Acute calculous cholecystitis: Clinical features and diagnosis

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INTRODUCTION

Cholecystitis refers to inflammation of the gallbladder. Acute cholecystitis predominantly occurs as a complication of gallstone disease and typically develops in patients with a history of symptomatic gallstones. Less often, acute cholecystitis may develop without gallstones (acalculous cholecystitis). (See "Overview of gallstone disease in adults", section on 'Natural history and disease course'.)

This topic will review the pathogenesis, clinical manifestations, and diagnosis of acute calculous cholecystitis. Separate topic reviews on gallstone disease and the management of acute calculous cholecystitis include the following:

- (See "Overview of gallstone disease in adults".)
- (See "Approach to the management of gallstones".)
- (See "Acalculous cholecystitis: Clinical manifestations, diagnosis, and management".)
- (See "Treatment of acute calculous cholecystitis".)

DEFINITIONS

Acute cholecystitis — Acute cholecystitis refers to a syndrome of right upper quadrant pain, fever, and leukocytosis associated with gallbladder inflammation. In the majority of patients,

acute cholecystitis is caused by gallstones, while acalculous cholecystitis accounts for about 5 to 10 percent of cases. (See "Acalculous cholecystitis: Clinical manifestations, diagnosis, and management".)

Chronic cholecystitis — Chronic cholecystitis is the term used to describe chronic inflammatory cell infiltration of the gallbladder seen on histopathology. The clinical significance of chronic cholecystitis is questionable. It is almost invariably associated with the presence of gallstones and is thought to be the result of mechanical irritation or recurrent attacks of acute cholecystitis leading to fibrosis and thickening of the gallbladder [1-3]. However, its presence does not correlate with symptoms since patients with extensive chronic inflammatory cell inflammation may have only minimal symptoms, and there is no evidence that chronic cholecystitis increases the risk for future morbidity [4]. (See "Overview of gallstone disease in adults".)

Some authors use the term "chronic cholecystitis" to describe gallbladder dysfunction as a cause of abdominal pain [5]. It is more appropriate in this instance to refer to the condition as pain due to biliary dyskinesia (which is attributed to sphincter of Oddi dysfunction), or pain due to functional gallbladder disorder (also called gallbladder dyskinesia). (See "Functional gallbladder disorder in adults".)

EPIDEMIOLOGY

Acute cholecystitis is the most common complication of gallstone disease and typically develops in patients with a history of symptomatic gallstones. In one systematic review, acute cholecystitis developed in 6 to 11 percent of patients with symptomatic gallstones over a median follow-up of 7 to 11 years [6].

PATHOGENESIS

Acute calculous cholecystitis occurs in the setting of cystic duct obstruction. However, in contrast to biliary colic, the development of acute cholecystitis is not fully explained by cystic duct obstruction alone [7,8]. Studies suggest that an additional irritant is required to develop gallbladder inflammation. One such irritant used in experimental models, lysolecithin, is produced from lecithin, a normal constituent of bile. The production of lysolecithin from lecithin is catalyzed by phospholipase A, which is present in gallbladder mucosa. This enzyme may be released into the gallbladder following trauma to the gallbladder wall from an impacted

gallstone [8]. Supporting this hypothesis is the observation that lysolecithin (normally absent in bile) is detectable in gallbladder bile in patients with acute cholecystitis [9].

Inflammatory mediators are released in response to gallbladder inflammation and further propagate the inflammation [10]. Prostaglandins, which are involved in gallbladder contraction and fluid absorption, probably play a central role in this process [11]. The prostaglandin hypothesis is supported by the observation that prostaglandin inhibitors can reduce intraluminal gallbladder pressure and relieve biliary colic [12-14].

Infection of bile within the biliary system probably has a role in the development of cholecystitis; however, not all patients with cholecystitis have infected bile. This observation was illustrated in a study of 467 subjects in whom bile samples were obtained from the gallbladder and common bile duct for aerobic and anaerobic culture [15]. Patients with a variety of hepatobiliary diseases and a healthy control group were included. Patients with gallstones, acute cholecystitis, and hydropic gallbladder had similar rates of positive cultures in the gallbladder and common bile duct, ranging from 22 to 46 percent; cultures were generally sterile in healthy subjects. The main species isolated were Escherichia coli, Enterococcus, Klebsiella, and Enterobacter.

Histologic changes of the gallbladder in acute cholecystitis can range from mild edema and acute inflammation to necrosis and gangrene. Occasionally, prolonged impaction of a stone in the cystic duct can lead to a distended gallbladder that is filled with colorless, mucoid fluid. This condition, known as a mucocele with white bile (hydrops), is due to the absence of bile entry into the gallbladder and absorption of all the bilirubin within the gallbladder.

CLINICAL MANIFESTATIONS

History — Patients with acute cholecystitis typically complain of abdominal pain, most commonly in the right upper quadrant or epigastrium. Characteristically, acute cholecystitis pain is steady and severe and is typically prolonged (greater than four to six hours). The pain may radiate to the right shoulder or back. Associated complaints may include fever, nausea, vomiting, and anorexia. There is often a history of fatty food ingestion one hour or more before the initial onset of pain.

Physical examination — Patients with acute cholecystitis are usually ill-appearing, febrile, tachycardic, and lie still on the examining table because cholecystitis is associated with true local parietal peritoneal inflammation that is aggravated by movement. Abdominal examination usually demonstrates voluntary and involuntary guarding. Patients frequently will have a

positive Murphy's sign. To check for a Murphy's sign, the patient is asked to inspire deeply while the examiner palpates the area of the gallbladder fossa just beneath the liver edge. Deep inspiration causes the gallbladder to descend toward and press against the examining fingers, which in patients with acute cholecystitis commonly leads to increased discomfort and the patient catching his or her breath. Murphy's sign has a high sensitivity for acute cholecystitis but is not specific. In one study, using cholescintigraphy as the gold standard, the sensitivity and specificity of a positive Murphy's sign were 97 and 48 percent, respectively [16]. However, the sensitivity may be diminished in older adults [17].

Patients with complications may have signs of sepsis (gangrene), generalized peritonitis (perforation), abdominal crepitus (emphysematous cholecystitis), or bowel obstruction (gallstone ileus). (See 'Complications' below and "Sepsis syndromes in adults: Epidemiology, definitions, clinical presentation, diagnosis, and prognosis", section on 'Sepsis' and "Gallstone ileus".)

Laboratory findings — Patients typically have a leukocytosis with an increased number of band forms (ie, a left shift). Elevation in the serum total bilirubin and alkaline phosphatase concentrations are **not** common in uncomplicated acute cholecystitis since obstruction is limited to the gallbladder; if present, they should raise concerns about biliary obstruction and conditions such as cholangitis, choledocholithiasis, or Mirizzi syndrome (a gallstone impacted in the distal cystic duct causing extrinsic compression of the common bile duct) (image 1). However, mild elevations in serum aminotransferases and amylase, along with hyperbilirubinemia and jaundice, have been reported even in the absence of these complications and may be due to the passage of sludge, or pus [18]. (See "Acute cholangitis: Clinical manifestations, diagnosis, and management" and "Choledocholithiasis: Clinical manifestations, diagnosis, and management" and "Mirizzi syndrome".)

In patients with emphysematous cholecystitis, mild to moderate unconjugated hyperbilirubinemia may be present because of hemolysis induced by clostridial infection. (See 'Emphysematous cholecystitis' below.)

DIAGNOSTIC APPROACH

Clinical suspicion and diagnosis — Acute cholecystitis should be suspected in a patient presenting with right upper quadrant or epigastric pain, fever, and a leukocytosis. A positive Murphy's sign on physical examination supports the diagnosis [19]. However, history, physical examination, and laboratory test findings are not sufficient to establish the diagnosis.

The diagnosis of acute cholecystitis requires demonstration of gallbladder wall thickening or edema, a sonographic Murphy's sign, or failure of the gallbladder to fill during cholescintigraphy (algorithm 1). In most cases, the diagnosis can be established with an abdominal ultrasound. We perform cholescintigraphy if the diagnosis remains unclear despite ultrasonography.

