Novel use of percutaneous thrombosuction to rescue the early thrombosis of the conduit vein graft after living donor liver transplantation

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ABSTRACT
A 54-year-old woman with liver cirrhosis and hepatocellular carcinoma received a living donor liver transplant. Thrombosis of the segmental hepatic vein occurred on postoperative day 7. We undertook percutaneous catheter thrombosuction under local anesthesia to extract the thrombus successfully without re-exploration. Thrombosuction has been used for thrombosis of the cardiovascular system, limbs, and brain. We first used it in hepatic venous thrombus after liver transplantation. This procedure is simple, less invasive, feasible, safe, repeatable, and effective. (J Vasc Surg Cases and Innovative Techniques 2018;4:204-9.)

Keywords: Liver transplantation; Thrombosis; Hepatic venous outflow; Thrombosuction

It is necessary and important to maintain patency of hepatic venous outflow of the liver graft after liver transplantation to prevent hepatic congestion.1-4 The incidence of hepatic venous outflow obstruction is not low after a living related donor liver transplantation because of the multiple venous anastomoses.1,2,4

With approval of the research ethics review committee of our hospital, we report the first case of treatment of early thrombosis of post-transplantation hepatic vein conduit using percutaneous catheter thrombosuction to extract the thrombus successfully without re-exploration. The patient consented to publication of this report.

CASE REPORT
A 54-year-old woman with hepatitis B viral infection-related liver cirrhosis and two hepatocellular carcinomas (cT2NOM0, stage II; AJCC Cancer Staging Manual, 7th edition) underwent living donor liver transplantation (LDLT) with a right hepatic lobe donated by her daughter. The donor’s liver had no steatosis (density ratio. 119:1:32). The graft was 998 mL and the graft to recipient weight ratio was 1.10%. The remnant liver of the donor was 34.9%. Severe micronodular cirrhotic liver change with a massive amount of ascites (around 1600 mL) was found during exploration of the recipient. The diameter of the venous tributaries V5 and V8 of the middle vein of the donor liver was 6.2 mm and 7.2 mm, respectively (Fig 1). During LDLT, we used a ringed polytetrafluoroethylene (PTFE) graft as a conduit to reconstruct V5, V8, right inferior hepatic vein, and right hepatic vein. A piece of a cryopreserved iliac vein segment was used as a patch for widening the orifice of V5 and V8 to become an artificial wall for the anastomosis with the PTFE graft. Each end-to-side anastomosis for V5, V8, and the inferior hepatic vein was performed with 6-0 Prolene (Fig 2). End-to-end anastomosis between the end opening of the conduit vein graft and the right hepatic vein stump on the inferior vena cava of the recipient was undertaken with 4-0 Prolene continuous suture. End-to-end anastomosis of graft right portal vein stump to recipient right portal vein stump with 5-0 Prolene continuous suture was then undertaken before the release of clamping. An end-to-end anastomosis between graft right hepatic artery stump and recipient right hepatic artery stump with 7-0 Prolene continuous suture was performed. Flow patency was then checked with a flow meter. Finally, end-to-end anastomosis between graft right bile duct stump and recipient right hepatic bile duct stump with 6-0 Prolene interrupted suture was undertaken. The left hepatic bile duct of the recipient was closed. After hemostasis and warm saline irrigation, two 10-mm Jackson-Pratt drains were placed in the right subphrenic space and in the Morrison pouch, respectively. The wound was closed in layers.

The intraoperative course was smooth. The anastomosis time was as follows: right hepatic vein, 10 minutes; right portal vein, 7 minutes; right hepatic artery, 11 minutes; and right bile duct, 40 minutes. The cold ischemia time was 60 minutes.

However, abnormal liver function (ammonia, 88 μmol/L; total bilirubin, 2.3 mg/L; aspartate transaminase, 194 IU/L; alanine transaminase, 405 IU/L; total bilirubin, 2.3 mg/L; aspartate transaminase, 194 IU/L; alanine transaminase, 405 IU/L) occurred from postoperative day 7 (Fig 3). Computed tomography scan showed thrombosis of the orifices of both V5 and V8 onto the conduit vein graft, resulting in
hypoenhancement of the corresponding liver parenchyma (Fig 4, a and b). The hepatic artery and the portal vein remained patent.

To avoid reoperation and repeated general anesthesia, we tried a new strategy using percutaneous catheter thrombosuction to rescue the conduit vein thrombus.

**Procedure of percutaneous catheter thrombosuction.** Heparin 5000 units was given intravenously first. The patient was in the supine position. After local anesthesia, a 7F sheath was inserted through the right femoral vein, then a 7F Judkins guide catheter to approach the hepatic vein through the vascular conduit, wiring to distal V5 branch with 0.014-inch Hydro ST wire (Cook Medical, Bloomington, Ind). Thrombus was then extracted repeatedly by thrombosuction using 7F Pronto V4 (Vascular Solutions, Minneapolis, Minn) smoothly (Fig 4, c). After thrombosuction as clear as possible, follow-up balloon catheter angiography showed a good flow from V5 to the inferior vena cava (Fig 4, d).

