

Portal vein stent placement for malignant portal vein occlusion

Eslam W. Youssef, Mario Ghosn, Mohamad El Hawari, Gabriel C. Fine, Majid Maybody, Hooman Yarmohammadi, Karen T. Brown, F. Edward Boas *

* corresponding author; fboas@coh.org

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Abstract

Background

Portal vein stents can be placed for malignant occlusion, but not all patients benefit from the procedure.

Purpose

To evaluate the safety and efficacy of stenting malignant portal vein occlusion or stenosis, to treat bleeding varices or portal hypertensive ascites.

Material and Methods

25 patients with malignant compromise of the portal vein were referred for percutaneous portal vein stent placement to treat bleeding varices ($n=16$), portal hypertensive ascites ($n=7$), or both varices and ascites ($n=2$). Technical and clinical success rates, complications, survival, and stent patency were evaluated.

Results

Mean age was 66, and 18 of 25 (72%) patients had pancreatic cancer. Portal vein stent placement was technically successful in 24 of 25 (96%) patients. One patient (4%) died 10 days post procedure, due to bleeding after tPA thrombolysis of immediate stent thrombosis. Median overall survival after stenting was 4.4 months for patients with ascites, and 8.6 months for patients with bleeding. Primary stent patency at 5 months was 56%. There was a trend towards improved stent patency when the stent diameter was at least 10 mm. 10 of 17 (59%) patients who underwent stenting for variceal bleeding had no further bleeding episodes. 3 of 9 (33%) patients who underwent stenting for ascites had decreased ascites, and no further interventions needed for ascites.

Conclusion

Transhepatic stenting of the portal vein is safe and potentially effective for treating variceal bleeding secondary to malignant portal vein compromise. Patients with ascites had limited benefit and poor survival after portal vein stenting.

Keywords: portal vein stent, pancreatic cancer, portal hypertension, varices, ascites

Introduction

Pancreatic cancer and other malignancies can occlude the portal vein, causing prehepatic portal hypertension. This can result in life-threatening complications such as variceal bleeding, or lead to refractory ascites that reduces the patient's quality of life.

Portal vein stent placement can be performed for malignant portal vein occlusion [1-7], resulting in decreased varices and ascites for many patients. However, not all patients benefit, and optimal patient selection remains unclear.

In this study, we evaluated the safety and efficacy of stenting malignant portal vein occlusion or stenosis, to treat bleeding varices or portal hypertensive ascites. We examined technical success, clinical success (by indication), complications, factors associated with primary stent patency, and survival.

Materials and Methods

Study population

Institutional Review Board approval was obtained for this single institution retrospective study, and the need for informed consent was waived. From January 2011 to September 2020, 25 patients with malignant compromise of the portal vein were referred for percutaneous portal vein stent placement to treat bleeding varices ($n=16$), portal hypertensive ascites ($n=7$), or both varices and ascites ($n=2$). Portal hypertensive ascites was diagnosed based on negative cytology, no peritoneal carcinomatosis, and serum ascites albumin gradient > 1.1 g/dL [8].

Portal vein stenting technique

Procedures were performed under general anesthesia by fellowship-trained interventional radiologists. A peripheral branch of the portal vein or splenic vein was accessed under ultrasound guidance, and a sheath was placed. Initial portal venogram was performed. The portal vein occlusion was crossed with a guidewire and catheter, and a self-expanding stent was placed: SMART stent (Cordis, Miami, FL) or WALLSTENT (Boston Scientific, Marlborough, MA). The nominal stent diameter was 10-20% larger than the target vessel. Post-stent venogram was performed. As the sheath was removed, the tract was embolized using gelatin foam pledgets (SURGIFOAM; Ethicon, Raritan, NJ) in the liver, and coils or vascular plugs (Amplatzer vascular plug 4; Abbott, Chicago, IL) in the spleen. See Figure 1.

Outcomes and statistical analysis

Technical success was defined as the ability to cross the portal stenosis or obstruction and deploy a stent. For patients with variceal bleeding, clinical success was defined as no further episodes of variceal bleeding. For patients with ascites, clinical success is defined as decreased ascites, and no further paracentesis or other interventions needed for ascites.

Complications were classified using guidelines from the Society of Interventional Radiology [9].

Overall survival and stent patency were evaluated using Kaplan Meier analysis. Kaplan Meier curves were compared using a log rank test. $p < 0.05$ was considered statistically significant. Statistical tests were performed in Mathematica 12 (Wolfram Research, Champaign, IL).

