

Microsurgical reconstruction of hepatic artery in A-A LDLT: 124 consecutive cases without HAT

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Abstract

AIM: To retrospectively investigate microsurgical hepatic artery (HA) reconstruction and management of hepatic thrombosis in adult-to-adult living donor liver transplantation (A-A LDLT).

METHODS: From January 2001 to September 2009, 182 recipients with end-stage liver disease underwent A-A LDLT. Ten of these patients received dual grafts. The 157 men and 25 women had an age range of 18 to 68 years (mean age, 42 years). Microsurgical techniques and running sutures with back-wall first techniques were performed in all arterial reconstructions under surgical loupes (3.5 ×) by a group of vascular surgeons. Intimal dissections were resolved by interposition of the great saphenous vein (GSV) between the donor right hepatic artery (RHA) and recipient common HA (3 cases) or abdominal aorta (AA) (2 cases),

by interposition of cryopreserved iliac vessels between the donor RHA and recipient AA (2 cases).

RESULTS: In the 58 incipient patients in this series, hepatic arterial thrombosis (HAT) was encountered in 4 patients, and was not observed in 124 consecutive cases (total 192 grafts, major incidence, 2.08%). All cases of HAT were suspected by routine color Doppler ultrasonographic examination and confirmed by contrast-enhanced ultrasound and hepatic angiography. Of these cases of HAT, two occurred on the 1st and 7th d, respectively, following A-A LDLT, and were immediately revascularized with GSV between the graft and recipient AA. HAT in one patient occurred on the 46th postoperative day with no symptoms, and the remaining case of HAT occurred on the 3rd d following A-A LDLT, and was cured by thrombolytic therapy combined with an anticoagulant but died of multiorgan failure on the 36th d after A-A LDLT. No deaths were related to HAT.

CONCLUSION: Applying microsurgical techniques and selecting an appropriate anastomotic artery for HA reconstruction are crucial in reducing the high risk of HAT during A-A LDLT.

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Key words: Adult-to-adult living donor liver transplantation; Hepatic arterial thrombosis; Microsurgical reconstruction

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INTRODUCTION

In the face of a constant shortage of cadaveric livers and the increasing size of the transplantation waiting list, living donor liver transplantation (LDLT) has become an accepted therapy for many patients with end-stage liver disease. However, despite improvements in LDLT, vascular complications related to the small diameter of the vessels in the partial liver graft and complex arterial reconstruction remain an important cause of morbidity and mortality^[1-3]. Hepatic artery thrombosis (HAT), which is the most common vascular complication after LDLT, can result in graft loss associated with septic hepatic infarction and bile duct ischemia^[4,5]. The incidence of HAT after LDLT varies widely, with a reported frequency of 4%-25%^[6-10].

Retransplantation used to be an option for the treatment of HAT^[11]. However, retransplantation is limited by both organ availability and the patient's condition. Urgent revascularization with thrombectomy or revision of anastomosis with thrombectomy has been successful in some patients with an early diagnosis^[8,9]. Color Doppler ultrasonography^[12] and contrast-enhanced ultrasound (CEUS)^[13] have provided an accurate noninvasive method for the detection of HAT before nonreversible ischemic damage of the graft occurs.

The application of microsurgical techniques in arterial reconstruction has helped overcome the high risk of HAT following liver transplantation, and the effectiveness of this method has been demonstrated in numerous reports^[14-17]. Because of the short donor hepatic artery (HA) stump which is small in diameter and vessel size discrepancy between the graft and recipient hepatic arteries, reconstruction of hepatic arteries is a challenge to surgeons in LDLT.

To outline our experience in hepatic reconstruction and the management of HAT after adult-to-adult living donor liver transplantation (A-A LDLT), the clinical data of patients with end-stage liver disease who underwent A-A LDLT at our center, were retrospectively reviewed.

MATERIALS AND METHODS

Recipients

From January 2001 to September 2009, we performed 182 consecutive A-A LDLTs in adult recipients with a mean age of 42 years (157 men and 25 women, aged 18 to 69 years) suffering from end-stage liver disease. Informed consent from both donor and recipient and the approval of the Ethics Committee of West China Hospital of Sichuan University were obtained. Of these cases, 10 recipients underwent dual graft liver transplantation.

