

Transjugular intrahepatic portosystemic shunt for portal vein thrombosis with and without cavernous transformation

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SUMMARY

Background

Treatment options for patients with portal vein thrombosis are limited.

Aim

To evaluate the feasibility and efficacy of transjugular intrahepatic portosystemic shunt for portal vein thrombosis with/without cavernomatous transformation.

Methods

A survey of such patients, referred for transjugular intrahepatic portosystemic shunt between 1994 and 2005, was performed. Success rates, complications, transjugular intrahepatic portosystemic shunt patency and clinical progression were examined.

Results

Transjugular intrahepatic portosystemic shunt was attempted in 28 patients (13 cirrhotics). Indications were: presurgery/transplantation (2), worsening of ascites (2), variceal bleeding (15 – 8 elective), refractory ascites (3), portal biliopathy (3) and portal vein thrombosis complicating Budd–Chiari syndrome (2). Transjugular intrahepatic portosystemic shunt was placed successfully in 19 of 28 (73%); 23 of 28 had complete portal vein thrombosis and 9 of 23 had cavernous transformation and transjugular intrahepatic portosystemic shunt was successfully placed in six of these. In the 19 patients with transjugular intrahepatic portosystemic shunt, the mean follow-up was 18.1 months (range 5–70): six patients had stent revisions; three had liver transplantation, one died of bleeding. Most cirrhotic patients had an improvement in the Child–Pugh score. In the failed transjugular intrahepatic portosystemic shunt group, two of nine died, and three had further bleeding.

Conclusions

Transjugular intrahepatic portosystemic shunt should be considered for selected patients with symptomatic complete portal vein thrombosis with/without cavernous transformation, as clinical improvement and less rebleeding occur when transjugular intrahepatic portosystemic shunt placement is successful.

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INTRODUCTION

Portal vein thrombosis (PVT) in the absence of cirrhosis is rare. In adults a thrombophilic tendency should be suspected, whilst in children umbilical vein sepsis and intra-abdominal infection are the predominant causes.^{1, 2} PVT can also complicate Budd–Chiari syndrome in 20% of cases leading to increased mortality.³

In the absence of cirrhosis, liver function is usually well preserved, but the significant portal hypertension may result in serious/life-threatening complications, including variceal bleeding, intestinal venous congestion and ischaemia,^{1, 4} as well as ascites in elderly patients according to our clinical experience. Rarer complications include growth failure in children, bile duct compression and cholangitis from hilar varices, and protein-losing enteropathy.^{1, 5, 6} In addition, portal hypertension increases the risk of abdominal surgery.

When there is cirrhosis (in the absence of hepatocellular carcinoma), PVT has a prevalence of between 1% and 16%.^{7, 8} It is reported to be more frequent with autoimmune cirrhosis and more advanced liver disease,^{9, 10} and is thought to occur as a consequence of reduced portal vein flow secondary to raised hepatic resistance. The onset of PVT leads to life-threatening complication such as mesenteric infarction, and/or variceal bleeding and worsening of ascites.⁹

Whilst PVT is not a contraindication to liver transplantation, extension of thrombus into the remaining splanchnic vessels can severely reduce splanchnic vein inflow into a transplanted liver and result in graft failure.^{11–14}

There is no established management algorithm for patients with PVT. Early anticoagulation is recommended when the thrombus is recent and may reestablish the patency in some.¹⁵ Local and systemic thrombolysis has also been used¹⁶ but are only applicable in the presence of recent thrombus, and in the absence of bleeding. Children with localized thrombosis may be treated with meso-portal bypass shunts,¹⁷ and likewise adults with limited thrombosis may be treated by surgical shunts.¹

The advent of transjugular intrahepatic portosystemic shunt (TIPS) has enabled non-surgical shunting to be considered, and access to the portal vein has allowed localized thrombosis to be mechanically disrupted. There are case reports where TIPS (sometimes combined with a percutaneous approach) has been

reported to re-establish portal vein patency, but these are limited in numbers,^{18–24} and indeed PVT in association with cavernous transformation has been seen as a contraindication to TIPS.²⁵

We report our experience of TIPS in patients with PVT including cavernomas, the changing technique over time, and factors that appear to be associated with a successful outcome.

PATIENTS AND METHODS

From January 1994 to June 2005, 450 patients with portal hypertension underwent TIPS placement at our hospital: 28 patients (6%) had PVT. The diagnosis of PVT was established before the procedure, based on Doppler ultrasound, computed tomography scan or magnetic resonance imaging of the liver and in a few patients a suspected diagnosis was confirmed during the TIPS procedure by either angiography or CO₂ portography. No patients included in this series had evidence of hepatocellular carcinoma.

