ORIGINAL ARTICLE

Early Posttransplant Hepatic Venous Outflow Obstruction: Long-Term Efficacy of Primary Stent Placement

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Although balloon angioplasty has been accepted as the safe and effective initial treatment to manage hepatic venous outflow abnormalities, it may induce rupture of the fresh anastomosis but also may be ineffective to eliminate various etiologies of venous outflow abnormalities in the early post-transplant period. Therefore, we performed primary stent placement in 108 patients to treat early-onset (≤4 weeks) post-transplant hepatic venous outflow abnormality. The following parameters were documented retrospectively: technical success and complications: clinical success; recurrence; and patency of stent-inserted hepatic veins.

Technical success was achieved in 166 (97.6%) of 170 anastomoses (107 patients). Major complications occurred in 5 (4.6%) patients: partial stent migration (n = 2) and stent malposition (n = 3). Clinical success was achieved in 83 (82.2%) of 101 patients who had abnormal liver enzymes or clinical symptoms. Seven patients without initial clinical symptoms have remained healthy. Restenosis or occlusion of the stent-inserted hepatic veins was documented in 22 patients at a mean of 9.6 ± 8.6 months after stent placement. Four of them underwent stent replacement or retransplantation due to liver function deterioration. Overall 1-, 3-, and 5-year primary patency rates were 82.3 ± 0.3%, 75.0 ± 0.4%, and 72.4 ± 0.5%, respectively. Multivariate Cox regression analysis showed that diameter of stents was an independent factor associated with patency of stents (p = 0.001).

Primary stent placement seems to be an effective treatment modality with an acceptable long-term patency to treat early post-transplant hepatic venous outflow obstruction. ***Liver Transpl 14:1505-1511, 2008.*** **© 2008 AASLD.**

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Living donor liver transplantation (LDLT) is now accepted as one of the therapeutic options for overcoming graft shortage in adult patients waiting for liver transplantation. In LDLT, however, there are several technical difficulties due to multiple anastomoses, including hepatic venous anastomoses, and the limited volume of the donor liver segments.

Patency of the hepatic venous outflow may be crucial to graft survival, especially in the early posttransplant period.[1,2] Balloon angioplasty has been accepted as a safe and effective initial treatment for managing hepatic venous outflow abnormalities following liver transplantation.[3-5] However, balloon angioplasty may induce rupture of the fresh anastomosis and also may be ineffective in eliminating various etiologies of venous outflow abnormalities in the early posttransplant period.[6] Stents have usually been used to treat elastic or recurrent stenosis following balloon angioplasty,[7-10] and there have been a few reports dealing with the long-term (>1 year) patency of stent-inserted hepatic veins in LDLT.[7,11-13] We believe that primary stent placement is the treatment of choice to manage early (≤4 weeks) posttransplant hepatic venous outflow abnormalities.

In this article, we describe the efficacy and long-term patency of primary hepatic venous stent placement following LDLT.

Abbreviations: CT, computed tomography; HBV, hepatitis B virus infection; HCC, hepatocellular carcinoma; HCV, hepatitis C virus infection; LDLT, living donor liver transplantation; MjHV, major hepatic vein; SHV, short hepatic vein; US, ultrasonography.

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TABLE 1. Demographic Data of 108 Patients

Item	Number of Patients
Males versus females	78:30
Age	3-67 years (mean, 48 ± 11)
Underlying diseases	
Liver cirrhosis (HBV)	66 (61.1%)
HCC (HBV or HCV)	21 (19.4%)
Fulminant hepatitis	8 (7.4%)
Liver cirrhosis (HCV)	7 (6.5%)
Others*	6 (5.6%)
Graft lobes	
Right lobe	75 (69.4%)
Left lobe	2 (1.9%)
Dual left lobes	29 (26.9%)
Left lateral segment	2 (1.9%)
Interval between LDLT and stent placement	0-28 days (mean, 6.4 ± 7.1)
Main symptom or sign	
Abnormal liver enzymes	90 (83.3%)
Ascites	9 (8.3%)
Effusion	2 (1.9%)
None†	7 (6.5%)
Treated hepatic veins (n = 166)	
Major‡	77 (46.4%)
Minor§	89 (53.6%)

