Update on Shunt Surgery

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Keywords

Portal hypertension · Portal/mesenteric vein thrombosis · Surgical portocaval shunt · Cirrhosis · Esophageal varices

Abstract

Background: Bleeding from esophagogastric varices is a life-threatening complication from portal hypertension. It occurs in 15% of patients and has a mortality rate of 20–35%.

Summary: The primary therapy for variceal bleeding is medical. In cases of recurrent bleeding, a definitive therapy is required. In cases of parenchymal decompensation, liver transplantation is the causal therapy, but if liver function is preserved, portal decompression is the therapy of choice. The use of the transjugular intrahepatic portosystemic shunt (TIPS) has achieved widespread acceptance, although evidence for surgical shunts is comparable or better in patients with good hepatic reserve. The type of surgical shunt depends on the patent veins of the portomesenteric system. If total occlusion is present, a devascularization procedure might be indicated. **Key Messages:** Therapy, taking into account liver function, morphology of the portovenous system, and imminent liver transplantation, should be performed by an interdisciplinary team of gastroenterologists, interventional radiologists, and gastrointestinal surgeons.

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Introduction

Portal hypertension (PH) is a frequent complication of liver cirrhosis and portal/mesenteric vein thrombosis [1]. In western countries, liver cirrhosis accounts for 90% of PH cases, the others being mainly extrahepatic portal vein thrombosis (PVT) and idiopathic PH [2]. The most significant complication arising from PH is variceal bleeding (VB), which occurs in 15% of patients independently of the underlying disease [3–5]. The primary therapy for VB is medical according to the Baveno VI guidelines, and even early TIPS should be considered in patients with a high risk of treatment failure [6]. The 6-week mortality after VB is still 20–35% [7] and rebleeding occurs in 20–30%, this is why patients with VB require variceal decompression as the only effective causative therapy [8–11]. Since the introduction of the transjugular intrahepatic portosystemic shunt (TIPS) in 1988 [12], the therapy of PH has profoundly changed. TIPS is nowadays widely available, and the Baveno VI consensus recommends TIPS when primary therapy with nonselective β -blockers and endoscopic ligation fails (the option of a surgical shunt is not even mentioned) [6]. This has led to a significant reduction in the frequency of surgical shunt procedures (12 times more TIPS than surgical shunts were deployed in the USA in 2002–2005 [13]).

The reduced involvement of gastrointestinal surgeons in the therapy of PH has led to a comprehensive decline in the expertise in portocaval shunt surgery in many surgical departments [13–15]. Despite the widespread acceptance of TIPS, its superiority over surgical shunting techniques has never been shown [11, 16–19]. Surgical shunts perform better in terms of shunt patency, long-term survival, and economic aspects [1]. One significant objection in most studies comparing TIPS and surgical shunts is the use of uncovered stents, but even when polytetrafluoroethylene (PTFE)-covered stents are used, the surgical shunt shows better results [20]. The correct therapy algorithm for variceal decompression, taking into account liver function, rebleeding frequency, long-term outcome, graft patency, and cost-effectiveness, should be chosen by an interdisciplinary team with experience in the therapy of PH. Patients with noncirrhotic PH, in particular, benefit from surgical portocaval shunts, which provide excellent secondary prophylaxis for VB but also prevent ongoing thrombosis of the portovenous system, i.e., “panthrombosis.”

Definition and Classification of PH

Clinically significant PH is defined as a hepatic venous pressure gradient (HVPG) of ≥ 10 mm Hg [6]. Above this value, gastroesophageal varices develop, and bleeding occurs at 12 mm Hg [2]. The leading cause for PH in Western countries is liver cirrhosis (90%), predominantly caused by alcoholic liver disease and viral hepatitis. Less common is extrahepatic PVT (8–10%) in which liver function is normally not impaired [2]. Rare causes are posthepatic disorders like Budd-Chiari syndrome [21]. Complications from PH in cirrhosis like ascites, bacterial peritonitis, hepatorenal syndrome, portopulmonary hypertension, hepatopulmonary syndrome, hepatic encephalopathy, portal hypertensive gastropathy, enteropathy, colopathy, and the formation of esophagogastric varices dominate the further course of the disease [2]. Two-thirds of cirrhotic patients develop varices, with bleeding occurring in 30–40% of these patients [7]. The impairment in liver function dominates the outcome after the first bleeding episode; average mortality is 20–35% but can be as high as 50% in Child-Pugh class C patients [7, 22, 23]. Impaired liver function also increases the risk of early rebleeding [23].

