic techniques, such as ultrasonography (US)-guided drainage, have also been used to treat refractory cases that do not respond to drainage via the ampulla of Vater; however, only few reports have described these techniques. These new techniques may also be less invasive than surgical treatment[21-22].

In conclusion, we described 2 cases of pancreatic fistula after LDLT that were not responsive to conservative therapy. In each case, the patient recovered within approximately 40 days after ENPD. Thus, endoscopic treatment for pancreatic fistula after LDLT should be adopted because of its high recovery rate and low invasiveness.

REFERENCES

11

1. Freise CE, Gillespie BW, Koffron AJ et al. Recipient morbidity after living and deceased donor liver transplantation: findings from the A2ALL Retrospective Cohort Study. Am J Transplant 2008; 8:2569-2579. PMID: 18976306

2. Ho MC, Wu YM, Hu RH et al. Surgical complications and outcome of living related liver transplantation. Transplant Proc 2004; 36:2249-2251. PMID: 15561208

3. Marsh JW, Gray E, Ness R et al. Complications of right lobe living donor liver transplantation. J Hepatol 51:715-724. 2009; PMID: 19576652

4. Emiroglu R, Sevmis S, Moray G et al. Living-donor liver transplantation: results of a single center. Transplant Proc 2007; 39:1149-1152. PMID: 17524917

5. Tanaka K, Miyashiro I, Yano M et al. Accumulation of excess visceral fat is a risk factor for pancreatic fistula formation after total gastrectomy. Ann Surg Oncol 2009; 16:1520-1525. PMID: 19267237

6. Parr ZE, Sutherland FR, Bathe OF et al. Pancreatic fistulae: are we making progress? J Hepatobiliary Pancreat Surg 2008; 15:563-569.

12

7. Pratt WB, Maithel SK, Vanounou T et al. Clinical and economic validation of the International Study Group of Pancreatic Fistula (ISGPF) classification scheme. Ann Surg 2007; 245:443-451. PMID: 18987924

8. Lipsett PA, Cameron JL. Internal pancreatic fistula. Am J Surg 1992; 163:216-220. PMID: 1739176

9. Tung BY, Kowdley KV, Kimmey MB. Pancreatic fistula without pancreatitis after endoscopic biliary stent placement for bile leak after orthotopic liver transplantation. Gastrointest Endosc 1999; 49:647-651. PMID: 10228269

10. Kozarek RA, Ball TJ, Patterson DJ et al. Endoscopic transpapillary therapy for disrupted pancreatic duct and peripancreatic fluid collections. Gastroenterology 1991; 100:1362-1370. PMID: 2013381

11. Boerma D, Rauws EA, van Gulik TM et al. Endoscopic stent placement for pancreaticocutaneous fistula after surgical drainage of the pancreas. Br J Surg 2000; 87:1506-1509.

12. Bracher GA, Manocha AP, DeBanto JR et al. Endoscopic pancreatic duct stenting to treat pancreatic ascites. Gastrointest Endosc 1999; 49:710-715. PMID: 11091237

13

13. Telford JJ, Farrell JJ, Saltzman JR et al. Pancreatic stent placement for duct disruption. Gastrointest Endosc 2002; 56:18-24. PMID: 12085030

14. Varadarajulu S, Noone TC, Tutuian R et al. Predictors of outcome in pancreatic duct disruption managed by endoscopic transpapillary stent placement. Gastrointest Endosc 2005; 61:568-575. PMID: 15812410

15. Saeed ZA, Ramirez FC, Hepps KS. Endoscopic stent placement for internal and external pancreatic fistulas. Gastroenterology 1993; 105:1213-1217. PMID: 8405869

16. Kozarek RA, Ball TJ, Patterson DJ et al. Transpapillary stenting for pancreaticocutaneous fistulas. J Gastrointest Surg 1997; 1:357-361. PMID: 9834370

17. Costamagna G, Mutignani M, Ingrosso M et al. Endoscopic treatment of postsurgical external pancreatic fistulas. Endoscopy 2001; 33:317-322. PMID: 11315892

18. Fischer A, Benz S, Baier P et al. Endoscopic management of pancreatic fistulas secondary to intraabdominal operation. Surg Endosc 2004; 18:706-708. PMID: 15026901

19. Le Moine O, Matos C, Closset J et al. Endoscopic management of

14

pancreatic fistula after pancreatic and other abdominal surgery. Best Pract

Res Clin Gastroenterol 2004; 18:957-975. PMID: 15494289

20. Goasguen N, Bourrier A, Ponsot P et al. Endoscopic management of

pancreatic fistula after distal pancreatectomy and enucleation. Am J Surg

2009; 197:715-720. PMID: 18789426

21. Romano A, Spaggiari M, Masetti M et al. A new endoscopic treatment for

pancreatic fistula after distal pancreatectomy: case report and review of

the literature. Gastrointest Endosc 2008; 68:798-801. PMID: 18514650

22. Arvanitakis M, Delhaye M, Bali MA et al. Endoscopic treatment of

external pancreatic fistulas: when draining the main pancreatic duct is not

enough. Am J Gastroenterol 2007; 102:516-524. PMID: 17335445

Figure Legend

Fig 1: Computed tomography (CT) performed on postoperative day (POD) 13

showing fluid collection (circle) at the lower edge of the pancreas. The

hematoma was considered to be ruptured.

