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SURGICAL TREATMENT OF PORTAL HYPERTENSION

HIRURŠKO LEČENJE PORTNE HIPERTENZIJE

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Summary

Portal hypertension (PHT) is one of the main causes of morbidity and mortality in patients with severe liver diseases. The most serious complication of PHT is haemorrhage due to rupture of the gastric and esophageal varices and requires primary prophylaxis, acute bleeding management and secondary prophylaxis of re-bleeding. Surgical treatment of PHT developed from invasive total shunt surgery to recent minimal-invasive procedures, but the best therapeutic solution is still a subject of many clinical studies. In this review, we investigated positives and downsides of current surgical solutions for dealing with portal hypertension.

Key words: portal hypertension; surgical treatment; non-invasive procedures

Sažetak

Portna hipertenzija (PHT) predstavlja jedan od glavnih uroka mortaliteta i morbiditeta kod pacijenata sa teškim oboljenjem jetre. Najozbiljnija komplikacija PHT je krvarenje, koje nastaje kao poslednica rupture gastričnih i ezofagijalnih varikoziteta i zahteva primarnu profilaksu, zaustavljnje aktivnog krvarenja i sekundarnu profilaksu ponovnog krvarenja. Hirurški tretman PHT se razvijao od invazivne hirurgije da minimalno invazivnih tehnika, ali najbolji terapijski izbor je i dalje predmet velikog broja kliničkih studija. U ovom prikazu literature smo istraživali pozitivne i negativne strane trenutnih hirurških tehnika u lečenje portne hipertenzije.

Ključne reči: portna hipertenzija; hirurško lečenje; minimalno invazivne tehnike.

Introduction

Portal hypertension (PHT) is one of the complications of liver diseases that most frequently requires surgical treatment, in up to 15% of patients. In a healthy subject at rest, portal pressures range from 60 to 140 mm saline (4 to 10 mmHg), while inferior vena caval (IVC) pressures range from 7-55 mm saline (0.5 to 4 mmHg). Clinically meaningful PHT exists when the difference between portal and IVC pressure (corrected portal pressure) exceeds 150 mm saline (11 mmHg) [1]. The elevated portal vein

pressure leads to an enlargement of all the collateral venous connections between the portal and systemic circulation and to development of varicosities and splenomegaly.

Classification of portal hypertension

Based on the anatomic location of the resistance flow, PHT could be classified as presinusoidal or postsinusoidal [2].

Extrahepatic obstruction of the portal vein is responsible for less than 5% of the cases of PH, whereas intrahepatic obstructive disease accounts for more than 95% of cases of PH. Intrahepatic blockages occur in cirrhosis, inborn errors of metabolism, and schistosomiasis. Alcoholic cirrhosis is the most common cause of PHT and produces resistance that is primarily intrasinusoidal or postsinusoidal, while schistosomiasis and biliary cirrhosis produces a presinusoidal blockage. On the other hand, prehepatic obstruction of the portal vein results from congenital atresia, thrombosis, or extrinsic compression. Posthepatic portal hypertension is associated with the rare Budd-Chiari syndrome (thrombosis of the hepatic veins or obstruction of the retrohepatic vena cava). The underlying cause of portal hypertension influences the options for therapy. The most common causes of PHT are shown in Table 1.

Treatment options for portal hypertension

The management of PTH includes two methods: operative or non-operative method. Application of the surgical shunts in the 1940s has significantly improved the treatment results of PTH and became gradually the major modality in the treatment. In recent years, the investigation of mechanism of PTH and the development of modern endoscopy have improved the results of medication and endoscopic treatment. At present, shunt and disconnection are commonly used for the treatment of PTH.

The most urgent indication for the surgical treatment of portal hypertension is hemorrhage. Life-threatening hemorrhage results from the rupture of submucosal esophageal varices as a consequence of increased portal pressure and flow. Other causes of upper gastrointestinal tract bleeding are portal hypertensive gastropathy, gastritis, peptic ulcer and

Mallory-Weiss syndrome. However, the severity of varices or the probability of the varices ruptures are not directly related to the measured portal pressure. Each episode of hemorrhage carries a mortality of about 50%. After bleeding stops, the likelihood of recurrent bleeding without specific therapy to reduce portal pressure is about 75% [2]. In recently published meta-analysis [3], the portosystemic shunting procedures were compared with the endoscopic therapy (ET) of variceal haemorrhage. Researchers found that shunt therapy compared with ET demonstrated significantly less re-bleeding (OR 0.24, 95% CI 0.18 to 0.30) at the cost of significantly increased acute hepatic encephalopathy (OR 2.07, 95% CI 1.59 to 2.69) and chronic encephalopathy (OR 2.09, 95% CI 1.20 to 3.62). Also, differences regarding mortality were not significant, while the proportion of the patients with shunt occlusion or dysfunction was 3.1% (95% CI 0.4 to 10.7%) for total shunt, 7.8% (95% CI 3.8 to 13.9%) for distal splenorenal shunt, and 59% (range 18% to 72%) for TIPS.

