# ORIGINAL ARTICLE

# Endovascular management of early hepatic artery thrombosis after living donor liver transplantation

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#### **Conflicts of Interest**

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#### Summary

To study the feasibility of endovascular management of early hepatic artery thrombosis (HAT) after living-donor liver transplantation (LDLT) and to clarify its role as a less invasive alternative to open surgery. A retrospective review of 360 recipients who underwent LDLT. Early HAT developed in 13 cases (3.6%). Diagnosis was performed using Doppler, CT angiography, and digital subtraction angiography. Intra-arterial thrombolysis (IAT) was performed using streptokinase or tPA. In case of underlying stricture, PTA was attempted. If the artery did not recanalize, continuous infusion was performed and monitored using Doppler US. Initial surgical revascularization was successful in 2/13 cases. IAT was performed in 11/13 cases. The initial success rate was 81.8% (9/11), the failure rate was 18.2% (2/11). Rebound thrombosis developed in 33.3% (3/9). Hemorrhage developed after IAT in 2/11 cases (18.2%). Definite endovascular treatment of HAT was achieved in 6/11 cases (54.5%) and definite treatment (surgical, endovascular or combined) in 9/13 cases (69%). (Follow-up 4 months-4 years). Endovascular management of early HAT after LDLT is a feasible and reliable alternative to open surgery. It plays a role as a less invasive approach with definite endovascular treatment rate of 54.5%.

## Introduction

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 [1]. Early HAT occurs within the first 2–4 weeks of transplantation and is often catastrophic. The mortality because of early thrombosis is high as 55.6% compared with 15–22.6% because of late thrombosis. It requires early diagnosis and revascularization to avoid graft loss [2,3].

The most effective treatment approach is still controversial. Urgent re-transplantation has been considered as the mainstay therapy. Surgical revascularization is an effective alternative for graft salvage, or may help as a bridging measure for a re-transplantation in a less emergent setting [4–6].

Endovascular management including intra-arterial thrombolysis (IAT), percutaneous transluminal angioplasty (PTA), and stent placement has emerged as an attractive and less invasive alternative to surgical intervention in recent years; however, their use remains controversial because of potential risk of hemorrhage [7–10].

The purpose of this study is to review the different strategies in the management of early HAT after living donor liver transplantation (LDLT) and to clarify the feasibility of IAT with or without PTA and stenting as a less invasive alternative to open surgery. We present the preliminary results of 13 cases with early HAT after LDLT in a consecutive 360 cases.

## Materials and methods

Approval of this study was obtained from the institutional review board, and informed consent was obtained from all patients including approval of the protocol of treatment and the anonymous use of the data for research purposes.

The medical records of 360 patients who underwent LDLT between August 2001 and April 2011 in Cairo University Hospitals and Dar Al Foad Hospital were retrospectively evaluated. A total of 13 male patients ranging in age from 38 to 62 years (mean: 54 years) developed early HAT within 2 weeks post-operative (PO) and were treated by urgent revascularization.

All the patients had previous HCV related cirrhosis and received right lobe graft from a living donor.

## Management protocol of the patients

A multidisciplinary team including micro surgeons, transplant surgeons, interventional radiologists, and hepatologists decided the protocol of management.

### Technique of hepatic artery reconstruction

Our technique of hepatic artery reconstruction is performing an end-to-end anastomosis between the graft artery and the recipient right hepatic artery or one of its smaller terminal branches, the left or the middle hepatic artery if present. We choose the recipient artery nearest in size to the graft artery to minimize the size mismatch. The length must be adequate to allow a tension-free anastomosis, but if it is too long it must be trimmed to avoid kinks and rotations. We use the microscope with a magnification of 4× to 8× depending on the need. A double clamp is used. The suture material used is black monofilament nonabsorbable nylon 8-0 on a taper point 3/8 circle curved needle either 6.5 mm (BV-135-5) or 5 mm (BV 135-4) long. The whole anastomosis is performed using interrupted stitches. Intra-operatively aspirin is started as a suppository and continued until oral medications can be given. Parameters for holding aspirin postoperatively are a platelet count below 30 000 or severe postoperative bleeding. Low molecular weight heparin is administered postoperatively when the international normalized ratio (INR) drops below 2.