Fig 2: Pancreatographic examination of the drain at the tail of the pancreas (Drain) in case 1 reveals the disrupted (arrows) main pancreatic duct (MPD) with flow of contrast into the duodenum.

Fig 3: A radiograph showing postprocedure endoscopic naso-pancreatic drainage (ENPD) in case 1. Excellent drainage of the pancreatic duct is noted.

Fig 4: Upper chart shows the body temperature (BT) and serum C-reactive protein (CRP) level. Lower one shows daily output of the endoscopic naso-pancreatic drainage (ENPD) tube and the drain at the tail of the pancreas (Tail) in case 1. The patient had an episode of fever caused by the occlusion of the ENPD tube. After the tube was replaced, the pancreatic fistula healed completely.

Fig 5: Contrast examination from the drain at the tail of the pancreas (Drain) in case 1 on postoperative day (POD) 363 reveals the closure of fistula.

16

Fig 6: Computed tomography (CT) performed 2 days after removal of the drain in case 1 showing no fluid collection around the pancreas.

Fig 7: A radiograph showing postprocedure endoscopic naso-pancreatic drainage (ENPD) in case 2. The image shows drains placed at both the right and left edges of the pancreas (Right and Left) and ENPD.

Fig 8: Upper chart shows the body temperature (BT) and serum C-reactive protein (CRP) level. Lower one shows daily output of endoscopic naso-pancreatic drainage (ENPD) tube and the drains at both the edges of the pancreas (Right and Left) in case 2. The pancreatic fistula healed completely on post-ENPD day 38.

Fig 9: Computed tomography (CT) performed 14 days after removal of the drains in case 2 showing no fluid collection around the pancreas.

17

Three-Dimensional Computed Tomography Scan Analysis of Hepatic Vasculatures in the Donor Liver for Living Donor Liver Transplantation

Koichiro Uchida,[1] **Masahiko Taniguchi,**[2] **Tsuyoshi Shimamura,**[3] **Tomomi Suzuki,**[2] **Kenichiro Yamashita,**[4] **Minoru Ota,**[3] **Toshiya Kamiyama,**[1] **Michiaki Matsushita,**[1] **Hiroyuki Furukawa,**[2] **and Satoru Todo**[1]

[1]*Department of General Surgery,* [2]*Department of Organ Transplantation and Regeneration,* [3]*Division of Organ Transplantation, and* [4]*Department of Molecular Surgery, Graduate School of Medicine, Hokkaido University, Sapporo, Japan*

Because hepatic vasculatures exhibit variations, a preoperative evaluation of the vascular anatomy and an estimation of the volume of the liver graft are essential for successful adult living donor liver transplantation. Using 3-dimensional (3D) computed tomography (CT), we analyzed the volumetric and anatomical relationship of the hepatic vasculatures of liver grafts. The livers of 223 potential donors were analyzed by 3D CT. Volumetric analysis was performed for each hepatic vein and its tributaries. The anatomy of the portal vein and hepatic artery was assessed along with the biliary system via intraoperative cholangiography in 110 recipients. On the basis of the anatomical presentation of the inferior right hepatic vein (IRHV), the hepatic veins were classified as follows: in type I, the IRHV was absent; in type II, the IRHV was smaller than the right hepatic vein (RHV); and in type III, the IRHV was greater than or equal to the RHV in size. The drainage volume of the middle hepatic vein (MHV) and especially its tributaries in the right lobe increased with the size of the IRHV ($P < 0.001$). In type III hepatic veins with a large IRHV (17% of the donors), the MHV tributaries had the largest drainage volume in the right lobe (41.2% $\pm$ 11.8%). Furthermore, type III hepatic veins typically exhibited biliary variations in 75% of the donors. No correlation was observed between variations in the hepatic artery and portal vein. In conclusion, a right lobe graft with a large IRHV is accompanied by a large drainage volume via the MHV and by bile duct variations in 17% of livers. Therefore, anatomical and volumetric analysis is important for preoperative evaluations. *Liver Transpl 16:1062-1068, 2010.* © 2010 AASLD.

Living donor liver transplantation (LDLT) is an alternative to deceased donor liver transplantation for the treatment of patients with end-stage liver disease.[1] However, its application in adult recipients has been complicated by the problems associated with anatomical variations of hepatic vasculatures[2] and insufficient graft volume caused by venous outflow disturbances.[3] This is particularly true when the right lobe is selected for transplantation.

To successfully perform right lobe LDLT, the drainage volume of the middle hepatic vein (MHV) tributaries and the inferior right hepatic vein (IRHV) must be

considered.[4] Liver atrophy or almost no liver regeneration has been noted in congested areas.[5] Moreover, large congested areas can also result in infarction and lead to septic shock.[3] Complications caused by potentially congested areas can be prevented if an adequate volume is provided by venous reconstruction, even in a right lobe graft with large congested areas.[6] Meanwhile, extended right lobe grafts[7] or right lobe grafts in which venous collaterals are preserved without reconstruction of MHV branches[8] have been used to manage graft congestion and to provide consistent graft function. However, an extended right lobe graft leads to congestion of the left medial segment in the donor. Because of these findings, a right lobe graft with reconstruction of the MHV is still more common.

