Real-time sonography remains the screening study of choice for the evaluation of jaundice in infants and children and it is an important tool in differentiating between obstructive and nonobstructive causes of jaundice [1,2]. The causes of cholestasis are multiple, but the three major causes are hepatitis, biliary atresia, and choledochal cyst. Other causes include neoplastic processes, cirrhosis, and strictures.

This article reviews the common congenital and acquired causes of jaundice in the pediatric patient and describes the sonographic findings associated with these conditions. The role of correlative imaging studies is also reviewed.

**Sonographic examination**

The sonographic examination of infants and children who have jaundice includes a detailed examination of the liver, bile ducts, gallbladder, and pancreas. Hepatic size and echotexture should be thoroughly assessed. The right hepatic lobe should extend to or just below the right costal margin in a patient without hyperinflated lungs. The echogenicity of the normal liver is low to medium and homogeneous, and the central portal venous vasculature is easily seen (Fig. 1). In the neonate and young infant, the hepatic parenchyma and renal cortex are equally echogenic. In individuals 6 months of age and older, the liver usually is more echogenic than the kidney. The patency and flow direction of the hepatic vessels should be documented with pulsed and color Doppler interrogation. The liver and adjacent area should also be evaluated for evidence of end-stage liver disease, including collateral channels (varices), hepatofugal flow, and ascites.

The diameter of the common duct should be measured on the sagittal scan to confirm the presence or absence of ductal dilatation. The upper limits of the common duct should not exceed 1 mm in neonates, 2 mm in infants up to 1 year of age, 4 mm in children 1 to 10 years of age, and 6 mm in adolescents and young adults [3]. The distal portion of the common duct is typically larger than the proximal portion. Ductal size may increase by 1 mm or more during deep inspiration and the Valsalva maneuver [4]. The cystic duct in children
is not routinely seen unless it is dilated, and then usually only the distal part of the duct near its insertion into the common bile duct is seen.

Gallbladder size usually can be assessed subjectively, but measurements may be helpful in equivocal cases. The normal gallbladder length is 1.5 to 3.0 cm in neonates and young infants (younger than 1 year old) and the width is approximately 1 cm. In older children and adolescents, gallbladder length is 3 to 8 cm and width is less than 3.5 cm. The wall of the gallbladder should be thin, hyperechoic, and well defined. The upper limits of wall thickness in the fasting state are 3 mm [5]. Feeding of a fatty meal may be helpful in patients who have enlarged gallbladders to assess cystic duct patency. In healthy individuals, maximum emptying of the gallbladder occurs between 45 and 60 minutes after the fatty meal, and the mean volume decreases approximately 60%. Contraction of the gallbladder after a fatty meal supports the diagnosis of a patent cystic duct.

Pancreatic size, echotexture, and ductal size should be evaluated. Pancreatic size increases with increasing age of the child [6]. The mean cross-sectional diameter of the pancreatic head ranges between 1 and 2 cm, the body between 0.6 and 1.1 cm, and the tail between 1 and 2 cm. The normal pancreas is isoechoic or minimally hyperechoic compared with the liver. The cross-sectional diameter of the pancreatic duct should not exceed 1 to 2 mm.

Overview of cholestatic diseases

Causes of cholestasis vary with patient age. For this review, diseases are classified into two main classes: (1) neonatal and (2) older child and adolescent. In the neonate, biliary atresia, the neonatal hepatitis syndrome, and choledochal cyst are the most common causes of jaundice [7–9]. Other causes include syndromic and nonsyndromic bile duct paucity, inspissated bile syndrome, and spontaneous perforation of the extrahepatic bile duct. In older children, jaundice is most often caused by hepatocellular disease, including hepatitis and cirrhosis. Biliary tract obstruction is a less common cause of childhood jaundice. The possible causes of obstructive jaundice include choledochal cyst, cholangitis, stricture, stones, and neoplasms.

The results of various laboratory tests of liver function, in conjunction with the pertinent historical and physical findings, generally suffice to differentiate between obstructive and nonobstructive causes of jaundice. Imaging examinations are used to confirm the clinical impression. In patients who have obstructive jaundice, these studies may also often show the level and cause of obstruction.

