OUR EXPERIENCE OF LIVING DONOR LIVER TRANSPLANTATION IN COMPLETE PORTAL VEIN THROMBOSIS; TECHNIQUES AND RESULTS

Rajesh D., Subhash G.

Max center for liver and biliary sciences,
Max Superspeciality Hospital, Saket, Delhi, India.

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Rajesh D.,
https://orcid.org/0000-0002-0801-6205
Subhash G.,
https://orcid.org/:0000-0002-7782-2593

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Author for correspondence:
Rajesh D.,
Centre for Liver and Biliary Sciences, Max Superspeciality Hospital, Saket, 110017, New Delhi, India.
Email: int.query@maxhealthcare.com; +91 81 307 00436; +91 88604 44888

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Abstract

Background: Combined liver-intestine transplantation for complete portal vein thrombosis is an acceptable solution, most groups have not achieved good long term results.

Materials and methods: A retrospective analysis of database for adult patients who underwent living donor liver transplantation at our centre between 1st September 2006 and 31st December 2014 was carried out. Patients were divided into three groups: without and with complete portal vein thrombosis and transplant techniques and outcomes after transplant were analyzed. A total of 79 / 1,288 adult patients who underwent living donor liver transplantation. 11 out of 79 patients had complete portal vein thrombosis and the incidence of major complications was similar in both the groups.

Results: Portal vein thrombosis often accompanies liver cirrhosis and can affect as many as 25% of the patients. Results of transplantation in presence of portal vein thrombosis is inferior even after successful thrombectomy and it is not clear what is the best surgical option in cases of complete obliteration of portal vein lumen. There is paucity of data on living donor liver transplantation in patients with complete portal vein thrombosis. It is possible that careful surgical technique may allow liver transplant in patients of partial and complete portal vein thrombosis and avoid multi-visceral transplantation.

Conclusion: There was no survival difference between those with and without portal vein thrombosis (p = 0.569). Out of the 11 patients, 3 patients died post transplant, one from failure of obtaining adequate portal venous flow and 2 patients from small for size syndrome.

Introduction

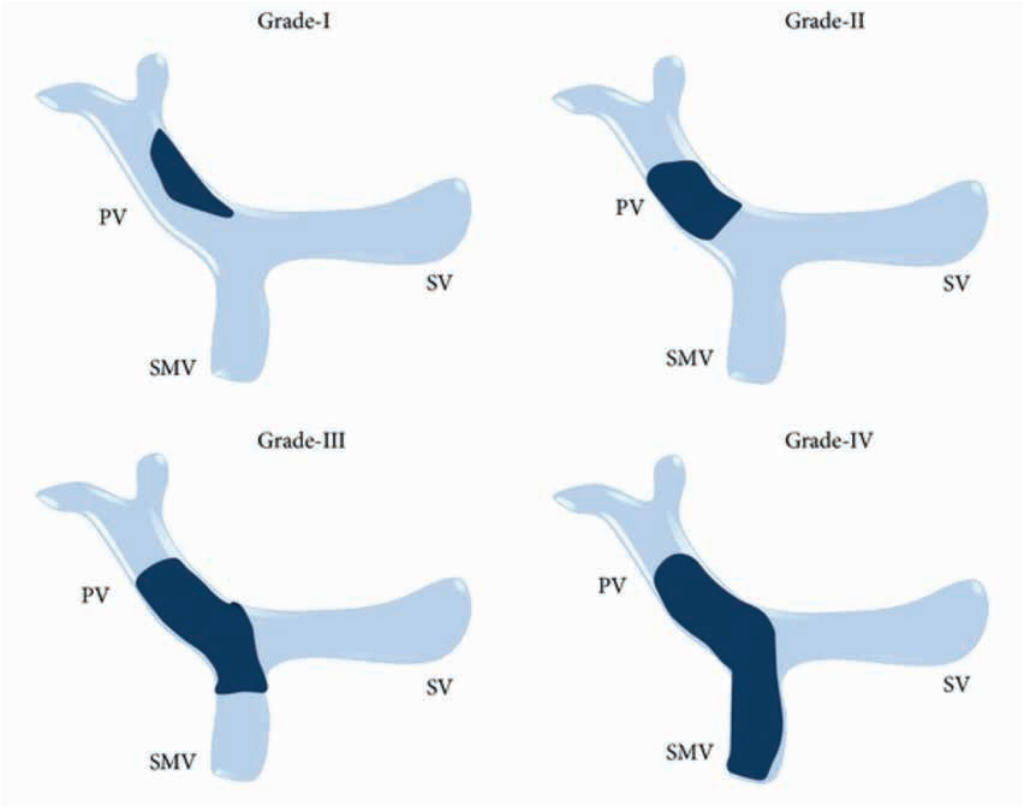
Non-neoplastic portal vein thrombosis (PVT) has a prevalence of 10-25% in cirrhotics and is currently not considered a contraindication for living donor liver transplantation (LDLT).[1,2] The complex interplay of portal hypertension, diminished portal flow, hypercoagulability and periportal lymphangitis contribute to the development of PVT. The relation of PVT to severity of liver disease (Child class and MELD score) is debated.[2-4] Among the numerous attempts to classify the types of PVT, Yerdel's classification system,[5] based on the extent and location of thrombus, is widely used. Complete PVT, though can be approached with various new surgical techniques, may lead-to increased perioperative morbidity and mortality. Even with complete PVT, large collaterals may exist to allow for graft revascularisation. With triple phase CT angiogram they can be picked up and provide inflow to the liver graft. The occluded PV results in a difficult portal dissection with bleeding leading to acidosis and coagulopathy during the anhepatic phase. This ultimately affects the postoperative outcome with rethrombosis of PV and graft failure. PV thrombectomy, the most commonly employed disobliteration technique has a success rate of 31-95%.[1,4] The major concerns for PV thrombectomy include a residual thin walled PV with questionable quality following surgical trauma.[6] Other techniques in the surgical armamentarium include cavoportal hemitransposition, renoportal bypass, PV resection with or without venous graft interposition, portal revascularization from

