Cholecystitis

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Overview

Cholecystitis in its varied forms is the most prevalent surgical entity afflicting populations of industrialized countries. The most common cause of cholecystitis and biliary colic is choledolithiasis. Autopsy findings show that 11% to 35% of American adults, or roughly 25 million people, have gallstones. Some 1% to 2% of people who have choledolithiasis develop symptoms or complications per year [1]. These complications include biliary colic, acute or chronic cholecystitis, choledocholithiasis, cholangitis, pancreatitis, and gallbladder carcinoma. It has been estimated that nearly 700,000 cholecystectomies are performed yearly in the United States. This article addresses the pathophysiology and clinical management of symptomatic choledolithiasis, acute calculous and acalculous cholecystitis, chronic cholecystitis, and complications of cholecystitis.

Symptomatic cholelithiasis: gallstones

Symptomatic cholelithiasis is defined as gallbladder pain in the presence of gallstones. Gallstones arise from the precipitation of cholesterol and calcium salts in supersaturated bile. They are classified by their content of cholesterol as either cholesterol stones or pigmented stones. Pigmented stones receive their color from their concentration of calcium bilirubinate. Black stones are small and tarry and are typically found associated with cirrhosis and hemolytic disorders, such as sickle cell disease and hereditary spherocytosis. Brown gallstones are more common in Asian populations and are associated with disorders of biliary motility and bacterial infection. In the United States, 70% to 80% of gallstones are of the cholesterol variety.

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Clinical manifestations and diagnosis

It is estimated that 20% of individuals who have gallstones have biliary colic. This term describes the constellation of symptoms experienced by a patient when the gallbladder contracts against an outlet obstruction, usually a gallstone lodged in the gallbladder neck or in the Hartman pouch. It is typically described as a sharp cramping pain localized to the right hypogastrium, often radiating to the right scapula or interscapular area. The symptoms commonly occur following large or fat-rich meals, and often at night when they awaken the patient from sleep. Associated symptoms include nausea, chills, malaise, bloating, belching, and occasionally diarrhea. Biliary colic is frequently indistinct or mild, limited to waves of nausea or gastric reflux symptoms. Uncomplicated biliary colic usually resolves spontaneously within 30 minutes to 6 hours, or with the administration of an analgesic. Nevertheless, once individuals begin to experience symptoms, the symptoms tend to become recurrent.

On physical examination, the patient may have localized right upper quadrant tenderness. Between episodes of biliary colic, however, the physical examination may be unimpressive and without tenderness. The workup for symptomatic cholelithiasis, particularly in the emergency room setting, oftentimes needs to exclude from the differential diagnosis angina, nephrolithiasis, pancreatitis, gastritis, and peptic ulcer disease. Laboratory studies, including leukocyte count, are generally normal. The transabdominal ultrasound of the right upper quadrant is the gold standard for diagnostic imaging. Although sensitive for identifying gallstones or sludge, ultrasound is limited by obesity, bowel gas, and operator skills. Gallbladder wall thickening or pericholecystic fluid suggests acute or chronic cholecystitis. Given that 20% of gallstones are radiopaque, some stones may occasionally be identified on CT scan or plain radiographs of the abdomen.

Treatment

Treatment of symptomatic cholelithiasis is electively scheduled routine laparoscopic cholecystectomy. An alternative approach described by some centers is the mini-cholecystectomy performed through a 5-cm midline incision [2]. Before surgery, the patient is advised to maintain a strictly low-fat diet and avoid heavy meals. Patients must also be counseled regarding the signs and symptoms that suggest progression to cholecystitis and the need for more urgent therapy.

Acute calculous cholecystitis

Pathophysiology

The primary cause of obstructive cholecystitis is gallstones. Of all individuals who have gallstones, 1% to 3% develop cholecystitis. Other causes of
obstructive cholecystitis include primary tumors of the gallbladder or common duct, benign gallbladder polyps, parasites, metastatic tumors to the gallbladder or the periportal lymph nodes, and even foreign bodies, such as bullets [3–6]. Prolonged gallbladder outlet obstruction leads to acute cholecystitis. Obstruction at the neck of the gallbladder causes increased intraluminal pressure leading to venous congestion, compromised blood supply, and impaired lymphatic drainage. The mucosa becomes ischemic, releasing inflammatory mediators, such as prostaglandins I₂ and E₂. Localized mucosal trauma causes lysosome release of phospholipase, converting lecithin in the supersaturated bile to lysolecithin. Lecithin normally protects the mucosa against bile acids, but lysolecithin is a detergent and toxic to the mucosa. Wall thickening occurs with edema, vascular congestion, and intramural hemorrhage. Mucosal ulcers develop with focal areas of wall necrosis. Histologically, there is dense infiltration of neutrophilic leukocytes, microabscesses, and secondary vasculitis [7]. Eventually there may be secondary bacterial infection, accumulation of purulent fluid with the formation of an empyema, and perforation with widespread peritonitis with sepsis. Other complications of acute cholecystitis include hepatic abscess and intra-abdominal abscess.

