RadioGraphics

Hepatic Capsular and Subcapsular Pathologic Conditions: Demonstration with CT and MR Imaging[1]

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LEARNING OBJECTIVES

After reading this article and taking the test, the reader will be able to:

- Discuss the anatomic and hemodynamic characteristics of the subcapsular regions of the liver.
- Describe the pathophysiologic features of lesions that can occur at the hepatic capsular and subcapsular regions.
- Identify the CT and MR imaging findings of various pathologic conditions at the hepatic capsular and subcapsular regions.

TEACHING POINTS

See last page

A variety of pathologic conditions and pseudolesions occur at the capsular and subcapsular regions of the liver and are detected with cross-sectional abdominal imaging. These entities are related to anatomic and hemodynamic characteristics of the liver such as negative subdiaphragmatic pressure, connection with other viscera and extraperitoneal sites by the perihepatic ligaments, and a "third inflow" of blood from sources other than the usual hepatic arterial and portal venous sources. Pathologic conditions can affect the hepatic capsular and subcapsular regions by way of peritoneal, hematogenous, biliary, and perihepatic ligamentous routes. Pseudolesions or benign conditions may also be identified on the basis of altered hemodynamics of the liver. Computed tomography and magnetic resonance imaging with a multiphasic approach can be used to identify and characterize these entities. Familiarity with the wide spectrum of pathologic conditions and pseudolesions at the hepatic capsular and subcapsular regions and precise knowledge of the anatomic and hemodynamic characteristics of the liver will aid the radiologist in diagnosing pathologic conditions and differentiating pseudolesions from true lesions.

Abbreviations: IVC = inferior vena cava, THAD = transient hepatic attenuation difference, VIBE = volumetric interpolated breath-hold examination

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Introduction

The hepatic capsular and subcapsular regions account for a relatively small portion of the liver. They may be affected by pathologic conditions that affect the entire liver or are restricted to just this area. Because of certain anatomic and hemodynamic characteristics of the liver (eg, negative subdiaphragmatic pressure that preferentially draws infected material or malignant cells toward the diaphragm, connection with other viscera and extraperitoneal sites by the perihepatic ligaments, "third inflow" of blood from sources other than the usual hepatic arterial and portal venous sources), a variety of pathologic conditions and pseudolesions can occur at the hepatic capsular and subcapsular regions. The pathologic conditions at these sites may be caused by infectious and inflammatory diseases (perihepatitis, parasitic diseases), infiltrative diseases (lymphoma, extramedullary hematopoiesis), and metastatic diseases (seeded metastasis, invasion via the perihepatic ligaments, direct invasion by malignancy from an adjacent organ). A wide variety of pseudolesions or benign conditions may also be identified on the basis of altered hemodynamics of the liver (third inflow, arterioportal shunt, compression of the liver, portal vein obstruction, hepatic vein or inferior vena cava [IVC] obstruction, hepatic infarction, intrahepatic vascular shunts). Miscellaneous lesions include focal fatty sparing or infiltration, surface hepatic and portal veins, pseudolipoma of the Glisson capsule, confluent hepatic fibrosis, and portal vein gas. In this article, we review the anatomic and hemodynamic characteristics of the subcapsular region of the liver and discuss and illustrate the pathophysiologic-radiologic features of the aforementioned pathologic conditions, pseudolesions, and benign conditions.

Anatomic and Hemodynamic Considerations at the Hepatic Subcapsular Region

The upper abdomen around the liver is especially rich in peritoneal reflections. The liver is fixed by the coronary, gastrohepatic, hepatoduodenal, and falciform ligaments. It is connected with other viscera and extraperitoneal sites by these perihepatic ligaments (1). Small areas of the subcapsular region of the liver are known to be supplied by a third inflow of blood from sources other than the usual hepatic arterial and portal venous sources (2). The hemodynamics at the subcapsular region can be altered by obstruction of the portal or hepatic vein. With portal vein obstruction, a homogeneous regional increase or inhomogeneous peripheral increase in arterial flow has been observed during the arterial phase at computed tomography (CT) or magnetic resonance (MR) imaging (3,4). Occlusion of the hepatic veins results in increased sinusoidal pressure and reverses the pressure gradient between the sinusoidal and portal veins. The portal vein then becomes a draining vein. Reversal of the portal venous flow direction results in a decrease in portal venous flow and an increase in arterial flow in the peripheral regions of the liver (5).

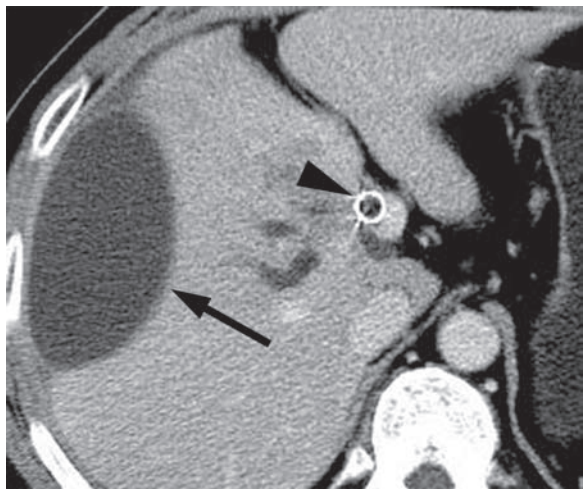
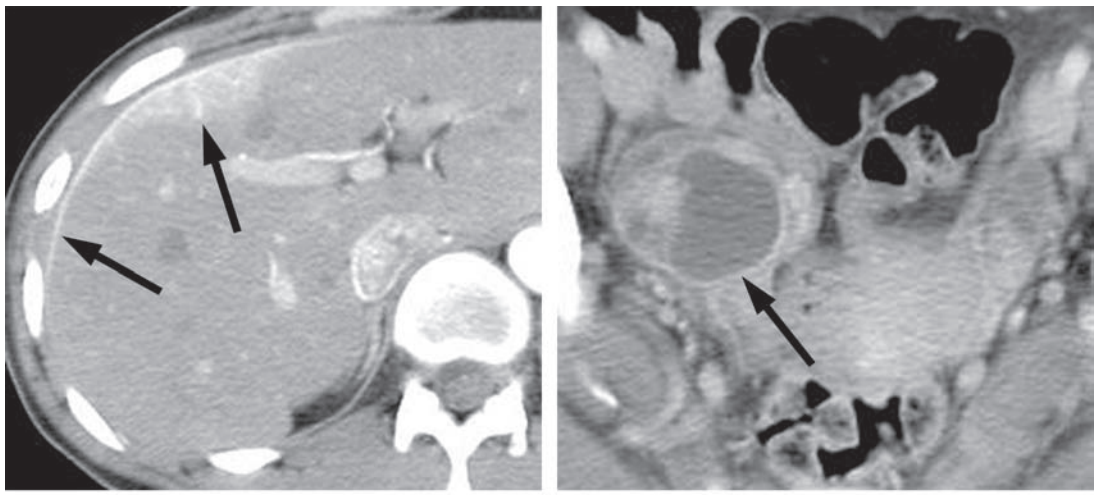


Figure 1. Subcapsular biloma in a 54-year-old man with bile duct cancer. Contrast material–enhanced CT scan shows a lentiform fluid collection and indentation of the underlying liver margin (arrow). Note the metallic stent at the common hepatic duct (arrowhead).

Pathologic Conditions at the Hepatic Capsular and Subcapsular Regions

The liver capsule is composed of two adherent layers: a thick, fibrous inner layer called the Glisson capsule; and an outer serous layer that is derived from the peritoneum. The Glisson capsule covers the entire surface of the liver, whereas the serous layer covers most but not all of the liver surface, excluding the bare area near the diaphragm, the porta hepatis, and the area where the gallbladder is attached to the liver. The subcapsular space is the potential space that is deep relative to the Glisson capsule and superficial relative to the liver parenchyma (6). Fluid, blood, and other benign and malignant entities may occupy the subcapsular space. A subcapsular hematoma appears as a low-attenuation, elliptic collection

Teaching Point



a. b.

Figure 2. Fitz-Hugh–Curtis syndrome in a 33-year-old woman with right upper quadrant pain. **(a)** Contrast-enhanced arterial phase CT scan shows enhancement of the liver capsule and of the subcapsular portion of the liver (arrows). **(b)** Portal venous phase CT scan of the pelvis shows an ovoid hypoattenuating mass with wall enhancement (arrow) at the right adnexa, a finding that is consistent with a tubo-ovarian abscess. The findings in both **a** and **b** are suggestive of Fitz-Hugh–Curtis syndrome.

of blood between the liver capsule and the liver parenchyma at CT caused by the compression of the underlying liver margin by the thick, fibrous Glisson capsule (Fig 1); in contrast, free intraperitoneal blood in the perihepatic space does not cause indentation or flattening of the underlying liver margin. Parenchymal hematomas or contusions appear as focal low-attenuation areas with poorly defined irregular margins in the liver parenchyma at CT.

