Portal Hypertension and Shunting

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Algorithmic Approach

A. The diagnosis of portal hypertension typically occurs with imaging demonstrating cirrhosis or secondary signs such as hypersplenism, ascites, and/or bleeding from varices. It is important to perform a history and physical examination, obtain blood work, and perform dedicated liver imaging. Portal hypertension is broadly categorized as related to cirrhosis or noncirrhosis etiologies.

B. Noncirrhotic etiologies are classified into three general groups: pre-hepatic, intrahepatic, and posthepatic. Prehepatic causes include splenic vein thrombosis or portal vein thrombosis. Left-sided portal hypertension (sinistral hypertension) may be related to severe pancreatitis with splenic vein thrombosis or postsurgical splenic vein ligation. In patients with varices and bleeding, splenectomy should be considered; asymptomatic patients can be monitored [1]. Portal vein thrombosis can be classified as acute or chronic and may be related to malignancy, cirrhosis, or a hypercoagulable state. In noncirrhotic patients, early anticoagulation is important to prevent varices from forming [2].

C. Intrahepatic causes of portal hypertension include schistosomiasis, a parasitic disease caused by trematode flukes, particularly *S. japonicum* and *S. mansoni*. Because of the immune response to parasite egg antigens, extensive fibrosis and hepatosplenic disease with periportal fibrosis can occur. Praziquantel is the treatment of choice, although oxamniquine is also effective [3]. Other intrahepatic causes include biliary disease such as biliary cirrhosis, neoplastic occlusion of the intrahepatic portal veins, developmental abnormalities such as polycystic liver disease or congenital hepatic fibrosis, and acquired diseases such as nonalcoholic fatty liver disease or inflammatory viral hepatitis. There are a multitude of intrahepatic etiologies; treatment generally centers on the prevention of severe complications such as variceal bleeding.

D. Posthepatic etiologies include Budd-Chiari syndrome or hepatic vein outflow obstruction. Treatment options include anticoagulation, short segment angioplasty, or transjugular intrahepatic portosystemic shunt (TIPS) in patients not in liver failure. If the inferior vena cava is patent and there is not a significant pressure gradient between the infrahepatic and suprahepatic portions, surgical shunting can also be offered, such as portacaval, splenorenal, or mesocaval shunts.

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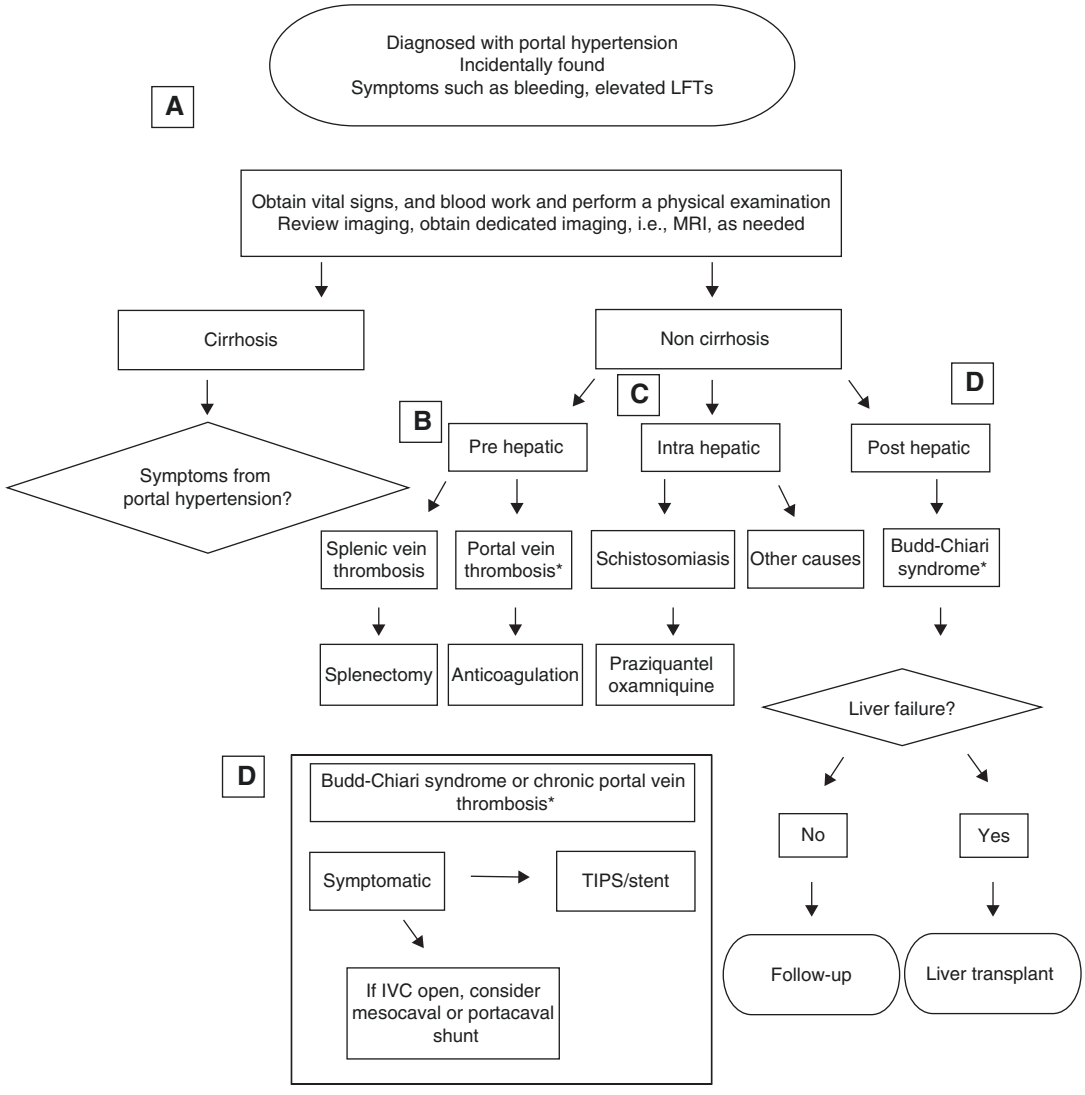
In patients with symptoms of liver failure, liver transplantation is offered [4].

