# 17 Choledocholithiasis and Cholangitis: Incidence, Initial Management, and Surgical Management

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## Choledocholithiasis

### Epidemiology

Nearly 15% of the adult population in the United States has underlying cholelithiasis [1]. Of this number, 10–20% are estimated to have concomitant non-obstructing choledocholithiasis [2, 3]. Exact figures of the prevalence of choledocholithiasis are difficult to ascertain as many stone formers may pass clinically silent stones [4]. While ductal stones typically arise in and are secreted from the gallbladder, choleliths may arise *de novo* in the biliary tree. A history of cholecystectomy, therefore, does not preclude the development of choledocholithiasis. Risk factors for primary stone formation include bile stasis (as in cystic fibrosis), periampular diverticula, and East Asian heritage [5, 6].

### Clinical Presentation

Choledocholithiasis causes symptoms only when stones result in obstruction. These patients present with symptoms similar to those of biliary colic. There is crampy, intermittent right upper quadrant pain and associated nausea and vomiting. Pain typically persists for longer periods than is seen with simple biliary colic, up to several hours per episode [7]. The patient may appear jaundiced and endorse right upper quadrant tenderness to palpation. There may also be a history of acholic stools, generalized pruritus, and darkened urine secondary to altered bilirubin excretion. Patients with intermittent obstruction and subsequent passage of stones may describe a history of repeated bouts of abdominal pain associated with jaundice. A palpable gallbladder (Courvoisier's sign) has been described, but this is more commonly associated with the progressive obstruction of malignant disease than with the acute process of stone blockage [8].

If the offending stone is located distal to the junction of the common bile duct and main pancreatic duct, the presenting signs and symptoms may instead be those of pancreatitis, namely, epigastric pain with radiation to the back [9]. Though this clinical entity is a potential complication of choledocholithiasis, it will be discussed in another section.

### Laboratory Findings

The earliest laboratory abnormality is a transaminitis with elevations of alanine aminotransferase

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(ALT) and aspartate aminotransferase (AST) up to 25 times the upper limit of normal, though these findings have poor specificity. A cholestatic picture then predominates with elevations of serum bilirubin, alkaline phosphatase, and gamma-glutamyl transpeptidase (GGT) [10]. Hyperbilirubinemia is seen in a broad range of hepatic and biliary derangements as well as hemolysis and therefore should not be considered a specific finding. Fractionation of the serum bilirubin allows for the determination of conjugated hyperbilirubinemia which generally supports the determination of a biliary rather than a hepatocellular source. However, conjugated hyperbilirubinemia may also be seen in Rotor syndrome and Dubin-Johnson syndrome; in these cases serum GGT and alkaline phosphatase should be normal. The presentation of acute-onset right upper quadrant pain and marked transaminitis in the absence of concomitant elevation in alkaline phosphatase, GGT, and conjugated bilirubin should raise suspicion for an acute hepatitis. In gallstone pancreatitis, lipase will be elevated.

## Imaging

Ultrasonography (US) of the right upper quadrant is often obtained early in the course of the clinical presentation concerning for choledocholithiasis. Though several signs, namely, visualized stones and common bile duct dilation (Fig. 17.1), may be observed that support the diagnosis of choledocholithiasis, this modality has generally poor sensitivity and may not be relied upon to exclude the diagnosis of choledocholithiasis [11].

Normal common bile duct diameter varies depending both on the age and the surgical history of the patient [12]. Up to the 5th decade of life, 4 mm should be considered a mean measurement in the healthy population. For each decade thereafter, the average increases by 1 mm [13]. In the patient who has undergone cholecystectomy, duct diameters up to 10 mm are routinely found in patients without obstruction [14, 15]. When there is high clinical suspicion for intraductal stone based on presentation and laboratory findings, further imaging is not warranted prior to proceeding to endoscopic retrograde cholangiopancreatography (ERCP), as this intervention allows for simultaneous diagnosis and treatment. When the diagnosis of choledocholithiasis is uncertain, magnetic resonance cholangiopancreatography (MRCP) is called for and has virtually 100% sensitivity for stones large enough to be clinically significant [16]. In patients in whom cholecystectomy is indicated for symptomatic cholelithiasis or acute cholecystitis, and there is low but nonzero concern for ductal stones, intraoperative cholangiography (IOC) may be used to exclude the diagnosis of concomitant choledocholithiasis.

