Complications after endovascular treatment of hepatic artery stenosis after liver transplantation

Leighton E. Goldsmith, MD, Kristy Wiebke, DO, John Seal, MD, Clayton Brinster, MD, Taylor A. Smith, MD, Hernan A. Bazan, MD, and W. Charles Sternbergh III, MD, New Orleans, La

ABSTRACT
Background: Hepatic artery stenosis (HAS) after liver transplantation can progress to hepatic artery thrombosis (HAT) and a subsequent 30% to 50% risk of graft loss. Although endovascular treatment of severe HAS after liver transplantation has emerged as the dominant method of treatment, the potential risks of these interventions are poorly described.

Methods: A retrospective review of all endovascular interventions for HAS after liver transplantation between August 2009 and March 2016 was performed at a single institution, which has the largest volume of liver transplants in the United States. Severe HAS was identified by routine surveillance duplex ultrasound imaging (peak systolic velocity >400 cm/s, resistive index <0.5, and presence of tardus parvus waveforms).

Results: In 1129 liver transplant recipients during the study period, 106 angiograms were performed in 79 patients (6.9%) for severe de novo or recurrent HAS. Interventions were performed in 99 of 106 cases (93.4%) with percutaneous transluminal angioplasty alone (34 of 99) or with stent placement (65 of 99). Immediate technical success was 91%. Major complications occurred in eight of 106 cases (7.5%), consisting of target vessel dissection (5 of 8) and rupture (3 of 8). Successful endovascular treatment was possible in six of the eight patients (75%). Ruptures were treated with the use of a covered coronary balloon-expandable stent graft or balloon tamponade. Dissections were treated with placement of bare-metal or drug-eluting stents. No open surgical intervention was required to manage any of these complications. With a median of follow-up of 22 months, four of eight patients (50%) with a major complication progressed to HAT compared with one of 71 patients (1.4%) undergoing a hepatic intervention without a major complication (P < .001). One patient required retransplantation. Severe vessel tortuosity was present in 75% (6 of 8) of interventions with a major complication compared with 34.6% (34 of 98) in those without (P = .05). In the complication cohort, 37.5% (3 of 8) of the patients had received a second liver transplant before intervention compared with 12.6% (9 of 71) of the patients in the noncomplication cohort (P = .097).

Conclusions: Although endovascular treatment of HAS is safe and effective in most patients, target vessel injury is possible. Severe tortuosity of the hepatic artery and prior retransplantation were associated with a twofold to threefold increased risk of a major complication. Acute vessel injury can be managed successfully using endovascular techniques, but these patients have a significant risk of subsequent HAT and need close surveillance. (J Vasc Surg 2017;66:1488-96.)

A devastating and frequent vascular complication after orthotopic liver transplant is hepatic artery thrombosis (HAT), which can lead to severe cholangiopathy and graft loss.19 Hepatic artery stenosis (HAS), which can predispose the artery to subsequent thrombosis, has a reported incidence of 5% to 11% in adults after liver transplantation.1569-18 However, the true incidence is not well defined because most patients are asymptomatic.58

Untreated HAS has a reported incidence of subsequent thrombosis as high as 65%.18 A number of different therapeutic options can be used to treat HAS after liver transplantation, ranging from conservative treatments, such as anticoagulation, to more invasive options, ranging from surgical revascularization to retransplantation.2468,10,13,14,16,17 Early HAT (<7 days after transplant) is still typically treated by open surgical revascularization.1 In the last decade, endovascular treatment has emerged as a less invasive and primary option for management in posttransplantation HAS. Although the risk of mortality and morbidity is significantly decreased in percutaneous treatment compared with surgical revascularization and retransplantation, these complex interventions are not risk free.

