Diagnosis and management of cholecystitis and cholangitis

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The main biliary tract emergencies are acute cholecystitis, ascending cholangitis, and acute pancreatitis. These conditions usually arise as a consequence of calculi obstructing the biliary tree or gallbladder. Less frequently they may result from benign or malignant strictures of the biliary tree, biliary tree manipulation, or biliary sludge [1]. Despite therapeutic advances these conditions still cause significant morbidity and mortality, particularly in the elderly [2]. This article reviews acute cholecystitis and cholangitis with a focus on the clinical presentation, diagnosis, and state-of-the-art management. Acute pancreatitis is dealt with elsewhere in this issue.

Acute calculous cholecystitis (ACC)

Acute cholecystitis is an acute inflammation of the gallbladder. Gallstones are present in over 90% of cases, and cause persistent obstruction of the gallbladder outlet because of impaction in the neck of the gallbladder, Hartmann’s pouch, or cystic duct [2,3]. In the remaining 5% to 10% of cases, gallstones are not identified, so-called “acalculous cholecystitis” [4].

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Natural history and prevalence of cholelithiasis and cholecystitis

Gallstones are ubiquitous; however, because most patients are asymptomatic, their precise incidence is uncertain. It is estimated that 10% to 15% of the United States adult population have gallstones [5], and studies from other westernized societies describe a prevalence of 5% to 20% [6–8]. Only a minority of patients with gallstones become symptomatic or develop cholecystitis [9]. The GREPCO investigators estimated the cumulative probability of developing a complication in patients with asymptomatic cholelithiasis after 10 years of follow-up was 3% [10] and other studies have confirmed a low rate of 0.5% to 3% per year of developing complications [9,11–13]. Once the patient is symptomatic, the rate of biliary complications rises [9,10,12,13]. The natural history may be less benign in selected subgroups, such as diabetics and organ transplant recipients [14]. Because the epidemiology and natural history of gallstones is different in Asian populations, this discussion is largely limited to Westernized societies [6].

Pathophysiology

The initial event in ACC is believed to be obstruction to gallbladder drainage [3]. This causes an increase in intraluminal pressure, gallbladder distention, and wall edema that may progress to venous and lymphatic obstruction, ischemia, and necrosis. A number of potential mediators have been identified including cholesterol-supersaturated bile, lysolecithin, phospholipase A, and prostaglandins [2,3,6]. Enhanced production of prostaglandins is believed to play a key role in mediating inflammation, and agents that reduce prostaglandin production have been shown to block the inflammatory response and reduce the pain of cholecystitis [15,16]. Bile is sterile in the early stages of acute cholecystitis and infection is believed to be a secondary event [17,18]. Indeed, although ACC is often considered an infection, bile cultures are positive only in 20% to 75% of patients [17–19]. The organisms most commonly cultured are enteric bacteria including *Escherichia coli*, *Klebsiella*, and *Enterococcus* [17–19].

Clinical presentation

Patients with ACC usually present with abdominal pain associated with fever, nausea, and vomiting. Pain and tenderness are usually localized to the right upper quadrant and the pain may radiate to the back, right scapula, or right clavicular area [2,20]. Three quarters of patients with ACC report a previous attack of biliary colic [21]. The pain is distinguished from biliary colic by its prolonged duration and the presence of Murphy’s sign (where mid inspiration is inhibited by pain on palpation of the right upper quadrant). Singer et al [22] found that a positive Murphy’s sign was highly sensitive (97%) and predictive (positive predictive value 93%) of ACC.
Presentation in elderly patients may be atypical. Hafif et al [23] reported that among 131 patients over 70 years with ACC, right upper quadrant pain was absent in 27% and fever was absent in 45% [23].