In the right lobe graft, multiple branches of the portal vein (8.1%-13.6%),[9-11] hepatic artery (18%),[12] and bile duct (41.5%-67.5%)[13] can be exposed on the cut surface. In such cases, multiple or complex reconstructions are necessary, and this may increase the attendant risk of anastomotic complications.[12,14-16]

In recent years, 3-dimensional (3D) computed tomography (CT) scans have assisted doctors in preoperative surgical planning. We have reported the contribution of 3D CT scans in evaluating the morphology of hepatic vessels and tumors and in estimating the volume that is drained from the liver by the hepatic vein.[17-19] Previously, the morphology of each hepatic vein pattern has been classified individually as surgical anatomy for hepatectomy. Reichert et al.[20] classified the left hepatic vein (LHV), Marcos et al.[4] and Neumann et al.[21] classified the MHV, and Radtke et al.[22] classified the IRHV. Nakamura and Tsuzuki[23] reported the size of the right hepatic vein (RHV) in a case study. The RHV is generally the largest draining vein in both the right lobe and the whole liver, although the size of the RHV varies according to the size of the IRHV.[23] The morphology or interrelationship of each hepatic vein (especially the RHV and MHV) is important for successful reconstruction in LDLT.

In the current study, we attempted to analyze the volumetric and anatomical interrelationships of the hepatic veins (RHV, MHV, and IRHV) and other hepatic vasculatures (portal vein, hepatic artery, and bile duct) in LDLT. Our results indicate that a knowledge of these associations contributes to preoperative evaluations of the donor liver in LDLT.

PATIENTS AND METHODS

Patients

From August 2002 to May 2009, 223 potential liver donors were analyzed with 3D analysis software at Hokkaido University Hospital (Sapporo, Japan). One hundred thirteen were donor candidates, and 110 subsequently underwent surgery as donors for graft harvesting. None of the analyzed potential donors had a fatty liver on the CT scan.

CT Scan and Volume Rendering

All CT scans were obtained with a 64-slice multidetector CT scanner (Aquilion, Toshiba, Tokyo, Japan). All data were transferred to a 3D workstation (Virtual Place Lexus, Medical Imaging Laboratory, AZE, Tokyo, Japan). A virtual dissection line was drawn between the left and right lobes of the liver (determined from the Rex-Cantlie line) just above the main trunk of the MHV. Both the volume of the whole liver and the right and left lobes and the drainage volume of each hepatic vein and its branches were calculated.

Hepatic Venous Anatomy and Terminology

The names of the hepatic veins and their branches were described according to Couinaud's segmentation[24]; for example, the vein draining segment 5 (the inferior part of the anterior sector) was named V5. Each branch described by 3D CT was more than 2 mm in diameter. The MHV was divided into MHV tributaries in the right lobe and in the left lobe. Assuming right lobe graft LDLT, we analyzed the morphology, drainage volume, and relationships of the hepatic veins (eg, RHV, MHV, and IRHV).

Portal Vein (Fig. 1)

The branching patterns of the portal vein were classified with 3D CT images as follows: normal branching of the portal vein (PV1), trifurcation of the portal vein (PV2), and independent branching of the posterior branch from the main portal trunk (PV3).

Hepatic Artery (Fig. 1)

The branching patterns of the hepatic artery were detected by 3D CT scanning and were denoted as follows: normal branching of the hepatic artery (A1), replacement of the left hepatic artery from the left gastric artery (A2), and replacement of the right hepatic artery from the superior mesenteric artery (A3). The branching patterns of the middle hepatic artery (MHA) were also classified: the MHA arising from the left hepatic artery (A4) and the MHA arising from the right hepatic artery (A5).

Biliary Duct (Fig. 1)

The evaluation of the biliary duct was performed in only 110 donors who underwent intraoperative cholangiography. Branching of the biliary tree was classified as follows: normal branching of the biliary duct (B1), trifurcation of the biliary duct (B2), posterior branching from the left hepatic duct (B3), independent posterior branching from the common hepatic duct (B4), and a segment 3 or 4 bile duct from the right hepatic duct (B5).

Definitions of the Normal and Variation Groups

Considering right and left lobe graft liver transplantation, we classified each branching pattern of the
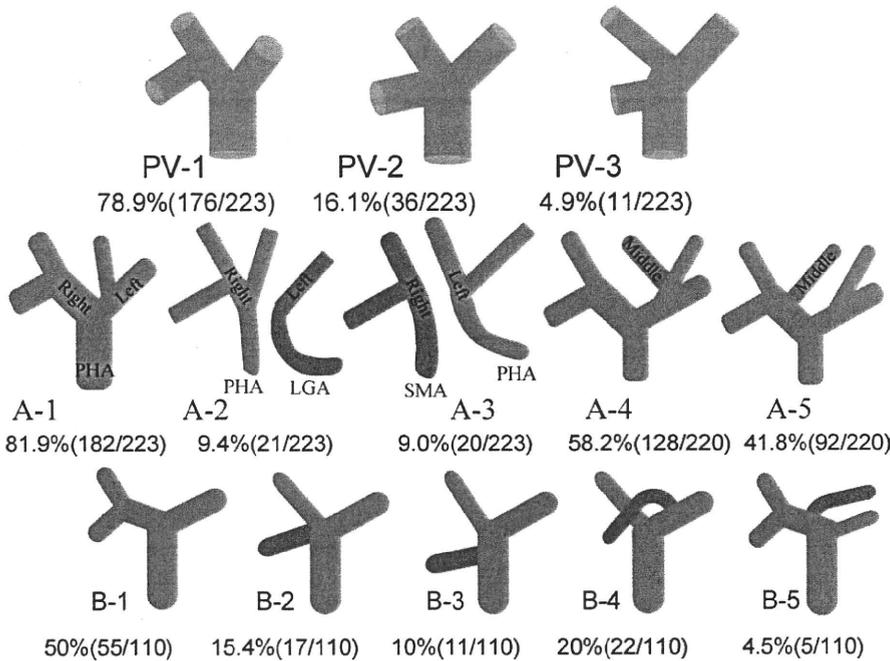
Figure 1. Branching patterns of the portal vein (PV1-PV3), hepatic artery (A1-A5), and biliary duct (B1-B5).

