

# Management of vascular complications in adult-to-adult living donor liver transplant recipients: a single-group experience with 1000 cases

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## Background

Liver transplantation is currently the method of choice for treatment of cases with irreversible severe liver dysfunction. In living donor liver transplantation (LDLT), vascular complications are more frequently encountered than in deceased donor transplantation. Satisfactory outcomes of liver transplantation are critically dependent on sufficient venous outflow and uncompromised inflow to the liver graft. The aim of this study was to discuss the complications of vascular reconstruction in our study cases and the different modalities of their management.

## Patients and methods

This is a retrospective study evaluating vascular complications in adult-to-adult living donor liver transplant recipients that occurred in Ain Shams University Specialized Hospital and Wadi El-Neel Hospital from October 2001 to December 2020 and their management.

## Results

The recipients comprised 819 males and 181 females. Pediatric cases were excluded from this study. The indications for liver transplantation were chronic hepatocellular liver diseases due to HCV infection in 48.8%, hepatocellular carcinoma in 33.9%, cryptogenic cirrhosis in 5.9%, fulminant hepatic failure in 0.3%, and other causes in 7.9%. Vascular complications were 9.5% (7.9% occurred during the first 3 months after transplantation and 1.6% occurred late after the first 3 months from transplantation). Hepatic artery complications were seen in 2.2%, portal vein complications were seen in 1.0%, hepatic vein complications were seen in 0.5%, whereas V5, V8, and the inferior right hepatic vein (Makuuchi) complications were seen in 5.8% of cases.

## Conclusion

Careful preoperative assessment of both the recipient and the donor with proper intraoperative vascular reconstruction techniques with microsurgical technique ultimately prevents vascular complications. Routine posttransplant Doppler assessment should be performed at least once a day for the first week postoperatively. Immediate surgical intervention is required for acute vascular complications, whereas late complications may be managed by means of interventional radiology in the form of balloon angioplasty and end-luminal stent to avoid late complications and mortality.

## Keywords:

liver transplantation, living donor, vascular complications

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## Introduction

Liver transplantation is currently considered worldwide a definite therapy for end-stage liver disease. Vascular complications in liver transplantation are one of the most serious complications that can affect graft viability; therefore, early recognition is critical for proper management. Insufficient liver blood supply can lead to early graft failure; therefore, intraoperative and early postoperative diagnosis is crucial for graft survival [1]. Clinical assessment assisted with laboratory investigations in the post-transplantation period can raise the suspicion of occurrence of vascular

complications, whereas definitive diagnosis of these complications can be achieved with radiological investigations. Doppler ultrasonography and computed tomography angiography are noninvasive useful techniques for surveillance; furthermore, conventional angiography can be used for both diagnostic and interventional purposes, and so, radiological modalities

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play a vital role in both diagnosis and treatment of vascular complications [2].

Although bleeding, stenosis, or thrombosis may occur at any of the sites of vascular anastomoses (hepatic veins, portal vein, and hepatic artery), hepatic artery thrombosis (HAT) and portal vein thrombosis (PVT) are the most common and also the most serious. HAT is reported to complicate 4–15% of orthotopic liver transplantations (OLTs) and is more common after pediatric liver transplantation [3].

PVT complicates 3–7% of OLTs and, similar to HAT, can have a lethal outcome for both the allograft and the patient. Factors related to posttransplant PVT include technical issues (redundancy), preexisting PVT requiring thromboendovenectomy intraoperatively, small portal vein size (5 mm), earlier splenectomy, and use of venous conduits for portal vein reconstruction [4].

Hepatic vein stenosis (HVS) is less common, with incidences less than 1%. It is more common in cases of retransplantation and in pediatric liver transplantation. HVS should always be suspected when we encounter unexplained increasing ascites or graft dysfunction. Percutaneous angioplasty is the method of choice for management of cases with HVS [5].

## Patients and methods

After taking approval of the ethical committee, written informed consent from each donor and each recipient, approval from the ethics and indication committees at our institution for each living donor liver transplantation (LDLT) procedure, and permission of the supreme committee of organ transplant, MOH, Egypt, this retrospective study was conducted on 1000 LDLT recipients. All of the recipients included in our study underwent LDLT during the period from October 24, 2001 to December 21, 2020 in Ain Shams University Specialized Hospital and Wadi El-Neel Hospital.

