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ORIGINAL ARTICLE

Surgical Complications Following Liver Transplantation in Patients with Portal Vein Thrombosis—A Single-Center Perspective

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Abstract
Introduction Portal vein thrombosis (PVT) was once considered a contraindication for liver transplantation (LTx) because of technical difficulties. Though no longer a contraindication, it remains a risk factor.
Aim A study of surgical complications following LTx in patients with and without PVT.
Patients and methods A retrospective review of 1,171 consecutive patients who underwent LTx between June 1995 and June 2007 was performed, and 78 recipients with PVT (study group) were compared with a stratified random sample of 78 contemporous recipients without PVT (control group) for postoperative complications. Both groups were comparable with respect to age, sex, race, and other confounding variables.
Results The rate of primary nonfunction (PNF) in the study and control groups was 9.0% and 1.3%, (p=0.063), while that of retransplantation was 17.9% and 7.7% (p=0.055), respectively. The mean donor risk index (DRI) among the patients with and without PNF in the study group was 2.58±0.44 and 2.08±0.42, respectively (p=0.014). A significantly higher number of packed red blood cells and fresh frozen plasma transfusions were observed in study group compared to controls (p=0.012, 0.007, respectively).
Conclusion A higher rate of PNF was related to the complexity of the surgical procedure and the use of donor livers with a high DRI. Higher rates of PNF eventually led to a higher rate of retransplant. A strategy of offering donor livers with a low DRI might be helpful in decreasing the rate of PNF. Further, a PV interposition graft in difficult cases instead of thrombectomy could lead to a lower rethrombosis rate.

Keywords Liver transplant · PVT · Infection · Biliary complications · Thrombotic complications

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Introduction

The incidence of portal vein thrombosis (PVT) in patients with end-stage liver disease varies from 5% to 15%.[1,2] When recognized in advance, PVT was once considered an absolute contraindication to liver transplantation,[2–4] until good results were obtained with thrombectomy or a venous interposition graft between the donor portal vein and splenomesenteric confluence.[5] With the evolution of new surgical techniques,[6] many of the technical difficulties were overcome, and encouraging results of liver transplantation (LTx) have been reported in patients with PVT.[7–9] Although

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no longer considered a contraindication, PVT is still considered a risk factor for LTx.

The consequences of portal vein thrombosis are related to the extension of the thrombus. Upstream from the thrombus, there is little effect on the intestine as long as the mesenteric venous arches remain patent. Ischemia results from extension of the thrombus into the mesenteric veins and the mesenteric venous arches.[10] When ischemia is prolonged for several days, intestinal edema may follow, and translocation of intestinal bacteria may lead to sepsis. Downstream from the portal vein thrombus, the consequences for the liver are hardly discernible[11–13] due to the arterial "buffer" response, which consists of immediate vasodilatation of the hepatic arterial bed in response to a decreased portal vein flow[14] and a rapid development of collateral veins bypassing the thrombosed portion of the portal vein.[15] Portal pressure, however, is increased.[16] In other words, portal perfusion is maintained at the expense of portal hypertension.

Aim

The purpose of this paper is to study the surgical complications following LTx in patients with and without PVT at our center over a period of 12 years at our center.

Patients and Methods

Design

This is a retrospective cohort study.

Primary end-points:

- Rate of PV rethrombosis,
- Rate of primary non-function (PNF)
- Rate of retransplantation,
- Infectious complications,

A retrospective review of 1,171 consecutive patients who underwent LTx between June 1995 and June 2007 was performed, and 84 patients with PVT were identified with an incidence of 7.15%. The diagnosis was made on the basis of preoperative imaging and confirmed on the operative report. Six patients with live donor liver transplant were excluded from the study to minimize bias, so that 78 deceased donor liver transplant (DDLT) recipients (study group) with PVT were compared with a stratified random sample of 78 DDLT recipients without PVT (control group) for postoperative morbidity. The controls were chosen from all the remaining patients without PVT who underwent primary liver transplant during the same time period. One control was randomly selected for each patient from the remaining patients without PVT who underwent primary liver transplantation during the same study period. The baseline characteristics of the two groups were comparable with respect to age, sex, race, Model for End-Stage Liver Disease score (MELD), donor risk index (DRI; calculated, as described by Feng et al.[17]), cold ischemia time, warm ischemia time, and the primary indication for liver transplant (Table 1). Thus, at the outset, the two groups were equally susceptible to develop complications following LTx.