Abbreviations: HBV, hepatitis B virus infection; HCC, hepatocellular carcinoma; HCV, hepatitis C virus infection; LDLT, living donor liver transplantation.
*These include biliary cirrhosis (n = 3) and biliary atresia (n = 3).
†These patients had a monophasic waveform in major hepatic veins on Doppler ultrasonography and a low attenuation area on computed tomography.
‡These include the right and left hepatic veins.
§These include the segmental hepatic veins and the right inferior hepatic vein.

PATIENTS AND METHODS

Patient Population

From January 2000 to October 2006, 108 (9.5%) of 1135 patients who had undergone LDLT underwent percutaneous primary stent placement for treating hepatic venous outflow abnormalities. Three patients were younger than 15 years of age. The demographic data of these patients are summarized in Table 1.

In general, major hepatic veins (MjHVs; the right or left hepatic veins) of the graft were anastomosed with those of the recipient in an end-to-end fashion. Also, major short hepatic veins (SHVs; the right inferior hepatic vein and the middle hepatic vein tributaries that drain the right liver; ≥5 mm in diameter) were anastomosed to the recipient's inferior vena cava with various methods such as end-to-end interposition of the recipient's portal bifurcation or an interposed autologous or cadaveric vein or artery graft between the SHV and the recipient's middle hepatic vein.

A hepatic venous outflow abnormality was diagnosed when Doppler ultrasonography (US) showed a persistent monophasic waveform or slow flow of less than 10 cm/second or computed tomography (CT) showed nonopacified hepatic veins, focal luminal narrowing greater than 50% of the adjacent normal hepatic venous diameter, or a geographic low attenuation area in the liver in the clinical setting of a hepatic venous outflow abnormality. A hepatic venous outflow abnormality was confirmed by hepatic venography, and we defined significant hepatic venous outflow stenosis when the venography revealed stasis of the contrast medium due to anastomotic occlusion or a pressure gradient across an anastomosis was more than 5 mm Hg.

Stent Placement

Written informed consent was obtained from each patient or each patient's legal guardian. Our institutional review board approved this study protocol.

Selective hepatic venography and stent placement were performed from the right internal jugular vein (n = 99), right common femoral vein (n = 5), both the internal jugular and femoral veins (n = 3), and both the internal jugular vein and transhepatic (n = 1). A 0.035-inch hydrophilic guide wire (Terumo, Tokyo, Japan) and a 5F cobra catheter (Cook, Bloomington, IN) were used to select each hepatic vein. The pressure gradient across the anastomosis was obtained in most patients, except those with anastomotic occlusion or those who had stenosis that was very difficult to negotiate. Primary stent placement was then performed with a Wallstent (Boston Scientific, Galway, Ireland) or a Zilver stent (Cook, Bloomington, IN). The stents were 6 to 14 mm in diameter and 2.8

to 8.0 cm long. The stent diameter for each case was intentionally oversized by approximately 1 to 2 mm with respect to the measured normal hepatic vein diameter adjacent to the stenosis. In 2 cases, balloon angioplasty following stent placement was performed because of underexpanded stents that were less than 50% of their normal diameter. Following stent placement, a repeat hepatic venogram and the pressure gradient were obtained. Anticoagulants were not administered in all patients during or following the procedure.