Primary bacterial infection is not believed to play an initial role in cholecystitis, but secondary infection may complicate up to 50% of clinical courses. Some 40% to 50% of acute cholecystitis cases have been shown to have positive bile cultures. Bacteria that infect the bile include gram-negative bacilli (Escherichia coli, Klebsiella spp, Enterobacter spp), anaerobes (Bacteroides, Clostridia spp, Fusobacterium spp) and gram-positive cocci (enterococci) [8]. Overgrowth of gas-producing bacteria within the gallbladder can lead to emphysematous cholecystitis.

Clinical manifestations

The average patient presenting with acute cholecystitis ranges from 40 to 80 years old. Most men presenting with biliary colic present with acute cholecystitis. Because the incidence of stones in females is higher, however, overall there are more female patients who have acute cholecystitis. The female/male ratio is approximately 3:1.

Patients present with right upper quadrant or epigastric colic that either persists or escalates over 12 to 24 hours. Although it may be ameliorated by administration of analgesia, it does not typically resolve entirely. Commonly, patients have a history of more mild, sometimes escalating antecedent episodes of biliary colic. Other symptoms include chills, malaise, nausea, vomiting, and anorexia. The examiner must remember to question the patient about icteric orange-tea-colored urine or clay-colored stool, which would raise the suspicion of a common duct obstruction.

On physical examination, patients often have low- to moderate-grade fever, tachycardia, and marked right upper quadrant tenderness. Up to 25%
of patients have a palpable distended gallbladder. The classic Murphy sign, the abrupt inhibition of inspiration with palpation directly over the gallbladder fossa, is commonly seen. Abdominal wall guarding or rigidity must raise the suspicion of gangrenous cholecystitis or perforation.

Laboratory and radiologic diagnosis

Laboratory examination often demonstrates a mild to pronounced leukocytosis with left shift. The bilirubin, alkaline phosphatase, transaminases, and the amylase may be mildly elevated. A total bilirubin greater than 3 mg/dL should raise concerns for choledocholithiasis. Generally laboratory values are nonspecific, but they can be useful in ruling out alternative diagnoses, such as acute pancreatitis.

Plain films of the abdomen are of minimal value in diagnosing acute cholecystitis. In the global evaluation of the acute abdomen, however, plain films assist in ruling out intra-abdominal free air that can be seen with diverticulitis or a perforated peptic ulcer. Ultrasound of the right upper abdomen is sensitive and specific for the diagnosis of acute calculous cholecystitis. The identification of choleliths or sludge, wall thickening (>4 mm), or pericholecystic fluid all support the diagnosis of acute cholecystitis. The sonographic Murphy sign, which involves painful replication of the biliary colic and inhibition of inspiration by palpating the gallbladder in real time while visualizing it, is helpful in the diagnosis of cholecystitis. The measured common bile duct diameter is also important to consider. Although normal common bile duct caliber is often slightly increased in the elderly population, a diameter greater than 8 mm must raise concern over common duct obstruction. CT scan of the abdomen also demonstrates many of the radiologic features of cholecystitis, but is a much less sensitive and more time-consuming and expensive imaging modality.

The HIDA scan (99mTc-HIDA cholescintigraphy) is the most accurate test for cholecystitis, with a sensitivity of 97% and specificity of 87% [9]. The gallbladder is usually visualized within 30 minutes, and the absence of radiotracer uptake by 4 hours is considered positive for cystic duct occlusion. A prolonged fasting state diminishes the accuracy of the HIDA scan, and a false-positive rate of up to 40% has been seen after 5 days of non per os (NPO) status. Identification of radiotracer in the pericholecystic space indicates perforation. Generally, the HIDA scan is unnecessary in most cases of acute calculous cholecystitis given the availability of an ultrasound examination. Most practitioners reserve use of the HIDA scan for clinical situations in which there is diagnostic ambiguity. One example is the evaluation of incidental cholelithiasis and gallbladder wall thickening identified by ultrasound in a patient who does not have signs or symptoms of cholecystitis. A second example can occur during a sepsis evaluation in an obtunded or heavily sedated ICU patient who cannot provide an accurate history or physical examination. Third, patients who have hepatitis or cirrhosis
commonly have abdominal complaints mimicking cholecystitis, and ultrasound findings of gallbladder wall edema, ascites, and gallbladder distension when fasting. In these patients, the HIDA scan can help to rule out cholecystitis, thereby avoiding consideration of cholecystectomy or unnecessary antibiotic treatment.

