Diagnostic and Interventional Imaging (2019) **100**, 147—156

ORIGINAL ARTICLE /*Interventional imaging*

Percutaneous portal vein recanalization using self-expandable nitinol stents in patients with non-cirrhotic non-tumoral portal vein occlusion

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KEYWORDS
Portal vein occlusion;
Portal hypertension;
Portal vein recanalization

Abstract
Purpose: The purpose of this study was to evaluate the feasibility, safety, and efficacy of portal vein recanalization (PVR) and propose a new classification for better selecting candidates with portal vein occlusion (PVO) in whom PVR could be feasible.
Materials and methods: The charts of 15 non-cirrhotic patients in whom stent placement using a trans-hepatic approach was attempted for the treatment of PVO with cavernous transformation were reviewed. There were 12 men and 5 women with a mean age of 47 ± 12 years (range: 22—60 years). Intrahepatic involvement was classified into 3 groups according to the intrahepatic extent of PVO: type 1 included occlusions limited to the origin of the main portal vein and/or the right or left portal branches, type 2 included type 1 plus extension to the origin of segmental branches, type 3 included type 2 plus extension to distal branches.

Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging; PVO, portal vein occlusion; PVR, portal vein recanalization; TIPS, transjugular intrahepatic portosystemic shunt.

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https://doi.org/10.1016/j.diii.2018.07.009

Results There were 6 patients with PVO type 1, 7 patients with PVO type 2, and 2 patients with PVO type 3. Indications for PVR were gastrointestinal bleeding ($n = 6$), portal biliopathy ($n = 2$), reduce portal pressure before surgery ($n = 4$), or other ($n = 3$). PVR was successful in 13 patients (87%) with no severe side effects. Failure of PVR or early stent thrombosis occurred in 100% of type 3 vs. 8% of type 1 and 2 patients ($P = 0.03$). During a mean follow-up of 42 ± 28 months (range: 6—112 months), patients with a permeable stent had resolution of portal hypertension-related manifestations. In 13 patients in whom PVR was feasible, stent permeability was 77% at 2 years (87% vs. 60% in patients who received anticoagulation or not, respectively; $P = 0.3$).
Conclusion: PVR is feasible in most patients with non-cirrhotic, non-tumoral portal vein occlusion when there is no extension of the occlusion to distal branches.

Introduction

Non-tumoral chronic portal vein occlusion (PVO) occurring outside the setting of cirrhosis is a rare condition, which is thought to account for 5% to 10% of all patients with portal hypertension in developed countries [1]. It can be associated with severe complications such as gastrointestinal bleeding, portal biliopathy, and intestinal ischemia [2,3]. The management of patients with complications related to chronic PVO is a challenge as issues related to both portal hypertension and thrombosis recurrence must be addressed. In patients with symptoms despite conservative treatment with non-specific β-adrenergic blockade, endoscopic band ligation, and/or anticoagulation therapy, shunting procedures may be considered. Due to the high morbidity rates related to surgical techniques, transjugular intrahepatic portosystemic shunt (TIPS) has been used in both cirrhotic [4] and non-cirrhotic patients with PVO [5—7]. However, rates of success are limited and long-term patency is a challenging issue in cases of cavernous transformation of the portal vein [8—10].

Portal vein recanalization (PVR) has been proposed for treating complications related to portal hypertension by addressing PVO itself [11]. Initially, PVR was used in combination with TIPS insertion with the hope that TIPS would help to maintain long-term patency of the portal vein [7,12,13]. However, the benefit of shunting the liver in addition to PVR is not clear in non-cirrhotic patients with no intrahepatic portal hypertension. On the other hand, failure of recanalization and/or early intra-stent thrombosis may occur and depend on the magnitude of intrahepatic extension of PVO. Currently, data on the use of PVR without TIPS are available for only a limited number of patients who were followed-up for a short period of time [11,14,15].

