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Idiopathic portal hypertension with regard to thiopurine treatment

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Dear Editor,

We read with interest the paper "Idiopathic portal hypertension regarding thiopurine treatment in patients with inflammatory bowel disease" (1), which was recently published in this journal. The paper reported four cases of idiopathic portal hypertension (IPH). In this letter, we would like to comment on a recent case treated at our unit.

Case report

We present the case of a 76-year-old male with ileocolic Crohn's disease treated with azathioprine. After ten years of treatment, he presented with a severe outbreak that required a resection of the terminal ileum. During the intervention, non-purulent ascites were observed which were culture negative. One month later, he presented with low-grade fever, abdominal pain and grade 2 ascites. The paracentesis showed a transudate without infection criteria, negative ascites and blood cultures. Empirical treatment with broad-spectrum antibiotics was initiated. The ascitic fluid had a serum ascites albumin gradient of 11 g/l that was compatible with portal hypertension. A

computed tomography (CT) scan identified a homogeneous liver of a normal size and appearance, a splenomegaly of 13 cm and ascites. Endoscopy revealed small esophageal varices. The portal hemodynamics had a normal portosystemic gradient of 5 mmHg, compatible with presinusoidal portal hypertension. A liver biopsy identified hepatoportal sclerosis with mild changes due to nodular regenerative hyperplasia (NRH) (Fig. 1).

Discussion

Hepatoportal sclerosis and NRH are part of the spectrum of IPH. The lesion usually occurs insidiously and thrombocytopenia is usually the first manifestation (2). The natural history is not well established. However, cases of histological regression have been described with the withdrawal of the drug (3). However, it is usually diagnosed late due to its asymptomatic nature. Some studies show that lower levels of 6-TGN indicate a lower incidence of IPH and therefore, monitoring is recommended (4). It is important to guide the pathologist in a case of clinical suspicion; one study reported a k = 0.20 for NRH when the clinical information was inadequate (5).

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Fig. 1. A. Hematoxylin-eosin staining, 400x. Hepatoportal sclerosis. Portal space with the presence of a bile duct and an arteriole (*) with the absence of the portal vein. B. Hematoxylin-eosin staining, 200x. Mild changes of nodular regenerative hyperplasia, with hypertrophic hepatocytes that seem to compress other surrounding hepatocytes of an atrophic appearance.