CT of Nonneoplastic Hepatic Vascular and Perfusion Disorders

Maha Torabi, MD • Keyanoosh Hosseinzadeh, MD • Michael P. Federle, MD

The unique dual blood supply of the liver (75% portal venous, 25% hepatic arterial) makes multiphase helical computed tomography (CT) a highly suitable technique for hepatic evaluation with imaging in two (arterial and portal venous) or more phases. Multiphase helical CT has become an important tool in the detection and characterization of hepatic tumors. In some situations, hemodynamic changes might mimic neoplastic or inflammatory lesions and evoke diagnostic uncertainty. To confidently identify hepatic conditions such as venous outflow obstruction (Budd-Chiari syndrome), arteriportal shunts, hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome), peliosis hepatis, passive congestion, and hepatic infarction, radiologists must be familiar with the disease-specific CT appearances and related clinical manifestations.

Introduction

The liver has a unique dual blood supply, with a compensatory relationship existing between the two sources so that the arterial flow increases when the portal venous flow decreases. Multiphase helical computed tomography (CT) allows evaluation of the liver during both the arterial and the portal venous phases of contrast enhancement and therefore has become an important modality for the detection and characterization of hepatic neoplasms. After the advent of rapid bolus-infusion multidetector CT technology, the rate of detection of nonneoplastic hypervascular lesions, intrahepatic vascular shunts, and perfusion abnormalities increased. When vascular compromise occurs, the dual blood supply system may cause changes in both the volume and the direction of blood flow. It is important to realize that the arterial and portal venous supplies to the liver are not independent; communications exist between the two systems. The article surveys the CT features and related clinical manifestations of various vascular disorders, including Budd-Chiari syndrome, arteriportal shunt, hereditary hemorrhagic telangiectasia, passive congestion, peliosis hepatis, and hepatic infarction. Familiarity with the characteristic helical CT appearance and pathophysiologic mechanisms of these disorders may result in more accurate diagnosis.

Abbreviation: IVC = inferior vena cava

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1From the Department of Radiology, University of Pittsburgh Medical Center (Presbyterian Campus), 200 Lothrop St, CHP MT Suite 3850, Pittsburgh, PA 15213. Recipient of a Certificate of Merit award for an education exhibit at the 2007 RSNA Annual Meeting. Received March 25, 2008; revision requested April 23; final revision received June 6; accepted June 13. All authors have no financial relationships to disclose. Address correspondence to K.H. (e-mail: hosseinzadeh@upmc.edu).
accurate diagnoses, helping reduce false-positive findings of neoplastic or inflammatory lesions. Furthermore, the consideration of relevant clinical manifestations that may be correlated with disease-specific CT features may help radiologists identify many of these disorders with greater confidence.

**Budd-Chiari Syndrome**

Budd-Chiari syndrome is defined as lobar or segmental hepatic venous outflow obstruction at the level of the hepatic veins or inferior vena cava (IVC) and may be primary or secondary (1,2). Hepatic venous outflow obstruction leads to elevation of sinusoidal pressure and diminished portal venous flow, which culminate in centrilobular congestion followed by necrosis and atrophy. The acuteness of obstruction will determine the degree of hepatocellular necrosis. In the acute form, collateral veins have not formed, and hepatocellular necrosis develops rapidly. In the subacute or chronic form, hepatic venous outflow is not obstructed completely; either venous thrombosis is incomplete, or various accessory hepatic veins drain the central region of the liver into the IVC. In addition, obstruction of hepatic venules leads to shunting of blood from hepatic arteries to portal veins, especially at the periphery of the liver, which helps to reduce hepatocellular congestion but does not prevent peripheral atrophy. 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 1) (1–4).

Primary causes of venous outflow obstruction include congenital causes such as webs and diaphragms, injury, and infection. Secondary causes most commonly are thrombotic (5). Thrombotic causes include obstruction of central and sublobular veins after chemotherapy and radiation, obstruction of small centrilobular veins in venoocclusive diseases after bone marrow transplantation and antineoplastic drug therapy, and, most commonly, obstruction of the major hepatic veins in patients with hypercoagulability due to oral contraceptive use, pregnancy, polycythemia, or protein C deficiency. Nonthrombotic venous obstruction may result from a hepatic or extrahepatic mass.

