Longterm follow up of transjugular intrahepatic portosystemic stent shunt (TIPSS) for the treatment of portal hypertension: results in 130 patients

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Abstract

Background—Transjugular intrahepatic portosystemic stent shunts (TIPSS) are increasingly being used to manage the complications of portal hypertension. This study reports on the follow up on 130 patients who have undergone TIPSS.

Patients and Methods—One hundred and thirty patients (81 male), mean (SD) age 54.7 (12.5) years underwent TIPSS. The majority (64.6%) had alcoholic cirrhosis and 53.2% had Childs C disease. Indications were: variceal haemorrhage (76.2%), refractory ascites (13.1%), portal hypertensive gastropathy (4.6%), others (6.1%). Shunt function was assessed by Doppler ultrasonography and two then six monthly portography and mean follow up for survivors was 18.0 months (range 2–43.5).

Results—The procedure was successful in 119 (91.5%). Sixty three episodes of shunt dysfunction were observed in 45 (37.8%) patients. Variceal rebleeding occurred in 16 (13.4%) patients and was always associated with shunt dysfunction. Twenty (16.8%) patients had new or worse spontaneous encephalopathy after TIPSS and 11 (64.7%) patients had an improvement in resistant ascites. Thirty day mortality was 21.8% and one year survival 62.5%.

Conclusion—TIPSS is an effective treatment for variceal bleeding, resistant ascites, and portal hypertensive gastropathy. Rebleeding is invariably associated with shunt dysfunction, the frequency of which increases with time, therefore regular and longterm shunt surveillance is required.

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recurrent bleeding. Immediate control of bleeding can be achieved in 90% of patients by balloon tamponade,⁴ vasoactive drug therapy,⁵ sclerotherapy,⁶ variceal band ligation⁷ or surgery,^{8,9} with the greatest reduction in rebleeding rates achieved by surgical shunts.¹⁰ The main limitations of shunt surgery are its high perioperative mortality and frequency of debilitating postoperative encephalopathy, which approaches 30% in some series.¹¹ TIPSS has been shown to control active variceal haemorrhage and reduce rebleeding rates while having lower procedure related complications and probably less post-treatment encephalopathy¹² compared with surgical shunts. Follow up of patients with TIPSS is however limited, which is important as encephalopathy following surgical shunts commonly was delayed and shunt patency after TIPSS may decrease with time.

Refractory ascites is associated with advanced liver disease and a poor prognosis.¹³ The current therapeutic options of repeated paracentesis and a peritoneovenous shunt are far from ideal and associated with significant morbidity and prolonged hospital stay and do not affect survival.¹⁴ TIPSS permits better control of ascites and improves renal sodium excretion¹⁵ although the mortality seems unchanged in the limited reported data. TIPSS has also been used and found to be effective in the control of other conditions such as portal hypertensive gastropathy, splenomegaly,¹⁶ Budd-Chiari syndrome,¹⁷ and hepatic hydrothorax.¹⁸ The present available literature is limited both by short duration of follow up and small patient numbers. Up until August 1995, we had carried out 130 TIPSS procedures in our unit and the aim of this paper is to present our results and more importantly the longterm follow up.

Methods

PATIENTS

From 1991 to 1995, TIPSS insertion was attempted in 130 patients, with successful placement of the stent in 119 cases (91.5%). Table I shows the details of the patients. Eighty one patients were male with mean (SD) age of 54.7 (12.5) years (range 9 to 83 years). Over the same period, a total of 220 patients were treated for variceal haemorrhage at our institution.

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Since their introduction into clinical practice in 1989,¹ transjugular intrahepatic portosystemic stent shunts (TIPSS) are being increasingly used in the management of both variceal haemorrhage and refractory ascites.