Follow-Up and Definitions

Patient follow-up included medical records, clinical and laboratory data, and imaging findings. Doppler US was routinely performed on posttransplant days 0, 1, 2, 3, and 7 and then weekly until the patient was discharged. CT was also routinely performed at 7- to 10-day intervals until the patient was discharged. Thereafter, CT and Doppler US were randomly performed 1, 6, and 12 months after discharge. Patient symptoms and biochemical data, including serum aspartate aminotransferase, alanine aminotransferase, and bilirubin, were routinely assessed on a daily basis until the patient was discharged and then were randomly assessed every 8 to 12 weeks at the liver transplant outpatient clinic.

The following parameters were documented retrospectively: technical success and complications, clinical success, recurrence, and patency of stent-inserted hepatic veins. Technical success was defined as fluent hepatic venous outflow with a postprocedural pressure gradient of less than 6 mm Hg. However, when the pressure gradient was not measured, a fluent hepatic venous outflow on hepatic venography and a biphasic or triphasic waveform on follow-up Doppler US were considered to constitute technical success. Clinical success was defined as amelioration of presenting signs/symptoms and recovery of liver function with the disappearance of a geographic low attenuation area in the liver on follow-up CT. Recurrence was defined as a relapse of clinical signs/symptoms or liver function deterioration associated with a hepatic venous outflow abnormality. Patency was defined as biphasic or triphasic waveform on follow-up Doppler US or well-enhancing stent-inserted hepatic veins without stenosis greater than 50% of the stent diameter on follow-up CT. Complications were classified as major and minor according to the guidelines of the Society of Interventional Radiology Standards of Practice Committee.[14] Major complications were defined as those necessitating major therapy, those necessitating an unplanned increase in the level of care or prolonged hospitalization (>48 hours), and those resulting in permanent adverse sequelae or death. Minor complications were defined as those requiring no therapy or nominal therapy, including overnight admission for observation only.

Statistical Analysis

The paired *t* test was performed to analyze the difference between prestenting and poststenting pressure gradients. The Kaplan-Meier method was used to analyze the cumulative patency rate of stent-inserted hepatic veins. In the calculation of the stent patency period, patients were censored if cessation of stent patency did not occur during a patient's life. Multivariate Cox proportional hazards modeling was planned to find factors related to the patency of stents. The following factors were included in this model: age, sex, types of placed stents, location of stent-placed hepatic veins, interval between LDLT and stent placement, and stent diameter. Variables were selected in a stepwise forward selection manner, with entry and retention set with a *P* value of 0.05 considered to indicate a significant difference. A variable's risk was expressed as a hazard ratio with a corresponding 95% confidence interval. All analysis was conducted with SPSS software (version 14.0, SPSS, Chicago, IL).

RESULTS

In all, stent placement was achieved successfully in 166 anastomoses (2 anastomoses in 28 patients, 3 anastomoses in 10 patients, 4 anastomoses in 1 patient, and 5 anastomoses in 2 patients) in 107 patients. In 1 patient who suffered from delayed recovery of liver function, negotiation of an SHV failed because of occlusion. In another 3 patients, stent placement was successful in 1 SHV but failed in the other SHV because of acute angulation. Thus, technical success was achieved in 166 (97.6%) of 170 anastomoses (Figs. 1 and 2).

The pressure gradient was measured in 125 anastomoses. Preprocedural and postprocedural pressure gradients across stenoses were 9.7 ± 4.1 mm Hg (range, 3-27) and 1.4 ± 1.3 mm Hg (range, 0-5), respectively ($P < 0.001$). The preprocedural pressure gradient was less than 6 mm Hg in 5 patients; however, these patients also underwent stent placement prophylactically on postoperative day 1 or 2, respectively, because they had abnormal Doppler US findings, a single hepatic venous anastomosis, and unusually elevated liver enzymes.