Infectious and Inflammatory Diseases

Teaching Point

A wide spectrum of infectious and inflammatory diseases can affect the hepatic capsular and subcapsular regions by way of peritoneal, hematogenous, biliary, and perihepatic ligamentous routes. The liver capsule is not visible at CT or MR imaging under normal conditions; however, it becomes visible in the presence of liver disease.

Perihepatitis.—Perihepatitis is defined as inflammation of the peritoneal capsule of the liver and is classically described as being associated with pelvic inflammatory disease (so-called Fitz-Hugh–Curtis syndrome). Fitz-Hugh–Curtis syndrome is thought to result from the peritoneal spread of infection from the pelvic cavity. CT has been reported to demonstrate intense capsular enhancement along the anterior surface of the liver (Fig 2). Capsular enhancement on early-phase images may reflect increased blood flow at the inflamed liver capsule, whereas enhancement on delayed images may reflect the early events of capsular fibrosis (7,8). Although an increase in capsular blood flow caused by inflammation may reach the subcapsular region, resulting in a thick enhancing zone, the details of this process are unknown.

Parasitic Diseases.—Amebiasis, hydatid disease, and schistosomiasis of the liver are relatively common parasitic diseases seen throughout the world. Hepatic involvement by these parasites occurs via the portal venous system and is randomly distributed in the liver. Other major parasitic diseases include fascioliasis, paragonimiasis, and sparganosis. The responsible parasites penetrate the intestinal wall and may migrate through the peritoneal cavity to the liver after perforating the Glisson capsule; thus, the lesions are located mainly at the subcapsular region initially. The parasites migrate toward the central portion of the liver, causing hemorrhage, necrosis, inflammation, and subsequent fibrosis. Thickening of the liver capsule, capsular enhancement, and subcapsular hematoma have been reported. Capsular hyperintensity, ill-defined hyperintense areas, lesions with a tracklike appearance, and nodular areas at the liver may be seen at MR imaging as an inflammatory response, migration route, and

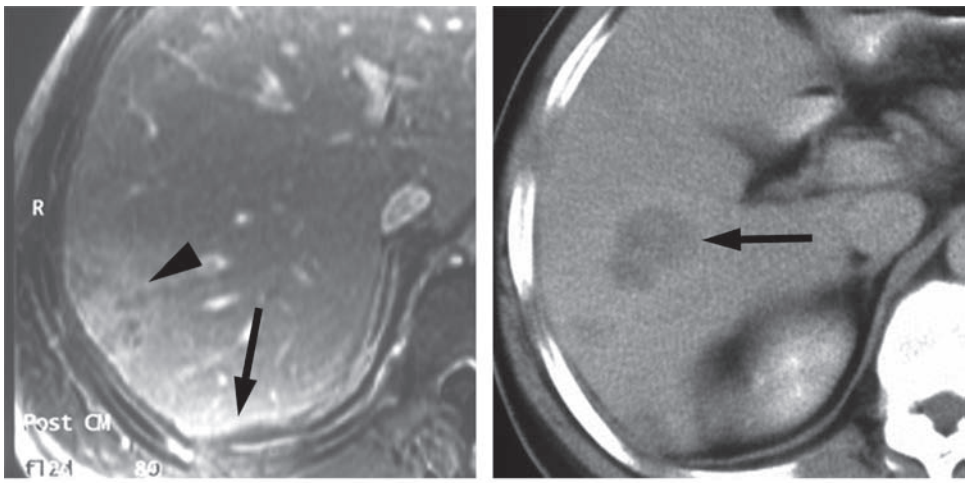


Figure 3. Fascioliasis of the liver in a 49-year-old man. **(a)** Gadolinium-enhanced gradient-echo T1-weighted MR image (repetition time msec/echo time msec = 160/4, 80° flip angle) shows a heterogeneously enhancing lesion (arrowhead) and capsular enhancement (arrow). **(b)** On a contrast-enhanced CT scan obtained 21 days later, the lesion has migrated toward the center of the liver and contains a cluster of microabscesses with a tracklike appearance (arrow).

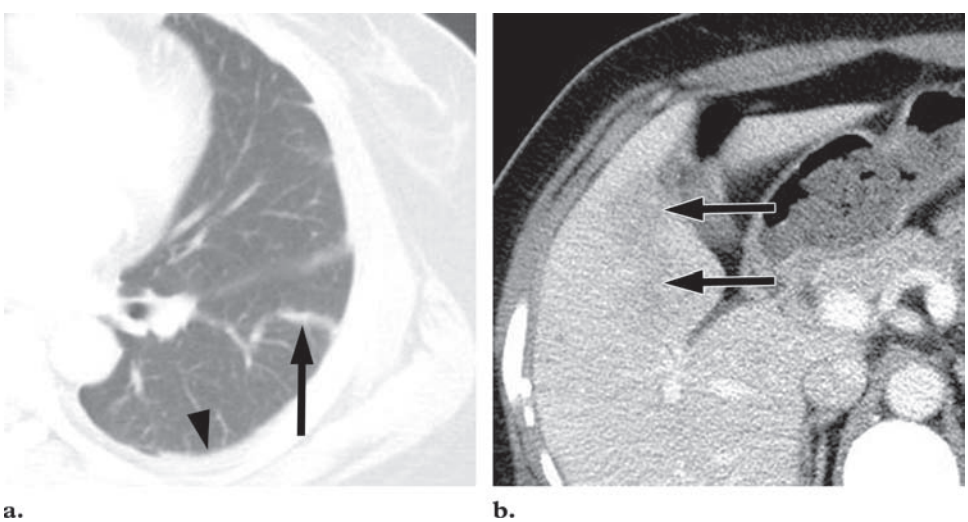


Figure 4. Paragonimiasis involving the lung and liver in a 43-year-old woman. **(a)** Chest CT scan (lung window) shows a parenchymal band at the subpleural portion (arrow). A coexisting left pleural effusion is also seen (arrowhead). **(b)** Portal venous phase CT scan shows an ill-defined hypoattenuating area (arrows) at segment V of the liver due to parasites that have migrated toward the center of the liver, causing hemorrhage, necrosis, inflammation, and subsequent fibrosis. CT scans obtained at a lower level demonstrated abdominal wall involvement.

fibrosis (9). Although these are nonspecific findings and are observed in numerous hepatobiliary diseases, the concomitant presence of lesions in other organs suggests a specific parasitic infestation: A cluster of microabscesses with a tracklike appearance in the subcapsular portion of the liver suggests *Fasciola hepatica* infestation (Fig 3), and pleural effusion and lung cysts suggest paragonimiasis (Fig 4) (10). The diagnosis of a parasitic infestation is made on the basis of clinical symptoms, absolute eosinophilia, positive serologic test results, or detection of ova or worms in the feces or pathologic specimens.

Clonorchiasis is another common parasitic disease of the bile ducts that occurs after ingestion of raw flesh and freshwater fish in endemic areas

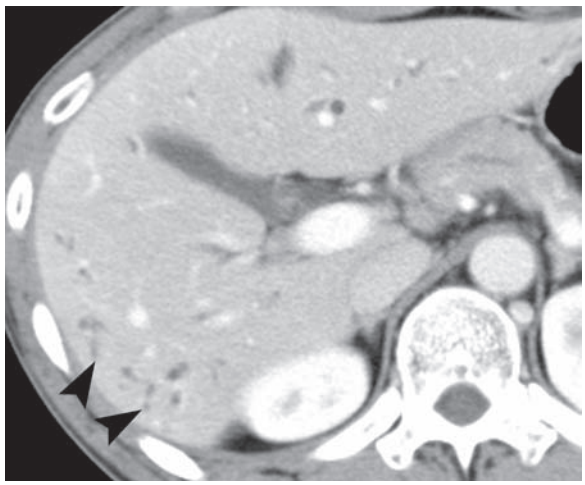


Figure 5. Clonorchiasis of the liver in a 39-year-old man. Contrast-enhanced CT scan shows dilatation of the peripheral intrahepatic bile duct (arrowheads) but no dilatation of the central duct.