The potential risks of hepatic artery interventions are not well described in the literature. Potential complications include hepatic artery rupture, perforation, dissection, pseudoaneurysm formation, and thrombosis.146,11,12,14-18 The reported risk of such complications

From the Section of Vascular and Endovascular Surgerya and Section of Transplant Surgeryb Department of Surgery, Ochsner Clinic. Author conflict of interest: none.
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Correspondence: W. Charles Sternbergh III, MD, Professor and Chief, Vascular and Endovascular Surgery, Ochsner Clinic, 1514 Jefferson Hwy, New Orleans, LA 70115 (e-mail: csterbergh@ochsner.org).
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during endovascular treatment ranges from 5% to 19%.3-6 However, most publications give scant specific information regarding the type of complication, its management, or patient outcomes.

Here, we identify the type and incidence of major complications of endovascular treatment of HAS after liver transplantation, assess possible anatomic risks that can lead to these complications, and discuss endovascular therapeutic rescue methods.

METHODS

This was a single-institution retrospective review of a prospective database of all patients undergoing hepatic angiography and intervention for HAS after liver transplantation between August 2009 and March 2016. Institutional Review Board approval was obtained, patient records were deidentified, and patient consent was waived. Notably, this institution has the largest volume of liver transplantation in the United States (U.S.) since 2011.

Severe HAS was identified on routine duplex ultrasound imaging and defined as main hepatic resistive index <0.5, a peak systolic velocity >400 cm/s, and presence of tardus parvus waveforms. For patients with normal renal function, most patients with HAS subsequently underwent confirmation of these findings on computed tomography angiography (CTA). HAT was defined as lack of arterial flow identified on angiography or on duplex ultrasound imaging. Significant tortuosity of the hepatic artery was defined as a >90° acute bend in the vessel measured on angiography or CTA.

The standard protocol for vascular surveillance after transplant included routine ultrasound assessments, which were performed on postoperative days 1 and 7 and at 1, 6, and 12 months after transplant. Owing to the potential increased risk for vascular complications for recipients of donation-after-circulatory death livers, closer surveillance was performed with additional ultrasound imaging at 3 and 9 months after transplant. If significant abnormalities were identified during surveillance ultrasound imaging, closer surveillance was performed at the discretion of the surgery team.

Initial technical success was defined as <30% residual stenosis after treatment. Patients with major complications were considered technical successes only if the hepatic stenosis was successfully treated. Patients with successful treatment of a complication but unsuccessful or aborted treatment of the HAS were considered technical failures.

The decision to intervene was based exclusively on imaging studies. Isolated HAS rarely caused acute clinical abnormalities in liver function (such as enzyme elevations), and thus, liver function test results were not a useful marker for HAS.

ARTICLE HIGHLIGHTS

- **Type of Research:** Single center retrospective cohort study
- **Take Home Message:** Endovascular management of 99 hepatic artery stenoses after liver transplantation was technically successful in 91%. Major complications (dissection, rupture) occurred in 7.5%, and they were associated with hepatic artery tortuosity and retransplantation. Complications ultimately led to hepatic artery thrombosis in 50%.
- **Recommendation:** This study suggests that endovascular management of hepatic artery stenosis after liver transplantation is safe and effective, with a major complication rate <10%, with half of these ultimately developing hepatic artery thrombosis.

**Technique.** Detailed technique for HAS has been previously described.4,5 In brief, femoral or brachial access was performed depending on the angulation of the celiac artery, with the femoral approach used in most patients. A 6F RDC (Boston Scientific, Marlborough, Mass) or RDC-1 guide was favored when femoral access was used, and a 5F multipurpose sheath was used in cases performed from a brachial approach. A 0.035-inch stiff angled Glidewire (Terumo Medical Corp, Somerset, NJ) was typically used to select the celiac, superior mesenteric artery, or aortohepatic conduit, depending on transplant anatomy. After this wire was placed into the gastroduodenal artery or distal common hepatic artery, a 5F Glide catheter (Terumo) was advanced for more support, allowing the working guide or sheath to be telescoped over the catheter into the terminal common hepatic artery. Advancement of the working platform well into the hepatic vasculature was needed to provide adequate support in delivering the balloon and stents through frequently tortuous distal anatomy. All patients received therapeutic weight-based bolus heparin (100u/kg) at the beginning of the procedure, and protamine was used to reverse anticoagulation at the end of the procedure.

