Hepatic Perfusion and Vascular Lesions

Terry S. Desser[1]

The liver has a dual afferent blood supply, with inflow provided by the portal vein and hepatic artery in a ratio of approximately 3:1. Localized alterations in the balance between arterial and venous supply are frequently seen as perfusion abnormalities at contrast-enhanced CT and MRI. Causes include various entities that increase hepatic sinusoidal pressure or reduce portal venous inflow, hypervascular tumors, and tumors or vascular malformations with arterioportal or arteriovenous shunting. Vascular neoplasms may be benign or malignant, and, although the latter are rare, radiologists should be familiar with their appearance to suggest the diagnosis.

The detection and characterization of focal liver lesions depends on administration of IV contrast material to augment intrinsic liver-to-lesion contrast and to differentiate lesions by the temporal evolution of their contrast enhancement patterns. An important caveat, however, is that macroscopic abnormalities in hepatic vascular structures or microscopic disturbances in vascular function may lead to focal regions of increased or decreased hepatic enhancement that do not necessarily represent mass lesions, or that represent vascular malformations rather than neoplasms. The purpose of this chapter is to assist the imager in recognizing enhancement patterns of vascular lesions and perfusion abnormalities of the liver, and in using them to make inferences about the status of the liver in the regions encompassed by them.

The Hepatic Circulation

The hepatic circulation is the most complex in the body by virtue of its dual blood supply, with 75% of afferent blood flow provided by the portal vein and the remaining 25% provided by the hepatic artery. Within the liver parenchyma, portal veins and hepatic arteries arborize together along the efferent bile ductules within the portal triad, which are situated at the vertices of the hexagonal hepatic lobules. Terminal arterioles and venules both supply the hepatic sinusoids, which run along radiating rows of hepatocytes and ultimately coalesce to form the efferent hepatic venules and then hepatic veins. Twigs arising from the hepatic arterioles also supply the vasa vasorum of the portal venules and branch to a vascular plexus that surrounds the bile ductules within the portal triad. The efferent venules of the latter—that is, the peribiliary plexus—drain into both the portal venules and the hepatic sinusoids [1]. These structural interconnections at the microscopic level thus provide potential routes of communication between the arterial and portal circulations, with implications as we will see later in this chapter.

Unlike other organs, regulation of liver blood flow seems not to involve extrinsic innervation or systemic vasoactive molecules, but instead involves reciprocity between the hepatic arterial and portal venous flows. The hepatic artery supplies the liver at a mean arterial pressure of 100 mm Hg, whereas normal pressure in the valveless portal venous system is 5–8 mm Hg. High-volume antegrade flow in the portal circulation depends on the difference between the splanchnic arteriolar pressure and the hepatic vascular resistance, which is normally quite low at the level of the portal venules and hepatic sinusoids [2]. In response to decreases in portal venous flow, flow in the hepatic artery increases by a mechanism known as the hepatic arterial buffer response [3]. The converse—namely, an increase in portal flow in response to decreased arterial flow—seems not to occur.

CT and MRI protocols for dedicated liver imaging usually involve multiple phases of image acquisition. During the arterial phase, the normal liver enhances relatively little, because the iodinated contrast agent delivered by the hepatic artery is diluted 3:1 by unopacified portal venous blood. In contrast, lesions with a robust arterial supply enhance significantly more than the normal liver and will therefore be visible as hyperdense lesions during arterial phase imaging. Subsequently, beginning approximately 10 seconds later, the IV contrast agent bolus completes its transit through the splanchnic organs (spleen and gut) and opacifies the portal inflow to the liver [4]. Given the duration of the contrast agent injection in most current protocols, the hepatic arterial inflow also contains contrast agent during the portal venous phase, and the normal liver parenchyma thus enhances maximally, whereas the hypervascular lesions may wash in, wash out, or disappear entirely.

Perfusion Abnormalities

Arterioportal Shunts

Normal hepatic perfusion consists of hepatopetal flow in the hepatic arterial and portal venous branches throughout the

[1]Department of Radiology, Stanford University School of Medicine, 300 Pasteur Dr, Mail Code 5621, Stanford, CA 94305. Address correspondence to T. S. Desser (desser@stanford.edu).

liver. Given the perfusion pressure difference between these two systems and the existence of microscopic sites of communication at the peribiliary plexus and hepatic sinusoids, mechanisms must exist in the normal liver to prevent blood from always shunting from the hepatic arterioles into the portal venules. A detailed discussion of these regulatory mechanisms is beyond the scope of this chapter, and many such mechanisms are still being elucidated, but it may be stated that they are complex [1–3]. Nevertheless, many structural and functional changes within the liver can develop that disturb this balance and cause blood to shunt from the hepatic arterial into the portal venous circulation, at either the macroscopic or microscopic level. Communication between the hepatic arterial system and portal venous system may arise within hypervascular tumors, which appropriate portal venous structures for drainage, within the cirrhotic liver, or in association with physiologic conditions resulting in elevated sinusoidal pressure [5, 6]. The result is flow of arterial blood at systolic pressures into the low-pressure portal venous system with localized reversal of portal venous flow. Supply of the sinusoids is then solely by the hepatic artery, with the portal venules repurposed as efferent channels.