The purpose of this study was to evaluate the feasibility, safety, and efficacy of PVR and propose a new classification for better selecting candidates with PVO in whom PVR could be feasible.

Materials and methods

Patients

The charts of 15 non-cirrhotic patients in whom stent placement was attempted for the treatment of PVO with cavernous transformation between February 2006 and September 2016 were reviewed. There were 12 men and 5 women with a mean age of 47 ± 12 years [range: 22—60 years) (Tables 1 and 2). The mean interval between diagnosis of PVO and PVR was 26 ± 25 months (range: 0—73 months). Indications for PVR were gastrointestinal bleeding ($n = 6$), portal biliopathy ($n = 2$), the need for reduction in portal pressures before abdominal surgery ($n = 4$), and other reasons ($n = 3$). Patients with tumoral PVO or with cirrhosis were excluded. Written informed consent was obtained from all patients.

Diagnosis of PVO was based on the results imaging techniques including ultrasound, computed tomography (CT), and/or magnetic resonance imaging (MRI). Cavernous transformation of the portal vein was defined as the presence of numerous venous collaterals of the helium in the presence of extra-hepatic portal vein thrombosis on the basis of imaging studies. The intra-hepatic extension of the occlusion was assessed by contrast-enhanced CT and/or MRI. All morphological examinations were reviewed by the same trained investigator (AD) and the decision to attempt PVR was made during a multidisciplinary meeting of interventional radiologists, surgeons, and hepatologists aiming at identifying the best therapeutic option in each individual case.

Classification of PVO

Intrahepatic involvement was classified into 3 groups according to the vascular extent of PVO: ''type 1'' included occlusion limited to the origin of the main portal vein and/or to the right or left portal branches, ''type 2'' included type 1 plus extension to the origin of segmental branches, and ''type 3'' included type 2 plus extension to distal branches (Fig. 1).

Recanalization procedure

Percutaneous transhepatic access to the portal vein was performed in all patients using a Neff® introducer set (Cook Medical) (Fig. 2). Access to the portal vein was made through segment V or segment VIII because these segments always represent more than 10% of the whole liver volume and they are the largest segments though which PVR would allow sufficient outflow through the portal vein. For this reason, intrahepatic involvement of PVO was only assessed in these segments. A 5f 40 cm biliary catheter (Soft-vu Berenstein®, AngioDynamics) was inserted into the intrahepatic portal branches. Recanalization was performed using a 0.035 inch angulated stiff hydrophilic guidewire (Terumo). When the

Table 1 Characteristics of 15 non-cirrhotic patients.

	Age, Sex	Etiology of PVO	Clinical presentation and indication for PVR	Management before PVR	Delay between the diagnosis of PVO and PVR (months)	Extension of PVO[a]	PVO extended to mesenteric and/or splenic veins	Feasibility of PVR	Gradient of portal venous pressure above/below the PVO before the PVR (mmHg)	Correction of the gradient of portal venous pressure above/below the PVO after the PVR	Duration of follow-up after PVR (months)	Permeability of the stent at the end of the follow-up	Clinical outcome after PVR
1	39, M	Local inflammation related to chronic pancreatitis	Recurrent bleeding from jejunal varices	Endoscopic treatment, no anticoagulation therapy	15	Type 1	Yes	Yes	4	Yes	20	Yes	No further episode of bleeding
2	53, M	Local inflammation related to chronic pancreatitis	Abdominal pain, PVR before surgery	No anticoagulation therapy	6	Type 1	No	Yes	7	Yes	73	Yes	Abdominal surgery done
3	53, M	Unknown	Jaundice, abdominal pain	Anticoagulation therapy	36	Type 3	Yes	No	—	—	28	NA	Bleeding, cholangitis
4	57, M	Local inflammation related to chronic pancreatitis	Abdominal pain, PVR before surgery	No anticoagulation therapy	6	Type 2	No	Yes	9	Yes	22	No, development of partial intra-stent thrombosis after 2 months and a complete thrombosis after 6 months	Abdominal surgery done
5	55, M	Local inflammation related to chronic pancreatitis	Recurrent variceal bleeding	No anticoagulation therapy	3	Type 1	No	Yes	NA	—	47	Yes	No further episode of bleeding after PVR
6	60, F	Unknown	Recurrent bleeding from gastric varices	Persistence of an occlusion of the main portal vein despite anticoagulation therapy	18	Type 1	Yes	Yes	7	Yes	10	Yes	No further episode of bleeding after PVR