In cases of noncirrhotic PH due to PVT, it is important to determine the stage of PVT (recent vs. chronic), which can sometimes be difficult. Recent PVT can be assumed when patients present with symptoms like abdominal pain, ascites or fever in the absence of portosystemic collaterals, or portal cavernoma. The initial phase of chronic PVT can be asymptomatic, and the disease and its underlying factors are often not diagnosed until the first bleeding episode [14].

Diagnostics

Aside from the underlying disease (liver function?), the extent of PVT is crucial for the planning of a surgical therapy. Medical history includes the typical complications of PH and the history of gastrointestinal hemorrhages. Upper gastrointestinal endoscopy can diagnose esophagogastric varices and exclude other sources of gastrointestinal bleeding. Bleeding risk is classified by the localization, size, color, and stigmata of the varices [7]. The exact quantification of liver function is crucial for the following therapy. Child-Pugh class, MELD score, and transient elastography (Fibroscan®) [24, 25]) can assess the severity of liver fibrosis/cirrhosis, and in special cases, liver biopsy can provide histological information [26]. In known cirrhosis, new-onset PVT should lead to imaging combined with α -fetoprotein levels to exclude hepatocellular carcinoma formation. Especially in noncirrhotic PH, a hypercoagulable syndrome or myeloproliferative neoplasm must be evaluated. This includes measurements of protein C, protein S, antithrombin III, total homocysteine serum levels, anti-cardiolipin antibodies, lupus-like anticoagulant, and anti- β_2 glycoprotein antibodies [27]. Genetic studies for factor V Leiden, prothrombin G20210A, and *JAK2* gene mutations should be conducted, and bone marrow biopsy can be useful [28, 29]. Abdominal sonography is the first-line imaging technique for the diagnosis of cirrhosis and PVT, with a high sensitivity. In compensated patients, sensitivity for PH is lower, but indirect signs like splenomegaly or ascites can be detected [2]. CT and MRI (sometimes combined) are used to visualize the portal system including the extent of the PVT. They can also map the collateral circulation and identify rare causes for PH (inflammation or tumor growth) [2, 28]. Visceral angiography is no longer standard, but can still be used in selected cases to assess the direction of blood flow and map collateral circulation precisely, in order to evaluate the feasibility of a selective surgical shunt [10].

Indication for Portal Decompression

There is no indication for prophylactic portal decompression (either TIPS or surgical shunt) in asymptomatic varices [6, 9, 30, 31]. If VB occurs, the primary therapy is the combination of blood volume restitution, antibiotic prophylaxis if higher grade cirrhosis is present, vasoactive drugs (terlipressin, somatostatin, or octreotide), and endoscopy [6]. Emergency portal decompression is seldom indicated. Nonetheless, some groups report excellent outcomes with early TIPS [32], which is also recommended in the current Baveno consensus statement [6] for patients at a high risk of treatment failure (Child-Pugh class C/B

Table 1. Studies comparing surgical shunt procedures with TIPS (modified according to [1, 15])

	Khaitiyar et al. 2000 [16]		Helton et al. 2001 [17]		Henderson et al. 2006 [11]		Rosemurgy et al. 2012 [18]		Orloff 2012 [19]		Hosokawa et al. 2017 [20]	
	TIPS	DSRS	TIPS	14 PCS and 6 DSRS	TIPS	DSRS	TIPS	HGPCS	TIPS	EPCS	TIPS	PCS
N	35	32	20	20	67	70	66	66	78	76	31	31
Child-Pugh class A/B, %	100		100		100		56	50	71	68	94	94
Follow-up	30 months		19 months	23 months		45 months	13.4–18 years			7 years	51 months	
30-Day mortality, %	6	6	20	0	2	6	15	20	22	23	3	3
2-Year survival, %	80	81	80	90	88	81	53	68*	49	68*	93 ^a	88 ^a
5-Year survival, %	–	–	–	–	61	62	30	0*	80	39*	58	7*
Recurrence of VB, %	26	6	50	5*	11	6	48	11*	78	3*	19	0
Shunt revision/stenosis, %	69	6*	60	10*	82	11*	48	11*	84	3*	32/26	3/0*
Total cost, USD			111,573	61,934*	74,267	54,975*			264,800	39,000*		