Interventions were typically performed using a 0.014-inch wire platform, with preferential use of regular or the ChoICE PT extra support guide wire (Boston Scientific). In cases of ostial left and right HAS, separate wires were used for each vessel. Low-profile coronary angioplasty balloons (2.0- to 5.0-mm diameter × 12- to 30-mm length) were used in most cases to dilate the lesion. Subsequent stenting was selectively performed when stent placement was technically feasible and in cases of >30% residual stenosis after angioplasty. Balloon-expandable (bare-metal and drug-eluting) coronary stents were used in >95% of cases. Initial balloon sizing was conservative, generally choosing a diameter thought to be at least 1 mm smaller than the reference vessel. Larger balloons were then used as needed to match
the reference vessel. Stent diameters were also chosen to match the reference vessel without oversizing.

**Treatment of complications.** Arterial dissections occurred with the initial cannulation attempt of vessels or during attempted advancement of a balloon or stent. If a wire could be placed into the true lumen distally, these patients were generally treated with stent placement. In cases of rupture, initial extended balloon tamponade was used. If this was not successful, Jomed (Cardinal Health, Dublin, Ohio) covered balloon-expandable coronary stents (2.5-3.5 mm) were selectively used on an emergency compassionate-use basis.

**After the intervention.** Patients treated with percutaneous transluminal angioplasty (PTA) alone generally received aspirin (81 mg), whereas patients who received bare metal stents received dual-antiplatelet therapy with (81 mg of acetylsalicylic acid [ASA] and 75 mg of clopidogrel) for 1 month postintervention, with continued use of ASA long-term. For patients who received a drug-eluting stent, dual antiplatelet therapy was continued for 6 to 12 months with subsequent ASA long-term.

**Statistical analysis.** A Student t-test was performed to compare continuous variables, which are presented as a median with interquartile range (IQR). A Fisher exact test was performed for categorical variables. Primary patency rates with angioplasty alone compared with stent and assisted primary patency were analyzed and reported as described previously.  

**RESULTS**

During the 79-month study period, 1129 orthotopic liver transplants were performed. From this patient cohort, 106 angiograms were performed in 79 patients (6.9% [79 of 1129]) for severe de novo or recurrent HAS. Fifteen percent (12 of 79) of these patients had undergone a second liver transplant before intervention.

The mean time from transplantation to vascular intervention was 71 ± 224 days (range, 5 days-45 months). The median time to intervention in the noncomplication cohort was 69.5 days compared with 90 days in the complication cohort. The mean recipient age was 51 ± 13 years (range, 11-72), and 24% (19 of 79) of patients were women. The mean follow-up period in this series was 22.3 ± 21.7 months (range, 0-82 months).

Interventions were performed in 99 of 106 (93.4%) of these cases, with PTA alone (34 of 99) or with stent placement (65 of 99). Drug-eluting stents were used in 18% (12 of 65) of cases. Eighty-six percent (85 of 99) of interventions were performed on the proper hepatic artery, and 11% (11 of 99) of cases were performed exclusively on the right or left hepatic artery, or both. Multiple areas of intervention were required in 9.9% (10 of 99) cases. In three patients, isolated severe celiac artery stenosis was treated because it was causing poor hepatic arterial flow. Seven angiograms were only diagnostic secondary to inability to gain access (n = 1), no flow-limiting stenosis seen on the angiogram (n = 5), or need for surgical revision (n = 1). Femoral access was used for 77 of 106 (72.6%) angiograms. Brachial access was used when there was marked caudal angulation of the celiac artery.

**Reinterventions.** The reintervention rate was 26.5% (21 of 79) in this series. There were 27 reinterventions in 21 patients. One patient underwent five reinterventions, and two other patients required two reinterventions each. Reinterventions occurred in 22% (10 of 46) of the patients who initially had stent placement and in 38% (10 of 26) of patients who initially had PTA alone (P = .182). The overall time to the initial reintervention was 97 ± 101 days. The median time to the initial intervention was 40 days (IQR, 8-156 days) in the 10 patients treated with PTA initially compared with a median of 101 days (IQR, 37-158 days) in the 10 patients treated with a stent (P = .64). All initial reinterventions in the PTA group were done with stent placements.

