Ultrasound Imaging of the Biliary Tract

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Patients who have disease of the biliary tract commonly present with acute right upper quadrant pain, nausea or vomiting, mid-epigastric pain, and/or jaundice. Etiologies include inflammation with or without infection, noninflammatory disorders, and benign or malignant neoplasms of the gallbladder or bile ducts. Ultrasound (US) is now accepted as the initial imaging modality of choice for the work-up of suspected biliary tract disease.

This article reviews the most common diseases of the gallbladder and bile ducts, strategies for evaluating the biliary tract with ultrasound, and specific imaging patterns that aid in diagnosis.

Inflammatory disorders: cholecystitis

Acute cholecystitis most often occurs secondary to obstruction of the gallbladder with resultant inflammation of the gallbladder wall. There may or may not be associated infection and necrosis. Ninety to ninety-five percent of all cases of acute cholecystitis are caused by obstruction of either the cystic duct or the neck of the gallbladder by gallstones [1]. Acute cholecystitis, however, occurs in only approximately 20% of patients who have gallstones [2]. This means that most gallstones are asymptomatic. Thus, right upper quadrant pain in a patient who has gallstones often is caused by something other than acute cholecystitis [3]. Furthermore, studies have shown that only 20%–35% of patients presenting with right upper quadrant pain are subsequently shown to have acute cholecystitis [1,2]. Therefore, it is important to understand the sensitivity and specificity of common US findings in patients who have acute cholecystitis, because the presence of gallstones alone is not adequate to
make the diagnosis of acute cholecystitis. The combination of US findings that is most predictive of acute cholecystitis is the presence of a positive sono-

graphic Murphy sign plus the presence of gallstones. Secondary signs on US examination of acute chole-
cystitis include gallbladder wall thickening (>3 mm), a distended or hydropic gallbladder (loss of the normal tapered neck and development of an elliptic or rounded shape), and pericholecystic fluid.

The sonographic Murphy’s sign

The sonographic Murphy sign is defined as repro-
ducible point tenderness specifically over the gall-
bladder upon application of pressure by the transducer. Ralls and colleagues [4] wrote a classic article that reported a sonographic Murphy sign was 87% specific for the diagnosis of acute cholecystitis, in a patient population which only included patients who had right upper quadrant pain, fever and an elevated white blood cell count. Laing and colleagues [5] reported that the presence of a positive sonographic Murphy’s sign in combina-
tion with the presence of gallstones has a positive predictive value of 92% for the diagnosis of acute cholecystitis. In order to avoid false positive exami-
nations, one must be careful to elicit pain directly

over the gallbladder, not diffusely in epigastrium, or over the liver edge. False negative examinations may occur in patients who have received pain med-

cine, patients who are taking steroids, para or quadriplegics, or any patient who is not able to give a reliable history or pain response. In addition, the sonographic Murphy’s sign may be absent in de-
nervated gallbladders, for example, in patients who have diabetes or gangrenous cholecystitis. A sono-
graphic Murphy’s sign also may be significantly diminished if the gallbladder ruptures because this will relieve the obstruction. Therefore, careful attention to the patient’s clinical status is important when assessing for a sonographic Murphy’s sign.

Gallstones

Gallstones are diagnosed on US by the presence of gravity-dependent, mobile, echogenic foci within the gallbladder lumen that cast a posterior shadow (Fig. 1). Although ultrasound has been demon-

strated to have an accuracy (>95%) for the identifica-
tion of gallstones, stones that are too small, (usually <1 mm to cast a posterior shadow soft stones lacking strong internal echoes [1], or gallstones impacted in the gallbladder neck or in the cystic duct that may not be as readily detectable on US examination as they silhouette with the surrounding echogenic bowel gas or intraperitoneal fat (see Fig. 1) [5]. If the gallbladder is focally tender but no gallstones are appreciated, the patient should be examined from multiple positions and scanning planes, including prone, upright and decubitus positions and intercostal scanning, to facilitate complete visualization of the neck of the gallbladder [3,6].

Harmonic imaging significantly improves visualization of small gallstones. This type of ultrasound transmits the insonating US beam at a fundamental frequency, such as 2.5 or 3 MHz, and receives the returning echoes not only at the fundamental fre-

quency but also at the second harmonic frequency that is twice the fundamental frequency creating the image with the higher harmonic frequency

[7–9]. By eliminating the fundamental frequency, this technique significantly reduces degradation of the image by noise, since lower frequencies easily can be filtered out. In addition, scattering of the US beam from fat in the anterior abdominal wall is diminished because the harmonic frequencies are generated after the beam enters the body. The narrower harmonic beam also has fewer side lobes, and therefore, improved lateral resolution and sig-
nal to noise ratio. Harmonic imaging increases the echogenicity of gallstones and strengthens their posterior shadows, permitting visualization of stones not seen with conventional grayscale ultrasound (see Fig. 1). Another technique that im-

proves visualization of stones is spatial compounding. Multiple images are acquired slightly off axis from one another, which increases the signal from the persistent echoes that comprise the image and blurs out some of the random noise. The disadvan-
tage of compounding is that posterior shadowing is diminished, which may be a better visual cue to detect typical gallstones than the actual echoes.