the superior mesenteric vein (SMV) and revascularization from a collateral vein, the selection depending on the extent of PVT and surgical expertise. In contrast to a cadaveric donor where vessel grafts are readily available, LDLT necessitates the use of autologous vessel graft, cryopreserved vessel graft or a prosthetic graft with various graft related limitations.[1] Moreover non-physiological PV reconstruction has been associated with comorbidities related to residual portal hypertension, altered cardiopulmonary dynamics and prolonged postoperative recovery.[1]

Portal vein thrombosis in patients with cirrhosis may be a result of low portal flow from increased resistance in the hepatic sinusoids from the cirrhotic process, from development of portosystemic shunting, after splenectomy and in a small number of cases from myelodysplastic syndromes or thrombotic states such as deficiency of protein C, S and antithrombin III. However it is very rare to find a contributing factor in a patient of advanced cirrhosis who has portal vein thrombosis.

Materials and methods

Classification: PVT was classified according to Yerdel's classification[5] i.e. grade 1 was <50 % occlusion of main portal vein, grade 2 were those with > 50% occlusion including total occlusion of main portal vein, grade 3 were those with complete occlusion of portal vein as well as proximal portion of the superior mesenteric vein and grade 4 were labeled as those with complete occlusion of portal as well as superior mesenteric veins.



Grade I

<50% of lumen, with no or minimal obstruction of the superior mesenteric vein

Grade II

Grade Iwith obstruction > 50%, including total obstruction with no or minimal obstruction of the superior mesenteric vein

Grade III

Complete obstruction of the portal vein and proximal superior mesenteric vein

Grade IV

Complete obstruction of the portal vein and superior mesenteric vein

Assessment: Assessment of portal vein thrombosis is often difficult in cirrhotic patients unless a CT triple phase liver angiogram is carried out. This may sometimes be difficult as the renal function in advanced cirrhosis is deranged. If the ascites is not massive, a plain MRI scan of the liver may help delineate

the portal vein anatomy. An ultrasound Doppler examination may often suggest thrombosis as the flow is very sluggish in advanced cirrhosis. Partial portal vein thrombosis is often underestimated by current imaging techniques as portal vein typically dilates in portal hypertension initially and then the flow decreases as cirrhosis progresses and wall may become thickened eccentrically.