### Ethical considerations

All patients included in the study were meticulously assessed and informed about the operation and its risks, techniques, and postoperative course.

### Study procedures

All patients were subjected to the following:

#### Preoperative workup

- (1) Full clinical assessment.
- (2) Laboratory investigations, including CBC, coagulation profile, liver function tests, kidney

function tests, lipid profiles, diabetes profile, serum electrolytes, viral markers, and tumor markers, especially alpha-fetoprotein, and laboratory tests for Bilharzias, autoimmune diseases, and metabolic liver diseases.

- (3) Radiological investigations: tri-phasic pelviabdominal computed tomography with portography venography and arteriography and computed tomography chest with contrast for cases with hepatocellular carcinoma.
- (4) Endoscopy: upper gastrointestinal and colonoscopy.
- (5) Medical consultations: cardiological, chest, psychological, ENT, dental consultations, and gynecological consultation for female cases.
- (6) Calculation of MELD score and Child–Pugh classification.

#### Intraoperative workup

The following were assessed:

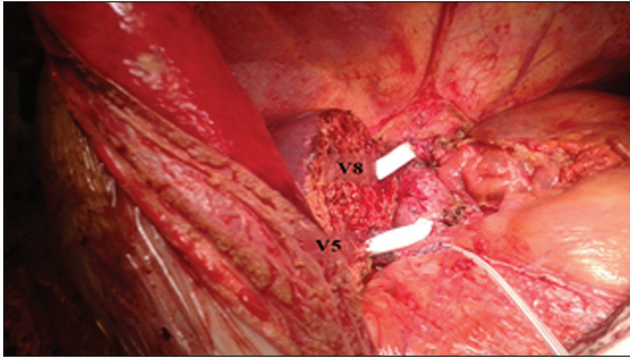
- (1) Operation time.
- (2) Operation cold and warm ischemic times of the graft.
- (3) Graft weighting for assessing graft for recipient weight ratio.
- (4) Number of veins that needed reconstruction on the back table.
- (5) Intraoperative duplex after vascular anastomosis to assess both inflow and outflow through the graft.

A J-shaped hockey stick incision was used to enter the abdomen followed by mobilization of the liver with the piggyback technique. Hilum dissection was performed with section of right and left branches of HA, then dissection and section of bile duct, so total hepatectomy is done with preservation of the RHV and common trunk of MHV and LHV.

Before starting vascular reconstruction of the graft in the recipient, it is assessed at the back table considering the weight, the right hepatic artery stump, the right portal vein stump, the right hepatic duct, the right hepatic vein stump, and number of veins to be anastomosed in the recipient according to diameter (>4 mm). If V5 and/or V8 were significant after the assessment, reconstruction was done using ePTFE synthetic grafts and anastomosis to IVC in case of V5 alone and MHV in case of V8 alone. In some cases if both V5 and V8 were present, anastomosis may be held through two separate grafts for each vein with an end-to-end anastomosis or through a single graft by end-to-side anastomosis for both veins (Figs 1 and 2).

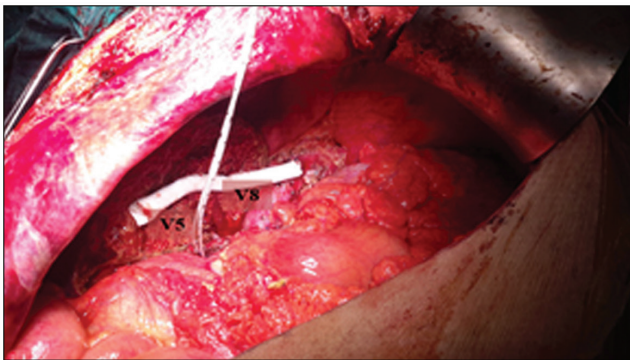
The ready graft for transplantation was brought out to the recipient, in cases with RT lobe graft, RHV

Figure 1



V5 anastomosis to IVC and V8 anastomosis to MHV stump with two separate grafts through an end-to-end anastomosis.

