Ann Med Res

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Early portal vein thrombosis after pediatric liver transplantation: Assesment of risk factors

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ARTICLE INFO

Keywords:
Pediatric liver transplantation
Portal vein
Thrombosis

Received: Nov 01, 2022
Accepted: Jan 30, 2023
Available Online: 27.02.2023

DOI:
10.5455/annalsmedres.2022.11.330

Abstract

Aim: Despite advancements in surgical techniques, early portal vein thrombosis (ePVT) continues to be one of the major complications of liver transplantation (LT) in pediatric age group. Possible risk factors are portal vein diameter < 5 mm, infancy, patient body weight < 10 kg and high graft recipient weight ratios (GRWR > 4.0). We retrospectively evaluated our records of pediatric LTs' in terms of ePVT and possible risk factors determining development of this dreaded complication.

Materials and Methods: Between January 2018 and January 2022, 228 LTs were performed for pediatric age (under the age of 18) group at Inonu University, Liver Transplantation Institute. Among these patients, 212 were eligible for the study. Patients with ePVT were defined as Portal Vein Thrombüs Group (PVTG) and patients with no Portal Vein thrombosis were defined in control group (CG). ePVT was described as detection of impeded portal venous outflow with imaging studies either perioperatively or within postoperative 3 days . Demographic, clinical and operative variables were retrospectively evaluated.

Results: Among 212 LTs, 24 cases were complicated with ePVTs (11.3 %). Preoperative platelet counts, etiology of Budd-Chiari, postoperative hepatic artery thrombosis (HAT) and lower age were significantly higher for early PVT. In multivariate analysis, preoperative platelet levels, etiology of Budd-Chiari and postoperative HAT were significantly higher for PVT. One and 5 years overall survivals (OS) for PVTG and CG were 50.0 % - 50.0 % and 69 % - 63 % respectively. No significant OS difference was observed despite much more patients were died in PVTG.

Conclusion: High preoperative platelet counts, Budd-Chiari syndrome and postoperative HAT are predictive factors for ePVT. Anti-thrombotic prophylaxes can be considered in high-risk patients. Venous jump grafts seem to have no effect on ePVT. Despite PVT increases the mortality rates, it can be resolved easily with immediate reoperation.

Introduction

Liver transplantation is the only means of cure for the patients with end-stage liver disease. Living donor liver transplantation (LDLT) is a strategy to reduce the organ demand of the patients in the waiting list. It is especially important for countries where deceased donor organ supply is limited [1]. It offers a sizable graft suitable for the patient's body weight and enables the operation to be a planned procedure because the graft is accessible on demand. Furthermore, recent studies have shown that the results of LDLT have been better than deceased donor LT in terms of patient/graft survival and perioperative complications [2,3].

In general, LT in pediatric patients have specific technical pitfalls and complications because of the discordance between the caliber of the vascular structures of the graft and recipient [4]. Bezinover et al. have stated that pediatric LTs' are especially prone to thrombotic perioperative complications when compared with adult patients [5]. Most frequent vascular complications following pediatric LT are hepatic artery and portal vein thrombosis (PVT) which are responsible for graft loss and patient morbidity [6].

PVT is still a dreaded complication of pediatric LT, despite decreasing rates with the improvement of surgical

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techniques [7, 8]. Hypoplastic or narrow portal vein, low recipient body weight (< 10 kg), infancy and younger age of the recipient (especially less than 12 years of age), high graft recipient weight ratios (GRWR > 4.0), etiologic factors such as biliary atresia and previous abdominal operations (Kasai procedure) are reported as the main contributory factors for this entity [9]. Other risk factors are hypercoagulable states, such as patients with Budd-Chiari syndrome, patients who have undergone splenectomy, and patients with inadequate anti-thrombotic prophylaxis [10, 11].

Early PVT is defined as thrombosis of Portal Vein within the 10 days of postoperative period [8]. Although late PVT is more frequent than early PVT (ePVT), ePVT is associated with increased mortality and fortunately is amenable to revisional surgery. Therefore, we aimed to focus on ePVT among pediatric LT patients to evaluate our diagnostic and management protocol and also to evaluate the risk factors associated with ePVT.