Treatment

Treatment of acute calculous cholecystitis commences with fluid resuscitation, making the patient NPO, providing analgesia, and initiating antibiotic therapy. The role of antibiotics in uncomplicated acute cholecystitis has not been fully determined when surgical treatment is completed in a timely fashion [8]. It is often difficult to clinically evaluate whether secondary bacterial infection has occurred, however, or whether the cholecystitis has evolved to gangrene and perforation. Broad-spectrum antibiotic coverage is therefore recommended. Commonly used regimens are ampicillin with gentamicin, ampicillin-sulbactam, piperacillin-tazobactam, a third- or fourth-generation cephalosporin, or a third-generation fluoroquinolone, such as moxifloxacin.

Laparoscopic cholecystectomy is currently the standard definitive management for acute calculous cholecystitis. It is well recognized to be more difficult in the acute setting, but with adequate experience it has been shown to be effective and safe [10]. Thickening of the gallbladder wall and friability can make the gallbladder difficult to grasp and limit the surgeon’s ability to elevate the fundus or retract the infundibulum for exposure. Inflammation around the triangle of Calot can impair visualization of the ductal anatomy and the cystic artery. Conversion to an open cholecystectomy must be considered if there is any uncertainty about the anatomy before clipping or dividing ductal or arterial structures. Conversion to an open surgery must not be considered a failure and the possibility of conversion should be discussed preoperatively with the patient and included on the surgical permit.

Even during an open cholecystectomy, there may be such a dense inflammatory reaction at the base of the gallbladder as to preclude safe ligation of the cystic duct without endangering the portal structures. In these cases, one can consider performing a partial cholecystectomy, where a small portion of the gallbladder wall at the base is left behind. A permanent stitch can be carefully placed within the gallbladder to occlude the orifice of the cystic duct. The remnant mucosa is fulgurated and a surgical drain placed. If a postoperative bile leak occurs, an endoscopic retrograde cholangiopancreatography (ERCP) with common duct stent placement can be performed.

The timing of cholecystectomy for acute calculous cholecystitis has been a matter of much discussion in the literature. It is recognized that the presence of fever, marked leukocytosis, or diffuse abdominal tenderness portends possible necrosis, empyema, or rupture, and that emergent surgery within 12 to 24 hours is indicated. Patients who have diabetes often present later secondary to neuropathic impairment of pain sensation, and they have
rapid disease progression and greater infectious complications [11]. These patients warrant early cholecystectomy. Similarly, urgent cholecystectomy for acute cholecystitis is indicated in the elderly and the immunocompromised, because they often present with vague, nonspecific symptoms and comorbid medical conditions, and they have a higher incidence of complications [12].

Most patients who have uncomplicated acute cholecystitis can be treated supportively and scheduled for urgent cholecystectomy within 24 to 48 hours of presentation. Patients who undergo cholecystectomy within 48 hours of symptom onset have a 4% chance of conversion to an open procedure compared with a 23% chance of open cholecystectomy if surgery is delayed [13]. It has been recognized that patients presenting with symptom duration greater than 72 hours have higher rates of complications and conversion to open cholecystectomy. The practice of treating patients with acute cholecystitis with antibiotics and then a planned interval cholecystectomy 4 to 8 weeks later with the hope of operating when the acute inflammation has abated is no longer supported. Of those patients treated with antibiotics with intention of doing an interval cholecystectomy, 20% to 30% will re-present and require urgent surgery during the interval period [14–16]. The conversion rate can still be as high as 30%, even with an interval cholecystectomy [17]. Delayed interval surgery does not reduce the morbidity or conversion rate, and it increases the overall hospital stay [18].