Figure 2



V5 and V8 anastomosis to MHV through a single graft by end-to-side anastomosis.

anastomosis to the RHV stump in the IVC was started with prolene 4/0, and the inferior right hepatic vein (Makuuchi) was anastomosed to the IVC directly using prolene 5/0 if preserved. After that, anastomosis of the portal vein was followed using prolene 6/0 sutures. Then, venous declamping was done and the graft was flushed with the portal blood. V5, if present, was anastomosed to IVC via synthetic graft, and V8 as well, if present, in the graft was anastomosed to MHV of the recipient via synthetic graft with prolene 5/0. In cases with left lobe graft, LHV was anastomosed to MHV, and left portal vein was anastomosed to the main portal vein using prolene 6/0 sutures.

Hepatic artery reconstruction was performed by end-to-end anastomosis after reconstructing of both hepatic and portal veins followed by the reperfusion of the graft using donor right HA to recipient right HA in case of right lobe graft and using donor left HA to recipient left HA in case of left lobe graft with interrupted prolene 8-0 sutures. In some cases with insufficient flow in the right HA or cases with intimal injury of the right HA, arterial reconstruction was performed using left HA or reversed splenic artery

was done. Intraoperative Doppler ultrasonography was performed for all cases after vascular anastomoses to check that there is a sufficient flow for survival of the graft. Then, reconstruction of the biliary tree was done using 6/0 PDS sutures with or without stent applied, and intraoperative cholangiography was done to assess the patency of the biliary tree. Finally, anatomical closure of the anterior abdominal wall was done after application of intra-abdominal drains in the determined sites (Morrison pouch, hepatic pedicle, and left subdiaphragmatic space).

#### Postoperative follow-up

All of the patients in our study were subjected to the following:

- (1) Early follow-up (during the first 3 months after transplantation):

Daily follow-up full laboratory investigations, including full liver profile and abdominal duplex ultrasonography to assess portal venous inflow, hepatic venous outflow, and hepatic artery inflow were done daily for the first week postoperatively, then every other day for the second week, then twice weekly for the third and fourth weeks, and after that, once weekly for 2 months.

- (2) Late follow-up :(after the first 3 months after transplantation):

Follow-up laboratory investigations and ultrasound were done every 2–4 weeks according to patients' demands. In cases with hepatocellular carcinoma, follow-up was done for tumor markers every 3 months and abdominal computed tomography every 6 months.

#### Informed consent

Informed consent was taken from patients in the research. All of the patients' data were kept confidential, and the patients were not mentioned by name in any published paper.

#### Statistical analysis

The data were collected, tabulated, and statistically analyzed. Description of quantitative variable was done using mean and SD and qualitative data as frequency.  $\chi^2$  test was used to compare the groups regarding qualitative variables. Student *t* test was used to compare groups regarding quantitative variables in parametric data. The results were considered significant with *P* value less than 0.05 and highly significant with *P* value less than 0.01. *P* value more than or equal to 0.05 was considered nonsignificant. Analysis of data was done using IBM SPSS software (Statistical Package for the Social Sciences version 21, Chicago, IL, USA).

## Results

The median age was 47 years (range, 17–68 years). The median body weight was 81.3 kg (range, 50–170 kg). The median BMI was 27.05 kg/m<sup>2</sup> (range, 17–44 kg/m<sup>2</sup>). The donor was related to the recipient in 60.9% of cases and not related in 39.1% of cases (Table 1).

Other causes include Budd–Chiari syndrome, fulminant hepatic failure, bilharzial cirrhosis, nonalcoholic steatohepatitis, hypercholesterolemia, Wilson disease, and alcoholic liver cirrhosis (Tables 2 and 3).

Left lobe liver transplantation was performed in 17 cases, and right lobe liver transplantation was performed in 983 cases.

The operation was done electively in 937 (93.7%) cases and as emergency in 63 (6.3%) cases. The median operative time was 10.83 h (range, 5.47–17.25 h). The median blood loss was 1750 ml (range, 120–19 000).