**Diagnosis**

The combination of prolonged, constant right upper quadrant pain and tenderness with fever are highly suggestive of ACC. A leukocytosis with increased neutrophils and band forms is usually present. There may be mild elevation of the transaminases or bilirubin. A significantly elevated bilirubin should raise the possibility of choledocholithiasis or Mirrizzi syndrome (obstruction of the common hepatic duct by a calculus impacted in Hartmann’s pouch) (Fig. 1) [3,24]. The diagnosis can usually be made by either ultrasound scanning or biliary scintigraphy. Ultrasonography is highly sensitive and specific for the presence of gallstones greater than 2 mm, but does not always confirm the diagnosis of ACC [6,25]. The sonographic features of ACC include gallbladder wall thickening, pericholecystic fluid,
gallbladder distention, and a sonographic Murphy’s sign (Fig. 2) [25–27]. The reported test performance of ultrasound varies with sensitivities of 48% to 100% and specificities of 64% to 100%; the presence of multiple criteria increases its diagnostic accuracy [26–32]. Biliary scintigraphy is highly accurate and is the gold standard for the diagnosis of cystic duct obstruction. Multiple studies have confirmed that scintigraphy has a sensitivity and specificity for the diagnosis of ACC of over 94% [31–36]. Scintigraphy, however, displays reduced specificity in the presence of hepatic impairment, parenteral nutrition, or when the patient is in the fasting state [2]. A positive scan may also be seen in chronic cholecystitis [6]. Scintigraphy gives little information about nonobstructing cholelithiasis and cannot detect other pathologic states. In a rigorous meta-analysis, Shea et al [37] reported that scintigraphy was more sensitive and specific than ultrasonography in ACC, and recommended it be the preferred test in patients with otherwise equivocal signs of ACC. Nonetheless, the choice of initial imaging modality remains controversial and varies with institution. The authors’ practice is to use ultrasonography as the screening test for ACC given its

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**Fig. 2. Cholecystitis.** Ultrasonographic study of the gallbladder showing a thickened gallbladder wall (solid arrows) and pericholecystic fluid (open arrow). Gallstones are seen in the gallbladder (S) with the characteristic acoustic shadowing (right brace).
relatively high accuracy, availability, portability, low cost, quickness, and ability to detect other pathology. Scintigraphy is usually reserved for cases where the ultrasound is normal and the clinical suspicion remains high. Magnetic resonance imaging (MRI) (Fig. 3) and computed tomography (CT) as an adjunct to ultrasound have been reported as being useful in the diagnosis of ACC but scanning an acutely ill patient may be difficult and the added time and expense are infrequently justified [30,38].

Management

The immediate management of ACC includes having the patient fast; fluid and electrolyte resuscitation; and parental (narcotic) analgesia. If the patient is vomiting, or if there is evidence of an ileus or gastric distention, nasogastric tube placement is appropriate [1]. Indomethacin has been reported to reverse the inflammatory changes in ACC and improve gallbladder contractility when given early, and diclofenac has been reported to reduce the rate of progression to ACC in patients with symptomatic

Fig. 3. Cholecystitis. MRI study (heavily T2-weighted) showing a thickened gallbladder wall (A, solid arrows), sloughed mucosa in the gallbladder lumen (A, open arrow), as well as pericholecystic fluid (B, open arrow).
gallstones; however, neither of these agents is used widely for this purpose [15,16,39]. Intravenous antibiotics are usually commenced empirically when there are systemic signs (eg, high fever, tachycardia, and hypotension) or if there has been no improvement over 12 hours with conservative management [3]. The favorable results of antibiotic treatment that reduces septic complications seems to depend more on adequate serum rather than tissue concentrations [30]. The choice of antibiotic varies with local experience and patterns of bacterial resistance. Single-agent therapy with an extended spectrum cephalosporin provides safe, appropriate coverage in most cases; however, in severe or high-risk cases, anaerobe cover is required (ie, by adding a nitroimidazole) [41,42]. In patients undergoing early uncomplicated cholecystectomy, prolonging the antibiotic course for more than 1 day postoperatively has not shown to be of benefit [43]. It must be emphasized that the optimal therapy in ACC is surgery, not antibiotic therapy.

**Cholecystectomy**

Surgical cholecystectomy has been the treatment of choice for ACC since Langenbuch first described it in 1882. Key issues regard the timing and the type of surgery and the treatment of those unfit for surgery. Approximately 20% of patients with ACC require emergency surgery because of clinical deterioration or other features of complicated disease, such as peritonitis or perforation [2,3]. If the nature of the symptoms is uncertain, exploratory surgery may be indicated [6]. For the remaining patients, early surgery has been shown to be superior to delayed surgery. Six randomized controlled trials before and after the advent of laparoscopic cholecystectomy (LC) have compared early surgery (within days of admission) with delayed surgery (performed 6 to 8 weeks after the event) [4–49]. These studies have found that mortality and complication rates of early cholecystectomy are no different than for delayed surgery, and that early surgery is preferable because of reduced total hospitalization costs and morbidity because approximately 10% to 25% of patients are admitted with recurrent complications of gallstone disease.