Materials and Methods

Study population

Total of 228 pediatric LTs (Under the age of 18) were performed between January 2018 and January 2022 in our institute. Patients with insufficient medical records were excluded. After exclusion, total of 212 cases were studied. The study was performed by a retrospective analysis of the prospectively collected database. Patients with ePVT with complete follow up parameters were included for the analysis in the study and named as Portal Vein Thrombus Group (PVTG). To determine the risk factors for ePVTs, a control group (CG) was created. CG consisted of the cases other than the ePVTs.

Study design and the study parameters

ePVT is defined as any disturbance in portal venous flow detected intraoperatively or within the first postoperative 10 days that is caused by a thrombosis at the portal vein anastomosis site. Therefore, according to timing of the diagnosis of the PVT, there are two clinical scenarios: a) The patients that are detected perioperatively, are usually detected during the intraoperative surveillance Doppler ultrasound, performed just after the implantation of the liver graft or during the first Doppler ultrasound examination performed in the intensive care unit (ICU) within the postoperative 24 hours, b) Within the postoperative 10 days, if the patient developed graft dysfunction and the imaging studies (such as Doppler ultrasound or multidetector computerized tomography) showed a PVT impeding the portal flow.

The study parameters included gender, age, etiology, recipient weight, height, type of LT, GRWR, pediatric end stage liver disease (PELD) or model for end stage liver disease (MELD) scores, warm and cold ischemia times (WIT, CIT), intraoperative blood transfusion, duration of surgery, intraoperative use of cryopreserved venous grafts (CVG) for portal vein inflow reconstruction, presence of previous abdominal surgery, preoperative platelet counts, total bilirubin levels and preoperative ICU stay.

Ethical approval was obtained from the Inonu University Health Sciences Clinical Research Ethical Commitee (Decision number: 2022/4001).

Surgical technique

Our institute is a center of excellence for LDLT and majority of the cases were LDLTs (195 of the 212 patients). Majority of the living donor liver grafts were segment 2-3 (n: 106) or reduced segment 2-3 liver (n: 34) grafts. Briefly, we start the recipient hepatectomy by division of the triangular ligaments suspending the right and left lobes of the liver. After the dissection of the bare area of the liver and separation of the right adrenal gland from the right lobe of the liver; full length of retro-hepatic vena cava is dissected. We use the piggy-back technique for reconstructions of the venous out-flow of the liver graft and for this reason the native vena cava is always preserved. The hilar dissection is usually started from the right side of the hepatoduodenal ligament. The right hepatic artery is visualized, and the terminal branches are ligated and divided. The left hepatic artery is also identified and mobilized from the surrounding tissues. Common hepatic artery is routinely identified together with the gastroduodenal artery. The peri-choledochal lymph nodes are occasional dissected for better visualization. The portal vein is identified from the right side of the hepatoduodenal ligament, and it is dissected throughout its whole length from the hilar plate above the bifurcation to the junction of the SMV and splenic vein in the retro-pancreatic region. Hilar structures are divided first and followed by the division of the hepatic veins during recipient hepatectomy. After the recipient hepatectomy is completed, the hepatic vein anastomosis is performed followed by the portal anastomosis.

When the GRWR is about 4 and graft artery is too short, we perform hepatic artery anastomosis before portal vein reconstruction. In patients with hypoplastic or occluded portal veins, (diameter < 5mm) CVGs derived from cadaveric veins are used to reconstruct porta inflow. The portal CVG reconstruction is performed between the superior mesenteric vein (SMV) - splenic vein confluence and the graft portal vein (Figure 1).

CVG was anastomosed to SMV - splenic vein confluence with 8/0 polypropylene sutures (Ethicon USA) interrupted sutures and to graft portal vein with 7/0 polypropylene interrupted sutures respectively. We usually infuse about 100 ml of warm saline into the SMV to provide optimal reperfusion pressure in pediatric patients with hypoplastic or occluded portal vein just before portal reperfusion. In patients with portal vein diameter is about 5 mm or above, the flow is evaluated by temporarily removing the portal clamp to observe the flow. If the portal flow is insufficient, then a CVG is also anastomosed to the confluence of SMV and splenic vein with the resection of the hiler part, regardless of the native portal vein diameter. Biliary reconstruction is the last step and can either be duct-to-duct anastomosis or bilio-enteric anastomosis depending on the underlying etiology of liver failure (such as biliary atresia) or the age of the patient (<2 years). We primarily close the skin but not the fascial structures and occasionally, some patients need Bogota bag reconstruction to prevent abdominal hypertension and compression of hilar structures.

