

Arterial Complications after Living Donor Liver Transplantation

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Abstract: Background: Liver transplantation (LT) has become the treatment of choice for pediatric and adult patients with end-stage liver disease (ESLD). **Objectives:** The aim of this study is to know the incidence of arterial complications after living donor liver transplantation and management of these complications and their effect on graft failure and mortality. **Patients and Methods:** We retrospectively reviewed the early postoperative arterial complications in patients who underwent LDLTx in 2017 and 2018, All patients were admitted, living donor liver transplantations were performed and followed up at the Ain shams university specialized hospital in Cairo, Egypt. Doppler ultrasound and detailed biochemical monitoring of graft function were performed once daily for 7 to 10 days after transplantation. And when indicated after that. **Results:** the incidence of arterial complications was 7 cases from total 85 cases studied retrospectively (8.2 %). hepatic artery thrombosis in 5 cases (5.9 %) and hepatic artery stenosis in 2 cases (2.4 %) and there is no documented cases complicated by pseudo aneurysm or splenic artery steel syndrome. There is graft failure of 8 cases (10.3%) in non complicated group and 2 cases (28.5%) in complicated group, And no graft failure in 70 cases (89.7%) in non complicated group and 5 cases (71.5%) in complicated group. There is mortality of 12 (15.4%) in non complicated group and 4 (57.1%) in complicated group, And no mortality in 66 (84.6%) in non complicated group and 3 (42.9%) in complicated group. **Conclusion:** Arterial complications are still a major source of morbidity and mortality after OLT. Hepatic artery complications after OLT include hepatic artery thrombosis (HAT), hepatic artery stenosis, hepatic artery pseudoaneurysm (HAP) and splenic artery steel syndrome (SASS). HAS and HAT are the most common hepatic arterial complications, with high rates of morbidity and mortality.

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1. Introduction

Liver transplantation (LT) has become the treatment of choice for pediatric and adult patients with end-stage liver disease (ESLD) (1).

The indications for LDLT included hepatocellular diseases such as hepatitis B or C virus-associated cirrhosis or alcoholic liver cirrhosis, hepatocellular carcinoma, progressive intrahepatic cholestatic diseases, including primary biliary cirrhosis and primary sclerosing cholangitis, retransplantation due to graft loss, cryptogenic cirrhosis, fulminant hepatic failure, autoimmune hepatitis, and metabolic liver disease (2).

Vascular problems such as thrombosis and stenosis of the hepatic artery, portal vein, and hepatic vein are among the most serious complications reported after liver transplantation and are more frequently seen among recipients of living donor transplantations (LDLT). complications can lead to increased morbidity, graft loss, and patient death (3).

Early diagnosis and appropriate management of these complications result in longer survival. Close surveillance of all vascular anastomoses using Duplex ultrasonography facilitates early detection and treatment of these complications before irreversible

graft failure. Treatment options usually include surgical revascularization, percutaneous thrombolysis, percutaneous angioplasty, retransplantation, or less commonly, a conservative approach (4).

Hepatic arterial reconstruction in liver transplantation (LT) is a crucial, yet delicate procedure. Particularly in living donor liver transplantation (LDLT), Hepatic arterial complications are often associated with high graft loss and mortality rates if they occur in the early phase after LT and are left untreated. Hepatic arterial complications include hepatic artery thrombosis (HAT), hepatic arterial stenosis, bleeding from anastomotic sites, splenic artery steel syndrome and rupture of hepatic artery aneurysms (5).

Various factors contributing to development of vascular thrombosis have been proposed: ABO incompatibility, multiple anastomoses, prolonged cold ischemic time, acute rejection. and previous vascular thrombosis (6).

Hepatic artery thrombosis represent more than 50% of all arterial complications. It is the most frequent and severe vascular complication following LDLT ((7).

It is the first cause of primary non-function of the liver transplant, which can lead to allotransplant loss and patient death in the early postoperative period. HAT is associated with a high incidence of liver transplant failure (more than 50%) and carries a mortality of more than 50% in the absence of revascularization or retransplantation. In recent years, early revascularization by means of endovascular catheter-based intervention has been a viable option for graft salvage before considering retransplantation (8).