Table 1. The common causes of PHT

Extrahepatic Disease	Intrahepatic Obstructive Disease
Portal vein obstruction	Alcoholic cirrhosis
Thrombosis due to infection or trauma	Posthepatic cirrhosis
Cavernomatous transformation	Biliary cirrhosis (primary and secondary)
Congenital atresia or stenosis	Toxic (chemical or drug) cirrhosis
Extrinsic compression	Metabolic (genetic) cirrhosis
Hepatic vein (outflow) obstruction	(hemochromatosis)
Budd-Chiari syndrome	Nutritional cirrhosis (after intestinal bypass)
Constrictive pericarditis	Other forms of cirrhosis (cryptogenic, congestive, etc.)
Excessive portal blood flow	Alcoholic hepatitis
Arteriovenous fistula between hepatic artery and portal vein	Neoplasms and granulomas
Arteriovenous fistula between splenic artery and vein	Schistosomiasis
	Venoocclusive disease
	Congenital hepatic fibrosis
	Hepatoportal sclerosis

PHT- Portal hypertension

The portal pressure can be reduced below the critical threshold by various operative techniques. All shunts are aimed to reduce variceal pressures sufficiently to arrest or prevent bleeding. Extrahepatic portal hypertension is one of the most common entities wherein shunt surgery is performed with success. Surgical shunts are classified as: 1. Total; 2. selective or; 3. partial shunts based on their specific hemodynamic properties. An angiographically placed

stent or transjugular intrahepatic portacaval shunt (TIPS), are also treatment options.

1. Total shunts

Total shunts completely divert portal blood flow into the systemic circulation and arrest hemorrhage in about 95% of cases, but with a 40% to 50% incidence of hepatic encephalopathy. In emergency situations, when bleeding cannot be controlled by less invasive means, total shunts may be lifesaving and encephalopathy becomes a secondary consideration. Another advantage of total shunts is that these shunts have a wider diameter with rare shunt thrombosis incidence. However, total shunts have several disadvantages: no portal vein flow to the liver results in diversion of hepatotropic factors and leading to deterioration of liver functions, and up to 40% of patients develop encephalopathy over the long time. Also, thrombosis of the shunt is frequent if graft is utilized and ascites does not get relieved after the shunt surgery. In four controlled trials in the United States that compared the use of total shunts with controls, three demonstrated poorer survival in shunted than in non-shunted patients, [4-6] and one showed no significant difference [7]. Whereas shunting decreased the risk of death from hemorrhage, deaths from liver failure overshadowed this advantage. For this reason, shunts are performed after (or sometimes during) the first episode of hemorrhage [8].

1.1. Portacaval shunt

The two different surgical techniques in portacaval shunts are the end to side portacaval shunt as first described by Eck [9] and the side to side shunt. Portal blood is completely redirected into the IVC below the liver and the hepatic end of the portal vein is oversewn in the end to side shunt. Another total shunt is the side-to-side portacaval shunt. The difference from the end-to-side shunt is that the portal vein is not transected in the side-to-side shunt. The side to side portacaval shunt allows blood from the intestine and the spleen to flow easily into the IVC. In addition, the hepatic end of the portal vein is changed into an outflow tract thus decreasing sinusoidal hypertension. Therefore it is the shunt of choice in Budd-Chiari post hepatic PHT, resulting in long term palliation of the disease with arrest or delay in the progression of hepatic fibrosis and ultimate failure [10].

In patients with an unsuitable splenic vein, a portocaval H-grafts shunts are preferred [11] over mesocaval shunts [12] since a shorter graft is needed, probably has a lower occlusion and postoperative ascites rate, and can easily be taken down during liver transplantation.

Collins et al. [11] reported 95% cumulative shunt patency over 7-years follow-up period and 65% survival after 5 years, with re-bleeding in 8% of the

patients. However, 30-day mortality in this cohort was 7.7%. Rosemurgy et al. [13] in a study group of 33 patients noted re-bleeding in 5.4 % of patients and 97% cumulative shunt patency during 4-years follow-up with de novo encephalopathy in 5% of their patients.

1.2. Mesocaval shunt

Mesocaval shunt is either constructed directly as a side to side anastomosis between the two veins or with the interposition of a short autologous vein graft - internal jugular vein or a prosthetic graft [14]. This shunt is indicated in patients with central thrombosis of portal vein. Vein graft is preferred as the incidence of thrombosis is particularly less. It has been used in children in a variety of settings and diseases with uniformly acceptable results [15]. This option is preferred by some surgeons as it allows an anastomosis between two large diameter vessels and because the length of the anastomosis can be increased to some extent to facilitate the creation of a large venous fistula. Splenectomy necessarily need not be done, except in cases with hypersplenism and so preventing post splenectomy sepsis [16]. A disadvantage of the mesocaval H graft is that the graft is relatively long, so there is a greater risk of graft occlusion by kinking or thrombosis.