The initial technical success rate was 91% (90 of 99). Initial failures included those with major complications that could not be treated (n = 2) and those whose initial complication was successfully treated but the HAS treatment was aborted (n = 2). The remaining technical failures were those patients who had a >30% residual stenosis despite intervention (n = 5).

**Complications.** Major complications occurred in eight of 106 cases (7.5%), consisting of target artery dissection (5 of 8) or rupture (3 of 8), outlined in Table I. For these major complications, successful acute endovascular treatment was possible in six of eight patients (75%). No open surgical intervention was required to manage any of these complications. However, four of eight of these patients (50%) progressed to HAT compared with one of 71 patients (1.4%) undergoing intervention without a major complication (P < .001). One patient required retransplantation 1 month after failed intervention.

**Risk factors for complications.** Severe vessel tortuosity was present in 75% (6 of 8) of interventions with a major complication compared with 34.6% (34 of 98) in those without (P = .05). Patients with a second liver transplant had a threefold increase in the risk of a major complication in the complication cohort. 37.5% (3 of 8) of the patients had received a second liver transplant before intervention compared with 12.6% (9 of 71) of the patients in the noncomplication cohort (P = .097).

**Ruptures.** Hepatic artery rupture occurred in patient 1, who had an intervention 6 months earlier for HAS. The rupture was contained and was treated with a placement of a bare-metal self-expanding stent and prolonged balloon tamponade. Notably, her stenosis was at the apex of a 360° corkscrew of the hepatic artery. CTA
performed 4 days postoperatively revealed resolution of the rupture without hepatic artery pseudoaneurysm. At 56 months postprocedure, she continued to have good liver function with no HAS.

Hepatic artery rupture in the second patient also occurred during a reintervention. His initial hepatic stent placement was performed 24 days after liver transplant. During the reintervention, a high-grade stenosis (99%) was noted in the main hepatic artery just proximal to the previously placed stent. After predilation with a balloon, deployment of a second balloon-expandable stent caused rupture of the hepatic artery into the bile duct, causing significant hemobilia. After unsuccessful balloon tamponade, a coronary covered balloon-expandable (Jomed) stent was placed with complete resolution of extravasation and no residual stenosis. However, the stent had early thrombosis. At the 9-month follow-up, he had not required retransplantation.

Patient 3 was an 11-year-old boy who had undergone his second transplant only 2 days after the first transplant failed secondary to hepatic artery and portal vein thrombosis. After unsuccessful transluminal angioplasty, a PT ChoICE wire was used to cannulate the main to a 180° hairpin turn. Balloon angioplasty treated the stenosis without complication. After placement of a balloon-expandable coronary stent, localized dissection was identified. This was unsuccessfully treated with another balloon-expandable stent. The patient ultimately progressed to HAT and subsequent graft failure, and underwent a successful retransplant 1 month later. This patient died ≤1 year from the second transplant. This was the one of two patients in the series whose major complication was not successfully treated.

Dissections. Patient 4 experienced two hepatic artery dissections. Notably, this patient’s stenosis was 1 cm proximal to a 180° hairpin turn. Balloon angioplasty treated the stenosis without complication. After placement of a balloon-expandable coronary stent, localized dissection was identified. This was unsuccessfully treated with another balloon-expandable stent. The patient ultimately progressed to HAT and subsequent graft failure, and underwent a successful retransplant 1 month later. This patient died ≤1 year from the second transplant. This was the one of two patients in the series whose major complication was not successfully treated.

Patient 5 developed rapidly worsening HAS, with peak systolic velocity increasing from 365 to >600 cm/s in 1 week. In addition, the patient’s stenosis on angiography appeared at a 360° hairpin turn. After a Glidewire was directed into the hepatic artery proximal to the stenosis, a follow-up arteriogram revealed dissection. An Xpert stent (Abbott Vascular, Abbott Park, Ill) was deployed. This successfully treated the dissection; however, the flow of the distal hepatic artery did not improve. The patient progressed to HAT but continued to have a functional graft secondary to collateralization at a follow-up of 55 months.