## Intra-operative evaluation of the hepatic artery

The normal spectral Doppler waveform of the HA should have a biphasic pattern with resistivity index (RI) ranging 0.5–0.8. Anastomotic flow jet (2–3 folds compared with the preanastomotic flow) was considered a normal find-

ing in the presence of size mismatch so far there was normal intrahepatic flow pattern. We do not rely on absolute values of the PSV as it is influenced by many factors, including the systemic pressure, source of the recipient artery, size mismatch, kinks, and spasm. A high anastomotic jet (>3 folds) with high pitched sound and damped intrahepatic PSV and increased systolic acceleration (Tardus Parvus waveform) were considered as signs of HAS.

## Diagnosis of HAT

Serial Doppler US were performed for all patients every 12 h during the 1st w PO then once daily till hospital discharge.

If abnormal arterial flow whether damped flow or abnormal resistivity index (RI <0.55 or RI >0.80) was detected with no clinical or laboratory data suggestive of HAT, follow-up was performed using Doppler US to confirm that the hepatic artery waveform had normalized.

When Doppler Ultrasound revealed the absence of arterial flow, confirmed by two sonographers and the clinical and laboratory picture were suggestive of HAT, Digital subtraction angiography (DSA) was performed to rule out HAT. Multi-detector CT angiography (MDCTA) had been used in some cases to confirm the diagnosis prior to conventional angiography.

#### Technique of intra-arterial thrombolysis

Diagnostic arteriography was performed with standard catheter techniques using the right femoral artery access with selective catheterization of the celiac trunk using 5 F cobra or Simon catheters. Once HAT was confirmed, a micro catheter was manipulated into the thrombus. Thrombolysis was then performed with bolus dose of a 150 000 µ streptokinase, if the artery did not recanalize, continuous infusion at a rate of 100 000 µ/h. was performed for maximum of 12 h. Tissue plasminogen activator (tPA) was used in two cases. The total dose used was 0.9 mg/kg (10 mg bolus dose and continuous infusion of the rest over 24 h). If the angiogram after recanalization revealed an underlying stricture, PTA and stent placement were performed. The endovascular interventions were monitored using Doppler US to ensure good arterial flow in the graft during the procedures. During continuous infusion, Doppler US was performed every 2 h. If the Doppler revealed arterial flow in the hepatic artery at the graft hilum as well as the in the intrahepatic branches, the infusion was stopped, then control angiography was performed.

After recanalization, follow up was performed using daily Doppler US every 12 h for 3 days, then was performed daily till hospital discharge. Successful IAT was defined as complete or partial resolution of the thrombus with delineation of the intrahepatic branches. Definite endovascular treatment was defined as complete resolution of the thrombus without underlying stricture. Definite treatment was defined as complete resolution of the thrombus without underlying stricture by surgical or endovascular revascularization or using them in conjunction.

## Results

Early HAT within 2 weeks PO occurred in 13 out of 360 cases. Eight cases in the 1st week (61.5%) and five cases in the 2nd week (38.5%), ranging 1–11 days, mean 5.4 days. At the time of diagnosis, shooting of the liver enzymes was noticed in eight cases (61.5%), being stable in three cases (23%) and mildly elevated in two cases (15.4%).

IAT was attempted in 11/13 cases. Streptokinase was used in nine cases and tPA used in two cases. Initial successful IAT was achieved in 9/11 cases (81.8%). Successful recanalization was achieved after the bolus dose in 7/9 cases (six cases using streptokinase and one case using tPA), whereas two cases required continuous infusion of tPA for 8 h and streptokinase for 12 h to recanalize.