Sonography is the preliminary imaging procedure. If the extrahepatic ducts are well visualized by sonography and are normal in caliber and there is no evidence of intraductal dilatation, further radiologic evaluation is rarely needed. Sonography is supplemented by radionuclide studies using hepatobiliary agents (99mTc-IDA analogs) when functional information is needed. Hepatobiliary scintigraphy currently is used primarily to confirm suspected diagnoses of choledochal cysts, biliary atresia, and neonatal hepatitis. CT or MR imaging are reserved for cases in which more anatomic detail is needed for surgical planning or the level or cause of obstruction cannot be determined by sonography [10–15].

Neonatal jaundice

Biliary atresia

Biliary atresia is a rare disease with an incidence of 1 in 8,000 to 10,000 live births. It is, however, the single most common cause of neonatal cholestasis, accounting for nearly 90% of the surgical causes and for approximately 40% of all causes of cholestasis [16]. The cause is unclear, but it is believed to be caused by in utero inflammation that results in failure of the remodeling process at the hepatic hilum [7]. Histologically, it is characterized by absence of the extrahepatic bile ducts, proliferation of the small intrahepatic bile ducts, periporal fibrosis, and occasionally multinucleated giant cells. There is a spectrum of changes, depending on the extent of the obliterative process. Complete atresia is present in 75% to 85% of cases. In the remaining cases, there may be patency of the gallbladder and cystic duct or patency of only the gallbladder. Associated
anomalies are common (10%–20% of patients) and include choledochal cyst, polysplenia, pre-duodenal portal vein, azygous continuation of the inferior vena cava, diaphragmatic hernia, situs inversus, and hydronephrosis [16–18].

Patients who have biliary atresia and neonatal hepatitis usually present at 1 to 4 weeks of age with jaundice. Distinguishing between neonatal hepatitis and biliary atresia is important, because biliary atresia requires early surgical intervention to prevent biliary cirrhosis, whereas neonatal hepatitis is managed medically. Surgical treatment varies with the level of obstruction [7,8]. When there is extrahepatic biliary obstruction (15%–25% of cases), a direct anastomosis between the patent portion of the extrahepatic bile duct and intestine is performed. When there is intrahepatic biliary atresia, a Kasai hepatoportoenterostomy (anastomosis of a segment of small bowel to the portal region) is performed [7,8,19]. The success rate of the Kasai procedure is inversely proportional to patient age. Bile flow can be re-established in up to 90% of infants who are younger than 2 months of age at the time of hepatopportoenterostomy and in approximately 50% in those who are 2 to 3 months. The success rate decreases to less than 20% when surgery is performed after 90 days of age because of the presence of cirrhosis [7–9]. Liver transplantation is often required in older infants and children who have intrahepatic biliary atresia.

A spectrum of findings may be seen sonographically, reflecting the underlying histology. The liver size and parenchymal echogenicity may be normal or increased [1]. The intrahepatic ducts are typically not dilated. The extrahepatic duct is typically not visualized. A remnant of the extrahepatic duct, however, may be noted in the porta hepatitis [20–23]. This remnant appears as a triangular or tubular echogenic structure just superior to the portal vein bifurcation. This finding has been termed the triangular cord sign and correlates with fibrous tissue in the porta hepatitis at histologic examination (Fig. 2). The sign is reliable for the diagnosis of extrahepatic biliary atresia and has a specificity approaching 100% and a sensitivity of approximately 85%.

In biliary atresia, the gallbladder is usually small or absent (Fig. 3), although a normal-sized gallbladder may be seen when the atresia is distal to the insertion of the cystic duct (approximately 10% of cases). The finding of a small gallbladder (<1.5 cm in diameter) is nonspecific and may be seen with biliary atresia or neonatal hepatitis. Contractility and changes in gallbladder size after a milk feeding are rare in patients who have biliary atresia (<10% of cases) [24,25].

**Neonatal hepatitis syndrome**

The neonatal hepatitis syndrome is the term given to nonspecific hepatic inflammation that develops secondary to several different causes, including infection (cytomegalovirus, herpes simplex, toxoplasmosis, protozoa, syphilis), metabolic defects (alpha 1-antitrypsin deficiency, galactosemia, glycogen storage disease, tyrosinosis), and Alagille syndrome.
Histologic examination shows multinucleated giant cells with hepatic parenchymal disruption and little bile within the bile duct canaliculi. Similar to biliary atresia, the cause is believed to be an in utero inflammatory process and the disease process usually manifests with jaundice at 3 to 4 weeks of life.

