

# Multidetector CT of Emergent Biliary Pathologic Conditions<sup>1</sup>

## SA-CME

See pp 2117–2124.

## LEARNING OBJECTIVES FOR TEST 1

After completing this journal-based SA-CME activity, participants will be able to:

- Identify the CT imaging appearances of emergent biliary pathologic conditions.
- Discuss clinical scenarios that may lead to the development of biliary disease.
- Describe how CT findings can guide treatment referral for emergent biliary pathologic conditions.

## TEACHING POINTS

See last page

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Various biliary pathologic conditions can lead to acute abdominal pain. Specific diagnosis is not always possible clinically because many biliary diseases have overlapping signs and symptoms. Imaging can help narrow the differential diagnosis and lead to a specific diagnosis. Although ultrasonography (US) is the most useful imaging modality for initial evaluation of the biliary system, multidetector computed tomography (CT) is helpful when US findings are equivocal or when biliary disease is suspected. Diagnostic accuracy can be increased by optimizing the CT protocol and using multiplanar reformations to localize biliary obstruction. CT can be used to diagnose and stage acute cholecystitis, including complications such as emphysematous, gangrenous, and hemorrhagic cholecystitis; gallbladder perforation; gallstone pancreatitis; gallstone ileus; and Mirizzi syndrome. CT also can be used to evaluate acute biliary diseases such as biliary stone disease, benign and malignant biliary obstruction, acute cholangitis, pyogenic hepatic abscess, hemobilia, and biliary necrosis and iatrogenic complications such as biliary leaks and malfunctioning biliary drains and stents. Treatment includes radiologic, endoscopic, or surgical intervention. Familiarity with CT imaging appearances of emergent biliary pathologic conditions is important for prompt diagnosis and appropriate clinical referral and treatment.

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**Abbreviations:** CPR = curved planar reformatted, ERCP = endoscopic retrograde cholangiopancreatography, MPR = multiplanar reformatted, MRCP = magnetic resonance cholangiopancreatography

RadioGraphics 2013; 33:1867–1888 • Published online 10.1148/rg.337125038 • Content Codes: **CT** **ER** **GI**

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## Introduction

Computed tomography (CT) is used to evaluate approximately one-third of patients aged 60 years and older who present to the emergency department with abdominal pain, and biliary disease is diagnosed in approximately 10% of these patients (1). A wide spectrum of disease affects the biliary system, varying from uncomplicated acute cholecystitis to liver transplantation-associated hepatic artery thrombosis and biliary necrosis. Acute cholecystitis typically is the first diagnostic consideration in patients presenting with right upper quadrant pain, and ultrasonography (US) is the primary imaging modality for this condition. US also is helpful in identifying biliary obstruction but may not always allow characterization of the cause of the obstruction. Both magnetic resonance cholangiopancreatography (MRCP) and CT allow detailed evaluation of the biliary system. CT often is chosen because of its widespread availability, decreased operator dependence versus US, faster imaging speed, decreased artifacts, and increased anatomic coverage during a single breath hold.

We review CT techniques useful for imaging the biliary tract in patients with acute abdominal pain. We describe clinical signs and symptoms; typical imaging findings; and interventional radiologic, endoscopic, and surgical treatment of common and uncommon acute biliary pathologic conditions. Specific topics include inflammatory diseases of the gallbladder, acute sequelae of benign and malignant biliary obstruction, iatrogenic biliary tract complications, and uncommon causes of biliary disease.

## Protocol Considerations

The CT protocol should be designed to optimally image the biliary system to increase diagnostic accuracy. The imaging protocol corresponds to an appropriately designed clinical question. Parameters of the CT protocol include the number of imaging phases, extent of anatomic coverage, and use of oral or intravenous contrast agents.

Use of at least 64 detector rows at CT imaging will reduce scan times and motion artifact and increase anatomic coverage during a single breath hold. Thinner collimation allows increased spatial resolution and lesion detection but also creates increased image noise. The potentially increased patient radiation dose is a disadvantage that can be minimized by optimizing individual scan parameters. Isotropic voxel datasets obtained with CT allow the creation of multiplanar reformatted

(MPR) images in the axial, coronal, sagittal, or any user-defined plane. Curved planar reformatted (CPR) images are particularly helpful for visualizing the entire common bile duct on a single image and assessing the ampulla of Vater.

At our institution, axial CT images for non-pancreaticobiliary indications are obtained in the portal venous phase with a positive oral contrast agent, and coronal and sagittal reformations are produced from isotropic source data. If pancreaticobiliary disease is suspected, nonenhanced, pancreatic parenchymal, and portal venous phase images are obtained with a neutral oral contrast agent (VoLumen; Bracco Diagnostics, Princeton, NJ). MPR and CPR images created from thin-section (<1 mm) isotropic datasets allow detailed evaluation of the biliary system and improve detection of choledocholithiasis and potential obstruction sites (2). Additionally, use of a neutral oral contrast agent reduces false-positive detection of choledocholithiasis that results from reflux of a positive oral contrast agent into the biliary tree (3). A combination of pancreatic parenchymal and portal venous phase images with coronal and sagittal MPR images can maximize diagnostic sensitivity and the staging of pancreatic adenocarcinoma (4).

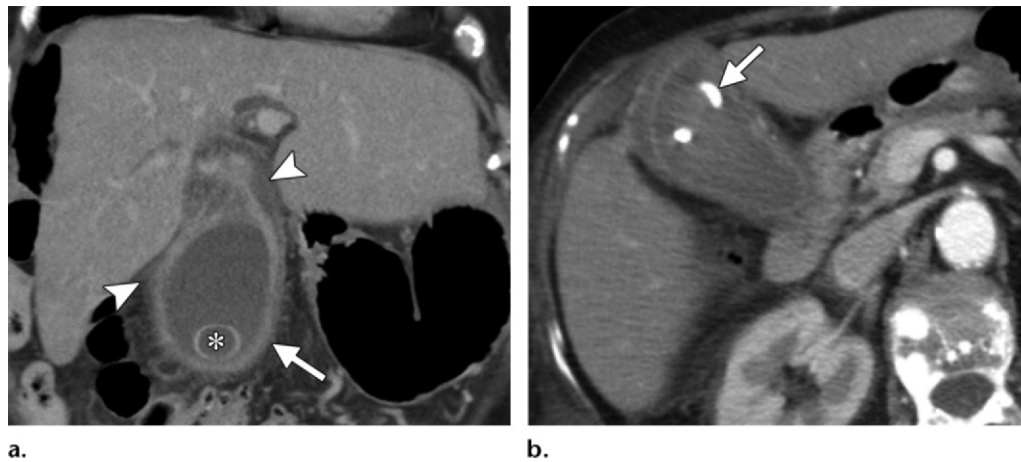
Delayed phase imaging of the biliary system is not routinely performed; however, if there is a clinical concern for cholangiocarcinoma, an additional 10- to 15-minute delayed phase is added to enhance visualization of contrast agent-retaining fibrous tissue. Nonenhanced, arterial phase, and maximum intensity projection CT images are obtained if vascular disease such as hepatic artery thrombosis is suspected.

## Gallbladder Disease

### Acute Cholecystitis

Gallstones are implicated in over 90% of cases of acute cholecystitis. Gallstones obstruct the cystic duct or gallbladder neck and irritate the gallbladder mucosa, leading to the release of several inflammatory mediators and progressive gallbladder wall inflammation. Acute acalculous cholecystitis is the cause in 5%–10% of cases of acute cholecystitis. This condition typically occurs in severely ill patients and is associated with higher mortality and morbidity.

Although US is the recommended initial imaging modality for evaluation of acute cholecystitis, CT often is used in the clinical setting because other disease processes such as pancreatitis, gastritis, and bowel obstruction may mimic right up-



**Figure 1.** Acute calculous cholecystitis in an 87-year-old woman with metastatic breast cancer. **(a)** Coronal contrast-enhanced CT image shows a hydropic gallbladder with a large gallstone (\*). Circumferential gallbladder wall thickening (arrow) and pericholecystic inflammatory changes and fluid (arrowheads) are seen. **(b)** Axial contrast-enhanced CT image shows interval placement of a cholecystostomy tube (arrow), a procedure necessary because of the patient's high surgical risk and comorbidities. The cholecystostomy tube was removed 2 months later after confirming the patency of the cystic and common bile ducts and consulting with the surgeon.

#### Teaching Point

per quadrant pain. **The negative predictive value of CT for diagnosis of acute cholecystitis is 89%, and although this value is lower than that of US, CT can exclude the diagnosis in patients with a low clinical suspicion for gallbladder disease and nonspecific clinical symptoms (5).**

Gallstones are classified by their concentration of cholesterol, a factor that correlates well with their appearance at CT (6). Cholesterol stones typically contain more than 70% cholesterol, while pigment stones contain less than 25% cholesterol and are composed primarily of calcium bilirubinate. A pure cholesterol or pigment stone is uncommon. Gallstones can be visualized at CT only if they have a different attenuation than surrounding bile. When CT is performed at 140 kVp, its sensitivity for gallstone detection is 81%–86%, versus 52%–67% at lower voltage settings. The absolute attenuation of higher energy x-rays decreases more with water than with gallstones, thereby widening the difference in attenuation between the two and improving visualization (7).

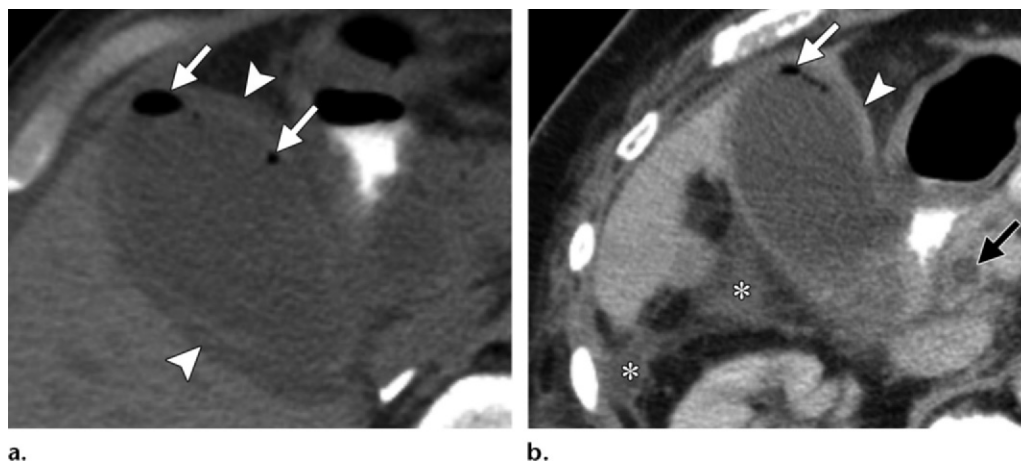
CT findings of acute cholecystitis include gallstones, gallbladder wall thickening more than 3 mm, gallbladder distention more than 5 cm in the short axis or more than 8 cm in the long axis, pericholecystic fluid, inflammatory stranding, and subserosal edema (Fig 1) (8). Imaging performed in the late arterial or early portal venous phase may show transient curvilinear increased hepatic parenchymal enhancement adjacent to the inflamed gallbladder, a finding caused by reactive hepatic arterial hyperemia (9).