Abdominal computed tomography (CT) and magnetic resonance cholangiopancreatography (MRCP) are not usually required but may be performed in patients with suspected complications or to rule out alternate diagnoses. Oral cholecystography has no role in the diagnosis of acute cholecystitis since it cannot show gallbladder wall edema and requires days to complete. (See 'Additional evaluation in selected patients' below.)

Laboratory studies — In addition to a complete blood count, we evaluate levels of serum lipase and amylase, electrolytes, alanine aminotransferase, aspartate aminotransferase, bilirubin, calcium, and albumin to rule out other causes of acute abdominal pain or complications of acute cholecystitis. A pregnancy test should be performed in all women of childbearing age. (See 'Laboratory findings' above and 'Complications' below.)

Diagnostic imaging

Ultrasonography — The presence of stones in the gallbladder in the clinical setting of right upper quadrant abdominal pain and fever supports the diagnosis of acute cholecystitis but is not diagnostic. Additional sonographic features include:

- Gallbladder wall thickening (greater than 4 to 5 mm), pericholecystic fluid, or edema (double wall sign) (image 2).
- A "sonographic Murphy's sign" is similar to the Murphy's sign elicited during abdominal palpation, except that the positive response is observed during palpation with the ultrasound transducer. This is more accurate than hand palpation because it can confirm that it is indeed the gallbladder that is being pressed by the imaging transducer when the patient catches his or her breath.

Several studies have evaluated the accuracy of ultrasonography in the diagnosis of acute cholecystitis [19-25]. A particularly informative systematic review summarized the results of 30 studies of ultrasonography for gallstones and acute cholecystitis [22]. Adjusted sensitivity and specificity for diagnosis of acute cholecystitis were 88 percent (95% confidence interval [CI] 0.74 to 1.00) and 80 percent (95% CI 0.62 to 0.98), respectively.

The sensitivity and specificity of ultrasonography for detection of gallstones are approximately 84 (95% CI 0.76 to 0.92) and 99 percent (95% CI 0.97 to 1.00), respectively [22]. Ultrasonography may not detect small stones or sludge as illustrated by a study that compared ultrasonography with direct percutaneous mini-endoscopy in patients who had undergone topical gallstone dissolution [26]. Ultrasonography was negative in 12 of 13 patients in whom endoscopy demonstrated 1 to 3 mm stones or fragments (picture 1) [26].

In patients with emphysematous cholecystitis, the ultrasound report may erroneously note the presence of "overlying bowel gas making adequate visualization of the gallbladder difficult," when in reality, this reflects air in the wall of the gallbladder. (See 'Emphysematous cholecystitis' below.)

Contrast enhanced ultrasonography performed in conjunction with intravenous injection of an ultrasound contrast agent containing microbubbles is advocated by some to allow the preoperative detection of gangrenous cholecystitis [27]. Theoretically, early diagnosis of this serious complication of acute cholecystitis would lead to better planning of early intervention to prevent further more serious complications; however, this test is not widely available [28].

Cholescintigraphy (hepatic iminodiacetic acid [HIDA] scan) — Cholescintigraphy using 99mTc-hepatic iminodiacetic acid (generically referred to as a HIDA scan) is indicated if the diagnosis remains uncertain following ultrasonography. Technetium-labeled HIDA is injected intravenously and is then taken up selectively by hepatocytes and excreted into bile. If the cystic duct is patent, the tracer will enter the gallbladder, leading to its visualization without the need for concentration. The HIDA scan is also useful for demonstrating patency of the common bile duct and ampulla. Normally, visualization of contrast within the common bile duct, gallbladder, and small bowel occurs within 30 to 60 minutes (image 3). When the gallbladder is not visualized within 60 minutes, delayed images (at three to four hours) or morphine augmentation is obtained. Nonvisualization of the gallbladder 30 minutes post-morphine or on delayed images is diagnostic of acute cholecystitis. This occurs because of cystic duct obstruction, usually from edema associated with acute cholecystitis or an obstructing stone (image 4).

Cholescintigraphy has a sensitivity and specificity for acute cholecystitis of approximately 90 to 97 percent and 71 to 90 percent, respectively [22,25,29,30]. Morphine increases sphincter of Oddi pressure, thereby causing a more favorable pressure gradient for the radioactive tracer to enter the cystic duct. This modification is thought to be particularly useful in critically ill patients, in whom standard HIDA scanning may be associated with false positive results [31-34].

False positive results can also occur in the setting of cystic duct obstruction due to a stone or tumor in the absence of acute cholecystitis. Other conditions that can cause false positive results despite a non-obstructed cystic duct include:

- Severe liver disease, which may lead to abnormal uptake and excretion of the tracer.
- Fasting patients (eg, receiving total parenteral nutrition) have no stimulation to contract the gallbladder. In such patients, the gallbladder is already full of viscous bile that prevents the entry of radiotracer.
- Biliary sphincterotomy, which may result in low resistance to bile flow, leading to preferential excretion of the tracer into the duodenum without filling of the gallbladder.
- Hyperbilirubinemia, which may be associated with impaired hepatic clearance of iminodiacetic acid compounds. Newer agents commonly used in cholescintigraphy (diisopropyl and m-bromotrimethyl iminodiacetic acid) have generally overcome this limitation [35].

False negative results are uncommon since most patients with acute cholecystitis have obstruction of the cystic duct. When they occur, they may be due to incomplete cystic duct obstruction.

Additional evaluation in selected patients

Magnetic resonance cholangiopancreatography or endoscopic ultrasound — Choledocholithiasis should be suspected in patients with acute cholecystitis and elevations of liver transaminases, total bilirubin, or evidence of common bile duct dilatation on ultrasound. Patients suspected of having choledocholithiasis are diagnosed with a combination of laboratory tests and imaging studies. The first imaging study obtained is typically a transabdominal ultrasound. Additional testing may include magnetic resonance cholangiopancreatography (MRCP) and endoscopic ultrasound (EUS). Endoscopic retrograde cholangiopancreatography (ERCP) is the gold standard but is reserved for therapeutic procedures given the risk of post-ERCP pancreatitis. (image 5). (See "Choledocholithiasis: Clinical manifestations, diagnosis, and management", section on 'Initial diagnostic evaluation'.)

The role of MRCP in the diagnosis of acute cholecystitis was evaluated in a series that included 35 patients with symptoms of acute cholecystitis who underwent both ultrasound and MRCP prior to cholecystectomy [5]. MRCP was superior to ultrasound for detecting stones in the cystic duct (sensitivity 100 versus 14 percent) but was less sensitive than ultrasound for detecting gallbladder wall thickening (sensitivity 69 versus 96 percent).

Computed tomography — We perform an abdominal CT to rule out complications of acute cholecystitis in patients with sepsis (gangrene), generalized peritonitis (perforation), abdominal crepitus (emphysematous cholecystitis), or bowel obstruction (gallstone ileus). (See 'Complications' below.)

Abdominal CT is not routinely required to diagnose acute cholecystitis but is often performed in the initial evaluation of patients with abdominal pain to rule out other etiologies. CT findings of acute cholecystitis include gallbladder wall edema (image 6), pericholecystic stranding and fluid, and high-attenuation bile [36,37]. The sensitivity of abdominal CT for acute cholecystitis is 94 percent but its specificity is low (59 percent). CT may also fail to detect gallstones as many stones are isodense with bile (image 7) [38,39]. (See "Overview of gallstone disease in adults", section on 'Complications'.)

DIFFERENTIAL DIAGNOSIS

- **Biliary colic** – As with acute cholecystitis, biliary colic causes pain in the right upper quadrant. However, the pain of biliary colic typically lasts at least 30 minutes, plateauing within an hour. The pain then starts to subside, with an entire attack usually lasting less than six hours. An episode of right upper quadrant pain lasting for more than six hours should raise suspicion for acute cholecystitis. Unlike acute cholecystitis, constitutional symptoms such as malaise or fever and peritoneal signs are absent in patients with biliary colic and laboratory studies are normal. (See "Overview of gallstone disease in adults", section on 'Biliary colic'.)