At sonography, the liver size and echogenicity may be normal or increased, and the biliary ducts are not dilated [1,2] (Fig. 4). The gallbladder may be small, normal, or increased in size. Changes in gallbladder size after a milk feeding can occur in patients who have neonatal hepatitis, reflecting patency of the common hepatic and common bile duct [24].

**Additional imaging studies to differentiate atresia and hepatitis**

**Hepatobiliary scintigraphy**

Because the sonographic findings of biliary atresia and hepatitis overlap, hepatobiliary scintigraphy is usually performed to assess the presence or absence of bile excretion into the bowel. Infants who have biliary atresia less than 3 months of age usually show normal hepatic extraction of tracer but no excretion of the radionuclide into the small intestine (Fig. 5A), whereas infants older than 3 months of age show decreased extraction of tracer and no excretion into the bowel. In neonates who have neonatal hepatitis, parenchymal extraction is diminished but there is some excretion into the bowel (Fig. 5B).

The sensitivity and specificity of scintigraphy for the diagnosis of biliary atresia in infants less than 3 months of age is approximately 95% and 80%, respectively. The presence of small bowel activity excludes biliary atresia as the cause of jaundice. Differentiation between biliary atresia and neonatal hepatitis is more difficult when there is poor hepatocellular function.

**Magnetic resonance imaging**

MR cholangiopancreatography may also be useful in assessing the patency of intra- and extrahepatic biliary ducts [10,12]. Complete visualization of the extrahepatic biliary system excludes biliary atresia as the cause of cholestasis [10].

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**Fig. 4.** Neonatal hepatitis. Longitudinal sonogram shows diffusely increased and coarsened echogenicity. The gallbladder is small and filled with sludge (arrow).

**Fig. 5.** Hepatobiliary imaging in neonatal jaundice. (A) Biliary atresia. Hepatobiliary scan obtained 6 hours after injection of Tc-99m disofenin demonstrates hepatic uptake but absence of excretion into the central bile ducts and intestine. (B) Neonatal hepatitis. Hepatobiliary scan obtained 3 hours after injection shows radioactivity in the gallbladder (*) and within bowel (arrows). On more delayed images, there was poor clearance of radioactivity from the liver.
Cholangiography
Cholangiography is performed when other clinical or imaging findings suggest the diagnosis of biliary atresia. It may be performed percutaneously, endoscopically, or intraoperatively. Contrast medium is injected into the gallbladder.

Alagille syndrome
Alagille syndrome (also known as arteriohepatic dysplasia) is a hereditary disorder, usually an autosomal dominant trait with variable penetrance [8,26]. A deletion in the short arm of chromosome 20 has been seen in some patients [26]. It is associated with abnormalities of the liver (cholestatic jaundice), heart (most commonly peripheral pulmonic stenosis), skeleton (butterfly vertebrae and hemivertebrae), eye, kidneys, and abnormal facies (frontal bossing, deep-set eyes, bulbous tip of the nose, and pointed chin). The associated findings help to distinguish Alagille syndrome from biliary atresia. Patients typically present with jaundice in the neonatal period. Histologic examination shows paucity and hypoplasia of the interlobular bile ducts. Imaging findings are similar to those described for neonatal hepatitis.

Choledochal cyst
Choledochal cyst is a congenital dilatation of the common bile duct, with 30% of cases found to occur in the first year of life, 50% between 1 and 10 years of age, and 20% in the second decade or later [27]. The classic clinical presentation is jaundice, abdominal pain, and mass, although this triad is present in only 20% to 50% of patients [27]. This abnormality is believed to be the result of an abnormal insertion of the common bile duct into the pancreatic duct, which allows reflux of pancreatic enzymes into the biliary system. This reflux results in a chemical cholangitis, which weakens the walls of the bile duct, eventually leading to ductal dilatation [28].

Four types of choledochal cysts have been described [29]. The type 1 cyst, accounting for 80% to 90% of cases, is subdivided into type 1A, cystic dilatation of the common duct; type 1B, focal segmental common duct dilatation; and type 1C, fusiform dilatation of the common bile duct. The type 2 cyst, accounting for approximately 2% of cases, is a true diverticulum arising from the common duct. The type 3 cyst, accounting for 1% to 5% of cases, is a choledochocele involving only the intraduodenal portion of the duct. The type 4 cyst is subdivided into type 4A, multiple intrahepatic cysts and an extrahepatic cyst, and type 4B, multiple extrahepatic cysts. The type 5 cyst, or Caroli disease, consists of multiple intrahepatic biliary cysts and is considered to be a separate disorder (see later discussion). Choledochal cysts in neonates and young infants may coexist with biliary atresia [17,18].