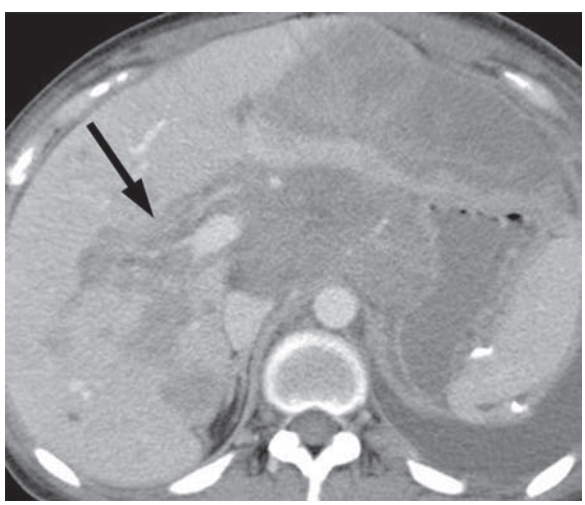


Figure 6. Periportal infiltration by lymphoma in a 24-year-old woman. Contrast-enhanced CT scan shows a homogeneous soft-tissue mass with a slightly expanded contour (arrow) along the right portal vein. Invasion of the hepatic subcapsular region is via the Glisson sheath. A conglomeration of lymph nodes is seen around the celiac axis.

and is most prevalent in the Far East. Clonorchiasis is a trematodiasis caused by infestation by the liver fluke *Clonorchis sinensis*. The adult flukes reside mainly in the medium-sized and small intrahepatic bile ducts; thus, the lesions are located primarily at the subcapsular regions. The flukes occasionally reside in the extrahepatic bile ducts, gallbladder, and pancreatic duct. CT and MR imaging findings include diffuse, uniform dilatation of the small intrahepatic bile ducts with minimal or no dilatation of the large bile ducts and no focal obstructing lesions (Fig 5). Clonorchiasis could be suspected with a high degree of confidence on the basis of dilatation of the peripheral intrahepatic bile duct without dilatation of the extrahepatic bile duct (11). Thickening of the bile ducts is seen at ultrasonography (US) and CT.

Infiltrative Diseases

The part of the Glisson capsule that surrounds the intrahepatic portion of the hepatic portal system is called the Glisson sheath. The Glisson capsule or sheath continues into the subperitoneal space of the gastrohepatic and hepatoduodenal ligaments. Infiltration by benign or malignant tumors or chronic inflammatory lesions can extend along the subperitoneum and progressively invade the subcapsular region of the liver.

Teaching Point

Lymphoma.—The imaging findings of secondary hepatic lymphoma have been reported as variable, ranging from single or multiple small nodules to diffuse infiltrative tumor patterns. Radiology reports of gross periportal infiltration by lymphoma are very rare. Imaging findings include a periportal infiltrating homogeneous mass with a slightly irregular expanded contour (Fig 6) (12). As the disease progresses, the mass may involve the subcapsular region of the liver centrally. These radiologic features should be differentiated from the "periportal halo" or "periportal tracking," which are characterized by circumferential thin halos of fluid or low attenuation around the portal vein branches.

Extramedullary Hematopoiesis.—Extramedullary hematopoiesis is a compensatory phenomenon in which red blood cells are produced outside the bone marrow when marrow production is not sufficient to meet the body's demands. It may be caused by either myeloproliferative disorders with lack of cell formation or hemolytic disease. When the marrow is incapable of producing an adequate number of blood cells, focal areas of hematopoiesis appear in the liver, spleen, and lymph nodes, probably arising from multipotent stem cells. Homogeneous hepatomegaly is commonly found in patients with myelofibrosis, but patterns of focal myeloid metaplasia of the liver have been described in a few case reports (13). The focal hepatic disease can manifest as solitary or multiple lesions. At US, focal intrahepatic myeloid metaplasia appears as well-defined, homogeneously hypoechoic lesions or a well-defined, homogeneously hyperechoic mass encircling the portal vein and its main branches. The lesion is

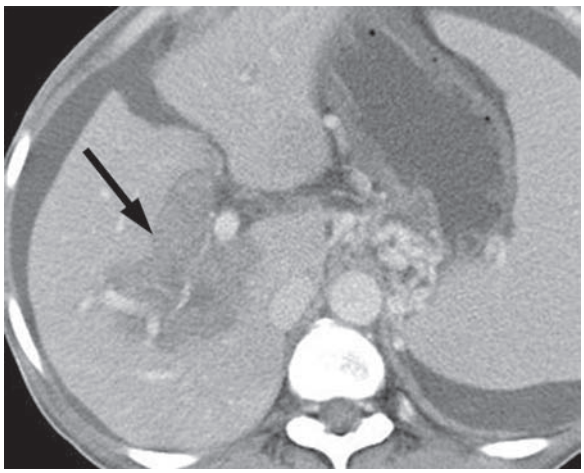


Figure 7. Periportal infiltration by extramedullary hematopoiesis in a 58-year-old woman with chronic myelogenous leukemia. Contrast-enhanced CT scan shows a well-defined soft-tissue mass encircling the right portal vein (arrow), along with ascites and splenomegaly.

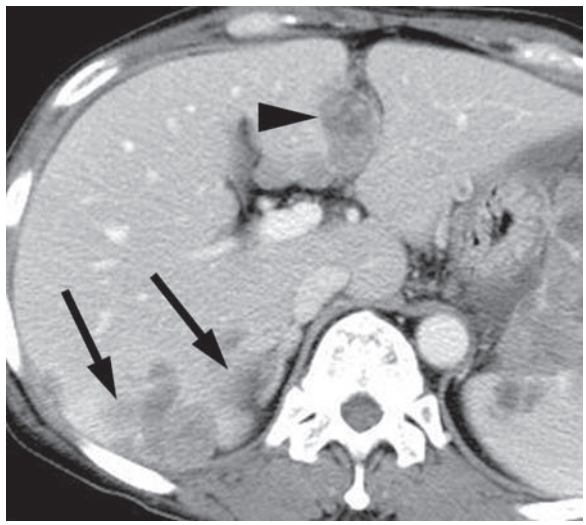


Figure 8. Capsular implants in a 52-year-old woman with ovarian cancer. Contrast-enhanced CT scan shows multiple tumor implants in the liver capsule (arrows). Note that part of the mass extends along the falciform ligament (arrowhead).

hypoattenuating at unenhanced CT with a well-defined edge and does not enhance after the intravenous administration of contrast material (Fig 7). These characteristics could be related to a high grade of intralesional fibrosis (14). Extramedullary hematopoiesis may involve the subcapsular region of the liver based on the progression of disease, similar to involvement of the subcapsular region of the liver by lymphoma.

Metastatic Diseases

Teaching Point

Metastatic diseases of the liver can occur at the capsular and subcapsular regions from seeded metastasis, invasion via the perihepatic ligaments, and direct invasion by malignancy from an adjacent organ. These metastatic lesions are usually much less well defined than are true parenchymal metastases and are not surrounded by liver parenchyma.

Seeded Metastasis.—The peritoneal cavity is a common site of metastatic spread from many different malignancies. In the abdomen, implants on the diaphragmatic surface appear as nodular or plaquelike thickening of the diaphragm. Involvement of the liver results in scalloping of the surface by masses that are lower in attenuation than the parenchyma on contrast-enhanced images (Fig 8) (15,16). These masses may extend along the falciform ligament and may potentially be mistaken for parenchymal metastases. In the case of liver metastasis from ovarian cancer, it

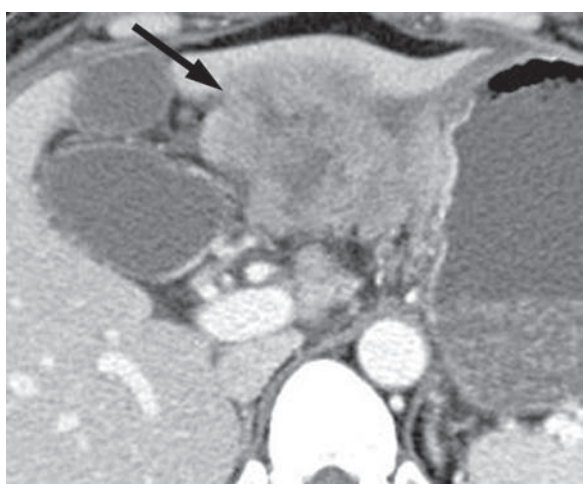


Figure 9. Invasion of the liver by stomach cancer in a 38-year-old woman. CT scan shows invasion of the liver by stomach cancer (arrow) via the gastrohepatic ligament.

is important to distinguish capsular implants or subcapsular invasion of the liver from true parenchymal metastases, since these findings have different implications with regard to prognosis and treatment. Capsular implants are still considered resectable, whereas parenchymal metastases generally are not (16).

Invasion via the Perihepatic Ligaments.—As mentioned earlier, the liver is connected with other viscera and extraperitoneal sites by the perihepatic ligaments. The liver is fixed posteriorly and superiorly by the coronary ligament,

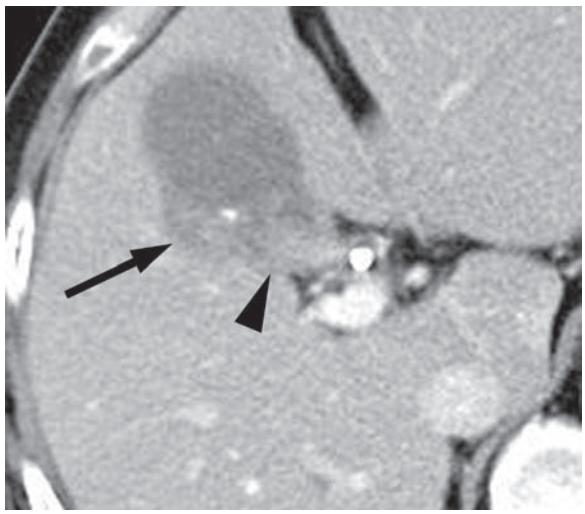


Figure 10. Direct invasion of the liver by gallbladder cancer in a 66-year-old woman. Contrast-enhanced CT scan shows a subcapsular hypoattenuating mass (arrow) near the gallbladder, which contains a gallstone and a mass (arrowhead).

inferiorly and medially by the gastrohepatic and hepatoduodenal ligaments, and anteriorly by the falciform ligament (1). Malignant tumors arising from the stomach (Fig 9), duodenum, pancreas, colon, anterior abdominal wall, or other organs can invade the liver along these perihepatic ligaments.