* Significant difference. TIPS, transjugular intrahepatic portosystemic shunt; DSRS, distal splenorenal shunt; PCS, portocaval shunt; HGPCS, H-graft portocaval shunt; EPCS, emergency portocaval shunt; VB, variceal bleeding.
^a Survived for 1 year.

with active bleeding), or operative portocaval shunt procedures [19]. Initial treatment failure with rebleeding occurs in 20–30% [8–11]. As stated earlier, the further therapy should be guided according to the hepatic reserve.

Liver Cirrhosis

Three factors should be included in the treatment decision: the hepatic reserve, imminent liver transplantation, and the morphology and patency of the portovenous system.

Operative decompression should only be undertaken in compensated liver function due to high procedural mortality in Child-Pugh class C patients of >50% as well as high encephalopathy rates [14]. Of special importance for the preservation of liver function is the maintenance of at least a partial hepatopetal blood flow. If liver transplantation is planned, TIPS, with the possibility of avoiding an abdominal operation and preserving the hilar anatomy, is widely favored. Nevertheless, transplantation complications due to TIPS misplacement have been described [33]. In patients with failed TIPS, a distal spleno-renal shunt can be applied without touching the hilar structures [34], or a mesocaval or mesorenal shunt should be used which can be easily ligated during transplantation. Of course, the planned shunt procedure depends on the patency of the portomesenteric veins. Undoubtedly, liver transplantation is the only causal therapy for cirrhosis. But 20–30% of the patients suffering from variceal rebleeding have an excellent liver function and do not require transplantation [31]. These patients are now widely treated with TIPS as a bridging procedure to transplantation, but <10% of patients are transplanted following TIPS [35, 36]. According to the scientific evidence, surgical shunting procedures show a comparable or even better outcome in patients with good liver function (Child-Pugh class A and B), but even after surgical shunt placement, liver transplantation is not more common [37, 38].

The portocaval H-graft interposition shunt (SARFEH) is widely used to preserve a hepatopetal flow of about 80% due to the 8-mm diameter of the PTFE graft used [14]. The encephalopathy rate is low at only 5%, and rebleeding occurs in 5%, with a graft patency of about 95% over 7 years [18, 39]. A selective decompression of the left upper quadrant can be achieved with the distal splenorenal shunt (WARREN), even if the portal and superior mesenteric vein are occluded. Hepatopetal flow is maintained in 70% of cases [14]. This shunt has a low rebleeding frequency of 5–8%, a low mortality of <5%, and a 3- to 5-year survival rate of 75–80% [11]. Shunt occlusion occurs in 6–11% of patients [11, 16]. It is of outmost importance to perform a complete splenopancreatic disconnection, in order to prevent newly developing collateralization which can be technically demanding especially when pancreatitis is apparent [10, 34].

Fig. 1. Algorithm for the secondary prophylaxis of variceal bleeding (modified according to Glowka et al. [1]). NSBB, nonselective β -blockers; Child, Child-Pugh class.

Noncirrhotic PVT

The outcome for shunting procedures after VB without impaired liver function is better than in cirrhotic PH, and late events like slow hepatic dysfunction and portal biliopathy can be avoided [40]. In children and young adults, particularly, other indications than the secondary prophylaxis of VB should be considered; hypersplenism, thrombocytopenia, and growth retardation are reasons for decompression of the portal system [40]. In recent PVT, the risk of ongoing thrombosis with intestinal infarction is as high as for VB, with an appositional thrombus of the superior mesenteric vein and splanchnic vein present in 37% of cases [41]. In cases of chronic PVT, shunting provides a patent portovenous system in addition to rebleeding prophylaxis, allowing an aggressive anticoagulation therapy when there is a myeloproliferative neoplasm or hypercoagulable syndrome. PVT caused by malignant tumor growth (e.g., pancreatic cancer and hepatocellular carcinoma)

is not a good indication for surgical shunt placement due to the poor prognosis [14].