Data were collected by retrospective chart review on postoperative infectious complications (bacterial, viral, fungal, or mixed), portal vein rethrombosis, and primary nonfunction (PNF). We looked at all postoperative (30-day) infections including wound infection, infected hematoma, peritonitis, urinary catheter-related infections, and line sepsis and, based on culture reports, categorized them into bacterial, fungal, viral, or mixed infections. Rate of retransplant was also calculated. In addition, data were collected on etiology of liver failure, MELD score, DRI, preoperative investigations (CECT scan and/or MRI), intraoperative confirmation of PVT, and its extent. Data were also collected on number of blood products transfused (packed red blood cells (PRBC), fresh frozen plasma (FFP), and platelets) and length of hospital stay.

Statistical analyses were performed using SPSS Windows-based version 15.0 (Statistical Package for Social Sciences, SPSS Inc., Chicago, IL, USA). Median and range are used to describe FFP, Platelet and PRBC transfusions and length of hospital stay whereas normal variables are described using mean and standard deviation. All the categorical variables are described using frequency and percentage. The FFP, Platelet and PRBC transfusions were compared between the cases and controls using t-test after taking square root transformation. However, the length of stay was compared using Wilcoxon rank sum test. All the categorical variables were compared between the cases and controls using Pearson's Chi-square test and Fisher's exact test. A p value of <0.05 was considered significant.

Results

Classification of PVT

We classified PVT clinically into partial, if there was some preservation of portal flow, and complete, if there was complete occlusion of the lumen. Fifty-four (69.2%) patients in study group had partial PVT (pPVT), and 24 (30.8%) had complete PVT (cPVT).

PVT was further sub-classified anatomically into four types based on anatomic location of thrombus. This classification was based on preoperative imaging (CT or MRI scans) and intraoperative confirmation.

Type 1 was a pPVT in right or left PV branch (n=12, 15.4%), type 2 was a pPVT in main PV alone (n=34, 43.6%), type 3 was a pPVT in the main PV along with a

Table 1 Demographics

	Study (n=78)	Control (n=78)	p Value
Demographics (study period—June 1995 to June 2007)			
Sex			0.867
Males	51	50	
Females	27	28	
Race			1.000
Caucasian	68	68	1.000
Hispanic	4	4	1.000
Asian	3	3	1.000
African–American	2	2	1.000
Native American	1	1	1.000
Diagnosis			
Laennec's cirrhosis	22 (28.2%)	22 (28.2%)	1.000
Cryptogenic cirrhosis	17 (21.8%)	14 (17.9%)	0.547
HCV	15 (19.2%)	18 (23.1%)	0.556
NASH	9 (11.5%)	4 (5.1%)	0.148
Primary biliary cirrhosis	3 (3.8%)	4 (5.1%)	0.699
HBV	3 (3.8%)	5 (6.4%)	0.468
Autoimmune hepatitis	3 (3.8%)	3 (3.8%)	1.000
Hemochromatosis	2 (2.6%)	2 (2.6%)	1.000
Hepatocellular carcinoma	1 (1.3%)	2 (2.6%)	0.560
α-1-antitrypsin deficiency	2 (2.6%)	0	0.155
Hepatoblastoma	1 (1.3%)	0	0.316
Primary sclerosing cholangitis	0	3 (3.8%)	0.080
Drug toxicity	0	1 (1.3%)	0.316
Other variables			
Age (years) (mean ± SD)	56.76±11.32	57.67±9.33	0.581
Donor age (years) (mean ± SD)	50.21±18.95	51.81±17.75	0.590
Cold ischemia time (hours) (mean ± SD)	11.49±3.24	10.81±3.20	0.215
Warm ischemia time (minutes) (mean ± SD)	45±16	45±18	0.956
DRI[a] (mean ± SD)	2.11±0.44	2.08±0.48	0.675
MELD[b] (mean ± SD)	19.12±7.89	21.06±10.88	0.249

NASH nonalcoholic steatohepatitis, *HCV* hepatitis C viral infection, *HBV* hepatitis B viral infection, *LDLT* live donor liver transplant, *DDLT* deceased donor liver transplant, *DRI* donor risk index, *SD* standard deviation, *ns* not significant, *MELD* model for end stage liver disease

[a] DRI not available for 14 patients in the study group and five patients in the control group

[b] MELD not available for 14 patients operated before Feb 2002 in study group and 12 patients in the control group

thrombus in right or left branch or both (type 3a: n=2, 2.6%) and/or a pPVT in main PV along with a thrombus in superior mesenteric vein (SMV) or splenic vein (SV) or both (type 3b: n=6, 7.7%; total type 3: n=8, 10.3%), and type 4 was a complete thrombus occluding the main PV alone (type 4a: n=15) with or without the right or left branch (type 4b: n=3), and SMV or SV or both (type 4c: n=6; total type 4, n=24, 30.8%, Fig. 1).