Hepatic artery stenosis following LDLT is defined as a narrowing of the transverse diameter of the HA, more or less extended, resulting in graft ischemia mainly revealed by elevated liver function tests. Significant HAS is usually defined as a narrowing of the transverse diameter > 50% on angiogram associated with clinical suspicion and a RI < 0.5 (defined by peak systolic flow/peak diastolic flow/peak systolic flow) and a peak systolic velocity > 400 cm/s detected by DUS (9).

Hepatic artery pseudoaneurysm is defined as a dilated hepatic artery, which occurs after iatrogenic injury in most cases, causing blood to leak and pool outside the artery wall into surrounding tissue, with a persistent communication between the HA and the resultant adjacent cavity (10).

Hepatic artery rupture is defined as a severe hemorrhage from the trunk or from a main branch of the HA. It is a very serious complication that results in the disruption of the arterial blood supply of the transplant. This is a very exceptional but a dramatic complication after LDLT which carries very high incidence of liver transplant loss and high mortality rate. In most cases, this condition complicates a pseudoaneurysm of the HA, leading to major bleeding that requires emergency operation (10).

Aim of the work

The aim of this study is to know the incidence of arterial complications after living donor liver transplantation and management of these complications and their effect on graft failure and mortality.

2. Patients and Methods

Patients

We retrospectively reviewed the early postoperative arterial complications in patients who underwent LDLTx in 2017 and 2018, All patients were admitted, living donor liver transplantations were performed and followed up at the Ain shams university specialized hospital in Cairo, Egypt.

Doppler ultrasound and detailed biochemical monitoring of graft function were performed once daily for 7 to 10 days after transplantation. And when indicated after that.

- **Inclusions Criteria:**

Patients underwent LDLTx

- **Exclusions Criteria:**

- ESLD with retransplantation
- ESLD with blood hypercoagulopathy

Methods

Donor work up

1- Selection policy

Donors should retain a remnant liver volume >35% of the original liver volume [based on preoperative computed tomography (CT) volume measurements], and the donation should be >40% of the recipient standard liver volume (SLV). All the grafts provided by the donors led to estimated graft to-recipient weight ratios (GRWRs) that exceeded (0.8).

Donor Absolute Contraindications

- BMI > 28 (donors with BMI = 30 is accepted if there is no fatty infiltration)
- Age over 50 or below 18
- Previous upper abdominal surgery
- Previous cholecystectomy

2- Evaluation process

The Evaluation Protocol starts with the simplest and most cost effective blood work (i.e. blood group, virology and serum biochemistries). Once a potential donor is determined to be compatible with the intended recipient, he or she will receive the "Informed Consent for Organ Donation Surgery as well as other written education materials. In the presence of negative virology, serum biochemistry values within normal limits and a compatible blood group. At this second step, if, for example, the potential donor has medical history consistent with some alcohol intake, the liver biopsy is taken first. On the other hand, if there is a question concerning his liver volume, the CT or MRI volumetry is done first. If the potential donor has issues concerning his cardiac or pulmonary status, for example heavy smoker of greater than 50 years of age, the cardio-pulmonary evaluation will be performed.

Recipient Preparation

Evaluation processes

A. Laboratory Testing

New patients have an extensive profile of laboratory blood tests performed:

- Complete Blood Count
- Lipid Profile
- Virology: CMV IgG, VDRL, and HIV
- Coagulation Studies
- Chemistry Profile
- Tumor Markers
- Iron Studies.
- Blood Bank: Two Blood Type and Screen tests at different times.
- Viral Hepatitis Screen: HAV Total Ab, HCV Ab, HBsAg, HbsAb, HBcore Ab.

B. Diagnostic Studies

1. Routine: all potential transplant candidates will undergo the following tests except in cases of medical urgency.

- Triple phase CT of the abdomen or MRI.
- Chest X-ray.
- ECG.
- Resting echocardiogram.
- Stress echocardiogram.
- Pulmonary function tests.
- Upper endoscopy.