Patients with noncirrhotic PH can largely benefit from the splenorenal side-to-side shunt (COOLEY), especially if only a segmental PVT and a patent superior mesenteric vein are present. The procedure decompresses the gastroesophageal varices and mesenteric compartment, and also shows excellent patency (87%) and a low rebleeding frequency (10%) [42]. If selective decompression of the left upper quadrant is warranted, the WARREN shunt can also be used for noncirrhotic PVT. The mesocaval shunt is a small-diameter shunt and can be applied with or without PTFE interposition. Results differ, with a rebleeding rate of 5–15% and a patency of 81–95% [43, 44]. It is a good alternative in cases of noncirrhotic PVT and patent superior mesenteric vein when the splenic and portal veins are occluded.

Generally, the patency of the portovenous branches dictate the type of surgical shunt procedure in extrahepatic

PVT. If the left gastric vein is patent, it can be anastomized with the inferior vena cava (the INOKUCHI shunt [45]). In cases of total occlusion of the portovenous system and recurrent VB with preserved liver function, a devascularization procedure can be performed with low rebleeding rates of 1.5–16% and a mortality rate of 4–22% [34, 46]. In cases of combined total portovenous occlusion and parenchymal decompensation, liver transplantation with cavoportal hemitransposition or multivisceral transplantation should be considered [10].

The end-to-side portocaval shunt is a total portosystemic shunt with no remaining hepatopetal flow. Despite its high patency rate and excellent prevention of rebleeding (<5%), it has a high rate of encephalopathy (approx. 40%) and is nowadays only used in emergency settings due to its easy technical feasibility [34, 47].

In the Baveno V consensus statement, PTFE-covered TIPS is the preferred therapy for patients failing medical treatment for the prevention of rebleeding. Surgical shunts are considered in Child-Pugh class A and B patients only if TIPS is unavailable [5]. The scientific basis for this decision is still lacking. Six studies (Table 1) have compared TIPS with surgical shunts. All these studies show comparable or better results for the surgical shunting procedures. In most studies, the cost-effectiveness of surgical shunt placement is better than that of TIPS, mostly because of the higher rate of reinterventions after TIPS. The common argument of a higher periprocedural mortality of surgery compared to TIPS can be contradicted with the 6 studies above. A German case series showed constantly better results for surgical shunts. There was a rebleeding rate of 10.5–30% for TIPS versus 1–11% for the surgical shunt, a reintervention rate of 48–82% (TIPS) versus 6.3–11% (surgical shunt), and a 30-day-mortality of 15–40% (TIPS) versus 8–20% (surgical shunt) [15]. TIPS supporters criticized the fact that most of these trials used bare-metal TIPS rather than the PTFE-covered TIPS now in use. Hosokawa et al. [20] included PTFE-covered

TIPS in their study and also found better results with surgical shunts. Most studies evaluating PTFE-covered TIPS are retrospective, case series only, or have poor follow-up rates [40–43]. Findings for an overall survival benefit of PTFE-covered versus uncovered TIPS vary [44, 45]. There are only a few studies on applying TIPS for noncirrhotic PVT, and they show a reintervention rate of 35% [46]. In our opinion, the decompressive therapy for each patient suffering from PH should be planned by an interdisciplinary board.

Conclusion

VB is a severe complication of PH, with a high mortality. The primary therapy is medical (including endoscopy), but when recurrent bleeding occurs an interdisciplinary approach should be followed. A team of gastroenterologists, interventional radiologists, and gastrointestinal surgeons should recommend a therapy that takes into account liver function, portovenous morphology, personal expertise, and the patient's preference (Fig. 1). Patients with noncirrhotic PVT, in particular, can benefit from the surgical shunt procedure.

Disclosure Statement

The authors have no conflicts of interest to declare.

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Author Contributions

T.R.G. and S.M. did the literature search, interpreted the data, and drafted the manuscript. All 3 authors revised the draft critically and gave their final approval.

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