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Review Article

Diagnosis and correction of hepatic vena caval obstruction following liver transplantation

Jeffrey Campsen[1,4], Charles Ray[2], Michael Zimmerman[1], M Susan Mandell[3], Maria Kaplan[1], Igal Kam[1]

[1]Division of Transplant Surgery, [2]Division of Interventional Radiology, [3]University of Colorado at Denver, Division of Anesthesia Surgery, Aurora CO, [4]University of Utah, Department of Surgery, Salt Lake City, UT.

INTRODUCTION

Clinically significant inferior vena caval (IVC) obstruction is a possible complication of malpositioning of the liver graft or of suprahepatic vena caval narrowing secondary to an anastomotic strictures. Outflow obstructions have been reported in the immediate postoperative period following vascular reconstruction of the whole cadaveric liver allografts using either the standard vena cava transposition or the piggy-back technique. The same complication has also been documented using both live donor and split liver grafts.[1–4] The resulting narrowing of the IVC causes a pressure gradient across the hepatic cava resulting in venous hypertension (HTN) below the liver affecting the kidneys and distal extremities. The severity of the gradient is dependent upon the degree of narrowing but leads to clinical symptoms of massive congestion of the lower body and less often parenchymal hepatic venous congestion with impaired graft function. The diagnosis of vena caval narrowing can be delayed because the clinical presentation is easily confused with the volume overload early post-orthotopic liver transplant (OLT).

This technical complication is difficult to diagnose. Liver function tests are usually normal or slightly elevated with adequate synthetic function because the hepatic vein outflow is not significantly compromised by the hepatic caval compression. Further, ultrasonography (U/S) has not been a reliable diagnostic technique, even when there is clinical suspicion of vena caval narrowing.[5] A confirmed diagnosis is necessary because the surgical treatment options are associated with a significant risk of patient morbidity.[6] In order to reduce the operative risk associated with vena caval repair, investigators have recently developed non-surgical options including balloon dilation and/or stenting of the IVC.[7,8]

This report describes the early clinical and radiographic diagnosis of IVC obstruction in a small group of liver transplant (LT) recipients. Further, we explore the clinical outcome following percutaneous stenting. Our data suggest that an early diagnosis and radiologic intervention provides a minimally invasive alternative to achieve symptomatic relief from clinical symptoms. Herein, we will show how to diagnosis and treat this serious problem.

RESULTS

The study time period included 266 patients that received a LT of which four (1.5%) were treated with a vena caval stent. The average age of the study group was 53.3 (46–59 years) and there were an equal number of males and females. The average model for end-stage liver disease (MELD) score was 30.5 (25–40). Two patients had disease due to combined hepatitis C and alcohol abuse. One patient had non-alcoholic steatohepatitis while the remaining patient had a diagnosis of cryptogenic cirrhosis. The remaining patient demographics are listed in Table 1. An U/S was only diagnostic in two of the four patients. The venogram showed a significant gradient (>8 mmHg) with obvious narrowing of the hepatic cava in all four patients (Table 2).

All patients underwent metal Z stent placement, and the average gradient pre- and post-stent deployment fell from 12 mmHg to 2.5 mmHg. The average pressure gradient was reduced by nearly 80%. Technical success was achieved in all four patients. All patients demonstrated significant improvement in clinical symptoms of ascites, leg edema, and glomerular filtration rate (GFR). No patient in this series experienced stent migration or hemorrhage, and stent patency was easily demonstrated by follow-up venogram.

Correspondence: *Dr. Jeffrey Campsen, email: Jeffrey.Campsen@hsc.utah.edu*
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Table 1 Patient demographics

Patient	*Gender*	*Age (yr)*	*MELD*	*BMI (kg/m^2)*	*Piggy-back*	*Disease*	*POD at venogram*	*Follow-up (days)*
1	Female	54	26	27	y	HCV/ETOH	58	870
2	Male	59	31	41	n	NASH	26	360
3	Male	46	40	35	n	HCV/ETOH	62	330
4	Female	54	25	24	y	Cryptogenic	17	280

BMI, body mass index; HCV, hepatitis C virus; MELD, model for end-stage liver disease; NASH, non-alcoholic steatohepatitis; POD, postoperative day. Postoperative venograms were performed to identify the patency and position of the stent.

Table 2 Pre/post-stent changes in clinical variables

	Pre-stenting	*Post-stenting*	*Percent change*
Massive ascites	Yes—100%	No—0%	–
Lower extremity edema	Yes—100%	No—0%	–
Average GFR (range) (mL/min/1.73 m^2)	64.2 (44–101)	88.9 (79–133)	+38.5(%)
Average pressure gradient (range) (mmHg)	12 (8–15)	2.5 (0–5)	–79.2(%)

GFR, glomerular filtration rate.

Similarly, no patient in this cohort required paracentesis following the intervention and all grafts survived for the follow-up period. We also found that the calculated GFR improved by nearly 40%.

DISCUSSION

Hepatic IVC obstruction in the early postoperative period after LT is a rare but serious technical complication that is difficult to diagnosis and to treat. There are recent reports of venous outflow obstruction in living donor liver transplant (LDLT).[1,4] We have not seen this in the LDLT population at our center[9]; however, we have identified four patients with hepatic IVC narrowing in adult whole LT both with piggy-back and with non-piggy-back techniques. Our data probably underestimate the incidence because we only identified affected patients by the severity of their postoperative symptoms. It is possible that there are additional patients who have milder symptoms but still have non-clinical venous HTN. Our study population represents those patients who have overt signs of venous HTN due to caval narrowing.

The previous reports in the radiologic literature demonstrate outflow obstruction correction following the placement of endovascular stents.[1,9] Often, the postoperative course after LT is complicated by a number of clinical problems. This may make it difficult to identify signs and symptoms suggestive of IVC compromise. Further, before transplant many patients have ascites that may continue after the transplant. After a thorough review of our recent patients, we discovered a cluster of clinical signs and symptoms that are suspicious for hepatic IVC obstruction after LT. A clinical picture of normal liver function by laboratory values without rejection, massive ascites (increased from the pre-operative period), lower extremity and/or truncal edema, and new onset renal insufficiency can be predictive. Our data suggest that if patients have the cluster of clinical signs and symptoms described above that do not improve with diuretics, an U/S should be obtained and then followed by a venogram. Ultrasonography is not sensitive for caval stenosis but can rule out other vascular complications.

At the time of venography, the interventional radiologist should evaluate the images for signs of stenosis of the vena cava (Figure 1A). However, the pressure gradient across the hepatic cava is diagnostic. Pressure measurements from the right atrium and the hepatic veins can be obtained if the transplant was a piggy-back. If a piggy-back technique was not used, the pressure measurements from the right atrium and the infrahepatic cava should be compared. Our data suggest that radiographic evidence of a hepatic IVC narrowing coupled with a gradient across the anastomosis of 8 mmHg or greater is sufficient evidence to establish a diagnosis of IVC obstruction. We hypothesize that this narrowing in the face of relatively normal liver function is from malpositioning of the graft or the weight of the graft on the hepatic IVC. However, if the liver has dysfunction, then a hepatic vein outflow obstruction should be suspected at the suprahepatic anastomosis.

Placement of the Gianturco Z stent in our series successfully relieved the narrowing in all patients (Figure 1B–D). Immediately after stent placement, there should be a reduction in gradients between the caval and the right atrial pressure. Successful reduction in the venous gradient improved clinical symptoms in all of our patients within 24–48 hours.