Fig. 1. Gallstones. (A) (Left) Gallstone in the gallbladder neck (arrow) casts no significant shadow and is nearly invisible. Gas in the duodenum (arrowhead) obscures the fundus of the gallbladder and casts a strong sharp shadow (asterisk). (Right) With patient in sitting position, stone (arrow) moves out of the neck and casts a clear shadow (asterisk). Adjacent duodenum (arrowheads) is now separate from the gallbladder but still casts a strong shadow, equivalent to the gallstone. (B) (Left) Multiple gallstones (arrowheads), some of which cast shadows (arrows), whereas others do not. (Right) Normal caliber common duct (6 mm at the porta) with stones (arrows) in the same patient. Choledocholithiasis may be difficult to detect, especially in the distal duct, if the stones do not shadow or are not outlined by fluid. (C) (Left) Longitudinal ultrasound shows a normal gallbladder. (Right) Harmonic imaging reveals multiple small stones (arrows). (From Rubens D. Hepatobiliary imaging and its pitfalls. Radiol Clin North Am 2004;42:257–78; with permission.)
from the stones themselves. So, while harmonic imaging definitely improves detection, spatial compounding remains optional on an individual case basis.

Other stones such as soft pigment stones may not shadow with any technique. Soft pigment stones are less echogenic than the more common cholesterol gallstones and may simulate soft tissue masses. Pigmented stones are commonly associated with recurrent pyogenic cholangiohepatitis and are more often seen in the bile ducts than in the gallbladder. Because of their lack of shadowing, they may be misinterpreted as sludge or debris and result in a false negative examination.

False positive US diagnosis of gallstones may occur secondary to side lobe artifacts that can generate echoes appearing to arise within the gallbladder lumen but actually originate from the wall or outside the wall [1]. Similarly, gas in adjacent bowel can create a brightly echogenic mass-like area with posterior shadowing, which appears to be within the gallbladder lumen because of a partial volume artifact and thereby mimics gallstones (Fig. 1A). A calcium bile salt precipitate may form in patients taking the antibiotic ceftriaxone and may mimic gallstones on sonographic examination. These precipitates resolve after the patient ends therapy.

Other fluid-containing structures such as the duodenum, gastric antrum, colon, hematomas, pancreatic pseudocysts (Fig. 2), or even dilated vascular collaterals may be mistaken for the gallbladder on US examination, especially if the gallbladder is out of its normal position or is small and contracted. Mistaking these structures for the gallbladder may result in missing pathology in the true gallbladder or a false-positive diagnosis of gallbladder disease (ie, obstructed gallbladder or acalculous cholecystitis).

**Gallbladder wall thickening and pericholecystic fluid**

Gallbladder wall thickening is defined as a wall diameter greater than 3 mm and is present in 50% of patients who have acute cholecystitis (Fig. 3) [1]. However, this is a very non-specific finding, because numerous other etiologies such as hepatic congestion or edema, congestive heart failure, or hypoproteinemia (often associated with renal disease or hepatic dysfunction) can cause thickening of the gallbladder wall. Adenomyomatosis and cancer of the gall bladder also may result in thickening of the gallbladder wall [3]. A thickened gallbladder wall also can occur in association with viral infections and adjacent inflammatory conditions, including hepatitis, peptic ulcer disease (Fig. 4), pancreatitis, perihepatitis (Fitz-Hugh-Curtis syndrome), and pyelonephritis (Fig. 5). In patients who have thickening of the gallbladder wall caused by etiologies other than acute cholecystitis, the gallbladder often is nondistended, implying a nonobstructive (non-biliary) cause of wall thickening (Fig. 6).

A thickened gallbladder wall demonstrating a striated appearance with alternating hyper- and hypoechoic layers in the setting of acute cholecystitis is strongly associated gangrenous cholecystitis [10]. However, striations in the gallbladder wall without
evidence of acute cholecystitis is a nonspecific finding and is often noted in patients who have hepatitis [11] (see Fig. 6).

Pericholecystic fluid is also a nonspecific finding, often occurring secondary to localized inflammation from other causes, such as peptic ulcer disease [3] (see Fig. 4) or identified in patients who have ascites. Teefey and colleagues [10] have described two specific patterns of pericholecystic fluid. Type I, a thin anechoic crescent-shaped collection adjacent to the gallbladder wall, is a nonspecific finding (see Fig. 4B). Type II, a round or irregularly shaped collection with thick walls, septations, or internal debris, is more likely to be associated with gallbladder perforation and abscess formation (Fig. 7).

**Acute acalculous cholecystitis**

Acute acalculous cholecystitis account for up to 5%–14% of cases of acute cholecystitis [11]. It is seen most commonly in critically ill patients often following trauma, surgery, or major burns. The exact etiology is unknown, but ischemia, hypotension or sepsis are likely contributing factors [12]. These critically ill patients are often medicated with narcotics, placed on ventilators, and receive hyperalimentation that contribute to biliary stasis and functional obstruction of the cystic duct obstruction. Gangrene of the gallbladder develops in approximately 40% to 60% of patients who have an associated increased risk for perforation [2]. Mortality ranges from 6% to 44% but can be reduced by
early diagnosis and therapy [12]. However, the diagnosis of acalculous cholecystitis is difficult to make clinically and by US, because gallstones are absent and the sonographic Murphy sign may not be detected because of diminished mental status, medication and co-morbid illness. In the series reported by Cornwall and colleagues [12], only 50% of patients who had acalculous cholecystitis had a positive Murphy’s sign. The diagnosis is, therefore, made by distension of the gall bladder in a suspicious clinical setting, the presence of intraluminal debris, gallbladder tenderness when present (~50%) and gallbladder wall thickening when other etiologies, such as hypoalbuminemia, congestive heart failure (CHF), or liver disease are considered unlikely to be the cause (Fig. 8). CT can be used to assess for pericholecystic inflammation to improve diagnostic specificity in patients who have a thick gallbladder wall and multiple potential etiologies [2,13].