Surgical Management

The portal flow was established using portal vein thrombectomy alone in 58 (74.4%) patients, which includes 30 (38.5%) patients with type 2 PVT, seven (8.9%) patients with type 3 PVT, and 21 (27.0%) patients with type 4 PVT.

In eight (10.3%) patients, we had to use a jump/interposition graft because of extensive long-segment occlusion of PVT. This included four (5.1%) patients with type 2 PVT, one (1.3%) patient with type 3, and three (3.8%) patients with type 4a PVT. A venous jump graft to SMV was used in three (3.84%) patients, and a PV interposition graft was used in five (6.41%) patients. One of these patients was retransplanted twice, first for PNF (45 days after primary transplant) and then for HAT (29 days after second transplant), but finally died. In this group of patients where a jump graft was used, three other patients were retransplanted: one for PNF (10 days after primary transplant), one for portal vein rethrombosis, and one for intrahepatic abscess.

In 12 (15.4%) patients, no additional procedure was required since the thrombus was in the right or left PV branch close to the hilum and was removed along with recipient hepatectomy (Table 2). A higher rate of PV rethrombosis, though not statistically significant (p=1.0), was observed in patients who underwent a thrombectomy

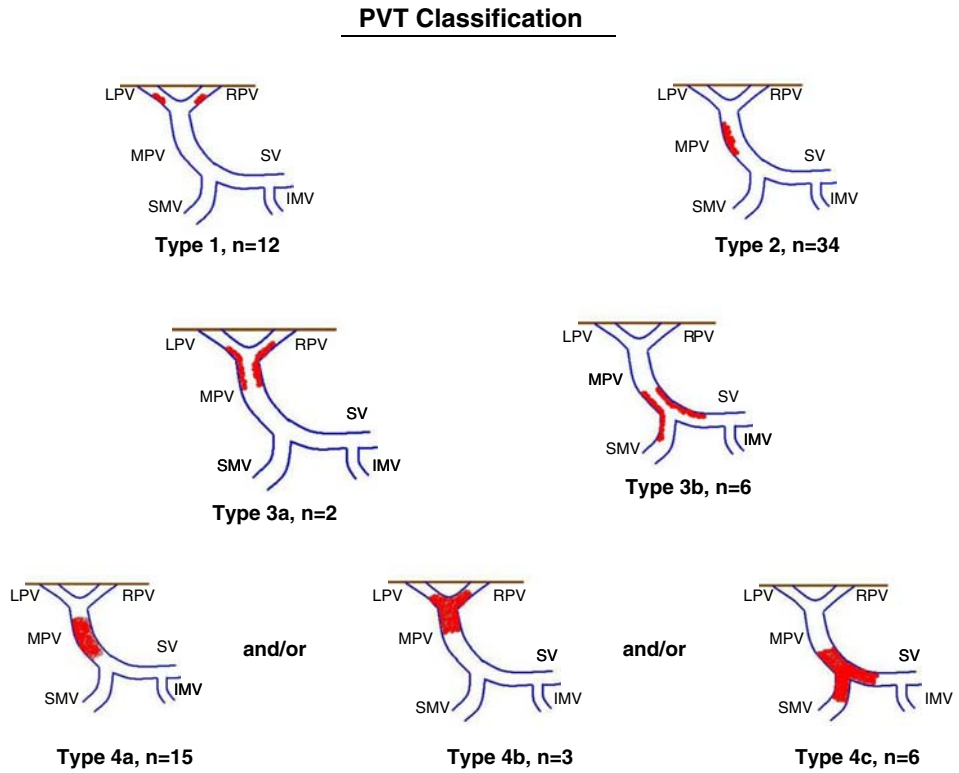

Figure 1 Anatomical classification of portal vein thrombosis. *Type 1* Partial PVT in right or left PV branch. *Type 2* Partial PVT in main PV alone. *Type 3* Partial PVT in the main PV along with a thrombus in right or left branch or both (*type 3a*) and/or a pPVT in main PV along with a thrombus in superior mesenteric vein (*SMV*) or splenic vein (*SV*) or both (*type 3b*). *Type 4* Complete thrombus occluding the main PV alone (*type 4a*) with or without the right or left branch (*type 4b*), and SMV or SV or both (*type 4c*).