Paquet et al. [12] reported 95% cumulative mesocaval H-graft shunt patency over 6-years follow-up period and 68% survival after 5 years. Also, Orozco and Mercado [17] reported 70% survival rate during 5-years follow-up period with 4.5% incidence of re-bleeding.

1.3. Proximal Splenorenal shunt (Linton)

It is valuable for the treatment of patients with severe hypersplenism and thrombocytopenia, because the spleen has been removed at the onset of the operation, facilitating thrombocytosis and coagulation. Extrahepatic portal hypertension in children, produced by the portal vein neonatal thrombosis, causing oesophageal variceal haemorrhage at any age from birth to adolescence can be relieved by a central splenorenal venous anastomosis, sometimes as small as 4 mm in diameter, with only an 8% chance of failure. The only issue is that children virtually never die from variceal bleeding, because their compensatory systems are so resilient.

Prasad et al. [18] performed 160 proximal splenorenal shunt surgery and reported overall operative mortality rate 1.9% (3/160); 0.7% (1/140) after elective operations and 10% (2/20) after emergency operations. Also, in this series, re-bleeding occurred in 17 patients (11%), but none of the patients developed encephalopathy. In addition, survival rate was good; 95% after 15 years follow-up.

2. Selective Shunts

The distal splenorenal shunt was developed to avoid the high rate of encephalopathy associated with

the use of total shunts [19]. Anastomosing the distal splenic vein to the left renal vein selectively decompresses the gastric and splenic veins, while maintaining relatively high pressures in the mesenteric and portal veins. Dividing the left gastric (coronary) vein and disconnecting the gastrosplenic and portomesenteric compartments by collateral ligation, remains an important part of the procedure [20,21].

Hepatopedal blood flow is preserved initially, with a low incidence of encephalopathy. In patients with alcoholism, collateral channels tend to dilate over time, eventually converting the selective shunt to a total one [22]. This shunt is rarely used in emergencies because portal decompression is selective, requiring time for bleeding to stop, and the procedure itself is time-consuming. A meta-analysis of four randomized trials that compared the use of the distal splenorenal shunt with that of sclerotherapy found that the Warren shunt reduced the risk of re-bleeding, did not worsen encephalopathy, and improved survival in nonalcoholic patients [23]. Patients with alcoholism did not have improved survival. As an elective procedure in patients with portal hypertension from causes other than alcoholic cirrhosis, the Warren shunt surgical procedure is an effective and durable operation with extensive application worldwide. In several series [24-26], control of hemorrhage is nearly equivalent to that for total shunts (> 89%) and the incidence of encephalopathy is less than 17%. Also, survival rate is similar as for total shunts; after 5 years follow-up is 60-85%. The Warren distal splenorenal shunt is particularly well suited for managing patients with extrahepatic portal vein thrombosis, of whom about 80% will have a patent splenic vein and thus be candidates for this procedure [27].

In children distal splenorenal shunting is used primarily in subject with extra hepatic portal vein thrombosis, stable Child class A or B cirrhosis or less common forms of intrahepatic portal hypertension such as congenital hepatic fibrosis with well preserved liver function but symptomatic variceal bleeding. It has also shown a lot of benefit in children with advanced hypersplenism [28].

3. Partial shunts

Partial shunts were first proposed by Bismuth and associates [29]. Variceal bleeding occurs above a corrected portal pressure threshold of 12 mm of mercury. Partial decompression of the portal vein to a pressure less than the critical threshold should stop variceal hemorrhage while preserving hepatopedal blood flow and preventing encephalopathy.

Sarfey and al. [30] reported a prospective, randomized trial comparing the use of large-diameter total shunts with that of small-diameter partial shunts interposed between the portal vein and vena cava. Both shunts were 100% effective in controlling hemorrhage

with long-term patency over 95%. Partial shunts preserved hepatopedal flow in 90% of patients and had a notably reduced incidence of encephalopathy.

The use of a small-diameter H graft is recommended for patients with Child class A or B alcoholic cirrhosis and at least one previous episode of variceal hemorrhage [31]. Although this procedure has succeeded in the emergency control of the bleeding and in patients with Child class C cirrhosis, the associated high mortality (around 50%) is unacceptable. In class C cirrhosis that could not be improved by medical treatment, a rational alternative is TIPS followed by liver transplantation.