On contrast-enhanced CT or MRI scans of the liver, the regions of the liver receiving either relatively increased (or exclusively) arterial inflow will appear as focal areas of higher attenuation during the arterial phase. This is due to both an increase in arterial perfusion occurring by the hepatic arterial buffer response and a lack of dilution of the iodinated contrast agent in the arterioles by unopacified blood in portal venules. During portal venous phase scans, iodinated contrast material is present in both the arterial system and the portal venous system. The perfusion of a region receiving only arterial inflow will then appear isodense compared with a region receiving both arterial and portal flow, because the total blood flow is equivalent in the two areas. This is the physiology of the transient hepatic attenuation difference (THAD) at CT and its MRI equivalent, the transient hepatic intensity difference (THID).

Branch portal venous thrombosis due to hypercoagulable states or septic pyelophlebitis decreases portal flow at the segmental or subsegmental level. Tiny peripheral thrombosed vessels are often visible as branching linear low densities. The decreased portal inflow causes arterioportal shunting via the plexus surrounding the bile ductules (transplexal route), resulting in THADs or THIDs [7]. In many instances, the perfusion abnormality is more conspicuous than the thrombosed vessel itself. Within the THAD or THID, normally arborizing vascular and biliary structures can often be seen, indicating an absence of mass effect.

In the cirrhotic liver, structural distortion of the microvascular anatomy by fibrosis results in occlusion of hepatic venules and retrograde filling of small portal branches via hepatic arterioles (i.e., arterioportal shunting) [7–9]. Peripheral arterioportal shunts in cirrhosis are a common cause of false-positive diagnoses of hepatocellular carcinoma (HCC) on contrast-enhanced CT or MRI scans of the liver [10]. They typically appear as wedge-shaped hypervascular lesions on the arterial phase that then become isodense or slightly hyperdense during the portal venous and delayed phases. No actual underlying mass lesion is present, and the lack of washout should permit differentiation from HCC.

Macroscopic arterioportal shunts (i.e., arterioportal fistulas) may occur as a result of trauma such as liver biopsy. A hypervascular nodule isodense with the aorta and early draining portal vein will be visible, as well as a THAD (Fig. 1).

Extraluminal processes may also decrease portal flow. Direct compression of portal venules by intrahepatic tumors or abscesses may result in arterioportal shunting and consequent hypervascular foci on imaging via the same transplexal route as intrinsic branch vessel intraluminal thrombi. In this instance, the resultant THAD may appear more rounded or irregular instead of wedge-shaped because of the presence of the obstructing mass. Arterioportal shunting may also occur if the

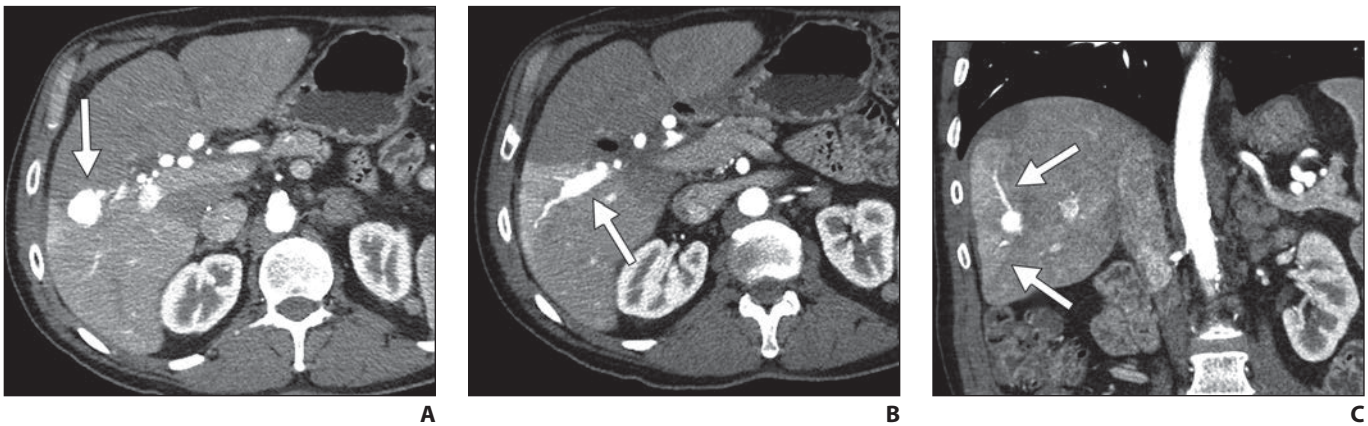


Fig. 1—59-year-old man with cirrhosis and large arterioportal fistula after liver biopsy.
A, CT image shows that round hypervascular nodule (*arrow*), isodense with aorta, is visible in liver periphery.
B, CT image shows that portal vein branch (*arrow*), also isodense with aorta and enhancing earlier than main portal vein, is evident.
C, Coronal CT image shows wedge-shaped transient hepatic attenuation difference (*arrows*), with nondisplaced vascular structures arborizing within.

tumor appropriates portal branches as draining veins.