In non-cirrhotic patients and in those who had a previous history of thrombosis a thrombophilic screening was performed.

The thrombus was defined as an occluding thrombus when occupying more than 90% of the circumference of the vessel. Age of the thrombus was estimated by the clinical history when possible or by comparison with previous imaging where available. Thrombus was arbitrarily defined as new if there was a recent episode of abdominal pain associated with radiological findings of PVT with no evidence of chronic portal hypertension, and absent portal collaterals on cross-sectional imaging. Cavernous transformation was defined as the presence of numerous venous collateral in the hilum in the presence of PVT.

The initial part of the TIPS procedure was performed in a standard fashion. The right internal jugular vein was punctured under ultrasound guidance and the TIPS sheath inserted into a selected hepatic vein. The portal venous system was accessed by puncturing a non-thrombosed intrahepatic branch in most cases with a combination of ultrasound guidance and CO₂ injection. Usually a hydrophilic wire was required to negotiate into the main portal vein. In the presence of cavernomatous transformation, the technique was similar, but required manipulation of a catheter behind the hydrophilic wire to dilate a channel within the collateral veins of the

cavernoma, leading to the main trunk of the portal vein.

Following dilatation of the intrahepatic tract, as well as the thrombosed portal vein, a stent was deployed, ensuring the distal end would not compromise future surgical procedures such as transplantation. When there was evidence of persistent portal, splenic or mesenteric thrombus, balloon dilatation as well as disruption of thrombus by manual rotation of a 5 french pigtail catheter was employed. At this time point, in selected patients a bolus of 5000 IU of heparin was administered into the portal circulation. Successful thrombectomy was defined as the complete absence of filling defects and optimal flow during contrast injection in the vessel examined.

If the clot remained adherent, and the flow was poor, then the stent was extended across the thrombus. If the tract from the hepatic vein puncture was not fully covered another stent was deployed. In patients with Budd–Chiari syndrome when there was no patent hepatic vein, the procedure was modified and direct puncture of the intrahepatic inferior vena cava was performed under ultrasound guidance.

Factors which may influence the successful rate were visualization of intrahepatic branches of PVT during the procedures, presence/absence of cavernoma, complete/partial PVT, aetiology (cirrhotic/non-cirrhotic PVT) and knowledge of the thrombus prior to the procedure.

We followed up all patients regardless of the technical success. Baseline characteristics such as severity of liver disease by Child–Pugh score, subsequent further episodes of bleeding or hepatic encephalopathy were recorded.

Stent patency was evaluated with Doppler ultrasound within 1 week after the procedure and every 6 months. A tipsogram was routinely performed at 12 months and every time was indicated by reduction of bleed flow at ultrasound or clinically (recurrence ascites/bleeding). The end points during follow-up were defined by liver transplantation or death.

RESULTS

Our cohort had 20 males and eight females with a mean age at procedure of 45 years (range 17–65). Underlying liver disease was present in 16 of 28 patients. The remaining 12 patients had primary PVT without underlying liver disease.

Indications for TIPS placement were emergency variceal bleeding in six, emergency bleeding from

colonic varices in one, elective control of variceal bleeding in eight having failed both endoscopic and medical therapy, refractory ascites in three, treatment of Budd–Chiari syndrome in two and portal-biliopathy in three patients. Five patients underwent TIPS procedure because of the PVT itself: one patient presented acutely and TIPS was attempted to unblock the portal vein, two further patients had worsening ascites, one patient had extensive thrombus and was on the liver transplant list, and the fifth patient had a TIPS attempted prior to undergoing hepatic resection. The patients' characteristics, symptoms and indications for TIPS are summarized in Table 1. The presence of thrombophilic disease, vessel involvement, extent and age of thrombosis for every patient are summarized in Table 2. Predisposing local factors and prothrombotic disorders were found in eight patients, seven were non-cir-

Table 1. Patient characteristics and indications for TIPS procedure

<i>Patient characteristics</i>	
No. patients	28
Sex ratio (m/f)	20/8
Mean age at procedure	45 years (range 17–65)
<i>Aetiology</i>	
Non-cirrhotic PVT	12
HCV-PNC	3
ALD-PNC	7
Cryptogenetic PNC	2
Budd–Chiari syndrome	2
Other	2
<i>Indications for TIPS placement</i>	
Emergency variceal bleeding	6
Emergency bleeding from colonic varices in	1
Elective – failed endoscopic and medical therapy for prevention of variceal rebleeding	8
<i>Treatment of PVT</i>	
Acute portal thrombosis	1
Sudden worsening of ascites	2
Pretransplant	1
Prior hepatic resection (non-malignant)	1
Refractory ascites	3
Budd–Chiari syndrome	2
Portal-biliopathy	3

TIPS, transjugular intrahepatic portosystemic shunt; PVT, portal vein thrombosis; HCV, hepatitis C virus; PNC, post necrotic cirrhosis.