hepatic vasculatures (portal vein and biliary duct) into group A (1 anastomosis in reconstruction) or group B (possible multiple or technically complicated anastomoses). The branching patterns of the hepatic artery were classified into group A (A1 and A3) or group B (A2) for right lobe grafts and into group A (A4) or group B (A5) for left lobe grafts according to the possibility of multiple anastomoses.

Statistical Analysis

Statistical analysis was performed with SPSS version 12.0 (SPSS, Inc., Chicago, IL). Volumetric results are expressed as arithmetic means and standard errors of the mean. Statistical comparisons of the hepatic vasculatures were performed with logistic regression analysis. Volumetric results were compared by 1-way analysis of variance (Tukey). All differences were considered to be statistically significant at $P < 0.05$.

RESULTS

Types of Hepatic Veins

Using 3D CT images, we calculated the drainage volume of each hepatic vein, and we found that the hepatic veins could be classified into 3 types on the basis of the size of the IRHV (Fig. 2). The size of the IRHV was determined by comparison with the RHV. In cases of multiple IRHVs, we evaluated their collective size by determining the drainage volume and comparing it with the RHV volume. The diameter of the IRHV detected by the CT scan in our analysis was >2 mm. The average total volume of the IRHV was 169.5 mL, and the volume ranged from 14 to 556 mL; the average number of IRHVs was 1.24, and the number ranged from 1 to 3.
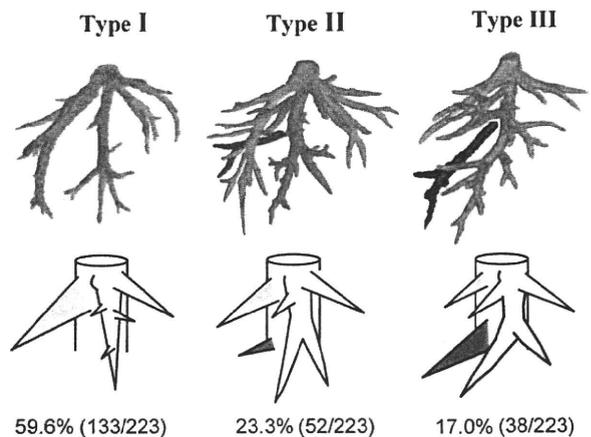
Figure 2. Classification of the hepatic vein by the RHV and IRHV sizes: in type I, the IRHV was absent; in type II, the IRHV was smaller than the RHV; and in type III, the IRHV was greater than or equal to the RHV in size.

Patients with type I hepatic veins had no IRHV, those with type II hepatic veins had a small IRHV (IRHV < RHV), and those with type III hepatic veins had a large IRHV (IRHV ≥ RHV).

Donors and Candidates (Table 1)

In the current study, we investigated the anatomy of the hepatic vasculatures of 113 donor candidates and 110 donors. Comparing the donors and candidates, we found no differences in the type of hepatic vein, the development of each hepatic vein, or the distribution of the branching patterns of the hepatic artery

TABLE 1. Comparison of Donors and Donor Candidates with Respect to the Percentage of Drainage in Whole Liver Grafts by Each Hepatic Vein and with Respect to the Distribution of Branching Patterns of the Hepatic Artery and Portal Vein

Drainage Percentage: Donor Whole Liver				
Vein	Total (n = 110)	Type I (n = 65)	Type II (n = 25)	Type III (n = 20)
RHV	35.6% ± 12.0%	42.4% ± 7.0%	32.2% ± 7.5%	16.3% ± 5.8%[*,†,‡]
IRHV	5.9% ± 7.2%	0.9% ± 1.5%	8.9% ± 2.9%	18.7% ± 4.1%[*,†,‡]
MHV	33.7% ± 8.0%	32.1% ± 6.7%	33.2% ± 8.2%	39.6% ± 9.2%[†]
LHV	20.1% ± 6.4%	20.9% ± 5.2%	19.1% ± 7.9%	18.9% ± 7.7%

Drainage Percentage: Candidate Whole Liver				
Vein	Total (n = 113)	Type I (n = 68)	Type II (n = 27)	Type III (n = 18)
RHV	35.6% ± 12.3%	42.5% ± 7.9%	31.3% ± 8.0%	16.0% ± 5.5%[*,†,‡]
IRHV	5.85% ± 8.19%	0.7% ± 1.6%	8.6% ± 3.1%	20.8% ± 7.9%[*,†,‡]
MHV	33.8% ± 8.1%	32.1% ± 6.8%	34.6% ± 8.5%	39.0% ± 8.1%[†]
LHV	19.6% ± 4.9%	19.9% ± 4.4%	19.8% ± 5.5%	18.4% ± 5.9%