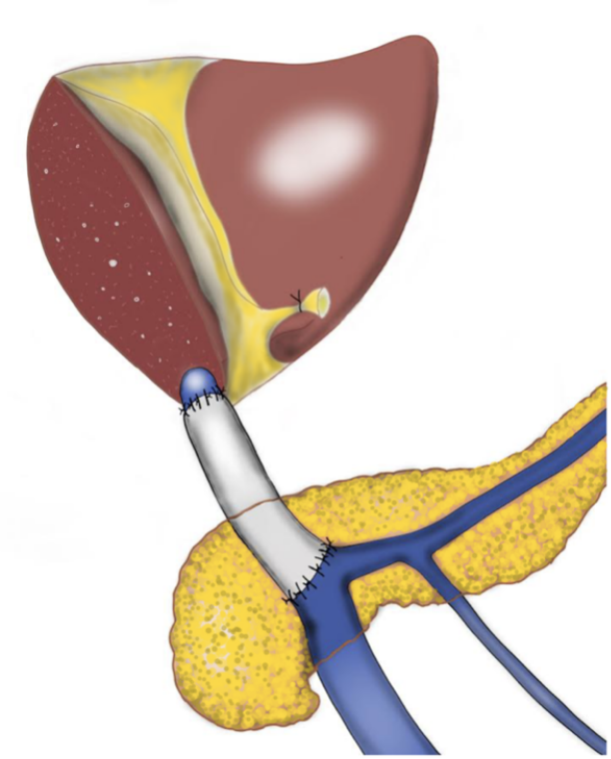

Figure 1. CVG anastomosis to Portal Vein root.

Postoperative follow-up

All patients received low molecular weight heparin starting from the first postoperative day to until postoperative 10th day as 50 IU/kg once a day. We do not use warfarin treatment in patients with preoperative portal vein hypoplasia or thrombosis. Acetyl- salicylic acid (5 mg/kg/day) is started on the postoperative 10th day with the ceasing of low molecular weight heparin. Doppler ultrasonography is performed every day for the postoperative 10 days. A routine control computerized tomography scan is performed on the postoperative course after 10 days. Our immune suppressive protocol includes initiation of methylprednisolone therapy after the completion of hepatic artery anastomosis (20 mg/kg). Then we taper steroid therapy to 5 mg/day.

Tacrolimus is started on the postoperative first day (0,05 mg/kg/day). We daily monitor the trough levels of tacrolimus and desired levels are kept between 7-10 ng/mL. Tacrolimus and low dose steroid regimen in the first 3 months is the main immunsupressant protocol. Survival was measured using the last out-patient clinic visit of the patients or date of death.

PVT was detected in two ways; First one is intraoperative detection using Doppler ultrasonography just after the all vascular anastomosis were completed. Second, during the routine Doppler ultrasonography in the postoperative period this is performed for the postoperative first 10 days. In all these scenarios, if the portal flow is not detected, we take down the portal anastomosis and thrombectomy is performed. For perioperative thrombosis, revision of the anastomosis is sufficient. However, if the thrombus is not just at the anastomosis site but also through the graft portal vein site (detection with both laparatomy and intrahepatic Doppler ultrasound), then we perform autotransplantation (explanation of the graft and re-implantation) of the graft, with perfusing the graft with 150 ml of saline including with 5000 IU of heparin to clear the intrahepatic branches of the portal vein. Partial thrombosis, seen at CT in the portal vein after day 10, that did not impede the portal flow and did not affect the biochemical parameters, were not defined as PVT. In this case, low molecular weight heparin followed by warfarin prophylaxis is our treatment of choice. Interventional radiology is only used for patients who have late PVTs; together with anticoagulant therapy.

