CT and MRI drive awareness of vascular liver disorders

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The liver receives blood from arterial (20%) and venous (80%) sources. This dual blood supply means that the organ is well-suited to contrast-enhanced cross-sectional imaging.

Imaging with state-of-the-art equipment achieves a clear-cut separation of the hepatic phases of liver enhancement. Perfusion abnormalities, however, can produce areas of abnormal liver enhancement. Vascular compromise may affect the volume and direction of flow in the dual blood supply. Radiologists need to remember that the arterial and portal systems are not independent and may communicate via intrahepatic anastomosis at the acinus level, using transplexal, transvasal, or even transtumoral routes.¹

The most important vascular disorders of the liver include transient hepatic attenuation difference, Budd-Chiari syndrome, passive hepatic congestion, peliosis hepatis, hepatic infarction, and hereditary hemorrhagic telangiectasia. An awareness of the imaging and pathophysiological features of each of these conditions is important when examining the liver.

**TRANSIENT DIFFERENCES**

Transient hepatic attenuation/intensity differences THADs can be seen during dynamic liver CT and MRI. This refers to an area or areas of increased attenuation/intensity on arterial phase images, unrelated to a tumor, which become isoattenuating/isointense on portal phase images. THADs are typically wedge-shaped with straight borders. They are not associated with a mass effect and normal vessels may be recognized within the triangle-shaped area. When observing such a finding, radiologists should carefully search for a lesion in the tip of the transient attenuation caused by arterioporal shunting secondary to portal vein obstruction.² The physiopathologic explanation rests on an increased compensatory arterial flow, mainly through the peribiliary plexus, due to the decreased portal flow (Figure 1). A similar phenomenon may be seen in other situations, such as hepatic parenchymal compression, where rib compression or a peritoneal implant locally diminishes portal flow because this is a low-pressure system.
Another cause might be related to local inflammatory processes (cholecystitis or abscess) that may affect the blood supply. Local hyperemia related to the inflammatory process itself increases arterial perfusion, while parenchymal compression exerted by the abscess contributes to reduce the amount of portal blood flow.\(^3\)

Transient hepatic attenuation differences can also be seen in the context of liver cirrhosis, since nodular regeneration and fibrosis reduce portal venous blood within the sinusoids causing functional arterioportal shunts to open between the two vascular systems. Since they can be difficult to recognize as such, confusion with hypervascular foci representing hepatocellular carcinoma (HCC) may happen.\(^4\)

Aberrant blood supply to the liver is another possible cause. Systemic, nonportal venous blood may enter the liver via vascular variants. Since the contrast-enhanced portal venous flow does not run into the systemic venous flow, the different concentration of contrast may result in a hyperattenuating foci on arterial phase images or be represented by a hypodense area on the portal venous phase of dynamic CT. This is most commonly seen in the subcapsular region, anterior to the porta hepatitis, adjacent to the gallbladder fossa and falciform ligament.\(^5\)

Steal phenomenon is another flowrelated occurrence that may be encountered with hypervascular liver tumors. The physiological explanation results from a siphoning effect that causes a disproportionate volume of the arterial opacified blood to be diverted to the liver tumor. This causes an area of transient hyperdensity around the lesion in the arterial phase that artificially increases tumor size. This finding has been reported in both benign and malignant liver tumors.\(^6\)

In some cases the underlying mechanism of THAD remains obscure. Sometimes, however, it is an indirect sign of disturbance of the blood flow at the sinusoidal level, as it may be in the case of intrahepatic portal microvessel involvement in metastatic disease.

**BUDD-CHIARI SYNDROME**

Budd-Chiari syndrome (BCS) is defined as lobar or segmental hepatic venous outflow obstruction at the level of the hepatic veins or inferior vena cava (IVC).\(^7\) Primary causes of venous outflow obstruction include congenital factors, such as webs and diaphragms, as well as injury and infection. Secondary causes are commonly thrombotic. These are usually linked to hypercoagulability due to oral contraceptive use, pregnancy, polycythemia, or protein C deficiency.

