Biliary Duct Dilatation: *AJR* Expert Panel Narrative Review

Daniel R. Ludwig, MD[1], Malak Itani, MD[1], David D. Childs, MD[2], Margarita V. Revzin, MD, MS[3], Koushik K. Das, MD[4], Mark A. Anderson, MD[5], Hina Arif-Tiwari, MD[6], Mark E. Lockhart, MD, MPH[7], Ann S. Fulcher, MD[8]

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Biliary duct dilatation is a common incidental finding in practice, but it is unlikely to indicate biliary obstruction in the absence of clinical symptoms or elevated levels on liver function tests (LFTs). However, the clinical presentation may be nonspecific, and LFTs may either be unavailable or difficult to interpret. The goal of this *AJR* Expert Panel Narrative Review is to highlight a series of topics fundamental to the management of biliary duct dilatation, providing consensus recommendations in a question-and-answer format. We start by covering a basic approach to interpreting LFT results, the strengths and weaknesses of the biliary imaging modalities, and how and where to measure the extrahepatic bile duct. Next, we define the criteria for biliary duct dilatation, including patients with prior cholecystectomy and advanced age, and discuss when and whether biliary duct dilatation can be attributed to papillary stenosis or sphincter of Oddi dysfunction. Subsequently, we discuss two conditions in which the duct is pathologically dilated but not obstructed: congenital cystic dilatation (i.e., choledochal cyst) and intraductal papillary neoplasm of the bile duct. Finally, we provide guidance regarding when to recommend obtaining additional imaging or testing, such as endoscopic ultrasound or ERCP, and include a discussion of future directions in biliary imaging.

Biliary duct dilatation is a common finding in clinical practice and is often incidental. In the absence of clinical symptoms or elevated levels on liver function tests (LFTs), a dilated bile duct is unlikely to imply biliary obstruction. However, the clinical context and relevant laboratory data are often unavailable to the interpreting radiologist. Even if available, the clinical symptoms and pattern of liver chemistry abnormality may be nonspecific or challenging to interpret. The American College of Radiology (ACR) provides general guidance for managing incidental biliary duct dilatation [1], which is helpful but unfortunately is not applicable in many common clinical contexts. Furthermore, the ACR does not define a management algorithm for further workup when clinically indicated.

The goal of this *AJR* Expert Panel Narrative Review is to present consensus recommendations for the management of biliary duct dilatation, including how to incorporate liver chemistries, what defines biliary duct dilatation, and when to consider dilatation suspicious for or even consistent with biliary obstruction. We also provide guidance regarding when to obtain additional imaging or testing, such as endoscopic ultrasound (US) or ERCP. Our recommendations will be presented through a series of questions and answers. This article is not intended to serve as a comprehensive review or to provide an initial imaging strategy for patients with elevated levels on LFTs or right upper quadrant pain; such topics are covered in the ACR Appropriateness Criteria and clinical society guidelines [2–5].

Why Is Biliary Duct Dilatation Challenging in Clinical Practice?

Biliary duct dilatation can be challenging for a number of reasons. First and foremost, in many conditions, the bile duct may be dilated but not obstructed (Table 1). The two

[1]Mallinckrodt Institute of Radiology, Washington University School of Medicine, 510 S Kingshighway Blvd, Campus Box 8131, Saint Louis, MO 63110. **Address correspondence to** D. R. Ludwig (ludwigd@wustl.edu).

[2]Department of Radiology, Wake Forest University School of Medicine, Winston-Salem, NC.

[3]Department of Radiology, Yale School of Medicine, New Haven, CT.

[4]Division of Gastroenterology, Washington University School of Medicine, Saint Louis, MO.

[5]Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA.

[6]Department of Radiology, University of Arizona College of Medicine, Tucson, AZ.

[7]Department of Radiology, The University of Alabama at Birmingham, Birmingham, AL.

[8]Department of Radiology, Medical College of Virginia/Virginia Commonwealth Medical Center, Richmond, VA.

most common such reasons encountered in practice are prior cholecystectomy, in which the bile duct functions as the biliary reservoir, and advanced age [6]. Prior liver transplant is associated with biliary duct dilatation in the absence of an underlying stricture, which may relate to donor or recipient cholecystectomy and/or sphincter of Oddi denervation [7]. Patients with gallbladder dyskinesia, chronic cholecystitis, or other forms of gallbladder dysfunction may also have dilated bile ducts, through mechanisms similar to those of cholecystectomy. Prior biliary obstruction may result in persistent duct dilatation due to a patulous system after the obstruction has resolved [8]. Chronic opiate use is an increasingly common cause of biliary duct dilatation given its myogenic effect on the sphincter of Oddi [9, 10]. Periampullary duodenal diverticula are ubiquitous in older patients and very rarely may cause biliary obstruction when impacted (i.e., Lemmel syndrome) [11]. However, much more commonly, they exert mass effect on the distal bile duct and are associated with nonobstructive biliary duct dilatation [12, 13].

The duct is pathologically dilated but not obstructed in two conditions: congenital cystic dilatation (i.e., choledochal cyst) and intraductal papillary neoplasm of the bile duct (IPN-B); these will be covered in detail in subsequent sections. To further complicate matters, patients with biliary obstruction may not have significant biliary duct dilatation. For example, a patient with early or intermittent obstruction may not have dilated bile ducts at the time of presentation. In addition, in primary sclerosing cholangitis (PSC), periductal fibrosis may limit the degree to which obstructed bile ducts dilate [14].

What Are the Biliary-Associated Enzymes?

LFTs are usually the first laboratory tests performed to assess for hepatobiliary disease, and they frequently determine the need for abdominal imaging. The serum albumin level and prothrombin time are true markers of hepatocellular synthetic function, whereas the popularly termed "liver function tests" are standard serum tests that are used to investigate hepatocyte derangement (transaminitis) using alanine transaminase (ALT) and aspartate transaminase (AST) levels, assess for biliary insult using alkaline phosphatase (ALP) and γ-glutamyl transpeptidase (GGT) levels, and evaluate impaired bile transport or excretion or increased bile production due to hemolysis using the serum bilirubin (conjugated or unconjugated) level [2, 15]. Patterns of elevations in LFT levels may classify the hepatic insult as hepatocellular predominant, cholestatic predominant, or biliary metabolism predominant (Table 2).

ALP, GGT, and conjugated bilirubin levels are elevated when the liver injury pattern is cholestatic. When biliary-associated enzyme levels are elevated, imaging is performed to assess for biliary duct dilatation, which will aid in the distinction between intrahepatic and extrahepatic cholestasis (i.e., biliary obstruction) [4, 16]. Elevation of these enzyme levels is sensitive for obstruction but may not be specific. Thus, in a patient with incidental biliary duct dilatation, correlation with biliary-associated enzymes is critical to guide further management.

What Are the Biliary Imaging Modalities and Their Trade-Offs?

The biliary imaging modalities include US, CT, MRI/MRCP, endoscopic US, and ERCP; the advantages and disadvantages of each are summarized in Table 3.

Ultrasound

US is often considered the initial modality of choice for assessment of the biliary system in patients with suspected biliary obstruction [2, 3]. It is cost-effective, does not deliver ionizing radiation, provides real-time images, and can be performed portably in unstable patients [17]. It is highly sensitive for the detection of cholelithiasis and has a very high NPV for biliary obstruction in a patient with bile ducts with a normal caliber [18]. However, US is operator dependent and may be limited by the presence of overlying bowel gas or body habitus, leading to limited sensitivity (72%) for choledocholithiasis [19]. Even when biliary obstruction is present, US may not reveal the level and cause. US is an effective screening test, although the presence of biliary duct dilatation or indeterminate findings often requires further characterization with MRI/MRCP or CT.

CT

CT provides superior visualization of the entire bile duct, particularly when IV contrast media are administered, and can reliably exclude biliary obstruction or show the location and cause of obstruction when present [20]. CT is widely available, and images can be acquired in a rapid and noninvasive fashion. An important

TABLE 1: Differential Considerations for Nondilated and Dilated Bile Ducts in Conditions With or Without Biliary Obstruction

Duct	No Obstruction	Obstruction
Nondilated	Normal	Early or intermittent obstruction Primary sclerosing cholangitis
Dilated	Advanced age Prior cholecystectomy Liver transplant Gallbladder dysfunction (dyskinesia, chronic cholecystitis, etc.) Prior, resolved biliary obstruction Chronic opiate use Periampullary duodenal diverticulum Choledochal cyst[b] Intraductal papillary neoplasm of the bile duct[b]	Choledocholithiasis Mirizzi syndrome Portal biliopathy Ampullary masses Papillary stenosis or sphincter of Oddi dysfunction[a] Pancreatic masses Metastatic disease Other causes of benign and malignant strictures

[a]These are considered clinical diagnoses and not imaging diagnoses.
[b]These represent conditions in which the duct is pathologically dilated but not obstructed, whereas the additional conditions are not necessarily pathologic.