**Complicated cholecystitis**

Gangrenous cholecystitis, emphysematous cholecystitis, and perforation of the gallbladder occur in up to 20% of patients who have acute cholecystitis [5]. These complications are important to recognize, because they are associated with increased
morbidity (10%) and mortality (15%) [14] and require emergency surgery [2]. There is also approximately a 30% conversion rate for laparoscopic cholecystectomy to an open procedure in the setting of complicated cholecystitis [14].

Gangrenous cholecystitis

Gangrenous cholecystitis is defined histologically as coagulative necrosis of the mucosa or the entire gallbladder wall associated with acute or chronic inflammation [10]. It occurs in up to 20% of patients who have acute cholecystitis and has an increased risk for perforation [3]. Unfortunately ultrasound is nonspecific for the diagnosis of gangrenous cholecystitis. This is because the sonographic Murphy sign is absent in up to two thirds of patients [15]. A specific finding is the presence of intraluminal membranes or stranding caused by sloughing of the gallbladder mucosa, necrosis of the gallbladder wall or fibrous exudate (Fig. 9). This finding is present on US examination, however, in only 5% of patients [10].

Gallbladder perforation

Perforation of the gallbladder occurs in 5% to 10% of patients who have acute cholecystitis, most often in association with gangrenous cholecystitis [3]. The fundus is the most common site for perforation, because it has the least blood supply. Acute gallbladder perforation with an intraperitoneal bile leak will result in peritonitis but is much less common than subacute perforation, which typically leads to pericholecystic abscess formation [2]. These abscesses may occur within or adjacent to the gallbladder wall in the gallbladder fossa, within the liver, pararenchyma, or along the free margin of the gallbladder within the peritoneal cavity [10]. These are complex fluid collections. Inflammatory changes in the adjacent fat can be detected on ultrasound or CT (Fig. 7C) [2]. Patients who have intraperitoneal abscesses require immediate surgery, although liver abscesses can be treated effectively with percutaneous drainage. Abscesses in the gallbladder wall or gallbladder fossa may respond to conservative management [16].

Pericholecystic fluid adjacent to the gallbladder wall may mimic perforation. However, with careful inspection, the gallbladder wall will be intact, and the fluid is typically anechoic (see Fig. 4B). Fluid collecting within the gallbladder wall has been reported in one case to precede perforation [17]. However, no other specific ultrasound features have been identified that will accurately predict which inflamed gallbladders will perforate.

Emphysematous cholecystitis

This is a rare complication of acute cholecystitis, accounting for less than 1% of all complicated cases of acute cholecystitis, and is caused by gas-forming bacteria in the gallbladder lumen or in the gallbladder wall. As many as 40% of patients who have emphysematous cholecystitis have diabetes [2]. Emphysematous cholecystitis is more common in men and patients often do not have gallstones. The clinical course is rapidly progressive, with a 75% incidence of gallbladder gangrene and a 20% incidence of gallbladder perforation [18]. Emphysematous cholecystitis can be recognized on US examination by the extremely echogenic gas which casts a distal shadow and layers nondependently within the gallbladder lumen (Fig. 10). Intramural gas is more difficult to identify, because it may mimic the mural calcification seen in a porcelain gallbladder. The type of shadowing (ie, “clean” versus “dirty”) does not differentiate between calcium and air. The nondependent location of the mobile echoes within the lumen or mobile bubbles within the wall can document gas. If the US findings are equivocal, either CT or plain film radiography can be used to differentiate between gas and calcification [19].
Chronic cholecystitis is defined histologically as chronic inflammation of the gallbladder wall and is routinely associated with gallstones. It can generally be differentiated from acute cholecystitis by the absence of acute clinical symptoms, although it can be exacerbated by episodes of superimposed acute cholecystitis. The chronic inflammation causes thickening and fibrosis of the gallbladder wall and, ultimately, contraction of the gallbladder which when severe can result in almost complete obliteration of the gallbladder lumen. This produces an US image with two brightly colored arcs and a posterior shadow, the so-called “double arc” sign or wall-echo-shadow (WES) complex [20] (Fig. 11). The first echogenic arc of the WES complex is created by the near wall of the gallbladder and the second by the gallstone. The two echoes are discernible because they are separated by a thin crescent of anechoic bile in the residual gallbladder lumen. The WES complex can be mimicked by a collapsed duodenum (Fig. 12) or, rarely, by a porcelain gallbladder. Porcelain gallbladder is more common in males, is seen in conjunction with chronic cholecystitis, and is the result of mural calcification of the gallbladder wall. It is a rare disorder, seen in 0.06% to 0.8% of cholecystectomy specimens [2]. The calcification pattern on ultrasound may involve the entire wall or only a portion

**Fig. 7.** Complicated cholecystitis with gallbladder perforation. (A) Longitudinal ultrasound of the gallbladder with adjacent irregularly marginated pericholecystic intrahepatic fluid (arrows). This patient presented with sepsis 2 weeks after prostate surgery and was found to have acute cholecystitis with an adjacent liver abscess. (B) Longitudinal ultrasound of gallbladder containing stones shows a pericholecystic collection (arrows) containing debris. The collection abuts the free wall of the gallbladder and is not contained within the gallbladder wall (double arrow). (C) CT shows an enhancing rim around the fluid (arrows) and inflammatory edema in the adjacent fat consistent with abscess (arrowheads). (From Rubens D. Hepatobiliary imaging and its pitfalls. Radiol Clin North Am 2004;42:257–78; with permission.)
of it. In either case, the calcified wall causes only a single echogenic arc, not the double arc seen in the WES complex. If calcification in the wall is heavy, there is a single echo with a strong posterior shadow obscuring the gallbladder. With lesser degrees of calcification, the gallbladder lumen may be discerned posteriorly (Fig. 13). Another manifestation of chronic cholecystitis is xanthogranulomatous cholecystitis (XGP), in which the gallbladder wall is infiltrated by foamy histiocytes, lymphocytes, polymorphonuclear leukocytes, fibroblasts, and giant cells [19]. It presents sonographically as diffuse or focal thickening of the gallbladder wall, with mural nodularity (Fig. 14). Although this is rare, occurring in only 2% of cholecystectomy specimens [21], the imaging appearance is often difficult to distinguish from noninflammatory lesions, such as adenomyomatosis and gallbladder carcinoma. Because the hepatic surface of the gallbladder lacks a serosal layer, the inflammatory process more easily extends to the adjacent liver, and the liver–gallbladder margin is frequently indistinct [2,19].