At sonography, the choledochal cyst appears as a fluid-filled cystic mass in the region of the porta hepatitis that is separate from the gallbladder (Fig. 6). Intrahepatic biliary duct dilatation is present in approximately half of affected patients and typically is limited to the central portions of the left and right main hepatic ducts. Generalized ductal dilatation, typical of acquired obstruction, is absent. The cysts tend to be smaller and ductal dilatation is absent when there is concomitant biliary atresia [17,18]. Complications associated with choledochal cysts include cholelithiasis, choledocholithiasis, ascending cholangitis, intrahepatic abscesses, biliary cirrhosis, portal hypertension, and hepatobiliary malignancy, usually adenocarcinomas. The risk for malignancy increases with age [27].

When a choledochal cyst is demonstrated sonographically, scintigraphy with hepatobiliary agents is performed to confirm that the cystic mass communicates with the biliary system. Preoperative CT is acquired to further define the anatomy of the intrahepatic biliary tree and the distal common bile duct [13]. MR cholangiography may also be useful in the preoperative anatomic assessment of these lesions [10–12].

Spontaneous perforation of the extrhepatic bile ducts
Spontaneous perforation of the extrahepatic bile ducts is a cause of neonatal jaundice and ascites, usually affecting infants between 1 week and 4 months of age. The clinical findings include ascites, mild jaundice, failure to thrive, and abdominal distension. The serum bilirubin level is elevated, whereas other liver function tests are normal. The latter feature is helpful in differentiating perforation from neonatal hepatitis and biliary atresia, which have similar clinical findings but abnormal liver function tests. The most frequent site of perforation is the junction of the cystic and common bile ducts. Rarely the perforation involves the common hepatic duct, gallbladder, or junction of the cystic duct and gallbladder [30].

Sonography shows generalized ascites or a loculated fluid collection in the porta hepatitis [30] (Fig. 7). Echogenic debris or fine septations may be present within the ascitic fluid. The biliary tree is not dilated because it is not obstructed. Gallbladder or distal common duct calculi may be associated findings. Hepatobiliary scintigraphy is useful to confirm the diagnosis by showing leaking of radioactive tracer into the peritoneal cavity. Surgical
placement of a drainage tube in the area of perforation usually results in spontaneous closure.

**Inspissated bile syndrome**

The inspissated bile or bile-plug syndrome refers to an extrahepatic obstruction of the bile ducts by biliary sludge [2]. This condition typically affects full-term infants. Inspissated bile syndrome has been associated with massive hemolysis, hemorrhage, total parenteral nutrition, cystic fibrosis, and various intestinal diseases (Hirschsprung disease, intestinal atresias, and stenoses). Sonography shows moderately or highly echogenic bile within the gallbladder and often within dilated intra- or extrahepatic bile ducts. Although the bile is echogenic, it does not cause acoustic shadowing. The ductal dilatation may be difficult to recognize if the echogenicity of the inspissated bile and liver are similar [1,2].

**Jaundice in older infants and children**

The causes of jaundice in older children and adolescents include cystic diseases, including choledochal cyst (see earlier discussion) and Caroli disease, diseases of the hepatocytes, and inflammatory and obstructive lesions of the biliary ducts.

**Caroli disease**

Caroli disease, also known as congenital cystic dilation of the intrahepatic biliary tract, has two forms. One form is characterized by segmental, saccular dilation of the intrahepatic bile ducts, an increased frequency of calculus formation and cholangitis, and the absence of cirrhosis and portal hypertension. The other form is characterized by hepatic fibrosis, cirrhosis, and portal hypertension. Both forms of Caroli disease are associated with renal cystic disease, including renal tubular ectasia (medullary sponge kidney), cortical cysts, and autosomal recessive polycystic disease. Patients who have Caroli disease, like those who have choledochal cysts, have an increased risk for developing cholangiocarcinoma. Patients may present in the neonatal period [31], but the vast majority present as young adults who have abdominal pain, fever, and jaundice or with portal hypertension.