Table 2. Predisposing factors to thrombosis, characteristic of portal vein thrombus and associated thrombosed vessels

Case no.	Predisposing factor	Known thrombus TIPS	Age before thrombus	Portal thrombus	Cavernoma	SMV thrombosis	Splenic thrombosis
1*	Cirrhosis	No	Old	Total	-	-	-
3*	Paroxysmal nocturnal haemoglobinuria	Yes	3 months	Total	-	+	+
4*	Cirrhosis	Yes	15 days	Total	-	+	-
6*	Cirrhosis	Yes	Unknown	Partial	-	+	-
9*	Cirrhosis	No	9 months	Partial	-	-	-
10*	Cirrhosis	Yes	Unknown	Total	+	-	-
11*	Cirrhosis	No	Unknown	Partial	-	-	-
12*	Unknown	Yes	1 year	Total	+	+	-
13*	Unknown	Yes	6 months	Total	+	+	-
14*	Unknown	Yes	3 years	Total	+	-	+
15*	Cirrhosis	No	Unknown	Partial	-	-	-
16*	Cirrhosis	No	Unknown	Partial	-	-	-
18*	Unknown	Yes	Unknown	Total	-	+	-
21*	Myeloproliferative disease	Yes	Old	Total	-	+	+
23*	Chronic pancreatitis	Yes	Old	Total	+	-	+
24*	Cirrhosis	Yes	2 months	Total	-	+	-
25*	Cirrhosis	Yes	1 month	Total	-	-	-
27*	Factor V mutation/ hepatic abscess	Yes	13 years	Total	+	+	-
28*	Sarcoidosis	Yes	7 years	Total	-	+	-
2	Cirrhosis	No	Unknown	Total	-	-	-
5	Increase factor VIII	Yes	Old	Total	-	-	+
7	Myelodysplasia deficit protein C and S	Yes	Unknown	Total	-	-	-
8	Protein C deficit	Yes	Unknown	Total	-	+	-
17	Cirrhosis	Yes	11 months	Total	+	+	-
19	Cirrhosis	Yes	2 years	Total	+	-	+
20	Recurrent local infections	Yes	10 months	Total	-	+	+
22	Unknown	Yes	Old	Total	+	-	+
26	Unknown	Yes	Unknown	Total	-	-	-

TIPS, transjugular intrahepatic portosystemic shunt; SMV: superior mesenteric vein

* TIPS placed successfully.

rhotics and one had Budd–Chiari syndrome. Prothrombotic disorders were paroxysmal nocturnal haemoglobinuria in one, increased factor VIII in one, myelodysplasia associated with protein C and S deficiency in one, protein C deficiency in one, myeloproliferative disorder in one and local prothrombotic factors in three patients (recurrent local infections in one, chronic pancreatitis in one and multiple hepatic abscesses in one). Only two patients received anticoagulation before TIPS, as those referred with active or recent bleeding had a contraindication to anticoagulation or being cirrhotic their International Normalized Ratio (INR) was already above 2.

Total occlusion (90% of the lumen) was found in 23 patients, and 9 of 23 had evidence of cavernous transformation.

Transjugular intrahepatic portosystemic shunt was successfully placed in 19 of 28 patients (73%) (Figures 1 and 2). In cases 2, 20 and 22, we successfully punctured a non-thrombosed intrahepatic branch of the portal vein, but it was impossible to negotiate the wire into the main PV. In cases 5, 7, 8, 19 and 26, no patent intrahepatic branches of the PV were visualized during the procedure. In case 17, because of a shrunken liver, a biliary and capsular puncture occurred and the procedure was abandoned because of the high risk of complications with