Table 1 (*Continued*)

	Age, Sex	Etiology of PVO	Clinical presentation and indication for PVR	Management before PVR	Delay between the diagnosis of PVO and PVR (months)	Extension of PVO[a]	PVO extended to mesenteric and/or splenic veins	Feasibility of PVR	Gradient of portal venous pressure above/below the PVO before the PVR (mmHg)	Correction of the gradient of portal venous pressure above/below the PVO after the PVR	Duration of follow-up after PVR (months)	Permeability of the stent at the end of the follow-up	Clinical outcome after PVR
7	25, M	Presence of anticardiolipin antibody positivity	Abdominal pain	Progression of the thrombosis despite antico-agulation therapy	48	Type 2	Yes	Yes	5	Yes	47	Yes	No clinical manifestation after PVR
8	59, M	Local inflammation related to chronic pancreatitis	Abdominal pain, recurrent bleeding from gastric varices	No anticoagu-lation therapy	18	Type 3	No	Yes	3	No	50	No, occlusion of the stent shortly after the procedure	Persistent variceal bleeding treated endoscopically
9	49, M	Unknown	Abdominal pain, PVR before surgery	No anticoagu-lation therapy	96	Type 2	No	Yes	NA	—	51	No, development of an intra-stent thrombosis 3 years after the procedure	Abdominal surgery done
10	49, M	Factor V Leiden and prothrombin gene mutations	Recurrent variceal bleeding	Anticoagulation therapy	144	Type 2	No	Yes	11	Yes	60	Yes	No clinical manifestation after PVR
11	33, F	Hormonal therapy	Abdominal pain, presence of portal hypertension and desire for pregnancy	Anticoagulation therapy	18	Type 2	No	No	—	—	60	NA	Endoscopic treatment of the varices

Table 1 (*Continued*)

	Age, Sex	Etiology of PVO	Clinical presentation and indication for PVR	Management before PVR	Delay between the diagnosis of PVO and PVR (months)	Extension of PVO[a]	PVO extended to mesenteric and/or splenic veins	Feasibility of PVR	Gradient of portal venous pressure above/below the PVO before the PVR (mmHg)	Correction of the gradient of portal venous pressure above/below the PVO after the PVR	Duration of follow-up after PVR (months)	Permeability of the stent at the end of the follow-up	Clinical outcome after PVR
12	45, F	Local inflammation following intra-abdominal surgery	Recurrent bleeding from esophageal varices	No anticoagulation therapy	9	Type 1	No	Yes	10	Yes	38	Yes	No further episode of bleeding after PVR
13	58, M	Local inflammation related to chronic pancreatitis	Abdominal pain, jaundice, PVR before surgery	No anticoagulation therapy	3	Type 1	Yes	Yes	14	Yes	112	No, development of an intra-stent thrombosis 28 months after the procedure	Abdominal surgery done
14	22, M	Mutation of MTHFR	Recurrent bleeding from gastric varices	No anticoagulation therapy	96	Type 2	Yes	Yes	20	Yes	6	Yes	Disappearance of gastric varices 1 month after the PVR. No further episode of bleeding after PVR
15	43, M	Antithrombin III deficiency	Jaundice, symptoms related to portal biliopathy	Anticoagulation therapy	120	Type 2	Yes	Yes	5	Yes	7	No, development of an intra-stent thrombosis 6 months after the procedure	No symptoms related to cholantitis after PVR

AE: adverse event; F: female; M: male; MPV: main portal vein; MTHFR: Methylenetetrahydrofolate Reductase; NA: not available; PVO: portal vein occlusion; PVR: portal vein recanalization.
[a] Type 1, Occlusion limited to the origin of the main portal vein with or without right and/or left portal branches; Type 2, Type 1 + extension to the origin of segmental branches; Type 3, Type 2 + extension to distal branches.