Noninflammatory non-neoplastic gallbladder disorders: the hyperplastic cholecystoses—cholesterolosis and adenomyomatosis

Hyperplastic cholecystoses are common, often asymptomatic processes that involve various layers of the gallbladder wall. Cholesterolosis, which may be diffuse or polypoid, has been reported in up to 25% of surgical specimens [2], whereas adenomyomatosis (diffuse, focal, or polypoid) has been reported in 8.7% [22]. Cholesterolosis is caused by deposition of lipid-laden macrophages in the lamina propria, beneath the normal epithelium in the mucosa of the gallbladder wall. The diffuse form, which is more common, is difficult to appreciate on imaging [2]. Cholesterol polyps represent 20% of cholesterolosis but comprise approximately one half of all gallbladder polyps [2,19]. They are usually less than 1 cm in size, often multiple, and have no malignant potential. On ultrasound they appear brightly echogenic, round or lobulated, immobile, non-shadowing masses abutting the gallbladder wall (Fig. 15). Adenomyomatosis, also known as adenomyomatous hyperplasia, involves the mucosa and the muscular and connective tissue layers of the gallbladder wall. The epithelium and muscular layers proliferate, and invagination of the epithelial-lined spaces into the gallbladder wall produce intramural diverticula, termed Rokitansky-Aschoff sinuses. These may accumulate bile, cholesterol crystals, or even stones. On US examinations they may be anechoic if large enough and bile containing but more frequently are small and contain cholesterol, biliary sludge, or gallstones that create echogenic foci (Fig. 16), often with ring-down or comet tail reverberation artifacts [23]. The most common form of adenomyomatosis is a focal polypoid lesion, also known as an adenomyoma, typically located at the tip of the gallbladder fundus. The segmental form consists of localized gallbladder wall thickening that typically narrows the gallbladder body in an hourglass configuration. Diffuse adenomyomatosis involving
the entire gallbladder wall is less common than focal or segmental diseases.

**Benign neoplasms of the gallbladder**

Adenomas are well-demarcated, polypoid gallbladder lesions, usually less than 2 cm in size, and are rare compared with cholesterol polyps or polyps of adenomyomatous hyperplasia. They are found in 0.3% to 0.5% of cholecystectomy specimens and are usually solitary. They are classified as tubular, papillary, or tubulopapillary, depending on their growth pattern. On US they are typically echogenic when small, but become more heterogeneous as they enlarge [19]. Their premalignant potential is believed to be low, although this is somewhat controversial. There is a much stronger relationship between chronic cholecystitis and gallbladder carcinoma than between adenoma and carcinoma. On imaging alone, however, it is impossible to identify coexistent dysplasia or carcinoma in situ within an adenoma, or to determine whether a polypoid mass is benign or malignant (Fig. 17). The current surgical literature therefore recommends excision of polyps greater than 1 cm in size in patients older than age 50 years or any polyp that is clearly growing, even if less than 1 cm [24,25].

**Malignant neoplasms of the gallbladder**

Gallbladder carcinoma represents 98% or more of all gallbladder malignancies; the rest are comprised of nonepithelial tumors arising from the muscular or neurologic components of the wall, metastases or lymphoma (lymphoma) [26]. The median age at presentation is 72 years, with a 2:1 female-to-male ratio [26]. The major risk factor is
chronic cholecystitis, which is associated with dysplasia and carcinoma in situ, and which subsequently progresses to invasive carcinoma [27]. Other risk factors include primary sclerosing cholangitis, anomalous junction of the pancreaticobiliary ducts, and choledochal cysts [27]. Gallbladder carcinoma is found incidentally in 1% of cholecystectomy specimens and in up to 6% of cholecystectomies performed for polypoid lesions [25]. It is usually not suspected clinically nor detected on preoperative imaging. When gallbladder carcinoma is diagnosed by imaging, it is usually advanced [27,28]. The lack of serosa in the portion of the gallbladder wall adjacent to the liver means that the connective tissue of the gallbladder wall in this region is in direct continuity with the hepatic interlobar fissure. This permits early tumor invasion into the bloodstream, lymphatics, adjacent liver parenchyma, and hepatoduodenal ligament [27]. The most common imaging presentation of the gallbladder is that of a mass completely replacing the gallbladder (40%–65%). Less commonly focal or diffuse wall thickening (20%–30%) or an intraluminal polypoid mass (15%–25%) may be seen [27]. Carcinomas that replace the gallbladder are often heterogeneous, obliteration of the normally distinct plane which separates the gallbladder from the adjacent liver, caused by direct invasion of the adjacent hepatic parenchyma. Immobility of gallstones displaced by the mass may be a clue to diagnosis. Hepatic arterial Doppler waveforms within an intraluminal mass are also suspicious for malignancy (Fig. 18). Any solitary mass greater than 1 cm with internal vascularity should raise suspicion for carcinoma [28] (Fig. 17). Wall thickening alone may be difficult to distinguish from more common benign disorders, such as chronic cholecystitis or adenomyomatosis. Pronounced wall thickening greater than 1 cm or loss of the normal mural layers of the wall [27,28] should raise concern for malignancy (Fig. 18). If adenomyomatosis is suspected, the characteristic echoes from the Rokitansky-Aschoff sinuses should be sought, or MR imaging can be used to improve specificity [27]. Similarly, if a focal polypoid mass is present, the characteristics of adenomyomatosis should be absent.