Medical management Medical management has been proposed in recent years for cirrhotic with PVT particularly in the subgroup awaiting LT. However, the efficacy of medical management in treating PVT or preventing its progression has not been conclusively proven. The objective is to recanalize the portal vein or, if recanalization is not achievable, to prevent the extension of the thrombus so that a splanchnic vein can be used as the inflow vessel to restore physiological blood flow to the allograft.[4]

It has also been proposed recently that systematic anticoagulant therapy could help prevent PVT in advanced cirrhosis though results of such studies need to be validated.[4]

Ethical approval. The study was approved by the BioEthics Committee. All participants provided written informed consent prior to enrollment. The study procedures complied with the ethical standards of the Declaration of Helsinki of the World Medical Association (WMA).

Statistical analyses. Study subjects were followed from the time of transplant to death or the last available follow-up. Descriptive statistics were presented as means and standard deviations for continuous variables or as proportions for categorical variables. Statistical analyses were performed using SPSS Statistics v25.0. Between-group comparisons were assessed for numerical variables, and the Chi square test and Fisher's exact test were used for categorical variables. $P \leq 0.05$ was considered statistically significant. Analysis of main risk factors and the corresponding causal relationship was evaluated by calculating the odds ratio (OR).

Results

Data was collected retrospectively from a prospective maintained database for all adult patients (> 18 years) who underwent living donor liver transplant at ANG Centre for Liver and Biliary sciences, Indraprastha Apollo Hospital, New Delhi India, between 1st September 2006 and 31st December 2014. All patients underwent protocol CT angiogram of the Liver prior to their surgery and repeat imaging was done it the scan was older then 3 months at the time of their liver transplant. Color doppler was done one day prior to surgery for all patients. Those with portal vein tumour were turned down for transplant. No patient was refused transplant based on the extent of thrombosis. However risk counseling was done in these patients.

Surgical technique for patients All patients underwent living donor orthotropic liver transplantation with preservation of Inferior vena cava. In patients with patent portal vein and incomplete portal vein thrombosis (grade 1), standard hilar dissection technique was used and bile duct was dissected completely away from the underlying portal vein together with the right hepatic artery. A modified approach for hilar dissection was introduced following encouraging results in pediatric liver transplantation for patients with grades 2, 3 and 4 portal vein thrombosis. No attempt was made to dissect bile duct and surrounding tissues from the main trunk of the portal vein and the portal was divided beyond the division of right and left branches. This technique is of extreme help in allowing effective thrombectomy either with a suction catheter or with the index finger along the length portal vein without the risk of tearing the fragile vein. In patients of grade 3 and grade 4 PVT, eversion thromboendovenectomy with a sharp scissor was done and suction was used additionally till surgeon was able to feel the junction of splenic vein and superior mesenteric vein.

Eversion thrombectomy with end-to-end anastomosis was our preferred surgical technique. In patients with grade 4 portal vein thrombosis and failed thrombectomy, measures included inflow from a large collateral or renoportal anastomosisif a large shunt could be identified draining in to the left renal vein. Successful thrombectomy in our series was possible in most cases. Portocaval shunt was made soon after thrombectomy in all patients. The flow through the shunt was measured using doppler ultrasound

by placing the ultrasound probe on the head of the pancreas. The left renal vein or shunt itself was looped in all cases with a shunt diameter > 10 mm and when left renal vein was bigger in size then the portal vein. Ligation if required of left renal vein was preferred as it was technically easier to loop and could be done without mobilization of colon and thereby avoiding bleeding from retroperitoneal collaterals. However ligation of left renal vein was not done in patients with renal impairment.

Intra operative doppler of the grafted liver was done in all cases and ligation of shunt or left renal vein was done when the portal flow was < 100 ml / 100 gm of liver. Color doppler was done twice a day for 5 days and once a day for another 2 days and thereafter as when it was required. CT angiogram of the liver was performed in all cases of doubtful color doppler examination or clinical suspicion of compromised inflow to liver parenchyma. Use of antithrombolytic therapy was reserved for the cases developing rethrombosis. All patients underwent a protocol CT angiogram prior to their discharge from the hospital.