- **Acute cholangitis** – Acute cholangitis may present a right upper quadrant pain, fever, and leukocytosis. In contrast to patients with acute cholecystitis, patients have evidence of cholestasis on laboratory testing and biliary dilation on imaging. It is important to note that patients can have both acute cholangitis and acute cholecystitis. (See "Acute cholangitis: Clinical manifestations, diagnosis, and management", section on 'Diagnosis'.)

- **Fitz-Hugh Curtis syndrome** – In women with acute pelvic inflammatory disease, perihepatitis can cause right upper quadrant abdominal pain. Pericholecystic fluid on imaging studies may be confused with acute cholecystitis, but a hepatic iminodiacetic acid scan is negative. (See "Pelvic inflammatory disease: Clinical manifestations and diagnosis", section on 'Perihepatitis'.)

Other conditions that can give rise to pain in the epigastrium or right upper quadrant abdomen (eg, acute pancreatitis, peptic ulcer disease) can usually be differentiated from acute

cholecystitis by the clinical setting in which they occur and by obtaining the appropriate diagnostic studies. These are discussed in detail, separately. (See "Causes of abdominal pain in adults", section on 'Right upper quadrant pain'.)

COMPLICATIONS

Left untreated, symptoms of cholecystitis may abate within 7 to 10 days. However, as complications are frequent and have the potential of being severe, patients with suspected acute cholecystitis require definitive treatment. (See "Treatment of acute calculous cholecystitis".)

Gangrenous cholecystitis — Gangrenous cholecystitis is the most common complication of cholecystitis (up to 20 percent of cases), particularly in older patients, patients with diabetes, or those who delay seeking therapy (image 8) [40]. The presence of a sepsis-like picture in addition to the other symptoms of cholecystitis suggests the diagnosis, but gangrene may not be suspected preoperatively. (See "Sepsis syndromes in adults: Epidemiology, definitions, clinical presentation, diagnosis, and prognosis", section on 'Sepsis'.)

Perforation — Perforation of the gallbladder results in approximately 10 percent of cases and usually occurs in patients with a delay in diagnosis or failure to respond to initial therapy. The perforation is often localized and occurs at the fundus of the gallbladder after the development of gangrene [41]. The resulting pericholecystic abscess may be palpable and can be visualized on abdominal computed tomography (image 9). Less commonly, there is free perforation into the peritoneum, leading to generalized peritonitis and associated with a high mortality (image 10).

Emphysematous cholecystitis — Emphysematous cholecystitis is caused by secondary infection of the gallbladder wall with gas-forming organisms (such as Clostridium welchii) (image 11) [42,43]. Like other patients with acute cholecystitis, patients with emphysematous cholecystitis usually present with right upper quadrant pain, nausea, vomiting, and low-grade fever. Peritoneal signs are usually absent, but crepitus in the abdominal wall adjacent to the gall bladder may rarely be detected. When such crepitus is present, it is an important clue to the diagnosis.

Affected patients are often men in their fifth to seventh decade, and approximately one-third to one-half have diabetes [43-45]. Mild to moderate unconjugated hyperbilirubinemia may be present (caused by hemolysis induced by clostridial infection). The ultrasound report may

erroneously note the presence of "overlying bowel gas making adequate visualization of the gallbladder difficult", when in reality, this reflects air in the wall of the gallbladder.

Emphysematous cholecystitis often heralds the development of gangrene, perforation, and other complications [43-45]. Other organisms that may be isolated include Escherichia coli (15 percent), staphylococci, streptococci, Pseudomonas, and Klebsiella [43].

Cholecystoenteric fistula — A cholecystoenteric fistula may result from perforation of the gallbladder directly into the intestinal lumen. Fistula formation is more often due to long-standing pressure necrosis from stones than due to acute cholecystitis. It complicates 2 to 3 percent of all cases of gallbladder stone disease but it can rarely occur as a result of acute calculous cholecystitis. While the majority of fistulas are cholecystoduodenal fistulas, approximately 15 percent are cholecystocolonic, most commonly to the hepatic flexure [46]. Symptoms of a cholecystocolonic fistula include bile acid diarrhea and rarely intestinal obstruction due to gallstone ileus.

Gallstone ileus — Passage of a gallstone, usually larger than 2.5 cm, through a cholecystoenteric fistula may lead to the development of mechanical bowel obstruction, usually in the narrowest part of the terminal ileum which is approximately two feet proximal to the ileocecal valve (image 12) [47]. (See "Gallstone ileus" and "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Clinical presentations' and "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Gallstones or foreign body'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Cholecystitis and other gallbladder disorders".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading

level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Gallstones (The Basics)" and "Patient education: Choosing surgery to treat gallstones (The Basics)")
- Beyond the Basics topics (see "Patient education: Gallstones (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Epidemiology and pathogenesis** – Acute cholecystitis predominantly occurs as a complication of gallstone disease and typically develops in patients with a history of symptomatic gallstones. Acute calculous cholecystitis occurs in the setting of cystic duct obstruction. However, the development of acute cholecystitis is not fully explained by cystic duct obstruction alone and an additional irritant may be required to develop gallbladder inflammation. In some cases, infection of bile within the biliary system may also have a role in the development of acute cholecystitis. (See 'Epidemiology' above and 'Pathogenesis' above.)
- **Clinical presentation** – Patients with acute cholecystitis typically complain of abdominal pain, most commonly in the right upper quadrant or epigastrium. The pain may radiate to the right shoulder or back. Characteristically, acute cholecystitis pain is prolonged (more than four to six hours), steady, and severe. Associated complaints may include nausea, vomiting, and anorexia. (See 'Clinical manifestations' above.)
- **Diagnosis** – Acute cholecystitis should be suspected in a patient presenting with right upper quadrant or epigastric pain, fever, and a leukocytosis. A positive Murphy's sign on physical examination supports the diagnosis. However, history, physical examination, and laboratory test findings are not sufficient to establish the diagnosis. The diagnosis of acute cholecystitis requires demonstration of gallbladder wall thickening or edema, a sonographic Murphy's sign, or failure of the gallbladder to fill during cholescintigraphy (algorithm 1). In most cases, the diagnosis can be established with an abdominal ultrasound. We perform cholescintigraphy if the diagnosis remains unclear despite ultrasound. (See 'Diagnostic approach' above.)

- **Additional evaluation in selected patients** – We perform additional imaging (eg, endoscopic ultrasonography, magnetic resonance cholangiopancreatography) to evaluate for choledocholithiasis in patients with elevations of liver transaminases, total bilirubin, alkaline phosphatase, or common bile duct dilatation on ultrasound. We perform abdominal computed tomography to rule out specific complications of acute cholecystitis in patients with sepsis (gangrene), generalized peritonitis (perforation), abdominal crepitus (emphysematous cholecystitis), or bowel obstruction (gallstone ileus). (See 'Additional evaluation in selected patients' above.)

- **Complications** – Left untreated, symptoms of cholecystitis may abate within 7 to 10 days. However, as complications are frequent and have the potential of being severe, patients with suspected acute cholecystitis require definitive treatment. Gangrenous cholecystitis is the most frequent complication of acute cholecystitis (up to 20 percent of cases). Other complications include perforation, emphysematous cholecystitis, cholecystenteric fistula, and gallstone ileus. (See "Treatment of acute calculous cholecystitis".)