**Fig. 11.** Wall-echo-shadow complex. Transverse image of the gallbladder demonstrates the anterior echo of the gallbladder wall (arrow), the small space of the residual lumen containing bile (asterisk) and the second, posterior parallel echo of the stone (arrowheads).

**Fig. 12.** WES mimic. (A) Longitudinal ultrasound image shows a sharp shadow (asterisk) behind a strong echo (arrow) that could be mistaken for a WES complex. (B) Transverse image confirms a normal gallbladder (GB) laterally. Unlike the gallbladder wall, which is echogenic, the duodenal wall (arrowhead) anterior to the echo of the luminal gas is actually hypoechoic.
Fig. 13. Porcelain gallbladder. (A) Sagittal image of the gallbladder shows a densely echogenic anterior wall (arrow) with a sharp shadow that obliterates the gallbladder lumen and posterior wall. (B) Transverse ultrasound of the gallbladder in the same patient. The anterior wall is bright, but, without enough reflection or attenuation to eliminate visualization of the lumen and posterior wall (arrow), which is also echogenic and casts a posterior acoustic shadow. (From the Armed Forces Institute of Pathology/American Registry of Pathology; with permission.)

Fig. 14. Xanthogranulomatous cholecystitis. (A) Transverse image of the gallbladder with a compressed lumen (asterisk). The walls are markedly thickened with multiple hypoechoic mural inflammatory nodules (arrowheads) that envelop the adjacent stone (arrow). (B) CT axial image of the gallbladder shows the compressed lumen (asterisk) and the multiple large mural nodules (arrowheads) separated by enhancing margins. The stone is not visualized. (From the Armed Forces Institute of Pathology/American Registry of Pathology; with permission.)

Fig. 15. Cholesterol polyps. (A) Initial longitudinal ultrasound shows two nondependent and non-shadowing 5-mm moderately echogenic nodules (arrows) abutting the gallbladder wall. (B) Two years later the nodules are unchanged in size, appearance, and location (arrows).
Biliary ducts

Disease of the bile ducts usually is manifested by obstruction of the bile ducts with resultant dilatation of either the intrahepatic or extrahepatic ducts or both. Clinical presentation is varied and may include right upper quadrant or epigastric pain, fever, and jaundice. Biliary ductal dilatation may be attributable to multiple causes, including stones, intrinsic tumor, stricture, or compression by extrinsic masses. Ultrasound ideally is suited to screen both the intrahepatic and extrahepatic ducts for duct size and continuity. Either or both may be dilated, depending on the level of obstruction.

**Ultrasound diagnosis of biliary ductal dilatation**

The extrahepatic common duct is measured from inner wall to inner wall at the level of the crossing of the right hepatic artery. The diameter at this level should not exceed 6 mm [1]. The diameter of the common duct is slightly greater distally as it approaches the pancreas, sometimes by as much as 10 mm.

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**Fig. 16.** Adenomyomatosis. Transverse ultrasound shows multiple discrete 1-mm echogenic cholesterol crystals in the intramural diverticula (Rokitansky-Aschoff sinuses) (arrows) within a thickened gallbladder wall in this asymptomatic patient who had adenomyomatosis. (From Harrow A. The gallbladder and biliary tree. In: Dogra V, Rubens D, editors. Ultrasound secrets. Philadelphia: Hanley and Belfus; 2004. p. 113–29; with permission.)

**Fig. 17.** Gallbladder adenoma. Transverse color Doppler ultrasound in a 67-year-old man presenting with a 2-week history of RUQ pain shows a 2-cm polypoid mass at the fundus with internal vascularity. Histology revealed a tubular villous adenoma with foci of carcinoma in situ. (From Harrow A. The gallbladder and biliary tree. In: Dogra V, Rubens D, editors. Ultrasound secrets. Philadelphia: Hanley and Belfus; 2004. p. 113–29; with permission.)

**Fig. 18.** Gallbladder carcinoma. (A) Transverse color Doppler image of the gallbladder shows stones (arrows) anterior to a 2-cm thickened hypoechoic wall with no discrete features but demonstrating some vascularity. (B) Spectral Doppler shows high-velocity arterial flow within the wall, concerning for tumor.
as 1 to 2 mm. There is still debate in the literature as to whether the bile duct dilates with age and whether it dilates postcholecystectomy [1,28]. Most laboratories consider a common bile duct (CBD) less than 6 mm normal, and a CBD that measures greater than or equal to 8 mm [1,29] abnormal. Clinically, if the patient has dilated ducts but no accompanying symptoms, such as elevated bilirubin, pain, sepsis, or elevated liver enzymes, including alkaline phosphatase, the dilated ducts are unlikely to be clinically relevant. Hence, the clinical scenario is of paramount importance when assessing the bile ducts. Intrahepatic bile ducts are normal if they measure 2 mm or less in the porta or no more than 40% of the diameter of the accompanying portal vein [1,30]. With the advent of newer equipment, however, it is now possible to see intrahepatic biliary ducts in normal