Statistical analysis

Continuous variables were expressed as median (range: min-max) if the data distribution was homogenous and as mean (± SD) if the data distribution was heterogeneous. Categorical variables were expressed as percentage of the study cohort including the number of individuals that were affected. Categorical variables were compared with Fisher exact test and Chi-square test. Continuous variables were compared Mann-Whitney U test. Any p value smaller than 0.05 is considered to be statistically significant. Kaplan-Meier analysis was performed to evaluate the patient survival. Significant variables in the univariate analysis were subjected to Cox regression model for multivariate analysis in order to determine the independent risk factors for complications in the portal inflow reconstruction. Receiver Operating Characteristics curve analysis was performed to determine the optimal cut-off value for continuous variables in accord with the development of postoperative PVT and continuous variables were converted to categorical variables for multivariate analysis. Statistical analysis was performed with "Statistical software Package for Social Sciences, SPSS version 24.0".

Results

General characteristics of the study population

LDLT was the main operation that performed to 195 of 212 (91.9 %) patients. Among LDLTs, 47 cases (24.1%) were left, 106 were segment 2-3 (54.4%), 34 were reduced segment 2-3 (17.4%) and 8 cases (4.1%) were right lobe LTs. In the remaining 17 cases, 12 cases were full size and 5 cases were split cadaveric liver grafts. Median GRWR was 2.2 % in all study population (min: 0.7 - max: 5.7). It was 2.3 % for PVT group and 2.2 % for CG. There were total of 122 male and 90 female patients. Majority of the study population was between 0-5 years of age (n:118) (55.7 %). Most common etiology was biliary atresia (n: 53). There were total of 31 fulminant etiology and 2 of them were complicated with ePVT (Table 1). Four of the 7 patients with Budd-Chiari syndrome were complicated with ePVT (57.1 %). Iliac CVG was used in 32 of 212 patients (15 %). Demographic, clinical and operative data of the study cohort are summarized in are shown at Table 1.

Evaluation of the patients with and without ePVT

Among 212 patients that were included in the study, 24 cases had ePVT (11.3 %). There was no difference between groups in the manner of sex, weight, height, PELD/MELD

Table 1. Demographic and clinical parameters of study groups (ePVT: Early Portal Vein Thrombosis, CG: Control Group).

Parameter	ePVT Group	CG (Control Group)	p value
Gender			
Male (n)	14 (58.3 %)	108 (57.4 %)	1.00
Female (n)	10 (41.7 %)	80 (42.6 %)	
Age (Mean)	45.9 (± 48.0)	53.9 (± 49.1)	0.45
Weight (kg) (Mean)	17.4 (± 14.8)	19.9 (± 16.2)	0.49
Height (cm) (Mean)	93.8 (± 35.4)	102.3 (± 34.1)	0.25
PELD/MELD score (Mean)	20.3 (± 6.4)	21.1 (± 7.4)	0.62
Etiology			
Fulminan	2 (8.3 %)	29 (15.4 %)	0.001 *
Chronic	18 (75.1 %)	156 (83.0 %)	
Budd-Chiari *	4 (16.6 %)	3 (1.6 %)	
Transplant Type (n)			
DDLT	0 (0 %)	17 (100 %)	0.23
LDLT	24 (12.3 %)	171 (87.7 %)	

scores, GRWRs and LT type (Table 1). Age, the etiology of the liver disease (Table 1), hepatic artery trombosis (HAT) and preoperative platelet counts were significantly different among the groups (Table 2). The emergency LTs due to fulminant liver failure or other causes of chronic liver failure did not show significant difference among the groups.

All of the ePVTs were detected either within the operation or within the postoperative 48 hours. There were no ePVTs were detected after 2 days. Eleven ePVTs were detected intraoperatively, 11 ePVTs were detected within postoperative 24 hours and 2 were detected within postoperative 48 hours. Immediate reoperation and primary portal re-anastomosis, CVG reconstruciton to portal vein root or reimplantation of the liver were the main surgical treatments for ePVT.

There were 31 CVGs used for hypoplastic or insufficient flowed recipient portal veins (14.6 %). No difference was observed between PVTG and CG in the manner of CVG usage (Table 2). Despite CVG did not affect the PVT situation in the cohort, CVGs were used more in the early age group than the olders (p= 0.001) (HR: -31.7, 95 % CI: -49.9, -13.4).