It is not infrequent that the surgeon is asked to evaluate a patient who has acute cholecystitis in the critical care setting with overwhelming medical comorbidities that preclude safe surgical intervention. Often the cholecystitis is a major contributor to the patient’s critical status. For the patient who is a poor surgical candidate, antibiotic and supportive care and percutaneous transhepatic cholecystostomy is adequate intervention. This procedure can be performed under local anesthesia with CT scan guidance or by ultrasound guidance at the bedside. For severe acute cholecystitis, percutaneous cholecystostomy is superior to gallbladder aspiration [19]. Eventually definitive treatment should be considered when possible; roughly half of patients reaccumulate gallstones within 5 years, even if all the gallstones are removed by cholecystostomy. If the patient’s condition improves and he or she can tolerate surgery later, interval cholecystectomy can be performed in 3 to 6 months.

**Acute acalculous cholecystitis**

**Epidemiology**

Acute inflammation of the gallbladder in the absence of cholelithiasis accounts for roughly 2% to 15% of acute cholecystitis cases and is the indication for 1% to 2% of laparoscopic cholecystectomies. Risk factors include old age, critical illness, burns, trauma, major surgical operations, long-term total parenteral nutrition, diabetes, immunosuppression, and
childbirth. Acute acalculous cholecystitis occurs in 0.2% of surgical intensive care admissions and has a mortality rate as high as 40% [20]. A higher proportion of patients who have acute acalculous cholecystitis are male. Children can be affected, especially after severe viral infections. Patients who have HIV and opportunistic infections, such as cytomegalovirus, cryptosporidium, *Mycobacterium tuberculosis*, *Mycobacterium avium intracellulare*, or mycotic infections, have been reported to develop acute acalculous cholecystitis. Complications, such as gangrene, empyema, and perforation, are more common with acalculous cholecystitis than with calculous cholecystitis. Deterioration of clinical status can be rapid.

**Pathophysiology**

The precise pathophysiology of acute acalculous cholecystitis remains poorly understood. Most known risk factors are associated with bile stasis within the gallbladder, leading to increased viscosity and the formation of sludge, which likely contributes to bacterial overgrowth. In elderly patients and patients on vasoconstrictor medications to support blood pressure, mucosal ischemia likely contributes to local inflammation and necrosis of the mucosal barrier. Histologically there are no specific differences between acute calculous and acalculous cholecystitis.

**Clinical manifestations**

Timely diagnosis of acute acalculous cholecystitis can be difficult and requires a high index of suspicion. Patients may present with biliary colic and fever, or they may have nonspecific or subtle complaints, such as fatigue, indigestion, or nausea. Acalculous cholecystitis may be uncovered during an evaluation of fevers of unknown cause, especially in ICU patients for whom reliable physical examination is difficult. Laboratory findings are similar in acalculous and calculous cholecystitis. Ultrasound usually demonstrates gallbladder wall thickening and pericholecystic fluid, but no gallstones. It is difficult to interpret the findings of gallbladder wall edema or localized ascites in critically ill patients who have congestive heart failure, renal insufficiency, or hepatic disease, in which there is generalized anasarca. In these situations, the HIDA scan can be critical to determining the diagnosis. Often, however, the surgeon must rely on imperfect data and overall clinical impression.

**Treatment**

Treatment of acute acalculous cholecystitis is similar to that of calculous cholecystitis; supportive care, antibiotics, and emergent laparoscopic cholecystectomy. With acalculous cholecystitis, however, a greater proportion of patients are too ill to undergo anesthesia and surgery. In these situations, percutaneous transhepatic cholecystostomy is advised. Ninety percent of
these patients demonstrate clinical improvement [21]. Once the patient has recovered, the cholecystostomy tube can be removed without sequelae, as long as there is confirmation that the gallbladder contains no stones. Interval cholecystectomy is not necessary.

Although mentioned earlier as a possible complication of calculous cholecystitis, emphysematous cholecystitis most often occurs in the absence of gallstones. Men are affected three times as often as women, and it typically occurs in patients who have diabetes mellitus and are between 50 and 80 years of age [7]. Small vessel occlusive disease involving the cystic artery is believed to be a major contributing factor. Nearly half of bile cultures from these patients contain *Clostridia welchii*. The gallbladder characteristically contains malodorous gas and purulent bile. CT scan, and occasionally abdominal plain films, demonstrates gas within the gallbladder and gallbladder wall pneumatosis. Emergency intervention is indicated. Emphysematous cholecystitis can lead to rapid clinical deterioration with mortality as high as 15% because of gangrene or perforation.