Variceal haemorrhage is the most dramatic complication of portal hypertension, occurring in 30% patients with cirrhosis during their lifetime.² Mortality from the first bleed approaches 50%³ and 70–100% patients have

TABLE I Patient characteristics (n=130) before TIPSS

Sex (M/F)	81/49
Age (y)	
mean (SD)	54.7 (12.5)
range	9-83
Aetiology of liver disease (%)	
alcoholic cirrhosis	84 (64.6)
cryptogenic cirrhosis	12 (9.2)
primary biliary cirrhosis	11 (8.5)
hepatitis C	5 (3.8)
hepatitis B	5 (3.8)
cystic fibrosis	3 (2.3)
primary sclerosing cholangitis	2 (1.5)
autoimmune hepatitis	2 (1.5)
other	6 (4.6)
Childs-Pugh score (mean (SD))	9.9 (2.7)
Childs-Pugh grade (%)	
A	10 (7.9)
B	49 (38.9)
C	67 (53.2)
Indication (%)	
oesophageal varices	73 (56.2)
gastric varices	26 (20.0)
refractory ascites	17 (13.1)
portal hypertensive gastropathy	6 (4.6)
ectopic varices	5 (3.8)
other	3 (2.3)
Emergency procedure (%)	35 (26.9)
Clinical features (%)	
artificially ventilated	19 (14.6)
haemodynamic compromise	10 (7.7)
ascites	86 (66.2)
hepatic encephalopathy	44 (33.8)

The aetiology of portal hypertension was alcoholic cirrhosis (ALD) in 84 (64.6%) patients, with other aetiologies shown in Table I. One patient had α_1 antitrypsin deficiency and one Budd-Chiari syndrome. Four patients were non-cirrhotic (one each with amyloid, idiopathic portal hypertension, polycystic disease, and nodular regenerative hyperplasia). Most cirrhotic patients (53.2%) had Childs-Pugh grade C disease at time of TIPSS with a mean (SD) overall Childs-Pugh score of 9.9 (2.7).

The indication for TIPSS was oesophageal variceal bleeding in 73 (56.2%) patients who had either continued bleeding despite two sessions of sclerotherapy or were part of a trial comparing band ligation with TIPSS in the prevention of rebleeding (see Table I). The indication was ectopic varices in five (3.8%) patients (two rectal, two stomal, and one duodenal) and painful splenomegaly, hypersplenism and embolisation of a spontaneous shunt in one patient each. Thirty five procedures (26.9%) were carried out as an emergency with 19 (14.6%) patients receiving assisted ventilation and 23 (17.7%) patients treated with balloon tamponade prior to TIPSS.

STUDY DESIGN

The technique of TIPSS placement was based on the original method described by Richter¹⁹ and is described in detail elsewhere.²⁰ Routine pre-procedural mesenteric angiography was undertaken in the first 32 patients to guide portal vein puncture and in 27 subsequent patients, Doppler ultrasonography was used to identify the site of portal vein bifurcation. In the last 71 patients however, no routine imaging was undertaken to localise the portal vein pre-TIPSS, although ultrasonography was used prior to the procedure to exclude portal vein thrombosis. These changes have evolved as a result of ongoing audit at our unit.

Once successful puncture of the portal vein was achieved, 2-3 Palmaz stents (Johnson and Johnson) (20 patients), or 1-2 Wallstents (Schneider US Stent Division) (99 patients) were inserted to reduce the portal pressure gradient (defined as: (portal pressure) - (inferior vena-caval pressure)) to less than 12 mm Hg. Three patients subsequently had Angiomed stents (Angiomed, Karlsruhe, Germany) inserted to reduce the shunt size. In two patients in whom a thrombus was noted within the portal vein at the end of the procedure, a catheter was left within the shunt for regional infusion of low dose streptokinase for 24 hours. Prophylactic antibiotics (cefotaxime and amoxycillin) were given one hour before the procedure and continued for 48 hours thereafter and Doppler ultrasonography was performed prior to discharge to ensure shunt patency. Routine portography was undertaken at one to three months and six months thereafter to assess shunt function, or earlier in the event of rebleeding or reaccumulation of ascites. Early in our experience however, several shunts were left six to 12 months before initial angiographic assessment.

Encephalopathy was assessed clinically after six weeks then at three monthly intervals during outpatient review. Prophylactic lactulose and protein restriction were not routinely applied. Variceal rebleeding was defined as endoscopically confirmed variceal haemorrhage occurring more than 24 hours after TIPSS insertion. All shunt complications were confirmed angiographically with occlusion defined as absent flow through the shunt. Pseudo-intimal hyperplasia, hepatic vein stenosis, portal vein and shunt thrombosis were defined by the angiographic appearance in conjunction with either a 20% rise in portal pressure gradient or an increase in portal pressure gradient to 12 mm Hg or more. Primary shunt patency was defined as the (pre-intervention) absence of any of the above shunt complications. Mean (SD) follow up (defined as time to death, most recent clinical review or liver transplantation) for all patients was 10.7 (11.0) months and for survivors (patients alive up to October 1995) 18.0 (11.9) months.