Laparoscopic cholecystectomy has changed the approach to biliary disease in the last two decades and it has been become the procedure of choice for elective cholecystectomy. Three randomized controlled trials [48–50] and multiple nonrandomized trials [51–56] studied the use of LC in ACC. These trials confirm that early LC is a safe and feasible option in ACC. Furthermore, these trials suggest LC within 48 to 72 hours of presentation (early surgery) is preferable to surgery after 3 to 5 days of conservative therapy (interval surgery) and surgery delayed 6 to 12 weeks after the attack (delayed surgery). The main reason for this conclusion is reduced conversion rates in the early surgery group when compared with the interval surgery group and reduced hospital stay in the early surgery
group. Indeed, early in the course of ACC inflammation and fibrosis are limited and tissues planes are easier to find, facilitating laparoscopic dissection. There may, however, be an increased risk of gallstone spillage with LC and this may increase the potential risk of subsequent abscess formation [57].

Percutaneous cholecystostomy

In high-risk patients, where the risks of surgery may outweigh the benefits, percutaneous cholecystostomy has been reported as an acceptable option with technical success in most series of over 90% [58–64]. This procedure can be performed at the bedside under ultrasound control and gives clinical improvement in 75% to 90% of patients with reported mortality rates of 5% to 20% (usually related to underlying condition) [58,59,63,64]. An incomplete or poor response after 48 hours should prompt a search for alternate sources of sepsis, complications, or tube dislodgment [3]. Once the patient has recovered, elective cholecystectomy should be performed [65]. An attempt at elective LC is reasonable in this setting [64,66,67]. Alternatively, if the patient remains unfit for surgery, percutaneous extraction of the calculi subsequently can be performed under radiologic or cholecystoscopic guidance [62,68,69]. In selected cases gallstone dissolution (with methyl tert-butyl ether) and disruption (with extracorporeal lithotripsy) have been reported [70,71].

Outcomes and complications of ACC

The reported mortality rate of ACC treated with LC is less than 0.5% with reported morbidity rates of 5% to 20% [48,49,52–55,72–75]. A number of complications of ACC may occur including gallbladder perforation, gallbladder gangrene, emphysematous cholecystitis, and empyema. The presence of any of these calls for urgent surgery. Risk factors for complications include advanced age; male gender; and associated diseases, such as diabetes, high temperature, and significant leukocytosis [76]. Gangrenous cholecystitis occurs in 2% to 30% of cases of ACC [3,76]. Perforation of the gallbladder is present in 3% to 10% of patients with ACC [2]; the most common form of perforation is subacute, with walling off of the process as a pericholecystic abscess [77]. Free perforation is less common; occurring usually in institutionalized patients, it presents with general peritonitis, and has a high mortality rate [77]. Emphysematous cholecystitis is an uncommon condition occurring when there is secondary infection of the gallbladder wall with gas-forming organisms. It is most common in elderly, diabetic men and it is treated with immediate administration of antibiotics (directed at anaerobes, coliforms, and clostridia species) and urgent surgery because of the high incidence of free perforation [6]. A clinical algorithm for the management of ACC is presented in Fig. 4.
Acute acalculous cholecystitis

Acute acalculous cholecystitis (AAC) is an acute inflammation of the gallbladder in the absence of demonstrable cholelithiasis [78]. It has been suggested that a more appropriate name is “necrotizing cholecystitis” to distinguish it as a distinct entity [79]. AAC accounts for 2% to 12% of all cases of cholecystitis, and in the United States 5% to 10% of cholecystectomies are performed for AAC [4,78,80]. AAC most frequently occurs in those who are critically ill from trauma, sepsis, or burns [4,78,80]. Compared with ACC, AAC tends to have a more complicated course and a higher morbidity and mortality [80]. Reported mortality rates range from 10% to 50%, in part because of the severity of the underlying disease [78,80]. Other reported risk factors include vascular disease, male gender, systemic vasculitides, HIV infection, diabetes mellitus, acute renal failure, immunosuppression, prolonged fasting, and total parenteral nutrition [78,80–82]. It has also been reported in patients without identifiable risk factors [83,84].