TABLE 2: Patterns of Elevations in Liver Function Test Levels and the Most Commonly Associated Causes

Pattern of Hepatobiliary Insult	Elevated Level(s)	Associated Causes
Hepatocellular predominant	Predominant elevations in ALT and AST levels reflect injury to the hepatocytes	Mild increase in aminotransferase level: increase of two to five times greater than normal level; seen in NAFLD, alcohol-induced liver disease, and chronic liver parenchymal disease Increase in aminotransferase level defined as follows: moderate, more than five to 15 times the normal level; severe, > 15 times the upper reference limit; marked, > 75 times the normal level. The following causes can result in either a moderate or severe increase in transaminase levels: acute viral hepatitis, acute Budd-Chiari syndrome, ischemic hepatitis, drugs/toxins, autoimmune condition, hemochromatosis, Wilson disease, and α-antitrypsin deficiency
Cholestatic predominant	Predominant elevations in ALP, GGT, and conjugated bilirubin levels reflect impairment in the biliary system	Intrahepatic cholestasis can be seen with primary biliary cholangitis, primary sclerosing cholangitis, IgG4-associated cholangiopathy, hepatic abscess, hepatic sarcoidosis, and drug-associated cholestasis Extrahepatic cholestasis (biliary obstruction) can be seen with benign or malignant biliary strictures, extrahepatic biliary obstruction due to benign (choledocholithiasis) or malignant causes, cholangiocarcinoma, pancreatic ductal adenocarcinoma, gallbladder adenocarcinoma, ampullary tumors, primary sclerosing cholangitis, IgG4-associated cholangiopathy, or extrinsic compression
Biliary metabolism predominant	Elevated bilirubin level implies an insult to biliary metabolism	Prehepatic: large amount of hemolysis (sickle cell disease, thalassemia, hereditary spherocytosis, glucose-6-phosphate dehydrogenase deficiency) produces excessive bilirubin, which is unconjugated Intrahepatic: mildly elevated unconjugated hyperbilirubinemia can be seen in Gilbert syndrome Posthepatic: elevated conjugated bilirubin levels (see entry for extrahepatic cholestasis under Associated Causes column entry for cholestatic-predominant insults)

Note—ALT = alanine transaminase, AST = aspartate transaminase, NAFLD = nonalcoholic fatty liver disease, ALP = alkaline phosphatase, GGT = γ-glutamyl transpeptidase.

TABLE 3: Advantages and Disadvantages of Biliary Imaging Techniques

Modality	Advantages	Disadvantages
US	Initial test of choice for gallbladder and biliary pathology Very high (> 95%) sensitivity in detecting gallstones in the gallbladder Can be used to assess for sonographic Murphy sign Can assess mobility of intraluminal structures with changes in position Normal caliber CBD has very high NPV for biliary obstruction (> 95%) Readily available No ionizing radiation Low cost	Operator dependent May not depict the cause of biliary obstruction Limited evaluation of distal CBD due to overlying bowel gas Suboptimal images in obese patients Low to moderate (72%) sensitivity for choledocholithiasis [19]
CT	Sensitivity and specificity of > 90% for detection of biliary obstruction Frequently localizes the site and cause of obstruction Simultaneously evaluates other organs in the abdomen Readily available Rapid acquisition	Limited sensitivity (< 80%) for detecting stones in the gallbladder and bile duct Delivers ionizing radiation
MRI/MRCP	Highest sensitivity and specificity for biliary obstruction Almost always localizes the site and cause of obstruction No ionizing radiation	May be compromised by artifacts and patient motion Time intensive (> 30 min) Limited availability in emergency setting
Endoscopic US	Very high sensitivity and specificity for biliary obstruction Allows simultaneous FNA/biopsy and biliary drainage Can be performed simultaneously with ERCP	Requires sedation Narrow imaging field Risk of postprocedural complications if simultaneous intervention performed Higher cost Technically limited by postsurgical anatomy (e.g., gastric bypass, chronic calcific pancreatitis, or large periampullary diverticulum)
ERCP	Allows simultaneous intervention: sphincterotomy, stone extraction, biopsy, stenting, brushing	Risk of acute cholangitis by contamination of otherwise sterile biliary tree Risk of complications approximately 7% May not evaluate biliary tree upstream of significant stricture Technically limited by postsurgical anatomy (e.g., gastric bypass)

Note—US = ultrasound, CBD = common bile duct, FNA = fine-needle aspiration.

limitation of CT is its relatively low sensitivity for depicting noncalcified biliary stones, which are often isoattenuating to the adjacent bile [21]. Dual-energy CT may improve the performance of CT for isoattenuating stones, although it still may not show noncalcified stones when they are small [22]. The biliary system is visualized on CT examinations that are obtained for nonbiliary indications, and for this reason incidental biliary duct dilatation is commonly initially encountered on CT.

MRI/MRCP

MRCP revolutionized the evaluation of the biliary tract in general, and the obstructed bile duct in particular, by allowing noninvasive

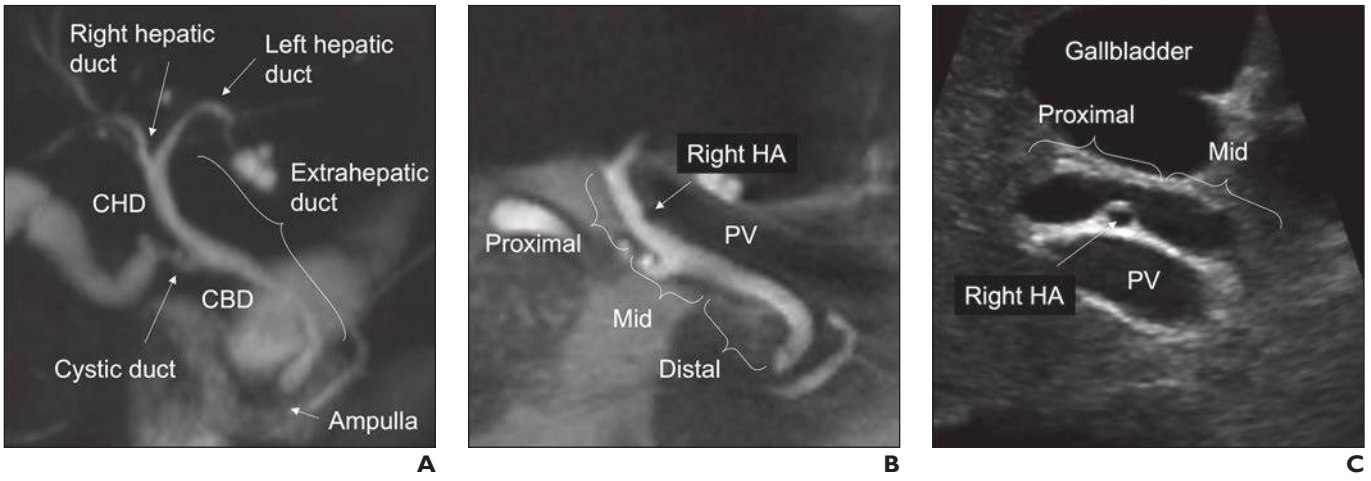

Fig. 1—Extrahepatic biliary anatomy of two patients.
A–C, Three-dimensional MRCP maximum-intensity-projection image (**A**) of 66-year-old patient shows right and left hepatic ducts converging to form common hepatic duct (CHD), which becomes common bile duct (CBD) distal to cystic duct insertion. CHD and CBD are often collectively referred to as extrahepatic duct given that cystic duct usually is not apparent on CT and ultrasound. Extrahepatic duct may be subjectively divided into proximal, mid, and distal segments, as shown on corresponding coronal T2-weighted SSFSE image (**B**) of same 66-year-old patient shown in **A** as well as on ultrasound image (**C**) of 52-year-old patient. Right hepatic artery (HA) crosses at level of proximal extrahepatic duct, whereas mid duct is conventionally defined as location where it is anterior and parallel to portal vein (PV).