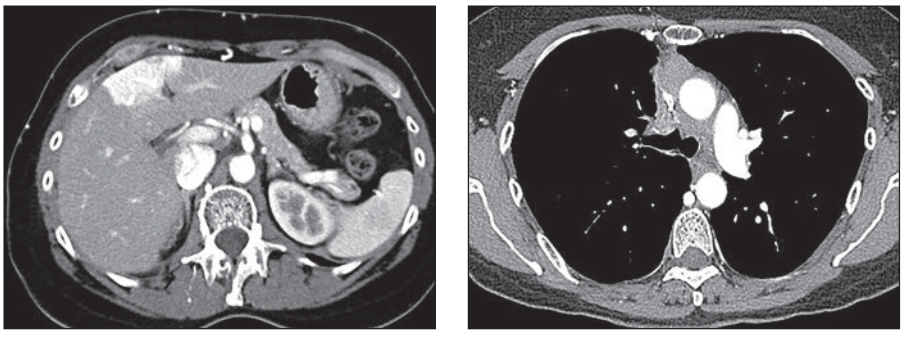
Indeed, localized increases in sinusoidal pressure from any cause will result in regionally diminished portal inflow, a reflex increase in hepatic arterial inflow, and a THAD seen at multiphasic CT or MRI [7, 8, 11]. Such entities include cholangitis or biliary obstruction [12], focal hepatic vein branch thrombosis, and extrinsic compression of the liver by the ribs, subcapsular hematomas, or masses [13].

Perfusion abnormalities caused by main portal vein thrombosis: zonal perfusion—The presence of a robust collateral venous network in the center of the liver results in a difference between the appearance of main portal vein thrombosis and peripheral portal vein thrombosis. When flow in the main portal vein is absent or reduced, collateral inflow pathways along the common bile duct (i.e., the parabiliary venous plexus) form gradually and supply the central (i.e., perihilar) region of the liver. By comparison, the periphery of the liver receives exclusively arterial perfusion [8, 14]. During the arterial phase of biphasic CT, there will be a crescentic region of increased enhancement in the periphery of the liver corresponding to the region of relatively increased arterial flow (particularly in the right lobe) and a large region of decreased enhancement in the perihilar central region of the liver. This lower attenuation central region, known as a central pseudotumor, should then enhance normally on portal and delayed phases and can thereby be differentiated from an infiltrating mass. Over time, the perfusion difference produced by main portal vein thrombosis results in atrophy of the peripheral subcapsular regions of the liver and hypertrophy of the central segments, producing a dysmorphic hepatic appearance that should not be mistaken for cirrhosis [15].

Perfusion abnormalities resulting from nonportal venous inflow—Aberrant or nonportal venous drainage into the liver will also produce a perfusion abnormality on contrast-enhanced CT or MRI scans if there is temporal separation between contrast agent inflow to the liver via this route and the main portal route. Obstruction of the superior or inferior vena cava causes collateral venous pathways to form, which run along the abdominal wall and into the liver along its embryologic mesenteries [7, 8, 16]. This paraumbilical venous system is familiar to radiologists as a common collateral pathway in portal hypertension [16–18]. The internal mammary and lateral thoracic veins communicate with the superficial epigastric and paraumbilical veins. These veins, in turn, connect to the left portal vein via the superior and inferior veins of Sappey, which drain into liver segments II, III, and IV adjacent to the falciform ligament. In superior vena cava obstruction, a THAD may be present in the superior aspect of segment IV of the liver as undiluted venous blood from the arm traverses the liver parenchyma en route to the intrahepatic inferior vena cava and then the heart (Fig. 2). The presence of densely opacified collateral vessels along the diaphragm should enable the radiologist to suggest the presence of superior vena cava obstruction even if only the abdomen is scanned. Similarly, a perfusion defect may be visible adjacent to the falciform ligament in inferior vena cava obstruction, when unopacified systemic venous drainage from the leg routed through the liver creates an area of decreased enhancement on portal venous phase scans. Low attenuation in this region, usually attributed to fatty infiltration, may in some cases represent a perfusion abnormality instead.

Nonportal inflow also occurs via the parabiliary venous system in the hepatic hilum along the common bile duct and through the pericholecystic veins draining the gallbladder. The veins of the gallbladder drain both directly into the adjacent liver parenchymal segments IV and V and into the veins of the venous system in the hepatic hilum [11, 16]. Cholecystitis produces gallbladder wall hyperemia and increased nonportal venous drainage into the adjacent liver segments, thereby producing a THAD [19, 20]. This same phenomenon of nonportal venous drainage into the liver segments around the gallbladder is thought to account for focal sparing of this region in diffuse fatty infiltration, because this region of the liver does not receive nutrient-rich venous drainage from the gut. Similarly, in the setting of diffuse fatty infiltration, perihilar segments may be spared in some patients by virtue of receiving blood flow via the peribiliary venous system running along the common bile duct, rather than superior mesenteric venous drainage.

Abnormal venous outflow—With the exception of small accessory short hepatic veins draining the caudate lobe, the three major hepatic veins are the sole route of egress for blood exiting the liver [21]. Functional or anatomic impairment of hepatic venous outflow may lead to perfusion abnormalities visible on contrast-enhanced MRI studies of the liver. Over the long term, structural abnormalities and mass le-



A B

Fig. 2—Patient with superior vena cava obstruction and transient hepatic attenuation difference produced by nonportal venous inflow.
A, Arterial phase CT image of liver shows area of increased enhancement in segment IV and portions of segment II. Note prominent subcutaneous collateral vessels.
B, CT image of chest shows anterior mediastinal mass representing malignant thymoma and slitlike superior vena cava. Note enlarged right internal mammary and dense azygous veins.

sions may also develop as a consequence of the chronically elevated sinusoidal pressures.

