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Letters to the Editor

Anticoagulation in a cirrhotic patient with acute portal vein thrombosis unrelated to malignancy. A case report

Key words: Acute portal thrombosis. Anticoagulation. Cirrhosis.

Dear Editor,

Anticoagulation is an accepted treatment in acute portal vein thrombosis (PVT) unrelated to cirrhosis. However, the usefulness of this therapy in cirrhotic patients has not been well documented. We report the case of a cirrhotic patient who received anticoagulant therapy.

Case report

A 46-year old man, with the antecedent of lung abscesses in 2003, was diagnosed with alcoholic cirrhosis in 2006, when he developed ascites. In October 2008, he was admitted to hospital again with ascites (ASAG 1.74). Doppler ultrasound revealed extrahepatic and intrahepatic portal vein thrombosis, with patent splenic vein and no collateral vessels nor portal cavernomatosis. It showed also enlarged spleen and ascites. MR ruled out the presence of hepatic nodules suggesting hepatocellular carcinoma. Small esophageal varices were seen by upper endoscopy. With the suspicion of acute thrombosis, anticoagulant treatment with acenocumarol was given. Laboratory investigation for prothrombotic disorders showed deficit of protein C and antithrombin III, probably related to decreased synthesis secondary to liver disease. After six months of treatment, Doppler ultrasound showed that intra- and extrahepatic portal vein was patent and spleen was smaller. Anticoagulation was discontinuated after one year of treatment, after assessing normalization of protein C and antithrombin III, as recommended by the haematologist. Doppler scan showed no signs of recurrence of thrombosis three months later.

Discussion

Portal vein thrombosis is not an uncommon complication of cirrhosis, with a prevalence of 25% in advanced liver disease (1). It is caused by an inbalance between procoagulant and anticoagulant factors synthesized in the liver, as well as by a decrease in portal flow speed. However, there are other etiologic factors such as prothrombotic disorders, especially mutations in Leiden V factor and prothrombine gene, frequently associated in cirrhotic patients with PVT (2). It is also well known the relationship between PVT and hepatocellular carcinoma (3), so malignancies should be ruled out by imaging techniques, such as MR or triphasic CT. PVT can induce decompensation of underlying liver disease, with ascites, hepatic encephalopathy, worsening of liver function, jaundice or variceal bleeding (2). In addition, in candidates for liver transplantation, PVT is likely to make technically difficult, or even impossible, to perform it. For that reason, Francoz et al. made a study in 29 cirrhotic patients with acute PVT unrelated to hepatocellular carcinoma; 19 of them were anticoagulated achieving portal recanalization in 42%, whereas none of the patients who did not receive anticoagulation did. No significant side effects were seen. Thus, they recommend that patients with PVTshould be given anticoagulation if they are candidates for liver transplantation, if they have upper mesenteric vein thrombosis (because of the risk of intestinal infarctation), and when a prothrombotic disorder exists (risk of thrombosis recurrence) (1). However, more recent guidelines do not recommend anticoagulation as a general rule in these patients, but an individual case by case

assessment, always taking into account the risk of variceal bleeding (4).

Our patient was admitted to the hospital because of ascites; PVT was demonstrated and hepatocellular carcinoma was ruled out by MR. Upper endoscopy was performed to assess the potential risk of variceal bleeding and laboratory investigations for prothrombotic disorders were made, before giving anticoagulation to the patient. The outcome of therapy is remarkably good, achieving complete resolution of intra and extrahepatic thrombosis, and the decrease of the spleen size, which shows the relationship between PVT and portal hypertension.

In summary, we think that anticoagulation in cirrhotic patients with PVT is a useful therapy in selected patients, given that hepatocellular carcinoma has been ruled out and the risk of variceal bleeding is low. Prothrombotic disorders must be investigated because long term treatment may be necessary in those cases. Nevertheless, more studies are required before this strategy can be generalized.

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