Usefulness of Artificial Jump Graft to Portal Vein Thrombosis in Deceased Donor Liver Transplantation

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Severe portal vein thrombosis (PVT) is often considered a relative contraindication for living donor liver transplantation due to high associated risks and morbidity. Meanwhile, improvement in operative techniques, resulting in higher success rates has removed PVT from the list of contraindications in deceased donor liver transplantation (DDLT). In this report, we describe a surgical technique for DDLT using polytetrafluoroethylene graft from the inferior mesenteric vein for portal inflow in patient with portomesenteric thrombosis.

Key Words: Liver transplantation, portal vein thrombosis, artificial graft, inferior mesenteric vein, polytetrafluoroethylene

INTRODUCTION

Portal vein thrombosis (PVT) is a common complication of end-stage liver disease with an incidence of 0.6–16% in patients with well-compensated disease, increasing up to 35% in cirrhotic patients with hepatocellular carcinoma.[1] As recently as the 1990s, PVT was associated with an increased incidence of complications and a higher mortality rate.[2] Additionally, blood loss volumes in patients with complete thrombosis were reported to be significantly higher than those in patients without PVT or with partial PVT.[3] Nevertheless, the presence of PVT is no longer considered an absolute contraindication for liver transplantation, as many improvements have been made in surgical technique and perioperative management.[2] In this report, we describe using polytetrafluoroethylene (PTFE) jump graft from the inferior mesenteric vein (IMV) for portal inflow in a patient with severe portomesenteric venous thrombosis.

CASE REPORT

A 64-year-old male patient was admitted to our hospital for deceased donor liver

transplantation. The patient's previous medical history revealed alcoholic liver cirrhosis and diabetes mellitus. Also, he had undergone transarterial chemoembolization with adriamycin three times in the past because of hepatocellular carcinoma in segment 8 with minimal portal vein thrombosis, as well as balloon occluded retrograde transvenous obliteration and/or endoscopic variceal band ligation six times, because of gastric and esophageal variceal bleeding. At the time of admission, his Child-Turcotte-Pugh score was 10 points (grade C), and his model for end-stage liver disease score was 9.0 points. Preoperative computed tomography revealed portomesenteric thrombosis from the portal vein to the proximal superior mesenteric vein with complete luminal obstruction, and venous collateral flow was not prominent (Fig. 1). The segment of portomesenteric thrombus was about 6.8 cm and the IMV had no thrombosis. The donor was a 56-year-old male who had brain death due to traumatic cerebral hemorrhage.

During exploration for recipient hepatectomy, portal venous flow was not detected by intraoperative doppler ultrasound and venous collateral flow was not prominent. Mechanical thrombectomy and low dissection of the portal vein was performed, and the portal venous inflow was improved. After the liver graft was placed in the recipient's abdomen, the donor's inferior vena cava was anastomosed to the recipient's inferior vena cava by applying the piggyback technique. Then, the portal vein of the recipient was anastomosed to the graft portal vein in an end_ to_ end fashion. Although the graft was recovered from ischemia after reperfusion, portal blood flow decreased gradually over a few minutes. Although repeated mechanical thrombectomy with portal venotomy was executed, the portal inflow did not improve. Thus we attempted to search for other area for portal inflow along more distal regions of the superior mes-

enteric vein. However, the peripancreatic area showed severe adhesion with superior mesenteric vessels. We had no choice except to perform a bypass from the IMV to the portal vein using PTFE (diameter: 7 mm) graft since the length of the iliac vessel of the donor was not sufficient for bypass and cryopreserved vein. After IMV was exposed below the ligament of Treitz, end_ to_ side anastomosis was performed using polypropylene 6-0 continuous running sutures between PTFE with IMV and end_ to_ side anastomosis between PTFE and the graft's portal vein (Fig. 2). The PTFE graft was passed through the mesentery above the pancreas, and anastomosis was performed in the same pattern between PTFE with distal portal vein. Intraoperative Doppler assessment after bypass showed a mean portal vein velocity 48.4 cm/second. We performed gauze packing with externalization at the mesentery of the pancreatic head area because of uncontrolled oozing-like bleeding. The weight of the graft was 1799 gram, the cold ischemic time was 318 minutes, the warm ischemic time was 40 minutes, and the anhepatic time was 194 minutes. Intraoperative transfusions were performed with 50 packs of packed red blood cells, 20 packs of fresh frozen plasma, and 20 packs of platelet concentrate. The total operative time was 14 hours and 5 minutes.