The median hospital stay for the patients was 23 days (range, 14–171 days).

In our study, RHV of the graft was anastomosed to RHV of the recipient alone in 438 (43.8%) cases, with

**Table 1 Baseline characteristics in the study group**

Baseline characteristics	Total (N=1000)
Sex [n (%)]	
Male	819 (81.9)
Female	181 (18.1)
Age (years)	
Range	17–68
Mean±SD	47.97 ± 7.89
Weight (kg)	
Range	50–120
Mean±SD	81.35 ± 10.08
BMI [wt/(ht) <sup>2</sup> ]	
Range	17–38
Mean±SD	27.05 ± 3.44
Related [n (%)]	
Yes	633 (63.3)
No	367 (36.7)

**Table 2 Diagnosis in the study group (N=1000)**

Diagnosis	n (%)
HCV	460 (46.0)
HCV and HCC	325 (32.5)
Cryptogenic cirrhosis	59 (5.9)
Autoimmune hepatitis	35 (3.5)
HBV and HCV	28 (2.8)
HBV and HCC	14 (1.4)
Others	79 (7.9)

HCC, hepatocellular carcinoma.

V5 to IVC in 213 (21.3%) cases, with V8 to MHV in 113 (11.3%) cases, with both of V5 and V8 in 68 (6.8%) cases, and Makuuchi to IVC in 151 (15.1%) cases (Table 4).

PVT was detected preoperatively in 89 (8.9%) cases and discovered accidentally intraoperatively in 14 cases. Eversion thrombectomy was done for all cases with PVT. RPV of the graft was anastomosed to MPV in 963 (96.3%) cases, whereas two branches of RPV were anastomosed to MPV in 20 (2.0%) cases. LPV was anastomosed to MPV in 17 cases with left lobe liver transplantation (Table 4).

For hepatic artery anastomosis, RHA of the graft was anastomosed to RHA of the recipient in 932 (93.2%) cases, RHA to LHA in 23 (2.3) cases, to reversed splenic artery in 26 (2.6%) cases, and to LT gastric artery in two (0.2%) cases. However, in cases with LT lobe liver transplantation (17 cases), LHA was anastomosed to common hepatic artery (Table 4).

**Table 3 Preoperative data in the study group (N=1000)**

Preoperative data	n (%)
Child class	
A	37 (3.4)
B	261 (24.2)
C	702 (70.2)
Child score	
Range	5–15
Mean±SD	10.06 ± 1.68
MELD score	
Range	6–38
Mean±SD	16.33 ± 4.27
PCR for HCV	
Positive	482 (48.2)
Negative	518 (51.8)

**Table 4 Vascular anastomosis in the study group (N=1000)**

Vascular anastomosis	n (%)
Hepatic veins anastomosis	
RHV to RHV alone	438 (43.8)
V5 to IVC	213 (21.3)
V8 to MHV	113 (11.3)
Both of V5 and V8	68 (6.8)
Makuuchi to IVC	151 (15.1)
Portal vein anastomosis	
RPV to MPV	963 (96.3)
2 branches of RPV to MPV	20 (2.0)
LPV to MPV	17 (1.7)
Hepatic artery anastomosis	
RHA to RHA	932 (93.2)
RHA to reversed splenic artery	26 (2.6)
RHA to Lt. gastric artery	2 (0.2)
RHA to LHA	23 (2.3)
LHA to common hepatic artery	17 (1.7)

Severe intraoperative bleeding was encountered in 6 cases for which packing and re-exploration were done.

Cardiac arrest occurred intraoperatively in four cases; successful CPR was done in two of the cases and death on the table occurred in two cases.

Vascular complications occurred in 9.5% of cases; HAT occurred in 17 (1.7%) cases, PVT occurred in seven (0.7%) patients, HVS occurred in five (0.5%) cases, and both HAT and PVT occurred in four (0.4%) cases.