Pathophysiology

The pathogenesis of AAC is likely multifactorial with gallbladder stasis, paresis, and ischemia possibly playing a role [1,78]. Most patients with this condition have fasted for prolonged periods, are immobile, and often have

**Fig. 4. Clinical algorithm for the management of ACC.**
experienced hemodynamic instability, and over 70% may have associated atherosclerotic disease [82]. It is postulated that concentrated, stagnant bile and reduced gallbladder perfusion lead to a chemical and ischemic injury to the gallbladder epithelium [78]. Cholecystectomy specimens from patients with AAC (but not ACC) demonstrate impaired gallbladder microcirculation suggesting that splanchnic vasoconstriction or intravascular coagulation may be important events [85,86]. As in ACC, infection is thought to be a secondary event [17].

**Clinical presentation and diagnosis**

The clinical features of AAC may be the same as ACC; however, some or all of these features may be absent [87]. Indeed, pyrexia may be the only symptom, and up to 75% of cases may initially not have symptoms referable to the right upper quadrant [78]. Diagnostic tests used are those outlined for ACC. Ultrasound is usually the first line of investigation because it is widely available, can be performed at the bedside, and can detect concomitant lesions [3,87]. The sonographic features are similar to those of ACC, but without calculi [87,88]. Reports of ultrasound sensitivity have varied from 29% to 92% with specificity being over 90% [80,88,89]. CT is often performed because it is superior to sonography in detecting other intra-abdominal pathology [90]. The role of hepatobiliary scintigraphy in AAC is debated [78]. It is highly sensitive in diagnosing AAC; however, it may lack specificity because fasting for prolonged periods may result in thick, viscous bile and a false-positive scan [4,91]. Morphine-augmented scintigraphy may overcome this shortcoming, and it may complement sonography in cases where the diagnosis is uncertain [80,89]. In practice multiple studies may be necessary before the diagnosis becomes apparent [80,89].

**Treatment**

Initial supportive management is similar to ACC. Thereafter the management depends on the patient’s condition. In patients fit for surgery, open cholecystectomy (OC) has been the traditional approach [2,3,80]. LC has also been reported as an acceptable alternative in the early stages of AAC [2,92]. In critically ill patients, cholecystostomy is often preferred. Percutaneous cholecystostomy has been reported in a number of uncontrolled trials with favorable success rates [93,94]. The catheter may be left in place for 6 to 8 weeks and if no gallbladder stones are present at postdrainage cholangiography, the catheter is removed and cholecystectomy is usually unnecessary. Endoscopic transpapillary gallbladder drainage has been described for AAC but this technique has few advantages and several potential disadvantages (including cost and need for sedation) compared with percutaneous cholecystostomy [95].
Acute cholangitis

Acute cholangitis occurs as a result of bacterial infection superimposed on obstruction of the biliary tree. Of the common complications of gallstones, cholangitis is the most rapidly lethal entity, making accurate diagnosis and early treatment imperative [6]. Patients who fail conservative therapy and do not have appropriate drainage have mortality rates approaching 100% [96–98].

Pathophysiology and etiology

Bile is normally sterile [99]. The sphincter of Oddi, bile flow, and bacteriostatic properties of bile help maintain this sterility [100]. It is believed that biliary obstruction reduces antibacterial defenses, causes a state of immune dysfunction, and increases small bowel bacterial colonization [98]. Bacteria are able to gain access to the biliary tree; however, the precise route of infection is uncertain. Ascent from the duodenum or seeding through the portal vein are the most likely sources, but other possibilities include spread through the lymphatics or hepatic secretion [98,101]. Once obstructed bile is colonized, stasis allows for enhanced bacterial multiplication, whereas increased ductal pressure facilitates spread into the lymphatics and bloodstream [98].