2. Disease Specific: testing performed on candidates with specific diagnoses, noted below.

1. Suspected HCC - Chest CT (without contrast) and bone scan.
2. PBC & PSC - Bone Densitometry.

Arterial anastomosis:

- Hepatic artery anastomosis is performed under magnification loope minimum x 4.5.
- We do interrupted sutures in arterial anastomosis by using prolene 8/0 and must avoid kinking.
- The last three stitches in anastomosis should be untight until flushing the artery with saline heparin then we can do tightening of these stitches and close the anastomosis.
- Duplex ultrasound must be performed to evaluate blood flow in the hepatic artery.

Postoperative follow up (all patients):

All patients were followed up on a predefined protocol basis. Following discharge from the hospital, patients had a weekly follow up for 4 weeks, monthly follow up for the first six months and thereafter 3-monthly follow up for first year, then every 6 months. At each follow up the patient had;

- 1- A complete physical examination.
- 2- Laboratory tests including;
 - a) Complete blood counts.
 - b) Hepatic and renal function tests.
 - c) Fasting blood glucose test.
 - d) Cyclosporine or tacrolimus level 12 hours after the last drug dose was measured.
- Vascular flow in the graft or interposition vein patency was checked by Doppler ultrasound every day until the postoperative day 14 and once a

week thereafter until hospital discharge. Enhanced CT was performed 1 and 3 months after LDLT.

3. Results

Donor age was in average of (18 – 55) with a mean of 29.22 ± 8.46 . and mean recipient age is 47.22 ± 12.01 with range 10 – 65 and donor gender female was 20 (23.5%) and male 65 (76.5%) while in recipient gender female was 23 (27.1%) and male was 62 (72.9%). as shown in table 5.

The mean donor age was 29.41 ± 8.70 with range 18 – 55 in non complicated cases and 27.14 ± 5.05 with range 18 – 31 in complicated cases (P value = 0.500), mean recipient age was 46.82 ± 11.74 with range 10 – 65 in non complicated cases and 51.71 ± 15.02 with range 19 – 64 in complicated cases (P value = 0.305), donor gender was male in 58 (74.4%) and female in 20 (25.6%) in non complicated cases and male 7 (100.0%) and no female in complicated cases (P value = 0.126), recipient gender was male in 57 (73.1%) and female 21 (26.9%) in non complicated cases, male was 5 (71.4%) and female 2 (28.6%) in complicated cases (P value 0.925). as in table 8.

Mean MELD score was 15.67 ± 4.93 in non complicated cases and 14.43 ± 3.78 in complicated cases (P value = 0.520), PVT was 19 (24.4%) in non complicated cases and 2 (28.6%) in complicated cases (P value = 0.804). as in table 9.

In anastomosis RHA to RHA was 48 (61.5%) in non complicated cases and 5 (71.4%) in complicated cases, RHA to LHA was 16 (20.5%) in non complicated cases and 1 (14.3%) in complicated cases, RHA to CHA Was 10 (12.8%) in non complicated cases and 0 (0.0%) in complicated cases, LGA TO RHA was 0 (0.0%) In non complicated cases and 1 (14.3%) in complicated cases, RHA TO SA Was 1 (1.3%) in non complicated cases and 0 (0.0%) in complicated cases (p value = 0.027 significant. as in table 10.

There is mortality of 12 (15.4%) in non complicated group and 4 (57.1%) in complicated group, And no mortality in 66 (84.6%) in non complicated group and 3 (42.9%) in complicated group. as in table 12.

Table (1): Characteristics of donor age, gender, recipient age and gender

	Donor		Recipient	
Age	Mean \pm SD	29.22 ± 8.46	Mean \pm SD	47.22 ± 12.01
	Range	18 – 55	Range	10 – 65
Gender	Female	20 (23.5%)	Female	23 (27.1%)
	Male	65 (76.5%)	Male	62 (72.9%)

Table (2): Comparison between arterial complicated cases versus non complicated as regard different variables:

