Case Report

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Late Hepatic Venous Outflow Obstruction Following Inferior Vena Cava Stenting in Patient with Deceased Donor Liver Transplantation Using Modified Piggyback Technique

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Following liver transplantation, a few reports have documented hepatic venous outflow obstruction (HVOO) after inferior vena cava (IVC) stenting for the treatment of IVC stenosis. However, HVOO occurred early after IVC stenting and was mostly associated with living donor liver transplantation. Here, we report a case of HVOO that occurred 31 months after IVC stenting in a man who received deceased donor liver transplantation (DDLT) using a modified piggyback (PB) technique. The cause of HVOO was unclear, but one possible explanation is that the balloon-expandable IVC stent might have compressed the IVC chamber on the donor liver side, which would have changed the outflow hemodynamics, resulting in intimal hyperplasia. Therefore, simultaneous hepatic venous stenting with IVC stent placement could help prevent HVOO in patients receiving DDLT with the modified PB technique.

Key Words: Modified piggyback technique, Inferior vena cava stent, Heptic venous outflow obstruction
중심 단어: 변형 피기백 술, 하대정맥 스텐트, 간정맥 유출로 폐쇄

INTRODUCTION

The piggyback (PB) technique used in orthotopic liver transplantation is theoretically superior to conventional end-to-end cavo-caval anastomosis because it can maintain hemodynamic stability, shorten the warm ischemic period because of the shorter time taken for anastomosis, and there is no need of a veno-venous bypass(1,2). However, the classical PB technique has some drawbacks such as hepatic venous outflow obstruction (HVOO). In 1992, Belghiti et al.(3) developed a side-to-side cavo-caval anastomosis with preservation of the recipient retrohepatic vena cava, named the modified PB technique, to overcome this problem.

Stenosis of the inferior vena cava (IVC) during the early postoperative period results from technical factors and can be managed by interventional stent placement. Also, a few studies reported that IVC stenting induced HVOO(4-6). However, there is no report regarding late-onset HVOO after IVC stenting in the recipient using the modified PB technique. Herein, we report a case of HVOO that developed 31 months after balloon-expandable IVC stent placement for treating IVC stenosis in a man who received adult deceased donor liver transplantation (DDLT) with the modified PB anastomosis.

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CASE REPORT

In June 2012, a 53-year-old man who had been listed on the Korean Network for Organ Sharing was admitted to Kyungpook National University Hospital for receiving DDLT. He had been diagnosed with hepatitis B-related liver

cirrhosis in 2004, and thereafter he suffered from multiple episodes of hematemesis caused by esophageal varix bleeding and uncontrolled ascites, with conservative therapy being applied. Eventually, a transjugular intrahepatic portosystemic shunt (TIPS) stent was placed in 2006. In addition, he was diagnosed with hepatocellular carcinoma (HCC) in 2009, and radiofrequency ablation therapy or transarterial chemoembolization was performed for the treatment of HCC up to 5 months before the DDLT procedure.

A preoperative dynamic computed tomography (CT) scan of the abdomen showed no viable tumor or extrahepatic metastasis. However, the cranial end of the TIPS stent reached to just below the right atrium across the suprahepatic vena cava and the caudal end of the stent was located at the junction of the superior mesenteric and splenic veins (Fig. 1). During total hepatectomy of the recipient patient with preservation of the native retrohepatic vena cava, we paid particular attention not to injure the IVC, but the suprahepatic portion received a tangential tear about 1.8 cm long because of severe adhesion of the stent with the anterior wall of the IVC. This was repaired using a 5～0 polypropylene running suture. Hepatic outflow reconstruction was performed by the modified PB technique with a 39 mm orifice at the IVC.