Table 2 Baseline characteristics in 15 non-cirrhotic patients.

Age (years)[a]	47 ± 12 [22–60]
Gender	
Male	12
Female	3
Cavernoma	
Yes	15 (15/15; 100%)
No	0 (0:15; 0%)
Classification of PVO[b]	
Type 1	6 (6/15; 40%)
Type 2	7 (7/15; 47%)
Type 3	2 (2:15; 13%)
Extension of the occlusion to mesenteric and/or splenic veins	
Yes	7 (7/15; 47%)
No	8 (8/15; 53%)
Mean prothrombin ratio (%)	77 ± 27
Mean ASAT (IU/L)	33 ± 18
Mean ALAT (IU/L)	43 ± 34
Total bilirubin level (umol/L)	35 ± 82
Platelets count level (10^3/mm^3)	277 ± 121
Albumin level (g/L)	36 ± 6

PVO: portal vein occlusion.
[a] Data are expressed as mean ± standard deviation [range].
[b] Classification of PVO: Intrahepatic involvement was classified into 3 groups according to the vascular extent of PVO: ''Type 1'' included occlusion limited to the origin of the main portal vein and/or to the right or left portal branches, ''Type 2'' included type 1 plus extension to the origin of segmental branches, and ''Type 3'' included type 2 plus extension to distal branches.

mesenteric vein was reached, 5000 units of heparin were administered and pressures above and below the PVO were measured. A portogram was obtained below the PVO. The occluded segment was first dilated to 6 mm using a 6/40 mm balloon (Passeo® 0.035, Biotronik) and a self-expandable nitinol stent (S.M.A.R.T. control®, Cordis) was placed and then dilated to 10 mm. Post-stent portography and new portal pressure measurements were then obtained. Finally, the intrahepatic access route was embolized using 5/3 mm Tornado® coils to avoid bleeding. The length of the stent was chosen to cover the whole obstruction. When necessary, two stents were used.

Outcomes

The main end-point was to evaluate the feasibility of PVR in this setting. Secondary aims were to evaluate the efficacy of PVR, the patency of the stent during follow-up, and the safety of PVR. The patency of the stent was assessed by ultrasound and/or computed tomography (CT).

Statistical analysis

Variables with normal distribution were expressed as mean ± standard deviation (SD). Skewed variables were expressed as median with interquartile ranges. Analyses were conducted using variance analysis, the Chi^2 test, the two-sided Fisher exact test, the Mann–Whitney test, the Wilcoxon test, and a two-sample Student's *t*-test, when appropriate. All statistical testing was two-tailed at the 5% level. Follow-up started at the inclusion of patients. Stent permeability was assessed by the Kaplan–Meier method and compared using the log-rank test. Survival was expressed as percentage ± standard error. All statistical analyses were performed using NCSS 2016 software (NCSS, Kaysville, UT, USA).

