Transjugular Intrahepatic Portosystemic Shunt (TIPS) in Combination with Recanalization of Portal and Superior Mesenteric Vein Thrombosis Ensured Hassab’s Operation for High-Risk Esophago-Gastric Varices

Koichi Tomikashi1,3, Kensaku Kojima1, Yusuke Sugiyama1, Atsushi Ishimaru1, Koutaro Okuda1, Kazuyuki Toyoda1, Hisakazu Yamagishi2, Takeshi Okanoue1

1Department of Molecular Gastroenterology and Hepatology, 2Department of Surgery, Division of Digestive Surgery, Kyoto Prefectural University of Medicine Graduate School of Medical Science and 3Kohoku General Hospital

Portal vein thrombosis (PVT) is a well-recognized complication of liver cirrhosis (LC) and often induces severe portal hypertension, refractory ascites and variceal bleeding. We report on a case of refractory ascites and high-risk esophago-gastric varices due to extensive PVT. The patient’s refractory ascites prevented surgical intervention to improve his hemodynamic status. We thus performed a transjugular intrahepatic portosystemic shunt (TIPS) and balloon dilatation of the occluded segment. After this procedure, the patient’s ascites disappeared rapidly and we could carry out the required surgical procedure safely.

KEY WORDS: portal vein thrombosis, transjugular intrahepatic portosystemic shunt, portal hypertension, refractory ascites, variceal bleeding

INTRODUCTION

PVT (Portal vein thrombosis) is a well-recognized complication of various conditions including malignant disease, infection, hematological disease, and trauma, and it can occur following surgery. PVT is particularly associated with liver cirrhosis, and in fact, occurs in 5–19% of cases with end-stage liver cirrhosis.1–4 PVT often induces severe portal hypertension, which leads to esophageal varices that are unresponsive to endoscopic therapy. In addition, refractory ascites is a frequent complication.

The transjugular intrahepatic portosystemic shunt (TIPS) procedure is a commonly used percutaneous technique for patients who have portal hypertension, but it is not commonly used in patients with portal vein obstruction, including those with PVT. This is because in patients with PVT, the pressure of the intrahepatic portal vein is low, and thus TIPS has generally been considered a contraindication. However, Radosevich et al5) showed that TIPS is technically feasible in these patients. As familiarity with this procedure has grown, increasing numbers of patients have undergone successful shunt placement.

We report on a patient with high-risk esophago-gastric varices, portal vein (PV) and superior mesenteric vein (SMV) thrombosis, and refractory ascites. This patient was successfully treated by TIPS placement followed by balloon dilatation of the portal vein. After the procedure, his ascites became more controllable and the surgery for his high-risk varices could be performed safely.

CASE REPORT

In September 2000, a 78-year-old man with type B liver cirrhosis was admitted to our hospital for the treatment of esophago-gastric varices. He had a history of cholecystectomy for cholecystolithiasis 12 years earlier and of a percutaneous transluminal coronary angioplasty for acute myocardial infarc-
tion 11 years earlier. On physical examination, his conjunctiva showed evidence of anemia and his abdomen was slightly distended.

Laboratory investigations revealed markedly low levels of hemoglobin 9.3 g/dl (normal 10.8-16.9), hematocrit 29.1% (normal 32.4-50.2) and a platelet count of 101 $\times$ 10^9/l (normal 104-348 $\times$ 10^9). Liver function tests were as follows: aspartate aminotransferase 46 U/l (normal 12-35), alanine aminotransferase 41 U/l (normal 6-33), gamma-glutamyl transferase 20 U/l (normal 3-54), alkaline phosphatase 279 U/l (normal 120-362), total bilirubin 1.14 mg/dl (normal 0.2-1.0), albumin 3.9 g/dl (normal 3.9-5.2) and alpha-fetoprotein 3.3 ng/ml (normal below 20). Renal function was normal. Coagulation studies after admission were as follows: prothrombin time 13.2 s (control 11.8), fibrinogen degradation products (FDP) 25.8 $\mu$g/ml (normal below 5), D-dimer 12.6 $\mu$g/ml (normal below 1), antithrombin III activity 71% (normal 93-124), protein C activity 65% (normal 67-127) and protein S activity 61% (normal 65-135).

Endoscopy showed esophageal varices with multiple red spots and gastric varices on the cardia (Fig. 1a, b). Ultrasound showed minimal ascites and a partial thrombosis of the PV. Enhanced computed tomography revealed an extensive thrombosis from the PV to SMV (Fig. 2a, b). Indirect venography of the portal and splenic veins revealed a narrowing of the PV and severe stenosis of the splenic vein due to thrombosis (Fig. 3a). It was therefore apparent that the blood flow of the splenic vein could not enter the PV and esophago-gastric varices formed in the left gastric vein (Fig. 3b). In an attempt to recanalize the PV, we started thrombolytic therapy using urokinase (24 $\times$ 10^4 U/day) for 2 days. However, this was found
to be insufficient.

Due to the patient's hemodynamic status, endoscopic sclerotherapy of his varices was contraindicated. We, therefore, scheduled surgical treatment of his varices. However, the ascites, which were transudative and did not include any malignant cells, were refractory and gradually increased. Therefore, we opted for TIPS, to be followed by balloon dilatation of the PV in order to reduce the amount of ascites.