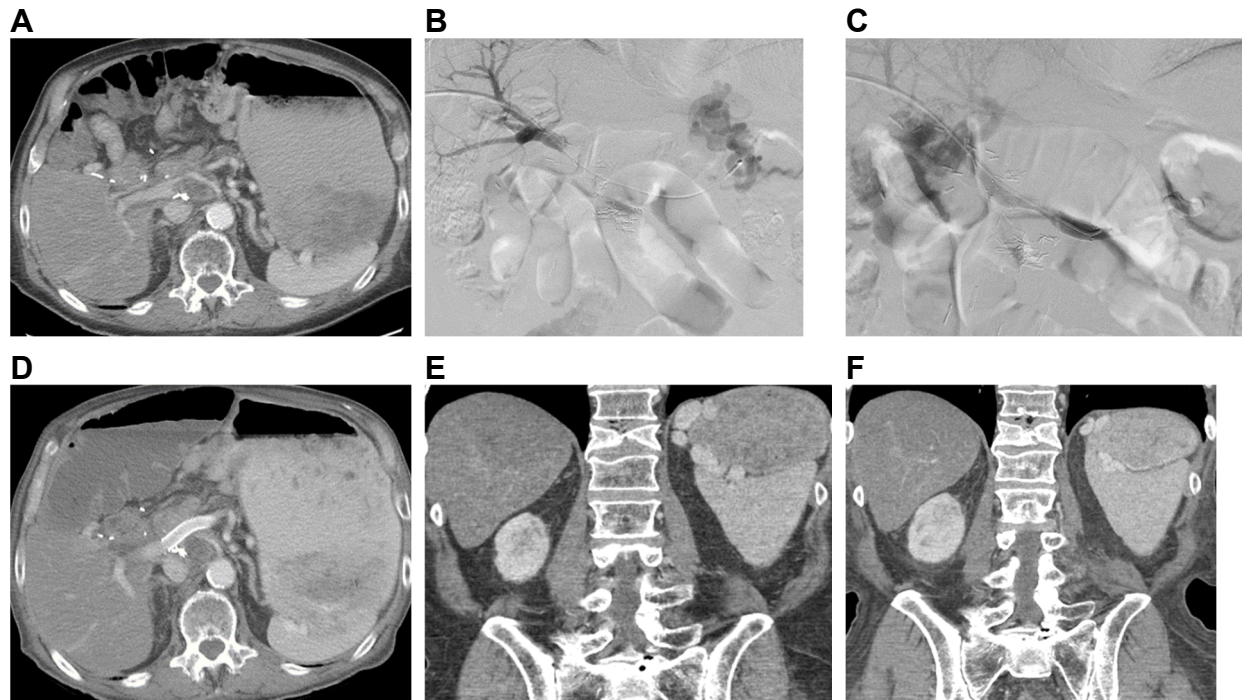


Figure 1. 77-year-old man with recurrent pancreatic cancer after Whipple, with tumor in the portal and splenic veins, causing sinistral portal hypertension and bleeding gastric varices. (A) CT shows tumor occluding the portal/splenic vein. (B) Simultaneous portal and splenic venograms show complete occlusion of the splenic vein extending to the main portal vein, with filling of gastric varices. (C) After portal vein stent placement, venogram shows good hepatopetal flow from the splenic vein through the stent into the portal vein, with no filling of gastric varices. (D) CT image 1 month after stent placement, showing a patent portal vein stent. (E) & (F) Coronal CT images before and 4 months after portal vein stenting, showing interval decrease in gastric varices.

Results

Patient characteristics

Mean age was 66, and 18 of 25 (72%) patients had pancreatic cancer. See Table 1.

Technical success

Portal vein stent placement was technically successful in 24 of 25 (96%) patients (Table 2). 23 of 24 patients were stented from a transhepatic approach. In one patient, a trans-splenic

approach was necessary to cross the occlusion. In the one technical failure, referred for variceal bleeding, the occlusion could not be crossed, and the patient underwent splenic artery embolization. Immediately after stent placement, portography showed improved hepatopedal flow in 22 of 24 (92%) patients, and decreased or no filling of varices in 14 of 17 (82%) patients with that indication.

Characteristic	<i>n</i> =25 patients
Age in years (range)	47-84
Gender	
Male	14 (56%)
Female	11 (44%)
Indication	
Ascites	7 (28%)
Variceal bleeding	16 (64%)
Ascites + Variceal bleeding	2 (8%)
Cancer type	
Pancreatic	18 (72%)
Gall bladder	2 (8%)
Breast	1 (4%)
Biliary	3 (12%)
Gastric	1 (4%)

Table 1. Patient demographics.