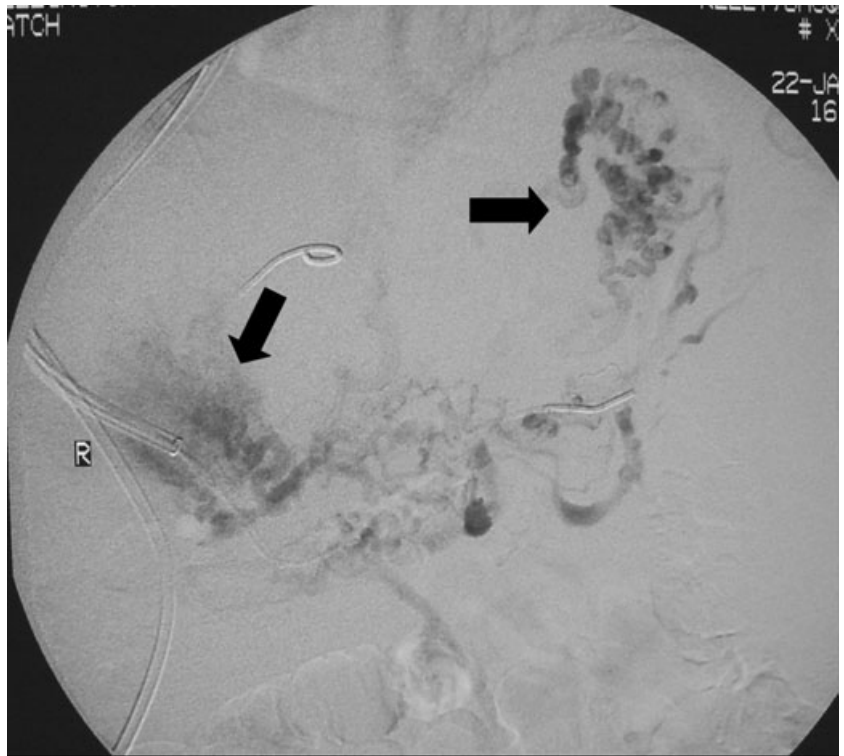


Figure 1. Portal venogram following transjugular intrahepatic puncture of the portal vein shows varices and contrast accumulation within the portal cavernoma (arrows).



Figure 2. Following transjugular intrahepatic portosystemic shunt placement, balloon dilatation and mechanical thrombectomy of the portal vein, vein variceal filling is reduced and contrast accumulation within the cavernoma is no longer seen indicating decompression.

further attempts. In three of the nine patients with technical failure there was cavernous transformation.

Procedural complications were four capsular perforations (three coils were placed without sequelae) and three biliary punctures (all without sequelae in the

failed TIPS group). In the patients in whom TIPS was placed, there were six capsule puncture without any sequelae (only one needed a coil) and one extra hepatic portal vein laceration which was successfully treated with a covered stent.

A total of 26 Memotherms (Angiomed GmbH and Co., Karlsruhe, Germany) and three Viator Gore (WL Gore and Associated, Flagstaff, AZ, USA) stents were used. In 10 patients, two stents were inserted during the first procedure to cover the entire tract from the punctured HV, and nine patients received only one stent. The stent was deployed in the portal vein in 15 patients, in the superior mesenteric vein in three and in a hilar collateral in one. Due to the absence of patent hepatic veins in case 21 the shunt was created puncturing the intrahepatic inferior vena cava (IVC). In all patients who were successfully stented we attempted to disrupt the thrombus with a pigtail catheter, balloon angioplasty or both. This aspect of the procedure was successful in 11 of 19 patients (58%), whereas in the others, the stent was deployed across the thrombus. In three patients heparin bolus of 5000 IU was infused during thrombus disruption.

Analysing the factor predicting success of the procedure, size of the thrombus (partial/total), age of thrombus, presence of cavernoma, aetiology of the PVT were not different between the failed and the successful TIPS groups, and so were not able to predict the failure. Only the absence of a visible patent intrahepatic portal vein branch during the procedure was associated with failure to access the PV; the difference was significant among the two groups ($P = 0.008$, Fisher exact test).

Ten patients (53%) were anticoagulated following TIPS placement. One who had Budd–Chiari syndrome received warfarin before the procedure. Nine other patients started anticoagulation after TIPS placement (one Budd–Chiari, four non-cirrhotic PVT and four cirrhotics), the others were not anticoagulated because of high INR or previous bleeding and in one non-cirrhotic PVT anticoagulation monitoring was not possible). During follow-up, three of the nine anticoagulated patients required stent revision vs. two of eight non-anticoagulated, thus the difference was not significant.