STATISTICAL ANALYSIS

Results are expressed as mean (SD) or range where indicated. Paired Student's *t* test was used to determine statistical significance and Kaplan-Meier method used for rates of variceal rebleeding, primary shunt patency, and survival.

Results

Tables II and III summarise the results.

SHUNT PROCEDURE

TIPSS placement was successful in 119 (91.5%) patients. The procedure failed in 11 patients (10 of whom had variceal haemorrhage) because a main branch of the portal vein could not be punctured: four of these patients subsequently underwent shunt surgery and five

TABLE II Results of TIPSS procedure and shunt complications

Successful TIPSS procedure (%)	119 (91.5)
PPG before TIPSS mean (SD) (mm Hg)	17.5 (5.9)
PPG after TIPSS (mm Hg)	8.6 (3.4)
Pre-intervention shunt patency (%):	
6 months	47 (71.2)
1 year	25 (58.1)
2 years	3 (21.4)
Shunt complications:	63 episodes in 45 (37.8) patients:
pseudo-intimal hyperplasia	29
hepatic vein stenosis	13
portal vein/shunt thrombosis	5
occlusion	16

PPG=portal pressure gradient.

had further endoscopic therapy. Thirty day mortality in the failed TIPSS group with variceal haemorrhage was 70.0%.

Two procedure related deaths occurred from intraperitoneal haemorrhage because of an extrahepatic tear of the portal vein in one and puncture of the liver capsule in the other. One patient developed an epidural haemorrhage, which was diagnosed after the procedure but the exact relation to TIPSS placement was unclear. Other complications included portal vein thrombosis in two patients (successfully treated by local streptokinase infusion), portal vein dissection in one (successfully managed by stenting), and shunt dislocation or migration into the splenic vein in two. There were no clinically significant groin or neck haematomas. Mean (SD) portal pressure gradient

was reduced from 17.5 (5.9) mm Hg pre-TIPSS to 8.6 (3.4) mm Hg ($p<0.001$) after successful stent placement.

A total of 63 episodes of shunt complications were seen in 45 patients (see table II). Twenty nine of these were clinically significant (associated with variceal rebleeding or reaccumulation of ascites), with the others detected on routine surveillance. Fifty per cent of the shunt complications occurring less than one month after TIPSS were associated with variceal rebleeding, as were 41.7% of those occurring from one to three months, 35% from three to 12 months, and 23.5% from 12–24 months. None of the shunt complications occurring more than two years after TIPSS were associated with variceal rebleeding. Taking follow up as most recent angiographic TIPSS assessment, pre-intervention shunt patency was 71.2% at six months, 58.1% at one year, and 21.4% at two years (see Table II and Fig 1). No Palmaz stent had primary patency at two years. All shunt complications but one (in a patient who was transferred to an endoscopic banding programme and remains well) were successfully treated by balloon dilatation, local thrombolysis, shunt extension or parallel shunt placement (nine patients). The causes of shunt dysfunction are described in Table II.

CLINICAL FOLLOW UP

Control of acute bleeding

Thirty five patients had TIPSS for acute variceal haemorrhage uncontrolled despite sclerotherapy with or without tamponade. Eighteen of these patients were mechanically ventilated and 10 were haemodynamically unstable at time of TIPSS. The procedure was technically successful in 32 of these patients and the other three patients died: two from massive haemorrhage and one shortly after oesophageal transection. Fourteen of the remaining patients died during the index hospital admission: three each from acute on chronic liver failure, sepsis, and end stage liver failure, two from continued bleeding (one from a sclerotherapy ulcer confirmed at necropsy and one with disseminated intravascular coagulation and acidosis), two from cardiac arrests/myocardial infarct, and one from a procedure related intraperitoneal bleed. Two further patients had subsequent variceal rebleeds, both related to shunt insufficiency and successfully treated by angioplasty and shunt extension.

Recurrent variceal bleeding

TIPSS was successful in reducing recurrent variceal haemorrhage. There were 24 episodes of variceal re-bleeding in 16 (13.4%) patients (see Fig 1), all of which were associated with shunt insufficiency and responded to dilatation or further stenting. Five patients underwent TIPSS for ectopic variceal haemorrhage (two rectal, two stomal, and one duodenal) and none of these patients have had a recurrent bleed. Non-variceal haemorrhage occurred in nine (7.6%) patients (see Table III).