Functional Outflow Obstruction: Right Heart Failure

Venous congestion in the hepatic sinusoids may occur when the volume of blood returned to the right heart exceeds its pumping capacity. Over the long term, this leads to compression, atrophy, and necrosis of centrilobular hepatocytes, producing first fatty infiltration and, eventually, perisinusoidal fibrosis (i.e., cirrhosis). CT scans of patients with passive congestion and venous outflow impairment may show a reticulated enhancement pattern of the hepatic parenchyma that is sometimes referred to as nutmeg liver [22] (Fig. 3). In right heart failure, the hepatic veins and inferior vena cava are typically distended, and there may be reflux of contrast material into the hepatic veins if tricuspid regurgitation is present. Similar hepatic venous drainage impairment occurs in constrictive pericarditis.

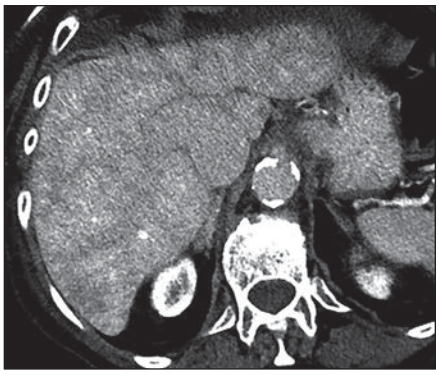


Fig. 3—77-year-old woman with right heart failure and passive congestion. Nutmeg liver pattern is seen on CT image. Note distended inferior vena cava and right hepatic vein.

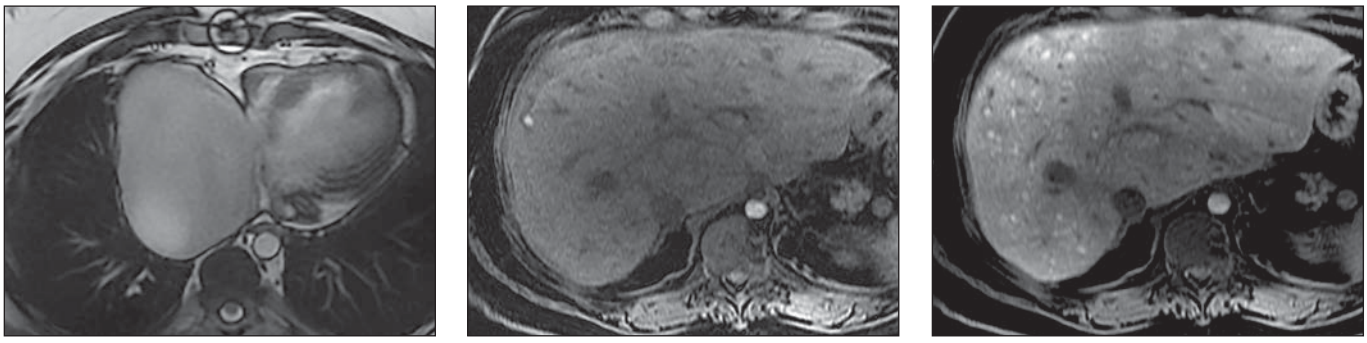
Hepatic congestion may also develop in patients who have undergone the Fontan procedure for congenital cardiac anomalies characterized by a single functioning ventricle (hypoplastic left or right heart and tricuspid atresia). In the Fontan circulation, either the right atrium or the vena cava is anastomosed to the pulmonary artery, bypassing the right heart. Although this procedure often permits patients to survive to adulthood without a heart transplant, transmission of the pulmonary vascular resistance directly into the systemic venous circulation (i.e., without an interposed ventricle to pump) causes vascular congestion in the liver and perisinusoidal atrophy and fibrosis. CT or MRI of these patients will show contrast enhancement patterns similar to those in patients with right heart failure. With chronic disease, the parenchymal damage produced by relentless hepatic vascular congestion leads to cirrhosis and formation of regenerative nodules. The latter typically appear as hypervascular nodules on arterial phase CT scans and retain contrast material on MRI scans performed with the hepatobiliary contrast agent gadoxetate disodium (Eovist, Bayer HealthCare), resembling focal nodular hyperplasia (Fig. 4).

Anatomic Venous Obstruction

The nutmeg liver enhancement pattern will also be present in the setting of anatomic obstruction of hepatic venous outflow. Commonly known as Budd-Chiari syndrome (BCS), the clinical symptoms consist of congestive hepatomegaly, abdominal pain (from hepatic capsular distention), ascites, and portal hypertension. Thrombosis is by far the leading cause of obstruction of the major hepatic veins and most often results from an underlying myeloproliferative disorder, hypercoagulable state, or other predisposing factor [23].

In addition to the mosaic heterogeneous perfusion pattern, contrast-enhanced CT or MRI of patients with BCS may show nonvisualization of the major hepatic veins [24]. Although the classic finding is preserved enhancement in the central pericaval portion of the liver due to intact venous drainage of the caudate lobe, in practice, the observed enhancement patterns will depend on the relative degree of obstruction of the various draining veins [25] and the specific collateral routes recruited, including lumbar, azygous, and hemiazygous veins.