The precise incidence and prevalence of Budd-Chiari syndrome are unknown. Budd-Chiari syndrome may manifest at any age and is more common in women (6). Cases with a congenital or membranous cause are more common in Japan, India, Israel, and South Africa; occurrences secondary to thrombosis and nonthrombotic causes are by far the most common in Western countries.

The clinical manifestations of Budd-Chiari syndrome may be fulminant, acute, and subacute or chronic (2,3). Fulminant manifestation, which is rare, involves the rapid development of hepatic encephalopathy within 8 weeks after the onset of jaundice. The acute form manifests with rapid onset of jaundice and ascites. The subacute or chronic form is more gradual and may be complicated by portal hypertension. Treatment is predicated on the underlying cause and severity of disease: For example, anticoagulant drug therapy is administered to treat a bland thrombus; for membranous occlusion, membranectomy or percutaneous stent placement with balloon angioplasty might be performed; or, in severe cases of portal hypertension, a surgical shunt or transjugular intrahepatic portosystemic shunt might be created in preparation for liver transplantation (2).

**CT Findings**

The imaging findings of Budd-Chiari syndrome are variable and depend on the disease stage (ie, acute or chronic). Acute Budd-Chiari disease at unenhanced CT is depicted as a diffusely hypodense, enlarged but morphologically normal liver, with narrowing of the IVC and hepatic veins (3). The IVC and hepatic veins may appear hyper-
attenuating on unenhanced CT images because of the increased attenuation of a thrombus. Ascites and splenomegaly also are usually present. In the chronic phase of Budd-Chiari syndrome, the liver is dysmorphic, with caudate lobe hypertrophy (ratio of caudate lobe width to right lobe width, ≥ 0.55:1) and atrophy of the peripheral segments (3). The IVC and hepatic veins often are not visible because of collapse or a diminished flow rate. At contrast-enhanced CT of acute phase disease, early enhancement of the caudate lobe and central portion of the liver around the IVC is observed in the arterial phase, with associated decreased peripheral liver enhancement caused by portal and sinusoidal stasis (Fig 2) (4,6,7). Diffuse peripheral hypoattenuation is a sign of a

Figure 2. Acute Budd-Chiari syndrome in a 70-year-old woman with hypercoagulability due to a factor V Leiden genetic mutation associated with an increased risk of venous thrombosis. (a) Axial contrast-enhanced CT image obtained during the portal venous phase demonstrates ascites and a heterogeneous diminished perfusion of the peripheral hepatic segments. The thrombosed hepatic veins (arrows) are distinguishable from the dilated bile ducts by the venous convergence to the narrowed IVC (arrowhead). (b) Axial contrast-enhanced CT image, obtained during the same phase as a, shows the thrombosed hepatic veins (arrows) and narrowed IVC (arrowhead) at a lower level. Perfusion of the caudate lobe is normal. (c, d) Axial contrast-enhanced CT images (at progressively lower levels than a and b) show hypertrophy as well as normal perfusion of the caudate lobe (⁎). In c, the thrombosed hepatic veins also are visible (arrows).
out a hypoattenuating rim, in the arterial phase (6) and remain slightly hyperattenuating in the portal venous phase (Fig 3) (8,9). They typically have a maximal diameter of 1–4 cm. By contrast, hepatocellular carcinoma is usually hypoattenuat-
oids, or peribiliary venules results in redistribution of arterial flow into a focal region of portal venous flow (Fig 4) (10). Arterioportal shunts may be posttraumatic, occurring after blunt or penetrating injury, biopsy, or instrumentation (eg, placement of a transhepatic biliary drainage catheter) (11,12). Spontaneous small arterioportal shunts may occur and subsequently resolve in the cirrhotic liver (13,14); a histopathologic explanation for this hemodynamic resolution has not been established. Such shunts also may occur in association with hypervascular tumors such as hepatocellular carcinoma (15,16) and small hepatic hemangiomas (17) and with the spontaneous rupture of a hepatic artery aneurysm into a portal vein.

The prevalence of arterioportal shunts in patients with large hepatocellular carcinomas is as high as 63% (15). In patients with cirrhosis, the majority of small arterioportal shunts are pseudolesions; that is, they involve no pathologic alteration (14).

