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Mimics of Hepatic Neoplasms

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Introduction

The normal liver receives a dual blood supply from the hepatic artery and the portal vein, each supplying approximately 25% and 75% of blood volume, respectively.[1] Consequently, there is low enhancement of the normal liver parenchyma during the arterial phase as the contrast agent transported by the hepatic artery is diluted by a ratio of 4:1 by unopacified portal venous blood. It should be noted that hepatic neoplasms are usually predominantly supplied by the hepatic artery. This distinction is the basis for the multiphase contrast-enhanced liver protocol for computed tomography (CT) and magnetic resonance imaging (MRI). It is important to realize that the arterial and the portal venous supplies are not independent vascular channels; communications exist between the 2 via the hepatic sinusoids, with blood flowing through the liver sinusoids and emptying into the central vein of each lobule. The central veins coalesce into hepatic veins, which drain into the inferior vena cava (IVC). When vascular compromise occurs, the dual blood supply can change in the volume and the direction of blood flow; when the portal venous flow decreases or reverses the direction of flow, arterial flow increases to compensate.

Disruption to the inflow or the outflow of blood to the liver may give rise to nonneoplastic hypervascular lesions, vascular shunts, and perfusion abnormalities within the liver. In patients with diffuse liver disease, the background liver architecture is altered and the hepatic blood flow and drainage are disrupted. Therefore, great attention should be taken when evaluating a focal lesion in the setting of diffuse liver disease to avoid misdiagnosing a vascular lesion as neoplastic. The distorted architecture of the hepatic parenchyma in cirrhosis is a predisposing condition to develop an arterioportal (AP) shunt. Focal nonneoplastic lesions in hepatic steatosis, iron overload, and the combination of both can present a diagnostic dilemma. Pyogenic, fungal, and parasitic infections in addition to inflammatory conditions can involve the liver. If there is a solid component from granulation or inflammatory tissue, these lesions can mimic metastatic disease.

In this review, we discuss the appearances of liver lesions that may mimic neoplasms in vascular and perfusion disorders, diffuse liver disease, and infectious and inflammatory conditions.

Vascular and Perfusion Disorders

AP Shunts

AP shunts result from the entry of blood from a high-pressure arterial system to a low-pressure portal venous system through an abnormal connection. This leads to an increase in the regional perfusion pressure, resulting in a local parenchymal perfusion abnormality.[2] AP shunts can be divided into spontaneous, posttraumatic, and iatrogenic. Spontaneous AP shunts are common in the setting of cirrhosis. They are frequently peripheral, but can be central, and usually measure less than 2 cm (Fig. 1). These lesions represent abnormal vascularity with no clinical significance or pathologic alterations and can resolve on subsequent imaging.[3-5] They can be associated with focal lesions such as hepatocellular carcinomas and hemangiomas.[6] In comparison with AP shunts, which are spontaneous, AP fistulas are most commonly iatrogenic and represent connections between a high-pressure hepatic artery and an adjacent low-pressure portal vein. They result from arterial injury from liver biopsy or biliary intervention[7] and may progress to hemodynamically significant fistulas requiring endovascular treatment.

AP shunts are usually subcapsular and do not exhibit a mass effect on adjacent vessels and bile ducts. They are usually not seen on unenhanced CT, as they are isoattenuating to the surrounding liver. At CT performed in the hepatic arterial phase (HAP), AP shunts typically appear as a peripherally located enhancing focus and are less commonly centrally located. On the portal venous phase (PVP), the shunt becomes isoattenuating when compared with the background liver.[8] In some cases, early enhancement of a dilated peripheral branch of the portal vein occurs during the HAP, before opacification of the main portal vein (Fig. 1).[9] On MRI, these lesions are

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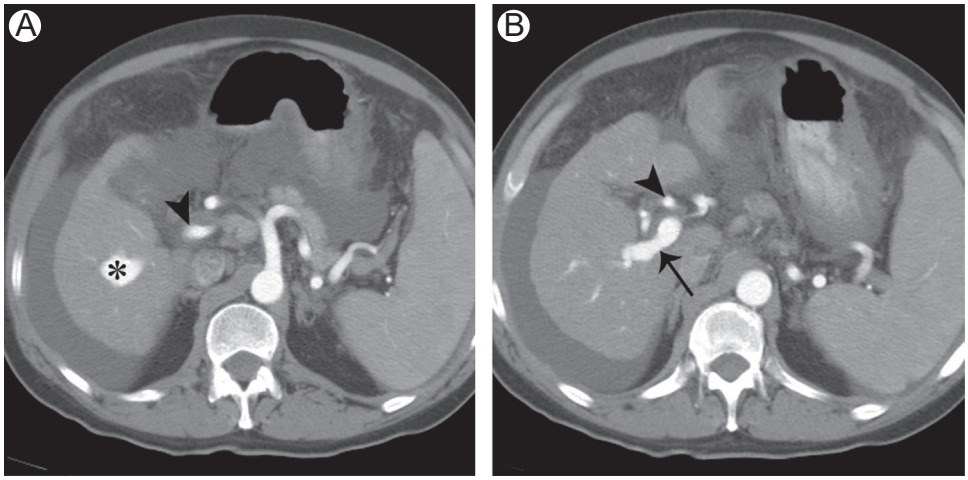

