

Portosystemic venous shunt: portocaval fistula in a patient with biliary cirrhosis secondary to a right hepatectomy for hydatidosis

Key words: Fistula. Hepatic encephalopathy. Cirrhosis.

Dear Editor,

Major intrahepatic venous shunts are rare conditions where a communication between hepatic veins and intrahepatic portal vessels is established. Potential spontaneous development has been proposed in patients with cirrhosis and portal hypertension (PHT).

Case report

We report the case of a 62-year-old male who underwent surgery in 2002 due to a hepatic hydatidosis consisting of a right partial hepatectomy, cholecystectomy and bilioduodenal bypass. The patient developed biliary cirrhosis with PHT (Child-Pugh B9, MELD 10) secondary to a benign left hepatic duct stricture following surgery. He had a first hydropic decompensation event in 2012 and has been on spironolactone ever since. He never developed encephalopathy during the course of the illness.

In 2015, he developed bradypsychia and confusion with no other clinical or laboratory changes with a similar liver function and treatment. Due to the absence of any obvious clinical triggers, a chest-abdominal computed tomography (CT) scan was performed which revealed a large portosystemic shunt from three right-side portal branches towards the inferior vena cava. He underwent a partial embolization using a 16 mm Amplatzer device and multiple coils.

No subsequent complications were observed and the patient remained free from encephalopathy (last follow-up in January 2016).

Discussion

Very few cases of large portosystemic venous shunt have been described in the literature. Shunts may be congenital or acquired (biopsy, trauma, liver surgery, chronic liver disease with PHT) (1,2). Interestingly, our patient had several of these acquired etiologic triggers. In the presence of liver cirrhosis, shunts usually manifest as hepatic encephalopathy, which may develop with a preserved liver function (3). Arterial portography allows selective embolization of the portosystemic shunt (4). In cirrhotic patients with PHT treatment, this may lead to the opening of both intra- and extra-hepatic new collateral vessels that may perpetuate the problem. Furthermore, complete shunt closure may increase PHT (5). Following a partial shunt closure, our patient has remained free from encephalopathy and other PHT-derived complications.

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