Distribution of Branching Patterns				
		Candidate	Donor	P Value
Hepatic artery	A1	80.5% (91/113)	82.7% (91/110)	NS
	A2	9.7% (11/113)	7.3% (8/110)	
	A3	9.7% (11/113)	9.0% (10/110)	
Portal vein	PV1	73.4% (83/113)	84.5% (93/110)	NS
	PV2	20.4% (23/113)	11.8% (13/110)	
	PV3	6.2% (7/113)	3.6% (4/110)	

[*]$P < 0.05$ for I versus II.
[†]$P < 0.05$ for I versus III.
[‡]$P < 0.05$ for II versus III.

and portal vein. Because there were no differences in the distribution of the types of hepatic veins or in the development of hepatic veins, the following analyses were performed for all 223 potential donors.

Hepatic Vein

Of the potential donors who were studied, 133 had type I veins (59.6%), 52 had type II veins (23.3%), and 38 had type III veins (17.0%; Fig. 2).

The drainage volume percentages in the whole liver according to the different types of hepatic veins are shown in Table 2. On the basis of the definitions of the hepatic vein types, the percentage of the volume drained by the IRHV increased from type I to type III, whereas the drainage volume of the RHV decreased in that order. In addition, the percentage of the volume that was drained by the LHV did not differ between the different types of veins; however, the volume of the MHV drainage increased similarly to that of the IRHV from type I to type III.

The drainage volume of each hepatic vein in right lobe grafts was compared. When the drainage volume of the IRHV increased in the right lobe, the percentage of the volume that drained from the right lobe by the MHV increased from type I to type III (Table 2). However, in the left lobe, the percentage of the volume drained by the LHV and MHV tributaries did not vary between the 3

types (Table 2). Moreover, with respect to the volume drained from the right lobe by the branches of the MHV [V5 and vein draining segment 8 (V8)], the percentages of both V5 and V8 varied significantly for all 3 types ($P < 0.05$ for V8 and $P < 0.05$ for V5) and increased significantly from type I to type III [for V5, 17.3% ± 8.5% on average, 15.6% ± 7.9% for type I, 18.4% ± 8.4% for type II, and 21.6% ± 9.1% for type III ($P = 0.259$ for I versus II, $P < 0.01$ for I versus III, and $P = 0.050$ for II versus III); for V8, 12.6% ± 5.9% on average, 12.0% ± 5.6% for type I, 12.6% ± 5.0% for type II, and 14.9% ± 7.2% for type III ($P = 0.958$ for I versus II, $P = 0.027$ for I versus III, and $P = 0.108$ for II versus III)].

With type III hepatic veins, the RHV drained 25.1% ± 8.8% of the right lobe, the IRHV drained 30.5% ± 9.3%, and the MHV drained 41.2% ± 11.8%. Each of these 3 veins drained almost one-third of the right lobe, and the MHV provided the greatest drainage.

Portal Vein

The proximal branching patterns of the portal vein were examined with 3D CT (Fig. 1). PV1 was the most frequent pattern (78.9%), and it was followed by PV2 (16.1%) and PV3 (4.9%). The type of hepatic vein was not related to the pattern of proximal portal vein branching. Variations in portal vein branching (group B) were detected in 21.0% of all cases. On the basis of

TABLE 2. Percentage of Drainage in Whole Liver Grafts and in Right and Left Lobe Grafts by Each Hepatic Vein and MHV Tributaries

Graft	Vein	Total (n = 223)	Type I (n = 133)	Type II (n = 52)	Type III (n = 38)
Whole liver (100%)	RHV	35.5% ± 12.2%	42.5% ± 7.5%	31.8% ± 7.8%*	16.2% ± 5.6%[†,‡]
	IRHV	5.9% ± 7.7%	0.8% ± 1.5%	8.7% ± 3.0%*	19.7% ± 6.2%[†,‡]
	MHV	33.7% ± 8.1%	32.1% ± 6.7%	33.9% ± 8.3%	39.3% ± 9.5%[†,‡]
Right lobe (100%)	RHV	55.0% ± 18.1%	65.4% ± 10.3%	50.1% ± 11.0%*	25.1% ± 8.8%[†,‡]
	IRHV	9.2% ± 11.9%	1.3% ± 2.4%	13.8% ± 4.5%*	30.5% ± 9.3%[†,‡]
	MHV tributaries	33.5% ± 10.7%	30.9% ± 9.28%	34.5% ± 10.5%*	41.2% ± 11.8%[†,‡]
Left lobe (100%)	LHV	56.5% ± 13.5%	58.3% ± 11.3%	53.9% ± 14.7%	53.4% ± 17.7%
	MHV tributaries	34.6% ± 10.6%	34.1% ± 9.0%	34.7% ± 10.7%	36.1% ± 14.9%

*$P < 0.05$ for I versus II.
[†]$P < 0.05$ for I versus III.
[‡]$P < 0.05$ for II versus III.