Major complications occurred in 5 (4.6%) patients: partial stent migration (n = 2) and stent malposition (n = 1) occurred, and they underwent stent replacement within 10 days after the initial stent placement. In the remaining 2 patients who had undergone dual left lobe transplantation, 2 stents were misplaced into the middle and left hepatic veins of a left-sided liver graft, although we initially planned to place 2 stents into right-sided and left-sided MjHVs, respectively. Fortunately, these patients' liver function normalized without additional stent placement, although the right-sided graft in 1 patient shrank progressively. As for a minor procedural complication, acute angulation of a stent-inserted hepatic vein at the junction of a stent and the distal hepatic vein occurred in 2 patients (1.9%): The angulation disappeared following a second stent placement to overlap the first stent and the angulation.

Initial clinical success was achieved in 83 (82.2%) of 101 patients who had abnormal liver enzymes, ascites, or effusion. Fifteen of 18 patients with clinical failure died of hepatic failure due to acute rejection (n = 6), hepatic arterial ischemia (n = 3), or massive hemor-

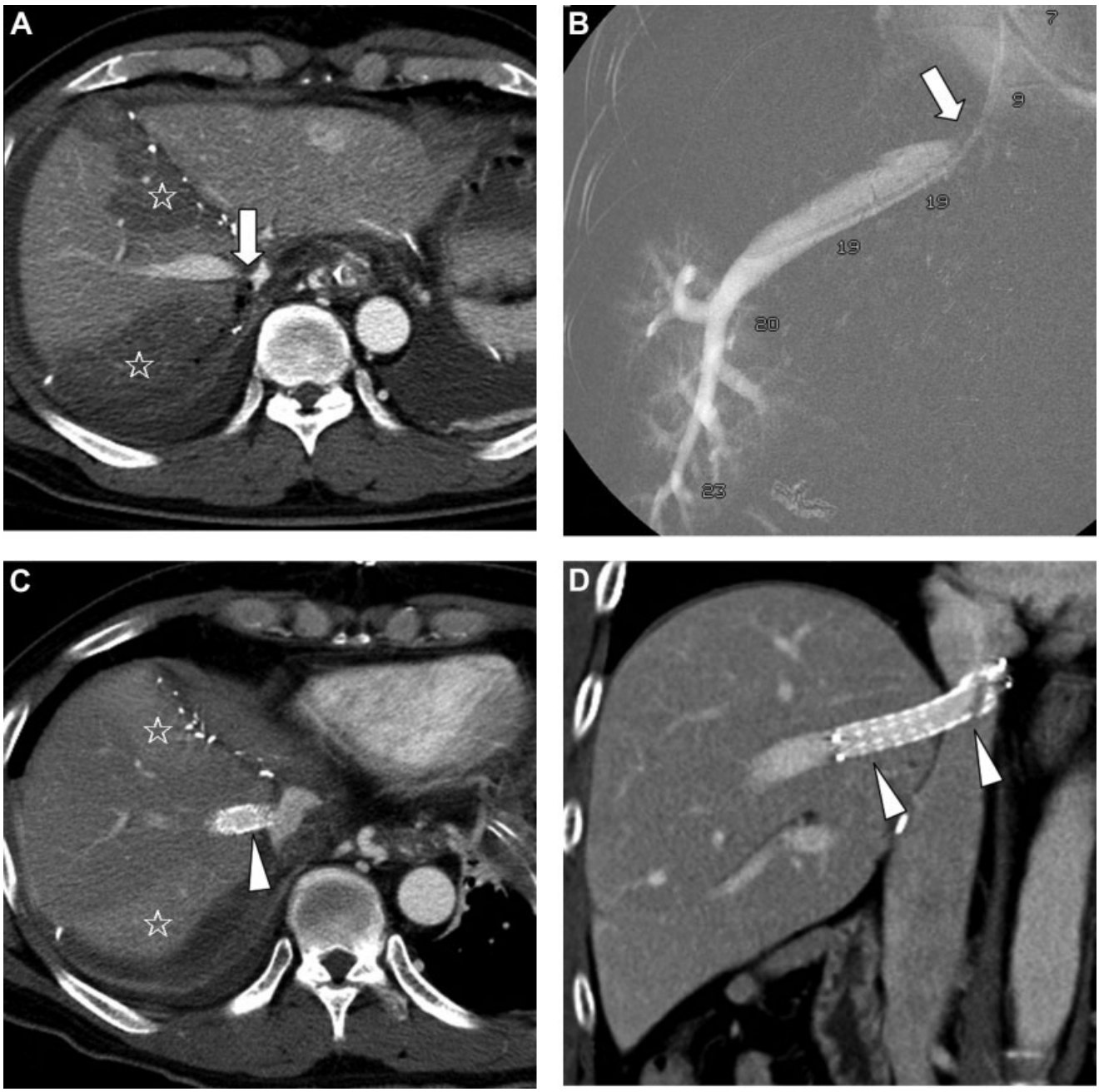