IAT failed in 2/11 cases (18.2%) even after continuous infusion of streptokinase for 12 h.

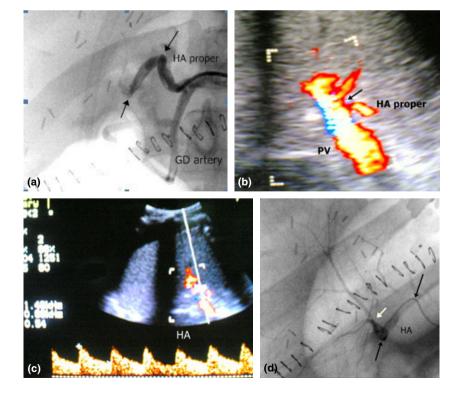
Successful IAT revealed underlying hepatic artery stenosis (HAS) in 4/9 cases (44.4%), significant kink and

size mismatch between the recipient and donor arteries in 2/9 cases (18.2%) (Fig. 1e) and did not reveal underlying anatomical defect in 3/9 cases (33.3%).

After successful recanalization, rebound thrombosis developed in 3/9 cases (33.3%). One case had adherent thrombus at the anastomotic site and rebound thrombosis developed after 4 h, for which successful surgical reconstruction was performed (follow up 2 years). In the 2nd case, IAT was performed after failed surgical revascularization in the 1st day PO, PTA was not attempted and rebound thrombosis developed after 72 h. The patient died after 2 weeks of graft failure. The 3rd case, rebound HAT developed after 72 h from successful IAT and stent placement. This was associated with portal vein thrombosis. Successful surgical revascularization (PV thrombectomy and saphenous graft of the HA) was performed. This patient died after 2 months of persistent graft failure after successful revascularization.

Initial surgical revascularization was performed in 4/13 cases. Successful revascularization was achieved in two cases, whereas rebound thrombosis developed in the other two cases. IAT was then attempted and was initially successful. In one case (Fig. 3), the intra-operative biopsy revealed acute rejection and the patient died after 1 week of graft failure. In the other case, rebound thrombosis developed because of failure to resolve the underlying HAS after successful thrombolysis and the patient died after 3 days of graft failure.

Figure 1 Fifty-four years old recipient 5th day post-LDLT with shooting liver enzymes and absent hepatic artery flow on Doppler US. (a) Selective hepatic arteriogram after bolus dose streptokinase shows occluded HA proper with failure to delineate the intrahepatic branches. Note two kinks in the HA proper (arrows). (b) Color US image 12 h after continuous streptokinase infusion shows recanalization of the hepatic artery. Note the kink in the hepatic artery proper (arrow). (c) Doppler US image shows normal Doppler waveform of the hepatic artery distal to the anastomosis. (d) Control hepatic arteriogram after stoppage of infusion shows patent HA and opacified intrahepatic branches. Note the size mismatch between the recipient and donor hepatic arteries (white arrow) and the two kinks in the HA (black arrows). (Follow-up 1 year).



Hemorrhage developed in two cases at the anastomotic site after 8 h of continuous infusion of tPA and after 12 h of continuous infusion of streptokinase. Both required blood transfusion, one of them survived, whereas the other died after 1 week of graft failure (Fig. 3e).

In 2/360 cases, Doppler US and MDCTA were suggestive of HAT with abrupt termination of the HA proper and absent intrahepatic arterial flow. Splenic artery steal syndrome (SASS) was diagnosed using conventional celiac arteriography. In one case (Fig. 4), the liver enzymes were stationary, celiac arteriography revealed moderate stenosis

 Table 1. Results of surgical and endovascular revascularization of 13 pts with early HAT after LDLT (S, successful; F, failed).