		No arterial complications No. = 78	Arterial complications No. = 7	Test value	P-value	Sig.
Donor age	Mean \pm SD	29.41 \pm 8.70	27.14 \pm 5.05	0.677•	0.500	NS
	Range	18 – 55	18 – 31			
Recipient age	Mean \pm SD	46.82 \pm 11.74	51.71 \pm 15.02	-1.033•	0.305	NS
	Range	10 – 65	19 – 64			
Donor gender	Female	20 (25.6%)	0 (0.0%)	2.347*	0.126	NS
	Male	58 (74.4%)	7 (100.0%)			
Recipient gender	Female	21 (26.9%)	2 (28.6%)	0.009*	0.925	NS
	Male	57 (73.1%)	5 (71.4%)			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

*: Chi-square test; •: Independent t-test

Table (3): Comparison between arterial complicated cases versus non complicated as regard different variables:

		No arterial complications No. = 78	Arterial complications No. = 7	Test value	P-value	Sig.
Hepatopathy	HCV	58 (74.4%)	5 (71.4%)	2.475*	0.963	NS
	Cryptogenic	5 (6.4%)	1 (14.3%)			
	PSC	3 (3.8%)	0 (0.0%)			
	AIH	7 (8.9%)	1 (14.3%)			
	HBV	1 (1.3%)	0 (0.0%)			
	HCV+HBV	2 (2.6%)	0 (0.0%)			
	Budd chiaris	1 (1.3%)	0 (0.0%)			
	Caroli disease	1 (1.3%)	0 (0.0%)			
HCC	No	51 (65.4%)	2 (28.6%)	3.708*	0.054	NS
	Yes	27 (34.6%)	5 (71.4%)			
Child-Pugh grade	A	9 (11.5%)	0 (0.0%)	4.55*	0.103	NS
	B	25 (32.1%)	5 (71.4%)			
	C	44 (56.4%)	2 (28.6%)			
MELD score	Mean \pm SD	15.67 \pm 4.93	14.43 \pm 3.78	0.646•	0.520	NS
	Range	4 – 35	9 – 20			
PVT	No	59 (75.6%)	5 (71.4%)	0.061*	0.804	NS
	Yes	19 (24.4%)	2 (28.6%)			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

*: Chi-square test; •: Independent t-test

Table (1): Comparison between arterial complicated cases versus non complicated as regard different variables:

		No arterial complications	Arterial complications	Test value	P-value	Sig.
		No. = 78	No. = 7			
Operation time (hour)	Mean \pm SD Range	8.62 \pm 1.86 5 – 15	9.14 \pm 1.77 8 – 13	-0.711•	0.479	NS
Cold ischemia time (min.)	Median (IQR) Range	50 (30 – 70) 20 – 150	60 (45 – 60) 40 – 160	-0.753 \neq	0.452	NS
Warm ischemia time (min.)	Median (IQR) Range	40 (30 – 60) 20 – 100	60 (45 – 75) 45 – 190	-2.678 \neq	0.007	HS
Anastomosis	RHA to RHA	48 (61.5%)	5 (71.4%)	12.625*	0.027	S
	RHA to LHA	16 (20.5%)	1 (14.3%)			
	RHA to CHA	10 (12.8%)	0 (0.0%)			
	LHA TO LHA	3 (3.8%)	0 (0.0%)			
	LGA TO RHA	0 (0.0%)	1 (14.3%)			
	RHA TO SA	1 (1.3%)	0 (0.0%)			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

*: Chi-square test; •: Independent t-test; \neq : Mann-Whitney test

Table (2): Comparison between mortality in arterial complicated group and non complicated group

Mortality	No arterial complications	Arterial complications	Test value*	P-value	Sig.
	No. = 78	No. = 7			
No	66 (84.6%)	3 (42.9%)	7.33	0.007	HS
Yes	12 (15.4%)	4 (57.1%)			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

*: Chi-square test

4. Discussion

Because the hepatic artery is the only route by which oxygenated blood is supplied to the transplanted liver, obstruction of the hepatic artery results in parenchymal and biliary ischemia, early diagnosis and prompt treatment of vascular complications result in longer graft and patient survival (11).

Hepatic artery complications after OLT include hepatic artery thrombosis, hepatic artery stenosis, hepatic artery pseudoaneurysm (HAP) and splenic artery steel syndrome. The early complications of hepatic artery were usually caused by technical problems (12).