Table 2 Operative Findings

Surgical procedure used *n* (%)	Study (*n*=78)
No additional procedure	12 (15.4%)
Type 1	12 (15.4%)
Thrombectomy	58 (74.3%)
Type 2	30 (38.5%)
Type 3	7 (8.9%)
Type 4	21 (27.0%)
Interposition graft	8 (10.3%)
To superior mesenteric vein	3 (3.8%)
Type 2	2 (2.6%)
Type 4a	1 (1.3%)
To portal vein	5 (6.4%)
Type 2	2 (2.6%)
Type 3b	1 (1.3%)
Type 4a	2 (2.6%)

PVT portal vein thrombosis, *CT* computed tomography scan

alone (*n*=4, 6.89%) compared to those with PV interposition graft (*n*=0).

The median number of PRBC, FFP, and platelets transfused were 12, 10, and 10, respectively, in the study group and 9, 9.5, and 10, respectively, in the control group (p=0.012, 0.007, 0.139, respectively). The median length of hospital stay was 19 days in the study group and 15.5 days in the control group (p=0.039) (Table 3). On subgroup analysis of patients in the study group with partial or complete PVT, no significant difference was observed in the rate of biliary, infectious, or thrombotic complications, PNF, or rate of retransplantation in the two groups (Table 4).

PV Rethrombosis

The rate of PV rethrombosis following LTx in the study group was 6.4% (*n*=5). It was managed with rethrombectomy in two patients, retransplant in two, and by anticoagulation in one patient. In the control group, the rate was 2.6% (*n*=2). The difference in incidence in the two groups was not statistically significant (p=0.246, Table 3).

Table 3 Comparison Between Cases and Controls

	Study (n=78)	Controls (n=78)	p Value
Blood products transfused (median)			
PRBC	12 (range 2–81)	9 (range 1–60)	0.012
FFP	10 (range 2–91)	9.5 (range 2–64)	0.007
Platelets	10 (range 2–75)	10 (range 5–40)	0.139
Hospital stay (median, days)	19.0 (range 7–173)	15.5 (range 8–219)	0.039
Portal vein thrombosis, n (%)	5 (6.4%)	2 (2.6%)	0.246
Infectious complications, n (%)	43 (55.1%)	37 (47.4%)	0.360
Bacterial	35 (44.8%)	29 (37.2%)	0.570
Fungal	3 (3.8%)	0 (0.0%)	0.093
Viral	1 (1.3%)	2 (2.6%)	0.513
Mixed	4 (5.1%)	6 (7.7%)	0.432
Primary nonfunction, n (%)	7 (8.9%)	1 (1.3%)	0.063
Rate of retransplantation, n (%)	14 (17.9%)	6 (7.7%)	0.055

Infectious Complications

In the study group, the rate of infectious complications following LTx was 55.1% (n=43), of which 35 (44.9%) were bacterial, three (3.8%) were fungal, one (1.3%) was viral, and four (5.1%) were mixed infections. In the control group, the rate of infectious complications was 47.4% (n=37), of which 29 (37.2%) were bacterial, two (2.6%) were viral, and ix (7.7%) were mixed infections (Table 3). No fungal infections were observed in the control group. The difference in incidence of infectious complications between the two groups was not statistically significant (p=0.360).

Primary Non-Function and Retransplant

The incidence of PNF was 9.0% (n=7) in the study population, while only one (1.3%) patient in the control group had a PNF (p=0.063, Table 3). The mean DRI in patients with PNF was 2.58±0.44, while in patients in the study group who did not have PNF, DRI was 2.08±0.42 (p=0.014).

The rate of retransplantation in the study group was 17.9% (n=14) and 7.7% (n=6) in the control group (p=0.055). The most common cause of retransplant was PNF (n=6, 7.7%) in the study group and HAT (n=2, 2.6%), recurrent HCV (n=2, 2.6%) in the control group (Table 5). The most common cause of death in both groups was sepsis (n=19, 24.4% in study group, n=9, 11.5% in control group, p=0.095). The causes of retransplantation and death are summarized in Table 5.

