

Portal venous stent placement for treatment of portal hypertension caused by benign main portal vein stenosis

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Abstract

AIM: To evaluate the value of endovascular stent in the treatment of portal hypertension caused by benign main portal vein stenosis.

METHODS: Portal vein stents were implanted in six patients with benign main portal vein stenosis (inflammatory stenosis in three cases, postprocedure of liver transplantation in another three cases). Changes in portal vein pressure, portal vein patency, relative clinical symptoms, complications, and survival were evaluated.

RESULTS: Six metallic stents were successfully placed across the portal vein stenotic or obstructive lesions in six patients. Mean portal venous pressure decreased significantly after stent implantation from (37.3 ± 4.7) cm H₂O to (18.0 ± 1.9) cm H₂O. The portal blood flow restored and the symptoms caused by portal hypertension were eliminated. There were no severe procedure-related complications. The patients were followed up for 1-48 mo. The portal vein remained patent during follow-up. All patients survived except for one patient who died of other complications of liver transplantation.

CONCLUSION: Percutaneous portal vein stent placement for the treatment of portal hypertension caused by benign main portal vein stenosis is safe and effective.

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Key words: Angioplasty; Stent; Portal vein stenosis; Hypertension; Portal

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INTRODUCTION

Portal venous hypertension is caused by extrahepatic obstruction or stenosis when the portal vein is blocked before blood reaches the liver. Patients with this condition account for 5-10% of all cases of portal hypertension^[1-3]. Benign portal venous stenosis or obstruction induced by pancreatitis, appendicitis and postsurgical adhesive portal venous stenosis are the most common extrahepatic portal venous occlusion^[4]. Portal hypertension resulted from the occlusion of main portal vein often results in the development of varices in esophagus, stomach, duodenum, small and large bowel, and gastrointestinal bleeding leading to death^[4,5]. The development of ascites due to portal hypertension affects the patient's quality of life. Therefore, it is crucial to restore the blood flow of portal vein and to prevent symptoms of portal hypertension.

To date, little is known about the role of stent placement in the treatment of benign portal venous stenosis or occlusion. The aim of the present study was to retrospectively assess the value of stent placement in the treatment of portal hypertension caused by benign main portal vein stenosis or occlusion.

MATERIALS AND METHODS

Patients

From July 2000 to July 2004, six male patients (mean age, 39.8 ± 14.7 years, range 21-65 years) underwent percutaneous transhepatic stent placement for benign portal vein stenosis or occlusion. The clinical characteristics of these patients are summarized in Table 1. Three cases were associated with procedure of liver transplants for posthepatic cirrhosis, two cases with necrotizing pancreatitis, and one case with suppurative appendicitis. The diagnosis of portal vein stenosis (five cases) and occlusion (one case) was established by Doppler ultrasound, and confirmed by transhepatic direct portography. Clinical signs or symptoms included gastrointestinal tract bleeding from varices in two patients, gastrointestinal tract bleeding and ascites in two patients, and ascites in two patients.

Stent placement

Informed consent was obtained from all patients prior to all procedures. After local anesthesia was performed with 2% lidocaine, the liver was punctured with fluoroscopic guidance using a 21-G needle (Chiba, COOK) or 18-G percutaneous cholangiographic needle (NPAS-100, COOK). A 7-F sheath was inserted into the portal vein through a guidewire. A 5-F catheter was advanced beyond the stenotic

Table 1 The clinical characteristics of six patients

Patient no.	Sex	Age (yr)	Symptoms	Etiology	Portal venous pressure (cm H ₂ O)		Stent style
					Before stent placement	After stent placement	
1	Male	33	Ascites, gastroesophageal varices, hematemesis	Postsurgical of liver transplantation	45	20	Wallstent
2	Male	35	Ascites	Postsurgical of liver transplantation	33	18	Wallstent
3	Male	21	Ascites, Melena	Necrotizing pancreatitis	38	20	Wallstent
4	Male	65	Gastroesophageal varices, Melena	Postsurgical of liver transplantation	35	18	Wallstent
5	Male	40	Ascites	Necrotizing pancreatitis	40	15	Symphony
6	Male	45	Gastroesophageal varices, Melena	Suppurative appendicitis	33	17	Wallstent