Figure 1. A 51-year-old man underwent dual left lobe living donor liver transplantation. (A) An axial computed tomography image obtained on postoperative day 3 reveals a hepatic venous anastomotic stenosis (arrow) in the right-sided graft. Note the geographic low attenuation areas (stars) in the right-sided graft. (B) A venogram in the right-sided graft reveals an anastomotic stenosis (arrow). The pressure gradient across the stenosis was 10 mm Hg. (C) Axial computed tomography obtained 5 days after stent placement shows a well-expanded stent (arrowhead). Note that the geographic low attenuation areas (stars) in the liver have disappeared. (D) A coronal reformatting computed tomography image obtained 41 months after stent placement still reveals the patent stent (arrowhead).

rhagic necrosis (n = 2), of sepsis (n = 3), or of intracerebral hemorrhage (n = 1) without recurrence of hepatic venous outflow obstruction. The remaining 3 patients died of hepatic failure following retransplantation due to primary hepatic failure (n = 2) or hemorrhagic necrosis with stent-inserted MjHV thrombosis (n = 1). Seven patients who had only abnormal Doppler US or CT findings of hepatic veins remained healthy at the time this article was completed.

Of the 83 patients with initial clinical success, 7 patients died of hepatic failure due to acute or chronic rejection (n = 3), recurrent hepatocellular carcinoma (n = 2), or reactivated hepatitis (n = 1) or of cerebrovascular accident (n = 1) 13.7 ± 8.3 (range, 5.0-28.3) months after stent placement. The remaining 76 patients were still alive 39.1 ± 22.3 (range, 13.0-90.7) months following stent placement.