Non Shunt surgery – Devascularisation

Various esophagogastric devascularization procedures have been used as both emergency and elective treatment of bleeding esophageal varices. These devascularisation procedures, also known as portal non-decompressive procedures or porto-azygos disconnection include procedures which aim to control the gastroesophageal varices either by direct on varices (variceal ligation of esophageal varices or esophageal/gastric transection) or by disconnecting varices from their feeding vessels. The main steps of the procedure described by Sugiura and Futagawa [32] involves transthoracic extensive devascularisation of lower esophagus from the level of the left inferior pulmonary vein up to the diaphragm, esophageal transection followed by end to end anastomosis, transabdominal devascularisation of upper half of the stomach, splenectomy, vagotomy and pyloroplasty. According to recommendations, indication for devascularization procedures are: idiopathic PTH, patients with Child class A or B cirrhosis, extrahepatic PTH, and patients who do not show shuntable venous vessels. Sugiura and Futagawa [32] in 1984 reported 4.3% operative mortality and 2.3% rebleeding rate, with 80% survival after 5 years follow-up. However, reported mortality rate of 16-60% and rebleeding rate of 20-64% [33], led the Sugiura procedure into disrepute because of high mortality and high incidence of rebleeding.

Transjugular intrahepatic portosystemic shunt (TIPS)

In essence TIPS is a side-to-side portosystemic shunt with decompression in the portal system similar as surgical shunts. Depending on the shunt diameter various amounts of portal flow volume are diverted from high-resistance liver parenchyma into the systemic venous circulation. Hemodynamically, TIPS differs little from surgical shunt procedures [34]. Therefore the rate of postshunt encephalopathy does not differ considerably in comparable stages of liver function and shunt diameter [34-36].

Indications for TIPS placement are:

- Acute variceal bleeding that cannot be successfully controlled with medical treatment, including sclerotherapy
- Recurrent and refractory variceal bleeding or recurrent variceal bleeding in patients who cannot tolerate conventional medical treatment, including sclerotherapy and pharmacologic therapy
- Therapy for refractory ascites [37]
- Portal decompression in patients with hepatic venous outflow obstruction (Budd-Chiari syndrome), [38] hepatic hydrothorax, or hepatorenal syndrome
- Contraindications include the following:
- Right-sided heart failure with increased central venous pressure
- Polycystic liver disease
- Severe hepatic failure
- Active intrahepatic or systemic infection
- Severe hepatic encephalopathy poorly controlled with medical therapy
- Hypervascular hepatic tumors

There are both advantages and disadvantages of TIPS over surgical shunts. TIPS can be performed under local anesthesia with a low procedure-related morbidity and mortality (0.5–4%). Hence TIPS represents the better option for emergencies, ascites, poor liver function and high operative risk. TIPS can be calibrated or occluded at any time if liver failure occurs. However, a high rate of dysfunction caused by stenosis and occlusion is still the main drawback to TIPS.

A major concern of a newly placed TIPS is new-onset or worsened encephalopathy, which occurs in about 25% of treated patients [39,40]. Patients with preprocedural hepatic encephalopathy or Child class C cirrhosis are more likely to have this complication. In a retrospective case analysis of 136 patients post-TIPS by Masson et al, hepatic encephalopathy developed in 34.5% of patients, and the frequencies were similar with covered and uncovered stents. The most significant predicting factor was the presence of pre-TIPS hepatic encephalopathy. Minimal encephalopathy occurred in 49% of patients at 26-month follow-up; and 10.3% of patients developed post-TIPS encephalopathy that required liver transplantation or contributed to death. The authors concluded that although post-TIPS hepatic encephalopathy is rather common, it is usually short-lived and well managed if patients are carefully selected for the procedure [39].

This requires close surveillance by Doppler-ultrasound and interventional correction to maintain or to restore shunt patency. The incidence of TIPS stenosis or occlusion which requires at least one re-

intervention is reported to be 30–70% after 1 year [34, 35]. The problem of late TIPS stenosis and dysfunction may be overcome by polytetrafluoroethylene (PTFE) covered stents (stent grafts). By improving the patency rate TIPS may evolve from a multistage to a one-step procedure in the future [34]. On the other hand, the mortality rates of elective shunt surgery in good-risk patients can be kept near zero in experienced centers [41–43].

In conclusion, portal hypertension is one of the main causes of morbidity and mortality in patients with severe liver diseases. The most serious complication of PHT is haemorrhage due to rupture of gastric and

esophageal varices and requires primary prophylaxis, acute bleeding management and secondary prophylaxis of re-bleeding. In past decades, management of PHT developed from invasive total shunt surgery to recent minimal-invasive procedures. However, due to lack of clear evidences, procedure effectiveness and relatively high procedure and post-procedures complications, best therapeutic solution is still subject of many clinical studies. Furthermore, future investigations also need to give strong criteria for appropriate patient selection for surgical therapy in different stage of this life-threatening disease.

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