Frongillo et al showed 421 liver transplant recipients who underwent serial ultrasound (US) color Doppler evaluations of the hepatic arteries after surgery. saw 48 hepatic arterial complications after liver transplantation (13 thrombosis, 29 stenosis, 2 kinking, 2 pseudo-aneurysm, and 2 pseudo-aneurysm

rupture). All subjects underwent US color Doppler examination periodically after surgery (11).

In our study, we reported seven cases of arterial complication from 85 cases underwent LDLTx in 2017 and 2018 in Ain shams university specialized hospital. Five cases were diagnosed by hepatic artery thrombosis and two cases diagnosed as hepatic artery stenosis. no pseudoaneurysm nor splenic artery steel syndrome.

In our study, There is mortality of 12 (15.4%) in non complicated group and 4 (57.1%) in complicated group, And no mortality in 66 (84.6%) in non complicated group and 3 (42.9%) in complicated group.

Scarinci et al., found that hepatic artery thrombosis (HAT) represents a devastating complication after liver transplantation, occurring in 1.6–9.2% of adult recipients and remains one of the major causes of graft failure and recipients mortality (13).

In our thesis, early hepatic artery thrombosis, occurred in five cases (5.8 %) of 85 adult grafts which is regarded as an acceptable incidence compared to other centres. The technique of microsurgical hepatic artery reconstruction contributed greatly to the reduction of incidence of HAT and hepatic stenosis.

Doppler ultrasound is the modality of choice for evaluating post liver transplant patients for vascular complications. Awareness of the normal postoperative Doppler findings and timely identification of various vascular complications is essential for improving outcome of liver transplantation (14).

In our study, five cases with early hepatic artery thrombosis was diagnosed, characterized by elevated hepatic enzymes, cholestasis, in the absence of acute rejection and drug toxicity. On Doppler ultrasound, HAT was diagnosed by the absence of flow in the hepatic artery; CT angiography was performed to confirm the diagnosis and revealed hepatic artery thrombosis.

In our study, Hepatic artery reconstruction was performed by loupe magnification (5.5×) in all cases using prolene 8/0.

Nonsurgical factors contributing to HAT include pro-coagulant states such as Janus kinase 2, anticardiolipin antibodies, a factor V Leiden deficiency, and a high hematocrit during the early postoperative course. a cytomegalovirus-negative recipient, a long cold ischemia time, a large liver graft (graft-to-recipient body weight ratio > 3%-4%), small-for-size liver syndrome (graft-to-recipient body weight ratio < 0.8%), and ABO incompatibility (15).

In our study in anastomosis RHA to RHA was 48 (61.5%) in non complicated cases and 5 (71.4%) in complicated cases, RHA to LHA was 16 (20.5%) in non complicated cases and 1 (14.3%) in complicated cases, RHA to CHA Was 10 (12.8%) in non complicated cases and 0 (0.0%) in complicated cases, LGA TO RHA was 0 (0.0%). In non complicated cases and 1 (14.3%) in complicated cases, RHA TO SA Was 1 (1.3%) in non complicated cases and 0 (0.0%) in complicated cases.

There are 3 treatment modalities for HAT: revascularization, retransplantation, and observation. The choice of any of these treatments, however, depends on the time of diagnosis. Retransplantation is the treatment of choice for most groups, offering the best results; however, this possibility is strongly conditioned by the donor shortage (16).

Urgent thrombectomy and revascularization is currently considered the treatment of choice in cases of early diagnosis of HAT. **Bekker et al.** looked at outcomes after various interventions. Surgical revascularization was reported for 257 of 510 cases from 47 published studies; for 163 of 315 cases, there was clear reporting of the type of intervention and the

clinical outcome. Revascularization was attempted in 75% of the adults and 54% of the children with an overall success rate of 56%. A correlation was noted between the early occurrence of HAT and a successful outcome after revascularization. Daily or frequent ultrasound examinations were associated with successful outcomes (66% versus 45%). Children were more likely than adults to have a successful outcome after early revascularization (61% of adults and 92% of children). Re-transplantation was performed for 30% of the attempted revascularization cases (17).