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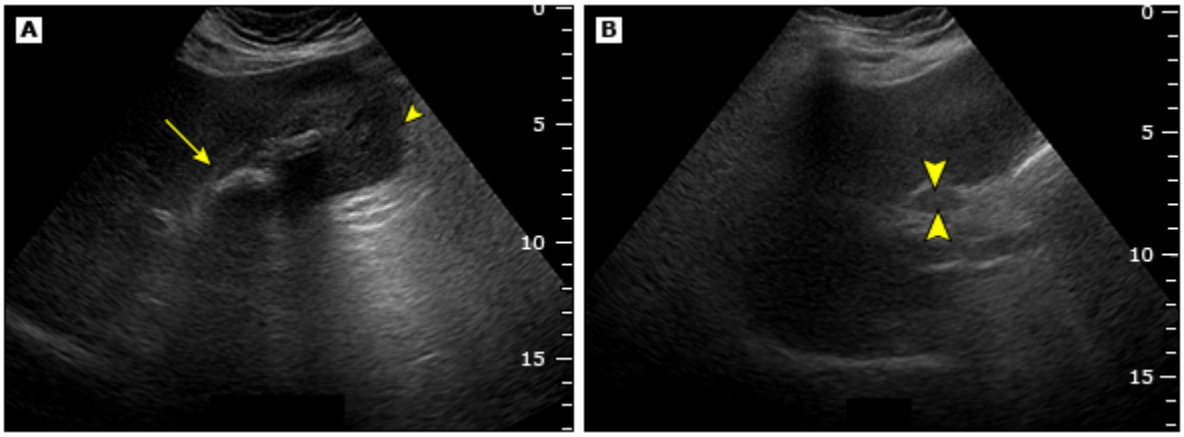
Topic 666 Version 37.0

GRAPHICS

Ultrasound from a patient with acute cholecystitis and Mirizzi syndrome



Transabdominal ultrasound from a patient with acute cholecystitis and Mirizzi syndrome. The patient presented with right upper quadrant pain and jaundice. Acute cholecystitis and Mirizzi syndrome are confirmed by the ultrasound findings of a positive sonographic Murphy's sign (pain with compression of the gallbladder by the ultrasound probe), a large shadowing stone impacted in the infundibulum of the gallbladder (arrow), cholestasis with sludge (small arrowhead), and a dilated common hepatic duct (large arrowheads in B).

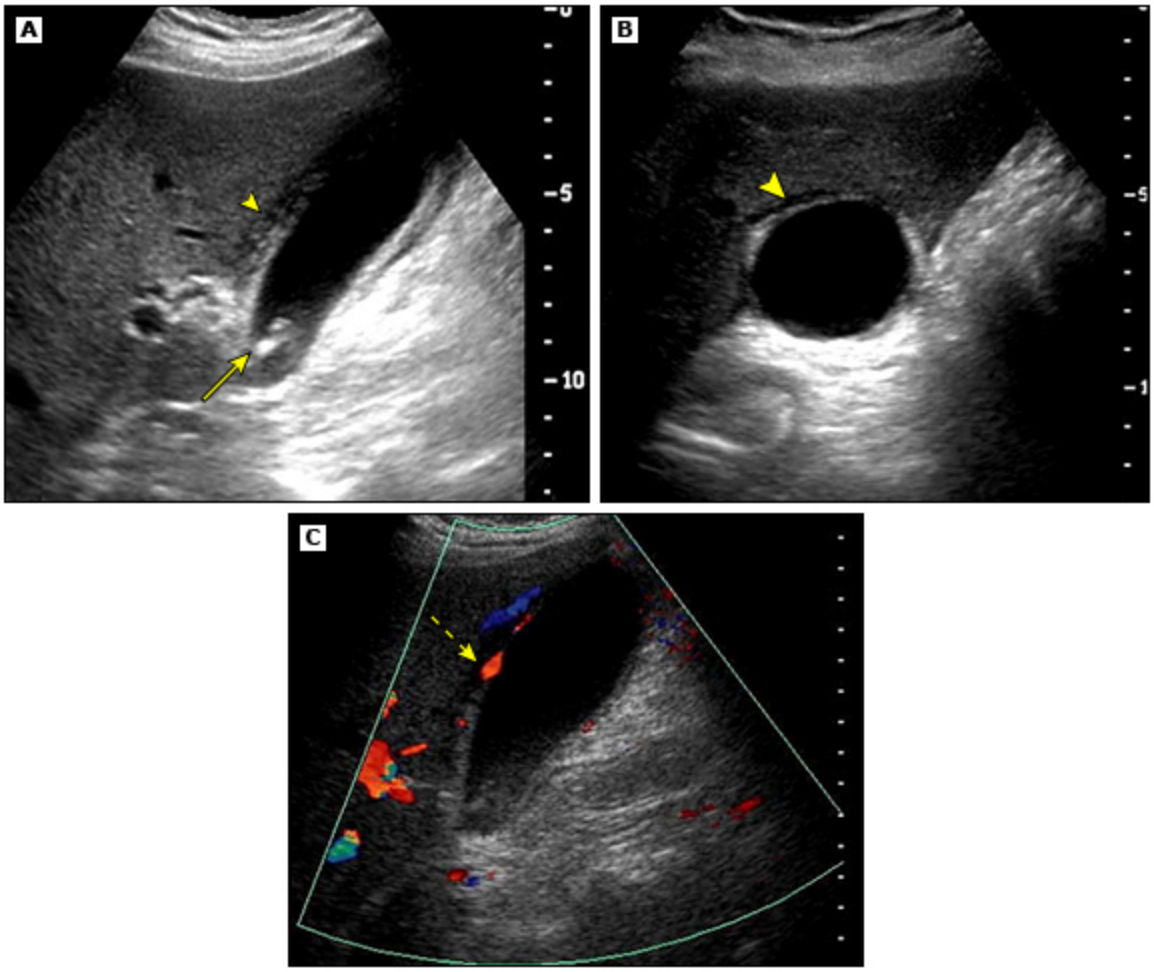
Graphic 86878 Version 2.0

Algorithm for the diagnosis of acute cholecystitis

GB: gallbladder; NSAIDs: nonsteroidal anti-inflammatory drugs; U/S: ultrasound.

Graphic 50032 Version 6.0

Acute cholecystitis with pericholecystic fluid seen on ultrasound



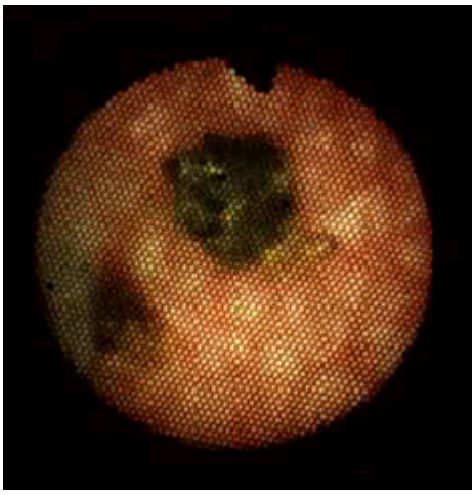
(A) Longitudinal view of the gallbladder showing small shadowing stones in the dependent part of the gallbladder (arrow). The ultrasound also shows a thickened wall in both the longitudinal projection (small arrowhead) and transverse projection (B).

(B) A small amount of pericholecystic fluid is noted (large arrowhead).

(C) The Doppler study shows an increase in blood flow to the wall (dashed arrow) reminiscent of the hyperemia of an inflammatory process. These findings are consistent with acute calculous cholecystitis.