TABLE 3. Distribution of the Branching Patterns of the Portal Vein, Hepatic Artery, and Biliary Duct by the Type of Hepatic Vein in Right and Left Lobe Grafts

			Right Lobe		
			Type I	Type II	Type III
Portal vein	Group A	PV1 (n = 176)	79.6% (106/133)	75.0% (39/52)	81.5% (31/38)
	Group B	PV2/PV3 (n = 47)	20.3% (27/133)	25.0% (13/52)	18.4% (7/38)
Hepatic artery	Group A	A1/A3 (n = 202)	93.2% (124/133)	86.5% (45/52)	86.9% (33/38)
	Group B	A2 (n = 21)	6.0% (8/133)	13.5% (7/52)	13.1% (5/38)
Biliary duct	Group A	B1 (n = 55)	55.3% (36/65)	56.0% (14/25)	25.0% (5/20)
	Group B	B2-B5 (n = 55)	44.6% (29/65)	44.0% (11/25)	75.0% (15/20)*[,†]
			Left Lobe		
			Type I	Type II	Type III
Portal vein	Group A	PV1/PV2 (n = 212)	96.2% (128/133)	94.2% (49/52)	92.2% (35/38)
	Group B	PV3 (n = 11)	3.8% (5/133)	5.8% (3/52)	7.8% (3/38)
Hepatic artery	Group A	A4	56.9% (74/130)	51.9% (27/52)	71.0% (27/38)
	Group B	A5	43.1% (56/130)	48.1% (25/52)	29.0% (11/38)
Biliary duct	Group A	B1-B3 (n = 83)	78.50 (51/65)	80.0% (20/25)	60.0% (12/20)
	Group B	B4/B5 (n = 27)	21.5% (14/65)	20.0% (5/25)	40.0% (8/20)

*$P < 0.05$ for I versus II.
[†]$P < 0.05$ for I versus III.

the type of hepatic vein, the percentage of portal vein variations was compared. The portal vein branching patterns in both right and left lobes showed no correlation with the patterns of hepatic veins (Table 3).

Hepatic Arteries

A1 was most frequent (81.9%), and it was followed by A2 (9.4%) and A3 (9.0%). The percentages of A4 and A5 were 58.2% and 41.8%, respectively (Fig. 1). The percentage of arterial variations was compared with respect to the type of hepatic vein. The arterial branching patterns in both the right and left lobes showed no significant correlation with the type of hepatic veins; however, in the left lobe graft, A4 tended

to be more frequent in the type III hepatic vein ($P = 0.06$).

Biliary Duct

B1 was most frequent (50%), and it was followed by B4 (20%), B2 (15.4%), B3 (10%), and B5 (4.5%; Fig. 1). There was no statistical relationship between the type of hepatic vein and each biliary variation, but B3 was more prevalent with type III veins than type I and type II veins (7.6% for type I, 8% for type II, and 20% for type III; intergroup $P = 0.315$). In right lobe grafts, group B biliary ducts (B2-B5) were detected in 50% of donors. A classification based on the type of hepatic vein indicated that B1 was found in 55.3% (36/65) of patients with type I veins, in 56.0% (14/25) with type

II veins, and in 25.0% (5/20) with type III veins (Table 3). In comparison with patients with type I and II hepatic veins, patients with type III veins had significantly fewer instances of normal branching in the right lobe graft ($P = 0.022$ for type II and $P = 0.041$ for type III). On the other hand, in left lobe grafts, there was no correlation between the type of hepatic vein and the branching patterns of the bile duct.

DISCUSSION

In the current study, we found that the size of the IRHV, which was compared with the RHV, was related to the development of the MHV tributaries in the right lobe. When the IRHV increased, the volume drained by MHV tributaries also increased. In addition, type III hepatic veins, characterized by a large IRHV, were shown to have some characteristics and were found in 17% of potential healthy donors. Type III MHV tributaries provided the largest drainage from the right lobe. Moreover, this type exhibited significantly more biliary duct variation, primarily in the form of an independent posterior branch that originated from the common hepatic duct (B3). On the other hand, we did not find other relationships between the type of hepatic vein and the branching pattern of the portal vein or hepatic artery.

On the basis of autopsies, Nakamura and Tsuzuki[23] revealed relationships between the RHV and the IRHV. They reported that 38.6% of the specimens were hepatic vein type I, 37.3% were type II, and 24.1% were type III; types II and III were more prevalent in their study versus ours. This may have occurred because 16 ± 9 IRHVs on average were counted in their study (even those with the diameter of a pin were included), and only veins with a diameter of 2 mm or larger can be depicted on CT scans.[25] In the current study, the type of hepatic vein was defined by a comparison of the collective volume of the IRHVs with that of the RHV when the CT scan showed multiple IRHVs. However, there were no cases in which the collective volume of multiple small IRHVs accounted for a significant volume in the liver graft. Meanwhile, this 3D CT classification of hepatic veins cannot be used as an indication for IRHV reconstruction. For technical reasons, the diameter of the hepatic vein needs to be sufficiently large for reconstruction (ie, >5 mm).[26]

