



Hepatic Venous Stenosis in Partial Liver Graft Transplantation Detected by Color Doppler Ultrasound Before and After Radiological Interventional Management

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ABSTRACT

Hepatic outflow insufficiency remains one of the major complications causing postoperative graft failure especially among partial liver graft transplantations (PLT) including living donor liver transplantation (LDLT), reduced size liver transplantation (RLT), and split liver transplantation (SLT). These procedures are different from the whole liver graft transplantations (OLT), which include multiple vascular anastomoses. Color Doppler ultrasound (CDUS) was used to evaluate the hepatic venous outflow from grafts before and after radiological interventional management and to document treatment effects. From June 1994 to March 2003, our 136 cases of PLTs included 131 LDLTs, two RLTs, and three SLTs. Seven cases (six children and one adult) showed postoperative hepatic vein outflow obstruction and persistent massive ascites, as detected by color Doppler ultrasound (CDUS) and confirmed by interventional angiography. The CDUS showed a monophasic flat waveform with a relatively low hepatic vein average peak velocity (Va) in all cases (mean 11 cm/s). Successful interventional procedures included balloon dilatation in three cases and metallic stent replacement in four cases. CDUS was used with guidance during the procedure to confirm restoration of normal hepatic vein flow with a multiphasic waveform and an objective increase of average flow velocity (high to average 66 cm/s). Ascites disappeared dramatically after the procedure. In conclusion CDUS is the prime modality to diagnose and document a treatment response.

PARTIAL LIVER GRAFT TRANSPLANTATIONS (PLT), including living donor (LDLT), reduced size (RLT), and split liver transplantation (SLT), are different from whole liver graft transplantation (OLT) with multiple major vascular anastomoses. Especially in the LDLT, intact hepatic inflow and outflow are necessary for normal liver function in both the graft and the remnant liver. Hepatic outflow insufficiency remains a major complication producing early graft failure (5.4% of our cases).¹⁻³ We used color doppler ultrasound (CDUS) to evaluate hepatic venous outflow insufficiency in liver grafts before and after radiological interventional by balloon dilatation and/or stent placement, as well as to document the treatment response.^{4,5}

MATERIALS AND METHODS

From June of 1994 to March of 2003, 136 cases of partial liver graft transplantation were performed in 90 pediatric and 46 adult recipients using either the left lateral lobe or the right lobe of the liver, including 131 cases of LDLTs, two cases of RLTs, and three

cases of SLTs. We used CDUS routinely to document normal graft inflow and outflow intraoperatively just after graft reperfusion and during the early postoperative days. In this study, CDUS was also performed to assess hepatic vein (HV) flow changes that required radiological interventions before or after treatment. We collected the data of HV average flow velocity (Va) and flow waveform for further analysis and correlation with clinical events.

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RESULTS

Among the 136 cases, 102 patients had a single HV anastomosis, including 81 left HVs (LHV) and 21 right HVs (RHV); 27 cases with double anastomoses including four cases of LHV + middle HV (MHV), eight cases of RHV + MHV, four cases of RHV + RIHV, and six cases of RHV + V5 or RHV + V8. There were seven cases with triple anastomoses or more, including four cases of MHV + RHV + RIHV, two cases of RHV + RIHV + V5, and one case of RHV + RIHV + V8 + LHV (dual grafts from two living donors). A total of 178 HV anastomoses were performed in these 136 cases. The waveforms were recorded as 30 monophasic, 116 biphasic, and 32 triphasic patterns, respectively. A high HV Va of 17 to 123 cm/s (mean 44.1) was noted in 111 cases with biphasic or triphasic patterns. Lower HV Va was noted among the 30 HVs monophasic waveforms in 25 cases, including 18 cases without significant complicated ascites with better HV Va fell in the range of 14 to 82 cm/s (mean 25); the other seven cases (including six children and one adult) with persistent massive ascites for more than 1 month had the worst HV Va of 2 to 18 cm/s (mean 11 cm/s).

Interventional angiography was performed in these seven cases to confirm the diagnosis of HV stenosis. Subsequent interventional management included three cases of balloon dilatation and four cases of metallic stent placement (Wallstent). CDUS studies were performed during the procedure to guide confirmation of restoration to a normal range. All returned to a normal biphasic or triphasic waveform with restitution of Va to 27 to 160 cm/s (mean = 66 cm/s). Ascites disappeared immediately and dramatically after the intervention. Two cases who first underwent balloon dilatation had recurrent HV stenosis with massive ascites. The lesions were again detected by CDUS by abnormally low Va and monophasic waveforms. These patients were further treated with Wallstent placements; there was no recalcitrant ascites thereafter.

DISCUSSION

Among 136 cases of PLTs, there were seven cases of postoperative HV outflow obstruction with clinically complicated, massive ascites. CDUS findings supported the diagnosis and correlated with angiography. Interventional procedures with balloon dilation in all seven cases were successful in three cases. Two of the remaining cases had recurrent HV stenosis requiring further metallic stent insertion, all of which were successful. Metallic stent insertion seems to be a better rescue procedure.⁶⁻⁹ The normal hepatic vein (HV) outflow should show average flow velocities (Va) > 10 cm/s with either biphasic or triphasic waveforms. Most of the hepatic venous outflow obstruction cases displayed flat waveforms and low flow velocity (Va < 10 cm/s) in the hepatic vein as previously reported by other authors.^{10,11} The hepatic vein waveforms usually reflect the pressure changes in the right atrium causing pulsatile biphasic or triphasic waveforms. In the presence of HV outflow insufficiency, cardiac pulses cannot be transmitted

to the hepatic vein, and only monophasic waveforms are seen using CDUS.¹² The velocity of flow in the hepatic vein may be affected by a number of parameters, mainly due to the patency of the outflow tract and the amount of total inflow. For 18 of 25 cases with monophasic HV waveforms with relative high Va of 14 to 82 cm/s (mean 25) without complicated clinical symptoms, some degree of HV outflow insufficiency occurred but was not sufficient to cause severe complications. For the other seven cases with prolonged massive ascites and proven HV stenosis, monophasic waveforms were accompanied by relatively low Va around 2 to 18 cm/s (mean 11). Their mean portal velocities were around 8 to 21 cm/s (mean 13).

The finding of abnormal monophasic waveforms was compatible with reported data from other authors. But the average velocities in HVs and their corresponding mean portal velocities were slightly higher (some up to 18 cm/s), so we believe that the criteria for the normal HV average velocity should be at least 20 cm/s and that decreased portal flow may also be observed. Some cases with acute HV thrombosis and almost complete outflow obstruction may develop reversed portal flow.⁷ Among our patients, 25 cases with monophasic waveforms but different HV Va showed different degrees of complicated clinical symptoms. The Va of HVs in the seven complicated cases were 2 to 18 cm/s (mean = 11) and that of the noncomplicated 18 cases, 14 to 82 cm/s (mean = 25). This result is higher than previous CDUS criteria. We believe that the reason is a difference in the severity of outflow obstruction. In our study, if the CDUS findings showed monophasic HV waveform changes and decreased Va below 20 cm/s, it was significant for some degree of HV outflow insufficiency. These cases with progressively worse HV outflow and persistent massive ascites (5.4% in 7 of 136 cases) were all successfully rescued by interventional angiography, which is the choice for management. CDUS provides a fast, easy, and objective method to ensure normal hepatic outflow dynamics before and after an interventional procedure.

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