Graphic 83042 Version 3.0

Percutaneous gallbladder endoscopy showing gallstone

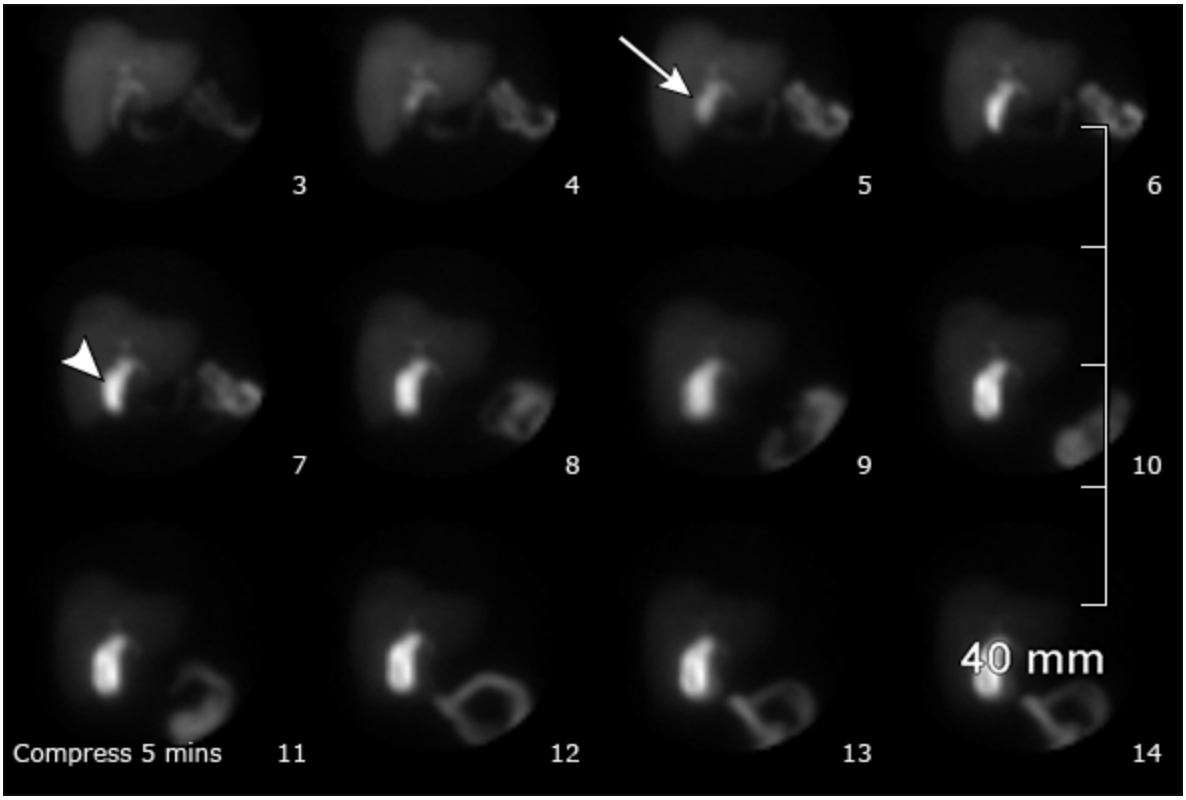


Percutaneous gallbladder endoscopy shows a small gallstone that was not detected on ultrasonography.

Courtesy of Salam Zakko, MD, FACP.

Graphic 65714 Version 2.0

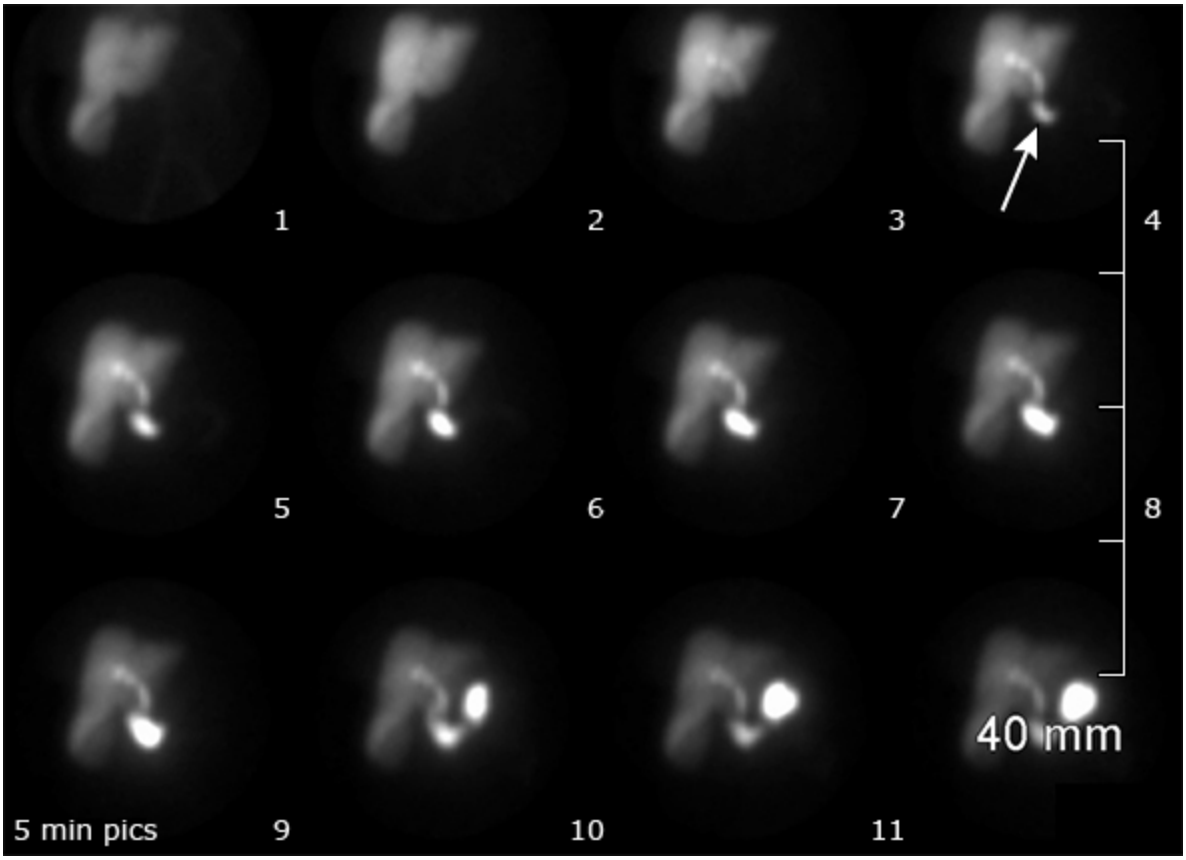
Normal HIDA scan



This is an example of a normal 99mTc-hepatic iminodiacetic acid (HIDA) scan and shows early filling of the gallbladder at 15 minutes (arrow) and complete filling by 25 minutes (arrowhead), indicating a patent cystic duct.

Graphic 59241 Version 4.0

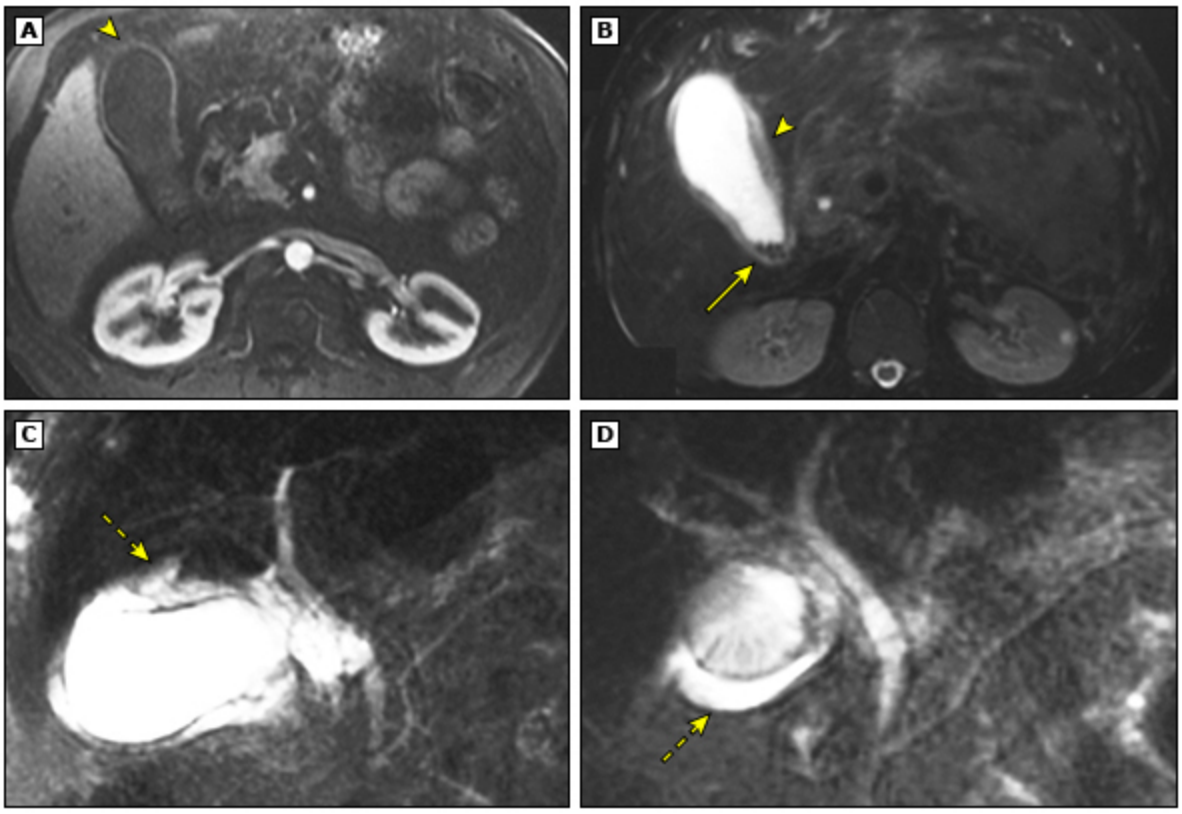
HIDA scan in a patient with acute cholecystitis