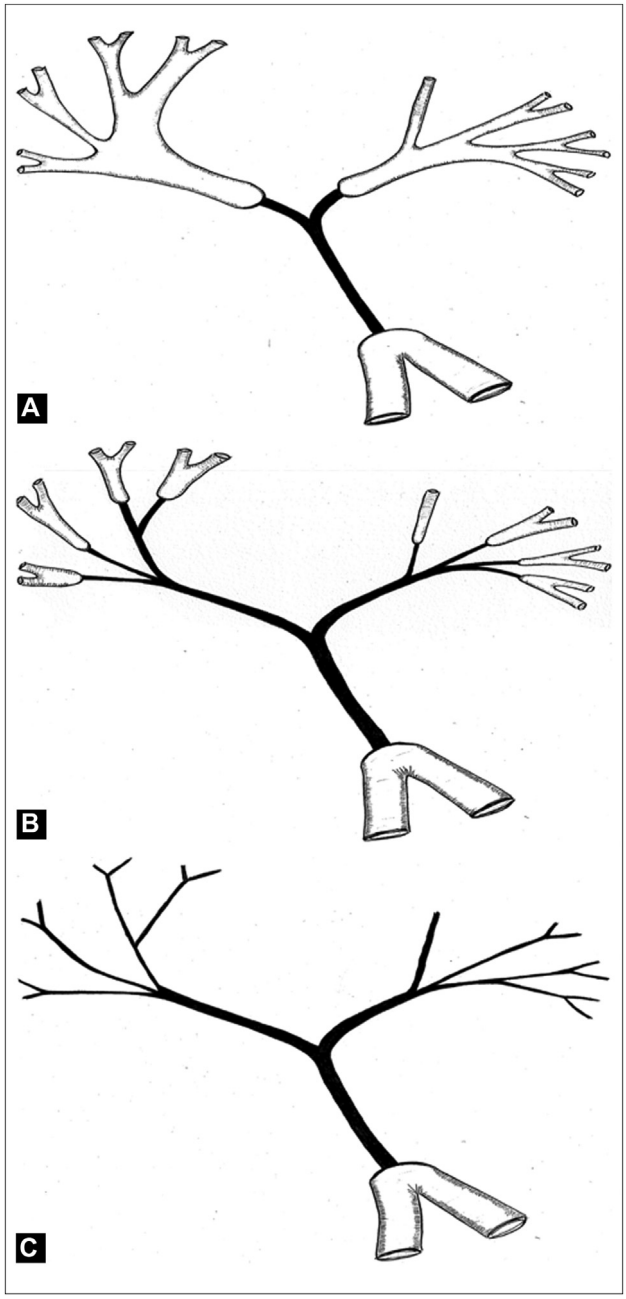

Figure 1. Drawings show new classification for portal vein occlusion (PVO). A. Drawing shows Type 1 PVO that corresponds to occlusion limited to the origin of the main portal vein with or without right and/or left portal branches. B. Drawing shows Type 2 PVO that corresponds to Type 1 + extension to the origin of segmental branches. C. Drawing shows Type 3 PVO that corresponds to Type 2 + extension to distal branches.