Total number	13 cases
Date of presentation	1st week: 8 pts
	2nd week: 5 pts
	Range 1–11 days
	Mean 5.45 days
Liver enzymes	Shooting: 8/13 (61.5%)
	Mildly elevated: 2/13 (15.4%)
	Stationary 3/13 (23%)
Management	
Initial surgery	4 cases
	(S): 2 pts with HAS
	(F): 2 pts (persistent HAS & rejection)
Initial IAT	9 cases
Combined surgery & IAT	4 cases
	(S): 2 pts. IAT was followed by surgery
	(F): 2 pts. Surgery was followed by IAT

Table 2. Results of IAT for 11 cases with HAT.

No of cases	11
Drug	Streptokinase: 9/11 tPA: 2/11
Outcome	Initial success: 9 /11 (81.8%) Failed: 2/11 (18.2%)
Underlying cause of HAT	HAS 4/9 (44.4%)
after successful IAT	Kink & mismatch 2/9 (18.2%) No anatomical cause 3/9 (33.3%)
PTA & stent	Performed in 2/4 with HAS (S)
Rebound thrombosis	3/9 (33.3%)
Complications	Bleeding 2/11 (18.2%)
Definite endovascular treatment of HAT	6/11(54.5%) follow up 4 months–4 years
Definite treatment (Surg/Endovasc or combined)	9/13 (69%)
Mortality (follow up	5/13 (38.5%)
4 months-4 years)	3 pts failed revascularization 2 pts persistent graft failure after successful revascularization

at the origin of the CHA with absent intrahepatic flow and splenic arterial steal. Successful PTA and stenting were performed with decreased splenic steal and normalization of the Doppler waveform (follow up 6 months). In the other case, the liver enzymes were shooting. Celiac arteriography revealed absent intrahepatic flow and splenic arterial steal. On selective hepatic arteriography, the HA was patent and showed significant kink and size mismatch between the donor and recipient arteries. Surgical ligation of the splenic artery was performed and complicated by splenic abscess and underwent splenectomy. This patient died after 2 months graft failure.

The results of surgical and endovascular revascularization of HAT are summarized in Tables 1 and 2.

## Discussion

Hepatic artery steno-occlusive disease is the most common arterial complication following orthotopic liver transplantation. This includes HAT, HAS, kinks, pseudoaneurysms, and arterial steal syndromes. Early diagnosis is mandatory to prevent graft loss [11–14]. Endovascular management has emerged as an attractive alternative to surgery in the management of these cases. Few case reports and series have described the endovascular revascularization after LDLT. Here in, we present our experience with 13 cases that developed early HAT after LDLT in which endovascular approach was attempted in 11 cases.

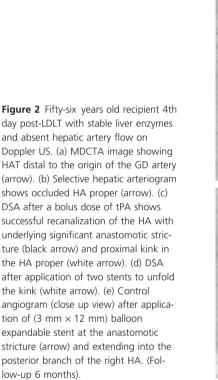
## Risk factors of HAT

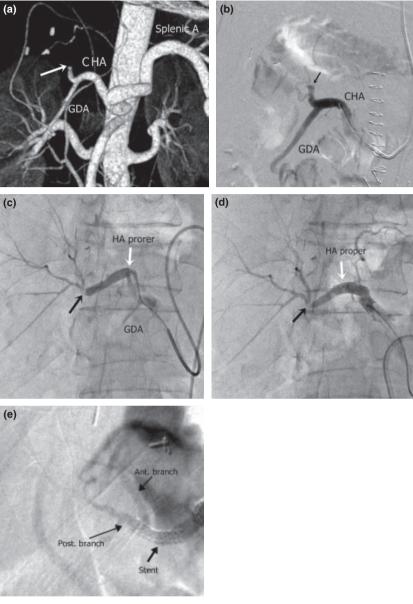
Potential risk factors for HAT have been identified in many series. It may be surgical (technical) or nonsurgical. We encountered underlying HAS in 6/13 cases (46.2%) that developed HAT (Fig. 2). The arterial anatomy of both recipient and donor/graft can affect the incidence of HAT. The smaller the arteries, the higher the incidence, as evidenced by the higher rate in pediatrics [15].