Patient 6 developed an extensive celiac artery dissection during attempts of wire placement into a very small and tortuous proper hepatic artery. Once the dissection was noted, a PT ChoICE wire was used to cannulate the

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Table I. Characteristics of all patients in whom a major complication occurred during endovascular treatment of hepatic artery stenosis (HAS)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, years/sex</th>
<th>Re-OLTx</th>
<th>Time to Inv after OLTx (days)</th>
<th>Re-Intv</th>
<th>Intv</th>
<th>Tortuosity</th>
<th>Complication</th>
<th>Complication treated</th>
<th>HAS treated</th>
<th>Follow-up US, months</th>
<th>Clinical outcome</th>
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<tbody>
<tr>
<td>1</td>
<td>64/F</td>
<td>No</td>
<td>424</td>
<td>Yes</td>
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<td>Yes</td>
<td>Rupture</td>
<td>Yes</td>
<td>Yes</td>
<td>56</td>
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</tr>
<tr>
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<td>61</td>
<td>Yes</td>
<td>PTA, stent ×2</td>
<td>No</td>
<td>Rupture</td>
<td>Yes</td>
<td>Yes</td>
<td>9</td>
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</tr>
<tr>
<td>3</td>
<td>11/M</td>
<td>Yes</td>
<td>32</td>
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<td>Yes</td>
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<td>Yes</td>
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<td>30/M</td>
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<td>77</td>
<td>No</td>
<td>Stent</td>
<td>Yes</td>
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<td>6</td>
<td>64/F</td>
<td>No</td>
<td>89</td>
<td>No</td>
<td>PTA, stent</td>
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<td>Dissection</td>
<td>Yes</td>
<td>No, aborted</td>
<td>0</td>
<td>Stable HAS, alive</td>
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<tr>
<td>7</td>
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<td>Yes</td>
<td>112</td>
<td>Yes</td>
<td>PTA, stent ×2</td>
<td>Yes</td>
<td>Dissection</td>
<td>Yes</td>
<td>No, aborted</td>
<td>NA</td>
<td>Death secondary to sepsis</td>
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<tr>
<td>8</td>
<td>33/M</td>
<td>Yes</td>
<td>98</td>
<td>No</td>
<td>PTA, stent</td>
<td>Yes</td>
<td>Dissection</td>
<td>No</td>
<td>Yes</td>
<td>11</td>
<td>No HAS, alive</td>
</tr>
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</table>

NA, not applicable; US, ultrasound.

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**Per-protocol set**

- **Participants**: 6 patients
- **Interventions**: 9 stent placements
- **Complications**: 9 stents
- **Deaths**: 2

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true lumen of the artery with a remaining PT ChoICE wire in the false lumen. The dissection was successfully treated with predilation with balloon angioplasty and a coronary balloon-expandable stent. Because of the size (1.5 mm) and severe tortuosity of the hepatic artery, no intervention on the stenosis was attempted. Follow-up ultrasound imaging showed the patient had stable HAS. Patient 7 underwent his second liver transplant 4 months before undergoing endovascular treatment of high-grade stenosis of his right hepatic artery. Notably, the patient was found to have a replaced left hepatic artery and very tortuous take offs of both left and right hepatic arteries on a prior angiogram (Fig 2, A). During his second endovascular intervention, proper hepatic artery dissection occurred during the attempt to cannulate the main hepatic artery (Fig 2, B). A steerable catheter was used to cannulate the true lumen, and angioplasty was performed. Two stents were deployed, with resolution of the dissection (Fig 2, C). The case was aborted after successful treatment of the proximal hepatic artery dissection, and the more distal HAS was not treated.

Patient 8 had undergone a second liver transplant in addition to having a 180° tortuosity of his proper hepatic artery. During the endovascular intervention, there was dissection of the hepatic artery just distal to a bare-metal stent that had just been deployed to successfully treat the high-grade stenosis. Because of the markedly severe tortuosity, a stent could not be delivered to treat the distal dissection. Fortunately, this was not a flow-limiting dissection. At follow-up of 11 months, this patient’s hepatic artery remained patent.