Direct Invasion by Malignancy from an Adjacent Organ.—Malignant tumors involving the stomach, gallbladder, adrenal gland, and kidney adjacent to the liver can invade the liver directly. Contiguous spread of gallbladder cancer is facilitated especially by the thin hepatic wall, which lacks a substantial lamina propria and has only a single muscular layer (Fig 10). In addition, the perimuscular connective tissue of the gallbladder is continuous with the interlobular connective tissue of the liver (17).

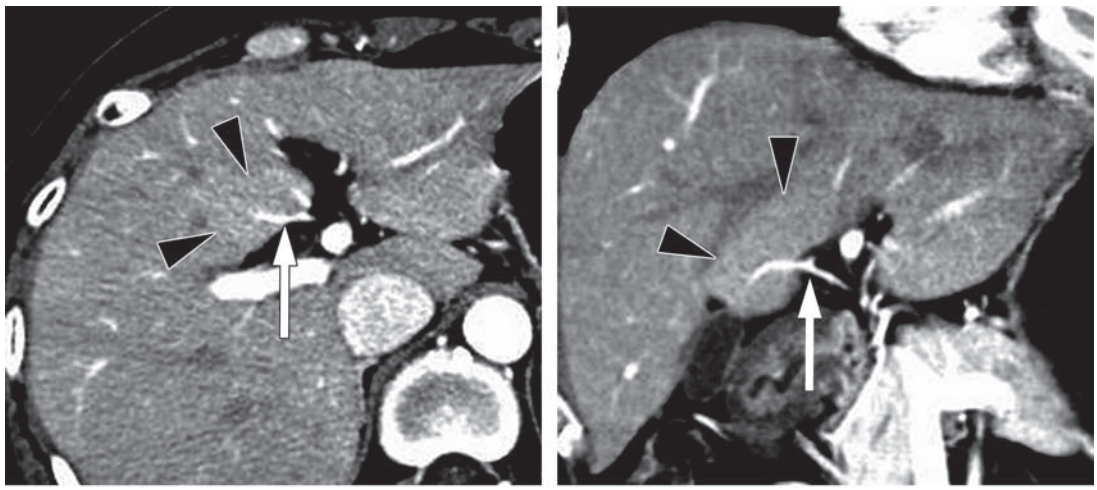
Pseudolesions and Benign Conditions Related to Altered Liver Hemodynamics

Teaching Point

A wide variety of pseudolesions and benign conditions may be identified in the subcapsular region on the basis of altered hemodynamics of the liver due to a third inflow, an arterioportal shunt, compression of the liver, portal vein obstruction, hepatic vein or IVC obstruction, hepatic infarction, or intrahepatic vascular shunts.

Pseudolesions.—Understanding pseudolesions of the liver seen at CT or MR imaging is important because of their close resemblance to primary liver cancers or metastases. Pseudolesions are caused by unusual hemodynamics or focal hemodynamic changes of the liver. The transient hepatic attenuation difference (THAD) is an attenuation difference of the liver appearing during dynamic bolus-enhanced CT and does not correspond to a mass. Nontumorous THAD is generally seen as an area of high attenuation at arterial phase hepatic imaging that returns to normal attenuation during the portal venous phase. A wedge shape, a straight-line margin, and the presence of normal vessels coursing through the lesion on hepatic arterial phase images make a diagnosis of THAD very likely.

Although most of the liver has a dual blood supply, small areas of the subcapsular portion of the liver are known to be supplied by another venous system, a third hepatic inflow (cholecystic, parabiliary, or epigastric-paraumbilical venous system) (18,19). The cholecystic veins enter the liver directly through the liver bed (segments IV and V) or run through the Calot triangle and join the parabiliary veins at the porta hepatis. The parabiliary venous system is within the hepatoduodenal ligament and collects venous blood from the head of the pancreas, the distal part of the stomach, and the bile duct system. The epigastric-paraumbilical venous system consists of small veins around the falciform ligament that drain the venous blood from the anterior part of the abdominal wall directly into the liver (19). At contrast-enhanced CT, the timing of visualization of the third inflow to the liver is variable. Blood enters the liver quickly via cholecystic veins coursing through the liver bed, and the pseudolesion appears as an enhanced area around the gallbladder during the hepatic arterial phase. Blood flowing through the parabiliary venous system enters slightly later, and the pseudolesion is enhanced during the arterioportal phase. The typical location of the pseudolesion—at the



a. b.

Figure 11. Pseudolesion in segment IV of the liver due to aberrant right gastric venous drainage in a 49-year-old woman. Arterial phase axial **(a)** and coronal reformatted **(b)** CT images show a focal enhancing area (arrowheads) in the posterior aspect of segment IV of the liver and inflow from an aberrant right gastric vein within the enhancing area (arrow).

dorsal aspect of segment IV—may be a key to the correct diagnosis (Fig 11). Blood flowing through the epigastric-paraumbilical venous system reaches the liver rather late (around the late portal phase), so that the pseudolesion is rarely enhanced (2). In the case of obstruction of the superior vena cava, contrast medium injected through an upper extremity reaches the liver directly via the internal thoracic vein, superior epigastric vein, and superior vein of Sappey and produces strongly enhanced areas in the cephalic portion of segment IV during the hepatic arterial phase (Fig 12) (20).

An arterioportal shunt is an organic or functional communication between a hepatic arterial branch and the portal venous system. It results in the redistribution of arterial flow into a focal region of the portal venous flow (21). The causes of arterioportal shunts include hepatic neoplasms, iatrogenic trauma such as a biopsy or biliary drainage, liver cirrhosis (nontumorous arterioportal shunt), and portal vein obstruction or compression (functional arterioportal shunt). At two-phase helical CT, an arterioportal shunt appears as an area of peripheral wedge-shaped high attenuation with or without internal branching structures during the hepatic arterial phase (Fig 13) and is slightly hyper- or isoattenuating relative to the liver during the portal venous phase (22).

Blood flow through the portal vein is reduced when there is increased pressure on the hepatic parenchyma and, consequently, increased arterial inflow to the affected hepatic segment. Rib compression, perihepatic peritoneal implants, pseudomyxoma peritonei, and perihepatic fluid collections are examples of potential causes of focal hepatic parenchymal compression (23). An ill-defined low-attenuation area appears in the subcapsular region on portal venous phase images (Fig 14). There should be a compressing structure outside the liver that creates a concave deformity on the hepatic surface just adjacent to the low-attenuation area.

Portal Vein Obstruction.—Portal vein obstruction is caused by vein thrombosis, tumor invasion, compression, or surgical ligation. Perfusion alterations are produced by increases in arterial flow through the transsinusoidal, transvasal, transtumoral, and especially transplexal (peribiliary) routes to compensate for the diminished portal venous flow (24). In portal vein obstruction, two patterns of increased arterial flow have been observed. A homogeneous regional increase in arterial inflow occurs in cases involving a tumor thrombus, thromboembolus, or compression or stricture of the portal vein. An inhomogeneous peripheral increase in arterial inflow occurs in

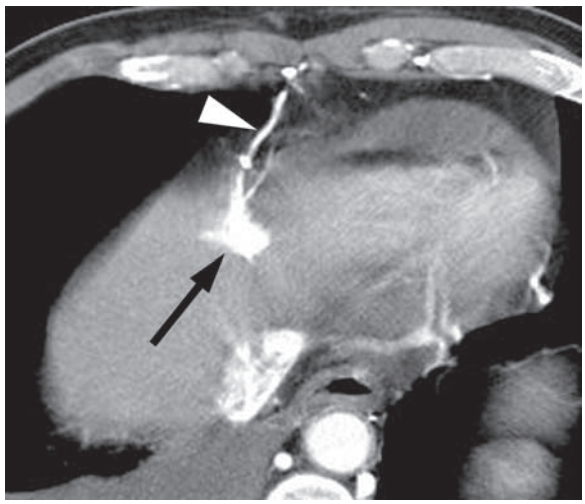


Figure 12. Pseudolesion in segment IV of the liver due to superior vena caval obstruction caused by lung cancer in a 57-year-old man. Arterial phase CT scan shows strong enhancement at the cephalic portion of segment IV (arrow) caused by the direct flow of contrast medium into the liver via the superior vein of Sappey (arrowhead).