TABLE III Results of rebleeding, encephalopathy, ascites, and sepsis after TIPSS

Variceal rebleeding	24 episodes in 16 patients (13.4%)
Non-variceal rebleeding	9 patients (7.6%)
	7 sclerotherapy ulcers
	1 duodenal ulcer
	1 Mallory-Weiss tear
Encephalopathy (%)	
before TIPSS	44 (33.8)
new/worse spontaneous encephalopathy	20 (16.8)
new/worse encephalopathy, secondary to sepsis	9 (7.6)
new/worse encephalopathy, secondary to bleeding	6 (5.0)
Ascites (%)	
before TIPSS	86 (66.2)
improved after TIPSS	56 (65.1)
reaccumulation	14 (25.0)
primary indication for TIPSS	17 (13.1)
Sepsis after TIPSS (%)	14 (11.8)

Figure 1: Kaplan-Meier analysis of patients free of variceal rebleeding and with primary shunt patency during follow up.

Ascites

Eighty six patients (66.2%) had ascites prior to TIPSS and this was the primary indication in 17 patients (seven of whom had established hepatorenal syndrome) (see Table III). The ascites improved (reduced or no diuretic requirement) in 56 (65.1%) of the 86 patients, but reaccumulated in 14. This was associated with shunt insufficiency in 11 patients and spontaneous bacterial peritonitis in two, but was responsive in all cases to shunt revision and antibiotics respectively. Of the 17 patients who had TIPSS performed for refractory ascites, improvement occurred in 11 and two have subsequently undergone successful liver transplantation. Those with biochemical evidence of renal dysfunction tended to respond less well after TIPSS insertion.

Other indications

TIPSS was performed for intractable bleeding from portal hypertensive gastropathy in six patients. Four of these have not required further transfusion and the other two have required one admission each for transfusion. Both of these patients had evidence of shunt dysfunction due to pseudo-intimal hyperplasia in association with a raised portal pressure gradient, which was successfully treated by angioplasty or shunt extension. TIPSS resulted in an improvement in platelet count from 13 000/l to 50 000/l in one patient with hypersplenism. Another patient had a large spontaneous shunt successfully embolised via the TIPSS for amelioration of intractable hepatic encephalopathy.

Hepatic encephalopathy

Forty four (33.8%) patients were clinically encephalopathic prior to TIPSS and this resolved in 24 (54.5%) after the procedure. Twenty (16.8%) patients developed new or worsening spontaneous encephalopathy during follow up (15 within the first six months). A further 15 (12.6%) patients developed encephalopathy secondary to sepsis or bleeding during follow up (see Table III). Reduction in shunt size was performed because of encephalopathy in four patients (successful in three), with all others responding to simple medical treatment.

Figure 2: Kaplan-Meier analysis of patient survival after TIPSS by Childs grade. (Childs A, $n=12$; Childs B, $n=44$; Childs C, $n=61$. At 12 months, $n=9$, 23, and 14 respectively.)

Sepsis

Fourteen (11.8%) patients developed clinically significant infections in the week after TIPSS (seven pulmonary, two spontaneous bacterial peritonitis, two related to central venous catheter, one cellulitis, and two of unknown origin). Sepsis was the cause of death in two patients during the index admission: one with pre-existing staphylococcal and fungal septicaemia and one with cystic fibrosis and pre-existing lung sepsis. All other infective episodes responded to antibiotics.

Liver function

Approximately one third of patients exhibited a transient deterioration in their liver function tests in the first week after TIPSS, usually with doubling of bilirubin and alanine aminotransferase values. Three patients died with clinical features of acute liver failure, characterised by hypotension, renal failure, and hypoglycaemia. Two of these patients also had evidence of raised intracranial pressure on extradural pressure recordings or computed tomography. Childs-Pugh score did however improve at three to six months to 7.6 (2.1) ($p=0.019$) largely because of a reduction in ascites.

Transplantation

Thirteen patients have undergone orthotopic liver transplantation after TIPSS. Transplant was undertaken a mean of 7.1 (5.4) months after TIPSS and 11 of these patients remain alive and well.