Mortality. At a mean follow-up of 22 months, there were 10 deaths, yielding an overall all-cause mortality rate of 12.6% (10 of 79). The 1-year mortality was 7.6% (6 of 79). The 1-year mortality rate for the complication cohort was 12.5% (1 of 8) compared with 7.0% (5 of 71) in the rest of the cohort ($P = .484$).

DISCUSSION

The diagnosis and management of HAS after liver transplantation remains controversial. Our center has adopted a practice of routine ultrasound surveillance and selective interventions with the aim of preventing late HAT. Our transplant program has an aggressive approach to organ utilization, with more than half of the donor allo grafts imported from outside of our local Organ Procurement Organization after being turned down by other centers. Many of these grafts have potential risk factors for vascular complications, including graft steatosis, donation-after-circulatory death, and advanced age. Despite our aggressive approach to donor utilization, we have not seen a significant increase in the rate of HAT. Much of this success we attribute to early diagnosis
and intervention for HAS after transplant. However, there are no prospective data comparing early intervention for HAS, and practice remains variable between transplant centers in the U.S. In this series, we aimed to clearly define the potential risks associated with early intervention for HAS and describe salvage techniques for endovascular repair.

The 99 endovascular interventions for HAS after liver transplantation is the largest reported experience to date. In this series, we observed a 7.5% rate of major complications. Although 75% of the complications were successfully treated acutely, these patients had a 50% risk of subsequent HAT compared with 1.4% in those patients with no complication during the initial endovascular treatment of HAS. Risk factors for a major complication included severe vessel tortuosity (twofold increased risk) and the presence of a second liver transplant (threefold increased risk). The cause of the increased rate of complications observed in retransplant patients is unclear but may be related to technical challenges in the arterial anastomosis at the time of retransplant, impaired recipient vessel quality from chronic inflammation, or the effects of chronic immunosuppression and steroids. Although vessel tortuosity and retransplants were associated with more complications, the intervention was successful in most of the patients with these risk factors. The data set is robust but is not large enough to make definitive conclusions about withholding intervention.

The location of the HAS was typically in the proper hepatic artery, presumably at the anastomotic site between the donor and recipient vessels. Rarely, stenosis was seen at the terminal proper hepatic artery or proximal right and left hepatic arteries, distal to the anastomotic site. Although speculative, the presumed etiology for the latter distribution of stenosis is due to ischemic injury of the donor vessel.

Other investigators have also found severe vessel tortuosity to be a risk factor for complications associated with endovascular HAS treatment. Saad et al. reported that PTA in patients with arterial kinks/tortuosity associated with HAS experience more complications in addition to tandem and distal stenosis. In patients with kinks/tortuosity, technical success and complication rates were 14% and 29%, respectively, compared with 94% and 10% in patients without such anatomy in their experience. In a retrospective review by Rajakannu et al., recipient age ≥60 years, female sex, preliver transplant, transarterial chemoembolization, redo hepatic artery anastomosis after liver transplant, more than one arterial anastomosis in the graft, and symptomatic HAS at diagnosis were statistically significantly associated with endovascular treatment failure.

The reported risk of procedural complications after endovascular treatment of HAS is quite variable, ranging from 0% to 23% in the literature (Table II). Rostambeigi et al. performed a meta-analysis in 2012 that analyzed 26 studies published between 1970 and 2011 performing interventional treatment of HAS in adult patients after liver transplant. In this pooled cohort of 263 interventions, the reported average periprocedural complication rate was 16%, with no significant difference between balloon...
angioplasty alone and stent placement. These complications included major complications, including immediate HAT, rupture/perforation, pseudoaneurysm formation, dissection, and distal embolization. The authors also included minor complications, such as femoral hematomas, but could not reliably break out the major and minor complications. Our reported complication rate of 7.5% compares favorably with this meta-analysis, but we did not include minor access complications in our analysis.