Cases with HAT are divided into two groups: early HAT in 13 cases and late HAT in four cases. Cases with early HAT were diagnosed with Doppler ultrasonography and were managed by means of surgical intervention; nine cases underwent thrombectomy and reanastomosis of the hepatic artery, whereas four cases underwent revision of the arterial anastomosis using reversed splenic artery. HAT occurred again in two patients for which retransplantation was done and three patients died owing to graft failure. In the retransplant, we used a right lobe graft with a 10% steatosis and graft-to-recipient weight ratio of 0.84, with no variations in the hepatic veins, arteries, and biliary and portal anatomy. Cases with late HAT were diagnosed by Doppler ultrasonography and confirmed by computed tomography angiography; recanalization of the hepatic artery after administration of anticoagulant was done in eight cases, whereas one patient developed intrahepatic biliary abscess at segment VIII, for which resection of segment VIII was done (Fig. 3).

Hepatic artery stenosis occurred in four (0.4%) patients: three patients were successfully treated by means of interventional radiology in the form of percutaneous angioplasty using balloon dilatation with stent insertion, and one patient died owing to graft failure.

One patient developed bleeding from the site of anastomosis on day 1 postoperatively, for which

re-exploration was done and the bleeding was controlled by stitches.

Two patients underwent urgent retransplantation; in the two cases, we used left gastric artery for arterial reconstruction, and one patient died on the fourth postoperative day.

Recurrence of HAT occurred in five (0.5%) cases; three patients died, and two patients underwent urgent retransplantation but one patient died. The success rate of the management of HAT was 13/17 (76.4%) cases. Combined HAT and PVT, which occurred in four cases, resulted in graft failure and death in these cases.

PVT occurred in seven (0.7%) patients and portal vein stenosis (PVS) occurred in three (0.3%) cases. PVT and PVS were diagnosed by Doppler ultrasonography. PVT was treated by intraoperative eversion thrombectomy, whereas PVS was treated by interventional radiology in the form of percutaneous balloon dilatation and stenting of the portal vein. The success rate of management of portal vein complications was 7/10 (70%) cases.

Hepatic venous outflow obstruction owing to HVS occurred in five (0.5%) cases. Both PVS and HVS occurred in one (0.1%) case. Cases with hepatic venous outflow obstruction were managed by means of interventional radiology in the form of percutaneous angiography, balloon dilatation, and stenting, whereas one case was managed by surgical revision of the anastomosis at V5 owing to marked elevation of liver enzymes with severe congestion of the liver graft.

During postoperative follow-up, occlusion of V5, V8, and the inferior right hepatic vein (Makuuchi) was found in 5.8% of cases, and despite that, no significant compromise of the venous drainage of the graft was noted. Hepatic venous outflow obstruction was diagnosed by Doppler ultrasonography. Failure of management of hepatic vein complications occurred in

**Figure 3**



(a) Abdominal CT showing intrahepatic (graft) biliary abscess. (b) Intraoperative intrahepatic abscess before and (c) after resection of segment VIII [6]. CT, computed tomography.

one case that developed graft failure. The success rate of management of hepatic vein complications was 4/5 (80%) cases.

## Discussion

LDLT is a far more sophisticated procedure as compared with cadaveric liver transplantation. A detailed acknowledgment of the segmental anatomy of the liver, hepatic arterial system, hepatic venous system, portal venous system, and biliary tree assisted with the ability to identify the anatomical variations are mandatory to perform successful and safe LDLT with acceptable outcomes [7].

In our study, vascular complications occurred in 95/1000 (9.5%) cases; HAT with or without HAS was present in 17/1000 (1.7%), PVT in 7/1000 (0.7%) cases, PVS in 3/1000 (0.3%) cases, HVS in 5/1000 (0.5%) cases, both HAT and PVT in 4/1000 (0.4%) cases, and both PVS and HVS in 1/1000 (0.1%) case.

HAT with or without HAS represented 17/95 (17.89%) cases of vascular complications. Failure of treatment occurred in five cases; three cases developed graft failure and died, whereas urgent retransplantation was performed to two patients. ABO-incompatibility was a major risk factor for HAT. HAT is considered the most common and the most lethal vascular complication that can endanger liver graft viability and affect its survival. It occurs in 12.0% of adult recipients [8].