Discussion

PVT is a well-recognized complication of end-stage liver disease and occurs in 5% to 15% of patients suffering from this condition.[1] An incidence of 7.15% was observed in our study. PVT has been reported traditionally as partial or complete.[18,19] We find this appropriate for comparison of clinical outcomes. Other authors have classified it into grades 1–4 depending upon the site of thrombus and percentage occlusion of the lumen,[20,21] which appears confusing and incomplete. We believe that an anatomic classification would be simple and more reasonable for reporting the findings and comparing results between institutions and have suggested one such classification (Fig. 1). However, for making clinical comparisons, classification of PVT into partial and complete appears appropriate.

When recognized in advance, PVT was once considered an absolute contraindication to liver transplantation[2–4] until

Table 4 Comparison Between Partial and Complete PVT

	pPVT (n=54)	cPVT (n=24)	p Value
Portal vein thrombosis, n (%)	3 (5.6%)	2 (8.3%)	0.644
Infectious complications, n (%)	32 (59.3%)	11 (45.8%)	0.271
Primary nonfunction, n (%)	5 (9.3%)	2 (8.3%)	0.895
Rate of retransplantation, n (%)	9 (16.7%)	5 (20.8%)	0.658

PVT portal vein thrombosis, *PRBC* packed red blood cells, *FFP* fresh frozen plasma, *DRI* donor risk index, *MELD* model for end-stage liver disease, *SD* standard deviation, *pPVT* partial portal vein thrombosis, *cPVT* complete portal vein thrombosis

Table 5 Causes of Retransplant and Death

Cause	Study (n=78)	Controls (n=78)
Retransplant n (%)		
Primary nonfunction	6 (7.7%)	0 (0.0%)
Hepatic artery thrombosis	2 (2.6%)	2 (2.6%)
Portal vein rethrombosis	2 (2.6%)	0 (0.0%)
Biliary cast syndrome	1 (1.3%)	1 (1.3%)
Recurrent HCV	1 (1.3%)	1 (1.3%)
Intrahepatic abscess	1 (1.3%)	0 (0.0%)
Recurrent HBV	1 (1.3%)	0 (0.0%)
Recurrent PSC	0 (0.0%)	1 (1.3%)
HBV	0 (0.0%)	1 (1.3%)
Death n (%)		
Sepsis	19 (24.4%)	9 (11.5%)
Unknown	3 (3.8%)	2 (2.6%)
Metastatic cancer	3 (3.8%)	4 (5.1%)
Cardiac arrest	3 (3.8%)	7 (9.0%)
Liver failure	1 (1.3%)	0 (0.0%)
Intracranial bleed	0 (0.0%)	1 (1.3%)

HCV hepatitis C viral infection, *PSC* primary sclerosing cholangitis, *HBV* hepatitis B viral infection

good results were obtained with thrombectomy or a venous interposition graft between the donor portal vein and splenomesenteric confluence.[5] Some of the recent studies have reported encouraging results of LTx in patients with PVT.[7–9] We did not exclude any patient, including those with complete thrombus extending to SMV or SV. The type of operative strategy depends on the extent of thrombosis. Thrombectomy and direct venous anastomosis is recommended in patients when the thrombosis is partial and involves portal vein with or without SMV.[6,20,22] In cases of complete PVT with patent SMV, venous jump graft to SMV is an alternative.[6,20,22] In cases where the portal vein is not amenable to thrombectomy, and the SMV is also thrombosed, the coronary vein can also be used for inflow.[22] For the patients with extensive and complete occlusion of the portal and proximal SMV as well as the distal SMV, cavoportal hemitransposition has been described;[23] however, the survival rate is significantly reduced when this technique is employed and the complication rate among survivors is significant.

In our study, during surgery, declotting of PV was done in all patients. If after declotting, good flow was established, nothing else was done; if not, then a jump graft to SMV or a PV interposition graft was used. Among 12 (15.4%) patients with type 1 PVT, no additional surgical procedure was needed since, being close to the liver hilum, this portion of portal vein was excised as a part of recipient hepatectomy. A good portal flow was achieved using thrombectomy alone in 58 (74.3%) patients (Table 2). In eight (10.3%) patients, we had to use a jump graft because of extensive long-segment occlusion of PVT. A jump graft to SMV was used in three and an interposition graft to PV in five patients.