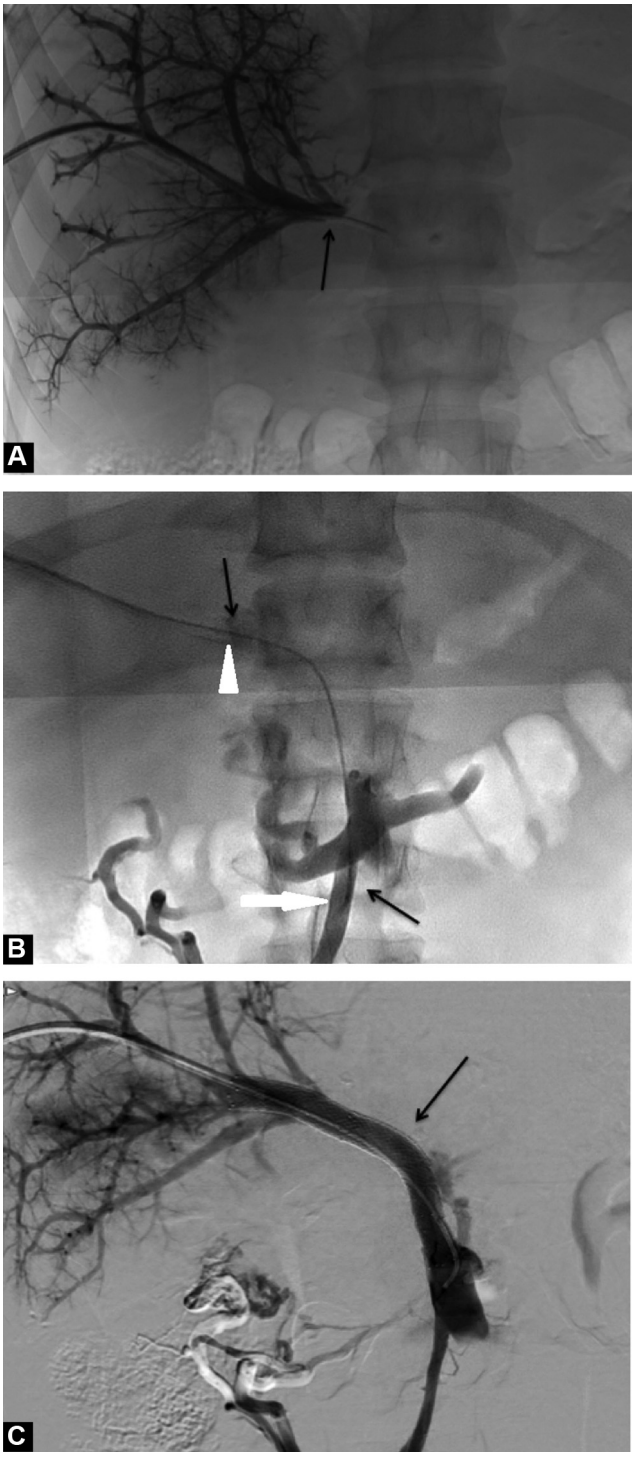

Figure 2. Portal vein recanalization (PVR) in a 22-year-old man (patient 14) with a complete portal vein occlusion (type 2 PVO). A. Percutaneous transhepatic access to the portal vein was performed through segment VIII of the liver. A 5Fr 40 cm biliary catheter (arrow) was placed into the intrahepatic portal branches. B. Portogram obtained below the PVO shows a patent superior mesenteric vein (white arrow) and the occluded main portal vein (arrowhead). Black arrows indicate catheter. C. Portogram after stent placement shows Nitinol self-expandable stent (Smart control®, Cordis) (arrow) in the occluded segment.

Results

Patients

The main characteristics of the 15 patients are presented in Tables 1 and 2. PVR was successful in 13 patients (87%). The rate of success of PVR was not related to the interval between diagnosis of PVO and PVR (19 vs. 25 months in patients in which PVR was successful compared to those in which it was not, respectively, $P = 0.9$). All patients had chronic occlusion of the main portal vein, either with ($n = 7$) or without ($n = 8$) occlusion of the mesenteric and/or the splenic veins. The intrahepatic extent of PVO was determined to be ''type 1'' PVO in 6 patients, ''type 2'' in 7 patients, and ''type 3'' for 2 patients. Intra-abdominal risk factors for thrombosis were identified in 8 patients (53%) and systemic risk factors for thrombosis were identified in 5 patients (33%).

Feasibility, clinical efficacy, and safety of PVR

Follow-up data are reported in Tables 1—3. The mean follow-up was 42 ± 28 months (range: 6 — 112 months). PVR was successful in 13 patients (87%). Technical failure was related to the failure of the placement of a guidewire through the occlusion to reach a patent segment of the portal venous system. The rate of success of PVR was not related to the duration of PVO (19 months in patients in which PVR was successful vs. 25 months in those in which it was not; $P = 0.9$). Failure of PVR occurred in 2 patients: one with Type 2 PVO and one with Type 3 PVO. The other patient with Type 3 PVO developed stent thrombosis 24 hours after recanalization. Overall, failure of PVR or early stent thrombosis occurred in 100% of Type 3 patients vs. 8% of Type 1 and Type 2 patients ($P = 0.03$). Extension of the occlusion to the mesenteric and/or the splenic veins had no impact on the feasibility of PVR or on stent patency during follow-up.