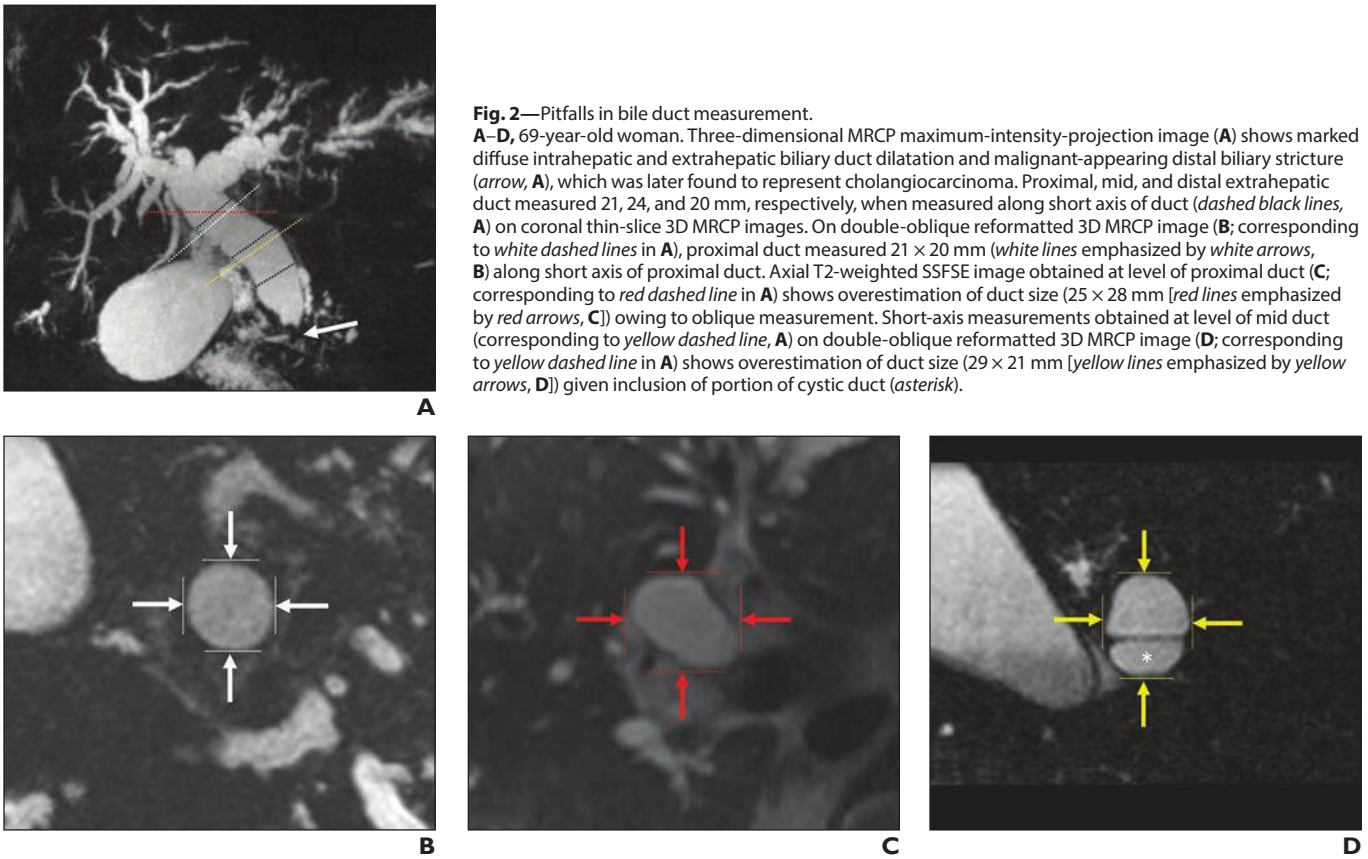

Fig. 2—Pitfalls in bile duct measurement.
A–D, 69-year-old woman. Three-dimensional MRCP maximum-intensity-projection image (**A**) shows marked diffuse intrahepatic and extrahepatic biliary duct dilatation and malignant-appearing distal biliary stricture (*arrow,* **A**), which was later found to represent cholangiocarcinoma. Proximal, mid, and distal extrahepatic duct measured 21, 24, and 20 mm, respectively, when measured along short axis of duct (*dashed black lines,* **A**) on coronal thin-slice 3D MRCP images. On double-oblique reformatted 3D MRCP image (**B**; corresponding to *white dashed lines* in **A**), proximal duct measured 21 × 20 mm (*white lines* emphasized by *white arrows*, **B**) along short axis of proximal duct. Axial T2-weighted SSFSE image obtained at level of proximal duct (**C**; corresponding to *red dashed line* in **A**) shows overestimation of duct size (25 × 28 mm [*red lines* emphasized by *red arrows*, **C**]) owing to oblique measurement. Short-axis measurements obtained at level of mid duct (corresponding to *yellow dashed line*, **A**) on double-oblique reformatted 3D MRCP image (**D**; corresponding to *yellow dashed line* in **A**) shows overestimation of duct size (29 × 21 mm [*yellow lines* emphasized by *yellow arrows*, **D**]) given inclusion of portion of cystic duct (*asterisk*).

depiction of the biliary tract without radiation exposure [23]. In most instances, MRCP has replaced diagnostic ERCP and its attendant complications while identifying patients who would benefit from therapeutic ERCP [24]. High-resolution images of the biliary tract are obtained with fluid-sensitive sequences that accurately confirm the presence or absence of biliary obstruction and its causes; the performance of MRI without and with IV contrast media in conjunction with MRCP aids in delineating the cause of obstruction, especially when that cause is malignant [25]. In selected cases, MRCP may be performed using a hepatobiliary contrast agent. As with all techniques, MRCP is associated with technical pitfalls such as incomplete ductal depiction as well as flow and pulsation artifacts, in addition to interpretive pitfalls such as pseudocalculus defects [26]. Despite these pitfalls, MRCP performed with MRI is considered by the ACR to be appropriate for evaluating possible obstruction [4].

Endoscopic Ultrasound

Although transabdominal US may be limited by bowel gas or habitus, endoscopic US provides excellent visualization of the extrahepatic biliary tree, the wall of the duodenum, and the papilla, allowing exclusion of an ampullary neoplasm, which is an important limitation of MRCP [27]. Endoscopic US has sensitivity greater than 90% and specificity approaching 100% for detection of choledocholithiasis [28–30], and it is superior to MRCP for detection of small stones or microlithiasis [29]. Guidelines have suggested use of endoscopic US, with concurrent use of ERCP if necessary, for patients with intermediate risk of choledocholithiasis (i.e., bile duct diameter > 6 mm with gallbladder present, a bilirubin level of 1.8–4 mg/dL, age older than 55 years, and abnormal LFT results) [31]. The proximity of the stomach and duodenum to the pancreas allows highly sensitive and accurate (> 90%) evaluation of pancreatic neoplasms and cysts causing biliary obstruction as well as the ability to perform simultaneous fine-needle aspiration (FNA) or biopsy to obtain a definitive diagnosis [30, 32]. Important limitations include a requirement for general anesthesia, the risk of adverse events if simultaneous intervention is performed (e.g., pancreatitis after FNA), and limitations in patients with abnormal anatomy (e.g., gastric bypass) [33].

ERCP

ERCP provides an anatomic view of the bile ducts as well as a functional assessment of bile drainage; however, in the era of MRCP and endoscopic US, purely diagnostic indications for ERCP have become sparse, with ERCP reserved for interventional settings, such as sphincterotomy with stone extraction, stenting, tissue sampling (i.e., brushing or biopsy), or tissue ablation (i.e., radiofrequency ablation) [34]. Peroral direct cholangioscopy, which involves insertion of an ultraslim endoscope into the bile duct, has allowed direct visualization of biliary strictures and targeted sampling of abnormal tissue [35, 36] as well as directed cannulation of severely obstructed or disconnected ducts. Important limitations include a requirement for general anesthesia, possible contamination of chronically obstructed sterile biliary segments [37], and postprocedural adverse events (e.g., post-ERCP pancreatitis) [38, 39].

How and Where Should the Extrahepatic Bile Duct Be Measured?