The liver function (ammonia, bilirubin, aspartate transaminase, and alanine transaminase) improved soon within 2 days (Fig 3), and the patient was discharged at the end of the third week after transplantation. Oral anticoagulant was given for 3 months. She was well until now, 14 months after operation.

**DISCUSSION**

To establish the hepatic venous outflow of the liver graft, we used the ringed PTFE grafts with a vessel patch to join V5, V8, and inferior hepatic veins. The PTFE graft has been recommended for its high patency rates, although a potential risk of postoperative occlusion of venous tributaries including V5, V8, and inferior hepatic veins exists. The cause of occlusion includes tight suture lines, torsion due to an inappropriate position of the liver graft, kinking, stretching, anastomotic discrepancy, graft regeneration with compression, and intimal fibrosis or hyperplasia around the anastomotic sites.
Gastaca et al\textsuperscript{7} considered graft malposition and kinking of venous anastomosis as the cause of hepatic venous outflow obstruction. Sakamoto et al\textsuperscript{8} attributed the acute hepatic venous outflow obstruction to two possible reasons, including mechanical obstruction of anastomosis stenosis and sinusoidal obstruction syndrome. Shirouzu et al\textsuperscript{9} found that graft regeneration tends to cause distortion and stretching of hepatic veins, especially of the left lobe liver grafts. Kitajima et al\textsuperscript{10} also found that the left lobe graft is an independent risk factor for hepatic venous outflow obstruction. For the graft of this patient, we used the right lobe, not the left lobe. In addition, Arshad et al\textsuperscript{11} hypothesized that a hypercoagulable state in the recipient may contribute to perioperative and early postoperative thrombotic complications. Feltracco et al\textsuperscript{12} reported that underestimated intrinsic hypercoagulation contributes to unpredictable onset of perioperative thrombotic complications. The possible factors that trigger it include inflammation, ischemia-reperfusion injury, vascular clamping, recipient to donor body weight ratio, and graft type. The possible reason for hepatic vein thrombosis of our patient may be multifactorial, related to material of the artificial graft (PTFE), mild degree of malposition, surgical suture problem, or some unavoidable factors. Neocollateralization that develops after a period could compensate for the gradual occlusion. However, early hepatic venous outflow obstruction results in significant morbidity and even graft loss.\textsuperscript{2,3,7,8,13,14} The common nonsurgical treatment options include various endovascular treatments, such as balloon angioplasty or stent placement (or both), percutaneous transluminal venoplasty, and stent placement.\textsuperscript{2-4,8,9,13,15-23} The potential complications of these treatments include failure, malposition, infection, lifelong anticoagulant therapy-related issues, stent migration, and reocclusion.\textsuperscript{2-4,12,15-17,20,21} Sakamoto et al\textsuperscript{13} emphasized that progression of fibrosis could not be prevented if the timing of stenting is too late. The reported surgical treatments include re-exploration to place a breast implant for repositioning, cavoplasty, and retransplantation.\textsuperscript{2,7,8,21,23}

Our thrombosuction of the V\textsubscript{8} thrombus failed because of the sharp angle. However, the patient’s liver function improved after clearance of V\textsubscript{5} thrombus. The liver congestion affected by the V\textsubscript{8} thrombosis was limited, and neocollateralization could compensate for it.

We did not use a stent. The obstruction site affecting the hepatic venous outflow is the orifice where both
Fig 3. The changing trend of liver function in the process. The arrow points to the date of catheter thrombectomy of the vein V5 thrombus. (a) Ammonia; (b) bilirubin; (c) aspartate transaminase (AST); (d) alanine transaminase (ALT).
segment V5 and segment V8 join the vascular conduit. Two problems affect the feasibility of stent placement. First, these obstructions are inside the vascular conduit; the diameter is too small. Second, the angle between the V5 orifice and the vascular conduit is perpendicular (about 90 degrees). To place a stent into such a small orifice with a 90-degree angle is difficult. Re-exploration to open the conduit to remove the thrombosis is another option. However, re-exploration has the risks of repeated general anesthesia, large surgical wound, adhesion after LDLT surgery, dissection difficulty, compliance of the family and patient, and potential of rethrombosis.

Thrombosuction has been used to treat thrombosis of the lower limbs, coronary vessels, hemodialysis fistula, and cerebral sinus or pulmonary embolism. Use of thrombosuction to treat early venous thrombosis after LDLT has not been reported.

The potential superiority of percutaneous thrombosuction to stent placement includes the following. First, placement of a stent has limitations of technical feasibility, such as small diameter or short length of the tributary veins or a sharp angle between them and the hepatic veins. Second, stent placement poses a potential compromise to the other hepatic venous drainage. Third, if the stent occludes, to clean it or to replace it meets with more difficulty. The thrombosuction avoids these limitations.

We recommend percutaneous catheter thrombosuction to treat early venous thrombosis after liver transplantation. This procedure is simple, less invasive, feasible, safe, repeatable, and effective.

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