Another major risk for liver transplant recipients with PVT is early rethrombosis. The incidence varies from 4.2% to 38.5%.[6,19,24–29] The use of therapeutic[28] or prophylactic[25] anticoagulation for 3 months to prevent thrombosis has been recommended. We routinely use anticoagulation after LTx to prevent rethrombosis. Partial rethrombosis might manifest with acute deteriorating liver function and complications secondary to portal hypertension, such as ascites or gastrointestinal bleeding. If detected early, it can be treated effectively with rethrombectomy. However, a delay in this diagnosis may lead to graft loss and retransplantation.[8] Frequent Doppler ultrasonography in the post-LTx period may prevent delay in the diagnosis of PVT and, therefore, the need for retransplantation and is recommended every 1 to 3 days during the first 2 weeks after LTx.[30,31]

The incidence of rethrombosis in our series was 6.4% (n=5). In two of these patients, there was a graft loss, and they had to be retransplanted. In one, it was managed with anticoagulation using heparin, and in another two patients, it required a re-exploration with rethrombectomy. One patient died following rethrombectomy, 1.4 months after re-exploration, the remaining four are alive and doing well. While in the control group, the observed incidence of portal vein thrombosis after LTx was 2.6% (n=2). The most important risk factor for rethrombosis is believed to be the extent of thrombus within the portal venous bed, and it has even been recommended to avoid retransplant in patients with complete PVT with extension throughout the portal venous bed.[8]

We found that four patients (6.9%) out of 58 who had undergone thrombectomy had a rethrombosis, while one patient out of three who received a jump graft to SMV had a rethrombosis and none of the five patients who received a PV interposition graft had rethrombosis. A probable explanation could be intimal injury following thrombectomy with resultant higher incidence of rethrombosis while patients with a PV interposition graft receive a healthy vessel with resultant no rethrombosis. The reason for rethrombosis in the patient who received a jump graft to SV was related to kinking of the vessel. A strategy of doing a PV interposition graft in difficult cases instead of thrombectomy could lead to a lower rethrombosis rate.

A significantly higher number of PRBC and FFP transfusion were recorded in study group as compared to the controls, highlighting the complexity of the surgical procedure with a difficult dissection and more intraoperative bleeding.

Interestingly, the incidence of PNF was 8.9% in the study population and 1.3% in the control group. This difference approached statistical significance. A possible explanation could be the fact that a difficult portal dissection in the presence of occluded portal vein and severe portal hypertension results in significant bleeding during hepatectomy. As a result, during the anhepatic phase, the patients develop severe acidosis and coagulopathy. The new liver graft is thus transplanted under less than optimal conditions in a compromised host, and the probabilities for failure are increased.[29] We postulate that this partly explains the high rate of PNF in the graft.

Out of the seven patients with PNF in the study group, six patients were retransplanted 3, 10, 29, 47, 8, and 5 days, respectively, after primary transplant. One died without intervention 45 days following LTx. The mean DRI in patients with PNF was significantly higher than patients in the study group who did not have PNF, which could also be a contributing factor for PNF.[17] The only patient with PNF in the control group died without intervention soon after primary transplant.

The rate of retransplantation in the study group was 17.9% (n=14), while in the control group, it was 7.7% (n=6). This difference could be accounted for by a higher incidence of PNF and portal vein rethrombosis in the study group compared to the controls.

Liver transplant recipients with PVT, especially the patients who have more than 50% of portal vein occlusion with or without SMV occlusion, are considered more prone to develop severe perioperative complications and a higher mortality rate.[8,20,32] Theoretically, one would assume a greater incidence of postoperative infectious complications in patients with complete and long-standing PVT as a result of mesenteric ischemia,[10] intestinal edema with consequent bacterial translocation, and sepsis. It is quite difficult, however, to attribute postoperative infection to PVT due to the involvement of several factors which can give rise to infection in the postoperative period. We looked at all postoperative (30-day) infections. We, however, did not find any statistically significant difference in postoperative infectious complications. However, there was a relatively higher incidence of fungal infections in the study group (n=3, 3.8%) compared to controls (n=0). This might suggest the need for prophylactic antifungal therapy in patients with PVT undergoing LTx, though the evidence in support for prophylactic antifungal therapy is not strong.

In conclusion, a higher rate of PNF was related to both the complexity of the surgical procedure and the use of donor livers with a high DRI. Higher rates of PNF eventually led to a higher rate of retransplant. A strategy of offering donor livers with a low DRI might be helpful in decreasing the rate of PNF in patients with PVT, though on the basis of this retrospective analysis alone, it is difficult to make a compelling argument. Further, a PV interposition graft in difficult cases instead of thrombectomy could lead to a lower rethrombosis rate. However, given the retrospective nature of the study, the evidence in support of these conclusions is not strong, and multicenter studies are needed to establish concrete recommendations.

Conflicts of Interest None

Prior Publication None

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