In patients who had undergone PVR for portal hypertension-related complications (gastrointestinal bleeding or symptomatic portal biliopathy), the only patient who developed recurrent manifestations of portal hypertension developed early stent thrombosis (patient 8 in Table 1). Another patient developed stent thrombosis 6 months after the procedure and was lost to follow-up thereafter without recurrent portal hypertension-related symptoms. All other patients ($n = 6$) did not develop intra-stent thrombosis and were asymptomatic during follow-up.

No patients developed severe adverse events related to PVR. The only complication related to PVR was a subcapsular hematoma without any harmful consequences. Two patients died during the follow-up, one from sepsis that was not related to the procedure or to complications of portal hypertension 26 months after the procedure, and one from unknown cause 42 months after the procedure.

Predictability of intrahepatic portal vein occlusion extension with preoperative imaging

Preoperative imaging performed within 3 months before PVR, poorly predicted intrahepatic portal extension. Preoperative imaging identified only 50% of the cases with distal

Table 3 Success and thrombosis rates following portal vein recanalization for portal vein occlusion in 15 non-cirrhotic patients.

Classification of PVO[a]	Feasibility of PVR (*n* feasible/*n* total; %)	Early (≤ 24 hours) stent thrombosis (*n* thrombosi/*n* performed; %)	Stent thrombosis at 2 years (*n* thrombosis/n performed; %)
Type 1	6/6 (100)	0/6 (0)	0/6 (0)
Type 2	6/7 (86)	0/6 (0)	2/6 (33)
Type 3	1/2 (50)	1/1 (100)	—

PVO: portal vein occlusion; PVR: portal vein recanalization.
[a] Type 1, Occlusion limited to the origin of the main portal vein with or without right and/or left portal branches; Type 2, Type 1 + extension to the origin of segmental branches; Type 3, Type 2 + extension to distal branches.

intrahepatic extension of PVO and had a negative predictive value of 33% (Table 4).

Patency of the stent during follow-up

In 13 patients in whom PVR was feasible, the actuarial probability of stent patency was 77% at 2 years. Anticoagulation was given to 10 patients after recanalization (77%, median duration: 180 days). The actuarial probability of stent patency at 2 years was 87% in patients who received anticoagulation and 60% in patients who did not receive anticoagulation ($P = 0.3$). No differences in portal pressure gradient at 2 years of follow-up was found between patients with patent stent (10 ± 5 [SD] mmHg; range: 4 — 20 mmHg) and those without patent stent (6 ± 3 [SD] mmHg; range: 3 — 9 mmHg) ($P = 0.2$).

Discussion

Despite conservative therapy, many patients with PVO suffer from complications related to portal hypertension. Current guidelines do not indicate which non-cirrhotic patients should be considered for PVR in cases of PVO [3,16]. In this article, we propose a new classification of PVO to help clinicians decide when PVR should be attempted.

The most important finding is that PVR was successful in all but one patient when at least a portion of the segmental branches of the largest liver segments (V or VIII) remained patent (type 1 or 2 PVO), regardless of the interval between diagnosis of PVO and PVR. By contrast, a deeper intrahepatic extension (type 3 PVO) was always associated with PVR failure or with early stent thrombosis due to insufficient blood outflow. Thus, PVR alone should not be considered in these patients. Of note, image fusion for guidance was not needed for the procedure since intrahepatic branches were always visible [17]. One unsolved issue is whether a TIPS should be inserted when PVR is performed [18,19]. As the aim of the PVR is to alleviate portal hypertension, the choice between PVR alone and the combined approach could take into account the existence of intrahepatic portal hypertension. TIPS would likely be useful when portal hypertension is also related to an intrahepatic block of the portal circulation, as in patients with type 3 PVO. However, as intrahepatic pressures are normal in most patients with PVO [20], the benefit of adding TIPS to PVR is unclear and should be evaluated on an individual basis.

