Ultrasound of the Extrahepatic Bile Duct

Issues of Size

Mindy M. Horrow, MD, FACR

Abstract: Ultrasound is a pivotal study for evaluation of the biliary tree. In particular, the size of the extrahepatic bile duct is a critical measurement and has been a contentious issue since the early days of diagnostic ultrasound. This article reviews the history and ongoing issues regarding sonoanatomy of the normal-size duct and a variety of factors that may affect its size, including age, prior surgery, congenital abnormalities, anatomical variations, and medications. Other related sonoanatomic issues are discussed including abnormal nondilated ducts and abnormal intraluminal contents such as sludge or air that make evaluation of the duct more difficult, particularly in patients with primary sclerosing cholangitis and prior liver transplantation. Ultimately, the luminal size of the extrahepatic duct should be considered as a single part of the entire assessment of the biliary tree that must also include the intraparenchymal and pancreatic ducts, the pattern of dilatation (variable vs progressively dilated to a single point of obstruction), any wall thickening, intraluminal sludge, calculi or mass, and extraluminal compression. Clinical symptoms and abnormal laboratory values should prompt further evaluation despite a normal appearance of the bile duct, whereas pursuit of an isolated clinical finding of an enlarged duct without supporting clinical data may not be warranted.

Key Words: extrahepatic bile duct, ultrasound, measurement, age, cholecystectomy

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Evaluation of the biliary tree continues to be one of the most preeminent uses of ultrasound imaging, even in the current era of endoscopic retrograde cholangiopancreatography (ERCP), magnetic resonance cholangiopancreatography (MRCP), and endoscopic ultrasound (EUS). In particular, the size of the extrahepatic duct is a critical measurement in the determination of biliary obstruction. Often, ultrasound is the first imaging study for patients with suspected biliary disease, and thus, high sensitivity for biliary dilatation is required. In many patients, biliary dilatation does indicate obstruction. However, such an assessment implies knowledge of the normal-size duct. A variety of factors, most prominently age and prior cholecystectomy, have been reported to have an effect on the size of the bile duct. Moreover, the appearance of the bile duct must be considered in addition to the luminal diameter. This article reviews the history and ongoing issues regarding sonoanatomy of the normal-size duct and includes a discussion with examples of abnormal nondilated ducts and pitfalls in evaluation of the bile ducts.

Does the Extrahepatic Bile Duct Dilate With Advancing Age?

In 1979, a landmark study by Parulekar1 established an upper limit of normal for the common bile duct of 7.0 mm in a population of 73 patients aged between 20 and 65 years. In the early 1980s, reports of increasing duct size with increasing age began to appear. In a population of 350 patients, Kaude2 reported a mean duct diameter of 2.8 mm at age 20 years and 4.1 mm at 71 years or older. The study of Wu et al3 of 256 patients aged between 10 and 70 years showed significant age dependence, with a range of 1 to 10 mm, increasing with age. Inclusion of pediatric patients in this cohort forced the regression analysis to show a significant effect of age. Nonetheless, this study was the basis for the widely accepted guideline of the normal bile duct increasing a millimeter in size for each decade of life so that 5 mm is normal in the 50s and 6 mm in the 60s, and so on.

More recent studies have revisited the age-size relationship and found a weaker correlation. In 2000, a large study4 of more than 1000 patients older than 60 years of age found the mean bile duct increased with age from 3.6 ± 0.26 mm at age 60 years to 4 ± 0.25 at 85 years or older. Despite this mild increase with age, 98% of ducts were less than 7 mm. In 2001, Horrow et al5 analyzed extrahepatic bile duct measurements in 258 people aged between 20 and 92 years to test the hypothesis of a slope of 1.0 mm per decade and found no association with age. The mean anteroposterior diameters of the proximal (porta hepatis), mid, and distal (head of pancreas) bile duct were 2.9, 3.5, and 3.5 mm. Finally, a study in 2003 by Bachar et al6 found a gradual dilatation of the bile duct at a rate of 0.04 mm/y. Even in their patients older than 80 years, the range was within 3.9 to 7.1 mm. However, because of 3 patients older than 60 years with ducts of 8.5 to 8.6 mm who were otherwise normal, they suggested that 8.5 mm be considered the upper limit of normal in older patients. In our experience, however, one must be skeptical of reporting an 8-mm bile duct as normal without correlating the clinical findings (Fig. 1).

