Surgical Shunt Versus TIPS for Treatment of Variceal Hemorrhage in the Current Era of Liver and Multivisceral Transplantation

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KEYWORDS

• Portal hypertension • Portomesenteric venous thrombosis
• Surgical shunt • Transjugular intrahepatic portosystemic shunt
• Multivisceral transplant • Hypercoagulable status

Variceal hemorrhage occurs as a result of portal hypertension due to different underlying diseases such as portal vein thrombosis (prehepatic), liver cirrhosis (hepatic), and Budd-Chiari syndrome (posthepatic).[1,2] The interplay between the portal hemodynamic changes and hepatic reserve (**Table 1**) plays a major role in the short and long-term management of these complex patients. Of the important observed hemodynamic changes, is the increase in the intrahepatic vascular resistance compounded by the vasoconstrictor effect of a deficient state of intrahepatic nitric oxide. Of the major sequelae are the development of portosystemic collaterals, gastroesophageal varices, and a chronic state of systemic hyperdynamic syndrome.[3–7]

The natural history of the underlying liver disease is another major factor that should be considered for the establishment of the management algorithm of these patients. Ethanol abuse, chronic viral hepatitis, and cholestatic/autoimmune disorders are the most common liver diseases that significantly influence the long-term outcome with the currently available different therapeutic modalities that are discussed herein. Another important factor that further complicates the management plan of these complex patients is the presence of extensive splenic and portomesenteric venous

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Table 1
Child-Pugh classification

	1	2	3
Albumin (g/dL)	>3.5	2.8–3.5	<2.8
Bilirubin (mg/dL)	<2	2–3	>3
Prothrombin time[a]	1–3	4–6	>6
Encephalopathy	None	1–2	3–4
Ascites	None	Slight	Moderate

Child-Pugh A = 5–6 points: excellent hepatic reserve; Child-Pugh B = 7–9 points: good hepatic reserve; Child-Pugh C = 10–15 points: poor hepatic reserve.
[a] Seconds prolonged.

thrombosis that develops in cirrhotics and in hypercoagulable patients with normal hepatic parenchyma.

Recent advances in pharmacologic and endoscopic management of gastroesophageal variceal bleeding has significantly shifted the main paradigm of therapy from surgical to medical management.[8–14] However, medical failure does occur with an incidence of 20%, which requires radiologic or surgical intervention to decompress the portal system as a palliative but life-saving procedure. Nonetheless, organ replacement with liver alone or a multivisceral graft is the ultimate and definitive treatment for these patients.[13–15] In this article, a management algorithm is outlined to guide and optimize therapy for acute and recurrent variceal bleeding in these unique patients.

EVALUATION GUIDELINES

The standard evaluation process of patients with variceal bleeding has been comprehensively addressed in the literature.[9–13] However, special emphasis should be placed on the history of the gastrointestinal hemorrhage including the number and severity of episodes. Documentation of the source of bleeding and the location of varices must be established to exclude other sources and guide therapy. The presence of gastric varices in the absence of Food and Drug Administration approval to use cyanoacrylate in the United States limits the therapeutic use of endoscopic therapy and calls for radiologic or surgical intervention.[11] The role of new endoscopic techniques including the recently introduced endoscopic capsule has increased the accuracy of variceal detection, particularly in patients with enteric and ectopic varices. The diagnosis of portal hypertensive gastropathy and/or colopathy, in the absence of gastrointestinal varices, may also shed some light on the source of bleeding and guide further therapy.

The status of portal hypertension can be semiquantitatively assessed by the degree of the pancytopenia, splenomegaly, and radiologic evidence of intra-abdominal visceral collaterals in addition to the endoscopic documentation of gastrointestinal varices, gastropathy, and/or colopathy. Noninvasive radiologic studies could also be helpful as a screening test for the possible coexistence of splenic or portomesenteric venous thrombosis. In persons with radiologic evidence of partial or complete visceral venous thrombosis, a hypercoagulable syndrome must be suspected and thoroughly evaluated. The evaluation process includes measurement of protein C, protein S, antithrombin III, and total homocysteine serum levels. In addition, genetic studies for factor V Leiden, prothrombin G20210A, and JAK-2 gene mutations should be conducted. Equally important are the diagnosis of paroxysmal nocturnal