The cumulative patency rates of the 166 stent-in-

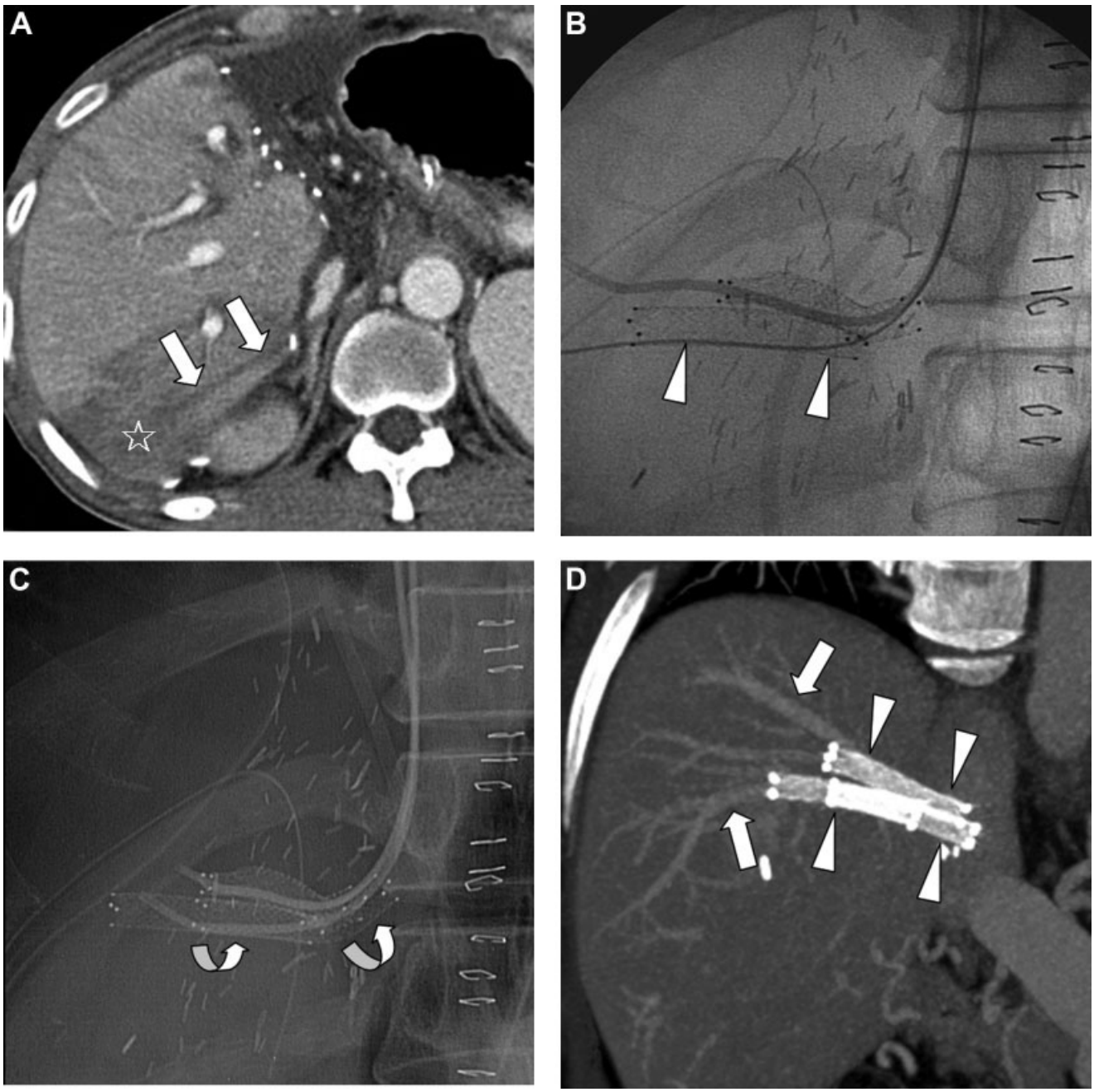

Figure 2. A 49-year-old man underwent right lobe transplantation. This patient had 1 right hepatic vein and 2 inferior hepatic vein anastomoses. (A) Computed tomography obtained on postoperative day 3 shows the nonopacified inferior hepatic vein (arrows) and geographic low attenuation area (star) in the liver. (B) Two 6-mm-diameter stents were placed in the upper and lower inferior hepatic veins, respectively. Note the peripherally misplaced stent in the lower inferior hepatic vein (arrowheads). (C) Another stent (curved arrows) was replaced in the lower inferior hepatic vein. (D) Oblique coronal reformatting computed tomography obtained 22 months after stent placement reveals the patent stents (arrowheads) with good contrast enhancement in the inferior hepatic veins (arrows).

serted hepatic veins are shown in Fig. 3. The overall 1-, 3-, and 5-year patency rates were 82.3% ± 0.3%, 75.0% ± 0.4%, and 72.4% ± 0.5%, respectively. Multivariate Cox regression analysis showed that the diameter of stents was an independent factor associated with the patency of stents ($P = 0.001$; hazard ratio, 0.668; 95% confidence interval, 0.531-0.841). Actually, 27 MjHVs into which stents 12 mm or larger in diameter were placed showed patent lumen in all cases during our follow-up period. However, 10 (32.6%) of 31 SHVs with 6-mm-diameter stents and 11 (26.8%) of 41 SHVs with 8-mm-diameter stents revealed occlusion during the follow-up period. Patency rates according to the diameter of stents are shown in Fig. 4.