Table 1 Details of recipients and diagnostic indications (*n* = 182)

	<i>n</i> (%)
Female	25 (13.74)
Male	157 (86.26)
Age (yr)	
18-60	173 (95.05)
60-69	9 (4.95)
Diagnostic indications	
Liver cirrhosis	74 (40.66)
Hepatitis B	52 (28.57)
Hepatitis C	5 (2.75)
Cholestasis	5 (2.75)
Alcoholic	4 (2.20)
Others	8 (4.39)
Hepatic carcinoma	77 (42.31)
FIF	10 (5.49)
Acute-on-chronic hepatic failure	9 (4.94)
Budd-Chiari ^[19]	4 (2.20)
DI IBS	4 (2.20)
Post-trauma hepatic failure	1 (0.55)
Hepatic echinococcosis	2 (1.10)
Polycystic liver	1 (0.55)
MELD	
1-13	86 (47.25)
14-24	63 (34.62)
≥ 25	33 (18.13)

FIF: Fulminant liver failure; DI IBS: Diffuse ischemic intrahepatic biliary stenosis; MELD: Model for end-stage liver disease.

In addition to dual graft A-A LDLT, 170 recipients received right grafts (right lobe without MHV, 160; right lobe with MHV, 10), 2 recipients received left grafts (extended left lateral segment, 1; left lobe without MHV, 1). The pre-transplant condition of the recipients was evaluated by the modified model for end-stage liver disease scores which has been documented to better predict the prognosis of patients^[18]. The details of recipients and the diagnostic indications for A-A LDLT are summarized in Table 1. All these patients underwent emergency A-A LDLT.

Donors

There were 190 donors, including 110 men and 80 women, with an age range of 19-65 years (mean age, 35 years). Details of the dual donors are described in Table 2. The donors and recipients were blood group identical in 151 cases and compatible in 31 cases. All donors in this group voluntarily donated part of their liver following informed consent.

Preoperative evaluation of donors and recipients

Physical examination and retrospective analysis of both donors' and recipients' medical records were performed before surgery. Hepatitis, syphilis, HIV, Epstein-Barr virus, cytomegalovirus, and tuberculosis infection constituted ineligibility as a potential donor. We did not use routine HA angiography to study the tracks and variations of the HA, but used computed tomographic arteriography (CTA) instead. The volume of the total

Table 2 Details of dual donors

No.	Donor 1			Donor 2		
	Gender	Age (yr)	Graft	Gender	Age (yr)	Graft
1	F	34	LL	F	31	LL
2	F	56	RL	M	27	Cadaveric LL
3	F	35	RL	M	55	LLS
4	F	29	RL	M	29	Cadaveric LL
5	M	42	RL	F	29	LL
6	M	58	LL	F	34	RL
7 ¹	M	45	RL	F	26	RL
8	F	34	RL	M	35	LL
9	F	20	RL	F	39	LLS
10 ²	F	28	RL	F	28	RL

^{1,2}These donors donated their right lobes, respectively, the others donated right lobes without the middle hepatic vein. LL: Left lobe; RL: Right lobe; LLS: Left lateral segment.

liver and right lobe were evaluated and calculated with 3-dimensional computed tomography. Graft mass to recipient body weight ratio (GRWR) of 0.8%^[20] or graft volume to recipient standard liver volume ratio of 40%^[21] and donor remnant liver volume of 35%^[22] were required for recipient and donor safety assurance.

Donor surgery and back table

The detailed surgical technique was as previously reported^[23]. The partial graft liver was harvested without vascular flow occlusion and without graft manipulation to maintain graft viability. The HA and portal vein (PV) were individually exposed and carefully divided, during hilar dissection. In right grafts, the right hepatic artery (RHA) was identified and isolated to the right side of the hepatic duct. In left grafts, the proper hepatic artery (PHA) was isolated up to the bifurcation of the left hepatic artery (LHA) and the RHA. We preserved the length of the arteries as much as possible and cut the RHA or LHA with sharp scissors or a microvascular appliance to avoid injury to the arterial intima and intimal dissection.

All grafts were placed into a container filled with 4°C University of Wisconsin (UW) solution, then removed to the back table. On the back table, we perfused the grafts from the PV with UW solution, rinsed the biliary tracts and flushed arterial ducts with heparin solution. All procedures mentioned below were performed by vascular surgeons. Plasty of the hepatic vein and PV was performed to make a single orifice for each. We anastomosed the great saphenous vein (GSV) from the recipient or cryopreserved vessels to the crassitude tributaries of the MHV (diameter, > 5 mm). We inspected the graft arteries to detect whether intimal injuries and dissection existed. When the graft arterial stump was shorter than 1 cm, we anastomosed the inverse GSV from the recipient to the graft arterial stump to prolong the stump and interposed the GSV between the graft artery and recipient artery. The graft HA including the origins of multiple tiny arteries was reshaped to achieve a single orifice of adequate diameter at the back table.