![Fig. 19. Normal ducts. (A) Normal intrahepatic bile ducts (cursors) in a postcholecystectomy patient. Multicolored vessel in the center of the color box is the hepatic artery (HA), and dark red adjacent vessel is the portal vein (PV). (B & C) Patient who had abdominal pain, nausea, and jaundice 1 month postcholecystectomy. Note multiple anechoic irregularly branching tubes converging at the porta hepatis. Color Doppler image (C) confirms that some are avascular and represent ducts (arrowheads), whereas the portal veins (red), hepatic veins (blue), and hepatic arteries (HA) are correctly identified. The inferior vena cava (IVC) can be recognized by its anatomic position. (From Rubens D. Hepatobiliary imaging and its pitfalls. Radiol Clin North Am 2004:42:257–78; with permission.) (D) Grayscale image (not shown) demonstrated a dilated tubular structure. Sagittal color Doppler image shows antegrade flow in the tube (arrow) and no flow in the smaller adjacent tube. The larger tube was shown to be the hepatic artery with spectral Doppler and the adjacent tube is the portal vein. The bile ducts are not seen at all. (From Rubens D, Carson N. Doppler Evaluation of the Liver and Transjugular Intrahepatic Portovenous Shunts In: Dogra V, Rubens D, editors. Ultrasound Secrets. Philadelphia: Hanley and belfus; 2004. p. 403–19; with permission.)](image)
patients, especially when using harmonic imaging that significantly improves resolution [9] or high frequency transducers in slender patients (Fig. 19A). In general, intrahepatic biliary duct dilation can be diagnosed when irregular angular branching, a central stellate configuration, and acoustic enhancement posterior to the ducts is observed (Fig. 19B) [1]. In addition, the use of color and power Doppler may be extremely valuable by demonstrating that dilated tubular structures in the liver are indeed ducts and not vascular structures (Fig. 19C). In the setting of cirrhosis, the hepatic arteries supply more blood to the liver and frequently dilate. As they also course parallel to the portal vein in the portal triads, they can be mistaken for dilated ducts (Fig. 19D).

**Diagnosis of biliary obstruction**

Assuming the patient has a dilated common duct (≥ 6 mm) associated with clinical signs of obstruction, including elevated bilirubin or elevated alkaline phosphatase, how well does ultrasound identify the level and cause of obstruction? With good US technique, the level of obstruction can be defined in up to 92% of patients and the cause in up to 71% [1]. Important technical factors include patient positioning in the semierect right posterior oblique (RPO) or right lateral decubitus positions to minimize shadowing from overlying bowel gas in the gastric antrum or the duodenum, and the use of a transverse imaging plane to completely follow the duct to the level of the pancreas [1]. Additional technical improvements sometimes can be achieved by having the patient drink water to displace gas or by using a large footprint curvilinear transducer to compress bowel and bowel gas away from the distal duct.

**Causes of biliary obstruction**

**Choledocholithiasis**

Common duct stones are the most common disorder of the biliary tract, occurring in 8% to 20% of patients undergoing cholecystectomy and 2% to 4% of patients postcholecystectomy [30]. On ultrasound, stones are identified as echogenic foci with a distal acoustic shadow. They are easiest to see, however, when surrounded by anechoic bile in a dilated duct and when large enough to cast an acoustic shadow. Stones impacted in the distal duct may be difficult to recognize when their margins are inseparable from the adjacent duct.

**Fig. 20.** Harmonic imaging of stones. (A) Standard longitudinal ultrasound imaging in the porta shows a dilated common duct obstructed by an incompletely visualized stone (arrow) with a faint posterior acoustic shadow (arrowheads) that is difficult to visualize. (B) Same view with harmonic imaging shows brighter, sharper echoes from the stone and a much sharper and darker shadow (arrowheads) posteriorly.

**Fig. 21.** Multiple intraductal stones. Longitudinal ultrasound shows a dilated duct (arrow) filled with multiple shadowing stones (arrowheads) in multiple ducts in this patient who had longstanding cholecystitis and cholangitis.
walls and surrounding echogenic fat. Additionally, small (<5 mm) stones may not cast an acoustic shadow, especially if they are deep in the abdomen or if spatial compounding is used. Harmonic imaging that decreases speckle and improves visualization of the acoustic shadow is a key ingredient to successful imaging [9] (Fig. 20). Similarly, transducer compression, patient positioning, and the use of the liver or water-filled bowel as an acoustic window to avoid interference from bowel gas are

**Fig. 22.** Cholangiocarcinoma. (A) Longitudinal ultrasound image through the right lobe shows multiple dilated ducts (arrows) anterior to the portal vein (asterisk) but no discrete mass. (B) More centrally a longitudinal image through the left lobe shows centrally dilated ducts (arrows) with contour deformity of the ducts and the posterior margin of the liver by an ill-defined space-occupying lesion (asterisk) at the level of the caudate lobe. No discrete margins are identified. (C) Axial CT at the level of the caudate lobe shows a discrete low attenuation mass (asterisk) with obstruction and dilatation of the ducts of the left lobe. The adjacent liver parenchyma is atrophied slightly, especially anteriorly, where it abuts the diaphragm. Localization of the tumor and recognition of atrophy are much more apparent on CT than on ultrasound.

**Fig. 23.** Pancreatic carcinoma. (A) Transverse ultrasound image shows a markedly dilated common duct (arrowheads) that terminates abruptly at the level of a slightly hypoechoic, rounded mass (arrows). (B) Transverse image through the body of the pancreas shows a dilated pancreatic duct (arrowhead), indicating that the level of obstruction is in the pancreatic head.
important maneuvers to improve visualization of stones in the extrahepatic duct. With good technique, Laing and colleagues achieved an overall sensitivity of 75% for US in the detection of cholecystolithiasis, with visualization of 89% of proximal and 70% of distal calculi as compared to surgery [31].