Early HAT is usually related to technical problems during anastomosis, whereas late HAT is usually related to a hypercoagulable state, overtransfusion of platelets and fresh frozen plasma intraoperatively, severe degrees of graft rejection, and bile leakage [9].

Hepatic artery complications after liver transplant are related to many factors. Some of these factors are the various anatomical variations in the hepatic arterial tree, the size of hepatic artery, damage to vessels during anastomosis as in prolonged clamping of hepatic artery, kinking of a long hepatic artery, and hematoma of artery wall due to vigorous flushing after clamping intraoperatively. Moreover, the quality of recipient vessels and the disproportion between donor and recipient arterial vessels should be carefully considered and managed intraoperative properly [10].

Early HAT was usually identified during routine postoperative Doppler ultrasound investigation before the development of complications. Surgical intervention for all of our cases with early HAT was done either in

the form of thrombectomy and reanastomosis of the hepatic artery or revision of arterial anastomosis using reversed splenic artery.

One of the important arteries that can be used in hepatic artery reconstruction is the right gastroepiploic artery owing to many factors. It can be delivered easily to the liver graft after being separated from the greater curvature of the stomach, and if there is a size mismatch, it can be properly enlarged after splenic artery ligation. If this artery cannot be used for retransplantation, there are many other ways such as interposition artery graft either from the inferior mesenteric artery or sigmoidal artery, or an arterial deceased donor graft [11].

In our study, we used the left gastric artery in retransplantation owing to lack of cadaveric vascular conduits and also the mismatch between the size of recipient gastroepiploic and donor right hepatic arteries even after splenic artery ligation.

HAT can lead to serious and fatal complications. Some of these complications are graft failure, biliary leakage or strictures, recurrent sepsis and septic shock, and ultimately death of the patients [12]. Therefore, early diagnosis with proper management is essential to avoid the need for retransplantation, which may be required for survival of the patient. In our study, two of our cases with HAT underwent retransplantation and one of the patients died.

The biliary tree depends only on the blood supply from the hepatic artery, and so if HAT occurred, it can lead to serious biliary complications such as biliary ischemia, necrosis, stricture, sepsis, and in late stages, graft failure and septic shock, which may lead to death of the patient [13]. In our study, one patient complained of recurrent attack of biliary cholangitis and liver necrosis in spite of development of collateral with anticoagulant treatment.

In our study, hepatic arterial stenosis (HAS) was detected in four (3.3%) of the cases with vascular complications. It was usually localized at the site of anastomosis. Management of the cases by the means of interventional radiology in the form percutaneous transluminal angioplasty, balloon dilatation, and stent insertion was done.

In most cases, HAS is caused by technical failure, which leads to damage of the vascular intima and then necrosis and scar formation. HAS can decrease the blood flow, which leads to arterial thrombosis. Therefore, HAS alone can be in late and complicated stages after failure of interventional radiology methods an indication of re-

exploration with reanastomosis, arterial reconstruction, or retransplantation [14]. If angioplasty was conducted within the early postoperative period, it can lead to rupture of the suture line or intimal dissection that can lead to catastrophic results [15].

PVT was detected preoperatively in 89 cases and discovered accidentally intraoperatively in 14 cases. Eversion thrombectomy was done for all cases with PVT. In the previous era of transplantation, PVT was an absolute contraindication for liver transplant. However, in 1985, two liver transplantations with preoperative PVT were reported with acceptable postoperative outcome and survival of patients [16]. Since then, advancements in LT have pushed the surgeons to step forward and utilize multiple techniques including eversion thrombectomy, extensive thromboendovenectomy up to splenomesenteric confluence, venous interposition graft, renoportal anastomosis, and cavoportal hemitransposition for dealing with PVT and restoring portal venous flow [17], taking into consideration that there are many other factors that can affect the success of management of PVT, such as the characteristics of portal venous thrombus (whether acute or chronic), degree (partial or complete), and also the extent of extension to the splanchnic venous system [18].