Significant factors for ePVT, which were the etiology of Budd-Chiari syndrome, early age, preoperative high platelet counts and postoperative HAT also re-evaluated in binary logistic analysis and etiology of Budd-Chiari, preoperative high platelet count and postoperative HAT were independently significant for ePVT group either (Table 3).

Survival analysis

Overall survival (OS) was 1115 ± 55. It was 687 ± 132 days for PVTG and 1146 ± 57 days for CG. OS was much better in patients without ePVT but not significant statistically (p=0.074) (Figure 2). One and 5 years OS for PVTG was 50.0 % - 50.0 % and for CG was, 69 % - 63 %.

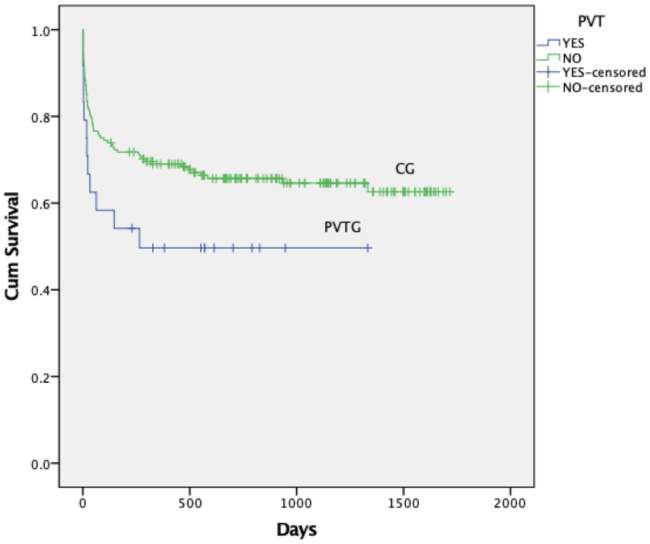

Figure 2. Survival for ePVT (p=0.074)

Discussion

PVT is a serious complication after pediatric LT, especially in small children. Risk factors for PVT have been stated as hypoplastic or narrow portal vein diameter (<5mm) low body weight, use of split cadaveric donors, young recipient age group and high GRWR [7, 12, 13]. Furthermore, biliary atresia and associated previous abdominal surgery such as Kasai procedure have been shown to be associated with hypoplastic portal vein and consequently PVT [14, 15]. As a consequence of the above-mentioned risk factors, ePVT is observed more frequently following LT in pediatric patients. Mainly, this is the result of portal vein hypoplasia and reduced flow in the portal circulation [16]. Vasavada et al. have stated that recipients who have a body weight lower than 10 kg had significant risk of ePVT following LT [17]. The results of our study showed that recipient weight and age did not have an impact on the development of ePVT. The current literature regarding PVT is abundantly from the western literature which usually includes older age pediatric patients who received full-size deceased donor liver grafts. In Turkey, LDLT is the preferred method for LT. Therefore, the graft type may have affected the incidence of ePVT regardless of the age and weight of the patients. In a review by Alvarez et al stated that risk factors for postoperative portal vein anastomotic complications are related to technical or flow related problems rather than the age of the patients [18].

In our opinion, dominancy of left lobe grafts of our series may be a possible reason for ePVT regardless of low age and weight. We think that, divergence of recipient portal veins anatomical direction from right upper direction to left upper direction leads the same divergence of portal flow and this may result in thrombosis.

It has been shown that diseases such as biliary atresia have higher risk of pre and post-transplant PVT. This

Table 2. Univariate analysis of clinical parameters for PVT (ePVT: Early Portal Vein Thrombosis, CG: Control Group).