**Arterioportal Shunts**
Communication between a hepatic arterial branch and the portal vein at the level of the trunk, sinusoids, or peribiliary venules results in redistribution of arterial flow into a focal region of portal venous flow (Fig 4) (10). Arterioportal shunts may be posttraumatic, occurring after blunt or penetrating injury, biopsy, or instrumentation (eg, placement of a transhepatic biliary drainage catheter) (11,12). Spontaneous small arterioportal shunts may occur and subsequently resolve in the cirrhotic liver (13,14); a histopathologic explanation for this hemodynamic resolution has not been established. Such shunts also may occur in association with hypervascular tumors such as hepatocellular carcinoma (15,16) and small hepatic hemangiomas (17) and with the spontaneous rupture of a hepatic artery aneurysm into a portal vein.

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**CT Findings**
Contrast-enhanced arterial phase CT usually shows small, peripheral, nonspherical, enhancing foci, which become isodense to the liver and vasculature in the portal venous phase (Figs 5, 6) (11). Early enhancement of the peripheral portal vein occurs during the hepatic arterial phase and before the opacification of the main portal
threatening portal hypertension or high-output cardiac failure (11). If radiologic imaging-guided intervention such as embolization fails, surgical intervention might be necessary.

**Pearls**

It may be difficult to distinguish an arterioportal shunt from a small hepatocellular carcinoma. In such situations, repeat imaging in 6 months usually demonstrates the resolution or stability of an arterioportal shunt, as opposed to growth for a hepatocellular carcinoma. Moreover, on portal venous and delayed phase images, a hepatocellular carcinoma usually becomes hypoattenuated to liver and vessels, but an arterioportal shunt has the same attenuation as the vessels.

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**Figure 6.** Iatrogenic arterioportal shunt in a 53-year-old man with cirrhosis who underwent a US-guided liver biopsy with a subxiphoid approach. (a) Axial contrast-enhanced CT image obtained in the arterial phase shows a nodular lesion (arrow) that is isoattenuating to the aorta in the lateral segment of the left hepatic lobe. (b) Axial contrast-enhanced CT image obtained in the arterial phase at a lower level than a shows early enhancement of the portal venous branch that drains this hepatic segment (arrow). Note the subtle peripheral region of hyperperfusion. (c) Axial contrast-enhanced CT image obtained in the portal venous phase shows neither the arterioportal shunt nor the perfusion abnormality seen in b.

Peripheral arterioportal shunts involve small peripheral branches of the portal vein and may be associated with a small peripheral area of transient high attenuation due to the passage of contrast material from a high-pressure arterial branch into a low-pressure portal vein branch, producing a wedge-shaped subsegmental area of enhancement before the adjacent hepatic parenchyma enhances. The wedge-shaped area of enhancement is transient, and normal parenchymal attenuation returns in the portal venous phase (18,19).

Infrequently, hepatic arterioportal fistulas occur after trauma or interventional procedures and may result in the rapid development of life-
cholangitis, and liver failure (23). Extrahepatic involvement may manifest with hemoptysis, hemothorax, cerebrovascular accident, or cerebral abscess. Most patients experience repeated episodes of epistaxis from nasal mucosal telangiectasias, which bleed spontaneously. The prognosis is usually good with supportive treatment with iron supplements and blood transfusion. Patients rarely require hepatic arterial coil embolization, surgical ligation of the hepatic artery, or, more rarely, liver transplantation.

**CT Findings**

Contrast-enhanced CT demonstrates prominent extrahepatic and intrahepatic arterial branches with dilated hepatic and portal veins with vascular shunts. The arterial phase is characterized by dilated and tortuous intrahepatic and extrahepatic arterial branches (mesenteric vessels); early filling of portal or hepatic venous trunks, indicating arterioporal or arteriovenous shunting; and possible focal bile duct dilatation due to external compression by vascular masses (Figs 8, 9) (23–25). In the arterial phase, liver enhancement is heterogeneous, with a mosaic pattern of perfusion characterized by multiple areas of transient hepatic attenuation difference indicative of arterioportal shunting (65% of cases), telangiectasias (63% of cases), and confluent vascular masses (25% of cases) (Fig 8) (23). Telangiectasias appear as small peripheral subcentimeter perfusion abnormalities in the arterial phase that are more readily recognizable on reconstructed multiplanar and maximum intensity projection images (23), which allow increased conspicuity of small hypervascular lesions adjacent to vessels. Confluent vascular masses appear as larger vascular pools with early and persistent enhancement during the arterial phase. Chest images may show cardiomegaly and prominent central pulmonary arteries; pulmonary arteriovenous malformations also may be detected.