**Chronic cholecystitis**

*Pathophysiology*

Chronic inflammation of the gallbladder is the indication for nearly 3% of cholecystectomies in adults. Chronic cholecystitis has many forms and the pathophysiology is poorly understood. It is believed that in most instances an evolving inflammatory process occurs with repeated episodes of low-grade gallbladder obstruction, resulting in recurrent mucosal trauma [7]. There is little correlation between the number of choleliths or their overall volume and the degree of gallbladder wall inflammation. In fact, 12% to 13% of patients who have chronic cholecystitis have no demonstrable stones. Bacterial infection of the bile does not seem to play a role; less than one third of bile cultures contain *E. coli* or enterococci. As each episode of acute inflammation resolves, neutrophilic infiltration is replaced with lymphocytes, plasma cells, macrophages, and eosinophils. Focal ulcerations and necrotic tissue are replaced by granulation tissue and collagen deposits. The gallbladder wall may become thickened or remain thin. The mucosa can remain intact, develop accentuated folds, or be flattened.

Particular mention should be made of several specific forms of chronic cholecystitis. Chronic obstruction by stones, tumor, fibrosis, or external compression of the cystic duct can lead to hydrops, wherein the gallbladder becomes markedly distended and the bile replaced with a clear or mucoid fluid. Gallbladder hydrops occurs in 3% of gallbladders removed in adults. When lymphocyte proliferation leads to the formation of prominent lymphoid follicles in the wall, the term “follicular cholecystitis” is applied. Xanthogranulomatous cholecystitis is found in 1.8% of removed gallbladders, wherein the gallbladder wall contains poorly demarcated firm yellow masses
that are histologically characterized by focal infiltrates of foamy histiocytes, plasma cells, lymphocytes, and fibrosis. Last, when there is chronic penetration of bile through ulcers or fissures in the mucosa into the subepithelium of the gallbladder wall, chronic scarification and deposition of dystrophic calcifications give the gallbladder a firm rock-hard quality. The phenomenon, called porcelain gallbladder, is associated with an increased risk for gallbladder carcinoma.

**Clinical manifestations**

The symptoms of chronic cholecystitis vary from classic severe biliary colic to vague or nonspecific complaints. Patients may report only intermittent episodes of nausea, reflux symptoms, food intolerance, or bloating. Symptoms may be as subtle as low-grade fever, mild upper abdominal discomfort, or chronic fatigue. Not infrequently patients who have chronic cholecystitis have been treated for gastritis, ulcer disease, or irritable bowel syndrome without appreciable improvement in their complaints.

The evaluation for chronic cholecystitis usually occurs in the outpatient or urgent care setting, often initiated by the primary care physician or gastroenterologist. The physical examination is typically unremarkable except when there is massive distension of the gallbladder associated with hydrops or a hard mass in the right upper quadrant associated with porcelain gallbladder. Specific laboratory abnormalities are unusual and ultrasound may show cholelithiasis with or without wall thickening. Dystrophic calcification of the gallbladder is well demonstrated by CT scan. Many patients are taken to the operating room with a diagnosis of symptomatic cholelithiasis only to find histologic evidence for chronic cholecystitis on pathologic examination. In cases with inconclusive constellations of symptoms and lack of objective studies to implicate the gallbladder, a cholecintigraphy study with cholecystokinin challenge, or emptying study, can help to identify biliary dyskinesia. Most patients diagnosed with significant biliary dysmotility experience symptomatic relief with cholecystectomy. There are frequently histopathologic changes of chronic inflammation present in these specimens. It remains uncertain whether impaired gallbladder emptying leads to chronic inflammation or vice versa. When the diagnosis is not straightforward, it is recommended to consider endoscopic gastroduodenoscopy, colonoscopy, CT scan, and in some instances, cardiology evaluation before scheduling a patient for cholecystectomy to exclude other causes, such as peptic ulcer disease, angina, tumor, or pancreatitis.