The hepatic iminodiacetic acid (HIDA) scan is abnormal and shows absence of filling of the gallbladder, indicating obstruction of the cystic duct. The duodenum starts to fill with radioisotope at about 20 minutes (white arrow). The radioisotope flows directly into the duodenum (white arrow) starting at 20 minutes. The gallbladder never fills during the course of the 60 minute examination. These findings are consistent with the diagnosis of acute cholecystitis.

Graphic 86879 Version 1.0

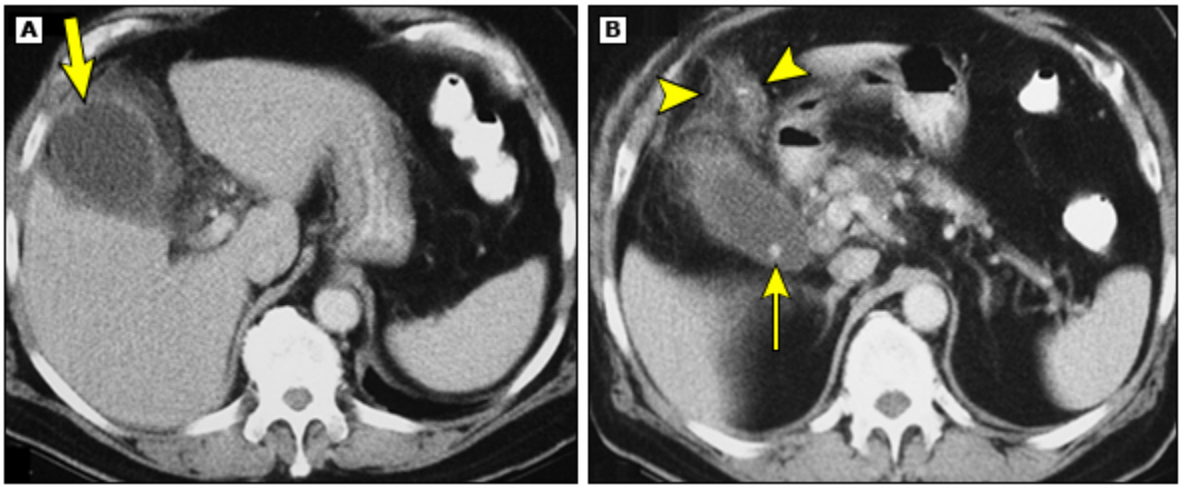
Magnetic resonance imaging (MRI) and magnetic resonance cholangiopancreatography (MRCP) in a patient with acute cholecystitis



MRI and MRCP images from a patient with acute cholecystitis. Contrast enhanced (A) and fat saturated T2-weighted imaging (B) reveal a hyperemic gallbladder wall (arrowheads). Multiple small stones are noted lying dependently in the base of the gallbladder (arrow). The MRCP in the coronal projections (C and D) reveals accumulation of pericholecystic fluid (dashed arrows). These findings are consistent with the diagnosis of acute cholecystitis.

Graphic 86864 Version 3.0

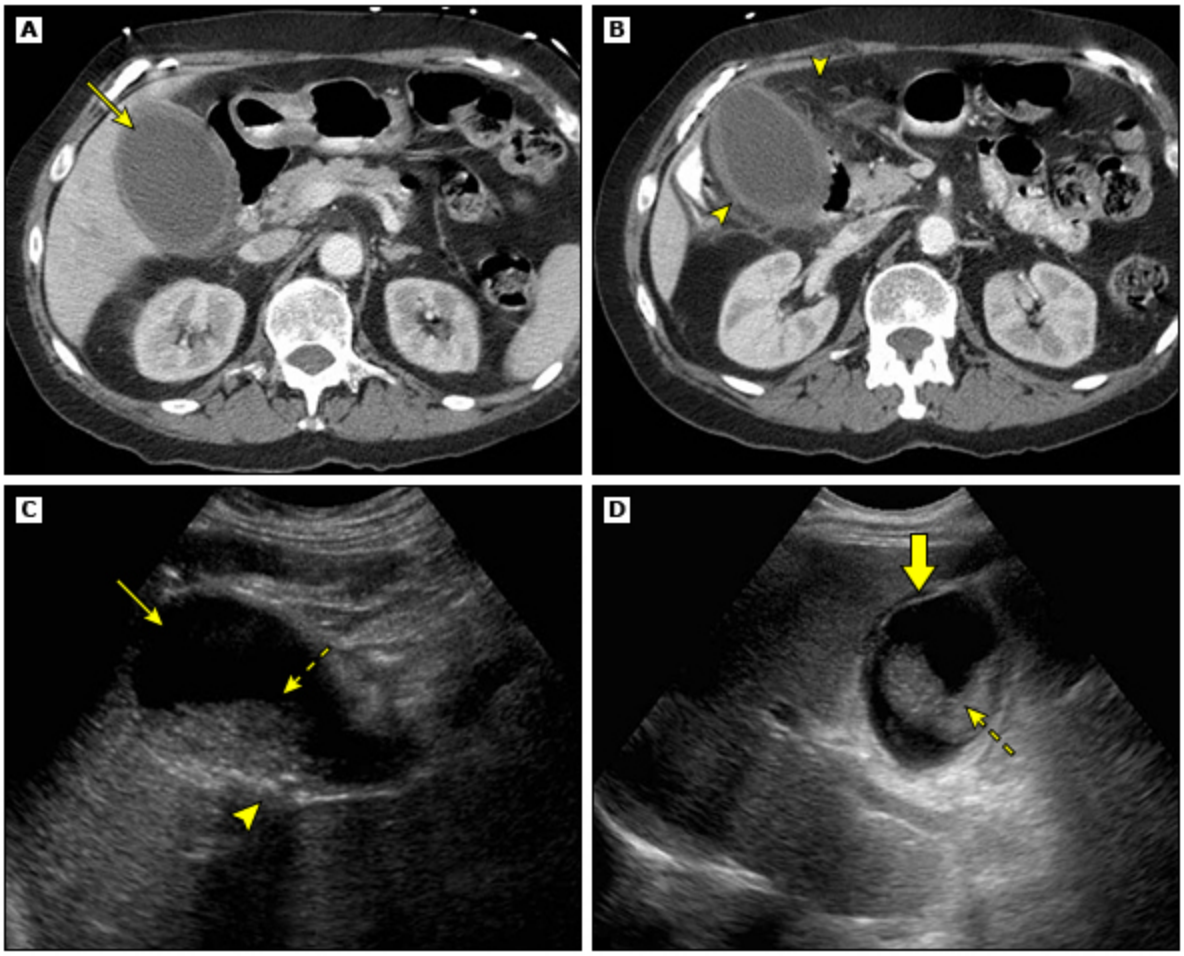
Computed tomographic (CT) scan from a patient with acute cholecystitis



The CT scan shows a distended gallbladder with an edematous and hyperemic wall (thick arrow) and inflammatory induration in the fat surrounding the gallbladder (arrowheads). A calcified stone is visible lying dependently at the base of the gallbladder (thin arrow).