**Pearls**

A detailed clinical history that includes specific questions regarding episodes of epistaxis or a family history of hereditary hemorrhagic telangiectasia may help achieve an accurate diagnosis.
Figure 8. Hereditary hemorrhagic telangiectasia in a 35-year-old woman. (a) Axial contrast-enhanced CT image obtained in the late arterial phase shows early opacification of the markedly dilated IVC (arrow) and hepatic veins because of an intraparenchymal arteriovenous shunt. The liver parenchyma appears heterogeneously enhanced, with multiple small telangiectasias (filled arrowheads) and vascular pools corresponding to large confluent vascular masses (open arrowhead). (b) Axial contrast-enhanced CT image obtained in the late arterial phase at a lower level than a shows a dilated tortuous left hepatic artery (arrow), a finding that helps distinguish hereditary hemorrhagic telangiectasia from passive congestion of the liver. (c) Axial contrast-enhanced CT image obtained in the portal venous phase depicts homogeneous enhancement of the liver and dilatation of the IVC and hepatic veins. (d) Early hepatic arteriogram shows dilated tortuous hepatic arteries and innumerable small telangiectatic vascular lesions (arrows). (e) Late hepatic arteriogram shows early filling of the right hepatic vein (arrowhead).
Figure 9. Multisystemic hereditary hemorrhagic telangiectasia in a 64-year-old woman. (a) Axial contrast-enhanced CT image obtained in the portal venous phase shows marked cardiomegaly and bilateral pleural effusions. (b) Axial contrast-enhanced CT image obtained in the portal venous phase at a lower level than a shows marked dilatation of the hepatic veins and IVC and heterogeneous enhancement of the hepatic parenchyma because of the presence of small telangiectasias. (c–e) Axial contrast-enhanced CT images obtained during the portal venous phase at successively lower levels than a and b show a dilated celiac axis (arrow in c), tortuous dilated intrahepatic arteries and veins, and an enormous mesenteric arteriovenous malformation (* in e). (f) Superior mesenteric angiogram shows multiple arteriovenous malformations.
The incidence and prevalence of passive hepatic congestion are unknown. The disease may remain subclinical and undiagnosed, and there is no known age or sex predilection.

Symptoms of congestive heart failure mask gastrointestinal symptoms. Patients may present with asymptomatic elevation of liver enzymes, jaundice, and right upper quadrant pain, hepato-tomely, and increased abdominal girth (26). Treatment is aimed at managing the patient’s elevated right-sided heart pressure and hepatic venous congestion. Decompensated or uncorrected venous congestion may prevent sufficient drainage of sinusoi-
dal blood flow, resulting in parenchymal atrophy, necrosis, and, ultimately, fibrosis or cardiac cirrhosis (26). Cardiac cirrhosis may be irreversible even after correction of the cardiac function.

**CT Findings**

In the arterial phase, there is early enhancement of a dilated IVC and central hepatic veins because of the reflux of contrast material from the right atrium into the IVC (Fig 11). Parenchymal phase images show a heterogeneous, mottled mosaic pattern of enhancement, with linear and curvilinear areas of poor enhancement due to delayed enhancement of small and medium-sized hepatic veins (Figs 11, 12) (27,28). There may be peripheral large patchy areas of poor delayed enhancement due to stagnant flow within the periphery of the liver. Perivascular lymphedema may be seen as linear low-attenuation regions encircling the intrahepatic IVC or portal veins and should not be confused with venous thrombosis (27). Hepatomegaly and ascites may be present. Chest images may show cardiomegaly, congestive heart failure, and pericardial and pleural effusion.

**Pearls**

An enlarged, heterogeneous liver may be seen as a manifestation of acute or early cardiac disease. The liver becomes small and cirrhotic with chronic passive congestion. It is important to distinguish passive hepatic congestion from Budd-Chiari syndrome. The latter is characterized by narrowed retrohepatic IVC or hepatic veins and by a classic flip-flop pattern of enhancement between the arterial and venous phases in the presence of acute disease. In chronic Budd-Chiari syndrome, the IVC and hepatic veins are obliterated, and large regenerative nodules may be present.
Peliosis Hepatis