Recipient surgery

The procedure was performed following the routine procedures of our hospital^[24,25]. All recipient hepatic arteries were isolated toward their insertion sites into the hepatic parenchyma. After removal of the recipient liver, the stump of the recipient HA was meticulously preserved for as long as possible. The implanted graft was reperfused after reconstructing both the hepatic and PVs. Thereafter, one group of vascular surgeons performed all the microvascular reconstructions of the HA. Hepatic arterial reconstruction was performed using microvascular techniques with (3.5 ×) surgical loupes, after adoption of systemic anticoagulation (heparin, 62.5 U/kg, intravenous, 5 min before anastomosis). Low dose heparin did not cause any remarkable adverse effects in our study. We used running 8-0 or 9-0 monofilament polypropylene (Prolene; Ethicon Inc.) sutures for the HA anastomosis depending on the arterial diameter. During running sutures, the assistant vascular surgeon persistently and delicately stretched the stitch to ensure intimal eversion, and the vascular surgeon could see the arterial lumen after each stitch. According to the amount of inflow, stump location and matching of diameters, the most appropriate inflow artery was selected. In all cases, end-to-end vessel anastomosis was carried out between the recipient and graft HA, and the back wall-first microsurgical anastomosis technique which avoided vessel twisting during suturing was applied. In the case of a hepatic graft with multiple stumps, we tried to reconstruct all of the stumps. The dominant HA was reconstructed first. If there was sufficient back bleeding, the other arterial stumps were ligated. If there was only weak back bleeding, we always reconstructed the stumps. The patency of the arterial anastomosis was evaluated by intraoperative Doppler ultrasonography.

In dual graft patients, the RHA of the graft was anastomosed to the RHA of the recipient to reconstruct the RHA. Finally, the living or cadaveric left lateral segment was orthotopically implanted to the left lobe position of the recipient, followed sequentially by end-to-end anastomosis. The primary candidates of the recipient HAs for reconstruction were the RHA, LHA, and PHA. There were 192 anastomoses in all A-A LDLTs. Details of the recipient arteries are described in Table 3. The caliber difference between the graft artery and the recipient artery was resolved by cutting the smaller artery obliquely. Because of recipient HAs intimal dissection, these arteries were not suitable for HA reconstructions, and interposition bypass using GSV was performed between the donor RHA and recipient common hepatic artery (CHA) in 3 cases. Bypass was performed between the donor RHA and recipient abdominal aorta (AA) using GSV in 2 patients and cryopreserved cadaveric iliac vessels in 2 cases. The gastroduodenal artery was not ligated to increase the blood flow through the anastomosis. Administration of alprostadil (20 µg) to maintain artery patency was used in all cases after the completion of hepatic arterial reconstruction.

Table 3 Details of hepatic arteries (*n* = 192)

	<i>n</i> (%)
RHA	62 (32.29)
LHA	20 (10.42)
PHA	90 (46.88)
CHA	14 (7.29)
Aberrant RHA ¹	2 (1.04)
AA	4 (2.08)

¹Aberrant right hepatic artery arising from superior mesenteric artery. Diameter of graft: mean (range), 1.91 (1.5-2.5) mm; diameter of recipient: mean (range), 2.12 (1.5-3.5) mm. RHA: Right hepatic artery; LHA: Left hepatic artery; PHA: Proper hepatic artery; CHA: Common hepatic artery; AA: Abdominal aorta.

Postoperative management and follow-up

All patients received immunosuppressive therapy including cyclosporine or tacrolimus. All patients underwent Doppler ultrasonography every 12 h during the first postoperative week and daily during the second postoperative week to confirm HA patency. The diagnosis of HAT after A-A LDLT was based on clinical presentation, color Doppler ultrasonography findings, and HA arteriography. If elevated hepatic enzymes, cholestasis, bile leakage, or high fever in the absence of acute rejection were detected, color Doppler ultrasonography, CTA, or conventional HA angiography was performed to establish the diagnosis in turn. If hepatic arterial inflow was not observed by color Doppler ultrasonography, CUES examination was performed in these cases^[12].

RESULTS

Donor outcome

There were no donor deaths, and the hospitalization period ranged from 7 to 30 d (mean 10 d). Only 4 donors had the following complications: portal venous thrombosis in 1 case who underwent second thrombectomy and leakage was mended with a patch of GSV; transient chyle leakage in 1 case, which healed after symptomatic treatment; subphrenic effusion in 1 case which was cured by surgical drainage; and pleural effusion, healed after repeated thoracic cavity puncture.