Pitfalls include patients who are obstructed without dilatation. This can occur from intermittent obstruction from stones. As many as one third of common bile duct calculi are found in nondilated bile ducts (Fig. 1B) [1]. In this group of patients, ultrasound is insensitive and MRCP or endoscopic retrograde cholangiopancreatography (ERCP) should be considered. Although the vast majority of stones occur in the distal duct, stones may also be identified in the intrahepatic ducts and may be the clue to more distal obstruction (Fig. 21).

**Neoplasm**

Benign neoplasms of the biliary tree are rare. The most common is the adenoma, which may be discovered incidentally at surgery or imaging or that may actually obstruct the biliary tree and present with pain or jaundice. On ultrasound these soft

![Image](image_url)

**Fig. 24.** Duodenal carcinoma with biliary, pancreatic, and bowel obstruction. Patient presented to the emergency department with nausea and jaundice. (A) Transverse ultrasound of the pancreas shows a 1.8-cm common duct (CD) and a dilated pancreatic duct (arrowheads). (B) Longitudinal ultrasound shows a distended gallbladder with a soft-tissue mass (arrows) behind it. (C) On transverse imaging, the mass (arrows) obstructs the duodenum (Duod), which has a fluid-filled proximal lumen. (D) CT confirms the circumferential duodenal tumor (arrows). Note distended gallbladder and common duct (CD). GB, gallbladder. (From Rubens D. Hepatobiliary imaging and its pitfalls. Radiol Clin North Am 2004;42:257–78; with permission.)
**Fig. 25.** Intraductal hepatocellular carcinoma with invasion of the biliary ducts. (A) Transverse ultrasound image at the porta shows an echogenic mass (arrows) obstructing the intrahepatic bile duct (arrowhead), which is markedly dilated. The echotexture of the left lobe of the liver is heterogeneous, nodular, and distorted (asterisk), lacking the normal vascular landmarks. (B) CT obtained at the same level shows the tumor (arrows) obstructing the intrahepatic duct (arrowhead). There is additional tumor (asterisks) involving the left lobe with multiple nodular metastases. The intraparenchymal extent of the tumor is much greater on CT than would be expected by the ultrasound images. Unlike the cholangiocarcinoma in Fig. 22, this tumor expands the left lobe.

**Fig. 26.** Sclerosing cholangitis. A 50-year-old woman presented with sepsis and abdominal pain. (A) Longitudinal ultrasound of the right lobe is normal, with the common duct ( cursors) measuring 2 mm. (B) Longitudinal ultrasound of the left lobe shows multiple markedly enlarged ducts (arrows), with enhancing walls, indicating inflammation. No central obstructing mass is identified. Emergent biliary drainage was performed, which alleviated the patient’s symptoms. (From Rubens D. Hepatobiliary imaging and its pitfalls. Radiol Clin North Am 2004;42:257–78; with permission.)
tissue masses are homogeneous, isoechoic to the liver parenchyma, and, if obstructing, cause proximal dilatation of the biliary tree [19]. Biliary cystadenomas are also rare and although benign histologically, tend to recur locally and may transform into cystadenocarcinomas [19]. They occur most commonly intrahepatically (83%) with the remainder in the extrahepatic bile ducts. These lesions tend to occur in middle-aged women and are large (3 to 40 cm), solitary cystic masses [19]. Ultrasound findings range from unilocular cyst to multilocular cysts sometimes with mural nodules and papillary projections. Calcification is common and may occur within the cyst wall or in the septations. Cystic fluid may be simple or complex, containing mucin, protein, or hemorrhagic debris. Differential diagnoses include echinococcal cysts, hemorrhagic cysts, cystic metastasis, and abscess.

Cholangiocarcinoma is the primary malignant neoplasm of the biliary tree. Risk factors include sclerosing cholangitis, choledochal cysts, and parasitic infection with liver flukes. Cholangiocarcinomas may arise anywhere along the biliary tree, but are most common at the hilum (50%–60%), otherwise known as a Klatskin tumors. Intrahepatic or peripheral cholangiocarcinomas comprise 10%, and extrahepatic or distal cholangiocarcinomas make up the remaining 30% [28,32]. Tumors are variable in their growth patterns and consequently in their ultrasound appearance. Tumor spread may be within or adjacent to the duct wall in a scirrhous pattern, leading to stricture and biliary ductal dilatation. This component of the tumor is fibrous. Walls of the ducts are thickened without a discrete tumor mass [33]. The tumor itself may not be directly visualized on US, only its resultant obstruction. However, careful imaging of the duct walls may reveal focal irregularity to establish a tumor diagnosis [34]. Lesions show decreased attenuation in comparison to the liver on the portal venous phase images of contrast-enhanced CT or MR imaging and are often much more apparent than on noncontrast ultrasound (Fig. 22). Glandular cells within the tumor produce mucin and contribute to greater mass effect than the stromal fibrous component [33]. Mass-like lesions are most common in the hilum or in the periphery of the liver. As the tumor extends outside the ducts and forms a mass, it may obstruct adjacent vessels, either the portal veins or hepatic arteries. As many as 50% of patients are reported to have associated portal venous involvement [34]. Chronic vascular obstruction caused by extrinsic compression or especially portal vein thrombosis may lead to lobar atrophy, a unique and common feature of peripheral cholangiocarcinoma [30,33]. The peripheral masses are large and present as indeterminate soft-tissue masses, indistinguishable on US from primary hepatocellular carcinoma or metastatic disease. Intraductal masses are uncommon within the liver and are more common in the extrahepatic duct, where they tend to be polypoid. A distal carcinoma may also be scirrhous and may locally thicken the duct wall, in which case it may be indistinguishable from the adjacent pancreas or ampulla. The echogenicity of cholangiocarcinomas is variable, ranging from hypoechoic to moderately echogenic.