On the first postoperative day, portal vein Doppler ultrasound revealed normal waveform (mean flow velocity of 34.9 cm/second) without thrombus, and the vital signs and laboratory findings were relatively stable. The patient underwent general anesthesia again on the 16th day after transplantation for perihepatic hematoma removal, but the portal vein and the PTFE graft were intact.

After the transplant operation, the amount of ascites was 500–1500 cc/day until post-transplant 3 weeks, and we compensated daily ascites by 5% albumin and Hartmann solu-
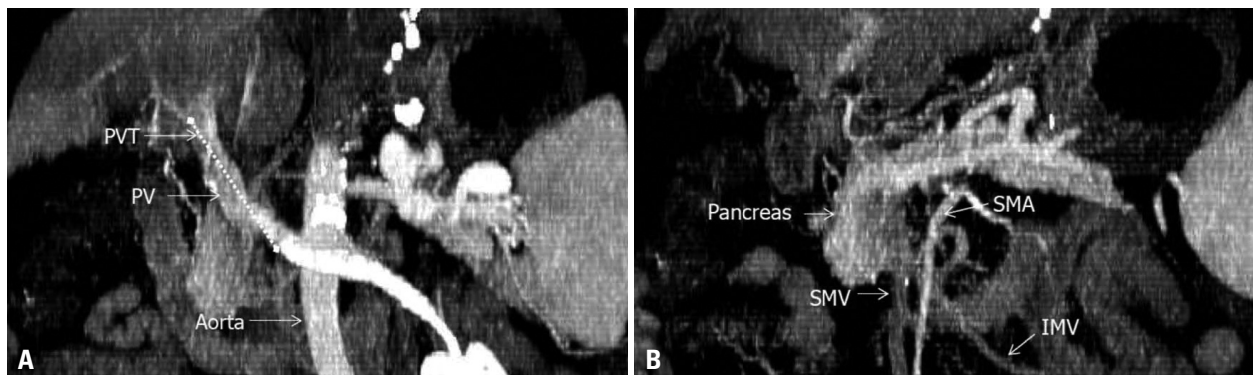
Fig. 1. Portal venous thrombosis is shown in preoperative computed tomography (A and B, maximum intensity projection of serial coronal view). Thrombosis was extended to the proximal superior mesenteric vein (dot line; length, 6.8 cm) for a Yedel classification Grade 3. PV, portal vein; PVT, portal vein thrombosis; SMV, superior mesenteric vein; SMA, superior mesenteric artery; IMV, inferior mesenteric vein.
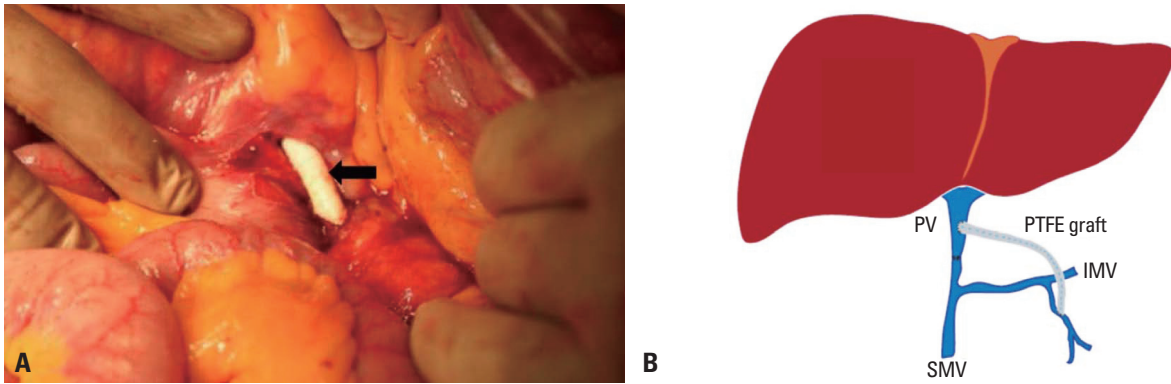
Fig. 2. After thrombectomy and lower dissection, portal vein anastomosis with polytetrafluoroethylene (PTFE; arrow) vascular artificial graft was performed with continuous running suture using polypropylene (A). Since massive bleeding and adhesion precluded the possibility of distal superior mesenteric vein interposition, we performed anastomosis between the proximal inferior mesenteric vein and distal portal vein using PTFE (B; schematic representation). PV, portal vein; SMV, superior mesenteric vein; IMV, inferior mesenteric vein.
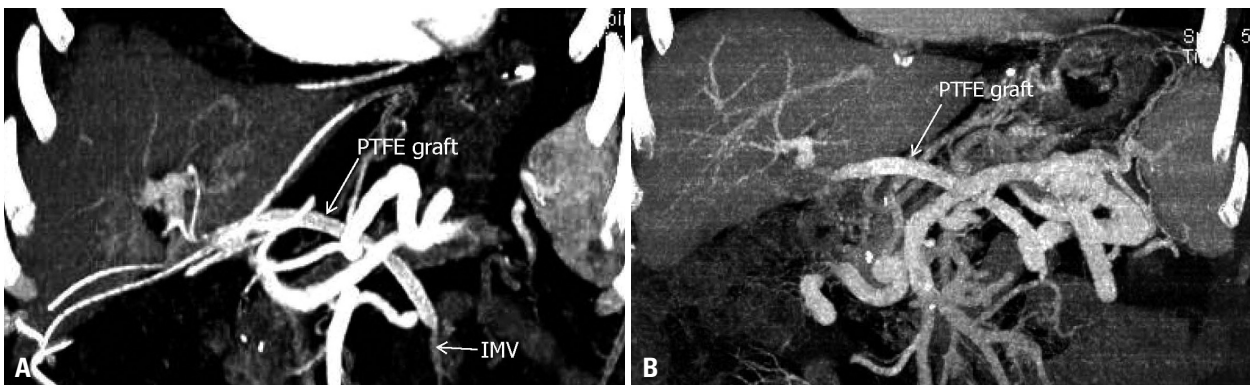
Fig. 3. Postoperative computed tomography at post operative 7 days (A) and 27 months (B) shows that the polytetrafluoroethylene graft has no thrombosis and no anastomotic stricture. PTFE, polytetrafluoroethylene; IMV, inferior mesenteric vein.