The common hepatic duct (CHD) and common bile duct (CBD) are often collectively referred to as the extrahepatic duct, which can be subjectively divided into the proximal, mid, and distal segments (Fig. 1). Measurements of all three segments may be performed using US, whereas the extrahepatic duct is typically measured solely at the largest-diameter location as seen on CT and MRI. Measurements of the extrahepatic biliary duct are most reliable when obtained with the patient in a fasting state. The duct diameter should be measured from inner wall to inner wall, with measurement performed perpendicular to the long axis of the bile duct.

In normal circumstances, the bile duct wall is imperceptible, measuring less than 1 mm. Certain conditions, such as cholangiopathy and/or cholangitis, are associated with increased thickness of the biliary wall, which may erroneously increase the size of the duct if included in the measurement. Another common pitfall is off-axis measurement of the bile duct on CT and MRI, which may result in overestimation of bile duct size (Fig. 2). Similar errors may also occur when the duct is measured in an oblique fashion on US. In addition, the extrahepatic duct may appear larger at the level of the cystic duct insertion due to measure-

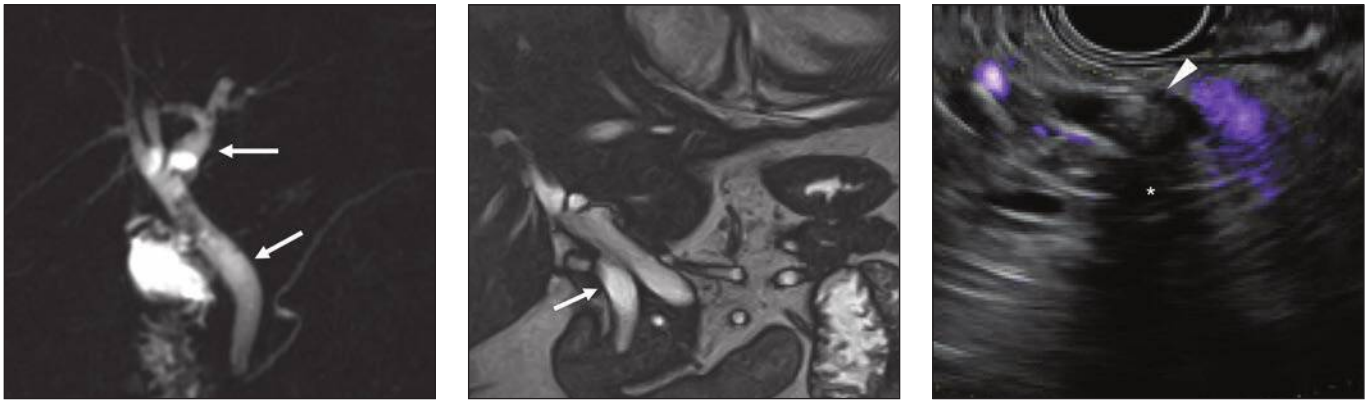

Fig. 3—51-year-old woman with occult bile duct stone and transiently elevated biliary enzymes.
A and **B,** Two-dimensional thick-slab MRCP image (**A**) and coronal balanced SSFP image (**B**) show mild diffuse intrahepatic and extrahepatic biliary duct dilatation, which measured up to 12 mm in mid segment (*arrows*), with gradual distal taper and no obstructing lesion.
C, Subsequently performed endoscopic ultrasound shows 8-mm echogenic stone (*arrowhead*) with posterior acoustic shadowing (*asterisk*). ERCP was performed, and stone was retrieved after sphincterotomy (not shown).

ment of the CHD and cystic duct together (Fig 2D). It is important to consider this pitfall, especially when there is a low insertion of the cystic duct and a long common channel between the cystic duct and CHD.

The literature has suggested different size thresholds for biliary duct dilatation, which vary according to the specific location where the duct is measured. For instance, an upper limit of normal of 4 mm has been reported for the proximal extrahepatic duct at the level of the right hepatic artery [18, 40]. On US, this segment is more reliably visualized than the mid duct, but it may not dilate as early in the setting of obstruction. The mid duct, where it is anterior to and parallels the portal vein, is less restricted, and a size measurement of 6 mm or less is considered normal [41]. However, insufficient strong data are available to provide distinct cutoffs for the CHD and CBD, and we recommend measuring the greatest diameter of the extrahepatic duct at any location along its course, with use of a threshold of 6 mm as the upper limit of normal in the general population. This approach is simpler than considering different thresholds at different biliary segments, is applicable to all imaging modalities, and is concordant with the report of the ACR Incidental Findings Committee II [1].

When Is Biliary Duct Dilatation Considered Obstruction?

Biliary duct dilatation is not synonymous with biliary obstruction (Table 1). However, clinical or laboratory evidence of biliary obstruction is a valuable clue and may trigger further investigation, even without an appreciable imaging abnormality apart from biliary dilatation. This situation may relate to an occult stone, which may be difficult to see on MRCP due to lack of surrounding bile (Fig. 3), or a periampullary lesion, which may not be apparent due to underdistention of the duodenum (Fig. 4). On the other hand, the most reliable finding of biliary obstruction is direct visualization of an obstructing lesion that is responsible for biliary duct dilatation (Fig. 5).

In cases in which no obstructing lesion is apparent, multiple ancillary findings may offer a degree of reassurance or raise suspicion for biliary obstruction. Coexistent intrahepatic duct dilatation, defined as size larger than 2 mm or 40% of the caliber of the adjacent portal vein [40], is more concerning than extrahepatic duct dilatation alone, especially when the peripheral intrahepatic ducts are involved [42]. This distinction may relate to the Laplace law, in which a higher pressure is required to dilate the peripheral ducts owing to either their smaller size (tension equals pressure multiplied by

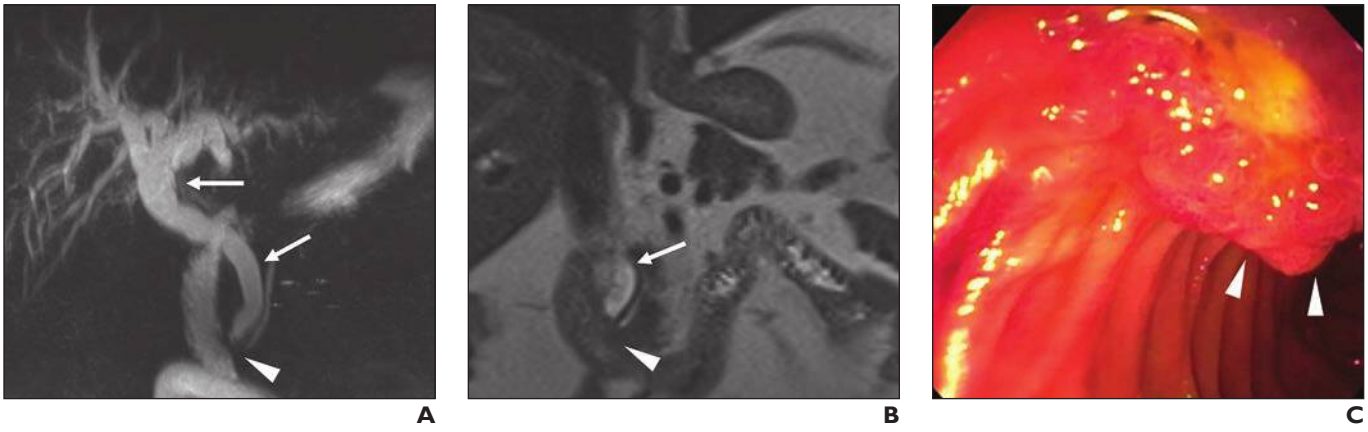

Fig. 4—43-year-old man with ampullary adenocarcinoma who presented with jaundice.
A and **B,** Three-dimensional MRCP maximum-intensity-projection image (**A**) and coronal T2-weighted SSFSE image (**B**) show moderate diffuse intrahepatic and extrahepatic biliary duct dilatation (*arrows*) to level of ampulla (*arrowheads*). Ampullary mass was present but difficult to visualize given underdistention of adjacent duodenum.
C, Endoscopic image confirmed presence of ulcerated ampullary mass (*arrowheads*).