Clinical manifestations of BCS may be fulminant, acute/subacute, or chronic. Obstruction of hepatic venous outflow results in severe centrilobular congestion with increased sinusoidal pressure, leading to delayed or reversed portal venous inflow. Regional disturbances in portal venous flow are primarily responsible for the characteristic findings seen on contrast-enhanced CT and MRI. Unenhanced CT demonstrates global hepatic enlargement with diffuse hepatic hypoattenuation in acute manifestations of BCS. Hyperattenuating thrombi may be identified in the IVC or hepatic veins.
veins. Ascitis and splenomegaly are usually present too. Dynamic contrast-enhanced CT and MRI of acute phase BCS shows patchy hepatic parenchymal enhancement with poor visualization of the hepatic veins. Early enhancement of the caudate lobe and central portion of the liver around the IVC is sometimes observed in the arterial phase, with associated decreased peripheral liver enhancement caused by portal and sinusoidal stasis. A “flip-flop” pattern is seen in the portal venous phase. Washout causes low attenuation in the central part of the liver, while attenuation in the peripheral part of the liver gradually increases corresponding to the progressive accumulation of contrast material. Intravascular thrombi may be identified as hypoattenuating masses within the hepatic veins or IVC. Hepatic infarcts appear as unenhancing peripheral wedge-shaped areas and are occasionally present, especially in subacute/chronic BCS.

The caudate lobe is often enlarged, reflecting its autonomous venous drainage into the IVC by small individual branches. Venous collaterals develop both inside and outside the liver in an attempt to bypass the obstructed hepatic veins and include systemic, portosystemic, and intrahepatic shunts. Intrahepatic collaterals typically appear as comma-shaped, branching vascular structures and are highly suggestive of chronic BCS (Figure 2).

CONGESTION, INFARCTION

Impaired hepatic venous drainage secondary to cardiac disease can lead to the stasis of blood within liver parenchyma. Patients with passive hepatic congestion may have hepatomegaly, liver tenderness, or elevated liver function tests. Uncorrected venous congestion may hamper drainage of sinusoidal blood flow, resulting in parenchymal atrophy, necrosis, and, ultimately, fibrosis and the so-called cardiac cirrhosis. Early enhancement of a dilated IVC and of the central hepatic veins is seen in the arterial phase owing to reflux of contrast from the right atrium into the IVC (Figure 3). Parenchymal phase images show a heterogeneous, mottled, mosaic pattern of enhancement, with linear and curvilinear areas of poor enhancement due to the slow flow of contrast material inside small- and medium-sized hepatic...
Peripheral large patchy areas of poor delayed enhancement may be observed due to stagnant flow within the liver’s periphery. Perivascular lymphedema may also be seen as linear regions of low attenuation encircling the intrahepatic IVC or portal veins. These should not be confused with venous thrombosis. Ancillary findings include cardiomegaly, hepatomegaly, pleural effusions, and ascites. The liver may become small and cirrhotic if passive congestion is chronic.

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Hepatic infarction is defined as areas of coagulation necrosis resulting from hepatocyte cell death caused by local ischemia. The ischemia is commonly due to a thrombus or embolus that has obstructed circulation to the affected area. Infarction due to hepatic artery thrombosis most often occurs after liver transplantation. In general, however, hepatic infarction is uncommon, given that the liver has a dual blood supply from the hepatic artery and portal vein, as well as extensive collateral vessels.

Hepatic infarcts have varied appearances at CT. They are more conspicuous on contrast-enhanced images, where they manifest as perfusion defects. These defects are typically distributed in a geographic or segmental pattern. Three major configurations of lesion have been described: wedge-shaped, rounded, and irregularly shaped, running parallel to the bile ducts. Wedge-shaped lesions (Figure 4) are usually seen at a peripheral location. The rounded configuration may be either
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peripheral or central.\textsuperscript{15} Gas formation resulting from necrosis has been reported in both sterile and infected infarcts. Chronic changes include atrophy of the involved segment and formation of cystic bile collections secondary to bile duct necrosis.