As noted already, prolonged venous outflow obstruction leads to hepatic congestion with ensuing loss of hepatocytes. If elevated pressure continues, fibrosis will occur, with the development of cirrhosis and regenerative nodules. Large regenerative nodules, composed of benign hepatocytes and histological-



A **B** **C**

Fig. 4—24-year-old man who underwent Fontan procedure for tricuspid atresia during infancy who later presented with regenerative nodules.
A, MR image obtained at lung bases shows massively enlarged right atrium, diminutive right ventricle, patent ventricular septal defect, and enlarged left ventricle.
B, Early arterial phase MR image shows hypervascular liver lesion in periphery of right hepatic lobe.
C, Liver MR image obtained 20 minutes after injection of hepatobiliary contrast medium gadoxetate disodium (Eovist, Bayer HealthCare) shows multiple regenerative nodules that retain contrast medium.

ly similar to focal nodular hyperplasia, may be seen in 60–80% of patients with BCS and should not be mistaken for HCC [26]. On the other hand, chronic BCS with the development of cirrhosis does indeed place patients at risk for HCC, so care must be taken to distinguish these two entities in this patient population [27–29].

Venoocclusive Disease: Sinusoidal Obstruction Syndrome

Venous outflow may also occur at the level of the sinusoids as a result of damage to sinusoidal endothelial cells and subsequent fibrosis. Formerly termed "venoocclusive disease" [30], the term "sinusoidal obstruction syndrome" is now preferred by several authors because the pathophysiology is a toxic insult that causes microvascular obstruction produced by sinusoidal endothelial cell swelling, sloughing, and embolization distally. Although the clinical picture of sinusoidal obstruction syndrome—hepatomegaly, ascites, and jaundice—may be similar to those of BCS and macroscopic vascular obstruction, the causes and patient populations differ. In the United States, sinusoidal obstruction syndrome is almost always seen in association with conditioning regimens for bone marrow or stem cell transplantation. Elsewhere, it may be seen with ingestion of teas or foods containing pyrrolizidine alkaloids.

Primary Vascular Lesions

Vascular shunts are usually easily recognized by their synchronous enhancement with the blood pool and prominent feeding or draining vessels. As discussed already, macroscopic arterioportal fistula most commonly occurs after penetrating trauma to the liver, such as that resulting from liver biopsy (Fig. 1). Portosystemic shunts are more common than shunts from the hepatic arteries to systemic veins and may be congenital or acquired. Congenital portosystemic shunts are rare and are thought to occur by either persistence of connections among tributaries of the vitelline vein (the precursor of the portal veins, hepatic veins, and portions of the inferior vena cava) [31, 32] or rupture of a portal vein aneurysm into the hepatic vein [5]. In contrast, acquired shunts are very common because they occur as sequelae of cirrhosis. These shunts provide collateral pathways for venous drainage of the liver in the setting of portal hypertension. Although the most commonly identified portosystemic shunts are extrahepatic, large intrahepatic portosystemic collaterals may be identified in the subcapsular area of the liver or may be seen draining directly into the inferior vena cava. Hepatic venovenous shunts may be seen in BCS and other causes of venous outflow obstruction.

Hereditary Hemorrhagic Telangiectasia

Hereditary hemorrhagic telangiectasia (HHT) is an autosomal dominant disorder caused by mutations in genes encoding receptors in the transforming growth factor–β signaling pathway, leading to mucocutaneous and visceral fibrovascular dysplasia and arteriovenous malformations [33, 34]. Also known as Osler-Weber-Rendu syndrome, it affects multiple organ systems, including the skin, lungs, oral mucus membranes, liver, and urinary and gastrointestinal tracts [6].

Liver involvement in HHT is diffuse, rather than segmental, and the imaging appearance depends on the types of vascular malformations present. The smallest lesions are focal dilations of postcapillary venules. In larger telangiectasias, the dilated venules may connect to dilated arterioles without intervening capillaries. Larger vascular nodules and masses may also be seen, including macroscopic arteriovenous malformations and hemangiomas. The hepatic artery feeding the liver may be markedly enlarged (Fig. 5). Hyperperfusion of the liver may induce parenchymal hyperplasia, and thus lesions of focal nodular hyperplasia may be seen. An increased frequency of benign hepatic masses, including focal nodular hyperplasia, regenerative nodules, and cavernous hemangioma, has been reported in HHT [35, 36]. The intrahepatic arteriovenous and arterioportal shunting can lead to complications of high-output cardiac failure and portal hypertension, respectively. Most commonly, the presenting symptom of HHT is epistaxis, and liver involvement is asymptomatic, but there is a female predominance among symptomatic patients [33].

Peliosis Hepatis

Peliosis hepatis is a rare vascular abnormality with a nonspecific imaging appearance found in association with a variety of conditions, including steroid use, immune deficiency, and wasting diseases such as tuberculosis and malignancy. Histologically, it is characterized by irregularly shaped blood-filled cavities, ranging in size from 1 mm to several centimeters, interspersed within the normal parenchyma [37]. At unenhanced CT, peliotic lesions are typically low attenuation, with occasional high attenuation representing hemorrhage. Calcification may be present. A variety of enhancement patterns may be present, including globular areas of blood-pool attenuation, sometimes including both a continuous peripheral ring of enhancement and central globular enhancement to form a target sign [37]. Alternatively, lesions may appear hypoenhancing compared with the adjacent parenchyma because of the presence of stagnant or clotted blood [38]. Late enhancement may be present, facilitating differentiation from other vascular lesions [37]. Signal characteristics at MRI are variable depending on the presence or absence of internal hemorrhage [37, 38]. Given the

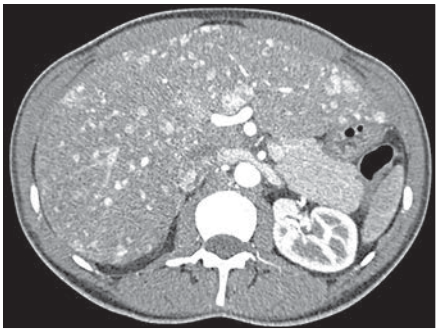


Fig. 5—43-year-old woman who presented with epistaxis, representing hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease). Arterial phase CT image shows innumerable hypervascular nodules and enlarged hepatic artery.

nonspecific appearance of peliosis hepatis, the clinical context will be required to suggest the diagnosis.