PVT grading preoperatively is crucial as it could affect posttransplant outcomes, as patients with complete PVT have much more less 1-year survival rates and postoperative outcome in comparison with those patients with partial PVT. Advanced preoperative PVT as in grade IV is still considered a critical risk factor of intraoperative blood loss and has much less postoperative outcome, with high rate of morbidity and mortality, even though it became no longer a contraindication for liver transplantation [19]. In our study, grade II PVT was the most common type with 68.7%, and four cases with grade IV PVT underwent liver transplantation, with survival of two cases.

In our study, PVT and PVS represented 10/95 (10.52%) cases of vascular complications, denoting 1.0% of all of our patients. The incidence of PVT ranges from 2 to 26% in various centers as in Cherqui *et al.* [20], and Davidson *et al.* [21]. Kim JD *et al.*, [26] reported a success rate of 93.3% regarding the management of patients with grades I and II PVT posttransplant.

Patients with PVT were treated by intraoperative eversion thrombectomy, whereas PVS was treated by interventional radiology in the form of percutaneous angioplasty, balloon dilatation, and stenting of the portal vein. The results of management of cases of PVT

and PVS were successful in 7/10 cases, with a success rate of 70%.

Hepatic venous outflow obstruction due to HVS occurred in 5/1000 (0.5%) cases. Both PVS and HVS occurred in one (0.1%) case. Post-LDLT hepatic venous outflow occlusion was reported to be ranged from 3.9 to 16.6% in cases with OLT [22]. In our study, cases with hepatic venous outflow obstruction were managed by means of interventional radiology in the form of percutaneous balloon dilatation and stenting, whereas one case was managed by surgical revision of the anastomosis at V5 owing to markedly elevated liver enzymes and severe congestion of the liver graft which would possibly lead to graft failure if it was left unmanaged properly. Early (during the first 30 days posttransplant) post-LDLT hepatic outflow obstruction, if it is neglected, can result in a congested graft, which will lead to compromise of liver functions, acute graft failure, and ultimately death of the liver transplant recipient; thus, early hepatic venous outflow obstruction is by far considered as a surgical emergency, and surgical correction is necessary for graft survival [23]. On the contrary, delayed hepatic venous outflow obstruction usually leads to gradual decline of liver function, ascites, with mild to moderate elevation of liver enzymes; furthermore, surgical correction is usually not feasible owing to severe perianastomotic fibrosis, rendering dissection more hazardous. It opens the door for interventional radiological modalities in the form of percutaneous angiography, balloon dilatation, and stenting to take the upper hand in management of these cases [24].

During postoperative follow-up, occlusion of V5, V8, and the inferior right hepatic vein (Makuuchi) was found in 5.8% of cases, and despite that, no significant compromise of the venous drainage of the graft was noted. Cases with hepatic venous outflow obstruction were diagnosed by Doppler ultrasonography. The success rate of management of hepatic vein complications was 4/5 (80%) cases.

Hepatic venous outflow obstruction can be caused by either stenosis, thrombosis, or presence of both stenosis and thrombosis at the anastomotic site or sites if there are multiple anastomoses. One of the crucial factors for prevention of anastomotic stricture is the design and shape of the orifice of the hepatic vein in both the graft and the recipient. Regarding HVS, several potential mechanisms could be encountered. Intraoperative improper technique is by far the most likely cause such as tight anastomoses causing purse-string phenomenon, stitches including the back wall of the vein, or additional hemostatic stitches. Moreover,

the structural stenosis of the hepatic vein in the postoperative period owing to enlargement of the graft during the process of regeneration can also be one of the factor [25].

In conclusion, thorough preoperative imaging combined with intraoperative Doppler assessment with early and regular postoperative follow-up imaging and careful surgical dissection allows identification and performance of satisfactorily vascular anastomosis, which significantly reduces vascular complications. Although arterial and venous problems are not common among the population of liver transplant recipients, they are of critical importance as they can gravely affect the liver graft with increased mortality and morbidity of LDLT recipients. Therefore, early surveillance for these complications with proper diagnosis and proper management is the key for getting the best possible outcome for graft salvage and patient survival after LDLT.

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#### Conflicts of interest

There are no conflicts of interest.

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