Peliosis hepatis is a rare benign disorder that causes sinusoidal dilatation and multiple blood-filled lacunae within the liver (29) that vary from 1 mm to several centimeters in diameter (30). Although the exact origins of this disorder are unknown, peliosis hepatis may be secondary to chronic wasting diseases (eg, tuberculosis, leprosy), various malignancies (eg, hepatocellular carcinoma), AIDS, drugs (eg, anabolic steroids, corticosteroids, tamoxifen, oral contraceptives, diethylstilbestrol, azathioprine), renal or cardiac transplantation, or toxin (eg, polyvinyl chloride, arsenic, thorium oxide) exposure (30). Bacillary peliosis hepatis is caused by Bartonella species infection in HIV-positive patients (31). Several conditions have been associated with peliosis hepatis, including diabetes mellitus, sprue, and necrotizing vasculitis. However, in 20%–50% of patients, no associated condition is identified.
The incidence and prevalence of peliosis hepatitis are unknown. Peliosis hepatitis is a rare entity, and most cases are incidentally discovered in surgical or autopsy specimens (32). There is an increasing incidence of cases of bacillary peliosis in immunocompromised patients.

The natural course of peliosis hepatitis is not well understood, but the condition is most often asymptomatic. Peliosis hepatitis regresses after drug withdrawal, cessation of steroid therapy, or resolution of an associated infectious disease (30). Antibiotic therapy with erythromycin has led to clinical improvement in patients with HIV-related peliosis hepatitis caused by *Bartonella* species. Peliosis hepatitis may be complicated by hepatic failure, cholestasis, and portal hypertension. Hepatic capsular rupture caused by a large mass may lead to intraperitoneal hemorrhage, requiring immediate embolization and surgical intervention.

**CT Findings**

At unenhanced CT, the lesions are typically hypoattenuating (30,32,33). At contrast-enhanced CT, a variable enhancement pattern is seen: In the arterial phase, the lesions may appear hypoattenuating in relation to the liver and progressively become hyperattenuating. The enhancement may be either complete or early globular vessel-like enhancement with progressive centrifugal (Fig 13) (32,34) or centrifugal (Fig 14) (33) enhancement on portal venous phase images without mass effect on adjacent hepatic vessels. In the delayed phase, diffuse increased attenuation may be seen (33). Thrombosed cavities resemble nonenhancing nodules and may simulate metastases or abscesses (31). Smaller lesions (with a maximal diameter of <1 cm) may not be visible on both the unenhanced and the contrast-enhanced images (31).

**Pearls**

*Careful history-taking is necessary for proper diagnosis. The differential diagnosis may include hypervascular metastases and hemangiomas.* Hypervascular metastases typically are hypo- to isoattenuating to liver parenchyma in the portal venous phase. Hemangiomas characteristically demonstrate discontinuous globular enhancement in the arterial phase, with a centripetal progression of enhancement, and mass effect on adjacent hepatic vessels. The distinction between a hepatic abscess and hepatic peliosis is important to avoid inadvertent aspiration of the peliotic lesions, which may be fatal (35). Pyogenic hepatic abscesses are typically multiseptate and may have a cluster-of-grapes appearance without enhancement of the pyogenic fluid components.
Hepatic Infarction

Hepatic infarction is defined as areas of coagulation necrosis from hepatocyte cell death caused by local ischemia, which in turn results from the obstruction of circulation to the affected area, most commonly by a thrombus or embolus (Fig 15). Hepatic infarction is uncommon because of the dual blood supply from the hepatic artery and portal vein, as well as extensive collateral vessels (36). In most cases, hepatic infarction results from either insult to the hepatic artery or portal vein thrombosis superimposed on hepatic arterial occlusion (36). Hepatic infarction may be iatrogenic (occurring after hepatobiliary surgery, intrahepatic chemoembolization, or a transjugular intrahepatic portosystemic shunt procedure) or posttraumatic (occurring after hepatic artery or portal vein laceration). It may occur as a complication of hepatic artery stenosis or thrombosis after liver transplantation, or it may be secondary to hypercoagulability (in sickle cell disease or antiphospholipid antibody syndrome), vasculitis (in polyarteritis nodosa or systemic lupus erythematosus), or infection (in sepsis and shock or rare “emphysematous hepatitis”) (5).

Hepatic infarction is uncommon, occurring at any age, without sex predilection. Hepatic artery thrombosis leading to infarction most often occurs after hepatic transplantation and has been reported in 3% of adult transplant recipients and 12% of pediatric transplant recipients (5,37).