Treatment of acute cholecystitis is based on symptom severity and the patient's surgical risk. Laparoscopic cholecystectomy is the preferred treatment in low-risk patients, while percutaneous cholecystostomy and antibiotic therapy are the initial treatments in high-risk patients or those with delayed diagnosis who do not improve with medical management (10,11).

Accurate characterization of acute cholecystitis at CT helps in planning the optimal surgical approach. A recent study found that an absence of gallbladder wall enhancement at CT (a finding suggestive of gangrenous cholecystitis) or the presence of a gallstone in the gallbladder infundibulum is associated with higher rates of conversion from laparoscopic to open cholecystectomy (12). Patients treated with percutaneous cholecystostomy undergo cholecystectomy after the acute inflammation subsides if they are suitable surgical candidates. Cholecystectomy also is indicated in patients whose disease course worsens despite percutaneous cholecystostomy drainage, a finding that suggests progression to complicated cholecystitis.

#### Emphysematous Cholecystitis

Emphysematous cholecystitis is a variant of acute cholecystitis, and both entities have similar clinical manifestations. Acute emphysematous cholecystitis occurs with secondary infection of the gallbladder



**Figure 2.** Emphysematous cholecystitis in two patients. **(a)** Axial nonenhanced CT image in a 70-year-old woman treated with sunitinib for a gastrointestinal stromal tumor shows intraluminal air in the gallbladder (arrows) and pericholecystic fluid (arrowheads). **(b)** Axial contrast-enhanced CT image in an 85-year-old man shows intraluminal air (white arrow) in the gallbladder, gallbladder wall thickening (arrowhead), pericholecystic fluid (\*), and dilatation of the common bile duct (black arrow).

wall by gas-forming organisms such as *Clostridium perfringens*, *Escherichia coli*, and *Bacillus fragilis* (13). Unlike acute cholecystitis, acute emphysematous cholecystitis affects men twice as commonly as women (13). It is more common in patients with diabetes, possibly because of poor microvasculature and perfusion of the gallbladder wall, and it carries a five times greater risk of perforation compared with uncomplicated acute cholecystitis (14). Emphysematous cholecystitis also has been reported in patients treated with sunitinib, a tyrosine kinase inhibitor used to treat metastatic renal cell carcinoma and gastrointestinal stromal tumor (15).

CT is the most sensitive and specific imaging modality for identification of gas in the gallbladder lumen or wall (Fig 2) because the US appearance of gas may mimic that of a porcelain gallbladder or multiple stones in a contracted gallbladder (16). In severe cases, acute emphysematous cholecystitis may progress to gangrene and perforation. Other nonspecific CT findings include pneumobilia, irregularity or discontinuity of the gallbladder wall, pericholecystic fluid, abscess formation, and free intraperitoneal air, a finding indicative of perforation.

Because of its high mortality and morbidity, emphysematous cholecystitis is treated with urgent cholecystectomy (13). In high-risk or unsuitable

surgical candidates, percutaneous cholecystostomy and antibiotic therapy may be used as temporizing measures until cholecystectomy is possible.

### Gangrenous Cholecystitis

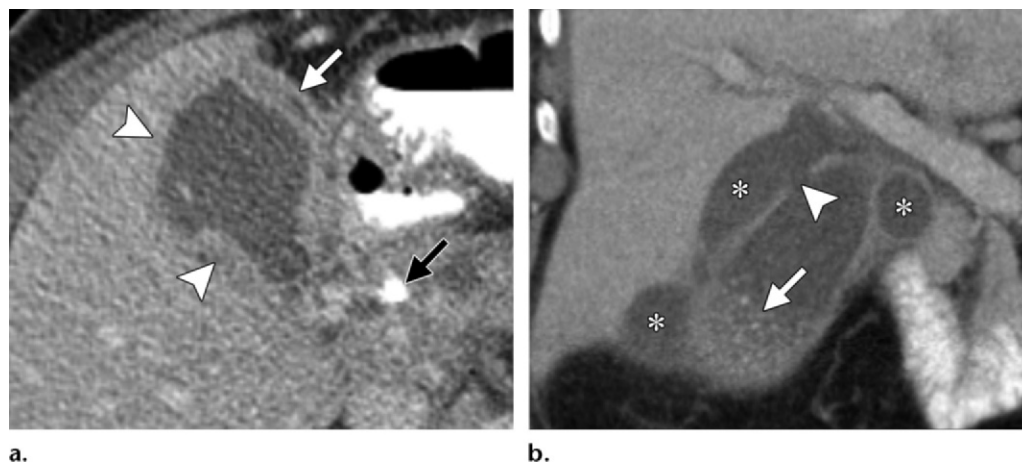
Gangrenous cholecystitis is a relatively common but severe complication of acute cholecystitis, affecting up to 26% of patients with acute cholecystitis (17). Gangrenous cholecystitis carries high morbidity and mortality and tends to occur in elderly patients or patients with diabetes (17).

**CT is insensitive but highly specific for the diagnosis of acute gangrenous cholecystitis. Specific CT findings include intramural or intraluminal gas, intraluminal membranes, irregular or absent gallbladder wall, lack of gallbladder wall enhancement, and pericholecystic abscess formation** (Fig 3) (18). Transient adjacent hepatic parenchymal enhancement, pericholecystic fluid, and striation of the gallbladder wall are less specific findings that may be seen with acute gangrenous or nongangrenous cholecystitis. These findings may indicate severe acute cholecystitis with possible gallbladder necrosis (18).

The definitive treatment for acute gangrenous cholecystitis, as with other variants of acute cholecystitis, is cholecystectomy. Both laparoscopic and open cholecystectomy are acceptable treatments, although the conversion rate from laparoscopic to open surgery is three times higher in

**Teaching Point**





**Figure 3.** Gangrenous cholecystitis in two patients. **(a)** Axial contrast-enhanced CT image in a 55-year-old woman shows irregular thickened gallbladder mucosa (arrowheads) with discontinuity and sloughed membranes (white arrow). A common bile duct stent (black arrow) is in place for biliary decompression from known pancreatic cancer. **(b)** Coronal contrast-enhanced CT image in a 70-year-old woman shows a hydropic gallbladder with dependent stones and sludge (arrow), focal disruption of the gallbladder wall (arrowhead), and multiple loculated pericholecystic fluid collections (\*).



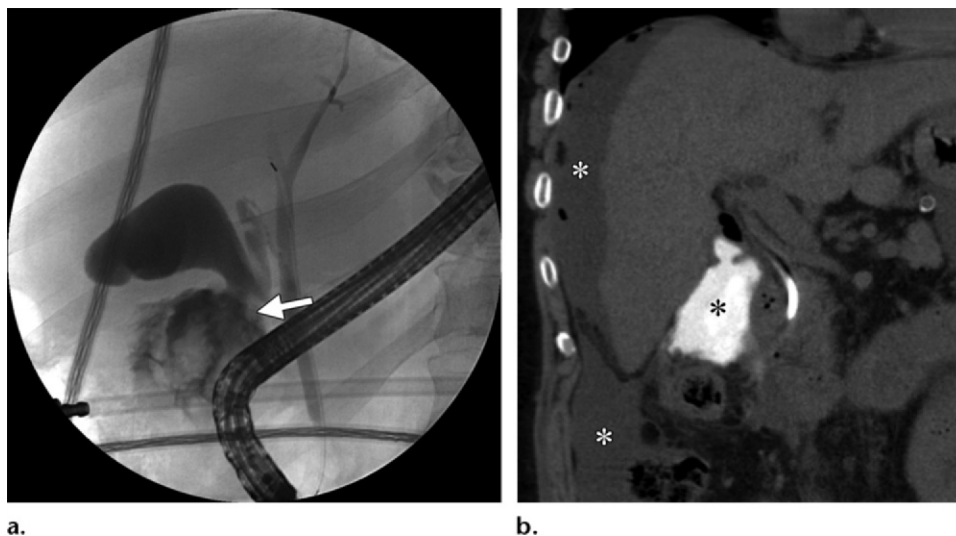
**Figure 4.** Hemorrhagic cholecystitis due to gallbladder necrosis after hepatic artery embolization for pseudoaneurysm in a 78-year-old woman. Axial nonenhanced CT image shows high-attenuation hemorrhage (\*) in the gallbladder and inflammation in the pericholecystic fat (arrows).

cases of gangrenous cholecystitis than in uncomplicated acute cholecystitis (19). As in treatment of other complicated forms of acute cholecystitis, temporary percutaneous cholecystostomy with antibiotic therapy is an acceptable treatment in patients with a high surgical risk.

### Hemorrhagic Cholecystitis

Hemorrhagic cholecystitis is a rare complication of acute calculous or acalculous cholecystitis and may be caused by trauma, anticoagulation, malignancy, or iatrogenic or other causes (20,21). Pathologically, hemorrhage may be caused by mural inflammation and subsequent mucosal infarction and erosion (21). Intraluminal hemorrhage may accumulate in the cystic duct and cause obstruction with subsequent inflammatory changes typical of acute calculous cholecystitis. Clinical symptoms of hemorrhagic and uncomplicated acute cholecystitis may be identical. Hemobilia caused by hemorrhagic cholecystitis may manifest as hematemesis or melena, uncommon findings not seen in uncomplicated acute cholecystitis (22).

CT findings include hyperattenuating bile with possible fluid-fluid level as well as typical findings of acute cholecystitis (Fig 4). Active contrast agent extravasation, a finding indicative of active bleeding, may be seen at arterial phase CT (22). Hemorrhage in the gallbladder should be differentiated from other causes of hyperattenuating bile, such as vicarious excretion from prior intravenous contrast agent administration, biliary sludge or layering gallstones, and milk of calcium bile. Hemorrhagic cholecystitis with gallbladder perforation may be



**Figure 5.** Gallbladder perforation in a 56-year-old man who fell down several stairs. **(a)** Endoscopic retrograde cholangiopancreatogram (ERCP) shows active contrast agent extravasation (arrow) from the inferior aspect of the gallbladder. **(b)** Coronal nonenhanced CT image after ERCP shows an irregularly shaped contrast agent collection in the gallbladder fossa (black \*), a finding compatible with contrast agent extravasation from a perforated gallbladder. The large amount of ascites with foci of air (white \*) is compatible with a biliary leak.

complicated by hemoperitoneum and hemodynamic instability. Urgent cholecystectomy is necessary because of the high morbidity and mortality associated with gallbladder perforation.