Accurate measurement of the extrahepatic bile duct requires strict attention to technique. In a fasting patient, one should obtain a longitudinal view of the duct at the porta hepatis where the duct is anterior and parallel to the portal vein. Measurements should be made with calipers from inner...
to inner walls with an appropriately sized image. The upper limit of 6 to 7 mm applies to this location, where one is actually measuring the common hepatic duct. Images and measurements should also be obtained in the mid duct and within the head of the pancreas, with the understanding that the duct may be slightly larger at these locations (Fig. 2). Usually, the cystic duct is so small that it cannot be imaged. In patients with a low insertion of the cystic duct into the common duct, care must be taken not to include the cystic duct in the measurement of the common duct (Fig. 3). Standard sonographic measurements of the extrahepatic bile duct represent the height of the duct. Wachsberg et al\(^7\) reported an oval shape of the duct in 70% of their study population so that a transverse (width) measurement may be slightly greater (Fig. 4).

Other imaging modalities may yield slightly greater bile duct measurements. On computed tomography (CT) and magnetic resonance imaging, the bile duct wall is included in the measurement, increasing it by 1 to 2 mm. Because the extrahepatic duct has an oblique course, reliance on the axial source images may result in inaccurate measurements. It may

**FIGURE 1.** A and B, An 83-year-old woman with 8.1-mm duct, without demonstrable cause on ultrasound, has distal calculus on MRCP (arrow).

**FIGURE 2.** A and B, Sagittal views of the common hepatic (A) and common bile duct (B). Duct size at porta (1) and in pancreatic head (3) was 3 to 4 mm. Mid duct (2) measured 8 mm in this patient without symptoms or signs of biliary disease.

**FIGURE 3.** Sagittal view of extrahepatic bile duct with low insertion of cystic duct (arrow) at level of pancreatic head (P).
also be difficult to separate a low cystic duct insertion. Measurements from ERCP and transhepatic cholangiography may be slightly greater than on ultrasound because of magnification and duct distention with contrast. In addition, measurements obtained from these modalities are the duct width, rather than the height. Because the duct is slightly oval, this tends to overestimate duct size. In addition, one must consider the acuity of symptoms. The bile duct can change rapidly in caliber as an obstruction occurs or clears. In summary, although there are multiple reports of normally increasing size of the bile duct with age, the degree of increase is probably slight and results in a wide spectrum of normal at advanced ages.

**Does the Extrahepatic Bile Duct Dilate After Cholecystectomy?**

The concept of postcholecystectomy bile duct dilatation is frequently referenced in imaging interpretations. A pre-ultrasound era study of cadaver dissections from the surgical literature in 1935 reported dilatation of the common bile duct after cholecystectomy. Ultrasound-based studies from the early 1980s to the present have examined this issue with varying conclusions. In 1980, Graham et al reported on 67 asymptomatic patients undergoing repeated ultrasound after cholecystectomy for up to 16 months. The common bile duct remained normal in size in the majority, but in 16%, the duct measured up to 10 mm in diameter without cause. Mueller et al, one year later reported on 40 patients studied both before and after cholecystectomy, with 38 of 40 showing no change, one duct enlarging, and one decreasing after surgery. Almost half of the patients had a common duct exploration. Both the Graham et al and Mueller et al studies obtained their measurements of the bile duct at the porta hepatis and did not evaluate the mid and pancreatic portions of the duct.

More recent studies with longer follow-up show minimal increases in bile duct size after cholecystectomy. In a large study of 234 patients imaged both before and after surgery, the mean diameter of the common bile duct before cholecystectomy was 5.9 mm and afterward was 6.1 mm. Although this difference was statistically significant, the authors conclude that most patients do not undergo significant postcholecystectomy duct dilatation. Majeed and Johnson followed a group of 59 patients with ultrasound studies before, at 3 and 6 months, and at 1 and 5 years after open cholecystectomy. The mean diameter before surgery was 3.43 mm and at 5 years was 3.96 mm. Using a 1-mm margin of error in measurement, there was no statistical difference. An editorial by Wilkinson that accompanied this article concluded that the bile duct tends to dilate very slightly after cholecystectomy, particularly in older patients. However, in asymptomatic patients with incidental dilatation, he recommends no further evaluation. Conversely, symptomatic patients, even with a normal-size duct, deserve further imaging. The anecdotal experience of this author and others is that the combination of cholecystectomy and advancing age tends to associate with larger extrhepatic bile ducts. This phenomenon occurs most often in the middle segment where the duct is not surrounded by either liver or pancreas (Fig. 2). This pattern of dilatation, effecting only or predominantly the mid portion of the extrhepatic duct, may actually be more typical of a nonobstructed, ectatic duct.