A total of 1288 adult cirrhotic patients underwent living donor liver transplant at our center between September 2006 to December 2014. Seventy-nine patients had PVT

Table 1. Pre operative characteristics and outcomes of patients with or without portal vein thrombosis

	PVT				
	Present (n=79)	**Absent (n=1209)**	**OR**	**95%CI**	**P value**
Age (years) (mean ± SD)	48.76 ± 9.7	47.98 ± 9.7	-	[2.9;1.4]	0.489
Sex (Male/female)	62 (78.5%) / 17 (21.5%)	1006 (83.2%) / 203 (16.8%)	0.736	[0.4;1.3]	0.281
Etiology					
HCV	33 (41.8%)	565 (46.7%)	0.817	[0.5;1.3]	0.392
HBV	13 (16.5%)	180 (14.9%)	1.126	[0.6;2.1]	0.701
Ethanol	11 (13.9%)	174 (14.4%)	0.962	[0.5;1.8]	0.908
Cryptogenic	10 (12.7%)	192 (15.9%)	0.768	[0.4;1.5]	0.447
NASH	6 (7.6%)	25 (2.1%)	3.893*	[1.5;9.8]	0.004*
Others	6 (7.6%)	73 (6.0%)	1.279	[0.5;3.0]	0.577
MELD score (mean ± SD)	17.97 ± 7.1	19.1 ± 5.9	-	[0.2;2.5]	0.104
Complications					
Sepsis	10 (12.7%)	205 (17.0%)	0.709	[0.4;1.4]	0.323
Renal dysfunction	2 (2.5%)	93 (7.7%)	0.312	[0.1;1.3]	0.107
Biliary complications	5 (6.3%)	111 (9.2%)	0.736	[0.3;1.9]	0.517
Cholestasis	5 (6.3%)	29 (2.4%)	2.749*	[1.0;7.3]	0.043*
Hemorrhage	2 (2.5%)	17 (1.4%)	4.565*	[1.6;12.7]	0.004*
PVT	2 (2.5%)	15 (1.2%)	5.169*	[1.8;14.6]	0.002*
Ascites	4 (5.1%)	28 (2.3%)	2.249	[0.8;6.6]	0.139
Mortality	10 (12.7%)	126 (10.4%)	1.246	[0.6;2.5]	0.532

PVT – Portal vein thrombosis; HCV - Hepatitis C virus; HBV - Hepatitis B virus; NASH - Non-alcoholic steatohepatitis; MELD - model for end stage liver disease
*OR: Odds ratio; OR > 1 means that the event is directly related and has a chance of occurring in the first group and P –value ⩽ 0.05 was considered statistically significant

Demographic data was similar for patients with PVT or without PVT. Age and gender distribution was similar in both the groups. Mean MELD of PVT group (17.97 ± 7.1) was again similar to those without PVT (19.1 ± 5.9) and failed to reach any statistical significance as suggested by a few reports (p = 0.085). Incidence of PVT was 5.8% for cirrhotic males while it was slightly higher (7.7%) for the female patients. Similarly incidence of PVT in HCV, HBV and ethanol was 5.5%, 6.7% and 5.9% respectively and failed to show any meaningful association with underlying disease.

Incidence of major complications was similar in both the groups. Post transplant cholestasis was higher in PVT group however due to small number of cases in group one it failed to reach any statistical significance. Post transplant PVT was experienced in two cases of pre op PVT and one of these patients could not be salvaged (renal failure and graft dysfunction). Fifteen cases in no PVT group developed post transplant PVT and 3 of these cases died due to causes directly attributed to inflow issues. Prolonged ascites following liver transplant was seen in 4 patients in PVT group and one of these patients required PV dilatation in post-operative period. There were 10 deaths in PVT group and 126 in non PVT however it failed to reach any statistical significance (p = 0.569).