Recipient outcome

In this series, HAT occurred in 4 cases of 192 grafts after A-A LDLT (2.08%). All thromboses occurred in the 58 incipient patients (63 grafts) with an incidence of 6.34%. Thereafter, HAT did not occur in 124 consecutive cases. HAT was suspected in 14 patients without arterial inflow by routine color Doppler ultrasonographic examination and confirmed in 4 patients by CEUS and HA angiography. These results are shown in Table 4. HAT occurred in 2 recipients on the 1st and 7th d following A-A LDLT, both of which were revascularized with GSV between the donor RHA and recipient AA immediately after thrombectomy. These patients were discharged with good liver function. The third HAT

occurred on the 3rd d after A-A LDLT, and urgent administration of thrombolytic therapy combined with anticoagulant treatment was performed and coagulation status was monitored. Despite recovering HA inflow detected by color Doppler ultrasonography with good liver function, the patient died on postoperative day 36 with multiorgan failure (MOF). The fourth HAT occurred on the 46th postoperative day, however, no ischemia-related complication was observed during the follow-up period. No special treatment for this patient was performed because liver function test results and general condition were good.

DISCUSSION

Hepatic arterial reconstruction, which is critical to successful outcome, is one of the most difficult procedures in A-A LDLT. Currently, many scholars emphasize the importance of microvascular techniques for hepatic arterial reconstruction in LDLT to overcome the risk of HAT^[14-17]. When comparing microsurgical techniques with conventional methods, the use of microsurgical techniques in HA reconstruction can result in a lower incidence of HAT compared with conventional procedures^[17].

Despite the improvements in surgical techniques, a liver graft with a fine HA less than 2 mm in diameter is regarded as a contraindication for LDLT because of the high risk of HAT^[15]. As shown in Table 3, the mean diameter of graft arteries and recipient arteries were equal to or less than 2 mm. In our study, all anastomoses were carried out by a group of vascular surgeons, including two experts in vascular surgery and two assistants, using atraumatic microvascular techniques during the reconstructions. Only 4 cases of HAT in recipients occurred which were in the 58 incipient patients following A-A LDLT. As we accumulated experience in microsurgical hepatic arterial reconstructions, no further cases of HAT occurred in 124 consecutive cases following A-A LDLT. On the basis of our experience in LDLT, the key points in HA reconstruction included: (1) selecting a reciprocal stump location for arterial reconstruction: a thicker arterial stump in the recipient was chosen for the first anastomosis. In 58 incipient cases, the primary candidates for the first anastomosis were the RHA and LHA, which are often small in diameter. To increase the blood flow through the anastomosis, a thicker arterial stump in the recipient, close to the donor stump, was used as the first option for anastomosis in the subsequent 124 cases. In order to expand the anastomotic stoma, the caliber discrepancy between the recipient and graft HA was resolved by cutting the smaller arterial end obliquely to change the effective caliber which was twice as wide as the diameter of the smaller vessel. Thereafter, the incidence of HAT decreased markedly. Other methods such as the fish-mouth method, funnelization method, or end-to-side anastomosis were not used in this study; (2) utilizing back wall-first anastomosis technique. Run-

Table 4 Management and outcome of HAT after A-A LDLT

Case	Gender	Age (yr)	Graft	Diagnosis and management time post A-A LDLT (d)	Management	Survival
1	Male	43	RL	1, 1	Revascularized	Alive
2 ¹	Male	57	RL + LLS	7, 7	Revascularized	Alive
3	Male	40	RL	3, 3	Medications	Dead
4	Female	33	RL	46	Observation	Alive

¹HAT occurred in right hepatic artery. HAT: Hepatic arterial thrombosis; A-A LDLT: Adult-to-adult living donor liver transplantation.

ning microsutures were placed in the back wall of the artery first without turning over the vessel. Compared with conventional sutures, running sutures can avoid repeatedly tied and twisting vessels, both of which can injure fragile intima. Running sutures were employed in all HA reconstruction procedures with a low incidence of HAT (2.08%) in our study. Controversially, another report suggested that interrupted sutures may be a better choice for HA anastomosis with a lower incidence of HAT than continuous sutures^[26]; and (3) treating intimal dissection of recipient HA. In our study, direct end-to-end anastomosis between the recipient and graft HA was employed in most procedures of A-A LDLT. In the case of intimal dissection, recipient HA is not suitable for hepatic arterial reconstruction. Several methods of revascularization have been reported, including application of recipient splenic or right gastroepiploic artery^[27-29], interposition of a vessel graft between the CHA and graft artery^[30,31], or between the AA and the graft artery^[32]. Despite several reports^[4,33] suggesting that interposition of a vessel graft increased the risk of HAT, interposition of GSV was performed between the graft RHA and recipient CHA in 3 cases, and bypass was performed between the graft RHA and recipient AA using GSV in 2 patients and cryopreserved cadaveric iliac vessels in 2 cases. No HAT occurred in the interposition cases. These cases provided a safe and effective approach for the management of intimal dissection. On account of a relatively small sample size in this study, further research is needed.