Recently, HAT has been successfully managed with total endovascular management including IAT, PTA, and stenting. Sixty-nine cases have been reported in 16 studies as both rescue and definitive therapy. Sixty-three patients (91%) underwent IAT following deceased donor LT and only six patients have been reported following LDLT. Successful IAT was achieved in 47 cases (68%). The initial success rate of IAT in this study (partial or total recanalization) was 81.8% (9/11 cases) and definite endovascular treatment rate of 54.5% (6/11 cases) (8).

Thrombolysis with restoration of flow without resolving underlying anatomical defects, including kinking or anastomotic stricture can lead to re-thrombosis, and often require PTA or stent placement. The inability to resolve underlying anatomical defects is a predictor of failed definitive endoluminal success (18).

In our study, one case with hepatic artery thrombosis had anticoagulant therapy with low molecular weight heparin, treatment was employed with success with no need for further intervention or retransplantation with follow-up from 3 to 42 months, the reasons for selecting therapeutic management were the presence of partial thrombus. The other four cases was treated by re exploration and urgent surgical thrombectomy or re transplantation of the hepatic artery if thrombectomy failed or rethrombosis occur.

Hepatic artery stenosis (HAS) is a relatively infrequent vascular complication after liver transplantation (LT) in adult patients. The true incidence of HAS, however, is poorly defined because many patients are asymptomatic (3).

Previous studies have suggested that early surgical revision of HAS may reduce the development of biliary necrosis. More recently, endovascular management of arterial stenosis disease has emerged as a less invasive alternative to surgical intervention (19).

There were 662 LT recipients, and HAS was identified in 54 (8.2%); the mean age was 50 years. Twenty-nine of the 54 patients with HAS underwent DSA, and 23 of these received endovascular treatment. Treatments included PTA for 15 patients

and PTA plus a stent for 8 patients. Postrevision hepatic artery patency was confirmed by Doppler ultrasound in all patients (20).

HAS is a relatively infrequent condition after adult LT with a low risk of progression to HAT. In this series, an intervention for HAS was associated with decreased chances of the development of biliary strictures. Endovascular therapy should be considered for patients with deranged LFTs/graft dysfunction or ischemic biliary strictures in an attempt to reduce further graft damage. In patients with late onset HAS (6 months) and asymptomatic patients, endovascular treatment is not warranted (20).

In our study, early hepatic artery STENOSIS, occurred in TWO case (2,4 %) of 85 adult grafts which is regarded as an acceptable incidence compared to other centres.

DUS findings suggestive of HAS included resistive indices (RIs) <0.5 and a systolic acceleration time >0.08 s. MDCTA was performed when HAS was suspected on DUS or for other non-HAS-related clinical reasons. DSA was performed when endovascular therapy was considered likely to be necessary (21).

Hepatic artery pseudoaneurysm is a rare complication after orthotopic liver transplantation with a reported incidence of 0.5e2.0%.^{1,2} This usually occurs within the first few months following a transplantation and are commonly reported to be associated with massive bleeding and localized infection (22).

We report the case of three patients who developed hepatic artery pseudoaneurysms after living donor liver transplantation. Two patients presented with massive duodenal bleeding secondary to erosion of the hepatic artery into the bile duct, and one patient presented with intra-abdominal bleeding. These patients were managed by catheter-based minimal invasive endovascular procedures including coil embolization and stent grafting. All the patients were treated successfully with uneventful recovery (23).

In our study, no cases were diagnosed by hepatic artery pseudoaneurysm

Splenic steal syndrome is a controversial diagnosis of hepatic artery hypoperfusion and may represent an underrecognized cause of graft ischemia in the United States. Splenic steal syndrome presents nonspecifically as graft dysfunction and, if overlooked, may lead to graft failure. Its incidence in the literature ranges widely from 0.6 to 10.1% in liver transplant recipients with some institutions performing prophylactic and/or posttransplant treatment procedures in up to 22 to 26% of their transplant recipients (24).

Patients with SASS may present with elevated liver enzyme levels, cholestasis and hepatic arterial

thrombosis, possibly, biliary complication and graft dysfunction in some severe cases (25).