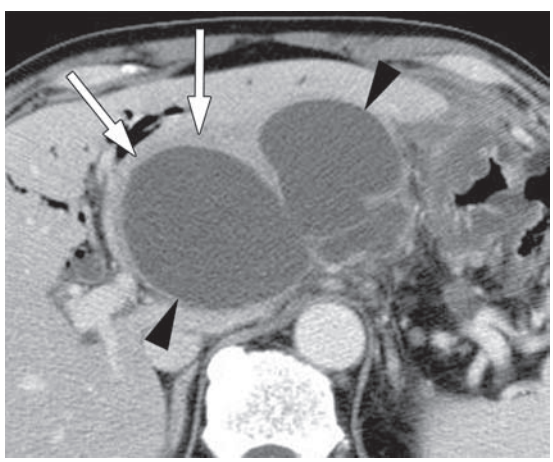


Figure 14. Pseudolesion due to compression by a pseudocyst in a 63-year-old man. Portal venous phase CT scan shows a bandlike low-attenuation area in the subcapsular region (arrows) compressed by a pancreatic pseudocyst (arrowheads).

cases involving cavernous transformation of the thrombosed main portal vein. This pattern occurs because the collateral vessels can supply the central (periportal) regions better than the peripheral regions. To compensate, arterial flow increases and thus gives rise to scattered areas of high attenuation in the periphery during the hepatic arterial phase (Fig 15) (25).

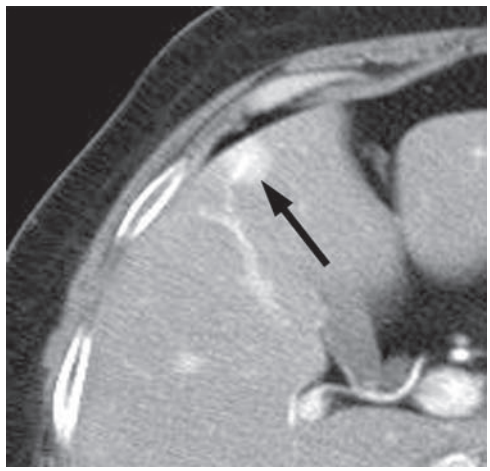


Figure 13. Nontumoral arterioportal shunt in a 45-year-old man with liver cirrhosis. Arterial phase CT scan shows a wedge-shaped area of high attenuation with an internal branching structure (arrow).

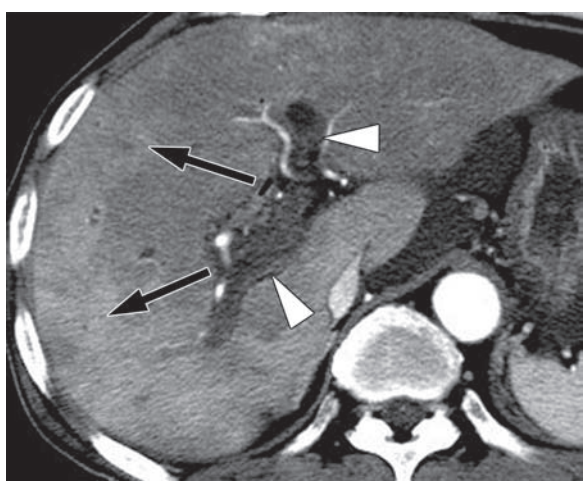


Figure 15. Portal vein thrombosis in a 60-year-old man who had undergone surgery for common bile duct cancer 5 months earlier. Arterial phase CT scan shows thrombosis of the right and left portal veins (arrowheads) and enhancement of the periphery of the right hepatic lobe (arrows) secondary to the compensatory increase in arterial flow.

Hepatic Vein or IVC Obstruction.—In cases of diminished hepatic venous flow, the hemodynamics are complex and can differ according to the acuteness, severity, and location of the obstruction. During the acute phase of hepatic vein obstruction, the portal vein becomes a draining

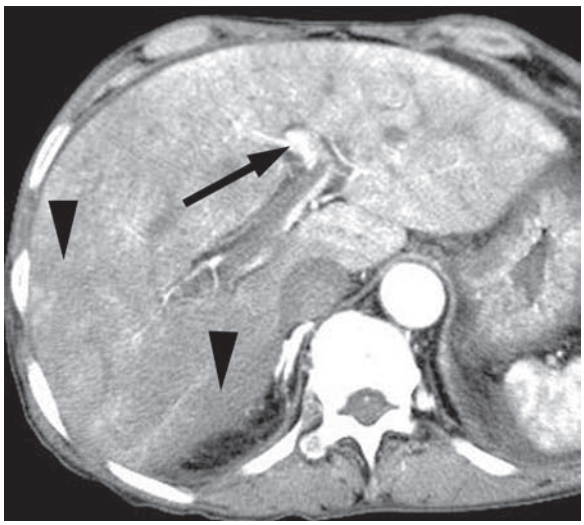


Figure 16. Acute IVC obstruction in a 77-year-old man. Arterial phase CT scan shows a central patchy area with poor enhancement of the peripheral areas of the liver (arrowheads) due to portal venous blood flowing away from the liver (arrow). The obstruction of the IVC was caused by hepatocellular carcinoma (not shown).

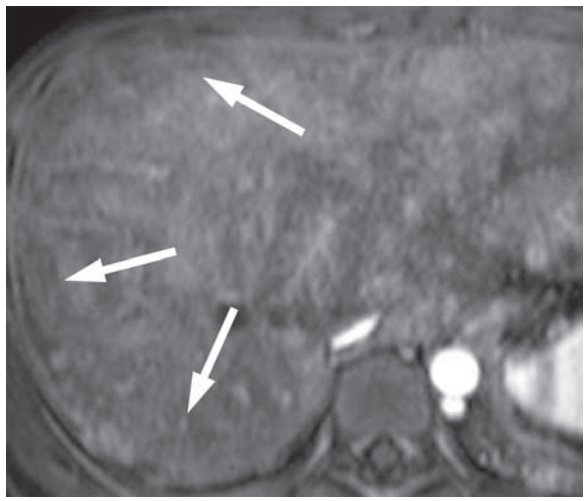


Figure 17. Chronic IVC obstruction in a 21-year-old woman with Budd-Chiari syndrome. Gadolinium-enhanced three-dimensional gradient-echo T1-weighted volumetric interpolated breath-hold examination (VIBE) MR image (3.8/1.7, 12° flip angle) shows heterogeneous enhancement with a reticular pattern (arrows), especially at the periphery of the liver.

vein, resulting in a functional arterioportal shunt, which appears as a central patchy area with poor enhancement of the peripheral areas of the liver during the hepatic arterial phase; these findings are manifestations of the underlying hepatic congestion (Fig 16). The caudate lobe may show increased enhancement and enlargement corresponding to the compensatory changes due to its separate venous drainage. Rarely, subcapsular enhancement is observed in patients with acute symptoms. The peripheral hepatic enhancement is probably related to the independent drainage of the subcapsular regions by their own capsular veins. In the chronic stage, necrotic areas resulting from hepatic vein congestion are replaced by fibrotic tissue, and reestablishment of hepatic venous drainage occurs through intrahepatic collateral vessels. Heterogeneous enhancement with a reticular pattern, especially at the periphery of the liver, is frequently seen during the portal venous and hepatic arterial phases (Fig 17) (26).

Hepatic Infarction.—Hepatic infarction was a rare event until recently. With the advent and increasing use of liver transplantation for end-stage liver disease and laparoscopic cholecystectomy (with their associated vascular complications),

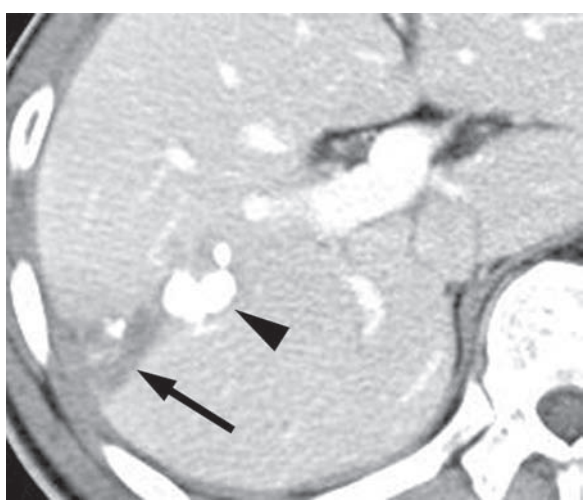


Figure 18. Hepatic infarction in a 53-year-old woman who had undergone transarterial chemoembolization 6 months earlier. Contrast-enhanced CT scan shows a wedge-shaped hypoattenuating lesion (arrow) extending to the liver surface peripheral to embolized hepatocellular carcinoma (arrowhead).

there has been an increase in the prevalence of hepatic infarction. Still, hepatic infarction is thought to be relatively rare because of the dual blood supply and extensive collateral pathways of the liver. At contrast-enhanced CT, it typically appears as sharply defined, wedge-shaped low-attenuation lesions extending to the liver surface (Fig 18) (27).