In comparison with the previous MHV classifications by Marcos et al.[4] and Neumann et al.,[21] the results of the present study permitted an interpretation of the relationship between the IRHV and MHV (Fig. 2). Type I in this study corresponds to an MHV with numerous small tributaries that flow into the main trunk (13%-20%); type II corresponds to an MHV type with symmetrical branching of the veins that drain the superior and inferior parts of segment 4, V5, and V8 (59%-70%); and type III corresponds to a MHV type with large V8 and V5 drainage to segment 6 (10%-18%). As indicated earlier, an increase in the area drained by the MHV from type I to type III was attributable to the fact that the areas draining seg-

ments 5 and 8 became larger than those draining segment 4. With type III veins, there was almost no venous flow from the dorsal portion of segments 5 and 8 into the small RHV and IRHV; the drainage of the area occurred via large branches of V8 and V5 flowing into the MHV. With type III veins, V5 accounted for approximately 21% of the drainage of the right lobe. Neumann et al.[21] reported that the volume of drainage differed according to the type of MHV branching and that drainage by V5 in particular differed; this is in agreement with the results of the current study. Therefore, the pattern of MHV branching and the percentage of drainage by the branches can be predicted by the size of the IRHV.

The current study revealed a significantly high prevalence of biliary variations with type III hepatic veins, but the type of hepatic vein did not correlate with the ramification of the portal vein and hepatic artery. Thus, if type III veins are used in a right lobe graft, 75% of patients might require multiple biliary reconstructions. On the other hand, portal vein variations may be related to biliary tree variations. Embryologically, intrahepatic ducts develop from ductal plates after development of the portal duct,[27] and a correlation with portal vein and biliary variations has been reported.[28] Radtke et al.[22] concluded that there is a correlation between a large IRHV and portal vein variations, including proximal and distal variations (we studied only proximal portal vein ramification in this study). Taking these facts into consideration, we should probably keep in mind the extent of the variations of the portal vein and the bile duct when preoperative image evaluations are performed for type III grafts.

In the context of LDLT, for the evaluation of the anatomy of the liver in a patient with a large IRHV before surgery, it is important to (1) confirm the characteristics of MHV branching and the sizes of the branches and (2) detect any possible abnormalities in biliary ramification. Indeed, 20 donors (17%) in our series showed type III hepatic veins. Seven of the 110 recipients (6%) underwent left lobe grafting, whereas 6 patients (5%) received a right lobe graft with a type III hepatic vein because of inadequate left lobe volume for grafting. We performed intraoperative cholangiography for the assessment of biliary anatomy; however, a recent report has suggested that preoperative CT cholangiography is useful for biliary assessment.[29] This study suggests that a preoperative evaluation of the biliary anatomy by CT cholangiography is important when a right lobe graft is being used, especially in the case of a liver with a type III hepatic vein. Meanwhile, the findings of our study suggest that multiple branches of the hepatic artery and bile duct are rarely exposed on the cut surface of the left lobe graft in the case of a type III hepatic vein. Hence, for simple reconstructions, a left lobe graft may be preferable for a liver with a type III hepatic vein if the graft volume is acceptable.

The use of 3D CT for the preoperative evaluation of hepatic vasculatures has become common in LDLT.[30] However, there have been very few reports on the

volumetric and anatomical relationship of hepatic vasculatures of liver grafts. This classification will help us to preoperatively determine the appropriate technique or form of reconstruction.

In conclusion, this study demonstrates the volumetric and anatomical interrelationship of hepatic veins and other hepatic vasculatures for LDLT with 3D CT scanning. According to our findings, a right lobe graft with a large IRHV is accompanied by a large drainage volume via the MHV and by bile duct variations in 17% of livers. Detailed preoperative evaluations are essential for the success of adult LDLT.