Many institutions reported that SASS should be treated by embolization with a coil. The patient with SASS usually has a coarse splenic artery, the diameter of which is more than 5 mm. The fast splenic arterial flow can often push the coil into the branch of the splenic artery, which could induce local ischemic necrosis of the spleen, infection and septicemia (26).

In our study, we reported no cases (0%) of arterial steal syndrome via the splenic artery.

Conclusion

Arterial complications are still a major source of morbidity and mortality after OLT. Arterial reconstruction is a frequent therapeutic option after the ligation of different collaterals until, finally, the celiac trunk remains the only arterial vascular supply to the transplanted liver.

Hepatic artery complications after OLT include hepatic artery thrombosis (HAT), hepatic artery stenosis, hepatic artery pseudoaneurysm (HAP) and splenic artery steal syndrome (SASS).

HAT represent more than 50% of all arterial complications. It is the most frequent and severe vascular complication following OLT. It is the first cause of primary non-function of the liver transplant, which can lead to allotransplant loss and patient death in the early postoperative period. Urgent thrombectomy and revascularization is currently considered the treatment of choice in cases of early diagnosis of HAT.

Hepatic artery stenosis following OLT is defined as a narrowing of the transverse diameter of the HA, more or less extended, resulting in graft ischemia mainly revealed by elevated liver function tests. Significant HAS is usually defined as a narrowing of the transverse diameter $> 50\%$ on angiogram associated with clinical suspicion and a RI < 0.5 (defined by peak systolic flow/peak diastolic flow/peak systolic flow) and a peak systolic velocity > 400 cm/s detected by DUS.

HAT and HAS are the most common hepatic arterial complications, with high rates of morbidity and mortality.

References

1. Karakayali H, Sevmis S, Boyvat F, et al. Diagnosis and treatment of late-onset portal vein stenosis after pediatric living-donor liver transplantation, *Transpl. Proc.* 2011; 43: 601e604.
3. Kaido T, Ogawa K, Fujimoto Y, Ogura Y, Hata K, Ito T, Tomiyama K, Yagi S, Mori A, Uemoto S. Impact of sarcopenia on survival in patients undergoing living donor liver transplantation.