Mortality

Of the 119 patients with successful TIPSS placement, 52 have died and 13 have undergone transplantation. Procedure related mortality was 1.5% and mean time to death was 6.0 (8.9) months (range 0.03–45.3). Mean follow up of survivors is 18.0 (11.9) months (range 2.0–43.5). Thirty day mortality was 21.8% (84.6% of whom had Childs C disease) and six months survival 69.2%. One and two year survival is 62.3% and 46.5% respectively. Mortality was dependent on Childs grade at time of TIPSS (see Fig 2).

Discussion

This is only the second study with large numbers of patients and longterm follow up and our results are similar to the two year follow up of 90 patients reported by Laberge *et al.*²¹ Shunt insertion was achieved with a reduction in the portal pressure gradient to below 12 mm Hg in 91.5% patients. Our recurrent variceal rebleeding rate (13.4% patients) and rate of new or worsening spontaneous encephalopathy (16.8% patients) compares favourably with most other series.^{12 21–23}

TIPSS has established a position as a 'rescue procedure' for uncontrolled variceal bleeding. Of the 35 patients who had TIPSS for

continued variceal haemorrhage despite sclerotherapy with or without balloon tamponade, shunt creation was successful in the vast majority (91.4%). Most (77.1%) of these patients had Childs C cirrhosis. Although only two patients had continued bleeding the inpatient mortality in this high risk group was 43.7%. The options of a surgical portosystemic shunt or transection in this situation have higher procedure related and early mortality.²⁴ In addition transection has higher rebleeding rates²⁵ and surgical shunting has higher rates of encephalopathy²⁶ while also compromising a subsequent liver transplant. It should be noted that in many cases TIPSS is performed in critically ill patients who would not be considered fit for surgery.

Similar to other investigators,²¹ we found shunt dysfunction in all patients with variceal rebleeding. There seems to be a steady attrition rate in shunt patency with time (Table II and Fig 1) with a primary (pre-intervention) patency of 21.4% at two years. We have previously shown that shunt dysfunction is more common with Palmaz stents than Wallstents,²⁷ although it should be noted that our early (often Palmaz stented) patients were left longer than three months prior to initial angiographic assessment, therefore especially in this group it is difficult to be sure of the exact timing of shunt dysfunction. Accepting this limitation, if it is believed important to assess the TIPSS prior to a 20% risk of dysfunction, it is necessary to check the shunt at two months and then approximately six monthly thereafter. It would seem that early shunt dysfunction is more likely to be associated with variceal rebleeding than that occurring later after TIPSS, therefore even closer shunt assessment in the first few months after placement may be necessary.

Although intervention maintained shunt patency in all but one patient when insufficiency was detected, most stents treated with balloon dilatation required repeat dilatation on follow up. This procedure would seem to have only a temporary effect and the longterm benefits of and exact indications for dilatation remain unclear. The current limitations of ultrasonographic methods of shunt assessment²⁸ mean that direct portography remains the 'gold standard' for surveillance. This is an important limitation of TIPSS but hopefully the development of new covered stents will tackle this problem.

An ability to identify early shunt dysfunction and minimise encephalopathy together with a low incidence of rebleeding makes TIPSS an alternative to sclerotherapy in the treatment of recurrent variceal bleeding. Trials are currently underway to compare these two treatments in this situation. In the case of gastric variceal haemorrhage where current endoscopic treatments are unsatisfactory, we believe TIPSS to be the treatment of choice, as indicated by the comparatively large proportion of patients in our series with gastric varices.

Nine (7.6%) patients developed non-variceal bleeding during follow up, mostly as a result of sclerotherapy ulcers and these were the cause of death in two patients. Sclero-

therapy ulcers have been recognised as a major problem by many investigators and remain a major potential cause of morbidity and mortality no matter how effective the shunt is. The alternative of band ligation should reduce the frequency of this complication.²⁹

New or worsening spontaneous encephalopathy was seen in 16.8% patients and a further 12.6% had new or worsening encephalopathy secondary to sepsis or bleeding after TIPSS. Considering however that 33.6% patients were encephalopathic prior to TIPSS, it is difficult to determine the exact role of the shunt in the development of encephalopathy in this group. In all but four patients (who went on to shunt reduction), the encephalopathy responded to simple medical treatment such as lactulose and protein restriction. This contrasts with the often debilitating encephalopathy seen in around one third of patients after non-selective surgical shunts¹¹ and is probably because of the small stent diameter and intrahepatic position that encourages continued portal flow into the liver.³⁰ A reduced incidence of encephalopathy has been reported in narrow portocaval H-grafts³¹ and selective splenorenal shunts³² however the associated risks of surgery and subsequent problems with transplantation must again be considered. Recent reports indicate that increasing age and a previous history of encephalopathy are the main predictors of encephalopathy after TIPSS³³ and perhaps these patients should have narrower stents placed.