Secondary malignant neoplastic involvement of the bile ducts occurs by extrinsic compression or direct extension. Causes of extrinsic compression
are myriad and include hepatic metastases displacing the intrahepatic ducts or extrahepatic obstruction caused by pancreatic carcinoma (Fig. 23), lymphadenopathy, or tumors of the adjacent gastrointestinal tract (Fig. 24). Intrinsic biliary duct obstruction is usually the result of hepatocellular carcinoma, which has a propensity to invade the biliary tree and the adjacent vessels [30] (Fig. 25). Differentiation from cholangiocarcinoma is made on contrast enhanced CT or MR imaging, which demonstrates marked arterial phase enhancement of hepatocellular carcinoma versus only moderate early and sometimes more intense delayed phase enhancement of typical cholangiocarcinomas [30].

**Inflammatory disorders of the biliary ducts**

Acute inflammatory processes include acute bacterial cholangitis, which occurs in concert with biliary

![Fig. 28. Biliary duct necrosis. (A) Transverse ultrasound of a liver transplant patient presenting with sepsis. Amorphous echogenic debris (arrows) is seen on grayscale. (B) Two months later the process has progressed. The echogenic areas (arrows) are more confluent and linear and cast acoustic shadows that obscure the adjacent parenchyma. (C) Color Doppler image shows echogenic debris in a ductal distribution (arrows) and a low resistive index (<0.5) in the hepatic artery, signifying hepatic arterial stenosis or thrombosis. (D) The extensive biliary duct necrosis (arrows) and the resulting liver abscess (arrowheads) are documented by CT. The abscess was obscured on the ultrasound because of shadowing from air in the ducts. (From Rubens D. Hepatobiliary imaging and its pitfalls. Radiol Clin North Am 2004;42:257–78; with permission.)
obstruction in the presence of common duct stones. Clinical presentation is nonspecific, with fever, right upper quadrant pain, and jaundice. US is useful to determine the level, and if possible, the cause of obstruction. Duct walls are thickened and ducts may contain internal debris. The ducts are usually dilated and there may be associated hepatic complications, including liver abscess. Other inflammatory processes adjacent to the ducts may result in obstruction without infection. Mirizzi syndrome involves acute cholecystitis caused by an impacted cystic duct stone that also compresses, inflames, and obstructs the adjacent common duct. Inflammation from pancreatitis can also inflame and narrow the common bile duct causing obstruction and jaundice. Recurrent pyogenic cholangiohepatitis (also known as oriental cholangiohepatitis) is a disease of unknown etiology characterized by strictures, stasis, and stone formation. It is most common in people of Asian descent. It is characterized on ultrasound by dilated ducts filled with stones and debris. Stones are typically noncalcified and of soft-tissue attenuation. Recurrent obstruction may result in focal atrophy of the surrounding liver parenchyma, often a key to the diagnosis [35].

Sclerosing cholangitis may be primary (idiopathic or associated with inflammatory bowel disease) or secondary to prior biliary infection. The pathophysiology is of inflammation followed by segmental fibrosis that leads to strictures and areas of biliary stasis. The latter may result in abscess formation with acute presentation. Chronically, patients progress to cirrhosis and liver failure. Sclerosing cholangitis gives rise to segmental dilation of the bile ducts, often only in one portion of the liver (Figs. 26). Bile duct walls are thickened and irregular, and the segmental strictures give the ducts a beaded appearance on ultrasound. Because there is no mass causing the obstruction, sclerosing cholangitis is difficult to distinguish from an infiltrating form of cholangiocarcinoma. Unfortunately these patients are also at increased risk for cholangiocarcinoma, which occurs in 10% to 30% of patients [36]. AIDS cholangitis, an end-stage infection usually caused by cryptosporidium or cytomegalovirus, also causes strictures and bile duct thickening indistinguishable from primary sclerosing cholangitis [28,30].

**Biliary air and biliary necrosis**

Air in the biliary tree may result from transient or prolonged communication with the gastrointestinal tract. Air from the duodenum is introduced into the bile duct as the sphincter of Oddi opens with passage of a stone (Fig. 27) or with ERCP. Sustained communication occurs following sphincterotomy, an endobiliary stent placement, or biliary enteric fistula. Gas can also arise in the biliary tree as a result of infection, from reflux, emphysematous cholecystitis, or biliary necrosis with secondary abscess formation. On US biliary air gives a typical appearance of smooth, linear, bright echoes that are located adjacent to the portal veins. There may or may not be associated biliary ductal dilatation. Biliary duct necrosis is a critical complication that may occur following liver transplantation, usually secondary to ischemia from hepatic artery stenosis or thrombosis. If biliary necrosis occurs, the ducts become enlarged and filled with pus or necrotic debris. On ultrasound they may appear echogenic, irregular, and enlarged without the usual anechoic bile. Echoes are frequently nodular in appearance (Fig. 28). Shadowing may occur if gas is also present.

**Summary**

US is currently recommended as the primary initial imaging modality for the evaluation of the gallbladder and bile ducts. Recent technical advances such as harmonic imaging and spatial compounding have improved detection of biliary stones, the most common disease of the biliary tract. Tumors and benign inflammatory conditions that mimic tumors may also be detected, but a specific diagnosis may not be readily apparent. If the margins of the gallbladder are indistinct, as in patients with gallbladder carcinoma or xanthogranulomatous cholecystitis, or if perforation or abscess are suspected in case of complicated cholecystitis, CT or MR imaging may be useful to assess the extent and character of the disease process. When the site or cause of biliary obstruction is not apparent, as in cholangiocarcinoma or distal common duct obstruction, further evaluation with contrast enhanced CT or MR imaging with MRCP also is indicated.

**References**


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