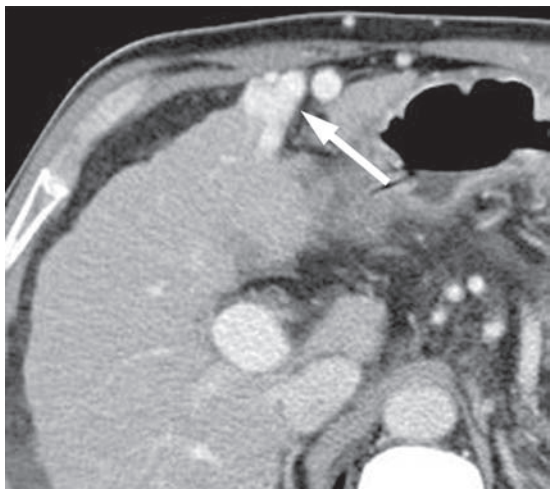
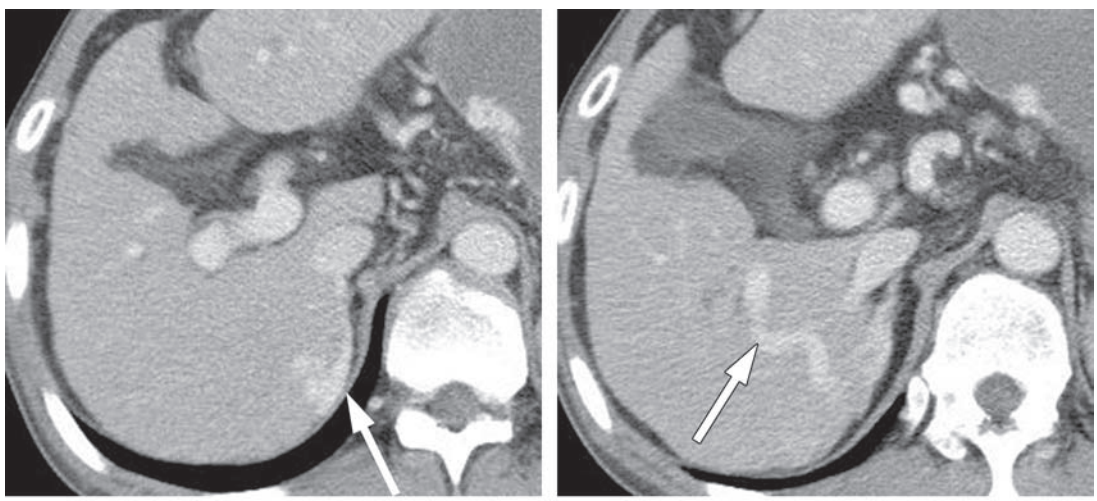


Figure 19. Falciform ligament type intrahepatic portosystemic shunt in a 57-year-old man with liver cirrhosis. Portal venous phase CT scan shows a dilated paraumbilical vein (arrow) at the falciform ligament.



a. **b.**

Figure 20. Right posterior portal vein type intrahepatic portosystemic shunt in a 47-year-old man with liver cirrhosis. **(a)** Portal venous phase CT scan shows shunting between the posterior portal vein and the IVC (arrow). **(b)** Portal venous phase CT scan shows tortuous dilatation of the posterior portal vein (arrow).

Intrahepatic Vascular Shunts.—Various intrahepatic portosystemic shunts or intrahepatic venous collateral vessels to systemic venous pathways may be identified in the subcapsular area of the liver.

Intrahepatic portosystemic venous shunts are uncommon. They may be either congenital (rare) or acquired (caused mainly by trauma and portal hypertension). Various intrahepatic portosystemic shunts may be identified in the subcapsular area of the liver. The falciform ligament type (umbilical and paraumbilical veins) is the best known of the various intrahepatic portosystemic shunts (Fig 19). The right posterior portal vein type is well recognized and is the most common type next to the paraumbilical vein type. The dilated right posterior portal vein runs across the posterior surface of the liver, takes a tortuous course, forms a venous aneurysm outside the liver, and then drains into the IVC either directly or through the adrenal vein (Fig 20). The apex type of vein is called the superior vein of Sappey. The vein arises, from the peripheral branch of the left medial portal vein, courses toward the apex of the liver, and drains into the internal thoracic

vein (Fig 21) (28). This pathway is sometimes observed in patients with superior vena cava syndrome. Contrast medium injected into the arm goes into the liver via the internal thoracic veins and this shunt.

Intrahepatic venovenous collateral vessels provide alternative pathways for venous return to the right heart when the IVC or hepatic veins are obstructed. One common collateral pathway connects the intrahepatic venous collateral vessels to the systemic venous pathways via the subcapsular veins and may be identified on the surface of the liver (Fig 22) (29).

Miscellaneous Lesions

Focal Fatty Sparing or Infiltration.—Focal fatty sparing or deposition characteristically occurs in specific areas (eg, adjacent to the falciform ligament or ligamentum venosum; in the porta hepatis, gallbladder fossa, or subcapsular portion). Locations of focal fatty sparing and focal fatty change are essentially the same because they are related to venous abnormalities in those areas of the liver parenchyma. Focal fatty sparing on a background of diffuse fatty infiltration may be misinterpreted as a focal tumor. The pathogenesis of focal fatty sparing is not clear, although a disturbance in the portal venous flow has been postulated. Focal fatty sparing is usually easy to recognize, being seen in the typical locations as hypoechogenic areas at US or hyperattenuation at CT or as a loss of signal intensity on opposed-phase as compared with in-phase chemical shift MR images (Figs 23, 24). The pathogenesis of focal fatty infiltration is still controversial. The abnormal accumulation of fat may be caused by relative ischemia due to decreased portal venous flow or decreased delivery of unknown substances via the portal vein. A key factor that has recently been linked to the development of fatty liver is increased levels of insulin in the portal venous blood. Typically, fatty change is subcapsular in location, has geographic margins, causes no mass effect, and contains normal nondistorted vessels (Fig 25) (30). For patients with renal failure and insulin-dependent diabetes, insulin can be delivered in the peritoneal dialysate rather than by the usual subcutaneous route. This route exposes the subcapsular hepatocytes to a higher concentration of insulin than the remainder of the liver. Insulin blocks the usual oxidation of free fatty acids in the hepatocytes, leading to preferential esterification into triglycerides, which

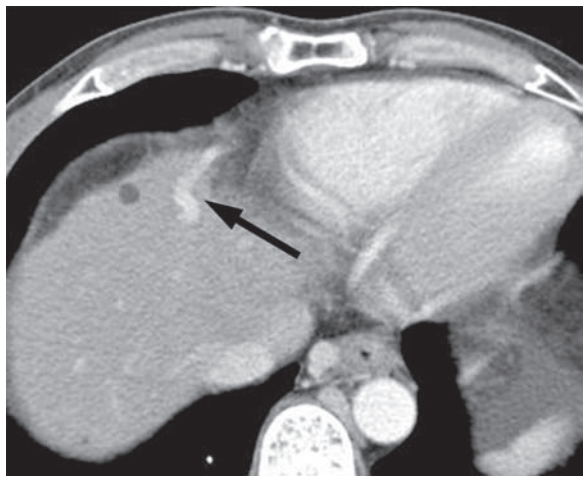


Figure 21. Superior vein of Sappey type intrahepatic portosystemic shunt in a 48-year-old man with liver cirrhosis. Portal venous phase CT scan shows a dilated left medial portal vein (arrow) arising from the surface of the medial segment of the left lobe and coursing toward the left anteriorly.