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**Fig. 6.** Choledochal cyst in a young boy with jaundice. (A) Longitudinal and (B) transverse sonograms through the liver demonstrate a cystic mass (C), representing the choledochal cyst, in the porta hepatis separate from the gallbladder (GB). P, pancreas. (C) Contrast-enhanced CT scan confirms the cystic mass (C), which is the dilated common bile duct.
Sonography shows multiple dilated tubular structures, typical of biliary radicals (Fig. 8). These can converge, creating larger saccular areas [31]. The portal radicals may be partially or completely surrounded by the dilated ducts (termed the central dot sign) [32] (Fig. 9). The extrahepatic bile ducts can be normal, narrowed, or associated with a choledochal cyst. Findings of portal hypertension may be observed in patients who have hepatic fibrosis.

**Byler disease**

Byler syndrome (also known as progressive familial intrahepatic fibrosis) is a familial intrahepatic cholestatic syndrome that is associated with cystic hepatic lesions and jaundice. Histologically, there is periportal fibrosis, micronodular cirrhosis, and periductal cysts. Symptoms, including jaundice, pruritus, and hepatomegaly, usually appear by the end of the first year of life. The sonographic findings

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*Fig. 7.* Spontaneous perforation of the common bile duct. (A) Transverse sonogram through the upper abdomen demonstrates ascites (A) in the perihepatic space. (B) On a more caudal image, a loculated fluid (F) collection and a calculus (arrow) are noted in the porta hepatis.

*Fig. 8.* Caroli disease. (A) Transverse Doppler sonogram shows dilatation of the intrahepatic bile ducts (arrows), which converge toward the porta hepatis. The color Doppler image helps confirm the absence of flow in the dilated ducts. Flow is seen in the portal vein (PV). (B) CT confirms saccular dilatation of the bile ducts. *(From Siegel MJ. Gallbladder and biliary tract. In: Siegel MJ, editor. Pediatric sonography. 3rd edition. Philadelphia: Lippincott Williams & Wilkins; 2002. p. 276–304; with permission).*
are multiple saccular cystic lesions, some of which may contain echogenic portal veins (the central dot sign) [33]. Unlike Caroli disease, the cysts in Byler disease do not communicate with the bile ducts.

**Hepatocellular diseases**

Hepatocellular disease can be classified into two major classes: infectious (acute and chronic hepatitis) and noninfectious (metabolic disorders, drugs, toxins, and autoimmune diseases). The sonographic appearance of the liver depends on the severity of the insult, rather than on the causative agent [1]. Sonography is usually normal in cases of mild acute infectious hepatitis. Sonographic findings in severe acute hepatitis include hepatomegaly, decreased parenchymal echogenicity, and increased echogenicity of the portal venule walls (starry sky liver) (Fig. 10). The gallbladder wall may be small, thick-walled, and filled with intraluminal sludge. In chronic active hepatitis, the liver often appears heterogeneous and hyperechoic with irregular margins and decreased visualization of the portal venous radicles (Fig. 11). The gallbladder may be small and contain thick bile, sludge, or stones, and collateral vessel formation may be noted.

Metabolic causes of jaundice include Wilson disease, cystic fibrosis, glycogen storage disease, tyrosinemia, and α1-antitrypsin deficiency. The sonographic appearance of these disorders is nonspecific and can be similar to that of acute or chronic hepatitis. A definitive diagnosis requires correlation with clinical information and laboratory results, and in many cases biopsy is needed to confirm the diagnosis.

**Inflammatory diseases of the biliary ducts**

**Sclerosing cholangitis**

Sclerosing cholangitis is a chronic cholestatic disorder characterized by inflammatory obliterative fibrosis of the extrahepatic and intrahepatic bile ducts leading to biliary cirrhosis and ultimately liver failure [34]. This entity has been associated with chronic inflammatory bowel disease, Langerhans histiocyotosis X, and immunodeficiency disorders [34]. Histologic examination shows multiple segmental strictures, diverticula formation between areas of stricture, and mural thickening of the bile ducts. Clinical manifestations include jaundice and right upper quadrant pain. Most affected patients are adolescents or adults.

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**Fig. 9.** Caroli disease. Transverse sonogram shows dilated ducts (arrows) that completely envelope the portal radicals. This appearance is termed the central dot sign.

**Fig. 10.** Acute hepatitis. Starry sky liver. Sagittal sonogram of the liver shows brightly echogenic portal venous triads. The liver was also moderately enlarged.