Some have suggested that there is a learning curve when using endovascular treatment of HAS, with improvement of technical success rate and decreased complication rates with increasing experience. We did not observe a learning curve in this series, perhaps partly because a single experienced operator performed all the interventions. Our current technical success rate of 91% after 99 interventions was actually slightly less than our earlier reported rate of 95% (n = 62). Of note, the earlier report included all early hepatic interventions performed by the operator. These can be technically challenging cases with potential complications, including flow-limiting dissection or rupture. Significant experience with visceral cannulation, 0.014-inch platforms, coronary stents, and bailout techniques are essential. Complications can be minimized by meticulous technique in delivering the working guide into the distal common hepatic artery, use of the least stiff wire that will allow tracking of the needed balloon/stent, and initial conservative choice in balloon diameter.

There is a paucity of literature on the endovascular treatment of ruptures of the hepatic artery after angioplasty. Historically, surgical intervention for hepatic artery ligation and subsequent retransplantation was usually required once a rupture had occurred. A case report by Rajan et al explicitly describes their salvage technique of a hepatic artery rupture. A patient with two previous transplants and an aortic infrarenal jump graft to the transplanted hepatic artery had >70% stenosis of the graft to hepatic artery on arteriography. Cannulation of the graft was performed, and an 18-mm-long uncovered balloon-expandable stent was deployed in the area of the stenosis. Hepatic artery rupture was immediately visualized. Conservative attempts to stop the extravasation with prolonged balloon tamponade failed. The area of rupture was traversed with a wire, and a Jomed coronary covered stent was deployed at the site of rupture, with resolution of the extravasation. In a pediatric cohort reported by Boyvat et al., five arterial ruptures occurred, all of which were managed with graft-covered stent placement except one, which required a reoperation.

The Jomed coronary covered stent is typically the only stent option in the U.S. for treatment of a ruptured hepatic artery after angioplasty. Other very small-caliber covered stents may be available worldwide. These stents, which are designed to treat rupture of a coronary bypass graft, are not commercially available in the U.S. and are used off-label on an emergency compassionate-use basis.

<table>
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<tr>
<th>Study</th>
<th>Publication date</th>
<th>Patient, No.</th>
<th>Intv, No.</th>
<th>Initial technical success, %</th>
<th>Complications, %</th>
<th>Re-Intv, No.</th>
<th>Restenosis rate, %</th>
<th>Late HAT, %</th>
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HAT, Hepatic artery thrombosis. Intv, intervention; NA, not available; OLTx, orthotopic liver transplant.
the Viabahn (W. L. Gore & Associates, Flagstaff, Ariz), typically have a diameter that is too large (5 mm is smallest). Other options for treating active extravasation include embolization of the hepatic artery when low-profile covered stents are not available or cannot be delivered to the site of vessel injury.

For arterial dissections, Saad et al.\textsuperscript{18} described treating with postangioplasty therapeutic anticoagulation, surgical revascularization, as well as stent placement to tack the dissection flap if not flow limiting and if technically feasible. One dissection and two arterial ruptures occurred in their study, giving an immediate complication rate of 7% and a delayed complication rate (HAT) of 5%. None of the complications were salvaged endovascularly, and one patient required retransplantation.\textsuperscript{18} In Sabri et al.,\textsuperscript{5} three major complications occurred, including two dissections. Both dissections were stented, with surgical revision required in one patient and resolution of dissection in the other. In Kodama et al.,\textsuperscript{15} complications occurred in 2 out of 30 interventions (6.7%) after living-donor liver transplant, including a hepatic artery dissection that was treated by tacking down the intimal flap with a balloon tamponade technique for 3 minutes and hepatic artery perforation with a guidewire on a third intervention that was treated with prolonged balloon tamponade.\textsuperscript{15}

This study has some limitations. As a single-institution study performed by a single operator, these results may not be generalizable. In addition, we did not have a control group of severe HAS who did not receive intervention and thus cannot comment on improvement from conservative management. However, this approach has been discussed in the literature.