Vascular Neoplasms

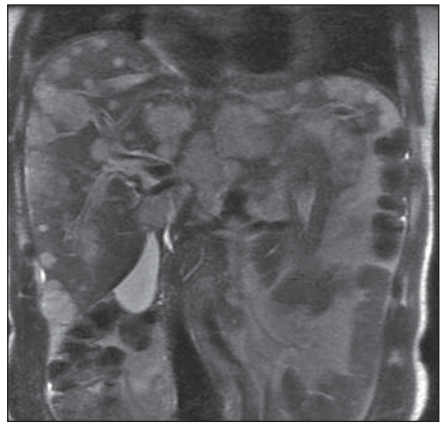
Cavernous Hemangioma

Hemangiomas are very common benign vascular neoplasms thought to arise from disordered angiogenesis [39]. Histologically, they consist of blood-filled spaces lined by a single layer of epithelial cells. The imaging features of typical and atypical cavernous hemangiomas have been well described elsewhere and will not be reiterated here [40, 41].

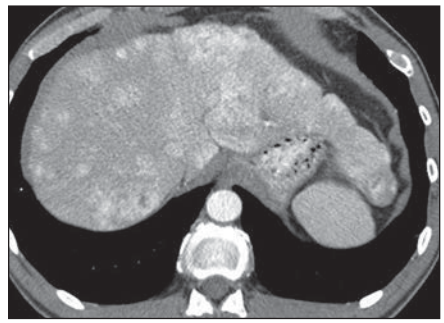
Epithelioid Hemangioendothelioma

Epithelioid hemangioendothelioma is a rare low-to-intermediate grade malignancy arising from endothelial cells of the liver. Most cases occur in women, and the cause is unknown. The clinical course is variable, with as many as 10–20% of patients being asymptomatic, so that the tumors may be discovered incidentally when scanning is performed for other indications. The lesion may be solitary or multifocal and has an infiltrative pattern of growth [42]. The multifocal nodules may coalesce and have a predilection for the subcapsular area of the liver. When associated with fibrosis, the peripherally located tumors may cause capsular retraction [43].

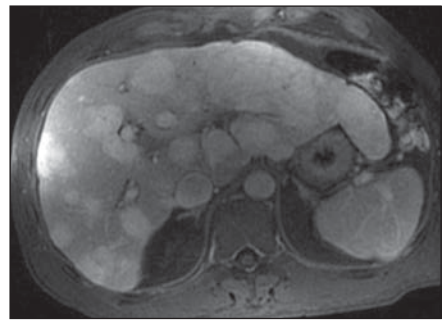
The CT or MR imaging appearance is variable, but the appearance of subcapsular confluent masses with capsular retraction should suggest the diagnosis. Before contrast agent administration, the tumor is hypodense; after contrast agent administration, a targetlike appearance has been described, with a low-attenuation outer ring representing avascular tissue, a ring interior to that representing the peripheral rim of the tumor, and a lower density center that may or may not enhance on delayed imaging. When the tumor is adjacent to a portal or hepatic vein, a lollipop sign may be produced [43]. Nevertheless, despite some of these vivid descriptors, no single feature is pathognomonic, and biopsy is usually necessary to exclude other entities, such as cholangiocarcinoma, metastatic disease, or atypical hemangioma.



A



B



C

Fig. 6—Patient with hepatic angiosarcoma. (Courtesy of Menias CO, Mayo Clinic, Scottsdale, AZ)
A, Coronal HASTE MR image shows innumerable intermediate-to-high signal intensity masses throughout liver in both peripheral and central locations.
B, Axial arterial phase CT image shows innumerable liver masses that appear hypervascular, but not as dense as aorta.
C, Axial MR image from delayed contrast-enhanced T1-weighted gradient-echo sequence shows that many masses retain contrast medium.

Angiosarcoma

At the opposite extreme from hemangiomas, hepatic angiosarcomas are very rare and highly aggressive vascular tumors. These malignancies constitute only 2% of primary hepatic neoplasms and 2% of soft-tissue sarcomas, although they are the most common mesenchymal hepatic malignancy [39, 44]. On pathologic examination, angiosarcomas are most often multicentric and composed of hemorrhagic masses of various size. Depending on the degree of differentiation, tumors may contain irregular vascular channels lined by abnormal endothelial cells (well differentiated tumor) or sheets of solid tumor cells resembling lymphoma or carcinoma (poorly differentiated tumor). Accordingly, the imaging appearance is variable. Enhancement is most commonly heterogeneous on arterial phase imaging, with lesions containing oddly shaped foci of enhancement that may be somewhat less dense than the aorta. Lesions may enhance centrally or peripherally, but when peripheral enhancement is present, it is typically continuous rim enhancement, rather than the globular discontinuous puddling characteristic of cavernous hemangioma. On later phases, progressive centripedal enhancement may be present. Lesions are typically multifocal, may be hyper- or hypoenhancing compared with adjacent parenchyma, and may be central or peripheral in location [39, 45] (Fig. 6). Different enhancement patterns may be present in different regions of the tumor because of its variable growth patterns, which include nodular, infiltrative, and periportal [46].