Biliary obstruction is necessary but not sufficient to cause cholangitis [6]. Partial obstruction is associated with a higher rate of infection than complete obstruction, and obstruction from calculi is associated with a much higher rate of cholangitis than neoplastic obstruction (which is associated with cholangitis in about 10% of cases) (Fig. 5) [98,101]. Biliary obstruction after biliary intervention carries a very high risk of cholangitis [2]. The organisms most commonly cultured in cholangitis are *E coli*, *Enterococcus*, *Klebsiella*,

Fig. 5. Impacted bile duct stone. Endoscopic image of a patient with acute cholangitis showing an impacted stone in the ampulla of Vater before (*A*) and following endoscopic sphincterotomy (*B*). (See also Color Plate 17.)
and Enterobacter [98,102]. Pseudomonas, skin, and oral flora may be found after biliary interventions and anaerobes are found in up to 15%, most commonly in elderly patients after biliary surgery [6,100,102]. The most common cause of cholangitis in the United States is choledocholithiasis secondary to cholelithiasis with common bile duct stones being found in up to 10% of patients who present with symptomatic gallstones [1,98]. Primary bile duct stones are unusual in North America but are common in Hong Kong and Southeast Asia where Oriental cholangiohepatitis is endemic [6]. Less common causes of cholangitis include obstructing primary tumors of the ampulla, bile ducts, or pancreas; metastatic tumors to the porta hepatis or peripancreatic lymph nodes; benign strictures; and primary sclerosing cholangitis. With increased biliary interventions, postprocedural cholangitis is becoming more common [2]. Rare causes of cholangitis include obstruction from hemobilia, parasites, and hereditary abnormalities of the biliary tree [1,100].

Clinical presentation and diagnosis

Presentation of cholangitis is variable with the classic Charcot’s triad of fever, right upper quadrant pain, and jaundice present in 50% to 70% of patients [6,98,103,104]. Fever is usually present in 90% of cases, with jaundice and abdominal pain present in approximately 60% and 70%, respectively [98]. A small proportion of patients present with altered mental status (10% to 20%) and hypotension (approximately 30%) (the so-called “Reynold’s pentad” when present with Charcot’s triad) [6,98,103,104]. Right upper quadrant tenderness is present in two thirds of patients but signs of peritoneal irritation are less common [1]. Severe cholangitis may be associated with hepatic microabscesses that usually carry a poor prognosis (Fig. 6).

Fig. 6. Hepatic abscesses. Patient with acute cholangitis and a cholangiogram that demonstrates a multitude of small hepatic abscesses in continuity with the biliary tree.
The diagnosis of acute cholangitis is often made clinically but laboratory and imaging confirmation is important to enable distinction from other conditions, such as acute cholecystitis, hepatic abscesses, and pancreatitis. Typically, laboratory investigations reveal leukocytosis, hyperbilirubinemia, elevated alkaline phosphatase, and mild elevation of transaminases [6,98,103]. Serum amylase may be elevated in up to 30% of patients [1]. Ultrasonography or CT scan are the most commonly used first-line imaging modalities. Ultrasound is highly sensitive for cholelithiasis [37] but less sensitive (approximately 50%) for choledocholithiasis [105]. Choledocholithiasis may, however, be inferred if there is associated biliary dilation in the presence of cholelithiasis. A normal ultrasound does not rule out cholangitis [6]. Most studies suggest that CT is superior to ultrasonography for determining the level of biliary obstruction [106–108]. MRI and MR cholangiopancreatography are accurate for detecting choledocholithiasis and determining biliary anatomy and specific features of cholangitis have been described [109], but this test is probably of limited value in acute cholangitis. Endoscopic retrograde cholangiopancreatography (ERCP) is highly accurate for determining the cause of biliary obstruction and allows appropriate intervention where required. Given its potential for complications and availability of accurate noninvasive imaging ERCP should not be used solely as a diagnostic modality. Rather, it should be used when the likelihood of intervention is high, as is often the case in patients with clinically suspected cholangitis. Indeed, if cholangitis is suspected in a patient with cholelithiasis and the likelihood of common bile duct stone is high [100,110,111], the authors believe investigations should be limited (to blood tests and transabdominal ultrasound) and early referral made for ERCP.