HAT is the most common vascular complication. Due to irreversible ischemic damage of hepatocytes and bile duct epithelial cells, HAT can cause devastating complications with a high mortality of 50%, including bile leakage, hepatic necrosis, graft loss, and sepsis^[4,5]. Several factors were suggested to cause HAT after LDLT: technical problems^[34-36], anatomic variation^[37], hypercoagulable state^[38], severe intraoperative hypotension^[38], long cold ischemic time^[39], occlusion of HA outflow owing to hepatic congestion^[10], and systemic infection^[10]. Diagnosis can be suggested by Duplex ultrasonography^[12], and confirmed by angiography^[40] or CEUS^[13]. In our study, among the 14 cases without arterial inflow at routine color Doppler ultrasonographic examinations, CEUS showed 4 cases of HAT that were confirmed by HA angiography which is the gold standard for diagnosing HAT.

In the case of early HAT, several methods^[8,9,11], including retransplantation, urgent revascularization, or

revision of anastomosis, have been performed to avoid irreversible damage of the allograft. In our study, two recipients underwent urgent revascularization immediately after HAT was confirmed on the 1st and 7th d after A-A LDLT, and were subsequently cured with satisfactory liver function. One HAT patient received thrombolytic and anticoagulant therapy with monitoring of coagulation status, and thereafter HA inflow returned to normal, however, this patient died of MOF on the 36th d after A-A LDLT. Thrombolytic therapy and anticoagulant therapy for HAT should be carried out cautiously due to the potential risk of hemorrhage. Late HAT, which occurs after the first postoperative month, can be well tolerated, and portal flow can support the liver until the collateral circulation has developed^[41]. No special treatment for the patient in our study with late HAT was performed because liver function test results and general condition were good. However, long-term results of late HAT patients will require further research.

In conclusion, selecting an appropriate anastomotic artery and applying microvascular techniques for HA reconstruction are crucial to reduce the high risk of HAT during A-A LDLT. The principles of HA reconstruction will be followed in our future clinical procedures which include: a settled group of vascular surgeons, running sutures with intimal eversion, sufficient large caliber of the stoma and atraumatic manipulation. Running sutures may be a reliable alternative for anastomosis of small diameter HAs with a low incidence of HAT following A-A LDLT. Nevertheless, randomized prospective controlled studies should be performed to validate these results.

COMMENTS

Background

Hepatic artery thrombosis (HAT), which is the most common vascular complication after living donor liver transplantation (LDLT), can result in graft loss and devastating consequences. Surgical techniques are suggested to an important factor in causing HAT. Despite improvements in surgical techniques, arterial reconstruction in LDLT has a high risk of thrombosis.

Research frontiers

Although microsurgical techniques in arterial reconstruction has helped overcome the high risk of HAT, because of the short donor hepatic artery (HA) stump, which is small in diameter, and vessel size discrepancy between the graft and recipient hepatic arteries, reconstruction of hepatic arteries is a challenge to surgeons in LDLT. The incidence of HAT after LDLT varies widely from 4%-25%.

Innovations and breakthroughs

With atraumatic microsurgical techniques, the reconstruction of HA was accomplished in 182 recipients (192 grafts) of adult-to-adult LDLT performed

from January 2001 to September 2009 by a settled group of vascular surgeons. Interposition of the great saphenous vein or cryopreserved vessel between the recipient and graft was performed for the treatment of 7 intimal dissections of recipient hepatic arteries. Both back-wall first techniques and running sutures with intimal eversion were employed in all anastomoses, and a good outcome with a low incidence of HAT (2.08%), was achieved.

Applications

Besides the use of microsurgical techniques, reconstruction of the HA by settled surgeons in LDLT provides an approach for decreasing the incidence of HAT. Based on our study results, running sutures can be used in the anastomoses of hepatic arteries of small diameter.

Peer review

This is a retrospective analysis of HA thrombosis after living donor liver transplantation.

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