

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 (cT2N0M0, 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, 1.19-1.32). 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) 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

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Fig 1. a, Computed tomography images of the donor's liver demonstrate the right hepatic vein and V_5 tributary of middle hepatic vein. **b**, Computed tomography images of the donor's liver demonstrate the right hepatic vein and V_8 tributary of middle hepatic vein.

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 V_5 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 V_5 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 V_5 , V_8 , and inferior hepatic veins. The PTFE graft has been recommended for its high patency rates,⁵ although a potential risk of postoperative occlusion of venous tributaries including V_5 , V_8 , and inferior hepatic veins exists.^{5,6} 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.⁴

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