Parameter	ePVT Group	CG (Control Group)	p value
GRWR (Mean)	2.38 (± 1.05)	2.31 (± 10.6)	0.75
Warm Ischemia Time (min) (Mean)	56.0 (± 21.4)	54.5 (± 18.2)	0.69
Cold Ischemia Time (Min)	109 (± 98)	110 (± 97)	0.99
Duration of Operation (min) (Mean)	426 (± 79)	420 (± 91)	0.76
Intraoperative Blood Transfusion (ml/kg) (Mean)	14.7 (± 18.7)	12.3 (± 26.4)	0.67
Cryopreserved Venous Graft			
Yes	4 (16.7 %)	28 (14.9 %)	0.77
No	20 (83.3 %)	160 (85.1 %)	
Previous Kasai Operation			
Yes	6 (25.0 %)	28 (14.9 %)	0.24
No	18 (75.0 %)	160 (85.1 %)	
Preoperative Platelet Count (µL) (Mean)	250.7 (± 125.2)	188.8 (± 156.6)	0.06
Preoperative Hematocrit Levels (Mean) (percent)	30.9 (± 5.7)	30.4 (± 5.7)	0.67
Preoperative Bilirubin Levels (Mean) mg/dl	13.0 (± 13.1)	16.1 (± 14.1)	0.31
Preoperative CRP Levels (Mean)	1.73 (± 2.04)	1.84 (± 3.43)	0.37
Preoperative Albumin Levels g/dl (Mean)	3.1 (± 0.84)	3.0 (± 0.80)	0.40
Preoperative Spleen (cm) (Mean) Diamater	10.3 (± 2.7)	11.9 (± 4.7)	0.12
Preoperative INR (Mean)	1.49 (± 0.49)	2.01 (± 1.70)	0.14
Postoperative HAT			
Yes	4 (16.7 %)	8 (4.3 %)	0.03 *
No	20 (83.3 %)	180 (95.7 %)	
Overall Survival (Days) (Mean)	687 (± 132)	1146 (± 57)	0.07

Table 3. Binary logistic analysis for ePVT.

Variables	% 95 CI	Hazard Ratio	p value
Preoperative Platelet Count	0.994 - 1.000	0.997	0.028 *
Etiology (Budd-Chiari)	0.005 - 0.440	0.046	0.008 *
Preoperative INR	0.747 - 4.853	1.904	0.177
Postoperative HAT	1.232 - 20.919	5.076	0.025 *
Spleen Diameter (cm)	0.862 - 1.219	1.025	0.781
Age	0.995 - 1.018	1.006	0.262

is due to significant perihilar inflammation that leads to portal hypoplasia [9]. Especially if the patients underwent Kasai procedure for biliary atresia, the risk of PVT increases nearly 5 times more than when compared to patients with biliary atresia who have not undergone Kasai portoenterostomy [19]. In the present study, although it was not statistically significant, our results support that of the current literature and we also have observed higher frequency of ePVT in patients with biliary atresia who have undergone Kasai procedure. Our results showed that patients transplanted for Budd-Chiari syndrome had higher rate of ePVT (16.7 % versus 1.6 %). This may due to the higher incidence of primary or secondary hypercoagulable states in these patients. In Budd-Chiari syndrome, 75 % of the patients have a congenital hypercoagulable disease such myeloproliferative diseases, presence Factor V Leiden mutation, presence of anticardiolipin antibodies, low levels of protein C, protein S, and antithrombin III [20]. Both McLin V et al and Hardikar W et al stated that, fresh frozen plasma infusions after hepatectomy phase resulted in significant low ePVT rates [21, 22]. They explained this issue as, anticoagulant factors such as anti-thrombin III, protein C and S in recipient do not come back immediately after implantation in infants but aPTT and aPT levels return. Also, while some thrombotic markers such as Von Willebrand Factor and Factor VIII are already significantly higher in infants but anti-thrombotic factors are physiologically low [23]. That's why, infusing FFP can supply anticoagulant factors such as anthi-thrombin III, protein C, protein S and tissue factor pathway inhibitor, which may balance the effect of already high concentrations of procoagulant factors. Unfortunately, we do not have any data on the hypercoagulable states of our patients with Budd-Chiari syndrome.