Conclusion

The complexity of hepatic vascular anatomy and physiology complicates interpretation of contrast-enhanced CT and MR images of the liver. Areas of increased or decreased enhancement may or may not represent mass lesions. However, by observing the temporal evolution of enhancement patterns and the presence or absence of anatomic abnormalities in the adjacent liver, such as mass effect, displacement of vascular structures, and biliary dilatation, the true nature of attenuation or intensity differences can usually be ascertained.

REFERENCES

1. Eipel C, Abshagen K, Vollmar B. Regulation of hepatic blood flow: the hepatic arterial buffer response revisited. *World J Gastroenterol* 2010; 16:6046–6057
2. Rocha FG. Liver blood flow: physiology, measurement, and clinical relevance. In: Jarnagin WR, Blumgart LH, eds. *Blumgart's surgery of the liver, biliary tract, and pancreas*. Philadelphia, PA: Elsevier Saunders, 2012:74–86
3. Lautt WW. The 1995 Ciba-Geigy Award Lecture: intrinsic regulation of hepatic blood flow. *Can J Physiol Pharmacol* 1996; 74:223–233
4. Young SW, Turner RJ, Castellino RA. A strategy for the contrast enhancement of malignant tumors using dynamic computed tomography and intravascular pharmacokinetics. *Radiology* 1980; 137:137–147
5. Lane MJ, Jeffrey RB Jr, Katz DS. Spontaneous intrahepatic vascular shunts. *AJR* 2000; 174:125–131
6. Torabi M, Hosseinzadeh K, Federle MP. CT of nonneoplastic hepatic vascular and perfusion disorders. *RadioGraphics* 2008; 28:1967–1982
7. Itai Y, Matsui O. Blood flow and liver imaging. *Radiology* 1997; 202:306–314
8. Itai Y. Hemodynamics in and around the liver. In: Okuda K, Mitchell DG, Itai Y, Ariyama J, eds. *Hepatobiliary diseases: pathophysiology and imaging*. Oxford, UK: Blackwell Science, 2001:228–246
9. Yu JS, Kim KW, Sung KB, Lee JT, Yoo HS. Small arterial-portal venous shunts: a cause of pseudolesions at hepatic imaging. *Radiology* 1997; 203:737–742
10. Kim TK, Choi BI, Han JK, Chung JW, Park JH, Han MC. Nontumorous arterioportal shunt mimicking hypervascular tumor in cirrhotic liver: two-phase spiral CT findings. *Radiology* 1998; 208:597–603
11. Breen DJ, Rutherford EE, Stedman B, Lee-Elliott C, Hacking CN. Intrahepatic arterioportal shunting and anomalous venous drainage: understanding the CT features in the liver. *Eur Radiol* 2004; 14:2249–2260
12. Pradella S, Centi N, La Villa G, Mazza E, Colagrande S. Transient hepatic attenuation difference (THAD) in biliary duct disease. *Abdom Imaging* 2009; 34:626–633
13. Desser TS. Understanding transient hepatic attenuation differences. *Semin Ultrasound CT MR* 2009; 30:408–417
14. Mathieu D, Vasile N, Dibie C, Grenier P. Portal cavernoma: dynamic CT features and transient differences in hepatic attenuation. *Radiology* 1985; 154:743–748
15. Tublin ME, Towbin AJ, Federle MP, Nalesnik MA. Altered liver morphology after portal vein thrombosis: not always cirrhosis. *Dig Dis Sci* 2008; 53:2784–2788
16. Yoshimitsu K, Honda H, Kuroiwa T, et al. Unusual hemodynamics and pseudolesions of the noncirrhotic liver at CT. *RadioGraphics* 2001; 21:S81–S96
17. Baba Y, Miyazono N, Inoue H, et al. Altered flow dynamics of intravascular contrast material to the liver in superior vena cava syndrome: CT findings. *Abdom Imaging* 2000; 25:146–150
18. Bashist B, Parisi A, Frager DH, Suster B. Abdominal CT findings when the superior vena cava, brachiocephalic vein, or subclavian vein is obstructed. *AJR* 1996; 167:1457–1463
19. Arai K, Kawai K, Kohda W, Tatsu H, Matsui O, Nakahama T. Dynamic CT of acute cholangitis: early inhomogeneous enhancement of the liver. *AJR* 2003; 181:115–118
20. Choi SH, Lee JM, Lee KH, et al. Relationship between various patterns of transient increased hepatic attenuation on CT and portal vein thrombosis related to acute cholecystitis. *AJR* 2004; 183:437–442
21. Sato TJ, Hirai I, Murakami G, Kanamura T, Hata F, Hirata K. An anatomical study of short hepatic veins, with special reference to delineation of the caudate lobe for hanging maneuver of the liver without the usual mobilization. *J Hepatobiliary Pancreat Surg* 2002; 9:55–60