**Is There Any Significance to Bile Duct Dilatation in Asymptomatic Patients?**

It is probably not surprising that there is scant literature about asymptomatic patients with a dilated bile duct of
unknown etiology on standard imaging studies. The collective clinical experience of most physicians and the inclination of most patients would be to ignore an imaging finding without clinical or laboratory abnormalities. A few small studies report on such patients with modest numbers of patients having a cause for dilatation, usually benign. A study by Kim et al\textsuperscript{14} of 77 asymptomatic patients with a bile duct diameter of greater than 7 mm found no cause in more than half (n = 40) of their cohort. The abnormalities in the other 37 patients included periampullary duodenal diverticulum (Fig. 5), benign stricture, choledochal cyst, anomalous ductal anatomy, and 2 unspecified ductal masses. Malik et al\textsuperscript{15} conducted a retrospective review of 47 patients referred for EUS with common duct dilatation (mean, 8.6 mm) unexplained by other imaging studies. This group was divided into 32 patients with normal serum liver enzymes and 15 with elevated enzymes. Abnormalities were found on EUS in 53% of those with elevated enzymes compared with 6% who were normal. The findings included choledocholithiasis, periampullary diverticulum, chronic pancreatitis, and an ampullary tumor. The tumor and most of the choledocholithiasis cases were in the group with abnormal enzymes. A prospective study of 90 patients by Songur et al\textsuperscript{16} compared EUS and ERCP after abdominal ultrasound failed to find a cause for biliary dilatation. All patients had a common bile duct 7 mm or greater with either right upper quadrant pain and/or abnormal liver function studies. Twenty-eight of these patients had a prior cholecystectomy. Ultimately, 24 patients (27%) had no cause for the dilatation. Of the remainder, the most common cause in 40 (44%) was choledocholithiasis. Other causes were tumors (n = 13), benign stricture (n = 8), choledochal cyst (n = 2), and ova of \textit{Ascaris} (n = 1). There was complete agreement between EUS and ERCP in 92.5%.

Occasionally, the cause of biliary dilatation may be functional. Sphincter of Oddi dysfunction is a disorder caused by spasm or stenosis of the biliary sphincter and/or the pancreatic sphincter. These patients have severe epigastric or right upper quadrant pain relieved by sphincterotomy.\textsuperscript{17} Mild biliary dilatation has also been reported in chronic opioid users, probably due to an effect on the sphincter of Oddi.\textsuperscript{18} A rare cause of biliary dilatation is a paraneoplastic syndrome associated with adenocarcinoma of the lung.\textsuperscript{19}

Choledochal cysts are a congenital cause of biliary dilatation and usually diagnosed in childhood. However, some patients, possibly as many as 25%, may remain undetected until adulthood. Visser et al\textsuperscript{20} reported a series of 38 adult patients with choledochal cysts. They propose that the current grading system of choledochoceles is unhelpful because it is a conglomerate of unrelated entities including choledochal diverticula, choledochoceles, and Caroli disease. Most patients in their series had choledochal cysts, which they postulate are due to an anomalous joining of the pancreatic and common bile ducts 1 to 2 cm proximal to the sphincter of Oddi. This common channel exposes the bile duct to reflux of pancreatic enzymes and eventual dilatation. The extremely high likelihood of developing cholangiocarcinoma leads these authors to recommend full excision of the duct with hepatojejunostomy.

Ultimately, the extent to which one investigates a patient with unexplained biliary dilatation depends on the presence of...
clinical symptoms and abnormal laboratory values, the probability of an underlying abnormality, and the appropriateness of further therapy. Patients with clinical symptoms and/or abnormal liver function studies are more likely than completely asymptomatic patients to have a discoverable cause for the dilatation. A reasonable recommendation, when more invasive investigation is unwarranted, is to obtain a follow-up ultrasound in several months. Stable findings would be reassuring and mitigate against further evaluation.

**Is There More to Sonography of the Bile Duct Than Size Alone?**

The major possibilities in this group are thickening of the wall of the bile duct or an intraluminal abnormality without dilatation. Thickening of the wall of the bile duct is relatively uncommon and probably underappreciated. Thickening of the wall of the bile duct may be associated with a normal, narrow, or dilated lumen. Because bile duct diameter usually refers to the size of the lumen, one must be vigilant to properly measure the lumen, the wall, and the entire duct. Wall thickening can be related to a variety of causes including pancreatitis, particularly autoimmune pancreatitis (Fig. 6); primary sclerosing cholangitis; acute infectious cholangitis (Fig. 7), including Oriental cholangiohepatitis; AIDS-related biliary disease usually associated with opportunistic infections; and cholangiocarcinoma. Eccentric wall thickening and a thickness greater than 5 mm are suggestive of neoplasm. Smooth biliary strictures with wall thickening can occur in liver transplant patients when the biliary tree is subject to ischemia (Fig. 8). Thickening of the bile duct can be simulated by intramural collaterals in patients with portal hypertension and portal vein thrombosis.