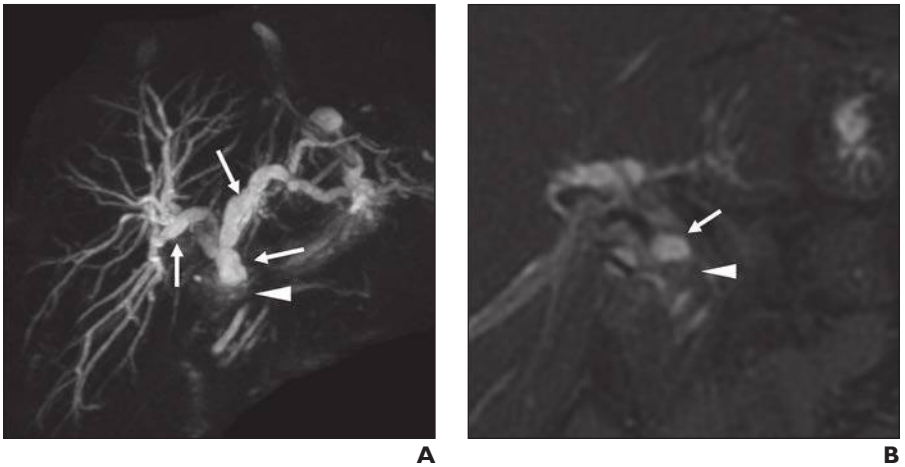

Fig. 5—60-year-old woman with extrahepatic cholangiocarcinoma who presented with jaundice.
A, Three-dimensional MRCP maximum-intensity-projection image shows marked intrahepatic and proximal extrahepatic biliary duct dilatation (*arrows*) with malignant-appearing mid- to distal extrahepatic duct stricture (*arrowhead*).
B, Corresponding coronal T2-weighted fat-suppressed SSFSE image shows small mass with intermediate signal intensity (*arrowhead*), which represented cholangiocarcinoma, with dilatation of proximal bile duct (*arrow*).

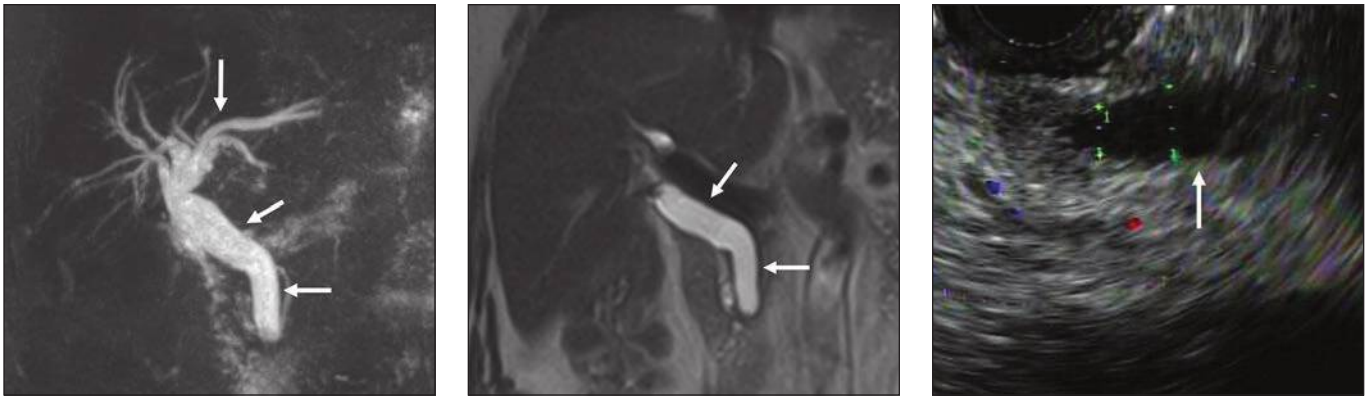

Fig. 6—73-year-old man with dilated but unobstructed bile duct who had undergone prior cholecystectomy.
A and **B,** Three-dimensional MRCP maximum-intensity-projection image (**A**) and coronal T2-weighted SSFSE image (**B**) show moderate diffuse intrahepatic and extrahepatic biliary duct dilatation (*arrows*) with gradual distal taper at level of ampulla. Patient had transient elevation in biliary enzyme levels that prompted performance of endoscopic ultrasound.
C, Ultrasound image shows dilated bile duct (*arrow*) but no obstructing lesion.

radius) or decreased compliance of the small intrahepatic ducts that are completely encircled by hepatic parenchyma [23]. The duct morphology also offers clues regarding the presence of obstruction; gradual tapering of the distal duct is a normal and reassuring finding, whereas an abrupt cutoff or stricture is more suspicious. On the other hand, multifocal areas of peripheral duct dilatation with stricturing disproportionate to the degree of central dilatation are usually indicative of cholangiopathy rather than obstruction [43]. In addition, new or progressive biliary duct dilatation is much more likely to indicate obstruction than stability over multiple years.

Pancreatic duct dilatation is another relevant finding in the setting of biliary duct dilatation (i.e., double duct sign) and may indicate obstruction due to an underlying pancreatic or periampullary malignancy. Patients with pancreatic and biliary duct dilatation but a normal bilirubin level have a significantly lower risk of malignancy (6% vs 86%), which again highlights the importance of biliary-associated enzymes [44]. Gallbladder distention in a patient with biliary duct dilatation also raises the level of concern for biliary obstruction [45, 46] and malignancy in particular (i.e., Courvoisier sign) [47]. However, the uncertain reliability of this finding and the high frequency of prior cholecystectomy among patients with biliary duct dilatation render this sign of questionable clinical utility.

Can Biliary Dilatation Be Attributed to Advanced Age or Reservoir Effect After Cholecystectomy?

To a certain extent, yes. Numerous studies have shown statistically significant dilatation of the common duct with age, albeit

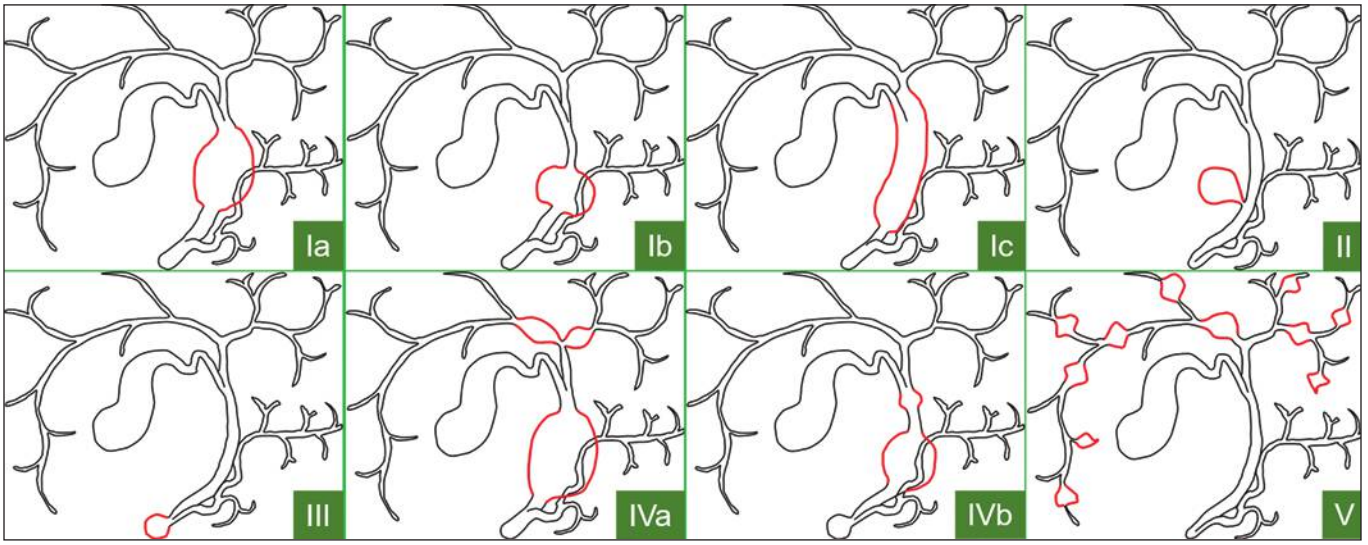

Fig. 7—Types of choledochal cysts according to revised Todani classification. Type Ia is defined by cystic dilatation of extrahepatic duct (*red*) with abnormal pancreaticobiliary junction (APBJ); type Ib, focal segmental dilatation (*red*) without APBJ; type Ic, diffuse fusiform dilatation (*red*) with APBJ, often with distal common bile duct (CBD) stricture; type II, extrahepatic duct diverticulum (*red*) without APBJ; type III (choledochocele), dilatation of intraduodenal CBD (*red*), no APBJ; type IVa, intrahepatic and extrahepatic biliary duct involvement (*red*), usually with APBJ but often with stricture at level of hilum; type IVb, multifocal cystic dilatations of extrahepatic duct (*red*) without APBJ; type V (Caroli disease), multifocal diffuse or localized saccular intrahepatic duct dilatation that may have funnel-shaped configuration (*red*).

to a lesser extent than the 1 mm per decade that has been classically taught [45, 48–50]. Indeed, newer studies indicate smaller increases in duct caliber with advanced age (0.3–0.7 mm per decade) and support a cutoff of 8.0–8.5 mm for maximal duct diameter in older patients [48, 50, 51]. The ACR Incidental Findings Committee II report provides recommendations for management of incidental biliary duct dilatation, which it defines as a duct diameter greater than 6 mm in a patient who has their gallbladder, but it also notes that this threshold only applies to patients younger than 60 years old and provides no guidance for older patients [1]. We recommend a threshold of 8 mm as the upper limit of normal in older patients (those 60 years old or older) with intact gallbladder unless cholestatic symptoms or abnormal biliary enzymes are present.