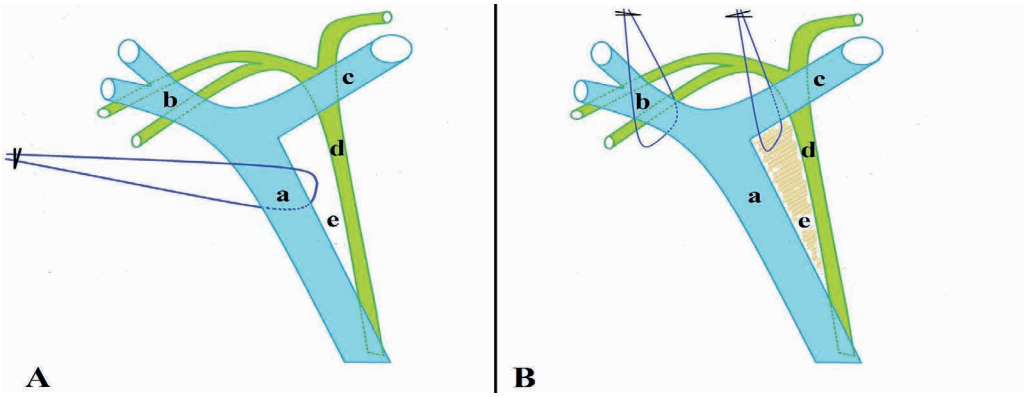


Figure 1.
Diagrammatic representation of hilar dissection technique:
A: Standard technique,
B: Modified approach

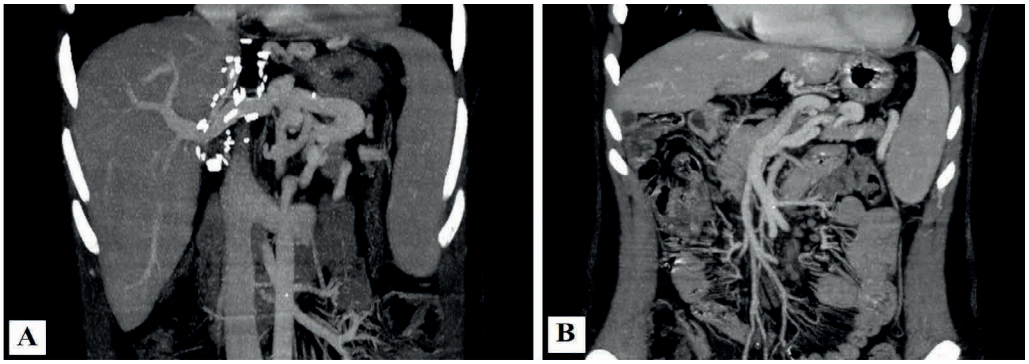


Figure 2.
Inflow from Coronary vein in a patient with complete PV thrombosis.

The patients with portal vein thrombosis were divided into 2 groups (Yerdel's grade 1 & 2 as group A and grade 3 and 4 as group B) to identify any co relation between pre operative characteristics and postoperative outcomes and results have been summarized in table 2

	Grade 1 and 2 (n=63)	Grade 3 and 4 (n=16)	OR	95%CI	Univariate P
Age (years) (mean ± SD)	50.62 ± 8.2	40.13 ± 14.8	-	[15.9;5.0]	0.0003*
Sex (male/ female)	47 (74.6%) / 16 (25.4%)	15 (93.8%) / 1 (6.2%)	0.196	[0.02;1.6]	0.129
MELD score (mean ± SD)	17.97 ± 7.5	18.0 ± 5.4	-	[3.9;4.0]	0.988

Table 2.
Comparison of patients with different grades of PVT

Major Shunts	32 (50.8%)	13 (81.3%)	0.238	[0.1;0.9]	0.003*
Shunt Ligation	9 (14.3%)	4 (25.0%)	0.50	[0.1;1.9]	0.308
Complications					
Sepsis	9 (14.3%)	1 (6.3%)	2.50*	[0.3;21.3]	0.402
Renal dysfunction	2 (3.2%)	0	1.34	[0.1;29.3]	0.852
Biliary complications	5 (7.9%)	0	3.10*	[0.2;59.1]	0.451
Cholestasis	4 (6.4%)	1 (6.3%)	1.03	[0.1'9.8]	0.988
Hemorrhage	2 (3.2%)	0	1.34	[0.1;29.3]	0.852
PVT	1 (1.6%)	1 (6.3%)	0.242	[0.01;4.1]	0.326
Ascites	2 (3.2%)	2 (12.5%)	0.229	[0.03;1.8]	0.158
Mortality	9 (14.3%)	1(6.3%)	2.5	[0.3;21.3]	0.402