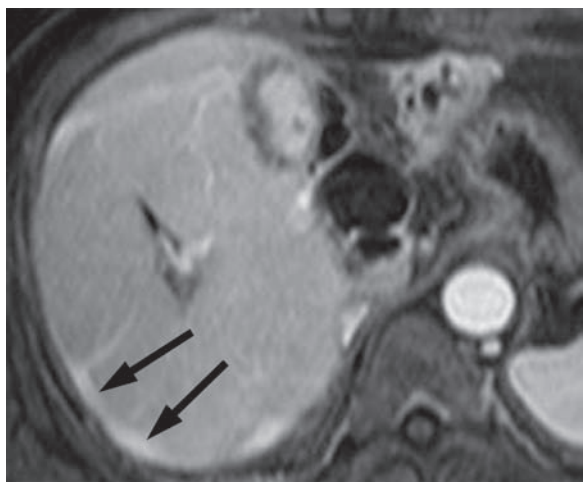
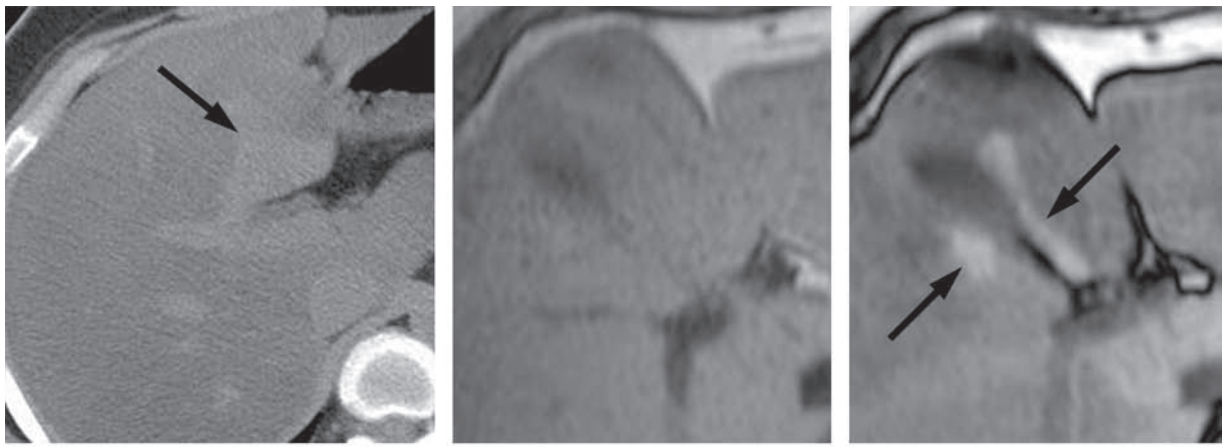
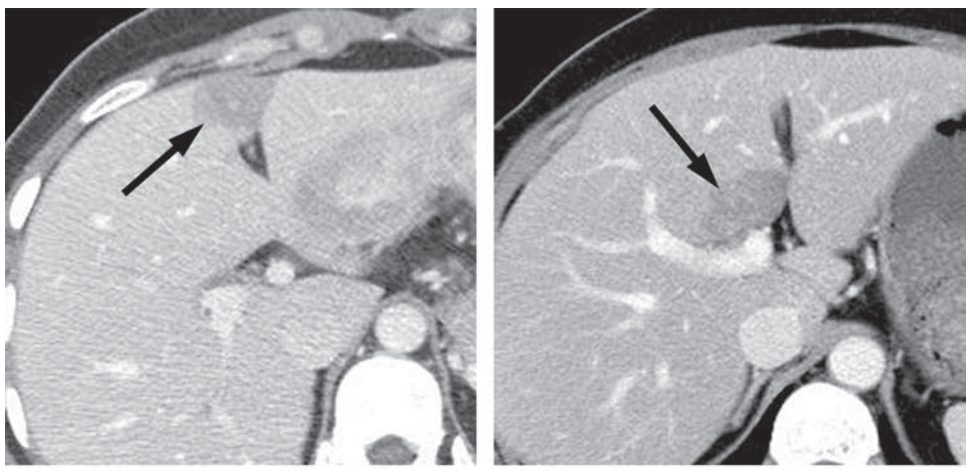


Figure 22. Hepatic venovenous shunt in a 59-year-old woman with portal cavernous transformation caused by recurrent pyogenic cholangitis. Gadolinium-enhanced three-dimensional gradient-echo T1-weighted VIBE MR image (3.8/1.7, 12° flip angle) shows dilatation of the hepatic capsular vein (arrows) as intrahepatic venous collateral vessels.



23. 24a. 24b.

Figures 23, 24. **(23)** Focal fatty sparing in a 33-year-old man. Unenhanced CT scan shows a wedge-shaped hyperattenuating lesion (arrow) at the posterior aspect of segment IV of the liver. **(24)** Focal fatty sparing in a 54-year-old woman. **(a)** Axial in-phase gradient-echo T1-weighted MR image (180/5) shows homogeneous signal intensity of the liver parenchyma. **(b)** Opposed-phase T1-weighted MR image (180/2.4) shows a loss of signal intensity in the liver parenchyma. Note the geography of the hyperintense areas (arrows) in segments IV and V near the gallbladder fossa.



a. b.

Figure 25. Focal fatty infiltration. **(a)** Portal venous phase CT scan shows a hypoattenuating area in segment IV of the liver (arrow) adjacent to the falciform ligament. **(b)** Portal venous phase CT scan shows an ovoid hypoattenuating area (arrow) at the posterior aspect of segment IV. Note that the lesion appears hypoattenuating, with no mass effect, and contains normal vessels.

then accumulate in the cell. The result is a unique pattern of fatty infiltration in a subcapsular location known as hepatic subcapsular steatosis (31).

Surface Hepatic and Portal Veins.—Unusual hepatic and portal veins located just beneath the liver capsule have been reported as anatomic variants (32). However, these unusual surface veins are not necessarily anatomic variants. The hepatic vessels may be seen in the subcapsular portion of the liver if the peripheral part of the parenchyma is collapsed (33). We have experience with several

cases involving hepatic vessels on the surface or in the subcapsular region of the liver after partial resection of the right hepatic lobe (Fig 26), a phenomenon that, to our knowledge, has not been described in the radiology literature.

Pseudolipoma of the Glisson Capsule.—Also known as hepatic pseudolipoma, pseudolipoma of the Glisson capsule was initially described by Rolleston in 1891. Pseudolipoma refers to an encapsulated lesion containing degenerated fat that is enveloped by a liver capsule. It is thought to represent a detached colonic epiploic appendix that develops a fibrous capsule and lodges in the peritoneal cavity. When this lesion is in proximity to the liver, it may become attached to the liver capsule. The differential diagnosis of this uncommon entity includes serosal metastasis and a fibrosing subcapsular necrotic nodule. At imaging, it appears as a well-circumscribed nodule on the liver surface with a center of either fat or soft-tissue attenuation (Fig 27) (34).

Confluent Hepatic Fibrosis.—Fibrosis can occur diffusely or focally in the setting of cirrhosis. Focal confluent fibrosis appears wedge shaped at the subcapsular portion and radiates from the hepatic portal. Most of the lesions are known to involve the medial segment of the left lobe, the anterior segment of the right lobe, or both. Focal abnormalities may be mistaken for malignant neoplasms. Focal confluent fibrosis appears as a hypoattenuating lesion with volume loss that becomes isoattenuating or minimally hypoattenuating at postcontrast CT (35). Fibrosis can appear as an area of low signal intensity on T1-weighted MR images and high signal intensity on T2-weighted images owing to the presence of edema and numerous vascular spaces (as in most hepatic neoplasms). It usually demonstrates delayed enhancement on contrast-enhanced images (Fig 28). This enhancement might be due to a pooling of contrast material within the edema or nonarterial vascular channels within the fibrotic lesions. The signal intensity characteristics and contrast enhancement of these lesions at MR imaging are not unique and do not allow differentiation from hepatic neoplasms. Retraction of the overlying liver capsule and a typical geographic pattern of involvement can be helpful in diagnosing this condition and in distinguishing it from hepatocellular carcinoma (36). A mass effect on the liver contour and on the adjacent

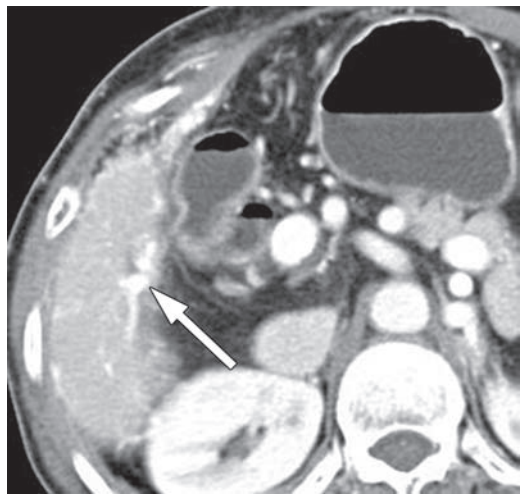


Figure 26. Dilated hepatic capsular vein in a 52-year-old woman who had undergone posterior segmentectomy of the liver for removal of intrahepatic stones. Contrast-enhanced CT scan shows tortuous dilatation of the hepatic capsular vein (arrow).

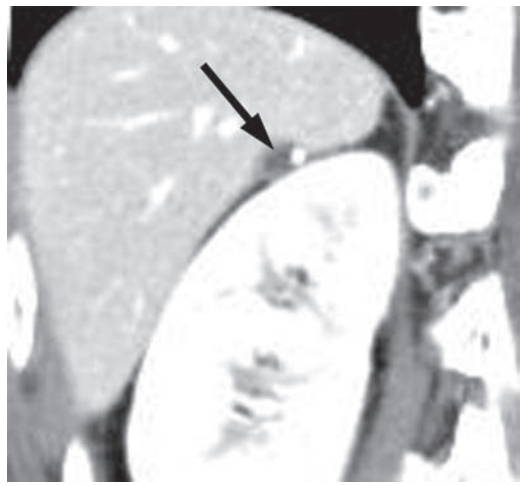


Figure 27. Pseudolipoma of the Glisson capsule in a 36-year-old woman. Portal venous phase coronal reformatted CT image shows a fatty mass with peripheral calcification (arrow) at the posterior notch of the liver.