22. Gore RM, Mathieu DG, White EM, Ghahremani GG, Panella JS, Rochester D. Passive hepatic congestion: cross-sectional imaging features. *AJR* 1994; 162:71–75
23. Valla DC. Hepatic vein thrombosis (Budd-Chiari syndrome). *Semin Liver Dis* 2002; 22:5–14
24. Gryspeerdt S, Van Hoe L, Marchal G, Baert A. Evaluation of hepatic perfusion disorders with double-phase spiral CT. *RadioGraphics* 1997; 17:337–348
25. Ueda K, Matsui O, Kadoya M, et al. CTAP in Budd-Chiari syndrome: evaluation of intrahepatic portal flow. *Abdom Imaging* 1998; 23:304–308
26. Brancatelli G, Federle MP, Grazioli L, Golfieri R, Lencioni R. Benign regenerative nodules in Budd-Chiari syndrome and other vascular disorders of the liver: radiologic-pathologic and clinical correlation. *RadioGraphics* 2002; 22:847–862
27. Flor N, Zuin M, Brovelli F, et al. Regenerative nodules in patients with chronic Budd-Chiari syndrome: a longitudinal study using multiphase contrast-enhanced multidetector CT. *Eur J Radiol* 2010; 73:588–593
28. Kobayashi S, Matsui O, Gabata T, et al. Radiological and histopathological manifestations of hepatocellular nodular lesions concomitant with various congenital and acquired hepatic hemodynamic abnormalities. *Jpn J Radiol* 2009; 27:53–68
29. Vilgrain V, Lewin M, Vons C, et al. Hepatic nodules in Budd-Chiari syndrome: imaging features. *Radiology* 1999; 210:443–450
30. DeLeve LD, Shulman HM, McDonald GB. Toxic injury to hepatic sinusoids: sinusoidal obstruction syndrome (veno-occlusive disease). *Semin Liver Dis* 2002; 22:27–42
31. Bhargava P, Vaidya S, Kolokythas O, Katz DS, Dighe M. Hepatic vascular shunts: embryology and imaging appearances. *Br J Radiol* 2011; 84:1142–1152
32. 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
33. Caselitz M, Wagner S, Manns MP. Liver involvement in Osler-Weber-Rendu Disease (hereditary hemorrhagic telangiectasia). In: Boyer TD, Manns MP, Sanyal AJ, eds. *Zakim and Boyer's Hepatology*, 6th ed., chapter 46. St. Louis, MO: Saunders, 2012:822–833
34. Guttmacher AE, Marchuk DA, White RI Jr. Hereditary hemorrhagic telangiectasia. *N Engl J Med* 1995; 333:918–924
35. Buscarini E, Danesino C, Plauchu H, et al. High prevalence of hepatic focal nodular hyperplasia in subjects with hereditary hemorrhagic telangiectasia. *Ultrasound Med Biol* 2004; 30:1089–1097
36. Milot L, Kamaoui I, Gautier G, Pilleul F. Hereditary-hemorrhagic telangiectasia: one-step magnetic resonance examination in evaluation of liver involvement. *Gastroenterol Clin Biol* 2008; 32:677–685
37. Iannaccone R, Federle MP, Brancatelli G, et al. Peliosis hepatis: spectrum of imaging findings. *AJR* 2006; 187:[web]W43–W52
38. Kim SH, Lee JM, Kim WH, Han JK, Lee JY, Choi BI. Focal peliosis hepatis as a mimicker of hepatic tumors: radiological-pathological correlation. *J Comput Assist Tomogr* 2007; 31:79–85
39. Bioulac-Sage P, Laumonier H, Laurent C, Blanc JF, Balabaud C. Benign and malignant vascular tumors of the liver in adults. *Semin Liver Dis* 2008; 28:302–314
40. Yamashita Y, Ogata I, Urata J, Takahashi M. Cavernous hemangioma of the liver: pathologic correlation with dynamic CT findings. *Radiology* 1997; 203:121–125
41. Vilgrain V, Boulos L, Vullierme M-P, Denys A, Terris B, Menu Y. Imaging of atypical hemangiomas of the liver with pathologic correlation. *RadioGraphics* 2000; 20:379–397
42. Goodman ZD, Terracciano LM, Wee A. Tumours and tumour-like lesions of the liver. In: Portmann BC, Ferrell LD, eds. *MacSween's Pathology of the Liver*, 6th ed., chapter 14. Edinburgh, UK: Churchill Livingstone, 2012:761–851
43. Azzam RI, Alshak NS, Pham HP. AIRP best cases in radiologic-pathologic correlation: hepatic epithelioid hemangioendothelioma. *RadioGraphics* 2012; 32:789–794
44. Kim HR, Rha SY, Cheon SH, Roh JK, Park YN, Yoo NC. Clinical features and treatment outcomes of advanced stage primary hepatic angiosarcoma. *Ann Oncol* 2009; 20:780–787
45. Peterson MS, Baron RL, Rankin SC. Hepatic angiosarcoma: findings on multiphasic contrast-enhanced helical CT do not mimic hepatic hemangioma. *AJR* 2000; 175:165–170
46. Koyama T, Fletcher JG, Johnson CD, Kuo MS, Notohara K, Burgart LJ. Primary hepatic angiosarcoma: findings at CT and MR imaging. *Radiology* 2002; 222:667–673