**Treatment**

Initial management consists of correction of fluid and electrolyte deficits; correction of coagulopathy (caused by vitamin K deficiency from prolonged jaundice or low platelets from sepsis); and analgesia. In all cases of suspected cholangitis, blood cultures should be obtained and empirical antibiotics commenced. Although particular agents (eg, fluoroquinolones) have better biliary penetration, this has not been shown to be of primary importance in affecting outcome [98]. An aminoglycoside and ampicillin are no longer the ideal regimen because of emerging gram-negative resistance and concerns regarding nephrotoxicity [98,112]. Multiple newer regimens have been shown to be effective, including combination therapy with an extended-spectrum cephalosporin, metronidazole, and ampicillin; single-agent or combination fluoroquinolones; and ureidopenicillins alone or with metronidazole [98,113–118]. The choice of agent needs to be guided by local sensitivities, costs, and the need for anaerobic coverage (eg, in the elderly and those with biliary manipulation). The authors’ practice is to use a single
broad-spectrum agent, such as a ureidopenicillin (eg, ticarcillin and clavulanic acid) where possible. All patients with ascending cholangitis require biliary drainage. In patients who respond to medical therapy this may be performed semi-electively during the same admission (and ideally within 72 hours). Approximately 10% to 15% of patients fail to respond (within 12 to 24 hours) or deteriorate after initial medical therapy and these patients need urgent biliary decompression [98,119,120]. Delay in this situation increases the chance of an adverse outcome [121].

Endoscopic biliary drainage

An ERCP with bile duct drainage or clearance is the treatment of choice for decompressing the biliary system in acute cholangitis (Fig. 7). It has a success rate of 90% to 98% and is superior to surgical or percutaneous drainage in multiple studies [122–127]. Lai et al [124] randomized 83 patients with acute cholangitis to undergo endoscopic or surgical decompression and found that mortality (10% versus 32%) was significantly lower in the endoscopic arm [124]. Other studies have confirmed this [123,126,128].

Fig. 7. Bile duct stones. Patient with acute cholangitis and a cholangiogram that demonstrates multiple common bile duct stones, and a basket that has been inserted for endoscopic stone removal (solid arrow).
ERCP has also been shown to have lower morbidity rates, shorter length of hospitalization, and a higher definitive success rate than percutaneous drainage [125,127].

Endoscopic options are biliary drainage with a plastic stent or nasobiliary catheter or endoscopic sphincterotomy (ES) and bile duct clearance. In the critically ill or in patients with a coagulopathy, there are concerns that ES may increase the risk of complications and procedure time [98]. Although this has never been shown to be the case in clinical trials the concern is legitimate, especially because biliary drainage without sphincterotomy is safe and effective in such patients [121,125,127]. In the only randomized trial directly to compare use of nasobiliary catheter with biliary stent for acute cholangitis, Lee et al [129] found both to be equally effective but the stent was associated with less postprocedural discomfort and avoided the potential inadvertent removal [129]. In patients who are not critically ill, ES and bile duct clearance should be attempted, and this is the definitive procedure in patients who have had a cholecystectomy. Complications of ERCP and ES include pancreatitis, bleeding, stone impaction, and perforation and occur in 5% to 10%, with bleeding being a particular problem in those with cholangitis [130]. Antibiotics should be continued after successful bile duct clearance because patients remain at risk of developing cholecystitis and empyema [131]. Total duration of antibiotic therapy depends on response and the authors’ practice is to continue therapy for 7 days. A Dutch study suggests, however, that in patients with an uncomplicated course, and good response to biliary drainage, 3 days of antibiotic therapy may be sufficient [132].

Percutaneous transhepatic biliary drainage

Percutaneous transhepatic biliary drainage can be performed successfully in up to 90% of patients with biliary tract obstruction [133]. It has higher rates of morbidity (30% to 80%) and mortality (5% to 15%) than endoscopic drainage [1,125,133,134]. Nonetheless, percutaneous transhepatic biliary drainage may be preferred over ERCP in specific situations, such as hepatolithiasis; intrasegmental cholangitis [135]; when the papilla is inaccessible endoscopically (eg, after Roux-en-Y formation); or when ERCP has failed. As with ERCP, caution must be taken to correct coagulopathies preprocedurally.

Surgical biliary drainage

Open surgery has been used to treat cholangitis for almost 100 years. It is associated with mortality rates of up to 40%, however, and is rarely used as the first-line method of biliary drainage [103,119,136]. When it is performed, emergency surgery may be limited to choledochotomy, decompression, and T-tube insertion [98,103,119]. A proportion of patients
treated endoscopically or percutaneously requires definitive surgical intervention, and the mortality is low in patients who are able to undergo this electively [1]. A clinical algorithm for the management of cholangitis is presented in Fig. 8.