### Gallbladder Perforation

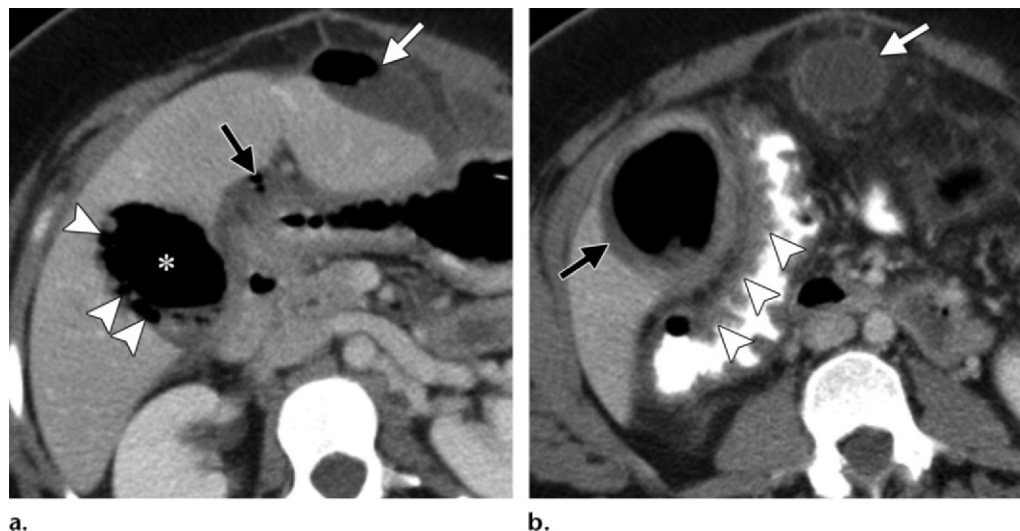
Gallbladder perforation is one of the most severe complications of acute cholecystitis, with mortality rates of up to 15% (23). Emphysematous, gangrenous, and hemorrhagic cholecystitis may progress to gallbladder perforation. Diagnosis is often difficult because the clinical symptoms of gallbladder perforation and variants of acute cholecystitis overlap. The high morbidity and mortality are attributed to delayed diagnosis and subsequent late surgical intervention. Gallbladder perforation is more common in men than in women and occurs at an average age of 60 years (24).

Three types of gallbladder perforation have been described: type I, or acute, refers to free

perforation and generalized peritonitis; type II, or subacute, refers to pericholecystic abscess formation and localized peritonitis; and type III, or chronic, refers to the formation of a cholecysto-enteric fistula (25).

The most common mechanism of gallbladder perforation involves cystic duct obstruction leading to gallbladder distention, vascular compromise, ischemia, necrosis, and ultimately perforation (26). Gallbladder perforation in the absence of gallbladder distention is likely related to infection of the Rokitansky-Aschoff sinuses with subsequent necrosis and rupture (27). Because of its poor blood supply, the fundus of the gallbladder is the most common site of perforation. Acute gallbladder perforation may also result from trauma (Fig 5).

An extraluminal gallstone is a finding specific for gallbladder perforation. Although it was originally described for US, the “hole sign,” which refers to a focal gallbladder defect, is more commonly seen at CT and is a reliable sign of gallbladder perforation (28). Complications include



**Figure 6.** Emphysematous cholecystitis with gallbladder perforation in a 36-year-old woman. **(a)** Axial contrast-enhanced CT image shows a gas-distended gallbladder lumen (\*) with multiple foci of air in the gallbladder wall (arrowheads). Air and fluid in the porta hepatis (black arrow) and a rim-enhancing air-containing abscess (white arrow) anterior to the left lobe of the liver indicate perforation. **(b)** Axial contrast-enhanced CT image at a more caudal level shows circumferential gallbladder wall thickening (black arrow) and pericholecystic inflammatory changes with reactive mural thickening of the ascending and transverse colon (arrowheads). An additional rim-enhancing abscess (white arrow) with surrounding inflammatory change is also seen.

free intraperitoneal air; bile leak; abscess formation in the liver, gallbladder fossa, or peritoneum; and small bowel obstruction (Fig 6).

Treatment of gallbladder perforation includes cholecystectomy, abscess drainage, peritoneal lavage, and antibiotic therapy. Type III gallbladder perforation may require additional surgery to close the cholecystoenteric fistula. Cholecystostomy and antibiotic therapy may be used if cholecystectomy is not possible because of the patient's high surgical risk.

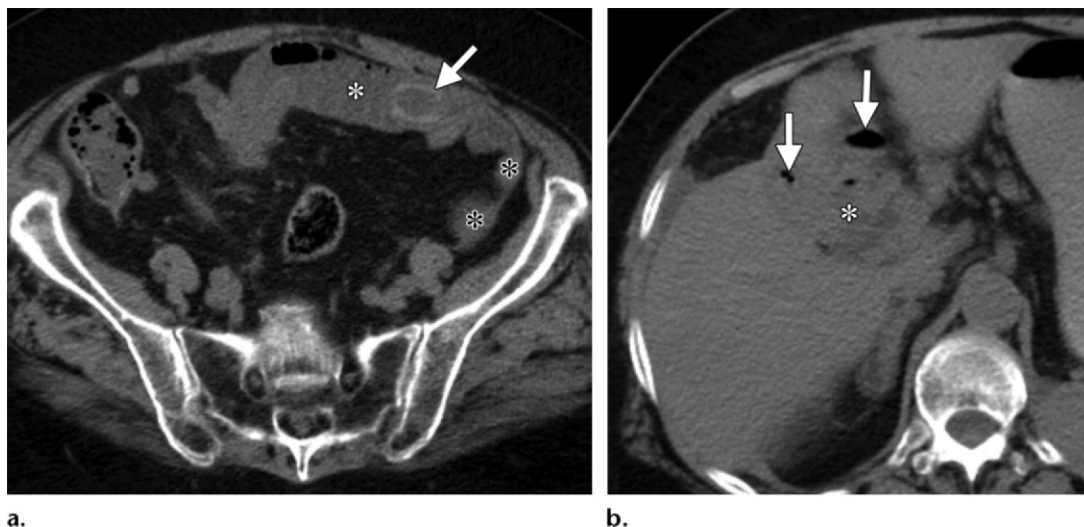
### Gallstone Ileus

Gallstone ileus is a rare complication of cholelithiasis and an infrequent cause of mechanical bowel obstruction, accounting for up to 3% of cases (29). Gallstone ileus predominantly occurs in women and has a mortality rate of up to 18% (30), presumably because of comorbidities in the elderly population. Gallstone ileus usually occurs in patients with subacute or chronic cholecysti-

tis. Long-standing obstruction leads to gallstone erosion into the small bowel, usually through a cholecystoenteric fistula (31). Gallstone ileus in the absence of a fistula may occur after passage of a large stone after endoscopic sphincterotomy or cholecystectomy (32).

Most obstructive gallstones are at least 2.5 cm in diameter and are located distally in the ileum and jejunum, with frequency rates of approximately 60% and 26%, respectively (29,32). The terminal ileum is the narrowest segment of the small bowel and thus is the most likely site of obstruction. Bouveret syndrome describes gastric outlet obstruction secondary to an impacted gallstone in the duodenum or pylorus. In a series of 27 cases of gallstone ileus proved at surgery, the Rigler triad of pneumobilia, bowel obstruction, and a gallstone in the lower abdomen (Fig 7) was observed in approximately 15% of radiographs

Teaching  
Point



**Figure 7.** Gallstone ileus in an 81-year-old woman. **(a)** Axial nonenhanced CT image shows dilated loops of small bowel (white \*) to the level of an obstructive gallstone (arrow) in the distal jejunum. Small bowel loops distal to the gallstone are collapsed (black \*). **(b)** Axial nonenhanced CT image centered on the porta hepatis shows a collapsed gallbladder (\*) containing foci of air (arrows), a finding that likely represents fistulous communication between the gallbladder and duodenum.

and 78% of CT images (29). CT is helpful in determining the multiplicity and location of ectopic gallstones and in detecting them before an obstruction develops (32).

Treatment is based on the patient's surgical risk and consists of a one- or two-stage procedure. A one-stage procedure for patients with low operative risk typically consists of enterolithotomy, cholecystectomy, and fistula repair. A two-stage procedure for patients with high operative risk includes enterolithotomy with later elective biliary surgery (33).

### Mirizzi Syndrome

Mirizzi syndrome describes an obstruction of the common hepatic duct due to extrinsic compression by an impacted cystic duct stone. Patients present with symptoms of obstructive jaundice, including fever, right upper quadrant pain, and jaundice. Repeated bouts of recurrent cystic duct stone impaction and inflammation may lead to a cholecystobiliary fistula (34).

CT findings of Mirizzi syndrome include an impacted gallstone in the gallbladder neck with proximal dilatation of the common hepatic duct and an abrupt change to normal caliber in the common bile duct below the gallstone (Fig 8) (35). MRCP may further delineate the extent of gallbladder inflammation. ERCP can be used for stone extraction, lithotripsy, and biliary stent

placement; to delineate the extent of obstruction; and to identify ductal anomalies, such as a low-lying cystic duct, before surgery (36).

Open surgery is the primary treatment of Mirizzi syndrome. Laparoscopic management is controversial and is associated with higher morbidity and mortality. At laparoscopy, distortion of the normal porta hepatis anatomy and landmarks caused by adhesions and inflammation increases the risk of biliary injury (36).

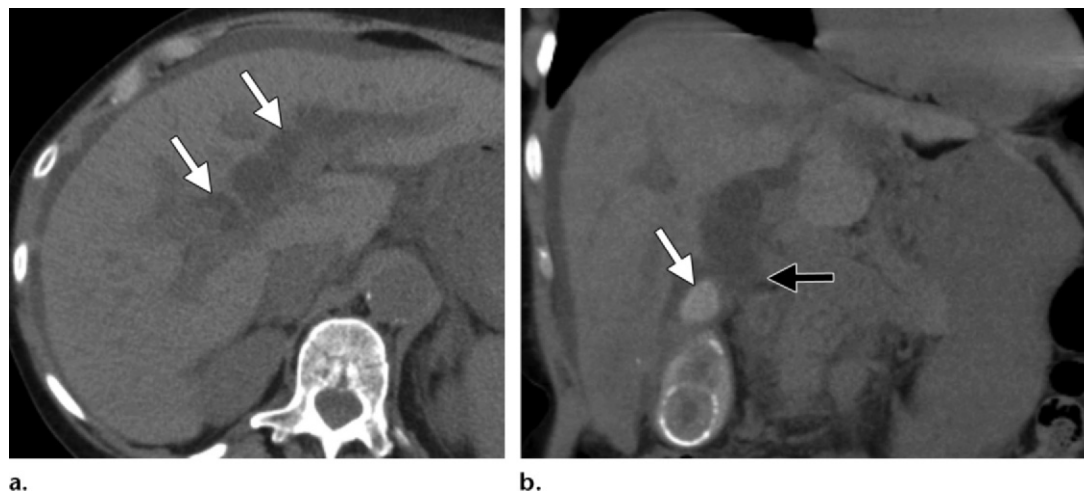
## Biliary Obstruction

### Choledocholithiasis

Choledocholithiasis refers to gallstones located in the common bile duct. Most gallstones pass from the gallbladder rather than forming *de novo* in the common bile duct. Although its exact incidence and prevalence are unknown, choledocholithiasis is found in up to 12% of patients at laparoscopic cholecystectomy (37). Most gallstones are asymptomatic unless they cause obstruction, which occurs typically at the ampulla of Vater. The most frequent complications of choledocholithiasis are acute cholangitis and gallstone pancreatitis.