In all, stenosis or occlusion of the stent-inserted hepatic veins was documented in 22 patients (31 hepatic

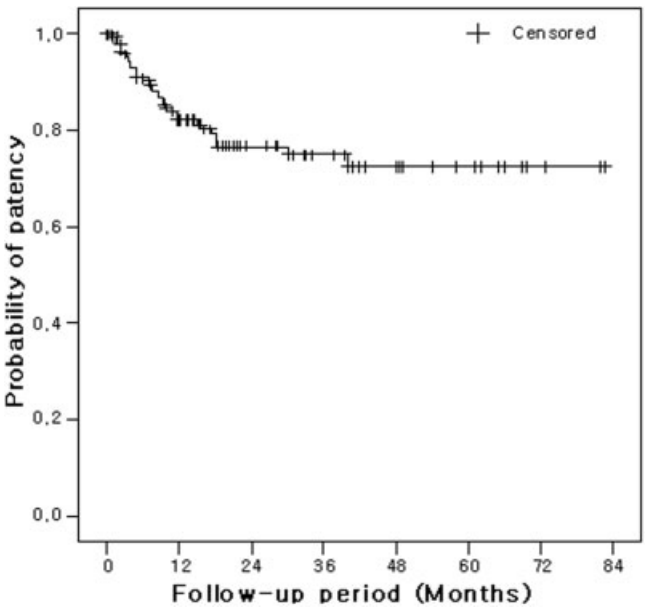

Figure 3. Cumulative patency of 166 stent-inserted hepatic veins.

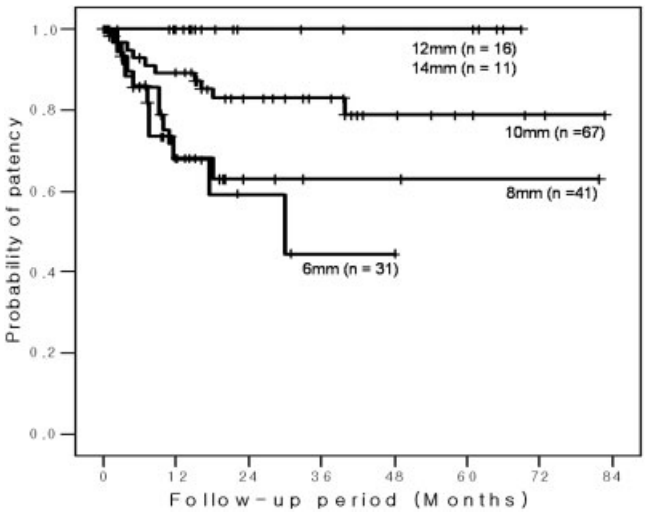

Figure 4. Patency of hepatic veins according to the diameter of the stents.

veins; 6 MjHVs and 25 SHVs) at a mean of 9.6 ± 8.6 (range, 0.8-40.0) months after stent placement. Four of 6 patients with stent-inserted MjHV restenosis or occlusion underwent stent replacement (n = 2) or retransplantation (n = 2) due to liver function deterioration. However, 2 patients with MjHV occlusion and the remaining 18 patients with SHV occlusion did not undergo any management because of normal liver function without clinical symptoms.

DISCUSSION

Although hepatic venous outflow occlusion following orthotopic liver transplantation is rare, it is not infrequent after LDLT because of the necessity of multiple hepatic venous anastomoses. Reported incidences of hepatic venous outflow occlusion following LDLT range from 3.9% to 16.6%.[3,15-17] In LDLT recipients, early posttransplant hepatic venous occlusion may induce liver function deterioration, graft failure, or death because of the graft's size insufficiency.[1,18] Thus, early adequate treatment of hepatic venous outflow occlusion is important for good graft function.