One common problem is the discrepancy in size between the graft and recipient arteries. Different techniques have been described for an anastomosis in such cases. Funnelization is our preferred technique. In two cases that developed HAT, there was significant kinks and size mismatch between the recipient and graft arteries (>2:1). However, they were considered as contributing factors as both cases were treated by IAT with no further intervention (Fig. 1). Also, kinks and size mismatch contributed to the splenic artery steal in one case.

Abnormal arterial anatomy in the graft requiring bench reconstruction, arterial conduit use and multiple anastomoses are all risk factors [16]. Intimal dissection is another risk factor [17]. This is especially problematic in

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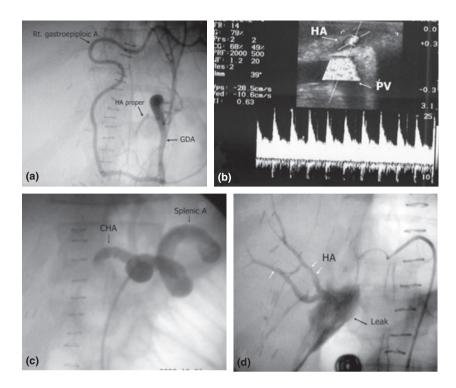




the graft artery where the flap is along the direction of blood flow, whereas proximal dissection of the recipient artery is self-limiting. If the duplex shows a single stream from the main lumen, the anastomosis can be performed safely. We encountered proximal dissection in one case that developed HAT during the surgical revision of the anastomosis and was self-limiting.

Interrupted sutures are associated with lower hepatic arterial complications [18]. Also, the use of magnification microscopes with continuous zoom as well as high magnification loupes reduces the incidence [19,20]. In the first 30/360 cases of our series, the hepatic artery reconstruction was performed using magnifying loop, the incidence of HAT was 4/30 (13.3%). In the next cases, all the hepatic artery reconstructions were performed by a microsurgeon using the operative microscope, the incidence of HAT improved to 2.1% (7/330 cases).

Nonsurgical potential risk factors, include hypercoagulable disorders (thrombophilias) in recipient and donor, cytomegalovirus mismatch, ABO incompatibility, and the arterial reperfusion time. Prolonged operation times are also suggested. Another important risk factor is acute rejection episodes, which increase the capillary bed resistance thus impending the arterial flow [15,16,21,22]. We encountered one case that developed HAT because of acute rejection in the 11 days PO (Fig. 3).



**Figure 3** Fifty-eight years old recipient 11th day post-LDLT with shooting liver enzymes and absent hepatic artery flow on Doppler US. (a) Selective DSA shows occluded hepatic artery distal to the hypertrophied GD artery. Selective catheterization of the HA proper for thrombolysis failed. Surgical revascularization was performed. (b) Intra-operative Doppler US image after thrombectomy and ligation of the GD artery shows good flow in the HA. However, re-thrombosis developed after 2 days. Biopsy proved acute graft rejection. (c) Selective celiac arteriogram shows thrombosed common HA. The artery did not recanalize after bolus tPA. (d) Control angiogram after 6 h of continuous tPA infusion shows recanalization of the HA and contrast extravasation at the anastomotic site. The patient died after 1 week of graft failure and sepsis.