CT is not the preferred imaging modality for detection of choledocholithiasis, but it often is ordered in the emergency department for patients who present with nonspecific abdominal complaints. The sensitivity of CT for detecting





**Figure 8.** Mirizzi syndrome in an 80-year-old woman with right upper quadrant pain. **(a)** Axial non-enhanced CT image shows intrahepatic biliary ductal dilatation (arrows). **(b)** Coronal nonenhanced CT image shows several hyperattenuating gallstones and an impacted stone (white arrow) in the gallbladder neck. Dilatation of the common hepatic duct (black arrow) is seen proximal to the stone. The common bile duct distal to the stone is normal in caliber.



**Figure 9.** Choledocholithiasis in a 61-year-old woman with right upper quadrant pain. Coronal contrast-enhanced CT image shows several hyperattenuating gallstones (white arrows) in a dilated common bile duct. A gallstone (black arrow) is seen in the gallbladder.

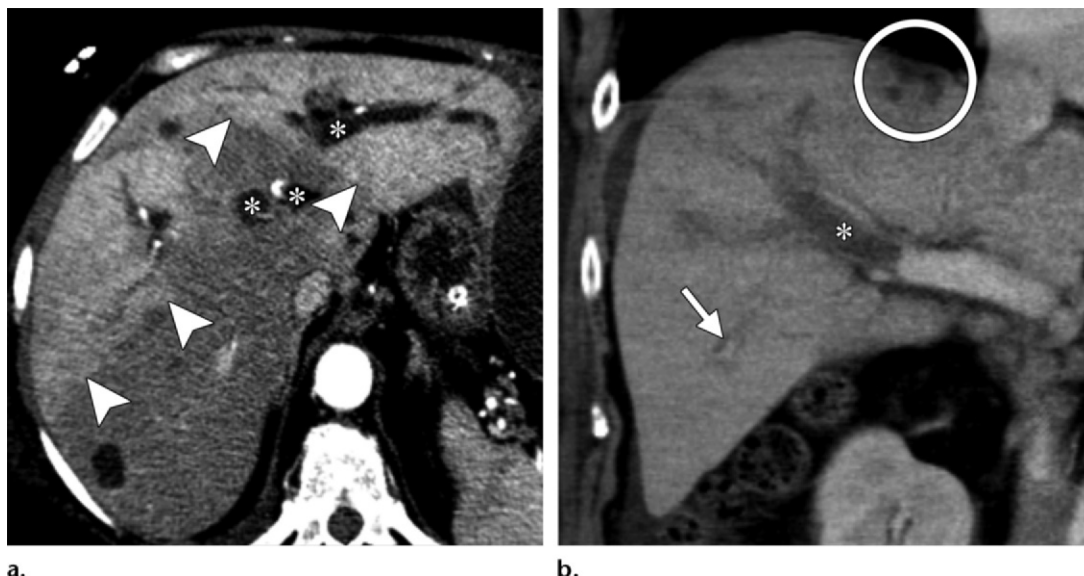
choledocholithiasis varies between 25% and 90% and is inherently limited because up to 24% of gallstones are isoattenuating to the surrounding bile (3,7,38–41). When visible on CT images, a biliary stone often appears angulated and geometric and is located dependently in the duct, with a rim of bile seen along the stone's anterior margin (42). Use of narrow window settings to

accentuate the attenuation difference between the stone and adjacent bile or soft tissue can improve detection (39). Use of coronal reconstructions can also help detect choledocholithiasis (Fig 9). MRCP is helpful in equivocal cases. Symptomatic choledocholithiasis is most commonly treated with ERCP and sphincterotomy to facilitate stone passage and extraction.

### Acute Cholangitis

Acute cholangitis is caused by bacterial overgrowth from biliary stasis and obstruction and manifests with abdominal pain, fever, and jaundice. Procedurally related cholangitis occurs in approximately 18% of transhepatic percutaneous biliary drainage catheter placements (43). Acute suppurative cholangitis refers to pus in the biliary tract, a condition that can lead to increased intraluminal pressure and precipitate biliary sepsis. Independent risk factors for this severe form of acute cholangitis include patient age older than 70 years, current smoker status, and an impacted biliary stone (44).

The most common bacteria isolated in infected bile without prior instrumentation are *E coli* (31%), *Klebsiella pneumoniae* (17%), *Enterococcus faecalis* (17%), and *Streptococcus* species (17%) (45). A separate study reported that *Enterococcus* and polymicrobial infections are found more commonly in the bile cultures of patients with plastic biliary stents (46).



**Figure 10.** Acute cholangitis in two patients. **(a)** Axial contrast-enhanced arterial phase CT image in a 62-year-old woman shows intrahepatic biliary ductal dilatation (\*) and wedge-shaped hepatic parenchymal enhancement (arrowheads). **(b)** Axial contrast-enhanced CT image in an 86-year-old man with pancreatic cancer and a biliary stent shows portal vein thrombosis (\*), intrahepatic biliary ductal dilatation (arrow), and an abscess in the dome of the liver (circle).

The most common CT finding of acute cholangitis is biliary obstruction. The extrahepatic biliary ducts may show diffuse and concentric thickening, often with enhancement (47,48). Purulent bile may have increased CT attenuation. Arterial phase CT may show nodular, patchy, wedge-shaped, or geographic inhomogeneous hepatic parenchymal enhancement (Fig 10a), a finding that is reversible with treatment (49). A recently described CT scoring system based on the extent of transient hepatic attenuation differences, degree of biliary dilatation, and presence of an obstructive lesion is highly sensitive (84%–90%) and specific (84%) for the diagnosis of acute cholangitis (50). Complications of acute cholangitis include pyogenic hepatic abscess, portal vein thrombosis, and biliary peritonitis (Fig 10b).

Treatment of acute cholangitis includes antibiotic therapy and biliary drainage (51). Most cases resolve with use of a broad-spectrum antibiotic that is modified after a specific organism is cultured. If the clinical course worsens, biliary decompression can be achieved with ERCP or

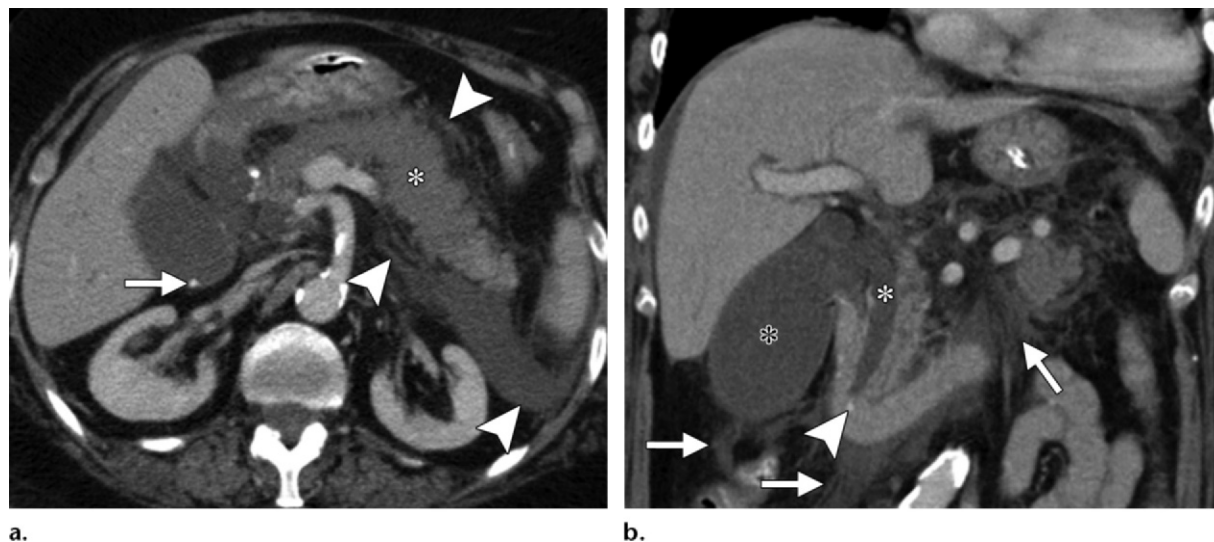
transhepatic percutaneous drainage; surgical decompression with a large-bore T tube is used in exceptional cases.

### Biliary Pancreatitis

Gallstone or biliary sludge impaction at the ampulla of Vater may cause ampullary spasm, pancreaticobiliary reflux, and obstruction of the common and pancreatic ducts leading to acute biliary pancreatitis and cholangitis. Anatomic variations such as a common pancreaticobiliary channel or pancreas divisum also raise the risk of acute pancreatitis.

Findings at contrast-enhanced CT include an obstructive stone in a dilated common bile duct associated with an edematous hypoattenuating pancreas with surrounding peripancreatic inflammation and fluid (Fig 11). Severe cases of pancreatitis may include findings of pseudocyst formation and parenchymal necrosis.

Treatment of biliary pancreatitis depends on associated complications such as biliary stasis and acute cholangitis. Biliary pancreatitis with associated acute cholangitis requires antibiotic treatment and removal of the obstructive stone



**Figure 11.** Gallstone pancreatitis in a 64-year-old woman. **(a)** Axial contrast-enhanced CT image shows an enlarged edematous pancreas (\*) with inflammation of the peripancreatic fat and a large amount of fluid tracking to the left anterior pararenal space (arrowheads). A gallstone is seen in the dependent aspect of the gallbladder (arrow). **(b)** Coronal contrast-enhanced CT image shows an obstructive gallstone in the distal common bile duct (arrowhead) with proximal intra- and extrahepatic biliary ductal dilatation (white \*) and a hydropic gallbladder (black \*). Fluid and inflammation are seen in the peripancreatic fat and right anterior pararenal space (arrows).

with early ERCP and sphincterotomy within 72 hours (52). Uncomplicated biliary pancreatitis can be treated conservatively without ERCP (53). Cholecystectomy is recommended within 2 weeks of resolution of gallstone pancreatitis to reduce the risk of recurrent gallstone-related complications.