Choledocholithiasis may occur in the absence of appreciable biliary dilatation. Usually, patients are asymptomatic. However, we have noticed outpatients without pain who have mobile calculi and/or tumefactive sludge within a normal-size duct (Fig. 9).
When Is It More Difficult to Detect Biliary Dilatation by Ultrasound?

When the bile ducts are not filled with anechoic bile or the pattern of dilatation is more unusual, it may be more difficult to appreciate biliary dilatation. This situation occurs particularly in 2 groups: liver transplant recipients and patients with primary sclerosing cholangitis. In both groups, the bile ducts may dilate and fill with echogenic sludge. In addition, both types of patients may have a pattern of intrahepatic and extrahepatic biliary dilatation consisting of alternating dilatation and stenosis, rather than a smoothly arborizing pattern of dilated ducts that progressively enlarge toward the extrahepatic bile duct.

The older transplant literature suggests that sonography is relatively insensitive and thus unreliable for biliary dilatation in liver transplant patients. Actually, one can visualize these dilated ducts by following the pattern of echogenic branching periportal tubules extending to the extrahepatic bile duct.

FIGURE 10. A and B, Liver transplant patient with proven hepatic artery thrombosis and collateralization of the hepatic artery has dilated sludge-filled intrahepatic ducts (arrows) (A) and extrahepatic duct (B).

FIGURE 11. A and B, A patient with primary sclerosing cholangitis has significantly dilated intrahepatic and extrahepatic ducts, filled with echogenic sludge (arrows), confirmed on ERCP.

FIGURE 12. Patient with lengthy stay in an intensive care unit has a dilated gallbladder (G) and a dilated extrahepatic bile duct (arrow). Both are filled with echogenic sludge.
duct. Scrolling through the liver in real time facilitates this process (Fig. 10). If biliary sludge is detected in a liver transplant patient, one must carefully evaluate the hepatic artery. Because the reconstructed hepatic artery is the only blood supply to the donor bile ducts, high-grade stenosis or thrombosis of the hepatic artery frequently results in ischemia of the bile ducts and sloughing of the mucosa, yielding “sludge.” This process can also occur with generalized hypotension, prolonged cold preservation of the donor liver, chronic rejection, and recurrent or ascending cholangitis.

Primary sclerosing cholangitis may cause inflammation and fibrosis of any portion of the biliary tree, resulting in strictures and dilatation with cholestasis. Eventually, this may progress to cirrhosis and hepatic failure. In addition to wall thickening, the ducts may fill with tumefactive sludge that blends with the liver, which may also be abnormally echogenic and heterogeneous (Fig. 11). Sludge-filled ducts can also be seen in adult patients with cystic fibrosis and occasionally in patients with sludge-filled gallbladders (Fig. 12).

Gas and blood and occasionally tumors can also fill the duct, making it more difficult to measure the lumen. Hemobilia can be secondary to trauma, inflammation, or coagulopathy. Pneumobilia is usually iatrogenic or due to sphincterotomy or surgical procedures (Fig. 13). Other causes include infection, trauma, and biliary enteric fistula. Intraductal tumors usually cause distal dilatation that is more easily appreciated (Fig. 14).

**CONCLUSIONS**

In no other imaging study of the biliary tree is a single measurement accorded such emphasis as in sonography of the extrahepatic bile duct. Interpretations from CT, MRCP, and ERCP usually are more descriptive, giving a global impression. Ultrasound interpretation should also take a wider view. Thus, the size of the bile duct must be evaluated as a single piece of data, to be interpreted as part of a complete examination. The interpretation must include an assessment of any intrahepatic biliary dilatation, the pattern of dilatation (gradually increasing to the level of obstruction vs isolated patches of dilated ducts), wall thickening or luminal contents (sludge, calculi or mass), and external compression of the duct. Further evaluation must take the clinical situation into account. The combination of advanced age and cholecystectomy may result in an ectatic, slightly dilated extrahepatic duct, especially in the mid portion. Symptoms and abnormal laboratory values should prompt further imaging of a seemingly normal duct on ultrasound. Conversely, pursuit of an isolated finding of biliary dilatation without supporting clinical data may not be justified.
REFERENCES