**CONCLUSIONS**

Although endovascular treatment of HAS is safe and effective in most patients, target vessel injury is possible. Severe tortuosity of the hepatic artery or prior retransplantation were associated with a twofold to threefold increased risk of major complications. Acute vessel injury can be managed successfully using endovascular techniques in most cases, but these patients have a significant risk of subsequent HAT and need close surveillance. Advanced endovascular skills and immediate availability of rescue devices are essential for good outcomes.

**AUTHOR CONTRIBUTIONS**

Conception and design: LG, KW, WS, JS, CB, TS, KB
Analysis and interpretation: LG
Data collection: LG, WS, JS
Writing the article: LG, WS
Critical revision of the article: LG, KW, WS, JS, CB, TS, KB
Final approval of the article: LG, KW, WS, JS, CB, TS, KB
Statistical analysis: LG
Obtained funding: Not applicable
Overall responsibility: WS

**REFERENCES**


DISCUSSION

Dr Mitchell H. Goldman (Knoxville, Tenn). Dr Goldsmith and his colleagues from Ochsner Clinic Foundation, one of the nation’s biggest hepatic transplant programs, report their experience with hepatic artery stenosis (HAS), focusing on the complications of the procedures of endovascular intervention. I want to thank Dr Goldsmith for a timely sent copy of his well-written paper.

In fact, these investigators had a laudable 91% success rate relieving greater than 30% stenosis and were able to rescue with adjunctive techniques 50% of their complications with the rest ending in hepatic artery thrombosis. The authors point out that HAS is associated with retransplantation and tortuosity of the vessels.

I have a few questions for Dr Goldsmith:

1. What was your noninvasive screening protocol? In the absence of enzyme changes or evidence of cholestasis was it a protocol driven routine? Also, were all three noninvasive criteria necessary before a computed tomography angiography was ordered or were one or two criteria more important?

2. Where were the majority of the stenoses? Were they at the anastomoses, implicating intimal hyperplasia secondary to intimal injury from suturing, handling, or perfusion cannulas, at a curvature or bifurcation implicating a turbulent phenomenon, or were there technical issues at the time of transplant from twists or excessive lengths of arterial segments?

3. While not exactly the scope of this excellent paper, you still have the largest cohort of HAS patients and I wonder if you noticed a correlation between immunologic match or viral infections and the incidence of HAS? Past President Scott Stevens, in his Presidential Award presentation at this meeting in Acapulco, Mexico in 1990, reported that in an animal model, histocompatibility match played a role in venous graft patency.1 Were there more mismatches, episodes of rejection or CMV infections in the HAS patients versus the rest of the transplant population?

I commend the authors for a well written, well thought out paper. It is a shame that we really truly don’t know what would happen to the HAS patients if they went untreated because these authors point out that intervention is not without, albeit small, consequence.

Dr Leighton E. Goldsmith. To answer your questions:

1. In terms of the noninvasive protocol, our transplant group aggressively uses ultrasound in the perioperative period to watch for vascular abnormalities. A hepatic artery ultrasound is performed intraoperatively and then again on the first postoperative day. Another ultrasound is performed between 2 to 4 weeks post-transplant and when any changes in clinical status prompts ultrasound. Your question about the HAS criteria — the answer is yes. Generally speaking, we look for all three criteria. This is present in >90% of cases in which we perform an arteriogram.

2. To answer your question about where the majority of the stenoses occur, the majority of the stenoses are in multiple locations, most commonly at the proper hepatic artery (86%) at the anastomosis site and in areas of kinks where there is major redundancy (also in the proper hepatic artery). Occasionally, we see high-grade stenosis or occlusions in the proximal right and left hepatic arteries. I think this accounted for about 10% of our HAS. The etiology at the hepatic confluence is most likely ischemia-related, whereas the anastomotic stenoses are usually intimal hyperplasia or technical issues.

3. In relation to finding a correlation between immunologic match or viral infections and HAS, that’s not something we looked for. That’s a great thought, but I don’t have that data; however, that would be a great area of investigation and one that our transplants surgeons are very interested in.

REFERENCE