**Fig. 11.** Chronic hepatitis. Longitudinal sonogram of the liver shows an enlarged liver with irregular margins and diffusely coarse echotexture.
Sonographic findings include a thick-walled gallbladder, thick-walled dilated intrahepatic ducts (Fig. 12), intrahepatic and intraductal stones, cholelithiasis, and segmental ductal narrowing secondary to strictureing. The strictures may be difficult to detect sonographically unless the ducts are dilated. In longstanding disease, sonography may show findings of biliary cirrhosis and portal hypertension.

**AIDS-related cholangitis**

The common biliary tract abnormalities in children who have AIDS are acalculous cholecystitis and cholangitis. The sonographic findings in AIDS-related cholangitis are similar to those of sclerosing cholangitis and include ductal dilatation and wall irregularity (Fig. 13), stricture of the intra- and extrahepatic bile ducts, and a dilated, thick-walled gallbladder wall [35–37]. An additional finding is a hyperechoic nodule in the distal end of the common bile duct caused by edema of the papilla of Vater [38].

**Biliary tract obstruction**

**Sonographic features**

Biliary obstruction resulting in jaundice is usually the result of stone disease. Acute pancreatitis, neoplasm, and benign strictures are less common causes of obstructive jaundice in children. The sonographic diagnosis of biliary obstruction is based on the demonstration of dilated intrahepatic or extrahepatic bile ducts. Dilated intrahepatic biliary radicles appear as multiple, anechoic branching structures that enlarge as they approach the porta hepatis. The dilated common hepatic and common bile ducts may appear as round or tubular anechoic structures near the porta hepatis or the head of the pancreas (Fig. 13).

**Cholelithiasis and choledocholithiasis**

Stones in the common bile duct usually originate in the gallbladder and migrate distally. Cholelithiasis in neonates has been associated with congenital anomalies of the biliary tract, total parenteral nutrition, furosemide therapy, phototherapy, dehydration, infection, hemolytic anemias, and short-gut syndrome [9]. In older children, causes of cholelithiasis include sickle cell disease, cystic fibrosis, malabsorption, total parenteral nutrition, liver disease, Crohn disease, bowel resection, and hemolytic anemia.

Choledocholithiasis typically manifests as brightly echogenic shadowing foci within the

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**Fig. 12.** Sclerosing cholangitis. Longitudinal sono-gram shows a dilated, thick-walled common bile duct (arrows) (diameter, 9 mm). PV, portal vein; SV, splenic vein.

**Fig. 13.** AIDS-related cholangitis. Longitudinal sono-gram at the level of the porta hepatitis demonstrates intrahepatic ductal dilatation (arrows). (Courtesy of Edward Lee, MD, Boston, MA).

**Fig. 14.** Choledocholithiasis. Longitudinal scan at the level of the pancreatic head (P) shows a stone (between calipers) without shadowing. At operation, calcium bilirubinate stones were found in the gallbladder and distal duct. Ductal diameter is 11 mm.
biliary ducts and is usually associated with ductal dilatation (Fig. 14). The calculi obstruct anywhere in the biliary duct, but most cause obstruction at the level of the pancreatic head. The sensitivity for detection of choledocholithiasis is lower for stones in the distal versus the proximal duct. A calculus impacted in the distal duct can be more difficult to detect because of adjacent or overlying bowel gas and because the calculus is surrounded by the echogenic pancreatic head.

**Cystic duct stones**

Mirizzi syndrome is a rare cause of biliary obstruction in children. It is secondary to an impacted cystic duct stone, which causes extrinsic compression or inflammatory stricture of the common duct. Sonographic findings include calculi in the gallbladder neck or in the cystic duct and dilated intrahepatic ducts, including the common hepatic duct (Fig. 15).

**Biliary neoplasms**

Rhabdomyosarcoma of the biliary tract is rare, but it is the most common neoplasm of the biliary tract in children. Most rhabdomyosarcomas arise in the porta hepatitis and involve the cystic duct. Sonographic findings are intra- and extrahepatic ductal dilatation and an echogenic mass without acoustic shadowing. Rhabdomyosarcoma spreads by direct extension to contiguous structures or by hematogenous or lymphatic dissemination to lymph nodes, lungs, bone, bone marrow, or liver.

**Biliary duct strictures**

Biliary stricture is an uncommon cause of distal obstruction, but the diagnosis needs to be considered in patients who have biliary obstruction in whom no calculus or other obstructing lesion can be visualized. An abrupt transition from a dilated duct to one of normal caliber is a finding suggestive of stricture.

**References**


