Portal Cavernomatosis on MR and CT: what to look for and how to report it?

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Learning objectives

- To review imaging findings in patients with Cavernous transformation of the portal vein (CTPV).

- To recognize the possible errors in interpretation.

- To elaborate a structured report as useful diagnostic tool for radiology residents and the clinician.
Background

CTPV is defined as a multitude of collateral vessels in the hepatic hilum surrounding an occluded segment of the extrahepatic portal vein, with or without higher extension along the right and/or left portal vein branches. The extension of CTPV is classified as extrahepatic (if it is extended from the confluence of the splenic vein and superior mesenteric vein to the bifurcation of the left branch of portal vein), intrahepatic (if it is extended above the bifurcation of the left branch of the portal vein), or both extra- and intrahepatic.

CTPV can profoundly modify liver morphology and vascular hemodynamic. Among the changes in liver morphology there is the atrophy-hypertrophy complex, with atrophy of II and III segments with hypertrophy of I and/or IV segments. Changes in vascular hemodynamic are not rare and among them there are the transient differences in hepatic enhancement, the presence of an enlarged hepatic artery (defined as a hepatic artery diameter at the hepatic hilum equal to or larger than that of the splenic artery) and the presence of focal nodular hyperplasia-like (FNH-like) lesions. These latters represent a benign hyperplasia of the hepatic parenchyma in response to increased arterial perfusion in areas with reduced or absent portal blood flow. These lesions can be often found in the peripheral zones of the liver and are characterized by an homogeneous enhancement during the hepatic arterial phase and lack of washout during the hepatic venous and interstitial phases.

Although biliary symptoms appear to be uncommon, in most of the patients with portal cavenoma it has been found a portal biliopathy, maybe due to the compression by the vein composing the cavernoma and ischemic cholangiopathy. It is represented by strictures mainly involving the suprapancreatic portion of the common bile duct, a calculus cholecystitis and ascending cholangitis.

The presence of intra- or peripancreatic portal cavernoma (defined as portal collaterals in the pancreas or around the pancreas) is associated with extension of the thrombosis to the splenic and superior mesenteric vein and causes changes in the pancreas, like enlargement and heterogeneity, and main pancreatic duct abnormalities (dilatation [ when the duct measured more than 3 mm in diameter at the level of the body of the pancreas], stricture and irregularities).

Signs of portal hypertension, such as splenomegaly (defined as longitudinal diameter of the spleen greater than 12 cm), para-umbilical and gastroesophageal veins, spontaneous splenorenal shunts and ascites have also been reported in CTPV.
Findings and procedure details

We retrospectively reviewed all reports of CT and MRI performed in our Department from November 2010 to October 2014. CTPV was found in 62 patients, but, of these, 29 were excluded because they had history of cirrhosis, 13 pancreas tumor, 2 cholangiocarcinoma and 4 had undergone just an ultrasound examination or an exam without contrast medium injection.

The remaining 14 patients (mean age 56±13,26 years) were 10 men and 4 women.

The images of 10 CT and 4 MR scans were reviewed by two expert radiologists and a radiology resident. MDCT scans were performed before and after intravenous administration of iodine contrast medium using 128-slice MDCT and MR scans were performed with a 1,5 Tesla system, before and after intravenous administration of gadolinium chelate.

The etiology of CTPV was recognized in 7 patients (mainly mieloproliferative syndrome). The extension of CTPV was extrahepatic in 4 and intra and extrahepatic in 10 patients.

Major complications observed were the atrophy - hypertrophy complex, mainly segment IV hypertrophy (using a segment IV diameter limit of 35 mm), caudate lobe hypertrophy (measured with CL/RL-r) and left lateral segment atrophy, found respectively in 12, 10 and 9 patients [figure 1] Just in one patient there was an hyperplasia of other hepatic segments (V-VI) [figure 2]

Among the changes in vascular hemodynamic, we noticed areas of transient differences in the hepatic enhancement in 9 patients, enlargement of the hepatic artery in 5 patients [figure 3] and FNH-like lesions just in 1 patient who had been submitted to CT [figure 4].

Regarding portal biliopathy, common bile duct stricture was encountered in 8 patients, ascending cholangitis and acute cholecystitis in 1 [figures 5 and 6]

In all patients we identified secondary signs of portal hypertension. Lastly, the presence of an intra or peripancreatic cavernoma, was detected in 8 patients, but in none of them there were changes of the pancreas or main pancreatic duct abnormalities.
Reviewing radiology reports, we recognized that there had been a failure or incomplete description of the morphology of the liver in 13 cases, of the enlargement of the hepatic artery in 4 cases, of vascular changes in 4 patients, of signs of portal biliopathy in 3, of splenic vein thrombosis in 1, of FNH -like lesions in 1 and of signs of portal hypertension in 2.

Therefore, a comprehensive structured report for an accurate identification and description of findings in patients with CTPV should include a description of the morphology of the liver, alterations in blood flow, which can mimic other hepatic disorders (mainly cirrhosis and Budd-Chiari syndrome) and the detection of focal liver lesions. Moreover there must be an evaluation of biliary tree, taking in mind that the solid tumor-like cavernoma occludes the bile duct lumen mimicking a tumor, the evaluation of hepatic artery width, of the extension of the thrombosis, of the signs of portal hypertension and of other abdominal organs, mainly of the pancreas and the main pancreatic duct, whose enlargement and dilatation may mimic a tumor.
Fig. 1: Abdominal CT in the portal phase shows caudate lobe hypertrophy and portal cavernomatosis.

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Fig. 2: Liver MR in the arterial phase shows segmentary hyperplasia (S5-S6) and portal cavernomatosis.

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Fig. 3: Abdominal CT in the portal phase shows an area of transient difference in the hepatic enhancement, enlargement of the hepatic artery, portal vein thrombosis and portal cavernomatosis.

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Fig. 4: Abdominal CT in the arterial phase shows subcapsular FNH-like lesion in S2.

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**Fig. 5:** MR-cholangiography shows dilatation of the biliary tree in a patient with portal cavernomatosis. Main pancreatic duct has a regular caliber.

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**Fig. 6:** Liver MR in the portal phase shows ascending cholangitis and dilatation of intrahepatic biliary tree.

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Conclusion

Considering the lack of description of many important findings related to CTPV, adoption of a standardized imaging reporting template could be useful for the radiologist and accordingly for the clinician.
References