Figure 1 Spontaneous AP shunt in a patient with cirrhosis and ascites. (A and B) Axial HAP CECT at 2 noncontiguous levels reveals the right hepatic artery (arrowhead) supplying a well-defined oval vascular shunt (asterisk) with early enhancement of a dilated right portal vein (arrow).

occult on unenhanced T1- and T2-weighted sequences, and the enhancement pattern parallels that of the CT.[10,11] AP shunts are typically not seen on ultrasound (US). AP fistulas are indistinguishable from AP shunts on imaging, unless there is evidence of an iatrogenic intervention such a gas-filled biopsy tract.

In patients with cirrhosis, AP shunts less than 2 cm are referred to as pseudolesions and can be difficult to distinguish from hepatocellular carcinoma. Distinguishing features of hepatocellular carcinoma include mass effect, washout on the PVP, and hypointensity in the hepatobiliary phase of MR images following administration of liver-specific contrast agents. However, if the pseudolesion is small and round, the specificity of these imaging features to differentiate AP shunts from well-differentiated hepatocellular carcinoma is low.[11] The American Association for the study of Liver Diseases recommends imaging follow-up for small indeterminate hypervascular lesions for assessment of imaging features and growth.[12] In patients with cirrhosis, a 6-month follow-up CT or MRI is recommended for small, indeterminate enhancing liver nodules.[13]

Budd-Chiari Syndrome

Budd-Chiari syndrome is the obstruction of hepatic venous outflow at the hepatic veins or hepatic IVC level. The outflow obstruction leads to increased sinusoidal pressure and diminished portal venous flow. This results in centrilobular congestion followed by hepatocellular necrosis and atrophy. It affects women more than men and can occur at any age.

It is classified into primary and secondary types. Primary causes include congenital webs and diaphragms in addition to injury and infection. Most of the secondary causes are thrombotic, most commonly secondary to a hypercoagulable state from contraceptive use, pregnancy, paroxysmal nocturnal hematuria, polycythemia, protein S and protein C deficiency, antithrombin III deficiency, and factor V Leiden mutations. In these cases, the thrombus is usually in a major hepatic vein or hepatic IVC. Secondary causes include chemotherapy and radiation therapy, where the thrombus is in a central or in a sublobular vein. In addition, bone marrow transplantation can cause thrombosis of small centrilobular veins.[14,15] Primary Budd-Chiari is more common in Asia, India, and Africa, whereas secondary causes are more common in western countries. The clinical presentation depends on the acuteness of the disease. The acute form has rapid onset of jaundice and ascites, whereas the subacute or the chronic form is more gradual. The less-common fulminant form has rapid progression over 8 weeks from disease onset to hepatic encephalopathy.

During the acute stage, hepatocellular necrosis develops rapidly, as collateral vessels have not yet developed. At unenhanced CT, the liver morphology is maintained, but the liver is enlarged and diffusely hypodense secondary to edema. There is narrowing of the hepatic veins and IVC, which may appear hyperattenuating when a thrombus is present.[16] An image obtained at the HAP shows decreased peripheral enhancement owing to diminished perfusion caused by decreased portal perfusion from increased sinusoidal pressure. There is early enhancement of the caudate lobe and the central liver because of separate venous drainage. An image obtained at the PVP may demonstrate a "flip-flop" pattern with hypoattenuation of the central liver owing to washout and increased attenuation of the periphery of the liver due to contrast agent flow from capsular veins.

In the subacute or the chronic phase, the peripheral areas are deprived of flow from the portal system and cannot regenerate. However, because of accessory venous drainage and preservation of the portal venous supply to the caudate lobe, the central region of the liver may undergo hypertrophy (Fig. 2).[16,17] Portosystemic and intrahepatic collateral vessels are formed (Fig. 2). These pathways include intrahepatic venovenous comma-shaped collaterals, systemic collaterals in the subcutaneous tissues, and capsular vein collaterals. Ascites (Fig. 2) and splenomegaly are frequently seen. The chronic thrombus in the IVC can calcify, and portal vein thrombus may form

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