1 cm H₂O = 0.098 kPa.

or occluded lesions. Then portography was performed and portal venous pressure was measured. After 3 000-IU heparin was directly injected into the portal vein, the stenotic or occluded segment was dilated with a 10-mm-diameter balloon catheter. The stents were placed across the lesions because of the persistence of stenotic lesions after repeated dilations. The implanted stents included 5 wallstents with a diameter of 10 mm and a length of 5-8 cm (Wallstent, Boston Scientific) in five patients (patients 1-4 and 6), and a Symphony stent with a diameter of 10 mm and a length of 8 cm (Symphony stent, Boston Scientific) in one patient (patient 5) (Table 1). Then portography and portal venous pressure measurement beyond the lesions were repeated. Finally, gelatin sponge or coils were placed in the liver parenchymal tract via the sheath or catheter to prevent intraperitoneal hemorrhage.

All the patients underwent anticoagulant therapy using fraxiparin 3-5 d after the procedure, followed by warfarin administration for 3 mo unless bleeding occurred.

Follow-up protocol

All the patients were followed up by Doppler ultrasound every month to evaluate the patency and blood flow in the portal vein, stent location and stenosis recurrence. Improvement of clinical symptoms, complications and survival was observed.

Statistical analyses

The data were expressed as mean±SD. The significance of differences in portal venous pressure before and after stent

placement was assessed with Student's *t* test. $P < 0.05$ was considered statistically significant.

RESULTS

Six stents were successfully implanted in the portal venous lesions of all six patients (Table 1, Figure 1). The mean portal venous pressure decreased significantly from (37.3 ± 4.7) cm H₂O (1 cm H₂O = 0.098 kPa) to (18.0 ± 1.9) cm H₂O (Table 1). There was a significant difference in portal venous pressure before and after stent placement ($t = 10.52$, $P < 0.001$).

The clinical signs or symptoms of gastrointestinal tract bleeding, gastroesophageal varices and ascites were eliminated after stent placement in all patients. The follow-up period ranged from 1 to 48 mo (mean 30 mo). During the follow-up period, all patients survived except for patient 4 who died of other complications of liver transplantation 1 mo after stent implantment. The follow-up Doppler ultrasound demonstrated that the portal vein remained patent and blood flow in the portal vein with a stent was good. All stents remained patent, and none required repeat dilation and migration of stents was not observed in all patients during follow-up.

After stent placement, all patients complained of mild abdominal pain at the puncture site. Transient fever (lower than 38 °C) occurred in three patients (patients 1, 3 and 4). These symptoms could be alleviated after symptomatic treatment. There were no other procedure-related and postprocedure complications in all patients.



Figure 1 Development of ascites and hematemesis 1 mo after liver transplantation in a 33-year-old man (A and B) and refractory ascites 4 mo after acute necrotizing

pancreatitis in a 40-year-old man (C, D).