Clinical course

Failed TIPS group

In the nine in whom there was a technical failure (four were cirrhotic), the mean follow-up was 22.9 months

(range 5–103). There were two deaths: one patient with severe oesophageal bleeding at presentation died 1 month later from a further bleeding episode and one (PVT as a primary indication) died 6 months after the attempt at TIPS placement because of complications of the underlying severe haematological disease. In the other seven, one had recurrent bleeding and underwent surgical devascularization with splenectomy (Sugiura); he is alive after 5 months. One with hepatitis C virus (HCV) cirrhosis had liver transplantation due to worsening of liver function 2 months later. At transplantation, surgical thrombectomy was performed without complications. In the remaining four, there were four episodes of bleeding in three patients.

Successful TIPS placement

In these 19, the mean follow-up was 18.1 months. Only one (with liver cirrhosis) died from bleeding 2 days after TIPS (stent placed in a collateral vessel); three of the seven cirrhotic patients were transplanted due to their underlying liver disease. There were no further episodes of bleeding among this group which had a significantly lower rebleeding rate compared with five patients rebleeding in the failed TIPS group ($P = 0.001$; Fisher exact test). TIPS resulted in a slight improvement in liver function according to Child–Pugh score in most patients, particularly with respect improving ascites. Only one patient had persistent fluctuating grade 1–2 hepatic encephalopathy 10 days

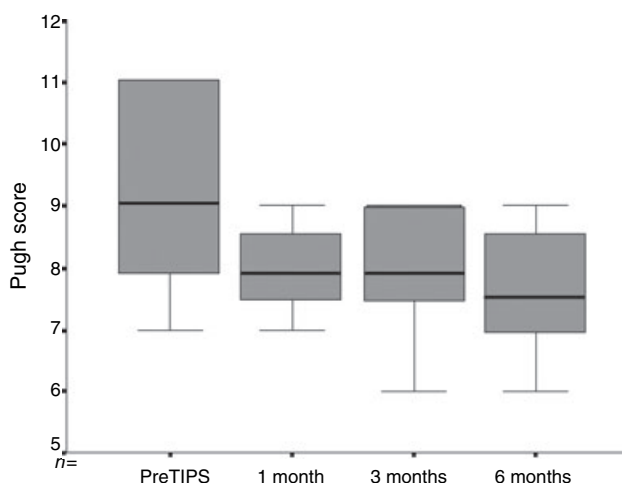


Figure 3. Boxplot showing mean (bold bars), distribution (grey area) and range (whiskers bars) of Child–Pugh score of cirrhotics before and after transjugular intrahepatic portosystemic shunt (TIPS).

post-procedure which responded well to medical treatment. The change in mean Pugh score in the cirrhotic subgroup is shown in Figure 3.

The primary patency was maintained in 14 of 19 patients (74%). Two of the five patients who had shunt thrombosis had Budd–Chiari syndrome, and despite anticoagulation, Doppler ultrasound showed stent thrombosis 2 and 4 days, respectively, after the procedure. Thrombus was disrupted by balloon angioplasty and patency re-established. Of the other three patients with shunt occlusion, one had very early shunt occlusion after 1 week and two new Viator Gore stents were placed with a good angiographic result. The final two patients underwent routine tipsogram at 7 days after the TIPS, and an intra-stent thrombus was disrupted with a balloon. Doppler follow-up showed reduction in intrastent flow in one other patient at 2 years post-TIPS, and a tipsogram was performed and the flow re-established. Thus during the follow-up, six revisions in six patients were performed.

DISCUSSION

Complications of PVT in non-cirrhotics have usually been treated surgically with shunting or devascularization. However, in cirrhotic patients even if well compensated, surgery is a relative contraindication, as transplantation may be required in future.²⁶ Therefore, simple thrombectomy has been used facilitating transplantation and reducing complications.^{10, 13, 27} With recent thrombosis, local or systemic fibrinolysis has been used, but this is clearly contraindicated if the patient has active variceal bleeding.²⁸

Non-surgical shunting with TIPS could provide good decompression at the same time as avoiding surgery, and in addition, it would not preclude further therapeutic options in cirrhotics.

Since the first description of TIPS,²⁹ the indications have steadily expanded. However, PVT was considered a contraindication for TIPS.^{25, 30} Our series and other centres' experience demonstrate that this is not always the case, and indeed for some patients PVT may be an 'indication' for TIPS.