Management of the gallbladder after bile duct clearance

Whether subsequent cholecystectomy is indicated for patients with cholelithiasis who have had an episode of cholangitis successfully treated with ES and bile duct clearance is a matter of debate. The rationale for proceeding to cholecystectomy is to prevent further biliary complications. Management strategies need to be individualized and guided by risk factors for surgery and further biliary complication. In retrospective and non-randomized prospective studies, the risk of patients with common bile duct stones developing subsequent biliary complications during follow-up has ranged from 4% to 12% [137–141]. Four randomized controlled trials have been reported that specifically address this issue [142–145]. Boerma et al [142] studied 120 patients aged 18 to 80 years with proved symptomatic common bile duct and concomitant gallbladder stones who underwent ES and bile duct clearance. Patients were randomized to LC within 6 weeks of endoscopic stone clearance or to a “wait and see” approach. During a mean follow-up period of 30 months, 47% of patients in the wait and see group had recurrent biliary symptoms compared with 2% in the LC group. Furthermore, 37% of the wait and see group needed cholecystectomy. Targarona et al [143] randomized 98 elderly and other high-risk patients with symptoms likely caused by bile duct stones either to ES alone or open surgery and found that after a mean follow-up of 17 months, biliary symptoms recurred in 20% of the ES group and 6% of the surgery group. Hammarstrom et al [144] randomized 83 patients with bile duct stones to ES and stone removal or open surgery (cholecystectomy and bile duct exploration) and found that after more than 5 years, 20% of the ES group underwent cholecystectomy because of recurrent biliary symptoms, whereas 2% of patients in the surgery group had recurrent symptoms from bile duct stones. During the follow-up period, nonbiliary mortality was significantly more common in the ES group. Panis et al [145] randomized 206 patients with common bile duct stones to endoscopic therapy alone or surgery and found early surgery was required in 19% in the endoscopic group, whereas only 2% of the surgical group needed reoperation. Taken together these studies strongly support the recommendation that patients with cholangitis should undergo elective LC after bile duct clearance if they are fit for surgery (unless an open approach is known to be required). This may not apply to Asian patients where bile duct stones may originate from intrahepatic stones and hence cholecystectomy may not prevent further biliary complications. Two nonrandomized prospective studies from Hong Kong have not shown a benefit of cholecystectomy over ES alone in
Fig. 8. Clinical algorithm for the management of ascending cholangitis. *In selected clinical scenarios PTBD may be preferred to ERCP (see text). ERCP, endoscopic retrograde cholangiopancreatography; ES, endoscopic sphincterotomy; ESWL, extracorporeal shock wave lithotripsy; LC, laparoscopic cholecystectomy; PTBD, percutaneous transhepatic biliary drainage.
preventing recurrent cholangitis or biliary complications in patients with bile duct stones [139,146]. In contrast a retrospective study from Hong Kong suggested that patients with bile duct stones treated with ES alone had an increased risk of recurrent biliary symptoms when compared with those who also underwent LC and the results of further prospective trials are awaited [147]. Similarly, a recent randomized trial from Hong Kong showed that, in patients with cholangitis and gallbladder stones but in the absence of common bile duct stones, ES resulted in decreased durations of cholangitis and hospital stay, but no reduced incidence of recurrent acute cholangitis [148].

Summary

Cholelithiasis is a prevalent condition in Western populations. Most cases are asymptomatic but complications can occur. Acute cholangitis, cholecystitis, and gallstone pancreatitis are the most common biliary tract emergencies and are usually caused by biliary calculi. Whenever possible, acute cholecystitis should be treated with early LC. AAC is an uncommon condition usually affecting patients with significant comorbidities. Treatment is usually with percutaneous cholecystostomy, which often is also the only required therapy. Endoscopic drainage is the preferred form of biliary decompression in acute cholangitis and these patients should subsequently undergo elective LC unless unfit for surgery. Effective and optimal management of biliary tract emergencies relies on close cooperation between gastroenterologist, surgeon, and radiologist.

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References