### Pyogenic Hepatic Abscess

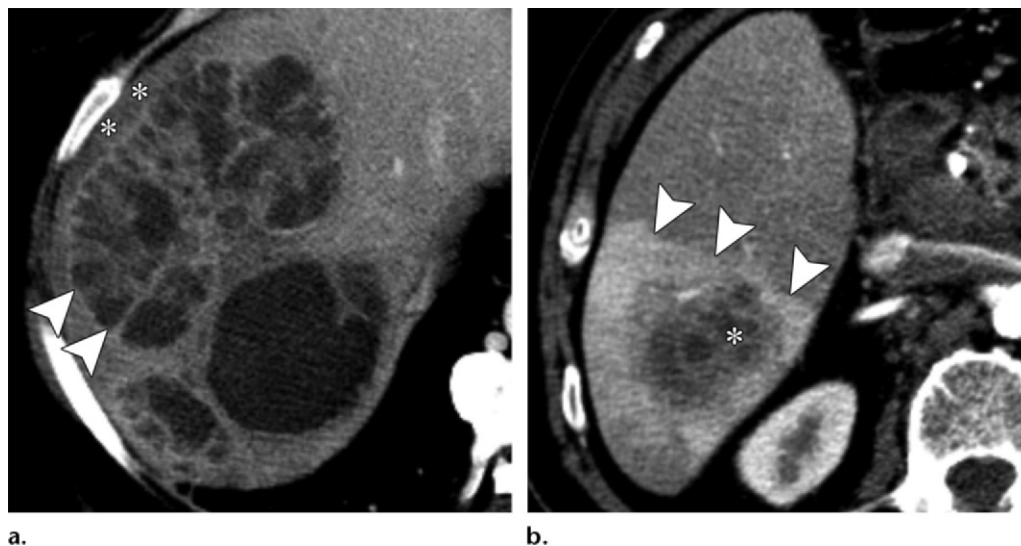
Pyogenic hepatic abscess typically manifests as fever and right-sided abdominal pain; other common symptoms include nausea, vomiting, weight loss, and malaise. The epidemiology, treatment, and mortality rates for pyogenic liver abscess have changed significantly over the past century. Mortality has decreased to 2%–10% from the 11%–31% reported in earlier studies (54,55). The mean patient age is approximately 56–64 years, and the risk of developing a pyogenic liver abscess is higher in men than in women.

Causes of pyogenic hepatic abscess include benign or malignant biliary obstruction, previous biliary instrumentation or stent placement, portal pyemia from peritonitis, and hematogenous spread from sepsis. The incidence of pyogenic

abscess due to appendicitis and diverticulitis has significantly decreased because of early detection and treatment of the primary process.

Although the potential etiology is diverse, many pyogenic hepatic abscesses are idiopathic (55). Approximately 1.4% of radiofrequency ablations and hepatic artery embolizations for primary and metastatic hepatic lesions are complicated by pyogenic liver abscess (56,57). The risk of abscess is substantially increased in patients with a prior bilioenteric anastomosis or an incompetent sphincter of Oddi caused by sphincterotomy or stent placement (56).

CT has more than 95% sensitivity for detection of hepatic abscess (58). Hepatic abscess tends to localize in the right lobe of the liver and is classified by size: a macroabscess is more than 2 cm in diameter and a microabscess is less than 2 cm in diameter. The cluster sign refers to multiple microabscesses in geographic proximity and likely represents an early stage of abscess evolution (59). Contrast-enhanced CT may show a single or multilocular hypoattenuating mass surrounded by a



**Figure 12.** Pyogenic hepatic abscess in two patients. **(a)** Axial contrast-enhanced CT image in a 59-year-old man shows multiple abscesses in various stages of maturity in the right lobe of the liver. A multilocular abscess cavity in the anterior segment shows the double target sign, with peripheral rim enhancement (arrowheads) surrounded by an outer hypoattenuating zone (\*). **(b)** Axial contrast-enhanced arterial phase CT image in an 84-year-old man shows a multiseptated hypoattenuating mass (\*) consistent with a hepatic abscess, with surrounding wedge-shaped parenchymal enhancement (arrowheads).

single enhancing rim (single target sign) or by an inner enhancing rim with an outer hypoattenuating zone (double target sign) (Fig 12a) (60,61).

Contrast-enhanced arterial phase CT may show transient segmental or wedge-shaped enhancement surrounding the hepatic abscess (Fig 12b), a finding caused by reduced portal venous flow due to inflammation and stenosis with a compensatory increase in hepatic arterial flow (60). Similar to transient arterial parenchymal enhancement in acute cholangitis, segmental or wedge-shaped enhancement with hepatic abscess is reversible with treatment that reduces periportal inflammation and restores portal venous flow (60). Pyogenic abscesses due to *K pneumoniae* have a higher association with thrombophlebitis of the hepatic veins and septic hematogenous complications (62).

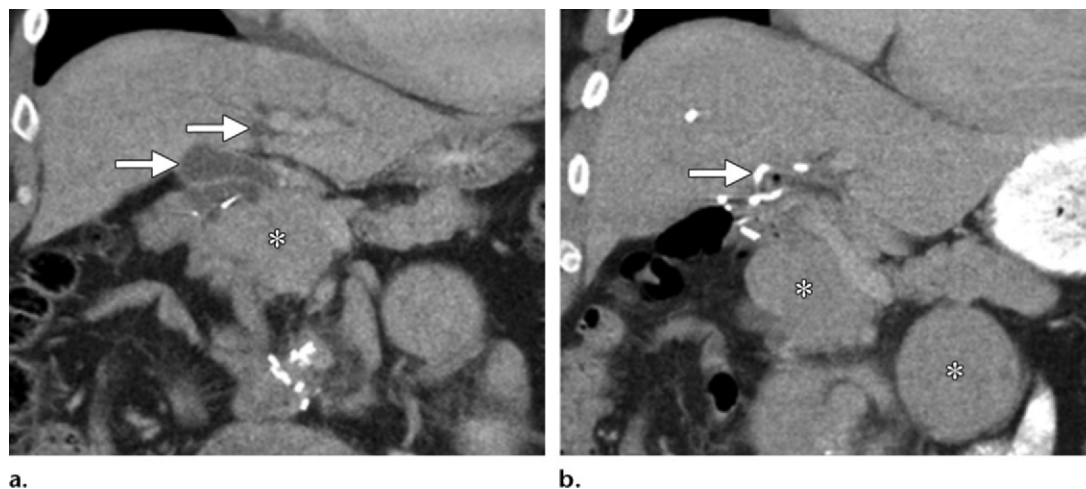
Treatment of pyogenic liver abscess includes antibiotic therapy and drainage. In a series of patients with a single pyogenic liver abscess and without coexisting biliary malignancy, percutaneous catheter drainage and percutaneous needle aspiration were equally effective for abscesses less than 5 cm in diameter, while percutaneous catheter drainage was more effective for multilocular

abscesses and abscesses more than 5 cm in diameter (63). Successful percutaneous drainage is possible with multiple abscesses regardless of their size and multilocularity (64). However, one study suggests that the lower failure rate of surgical drainage makes it preferable to percutaneous drainage in pyogenic hepatic abscesses larger than 5 cm in diameter, although there was no reported difference in morbidity or mortality (65).

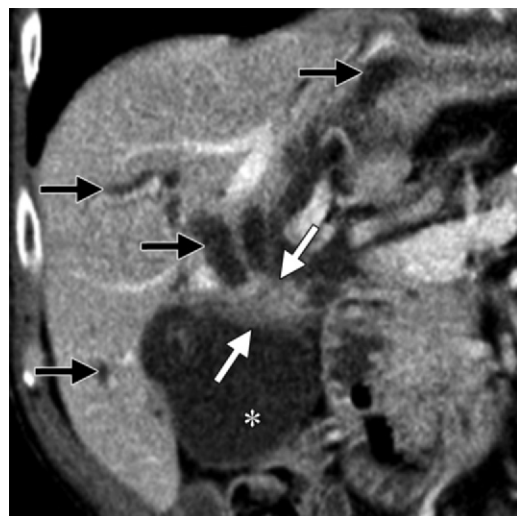
### Malignancy

Biliary obstruction from malignancy may manifest as right upper quadrant abdominal pain, fever, pruritis, dark urine, and clay-colored stools. Biliary obstruction may result from malignant tumors in the pancreas, duodenum, ampulla of Vater, gallbladder, or bile ducts or from the extrinsic compression caused by metastatic disease or lymphadenopathy. The pattern of biliary dilatation depends on the cause and location of the obstruction. A pancreatic head or ampullary carcinoma causes distal obstruction of the common bile duct and results in extra- and intrahepatic biliary dilatation. Peripheral cholangiocarcinoma causes segmental intrahepatic biliary dilatation. Lymphadenopathy in the porta hepatis may cause biliary dilatation at the confluence of the right and left intrahepatic ducts (Fig 13), mimicking a





**Figure 13.** Metastatic renal cell carcinoma in a 41-year-old man. **(a)** Coronal contrast-enhanced CT image shows extra- and intrahepatic biliary ductal dilatation (arrows) due to extrinsic compression of the common bile duct by metastatic lymphadenopathy in the porta hepatis (\*). **(b)** Coronal contrast-enhanced CT image after biliary catheter drainage (arrow) shows resolution of the extra- and intrahepatic biliary ductal dilatation. Metastatic porta hepatic and retroperitoneal lymphadenopathy remains (\*).



**Figure 14.** Common bile duct cholangiocarcinoma in a 72-year-old man. Coronal contrast-enhanced CT image shows an enhancing soft tissue lesion (white arrows) in the common bile duct associated with extra- and intrahepatic biliary ductal dilatation (black arrows). The gallbladder (\*) is distended because the cystic duct insertion is proximal to the obstructive common bile duct lesion.

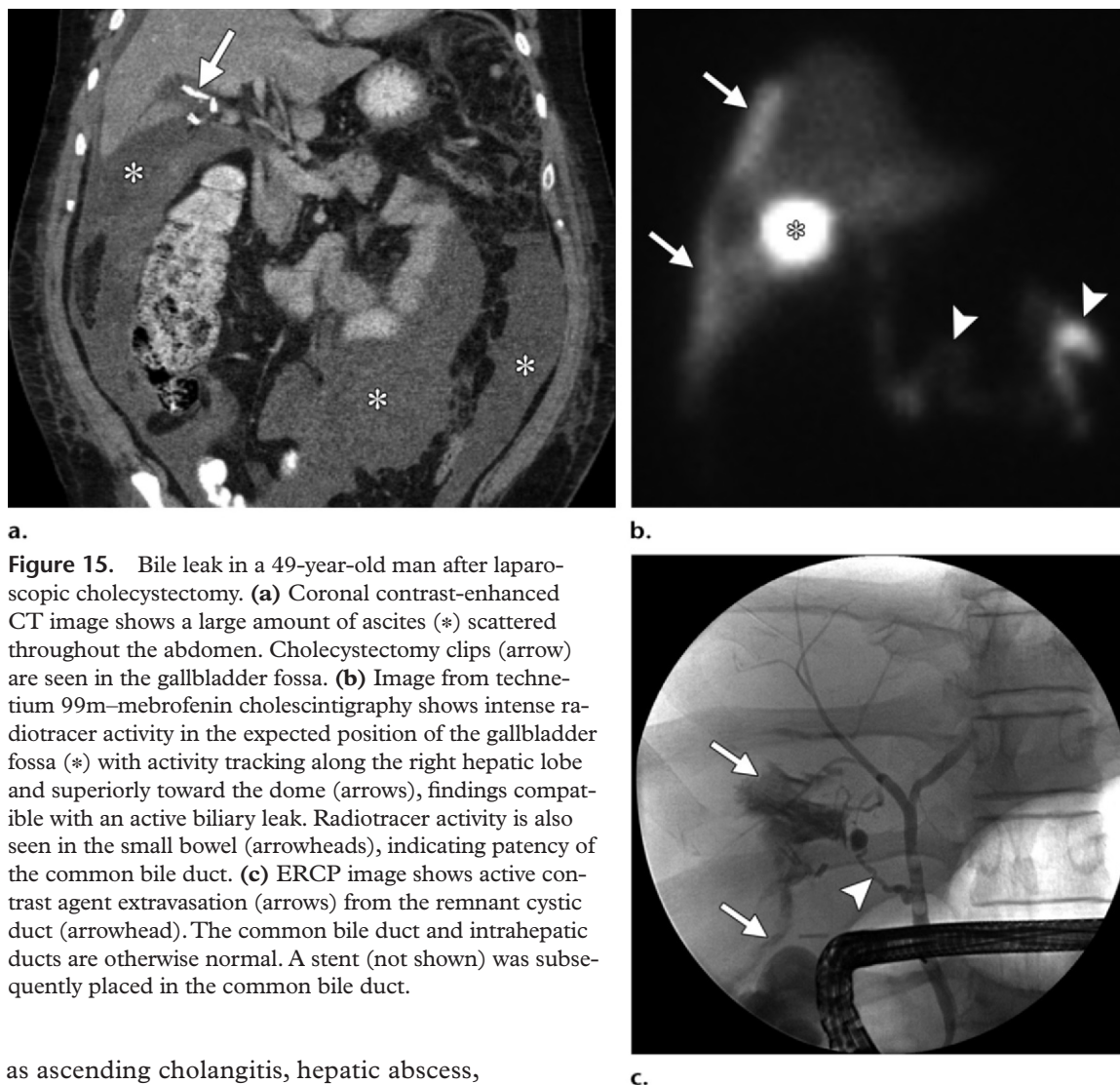
hilar, or Klatskin, cholangiocarcinoma. CT can help locate the obstruction and determine the organ of origin of the malignant neoplasm.