Although stents are susceptible to thrombosis and may interfere with retransplantation, there were several reasons to perform primary stent placement in our study. First, we were concerned about the risk of disruption of a relatively fresh anastomosis during balloon angioplasty because a hepatic venous outflow abnormality was diagnosed in the early posttransplant period (≤ 4 weeks). Second, although the etiology was not clearly defined in each patient, we assumed that kinking of a redundant hepatic vein itself or of an interposed vein graft, venous twisting by displaced liver grafts, extrinsic compression of hepatic veins by the liver graft edema, and regeneration that could not be resolved effectively by balloon angioplasty alone were the major causes of early hepatic venous outflow obstruction because the period was too short for perianastomotic fibrosis or intimal hyperplasia to form. Following primary stent placement in the current study, anastomotic disruption did not occur, and poststenting balloon angioplasty was needed in only 2 patients. Also, retransplantation was possible without major limitations in our 5 patients who had received stent placement. Therefore, we assume that primary stent placement is safe for treating early posttransplant hepatic venous outflow obstruction.

Third, although balloon angioplasty may be effective and safe for treating early posttransplant hepatic venous outflow stenosis, there still is a risk of elastic restenosis. Huang et al.[19] reported that 4 of 7 patients with hepatic venous outflow obstruction needed further stent placement following balloon angioplasty, and Ko et al.[12] also reported that 2 of 5 patients who underwent balloon angioplasty finally needed stent placement or surgical reposition because of recurrence.

There is a controversy about whether such SHVs should be effectively revascularized. Kaneko et al.[20] demonstrated intrahepatic venous collateral formation from the ligated middle hepatic vein tributaries to the right hepatic vein on Doppler US 6 days after right lobe LDLT. However, Sano et al.[21] reported that congestion in donor right livers was not relieved in 7 days in 8 (57%) of 14 patients following ligation of middle hepatic vein tributaries. That is, intrahepatic venous collaterals are often small and do not open up effectively in the early posttransplant period. In addition, Fan et al.[18] reported that middle hepatic vein occlusion was one of the independent significant factors determining hospital mortality following right lobe LDLT. Therefore, several investigators assumed that effective revascularization of SHVs might be necessary to avoid small-for-size graft syndrome because liver graft size in adult LDLT might be insufficient or marginal for the metabolic demands of recipients.[1,22]

We found that patency of SHVs following 6- or 8-mm-diameter stent placement was relatively poor. Interestingly, however, all patients who showed occlusion of these SHVs on follow-up US or CT did not experience recurrent symptoms or liver function change. This may be attributable to delayed development of intrahepatic collaterals to patent MjHVs and active regeneration of the remnant liver. Therefore, we assume that stent placement in SHVs in the early posttransplant period is also beneficial for shortening the restoration period of liver function following LDLT.

In our study, 7 patients who only had abnormal imaging findings in the MjHV also underwent stent placement. Although there may be controversy about placing a stent in these patients, we assumed that there might be a risk of progression of hepatic venous outflow obstruction due to extrinsic compression of hepatic venous anastomosis by rapid regeneration of the liver graft in the early posttransplant period. Lee[22] suggested that the regenerating enlarged liver graft may push on the vena cava and right hepatic vein anastomosis, and the resulting hepatic venous outflow obstruction could congest the liver graft, leading to a vicious cycle of further graft distension and dysfunction in the recipient.

In summary, our retrospective study revealed a high technical success rate and an acceptable clinical success rate of primary stent placement to treat hepatic venous outflow obstruction following LDLT. Although some patients needed repeated intervention because of stent thrombosis and small-diameter stents showed not encouraging long-term patency, most patients with initial clinical success were healthy with patent stents. In conclusion, primary stent placement seems to be an effective treatment modality with an acceptable long-term patency to treat early posttransplant hepatic venous outflow obstruction.

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