The patient's postoperative course was uneventful and the he was discharged with good liver function and a serum creatinine level of 1.33 mg/dL on postoperative day 28. However, 1 week after discharge, he complained of an abdominal distention that could not be controlled by conservative therapy. On contrast-enhanced CT scans, significant suprahepatic IVC stenosis was detected and a large amount of new ascites fluid was observed (Fig. 2). Subsequently, we placed a balloon-expandable Palmaz stent (20×45 mm; Cordis Corp.,

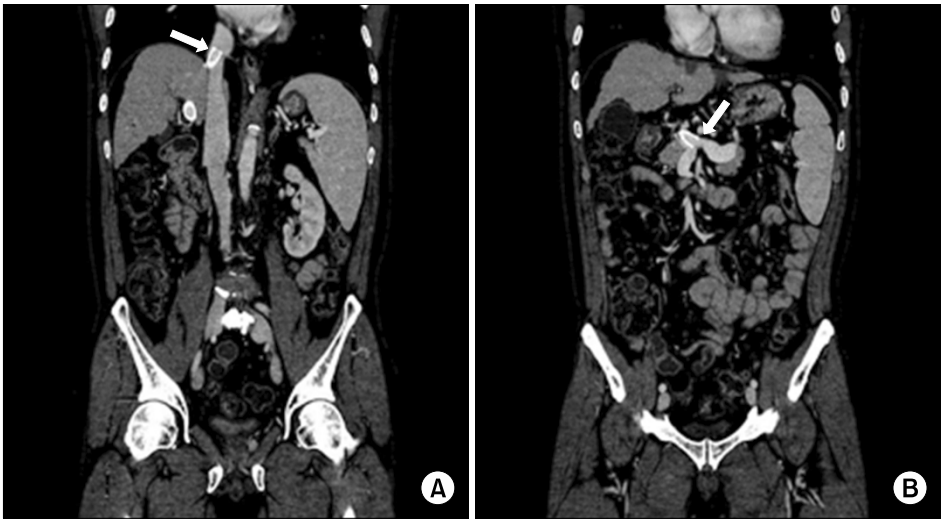

Fig. 1. Abdominal computed tomography scan of the patient performed prior to receiving deceased donor liver transplantation. (A) The cranial end of the transjugular intrahepatic portosystemic shunt (TIPS) stent was located just below the right atrium, across the suprahepatic vena cava (arrow). (B) The caudal end of TIPS stent was placed at the junction of the inferior mesenteric and splenic veins (arrow).

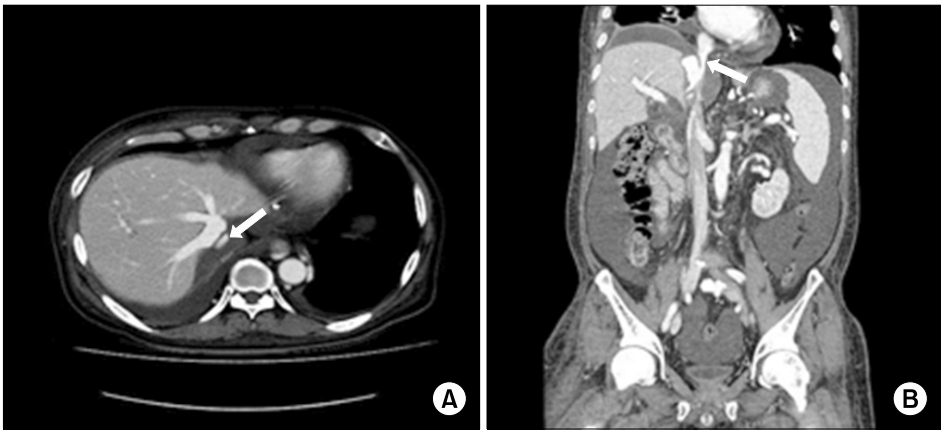

Fig. 2. This abdominal computed tomography scan of the patient performed 6 weeks after deceased donor liver transplantation shows stenosis of the suprahepatic vena cava (arrows). (A) Axial view. (B) Coronal view.

Johnson & Johnson, Fremont, CA, USA) in the stenotic site of the IVC over the side-to-side cavo-caval anastomosis through the right femoral vein, and the patient was discharged free of symptoms. Thereafter, he remained in a satisfactory condition for 31 months except for mild renal impairment. His serum creatinine level ranged from 1.32 to 2.52 mg/dL.

