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EDUCATION AND IMAGING

Hepatobiliary and Pancreatic: An uncommon cause of portal hypertension

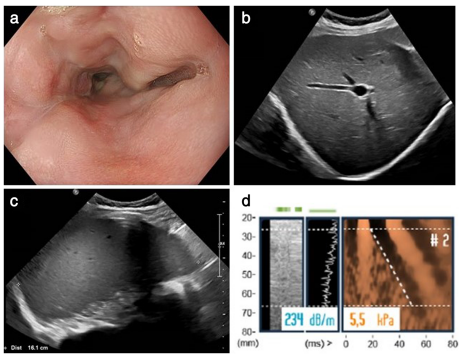

Figure 1 Upper gastrointestinal endoscopy showing Grade 2 esophageal varices (a); abdominal ultrasound revealing normal liver surface (b) and a splenomegaly (c) and vibration-controlled transient elastography results showing normal liver elasticity compatible with the absence of fibrosis (d).

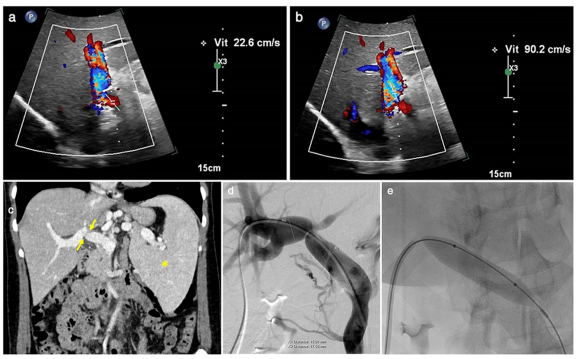

Figure 2 Focal stenosis of 2 mm long of the portal trunk before bifurcation: normal portal flow (22.6 cm/s) upstream the stenosis (a) *vs* accelerated flow (90.2 cm/s) downstream the stenosis (b); computed tomography, portal phase, axial section with portal vein kinking (arrows) and signs of portal hypertension, for example, splenomegaly (*) (c); portal vein catheterism and angiography (d) and balloon dilatation of the stenosis (e).

A 42-year-old female patient was referred to our hepatology clinic after identification of Grade 2 esophageal varices at upper gastrointestinal endoscopy (Fig. 1a). Her medical history is remarkable for a laparotomy cholecystectomy in Brazil back in 2013. The body mass index is normal. On clinical examination, the abdomen is diffusely tender. The spleen is palpated. A right subcostal laparotomy scar is visible. Blood analysis showed isolated thrombocytopenia (platelet count 91 000/mm^3) and mildly elevated transaminases (AST 37 UI/L, ALT 43 UI/L). There was no cholestasis nor prothrombin time prolongation. Abdominal ultrasound was remarkable for a slight elevation of hepatorenal index (suggestive of mild steatosis), normal liver contours (Fig. 1b), a splenomegaly (Fig. 1c), and mild ascites. Vibration-controlled transient elastography further confirmed slight steatosis (controlled attenuation parameter [CAP™] 221 dB/m) and excluded advanced fibrosis (elasticity [E] 5.5 kPa with a medium probe) (Fig. 1d). What is your diagnosis and what do you recommend?

The initial workup directed toward noncirrhotic portal hypertension. We repeated an ultrasound that highlighted a focal stenosis of 2 mm long of the portal trunk just before bifurcation. Doppler-analysis showed normal portal flow (22.6 cm/s) upstream the stenosis (Fig. 2a), contrasting with accelerated flow (90.2 cm/s) downstream the stenosis (Fig. 2b). Contrast-enhanced computed tomography confirmed the portal vein stenosis and kinking (Fig. 2c).

We concluded that the patient suffered from presinusoidal portal hypertension in the setting of a short and tight extrahepatic portal vein stenosis. Portal vein stenosis usually develops due to postoperative inflammation or oncological processes. Benign stenosis has been described in patients after duodenopancreatectomy or liver transplantation especially in children. In our patient, it most likely is the consequence of a cholecystectomy. To the best of our knowledge, this is the first report of portal vein stenosis complicating a cholecystectomy.

The treatment was based on existing literature regarding late-onset portal stenosis in liver transplant recipients and consisted in a percutaneous transhepatic endovascular balloon angioplasty from 5 up to 12 mm in diameter (Fig. 2d,e). Residual transstenotic gradient after the procedure was <2 mmHg. The procedure was well tolerated except for moderate abdominal pain for 3 days following the procedure and a transient elevation of bilirubin level. Portal vein thrombosis prophylaxis (fraxiparin 3800 IU od) was administered for 10 days after the intervention.

At the 2-month follow-up, the patient reported that her digestive symptoms had disappeared. The controls show marked regression of signs of portal hypertension. Indeed, thrombocytopenia had resolved (platelet count of 170 000/mm^3), the spleen long axis had shrunk to 13.9 cm, and the mild ascites had disappeared. An upper gastrointestinal endoscopy was performed 6 months after treatment, showing complete disappearance of the esophageal varices.

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