The second key finding of our study is that the feasibility of PVR was not predictable using preoperative imaging. In our experience, preoperative imaging lacked diagnostic accuracy in predicting when PVO was not feasible. Thus, portography performed at the beginning of the procedure is mandatory for deciding whether PVR should be attempted. In any case, the selection of candidates for PVR should be made during a multidisciplinary meeting that involves experts in the field of portal hypertension.

From a clinical perspective, all but one patient in whom PVR was decided upon for treating complications of portal hypertension experienced immediate relief of

Table 4 Predictability of feasibility of portal vein recanalization according to preoperative imaging in 15 non-cirrhotic patients.

Classification of PVO according to portography results[a]	Predicted type 1 or 2 PVO with preoperative imaging	Predicted type 3 PVO with preoperative imaging
Type 1 or 2	11/13 (83%)	2/13 (17%)
Type 3	1/2 (50%)	1/2 (50%)

PVO: portal vein occlusion; PVR: portal vein recanalization.
[a] Type 1, Occlusion limited to the origin of the main portal vein with or without right and/or left portal branches; Type 2, Type 1 + extension to the origin of segmental branches; Type 3, Type 2 + extension to distal branches.

symptoms when PVR was feasible. The only patient who developed recurrent manifestations of portal hypertension had no improvement of portal pressure gradient after PVR because of early stent thrombosis. Other patients had resolution of manifestations related to portal hypertension. Thus, it was not possible to assess whether the magnitude of the changes in portal pressure was correlated with clinical efficacy. In addition, there was no correlation between changes in portal pressure gradient before PVR and stent patency at 2 years. However, long-term follow-up was not available in all patients. It is likely that the length of the period free of portal hypertension-related complications depends on long-term stent patency. In line with this issue, the role of anticoagulation in preventing stent occlusion following PVR is unknown. As there is no recommendation on the use of anticoagulation therapy following PVR, this decision was made on a case-by-case basis. As a rule, long-term anticoagulation therapy was given in cases involving thrombophilia and/or past history of recurrent thrombosis, as is the case in PVO outside the setting of PVR [2,3]. While 2-year patency of the stent was not significantly different between patients receiving and those not receiving anticoagulation, the numerical difference observed between these 2 groups may indicate that anticoagulation could protect against recurrent thrombosis, at least in patients with thrombophilia and/or past history of recurrent thrombosis. Whether other factors, such as the velocity of the blood within the stent, should be taken into account should be addressed in prospective studies.

Another important clinical issue was related to the safety profile of PVR. Among the 15 patients, no severe adverse events occurred. A single patient developed subcapsular hematoma that did not require interventional procedure. Although our experience is still limited, PVR may be viewed as a safe procedure, assuming it is performed by trained physicians.

We acknowledge that this observatory has some limitations including the limited number of patients, its retrospective design, and the lack of data about the total number of patients with PVO during the study period as well as the reasons why a patient was not considered as a candidate for PVR. However, it was anticipated that PVR would be considered in only a few patients, even in a reference center. On the other hand, this observatory has several strengths. In addition to the fact that all procedures were performed by the same trained investigator, this series of patients without cirrhosis with PVO treated with PVR is the largest to date and assessment of stent patency was available over a sufficient duration of time to allow a first inspection.

In conclusion, PVR is safe and provides resolution of portal hypertension-related manifestations. The extension of intrahepatic involvement of PVO may help to select which patients could be considered for PVR.

Financial support

None.

Acknowledgment

We acknowledge the contribution of Sandy Field, Ph.D., for her assistance concerning English-language editing.

Disclosure of interest

The author declares that he has no competing interest.

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