Pancreatic adenocarcinoma occurs most often in the pancreatic head and typically appears as a hypoattenuating mass with decreased enhance-

ment relative to the surrounding pancreatic parenchyma. The tumor manifests with abrupt obstruction of the common bile and pancreatic ducts and upstream pancreatic parenchymal atrophy. Small pancreatic masses are difficult to visualize at CT, and the only hint for detection may be loss of the normal lobulated pancreatic contour or of the typical pointed configuration of the uncinate process.

Intrahepatic cholangiocarcinoma (peripheral and hilar) is the second most common primary hepatic tumor after hepatocellular carcinoma, and extrahepatic cholangiocarcinoma is the second most common biliary tumor after gallbladder carcinoma. Peripheral cholangiocarcinoma typically manifests as a large hepatic mass with peripheral rim enhancement and adjacent intrahepatic biliary ductal dilatation; delayed phase images show persistent and progressive contrast enhancement due to fibrous stroma (66). Hilar or extrahepatic cholangiocarcinoma typically manifests as a focally thickened, enhancing biliary duct with narrowing and obliteration of the biliary ducts (Fig 14) (66).

Surgical resection often is the only curative treatment but is possible in a minority of cases. Palliative treatment relieves obstruction when the disease is unresectable or metastatic. Prolonged biliary stasis from malignant biliary obstruction may cause acute complications



**Figure 15.** Bile leak in a 49-year-old man after laparoscopic cholecystectomy. **(a)** Coronal contrast-enhanced CT image shows a large amount of ascites (\*) scattered throughout the abdomen. Cholecystectomy clips (arrow) are seen in the gallbladder fossa. **(b)** Image from technetium 99m-mebrofenin cholescintigraphy shows intense radiotracer activity in the expected position of the gallbladder fossa (\*) with activity tracking along the right hepatic lobe and superiorly toward the dome (arrows), findings compatible with an active biliary leak. Radiotracer activity is also seen in the small bowel (arrowheads), indicating patency of the common bile duct. **(c)** ERCP image shows active contrast agent extravasation (arrows) from the remnant cystic duct (arrowhead). The common bile duct and intrahepatic ducts are otherwise normal. A stent (not shown) was subsequently placed in the common bile duct.

such as ascending cholangitis, hepatic abscess, and portal vein thrombosis. Distal common bile duct obstruction due to pancreatic carcinoma, extrahepatic cholangiocarcinoma, or metastatic lymph nodes is best treated with endoscopic metal stents, which offer high patency rates compared with endoscopic plastic stents or surgical bypass (67). Hilar bile duct obstruction due to Klatskin tumor may also be treated with endoscopic stents, but percutaneous transhepatic biliary drainage has higher technical and therapeutic success rates (68). Treatment preferences vary among institutions based on local expertise.

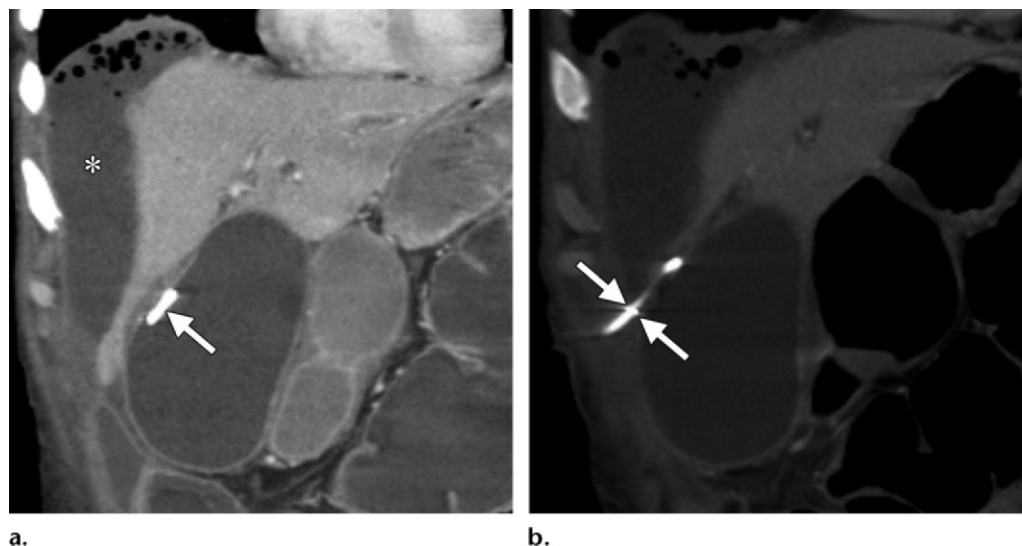
## Iatrogenic Biliary Tract Complications

### Bile Leak

Bile leaks may occur after orthotopic liver transplantation, pancreaticoduodenectomy, hepatic or biliary surgery, and cholecystectomy. A clinically symptomatic bile leak after normal find-

ings on an intraoperative cholangiogram is more common with laparoscopic cholecystectomy than with open cholecystectomy and occurs in up to 2% of patients (69). The leak appears during the first postoperative week and manifests as abdominal pain, fever, nausea, or vomiting. Subclinical or asymptomatic bile leaks after laparoscopic cholecystectomy have a prevalence of 7% (70).

Leaks typically occur from the cystic duct stump or the ducts of Luschka, also known as the subvesical or accessory biliary ducts. These ducts usually originate in the right hepatic lobe, course along the gallbladder fossa, drain variably into intra- or extrahepatic biliary ducts, and may be injured during surgical dissection or cauterization (71). The severity of bile extravasation is variable and depends on individual anatomic features. Ducts of Luschka that communicate with the extrahepatic biliary duct tend to re-



**Figure 16.** Migrated cholecystostomy tube in a 74-year-old man with acute cholecystitis. **(a)** Coronal contrast-enhanced CT image shows a loculated air-containing perihepatic fluid collection (\*). A cholecystostomy tube (arrow) is seen in the hydropic gallbladder. **(b)** Coronal contrast-enhanced CT image with wide window width and high level settings shows the radiopaque marker (arrows) of the cholecystostomy tube outside the gallbladder and in the peritoneum. The radiopaque marker denotes the position of the most proximal drainage side hole as outside the gallbladder, allowing bile spillage into the peritoneal cavity.

sult in larger volumes of bile extravasation than those that do not communicate with the central biliary system.

Postoperative fluid collections may be visualized at CT and typically are nonspecific findings because the origin of the fluid cannot be determined (Fig 15a). Cholescintigraphy can help determine the biliary origin of the fluid collection and the approximate rate of leakage (Fig 15b).

Treatment is based on the cause of the bile leak and the clinical course. Laparoscopic cholecystectomy complicated by a duct of Luschka leak requires ERCP (Fig 15c) with placement of a nasobiliary drain or an internal stent to restore normal biliary drainage and divert flow from the leakage site (72). A biliary leak from a hepaticojejunostomy or from the cut surface of the liver after liver transplantation can be successfully treated with percutaneous transhepatic biliary drainage (73). Large or inaccessible leaks may require surgical management.

A biloma, or encapsulated extrabiliary bile collection, may form as a consequence of biliary surgery, ERCP, laparoscopic cholecystectomy, radiofrequency ablation, transcatheter arterial chemoembolization, or percutaneous biliary drainage.

### Biliary Catheter or Stent Malfunction

Indications for percutaneous transhepatic biliary catheter drainage vary among institutions based on departmental expertise. Typical indications

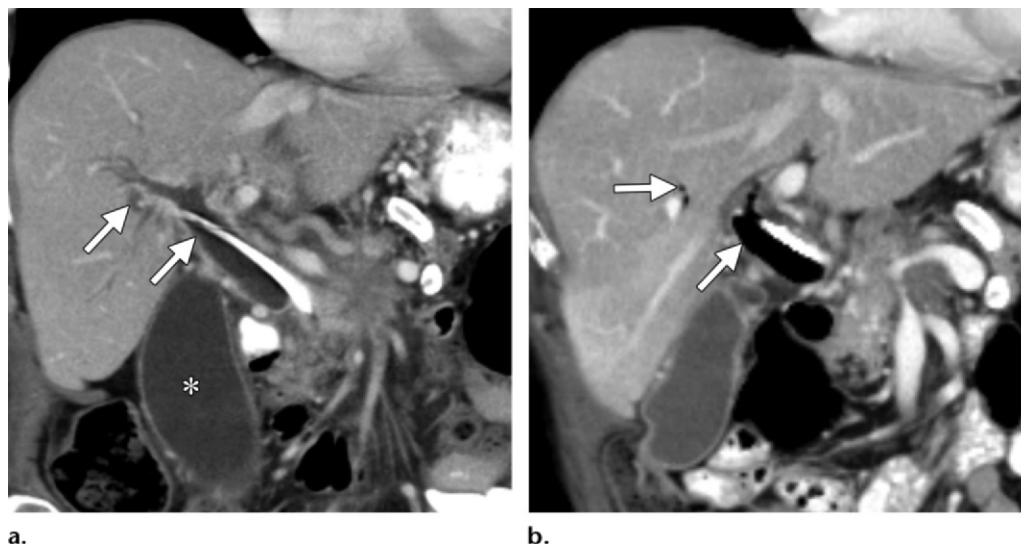
include failed endoscopic drainage, hepaticojejunostomy anastomotic leaks, or hilar obstruction.