**RARE FINDINGS**

Peliosis hepatis is a rare benign disorder that causes sinusoidal dilatation and multiple blood-filled spaces within the liver. Their size can vary from 1 mm to several centimeters in diameter.\textsuperscript{16} The disorder may be secondary to a wasting disease (for example, tuberculosis), cancer, AIDS, drug abuse, renal or cardiac transplantation, exposure to toxins (for example, polyvinyl chloride or arsenic), sprue, or necrotizing vasculitis. No associated condition can be identified, however, in 20% to 50% of patients. Peliosis hepatis less commonly results in hepatic failure, hemorrhagic necrosis, or spontaneous hepatic rupture.\textsuperscript{17}

Lesions are seen as areas of hypoattenuation on unenhanced CT, displaying a variable enhancement pattern on contrast-enhanced CT. Mass effect is not generally observed, aiding differentiation from other liver lesions. Enhancement is progressive over time and may be complete on delayed imaging, progressing from globular patches to a centripetal or centrifugal enhancement recognized on portal phase imaging.\textsuperscript{18}

Thrombosed cavities resemble nonenhancing nodules and may simulate metastases or abscesses. It may not be possible to depict smaller lesions (< 1 cm) on unenhanced or contrast-enhanced images.

Peliosis hepatis lesions are hyperintense on T2-weighted MRI. Signal intensity is variable on T1-weighted MRI, most likely reflecting the various stages of subacute hemorrhage.

Hereditary hemorrhagic telangiectasia, also known as Osler-Weber- Rendu syndrome, is a rare, autosomal dominant, multiorgan disorder. It is characterized by abnormal vascular remodeling in the form of fibrovascular dysplasia, with multiple telangiectasias accompanied by arteriovenous malformations.\textsuperscript{19} A high prevalence of hepatic involvement (74\%) has been reported.\textsuperscript{20}

Imaging findings reflect the predominant pattern of hepatic shunting in each patient as follows:\textsuperscript{21}

**FIGURE 5.** Contrast-enhanced CT of patient with hereditary hemorrhagic telangiectasia. A: Arterial phase CT depicts early opacification of enlarged hepatic veins (arrowheads) while IVC remains unenhanced, corresponding to presence of hepatic artery to hepatic vein shunts. B: Hepatic artery is markedly tortuous with enlarged caliber. Liver enhancement is diffusely heterogeneous due to presence of several small-sized, rounded vascular lesions corresponding to intrahepatic telangiectases.

**Hepatic artery to portal vein shunts:**
- early and sustained enhancement of the portal vein during the arterial phase;
- subcapsular, peripheral transient hepatic attenuation difference on arterial phase images;
- dilatation of the hepatic artery and its branches;
- dilatation of the portal vein (>13 mm), often with collateral vessels;
- splenomegaly (>13 cm); and
- biliary strictures, dilatations, and cysts.

**Hepatic artery to hepatic vein shunts (Figure 5):**
- early enhancement of one or more hepatic veins during the arterial phase;
• enlargement of early-filled hepatic vein(s);
• heterogeneous enhancement (mosaic pattern) during the arterial phase;
• hepatosplenomegaly;
• ascites; and
• biliary strictures, dilatations, and cysts.

**Portal vein to hepatic vein shunts:**
• dilated portal branches communicating with large-caliber hepatic or systemic veins on portal venous phase;
• intrahepatic telangiectasias (Figure 5) appear as rounded, strongly enhancing lesions (>10 mm) with a predominant peripheral distribution, more readily recognizable on reconstructed multiplanar and maximum intensity projection images; and
• large confluent vascular masses (>10 mm diameter) showing early filling during the arterial phase with persistent enhancement.

In conclusion, increasing use of dynamic cross-sectional imaging has allowed wider recognition of various pathologic conditions affecting hepatic blood vessels. Awareness of the imaging spectrum and knowledge of the mechanisms underlying vascular liver disorders prevents interpretative pitfalls, which increases diagnostic accuracy in this group of conditions, and helps reduce false-positive findings of neoplastic or inflammatory lesions.

**Disclosures:**

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