Graphic 77336 Version 3.0

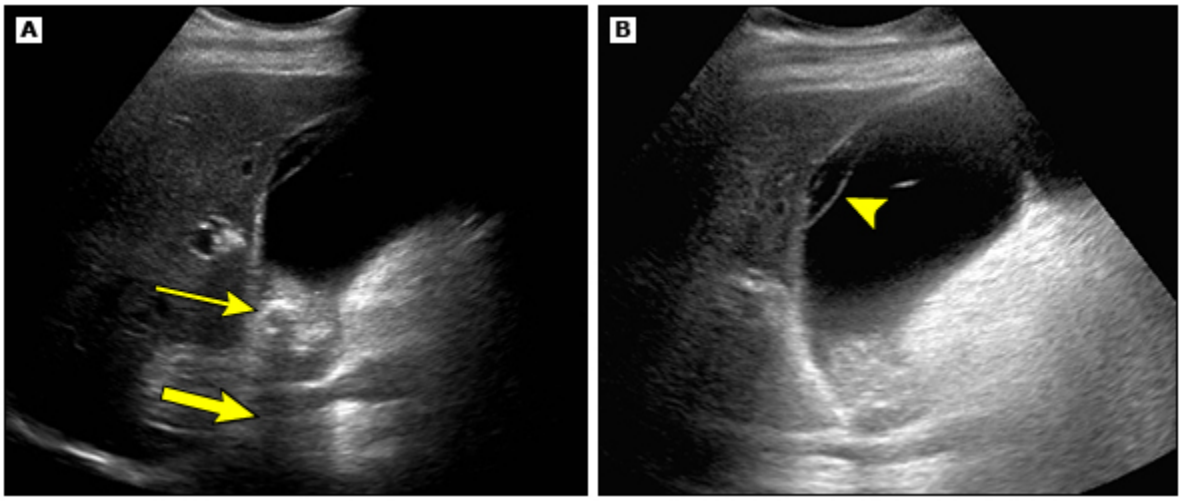
Computed tomographic (CT) scan and ultrasound in a patient with acute cholecystitis



The CT scan (A and B) shows a thick walled and distended gallbladder (arrow) with pericholecystic induration (small arrowheads). However, it does not reveal gallstones. The radiologic differential diagnosis includes acute calculous and acalculous cholecystitis. The ultrasound (C and D) confirms the presence of a distended gallbladder (arrow) with stones (large arrowhead), and the presence of tumefactive sludge (dashed arrow). The thick arrow shows a small amount of fluid in the gallbladder fossa. These findings are consistent with acute calculous cholecystitis.

Graphic 86866 Version 2.0

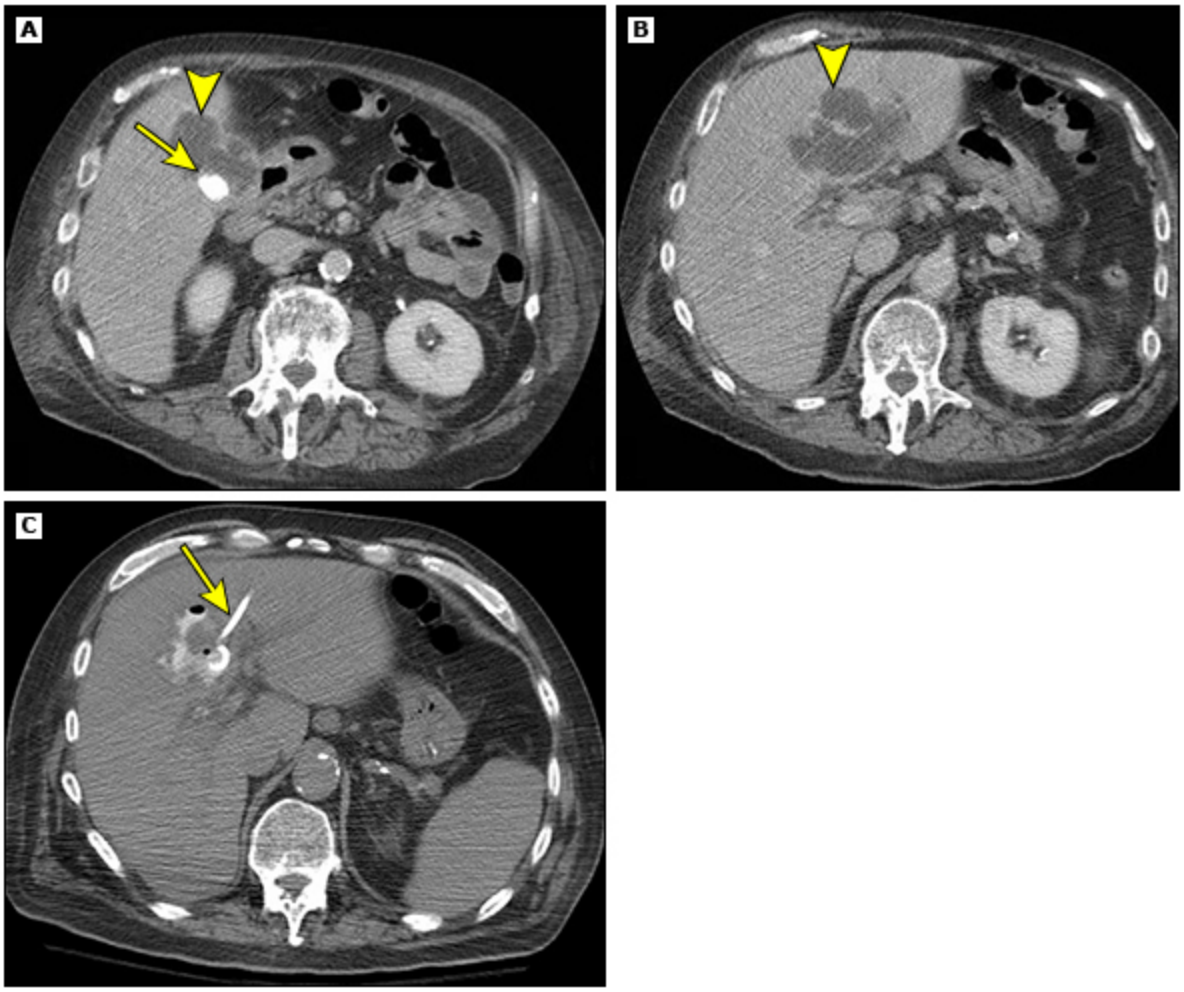
Gallbladder gangrene on transabdominal ultrasound



Transabdominal ultrasound of a gangrenous gallbladder showing evidence of necrotic and denuded mucosa. The longitudinal view of the base of the gallbladder (A) shows a distended gallbladder, with multiple stones within gallbladder sludge (thin yellow arrow) casting an acoustic shadow (thick yellow arrow). The longitudinal view of the fundus (B) demonstrates denuded mucosa (yellow arrowhead), indicating early necrosis of the gallbladder wall.

Graphic 86863 Version 1.0

Liver abscess complicating acute cholecystitis



Computed tomographic (CT) scan in a patient with acute cholecystitis complicated by perforation and liver abscess formation.