A percutaneous transhepatic biliary catheter contains a radiopaque marker that designates the most proximal end of the catheter drainage side holes. The radiopaque marker should be placed above the site of obstruction but within the biliary system for effective biliary drainage. A misplaced or migrated catheter with side holes outside the biliary system but within the subcutaneous tract, peritoneum, or pleural space can lead to a biliary leak (Fig 16a), perihepatic biloma formation, a choledochal cyst, or bleeding if a catheter side hole traverses a hepatic vessel. Percutaneous transhepatic biliary catheters also migrate with respiration and require correct fixation to the skin to prevent abnormal side-hole movement and the development of a biliary leak or bleeding.

Streak artifact from the radiopaque marker of a percutaneous transhepatic biliary catheter at typical window width and level settings at abdominal CT limits precise localization of the marker, and a migrated or misplaced catheter may not be visualized at CT. Use of higher window width and level settings reduces the streak artifact from the radiopaque marker and allows more precise scrutiny of catheter position and increased sensitivity for detecting catheter malposition and potential complications (Fig 16b).

**Teaching Point**





**Figure 17.** Occluded biliary stent in a 74-year-old woman with pancreatic cancer. **(a)** Coronal contrast-enhanced CT image shows a malfunctioning common bile duct stent with intra- and extrahepatic biliary ductal dilatation (arrows) and a hydropic gallbladder (\*). No pneumobilia is seen. **(b)** Coronal contrast-enhanced CT image obtained 6 weeks earlier shows a normal-caliber biliary system, a normal gallbladder, and pneumobilia (arrows), findings suggestive of patency of the common bile duct stent.

Biliary stents can relieve obstruction and minimize the risk of cholangitis caused by primary pancreaticobiliary malignancy, metastatic disease, or compressive porta hepatis adenopathy. Stents may be occluded by biliary sludge or tumor ingrowth, leading to cholestasis, acute cholangitis, and possibly acute cholecystitis if the cystic duct becomes obstructed. CT findings include intra- or extrahepatic biliary ductal dilatation proximal to the stent, depending on whether the stent is distal or hilar. **Pneumobilia, a common finding in patients with patent biliary stents, may not be seen with an occluded stent because there is no communication with the duodenum** (Fig 17).

Stent migration occurs more commonly with plastic and covered metal stents than with uncovered metal stents because of decreased mobility from increased tissue ingrowth with the latter (74). Migrated stents typically pass through the rectum without complication, but impaction in the bowel may occasionally cause obstruction or rarely perforation.

### Postcholecystectomy Clip Migration

Postcholecystectomy clip migration is an uncommon late complication of cholecystectomy and refers to the migration of cholecystectomy clips into the biliary tree with resultant biliary obstruction. In a recent review of 69 case reports, the median patient age at presentation was 60 years and the median time of presenta-

tion after cholecystectomy was 26 months (75). Postulated mechanisms for postcholecystectomy clip migration include inadvertent clip placement in the biliary tree, clip slippage due to wound dehiscence, placement of too many clips, bile duct injuries from incorrect clip placement, and complicated surgical course (75). A cholecystectomy clip may serve as a nidus for stone formation, and clinical symptoms are no different than those of primary or secondary choledocholithiasis (75).

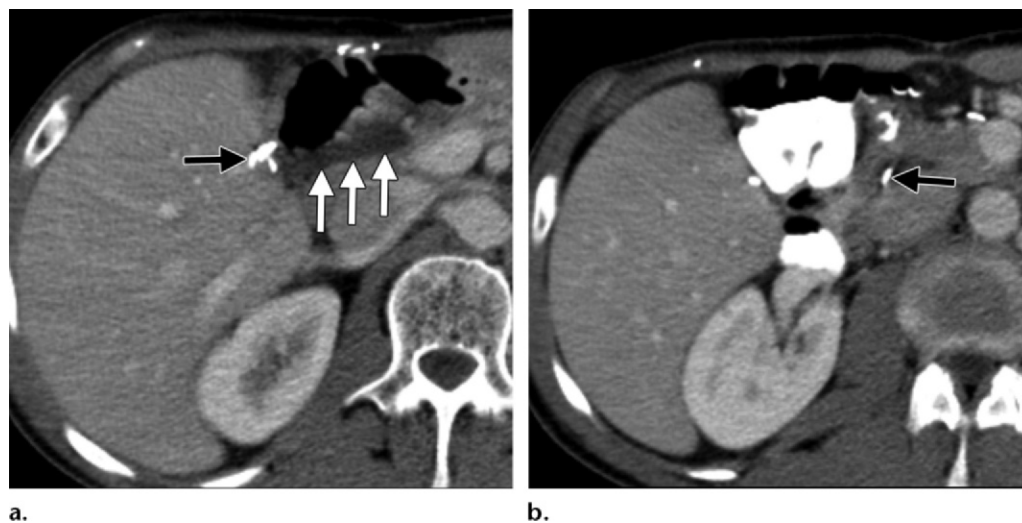
Imaging is required to distinguish postcholecystectomy clip migration from postcholecystectomy choledocholithiasis because of their similar clinical presentations. CT images may show a cholecystectomy clip, with or without stone formation, in a dilated common bile duct (Fig 18). ERCP is used to remove the migrated clip. Surgery and percutaneous biliary drainage are reserved for complicated cases.

## Uncommon Causes of Biliary Disease

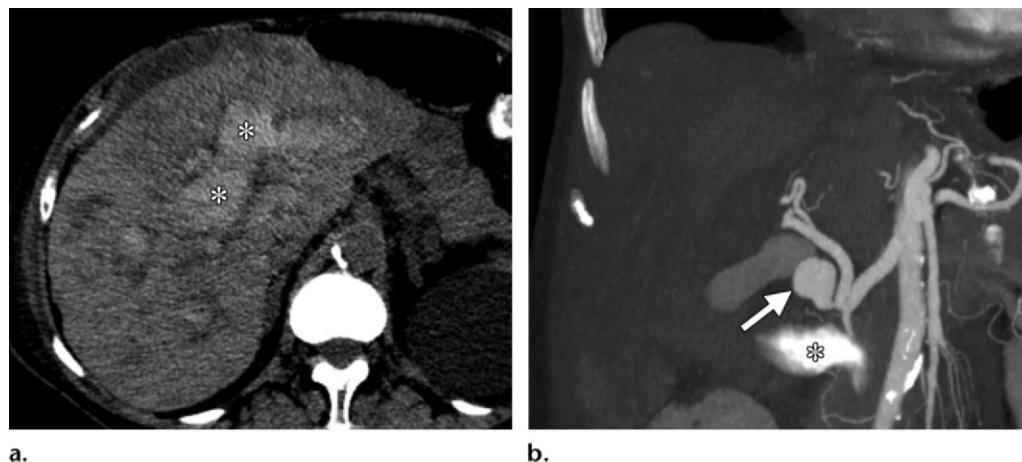
### Hemobilia

Hemobilia may manifest as right upper quadrant pain and jaundice; hematemesis or melena may occur if enough blood passes through the ampulla of Vater. High-volume hemobilia may precipitate anemia, hypotension, and shock. Low-volume hemobilia may result in biliary obstruction and ascending cholangitis. Possible causes include blunt or penetrating hepatic trauma, anticoagulation, and hepatic artery aneurysm or





**Figure 18.** Postcholecystectomy clip migration in a 57-year-old man. **(a)** Axial contrast-enhanced CT image shows a cholecystectomy clip (black arrow) in the gallbladder fossa and dilatation of the common bile duct (white arrows). **(b)** Axial contrast-enhanced CT image at a more caudal level shows a cholecystectomy clip in the distal common bile duct (arrow).



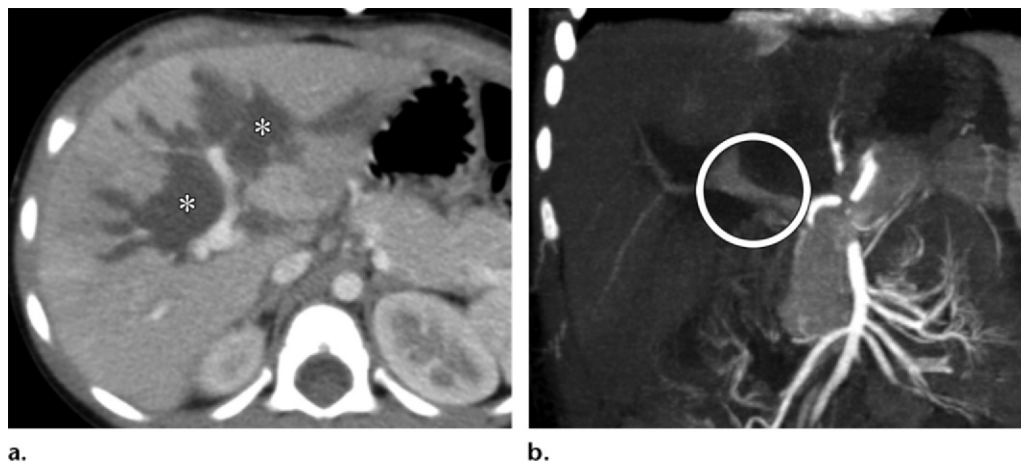
**Figure 19.** Hemobilia in a 72-year-old woman. **(a)** Axial nonenhanced CT image shows high-attenuation material compatible with acute hemorrhage (\*) in massively dilated intrahepatic biliary ducts. **(b)** Coronal arterial phase maximum intensity projection CT image shows a large tubular outpouching with a short, narrow neck (arrow), consistent with a pseudoaneurysm, arising from the proper hepatic artery just distal to the origin of the gastroduodenal artery. Attenuating contrast agent is seen in the dilated common bile duct (\*), a finding indicative of active extravasation.

pseudoaneurysm. The incidence of hemobilia has recently increased, likely because of iatrogenic factors related to the increased number of diagnostic and therapeutic interventional procedures such as radiofrequency ablation, liver biopsy (transjugular or percutaneous), sphincterotomy, endoscopic biliary stent placement, and percutaneous biliary drainage.

Hemobilia may complicate 4%–18% of percutaneous transhepatic biliary drainage catheter placements (43) and occurs when the catheter side holes traverse both the biliary and arterial or venous systems, allowing direct communication between the two. Patients who present with evi-

dence of hemorrhage in the catheter drainage bag typically are evaluated with interventional radiology without first undergoing cross-sectional imaging. Venous bleeding is treated by replacing the catheter with one of a larger diameter, a procedure that often tamponades the bleeding. Persistent bleeding after catheter upsizing indicates an origin at the hepatic artery and usually requires angiography and transarterial embolization.