## Diagnosis of HAT

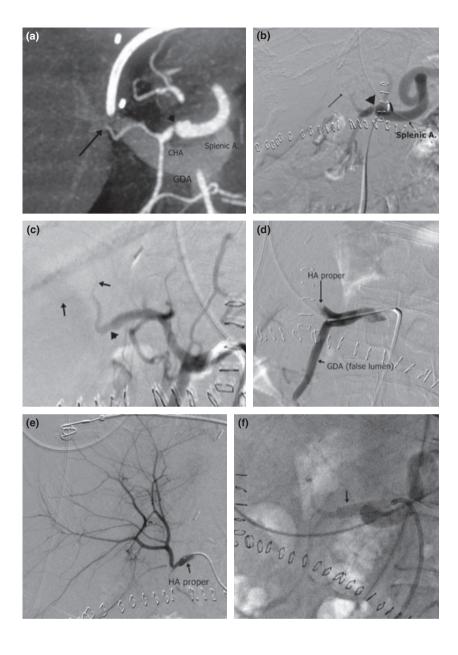
Early HAT always manifest clinically with fever, leukocytosis, severe elevation in liver enzyme levels, or septic shock [6]. By performing serial US examinations, HAT can be discovered when it is not yet suggested on clinical grounds. This saves precious time [23,24]. We diagnosed HAT prior to the clinical manifestations in 3/13 cases (23%) when Doppler US was performed twice daily in the 1st week PO. The most common finding on Doppler US was the absence of intrahepatic arterial flow in 12/13 cases. In one case, we relied on the sign of impending thrombosis described by Nolten and Sproat [25] in which there was no diastolic flow (RI = 1) and damped systolic peak. The liver enzymes were stable at that time. This phenomenon (no diastole) was also encountered in other patients with high portal flow and cases of small-for-size grafts [19]. In these equivocal cases, HAT should be ruled out by MDCTA or DSA.

Although DSA is the gold standard, MDCTA has emerged as a noninvasive imaging modality for the diagnosis of HAT. A sensitivity rate of 100% with a specificity rate of 89% and accuracy of 95% has been demonstrated. Conventional catheter angiography can be used as a next step possibly if any interventional treatment is contemplated [26,27]. We encountered false positive results using Doppler US in 3/360 cases when MDCTA was not performed and DSA revealed patent HA.

#### Management of HAT

In general, there are three different treatment modalities for HAT: Re-transplantation, surgical revascularization, and endovascular revascularization. However, the most effective treatment approach remains controversial. Traditionally, retransplantation has been the first choice of therapy [4].

Our criteria for management of HAT depend on many factors: The timing of thrombosis, the possible underlying cause, the graft function, and the opinion of the surgeon who performed the anastomosis. If the onset is within the first 3 days PO, we prefer surgical revascularization as the underlying cause is probably technical and the exposure is relatively easy as adhesions have not developed yet. Also, we do not recommend thrombolysis that early after surgery, especially if the drain output signifies early ongoing Figure 4 Fifty-five years old recipient 5th day post-LDLT with stationary liver enzymes and absent HA flow on Doppler US. (a) MDCTA shows CHA stenosis (arrowhead) and abrupt termination of the HA proper (long arrow). (b) DSA shows CHA stenosis (arrowhead), patent HA proper (arrow) and nonopacified intrahepatic branches. The splenic artery was hypertrophied with contrast steal noted during angiography. (c) Selective hepatic angiogram showed patent HA (arrowhead) and intrahepatic branches (arrows). Dissection occurred at the origin of the CHA during selective catheterization. (d) DSA shows dissected CHA with stasis of contrast in the false lumen of the CHA and GD artery (short arrow). The flap occluded the origin of the HA proper (long arrow). (e) Selective hepatic arteriogram after successful catheterization of the true lumen of the HA shows patent intrahepatic branches. (f) Control angiogram after PTA shows patent stent in the CHA (arrow) with filling of the intrahepatic branches during celiac angiography. Improved HA flow was confirmed using Doppler. (Follow-up 4 months).



bleeding. In addition, the endovascular approach may carry an increasing risk of complications as spasm, dissection, hemorrhage or anastomotic rupture in that early PO period.

After the 3rd day PO, the cause of HAT is less likely to be technical error and may be related to hemodynamic or immunological abnormality, so we prefer to attempt IAT. In addition, surgical re-exploration after the 3rd day PO is more difficult and carries the risk of dislodgment of the biliary stents or injury to the vascular pedicle that by now had formed adhesions around them.

