An unusual cause of hepatic encephalopathy

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A 72-year-old patient with compensated liver cirrhosis caused by chronic hepatitis C virus infection was admitted to our department because of malaise and aggravating mental confusion without pathological neurological signs. The results of liver function tests were similar to those obtained 6 months earlier (the Model for End-Stage Liver Disease [MELD] score was 13). The most frequent factors triggering hepatic encephalopathy (HE) such as bacterial infection, gastrointestinal hemorrhage, diuretic overdose, dyselectrolytemia, or protein-rich diet were excluded. A few months earlier, the patient underwent 3 consecutive sessions of endoscopic rubber banding. A liver ultrasound (Siemens Acuson X300, Siemens Medical Solutions, Inc, Malvern, Pennsylvania, United States) showed a subcapsular tumor with mixed echogenicity and a diameter of 2.5 cm, localized in segment 6 (FIGURE 1A). Further diagnostic workup with color Doppler ultrasound showed a portovenous shunt (FIGURE 1B), and helical 64-row computed tomography (Aquillon 64, Toshiba, Japan) revealed an aneurismal connection between the peripheral branch of the portal vein and hepatic vein (FIGURE 1CD).

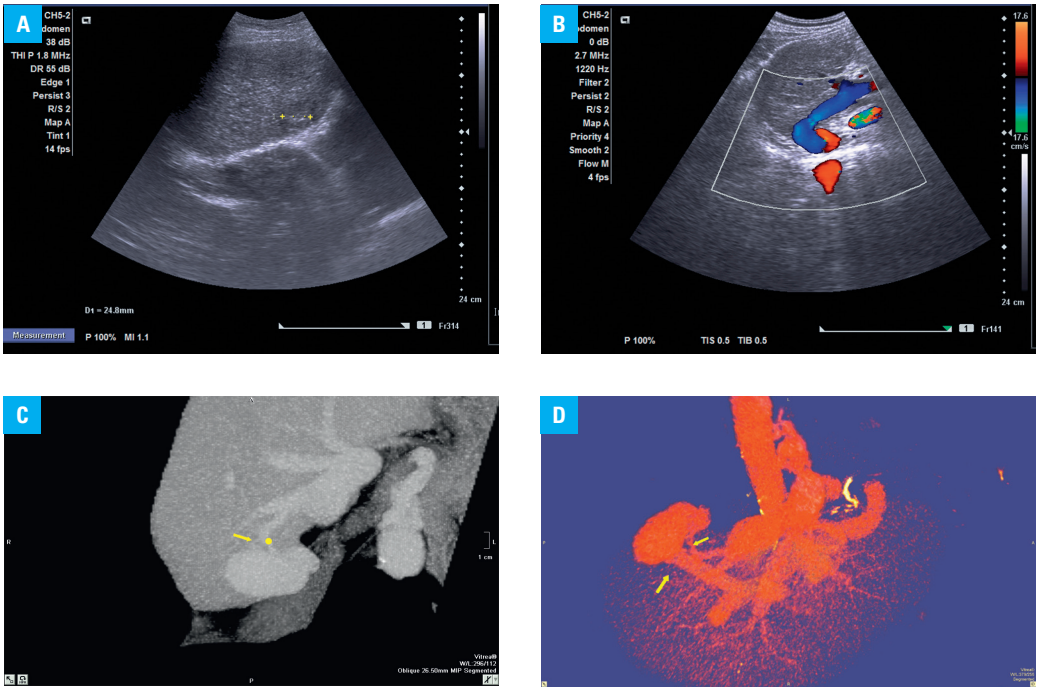

FIGURE 1 **A** – 2-dimensional ultrasound images of a portovenous shunt showing a liver tumor with mixed echogenicity; **B** – a color Doppler sonogram showing blood flow in a vascular (aneurismal) lesion located in the right liver lobe; **C** – an axial computed tomography (CT) image in the portal phase demonstrating a vascular lesion connecting the right branch of the portal vein with the right hepatic vein (scanning range, 1.0 mm); **D** – a CT image after reconstitution and multiplanar reformation showing the portovenous shunt (arrows)

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Nontumorous vascular malformations are rarely found in the liver, and their clinical manifestations depend on the type of the shunt. The shunt may remain asymptomatic or may lead to HE or heart failure.[1] HE is a serious but ususally reversible complication of liver cirrhosis, associated with an overdose of diuretics, bacterial infection, or gastrointestinal bleeding. Persistent HE is mostly linked to intra- and extrahepatic portosystemic shunts.[2] A classification by Park et al.[3] distinguishes 4 types of portohepatic venous shunts: 1) single large vessel linking the portal vein to the intrahepatic portion of the inferior vena cava; 2) a subcapsular communication between the peripheral portal and the hepatic vein branch situated in a single segment of the liver; 3) an aneurysmal connection between the peripheral portal and hepatic veins; and 4) multiple connections in both hepatic lobes.

A portohepatic shunt may be a complication of liver biopsy; however, in our patient, the biopsy was not performed. In theory, an aneurysmal connection might develop following the obliteration of esophageal varices, possibly leading to a significant increase in portal pressure. The clinical effect of an aneurysmal portohepatic shunt is similar to that of a transjugular intrahepatic portosystemic shunt, which is associated with an increased serum level of ammonia and increased risk of HE. The therapeutic option for the portohepatic shunt is intravascular embolization, but the method is limited to patients with preserved liver function defined as a MELD score of 11 or less. In conclusion, in patients with unexplained HE, the presence of tumor-like intrahepatic portosystemic shunts should be taken into account.

REFERENCES

1 Bodner G, Peer S, Karner M, et al. Nontumorous vascular malformations in the liver; color Doppler ultrasonographic findings. J Ultrasound Med. 2002; 21: 187-197.

2 Laleman W, Simon-Talero M, Maleux G, et al. Embolization of large spontaneous portosystemic shunts for refractory hepatic encephalopathy: amulticenter survey on safety and efficacy. Hepatology. 2013; 57: 2448-2457.

3 Park JH, Cha SH, Han JK, et al. Intrahepatic portosystemic venous shunt. AJR Am J Roentgenol. 1990; 155: 527-528.