REFERENCES

1. Hashikura Y, Makuuchi M, Kawasaki S, Matsunami H, Ikegami T, Nakazawa Y, et al. Successful living-related partial liver transplantation to an adult patient. Lancet 1994;343:1233-1234.
2. Nakamura T, Tanaka K, Kiuchi T, Kasahara M, Oike F, Ueda M, et al. Anatomical variations and surgical strategies in right lobe living donor liver transplantation: lessons from 120 cases. Transplantation 2002;73:1896-1903.
3. Lee S, Park K, Hwang S, Kim K, Ahn C, Moon D, et al. Anterior segment congestion of a right liver lobe graft in living-donor liver transplantation and strategy to prevent congestion. J Hepatobiliary Pancreat Surg 2003;10:16-25.
4. Marcos A, Orloff M, Mieles L, Olzinski AT, Renz JF, Sitzmann JV. Functional venous anatomy for right-lobe grafting and techniques to optimize outflow. Liver Transpl 2001;7:845-852.
5. Hata S, Sugawara Y, Kishi Y, Niiya T, Kaneko J, Sano K, et al. Volume regeneration after right liver donation. Liver Transpl 2004;10:65-70.
6. Yonemura Y, Taketomi A, Soejima Y, Yoshizumi T, Uchiyama H, Gion T, et al. Validity of preoperative volumetric analysis of congestion volume in living donor liver transplantation using three-dimensional computed tomography. Liver Transpl 2005;11:1556-1562.
7. Kasahara M, Takada Y, Fujimoto Y, Ogura Y, Ogawa K, Uryuhara K, et al. Impact of right lobe with middle hepatic vein graft in living-donor liver transplantation. Am J Transplant 2005;5:1339-1346.
8. Campsen J, Hendrickson RJ, Zimmerman MA, Wachs M, Bak T, Russ P, et al. Adult right lobe live donor liver transplantation without reconstruction of the middle hepatic vein: a single-center study of 109 cases. Transplantation 2008;85:775-777.
9. Hwang S, Lee SG, Ahn CS, Kim KH, Moon DB, Ha TY, et al. Technique and outcome of autologous portal Y-graft interposition for anomalous right portal veins in living donor liver transplantation. Liver Transpl 2009;15:427-434.
10. Kasahara M, Egawa H, Tanaka K, Ogawa K, Uryuhara K, Fujimoto Y, et al. Variations in biliary anatomy associated with trifurcated portal vein in right-lobe living-donor liver transplantation. Transplantation 2005;79:626-627.
11. Varotti G, Gondolesi GE, Goldman J, Wayne M, Florman SS, Schwartz ME, et al. Anatomic variations in right liver living donors. J Am Coll Surg 2004;198:577-582.
12. Marcos A, Orloff M, Mieles L, Olzinski A, Sitzmann J. Reconstruction of double hepatic arterial and portal venous branches for right-lobe living donor liver transplantation. Liver Transpl 2001;7:673-679.
13. Kasahara M, Egawa H, Takada Y, Oike F, Sakamoto S, Kiuchi T, et al. Biliary reconstruction in right lobe living-donor liver transplantation: comparison of different techniques in 321 recipients. Ann Surg 2006;243:559-566.
14. Kyoden Y, Tamura S, Sugawara Y, Matsui Y, Togashi J, Kaneko J, et al. Portal vein complications after adult-to-adult living donor liver transplantation. Transpl Int 2008;21:1136-1144.
15. Lerut J, Tzakis AG, Bron K, Gordon RD, Iwatsuki S, Esquivel CO, et al. Complications of venous reconstruction in human orthotopic liver transplantation. Ann Surg 1987;205:404-414.
16. Gondolesi GE, Varotti G, Florman SS, Munoz L, Fishbein TM, Emre SH, et al. Biliary complications in 96 consecutive right lobe living donor transplant recipients. Transplantation 2004;77:1842-1848.
17. Jin MB, Onodera Y, Furukawa H, Todo S. Three-dimensional volumetric analysis for reconstruction of middle hepatic vein tributaries in living donor liver transplantation. J Am Coll Surg 2005;200:468-469.
18. Taniguchi M, Furukawa H, Shimamura T, Suzuki T, Oota M, Onodera Y, et al. Hepatic venous reconstruction of anterior sector using three-dimensional helical computed tomography in living donor liver transplantation. Transplantation 2006;81:797-799.
19. Kamiyama T, Nakagawa T, Nakanishi K, Kamachi H, Onodera Y, Matsushita M, et al. Preoperative evaluation of hepatic vasculature by three-dimensional computed tomography in patients undergoing hepatectomy. World J Surg 2006;30:400-409.
20. Reichert PR, Renz JF, D'Albuquerque LA, Rosenthal P, Lim RC, Roberts JP, et al. Surgical anatomy of the left lateral segment as applied to living-donor and split-liver transplantation: a clinicopathologic study. Ann Surg 2000;232:658-664.
21. Neumann JO, Thorn M, Fischer L, Schobinger M, Heimann T, Radeleff B, et al. Branching patterns and drainage territories of the middle hepatic vein in computer-simulated right living-donor hepatectomies. Am J Transplant 2006;6:1407-1415.
22. Radtke A, Sotiropoulos GC, Molmenti EP, Nadalinl S, Schroeder T, Schenk A, et al. The influence of accessory right inferior hepatic veins on the venous drainage in right graft living donor liver transplantation. Hepatogastroenterology 2006;53:479-483.
23. Nakamura S, Tsuzuki T. Surgical anatomy of the hepatic veins and the inferior vena cava. Surg Gynecol Obstet 1981;152:43-50.
24. Couinaud C. Surgical Anatomy of the Liver Revisited. 2nd ed. Paris, France: Couinaud; 1989.
25. Radtke A, Sgourakis G, Sotiropoulos GC, Molmenti EP, Saner FH, Timm S, et al. Territorial belonging of the middle hepatic vein in living liver donor candidates evaluated by three-dimensional computed tomographic reconstruction and virtual liver resection. Br J Surg 2009;96:206-213.
26. Sugawara Y, Makuuchi M, Sano K, Imamura H, Kaneko J, Ohkubo T, et al. Vein reconstruction in modified right liver graft for living donor liver transplantation. Ann Surg 2003;237:180-185.
27. Desmet VJ. Ludwig symposium on biliary disorders—part I. Pathogenesis of ductal plate abnormalities. Mayo Clin Proc 1998;73:80-89.
28. Lee VS, Morgan GR, Lin JC, Nazzaro CA, Chang JS, Teperman LW, et al. Liver transplant donor candidates: associations between vascular and biliary anatomic variants. Liver Transpl 2004;10:1049-1054.
29. Schroeder T, Malago M, Debatin JF, Goyen M, Nadalin S, Ruehm SG. "All-in-one" imaging protocols for the evaluation of potential living liver donors: comparison of magnetic resonance imaging and multidetector computed tomography. Liver Transpl 2005;11:776-787.
30. Cheng YF, Lee TY, Chen CL, Huang TL, Chen YS, Lui CC. Three-dimensional helical computed tomographic cholangiography: application to living related hepatic transplantation. Clin Transplant 1997;11:209-213.