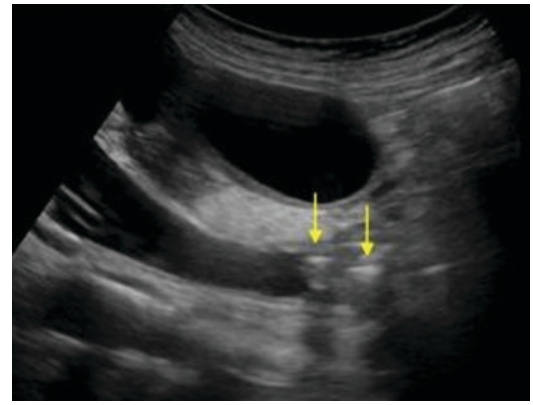

**Fig. 17.1** Right upper quadrant ultrasound image demonstrating common bile duct stones (yellow arrows) visualized obstructing a dilated common bile duct. (Image used courtesy of *Dr. T.S.A. Geertsma*)

## Management

Given the potential for development of cholangitis, as discussed below, all patients with confirmed choledocholithiasis require intervention. ERCP with stone extraction is indicated for clearing the offending stone. Contemporaneous sphincterotomy decreases the likelihood of recurrence; however it remains common enough that laparoscopic cholecystectomy is indicated within 72 hours of ERCP to decrease the potential for further episodes. Cases of post-sphincterotomy stenosis of the sphincter of Oddi with subsequent ascending cholangitis have also been described [17, 18].

ERCP fails in retrieving the offending stone in approximately 4.7–6% of cases depending of the volume of procedures performed at the institution in question [19]. In these cases, biliary distention may be temporized through the use of percutaneous transhepatic cholangiography (PTC), wherein the intrahepatic biliary ducts, dilated by downstream obstruction, are cannulated via a percutaneous approach. This allows for direct administration of contrast for the purposes of imaging (Fig. 17.2), as well as relieving the obstructed flow of bile. While not definitive, PTC offers relief of upstream obstruction and can greatly improve symptoms. The offending stone may then be removed either via a second attempt at ERCP or via a common bile duct exploration at the time of cholecystectomy [20, 21].

For the patient in whom ERCP is successful but comorbid conditions delay proceeding to surgery for cholecystectomy, biliary stenting at the time of ERCP can be considered [22]. Stents should also be considered if there have been repeated bouts of choledocholithiasis or if obstructing stones are removed but smaller stones remain in situ in the common bile duct. Definitive surgical therapy is still required as long-term stent placement is associated with 6–16% mortality, likely due to the stent acting as a nidus for bacterial cholangitis [23]. The stent may also serve as a nidus for the generation of further common duct stones, and for these reasons, proper patient selection is important.

Though ERCP carries with it a connotation of being a safer "nonsurgical" option in the minds of many providers, it bears the risk of several inherent complications. These include pancreatitis in 3.5% of cases, as well as bleeding and perforation in 1.3% and 0.6% of cases, respectively [24]. Additionally, the cardiovascular risks intrinsic to the induction of general anesthesia persist, including a remote risk of mortality in 0.07% of cases.

If choledocholithiasis is not discovered until IOC in the setting of cholecystectomy, intraoperative ERCP is indicated. If intraoperative ERCP is unavailable, two approaches are reasonable. Either the surgeon must elect to complete the cholecystectomy and allow for postoperative ERCP or a common bile duct exploration must be performed to remove the stone [25]. Common duct exploration will be discussed further in the section on management of cholangitis below. The decision to defer to postoperative ERCP carries with it the risk of ERCP failure and the need for a third procedure to relieve the obstruction.

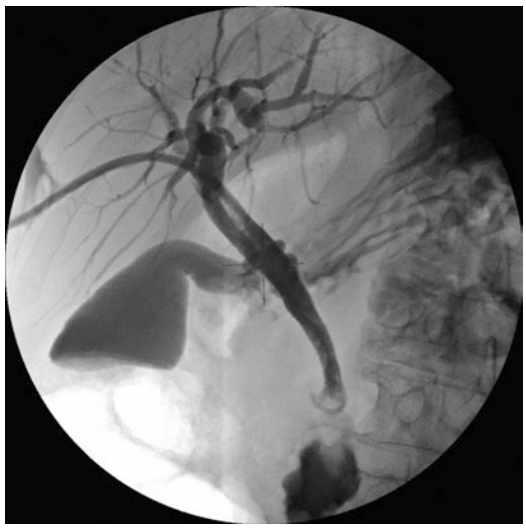

**Fig. 17.2** Fluoroscopic mage taken at the time of percutaneous transhepatic cholangiography demonstrating distal obstruction of the common bile duct and proximal dilation of the intrahepatic ductal system

## Cholangitis

### Epidemiology

Cholangitis is the presence of infection and inflammation within the biliary tree. Cholangitis classically arises in the setting of ductal obstruction secondary to choledocholithiasis. Roughly half of cholangitis cases arise via this etiology. Possible alternative causes include biliary stricture, biliopancreatic malignancies, indwelling foreign bodies (e.g., PTC catheter or stent), and choledocal cysts.