tion. The patient needed continuous renal replacement therapy for oliguria for 3 days, and we transported the patient to a general ward at post-transplant 10 days. After hematoma removal at post-transplant 16 days, the patient was admitted again to the surgical intensive care unit. At this time, the patient's mental status was deep drowsy for 3 weeks, and we performed tracheostomy at post-transplant 27 days for continuous ventilation. Brain magnetic resonance imaging revealed an acute ischemic lesion in the mid pons. At post-transplant 56 days, the patient was transported to a general ward; however, his lower extremities revealed muscle atrophy, which required rehabilitation for about 4 months. The patient stayed in the intensive care unit for 50 days, and the total hospital stay was 179 days. The patient was managed with prostaglandin E1 (0.01 lg/kg/h) and a protease inhibitor (mesilate gabexate; 1 mg/kg/h) for 7 days after transplantation. Immunosuppressive agents included a triple therapy of calcineurin inhibitor (tacrolimus), mycophenolate mofetil, and steroid. Anti-platelet agent (aspirin 100 mg/day) was prescribed at post-transplant 30 days, and is still being used. Follow-up computed tomography showed normal portal flow without thrombus on the 7th day and 27 months after liver transplantation (Fig. 3). Now, he is healthy without transplant-related complication, although he still sometimes reports paralytic ileus.