Similar to aging, cholecystectomy has been shown in multiple studies to result in statistically significant common duct dilatation [42, 52–54]. This dilatation is generally attributable to the reservoir effect in which the bile duct dilates to act as a bile reservoir after the gallbladder is removed [55]. However, dilatation likely happens within the first few years postoperatively rather than gradually over decades, as occurs with aging. Although biliary duct dilatation after cholecystectomy is common and in some cases may be pronounced (Fig. 6), it does not affect all patients, and most asymptomatic patients, with or without cholecystectomy, have a normal common duct diameter (i.e., ≤ 6 mm) [53]. In a subset of patients (e.g., those with chronic cholecystitis), biliary dilatation may happen preoperatively before cholecystectomy and remain persistent, rather than occur postoperatively [8]. Based on our experience and support from the literature, and in concordance with the ACR Incidental Findings Committee II report, a threshold of 10 mm should be used as the upper limit of normal when incidentally detected in the postcholecystectomy state [1, 51, 56, 57].

Should a Radiologist Attribute Biliary Dilatation on Imaging to Sphincter of Oddi Dysfunction or Papillary Stenosis?

Generally, no. Sphincter of Oddi dysfunction (SOD) and papillary (or ampullary) stenosis are conditions in which bile or pan-

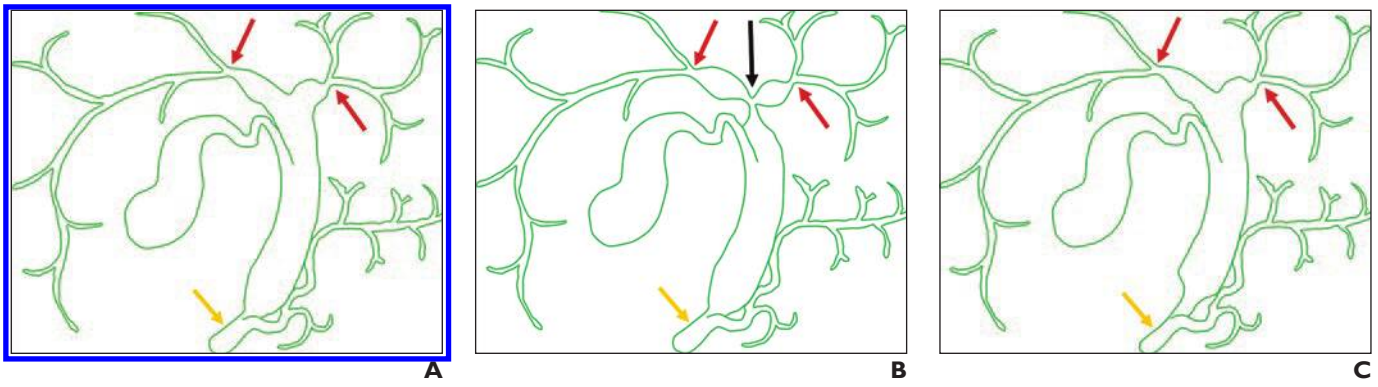

Fig. 8—Types of choledochal cysts (CCs) that can be mistaken for biliary obstruction. Distinguishing features help to favor CC over obstruction for each type.
A, Diagram shows type Ic (or IVa) CC with fusiform intrahepatic dilatation. Distinguishing features include abnormal pancreaticobiliary junction (APBJ) or long channel (*yellow arrow*) as well as abrupt transition of intrahepatic bile ducts (*red arrows*).
B, Diagram shows classic IVa CC with hilar narrowing. Distinguishing features include APBJ or long channel (*yellow arrow*), abrupt transition of intrahepatic ducts (*red arrows*), and hilar stricture (*black arrow*).
C, Diagram shows type Ic CC with fusiform intrahepatic dilatation and stricture. Distinguishing features include APBJ or long channel (*yellow arrow*) or abrupt transition of intrahepatic ducts (*red arrows*).

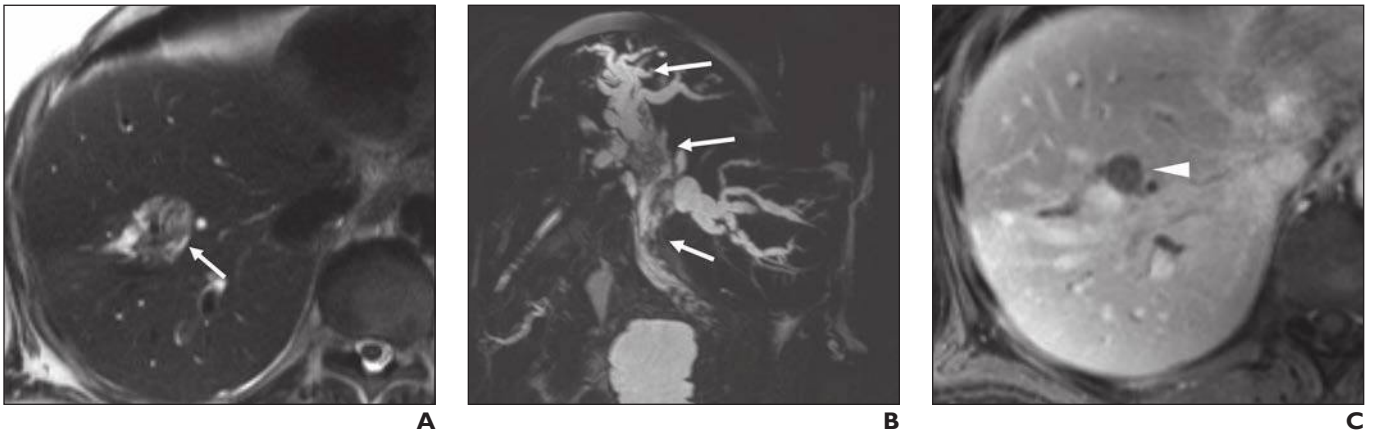

Fig. 9—74-year-old man with intraductal papillary neoplasm of bile duct (IPN-B).
A and **B,** Axial T2-weighted SSFSE image (**A**) and 3D MRCP maximum-intensity-projection image (**B**) show diffuse intrahepatic biliary duct dilatation, which is most pronounced in segment VIII, with multiple string and frondlike filling defects representing mucin and papillary tumor (*arrows*), respectively.
C, Corresponding axial T1-weighted fat-suppressed image shows enhancing mass (*arrowhead*) in proximal segment VIII duct. Patient's condition was managed with cholangioscopic radiofrequency ablation.

creatic fluid flow is obstructed at the ampulla in the absence of an underlying stone or mass [58]. SOD requires the presence of right upper quadrant or biliary-type pain and is divided into three types: type I, which is characterized by dilated bile duct and abnormal biliary enzyme levels; type II, which is characterized by a dilated bile duct or abnormal biliary enzyme levels (but not both); and type III, which is characterized by the absence of abnormalities [59]. Persistent right upper quadrant pain syndromes are reported in 10–40% of patients after cholecystectomy, frequently in association with a dilated bile duct on imaging [60–62]. Many of these patients have SOD diagnosed and may undergo ERCP and sphincterotomy that have doubtful clinical benefit. Indeed, a randomized, multicenter sham-controlled trial of endoscopic sphincterotomy in type III SOD failed to improve clinical symptoms and was associated with high rates of pancreatitis (11%) [63]. Furthermore, sphincter manometry has not been shown to predict the outcome of sphincterotomy and has generally fallen out of use [64]. Some authors advocate for the use of secretin-enhanced MRCP [65, 66] or gadoxetate disodium Eovist, Bayer HealthCare)–enhanced MRCP [67] in the diagnosis of SOD, although both techniques have only limited support in the literature and the relationship between imaging findings and outcomes after sphincterotomy have not been reported [68].