DISCUSSION

Etiology of portal vein stenosis or occlusion

Portal vein stenosis or occlusion is associated with neoplastic and non-neoplastic conditions. Malignant portal vein stenosis usually results from portal vein tumor thrombus or compression of neoplasms, accounting for 15-24% of patient with portal venous stenosis or occlusion^[4,6-9]. Non-neoplastic conditions are the most common causes of portal venous stenosis and occlusion. The following factors are associated with benign portal vein stenosis or occlusion: inflammatory diseases such as pancreatitis and appendicitis, which can result in thrombosis or elastic portal vein stenosis^[10-12]; portal hypertension resulted from liver cirrhosis during which stasis of blood flow in main portal vein and its branches caused by portal hypertension may result in portal vein stenosis or occlusion due to thrombosis^[13,14], and anticardiolipin antibodies may play a role in the development of portal vein thrombosis in cirrhosis^[15]; abdominal surgeries or trauma such as liver transplantation surgery, splenectomy, cholecystectomy^[4,16,17]; other conditions such as primary portal vein thrombosis and congenital absence of portal vein^[18]. Local portal vein stenosis secondary to fibrosis adhesion is usually associated with the inflammatory factors or abdominal surgery at the region of liver hilum. However, not all portal vein stenoses would cause symptoms related to portal hypertension. Some studies indicated that there is no clinical symptom if the stenosis is less than 50% of the diameter of portal vein. If the stenosis of portal vein was greater than 80% of its diameter, symptoms of portal hypertension (e.g, gastrointestinal bleeding, refractory ascites, and thrombocytopenia) develop and liver failure and transplant recipient liver dysfunction occur^[19,20].

In our series, three patients were associated with liver transplant surgery, and the other three patients were associated with inflammation (necrotizing pancreatitis in two cases and suppurative appendicitis in one case). The stenosis in our series was greater than 50% of the diameter of portal vein and occluded completely in one case. Because the clinical symptoms of portal hypertension in these patients were significant, portal venoplasty or stent placement was necessary to decrease the portal venous pressure.

Clinical value of portal venous stent placement

Portal venous stent placement is useful for most patients with portal vein stenosis or occlusion caused by malignant neoplasms. Yamakado *et al*^[6,21], reported the clinical effectiveness of portal venous stent placement in patients with hepatocellular carcinoma (HCC), pancreatic and biliary neoplasms invading portal vein, and found that portal venous stent placement decreases portal venous blood pressure. The clinical outcomes, however, are quite different depending on the invasion site. When portal venous blood flow is blocked and the splanchnic vein is intact, the stents remain patent and portal hypertension symptoms subside. If splanchnic vein is involved, stents patency would become worse.

To our knowledge, there are only few case reports about the stent placement for portal hypertension caused by benign portal vein stenosis or occlusion. Funaki *et al*^[6], considered that the first choice of treatment for most patients with

benign portal vein stenosis is venoplasty with balloon dilation, and stent placement is important for the elastic and recurrent stenosis to maintain the portal vein blood flow. In their study, intravascular stents were placed in 12 patients with segment hepatic transplants for 'elastic' or 'recurrent' portal vein stenosis, which remained patent for 5-61 mo (mean time, 46 mo). Cherukuri *et al*^[22], placed stents after thrombolysis in two patients with hepatic transplants and portal vein stenosis and thrombosis, and reported that the portal venous patency is good during follow-up. Some authors considered that the management of thrombolysis after stent placement is crucial and effective for patients with portal vein thrombosis^[23,24]. In our series, portal stenosis ($n = 5$) and occlusion ($n = 1$) were elastic. Balloon dilation was not enough for these patients, stent implantation was performed for all patients to decrease the portal vein pressure. Our results showed portal venous stent placement was useful in decreasing the portal vein pressure, and all stents remained patent in the follow-up. All patients survived and were asymptomatic except for one case who died of complications of hepatic artery thrombosis and bile leakage related to liver transplant.

Complications of percutaneous transhepatic portal venous stent placement

Abdominal pain at the puncture site is the most common complication of percutaneous transhepatic portal venous stent placement^[6,16], which occurred in all cases of our series. Transient fever (lower than 38 °C) occurred in three of six patients. The symptoms of abdominal pain and fever are usually mild and disappear 2-3 d after symptomatic treatments. Complication of liver abscess has been reported^[16]. Percutaneous drainage is necessary in these patients. The potential complications of intraperitoneal bleeding, bile injury or biliary bleeding, have been reported in percutaneous portal vein embolization or percutaneous transhepatic measurement of portal pressure^[25-29]. There were no severe complications in our series.

In conclusion, percutaneous portal vein stent placement for the treatment of portal hypertension caused by benign main portal vein stenosis is safe and effective.

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