Nonenhanced CT images may show high-attenuation layering material in the gallbladder or biliary tree (Fig 19a). The differential diagnosis



**Figure 20.** Early hepatic artery thrombosis in an 18-month-old girl 3 weeks after liver transplantation. **(a)** Axial contrast-enhanced CT image shows marked branched biliary ductal dilatation (\*) adjacent to the portal vein branches in the hepatic hilum, a finding compatible with biliary necrosis. **(b)** Coronal arterial phase maximum intensity projection CT image shows absence of the proper hepatic artery (circle) and lack of hepatic arterial vasculature, findings suggestive of hepatic artery thrombosis.

for high-attenuation bile includes vicarious excretion of intravenous iodinated contrast agent, retained contrast agent from cholangiography, biliary sludge, purulent bile, and malignancy. Arterial phase CT may show active extravasation into the biliary system if the cause is an aneurysm or pseudoaneurysm (Fig 19b).

### Biliary Necrosis

Biliary necrosis refers to destruction of the intrahepatic biliary duct epithelium and usually manifests as hepatic infarction caused by hepatic artery thrombosis. The intrahepatic biliary ducts are especially vulnerable to necrosis because of their exclusive vascular supply from the hepatic artery via the peribiliary plexus. Hepatic artery thrombosis is a serious complication of hepatic transplantation but also may result from atherosclerosis, hypercoagulable state, embolic disease, vasculitis, or traumatic laceration or iatrogenically from hepatic artery ligation during cholecystectomy or occlusion after transarterial chemoembolization.

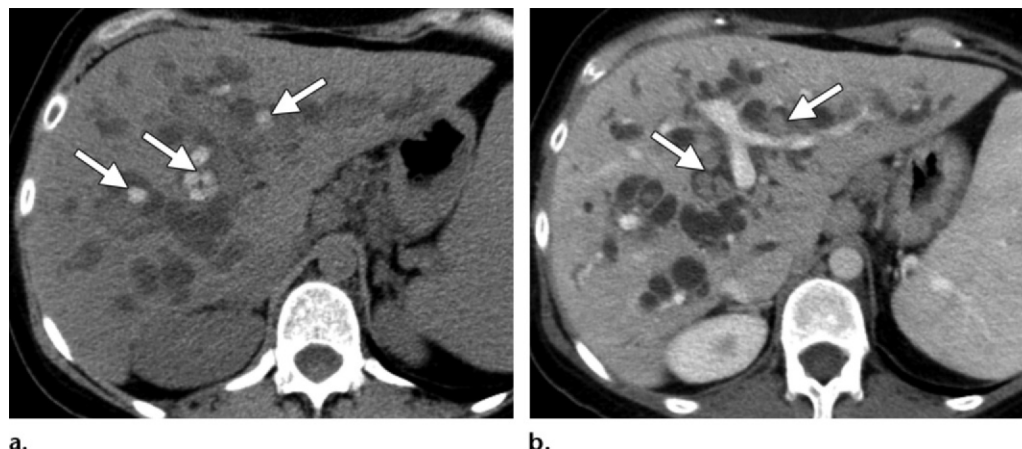
CT findings of biliary necrosis include marked regional branched or beaded biliary dilatation with adjacent parallel low attenuation in the liver parenchyma (Fig 20a) (76). Focal bile lakes may be seen as a late sequela of hepatic artery thrombosis (77). In one series of patients, hepatic infarction most commonly involved segments IV and VIII, while it occurred most commonly in segments II and III in an-

other series (76,78). Superinfection of hepatic infarction, although uncommon, tends to occur in the right lobe in patients who have undergone transplantation or pancreaticoduodenectomy and can be managed with percutaneous drainage (78). Although the imaging appearance of an infected hepatic infarct overlaps with that of a typical pyogenic abscess, the former is often complicated by communication with the biliary system (78).

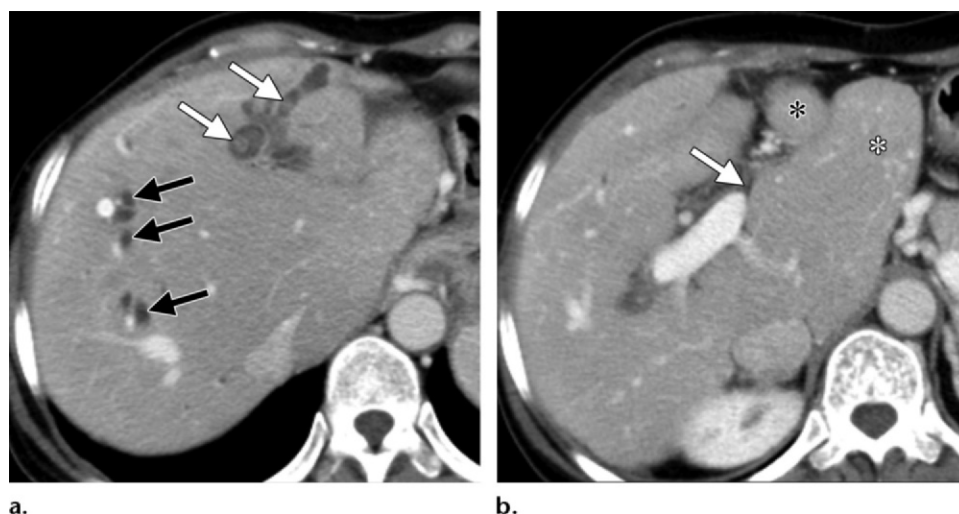
Early hepatic artery thrombosis within 30 days of transplantation (Fig 20b) is associated with higher mortality because of bile duct necrosis and sepsis due to lack of collateral blood supply (79). Treatment is usually retransplantation or surgical thrombectomy, but with a limited organ donor pool, interventional thrombolysis is also possible (79). Late hepatic artery thrombosis may manifest asymptotically or with nonspecific symptoms, and complications such as biliary necrosis and hepatic abscess can be diagnosed at imaging. Treatment involves managing complications rather than addressing hepatic artery thrombosis because collateral vessels have already formed and restoration of flow in the hepatic artery is usually of limited benefit.

### Hepatolithiasis and Recurrent Pyogenic Cholangitis

Hepatolithiasis refers to stones in the intrahepatic biliary ducts proximal to the common hepatic duct (Fig 21). Recurrent pyogenic cholangitis, also known as Oriental cholangiohepatitis, Oriental infestational cholangitis, or intrahepatic



**Figure 21.** Hepatolithiasis in a 44-year-old woman. **(a)** Axial nonenhanced CT image shows multiple high-attenuation gallstones (arrows) in a diffusely dilated intrahepatic biliary system. **(b)** Axial contrast-enhanced CT image shows multiple intrahepatic gallstones (arrows) with reduced conspicuity due to decreased attenuation difference with adjacent enhanced hepatic parenchyma.



**Figure 22.** Recurrent pyogenic cholangitis in a 51-year-old woman. **(a)** Axial contrast-enhanced CT image shows intrahepatic ductal dilatation (black arrows) with intrahepatic stones (white arrows) in the left lobe of the liver. **(b)** Axial contrast-enhanced CT image shows atrophy of the lateral segment of the left lobe (black \*) and marked hypertrophy of the caudate lobe (white \*). The left portal vein is not visualized (arrow), a finding suggestive of obstruction.

pigmented stone disease, is characterized by recurrent episodes of cholangitis associated with hepatolithiasis. Stones are of the intrabiliary pigmented or calcium bilirubinate subtype. Presenting symptoms include recurrent bouts of cholangitis characterized by right upper quadrant pain, fever, and jaundice.

Recurrent pyogenic cholangitis is found primarily in residents and former residents of East Asia and occasionally in Asian immigrants in western societies. Its etiology is uncertain, although a higher prevalence of cases in regions with endemic biliary parasites has implicated liver trematodes as causative parasites.

CT findings include first- and second-order intrahepatic biliary ductal dilatation with abrupt nonvisualization of the peripheral branches, most commonly in the lateral segment of the left lobe (Fig 22a) (80). In one series of patients, intrahepatic calculi existed in 74% of patients, and more than 90% of the calculi were higher in attenuation than the surrounding unenhanced hepatic parenchyma; pneumobilia, more frequently in the left lobe, was also seen in more than 50% of patients (80). Segmental atrophy may be seen in the lateral segment of the left lobe or the posterior segment



of the right lobe with distortion of the hepatic and biliary architecture (Fig 22b) (80). In an angiographic study of recurrent pyogenic cholangitis, the degree of atrophy correlated positively with the degree of portal vein obstruction (81).

Complications of recurrent pyogenic cholangitis include hepatic abscess, biloma, acute pancreatitis, and cholangiocarcinoma. Hepatic abscess occurs in approximately 20% of patients and has typical CT imaging characteristics (80). The prevalence of cholangiocarcinoma with recurrent pyogenic cholangitis is approximately 6% (82). Peripheral cholangiocarcinoma, which arises in second-order to the more distal intrahepatic branches, accounts for 80% of cases, and hilar or extrahepatic cholangiocarcinoma accounts for the remaining 20% (82). The differential diagnosis should also include inflammatory pseudotumor, a localized mass composed of fibrous stroma and chronic inflammatory cells without anaplasia, which may be impossible to distinguish from peripheral cholangiocarcinoma (83).

Management of recurrent pyogenic cholangitis is complicated and usually requires percutaneous, endoscopic, and surgical intervention to effectively treat acute bouts of cholangitis and eliminate biliary calculi to avoid long-term complications of hepatic abscess, recurrent cholangitis, and cholangiocarcinoma.

## Conclusion

Various biliary pathologic conditions cause abdominal pain in the acute clinical setting. Although US is the primary imaging modality used for evaluating right upper quadrant pain, CT can provide additional characterization when US findings are equivocal. Recognition of CT imaging features of various biliary disorders is essential for proper diagnosis and treatment referral.

**Disclosures of Conflicts of Interest.**—A.O.: *Related financial activities:* none. *Other financial activities:* consultant for Oxford Analytica, lecturer for Philips Healthcare.

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## Multidetector CT of Emergent Biliary Pathologic Conditions

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RadioGraphics 2013; 33:1867–1888 • Published online 10.1148/rg.337125038 • Content Codes: CT ER GI

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### Page 1869

The negative predictive value of CT for diagnosis of acute cholecystitis is 89%, and although this value is lower than that of US, CT can exclude the diagnosis in patients with a low clinical suspicion for gallbladder disease and nonspecific clinical symptoms.

### Page 1870

CT is insensitive but highly specific for the diagnosis of acute gangrenous cholecystitis. Specific CT findings include intramural or intraluminal gas, intraluminal membranes, irregular or absent gallbladder wall, lack of gallbladder wall enhancement, and pericholecystic abscess formation.

### Page 1873

Most obstructive gallstones are at least 2.5 cm in diameter and are located distally in the ileum and jejunum, with frequency rates of approximately 60% and 26%, respectively. The terminal ileum is the narrowest segment of the small bowel and thus is the most likely site of obstruction.

### Page 1881

A percutaneous transhepatic biliary catheter contains a radiopaque marker that designates the most proximal end of the catheter drainage side holes. The radiopaque marker should be placed above the site of obstruction but within the biliary system for effective biliary drainage.

### Page 1882

Pneumobilia, a common finding in patients with patent biliary stents, may not be seen with an occluded stent because there is no communication with the duodenum.