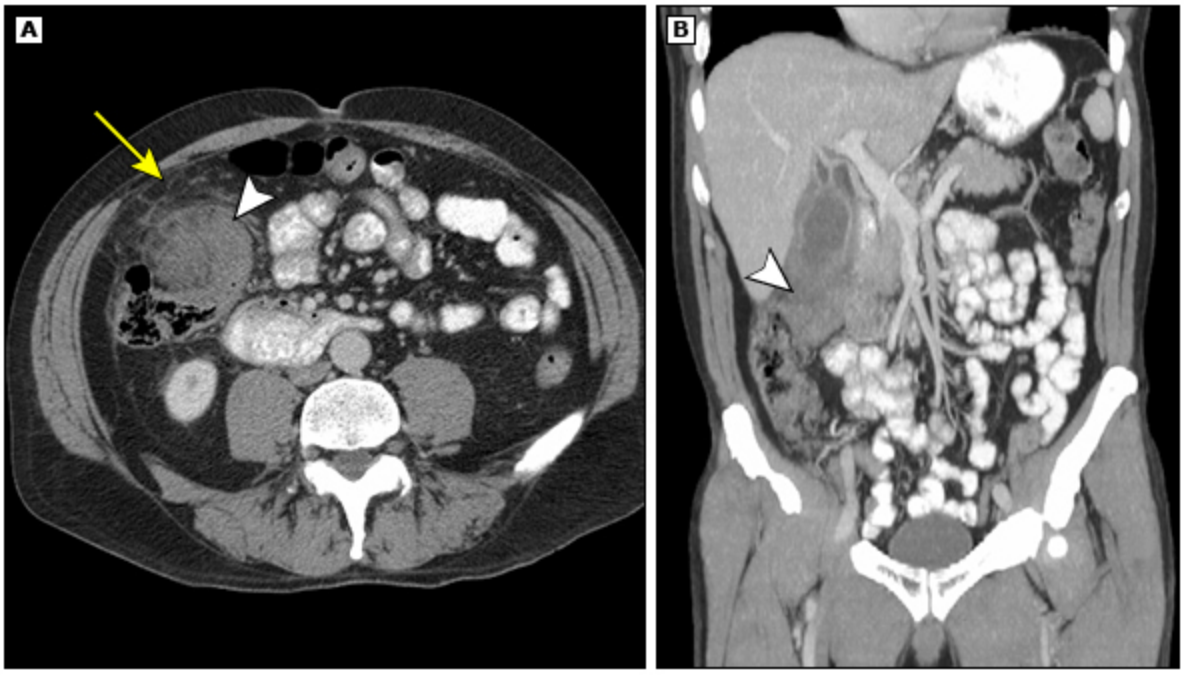
(A) The CT scan shows a gallstone (arrow) surrounded by an irregular accumulation of fluid with a hyperemic rim (arrowhead).

(B) The fluid can be seen extending from the gallbladder fossa into the liver parenchyma (arrowhead).

(C) Percutaneous aspiration of pus confirmed the presence of an abscess, and a drain was placed (arrow). Following drainage of the abscess, a small amount of contrast was injected into the decompressed cavity.

Graphic 86867 Version 2.0

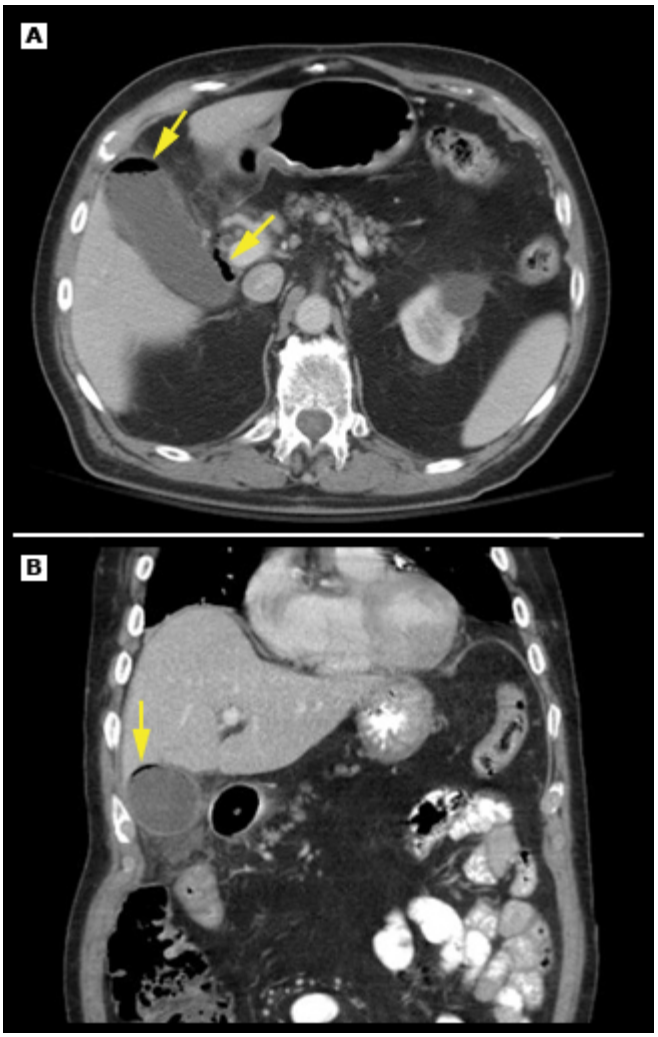
Contained gallbladder perforation in a patient with acute cholecystitis



Computed tomographic (CT) scan showing a sealed perforation of the gallbladder. Transverse (A) and reformatted coronal projections (B) of the CT scan demonstrate a focal accumulation of fluid near the fundus of the gallbladder (white arrowhead) with significant induration in the pericholecystic fat (yellow arrow). At surgery a necrotic and perforated gallbladder was identified.

Graphic 86865 Version 1.0

Emphysematous cholecystitis seen on computed tomographic scan



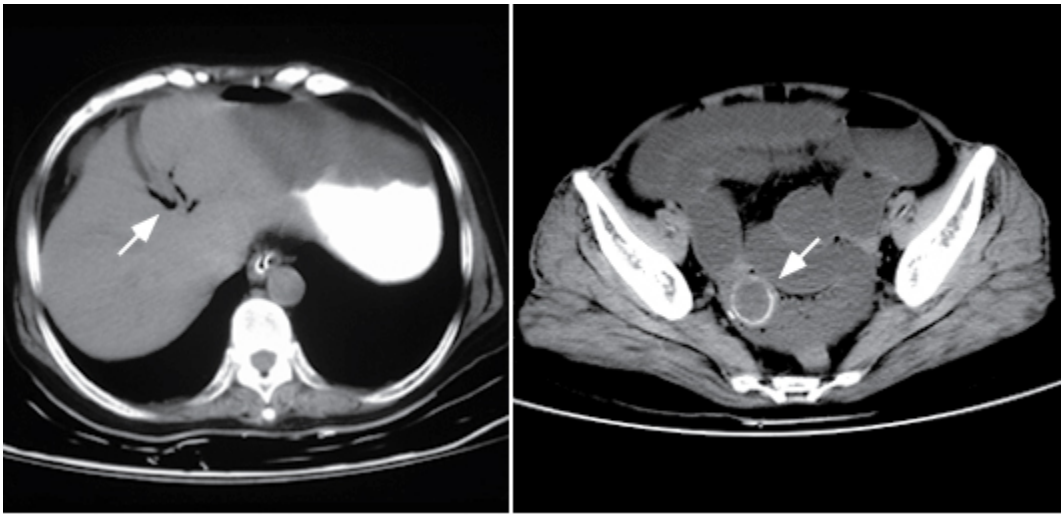
(A) An axial image of a contrast enhanced computed tomographic (CT) scan demonstrating air within the gallbladder lumen (arrows).

(B) A coronal reformat of a contrast enhanced CT demonstrating air within the wall of the gallbladder (arrow).

Courtesy of J Pierre Sasson, MD.

Graphic 56763 Version 3.0

Gallstone ileus seen on computed tomography (CT) scan



CT scan in a 75-year-old woman with small bowel obstruction due to gallstone ileus. Left panel: Free air is seen in the biliary tree and gallbladder (arrow). Right panel: Dilated loops of small bowel with large gallstone with a calcified rim (arrow) impacted in the terminal ileum.

Courtesy of Nezam Afdhal, MD.

Graphic 80522 Version 5.0

Contributor Disclosures

Salam F Zakko, MD, FACP, AGAF No relevant financial relationship(s) with ineligible companies to disclose. **Nezam H Afdhal, MD, FRCPI** No relevant financial relationship(s) with ineligible companies to disclose. **Sanjiv Chopra, MD, MACP** No relevant financial relationship(s) with ineligible companies to disclose. **Shilpa Grover, MD, MPH, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

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