**Two complications of cholecystitis: Mirizzi syndrome and gallstone ileus**

Two clinical entities result from long-term inflammation of the gallbladder and deserve mention in a discussion of cholecystitis: Mirizzi syndrome and gallstone ileus.
Mirizzi syndrome

Mirizzi syndrome is the partial obstruction of the common hepatic bile duct secondary to stone impaction and chronic inflammation in the adjacent gallbladder Hartman pouch. This inflammatory process can evolve into an erosive fistula to the anterior or lateral wall of the duct. The Csendes classification stratifies the degree of injury, from type I or simple external compression of the common hepatic duct, to type IV, or complete destruction of the entire wall of the duct [22]. Hepatic duct involvement by inflammation has been demonstrated to be present in 0.3% to 3% of patients undergoing cholecystectomy. It is only diagnosed preoperatively in 8% to 62.5% of cases. Mirizzi syndrome can lead to an increased risk for bile duct injury during cholecystectomy (up to 22.2%) and to an increased risk for conversion to open cholecystectomy. Preoperatively, ultrasound imaging may show a large immovable stone in the neck of a contracted gallbladder and proximal biliary dilatation. On CT scan there may be an irregular cavity near the neck of the gallbladder with stones outside of the lumen. ERCP, MRCP, or percutaneous transhepatic cholangiography can usually delineate the cause and level of a fistula.

Open cholecystectomy is the standard surgical approach to repair in Mirizzi syndrome. If no fistula is present, a subtotal cholecystectomy is adequate. With a small fistula (type II), a subtotal cholecystectomy can be performed and a T tube inserted into the fistula, closing the gallbladder remnant around the tube. For larger fistulae (types III and IV), the standard treatment is hepaticojejunostomy. In 6% to 27.8% of patients determined to have Mirizzi syndrome preoperatively, carcinoma of the gallbladder was the final diagnosis [23]. Frozen section histology should be performed intraoperatively in all cases of Mirizzi syndrome and preoperative preparations made for possible radical cholecystectomy.

Gallstone ileus

The second clinical entity, gallstone ileus, occurs after the gallbladder has spontaneously fistulized to the bowel. Gallstones may then pass directly into the gastrointestinal tract, and if large enough, cause a mechanical obstruction. In 68% of cases the fistula is a cholecystoduodenal fistula, in 5% cholecystocolonic, and in 5% cholecystoduodenocolonic [24]. Typically the obstruction occurs at the narrow ileocecal junction. The term Bouveret syndrome is applied when the stone enters and obstructs the duodenum, creating a gastric outlet obstruction.

Gallstone ileus accounts for 1% of all small bowel obstructions. It is implicated in 25% of patients older than the age of 65 years when obstruction is not attributable to adhesive disease or hernias [25]. Most of the patients have no known history of cholecystitis. The diagnosis can be made preoperatively 73% of the time. Findings on plain abdominal films include dilated loops of small bowel consistent with obstruction, and in 40% of cases,
intrabiliary gas. The latter finding is pathologic in any patient who has not had common duct instrumentation or a choledochoenterostomy. Unless the passed stone contains significant calcium, it is not appreciable on plain films or CT scan. In addition to intrabiliary gas, CT scan may demonstrate a small contracted gallbladder and a short bezoar at the obstruction transition point.

At laparotomy, the obstructing cholelith is identified by manual inspection of the bowel. It is extracted by way of a transverse enterotomy or by performing a small bowel resection if there is evidence for pressure necrosis of the bowel wall. Care must be taken in searching for additional stones proximally, because in 16% of cases there is more than one stone. Missing a second intraluminal cholelith risks a recurrent obstruction in the immediate postoperative period.

Considerable debate in the literature has focused on the appropriate approach to treatment of the cholecystenteric fistula. Several reports describe chronic cholecystitis or cholangitis if the fistula is left intact. Others have found that the fistula can close spontaneously. Typically there is a dense cicatrix involving the contracted and shrunken gallbladder and the involved bowel. There is appreciable risk for ductal injury, and the need to repair a defect in the duodenum or colon in a setting of acute or chronic inflammation. A single-staged procedure to treat the acute bowel obstruction and the fistula is feasible if the patient is stable and able to tolerate an extended operation. In one study, the associated mortality was 16.9%. Only 10% of patients who had the gallbladder left in place had continued biliary symptoms and the recurrence of gallstone ileus was less than 5%. If no large choleliths are palpable in the gallbladder fossa at the time of initial exploration for obstruction, it is safe to leave the fistula and decide future surgical intervention based on the patient’s clinical course.

**Summary**

Rather than a single clinical entity, cholecystitis is a class of related disease states with different causes, degrees of severity, clinical courses, and management strategies. Appropriate care of the patient who has a diseased gallbladder requires a broad understanding of the acute, chronic, and acalculous cholecystitis syndromes, and awareness of their particular clinical nuances and potential complications.

**References**