

Portal Vein Thrombosis in Decompensated Cirrhosis: Rationale for Treatment

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We thank Dott. Girleanu and coauthors for the comments and the interest in our article. We agree with the authors that patients with decompensated cirrhosis are a different population compared with patients with compensated liver disease (i.e., Child A class) when they develop portal vein thrombosis (PVT) (1). In fact, a recently published article that demonstrated that the occurrence of PVT does not influence the natural history of cirrhosis included only very stable patients with almost normal liver tests (2).

In this article, although the group of patients in Child C class was not very numerous, we were able to demonstrate for the first time that patients with cirrhosis with decompensated liver disease who do not resolve PVT are characterized by a higher risk of death at 2 years of follow-up; this was not the case in Child A patients.

This study was not interventional, and the therapeutic choice was left to the clinician's judgment. Actually, in the present cohort, the patients experienced the same number of bleeding episodes if PVT was not treated and during anticoagulation treatment, but significantly less after withdrawal of anticoagulation.

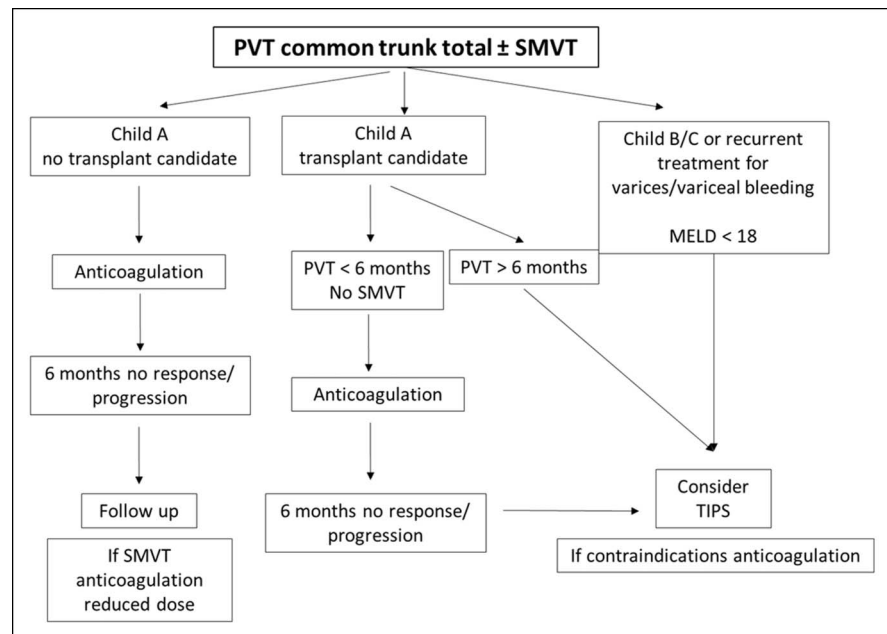


Figure 1. Treatment algorithm for complete PVT.

We interpreted this finding in relation to the possible decrease in portal pressure in those who resolved PVT. The period of anticoagulation was shorter than that in the study by Villa et al. (3), and the dose was mostly therapeutic, which can justify the higher percentage of bleeding during treatment.

Moreover, the best therapeutic approach in patients with PVT and decompensated liver disease with severe portal hypertension may not be anticoagulation as first-line treatment in all the cases.

We agree that acute PVT is difficult to catch, especially in patients with cirrhosis, probably because collateralization of the splanchnic venous system is already present, and the acute occlusion of PVT may be less symptomatic in the short term. We agree with the authors that classification of the PVT should not be merely anatomical, but based on the chance of response to anticoagulation (4), underlying the stage of cirrhosis, presence or complications of portal hypertension, and eventual candidacy of the patient to liver transplant (5).

A new classification was recently proposed (6), which may be the base to

create a treatment algorithm. Probably, a transjugular intrahepatic portosystemic shunt should be considered early in the course of PVT in some patients, in particular in liver transplant candidates. A proposal of treatment algorithm for complete PVT incorporating radiological intervention is represented in Figure 1.

CONFLICTS OF INTEREST

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