SOD is often considered functional, whereas papillary stenosis is considered structural and may occur after inadequate sphincterotomy or prior passage of gallstones or debris and resultant scarring. Papillary stenosis is generally treated with sphincterotomy extension or dilatation (i.e., sphincteroplasty). However, similar to SOD, a judgment of papillary stenosis is often highly subjective and at the discretion of the gastroenterologist, especially in a patient with normal biliary-associated enzyme levels. SOD and papillary stenosis are considered clinical diagnoses, and although biliary duct dilatation may be seen in a subset of patients with SOD or papillary stenosis, it is not sufficient for diagnosis. Thus, we recommend against using the terms "SOD" and "papillary stenosis" in imaging reports, as the terms do not effectively guide management. Instead, we recommend describing the duct morphology and indicating whether an obstructing lesion is evident (e.g., intrahepatic and extrahepatic biliary duct dilatation, with a gradual distal taper at the level of the ampulla but with no obstructing stone or mass). If there are elevated biliary-associated enzyme levels, a patient with such findings may be more likely to have an occult stone or periampullary mass than papillary stenosis; on the other hand, such findings are unlikely to be clinically significant in a patient with normal biliary-associated enzyme levels.

Can Biliary Dilatation Be Attributed to Congenital Cystic Dilatation?

On occasion, it may be difficult to distinguish a congenitally dilated bile duct segment (i.e., a choledochal cyst [CC]) from biliary obstruction. The pathogenesis of CCs is not entirely clear, although a commonly cited source is an abnormal pancreaticobiliary junction (APBJ) with a long common channel, which predisposes to pancreaticobiliary reflux and congenital destruction of the bile duct wall [69]. The most widely used classification system for CCs is the revised Todani system [70, 71] (Fig. 7). Most CCs (80%) are diagnosed in childhood on the basis of characteristic symptoms; however, a minority occur in adults with no symptoms or undifferentiated abdominal pain [72, 73].

The CC types most likely to be mistaken for biliary obstruction are types Ic and IVa, in which fusiform dilatation can involve both the extrahepatic and central intrahepatic ducts, mimicking distal biliary obstruction (Fig. 8). Unfortunately, no biliary size thresholds for CCs have been accepted, leaving duct morphology and an APBJ as the key diagnostic criteria [74]. Relative hilar narrowing, abrupt transition from dilated central to normal-caliber peripheral intrahepatic ducts, and an APBJ strongly favor type IVa CC over obstruction [71]. Marked dilatation of the extrahepatic duct without intrahepatic dilatation strongly suggests type Ic CC; however, when coexistent narrowing of the distal CBD and intrahepatic dilatation are present, distinguishing it from an obstructing stricture can be difficult. In this context, abrupt transition of the intrahepatic ducts and an APBJ both favor type Ic CC. Given the rarity of CCs in adult patients, we recommend that CC be considered in the

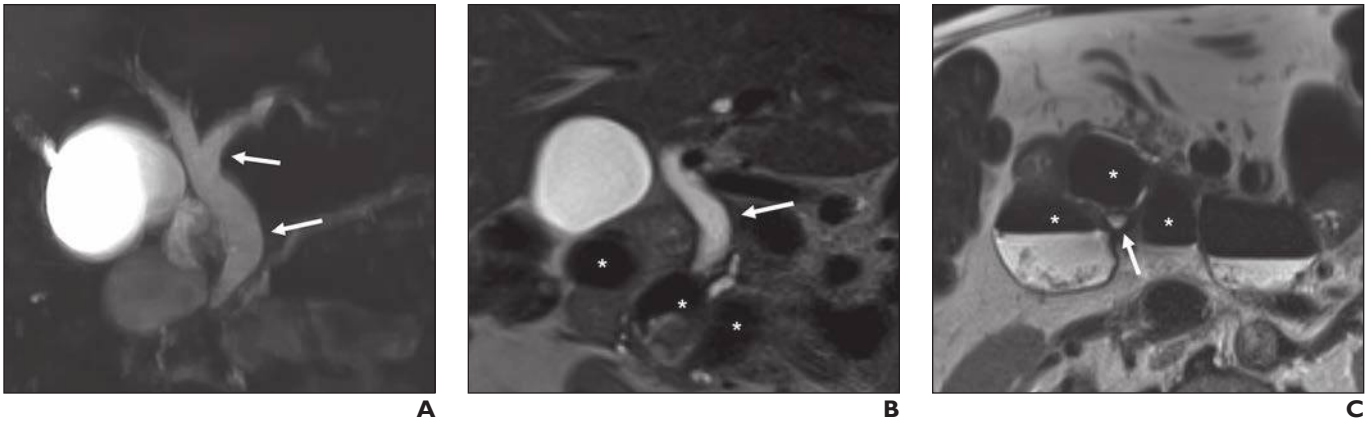

Fig. 10—74-year-old man with dilated bile duct in setting of large periampullary duodenal diverticula.
A, Three-dimensional MRCP maximum-intensity-projection image shows moderate central intrahepatic and extrahepatic biliary duct dilatation with smooth distal tapering (*arrows*) near level of ampulla.
B and **C,** Coronal (**B**) and axial (**C**) T2-weighted SSFSE images show multiple large periampullary duodenal diverticula (*asterisks*), which exert mass effect on distal common bile duct (*arrows*). Patient had elevated alkaline phosphatase level, which was considered to occur secondary to small-duct primary sclerosing cholangitis (diagnosis made after prior liver biopsy).

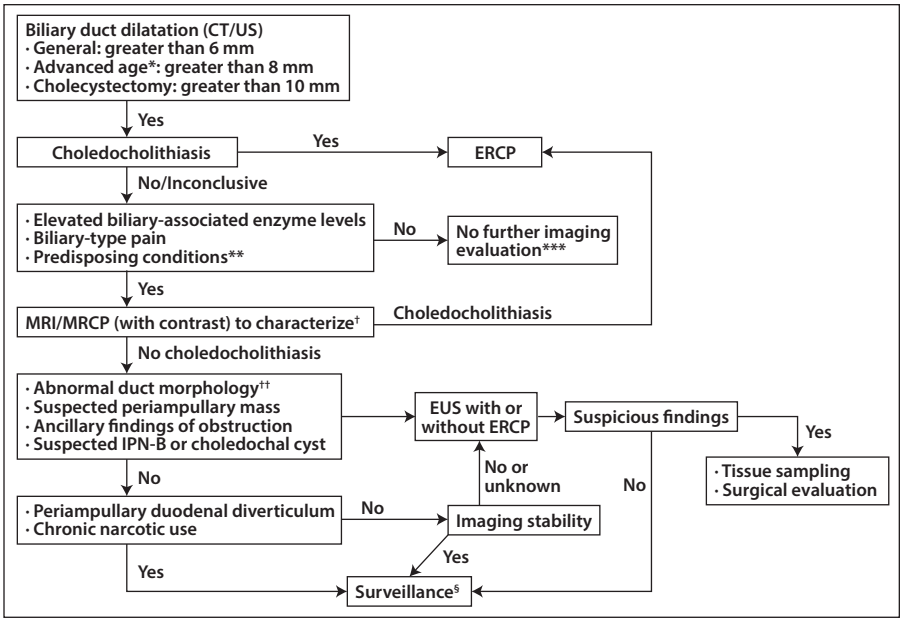

Fig. 11—Flowchart for management of biliary duct dilatation identified on CT or ultrasound (US). Asterisk denotes advanced age (defined as 60 years old or older); double asterisk, history of malignancy or genetic predisposition (e.g., familial pancreatic cancer); triple asterisk, clinical follow-up and/or repeat laboratory testing may be considered; dagger, comprehensive protocol (i.e., not abbreviated) with IV contrast media should be used when feasible; double dagger, endoscopic US with or without ERCP may not be necessary if findings are characteristic of benign or inflammatory process (e.g., primary sclerosing cholangitis); and section mark, first follow-up MRI/MRCP should be performed after 6–12 months, followed by annual surveillance imaging. Length of follow-up to exclude underlying malignancy is not clearly defined in literature. EUS = endoscopic ultrasound, IPN-B = intraductal papillary neoplasm of bile duct.

differential diagnosis of biliary duct dilatation only if the duct morphology is highly suggestive and/or an APBJ is present.