Procedural detail	<i>n</i> =25 patients
Access	
Transhepatic	24 (96%)
Trans-splenic	1 (4%)
Technical success	24 (96%)
Stent length (mm)	40-80
Stent diameter (mm)	6-14
Stent location	
PV	6 (25%)
PV to SMV	13 (54%)
PV to splenic vein	4 (17%)
PV to SMV and splenic vein (kissing stents)	1 (4%)
Post-procedural antithrombotics	
Plavix	4 (17%)
Lovenox	6 (25%)
None	14 (58%)

Table 2. Procedure details

Clinical success

10 of 17 (59%) patients who underwent stenting for variceal bleeding had no further bleeding episodes (median follow up interval of 7.4 months). 3 of 9 (33%) patients who underwent stenting for ascites had decreased ascites, and no further interventions needed for ascites (median follow up interval of 2.1 months).

Complications

One patient (4%) died after portal vein stent placement: immediate in-stent thrombosis was treated using tPA thrombolysis, followed by portal vein rupture, treated by covered stent placement, followed by ICU admission for hemothorax, and death 10 days post-procedure. There were no other adverse events.

Survival and stent patency

Median overall survival after stenting was 5.5 months overall, 4.4 months for patients with ascites, and 8.6 months for patients with bleeding.

Primary stent patency at 5 months was 56%. There was a trend towards improved stent patency when the stent diameter was at least 10 mm ($p=0.07$; Figure 2). No difference in stent patency for patients receiving versus not receiving anticoagulation or antiplatelets ($p=0.68$).

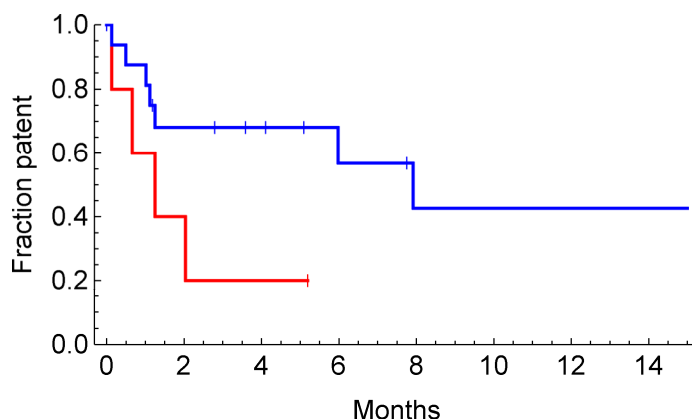


Figure 2. Primary stent patency for ≤ 8 mm (red) and ≥ 10 mm (blue) portal vein stents. $p=0.07$.

Discussion

Portal vein stent placement is a safe and potentially effective treatment for bleeding varices due to malignant portal vein occlusion. If the portal vein occlusion can not be stented, splenic artery

embolization can be performed to reduce splenic vein flow and thus reduce portal pressure. Splenic artery embolization is more effective than endoscopic glue injection into bleeding gastric varices [10]. Sclerotherapy of varices does not address the underlying prehepatic portal hypertension.

On the other hand, patients with ascites had limited benefit and poor survival after stenting the malignant portal vein occlusion. The etiology for ascites can be multifactorial (both portal hypertensive and malignant). In this study, we rigorously excluded malignant ascites, based on negative cytology, no peritoneal carcinomatosis, and serum ascites albumin gradient > 1.1 g/dL. Even in this highly selected patient population, most patients continued to have symptomatic ascites after portal vein stenting. One possible explanation is that ascites might not resolve immediately after portal vein stent placement. After TIPS, ascites can take 3 months to resolve [11]. Patients with ascites and malignant portal vein occlusion have significant mortality from their underlying disease, and median survival is only 4.4 months. Many patients with ascites may not live long enough to see a benefit from portal vein stenting.

There was a trend towards improved stent patency when the stent diameter was at least 10 mm (nominal stent diameter was 10-20% larger than the target vessel). There was no difference in stent patency for patients receiving versus not receiving anticoagulation or antiplatelets. Brisk blood flow through the stent is likely a key determinant of stent patency.

Limitations of this study include the small number of patients, and single center retrospective design. Other possible indications for portal vein stenting, such as thrombocytopenia and liver failure, were not examined.

In conclusion, portal vein stenting is safe and potentially effective for treating variceal bleeding secondary to malignant portal vein compromise. Portal vein stents smaller than 10 mm had poor patency, and those patients could be treated with splenic artery embolization instead. Patients with ascites had limited benefit and poor survival after portal vein stenting.

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KTB is on an advisory board for Instylla.

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