TIPS was performed successfully according to the following procedure. The right internal jugular vein was punctured and a 9-Fr sheath inserted into the inferior vena cava. After hepatic venography, TIPS was performed using the ROSCH-UCHIDA transjugular liver access kit (Cook, Bloomington, IN). After recording the pressures of the portal system, a stent measuring 6 cm in length and 8 mm in diameter was placed following dilatation of the TIPS tract using a percutaneous transluminal angioplasty (PTA) catheter 8 mm in diameter (10 atm for 30 s, 6 times) (Fig. 4a). Then, the occluded segment of the PV and the SMV were dilated using the PTA catheter (10 atm for 45 s, twice) (Fig. 4b). After the procedure, pressures in the portal system were reduced (PV: from 33 to 26 mmHg, SMV: from 37 to 23 mmHg, splenic vein: from 30 to 23 mmHg). After
this procedure, his liver function was stable and encephalopathy was not induced by TIPS.

Although the ascites disappeared rapidly after the procedure, the form of esophageal varices hardly improved. Furthermore, the pressure of the portal system did not decrease sufficiently. And so, we decided to perform Hassab's operation (splenectomy and devascularization of the lower esophagus) for him. Postoperative endoscopy revealed that the esophageal varices were reduced in size, and that the gastric varices had completely disappeared (Fig. 5a, b).

DISCUSSION

PVT is well-known to be associated with liver cirrhosis and can be secondary to a benign or malignant process. PVT is found in 34.8% of patients with hepatic malignancy, 22.2% of patients with Budd-Chiari syndrome (BCS) and from 5–19% of patients with liver cirrhosis.1-4) PVT is difficult to diagnose clinically but it is found in 71.6% of patients with hepatoma on autopsy during gross examination.5) In patients with cirrhosis, it is generally accepted that the low flow state in the PV system is the main predisposing condition for thrombus formation.7-10)

TIPS is now the generally accepted treatment for patients with portal hypertension and refractory variceal bleeding. However, the indication for TIPS in refractory ascites remains controversial. Obstruction of the PV, including that caused by PVT, has been considered a contraindication for TIPS. In patients with severe PVT, PVT itself needs treatment either with thrombolytic therapy or dilatation of the occluded PV. However, since most of these patients have some degree of ascites, the percutaneous transhepatic approach to PVT can be very risky. Conversely, TIPS is a procedure that does not injure the liver surface and makes the approach to the treatment of PVT safe. Therefore, there have been recent reports of the successful use of TIPS in the treatment of Budd-Chiari syndrome or PVT.5,11-16)

In most cases, no major complications related to TIPS were reported, although Ganger et al15) reported a fatal case in which TIPS immediately occluded. Walser et al17) suggested that patients with PVT treated with TIPS require a closer follow-up and probably experience shorter survival rates than patients without PVT. Thus, TIPS placement in a patient with PVT requires greater vigilance for complications.

In our patient, enhanced computed tomography done 3 years earlier had already revealed a small PVT on the wall of the hilar PV. This would suggest that both the cholecystectomy and the congestion of the PV were the causes of PVT growth. To reduce the patient's massive ascites, we performed TIPS and dilatation of the occluded PV (through TIPS) using a PTA catheter. This procedure recanalized the PV and reduced the pressure in the portal system. This rendered the patient's refractory ascites more controllable, allowing us to safely perform the appropriate surgical treatment for his high-risk varices. Postoperatively, the patient's esophageal varices were greatly reduced in size, while the gastric varices disappeared completely. During this treatment, his liver function
was stable, and encephalopathy was not induced by TIPS.

Although splenectomy is a recognized factor that may lead to deterioration of PVT, we performed this procedure. The patient's variceal blood flow was supplied from the splenic vein directly through the left gastric vein. Therefore, to decompress his high-risk varices, it was necessary to dilate the junction of the SMV and splenic vein. Since thrombosis was widespread, if we had dilated the splenic vein at that point, the SMV might have narrowed, increasing its pressure. Therefore we decided to resect the spleen.

Although the prophylactic treatment of high-risk varices remains controversial in the United States and Europe, it is widely accepted in Japan. This is because viral liver cirrhosis is largely responsible for the high-risk varices found in Japanese patients.

Our case is the first in which TIPS placement and angioplasty of the portal vein was used to control refractory ascites as a prophylactic measure in order to ensure safe surgical treatment for the high-risk varices.

The procedures described in this report are relatively invasive. Nevertheless, if high-risk varices due to PVT were to rupture, the portal blood flow to the liver could decrease further and liver function could deteriorate more rapidly than in patients without PVT. Alternatively, if a laparotomy was to be performed for the patient with PVT, the development of collateral veins in the peritoneal cavity may interfere with the safety of the operation. Thus TIPS followed by dilatation is of value in patients with PVT, as a preventive treatment for bleeding from high-risk varices and as a pretreatment for subsequent surgery.

In conclusion, we have found that TIPS followed by dilatation of the occluded PV or SMV using a PTA catheter is a highly effective procedure for patients with PVT complicated by refractory ascites or high-risk varices.

REFERENCES