Can Biliary Dilatation Be Attributed to an Intraductal Papillary Neoplasm of the Bile Duct?

One cause of biliary dilatation that should be considered is a mucin-producing IPN-B. Identification of these lesions is important given that they are a precursor to cholangiocarcinoma and are often managed surgically [75, 76]. IPN-Bs are predominant in male individuals and occur more frequently in individuals from Asia than in those from Europe or North America. They may present incidentally or be symptomatic with pain, cholangitis, or jaundice [76]. Chronic inflammation likely plays a role in pathogenesis given the overlap in incidence with regions of hepatolithiasis and infection with *Clonorchis sinensis* [76–78]. IPN-Bs have a spectrum of histologic behaviors and immunohistochemical phenotypes [79], although their appearance on imaging primarily depends on the amount of mucin production relative to solid papillary growth. Accordingly, IPN-Bs may be classified into one of four imaging patterns: type 1, denoting an intraductal mass associated with upstream duct dilatation only; type 2, disproportionate duct dilatation without a visible mass; type 3, an intraductal mass with both upstream and downstream duct dilatation; and type 4, focal cystic dilatation of the bile duct with a papillary mass [76].

When consideration is given to whether biliary dilatation might be attributed to IPN-B, identification of an enhancing intraductal papillary mass is a primary feature for establishing the diagnosis of IPN-B types 1, 3, and 4 [80, 81]. Type 2 IPN-Bs, in which there is segmental or diffuse duct dilatation without a visible mass, can present a diagnostic conundrum given that mucin is attenuating on CT and isointense on MRI compared with bile, mimicking duct dilatation of any cause [82]. For several reasons, MRI/MRCP has a far higher sensitivity for detecting IPN-Bs than CT or US [76]. First, MRI may identify intraductal curvilinear hypointense striations corresponding to concentrated mucin bundles within dilated ducts (the thread sign), which has a high specificity (99–100%) for IPN-B, albeit with limited sensitivity (45–53%) [83] (Fig. 9). Second, MRI with hepatobiliary phase imaging may allow identification of mucin as a filling defect and aid in the delineation of enhancing intraductal soft-tissue components [84]. Last, DWI sequences on MRI can be highly useful in identifying the presence of a papillary mass [85]. We recommend that a diagnosis of IPN-B be considered in a patient with biliary duct dilatation only when there is a visible intraductal mass or mucin on imaging.

What Are the Next Steps in Management When Incidental Biliary Duct Dilatation Is Present?

The initial step is to determine whether an enlarged bile duct meets the size criteria for biliary duct dilatation, defined as a diameter larger than 6 mm in the general population, larger than 8 mm in older patients (age 60 years old or older), and larger than 10 mm in those who have undergone prior cholecystectomy. If a bile duct stone is identified on initial evaluation, ERCP is performed for diagnosis and stone retrieval, and additional imaging is considered unnecessary according to American Society for Gastrointestinal Endoscopy guidelines [31]. If no choledocholithiasis or other obstructive lesion is present, the next step is correlation with clinical factors such as elevated biliary-associated enzymes, biliary-type pain, or predisposing factors for biliary obstruction (e.g., history of malignancy). Correlation with biliary enzymes is particularly critical, as the yield for abnormal findings on endoscopic US in a patient with biliary duct dilatation exceeds 50% when biliary-associated enzyme levels are elevated, compared with approximately 6% in patients with normal biliary enzyme levels [86]. If any of these clinical factors are present, and under the assumption that the initial evaluation is performed with US or CT, we recommend MRI/MRCP for further evaluation, with IV contrast media used when feasible, in accordance with comprehensive (i.e., not abbreviated) protocol meeting ACR–Society of Abdominal Radiology–Society of Pediatric Radiology practice parameters and technical standards [87]. In the absence of any of these clinical factors, incidental biliary duct dilatation generally requires no further evaluation.

The next step after performing MRI/MRCP is to determine whether the duct has abnormal or suspicious morphology, whether a periampullary mass is suspected, or whether there are ancillary features of biliary obstruction. As previously mentioned, this determination may reflect findings including an abrupt cutoff or stricture, coexistent pancreatic duct dilatation, or concomitant dilatation of the peripheral intrahepatic bile ducts. It is also important to determine whether biliary duct dilatation may be related to congenital cystic dilatation or an IPN-B. If any of these features are present, we recommend further evaluation with endoscopic US without or with simultaneous ERCP. For this reason, particularly for patients with elevated biliary-associated enzyme levels, endoscopic US should be performed in rooms where fluoroscopy is also available given the increased likelihood that concomitant intervention with ERCP will be required. Suspicious findings on endoscopic US and/or ERCP invariably prompt the need for tissue sampling and, usually, surgical evaluation.

If none of the aforementioned conditions are met, the next step is to ascertain whether a periampullary diverticulum may be causing biliary duct dilatation or whether the patient has a history of chronic opiate use [88]. In our experience, biliary duct dilatation in many patients with normal biliary-associated enzyme levels can be attributed to one of these two causes (Fig. 10). If either of these conditions are met, or if there is prior imaging available that establishes temporal stability, we recommend surveillance imaging rather than further evaluation with endoscopic US. The length of follow-up for excluding underlying malignancy as the explanation for biliary duct dilatation is not clearly defined in the literature. Our recommended management approach is outlined in Figure 11.

What Are the Future Directions of Biliary Imaging?

The use of specific size cutoffs and an algorithmic approach to biliary duct dilatation, as we have discussed in this *AJR* Expert Panel Narrative Review, may be clinically useful but does not consider all relevant factors when assessing the likelihood of biliary obstruction. Furthermore, many of these parameters are considered in dichotomous fashion, yet almost all exist on a continuum. For instance, a patient with a markedly dilated bile duct and a mildly elevated alkaline phosphatase level is much less likely to have biliary obstruction than is a jaundiced patient who has only mild bile duct dilatation. Comprehensive predictive models derived from data in large cohorts of patients are needed to better assess the likelihood of biliary obstruction. Clinical scoring systems and/or predictive models in choledocholithiasis, for instance, have shown preliminary success [89, 90].

Newer functional and/or quantitative imaging techniques may also provide useful information regarding the significance of biliary duct dilatation and further inform risk stratification. For instance, cine MRCP performed using a spatially selective inversion recovery pulse may show alterations in bile flow dynamics that imply a higher likelihood of biliary obstruction in a patient with biliary duct dilatation [91, 92]. MRI with gadoxetate disodium also has potential utility by providing functional information and helping to ascertain the significance of an underlying lesion or stricture [67, 93]. Finally, quantitative parameters of the biliary tree on MRCP may identify features in biliary duct dilatation that confer a higher likelihood of obstruction or may aid in the detection of subtle changes over time [94, 95].

Consensus Statements

- ALP, GGT, and conjugated bilirubin levels are elevated when the liver injury pattern is cholestatic.
- The extrahepatic duct should be measured at its greatest diameter at any location along its course; a threshold of 6 mm should be considered in the general population, 8 mm in patients 60 years old or older, and 10 mm after cholecystectomy.
- Avoid use of the terms "SOD" and "papillary stenosis" in imaging reports, as these are clinical diagnoses. Instead, describe the duct morphology and whether an obstructing stone or mass is evident.
- Given the rarity of choledochal cysts in adult patients, they should only be considered in the differential diagnosis if the duct morphology is highly suggestive and/or an APBJ is present.
- A diagnosis of IPN-B should be considered in a patient with biliary duct dilatation only if an intraductal mass or mucin is visible on imaging.
- If biliary duct dilatation without obvious cause is identified on US or CT in a patient with elevated biliary-associated enzyme levels, we recommend performing comprehensive MRI/MRCP for further evaluation, with IV contrast media used if feasible.
- For suspicious findings on MRI/MRCP, endoscopic US should be performed for further evaluation, with concomitant ERCP performed only if intervention is required.
- If biliary duct dilatation is present but there are no suspicious findings on MRI/MRCP, or if an explanation for duct dilatation (e.g., periampullary duodenal diverticulum or chronic opiate use) is present, surveillance imaging to assess for temporal stability, rather than endoscopic US, is sufficient.
- The length of follow-up for excluding underlying malignancy as the explanation for biliary duct dilatation is not clearly defined in the literature.

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