DISCUSSION

PVT is caused by decreased portal flow from progressive liver cirrhosis and the development of periportal lymphangitis and fibrosis.[1] PVT is more frequent in patients with autoimmune, cryptogenic, and alcoholic cirrhosis than in patients with hepatitis C.[2] The pathophysiology of PVT is complex, but related to liver cirrhosis, which causes the elevation of portal pressure in relation to endothelial injury and thrombus formation.[4] Enhanced coagulability as a result of decreased levels of natural anticoagulants, such as protein C, protein S, and antithrombin III, as well as coagulation factors are also observed in patients with PVT.[1]

Yerdel, et al.[5] classified PVT into four grades according to its extent and the severity of luminal occlusion by the throm-

bus. Our case, exhibited grade 3 PVT, showing complete thrombosis on both the PV and the proximal superior mesenteric vein (SMV).

Although an optimal technique for managing PVT during liver transplantation does not exist, the most preferred method of portal vein reconstruction is eversion thrombectomy, especially in grade 1 or 2 PVT.[1,2,5,6] Grade 2 and 3 PVT can be managed with either thrombectomy or anastomosis to the splenomesenteric confluence with or without graft, or anastomosis onto a varix, depending on the anatomy and thrombus severity, as well as extent in the patient.[2] Lladó, et al. recommend a venous graft or anastomosis to a collateral vein for the patients with grade 3 thrombosis and involvement of the SMV.[2,5] Especially, a jump venous graft from the SMV is indicated only when the portal flow can be restored in cases of extensive PVT or when there is no suitable engorged collateral coronary vein available.[4] However, prosthetic vessel grafts are the only choice if a venous conduit is not possible. The indications for prosthetic vessel grafts for the splanchnic venous system have been limited because the blood flow in this system is relatively slow, that is, highly thrombogenic. Moreover, little clinical experience with the use of PTFE grafts in the splanchnic venous system has been accumulated.[7] PTFE seems to be a good alternative for outflow venoplasty, when autologous or allogeneic vascular patch is not available or when in critical situations.[8] The hematological condition of patients undergoing liver transplantation is less thrombogenic than that in canine models, as shown in the impairment of coagulation profiles, such as moderate-to-severe thrombocytopenia, the low viscosity from lowered hematocrits, and the concurrent use of antiplatelet agents.[7] Luminal thrombus formation is uncommon when a prosthetic graft is used as a conduit for large arteries, such as the inferior vena cava, or even the portal vein. These vessels can be classified as high-flow vessels regardless of the pressure, so that there is a low risk of luminal thrombus formation.[7] Another problem with the use of PTFE is infection. Pomposelli, et al.[9] reported no infection complications cases when PTFE was used for venous reconstruction. Due to the inert nature of PTFE, the result of any infection is likely to be due to early conduit thrombosis and eventual encapsulation of the graft. Therefore, decisions regarding the type of conduit can be made based on availability of resources and preference of the surgeon. Moreover, one advantage of the synthetic graft material is reduced cost, which is approximately 10% less than that of cryopreserved vessels.[9]

The use of a temporary porto-caval shunt is associated with better hemodynamic stability and improved renal function, as well as decreased transfusion requirements in piggy back liver transplantation.[10,11] In our case, we performed bypass from the IMV to the graft portal vein after reperfusion of the liver graft. In fact, preoperative CT scan did not clearly show the range of portomesenteric thrombosis. Moreover, we thought that first mechanical thrombectomy was done successfully because of sufficient portal back flow. If the porto-caval shunt was performed before portal reconstruction and reperfusion, the patient may have shown better progression during hospitalization.

There are only a few reports associated with IMV for portal vein inflow. Kobayashi, et al. reported on IMV to the left gonadal vein shunt for gastroesophageal varices and extrahepatic PVT after liver transplantation.[12] In our case, the SMV was difficult to use for portal vein reconstruction because of severe adhesions and friable vessel walls. After the successful portal vein reconstruction using the IMV with PTFE, the patient had no vascular problems for 27 months.

In conclusion, PTFE jump graft from the IMV to the portal vein may be a feasible option in patients with portomesenteric thrombosis, and the IMV can be a potential source of portal inflow in liver transplants.

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