- American Journal of Transplantation. 2013; 13(6):1549-56.
4. Duffy JP, Hong JC, Farmer DG et al. "Vascular complications of orthotopic liver transplantation: experience in more than 4,200 patients," Journal of the American College of Surgeons, 2009; 208(5):896-903.
 5. Bonnet S, Sauvanet A, Bruno O, Sommacale D, Francoz C, Dondero F, Durand F, Belghiti J. Long-term survival after portal vein arterialization for portal vein thrombosis in orthotopic liver transplantation. Gastroenterol Clin Biol 2010; 34: 23-28.
 6. Iida T, Kaido T, Yagi S, Hori T, Uchida Y, Jobara K, Tanaka H, Sakamoto S, Kasahara M, Ogawa K, Ogura Y, Mori A, Uemoto S. Hepatic arterial complications in adult living donor liver transplant recipients: a single-center experience of 673 cases. Clin Transplant 2014; 28: 1025-1030.
 7. Khalaf H. Vascular complications after deceased and living donor liver transplantation: a single-center experience, Transpl. Proc. 2011; 42: 865e870.
 8. Hejazi-Kenari SK, Zimmerman A, Eslami M, Saidi FR. Current state of art management for vascular complications after liver transplantation. Middle East J Dig Dis 2014; 6: 121-130.
 9. Singhal A, Stokes K, Sebastian A, et al. Endovascular treatment of hepatic artery thrombosis following liver transplantation. Transpl Int 2010; 23: 245.
 10. Sabri SS, Saad WE, Schmitt TM, Turba UC, Kumer SC, Par AW, Matsumoto AH, Angle JF. Endovascular therapy for hepatic artery stenosis and thrombosis following liver transplantation. Vasc Endovascular Surg 2011; 45: 447-452.
 11. Boleslawski E, Bourse AF, Truant S, et al. Hepatic artery ligation for the arterial rupture following liver transplantation: a reasonable option. American Journal of Transplantation, 2013; 13(4):1055-1062.
 12. Frongillo F, Grossi U, Lirosi MC, Nure E, Sganga G, Avoli AW, Inchingolo R, Di Stasi C, Rinaldi P, Agnes S. Incidence management, and results of hepatic artery stenosis after live transplantation in the era of donor to recipient match. Transplant Proc 2013; 45: 2722-2725.
 13. Turrion VS, Alvira LG, Jimenez M, Lucena JL, Ardaiz J. Incidence and results of arterial complications in liver transplantation: experience in a series of 400 transplants. Transplant Proc, 2002; 34:292-293.
 14. Scarinci A, Sainz-Barriga M, Berrevoet F, et al. Early arterial revascularization after hepatic artery thrombosis may avoid graft loss and improve outcomes in adult liver transplantation. Transplant Proc 2010; 42: 4403.
 15. Bhargava P, Dakshina MG, Chandana GL, Mark DL, Rupan S, Jessica GZ. Postoperative Doppler evaluation of liver transplants. Indian Journal of Radiology and Imaging; 2014; 24(4):360-366.
 16. Heaton Institute of Liver Studies, King's College Hospital, London, United Kingdom Hepatic Artery Thrombosis: Conservative Management or Retransplantation? Liver Transplantation, 2013; 19(11): S14-S16.
 17. Pareja E, Cortes M, Navarro R, Sanjuan F, López R, and Mir J. Vascular Complications after Orthotopic Liver Transplantation: Hepatic Artery Thrombosis Transplantation Proceedings, 2010; 42: 2970-2972.
 18. Bekker J, Ploem S, de Jong KP. Early hepatic artery thrombosis after liver transplantation: a systematic review of the incidence, outcome and risk factors. Am J Transplant, 2009; 9:746-757.
 19. Lopez BR, Schlieter M, Hallscheidt PJ, et al. Successful arterial thrombolysis and percutaneous transluminal angioplasty for early hepatic artery thrombosis after split transplantation in a 4-month-old baby. Pediatr Transpl 2008; 12: 606.
 20. Rostambeigi N, Hunter D, Duval S, et al. Stent placement versus angioplasty for hepatic artery stenosis after liver transplant: a meta-analysis of case series. Eur Radiol. 2013; 23(5):1323-34.
 21. Pulitano C, Joseph D, Sandroussi C, Deborah V, Simone IS, Nicholas AS, Geoffrey WM, and Michael C. Australian National Liver Transplant Unit, Royal Prince Alfred Hospital, Sydney, Australia; and 2Centenary Research Institute, University of Sydney, Sydney, Australia.
 22. Park K, Kim TE, Park KW. Analysis of potential cost-savings after introduction of drug-eluting balloon angioplasty for in-stent restenosis or small vessel disease. Korean Circ J. 2011; 41:705-11.
 23. Leelaudomlipi S, Sugawara Y, Kaneko J, et al. Volumetric analysis of liver segments in 155 living donors, Liver Transpl., 2002; 8(7):612-4.
 24. Thorat A, Long-Bin J, Horng-Ren Y, Chun-Chieh Yeh, Shih-Chao H, Te-Hung C, and Kin-Shing P Organ Transplantation Center, China Medical University Hospital, Departments of 2Surgery and 3Anaesthesiology, China Medical University Hospital, Taichung, Taiwan Ann Hepatobiliary Pancreat Surg 2017; 21:205-211.
 25. Grieser C, Denecke T, Steffen IG. et al. Multidetector computed tomography for preoperative assessment of hepatic vasculature and prediction of splenic artery steal syndrome

- in patients with liver cirrhosis before transplantation. *Eur Radiol* 2010; 20(1):108–117.
26. Ishii E, Shimizu A, Kuwahara N, Kanzaki G, Higo S, Kajimoto Y, Arai T, Nagasaka S, Masuda Y, Fukuda Y. Hepatic artery reconstruction prevents ischemic graft injury, inhibits graft rejection, and mediates long-term graft acceptance in rat liver transplantation. *Transplant Proc* 2013; 45: 1748-1753.
 27. Sevmis S, Boyvat F, Aytekin C, Gorur SK, Karakayali H, Moray G, Haberal M. Arterial steal syndrome after orthotopic liver transplantation. *Transplant Proc* 2006; 38: 3651-3655.

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