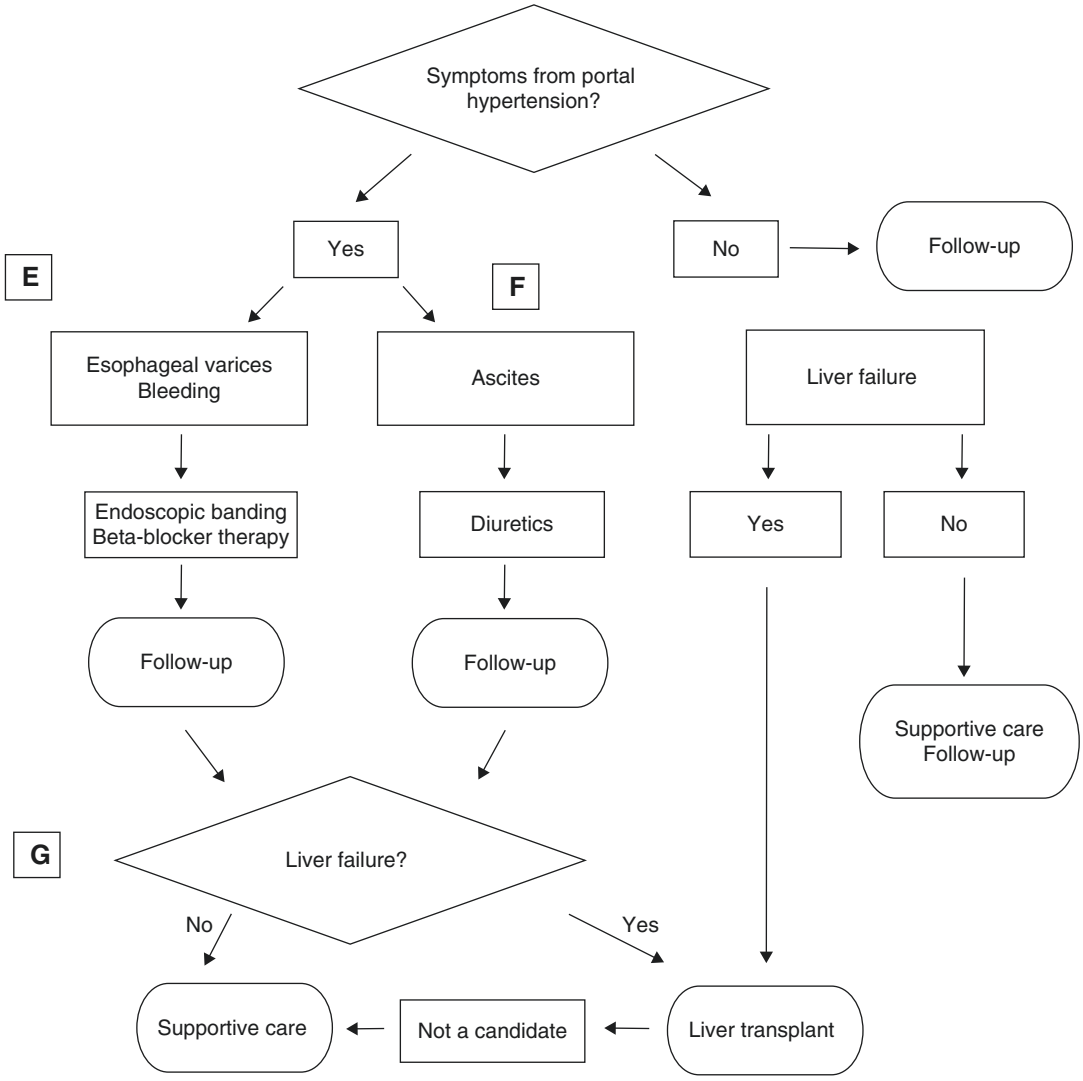
E. Care of the patient with portal hypertension is often directed toward management of symptoms. Patients with cirrhosis or platelet count <150,000 should undergo screening endoscopy. Prophylaxis of bleeding with a nonselective beta-blocker or endoscopic variceal ligation should be considered. Those with upper gastrointestinal bleeding from esophageal varices should undergo endoscopy with sclerotherapy, which controls bleeding in more than 90% of patients. If sclerotherapy fails, balloon tamponade can be used for temporary control, up to 24 h, followed by repeat endoscopy or TIPS [5].

F. For patients with ascites, paracentesis should be performed to evaluate cell count and differential, total protein, and serum-ascites albumin gradient. Typical management starts with sodium restriction (2gm/day) and diuretics (spironolactone, furosemide). For those with refractory ascites, serial paracenteses can be offered. For ongoing refractory ascites, liver transplantation should be considered [6].

G. For patients with refractory symptoms or development of end-stage liver disease, hepatorenal syndrome, or hepatopulmonary syndrome, liver transplantation is an important option, and referral to transplantation should be done, unless there is an underlying psychiatric or medical contraindication.



Algorithm 83.1



Algorithm 83.1 (continued)

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