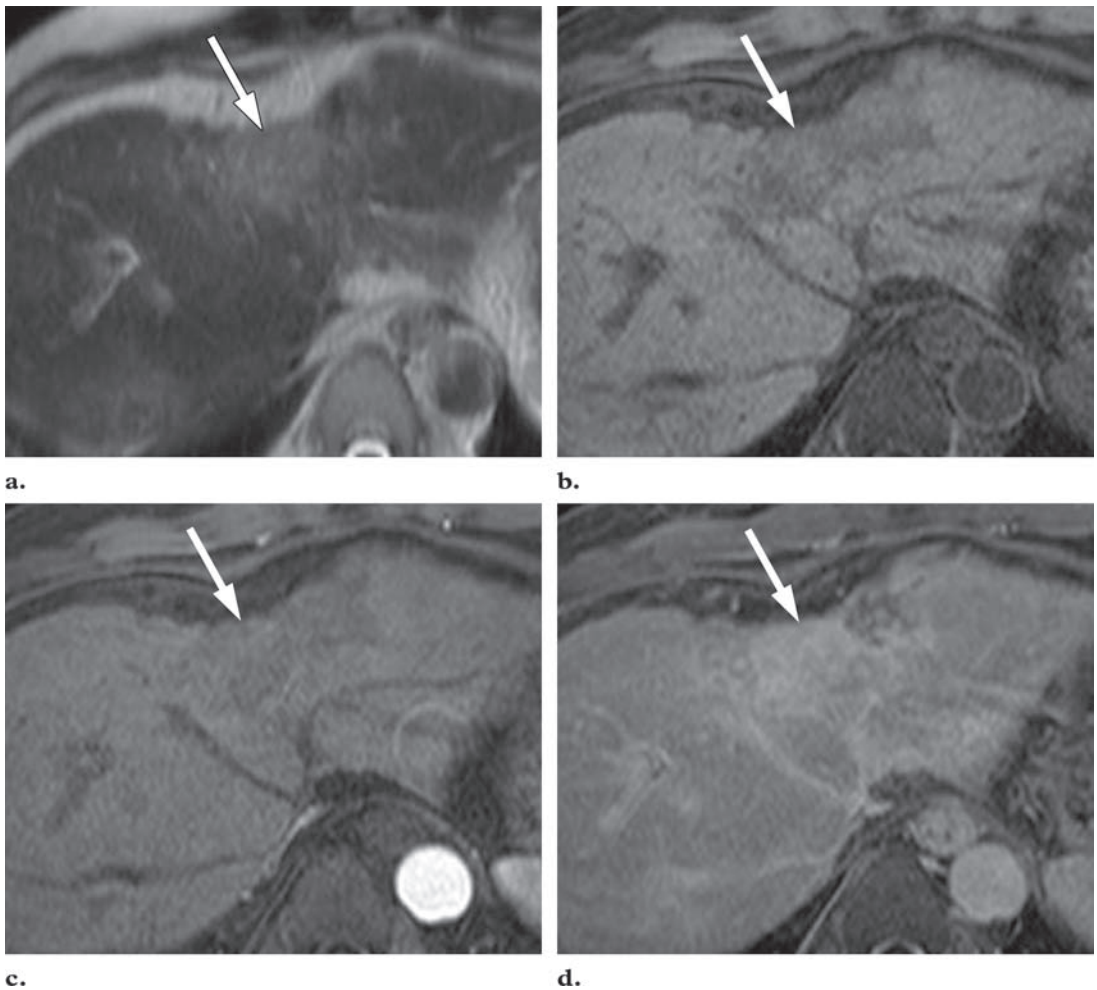
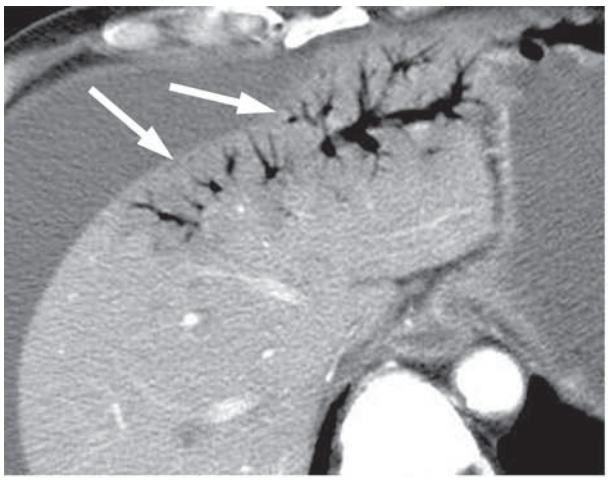


Figure 28. Confluent hepatic fibrosis in a 45-year-old man with liver cirrhosis. Axial T2-weighted (1000/60) **(a)**, unenhanced **(b)**, and gadolinium-enhanced three-dimensional gradient-echo T1-weighted VIBE (3.8/1.7, 12° flip angle) **(c, d)** MR images show a large heterogeneous area at segment IV of the liver. The lesion exhibits low signal intensity on the T1-weighted images and high signal intensity on the T2-weighted image. Delayed enhancement is seen in **d**, with no significant early contrast enhancement in **c**. Note also the capsular retraction (arrow).

blood vessels, as well as a more nodular appearance without retraction of the overlying liver capsule, are usually present in hepatocellular carcinoma.

Portal Vein Gas.—Portal vein gas is most commonly caused by mesenteric ischemia but may have a variety of other causes, such as infectious and inflammatory abdominal diseases, interventional procedures, trauma, and transplantation. At CT, portal vein gas appears as tubular areas of decreased attenuation in the liver periphery, predominantly in the left lobe (Fig 29). The radiographic criterion for portal vein gas is a radiolucent branching area extending to within 2 cm of the liver capsule. Intrahepatic portal vein gas must be differentiated from air in the biliary tree. In the latter condition, the distribution of hepatic gas is central, around the portal hilum, and does not extend to within 2 cm of the liver capsule (Fig 30) (37).



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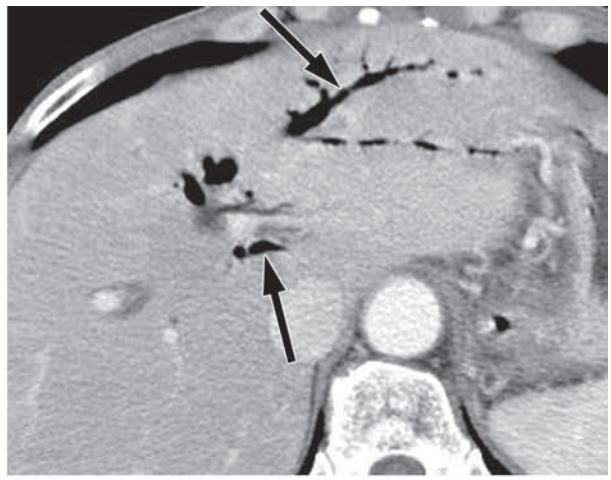

30.

Figures 29, 30. **(29)** Portal vein gas in a 63-year-old man with a strangulating obstruction of the small bowel. Contrast-enhanced CT scan shows branching areas of low attenuation (arrows) extending to within 2 cm of the liver capsule. The collections of portal vein gas are smaller and more numerous than those seen in pneumobilia. **(30)** Pneumobilia in a 55-year-old man who had undergone endoscopic sphincterotomy for removal of a common bile duct stone. Contrast-enhanced CT scan shows air (arrows) in the central and peripheral regions of the liver. The air does not extend to within 2 cm of the liver capsule.

Conclusions

A variety of pathologic conditions and pseudolesions occur at the hepatic capsular and subcapsular regions and can be identified with CT or MR imaging. Precise knowledge of the anatomic and hemodynamic characteristics of the liver aids in diagnosing pathologic conditions and differentiating pseudolesions from true lesions. It is important that the radiologist be familiar with the wide spectrum of pathologic conditions and pseudolesions in the capsular and subcapsular regions of the liver to ensure correct diagnosis.

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Hepatic Capsular and Subcapsular Pathologic Conditions: Demonstration with CT and MR Imaging

Jun Woo Lee, MD, et al

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Page 1308
The subcapsular space is the potential space that is deep relative to the Glisson capsule and superficial relative to the liver parenchyma.

Page 1309
A wide spectrum of infectious and inflammatory diseases can affect the hepatic capsular and subcapsular regions by way of peritoneal, hematogenous, biliary, and perihepatic ligamentous routes.

Page 1311
Infiltration by benign or malignant tumors or chronic inflammatory lesions can extend along the subperitoneum and progressively invade the subcapsular region of the liver.

Page 1312
Metastatic diseases of the liver can occur at the capsular and subcapsular regions from seeded metastasis, invasion via the perihepatic ligaments, and direct invasion by malignancy from an adjacent organ.

Page 1313
A wide variety of pseudolesions and benign conditions may be identified in the subcapsular region on the basis of altered hemodynamics of the liver due to a third inflow, an arterioportal shunt, compression of the liver, portal vein obstruction, hepatic vein or IVC obstruction, hepatic infarction, or intrahepatic vascular shunts.