### Pathogenesis

Under normal conditions, the continuous flow of bile, IgA secretion by the biliary epithelium, and

isolation of the common bile duct from enteric contents by the sphincter of Oddi all promote sterility of the biliary tree. Regardless of the etiology of common bile duct obstruction, resultant biliary stasis and rising intraductal pressure undermine these defenses and allow the ascent of duodenal pathogens, commonly *E. Coli* and *Klebsiella* [26]. Pathogenic infection of bile and inflammation from rising pressure due to obstructed bile flow contribute to a systemic inflammatory response.

## Clinical Presentation

The classic presentation associated with cholangitis is that of fever, right upper quadrant abdominal pain, and jaundice, termed Charcot's triad. Though the modified Tokyo guidelines support that Charcot's triad achieves >95% specificity in the diagnosis of cholangitis, its sensitivity is poor and captures only one quarter of patients [27]. Hypotension and altered mental status (Reynolds' pentad when found together with Charcot's triad) are late clinical findings that suggest septic shock and portend poor outcomes. Diagnosis, therefore, cannot rely on these classic findings [28].

## Laboratory Findings

Findings are similar to those associated with choledocholithiasis; elevated transaminases evince hepatocellular irritation, and an obstructive pattern emerges with elevations of conjugated bilirubin, alkaline phosphatase, and GGT. Unlike choledocholithiasis, leukocytosis and a relative neutrophilia are expected.

## Imaging

If acute cholangitis is suspected, diagnostic imaging should not delay therapeutic intervention. If imaging is obtained, the diagnosis is confirmed with evidence of bile duct dilation or visualized obstructing lesion such as a stone or malignancy. Thickening of the common bile duct wall is also supportive of the diagnosis (Fig. 17.3).

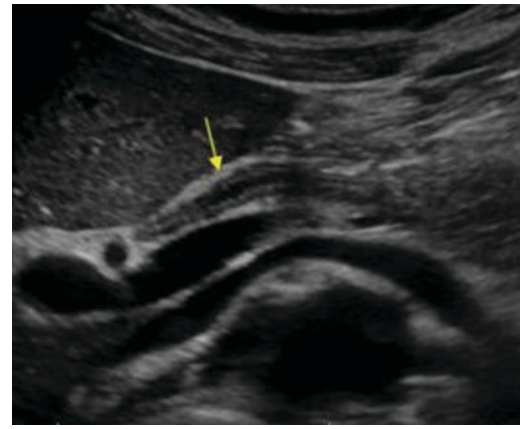

**Fig. 17.3** Thickened common bile duct wall found on ultrasonography in a patient with acute cholangitis. No intraductal stones are visualized. (Image used courtesy of *Dr. T.S.A. Geertsma*)

## Management

As in other etiologies of septic shock, early antibiotics and goal-directed resuscitation are vital. Blood cultures should be obtained but should not delay initiation of antimicrobial therapy. Given the ascending etiology of cholangitis, broad antibiotic therapy should be directed against gram-negative enteric organisms with an agent such as ampicillin/sulbactam or piperacillin/tazobactam [29]. In the great majority of patients, response to antimicrobial therapy is sufficient to allow a delay in ERCP up to 24 hours. If no improvement is evident and signs of suppurative cholangitis persist, emergent biliary decompression is indicated. This is ideally performed with immediate ERCP and sphincterotomy. When ERCP is unavailable or unsuccessful, biliary decompression is achieved with PTC, as discussed above.

Operative management of cholangitis should be considered as a last resort when the above interventions have failed. Choledochotomy may be attempted using a laparoscopic or open approach. The technique of a common bile duct exploration will be discussed in another chapter. The administration of glucagon may aid the surgeon in stone removal as it allows further dilation of the ductal system. The placement of a T-tube is advantageous as it allows for postopera-

tive contrasted imaging of the biliary tree as well as manipulation of the common bile duct postoperatively without the need for reoperation [30]. A more in-depth discussion of bile duct exploration occurs elsewhere in this text.

Provided the ascending infection arose in the setting of choledocholithiasis, cholecystectomy should again be performed as soon as the patient is clinically stable so as to prevent recurrence. However, in patients whose comorbidities preclude safe surgery, namely, the elderly and the frail, ERCP with sphincterotomy alone may be the best possible solution. In these patients, stents should be placed as leaving the gallbladder in situ increases the risk of recurrent episodes of choledocholithiasis and possibly subsequent cholangitis.

## Conclusion

Choledocholithiasis and cholangitis represent two distinct clinical entities. Though choledocholithiasis is often to blame for the development of cholangitis, this is not always the case. In either event, clinical outcomes can be devastating without prompt recognition and intervention. Multidisciplinary management is necessary as these cases often require the involvement of surgical, endoscopic, and sometimes interventional radiology teams. With rising incidence of cholelithiasis, more cases of choledocholithiasis and subsequent cholangitis should be anticipated. Clinicians of all specialties must be familiar with the presentations of these disorders and maintain healthy suspicion in the patient with right upper quadrant pain and an obstructive biochemical laboratory pattern.

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