In February 2015, the patient developed symptoms of abdominal distension without lower extremity edema. The laboratory test results were within the normal ranges except for serum albumin concentration as follows: aspartate aminotransferase 12 IU/L, alanine aminotransferase 15 IU/L, prothrombin time-international normalized ratio 1.1, and serum albumin 2.8 g/dL. Doppler ultrasonography showed monophasic waveforms of the hepatic vein and an abdominal CT scan showed liver graft congestion and a collapsed IVC on the donor liver side (Fig. 3). Subsequent venography confirmed stenosis at the cavo-caval anastomosis site with a 20 mmHg pressure gradient and edge stenosis of the IVC just below the Palmaz stent (Fig. 4). Balloon angioplasty was performed at both lesions and the pressure gradient improved. We considered additional stent placement for treating the stenosis, but the abdominal distension improved dramatically, which resulted in a loss of 4 kg of body weight during the first 2 weeks after the procedure. Also, serum albumin showed a value of 4.4 g/dL at 2 months after radiologic intervention. Therefore, we decided to "wait and see," and planned to insert the stent across the

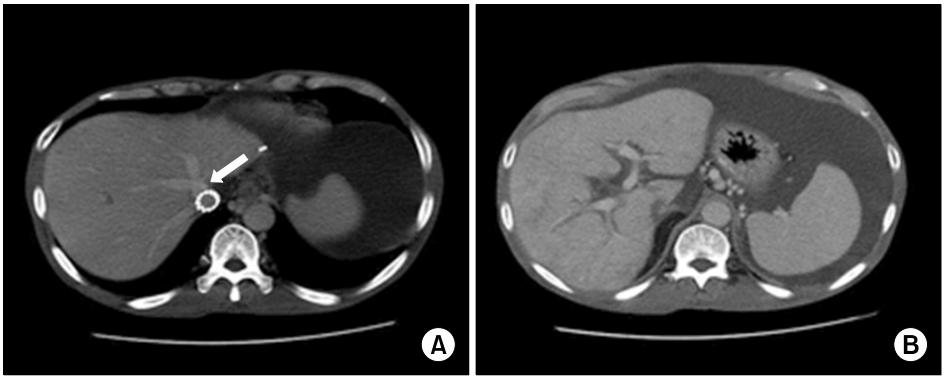

Fig. 3. This contrast-enhanced abdominal computed tomography (CT) scan shows a collapsed inferior vena cava (IVC) chamber on the donor side, a large amount of ascites fluid and liver graft congestion. (A) In comparison with the previous CT scan, the donor side IVC chamber had collapsed from extrinsic compression of the Palmaz stent (arrow). (B) A mottled pattern of contrast enhancement was observed in the portal venous phase.

