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Portal Vein Thrombosis in Cirrhotic Patients: Decision to Anticoagulate

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Description

The portal vein is formed by the confluence of the splenic and superior mesenteric veins. Portal vein thrombosis (PVT) is caused by the formation of a blood clot within the extra-hepatic portion of the portal vein. Occlusion of the portal vein by thrombus typically occurs in patients with cirrhosis and/or prothrombotic disorders including hepatocellular carcinoma, myeloproliferative disorder, inherited thrombophilia and abdominal trauma [1]. Chronic portal vein thrombosis (PVT) may have a myriad of presentations including worsening clinical symptoms of portal hypertension i.e. ascites, gastroesophageal variceal bleeding or even completely asymptomatic, detected in routine abdominal imaging [1]. The management of portal vein thrombosis (PVT) includes differentiating acute from chronic, cirrhotic from non-cirrhotic causes, considering risk of variceal bleeding from anticoagulation, risk of bowel ischemia from clot extension and possibility of liver transplant [2].

The incidence of PVT is proportional with the severity of chronic liver disease: less than 1% in well-compensated cirrhosis, 7.4%-16% in advanced cirrhosis [3].

The differential diagnosis of chronic PVT include abdominal malignancy invading portal vein and constriction of the portal vein by a tumor [2,4]. Chronic PVT, unlike acute PVT, develops collateral circulation around the area of obstruction known as portal cavernoma and minimal chance of causing bowel ischemia [2]. Chronic PVT can be differentiated from acute PVT by the presence of cavernoma of porto-portal collateral vessels on US or CT imagings [3]. Chronic PVT is mostly associated gastroesophageal varices. Patients with chronic PVT should be screened for esophageal varices before deciding anticoagulation.

There has been no published consensus about management of non-malignant PVT in cirrhosis patients. The decision to start anticoagulation is to be made on a case-by-case basis. The goal of anticoagulation is to prevent recurrent thrombosis, prevent thrombus extension, and promote recanalization. Chronic PVT patients with inherited prothrombotic disorders and awaiting liver transplant, who do not have significant risk for variceal bleeding, are usual candidates for anticoagulation [4]. In advanced cirrhosis patients awaiting liver transplantation, the proportion of partial or complete recanalisation was found to be significantly higher in patients received anticoagulation (8/19) than in those who did not receive (0/10, p=0.002). Anticoagulation also reduced post-operative complications and improved overall survival [5].

Acute PVT does not get a chance to develop collateral, hence acute obstruction of the superior mesenteric vein and mesenteric arches can lead to intestinal ischemia, and life-threatening infarction [6]. Acute PVT patients need to be anticoagulated with a goal of preventing clot extension, bowel ischemia and development of portal hypertension.

A recent study on 56 chronic PVT patients (35 received anticoagulation and 21 control), 36% recanalization rate was achieved with low molecular weight heparin for at least 6 months in the treatment group, compared with 5% in control group. There was also a significant difference in thrombus progression between non-anticoagulated and anticoagulated groups (15/21 non-anticoagulated patients and in 5/33 anticoagulated patients). Five patients were reported to have variceal bleedings and two intestinal venous ischemia episodes in the control group, compared with one variceal bleeding episode in the study group [7].

Our patient developed portal vein thrombosis, in spite of having an INR of 2.1. Naturally occurring anticoagulants levels including Protein C and protein S go down in cirrhotic patients, making INR a less reliable indicator for anticoagulation monitoring. There is no consensus regarding the best choice of anticoagulation. LMWH usage is associated with administration of the injection, whereas coumadin requires INR monitoring and may impact MELD score [3]. Anticoagulation in PVT cases may be started with LMWH, followed by coumadin therapy for 3-6 months [4,6]. Every patient chosen for anticoagulation should undergo screening for large varices and endoscopic obliteration of varices should be provided before commencement of anticoagulation. However, some experts prefer beta blocker over the endoscopic obliteration of varices. There is a high risk of bleeding from an esophageal ulcer in case the clips slip from the varices.

Large randomized control trials are required before consensus guidelines could be formulated. Factor Xa inhibitors may become future choice of PVT treatment. However, safety of factor Xa inhibitors have not been established in decompensated liver disease patient and none of the currently available factor Xa inhibitors is FDA approved for this indication.

Learning Objectives

Portal vein is formed by confluence of superior mesenteric and splenic veins. Portal vein thrombosis is seen in decompensated cirrhotic patients as well as patients with hepatocellular carcinoma, inherited thrombophilia and myeloproliferative disorders.

Acute PVT is more likely cause bowel ischemia. On the contrary, chronic PVT develops well-formed collateral and portal cavernoma.

The decision to anticoagulate PVT patient is individualized and taken on case by case basis. Chronic PVT is associated with gastroesophageal varices and risk of GI bleeding if anticoagulated.

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