At first we thought that the age of the thrombus would be important in determining success or failure of TIPS, but this was not the case. Thus some patients with well established and extensive PVT had successful TIPS. The main factor predicting success was the presence of a visible intrahepatic portal vein on either ultrasound or carbon dioxide portography which could

Table 3. Published studies on TIPS for portal vein thrombosis

Author	Patients considered for TIPS	Cavernous transformation	Successful TIPS (%)	Percutaneous approach	Complications procedure	Mean follow-up (months)	Anticoagulation	Reintervention (n)
Radosevich <i>et al.</i> (36)	10	1 (failed 1)	7/10 (70)	4/7	No	12	ND	1
Blum <i>et al.</i> (31)	7	No	7/7 (100)	0/7	No	8	ND	0
Walser <i>et al.</i> (25)	14	2 (failed 2)	12/14 (85)	2/12	No	10.4	ND	12
Ganger <i>et al.</i> (32)	11	No	9/11 (81)	0/9	2 (1 died)	13	ND	'Several' ND
Bilbao <i>et al.</i> (24)	6	No	6/6 (100)	5/6	No	15	ND	ND
Jiang <i>et al.</i> (33)	14	4 (failed 4)	10/14 (71)	0/10	No	ND	ND	ND
Current series	28	9 (failed 3)	19/28 (71)	0/17	1	16	9/17	7

TIPS, transjugular intrahepatic portosystemic shunt; ND, not defined.

then be punctured by the transjugular needle. An element of serendipity was then required if a hydrophilic wire was to find its way through a thrombus or area of cavernous transformation. If this did occur, it was relatively straightforward to dilate a tract and re-establish hepato-petal blood flow. No other technical elements were identified that predicted success in the presence or absence of cavernous transformation.

In our population prothrombotic disorders were found in 8 of 28 patients, exclusively in patients with Budd–Chiari syndrome and non-cirrhotic portal vein obstruction, but thrombophilic states were not examined in all our cirrhotics. The non-cirrhotic patients were anticoagulated routinely, although not immediately as we have previously experienced patients developing intrahepatic haematomas if they are aggressively and immediately heparinized post-TIPS.

This is not the first report of TIPS for PVT,^{22, 24, 25, 27, 31–36} but is the largest series, and also includes patients with cavernous transformation. Radosevich *et al.* successfully performed TIPS placement in 7 of 10 patients with total PVT with transjugular catheterization with or without a transhepatic approach.³⁶ Blum *et al.* treated seven patients with non-cavernous portal occlusion with TIPS placement and local fibrinolysis³¹ and recently Bilbao *et al.* reported successful TIPS placement in six patients with PVT with a combination of various percutaneous approaches.²⁴ Ganger *et al.* reported successful TIPS in 9 of 11 patients with PVT;^{25, 32} however, no information about the features of the thrombus were given,³² and in the series described by Walsner *et al.* 12 of 14 patients were successfully treated, all without cavernous transformation which was considered a contraindication and was responsible for failure in two.²⁵

A further paper by Jiang *et al.* reporting 14 patients also concluded that cavernous transformation was a contraindication to TIPS as it was responsible for all of the four failed procedures.³³ A summary of all the series published in the literature about TIPS for PVT is represented in Table 3.

All of our patients in whom the procedure was unsuccessful had complete PVT. However, in those with cavernous occlusion of PVT, the success rate was 6/9 (67%). There has been only one previous case report of a successful TIPS placement with PV cavernoma.³⁴

We used anticoagulation if there was an underlying prothrombotic disorder and in selected cirrhotics providing there were no contraindications. Thus more patients were anticoagulated than in other series. This may account for only six revisions in six patients in our series compared with the 'several revisions' reported by Ganger *et al.*,³² and two early shunt thrombosis and nine revisions in 12 patients.²⁵

The absence of bleeding except in one patient in whom the stent was created between the hepatic vein and a collateral at hilum shows that with this exception, all shunts functioned well. The three patients who subsequently underwent orthotopic liver transplantation (OLT) had no thrombus at the time of the procedure, facilitating the procedure itself, with the added benefit that patients had improvement of liver function and ascites – similar to the finding by Ganger *et al.*³²

In conclusion, TIPS placement is a feasible and effective treatment for portal vein occlusion regardless of the presence of cirrhosis and should be seen as one of the therapeutic options, in units regularly performing this procedure. Cavernous transformation of the portal vein should not be seen as a contraindication to TIPS, but it does increase the technical difficulty of the procedure. We must stress that not every patient with PVT needs to be considered for TIPS and indeed procedural difficulties can occur; however, in patients with severe and life-threatening complications of portal vein thrombosis or in whom the thrombus may jeopardize liver transplantation the procedure should be considered.

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