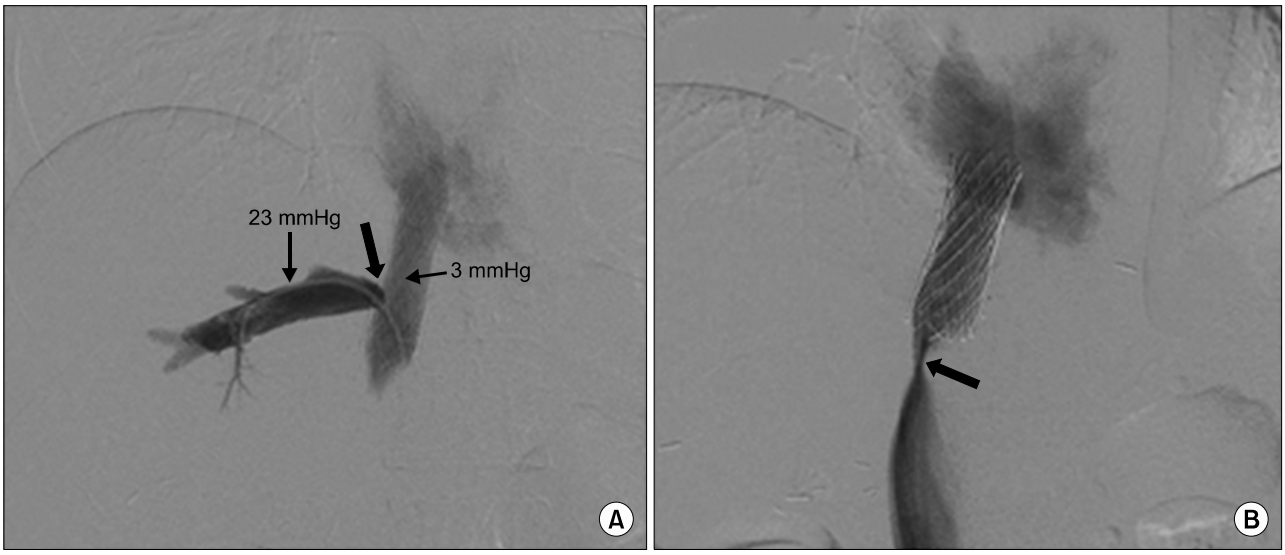

Fig. 4. The venogram demonstrated (A) hepatic venous outflow obstruction with a pressure gradient of 20 mmHg (arrow) and (B) edge stenosis at the inferior vena cava under the stent (arrow).

stenosis, when the symptoms recur. At last follow-up, the patient is doing well without suggestive symptoms 9 months after balloon angioplasty.

DISCUSSION

There are two possible causes of IVC stenosis following liver transplantation according to the time of occurrence after the operation. On one hand, during the early postoperative period, IVC stenosis is mainly caused by technical factors such as too tight an anastomosis of the outflow, kinking of the vessel, torsion of the graft, and other surgical errors. On the other hand, late stenosis most likely occurs in response to neointimal hyperplasia or perivascular fibrosis. For both early and late IVC stenosis, stent placement is the preferred treatment method in comparison with balloon angioplasty because of recoil or restenosis after this technique. In our case, the suprahepatic IVC was accidentally torn during the recipient's total hepatectomy because dense adhesion of the TIPS catheter with suprahepatic IVC made for a more complex transplant procedure, and this resulted in suprahepatic IVC stenosis. Although self-expandable stents are generally preferred in cases of IVC stenosis, we opted to use a balloon-expandable stent for precise placement and better radial strength.

Weeks et al.(4) reported a case of HVOO after a modified PB anastomosis that occurred 6 days after IVC stenting, and they managed it by placement of an additional stent across the modified PB anastomosis. Lee et al.(5) also reported excellent outcomes after IVC stent placement for treating IVC stenosis and emphasized that the possibility of hepatic venous outflow abnormalities should be considered following IVC stent placement. In addition, they assumed that the IVC stent itself could hinder the hepatic venous outflow. However, they performed end-to-end cavo-caval anastomosis for hepatic outflow reconstruction in whole liver transplantation, which was different from our technique, and most hepatic venous outflow abnormalities after IVC stenting have occurred early after stent placement in patients receiving living donor liver transplantation (LDLT). In our case, HVOO developed 31 months after IVC stenting in the recipient of DDLT with the modified PB anastomosis. Given that the modified PB technique was performed for achieving hepatic outflow, the diameter of the hepatic outflow anastomosis is wider than that seen in cases of LDLT. Therefore, we think that the IVC stent itself has less influence on hepatic outflow immediately after stenting. Conversely, one possible cause of late HVOO after IVC stenting among recipients of the modified PB technique is that the donor side IVC chamber can be collapsed over time by extrinsic compression of the stent, which might alter hepatic outflow velocity and patterns followed by some endothelial damage leading to intimal hyperplasia.

In the present case, edge stenosis was observed at the inferior border of the IVC stent. Another study suggested that the impact of intimal hyperplasia on patency could be minor after large diameter stenting in the IVC(7). However, given this case, altered hemodynamic conditions around the IVC stent might also cause neointimal hyperplasia, inducing edge stenosis.

In conclusion, when an IVC stent was placed after DDLT using the modified PB technique, the donor side IVC chamber can be compressed by the stent, and this might provoke the development of intimal hyperplasia. Therefore, simultaneous placement of stents at both the IVC stenosis and cavo-caval anastomosis sites might be a good alternative to prevent HVOO.

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