Finally, in our protocol of management, cases that failed arterial revascularization or had successful revascu-

larization after a prolonged interval of ischemia with clinical and laboratory manifestations of graft failure are considered as candidates for re-transplantation. In Egypt, deceased donor liver transplantation is not available therefore surgical or endovascular revascularization remain the primary options because of difficulties of finding a suitable donor in an emergency setting.

In a systematic review by Bekker *et al.* [16], an overall success rate with surgical revascularization of about 50% was reported with similar rates among adults and children. The success rate was even higher (66.1%) with early HAT. This is in contrast with success rate of 10.5% reported by another study [28]. Twelve cases of surgical

revascularization in conjunction with IAT have been also reported in six studies [2,6,29–32]. The success rate of surgical revascularization in this study was 50% (2/4 cases). The failure of surgical followed by IAT in the other two cases was the persistence of acute graft rejection in one case and failure to resolve the underlying stricture in the other case.

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 [4]. 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).

Majority of studies indicated the preferred use of urokinase. We had to use streptokinase in 9/11 cases, as it was available on shelf. There is no consensus on the optimal technique for catheter-directed delivery of any thrombolytic agent as they have been successfully used as continuous infusion or bolus form [4,33–35]. We agree with the termination of thrombolysis, if there is a residual thrombus or persistent HAT. We allowed continuous infusion of streptokinase for max 12 h to avoid the potential risk of hemorrhage.

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 [34,36–40]. In this study, 4/11 cases that underwent IAT showed underlying HAS. PTA and stent application were successful in two cases (Fig. 2). The 3rd case underwent successful surgical revascularization, whereas the 4 h case failed with conservative treatment without angioplasty.

Hemorrhage was the most common reported complication of thrombolysis seen in about 20% of the patients [4]. Fatal hemorrhage was reported in three patients. In this study, 2/11 cases (18.2%) developed serious intraabdominal bleeding after 8 h of continuous infusion of tPA and after 12 h of continuous infusion of streptokinase. Both required blood transfusion and survived the bleeding episode.

## Splenic artery steal syndrome

The splenic artery steal phenomenon has been recognized as a potential threat to transplanted livers and has been already described in case reports or small series. In those cases, liver ischemia was related to reduced perfusion or a situation of low flow status through the hepatic artery rather than obstruction [41–44].

As it falls in the broader spectrum of graft ischemia, we present our limited experience with SASS in 2/360 cases. Their radiological picture using Doppler US and MDCTA were suggestive of HAT. The steal phenomenon was demonstrated during celiac and selective hepatic arteriography. In addition to the hypertrophied splenic artery, an underlying CHA stenosis had been demonstrated and contributed to the steal phenomenon in one case (Fig. 4). After angioplasty, we did not proceed to splenic artery embolization because of the significant improvement of the intrahepatic flow monitored using Doppler US. Although in the other case, there was a significant HA kink and size mismatch that might have contributed in the splenic steal and this case was treated by splenic artery ligation. We recommend splenic artery embolization in SASS, as it became the therapeutic procedure of choice, it is minimally invasive and successful for steal, usually resulting early clinical improvement [44-47].

## Conclusion

Early HA complications remain a major cause of morbidity and mortality after LDLT. Urgent revascularization is necessary to avoid graft loss. Endovascular approach, including IAT and PTA is emerging as a less invasive alternative to open surgery in the management of early HAT and can be used in conjunction. The choice of therapy depends on a variety of factors, including the timing of thrombosis, the graft function, the underlying cause, and the availability of organs for re-transplantation.

## Authorship

OA and MM: contributed equally to this work and designed the research. OA, KH, AA, SE-E, SU and MM: performed research and analyzed data. OA, KH and MM: wrote the paper.

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