Our results showed that high preoperative platelet levels were significantly associated with high ePVT. Our results are in accordance with the study by Li et al. [24]. They have hypothesized that Von Willebrand Factor (VWF) is 10 fold higher in cirrhotic patients and physiologically, VWF supports platelet adhesion. Some studies showed that, the activated platelets after LT can release active microvescular contents releasing high amount of vWF to the circulation. Furthermore, they form pseudopods on their surface that promote their interaction with neutrophils and other immune cells [25]. These interactions promote the thrombocyte induced inflammation, endothelial cell activation, which may lead to higher incidence of portal vein complications. Voulgarelis et al. [26] stated that, during the anhepatic phase of the transplantation, additional platelets become sequestered in the spleen. After immediate decrease in portal pressure, these platelets are

being redistributed in systemic circulation. Linares et al. [11] stated that number of units of platelets transfused during transplantation was significantly higher for PVT in their study. In our opinion, ischemia and reperfusion injury after anhepatic phase activates both thrombocytes and the endothelial cells. The postreperfusion period induces potent inflammation, endothelial cell and thrombocyte interaction; all of which leads to a hypercoagulable state. Another explanation of this finding is, when the patient has splenomegaly and after the implantation of new graft, a massive splenic flow goes through portal vein and PVT rates decreases. But when spleen is normal sizes, splenic vein flow is not too much. Therefore, low platelet levels due to splenomegaly with low PVT rates may be due to this condition and thrombocytes are the key elements of circulation which should be targeted for developing an effective therapy for posttransplant PVT.

Postoperative HAT was in accordance with ePVT significantly. There are studies that mention HAT and PVT may affect each other, especially when there is a preoperative PVT, there's a high probability of postoperative HAT [27, 28]. Although there are reports that mention both HAT and PVT occurred early postoperative period after LT [29]. In our aspect, accordance of postoperative HAT and PVT is in favour of our hypothesis that postoperative PVT is a hematologic thrombotic problem. If the problem was due to venous perihilar collateral circulation then there were not such more HAT cases along with PVT at the same time. On the other hand, one may assume that preoperative PVT should be evaluated for both postoperative PVT and HAT. But our most cases were not having a specifically ceasing of portal flow intrahepatic or extrahepatic site in preoperative images, but having a narrow portal flow with or without venous collaterals. That's why, we did not include the preoperative PVT factor to the analysis.

Infants have narrower portal vein than older ones. Suzuki et al reported that, portal vein diamater < 3.5 mm is a significant risk factor for portal vein complications [30]. In our study, we used CVGs to all patients with portal vein hypoplasia (narrow portal vein; PV diameter < 5 mm); though we are trying to revise our reconstruction technique. Our results regarding CVG reconstructions have previously been published [31]. Unfortunately, patients who require CVGs for portal vein reconstruction have multiple adverse factors including reduced splanchnic blood flow and presence of preoperative PVT thus intraoperative need for use of CVGs means that these patients have either hypoplasia or thrombosis and these reduce our success in maintaining adequate portal inflow. There are studies recommending not to use CVGs for portal vein because of their poor results for PVT; especially because of poor outcomes in the long-term [7, 32, 33]. In the present study, we found that, the needs for CVGs for reconstruction of the portal flow were not correlated with ePVT. Narrow (< 4mm) portal veins are a significant factor for ePVT but we used CVG to all narrow portal veins routinely. In accordance with our better results, we can conclude that CVGs are feasible for hypoplastic portal vein reconstruction in order to prevent ePVT. Thus, we recommend that in necessary cases if primary anastomosis failed or if there is portal vein hypoplasia, CVGs are important for reconstruction of the portal inflow.

Although the patients with ePVTs had worse survivals than the non-complicated patients, it was not statistically different. In our opinion, early and immediate surgical intervention can easily solve this complication and its effects on the liver and survival. This is a very important finding of our study.

Conclusion

In conclusion, ePVT is a frequent complication of pediatric LT. Rapid intervention including revision of the anastomosis or reimplantation increases the graft and patient survival. CVGs are effective in reconstruction of failed primary anastomosis or in patients with hypoplastic portal vein. High preoperative platelet counts, Budd-Chiari syndrome and postoperative HAT are considered as patients with high risk of ePVT. Anti-thrombotic prophylaxies should be considered in high-risk patients.

Ethics approval

Ethical approval was obtained for this study from the Inonu University Health Sciences Clinical Research Ethics Committee (Decision No: 2022/4001).

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