PVT – Portal vein thrombosis; MELD - model for end stage liver disease
* OR: Odds ratio; OR > 1 means that the event is directly related and has a chance of occurring in the first group and P –value ⩽ 0.05 was considered statistically significant

Multivariate analysis of mean age values in groups 1 and 2 grades with 3 and 4 showed statistically significantly equal odds OR = 0.915, 95% CI [0.856 - 0.978], p = 0.009. Whereas the development of the main shunt is 2.8 times statistically not significantly higher in grades 1 and 2 than in grades 3 and 4, OR = 2.782, 95% CI [0.676 -11.452], p = 0.156.

Patients in group 2 were younger as compared to group 1 (p = 0.014) on univariate analysis and also have higher proportion of large shunts on preoperative imaging (p = 0.046) however gender distribution was similar and males cirrhotic were equally distributed amongst early (group 1) and advanced (group 2) PVT groups. On multivariate analysis age (p = 0.009) was the only significant variable and younger patients had more advanced PVT at the time of liver transplantation. Ligation of shunts was similar in those with early and those with more severe PVT (p = 0.449). Major morbidity and mortality of two groups were similar and didn't reach any statistical significance.

Discussion

PVT is a well recognised complication of liver cirrhosis and the prevalence of non-neoplastic PVT has been reported between 10-25 %.[1,2] This variation may be secondary to differences in diagnostic methods, geographical distribution of disease and reporting bias. Patients denied surgery due to PVT were probably not included in final analysis. Incidence of PVT is our series was 6.1 % which is similar to previous reported literature.[7]

PVT was found to be more common in male patients, autoimmune and cryptogenic cirrhosis in few series.[8,9] Similarly an incidence of 16 % was reported with alcoholic and HBV related cirrhosis.[10] Incidence of PVT in HBV and alcohol related cirrhosis was 6.7 and 5.9% respectively in our experince and was no greater then incidence for the rest of study cohart. Corelation has been found between age of the pateint, severity of the disease [5,9] in few series, however in our experince, advanced PVT was much more common in younger age group as comapred to eldery recipeints suggesting a more severe change in hemodynamics in this group of patients.

In the past, the presence of PVT was considered a contraindication to liver transplantion.[11]

Following few reports in the early 90s, PVT no longer remained a contraindication for this procedure.[12] However a cautious approach was suggested by a survey report in year 2002 where

presence of Yerdel gardes 3 and 4 PVTs were considered as relative or absolute contraindications for LDLT by many centres.[13] Considerable progress has been made in the subsequent years and recent literature suggest that the LDLT is no longer a contraindication for recipients with complete PVT.[6] Nonetheless LDLT in the presence of PVT remains a major undertaking and adds unique challenges to this complicated procedure. PV is extremely short and friable and attempt at thrombectomy may introduce a tear in the venous wall with a torrential life threatening hemorrhage. Thrombectomy in the retropancreatic portion may become extremely difficult and incomplete, leading to poor portal flow and a tendency to re-thrombosis following anastomosis.

A number of techniques have been described in the literature for patients with PVT with each having its own merits and demerits. Eversion thrombectomy of the PV, whenever feasible with an end to end anastomosis is considered as the most physiological approach and has been the first line technique in most reported series.[14,15] However the risk of hemorrhage during thrombectomy remains a major concern and can be life threatening in some situations. Our proposed technique of PV thrombectomy involving non separation of bile ductal tissues from PV wall makes this procedure safe and a non collapsing vein wall at the end of the procedure minimizing the risk of post operative thrombosis. Incomplete thrombosis however may be encountered and has to be addressed by a colour doppler before undertaking it for re-anastomosis. Only four cases of venous wall tear were faced by us using this technique and the rate of early re-thrombosis (2.5%) despite avoiding routine anti-thrombolytics in our experience has been quite low. Making a portocaval shunt soon after thrombectomy in our experience is mandatory and helps to restore the prograde flow across the vein. Similarly addressing a large portocaval shunt is equally important and can act as a double edged sword. A selective approach for portosystemic shunt ligation has been adopted by us. LRV or portosystemic shunt is looped prior to liver implantation and the ligation is offered only to those with low portal flow (< 100 ml / 100 gm of liver) on intraoperative colour doppler examination. Similar number of pateints required shunt ligation (in both early and more severe PVT, (p = 0.0449) in our series signifying the role of sound sugical tecnique while during endovenothrobectomy. A hyperdynamic splanchnic circulation is common and the routine ligation of shunt in our opinion can lead to hyperperfusion injury to the transplanted liver.