Treatment of patients with refractory ascites remains a major clinical problem. Apart from liver transplantation, the main therapeutic options are repeated large volume paracentesis and peritoneovenous shunting, both of which are associated with significant morbidity and prolonged inpatient management and may aggravate functional renal failure. Recent reports^{14 15} describe the benefits of TIPSS for refractory ascites although data are limited. The mechanism of action seems to be a combination of reduced portal pressure gradient and increased natriuresis, probably secondary to increased effective circulating plasma volume and neurohumoral factors. Survival is poor in patients with refractory ascites and any trial assessing TIPSS in this situation would need to assess quality of life and cost-benefit factors.

The place of TIPSS in the treatment of hepatorenal syndrome also remains controversial. Several authors have reported improvement in functional renal failure^{15 34} and a preliminary study³⁵ showed a decrease after TIPSS in the levels of endothelin 1, which is thought to have an important role in the syndrome. However as we found in this study, results are generally disappointing in this group of patients and they have a very poor prognosis without liver transplantation.

Similar to other investigators,³⁶ we found a transient deterioration in liver function tests in a third of patients probably secondary to reduced hepatic perfusion. Three patients died soon after TIPSS with clinical features of acute liver failure, two of whom had evidence of raised intracranial pressure. This is unusual in patients with chronic liver disease but has been

reported.³⁷ All three of these patients were extremely ill at the time of TIPSS, however, with prolonged hypotension and requiring ventilation. The role of TIPSS in their clinical deterioration is therefore difficult to define, although any reduction in liver perfusion in such patients may be critical. Overall however there was a significant improvement in Childs-Pugh score at three to six months largely due to improvement in ascites.

Procedure related mortality was 1.5%, which is similar to most reported series and compares favourably with the operative mortality associated with surgical portosystemic shunts of around 10%.³⁸ We found a relatively high 30 day mortality of 21.8% compared with some groups,²² however mortality has been consistently shown to depend on Childs grade at time of TIPSS and our higher percentage of Childs B and C patients (38.9% and 53.2% respectively) and higher numbers of emergency procedures (26.9%) would account for this. Our one and two year survival of 62.3% and 46.5% respectively are similar to the recently reported series from San Francisco.²¹

Longterm survival after TIPSS will depend on the underlying severity of liver disease and improved survival due to reduced rebleeding will be difficult to detect. However the benefits of reduced morbidity, as a result of improved control of ascites and less variceal rebleeding should not be underestimated. We have previously shown that besides severity of liver disease, hyponatraemia and encephalopathy prior to TIPSS independently determine long-term survival.³⁹ Thirteen of our patients subsequently underwent liver transplantation and the benefits of TIPSS as a 'bridge to transplantation' have been previously reported.⁴⁰ In particular the ability to avoid surgery in the treatment of variceal bleeding before transplant is important.

In conclusion, TIPSS is a comparatively simple procedure that can be successfully performed even on critically ill patients. It is effective in the treatment of both acute and recurrent variceal haemorrhage (especially where they are not amenable to sclerotherapy), refractory ascites, and portal hypertensive gastropathy. Unlike surgical portosystemic shunts, it has the advantage of not compromising subsequent liver transplantation. Shunt dysfunction however is common and seems to increase linearly with time and variceal rebleeding and reaccumulation of ascites usually occur in the presence of shunt stenosis or occlusion. Regular surveillance by portography is therefore necessary and this represents an important limitation of TIPSS. Post-procedure encephalopathy is significant but generally easily treated and bleeding from sclerotherapy ulcers remains an important cause of morbidity and mortality. Trials are currently underway to compare TIPSS with sclerotherapy or banding in the treatment of first variceal haemorrhage and with paracentesis for refractory ascites, which will help define its exact role in these situations.

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