CT Findings

Unenhanced CT shows peripheral wedge-shaped, rounded, or irregularly shaped tubular areas of low attenuation paralleling bile ducts. Wedge-shaped areas are predominantly peripheral and extend to the capsular surface, whereas rounded areas tend to be central in location (38). Bile lakes may be seen as a late sequela of large infarcts from ischemic necrosis of bile duct epithelium. The extravasated bile is surrounded by fibrous tissue forming localized intrahepatic bile duct dilatation (38). Gas formation has been described in sterile infarcts as well as infected ones. Sterile gas is related to the release of intracellular gas from necrotic tissue, an origin similar to that of gas bubbles in hepatic tumors after embolization therapy or radiofrequency ablation (38–40).

The lesions are more conspicuous on contrast-enhanced images than on unenhanced images. They manifest as perfusion defects and typically are distributed in a geographic or segmental pattern with or without straight margins (36,38).

Figure 15. Hepatic infarction. Schematic of a cadaveric liver allograft shows wedge-shaped regions of infarction in the right hepatic lobe because of thrombosis of the hepatic artery anastomosis.

Their enhancement pattern is patchy and heterogeneous (Fig 16) because of nondisplaced vessels (portal veins or arterial or venous collaterals) in the infarcted areas (38). Some components of the lesions remain hypoattenuating in arterial, portal venous, and delayed phases, representing regions of necrotic tissue, hemorrhage, or fibrous tissue with little or no vascularization seen at histologic analysis. Other lesion components remain isoattenuating to the surrounding liver parenchyma in the portal venous phase, a finding suggestive of retained viable tissue or of revascularized fibrotic tissue.

Pearls

Infarction is a serious complication of liver transplantation that results in significant morbidity and mortality and often requires repeat transplantation. Preservation of portal tracts is a feature worthy of emphasis because it helps differentiate infarction from other causes of hypovascular foci in transplanted livers (eg, abscess, biloma, and postbiopsy hematoma). Focal steatosis may mimic hepatic infarction; however, steatosis tends to occur in characteristic locations, and the enhancement of vessels in focal regions of steatosis is preserved, with enhancement approaching that of normal liver parenchyma. Hepatic abscess, which also is included in the differential diagnosis, often has a cluster-of-grapes appearance with rimlike peripheral enhancement and a nonenhancing central pyogenic component.

Conclusions

The increasing use of bolus-enhanced, multiphase helical CT has resulted in more common recognition of various pathologic conditions of
the hepatic blood vessels. Radiologists who understand the pathophysiologic processes that are represented on CT images are often in a position to suggest a specific explanation for the observed hepatic abnormality. Correlation of imaging features with clinical findings is often essential for accurate diagnosis, because some lesion enhancement patterns may mimic mass lesions. Liver biopsy does not usually contribute diagnostically useful information and may be contraindicated in some of these conditions.

References
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CT of Nonneoplastic Hepatic Vascular and Perfusion Disorders

Maha Torabi, MD, et al

Characteristic findings of Budd-Chiari syndrome are hepatic venous outflow obstruction, intrahepatic and systemic collateral veins, and large regenerative nodules in a dysmorphic liver. Regenerative nodules must be distinguished from multifocal hepatocellular carcinoma. Hypertrophy of the caudate lobe and variations in attenuation should not be interpreted as a tumor.

It may be difficult to distinguish an arterioportal shunt from a small hepatocellular carcinoma. In such situations, repeat imaging in 6 months usually demonstrates the resolution or stability of an arterioportal shunt, as opposed to growth for a hepatocellular carcinoma. Moreover, on portal venous and delayed phase images, a hepatocellular carcinoma usually becomes hypoattenuated to liver and vessels, but an arterioportal shunt has the same attenuation as the vessels.

It is important to distinguish passive hepatic congestion from Budd-Chiari syndrome. The latter is characterized by narrowed retrohepatic IVC or hepatic veins and by a classic flip-flop pattern of enhancement between the arterial and venous phases in the presence of acute disease. In chronic Budd-Chiari syndrome, the IVC and hepatic veins are obliterated, and large regenerative nodules may be present.

Careful history-taking is necessary for proper diagnosis [of peliosis hepatis]. The differential diagnosis may include hypervascular metastases and hemangiomas. Hypervascular metastases typically are hypo- to isoattenuating to liver parenchyma in the portal venous phase. Hemangiomas characteristically demonstrate discontinuous globular enhancement in the arterial phase, with a centripetal progression of enhancement, and mass effect on adjacent hepatic vessels.

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