Use of large collateral (coronary vein or gastroepiploic vein) is a viable alternative and can be used when PV thrombectomy is not successful or incomplete.[5,16] Two cases of G-4 PVT required inflow from collateral in our series (one from coronary and another from gastroepiploic vein) due to incomplete thrombectomy and low portal flow. Renoportal bypass in the presence of large lienorenal shunt and jump graft from SMV have also been described as modes for liver inflow.[14] The technique of renoportal inflow was used in one of our patients and has been reported earlier.[17] However a large lienorenal shunt appears mandatory for good inflow across the renoportal anastomosis as well as for the effetive decompression of splanchnic circulation. Need for cryopreserved veins and prosthetic grafts for jump inflow from SMV makes it less practicaland more cumbersome in clinical practice in setting of LDLT.

Cavoportal transposition is another rescue method, however its non-physiological nature and poor long term survival makes it a less favouredoption.[7,15] Multivisceral transplant has recently been suggested for patients with diffuse PVT with a 5 year graft survival rates of 72%.[18] However such a strategy is not applicable for LDLT. Due to the morbidity and nature of the procedure, multivisceral transplant has been advocated as one of the last resort in these patients.

Several studies performed before 2000 have shown poor outcomes following liver transplantation in PVT albeit recent reports failed to demonstrate a significant difference in survival.[5,6] *Lendoire J. et al.*[19] noticed a significantly lower 1-year survival rate (59% vs. 80.5%) for PVT patients with a trend towards better 1- and 5-year survival rates for grade 1 PVT compared to higher grades.

Once the patients with PVT have survived the peri-transplant period, the outcome was noted to be identical to non-PVT patients. The current study however failed to address long term survival because a significant number of pateints travel from neighbouring nations to our center and long term follow up still remain a major hurdle. Nontheless we agree with the fact that long term survival of PVT patients remain identical to non PVT patients. As reported comparable 1- and 5-year survival rates (91% and 87% respectively) after LDLT for PVT and non-PVT patients. The same group couldn't demonstrate a difference in survival rate between partial and complete PVT.[6] Similarly, also reported comparable 1- and 3-year survival rates for partial and complete PVT.[1] However 82% patients in their series had PVT above the confluence of splenic vein and SMV. A recent audit of 174 cases showed significantly lower 1-, 5-, and 10-year patient survival rates and graft survival rates for patients who had non-physiological portal inflow as compared to physiological portal inflow or no PVT. The short to long term graft survival was not significantly different between patients with physiological portal inflow and no PVT.[7]

A recent meta-analysis did not show a statistically significant association of PVT with the in-hospital, 1-year or 5-year survival.[20] The improvement in the surgical techniques and peri-operative management might have contributed to increased in-hospital and 1-month survival rates in recent reports.

Limitations. This is a single-center study. A retrospective analysis was performed, allowing for a broad overview of the results; however, a further prospective multicenter study is necessary to obtain statistically significant results.

What's known? Potential risk factors for the development of complete portal vein thrombosis in living-donor liver transplantation are known.

What's new? This study evaluates our experience with varying degrees of portal vein thrombosis on patient outcome and potential complications.

Conclusions

Results of the current study support emrging evidnence of equally good survival in patients of PVT following liver transplant and survival of PVT group was simialr to those with a patent portal vein at the time of transplantation. Similarly surival of those with complete PVT appears identical to those with partial thrombosis. A sound surgical technique with incresing experince has helped us to overcome the burden imposed by PVT in liver cirrhosis pateints.

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Authors' contributions: S. G.: the study concept, design, supervision, and approval of the final manuscript version; R.D.: data collection, preparation, and primary verification, statistical analysis and drafted the manuscript sections on Materials and Methods and Results; R.D., S. G.: prepared the Introduction and Discussion sections. All authors reviewed and approved the final version of the manuscript.

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